Sep 26, 2022

Robert Salisbury, Senior Planner
Santa Clara County Department of Planning & Development
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CC: planning.commission@pln.sccgov.org

Re: Sargent Ranch Quarry, Environmental Impact Report, SCH # 2016072058

Dear Mr. Salisbury:

On behalf of San Francisco Bay Physicians for Social Responsibility (SF Bay PSR), we are writing to submit comments on the Draft Environmental Impact Report (DEIR) for the proposed Sargent Ranch Quarry Project.

SF Bay PSR is a public health education and advocacy non-profit organization that works to protect human life from the gravest threats to health and survival. We represent a network of hundreds of physicians and health professionals in the greater San Francisco Bay Area, including in Santa Clara County. We promote public policies that protect human health from the threats of nuclear war and other weapons of mass destruction, global environmental degradation, climate change, the epidemic of gun violence, and other social injustices in our society today.

We are writing to strongly urge the County to reject the conditional use permit for Sargent Ranch Quarry. As detailed below, our organization has numerous concerns regarding the proposed Sargent Ranch Quarry Project and its threat to cultural resources, air quality, open space, and water resources.

The proposed Project's irreversible impacts to the heart of the ancestral lands of the Amah Mutsun Tribal Band are unacceptable. We urge the Department to prioritize the sovereignty of the Amah Mutsun Tribal Band and the integrity of their cultural resources in your consideration of this project.

As health professionals, **we are deeply concerned by the Project's potential to negatively impact health.** Exposure to the Project's estimated particulate matter and NOx pollution will contribute to a wide range of potential adverse health harms, including increased risk of mortality from cardiovascular disease, asthma attacks, and preterm birth. Children, older adults, and individuals with heart or lung disease are especially vulnerable to these impacts.[1]

Overall, the adverse public health and cultural impacts of this Project lead us to **strongly urge the County to reject the conditional use permit for Sargent Ranch Quarry.** As all alternative projects identified in the DEIR inadequately address the negative impacts of the Project we support the "No Project Alternative".

Detailed Comments:

The Sargent Ranch Quarry project would have irreversible and adverse impacts on the health and well-being of Santa Clara County residents, community members living within the broader San Francisco Bay Area Air Basin, and most profoundly, members of the Amah Mutsun Tribal Band.

We provide detailed comments expounding upon our reasons for opposing this project in reference to the following sections of the DEIR:

I. **Cultural and Tribal Cultural Resources**
II. **Air Quality and Health**
III. **Aesthetics**

I. Cultural and Tribal Cultural Resources

The desecration of sacred sites has profound and adverse health impacts.

In Impact 3.5-5, the DEIR states that the Project would "cause a substantial adverse change in the significance of the Juristac Tribal Cultural Landscape." These adverse changes are not remediable: "Reclamation activities would not restore the JTCL to a condition that reflects its cultural significance." (3.5-41, p. 375). This impact statement effectively outlines the truly devastating potential impacts of this Project. However, the DEIR's assessment of cultural resources fails to account for the health impacts associated with industrial resource extraction on sacred and culturally important lands. It is imperative for the Department to also consider and explicitly document the mental, physical, and spiritual health effects of the destruction of what is considered the "heart of the ancestral lands" of the Amah Mustun Tribal Band.[2]

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[1] US EPA. (2016, April 26). *Health and Environmental Effects of Particulate Matter (PM)* [Overviews and Factsheets]. https://www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm
[2] About Protect Juristac: Sacred Grounds of the Amah Mutsun. *Protect Juristac*. https://www.protectjuristac.org/about/

Colonization has profoundly and adversely impacted the health of Indigenous communities in myriad ways.[3] Research has documented the deep connection between Indigenous health, well being, and sacred sites.[4] Subica et. al. provide further explanation of how cultural trauma perpetuates health disparities, for further reference of the interconnectedness of culture, structural violence, and health outcomes.[5]

We urge the Department to seriously consider the extensive, irreversible impacts the Project will have on this sacred site, and the associated direct impacts to the spiritual, physical, and mental health of members of the Amah Mutsun Tribal Band. We also urge the Department to consider the Native American Freedom of Religion Act, the United Nations Declaration on the Rights of Indigenous Peoples in your evaluation of this project's legality and compliance with federal and international agreements.

II. Air Quality and Health

The DEIR details the significant and unavoidable effect this project would have on air quality in the San Francisco Bay Area Air Basin (SFBAAB). Significant and unavoidable is defined by the DEIR as an impact that "would result in an adverse effect that meets or exceeds the applicable significance threshold but, even with the implementation of mitigation measures to lessen the impact, if available, the residual effect would not be reduced to less-than-significant levels." (3.1-3, p. 129)

We are especially concerned with the wide-ranging impacts on air quality that the DEIR estimates, not just for local populations, but for an entire region in which millions of Californians live and breathe. As illustrated on page 188 of the DEIR: "The primary sources of pollutants are on-site operation of off-road (mining) equipment and on-road vehicle traffic. Off-road equipment and most on-road vehicle traffic emissions would affect air quality in the SFBAAB". Table 3.3-6 details the polluting emissions that would be generated by the project. This table illustrates that "NOx, PM10, and PM2.5 emissions would exceed BAAQMD [Bay Area Quality Management District] significance thresholds for emissions within the SFBAAB resulting in a significant impact". (3.3-24, p. 192).

We agree with the baseline assessment of the DEIR which states that the Project will have significant adverse impacts on the health of communities in the SFBAAB by:

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[3] Gracey, M., & King, M. (2009). Indigenous health part 1: Determinants and disease patterns. *The Lancet*, *374*(9683), 65–75. https://doi.org/10.1016/S0140-6736(09)60914-4
[4] Cooper, D., Delormier, T., & Taualii, M. (2019). *"It's Always a Part of You": The Connection Between Sacred Spaces and Indigenous/Aboriginal Health*. 30.
[5] Subica, A. M., & Link, B. G. (2022). Cultural trauma as a fundamental cause of health disparities. *Social Science & Medicine*, *292*, 114574. https://doi.org/10.1016/j.socscimed.2021.114574

- Affecting the implementation of the applicable air quality plans (S-10, p. 18)
- Emitting criteria air pollutants ozone precursors (NOx and ROG), PM2.5, and PM10, for which the region is in nonattainment status (S-10, p. 18)
- Contribute nonattainment pollutants (ozone precursors, PM2.5, and PM10) to cumulative increases in air pollutants (S-12, p. 20).

Below, we offer additional information to expand the characterization of the associated adverse health impacts of this project; we also outline further limitations and questions regarding the feasibility of proposed air quality mitigation measures.

A wide range of health impacts must be considered when evaluating increases in particulate matter pollution.

The DEIR is correct in its recognition that there is strong scientific evidence supporting the association between increased levels of particulate matter and short- and long-term health impacts, including increased mortality and hospitalizations for respiratory and cardiovascular symptoms. (3.3-1, p. 169)

However, the DEIR fails to characterize the following additional adverse health outcomes which have been shown to be associated with exposure to particulate matter:

- **Low birth weight.** Epidemiologic evidence supports an association between prenatal exposure to particulate matter air pollution and a decrease in infant birth weight.[6]
- **Harm to children's health and development after maternal exposure.** Epidemiologic evidence documents an association between maternal exposure to particulate matter and various long-term, adverse impacts on their children's health, including the following conditions:
    - **Autism Spectrum Disorder:** There is limited but growing evidence that prenatal exposure to particulate matter is associated with autism spectrum disorder.[7] [8]

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[6] Uwak, I., Olson, N., Fuentes, A., Moriarty, M., Pulczinski, J., Lam, J., Xu, X., Taylor, B. D., Taiwo, S., Koehler, K., Foster, M., Chiu, W. A., & Johnson, N. M. (2021). Application of the navigation guide systematic review methodology to evaluate prenatal exposure to particulate matter air pollution and infant birth weight. *Environment International*, *148*, 106378. https://doi.org/10.1016/j.envint.2021.106378

[7] Lam, J., Sutton, P., Kalkbrenner, A., Windham, G., Halladay, A., Koustas, E., Lawler, C., Davidson, L., Daniels, N., Newschaffer, C., & Woodruff, T. (2016). A Systematic Review and Meta-Analysis of Multiple Airborne Pollutants and Autism Spectrum Disorder. *PLOS ONE*, *11*(9), e0161851. https://doi.org/10.1371/journal.pone.0161851

[8] Perera, F. (2018). Pollution from Fossil-Fuel Combustion is the Leading Environmental Threat to Global Pediatric Health and Equity: Solutions Exist. *International Journal of Environmental Research and Public Health*, *15*(1), 16. https://doi.org/10.3390/ijerph15010016

- **Brain development:** There is some evidence suggesting that prenatal exposure to PM2.5 disrupts brain development. [9]
- **Incidence and prevalence of type 2 diabetes (T2D).** There is strong evidence supporting the association between exposure to PM2.5 and the risk of diabetes.[10]
- **Cumulative impacts and environmental justice.** The impacts of air pollution are not equally distributed in communities across the San Francisco Bay Area Air Basin. [11] We did not identify anywhere in the DEIR that provided an estimate of how air pollution generated will, or will not increase environmental inequities in the region. To this end, the DEIR appears to be lacking a full environmental justice assessment, and as such we recommend utilizing tools such as the CalEnviroScreen 4.0 to conduct the DEIR air pollution estimation through a lens of environmental justice, in addition to considering the profound environmental justice impacts of implementing this project on land that is sacred to the Amah Mutsun Tribal Band.

Overall, the DEIR falls short in assessing the full range of health harms from air pollution, and the Project's full range of environmental justice impacts.

Mitigation Measure 3.3-2a establishes large loopholes that permit circumventing the mitigation measures.

Mitigation Measure 3.3-2a seeks to modify the impacts of toxic air pollution by requiring all off-road mobile equipment and trucks powered by diesel used during the construction and operation phases of the Project meet USEPA Tier 4 engine standards for NOx (3.3-26; p. 194) . However, the mitigation measure also provides significant opportunity for exceptions to this standard if meeting the requirement is determined to be infeasible. What does the Department determine to be grounds for an excusal of this requirement? As written, the DEIR falls short of actually ensuring that this mitigation measure would even have to occur in practice.

In contrast, the Greenhouse Gas Emission Mitigation Measure 3.8-1b (S-48, pg. 56) outlines that "the project must replace diesel and gasoline-powered vehicles with electric or other low or zero-GHG emissions equipment as feasible". Given the impact this decision would have on air quality issues identified in the DEIR, this mitigation measure should be consistent throughout the

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[9] Guxens, M., Lubczyńska, M. J., Muetzel, R. L., Dalmau-Bueno, A., Jaddoe, V. W. V., Hoek, G., van der Lugt, A., Verhulst, F. C., White, T., Brunekreef, B., Tiemeier, H., & El Marroun, H. (2018). Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children. *Biological Psychiatry*, *84*(4), 295–303. https://doi.org/10.1016/j.biopsych.2018.01.016

[10] Bowe, B., Xie, Y., Li, T., Yan, Y., Xian, H., & Al-Aly, Z. (2018). The 2016 global and national burden of diabetes mellitus attributable to PM 2·5 air pollution. *The Lancet Planetary Health*, *2*(7), e301–e312. https://doi.org/10.1016/S2542-5196(18)30140-2

[11] Southerland, V. A., Anenberg, S. C., Harris, M., Apte, J., Hystad, P., van, D. A., Martin, R. V., Beyers, M., & Roy, A. (2021). Assessing the Distribution of Air Pollution Health Risks within Cities: A Neighborhood-Scale Analysis Leveraging High-Resolution Data Sets in the Bay Area, California. *Environmental Health Perspectives*, *129*(3), 037006. https://doi.org/10.1289/EHP7679

report. Additionally, the proposed 5-year feasibility re-assessment should consider the impacts that electrification of Project vehicles would have on reducing the health risks associated with poor air quality.

The mitigation does not even require the mine operator to comply with the most recent engine standards developed by the California Air Resources Board (CARB). CARB is currently developing amendments to the off-road diesel engine standards, which will eventually be known as the Tier 5 rulemaking. These amendments are anticipated to be released in 2024-2025.[12]

It is, overall, unclear how the Sargent Ranch Quarry Project will be held to the highest standards of pollution mitigation under this measure, including how potential loopholes will be closed.

The DEIR lacks analysis on climate change in considering the impacts of air pollution and water usage.

California is in a climate emergency. Beyond the estimations outlined in the Greenhouse Gas Emissions section, this report lacks in its analysis of how climate change will impact air quality and water resource availability throughout the project lifetime. (S-48, p. 56).

A) Air Quality

The DEIR fails to consider the impacts of climate change in its air pollution estimations. The tentative schedule of construction, mining and reclamation (page 4-21, p. 633) estimates the Project will continue operating until 2053 and fully close in 2058. According to the description of “Approach to the Analysis” provided on page (3.3-16, p. 184), the Project’s air pollution estimations were calculated using the outputs and conditions during the first feasible year of production, 2024.

Average temperatures, as well as the frequency of heat waves, are already reaching historic levels, and these climate events are only expected to increase in frequency and severity in California in the coming decades. Santa Clara County is projected to see temperature increases of between 3.4-5.8° F by the end of this century, depending on emissions scenarios.[13] There is a growing body of research on the synergistic effects of heat and air pollution: a recent review of 39 studies examining the interactive effects of heat and air pollution on health outcomes found

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[12] California Air Resources Board. 2021.. *Tier 5 Rulemaking Workshop: Potential Amendments to the Off-Road Diesel New Engine Regulations*. 31.
https://ww2.arb.ca.gov/sites/default/files/classic/msprog/tier5/off_road_tier_5_rulemaking_overview.pdf

[13] Page 12, Maizlish N, English D, Chan J, Dervin K, English P. Climate Change and Health Profile Report: Santa Clara County. Sacramento, CA: Office of Health Equity, California Department of Public Health; 2017.
https://www.cdph.ca.gov/Programs/OHE/CDPH%20Document%20Library/CHPRs/CHPR085SantaClara_County2-23-17.pdf

sufficient evidence for synergistic all-cause mortality, cardiovascular, and respiratory effects of air pollution and heat (particularly for ozone and particulate matter).[14]

It is not enough to estimate air pollution impacts based on today's climate. The DEIR's assessment of the Project's air pollution impacts is lacking in that it does not consider the reality of climate change, specifically, projected increases in average temperatures and heat waves, and how this will impact pollution dispersion, and subsequent exacerbation of the already adverse health impacts of exposure to air pollution.

B) Water Usage

Dust control is specified as one of two mitigation tools that will be implemented to reduce the negative impacts to air quality that would be produced by the Project. Mitigation Measure 3.3-2b specifies that the Project must "develop and implement a comprehensive dust control plan for Project construction and operation and shall submit the plan to the County Department of Planning and Development" (3.3-26, p. 194). We are concerned with this mitigation measure's reliance on vast quantities of water given the anticipated impacts that climate change will have on water availability in California. The DEIR is unclear as to whether climate change impacts are considered in its estimation of the availability of the quantity of water needed to implement this measure.

The DEIR does not provide estimates of the quantity of water that is expected to be used through this mitigation measure, but it does estimate that dust mitigation will create the second highest demand for water usage, following aggregate processing. The project is estimated to use 86,135 gallons of water per day and 26,742,000 gallons per year during peak production times. (Table 2-5, 2-39, p. 105). For reference, the average California resident uses about 85 gallons of water a day in their home[15]; this operation anticipates using as much as 1,000 individuals' water consumption per day during peak production.

There is ample evidence that climate change will impact water availability across California. The California State Water Resources Board recently issued a report projecting that "hotter and drier weather could diminish our [state] water supply by 10% by 2040.[16] California's Fourth Climate Change Assessment of impacts to the Central Coast region estimated that "more extreme droughts and higher temperatures will also alter the natural recharge of groundwater and

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[14] Anenberg, S. C., Haines, S., Wang, E., Nassikas, N., & Kinney, P. L. (2020). Synergistic health effects of air pollution, temperature, and pollen exposure: A systematic review of epidemiological evidence. *Environmental Health*, *19*(1), 130. https://doi.org/10.1186/s12940-020-00681-z

[15] *Residential Water Use Trends and Implications for Conservation Policy*. (2017). Legislative Analyst's Office. https://lao.ca.gov/Publications/Report/3611

[16] Page 3, California Natural Resources Agency. (2022). *California's Water Supply Strategy, Adapting to a Hotter, Drier Future*. https://resources.ca.gov/-/media/CNRA-Website/Files/Initiatives/Water-Resilience/CA-Water-Supply-Strategy.pdf

potentially exacerbate groundwater overdraft." More local to the Project, the Valley Water's Urban Water Management Plan (UWMP) Report Section 3.10 "Hydrology and Water Quality", which looks at water demand through 2045 for normal and dry years, projects that "Statewide and local changes in precipitation and temperature could impact Valley Water's water supplies and operations, the effectiveness of potential water supply investments, and water demand patterns…". [17]

From our review of this DEIR, it is unclear whether estimations of water availability through 2045 – for dust suppression and other water usage – fully account for how climate change risks may change water supply and demand.

III. Aesthetics

The preservation of natural spaces is linked to public health

Preserving open space from industrialization has numerous direct and indirect benefits to human health. The DEIR states that the Project will pose "significant and unavoidable" impacts after mitigation to the "visual character of the Project site or scenic resources visible from U.S. 101, a County-designated scenic highway". (S-10, p. 18). Overall, the Project will disrupt nearly 300 acres of open space (2-1, p. 69). According to the American Public Health Association, the presence of natural features in our communities promote lower levels of "mortality and illness, higher levels of outdoor physical activity, restoration from stress, a greater sense of well-being, and greater social capital".[18] Preserving the natural elements of this area will have direct benefits to those who live near, drive past, and visit this area.

Conclusion

As health professionals, we are deeply concerned by this project's potential to adversely impact access to sacred, culturally important lands to the Amah Mutsun Tribal Band. The destruction of cultural and spiritual sites threatens the health and well-being of Indigenous communities. We are also concerned about this Project's anticipated health harms associated with diminished air quality across the Bay Area; impacts on water resources; and to the loss of open natural lands. We urge the County to **reject the conditional use permit for Sargent Ranch Quarry and choose the** "No Project Alternative."

Thank you for your consideration.

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[17] Valley Water. (2020). *2020 Urban Water Management Plan*. https://fta.valleywater.org/dl/pggls1SeCr
[18] American Public Health Association. Improving health and wellness through access to nature. 2013 https://www.apha.org/policies-and-advocacy/public-health-policy-statements/policy-database/2014/07/08/09/18/improving-health-and-wellness-through-access-to-nature.

Respectfully,

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Health and Environmental Effects of Particulate Matter (PM)

Health Effects

The size of particles is directly linked to their potential for causing health problems. Small particles less than 10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream.

Exposure to such particles can affect both your lungs and your heart. Numerous scientific studies have linked particle pollution exposure to a variety of problems, including:

- premature death in people with heart or lung disease
- nonfatal heart attacks
- irregular heartbeat
- aggravated asthma <https://epa.gov/asthma>
- decreased lung function
- increased respiratory symptoms, such as irritation of the airways, coughing or difficulty breathing.

People with heart or lung diseases, children, and older adults are the most likely to be affected by particle pollution exposure.

- AirNow <https://airnow.gov/> can help you monitor air quality near you, and protect yourself and your family from elevated PM levels.

Environmental Effects

Visibility impairment

Fine particles ($PM_{2.5}$) are the main cause of reduced visibility (haze) in parts of the United States, including many of our treasured national parks and wilderness areas. Learn more about visibility and haze <https://epa.gov/visibility>

Environmental damage

Particles can be carried over long distances by wind and then settle on ground or water. Depending on their chemical composition, the effects of this settling may include:

- making lakes and streams acidic
- changing the nutrient balance in coastal waters and large river basins
- depleting the nutrients in soil
- damaging sensitive forests and farm crops
- affecting the diversity of ecosystems
- contributing to acid rain effects <https://epa.gov/acidrain/effects-acid-rain>.

Materials damage

PM can stain and damage stone and other materials, including culturally important objects such as statues and monuments. Some of these effects are related to acid rain effects on materials <https://epa.gov/acidrain/effects-acid-rain#materials>.

Further Reading

Particle Pollution and Your Health (PDF)(2 pp, 320 K, About PDF <https://epa.gov/home/pdf-files>): Learn who is at risk from exposure to particle pollution, what health effects you may experience as a result of particle exposure, and simple measures you can take to reduce your risk.

How Smoke From Fires Can Affect Your Health <https://www.airnow.gov/air-quality-and-health/fires-and-your-health/>: It is important to limit your exposure to smoke -- especially if you may be susceptible.

EPA research on airborne particulate matter <https://epa.gov/air-research>: EPA supports research that provides the critical science on PM and other air pollutants to develop and implement Clean Air Act regulations that protect the quality of the air we breathe.

PM Home <https://epa.gov/pm-pollution>

Particulate Matter (PM) Basics <https://epa.gov/pm-pollution/particulate-matter-pm-basics>

Health and Environmental Effects

Setting and Reviewing PM Standards <https://epa.gov/pm-pollution/setting-and-reviewing-standards-control-particulate-matter-pm-pollution>

PM Standards Regulatory Actions <https://epa.gov/pm-pollution/national-ambient-air-quality-standards-naaqs-pm>

Implementing PM Standards <https://epa.gov/pm-pollution/applying-or-implementing-particulate-matter-pm-standards>

PM Implementation Regulatory Actions <https://epa.gov/pm-pollution/particulate-matter-pm-implementation-regulatory-actions>

SIP Checklist Guide <https://epa.gov/pm-pollution/pm-state-implementation-plan-sip-checklist-guide>

PM SIP Training Presentations <https://epa.gov/pm-pollution/pm-naaqs-implementation-training-and-assistance-state-and-local-air-agencies>

PM Data and SIP Status Reports <https://epa.gov/pm-pollution/technical-data-and-reports-particulate-matter-pm-measurements-and-sip-status>

Other Criteria Air Pollutants <https://epa.gov/criteria-air-pollutants>

Contact Us <https://epa.gov/pm-pollution/forms/contact-us-about-particulate-matter-pm-pollution> to ask a question, provide feedback, or report a problem.

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About

Juristac (Huris-tak) lies at the heart of the ancestral lands of the Amah Mutsun Tribal Band near Gilroy, California. For thousands of years, our Mutsun ancestors lived and held sacred ceremonies at this location in the southern foothills of the Santa Cruz Mountains, above the confluence of the Pajaro and San Benito rivers.

The sacred hills and open valleys at the heart of the Juristac Tribal Cultural Landscape are today bounded by the Sargent Ranch. An investor group based in San Diego purchased the land at a bankruptcy auction and is currently seeking to develop a 403-acre open pit sand and gravel mining operation on the property.

The Amah Mutsun Tribal Band vehemently opposes the proposed mining project. We are asking the public to join us in standing for the protection of our sacred grounds.

No Sargent Quarry

Over a 30-year operational period, the proposed Sargent Quarry would impact 403 acres of land. The plan includes a 62-acre processing plant area, three open pit quarry sites up to 250 ft deep[1], a 1.6-mile long conveyor belt, and a 22-foot wide access road[2]. An estimated 40 million tons[3] of sand and gravel aggregate would be produced over the life of the mine, primarily for use in local road building and general construction.

Existing quarry adjacent to the Sargent Ranch property, operated by Graniterock (photo: AMTB)

For property owner Debt Acquisition Company of America (DACA), the quarry project is an opportunity for financial gain. Doing business under the name Sargent Ranch Partners LLC, DACA hired a Palo Alto based firm, Freeman Associates LLC, to shepherd their proposed quarry through Santa Clara County's planning and environmental review process. A draft Environmental Impact Report (EIR) was published on July 22, 2022 by the County and a 60-day public comment period was opened that ends September 26.

Cultural and Spiritual Impacts

> *"The whole area around Juristac is a power place. Long ago, the people all jointly agreed that this was an area that had power. This is where our ancestors held healing ceremonies, this is where our spiritual doctors went, at La Brea, to prepare themselves for the dances."*
> —Ed Ketchum, Amah Mutsun Tribal Band

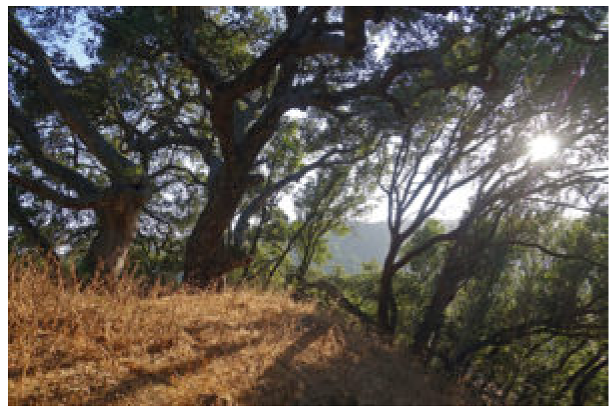
For Mutsun people, Juristac is the home of a powerful spiritual being known as Kuksui. Juristac translates to "place of the Big Head," and Big Head dances associated with Kuksui and other healing and renewal ceremonies took place in the area for centuries, often attended by neighboring tribal groups. The entire area now known as Sargent Ranch and previously named Rancho Juristac contains a complex of storied cultural sites and features of spiritual significance.

Today's Amah Mutsun Tribal Band are survivors of the destructive reign of Mission San Juan Bautista and Mission Santa Cruz. Many of our Mutsun ancestors were taken into the missions from villages at Juristac including Xisca, Pitac and La Brea. After the missions were closed in the 1830's, some Mutsun people returned to their homelands at Juristac, until a smallpox epidemic and pressures from

Bedrock mortars, Sargent Hills area (photo: E. Ketchum)

American settlers led to their relocation to surrounding towns and ranchos.

Our tribe, which now owns no land within our traditional territory, draws a clear connection between today's threats to sacred sites and the legacy of colonial violence our people have endured. "The destruction and domination of Amah Mutsun culture, spirituality, environment and people never ended," Chairman Valentin Lopez states. "It just evolved to the destructive and dominating projects that we see today."



The significance of the Juristac area is only further heightened by its pristine state in relation to the surrounding region. "When you look at our other ceremonial sites and our hunting, fishing and gathering places, the vast majority of these places have been lost to development," Lopez explains. "Juristac is one of the very last remaining undisturbed areas."

Our Amah Mutsun tribe maintains that once disturbed by mining, there will be no way to rehabilitate the cultural and spiritual aspects of the landscape. While the land and any cultural resources within the 403-acre footprint of direct impact is in obvious peril, the broader disruption of the spiritual integrity of the land as a result of mining cannot be quantified.

> *"We honor our ancestors by returning to those places where they had ceremony. For thousands and thousands of years they fulfilled their sacred responsibilities to manage and protect those lands. Through no fault of their own, they were violently interrupted. We cannot let them, or their responsibility be forgotten. We have a duty to continue to fulfill those responsibilities. Without these spiritual sites, we lose our purpose for being here."*
> —Chairman Valentin Lopez, Amah Mutsun Tribal Band

Ecological Impacts

The proposed quarry and processing plant represents a major intrusion into an otherwise relatively pristine area. Juristac's grasslands, oak woodland, riparian corridors, freshwater ponds and streams provide important habitat for an abundance of species.


California Tiger Salamander (photo: C. Steiner)

The project would eliminate approximately around 400 acres[4] of upland habitat for the California red-legged frog and California tiger salamander, both federally-listed threatened species, while also degrading breeding habitat in ponds adjacent to quarry operations. The loss of grasslands would also impact the American badger, a California listed species of special concern present in the Sargent Valley, and birds of prey that forage in the area such as the Golden Eagle, Northern Harrier, Prairie Falcon and Burrowing Owl. In addition, quarrying would destroy approximately 29 acres[5] of California live oak woodland, a valuable roosting and foraging habitat for many native species.

Seeps and springs line both sides of the Sargent Valley and are a vital component of the landscape, providing moisture year-round and recharging off-channel ponds and perennial pools in lower Sargent Creek. The aquifer that feeds these springs is likely to be impacted by quarry excavation pits and by the pumping of an estimated 83,300 gallons per day[6] from an onsite well for aggregate processing and dust control. Pit excavation would also directly eliminate approximately 7000 linear feet[7] of ephemeral stream drainages.


Lower Sargent Valley in late summer

The Sargent Hills have been identified in numerous landscape linkage studies as a critical point of habitat linkage between the Santa Cruz Mountains and the Diablo and Gabilan mountain ranges to the south. Sargent Creek provides valuable north-south passage for wildlife, and Juristac is the gateway to key under-crossings for wildlife passage beneath Highway 101. These wildlife corridors would be disrupted by the quarry and it’s processing plant, roads, and associated infrastructure.

In recognition its unique habitat values, the Santa Clara Valley Open Space Authority identified the Sargent Hills as a Conservation Focus Area in its 2014 Santa Clara Valley Greenprint. Local conservation organizations such as the Peninsula Open Space Trust and The Nature Conservancy also consider the Sargent Hills area as a top priority for protection.

Burrowing Owl (photo: M. Damiani)

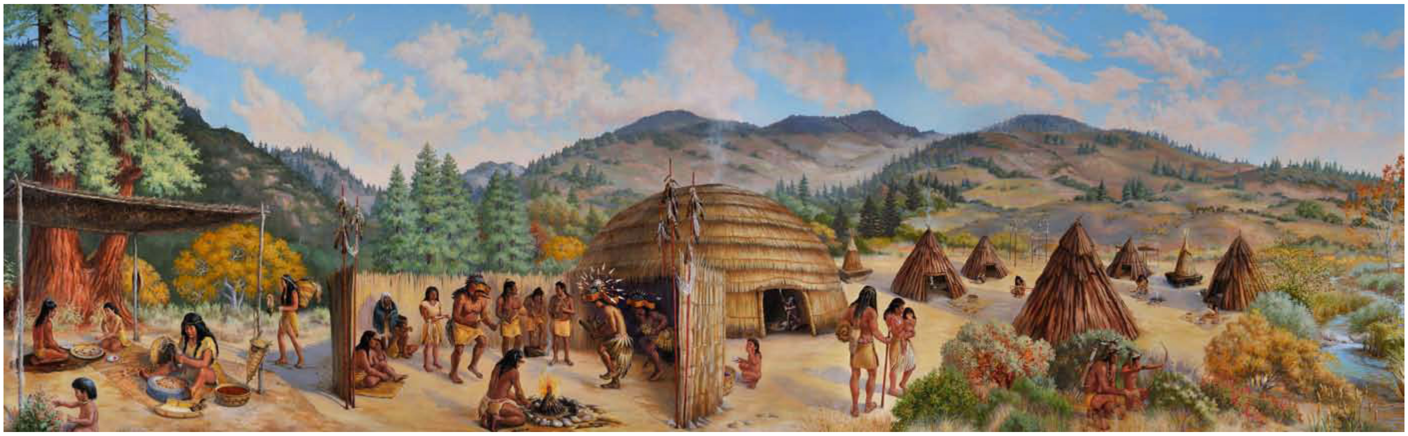
There is only one Juristac

Promoters of the Sargent Quarry point to the growing demand for local sources of aggregate, a necessary material for construction and road building, and herald the relative environmental benefits of quarrying upland locations like Sargent Valley, rather than riparian floodplains. Yet, it is clear that while there are many other potential upland sources of sand and gravel in our region, there is only one Juristac.

For the Amah Mutsun, who have already seen the loss and degradation of nearly all of the lands we once occupied, there is no room for another loss of this magnitude. Our very cultural survival hinges on the preservation of what little remains of our homeland.

We ask that you support our Amah Mutsun Tribal Band's effort to protect and conserve our sacred and cultural site, Juristac. For specific information on how you can help advocate for the preservation of Juristac, please visit our How to Help page.

> *"Our people have been destroyed and dominated for many generations. Juristac represents an opportunity to recognize the humanity of our ancestors and correct the wrongs that have been committed. It is time we fully acknowledge this difficult history and work together to protect the environment and its resources for generations to come."*
> —Valentin Lopez, Chairman, Amah Mutsun Tribal Band



1. Draft Sargent Ranch Quarry Environmental Impact Report, July 2022, Section 2-14 (p. 82)
2. Sargent Quarry Mining and Reclamation Plan —2022 updated, Section 3.6.1 (p. 58)
3. Sargent Quarry Mining and Reclamation Plan, —2022 updated, Section 3.5.2 (p. 45)
4. Draft Sargent Ranch Quarry Environmental Impact Report, July 2022, Section 3.4-62 (p. 266), Section 3.4-28 (p. 232)
5. Draft Sargent Ranch Quarry Environmental Impact Report, July 2022, Section 3.4-11 (p. 215), Table 3.4-1
6. Draft Sargent Ranch Quarry Environmental Impact Report, July 2022, Section 2-39 (p. 107), Table 2-5
7. Draft Sargent Ranch Quarry Environmental Impact Report, July 2022, Section 3.4-11 (p. 215), Table 3.4-1

AKKUYUT MISIMPI (WELCOME)

We invite you to stand with the Amah Mutsun Tribal Band in saying NO to sand and gravel mining at Juristac.

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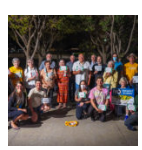

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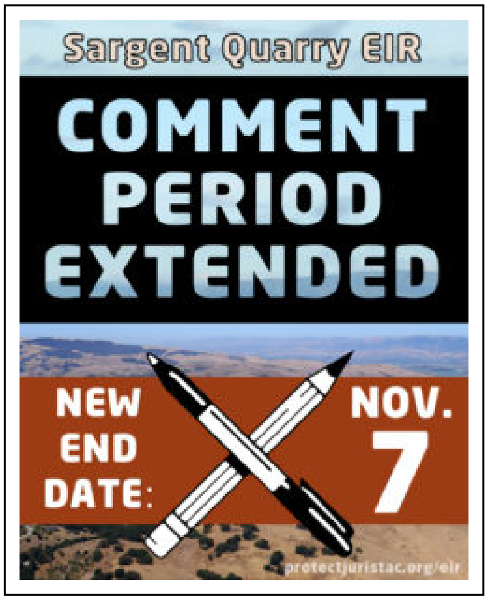

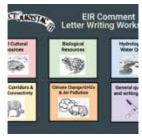

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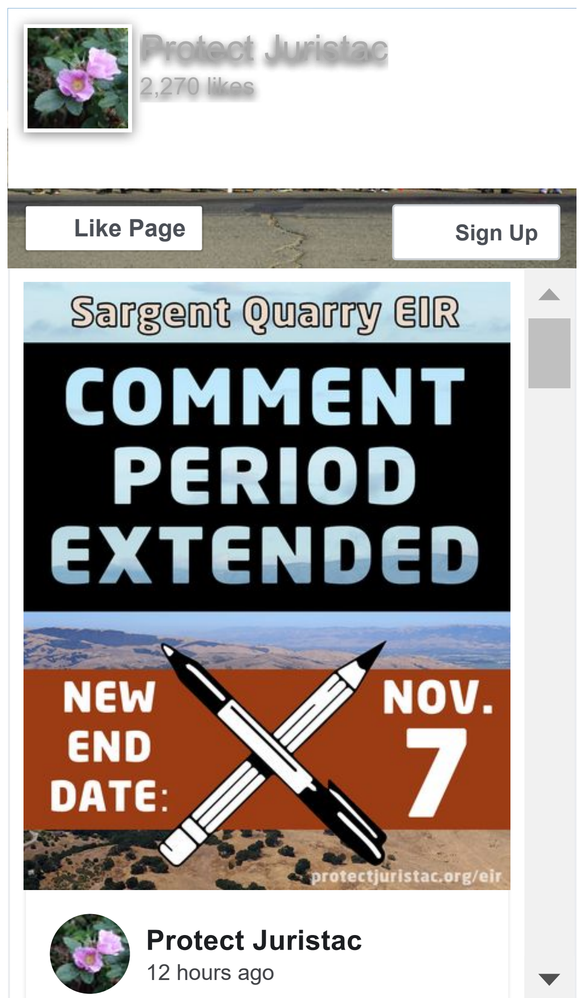

Indigenous health part 1: determinants and disease patterns

Michael Gracey, Malcolm King

The world's almost 400 million Indigenous people have low standards of health. This poor health is associated with poverty, malnutrition, overcrowding, poor hygiene, environmental contamination, and prevalent infections. Inadequate clinical care and health promotion, and poor disease prevention services aggravate this situation. Some Indigenous groups, as they move from traditional to transitional and modern lifestyles, are rapidly acquiring lifestyle diseases, such as obesity, cardiovascular disease, and type 2 diabetes, and physical, social, and mental disorders linked to misuse of alcohol and of other drugs. Correction of these inequities needs increased awareness, political commitment, and recognition rather than governmental denial and neglect of these serious and complex problems. Indigenous people should be encouraged, trained, and enabled to become increasingly involved in overcoming these challenges.

Lancet 2009; 374: 65–75

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See **Perspectives** page 19

See **Review** page 76

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Introduction

There are more than 370 million Indigenous people worldwide and they live in countries on every inhabited continent.[1,2] The definition of Indigenous can be difficult, even contentious;[2,3] panel 1 shows criteria that can be used to this end. Some Indigenous groups are easily identified, such as native Americans, Australia's Aboriginal people, Māori in New Zealand, and the original inhabitants of Pacific Ocean nations who were present long before Europeans.[3] Indigenous peoples are variously called Indigenous, Aboriginal, tribal, or minority groups or peoples.[3] Poor definition of Indigenous identification contributes to the groups' marginalisation and inadequate data for their numbers, health, and socioeconomic circumstances.[3] Most countries do not officially recognise their Indigenous groups, and have inaccurate or no published statistical data for these peoples. Therefore, systematic information about health, morbidity, and mortality is sparse.[2,3] Most reports relate to specific conditions and small groups. In this Review we discuss issues of worldwide importance and draw on Australian Indigenous experience as an example. Despite great diversity of Indigenous peoples, many similarities in their health and illnesses and their determinants exist.

Indigenous people come from thousands of cultures and are over-represented among the poor and disadvantaged. Overall, their health compares unfavourably with their non-Indigenous counterparts.[3] Their susceptibility to disease is exacerbated by poor living conditions and water supplies, often with restricted access to fresh and nutritious food, and inadequate health services. Panel 2 summarises their main health problems.

Effects of colonisation

Common to many Indigenous groups are the powerful effects of colonisation on their people and their lands by outsiders who later dominated societies and alienated them from their own ways of life. This colonisation adversely affected physical, social, emotional, and mental health and wellbeing in traditional societies. Extrapolation between different groups is unwise because local circumstances differ greatly.

We need to understand how colonisation affected the lives of Indigenous peoples to understand their health today. The effect of colonisation was and is profound. Many colonisers were European, including Belgian, British, Dutch, French, German, Italian, Portuguese, Russian, and Spanish; but there were also Asian colonists, including Chinese, Indonesian, Japanese, and Malaysians.[2] The biggest Indigenous populations are in the most populous countries, such as China, India, Indonesia, Asian Russia, and former Soviet Union countries. Some nations deny the existence of their Indigenous populations because of ignorance, embarrassment, or political expediency.

Foreign intruders introduced microorganisms to which traditional groups had not been exposed and were susceptible. The devastating entry of smallpox, measles, and tuberculosis into the long-isolated Indigenous inhabitants of Australia is a good example.[4] Likewise, infections introduced by colonists seriously affected Indigenous populations in the Americas and elsewhere.

Traditional Indigenous people were careful custodians of the environments that provided them and future generations with sustenance, including water, plants, animals that they hunted and fished, and from which they gathered eggs and tidal shoreline foods, such as shellfish and marine plants. Habitats of local foods and plants were protected to ensure that they were not spoiled by human or animal predators or pests and to maintain

Search strategy and selection criteria

We searched a combination of sources, including PubMed, concentrating on original publications and reviews from the preceding 10 years. The search was not confined to the English language. Keywords used included: "Indigenous", "Aboriginal", or "Aborigines", linked with "health", "nutrition", "malnutrition", "growth", "infants", "children", "pregnancy", "maternal health", "adolescents", "infections", "parasites", "hypertension", "cardiovascular disease", "diabetes", "renal disease", "dialysis", "alcohol", "drugs", "trauma", "accidents", "drowning", "poisoning", "homicide", "suicide", and "mortality". Information was obtained from other sources such as websites from international organisations, including UN and WHO. Some information came from earlier reviews and books of particular relevance; these works are in the public domain and are referenced here. We also had access to unpublished official reports about the health of Indigenous people in Australia.

Panel 1: Criteria to help to define Indigenous peoples[1]

- Self-identification as Indigenous peoples by individuals and acceptance as such by their community
- Historical continuity and land occupation before invasion and colonisation
- Strong links to territories (land and water) and related natural resources
- Distinct social, economic, or political systems
- Distinct language, culture, religion, ceremonies, and beliefs
- Tendency to form non-dominant groups of society
- Resolution to maintain and reproduce ancestral environments and systems as distinct peoples and communities
- Tendency to manage their own affairs separate from centralised state authorities

Panel 2: Major health problems of Indigenous peoples

- High infant and young child mortality
- High maternal morbidity and mortality
- Heavy infectious disease burdens
- Malnutrition and retarded growth
- Shortened life expectancy at birth
- Diseases and deaths associated with cigarette smoking
- Social problems, illnesses, and deaths linked to misuse of alcohol and other drugs
- Accidents, poisonings, interpersonal violence, homicide, and suicide
- Obesity, diabetes, hypertension, cardiovascular disease, and chronic renal disease (lifestyle diseases)
- Diseases caused by environmental contamination (eg, by heavy metals, industrial gases, and effluent wastes) and infectious diseases caused by faecal contamination

Panel 3: Nutritional deficiencies of Indigenous peoples

Besides hunger and general inadequacy of food and dietary energy (calories), specific deficiencies of various nutrients are widespread. Examples are iron deficiency, which can be caused by dietary inadequacy or secondary to blood loss, intestinal parasites, or malaria; hypothyroidism, shortness of iodine affects hundreds of millions of people; poor vitamin intake (eg, vitamins A and D, folic acid); and heavy metals, such as zinc. These deficiencies and any underlying causes, including poverty and inadequate food, should be corrected to reach satisfactory outcomes for those affected.

their long-term sustainability. Water supplies were protected from loss and spoilage, and for agriculturalist groups protection of water supplies was very important to support their crops.

Colonisation had a powerful effect on Indigenous populations. It blocked access to or destroyed traditional farming, food-gathering, or hunting and fishing places and practices.[5,6] This change made the previous inhabitants dependent on colonisers for foods that were often unfamiliar to them and of inferior nutrient quality (panel 3). Colonists introduced harmful substances such as tobacco and alcohol, which had serious long-term effects on health and caused severe social, psychological, and emotional damage.[6–8]

The fabric of traditional societies was shredded by colonisation. Traditional life was suppressed by alien regulations imposed on people who had lived, sometimes for many thousands of years, with well established traditional laws, languages, dress, religions, sacred ceremonies, rituals, healers, and remedies. This legalised disruption was worsened by socioeconomic and political marginalisation, and by racial prejudice which was often entrenched and institutionalised. This process was hastened by the often brutal dispossession of traditional lands, and subsequent poverty, undereducation, unemployment, exploitation by unscrupulous employers and landlords, and increasing dependence on social welfare or begging in cities and towns. Many Indigenous groups have to live on unproductive land or in towns, cities and their fringes, slums, or squatter camps that are environmentally degraded health hazards, contaminated by heavy metals and industrial waste (figures 1 and 2).[9]

These oppressive factors caused severe inequalities in Indigenous health status, unsatisfactory disease and vital statistics, impaired emotional and social wellbeing, and poor prospects for future generations.[10] These issues should be taken seriously to redress socioeconomic and health inequities for Indigenous populations worldwide. This redressal is an immense challenge, engaging different levels of governments, various international agencies, non-governmental organisations, clinicians, and health policy makers and administrators. Indigenous people should be meaningfully engaged rather than prejudicially excluded from these endeavours.

Health of children and mothers

Poor living conditions, inadequate nutrition, and exposure to high rates of infection cause a heavy burden of disease in infants and children.[11–13] These diseases are mainly skin infections, acute and chronic ear disease, dental caries, trachoma, diarrhoeal diseases, parasite infestations, upper and lower respiratory tract infections, urinary tract infections, and viral and bacterial infections affecting the nervous system. Indigenous children have high rates of low birthweight and being small-for-gestational age. These factors can affect development of cardiovascular disease, renal disease, and diabetes in adulthood.[14]

Unfavourable perinatal and neonatal health outcomes, including deaths, are pressing issues, especially in developing countries.[15] Several interventions could cost-effectively save many of these lives.[16] These interventions, including improved clinical care to poorly served groups, should engage families and communities and improve home-care practices. Better and well coordinated care and supervision for mothers and babies should be implemented simultaneously.[17]

Some diseases are prevalent in specific areas, such as tropical regions. These diseases include malaria, measles, dengue, haemorrhagic fevers, amoebiasis, ancylostomiasis, ascariasis, strongyloidiasis, schistosomiasis, and viral infections such as hepatitis and encephalitis. HIV infections affect many Indigenous infants, children, and adolescents. Childhood diseases are linked to substandard hygiene, nutrition, and immune status, worsened by heavy exposure to environmental microbial contamination such as contaminated water, food, utensils, or person-to-person or animal-vector-spread diseases such as giardiasis or salmonellosis. Infections are also associated with falling breastfeeding practices and contamination of non-human milks or other fluids.

Socioeconomic status is a major determinant of disparities in Indigenous health, irrespective of ethnicity.[18] Immunisation is effective against vaccine-preventable childhood viral infections in which strain variation is low and herd immunity is high, as in measles and hepatitis B.[19] However, universal vaccination is often not feasible in Indigenous populations, especially in remote areas. Vaccine-preventable diseases, including measles, mumps, diphtheria, rubella, pertussis, and tetanus have been controlled in most non-Indigenous populations but are still rife and potentially fatal in many Indigenous groups. This area should be a priority for action by governments and non-governmental organisations.

Many groups do not have access to traditional foods[5,6] and depend on commercial foods sold in Indigenous community stores or in small towns, villages, or at roadhouses; their infants and children are often malnourished. This malnourishment is frequently caused by poverty and insufficient food, and is worsened by inadequate facilities in the home to securely store and keep food cool and uncontaminated. Substandard nutrition of infants and young children can be associated with maternal ill-health and malnutrition, or both, which can negatively affect pregnancy and predispose to premature birth, low birthweight, and intrauterine growth retardation.[20] Childhood growth faltering and malnutrition are major challenges and are associated with increased mortality. About 15% of Aboriginal children aged less than 5 years in Australia's Northern Territory are underweight, 11% are stunted, and 9% are wasted.[21] Soundly based and community-delivered nutrition education, linked to interventions that enlist carers, community health workers, and community members, can help to prevent growth faltering.[21] Increased infant mortality is prevalent in many Indigenous populations and is related to social and economic circumstances and restricted access to adequate health care. Childhood malnutrition, impaired growth, and stunting are often associated with repeated or chronic infections. Gastrointestinal infections and parasite infestations are especially important because of their negative effects on intestinal digestion and malabsorption of nutrients, minerals, and vitamins.[22] Millions of increasingly urbanised Indigenous youngsters now face the consequences of overnutrition rather than undernutrition, including chronic lifestyle diseases.[23]

Pregnant women, nursing mothers, infants, and children form a large part of Indigenous populations.[2,24]

Getty Images

Figure 1: **Typical housing for Australian Aboriginal people in 2008**
Inadequate housing and overcrowding prevail in urban, periurban, rural, and remote Indigenous populations; many other Indigenous people are homeless or live in makeshift camps and shanties.

Figure 2: **Hazardous waste in an Aboriginal housing area in tropical northwest Australia in 2006**
Environmental contamination predisposes to high rates of recurrent and chronic infections in many communities.

Maternal health before and during pregnancy and while nursing their infants is essential for the health, nutrition, and growth of infants and young children. Many Indigenous mothers are ill-prepared for pregnancy. They can be very young or have been pregnant many times and, consequently, at high risk of complications to themselves or their infants. They also tend to have high rates of other risk factors, including (1) under-nutrition during pregnancy, which can be worsened by the necessity of many mothers to do strenuous physical work throughout pregnancy, such as labouring on farms, harvesting and carrying crops or traditional foods, and carrying water daily for many kilometres for domestic chores; (2) anaemia caused by nutrient deficiencies (eg, iron) and underlying disease, or both; (3) deficiencies of other nutrients (eg, iodine, zinc, and vitamins); (4) inadequate preparation, education, and prenatal and postnatal clinical care; (5) high rates of largely preventable urinary tract infections;[25] (6) gestational diabetes, which can pre-date permanent diabetes; and (7) scant human, clinical, and laboratory resources for safe pregnancy, delivery, and postnatal care. Many Indigenous women, especially those in poor countries, have little or no access to basic clinical staff and facilities that should be part of the routine care of women before, during, and after pregnancy. Gestational glucose tolerance,[26] obesity, pregravid weight, and weight gain during pregnancy can adversely affect maternal and fetal outcomes,[27] including HbA_{1c} and blood pressure measurements in later life.[28]

In many Indigenous societies, traditional midwives and healers give important advice and care to women before and during pregnancy and after parturition. Modern health professionals could usefully collaborate with them so that mothers and infants benefit from their experience.

Indigenous adolescents have many important health-related disadvantages that cause ill-health and disability (panel 4). These disadvantages are worsened by poor educational standards, inadequate knowledge of the determinants of health, and frequent absence of access to and use of good quality clinical care and preventive health services.

Burden of infectious disease

Indigenous people have much higher rates of infection than do their non-Indigenous counterparts, and these infections are likely to be more severe or more frequently fatal in Indigenous groups. The nature, frequency, and severity of infection depends on age, nutritional status, impaired immunity, presence of diabetes, personal living conditions and hygiene, exposure to infections and disease-carrying vectors, immunisation status, geography, and climate.

Skin infections are very common, especially in children. Some examples are: bacterial infections of abrasions, lacerations, vesicles, burns, pustules, and furuncles; superinfection of extensive lesions, such as impetigo; mycoses, including tinea of the head, body, feet, and skin folds (this can be very extensive); candidosis or moniliasis; parasitoses, including scabies, skin, and soft tissue infestation by larvae of flies, pediculosis, insect stings, and bites of fleas and ticks; cutaneous Larva migrans; and leprosy and yaws. These infections can cause permanent scarring and can allow entry of streptococcal infections and invasive diseases such as nephritis or endocarditis.[29–31]

Respiratory and gastrointestinal infections often coexist; they cause widespread illnesses and deaths, especially in infants and young children. Upper and lower respiratory tract infections are prevalent[32] and deaths from pneumonia are "a permanent global emergency".[33] Episodes often coincide with other infections such as gastroenteritis, meningitis, encephalitis, and locally endemic or epidemic diseases—eg, malaria. Respiratory infections can be drug-resistant such that mortality can be very high, especially in patients with malnutrition and impaired immunity. Measles can cause rapidly fatal pneumonia. Tuberculosis is still widely prevalent in many countries and should be suspected in patients with chronic symptoms.[34,35]

Immunisation can help to control some respiratory infections such as pneumococcal disease, *Haemophilus influenzae* type b, tuberculosis, pertussis, diphtheria, measles, and other viral infections.[19] There has been improvement in controlling measles. Vaccination programmes undertaken by the Pan American Health Organization have reduced Indigenous transmission of measles; political commitment was important in achieving this reduction and more effective control is feasible in future.[36] Overcrowding and indoor or outdoor air pollution (eg, from cooking and heating fires, cigarette smoke, and atmospheric pollution) predispose to airway disease and respiratory infections.[32,37,38]

Otitis is prevalent;[39] most episodes of otitis externa can be successfully managed conservatively but otitis media is often much harder to manage and surgery might be necessary. Hearing loss can be permanent if the inner ear is chronically damaged and if audiological management is unavailable. This loss can impede future education, training, and employment.

Diarrhoeal diseases are often accompanied by other infections, malnutrition, and specific nutrient deficiencies, especially in children. Causative agents include viruses, bacteria, parasites, protozoa, fungi, and yeasts. Symptoms range from mild to potentially fatal, as with cholera, shigellosis, and other enteroinvasive infections. Viral diarrhoeas can cause severe watery diarrhoea and widespread morbidity and mortality. Many diarrhoeal episodes do not respond to antibiotics—for example, those caused by non-bacterial agents. Rotavirus vaccine research has proceeded for many years, but vaccines are not yet generally available.[40] Such protection might not be effective in Indigenous populations for several years because of the multiplicity of strains involved, the technical problems and costs of developing effective stable polyvalent vaccines, and the logistical difficulties in their distribution.

Malaria is widespread in tropical countries and causes serious morbidity and millions of deaths. Many strains of plasmodium are multidrug-resistant and work is continuing towards producing vaccines to control the

Panel 4: Health-related problems of Indigenous adolescents

- Little knowledge of determinants of health and disease risk
- Increasing use of harmful substances such as tobacco, alcohol, and other drugs
- High-risk sexual activities
- High-risk, unplanned, and poorly supervised pregnancies
- Violence and trauma in crowded communities and urban environments
- Increasing rate of obesity in increasingly urban populations
- Mental and emotional disorders

infection.[41] Preventive measures, such as spraying mosquitoes, might be unavailable or ineffective. Indigenous populations are often unaware of the usefulness of simple protective equipment such as mosquito nets, and generally cannot afford them. To reduce the vast disease burden and deaths from malaria, many international non-governmental organisations are taking part in community-based programmes, including education, control of transmission of infection, and use of personal protective procedures.

Invasive meningococcal infections can cause potentially fatal illnesses, including pneumonia and septicaemia; vaccines exist[42] but are often inaccessible to Indigenous populations. Urinary tract infections[29] are very common and often have serious long-term sequelae, including renal failure.[43–45] Importantly, these infections are generally asymptomatic and in Indigenous men and women can cause long-term complications, such as chronic renal insufficiency, unless detected early and managed energetically.[25,46–48] Sexually transmissible infections are prevalent[49–51] and cause immense personal, family, and community damage. The introduction of a vaccine against human papillomavirus infection[52–54] might help to reduce the risk of cervical carcinoma, but only for those who have access to the vaccine. Immunisation against vaccine-preventable diseases in Indigenous populations should be very high priority. But there are inherent problems related to costs, production of sufficient vaccine stocks, adequate storage, transportation, and distribution facilities, and availability of trained staff to administer vaccines and to gather together people to be vaccinated. All these considerations assume access to epidemiological information to alert authorities of the need for vaccination.

HIV/AIDS has been called “the first postmodern pandemic”[55] and HIV infection is a continuing health crisis in racial and ethnic minorities, including Indigenous people.[56] This situation is interwoven with prevalent socioeconomic difficulties, including poverty, homelessness, substance misuse, and unequal access to health care. The AIDS epidemic disproportionately affects such populations, especially women.[56] Maternal HIV infection has particular importance to infants and young children because of increased risk of perinatal mortality, transplacental transmission of the virus, and the consequences of probable premature maternal death. Rates of HIV/AIDS are high in many Indigenous groups—for example, in American Indians, Alaskan Native populations,[57] Indigenous Canadians,[58] in African races,[6,59] and people in the Asia-Pacific region.[60,61] The HIV incidence in Indigenous Mayan Guatemalans, who represent 42% of the country’s population, may be three times as high as in the rest of the population.[62]

Such findings led UNAIDS to take action, by studying trends, vigorously promoting prevention, and engaging Indigenous representatives in decision making. Use of Indigenous languages is important in these processes. Transmigration to urban areas increases the risk of sexually transmitted infections, including HIV/AIDS, in Indigenous populations. The upsurge of these infections in the Indigenous peoples of Brazil, particularly those involved in agrarian conflicts or migrating to towns and cities, led to the establishment of Special Indigenous Health Districts. This system allowed improvement in disease surveillance, treatment, and control, and encouraged greater participation of Indigenous community health workers in the network’s activities and assessment.[63]

The AIDS epidemic is rapidly worsening in the Asia-Pacific region. Most of the Indigenous population does not have access to information, skills, methods, and infrastructure that are necessary for detection, treatment, and prevention, and mainstream health campaigns are inappropriate and ineffective.[60] The high rates of sexually transmitted infections in Aboriginal people in Western Australia are associated with high rates of HIV notifications.[64] From 1994 to 2002, the age-standardised rate ratios of HIV notifications, compared with non-Indigenous people, were 2:1 for men and 18:1 for women. Public health authorities are attempting to control these problems but “the clock is ticking”.[64]

Infections of the nervous system, such as meningitis and encephalitis, can have disabling long-term complications and are potentially fatal. Effective treatment might not be available or accessible. Some of the causative agents are bacteria (including tuberculosis), rickettsiae, viruses, and fungi. These infections can cause brain abscesses. Ophthalmic infections often cause visual impairment or blindness.

Soft tissue infections, such as pyomyositis, are often deep-seated and serious, especially in children. Osteomyelitis can be blood-borne and associated with penetrating injuries or fractures. Dental and periodontal diseases are common and can be associated with rheumatic and other forms of cardiovascular disease.[65,66]

Urbanisation and upsurge of lifestyle diseases

Urbanisation has had a profound effect in the past century. The process of urbanisation is usually regarded as the growth of cities and rural-to-urban migration. Millions of Indigenous people now live in urban or periurban areas. The effects of urbanisation are virtually worldwide and are not confined to large groups. These effects are caused by increasing commercialism, acculturation, and rapidly changing lifestyles. They include modern high-calorie, high-fat, high-salt, and low-fibre diets, changing infant feeding practices, decreased physical activity, overcrowding, and environmental contamination.[23] The effects of urbanisation on health, including chronic lifestyle diseases, have been intensifying in industrialised countries for many years and are a major international public health problem. These hazards have emerged more recently in Indigenous groups that seem prone to them, such as the Indigenous peoples of North America

Panel 5: Major difficulties, trends, and factors that affect Indigenous health

Persistent problems
- Poverty, hunger, environmental contamination, frequent infections, and parasites
- Infant and child malnutrition and growth failure
- High infant and young child mortality
- Maternal ill-health and high mortality
- Chronic ill-health and disabilities
- Shortened life expectancy
- Poor understanding of the complexities of Indigenous health by health professionals
- Widespread prejudice about perceived inadequacies of Indigenous people
- False expectations that medical strategies alone can overcome Indigenous health problems
- Government preoccupation with sickness services rather than wellness strategies
- Bureaucratic mishandling of culturally sensitive matters beyond their rigid protocols
- Insufficient chances for Indigenous people to be trained and take part in their health care
- Inadequate systematic data to allow surveillance and improvement of Indigenous health care
- Government indifference, ignorance, neglect, and denial about the poor state of Indigenous health

Areas with improvement
- Suppression of some vaccine-preventable diseases
- Improved pregnancy outcomes, including birthweights
- Lower rates of some infections and related deaths, especially in infants and young children
- Reduced maternal, infant, and young child mortality
- Increased life expectancy in some populations
- Improved education in some Indigenous groups and their employment in health-related fields
- Introduction of Indigenous components to education and training of health professionals
- Training of Indigenous people for careers in health professions
- Increased participation of Indigenous people and groups in policy-making and political affairs
- Widening awareness of the seriousness of health issues in Indigenous peoples
- Formal recognition by some national governments of Indigenous peoples' rights (eg, Australia, Canada, Japan)

Areas of deterioration
- Erosion of the authority of Indigenous Elders
- Illnesses associated with overcrowding and environmental contamination in squatter settlements, urban slums, and disaster situations
- The rapid upsurge of lifestyle diseases
- Respiratory and peripheral vascular disease associated with cigarette smoking
- Diseases and social problems associated with misuse of alcohol and other drugs
- Emotional, mental, and psychiatric illnesses
- Interpersonal and family violence, including, child abuse, homicide, and suicide
- Motor vehicle and other accidents and poisonings
- Sexually transmissible diseases, including HIV/AIDS

and Australia. The negative health effects of urbanisation now occur in *barrios* (small towns) and even in very remote Aboriginal communities in the Australian outback.[23] Misuse of alcohol and other drugs, injuries, poisonings, violence, and accidental deaths and injuries are also important hazards.[23,67]

The worsening epidemic of lifestyle diseases includes obesity, hypertension, cardiovascular disease, type 2 diabetes mellitus, chronic renal disease, and renal failure.[68,69] This epidemic is part of an international "crisis in public health".[70] These disorders are now prevalent in Indigenous populations—for example, in Australia[71–73] and the Americas.[74–78] These disorders have emerged recently in these groups, perhaps because of genetic predisposition and changed diet and lifestyle.[72,73,79–81] This issue is so serious in remote Aboriginal peoples living in northwest Australia that 40% of all adults and almost 60% of those aged 35 years or older have diabetes.[71] Some Indigenous children become overweight and hyperinsulinaemic as young as age 5 years.[82] Aboriginal children up to 17 years of age in Western Australia have a diagnosis rate of diabetes that is 18 times that of their non-Indigenous counterparts.[83] This disturbing upsurge has occurred in many Indigenous populations, especially in recent decades. Previously, the main childhood nutritional disorders in Indigenous Australians were malnutrition, stunting, and infections; now, increasingly, they are obesity and related risks of lifestyle diseases.[72] This nutrition transition has occurred in many countries, including in Chilean adults and children.[84]

Chronic diseases have become worldwide health problems that cause many millions of deaths every year.[85] This epidemic is worsening in low-income and middle-income groups and is driven by rapid social and environmental changes that aggravate the prevalence of preventable risk factors.[86] This situation helps to explain the importance of the risk factors in Indigenous populations. The main risk factors for chronic disease are unhealthy diet, decreased physical activity, and tobacco use. These factors apply in lower socioeconomic groups in industrialised, developing, and transitional societies.[85] Non-communicable diseases tend to increase as rates of infectious diseases lessen. The global burden of disease differs greatly around the world. Infectious and nutritional diseases are major problems in Africa, but are very much less so in high-income regions such as Europe. Non-communicable diseases are more important in higher-income regions, such as Europe, the Americas, and now the western Pacific and southeast Asia regions of WHO as these regions have become more affluent and urban.[87] However, such data can be misleading because they are aggregated and do not reveal variations within regions or countries. Chronic diseases and their risk factors need to be countered by promotion of healthy lifestyles, change in food habits, encouragement of physical activity and sport, discouragement of cigarette smoking and alcohol and drug misuse,[85] and by fostering of physical and emotional wellness (panel 5). Unless these changes take place chronic diseases will spread more widely as more and more Indigenous people adopt sedentary modern lifestyles. Related morbidity and mortality can be improved by control of blood glucose, blood pressure, and lipid concentrations.[88] Remote

Australian Aboriginal groups, given the opportunity, can collaborate through community-based programmes and with conventional clinical services to keep the devastating effects of these disorders to a minimum.[71] Indigenous people should be encouraged, trained, and enabled to become increasingly engaged in and take responsibility for their own health and wellbeing.[2,89]

Changing patterns of Indigenous health

Major difficulties, trends, and factors that affect Indigenous health are summarised in panel 5. Clearly, Indigenous people should have better health than they do at present, which will depend on recognition of the problems and resolute action to overcome them. Approaches should relate to local circumstances, interaction between Indigenous and non-Indigenous parts of society, and provision of improved health-related services.

Is Indigenous health changing? This important but sweeping question has no simple answer, mainly because of scarcity of reliable data. Such information is urgently needed to document present health status in Indigenous people, develop appropriate strategies and programmes, assess effectiveness of those activities and modify them if necessary, compare health standards between different groups of Indigenous and non-Indigenous people, and study changes in Indigenous health over time.

How can health or wellness be measured? Widely used indicators include infant mortality, mortality of children aged 0–5 years, incidence or prevalence of diseases and their risk factors, and life expectancy at birth. These are clinically-orientated statistical markers that give no indication of broader issues of physical wellness or social wellbeing. Indicators that use mortality rates measure the worst outcomes. More comprehensive indicators of health and wellness, presence or absence of disease or risk factors, and long-term outcomes are needed.

Disability-adjusted life year (DALY) assessment is a widely accepted single summary measure of population health. On the basis of this assessment to measure the main risk factors, diseases, and causes of excess mortality in Indigenous Australians,[90] the age-adjusted rate ratios of DALYs were higher in Indigenous Australians than in the total Australian population (see table). Among 20 diseases and injuries causing the greatest burdens in men were homicide and violence (relative risk 6·8), inflammatory heart disease (6·3), and lower respiratory tract infections (6·1). For women the greatest differentials were for rheumatic heart disease (26·4), homicide and violence (11·0), and alcohol dependence and harmful use (7·9).

11 risk factors collectively explained 37% of the Australian Indigenous disease burden. These factors were tobacco use, alcohol, illicit drug use, high body mass, inadequate physical activity, low intake of fruit and vegetables, high blood pressure, high cholesterol concentration, unsafe sex, child sexual abuse, and physical abuse of intimate partners. Some of these factors were major contributors to cardiovascular disease, diabetes, chronic respiratory disease, and injuries and violence, which shows their importance when designing and implementing strategies and interventions to lessen the burden of disease, injury, and premature deaths. Of 193 countries, all Australian men aged 15–60 years had the seventh lowest and all Australian women the 12th lowest probability of dying in 2003, yet Indigenous Australians were in a worse position than the East Timorese, whose probability of dying was worse than 130th in that list of 193 countries. These findings have many important implications. The major risk factors should be targeted more carefully, and better health-care facilities and services are needed because mortality in ill Indigenous Australians is worse than in other Australians. Furthermore, the disability and mortality gaps are greatest for young Indigenous people. Risk reduction in young people should have much higher priority than it does now.

The Millennium Development Goals expect that all people should benefit from development.[91] However, worldwide, Indigenous populations have higher mortality than their non-Indigenous counterparts.[92] The Indigenous versus non-Indigenous mortality gap is worse in Australia than in other Organisation for Economic Co-operation and Development nations with disadvantaged Indigenous populations, including Canada, New Zealand, and the USA.[92] This gap in Australia reached a stark peak of 17 years in 1996–2001,[93] and was partly responsible for the formal government apology to Indigenous Australians.[94] The federal government is now committed to closing this gap and other forms of long-term disadvantage that Indigenous Australians have.[95] These disadvantages consist of housing availability and standards, community infrastructure and services such as water supplies, environmental hygiene, educational attainments, training and employment opportunities, and accessibility to health care. These gaps will probably not be closed by the target date of 2030 despite our best efforts and irrespective of various strategies, social and medical, that have been proposed.[96] Regrettably, inadequate attention seems to have been given to potential gains that could be achieved through more meaningful involvement of Indigenous Australians and their communities in this task.

Close scrutiny of the use of mortality or life expectancy as a measure of the health of Indigenous peoples raises

	Men	Women
Cardiovascular disease	4·5	5·1
Diabetes	4·4	6·0
Intentional injuries	3·9	5·3
Unintentional injuries	2·4	2·9
Chronic respiratory disease	2·5	2·6

Table: **Age-adjusted rate ratios of Indigenous to total Australian DALYs[90]**

some important issues. In Canada, for example, there were substantial gains in life expectancy at birth in the Indigenous population from the 1940s onwards so that by 2000 the unfavourable gap for Indigenous Canadian men versus other Canadian men was only 7 years and for women was 5 years.[97] There were also steep declines in Canadian Indigenous infant mortality rates over a similar period, although the reduction was more striking for infants of First Nations people than for the Inuit, who still have infant mortality rates three times those of the national Canadian rates.[97] There have been substantial reductions in Indigenous infant mortality in Australia, too, over the past few decades, although the rate is still double or three-fold that of non-Indigenous Australian infants. Information from selected states has to be used because consistent, reliable, nationwide data are not available.[98] Between 1991 and 2005, the Indigenous infant mortality rate in Western Australia (WA) and South Australia (SA) fell from roughly 23 deaths per 1000 livebirths to about ten deaths per 1000 livebirths; the decline in WA was 39% and in SA was 26%.[98] Over the same period the relative rate ratios (Indigenous versus non-Indigenous) of infant mortality rates in those jurisdictions changed from more than 4 to about 2·5.[97] Over the same period Indigenous all-age mortality rates decreased somewhat but, despite that, in WA, SA, and the Northern Territory the mortality rate ratios (Indigenous versus non-Indigenous) increased because non-Indigenous mortality also declined substantially.[98] To regard Indigenous people, even in one country, as a single, homogeneous entity is hazardous. A major feature of Indigenous populations is their great diversity. Observers often think of Australian Aboriginal people as a single group. But there were hundreds of Aboriginal groups or nations before colonisation, and dozens of languages and cultures. Cultural diversity persists, especially in remote areas.

Figure 3: **Aboriginal Elders in the Kimberley region in the far northwest of Australia**
They are taking part in Indigenous-driven, community-based health programmes. The local spirit figure of the *Wandjina* is on the mural.

Conclusions

When considering Indigenous health worldwide, one can feel overwhelmed and discouraged by the great disparities in health and disease statistics. Using Indigenous Australians as an example,[90] we see that Indigenous peoples have higher rates of physical, mental, and emotional illness, injuries, disability, and earlier and higher mortality than do their non-Indigenous counterparts. The mortality gap between Indigenous and other Australians is considerably greater than the disability gap—ie, when they are ill, they are more likely to die than are non-Indigenous Australians. This discrepancy is probably caused by late presentation, frequent severe or complicated illnesses, inadequate access to good clinical care, and inadequate follow-up, compliance with drugs, and prevention of complications. Diabetes, cardiovascular disease, and tobacco use account for half the Indigenous health gap; as well as tobacco use, these diseases share other lifestyle risk factors. Rather than seeing this situation as a cause for despair, it should be seen as a potential target for greater health gains in the sicker group. Improvement could be achieved by addressing particular diseases and risk factors, targeting the most affected age groups, and providing effective interventions. Infections and neonatal disorders are examples in which targeted interventions should produce substantial health gains. These messages should be convincingly conveyed to politicians, policy makers, and community leaders. They have to be persuaded that Indigenous health demands priority and that prevention is better than cure.

There are some simple, affordable, and effective ways to improve Indigenous health. Basic hygiene could be improved through better personal, domestic, and community hygiene, disposal of dirty or stagnant water, sewage, and litter, and prompt treatment of skin sores. Clean drinking water should be provided to target communities and families, and local people should know the importance of it and relevant authorities should assist by making sure that supplies are clean. Heavy work, particularly by women and children, of carrying domestic water over long distances should be reduced. Local communities, their representatives, and health committees should be encouraged to contribute to and take responsibility for their health (figure 3). These groups might need financial, physical, and other support from governments and other organisations to be able to do so. Health and hygiene education should be provided to individuals, families, and communities, with a focus on community participation.

The basic causes of illness are similar in Indigenous and non-Indigenous peoples. But the burden of disease, disability, and death is consistently greater in Indigenous than in non-Indigenous people. The principles for improvement we mention require acknowledgment by governments of Indigenous peoples' special rights and needs; adequate, regularly collected data about Indigenous health and related factors; adequate resources to close the gaps in health, disease, disability, and mortality between Indigenous and other peoples; and addressing socioeconomic inequities between Indigenous and non-Indigenous populations to overcome these discrepancies (see panel 6).

These issues should all be addressed to overcome these problems. This process will need recognition and improved understanding of the issues, commitment by governments to contribute much more than previously, and acceptance that Indigenous people have to be more meaningfully engaged in these efforts. Most governments have given little attention to Indigenous health because of ignorance, indifference, political unpopularity with the grim realities of the situation, and failure to officially recognise or enumerate Indigenous people in official statistics. Agencies that have provided clinical and related health-care services for Indigenous people have often had little success because of an absence of awareness or acceptance of Indigenous cultural behaviours (including taboos) and needs, such as for families to be present during clinic visits and when patients are hospitalised, for female patients to have female clinical staff in attendance (and male staff for male patients). Furthermore, specific ethnic or tribal groups need culturally appropriate clinical carers, and insensitivities to Indigenous attitudes and behaviours such as not keeping to rigid timetables for clinic visits have contributed to this failure. Indigenous people often need long and painstaking explanations about the causes of their illnesses, how their drugs work, and why they should keep to the clinical instructions they have been given. Many conventional clinical-carers are unaware of these needs or are too impatient or busy to appreciate their importance.[99] Indigenous people often have difficulty understanding the language of the dominant society. Better communication is key to improvement of health. Large health bureaucracies often fail in many of these areas.[99]

Health standards of Indigenous peoples are unacceptably poor but there is no need to despair; correction of the present situation needs a radical reorientation of previous strategies that have been ineffective or virtually non-existent. Apart from the approaches we propose in this section, also important is to enable, train, and encourage Indigenous people to take responsibility for programmes and services that affect their health and for them to work closely with existing health-care systems. Emphasis on the urgent need for local, regional, national, and international statistics about Indigenous health is important to allow assessment of future trends and usefulness of interventions.

Panel 6: Key strategies to improve Indigenous health

Health of mothers and children
- Prenatal clinical care, health and nutrition education for pregnancy
- Avoidance of risks in pregnancy such as smoking, alcohol, and other drugs
- Detection of disease and disease risk promptly in pregnancy and treat as needed
- Avoidance and treatment of anaemia and other nutritional deficiencies
- Provision of adequate facilities and services for safe birthing
- Encouragement of breastfeeding and safe, nutritious weaning practices
- Regular monitoring of child growth and use of appropriate nutritional and clinical care
- Early referral of infants and children for clinical treatment
- Encouragement of healthy lifestyles, avoidance of high-risk health behaviours such as unsafe sex, smoking, and alcohol and drug misuse

Nutritional deficiencies
- Provision of enough nutritious and affordable food
- Targeting of vulnerable groups such as pregnant women, infants, and elderly people
- Provision of nutritional supplements as needed (eg, iron, iodine, zinc, and folic acid)
- Treatment of underlying causes such as malaria, intestinal parasites, and blood loss

Infectious diseases
- Provision of adequate housing, clean food and water supplies, and food storage places
- Encouragement of personal, family, and community hygiene at all times
- Disposal of rubbish, sewage, and solid waste, and draining of stagnant ponds and waters
- Prevention of contamination of water supplies and areas where people meet or eat
- Suppression of vectors of infections such as flies, mosquitoes, other insects, and larger animals
- Immunisation programmes against vaccine-preventable diseases
- Early and adequate treatment of infections

Urbanisation and lifestyle diseases
- Encouragement of nutritious eating habits throughout life
- Encouragement of regular exercise and weight control
- Discouragement of cigarette smoking and alcohol and drug misuse
- Regular and opportunistic screening, or both, for risk factors and follow up
- Encouragement and supervision of compliance with drug treatment and follow up
- Prevention of long-term complications—eg, by diet, exercise, weight control, and clinical care
- Encouragement of Indigenous involvement in community-based wellness programmes

Contributors

MG was the primary author and did most of the search of published work. MK contributed to the review of published work and writing.

Conflicts of interest

MG is a medical adviser to the Unity of First People of Australia, an Aboriginal-run not-for-profit organisation. He has no financial conflicts of interest. MK declares that he has no conflicts of interest.

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"It's Always a Part of You": The Connection Between Sacred Spaces and Indigenous/Aboriginal Health

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"It's Always a Part of You": The Connection Between Sacred Spaces and Indigenous/Aboriginal Health

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Abstract

Since colonization, Indigenous/Aboriginal Peoples (IAP) have fought for their inherent rights to follow their ways of life on their traditional territories. One continuing battle is the protection of sacred spaces. Sacred spaces are places recognized by IAP as deeply spiritually and powerful. Relationships to sacred spaces sustain spiritual connections integral to our concepts of holistic health/well-being and are vital for cultural integrity. Though all of the natural world is sacred to IAP, the particular cultural and spiritual significance of sacred spaces and impact on health merits attention. Drawing from qualitative research, this article investigates IAP's perspectives and experiences regarding the connection between Indigenous/Aboriginal and sacred spaces, and we conclude that the desecration of sacred spaces has negative impacts on IAP's health.

Keywords: Sacred Spaces; Indigenous/Aboriginal People; Indigenous health and well-being; environmental desecration; cultural identity

Introduction: IAP's sacred spaces, health, and research needs

Indigenous/Aboriginal People (IAP) around the world are uniting to protect their sacred spaces from desecration. In what is now the United States but was first Indigenous homelands, current examples are Mauna Kea in Hawai'i, Nuvatukya'ovi and Oak Flat in Arizona, and Standing Rock in North Dakota. The authors of this article maintain that sacred spaces are crucial to IAP's ways of life, and we argue that the desecration of these places has negative impacts on health. Most notably, because colonization is designed to terminate, assimilate, and relocate IAP, the elimination of their existence and connection to their land and culture remain constant threats. Examples are replete worldwide—from the United States' Code of Indian Offenses of 1883, the Revised Laws of Hawaii, to Canada's Indian Act of 1876, which outlawed traditional healing and ceremonies (First Nations in Canada, 2013; Department of the Interior, 1883; Medicine, n.d.). We assert that like a disease, colonization spreads and causes harm to the "physical, social, emotional, and mental health and well-being in traditional societies" (Gracey & King, 2009).

International agencies acknowledge these connections between health and environment. As early as 1946, the World Health Organization (WHO) stated,

> Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity. The enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being without distinction of race, religion, political belief, economic, or social condition. (2007)

Today, the United Nations Declaration on the Rights of Indigenous Peoples (UN Declaration) addresses Indigenous People's rights to sacred spaces and cultural practices. As Indigenous researchers, we claim that the highest standards of health and human rights possible are not upheld for IAP because of the ongoing, current, and emerging destruction of sacred spaces. Ongoing colonization has led to negative health effects, such as higher mortality and infectious disease rates, poor social determinants of health, and non-communicable diseases, which can be linked with racism, loss of languages and cultural practice ties, and spiritual, emotional, and mental disconnectedness linked with land removal (Gracey & King, 2009; King, Smith & Gracey, 2009). However, additional public health research is required to better understand these links.

We write as three Indigenous women whose goals are to improve the health and well-being of Indigenous People. Danelle Cooper, MPH, is Hopi, Tewa, Diné, and Mvskoke. She writes,

> Although I grew up mainly in the city, I consider my home Moencopi, Arizona. As an Indigenous woman my responsibility and intentions are to protect and heal my people, ancestors, future generations, all living beings, sacred spaces, and Mother Earth. From my family, I have learned that we are connected to Mother Earth and every living being, and that we have to care for and respect them. My responsibility to write this paper is to aid in the protection and healing of sacred spaces and IAP. As Indigenous People we understand that sacred spaces are a part of us and connected to our health, but some of the world does not. Therefore, I hope this article

> will help in spreading awareness around the issue of desecration of sacred spaces and IAP's health and prevent further destruction of sacred spaces.

Treena Delormier PhD, PDt, is a Kanien'keháka (Mohawk) woman and mother who was raised from birth on the reserve community of Kahnawake which is on the south shore of the St. Lawrence River, about 10 miles from downtown Montreal, in Quebec, Canada. She writes,

> I am a health professional and professor of public health and nutrition. My research training is both academic and community based, primarily in my home community. I believe research is a process of coming to an understanding through pursuits of knowledge that aim to improve the human condition and achieve social justice. As a supervisor of research trainees, I emphasize the through systematic inquiry. As an Indigenous researcher I endeavor to center Indigenous knowledges and methodologies in research and supporting the self-determination of Indigenous communities.

Maile Taualii, PhD, MPH, is Kanaka Maoli, a wahine (woman) and mother. She writes,

> I am a Clinical Transformation Healthcare Researcher for the Hawaii Permanente Medical Group, where I bring cultural, ethical, and community-oriented perspectives to clinical transformation. In 2015, I established the world's first global Indigenous Master of Public Health degree program and was awarded the University of Hawai`i, Board of Regents Excellence in Teaching Award. I live with my husband, five children, and three dogs on a 20-acre food forest with our ʻohana, who aim to feed the community traditional, plant-based food from the land.

In this article, we together investigate IAP's perspectives and experiences regarding the connection between IAP's health and sacred spaces, and we provide some specific discussion about Mauna a Wākea and Nuvatukyaʻovi as examples of sacred site desecration.

Background: Sacred spaces and Indigenous health and well-being

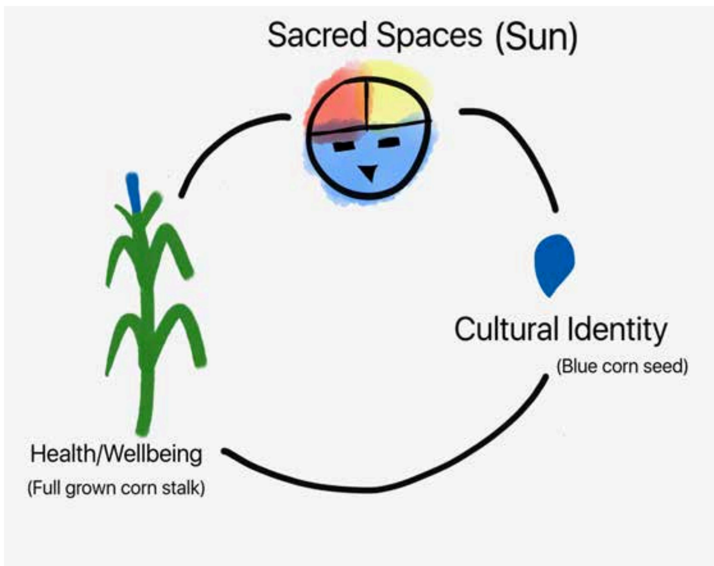
Sacred Spaces

We begin by sharing our understanding of sacred space and our usage of this notion throughout this article before providing specific snapshots of sacred spaces and their relationship to IAP's health and well-being.

IAP have relationships with vital parts of the world considered sacred spaces, which are sites, places, and areas that are believed by IAP to hold power. We understand this relationship as IAP philosophy that asserts, 1) sacred spaces are foundational to Indigenous/Aboriginal ways of life; 2) IAP are attached to sacred places; and 3) IAP express responsibility to sacred places. As Deloria Jr. (2003) states, "Sacred places are the foundations of all other beliefs and practices because they represent the presence of the sacred in our lives" (p. 285). Most IAP will articulate connection with the land and the natural environment in their homelands and consider natural elements sacred—that is, our environments *are* our cultural identities, origins, religions, and worldviews, and our relationships to our environments require actively bonding with elements that include mountains to forests to deserts (Tsosie, 2000). Tewa scholar Gregory Cajete further writes, that this active bonding means harmonizing with place as "a matter of spiritual, psychological, and cultural survival for Indigenous People" (1994, p. 81). Harmonizing is to connect with places not only on a physical level of being in a place, but bonding at the mental, emotional, and spiritual levels. In plain speak, to have a relationship with a place and to know this place for IAP is similar to knowing and relating to one's family.

We believe that as IAP, we have a responsibility to care for sacred spaces and for the Earth, and that this caring is a "sacred covenant with the land" (Cajete, 1994, p. 84). We illustrate our view of sacred spaces with a model (see Figure 1) based on Hopi beliefs about the centrality of corn. IAP are like the corn, illustrated by the blue corn seed. The sun represents sacred spaces, and just as corn needs the sun to grow, IAP are nourished and healed by the power of sacred spaces and the cultural practices that are associated with them. People grow like corn and gain their cultural identity through the cultural practices that connect them to these spiritual places. The fully-grown corn represents people's health and well-being, because when sacred spaces are thriving, people are healthy. This relationship with sacred spaces promotes IAP as mentally, emotionally, physically, and spiritually healthy. Without interaction with sacred spaces, IAP lose connection to their spirituality, ancestors, community, and the future generations.

Figure 1. Hopi corn model



Mauna a Wākea and Nuvatukya'ovi

Mauna a Wākea (Mauna Kea) in Hawai'i and Nuvatukya'ovi (Hopi for San Francisco Peaks) in Arizona serve as focal examples for our discussion on sacred places and Indigenous health connections. Kanaka Maoli Leon No'eau Peralto (2014), expresses that Kanaka Maoli (Native Hawaiian Peoples) have direct familial relationship with Mauna a Wākea, which means they are related to Mauna a Wākea, and the mountain is their family. Mauna a Wākea is the child of Papahānaumoku (Earth Mother) and Wākea (Sky Father), and this is where Poli'ahu (snow godess) other akua (god/goddess) live (Peralto, 2014; Nā Puke Wehewehe 'Ōlelo Hawai'i, 2003). Peralto states, "Mauna a Wākea is the piko [navel] that connects us to the heavens," and like the navel on human bodies, "Mauna a Wākea represents our physical and spiritual connections to past, present, and future generations" (2014, pp. 236-238). Peralto further shares,

> *We are the Mauna*, and our treatment of it reflects a deeply ingrained notion of the ways in which we now view and treat ourselves and each other. In neglecting our kuleana [responsibility] to mālama [to take care of] this ʻāina [land], we ultimately neglect our kuleana [responsibility] to the future generations of our lāhui [nation]. (Nā Puke Wehewehe ʻŌlelo Hawaiʻi , 2003, p. 241)

Kanaka Maoli are a part of Mauna a Wākea, and how they treat the mauna is how they treat themselves. Currently, there is proposed scientific development on Mauna a Wākea, which is desecration to Kanaka Maoli. The University of Hawaiʻi (UH) is the main proponent behind planned construction of a Thirty-Meter-Telescope (TMT), and as the colonial powers, they believe they have control over this area (Brown, 2016; KAHEA: Timeline of Mauna Kea Legal Actions Since 2011). Since the 1960s, Mauna a Wākea has held 13 telescopes overall (HNN STAFF, 2019; Andone, Jorgensen, Sandoval, 2019). Because Mauna a Wākea is considered "ceded crown lands," the State Land Department has been leasing this sacred space to UH (HNN STAFF, 2019; Andone, Jorgensen, Sandoval, 2019; see KAHEA: The Hawaiian Environmnetal Alliance). Furthermore, UH has then sub-leased Mauna a Wākea to other organizations through the Department of Land and Natural Resources (HNN Staff, 2019), including the TMT International Observatory LLC (TIO) members, which include a number of institutions—Caltech, University of California, Natural Institutes of Natural Sciences of Japan, the Natural Astronomical Observatories of the Chinese Academy of Sciences, the Department of Science and Technology of India, National Research Council of Canada, the Association of Universities for Research in Astronomy (AURA), and the Gordan & Betty Moore Foundation. Kanaka Maoli believe that Mauna a Wākea is already suffering desecration through the 13 telescopes presently occupying the space, and that the added TMT will cause further harm.

Across the ocean on the United States mainland is Nuvatukyaʻovi (San Francisco Peaks), a sacred space for Hopi People in Arizona being desecrated by a ski resort called the Snowbowl (Wilson v. Block, 1983; Hopi

Tribe v. Arizona Snowbowl, 2018). The San Francisco Peaks are sacred to 13 Indigenous Nations in the region, which include Diné, Zuni, Hualapai, Havasupai, Yavapai-Apache, Yavapai-Prescott, Tonto Apache, White Mountain Apache, San Carlos Apache, San Juan Southern Paiute, Fort McDowell Mohave Apache, Acoma, and Tohono O'odham (see Protect the Peaks for more information).

The Hopi People oppose the privately-owned ski resort, which leases the land from the United States Forest Service (Schlosberg & Carruthers, 2010). Furthermore, the Snowbowl is using reclaimed wastewater to make artificial snow for skiers (Wilson v. Block, 1983; Hopi Tribe v. Arizona Snowbowl, 2018). In Hopi, Nuvatukyaʻovi is central to Hopi culture, critical to ceremonies.

Indigenous/Aboriginal Concepts of Health/Well-being: Land and Identity

It is widely argued that IAP view health differently than western society. Western conceptualizations of health focus on physical elements related to the biomedical being, while Indigenous epistemologies focus holistically on the physical, mental, emotional, and spiritual being (King et al., 2009). IAP's ways of life and health incorporate philosophies of people in/striving towards harmony and spiritual relationships with the land, community, ancestors, and the spirit world (Gracey & King, 2009; Liu, Blaisdell, & Aitaoto, 2008). There is extant scholarship and testimony that supports the claims that connection to land is integral to IAP's health (King et al., 2009), and that IAP's notions of health include the overall wellbeing of family, community, and the Earth (Crivelli, Hautecouer, Hutchison, Llamas, & Stephens, 2013; Gracey & King, 2009).

For example, the Ojibwe peoples of the Northwoods of the United States and in Canada will refer to their health and well-being as resulting from balanced relationships between family, community, environment, and the mental, emotional, spiritual, and physical elements that must be engaged in those relationships (Malloch 1989; Richmond & Ross, 2013). Maori health models developed by Mason Durie (1994) are founded in the Whare Tapa Whā model, which incorporates the Taha tinana (physical), Taha wairua (spiritual), Taha hinengaro (mental), and Taha whānau (extended family). Along with these elements are the Te ao turoa (environment) and Te reo rangatira (identity) (Durie, 1994; Maori Public Health Action Plan, 2003-2004). Kanaka Maoli regard wellness as "lōkahi (oneness) and pono (harmony, balance)," with people and the world by maintaining "proper thoughts, feelings, and actions," toward everyone (Liu et al., 2008, p. 6). Thus, we advocate for embracing and understanding multi-dimensional concepts of health as critical and including the role that sacred spaces play in affecting holistic constructions of IAP's health.

To date, empirical research addressing links specifically between *sacred spaces* and IAP's health presents a gap in the literature. However, there is work on the relationship between IAP's health and land. Here, we delineate the two. For example, research linking IAP to their lands has introduced "solastalgia":

> the pain or sickness caused by the loss of, or inability to derive solace from, the present state of one's home environment. Solastalgia exists when there is recognition that the beloved place in which one resides is under assault (physical desolation). (Albrecht, 2006, p. 35)

Solastalgia is an "attack on one's sense of place, in the erosion of the sense of belonging (identity) to a particular place and a feeling of distress (psychological desolation) about its transformation" (Albrecht, 2005, p.45). Albrecht explains that solastalgia occurs when place-based distress transpires, and people feel homesick due to their environment being destroyed (2006). Relatedly, somaterratic illnesses refers to primarily physical illnesses related to environmental contaminants, while psychoterratic illnesses relate to mental well-being threatened through the disconnection between Indigenous peoples and their lands (Albrecht et al., 2007). We believe that these concepts also apply to the destruction of sacred sites. For example, we see testimonies of somaterratic illnesses from our participants, including a New Mexico Acoma Pueblo participant who observed, "There's exposure from extractive industries such as mining, which has led to cancer." This person then emphasized "unity" or the collective in upholding responsibilities and maintaining ceremonies linked with preventing illness:

> when we do not uphold this traditional based knowledge,
> when we allow our ceremonies to go unattended, and not

> participate in the full capacity…on numerous occasions you'll see physical ailments too…if we don't have that spiritual mental connection to our ceremonies, our spiritual calendar, and we assimilate to mainstream society, there's negative consequences there…Just even our change in lifestyle…Look at how many of us today are facing illnesses that we never faced historically in the past…High blood pressure, diabetes, heart disease.

Testimonies like these are critical today, and we also note the work of Richmond and Ross (2009) focusing on colonial policies and IAP's environmental dispossession, a process that results in Aboriginal people's reduced access to the resources of their traditional environments. They further argue that environmental dispossession leads to cultural disconnections between land and identity, which contributes to poor health experienced by Inuit and First Nations in Canada (Richmond & Ross, 2009). We add that the impacts of colonization through historical trauma cannot be emphasized enough here. Indigenous historical trauma is a worldwide phenomenon among IAP due to colonization (Wesley-Esquimaux, 2007). Historical trauma is defined as "cumulative emotional and psychological wounding across generations, including the lifespan, which emanates from massive group trauma" (Brave Heart, 2003, p. 7). Researchers further explain that historical unresolved grief stemming from colonial violence constitutes "the current social pathology, originating from the loss of lives, land, and vital aspects of Native culture promulgated by the European conquest of the Americas" (Brave Heart & DeBruyn, 1998, p. 60). Historical trauma results from oppression, negative dominant policy impacts, and the spiritual persecution of IAP's beliefs (Brave Heart & DeBruyn, 1998).

Additionally, Post-Traumatic Stress Disorder (PTSD) and Indigenous historical trauma have been associated with American Indians in the U.S. who face ongoing incursions (Manson, et al., 1996). A prominent example is the Exxon Valdez oil spill in Prince William Sound, Alaska, on March 14, 1989, which has been correlated with anxiety, depression, PTSD, and other negative health impacts among Alaska Natives (Palinkas, Peterson, Russell, & Downs, 1993). This environmental disaster affected Alaska Native traditional subsistence lifestyle, social relationships, and saw increased alcohol, drug abuse, domestic violence and decreased physical health (Palinkas et al., 1993; Palinkas, Downs, Peterson, & Russell, 1993). These environmental disasters and threats are widespread and ongoing, impacting Indigenous homelands and sacred spaces. However, their impacts can be countered through social, cultural, and political processes by which IAP are reclaiming their traditional lands and ways of life (Big-Canoe & Richmond, 2013).

Research on Sacred Spaces

In this section, we describe qualitative research conducted with IAP focusing on testimonies of sacred spaces, including Mauna a Wākea and Nuvatukyaʻovi. Data was collected between July 2015 to January 2016 in Hawaiʻi and Arizona through in-depth interviews. Participants included eight well-regarded IAP cultural experts from different Indigenous nations and between the ages of 31 and 65. Participants were selected based on extensive knowledge regarding sacred spaces in their respective homelands, and as this article focuses more specifically on Mauna a Wākea and Nuvatukyaʻovi, the Kanaka Maoli and Hopi participant testimonies are highlighted in this article. In addition to sharing their knowledge of sacred sites, participants were asked to provide insight on the nature of observed problems and to give recommendations for solutions. Twenty-eight questions comprised the interview protocol, and audio recordings were transcribed verbatim.

A qualitative thematic analysis of sacred spaces in relation to IAP's health was conducted in order to inform the interview process and to allow the researcher to listen for the following themes: sacred spaces (descriptions of), cultural identity (sacred spaces linked with notions of Indigenous identity), and health and well-being (related to sacred spaces).

What are sacred spaces, and how are they desecrated?

When asked, "What do you think makes a space sacred," participants responded using six characteristics. They explained that sacred spaces are,

1) places that have power (i.e. mana in the Hawaiian language) and where energy is embedded;

2) the home to "deities," "gods and goddesses," and "ancestors," as well as places where people can connect spiritually, mentally, emotionally, and physically with them;

3) places of worship, ceremony, and prayer, (similar to how other religions might view their churches, temples, or mosques); where ceremonies such as sweat ceremonies and vision quests take place, and link to ceremonies completed from afar. These places are where "shrines" and heiau [Kanaka Maoli for temple] are located;

4) places of origin, genealogy, and ancestors; participants expressed sacred sites as "there before them," and existing since "time immemorial." For Hopi, where women place "mother's wombs" [placenta or umbilical cord] connecting newborn children to these places, and place offerings such as "prayer feathers."

5) living places; participants called them, "living organisms," "natural," and used IAP's languages to describe them. All of the "land", the ʻĀina [Kanaka Maoli], is sacred. Water is also considered sacred with particular importance given to "rivers" and "springs." Along with "mountains," and "mountain tops" as important sacred spaces;

6) part of healing and ceremonies; sacred spaces are places of healing, and where people "harvest medicine" such as "plants," or "water."

Participants provided specific examples of the desecration of sacred spaces—through natural resource extraction and storing of toxins, including nuclear waste, as well as through extractive industry, including harvesting of timber, and "mining of uranium, copper, lead, molybdenite, and coal." Other examples of desecration include "non-Indigenous commercial and recreational activities," specifically the ski resort on Nuvatukyaʻovi and where the tourist/ski industry produces massive human waste, debris, and general disrespectful encroachment from visitors. In the case of Mauna Kea, Kanaka Maoli participants discussed harm transpiring through what they referred to as the ambitions of Western science, exemplified through telescopes on top of the mountain. Destruction of features of sites, such as an ahu [Kanaka Maoli for shrine] and temples were also noted as ways sites are desecrated.

Cultural Identity

Participants' connections to sacred spaces was conveyed through usage of language like stewardship, covenant, and kuleana [Kanaka Maoli for responsibility]. Participants communicated that their stewardship, covenant, and kuleana were dedicated to deities/spirits, the Earth/world/land, ancestors/people/future generations, and humanity/community. Cultural practices maintained connections to sacred spaces. For example, regarding stewardship to deities/spirits, one Hopi mentions relationship to spirits at Nuvatukyaʻovi:

> very simply that's where we believe the Kachinas spirits live, and our prayers are offered to the peaks and to the Kachina people there, to the cloud priest. And that's been part of our culture for thousands and thousands of years. You know the time the first clans began to arrive here in this part of country and the world, the San Francisco Peaks was experienced. Certain things happened a long time ago, and today thousands and thousands of years later, the Hopi People still I believe carry that relationship to the San Francisco Peaks or to Nuvatukyaʻovi, which is interpreted to mean, the "Peaks with the Snow," that's what it means, Nuvatukyaʻovi. So it becomes very special to you, and then as you grow up into the culture into adulthood and then later into levels of some cultural responsibility, it's daily for us. When you sit down in the kiva and you smoke and pray. And we look at all of the four cardinal directions, we think about it, we visualize it. And then towards the west then you visualize the peaks... *It's always a part of you* [emphasis added].

For the Hopi, Nuvatukyaʻovi is their cultural responsibility, and their relationship with the sacred spaces makes Nuvatukyaʻovi a part of who they are.

Stewardship to the earth, world, and land also represented connections participants carried with them to sacred spaces. The same participant explains,

> the San Francisco Peaks, Nuvatukyaʻovi. You know the significance of the place to the Hopi People. And see so I just want to talk about

> that background on how I look at space and really the environment and the special qualities of what we have naturally here that that gives us a feeling of being Hopi. You know because every clan has a stewardship responsibility. Ceremonies have an Earth stewardship responsibility. So for some of us who have now gone through time and have learned, we take that responsibility pretty seriously…it's not just at big events, it's daily for me.

Participants also expressed a sense of responsibility to both their ancestors and future generations:

> for Native Hawaiians, our connection to land goes back to our genealogy, our moʻo kūʻauhau, our genealogy. And in our genealogy we hear the stories of Wākea the Sky Father mating with Papahānaumoku our Earth Mother. And from their mating comes forth all of our islands… and so these islands are like our ancestors. In our genealogy we're directly connected to them. And so when people desecrate our lands, I try to tell my student this, "Imagine somebody punching your grandmother in the face. How does that make you feel?" That is how a Hawaiian feels when somebody desecrates land. It is as if somebody punched my grandma in the face, now I have a sense of anger, a sense of resentment, but also a sense to protect and to care for my tutu. And if my land is in my genealogy my tutu, or grandparent, I respect the land in that same way. Because the land has provided for my family for generations, and generations, and generations…If we know our genealogy, if we know who we are and where we're from, we will know where we're going. And I know that we come from these lands. That we come from this place. I know that the Kalo is our older sibling. I know that the islands and the stars are our older siblings. And if people begin to desecrate that, it is as if they are desecrating and fighting with my own family.

This participant shares how Kanaka Maoli are connected to the land through their genealogy—thus, desecrating the land is the same as harm to a respected relative. The person adds,

> But once we start to educate on some of these issues and once you know, then you have a kuleana or responsibility to act. But a kuleana

> also means a privilege too, cause now you're privileged to know, now you're privileged to act. It's very dual in that sense where it's a responsibility but it's also a privilege to have these kuleana. What we're training the kids here and in the community it becomes their kuleana, not only their rights, not only their responsibility, but also their privilege to uphold and to help pass on to the next generations and generations.

Kanaka Maoli state that they have a kuleana to take action once they know about issues. Kuleana is a privilege for Kanaka Maoli to uphold and to pass on to the next generation to ensure that future generations also carry on the responsibilities.

Lastly, participants described cultural identity in relation to sacred spaces as tied to humanity, including their communities and other people. Sacred spaces and the ceremonies connected to them are not only done for individual purpose or even the specific IAP group to which they are meaningful. They are for the benefit of *all* humanity. As one Hopi participant states,

> it's never an individual thing, when you go to these locations. It's always for a purpose. There's always a reason for these places that we go to for different offerings…I feel that with these places that are identified as sacred spaces, that those things be maintained so that we can continue to offer our prayers as Hopi People to that one location, and our prayers in turn are going to help in the well-being of everybody as whole, not just Hopi, but everybody in general.

For IAP, like the Hopi, sacred spaces help maintain cultural practices that include everyone. As one Pawnee participant states, "most of these ceremonies that are conducted by Indian people are for the good of humanity as a whole. To "preserve the continuity of the of the universe."

Cultural Practices

In this section, cultural practices are the ways IAP connect to sacred spaces and point to how IAP maintain their responsibilities and connect their cultural identities to these places. Completing cultural practices reinforces the covenant they undertake to protect and care for the Earth.

Cultural practices in direct relation to sacred spaces were defined by participants as ceremonies and rituals, offerings and prayers, seeking healing, and speaking their Indigenous languages. Speaking Indigenous languages is the mechanism for maintaining oral traditions and sharing traditional knowledge. In addition to ceremonies carried out at sacred sites, participants described making pilgrimages and special spiritual journeys to their sacred sites "since time immemorial." One Kanaka Maoli person explains,

> If we're going to the island of Kahoʻolawe for example, we're gonna go next week to honor our Makahiki ceremonies. It's the closing of our Makahiki season, which is like our our winter harvest season or our the birthing of the new year. The Makahiki ceremonies are specific to our Akua or our god Lono. And so when we go, we go and we bring offerings of growth in many different forms…What will bring these physical things along with our spritiual prayers in hopes that when we go to that space and do these ceremonies, Lono will bless us with rain, and not the heavy rains, but the nice soft rains that can help green our lands and green our spaces. So when we go to these lands, when we go to these sacred places, we will do different things according to the place. And so Kahoʻolawe is a very special place in that we will go to to ask the Akua for guidance, to ask the Akua who are gods, for blessings to continue throughout the year. And so to engage in that, we engage in it through chant, through prayer, through observation, through giving of physical offerings, through through sweating on the land and sharing that space with the land and getting dirty in that land. Through eating from that land in that space, but again it all goes back to the the proper way of entering that space and coming into it.

Makahiki for Kanaka Maoli is a winter harvest and New Year ceremony for the Akua Lono (Kanaka Maoli for one of their Gods). Furthermore, oli (chant), and mele (songs) are done at sacred spaces for the space, as well as offerings and prayers such as hoʻokūpū (ceremonial gift) provided to sacred spaces. Participants offer "prayer feathers," poi wrapped in ti leaf, and Hawaiian salt as offerings to sacred sites. Just as the place is honored, sacred sites are also sites considered in IAP's healing, and participants discussed traditional medicines gathered, which may include elements like water. While we do not delve into the details of these ceremonial activities in this article, our emphasis here is on cultural practice connections that are maintained, even from afar, reinforcing IAP's cultural connection to sacred spaces.

The living and essential link to Indigenous/Aboriginal health and well-being

While cultural practices are maintained in many Indigenous communities, our focus is on impacts to IAP's health. Participants were asked to describe this relationship, and they asserted first that the health of sacred spaces is interconnected with IAP's health. As IAP have physical, mental, emotional, and spiritual relationships with sacred spaces, desecration causes harm to people on all these levels. Harm is caused in many ways, including when IAP are obstructed from access to being able to fulfill their stewardship through cultural practices. Additionally, participants described desecration as disruptive, making it difficult (if not impossible) to connect to the spiritual power of places, deities, and their ancestors, all of which they perceived as having negative health effects, including the rise of social problems.

> It's pretty simple: it's harm forever. You know, for me personally, because I was in the thick of the legal fight, the political fight, and then being a practitioner, it's there forever, that experience. To know that the proponents and the Coconino National Forest got their way…the Hopis pray to nature. The Kachinas are a part of nature. So to substitute the natural with technology just doesn't fit in our way of looking at these spaces and landscapes. It just never existed…that kind of substitution never never existed in Hopi ways of thinking…what one of our elders testified that is that if the if snow-making came in, and now it's there because we legally lost. Then our generation who were in the thick of this fight today, it's harm directly. We feel it, we see the mountains there, but what our elders said, "What about over time?" See but the immediate harm for our generation, living generation today is forever. You know and then, in our principles of life, then when we live out our lives in this world

> right, the physical world, then we become spiritual people. So the harm in a secular and physical sense is up to my last day here. But you know what, when I say forever, it's also into my spiritual life. See that's what people really don't understand on this whole business of *emotional harm* for in this case the Hopi People [emphasis added]. (Hopi participant)

This participant was involved in the legal battle to protect their sacred space and described experienced harm on various levels—the physical and emotional harm of fighting, losing politically and legally in court, and by *seeing and feeling* the impact of desecration on their mountain. The Hopis carry the harm forever spiritually because they become "spiritual people" once they live out their physical life in this world. Thus, direct injury does not end on an emotional and physical health level *in this world* but goes on to the spirit world.

Participants importantly described social issues like drinking, drugs, suicide, and domestic violence resulting from desecration. A Kanaka Maoli participant explains,

> there's sort of a disconnection. You see sort of a break down in society. Erosion of cultural values, because you no longer have the places that you made ceremony or worship. You you lose that that knowledge of the place is lost. And then so then the cultural practice discontinues. And then you have following generations wondering why they have pain inside them, you know, it's an unarticulated pain. And it has to do with cultural loss. And it can manifest as maybe criminal behavior, domestic violence, crimes, health problems. It's really just being untethered to something that is, what was the foundational things within your culture. It's being just loosened and untethered from that root.

This participant described "unarticulated pain" when a sacred site is destroyed, because of the connection with the place being disrupted. This disconnection is linked with erosion of cultural values, due to loss of the place where the people made worship or ceremony. Without the sacred spaces and culture practices, IAP feel what participants referred to as "pain inside them" that can impact the future generations due to what is more broadly understood as cultural loss. The same person went on to explain,

> after a while it's like a thousand paper cuts, it's like if the things that are important to your culture are destroyed, then you start to think then, "I'm expendable. I don't matter." And you have you have modern society reflecting that you don't really matter. And then so then when you realize that you're just a throw away person, then your health suffers. Because then you believe that you're worthless, you know, that you lack any kind of value. And so what does that mean, what does that translate, in terms of your health? You get stressed, if you're a person that's trying so desperately to hold onto what's left of your culture, and every day you see an onslaught against it, you know from the dominant society, then there's this strain, this it's like you can never relax in that struggle. Sometimes you find meaning because at least you fighting for something, but if you constantly struggling, then that becomes like major stress, and then the diseases that are associated with stress, that's what manifests, you know for you physically, whether you realize it or not, so it's just sort of this compounding thing, a burden or a pain that you become accustomed to.

Indeed, research with IAP has shown that acculturation and racism physically affect health among Native Hawaiians, with higher levels of hypertension due to the stress of not being exposed to their culture, being disconnected from the Kanaka Maoli community, and perceived daily racism (Kaholokula, Iwane, & Nacapoy, 2010).

Discussion: IAP and Human Rights Frameworks

Through the testimonies shared, desecration of sacred spaces is seen as related to struggles for cultural continuity that are linked to IAP's health. We are reminded of what one participant from Acoma Pueblo stated with regards to desecration and rights: that desecration of these spaces constitutes "human rights violations to our traditional cultural worldview." This is an important reality for us to consider. Moreover, acknowledging their settler colonial contexts, participants described dealing with feelings of worthlessness and the "daily onslaught" against dominant society, which caused them significant stress manifesting as physical pain. These perceptions and realities of health impacts are critical areas for further research. With more public health research that takes on an IAP's sacred spaces lens, we believe that prevention and healing methods can be initiated for and by IAP and in relation to their sacred spaces.

Other forms of intervention include policy, and with the recognition of the distinct rights of IAP, laws and acts have been proposed as one approach to protecting IAP's rights. We assert that Indigenous access to and protection of sacred spaces are human rights, and IAP have been actively pursuing ways to protect them under this framework for decades. Indigenous Peoples strive for "place-based justice," which is based on their "responsibility to protect places important for survival" (Lorenzo, 2017, p. 2). More recently, the UN Declaration explicitly addresses Indigenous rights and can offer justifications for protecting sacred spaces. Laws in the United States that relate to IAP and sacred spaces are the American Indian Religious Freedom Act of 1978 (AIRFA), the National Environmental Policy Act of 1970 (NEPA), the National Historic Preservation Act, and the Native American Graves Protection and Repatriation Act 1990 (NAGPRA) (Lorenzo, 2017). However, although these are in place, they do not always assist and typically require concrete (Western scientific) evidence, which can diminish the firsthand testimonies of IAP's views on sacred spaces and health. For example, under the U.S. National Register of Historic Places certain areas can be designated as a Traditional Cultural Property (TCP), but do not include "intangible resources" (U.S. Department of the Interior, 2012, p. 1), which would include reliance on IAP's cultural practices as not necessarily justifiable for TCP designation. Generally speaking, the issue is much deeper here in that western views of land are focused on ideas of property and do not include discourse on the *sacredness* of land, water, and air. When land is seen solely as property, as Sumida Huaman states, this is "only for human gain, this is a parasitic relationship and not a reciprocal one" (Sumida Huaman, 2017, p. 8).

As Indigenous researchers, we are concerned about how to put into practice protections that consider the relationship between IAP, sacred spaces, and health, and we see human rights education (HRE) as one mechanism. For example, drawing from long-term and emerging United Nations frameworks, Bajaj (2011) writes that HRE is,

> education, training, and information aiming at building a universal culture of human rights through the sharing of knowledge, imparting of skills and moulding of attitudes directed to: (a) The strengthening of respect for human rights and fundamental freedoms; (b) The full development of the human personality and the sense of its dignity; (c) The promotion of understanding, tolerance, gender equality and friendship among all nations, indigenous peoples and racial, national, ethnic, religious and linguistic groups; (d) The enabling of all persons to participate effectively in a free and democratic society governed by the rule of law; (e) The building and maintenance of peace; (f) The promotion of people-centered sustainable development and social justice. (p. 484)

For us, this includes *all* people understanding that desecration has negative effects on humanity and Earth. As our participants have described, there is a ripple effect, with damage done to all living beings in this world and the spirit world. Using human rights education that is founded in our Indigenous knowledges, we hope that students and their families will gain respect for IAP's ways of life and the sacred spaces that we share. As IAP, we also have a right to care for and learn from sacred spaces, constituting what we believe is *IAP's human rights education*.

Conclusion

In this article, we emphasize that the desecration of sacred spaces points towards impacts on IAP's health in multiple ways. Our research considered IAP's definitions of sacred spaces, connections to sacred spaces, understandings of desecration, and perceptions of Indigenous health impacts. We listened to participants who asserted their kuleana to deities/spirits, world/Earth/land, ancestors/people/future generations, and humanity/community, and we understand that in order to fulfill stewardship roles, IAP maintain their relationship to sacred spaces.

IAP are involved in cultural practices associated specifically with their sacred spaces, which are living manifestations of their cultural identities. These include their origins, ancestors, and the future generations all at once. Participants shared that sacred spaces are our identities, part of them forever—from birth to when we enter the spirit world. We, along with our participants, know that we *are* our sacred spaces, *they are always a part of us*, and we are forever bonded to our Mother Earth. While desecration has a massive negative effect on IAP's abilities to access and engage our cultural practices, making it difficult to fulfill our covenant, it does not curtail our love for our sacred spaces and Earth Mother.

Colonization is continual and the driving force behind desecration and remains unrelenting in myriad ways. Knowledge shared from our study may help researchers and Indigenous community members to build health measurements that continue to analyze the relationships between (the desecration of) IAP's sacred spaces and Indigenous health. Possible interventions involve input from IAP, research support, and wide public health efforts rooted in IAP's human rights education. Furthermore, activism and social organizing leading to upholding laws and policies, while creating new ones is another approach key to protecting sacred sites.

As a final word, as Indigenous women, we see our responsibilities as ensuring the health of our people, all living beings, and Mother Earth. Our motivation to partake in this research was to assist our people who struggle daily with protecting our sacred spaces and to share their stories and experiences. Enduring injuries and traumas across time—from our past ancestors, to our present people, and to future generations—it is our collective energy and that of the people who shared their stories, as well as our communities, our ancestors, and our sacred spaces connecting to create what we have presented. This is our daily life—to know, feel, and experience desecration but to also follow our responsibility in protecting and healing our sacred spaces, as well as our commitment to preventing future harms to our people and Earth Mother. We hope you will join us in this responsibility.

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Cultural trauma as a fundamental cause of health disparities

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ABSTRACT

Health disparities disproportionately affect minority cultural groups (e.g., Indigenous, immigrant, refugee) worldwide; enduring across time, disease states, and risk factors despite co-occurring advancements in health and medicine. Fundamental cause theory holds that important social factors (e.g., socioeconomic status, stigma, racism) produce health disparities by restricting equitable access to health-protective resources. Yet, extant literature has not utilized fundamental cause theory to describe the health disparities impact of cultural trauma: an overwhelming, often ongoing physical or psychological assault by an oppressive dominant group on another group's cultural resources through force, threats of force, or oppressive policies. This paper presents a novel conceptual model detailing cultural trauma and the mechanisms through which it may disrupt health and create disparities by damaging three health-protective cultural resources: cultural modes, institutions, and lands. Following cultural trauma, we propose affected groups are socially disadvantaged and exposed to pervasive stress, stigma, and diminished resources, perpetuating health disparities across generations. Consequently, cultural trauma may represent an unrecognized fundamental cause of health disparities, offering potential avenues for promoting health equity through targeted research, interventions, and policies.

1. Introduction

Many minority cultural groups worldwide—including Indigenous, immigrant, refugee, and sexual minority populations—experience profound cultural traumas (e.g., colonization, genocide, hate crimes). Health disparities consist of inequities in health resulting from social disadvantage (Adler, 2009), and are caused by powerful social factors such as socioeconomic status, stigma, and racism that disadvantage and stress minority group individuals (Hatzenbuehler et al., 2013; Link and Phelan, 1995; Williams and Collins, 2016)—increasing their overall risk for illness and death despite stark changes in diseases (e.g., cholera, HIV/AIDs), risk factors (e.g., unsanitary living conditions, poor diet), and medical treatments over time (Link and Phelan, 1995). In this paper, we propose that because cultural trauma may also generate social disadvantage, stress, and mental and physical health problems in minority cultural populations, it may reflect an underrecognized driver of health disparities.

2. Collective trauma and culture

The purpose of this paper is to complement the existing literatures on collective, historical, intergenerational, and racial trauma—which have largely focused attention on these traumas' physical, psychological, or existential harms—by presenting a novel theoretical model linking cultural trauma to health disparities from a resource deprivation/loss and social disadvantage perspective; which has not been posited previously. To do so, we situate these collective traumas in fundamental cause theory (Link and Phelan, 1995); an important piece of canon in the social determinants of health literature (Clouston and Link, 2021). Consequently, this paper does not focus on the devastating physical (e.g., torture, death) or psychosocial (e.g., psychological injury) impacts of collective trauma (which are already well described), but will illuminate how these collective traumas may influence health disparities by disadvantaging cultural groups through the damaging/suppression of health-protective resources.

Conceptually, "culture" is a broadly defined construct labeled by Markus and Kitayama "an untidy and expansive set of material and symbolic concepts … that give form and direction to behavior" (Markus and Kitayama, 2010, p.422), with culture generally considered the shared beliefs, attitudes, norms, practices, institutions, and policies of a particular nation, people, or other social group (Betancourt and López, 1993; Markus and Hamedani, 2007). Thus, "cultural groups" refer to

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diverse social groups defined by shared characteristics including race/ethnicity, religion, nationality, or sexual orientation. Far from being static, cultures are dynamic entities that respond reflexively to changes in environment, resources, and threats; adapting fluidly to afford their members a shared source of resilience, identity, meaning, and connection. Cultures are also resilient, changing in response to migration, acculturation, globalization, and acts of oppression and violence from other groups.

Drawing from this conceptualization of culture as well as current definitions of collective and historical trauma, we define cultural trauma *as an overwhelming and often ongoing physical or psychological assault or stressor perpetuated by an oppressive dominant group on the culture of a group of people sharing a specific shared identity/affiliation (*e.g.*, race/ethnicity, nationality, religion)* (Evans-Campbell, 2008; Kohn and Reddy, 2006; Stamm et al., 2004). In the literature, current definitions of collective and historical trauma largely focus on the psychological or existential responses to a mass traumatic event with collective trauma being defined as the psychological reactions to a traumatic event shared by a group of individuals that becomes ingrained in the group's collective memories (Hirschberger, 2018). Similarly, historical trauma has been conceptualized as a multigenerational trauma inflicted on a group of people with a shared identity/affiliation that encompasses their psychological and social responses to the traumatic event (Evans-Campbell, 2008). Many collective and historical traumas inflicted upon specific groups (e.g., Indigenous, refugee) may meet our cultural trauma definition and criteria. If this occurs, cultural trauma is considered to encompass these collective and historical traumas. However, we differentiate cultural trauma from prior definitions/conceptualizations of collective and historical traumas by centering the focus and impact of the traumatic event on a group's *culture/cultural resources* vs. their psychological or physical well-being; as in collective and historical trauma (Evans-Campbell, 2008; Brave Heart and DeBruyn, 1998; Brave Heart, 1999; Hirschberger, 2018).

To differentiate *cultural* trauma from *interpersonal* trauma, whereas interpersonal trauma involves an assault by a person on an individual's health, cultural trauma involves an assault by a dominant group on an individual's culture—through force, threats of force, or oppressive policies—for the purposes of damaging, devaluing, or destroying that culture to advance the dominant group's interests in gaining key resources (e.g., natural, labor) or status/reputation (e.g., colonial empires). Cultural trauma may overlap with interpersonal trauma, as in culturally motivated physical violence (e.g., genocide, hate crimes) toward members of a minority cultural group. But cultural trauma diverges from interpersonal trauma by accounting for how culturally motivated physical violence *and* non-physical assaults/stressors (e.g., racial discrimination, internment), driven by animus toward a group's culture vs. a specific individual, may generate lasting health disparities that impact future generations not exposed to the original cultural violence/assault.

We introduce this new cultural trauma conceptualization for two reasons. First, current clinical definitions of trauma do not match the reported trauma experiences of many minority groups (Andermahr, 2015; Evans-Campbell, 2008; Krieg, 2009), which include ongoing persecution, discrimination, and retraumatization (Craps, 2013; Krieg, 2009). Second, the empirical evidence indicating that cultural trauma impacts health (Hartmann and Gone, 2014; Krieg, 2009; Whitbeck et al., 2004) is distributed across different constructs (e.g., collective vs. historical vs. intergenerational trauma), each describing separate aspects of the trauma experience that are often specific to different cultural groups (Sotero, 2006). We contend this lack of a unifying definition or model has arguably concealed cultural trauma's true influence as a fundamental cause of health disparities.

3. Fundamental causes

According to fundamental cause theory, health disparities persist due to underlying social factors that disadvantage certain groups in accessing resources for protecting health and avoiding disease (Gee and Ford, 2011; Link and Phelan, 1995; Williams et al., 2019). As these resources can be used flexibly to avoid risks for, or reduce the impact of, multiple diseases across multiple situations, Phelan et al. (2010) labeled them 'flexible resources'.

To comprise a fundamental cause, a social factor must satisfy three criteria (Link and Phelan, 1995; Phelan et al., 2010). First, it must impact multiple health outcomes in a population through multiple risk factors. Consequently, to be considered a fundamental cause, cultural trauma must be linked to numerous health disparities as in the case of American Indians, whose traumatic displacement and ensuing cultural loss instigated multiple health disparities that persist to this day (e.g., heart disease, suicidality, alcohol use disorders) (Brave Heart et al., 2011; Gone et al., 2019; Subica and Wu, 2018).

Second, the proposed factor must embody access to flexible resources (e.g., knowledge, money, power, prestige, beneficial social connections, freedom) (Phelan et al., 2010; Phelan and Link, 2015). For instance, during the past century, cultural traumas in the form of discriminatory U.S. housing practices (e.g., redlining, mortgage foreclosure, blockbusting) served to maintain health disparities and economic deprivation in African American communities by barring individuals from accruing critical health-protective resources such as property and intergenerational wealth (Jackson, 1980; Mehlhorn, 1998; Saegert et al., 2011; Satter, 2009).

Third, the proposed factor's production of health disparities must be reinforced over time through multiple replaceable mechanisms (Phelan and Link, 2015). Continuing with our example, after explicit forms of cultural trauma toward African Americans (e.g., slavery, Jim Crow laws, lynching) were outlawed through policy changes, other forms of cultural trauma such as mass incarceration, housing discrimination, and police violence that "*transmit the legacy of the oppressive past of slavery and Jim Crow*" (Feagin, 2000, p. 145) were instituted to perpetuate African Americans' unequal access to cultural and other flexible resources (Saegert et al., 2011; Satter, 2009). Additionally, because health disparities generated by fundamental causes are rooted in inequities in flexible resources, health disparities cannot be eliminated by intervening on a specific disease or risk factor (Phelan et al., 2010).

To date, several fundamental causes—e.g., socioeconomic status (SES; Link and Phelan, 1995), stigma (Hatzenbuehler et al., 2013), racism/racial segregation (Phelan and Link, 2015; Williams and Collins, 2016)—have been codified in the literature. Herein, we present evidence and a theoretical model implicating cultural trauma as an unrecognized fundamental cause of health disparities; harming health by assaulting a group's essential cultural resources.

4. Cultural resources

Central to our cultural trauma model (Fig. 1) is the empirically

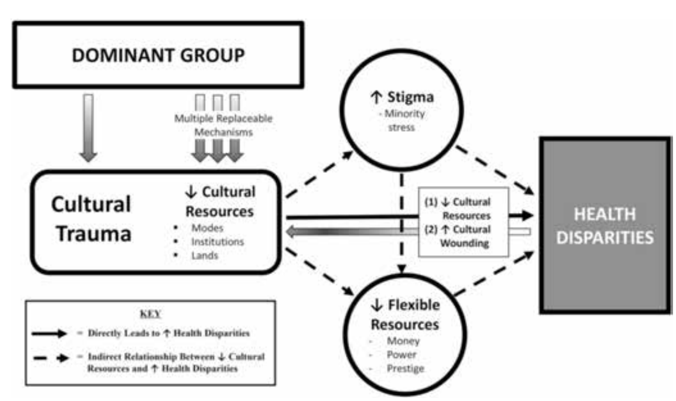

Fig. 1. Cultural trauma conceptual model.

grounded notion that culture represents an unrecognized flexible resource for health that is foundational to human survival. Accordingly, we identified from the literature three cultural resources that when disrupted via cultural trauma may trigger an intergenerational cascade of negative health outcomes. Like water to a fish, these cultural resources permeate human behavior and the social environment so deeply that they are often taken for granted. This paper closes this gap by merging existing research into a new model that recognizes three essential cultural resources for health: *cultural modes*, *institutions*, and *lands*.

4.1. Modes

Kitayama et al. (2007) define 'cultural modes of being' as a group's languages, norms, customs, values, and artifacts that construct both the internal and social worlds of group members. Modes are essential for healthy functioning as they organize and pattern one's thoughts, feelings, and actions, define one's place in the world relative to others (e.g., self-esteem, ethnic identity, cultural worldview), and frame one's social interactions to meet their basic, social, and health needs (Kitayama et al., 2007; Markus and Kitayama, 2010; Ryba et al., 2016). In essence, cultural modes encompass our ways of living, behaving, and experiencing the world to navigate society and fulfill our needs.

When cultural trauma damages a group's cultural modes, psychological studies have shown that healthy functioning can be disrupted as modes serve crucial psychological functions including protecting against stress and anxiety, promoting self-regulation, and facilitating effective adaptation/response to external stressors (Markus and Kitayama, 2010; Norris et al., 2002; Salzman, 2001; Savickas, 2005). For example, empirical studies of Terror Management Theory (Greenberg et al., 1986) strongly support the functioning of cultural modes as an important protective factor against anxiety (especially related to vulnerability and death), with the invalidation or loss of cultural modes having potentially devastating effects on individuals' self-esteem and anxiety (Greenberg et al., 1990, 1997; Pyszczynski et al., 2004). In addition, according to Evans-Campbell's (2008) historical trauma framework, the devaluing/destruction of cultural modes by a dominant group may profoundly shift cultural roles and identities, leading some survivors to experience elevated suicide, depression, substance use, chronic grief, and PTSD susceptibility (Brave Heart et al., 2011; Cook et al., 2003; Duran et al., 1998).

For instance, the Western colonization of Hawai'i wrought sweeping changes to Hawaiian social and cultural order through oppressive policies that destroyed native cultural modes including the banning of the Hawaiian language, prohibiting traditional spiritual ceremonies and rituals, and mixing sacred male and female work and living roles (Bushnell, 1993; Cook et al., 2003). According to Cook et al. (2003), this trauma to Hawaiian cultural modes generated cultural confusion and spiritual damage, which manifests as health disparities among present-day Native Hawaiians (Braun et al., 1995; Look and Braun, 1995; Mau et al., 2009).

4.2. Institutions

Institutions are the second cultural resource damaged by cultural trauma, and refer to the sociocultural systems—and policies upholding these systems—that establish social order in a society and govern people's behaviors and expectations (North, 1990). These systems cover all areas of social and community life and include family, economic, legal, educational, religious, political, and health systems (Miller, 2003; North, 1990). According to social status research, when individuals are afforded positive status within their cultural institutions, institutions protect against stress and support health (Demakakos et al., 2008; Hatzenbuehler et al., 2013; Sapolsky, 2004). But when institutions relegate individuals into lower statuses, institutions generate stress and derail health (Adler, 2009; Sapolsky, 2004). For example, Sapolsky's review of social status and health showed that forced subordination by dominant individuals within social hierarchies inhibits subordinates' health by imposing physical and psychological stressors *and* resource inequities on subordinates; which may be perpetuated through unequal institutionalized policies/actions including denial of flexible resources (Link and Phelan, 1995) and racism (Bailey et al., 2017; Williams et al., 2019).

Additionally, when a dominant group damages a group's cultural institutions via cultural trauma, survivors may be brought into the dominant group's institutions in subservient roles and status (Berry, 1989); exposing these individuals to potential acculturative stress, social disadvantage, and health-depriving discrimination and racism (Berry, 1989; Williams and Berry, 1991). For instance, after requiring Aboriginal groups to endure forced relocation, many Western nations consigned surviving children to residential schools to be "civilized" into Western institutions (e.g., family, religious, health systems); deepening poverty, unemployment, and ensuing health disparities such as tuberculosis, substance use, obesity, and early mortality in Aboriginal communities (Haskell and Randall, 2009; MacDonald and Steenbeek, 2015; Phillips-Beck et al., 2019; Walls and Whitbeck, 2012).

Additionally, research suggests systematic disadvantaging within cultural institutions may foster health disparities by fomenting racism; worsening health through multiple mechanisms including racial trauma (Comas-Díaz et al., 2019), social deprivation (Bailey et al., 2017), residential segregation (Williams and Collins, 2016), and unequal health care access (Flores, 2010; Smedley et al., 2003). For example, following extensive cultural trauma and disadvantaging in U.S. social, economic, educational, and criminal justice institutions, African Americans currently experience heavy disparities in morbidity and mortality (Cunningham et al., 2017; Huie et al., 2003; Orsi et al., 2010; Read et al., 2005; Williams et al., 2019).

4.3. Lands

Cultural lands is the third trauma-impacted resource, and refers to the material resources (e.g., physical property, housing, healthy foods, transportation, wealth) necessary to sustain health in a given society. As noted by scholars of colonialism and historical trauma, when a cultural group's lands gain a dominant group's interest, the dominant group may wield cultural trauma to dispossess the cultural group of its lands (Duran et al., 1998; Brave Heart and DeBruyn, 1998; Howe, 2002; Kohn and Reddy, 2006); potentially draining survivors of their health while restricting future opportunities to restore their cultural lands—and health—to pre-trauma levels (Evans-Campbell, 2008; Saegert et al., 2011).

Within the literature, this cultural trauma appears to occur both through physical dislocation from native lands via force, genocide, or disease (Evans-Campbell, 2008; Krieg, 2009), or discriminatory policies that strip groups of existing cultural lands (i.e., material resources) while blocking access to future lands and other flexible resources (Fullilove, 2001; Williams and Collins, 2016). For forcibly dislocated groups (e.g., Indigenous populations, refugees/asylum seekers), this traumatic displacement presents a massive stressor that impacts mental and physical health in the short-term and long-term (Bogic et al., 2015; Brave Heart and DeBruyn, 1998; Thomas and Thomas, 2004). For instance, American Indians' traumatic relocation from economically valuable lands to barren reservations (1) ruptured their cultural ways of life; (2) created a deep psychological injury and sense of traumatic loss; and (3) robbed them of health-protective flexible resources (Kirmayer et al., 2014; Walls and Whitbeck, 2012)—instigating a cultural genocide (Brave Heart et al., 2011) that underlies the glaring health disparities afflicting present-day American Indian communities (Jones, 2006; O'Connell et al., 2010; Subica et al., 2017).

Similarly, social psychiatrist Mindy Fullilove (2001) noted that the 1949 U.S. Urban Renewal Act-related dispossession of the lands of many flourishing Black communities intensified existing health disparities by

(1) generating prolonged stress, grief, and trauma-related symptoms in displaced residents (Fried, 1963); (2) segregating residents into areas of concentrated poverty with poor access to flexible resources (Williams and Collins, 2016); and (3) forcing residents to expend major financial capital to resettle, casting subsequent generations into economic disadvantage (Fullilove, 2001).

5. Multiple replaceable mechanisms

In fundamental cause theory, the mechanisms enacted by a dominant group to suppress access to flexible resources must be replaceable over time (Link and Phelan, 1995; Phelan and Link, 2015). More pointedly, when new opportunities to reinforce the effect of a fundamental cause on health disparities materialize, new mechanisms will reliably emerge to support or strengthen the relationship (Phelan and Link, 2015).

In cultural trauma, due to culture's dynamic nature and cultural groups' resilience in responding to cultural threats and oppression, we theorize dominant groups must continually implement 'multiple replaceable mechanisms' to maintain their advantage in flexible resources by quelling minority groups' efforts to reclaim their cultural resources and subsequent health (Fig. 1). As stated by Phelan et al. (2008), dominant groups achieve this by keeping other groups down (to exploit or dominate them), in (by enforcing dominant group cultural modes), or away (physically or socially distancing from cultural groups perceived as health threats).

There are multiple ways that dominant groups may prevent culturally traumatized groups from restoring or gaining needed cultural and other flexible resources. It can happen at the structural level through policies such as establishing American Indian boarding schools and reservations, or police surveillance of minority communities. It can happen at the interpersonal level via direct actions such as verbal threats, hate crimes, police violence, or discrimination in employment and housing. It can also be expressed by doing nothing when harm is experienced such as when low-income minority children suffer malnutrition and poor health due to food insecurity (Thomas et al., 2019) or American Indian women go missing or harmed while receiving minimal mainstream outcry or attention (Lucchesi and Echo-Hawk, 2018). What each mechanism achieves is a reinvigorated hierarchy in which the dominant group replaces older or failing mechanisms with new ones that maintain their ability to limit other groups from exerting their will and restoring their cultural and other flexible resources; keeping them down, in, or away. For example, following the repeal of policies that limited equal distribution of economic and social privileges to Asian Americans such as the Chinese Exclusion Act, anti-miscegenation laws, and the internment of Japanese Americans (Higginbotham and Kopytoff, 1988; Nagata, 1998; Soennichsen, 2011; Sohoni, 2007), new mechanisms of anti-Asian suppression and violence predictably emerged (Chang, 1998); gaining increased virulence and frequency during the COVID-19 era (Stop AAPI Hate, 2021).

6. Cultural trauma mechanisms

Fig. 1 displays three empirically supported mechanisms through which our model proposes cultural trauma influences health disparities. The first mechanism operates directly by damaging health-protective cultural resources and generating psychologically and physically harmful cultural wounding while the other mechanisms operate indirectly by stigmatizing traumatized groups with status loss and stereotyping, and denial/depletion of flexible resources, respectively. As our indirect mechanisms' influence on health disparities have been previously detailed in earlier papers by Hatzenbuehler et al. (2013) and Phelan et al. (2010), this paper will elucidate cultural trauma's role in exacerbating these mechanisms.

6.1. Direct mechanism: cultural resource loss and cultural wounding

By damaging a group's cultural resources (modes, institutions, lands), our model holds that cultural trauma denies members essential flexible resources that support and protect against stress, mental illness, and disease. As social scientists and Indigenous scholars have observed, damage to one's culture directly harms health by denying individuals vital culture-related health protective factors including (1) cultural/ethnic identity and pride (Crocker et al., 1994; Phinney and Ong, 2007); (2) cultural coping and healing strategies (Gone, 2013); and (3) supportive social, community, and family networks (Krieg, 2009; Norris et al., 2002).

Building on existing research, cultural trauma may also directly harm health by creating deep, intergenerational 'cultural wounding' (Evans-Campbell, 2008; Brave Heart and DeBruyn, 1998). Labeled a "soul wound" by Duran et al. (1998), this wounding has been characterized as a deep psychological injury that directly compromises people's sense of well-being, safety, self-regard, and coping. It is physically and emotionally destructive, manifesting in problems such as depression, anxiety, shared posttraumatic stress, and substance use (Brave Heart et al., 2011; Gone and Alcántara, 2007; Whitbeck et al., 2004) that lead to adverse health outcomes (e.g., diabetes, suicide, cancer) (Bezo and Maggi, 2018; Goodkind et al., 2012; Shrira et al., 2011). These wounds may be felt collectively across generations, impacting offspring through exposure to daily reminders of the cultural trauma including discrimination and loss of traditional languages, family systems, and spiritual/healing practices (Evans-Campbell, 2008; Subica and Wu, 2018; Whitbeck et al., 2004).

6.2. Indirect mechanisms: stigma and flexible resources

Our model further proposes that cultural trauma may affect health disparities via two indirect mechanisms (1) stigmatization of affected groups; and (2) decreased access to flexible resources.

Stigma is the labeling, stereotyping, separation, status loss, and discrimination of disadvantaged groups by those in power to achieve their desired goals (Link and Phelan, 2001). In our model (Fig. 1), we assert that dominant groups wield stigma to maintain dominance over minority groups' cultural and other flexible resources. According to Link and Phelan's (2001) theories on stigma, the dominant group may exercise stigma using multiple mechanisms such as anti-immigrant media and messaging (Morey, 2018) and resource-reducing discrimination in employment, housing, and education (Hatzenbuehler et al., 2013) to marginalize minority groups with status loss and negative labels/stereotypes (Link and Phelan, 2001). A vivid example is the descriptive labeling/othering of certain minority groups in Western countries as "immigrants," "refugees," "queer," or "terrorists" vs. their appropriate cultural affiliations (e.g., Latin American, sexual minority, Muslim).

From the psychological literature, we further posit dominant groups are motivated to wield stigma to justify their cultural trauma by propagating narratives that denigrate, blame, or cast as inferior affected groups—mimicking perpetrators' use of "victim-blaming" attributions to justify traumatizing behaviors (Harsey et al., 2017; Henning et al., 2005). Noteworthy dominant group justifications for inflicting cultural trauma in the literature include discriminatory narratives such as "We're civilizing the savages" by European colonists (Wallace, 2010), "We're the superior race" by the Third Reich (Tenenbaum, 1956), and "They don't belong here" against immigrant populations (Morey, 2018). Following stigma exposure, individuals may experience minority stress—a stigma-related chronic, additive stress (on top of general life stressors) associated with reduced mental and physical health (Hatzenbuehler et al., 2013; Meyer, 2003) in stigmatized cultural groups such as sexual minorities (Hatzenbuehler, 2009; Operario et al., 2015).

In our model's second indirect mechanism, cultural trauma may perpetuate health disparities by decreasing affected groups' access to flexible resources (e.g., money, power, prestige) (Link and Phelan, 1995;

Phelan and Link, 2015) through (1) cultural resource loss; and (2) stigma-related minority stress and disadvantaging. For example, the culturally traumatic denial of marriage rights (a valued cultural institution) to same sex couples denied millions of sexual minorities access to important marriage-related flexible resources (e.g., employer-sponsored health insurance, partner-inherited wealth) (Encarnación, 2014; Gonzales and Blewett, 2014; Perone, 2015).

7. Intergenerational cultural trauma

The intergenerational transmission of cultural trauma has been clearly observed in multiple populations including Holocaust survivors (Yehuda et al., 2008), refugees (Field et al., 2013; Sangalang and Vang, 2017), combat veterans (Dekel and Goldblatt, 2008), and Indigenous populations (Brave Heart, 2003; Kirmayer et al., 2014). Extensive research has highlighted several mechanisms through which intergenerational trauma transmission may affect health disparities including impaired parenting and parental modeling of maladaptive post-traumatic behavioral patterns (Auerhahn and Laub, 1998; Brave Heart, 2003; Krieg, 2009), secondary traumatization through transmission of trauma narratives to offspring (Brave Heart et al., 2011; Whitbeck et al., 2004), and potential epigenetic changes that increase susceptibility to mental and physical disorders/diseases (e.g., depression, metabolic syndrome, cardiovascular disease)(Bierer et al., 2014; Darity et al., 2001; Heckman and Payner, 1989; Yehuda et al., 2016).

Supported by this evidence, our model proposes that health disparities caused by cultural trauma are intergenerationally transmitted through the passing down of (1) cultural wounding and loss; (2) social disadvantage; and (3) biological vulnerabilities to disorders/diseases. These risk factors then increase members' exposure to persistent stress, stigma, racism, and diseases, which accumulate over time to perpetuate health disparities (Gone et al., 2019; Sotero, 2006).

8. Interventions

Several intervention approaches may play a role in addressing health disparities by targeting cultural trauma through direct intervention or policy change. The first seeks to restore damaged cultural modes via cultural/legacy interventions (Brave Heart et al., 2011; Gone and Alcántara, 2007; University of Calgary, 2012) that build cultural identity, pride, and knowledge, and facilitate cultural healing through racial socialization, traditional practices (e.g., diets, ceremonies), and cultural education (Anderson and Stevenson, 2019; Cook et al., 2003; Gone, 2013). An excellent example is Australia's Yiriman Project where Aboriginal elders take at-risk youth on 'back-to-country' walking trips containing cultural activities (e.g., fishing, land management, storytelling) to reinvigorate lost Aboriginal culture, lore, and identity (Palmer et al., 2020). Additionally, resilience interventions engaging at-risk Black youth in Africentric practices and rites of passages have led to increased self-esteem, ethnic identity, and decreased high-risk behaviors (e.g., substance use, aggression/violence) (Belgrave et al., 2004; Harvey and Hill, 2004; Jackson et al., 2010) while *Aloha ʻĀina* (caring for the land) interventions have strengthened cultural identity and self-esteem in Native Hawaiian youth (Mokuau, 2011).

A second approach employs community mobilization/capacity building interventions to restore groups' cultural resources and protective factors (e.g., community networks, collective efficacy) (Sotero, 2006). One promising example is community organizing-based health promotion (Subica, Grills, Douglas, et al., 2016), which engages and mobilizes disadvantaged residents to rectify community inequities in flexible resources by building community power to advocate for health-promoting policy changes to the social and built environment (Subica, Grills, Villanueva, et al., 2016). Increasing community power via grassroots interventions may also counteract the social paralysis/political disengagement of many traumatized groups that may sustain inequities in flexible resources and health over time (Degagné, 2007; Fullilove, 2001).

Unfortunately, targeting cultural trauma using these therapeutic and structural approaches alone places the onus on the *oppressed* to "fix the problems" created by dominant groups' traumatizing behaviors. Therefore, approaches should also intervene on *oppressors* through policy interventions that challenge the systems of domination/power that maintain health disparities through the suppression of cultural and other flexible resources. Examples include restoring disrupted cultural modes and institutions by establishing trusts and educational systems that uplift minority cultures/languages, as in Hawai'i's Kamehameha Schools that was formed in 1887 to counteract the severe socioeconomic and educational disadvantages facing Native Hawaiians (Serrano et al., 2007). To promote access to cultural lands and other flexible resources, policy interventions may also take the form of equality generators such as baby bonds (Hamilton and Darity, 2010; Zewde, 2020), universal basic income (Haagh, 2019; Hoynes and Rothstein, 2019), or reparations for culturally traumatized populations including Jewish Holocaust survivors (Ludi, 2012), the Indigenous Sámi of Europe (Errico and Hocking, 2012), Japanese Americans (Howard-Hassmann, 2004), and Africans (Spitzer, 2002).

Finally, an important intervention approach involves direct public actions (e.g., protests) that raise the dominant group's awareness of the harmful effects of ongoing traumas (e.g., racially-biased police violence) (Edwards et al., 2019; Nix et al., 2017; Williamson et al., 2018). This policy-focused approach is exemplified by the global Black Lives Matters movement (Jee-Lyn García and Sharif, 2015), which in disrupting existing law enforcement and political systems of domination has forced a reckoning and deeper understanding among many dominant group members of the entrenched systemic problems and oppressive policies (e.g., racism, police violence) (Political Polling U.S., 2020) that contribute to illness and death in many cultural populations.

9. Conclusion

Fundamental cause theory explains health disparities' persistence over time despite major changes in risk factors and diseases (Phelan et al., 2010). Presented evidence indicates cultural trauma meets all fundamental cause criteria, profoundly shaping the intergenerational social and health trajectory of affected populations by (1) impacting multiple mental and physical health outcomes through multiple mechanisms (e.g., cultural wounding, stigma, biological vulnerabilities); (2) restricting access to health-protective cultural and other flexible resources; and (3) reproducing health disparities over time through multiple replaceable mechanisms.

At the same time, we do not consider cultural trauma to be the sole driver or root cause of health disparities, as cultural trauma often overlaps and interacts with other fundamental causes (e.g., racism, stigma, socioeconomic status) and social or environmental factors (e.g., inadequate healthcare access, poor air and water quality) in a complex, synergistic fashion to produce and maintain health disparities over time. Notably, there may be cases where cultural trauma overlaps with structural racism—i.e., race-based discrimination practiced by a dominant group within racialized social systems (Bonilla-Silva, 1997). However, two key points serve to differentiate cultural trauma from structural racism. First, while racism is inherently race-based, cultural trauma is culture-based and can therefore account for the occurrence of health disparities in cultural groups affiliated by shared identities other than race such as sexual orientation, gender identity, immigration status, religion, and physical and psychiatric disability. Second, for cultural trauma to reflect a fundamental cause, it must (a) involve a clearly identifiable physical or psychological assault/stressor; (b) be produced by a dominant group motivated to achieve its desired ends (e.g., material resources, status); and (c) directly impact health by damaging the affected group's cultural modes, institutions, or lands. In contrast, racism (a) may be more insidious than cultural trauma and does not need to involve an overt assault/stressor (e.g., racial microaggressions); and (b) is caused by a dominant group primarily motivated by an ideology of inferiority, with the dominant group (c) exerting its power and control over resources by categorizing and ranking other people they perceive as inferior into social groups by "race" (Bonilla-Silva, 1997; Williams et al., 2019; Williams and Mohammed, 2013).

As a new model, limitations exist that should be addressed in future studies. First, because our goal was to introduce cultural trauma's influence on health disparities from a fundamental cause perspective, we could not review many of the biological, psychosocial, and existential effects/processes reported in the literature on collective, historical, and racial traumas. Second, we presented our model as a generalized theory of cultural trauma in order to allow others to tailor it to the unique trauma experiences and outcomes of specific groups in ways that we could not. We encourage researchers to test, challenge, and improve our model so as to better measure and apprehend the full impact of cultural trauma on diverse groups. We particularly encourage individuals to consider ways to conceptualize and assess the deleterious effects of cultural trauma not only on larger cultural groups, but also subcultures within these groups that may be differentially affected by cultural traumas. Yet, despite these limitations, we believe our model may offer researchers new insights into why health disparities persist across time, place, and generations, and potentially inform future studies and interventions to reduce health disparities.

In closing, our paper suggests that cultural trauma may be an unrecognized fundamental cause of health disparities for the numerous cultural groups worldwide that have endured traumatic assaults on their cultural modes, institutions, or lands. Therefore, reducing health disparities may require a multipronged approach focused on restoring access to cultural/flexible resources while also healing these traumas' intergenerational physical and psychological consequences. However, because oppressive mechanisms are replaceable, eradicating health disparities may require enacting policies designed to curtail dominant groups' ability to exploit and control the resources of others to prevent cultural trauma and advance health equity for all groups.

Author contributions

AS conducted the search, review, and analysis of the literature and conceptualized the initial cultural trauma theory and framework. BL advised on and helped refine subsequent cultural trauma models. AS wrote the first draft of the article and AS and BL refined subsequent drafts. Both AS and BL approved the final article.

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Review article

Application of the navigation guide systematic review methodology to evaluate prenatal exposure to particulate matter air pollution and infant birth weight

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Meta-analysis

A B S T R A C T

Low birth weight is an important risk factor for many co-morbidities both in early life as well as in adulthood. Numerous studies report associations between prenatal exposure to particulate matter (PM) air pollution and low birth weight. Previous systematic reviews and meta-analyses report varying effect sizes and significant heterogeneity between studies, but did not systematically evaluate the quality of individual studies or the overall body of evidence. We conducted a new systematic review to determine how prenatal exposure to $PM_{2.5}$, PM_{10}, and coarse PM ($PM_{2.5\text{-}10}$) by trimester and across pregnancy affects infant birth weight. Using the Navigation Guide methodology, we developed and applied a systematic review protocol [CRD42017058805] that included a comprehensive search of the epidemiological literature, risk of bias (ROB) determination, meta-analysis, and evidence evaluation, all using pre-established criteria. In total, 53 studies met our inclusion criteria, which included evaluation of birth weight as a continuous variable. For $PM_{2.5}$ and PM_{10}, we restricted meta-analyses to studies determined overall as "low" or "probably low" ROB; none of the studies evaluating coarse PM were rated as "low" or "probably low" risk of bias, so all studies were used. For $PM_{2.5}$, we observed that for every 10 μg/m^3 increase in exposure to $PM_{2.5}$ in the 2nd or 3rd trimester, respectively, there was an associated 5.69 g decrease (I^2: 68%, 95% CI: −10.58, −0.79) or 10.67 g decrease in birth weight (I^2: 84%, 95% CI: −20.91, −0.43). Over the entire pregnancy, for every 10 μg/m^3 increase in $PM_{2.5}$ exposure, there was an associated 27.55 g decrease in birth weight (I^2: 94%, 95% CI: −48.45, −6.65). However, the quality of evidence for $PM_{2.5}$ was rated as "low" due to imprecision and/or unexplained heterogeneity among different studies. For PM_{10}, we observed that for every 10 μg/m^3 increase in exposure in the 3rd trimester or the entire pregnancy, there was a 6.57 g decrease (I^2: 0%, 95% CI: −10.66, −2.48) or 8.65 g decrease in birth weight (I^2: 84%, 95% CI: −16.83, −0.48), respectively. The quality of evidence for PM_{10} was rated as "moderate," as heterogeneity was either absent or could be explained. The quality of evidence for coarse PM was rated as very low/low (for risk of bias and imprecision). Overall, while evidence for $PM_{2.5}$ and course PM was inadequate primarily due to heterogeneity and risk of bias, respectively, our results support the existence of an inverse association between prenatal PM_{10} exposure and low birth weight.

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1. Introduction

Prenatal exposure to particulate matter (PM) air pollution has been linked with adverse birth outcomes, namely infant low birth weight (LBW) (Dadvand et al. 2013). A number of studies have investigated the association between prenatal PM exposure and infant LBW, which is attributable to the regular collection and large-scale availability of birth weight data through birth records (Blencowe et al. 2019). Additionally, LBW has been associated with an increased risk of certain long-term health outcomes later in life (Belbasis et al. 2016). LBW is defined as infants born weighing<2,500 g (Cutland et al. 2017). A primary cause of LBW is preterm birth (PTB), delivery of a live born infant < 37 weeks of gestation (Cutland et al. 2017). Thus, mean birthweights across gestational age ranges has been used as a proxy for fetal growth. Specifically, small for gestational age (SGA) is defined as infants that fall within the smallest 10th percentile of infants of the same gestational age (Cutland et al. 2017). A subset of LBW and SGA infants require intensive neonatal care for immediate health issues and may have chronic health outcomes later in life (Belbasis et al. 2016).

In spite of the substantial evidence on the association between developmental PM exposure to PM and outcomes, including LBW, PTB, and SGA births, there have been inconsistencies in the conclusions on the magnitude of the effect (Lamichhane et al. 2015; Stieb et al. 2012). We applied the Navigation Guide systematic review methodology to assess the quality and strength of evidence on the effect of prenatal PM exposure on infant birth weight. The Navigation Guide was developed in 2011 to strengthen approaches for assessing evidence in environmental health sciences (Woodruff et al. 2011). The Navigation Guide is a systematic and transparent approach that draws from best practices in the clinical arena while accounting for differences in evidence and decision context involved in environmental health risk assessments, such as the reliance on human observational studies versus randomized controlled trials (Cumpston et al. 2019; Guyatt et al. 2008; Woodruff and Sutton 2014). To date, the Navigation Guide methodology has been applied in numerous reviews of environmental exposures, including the human evidence for effects of airborne pollutants on the diagnosis of autism spectrum disorder (Lam et al. 2016) and both the human and non-human evidence for effects of Perfluorooctanoic acids (PFOAs) on fetal growth (Johnson et al. 2014; Koustas et al. 2014; Lam et al. 2014). The results of these studies and others demonstrate the utility of this approach in applying rigor and transparency in support of evidence-based decisions to environmental health problems.

In this review, we evaluated the human evidence regarding prenatal PM exposure and infant birth weight. We assessed each study for the risk of bias and conducted a *meta*-analysis on a subset of studies to estimate the overall magnitude of effect. We focused on birth weight as a continuous outcome variable to determine the impact of bias on effect size estimates. Consideration as a continuous variable allows for the assessment of the effect on population distributions. Case studies have illustrated the importance of considering a continuous scale to provide added information about the exposure-disease continuum, inform population variability, and increase the predictive power of risk assessment (Woodruff et al. 2008).

2. Methods

2.1. Systematic review methodology

While systematic review methods have been used for decades in the clinical sciences, specific techniques for conducting a systematic review directly applicable to the decision context and evidence streams in environmental health have only recently been developed and utilized in the field of environmental health sciences (Rooney et al. 2014; Woodruff and Sutton 2014). We conducted our review using the Navigation Guide approach, which is based on the Cochrane Collaboration and Grading of Recommendations Assessment Development and Evaluation (Guyatt et al. 2008). We developed a protocol before initiating the study and registered it in PROSPERO [CRD42017058805].

2.2. Study question

Our ultimate objective was to evaluate whether ambient air pollution is "toxic" to the developing fetus in the sense of reducing birth weight with increasing exposure, since lower birth weight is a risk factor for both short- and long-term morbidities (Belbasis et al. 2016). The "Population," "Exposure," "Comparator," and "Outcome" (PECO) statement, is briefly outlined below with additional specifics available in our protocol. *Population*: Pregnant women. *Exposure*: Gestational exposure to ambient particulate air pollution. "Particulate air pollution" is defined as outdoor sources of inhaled airborne matter classified as $PM_{2.5}$ (mass concentration of particles with diameter smaller than 2.5 μm), PM_{10} (mass concentration of particles with diameter smaller than 10 μm), or $PM_{2.5\text{-}10}$ (mass concentration of particles with diameters 2.5–10 μm), excluding active and passive smoking. *Comparator*: Pregnant women exposed to lower levels of PM than the more highly exposed humans. *Outcome*: Birth weight measured as a continuous variable.

2.3. Data sources

We searched the databases Ovid Medline, Embase, and Global Health on November 23, 2015, using the search terms developed in collaboration with librarian (MF), shown in the Supplemental Materials, Table S1. Our search was not limited by publication date. We limited our search to English language and used the Medical Subject Headings (MeSH) database to compile synonyms for ambient particulate air pollution and birth weight (details in our protocol). We updated the search on February 27, 2020, to identify any new studies, applying the same strategies used in the original search. We also supplemented these results by hand-searching references of all included studies.

2.4. Study selection

We included original studies that evaluated ambient particulate air exposure and reported associations with birth weight. Three reviewers (MM, JP, IU) independently screened titles and abstracts of each reference in RefWorks to determine eligibility. In the event of a discrepancy between reviewers, the default was to move the article forward for full-text screening. We excluded studies if: 1) the article did not report birth weight outcomes; 2) the article did not report ambient particulate air pollution exposure; 3) the article contained no original data; 4) the article did not involve human subjects; or 5) other reason, with an explanation required. All duplicate articles were removed. At the full-text screening stage, the same reviewers (MM, JP, IU) independently screened references in RefWorks for inclusion using the same criteria as above. Additionally, at this stage, studies were excluded if the article did not report birth weight as a continuous variable. Studies reporting birth weight as z-scores were excluded.

2.5. Data extraction

Two reviewers (NO, AF) independently extracted data related to study characteristics and outcome measures into the Health Assessment Workspace Collaborative (HAWC) database (Supplemental Materials, Table S2). In the case of missing data, the protocol was to contact study authors; however, all relevant data was able to be extracted from the full text articles. Data extracted by each author was independently reviewed (WC, NMJ, IU) for quality assurance/quality control on all the studies to resolve any discrepancies between the two independent extractors and further ensure accuracy. We extracted all characteristics of the study population, including location and sample size, exposure period duration, pollutant class, methods used to estimate exposures, and all relevant estimates of association relating particulate air pollution exposure with birth weight, specifically recording estimates as related to exposure assessment technique or by spatial scale (i.e., city- or county-level versus < 5 km radius). For the *meta*-analysis, we extracted adjusted regression estimates and standard errors or 95% confidence interval limits and standardized to a continuous increment in exposure (i.e., per 10 $\mu g/m^3$ unit increase in pollutant). For instance, if change in birthweight was originally reported in grams per 1 ug/m^3 exposure, the effect and confidence interval limits were multiplied by 10.

Some studies reported the change in birthweight per IQR increase of exposure. For these the values were standardized by multiplying the (change in birthweight per IQR) by (10 ug/m^3 divided by the value of the IQR). For studies where a 95% confidence interval was not reported one was calculated from available p-values or standard errors assuming a normal distribution. For articles reporting multiple models adjusting for different sets of covariates, we selected estimates from the fully-adjusted model including the most confounders.

2.6. Assessing the risk of bias

We evaluated the risk of bias for each of the studies across the following domains: recruitment strategy, blinding, confounding, exposure assessment, incomplete outcome data, selective outcome reporting, conflicts of interest, or other problems that could put the study at risk of bias (Table 1). Ratings for each domain were "low," "probably low," "probably high," or "high" risk of bias, with customized instructions for each domain based on the type of evidence anticipated (Supplemental Materials, Table S3). For example, we determined for a study to be rated "low" risk of bias in the confounding domain, all five pre-determined potential confounders were accounted for. These included socioeconomic status, race/ethnicity, maternal tobacco use, maternal age, and season of conception/birth. Likewise, to determine if exposure assessment measurements were robust, reviewers took into consideration the validity and reliability of the monitoring or modeling methods employed. Review authors with subject-matter expertise from our team (NMJ, JL, XX, BT, MM, IU, ST, WC) independently determined the risk of bias across all domains. An additional QA/QC author was matched with each study to solve any discrepancies between ratings. An overall risk of bias rating was assigned as "low," "probably low," "probably high," or "high" risk of bias by evaluating the individual domain ratings. If any of the ratings were "high" or "probably high," the overall rating was automatically rated as "high" or "probably high," respectively. If the majority of domains were rated as "low" or "probably low," the overall rating was determined to be "low" or "probably low," respectively.

Table 1
Summary of risk of bias domains and criteria for low risk designation.

Risk of bias domain	Low risk of bias designation[a]
Recruitment strategy	Protocols for recruitment and inclusion/exclusion criteria applied similarly across study groups
Blinding	Knowledge of the exposure ensured when assessing outcome, or judgement that outcome measurement not likely to be influenced by lack of blinding
Exposure assessment	Confidence in the accuracy of the exposure assessment methods that minimizes exposure misclassification, i.e., validity and reliability measures specified for monitoring and modeling
Confounding	All five important potential confounders pre-specified by reviewers are accounted for (i.e., matched, stratified, multivariate analysis or otherwise statistically controlled for)
Incomplete outcome	No missing outcome data, balanced attrition across groups, or for continuous outcome data, plausible effect size among missing outcomes not enough to have a relevant impact on the observed effect size
Selective outcome reporting	All pre-specified outcomes outlined in the protocol, methods, abstract, and/or introduction reported in the pre-specified way
Conflicts of Interest	The study did not receive support from a company, study author, or other entity having a financial interest in the outcome of the study
Other bias	The study appears to be free of other sources of bias

[a] The complete criteria for determining risk of bias designations for individual studies are provided in Supplemental Material Table S3, "Instructions for Making Risk of Bias Determinations."

2.7. Meta-analysis

Details of the *meta*-analysis approach are in the study protocol. In brief, the analysis separately considered each of the three pollutant classes of $PM_{2.5}$, PM_{10}, and $PM_{2.5\text{-}10}$ and the four exposure windows, first, second, and third trimester, as well as entire pregnancy. The primary analysis utilized study results for the entire population in each study, using the exposure metric at the smallest spatial scale, analyzed using single pollutant models, adjusted for covariates. Additionally, due to the sufficient number of studies, the primary analyses for $PM_{2.5}$ and PM_{10} utilized only studies with "low" or "probably low" risk of bias. Studies were pooled using random-effects models with the Knapp-Hartung modification (Knapp and Hartung 2003). This approach accounts for uncertainty in the estimate of τ^2 in the standard error estimates, generally resulting in wider confidence intervals. Heterogeneity was evaluated using the I^2 metric. Sources of heterogeneity explored using subgrouping included the following: ethnicity (non-Hispanic White only, Hispanic only, Black only), geographic locale (Americas, Europe, Asia), spatial scale of exposure assessment, and risk of bias rating. Additionally, influence analysis was conducted by removing individual studies one at a time.

2.8. Rating the quality of evidence across studies

We rated the quality of the overall body of evidence as "high," "moderate," "low," or "very low." An initial rating of "moderate" quality was assigned based on the previously described rationale for rating human evidence according to the Navigation Guide approach (Johnson et al. 2014). We considered "downgrades" to the quality rating based on five categories of considerations: risk of bias, indirectness, inconsistency, imprecision, and potential for publication bias (Table 3). We considered "upgrades" to the quality rating due to a large magnitude of effect, dose–response, and whether residual confounding would minimize the overall effect estimate (Balshem et al. 2011). Possible downgrades or upgrades were: 0 (no change from initial quality rating), −1 (1 level downgrade) or − 2 (2 level downgrade), +1 (1 level upgrade) or + 2 (2 level upgrade). Review authors evaluated the quality of the evidence according to our protocol (Supplemental Materials, Table S4) and then compared ratings as a group to reach the final decision.

2.9. Rating the strength of the evidence across studies

We assigned an overall strength of evidence rating based on a combination of 4 considerations, outlined in Table 3 and detailed in Supplemental Materials, Table S4: (1) Quality of body of evidence (i.e., the rating from the previous step), (2) Direction of effect, (3) Confidence in effect (likelihood that a new study could change our conclusion), and (4) Other compelling attributes of the data that may influence certainty. Possible ratings were "sufficient evidence," "limited evidence," "inadequate evidence," or "evidence of lack of toxicity."

3. Results

3.1. Included studies

Fig. 1A depicts the screening of eligible articles: the original November 2015 search retrieved 532 unique records, of which 103 were screened at the full-text review stage. Of these, 32 met our pre-defined criteria for inclusion. Fig. 1B illustrates the February 2020 search which retrieved 223 additional studies, of which 50 were screened at

Table 2
Summary of study characteristics for studies included in the *meta*-analysis

Reference	Study location	Study design	Sample size	Pollutant(s) (exposure assessment method)	Exposure period	Overall ROB rating
(Basu et al. 2014)	California, USA (8 counties)	Cohort R	646,296	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Beland and Oloomi 2019)	southern USA	Cohort R	9,324,839	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy	Probably low
(Bell et al. 2007)	Connecticut and Massachusetts, USA	Cohort R	358,504	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy	Probably low
(Bell et al. 2010)	Connecticut and Massachusetts, USA (4 counties)	Cohort R	76,788	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Bijnens et al. 2016)	Flanders, Belgium	Cohort R	4,760	PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters, last month, last week	Probably high
(Darrow et al. 2011)	Atlanta, USA(5 Counties)	Cohort R	402, 627	$PM_{2.5}$, PM_{10}, $PM_{2.5\text{-}10}$ (Ambient monitoring)	Entire pregnancy, 3rd trimester only	Probably high
(Ebisu et al. 2016)	USA (224 Counties)	Cohort R	8,017,865	$PM_{2.5\text{-}10}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Erickson et al. 2016)	British Columbia, Canada	Cohort R	231,929	$PM_{2.5}$ (Modeling)	Entire pregnancy	Probably high
(Fong et al. 2019)	Massachusetts, USA	Cohort R	907,766	$PM_{2.5}$ (Modeling)	Entire pregnancy	Probably low
(Geer et al. 2012)	Texas, USA	Cohort R	1,548,904	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy	Probably low
(Giovannini et al. 2018)	Italy	Cohort R	3,614	PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	High
(Gouveia et al. 2004)	São Paulo, Brazil	Cross-sectional	179,460	PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Gray et al. 2010)	North Carolina, USA	Cohort R	350,754	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Gray et al. 2014)	North Carolina, USA	Cohort R	457, 642	$PM_{2.5}$ (Modeling)	Entire pregnancy	Probably low
(Guo et al. 2020)	Guangdong province, China	Cohort R	2,567,457	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy	Probably high
(Han et al. 2018)	Suzhou, China	Cohort R	10,915	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Hannam et al. 2014)	United Kingdom (Northwest England)	Cohort R	203,562	$PM_{2.5}$, PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(He et al. 2018)	Zhengzhou, China	Cohort P	591	PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Huang et al. 2015)	Beijing, China	Cohort R	50,874	PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	Probably high
(Hyder et al. 2014)	Connecticut and Massachusetts, USA	Cohort R	834,332	$PM_{2.5}$ (Ambient monitoring and modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Jedrychowski et al. 2009)	Krakow, Poland	Cohort P	481	$PM_{2.5}$ (Personal monitoring)	Entire pregnancy	Low
(Keller et al. 2017)	Georgia, USA	Cohort R	403,881	$PM_{2.5}$ (Modeling)	1st, 2nd' and 3rd trimesters	Probably low
(Kim et al. 2007)	Seoul, Korea	Cohort P	1,514	PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	Probably low
(Kirwa et al. 2019)	Puerto Rico	Cohort R	332,129	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy	Probably high
(Kumar 2012)	Chicago, USA	Cohort R	400,000	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Lamichhane et al. 2018)	South Korea	Cohort P	648	PM_{10} (Modeling)	1st, 2nd' and 3rd trimesters	Probably low
(Laurent et al. 2013)	California, USA (2 counties)	Cohort R	105,092	$PM_{2.5}$, PM_{10} (Ambient monitoring and modeling)	Entire pregnancy	High
(Lavigne et al. 2018)	Ontario, Canada	Cohort R	196,171	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Li et al. 2019)	Ningbo, China	Cohort R	170,008	$PM_{2.5}$, PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Mannes et al. 2005)	Sydney, Australia	Cohort R	138,056	$PM_{2.5}$, PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	Probably high
(Medeiros and Gouveia 2005)	São Paulo, Brazil	Cohort R	311,735	PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	High
(Merklinger-Gruchala and Kapiszewska 2015)	Krakow, Poland	Cohort R	84,842	PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Morello-Frosch et al. 2010)	California, USA	Cohort R	3,545,177	$PM_{2.5}$, PM_{10}, $PM_{2.5\text{-}10}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Parker and Woodruff 2008)	USA (excluding Alaska and Hawaii)	Cohort R	785,965	$PM_{2.5}$, $PM_{2.5\text{-}10}$ (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Parker et al. 2005)	California, USA	Cohort R	18,247	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy	Probably high
(Pedersen et al. 2013)	12 European countries	Cohort P	74,178	$PM_{2.5}$, PM_{10}, $PM_{2.5\text{-}10}$ (Modeling)	Entire pregnancy	High
(Rahmalia et al. 2012)	Poiters and Nancy, France	Cohort P	888	PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Rhee et al. 2019)	Boston, USA	Cohort R	3,366	$PM_{2.5}$ (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low

(*continued on next page*)

Table 2 (*continued*)

Reference	Study location	Study design	Sample size	Pollutant(s) (exposure assessment method)	Exposure period	Overall ROB rating
(Salam et al. 2005)	California, USA	Cohort R	3,901	PM_{10} (Ambient monitoring)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Santos Vde et al. 2014)	São José dos Campos, Brazil	Cross-sectional	21,591	PM_{10} (Ambient monitoring)	3rd trimester only	High
(Savitz et al. 2014)	New York, USA	Cohort R	252,967	$PM_{2.5}$ (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Schembari et al. 2015)	Bradford, United Kingdom	Cohort P	9,067	$PM_{2.5,}$ PM_{10} (Modeling)	Entire pregnancy, 3rd trimester only	Probably low
(Schwarz et al. 2019)	California, USA	Cohort R	2,768,898	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy	Probably low
(Sellier et al. 2014)	Poiters and Nancy, France	Cohort P	1,026	PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Stieb et al. 2016)	Canada	Cohort R	2,781,940	$PM_{2.5}$ (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(van den Hooven et al. 2012)	Netherlands	Cohort P	7,772	PM_{10} (Modeling)	Entire pregnancy	Probably low
(Vinikoor-Imler et al. 2014)	North Carolina, USA	Cohort R	322,981	$PM_{2.5}$ (Modeling)	1st, 2nd' and 3rd trimesters	Probably low
(Winckelmans et al. 2015)	Flanders, Belgium	Cohort R	525,635	PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Xiao et al. 2018)	Shanghai, China	Cohort R	132,783	$PM_{2.5}$ (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low
(Xue et al. 2018)	USA	Cohort R	18,317,707	$PM_{2.5}$ (Ambient monitoring)	Entire pregnancy	High
(Yang et al. 2003)	Kaohsiung, Taiwan	Cohort R	13,396	PM_{10} (Ambient monitoring)	1st, 2nd' and 3rd trimesters	Probably high
(Ye et al. 2018)	Taizhou, China	Cohort R	24,246	PM_{10} (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably high
(Yuan et al. 2020)	Shanghai, China	Cohort R	3,692	$PM_{2.5}$ (Modeling)	Entire pregnancy, 1st, 2nd' and 3rd trimesters	Probably low

R: retrospective cohort; P: prospective cohort

Table 3
Factors for evaluating the quality and strength of the body of evidence

Quality is rated across all studies. Evidence begins as "moderate" and may be downgraded (-1 or −2) or upgraded (+1 or + 2) according to factors.		Strength is rated across all studies. The final ratings represent the level of certainty of toxicity.	
Downgrade factors	Risk of bias across studiesIndirectnessInconsistencyImprecisionPublication bias	Considerations	Quality of body of evidenceDirection of effect estimatesConfidence in effect estimatesOther compelling attributes of the data that may influence certainty
Upgrade factors	Large magnitude of effectDose responseConfounding minimizes effect		
Quality rating	• High quality • Moderate quality • Low quality • Very low quality	**Strength rating**	• Sufficient evidence • Limited evidence • Inadequate evidence • Evidence of lack of toxicity

full-text review stage and 21 studies met our pre-defined criteria for inclusion into the final analysis, totaling 53 articles. A summary of the characteristics of these studies is detailed in Table 2. The included studies were largely cohort studies, with 44 using similar retrospective methods to investigate the relationship between air pollution and birth weight. Nine of the studies used prospective methods, enrolling pregnant mothers and collecting information to determine air pollution exposure during pregnancy. Studies varied in the pollutant measured, type of exposure assessment method, and exposure window (i.e., entire pregnancy or trimester specific) reported. Overall, 20 studies measured $PM_{2.5}$ exposure alone, 17 studies measured PM_{10} exposure alone, and only 1 study measured $PM_{2.5\text{-}10}$ alone. Several studies measured pollutants in combination, either all three (3 studies) or two of the three pollutant classes (12 studies). Exposure assessment methods included ambient monitoring as the primary technique (30 studies), followed by modeling (20 studies), a combination of monitoring and modeling (2 studies) or in one case personal modeling for a 48 h duration in the second trimester of pregnancy. In general, studies reported effect estimates for trimester-specific and entire pregnancy exposure windows (28 studies). In some cases, only estimates were reported for the entire pregnancy and not by trimester (15 studies) or just by trimester and not entire pregnancy (10 studies). Study locations ranged globally, and geographic location was taken into consideration in the *meta*-analysis.

3.2. Risk of bias for individual studies

Risk of bias designations generally were rated as "low" or "probably low" for most domains (Fig. 2). Individual study determinations are summarized in Figure S11 and individual study ratings are also available in HAWC (https://hawcproject.org/assessment/227/) and Figure S12. In a few cases, recruitment across study groups were determined to be "high" risk. For instance, Pedersen et al. 2013 investigated low birth weight in a large European cohort study, wherein study participants were recruited from different populations in varying proportions. Confounding was predominantly rated as "probably low" (58% of studies). In some cases, studies were rated as "high" or "probably high" risk in

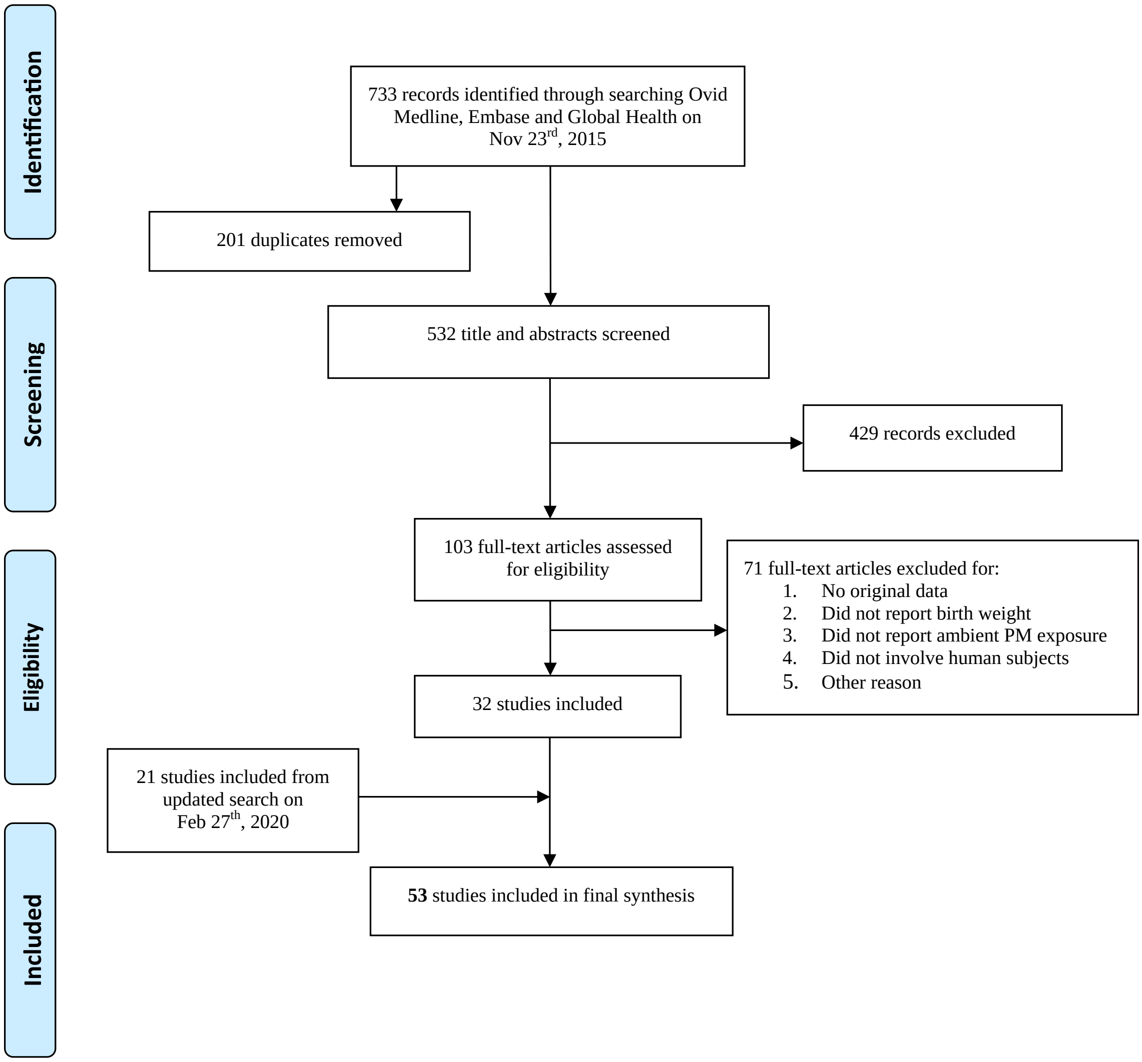

Fig. 1. A. Flowchart showing the literature search and screening process for studies relevant to prenatal particulate matter exposure and birth weight measured as a continuous variable. 1B. Flowchart showing the updated search and screening process (February 27th, 2020). The search terms used are provided in Supplemental Material, Table S1.

addressing confounding. In these cases, investigators only accounted for two or fewer of the pre-determined important potential confounders, which could have introduced bias into analyses. In a few cases reviewers determined a "probably high" risk of bias in the "other" category, defined as if the study appeared to be free of other problems that could put it at a risk of bias. For instance, regarding Mannes et al. 2005, reviewers determined a risk of residual confounding and over adjustment bias in the linear regression model, as authors adjusted for an intermediate on the pathway between exposure and outcome. In addition, authors also did not account for extreme values in birthweight for gestational age. In general, the domain with a considerable number of studies rated as "probably high" (43%) was related to the robustness of exposure assessment. This was mainly due to reliance on county-level monitoring data without adequate temporal coverage or spatial resolution. Overall, for $PM_{2.5,}$ 12 studies (out of a total of 30 studies measuring $PM_{2.5}$) were rated overall as "low" or "probably low" risk of bias. For PM_{10}, 10 studies (out of a total of 29 studies measuring PM_{10}) were rated overall as "low" or "probably low" risk of bias and used for subsequent *meta*-analysis. For studies on coarse PM, none of the 5 studies were given an overall rating of "low" or "probably low." This was largely the result of risk of exposure misclassification based on county-level measurements employed in most these studies (Darrow et al. 2011; Ebisu et al. 2016; Morello-Frosch et al. 2010; Parker and Woodruff 2008). Complete descriptions of risk of bias evaluations and their justifications are provided online in the HAWC workspace (https://hawcproject.org/assessment/227/).

3.3. Meta-analysis

We conducted a primary *meta*-analysis on studies rated as "low" or "probably low" risk of bias for exposures to $PM_{2.5}$ and PM_{10}. This included 18 total studies for $PM_{2.5}$ and 10 total studies for PM_{10}. For $PM_{2.5\text{-}10}$, there were a limited number of studies overall that measured this pollutant class, and none were rated as "low" or "probably low." Thus, we used the existing 5 studies rated as "high" or "probably high" in our primary *meta*-analysis. A summary of the *meta*-analysis results using a random effects model is shown in Table 4, separated by pollutant class and exposure window (trimester or entire pregnancy). For $PM_{2.5}$, the overall random effects estimates ranged from 5.69 g to 27.55 g decrease in birth weight per 10 µg/m^3 increase in $PM_{2.5}$ (Supplemental Figure S1A-D). The *meta*-estimate for the 1st trimester was not statistically significant, but those for the other exposure windows were. Substantial heterogeneity was evident in each exposure window (I^2 ranged from 68% to 94%). In each exposure window, at least one study reported

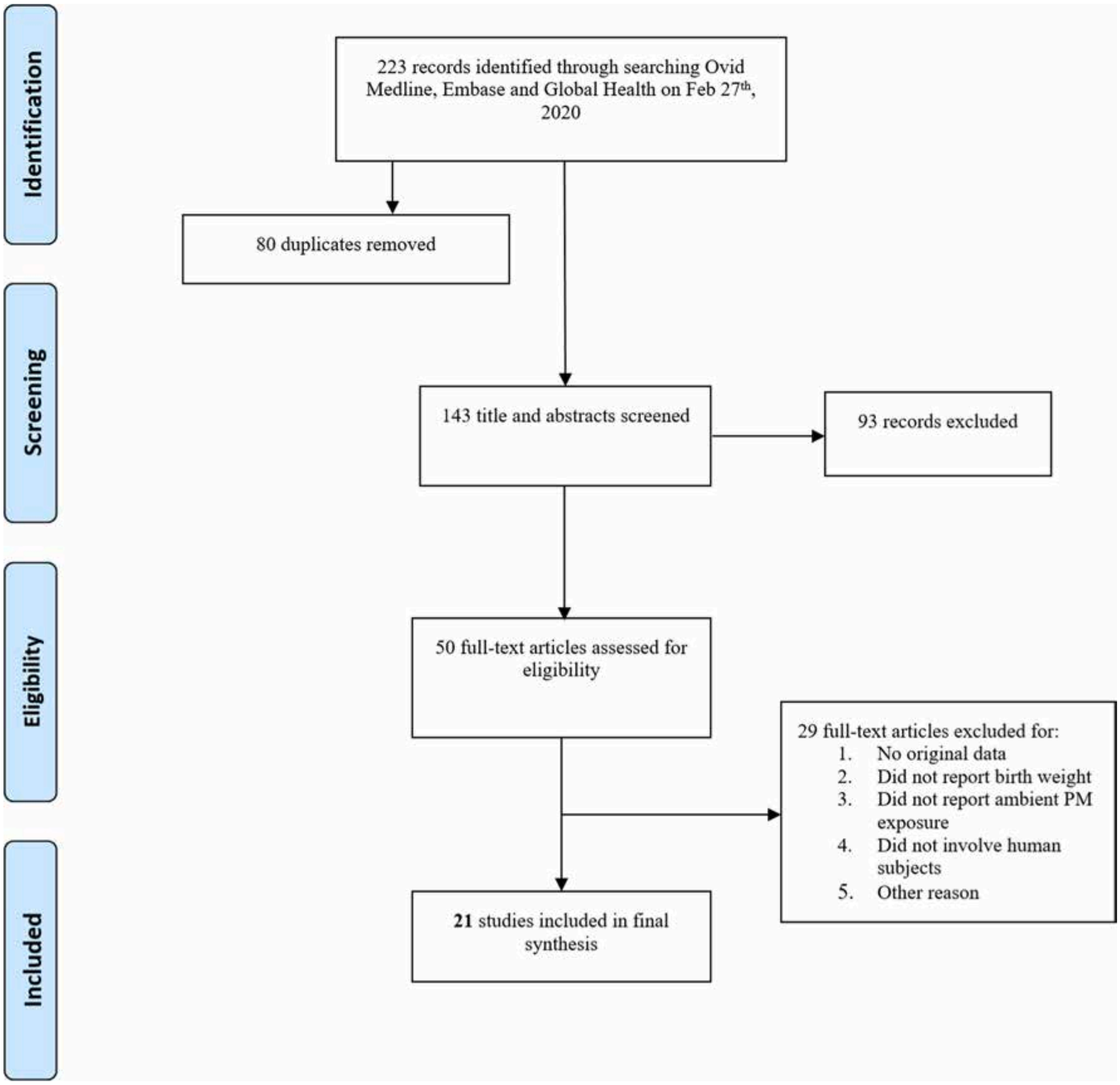

Fig. 1. (*continued*).

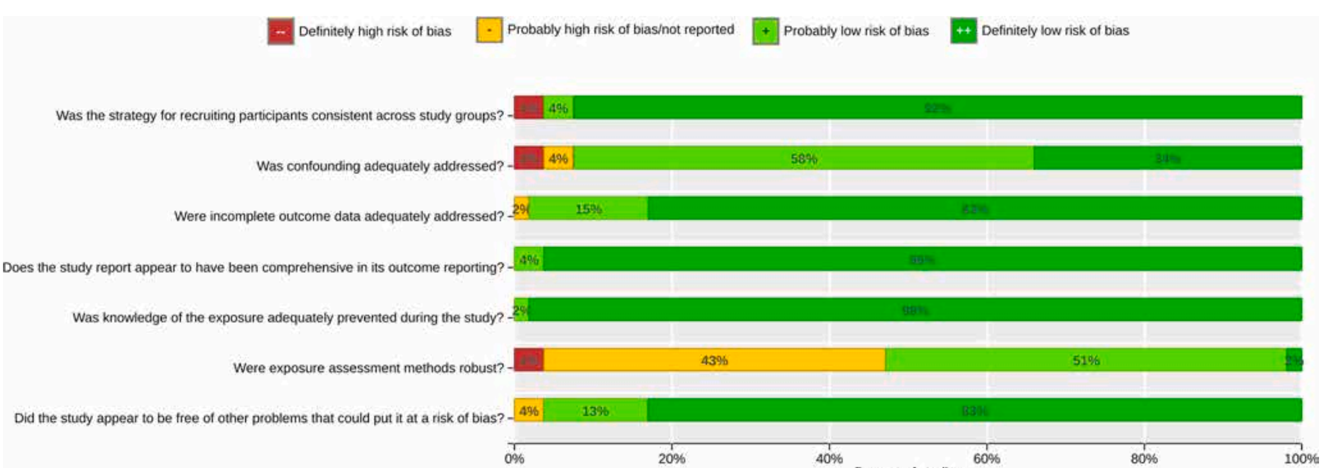

Fig. 2. Summary of risk of bias judgments. Determinations for each domain were assigned according to Supplemental Material, Table S3. In general, the domain with a considerable number of studies rated as “probably high” (43%) was related to the robustness of exposure assessment.

Table 4
Summary of main *meta*-analysis results and quality of evidence rating conclusions

Exposure Window	No. of studies	Effect estimate g per 10 μg/m^3 (95% CI)	I^2 (%)	Quality of evidence rating
$PM_{2.5}$				
1st Trimester	11	−6.50 (-15.07, 2.07)	87%	Very low (downgrades for imprecision and inconsistency)
2nd Trimester	12	−5.69 (-10.58, −0.79)	68%	Low (downgrade for imprecision)
3rd Trimester	12	−10.67 (-20.91, −0.43)	84%	Low (downgrade for inconsistency)
Full Pregnancy	15	−27.55 (-48.45, −6.65)	94%	Low (downgrade for inconsistency)
PM_{10}				
1st Trimester	6	3.22 (-3.13, 9.58)	14%	Low (downgrade for imprecision)
2nd Trimester	6	−3.37 (-8.22, 1.48)	0%	Low (downgrade for imprecision)
3rd Trimester	7	−6.57 (-10.66, −2.48)	0%	Moderate (no changes)
Full Pregnancy	8	−8.65 (-16.83, −0.48)	84%	Moderate (heterogeneity explained by single study with inverse effect)
$PM_{2.5\text{-}10}$				
1st Trimester	3	−2.70 (-3.90, −1.49)	0%	Very low (downgrades for risk of bias and imprecision)
2nd Trimester	3	−2.90 (-10.04, 4.23)	70%	Very low (downgrades for risk of bias, imprecision, inconsistency)
3rd Trimester	4	−4.93 (-10.82, 0.96)	76%	Very low (downgrades for risk of bias, imprecision, inconsistency)
Full Pregnancy	5	−8.81 (-10.32, −7.31)	0%	Low (downgrade for risk of bias)

For $PM_{2.5}$, we included 18 unique studies rated as "low" or "probably low" risk of bias. For PM_{10}, we included 10 studies rated as "low" or "probably low" risk of bias. For coarse PM ($PM_{2.5\text{-}10}$), there were no studies rated as "low" or "probably low" risk of bias, thus we included 5 studies rated as "high" or "probably high."

a positive relationship (increase in birth weight with increasing PM). Subgrouping based on ethnicity, spatial scale, or geographic location did not explain the observed heterogeneity (Supplemental Figures S6A, S7A-D, S8A-D); the only statistically significant subgroup differences were by geographic location for entire pregnancy (Supplemental Table S5). Including "high" and "probably high" risk of bias studies further increased heterogeneity (Supplemental Figure S4A-D), though subgroup differences by risk of bias were not in of themselves statistically significant (Table S5). Influence analysis showed that for the second trimester, heterogeneity is explained by a single study (Hyder et al. 2014) with a large effect size (Supplemental Figure S9B). Omitting this study reduced I^2 from 68% to 40% and reduced the *meta*-estimate from − 5.69 g (−10.58, −0.79) to − 3.81 g (−7.88, 0.25). For other exposure windows, heterogeneity could not be attributed to any single study (Supplemental Figure S9A, C-D). No evidence of publication bias (all p-values > 0.05) was found as assessed using funnel plots and tests for asymmetry (Begg and Mazumdar 1994; Egger et al. 1997; Sterne et al. 2011) (Supplemental Figures S10A-D).

For PM_{10}, the overall random effects estimates ranged from a 3.22 g increase to an 8.65 g decrease in birth weight per 10 μg/m^3 increase in PM_{10} (Supplemental Figure S2A-D). The *meta*-estimates for the 1st and 2nd trimesters were not statistically significant (effect estimate 3.22 g, 95% CI: −3.13, 9.58 and −3.37 g, 95% CI: −8.22, 1.48, respectively), but estimates for the other exposure windows were statistically significant. Low heterogeneity was seen in the trimester-based exposure windows (I^2 0–14%). However, substantial heterogeneity was evident for the entire pregnancy (I^2 84%). Subgrouping based on ethnicity was not possible due to too few studies, and subgrouping by spatial scale, or geographic location did not explain the observed heterogeneity (Supplemental Figures S7E-H, S8E-H); the only statistically significant subgroup differences were by geographic location for first trimester and entire pregnancy (Supplemental Table S5). Including "high" and "probably high" risk of bias studies increased heterogeneity in all cases (Supplemental Figure S5A-D), and subgroup differences by risk of bias were statistically significant for first and third trimesters (Table S5). Influence analysis showed that for the entire pregnancy, heterogeneity was explained largely by a single study (Geer et al. 2012) that reported a positive association, whereas all the other studies consistently showed an inverse association (Supplemental Figure S9H). Omitting this study reduced the I^2 from 84% to 0%, and changed the *meta*-estimate from − 8.65 g (−16.83, −0.48) to − 11.22 g (−13.17, −9.26). For the other exposure windows, similar results in terms of both heterogeneity and *meta*-estimates were obtained under influence analyses (Supplemental Figures S9E-G). No evidence of publication bias (all p-values > 0.05) was found as assessed using funnel plots and tests for asymmetry (Begg and Mazumdar 1994; Egger et al. 1997; Sterne et al. 2011) (Supplemental Figures S10E-H).

A smaller number of studies examined "coarse" PM ($PM_{2.5\text{-}10}$). None of these studies were rated as having "low" or "probably low" risk of bias, as discussed previously. Thus, when including all studies, overall random effects estimates ranged from a 2.70 g to 8.81 g decrease in birth weight per 10 μg/m^3 increase in $PM_{2.5\text{-}10}$ (Supplemental Figure S3A-D). The *meta*-estimates for the 2nd and 3rd trimesters were not statistically significant, −2.90 g (-10.04, 4.23) and −4.93 g (-10.82, 0.96) respectively, and each of these had high heterogeneity (I^2 70–76%). Due to the small number of studies, subgrouping based on ethnicity, spatial scale, or geographic location were not possible did not explain this observed heterogeneity (Supplemental Figures S6E-F, S7I-L, S8I-L). Heterogeneity was reduced to 55% for the 2nd trimester when omitting the most influential study (Parker and Woodruff 2008), though this left only two studies remaining with a pooled estimate that remained statistically non-significant (Supplemental Figure S9J). Similarly, in the 3rd trimester, omitting the most influential study ((Ebisu et al. 2016)) reduced heterogeneity to 64%, but the pooled estimate remained statistically non-significant (Supplemental Figure S9K). For the 1st trimester and the entire pregnancy, the *meta*-estimates were statistically significant, −2.70 g (-3.90, −1.49) and −8.81 g (-10.32, −7.31) respectively, with no observed heterogeneity in both cases (I^2 0%). For the 1st trimester, omitting any one study lead to *meta*-estimates that were either statistically non-significant or that were only barely significant (p = 0.0498) (Supplemental Figure S9I). For the entire pregnancy, *meta*-estimates remained statistically significant under influence analyses, with no heterogeneity (Supplemental Figure S9L). Insufficient studies were available to examine publication bias.

3.4. Quality of the body of evidence

In all cases, the initial rating for the quality of evidence was "moderate" based on Navigation Guide methods (Johnson et al. 2014). Using the factors for rating the quality of evidence (Table 3), we determined the following evaluations (Supplemental Table S6, Table 4). For $PM_{2.5}$ exposure in the first trimester, a downgrade of 2 levels was supported, based on "imprecision" due to the lack of a statistically significant *meta*-estimate, as well as a wide confidence interval indicating potential impact of random error. Moreover, a downgrade for "inconsistency" was due to the substantial heterogeneity that could not be explained. The resulting quality of evidence rating was "very low." For $PM_{2.5}$ exposure in the second trimester, a downgrade of 1 level was supported based on "imprecision". Heterogeneity was explained by a single study, and omitting this study lead to an effect estimate no longer statistically significant. The resulting quality of evidence rating was "low." Last, for $PM_{2.5}$ exposure in the third trimester, as well as exposure throughout entire pregnancy, a downgrade of 1 level was supported, based on "inconsistency" due to the substantial heterogeneity that could not be explained. Thus, the resulting quality of evidence rating was "low."

For PM_{10} exposure during the first trimester, a downgrade of 2 levels was supported based on "imprecision" due to a wide confidence interval and the lack of a statistically significant *meta*-estimate with low heterogeneity. The resulting quality of evidence rating was "low." For PM_{10} exposure during the second trimester, a downgrade of 1 level was supported based on "imprecision" due to the lack of a statistically significant *meta*-estimate with low heterogeneity. The resulting quality of evidence rating was "low." For PM_{10} exposure during the third trimester, no change in the quality of evidence was indicated, as the *meta*-estimate was statistically significant with low heterogeneity. The resulting quality of evidence rating was "moderate." Last, for PM_{10} exposure during the entire pregnancy, no change in the quality of evidence was indicated. Heterogeneity was explained by a single study and omitting that study lead to a precise, statistically significant *meta*-estimate. The resulting quality of evidence rating was "moderate." Meta-analysis results with "moderate" quality of evidence ratings are displayed in Fig. 3.

For exposure to coarse PM ($PM_{2.5-10}$) during the first trimester, a downgrade of 2 levels was supported based on "risk of bias" (all studies were rated "high" or "probably high"), "imprecision" due to few studies (n = 3), and a high degree of influence of any one study had on statistical significance. The resulting quality of evidence rating was "very low." For $PM_{2.5-10}$ exposure during the second and third trimesters, downgrades of 3 levels are supported based on "risk of bias" (all studies were rated "high" or "probably high"), "imprecision" due to the lack of a statistically significant *meta*-estimate, and "inconsistency" due to high, unexplained heterogeneity. The resulting quality of evidence rating was "very low." Last, for $PM_{2.5-10}$ exposure throughout the entire pregnancy, a downgrade of 1 level was supported based on "risk of bias" (all studies were rated "high" or "probably high"). The resulting quality of evidence rating was "low."

3.5. *Strength of the body of evidence*

Using the considerations for rating the strength of evidence in

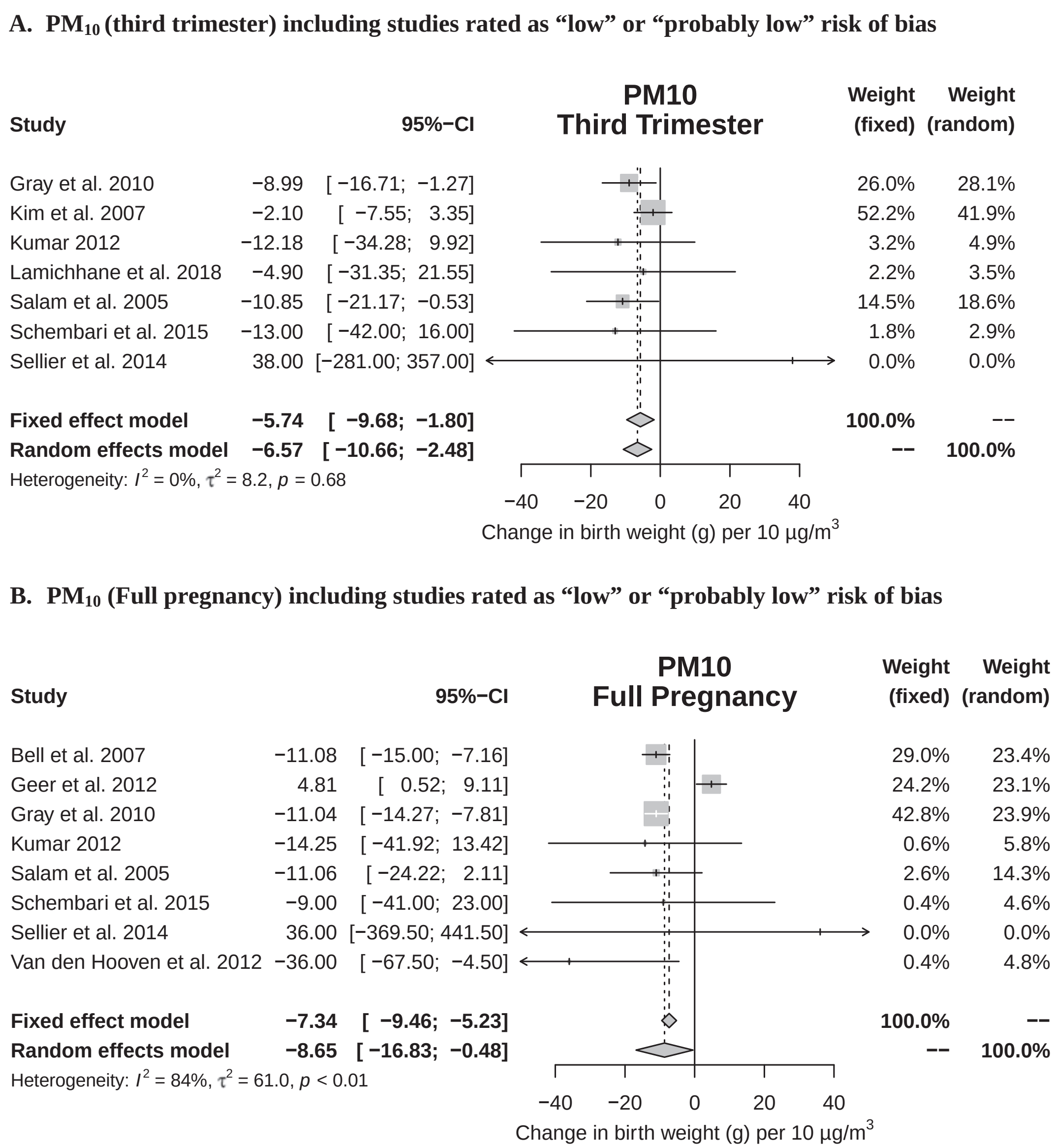

Fig. 3. Meta-analysis results for pollutants demonstrating "moderate" quality of evidence rating include (A) PM_{10} exposure during the 3rd trimester and (B) PM_{10} exposure throughout entire pregnancy.

Table 5
Summary of strength of evidence conclusions

Exposure Window	Quality of evidence rating[1]	Direction of effect estimates	Confidence in effect estimates	Other compelling attributes	Strength of evidence rating
$PM_{2.5}$					
1st Trimester	Very low	Adverse[2]	Low[3]	None	Inadequate
2nd Trimester	Low	Adverse[2]	Low[3]	None	Inadequate
3rd Trimester	Low	Adverse[2]	Low[3]	None	Inadequate
Full Pregnancy	Low	Adverse[2]	Low[3]	None	Inadequate
PM_{10}					
1st Trimester	Low	Adverse[2]	Low[3]	None	Inadequate
2nd Trimester	Low	Adverse[2]	Low[3]	None	Inadequate
3rd Trimester	Moderate	Adverse[2]	Limited[4]	None	Limited
Full Pregnancy	Moderate	Adverse[2]	Limited[4]	None	Limited
$PM_{2.5\text{-}10}$					
1st Trimester	Very low	Adverse[2]	Low[3]	None	Inadequate
2nd Trimester	Very low	Adverse[2]	Low[3]	None	Inadequate
3rd Trimester	Very low	Adverse[2]	Low[3]	None	Inadequate
Full Pregnancy	Low	Adverse[2]	Low[3]	None	Inadequate

[1] From Table 4.
[2] Decreasing birth weight with increasing exposure is considered an effect in the adverse direction.
[3] Results may be due to chance, bias, or confounding, so additional data are likely to alter the results.
[4] A credible association is observed, but chance, bias, and confounding cannot be ruled out with reasonable confidence, so additional data could alter the results.

Table 3, the following evaluations were made (Table 5). For $PM_{2.5}$, there is “inadequate evidence” for all exposure windows due to “low” or “very low” quality of evidence, based on either imprecision of the estimate or high and unexplained heterogeneity (none of the other considerations were influential in this evaluation). For PM_{10}, there is “limited evidence” that increasing exposure during the third trimester or during the entire pregnancy will lead to a reduction in birth weight. The quality of evidence for these exposure windows was rated as “moderate.” Although the direction of the effect estimate was in the “adverse” direction, confidence in the effect estimate is limited because chance, bias, and confounding cannot be ruled out with reasonable confidence, and additional data could alter this conclusion. No other compelling attributes of the data exist that would influence this evaluation. For other exposure windows, evidence for PM_{10} is “inadequate” due to “low” or “very low” quality of evidence, based on either imprecision of the estimate and/or the presence of a relationship in the opposite (non-adverse) direction (none of the other considerations were influential in this evaluation). For $PM_{2.5\text{-}10}$, there is “inadequate evidence” that increasing exposure is during any exposure window leads to a reduction in birth weight. The available evidence is insufficient to assess the effects of exposure, mainly due to high risk of bias in individual studies and the reliance on a small set of often heterogeneous studies. None of the other considerations from Table 3 were influential to this evaluation.

4. Discussion

Numerous case-control and cohort studies demonstrate an association between prenatal exposure to ambient air pollution and reduced fetal growth or infant birthweight. An early systematic review found an association between $PM_{2.5}$ exposure and LBW and SGA births, as well as PM_{10} exposure and SGA (Shah et al. 2011). Despite these observed associations, there have been inconsistencies in the conclusions about the association and magnitude of the effect. Initial systematic reviews based on a relatively small number of studies (n = 4), were not able to draw conclusions on effect size (Bonzini et al. 2010; Bosetti et al. 2010; Ghosh et al. 2007). More recent systematic reviews, which performed a *meta*-analysis on a larger number of studies (>30) showed that pooled estimates of effect size for LBW for a 10 μg/m^3 increase in $PM_{2.5}$ exposure during entire pregnancy ranged from − 15.9 g (−26.8, −5.0) (Sun et al. 2016) to − 22.17 g (−37.93, −6.41) (Lamichhane et al. 2015). Steib et al. also reported estimates per 10 μg/m^3 increase in $PM_{2.5}$ exposure to be − 23.4 g (−45.5, −1.4) (Stieb et al. 2012), all of which are consistent with our pooled estimate of −27.55 g (−48.45, −6.65) per 10 μg/m^3. This agreement is likely due to several of the same studies used across these *meta*-analyses. For PM_{10}, Lamichhane et al. reported estimates for a 10 μg/m^3 increase at − 10.31 g (−13.57 to − 3.13 g), whereas Stieb et al. published estimates for a 20 μg/m^3 increase at − 16.8 g (−20.2 to − 13.3) (Lamichhane et al. 2015; Stieb et al. 2012), both of which are also consistent with our pooled estimate of − 8.65 g (−16.83, −0.48) per 10 μg/m^3. These previous investigators cited that they were not able to rule out the consequences of specific biases that may be as a result of differences in study methodology, study design, population demographics, exposure period, characterization of confounding and data collection.

In our analysis, there was substantial heterogeneity across the different pollutant classes. Also, the spatial scale employed, large scale (at the city or county level or >/= 10 km) in comparison to medium scale (census tract, zip code, postal code, nearest monitor, <10 km and >/=5km) or small scale (<5km) led to greater heterogeneity. These findings underscore the complexity of estimating exposure across gestation. While one study (Jedrychowski et al. 2009) employed personal monitoring during pregnancy, the cost of adequate temporal coverage is great since it is infeasible for participants to carry monitors over time. Despite the significant heterogeneity, we still observed a decrease in birthweight for every 10 μg/m^3 increase in $PM_{2.5}$ across all trimesters (except the 1st) and entire pregnancy, as well as for every10 μg/m^3 increase in PM_{10} across the third trimester and entire pregnancy. The “inadequate” evidence rating for $PM_{2.5}$ reflects the quality, which received downgrades for inconsistency, driven mainly by heterogeneity. Similar conclusions were drawn by Lam et al. for the association between early-life exposure to air pollution as a whole and diagnosis of autism spectrum disorder (Lam et al. 2016).

Some limitations that may be associated with our study include the reliance on expert evaluation in the process used for the risk of bias, quality and strength ratings. However, this limitation was overcome by creating a diverse team of experts from relevant fields to participate in this process. Moreover, by publishing a pre-specified protocol and employing two independent reviewers for each study, our analysis includes a degree of transparency and robustness that is absent when using less structured approaches. Additionally, the rating of the quality of evidence across studies was dependent on the available data. For instance, PM_{10} and $PM_{2.5}$ are typically reported separately, but also likely occur in combination. Thus, models that consider multi-pollutant exposures may better represent gestational PM exposure. Furthermore, most studies fail to consider secondary/co-exposures like ultrafine particulate matter, gas phase pollutants, or heat, which can also affect birth weight. A recent systematic review including cohort and cross-sectional studies in U.S. populations demonstrated a significant association of air pollutant and heat exposure with adverse birth outcomes, such as preterm birth and low birth weight (Bekkar et al. 2020). There is also the potential for additional unmeasured confounding. For instance, (Wilson et al. 2017) noted that associations between infant health and with air pollution during individual trimesters may be biased unless all trimesters are included in the same model to fully address confounding and seasonal trends. Less than a quarter of the studies we identified addressed this issue, though subgrouping analyses revealed no statistically significant differences between studies that treated trimesters separately versus together in a single model. Recent studies also include measures of more temporality refined exposure windows, for instance, monthly or weekly averages. These studies may yield important insight into the critical windows of exposure ((Arroyo et al. 2019; Liu et al. 2019; Yuan et al. 2020). However, our analyses did not include enough studies to evaluate weekly exposure.

A major strength of our study is the transparency and thoroughness of the Navigation Guide systematic review process, which incorporates the GRADE system for assessing the quality of synthesized human evidence in environmental health research in the absence of randomized clinical trials (Woodruff and Sutton, 2014). Overall, our results support the vast evidence that prenatal PM exposure is associated with reduced infant birth weight. These implications on infant mortality burden were included for the first time in the State of Global Air report, which highlighted air pollution accounts for 20% of newborn deaths worldwide, mostly related to complications of low birth weight and preterm birth (Health Effects Institute). Thus, public health interventions to address infant birth weight suppression from PM may have a substantial impact on infant health, especially those at high risk for exposure. Future research and implementation strategies are recommended to help optimize interventions and policies to mitigate infant health effects.

5. Conclusions

Overall, we conclude that the existing evidence supports an association between prenatal exposure to ambient particulate matter air pollution and a decrease in birth weight, particularly for PM_{10}. However, our findings reveal the need to standardize and improve exposure assessment methods in air pollution research because the various forms of exposure measurement utilized in the studies contributed to the heterogeneity seen in the *meta*-analysis. Furthermore, some of the unexplained heterogeneity found in our study may be resolved with additional studies which could also strengthen the evidence.

CRediT authorship contribution statement

Inyang Uwak: Investigation, Formal analysis, Validation, Writing - original draft. **Natalie Olson:** Investigation, Formal analysis, Validation, Writing - review & editing. **Angelica Fuentes:** Investigation, Validation. **Megan Moriarty:** Investigation, Validation. **Jairus Pulczinski:** Investigation, Validation. **Juleen Lam:** Methodology, Validation, Writing - review & editing. **Xiaohui Xu:** Validation. **Brandie D. Taylor:** Validation, Writing - review & editing. **Samuel Taiwo:** Validation. **Kirsten Koehler:** Validation, Writing - review & editing. **Margaret Foster:** Methodology. **Weihsueh A. Chiu:** Conceptualization, Methodology, Formal analysis, Validation, Project administration, Supervision, Writing - review & editing. **Natalie M. Johnson:** Conceptualization, Validation, Project administration, Supervision, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2021.106378.

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RESEARCH ARTICLE

A Systematic Review and Meta-Analysis of Multiple Airborne Pollutants and Autism Spectrum Disorder

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Abstract

Background

Exposure to ambient air pollution is widespread and may be detrimental to human brain development and a potential risk factor for Autism Spectrum Disorder (ASD). We conducted a systematic review of the human evidence on the relationship between ASD and exposure to all airborne pollutants, including particulate matter air pollutants and others (e.g. pesticides and metals).

Objective

To answer the question: ªis developmental exposure to air pollution associated with ASD?º

Methods

We conducted a comprehensive search of the literature, identified relevant studies using inclusion/exclusion criteria pre-specified in our protocol (registered in PROSPERO, CRD # 42015017890), evaluated the potential risk of bias for each included study and identified an appropriate subset of studies to combine in a meta-analysis. We then rated the overall quality and strength of the evidence collectively across all air pollutants.

The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

Results

Of 1,158 total references identified, 23 human studies met our inclusion criteria (17 case-control, 4 ecological, 2 cohort). Risk of bias was generally low across studies for most domains; study limitations were related to potential confounding and accuracy of exposure assessment methods. We rated the quality of the body of evidence across all air pollutants as ªmoderate.º From our meta-analysis, we found statistically significant summary odds ratios (ORs) of 1.07 (95% CI: 1.06, 1.08) per 10-μg/m^3 increase in PM_{10} exposure (n = 6 studies) and 2.32 (95% CI: 2.15, 2.51) per 10-μg/m^3 increase in $PM_{2.5}$ exposure (n = 3 studies). For pollutants not included in a meta-analysis, we collectively evaluated evidence from each study in rating the strength and quality of overall evidence considering factors such as inconsistency, imprecision, and evidence of dose-response. All included studies generally showed increased risk of ASD with increasing exposure to air pollution, although not consistently across all chemical components.

Conclusion

After considering strengths and limitations of the body of research, we concluded that there is ªlimited evidence of toxicityº for the association between early life exposure to air pollution as a whole and diagnosis of ASD. The strongest evidence was between prenatal exposure to particulate matter and ASD. However, the small number of studies in the meta-analysis and unexplained statistical heterogeneity across the individual study estimates means that the effect could be larger or smaller (including not significant) than these studies estimate. Our research supports the need for health protective public policy to reduce exposures to harmful airborne contaminants among pregnant women and children and suggests opportunities for optimizing future research.

Introduction

Air pollution is a serious public health issue, responsible for over seven million deaths a year worldwide [1]. In addition to mortality, cardiovascular and respiratory diseases have been identified as primary health concerns related to exposure [2–4]. More recently, the central nervous system was proposed as another organ negatively affected by air pollutants [5], and prenatal air pollution has been identified as having potentially greater impacts than adult exposures [6].

Air pollution is composed of a large number of compounds coming from a wide variety of sources, notably vehicle and powerplant emissions. Compounds include well-characterized pollutants such as particulate matter (PM) and ozone, and lesser-characterized airborne chemicals like metals and pesticides. Studies of individual components of air pollution have found links to neurodevelopmental outcomes, including effects on intelligence quotient (IQ), language development, executive function, and psychomotor development [7–10]. Studies in animals have also found that developmental exposure to air pollution is related to functional and structural brain effects [11–15].

In particular, a number of studies have reported a relationship to Autism Spectrum Disorder (ASD). ASD is a group of complex neurodevelopmental disabilities defined by a spectrum of behaviors, characterized generally by difficulties with social interaction and communication accompanied by restricted or repetitive behaviors or interests. ASD has well-characterized comorbidities and increasing prevalence, estimated within the United States (US) at 1 in 68 children in 2010 (1.5%) [16, 17]. ASD has few and limited effective treatments and is associated with considerable financial and medical burden; research into its etiology has increased dramatically over the last decade to address the increasing prevalence. Many genetic, lifestyle, and environmental factors are being explored—current thinking suggests that multiple causes are likely to blame, including a number of genetic pathways and environmental chemical exposures, or their interaction [18–20]. The widespread availability of air pollution exposure data generated from decades of interest in air quality's other health effects has led to air pollution as one of the more-studied candidate ASD environmental risk factors.

However, a systematic review and meta-analysis of the evidence for the relationship between air pollution and ASD had been lacking. While systematic review methods have been used for decades in the clinical sciences [21, 22], such methods have only recently been developed and utilized in environmental health sciences [23–30]. Therefore, we applied the Navigation Guide review methodology [23, 26] to answer the question "does developmental exposure to air pollution affect diagnosis of ASD?"

Methods

The Navigation Guide Systematic Review Methodology

To conduct our review of ASD and air pollution we applied the Navigation Guide, a systematic and transparent methodology for synthesizing the available scientific evidence [23, 26]. The Navigation Guide systematic review methodology is based on Cochrane/GRADE methods [21, 22] and includes all the same elements (protocol, development, risk of bias evaluation, evidence evaluation, etc.) but accounts for the differences in evidence and decision context inherent to environmental health assessments, i.e., the reliance on human observational studies in the absence of randomized controlled trials (RCTs), and the fact that population exposure to exogenous chemicals precedes evidence of their safety. To date, the Navigation Guide method has been used in 3 case studies [25, 27, 28].

We assembled a diverse team of reviewers in August 2014 with expertise in epidemiology, air pollution/exposure assessment, ASD outcome assessment, biostatistics, library sciences, and/or systematic review methodology. We developed a protocol to outline the process for conducting the systematic review prior to initiating the study and registered the protocol with an international database for systematic reviews in March 2015. Each member of the review team also filled out a conflict of interest statement at the initiation of their involvement with the protocol to document any potential financial or other conflicts of interest, and these were appended to the protocol. Each of the protocol steps are described below and the protocol is available on PROSPERO (http://www.crd.york.ac.uk/PROSPERO/; CRD # 42015017890).

Specify the study question. Our objective was to answer the question: "Does developmental exposure to air pollution affect diagnosis of Autism Spectrum Disorder (ASD)?"

We developed a "Participants", "Exposure," "Comparator" and "Outcomes" (PECO) statement, as follows:

Participants: Humans

Exposure: Any developmental exposure to air pollution that occurred prior to the ASD assessment.

- *"Any developmental exposure" is defined as maternal or paternal exposure incurred any time "in proximity to" conception (as defined by authors of the included study), or exposures to offspring incurred in utero or in the perinatal or childhood period.*

- *"Air pollution" is defined as any indoor or outdoor source of any inhaled airborne environmental chemical, EXCLUDING active and passive smoking.*
- *Exposures "prior to the ASD assessment" include direct and proxy measures for this time period.*

Comparator: Humans exposed to lower levels of air pollution than the more highly exposed humans.

- *This definition is intended to include groups defined by ASD case-control studies; for instance comparing the air pollution exposure levels for people with ASD versus those without.*

Outcome: Any clinical diagnosis or other continuous or dichotomous scale assessment of ASD.

- *Clinical ASD diagnosis can be based on the International Classification of Diseases (ICD) 9, ICD 10, Diagnostic and Statistical Manual of Mental Disorders (DSM) 5, or DSM-IV criteria, including difficulties in social interaction, verbal and nonverbal communication and repetitive behaviors.*

Select the Evidence. *Search methods*. Our search was not limited by language or start-date of publication. We searched several online databases (PubMed, ISI Web of Science, Biosis Previews, Embase, Google Scholar, and Toxline) between November 3–5, 2014 using the search terms in S1 Table. We used the Medical Subject Headings (MeSH) database to compile synonyms for air pollution and ASD-related outcomes (http://www.ncbi.nlm.nih.gov/mesh/68000397, http://www.ncbi.nlm.nih.gov/mesh/68001321). We separated the exposure-related search into two general categories: one based on the route of exposure (air inhalation, along with appropriate synonyms in a Boolean search using the "OR" statement) and the other based on typical chemical composition of air pollution (ozone, particulate matter, etc. in a Boolean search using the "OR" statement). We intentionally crafted a broad search strategy for exposure to capture all studies evaluating any indoor/outdoor chemical air pollutant for inclusion, excluding those related to cigarette smoke. These two categories of search terms were then combined in a Boolean search using the "OR" statement to create the collection of exposure search terms. For the outcome, we combined "autism spectrum disorder" and its synonyms in a Boolean search using the "OR" statement (S1 Table).

We then combined the exposure and outcome terms using a Boolean search using the "AND" statement. We searched for terms in titles and abstracts (using the [tiab] function in PubMed, topic search in Web of Science and Biosis Previews; "ti,ab." function in Embase) or in MeSH headings (using the [mh] function in PubMed). We searched additional toxicological websites and grey literature databases (S2 Table) intended to capture papers and reports from the non-peer reviewed literature (November 6–13, 2014). We performed "snowball searching," which included hand-searching the reference lists of all included studies as well as review articles identified in the screening process, and using Web of Science to search for articles that cited the included studies. We also reached out to a group of recognized experts in this field from the Environmental Epidemiology of Autism Research Network (EEARN) in March 2015 to request review of the included studies to identify whether we had missed any relevant studies.

Study selection criteria. We included studies only if: 1) the report contained original data from human studies; 2) there was a measure or report of air pollution exposure prior to the diagnosis or assessment of ASD; and 3) there was a comparator (control group or exposure range comparison). References that did not meet all of these criteria were excluded from consideration (S3 File).

We screened references in duplicate for inclusion using structured forms in DRAGON (ICF International; available at: http://www.icfi.com/insights/products-and-tools/dragon-online-tool-systematic-review) and DistillerSR (Evidence Partners; available at: http://www.systematic-review.net). To determine eligibility, each reference from the literature search had the title and abstract independently reviewed by two of the five reviewers (ND, AH, AK, LD, GW) in a non-random assignment to ensure that the same two authors did not always screen the same references. In the event that an abstract was missing or there were discrepancies between the two reviewers, the default was to move the reference forward for full text review. Two of the five reviewers (ND, AH, AK, LD, GW) then independently performed a full-text review to evaluate inclusion criteria of each reference not excluded by title/abstract screening. An additional reviewer (PS) screened five percent of the titles/abstracts and full-texts for quality assurance.

Data collection and management. Four authors (ND, LD, PS, EK) and a UCSF research assistant (HT) independently extracted data relating to study characteristics and outcome measures (S3 Table) from all included articles into a DRAGON database. We contacted the corresponding author when information pertinent to our study question was missing or. A third author (JL) performed QA/QC on all of the studies by reviewing the studies and its extracted data to check for accuracy.

Reviewing the evidence. *Assessing the risk of bias for each included study*. We assessed risk of bias for each included study using a modified instrument we developed based on the Cochrane Collaboration's "Risk of Bias" tool and the Agency for Healthcare Research and Quality's (AHRQ) domains, i.e., selection bias, confounding, performance bias, attrition bias, detection bias, and reporting bias [21, 31]. We modified the wording and instructions for several of these domains beforehand to make it specific for our study question and the types of evidence or study characteristics that we anticipated.

In particular, because of the complexity of methods for assessing exposure to air pollution, we ultimately developed a novel risk of bias instructions for exposure assessment that specified a list of important considerations, i.e., modeling, monitoring, biomarkers, etc. Review authors developed this tool collectively and in collaboration with a known expert in the air pollution field (HC) as well as the EEARN working group. Review authors were instructed to separately rate the exposure assessment risk of bias for each air pollutant chemical or classes of chemicals reported in the study under review. The justification for this was based on empirical evidence demonstrating that similar exposure assessment methods for different air pollutant compounds are heterogeneous in terms of their internal validity [32, 33]. We also elicited expert opinions from the EEARN working group to develop a list of potentially important confounders or effect modifiers to include in the analysis. These are outlined in the protocol in the risk of bias tool, under the confounding domain, along with justification for inclusion and relevant citations.

We assigned each risk of bias domain as "low," "probably low," "probably high," or "high" risk of bias, or "not applicable" (risk of bias area not applicable to study) according to specific criteria as described in our risk of bias instruments (S2 File). Two of the eight review authors with subject-matter expertise (GW, AH, CN, AK, CL, TW, PS, EK) independently recorded risk of bias determinations for each included study. In the event that one of the review authors was a coauthor of the study in question, they recused themselves from rating the risk of bias for that particular study. We held an in-person meeting to review rationales and ratings for each study and come to consensus. Based on this discussion, two review authors (JL, EK) subsequently reviewed ratings for all included studies to ensure consistency across studies with similar study populations or study design.

Statistical analyses. We assessed study characteristics from included articles to evaluate comparability of findings based on pre-determined features as outlined in our protocol (i.e., study features, study population, exposure assessment method, and outcome assessment method) to determine which study results were potentially suitable for meta-analysis. In particular, review authors had decided beforehand that different air pollutant chemicals or classes of compounds (i.e., heavy metals, pesticides, and criteria air pollutants) should be analyzed separately in the absence of empirical evidence to suggest that we could combine these effect estimates. From the assessment of specified characteristics, we determined that two subsets of studies in which exposure was measured during pregnancy or prior to assessment of ASD met these criteria. The first was for particulate matter less than 10 μm (PM_{10}) (comprised of six studies), and the second was for fine particulate matter, less than 2.5 μm ($PM_{2.5}$) (comprised of three studies), from studies where exposure was measured during pregnancy prior to diagnosis or assessment of ASD.

PM_{10} or $PM_{2.5}$ concentrations and their standard errors (reported in the study or calculated from reported 95% confidence interval and sample sizes) were extracted from each study for the meta-analysis. We extracted adjusted odds ratio (OR) estimates reported for a continuous increment increase in exposure, then standardized effect estimates across all studies by computing adjusted OR estimates per 10 μg/m^3 increase in PM. In the event that such an estimate was unavailable or could not be calculated from the data available in the published article, we contacted study authors to request these data be made available to us. We then combined the standardized effect estimates from each study in a random effects model with inverse variance weighting. The result was an estimate of the combined summary OR per 10 μg/m^3 increase in PM, accounting for within- and between-study variability. We used R statistical software (version 3.0.1) and the "metafor" package for analyses.

We evaluated statistical heterogeneity across study estimates in the meta-analysis using Cochran's Q statistic with $p \leq 0.05$ as our cut off for statistical significance and I^2 [21], as previously described [24, 27, 30]. If statistical heterogeneity was present, we used leave-one-out analysis to identify the study or studies contributing, evaluated potential study characteristics (for example, study location, study population, study design, adjusted confounders, timing of exposure, etc.) to explain the source, and incorporated hierarchical cluster structures in the data analysis to statistically account for the heterogeneity.

Although data for other contaminants were not amenable to a meta-analysis due to insufficient number of studies and/or the existence of heterogeneity across study characteristics, we created scatterplots of effect estimates by contaminant to visually inspect these results and evaluate associations.

*Rating the **quality** of evidence across all included studies*. We rated the quality of the overall body of evidence as either"high,""moderate" or"low." The Navigation Guide approach follows that established by the Grading of Recommendations Assessment Development and Evaluation (GRADE) method used in the clinical field; i.e., by first assuming an initial quality rating to the body of evidence and then considering adjustments ("downgrades" or "upgrades") based on the characteristics of the included studies to reach a final quality rating [34].

We assumed an initial rating of "moderate" quality to the human bodies of evidence (observational studies), based on previously described rationale [26] and consistent with previous case studies [27, 28, 30], based on consideration of both the values and limitations of observational data in assessing associations between exposure and health outcomes in environmental health. We considered downgrades and upgrades to this initial quality rating based on 8 specific factors and instructions for consideration (S4 Table): risk of bias, indirectness of evidence, inconsistency of evidence, imprecision of evidence, potential for publication bias, large magnitude of effect, dose response relationship, and whether residual confounding would minimize the overall effect estimate. Possible ratings were 0 (no change from initial quality rating), -1 (1 level downgrade) or– 2 (2 level downgrade); +1 (1 level upgrade) or + 2

(2 level upgrade). Review authors independently evaluated the quality of the evidence and then compared their ratings and rationale for each quality category. We discussed our ratings as a group and recorded our rationale. Consistent with GRADE, we did not automatically add together the ratings for each downgrade and upgrade factor to create a score, e.g., a (-1) downgrade for each of 2 factors does not necessarily translate into a (-2) downgrade overall. We used our judgment to decide the weight of each downgrade or upgrade in the final overall quality rating.

*Rating the **strength** of the evidence across all included studies*. We rated the overall strength of the body of evidence based on 4 considerations: (1) Quality of body of evidence (i.e., the rating from the previous step); (2) Direction of effect; (3) Confidence in effect (likelihood that a new study would change our conclusion); and (4) Other compelling attributes of the data that may influence certainty. We used these considerations to assign the overall strength rating, according to the definitions specified in the Navigation Guide for "sufficient evidence of toxicity," "limited evidence of toxicity," "inadequate evidence of toxicity," or "evidence of lack of toxicity" (Table 1), which are based on categories used by the International Agency for Research on Cancer (IARC) [35], with the definitions of each category based on IARC, the U.S. Preventive Services Task Force, and U.S. EPA for evidence integration [35–38]. Review authors independently evaluated the strength of the evidence and compared their evaluations, resolved discrepancies by discussion, and recorded the collective rationale for decisions.

Table 1. Strength of evidence definitions.

Strength Rating	De®nition
Suf®cientevidence of toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable con®dence.[a] The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.[b]
Limited Evidence of Toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding cannot be ruled out with reasonable con®dence Con®dence in the relationship is constrained by such factors as: the number, size, or quality of individual studies, or inconsistency of ®ndingsacross individual studies[a]. As more information becomes available, the observed effect could change, and this change may be large enough to alter the conclusion.
Inadequate Evidence of Toxicity	The available evidence is insuf®cientto assess effects of the exposure. Evidence is insuf®cientbecause of: the limited number or size of studies, low quality of individual studies, or inconsistency of ®ndingsacross individual studies.[a] More information may allow an assessment of effects.
Evidence of Lack of Toxicity	No relationship is observed between exposure and outcome, and chance, bias and confounding can be ruled out with reasonable con®dence. The available evidence includes consistent results from more than one well-designed, well-conducted study at the full range of exposure levels that humans are known to encounter, and the conclusion is unlikely to be strongly affected by the results of future studies.[a] The conclusion is limited to the age at exposure and/or other conditions and levels of exposure studied.

[a] Language for the de®nitionsof the rating categories were adapted from descriptions of levels of certainty provided by the U.S. Preventive Services Task Force Levels of Certainty Regarding Net Bene®t[35]
[b]The Navigation Guide rates the quality and strength of evidence of human and non-human evidence streams separately as ªsuf®cientº,ªlimitedº, ªinadequateº or ªevidence of lack of toxicityº and then these two ratings are combined to produce one of ®vepossible statements about the overall strength of the evidence of a chemical's reproductive/developmental toxicity. The methodology is adapted from the criteria used by the International Agency for Research on Cancer (IARC) to categorize the carcinogenicity of substances [35] except as noted.

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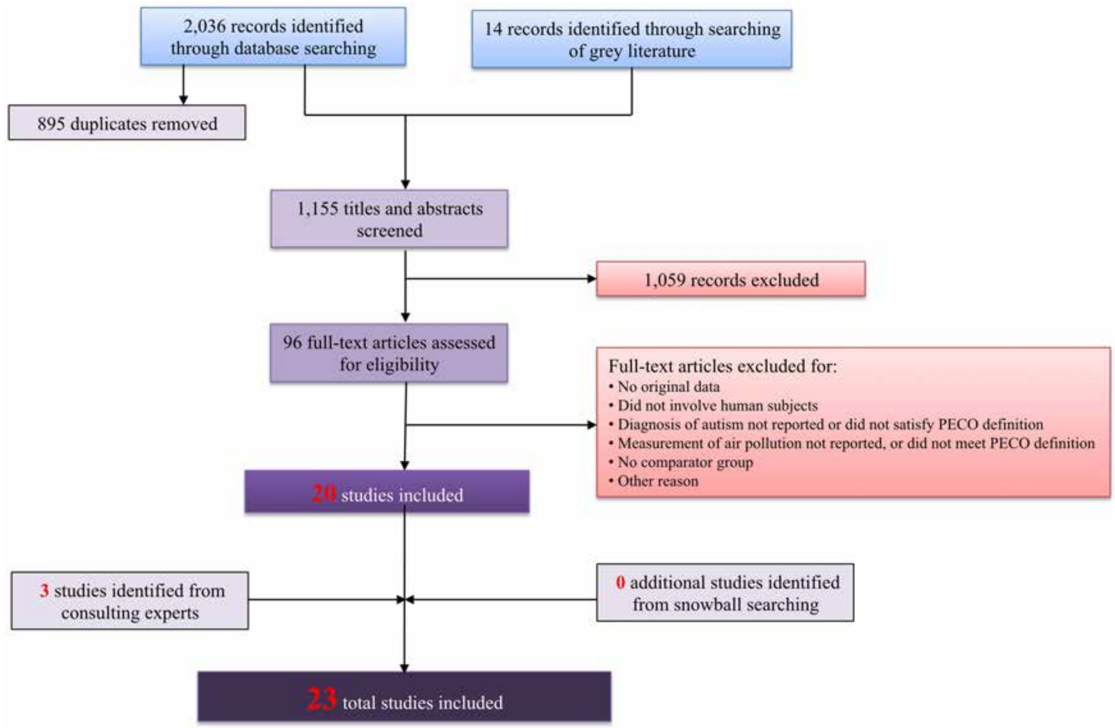

Fig 1. Search results for studies relevant to air pollution exposure and ASD outcome.

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Results

Included studies

We retrieved a total of 1,155 references from our literature search, of which 96 met inclusion criteria based on title and abstract screening and 20 further met inclusion criteria based on full text screening [39–58]. We also identified three additional studies [59–61] by consulting with experts in the field (those affiliated with the EEARN listserve) who reviewed our list of included studies to determine whether there were additional studies that had not yet been identified, bringing the total number of screened references to 1,158 and the total number of included studies to 23 (Fig 1). Some of these studies initially had multiple separate records—for instance, a conference abstract plus a subsequently published manuscript; the information from these records was ultimately collectively assessed using information from the peer-reviewed manuscript supplemented by further relevant details from the additional records if applicable. The 23 included studies were all published between the years 2006 to 2015, included 174 to 4,057,712 participants born between the early 1990s through 2005, were based in 4 different countries, and involved case-control, cohort, and ecological study designs (Table 2). All but one study published in Spanish [47] were in English.

Risk of bias assessment for individual studies

Overall, most studies were rated as "low" or "probably low" risk of bias in most domains other than the confounding and exposure assessment domains (Fig 2). Many of the studies rated as "probably high" (with one "high") for potential confounding were due to the failure to adjust for many of the important confounders that we had established beforehand in our protocol. We rated exposure assessment risk of bias separately for each air pollutant or class of components within each study. Overall, we rated 103 different air pollutant chemicals (such as formaldehyde, $PM_{2.5}$, ozone, etc.) and chemical classes (such as pesticides, fumigants, traffic-related pollutants, etc.). Only 21 of these chemicals or chemical classes were reported in $\geq$3 studies. Many of the contaminants that were ultimately rated as having “high” risk of exposure assessment bias used data from the US EPA National-scale Air Toxics Assessment (NATA), Toxic Release Inventory (TRI), or self-reported from surveys. We rated risk of exposure assessment bias for each air pollutant that used NATA data following published guidance that had already established the degree of confidence in each individual NATA contaminant estimate by comparing to monitored air pollution values [62, 63]. Following this guidance, our initial exposure assessment risk of bias ratings for NATA data were assigned as “probably low” for those with “higher confidence” (i.e., benzene, acrylonitrile, carbon tetrachloride, etc.), “probably high” for those with “medium” confidence (i.e., coke oven emissions, vinyl chloride, chloroform, ethylene dibromide, etc.), and “high” for those with “lower” confidence (i.e., arsenic compounds, beryllium compounds, cadmium compounds, lead compounds, etc.). All the air pollutants that were based on data modeled from Toxic Release Inventory (TRI) data were rated as “probably high” risk of exposure assessment based on concerns regarding the validity of extrapolating emission quantity data to individual or community-level exposures. Ultimately, only the studies involving air pollutants of PM_{10}, $PM_{2.5}$, ozone and methylene chloride were rated as “probably low” risk of bias (Fig 2b). Additional detail on individual study characteristics and risk of bias designation/rationale is presented in the S5 Table.

Statistical analysis

Of the 23 included studies, six studies that measured PM_{10} and three that measured $PM_{2.5}$ (a subset of the six with PM_{10} data) were amenable to meta-analyses. One additional included study [53] also reported effect estimates for both PM_{10} and $PM_{2.5}$ but ultimately was not included in the meta-analysis because upon personal communication with the study authors [64] it was determined that the study population was a subset of the population reported in a previous article [52] that was already included in the meta-analyses. Furthermore, although included studies evaluating ozone and methylene chloride exposure were rated as “probably low” risk of bias and therefore potentially could have been incorporated into a meta-analysis, upon review there were limited studies (4 each) measuring exposure using varied metrics (environmental monitoring, emissions-based modeling, using occupation as surrogate for exposure) that were deemed too heterogeneous to combine into a meta-analysis.

All studies included in the meta-analyses measured PM exposure levels either through national or state air quality monitoring stations (i.e., US Environmental Protection Agency Air Quality System, California Air Resources Board monitor stations, or Taiwan EPA monitoring stations) or through historical emissions databases, combined with dispersion models to estimate residential levels of pollutants. Study population sizes ranged from 524–83,229. Study populations came from a variety of cohorts and regions, primarily in the U.S., such as the Nurses’ Health Study II, U.C. Davis’ Childhood Autism Risks from Genetics and the Environment (CHARGE) study, the Center for Diseases Control and Prevention’s (CDC) Autism and Developmental Disabilities Monitoring (ADDM) Network, Child and Adolescent Twin Study in Sweden (CATSS), or identified through insurance claims or state departments tracking ASD diagnoses. The majority of studies adjusted for maternal age, parental level of education, race/ethnicity, gender of child, household income, and some measure of socio-economic status. We contacted the corresponding author from four of the six included studies to request additional information and received responses from all four authors.

Table 2. Characteristics of included human studies on air pollution and ASD, by publication date and study design.

Study	Study design	Study population & location	Sample size	Exposure assessment	Outcome assessment
Windham et al. 2006 [55]	Case-control	Children born in 1994 in 6 counties in the San Francisco Bay area	284 children with ASD and 657 controls	Modeled concentrations of 29 hazardous air pollutants in 1996,[a] assigned by census tract of maternal residence at delivery (birth cert)	ASD cases ascertained from multi-source records-based surveillance of children, conducted by California CADDRE (within CDPH)
Roberts et al. 2007 [48]	Case-control	Children born between 1996±1998 in 19 counties in the Central Valley of California	465 children with ASD and 6,975 controls	Modeled concentrations of 54 pesticides applied between 1995±1998,[b] assigned by maternal residence of maternal residence at delivery (birth cert)	ASD cases identi®ed(by CDPH) from CA Dept of Developmental Services (DDS) ®les
Windham et al. 2007 [56]	Case-control	Children born between 1996±1998 in 4 counties in Southern California	3,400 children with ASD and controls frequency matched on last menstrual period in a 1:10 ratio	Modeled concentrations of 29 hazardous air pollutants in 1996,[a] assigned by census tract of maternal residence at delivery (birth cert)	ASD cases identi®ed(by CDPH) from CA Dept of Developmental Services (DDS) client ®les
Lewandowski et al. 2009 [45]	Ecological	Students in Texas school districts for academic years 2000±2001 through 2005±2006	7,022 children with ASD and 4,050,690 controls for 2001; numbers not reported for other years	Modeled concentrations of 11 toxic release pollutants in 2001 and of mercury only between 2000±2005,[c] assigned by school district	Prevalence of ASD and other special education categories obtained from the Texas Education Agency Academic Excellence Indicator System
Kalkbrenner et al. 2010 [43]	Case-control	Children aged 8 years in North Carolina (born in 1994 and 1996) and West Virginia (born in 1992 and 1994)	383 children with ASD and 2,829 children with speech and language impairment as controls	Modeled concentrations of 35 hazardous air pollutants in 1996,[a] assigned by census tract of birth residence	ASD cases and controls with speech and language impairment identi®edfrom records-based surveillance of children conducted by ADDM
Trousdale et al. 2010 [50]	Case-control	All children aged 8 years in the US (speci®caly in MD for sub-analysis) during school years 2004±2005 and 2007±2008	Not reported	Modeled concentrations of 34 hazardous air pollutants in 1996 and 89 hazardous air pollutants in 1999,[a] assigned over entire U.S (and for MD state by county in sub-analysis)	ASD prevalence calculated using data from the U.S. Department of Education, Of®ceof Special Education Programs and control numbers using data from the National Center for Education Statistics enrollment data (Maryland sub-analysis from Maryland State Department of Education)
Blanchard et al. 2011 [40]	Ecological	Students in Bexar County, TX (all ages) and Santa Clara County, CA (elementary school ages) in 2008	Not reported	Modeled concentrations of mercury in 2002,[a] assigned by city block-level school districts	ASD rates obtained from the Texas Education Association and from http://www.kidsdata.org for California
Volk et al. 2011 [51]	Case-control	Children enrolled in the CHARGE study and born between 1997±2006 in California	304 children with ASD and 259 typically developing controls	Distance to freeways and major roads as proxy for traf®c-related pollutant exposure; assigned by residential address during pregnancy and at birth	ASD cases identi®edfrom California DDS and children evaluated and diagnosed by study staff using the ADI-R and ADOS tools; controls were selected based on SCQ
McCanlies et al. 2012 [46]	Case-control	Children enrolled in the CHARGE study and born between 1998±2003 in California	93 children with ASD and 81 typically developing controls	Self-reported and industrial hygienist-assessed parental occupational exposures to 49 chemical agents from three months prior to conception through to either birth or weaning for breast-fed children	ASD cases recruited by California DDS and children evaluated and diagnosed using the ADI-R and ADOS tools; controls were selected based on SCQ
Becerra et al. 2013 [39]	Case-control	Children born in 1994±2006 in Los Angeles County, CA	7,603 children with ASD and 75,782 controls	Modeled concentrations of 6 pollutants between 1993±2006,[d] assigned by residential address at delivery/birth	Autistic disorder cases identi®edfrom records of California DDS

(*Continued*)

Table 2. (*Continued*)

Study	Study design	Study population & location	Sample size	Exposure assessment	Outcome assessment
Pino-Lopez and Romero-Asuyo 2013 [47]	Case-control	Children aged 12±36 months evaluated by the Early Intervention Service in Ciudad Real, Spain between January 2009 and February 2011	70 children with ASD and 136 unaffected controls	Self-reported parental occupation to evaluate exposure to solvents during pregnancy	ASD cases and unaffected controls identi®edthrough the Early Intervention Service of Ciudad Real
Volk et al. 2013 [52]	Case-control	Children enrolled in the CHARGE study and born between 1997±2006 in California	279 children with ASD and 245 typically developing controls	Modeled concentrations to traf®c-related air pollution between 1997±2008 and monitoring data for 4 pollutants using regional air quality data between 1997±2009,[d,e] assigned by self-reported residence history address during pregnancy and the ®rst year of the child's life	ASD cases identi®edfrom California DDS ®les,children evaluated and diagnosed by study staff using the ADI-R and ADOS tools; controls were selected based on SCQ
Windham et al. 2013 [57]	Case-control	Children born in 1994 in 6 counties in the San Francisco Bay area	284 children with ASD and 659 controls	Self-reported parental occupation on birth certi®cate, coded by occupational medicine-certi®edphysician to categorize broad chemical exposures	ASD cases ascertained from multi-source records-based surveillance of children conducted by California CADDRE
Jung et al. 2013 [42]	Cohort	Children aged less than 3 years in 2000 enrolled in prospective cohort study in Taiwan	342 children with ASD and 48,731 non-ASD controls	Modeled concentrations of pollutants between 1996±2009,[d] assigned by post-code levels in the 1±4 years preceding ASD diagnosis	ASD and non-ASD children in cohort identi®edbased on diagnosis codes provided in the Taiwan National Insurance Research Database
Roberts et al. 2013 [49]	Cohort	Children of Nurses' Health Study II participants born between 1987±2002 in the US	325 children with ASD and 22,098 controls	Modeled concentrations of 14 ambient hazardous air pollutants between 1990±2002,[a] assigned by census tract by mother's address approximately around the year of birth	ASD cases identi®edbased on Nurses' Health Study II participant's response to questionnaire, validated by administration of the ADI-R to a small, random subset of case mothers
Gong et al. 2014 [41]	Case-control	Twins born after July 1, 1992 and enrolled in the CATSS longitudinal study in Stockholm, Sweden	109 children with ASD and 3,051 healthy controls	Modeled historical emissions to estimate exposures for two pollutants (PM_{10} and NO_x) between 1992±2009, assigned by residential address during pregnancy, child's ®rstyear of life, and the year before ASD diagnosis	ASD cases and controls identi®edafter assessment using A-TAC tool at 9 and 12 years of age conducted by the CATSS
Shelton et al. 2014 [58]	Case-control	Children enrolled in the CHARGE study and born after 2003 in California	486 children with ASD and 315 typically developing children as controls	Modeled concentrations of 4 classes of pesticides between 1997±2008,[b] assigned by prenatal and birth residential address	ASD cases identi®edfrom California DDS ®les,children evaluated and diagnosed using the ADI-R and ADOS tools by study staff; controls were selected based on SCQ
Volk et al. 2014 [53]	Case-control	Children enrolled in the CHARGE study in California	251 children with ASD and 156 controls	Modeled concentrations to traf®c-related air pollution and monitoring data for 4 pollutants using regional air quality data between 1997±2009,[e,f] assigned by prenatal and birth residential address	ASD cases identi®edfrom California DDS ®les,children evaluated and diagnosed by study staff using the ADI-R and ADOS tools; controls were selected based on SCQ
von Ehrenstein et al. 2014 [54]	Case-control	Children born between 1995±2006 in Los Angeles County	768 children with ASD and 147,954 controls	Monitoring data for 24 hazardous air pollutants within a 5-km radius of birth address	Cases identi®edfrom California DDS ®lesof children served for autistic disorder

(*Continued*)

Table 2. (*Continued*)

Study	Study design	Study population & location	Sample size	Exposure assessment	Outcome assessment
Raz et al. 2014 [61]	Case-control	Children of Nurses' Health Study II participants born between 1990±2002 in the US	245 children with ASD and 1,522 controls	Modeled concentration from monitoring data for two pollutants (PM_{10} and $PM_{10-2.5}$),[f] Assigned by prenatal, pregnancy and birth residential address	ASD cases identi®edbased on Nurses' Health Study II participant's response to questionnaire, and validated by administration of the ADI-R to a random subset of case mothers
Kalkbrenner et al. 2015 [44]	Case-control	Children born in North Carolina in 1994 (8 counties), 1996 (8 counties), 1998 (9 counties), and 2000 (10 counties) and born in 6 San Francisco Bay area counties in 1996	645 children with ASD and 12,434 controls for North Carolina and 334 children with ASD and 2,232 controls for California	Modeled concentration of one pollutant from monitoring data,[f] assigned by birth certi®cate address	ASD cases identi®edfrom multi-source records-based surveillance of children conducted by the ADDM in North Carolina and California CADDRE
Dickerson et al. 2015 [59]	Ecological	Children 8 years of age in 2000, 2002, 2004, 2006 and 2008 from Arizona, Maryland, New Jersey, South Carolina, and Utah	4,486 children with ASD from 2489 census tracts	Modeled concentrations of 3 toxic release pollutants between 1991±1999,[c] assigned by census tract using residence at the time of surveillance (8 years of age)	ASD cases identi®edfrom records-based surveillance of children conducted by ADDM network
Dickerson et al. 2016 [60]	Ecological	Children 8 years of age in 2000, 2002, 2004, 2006 and 2008 from Arizona, Maryland, New Jersey, South Carolina, and Utah	4,486 children with ASD from 2489 census tracts	Modeled concentrations of 3 hazardous air pollutants in 1999,[a] assigned by census tract at the time of surveillance (8 years of age)	ASD cases identi®edfrom records-based surveillance of children conducted by ADDM network

[a] Data from US EPA National-scale Air Toxics Assessment (NATA);

[b] Data from California Department of Pesticide Regulation (DPR);

[c] Data from US Toxic Release Inventory (TRI);

[d] Data from nearest air monitoring stations;

[e] Data from CALINE4 dispersion model;

[f] Data from US EPA Air Quality System (AQS)

Abbreviations: ADDM, Autism and Developmental Disabilities Monitoring; ADI-R, Autism Diagnostic Interview, Revised; ADOS, Autism Diagnostic Observation Schedule; AQS, Air Quality System; ASD, autism spectrum disorder; A-TAC, Autism Tics, ADHD, and other Comorbidities inventory; CADDRE, Centers for Autism and Developmental Disabilities Research and Epidemiology; CALINE4, California Line Source Dispersion Model, version 4; CATSS, Children from the Child and Adolescent Twin Study in Sweden; CHARGE, Childhood Autism Risks from Genetics and the Environment; CDPH, CA Department of Public Health; DDS, Department of Developmental Services; NIOSH, National Institute for Occupational Safety and Health; SCQ, Social Communication Questionnaire; TRI, Toxics Release Inventory; USC, University of Southern California

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The initial meta-analyses of PM_{10} yielded a pooled OR = 1.20 (95% CI: [1.00, 1.42]). However, there was considerable statistical heterogeneity in the pooled estimate (I^2 = 77%, p-value = 0.007). Although the overall number of studies was small, leading to uncertainty in the heterogeneity estimate, the 95% confidence interval for the I^2 estimate (20–98%) suggested that heterogeneity was of concern. Using a leave-one-out analysis, we identified one study in particular [52] that was contributing to the majority of this heterogeneity. We contrasted design characteristics of this study with those of the other included studies to determine if the statistical heterogeneity could be explained by differences on a key characteristic; however, we concluded that the statistical heterogeneity was largely unexplained. We added in a hierarchical cluster structure to include this study as a separate cluster from all other studies, and this greatly reduced the unexplained heterogeneity (I^2 = 2%, p-value = 0.41), indicating that with this clustered analysis statistical heterogeneity was of minimal concern. Based on this clustered

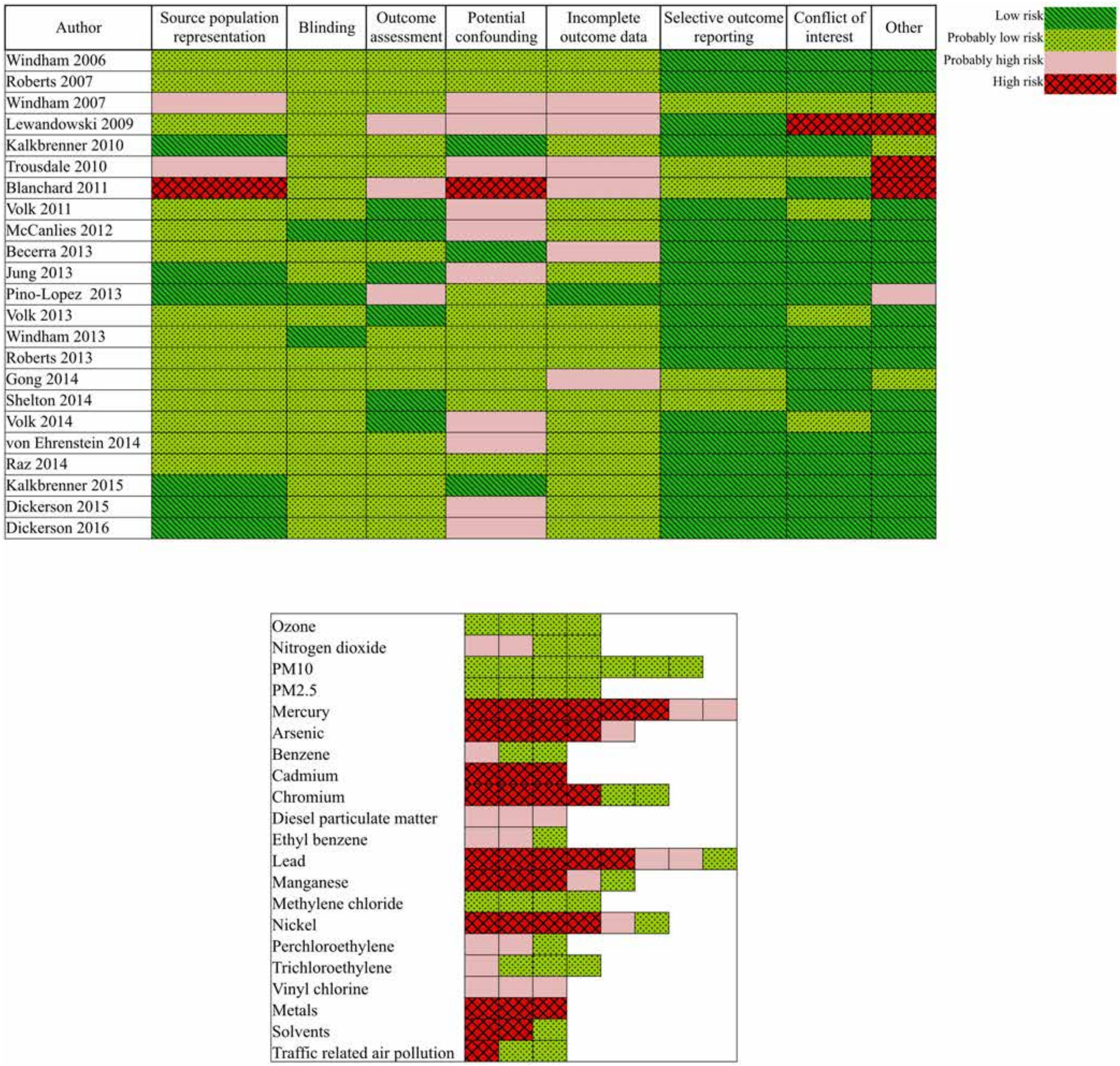

Fig 2. Risk of bias ratings for included human studies relevant to air pollution exposure and ASD outcome. A, All criteria except exposure assessment criteria. B, Exposure assessment criteria.

doi:10.1371/journal.pone.0161851.g002

meta-analysis, we found an overall effect estimate of OR = 1.07 (95% CI: [1.06, 1.08]) per 10-μg/m3 increase in PM_{10} (Fig 3).

We also conducted sensitivity analyses to assess the robustness of the positive relationship between PM_{10} exposure and risk for ASD. We estimated the hypothetical effect estimates needed from a single additional included study that would shift our meta-analysis to where: 1) the 95% confidence interval of the meta-analysis overlaps the value 1 (loses statistical significance), and 2) the summary effect estimate becomes less than 1. For both scenarios we assumed that the additional study would have a standard error of 0.04, equal to the smallest in our

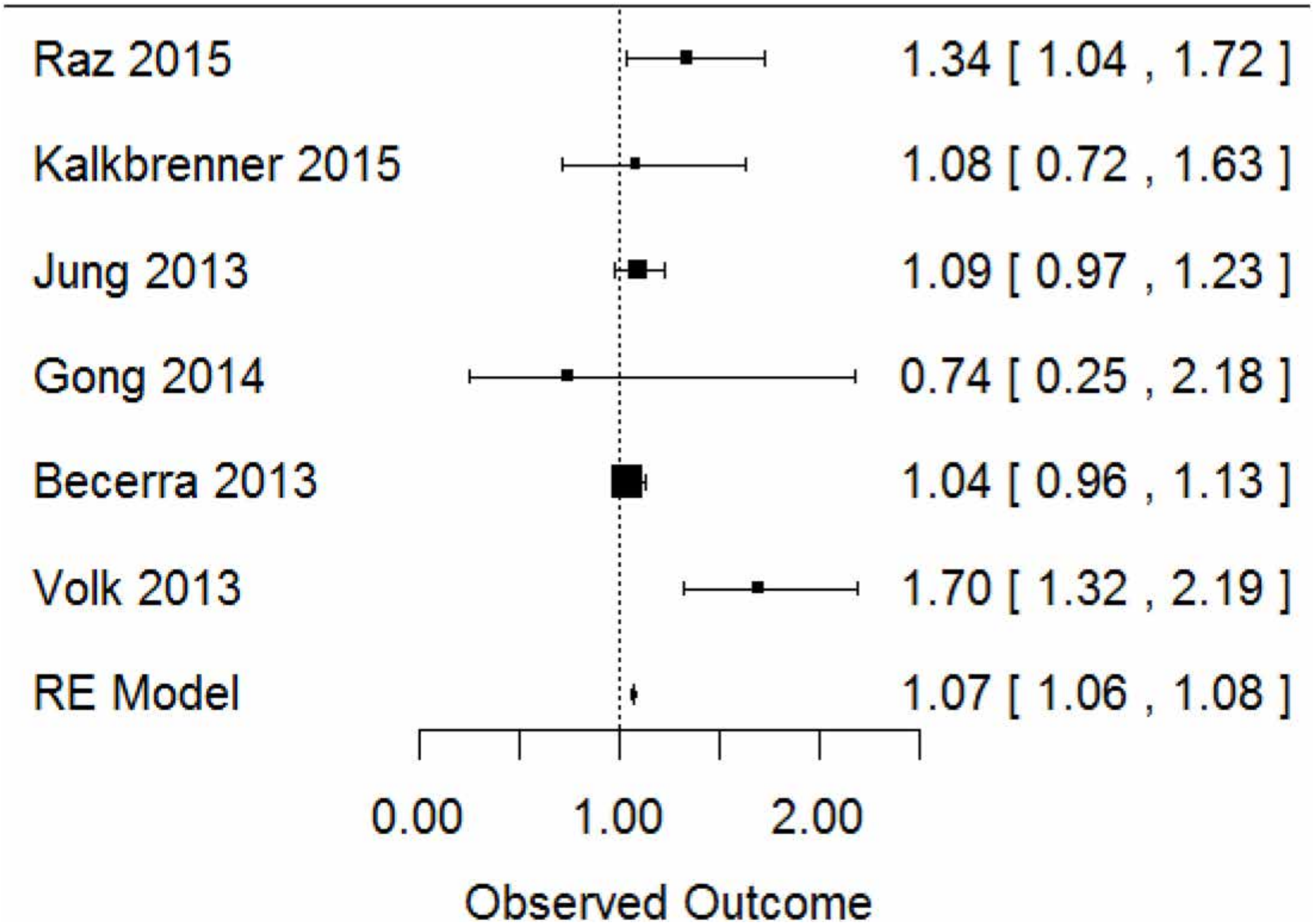

Fig 3. Meta-analysis of human studies; reported effect estimates [95% confidence interval] from individual studies (inverse-variance weighted, represented by size of rectangle) and overall pooled estimate from random effects (RE) model for PM_{10} exposure and ASD.

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group of studies [39]. We found that an additional new study would need to have an effect estimate of OR = 1.03 to enlarge our confidence interval to overlap 1, and OR = 0.44 to shift our overall effect estimate to OR<1. The former OR estimate is reasonably within the range of that reported by existing studies (thus, it might be possibly for a new study to easily shift our interpretation of statistical significance of the overall effect estimate), but the OR required to shift the overall effect estimate to OR<1 seemed rather unlikely.

For $PM_{2.5,}$ the summary effect estimate was OR = 1.88 (95% CI: [1.11, 3.20]) but, similar to PM_{10} analyses, we found evidence of considerable statistical heterogeneity (I^2 = 96%; p-value<0.0001). Again using a leave-one-out analysis, we identified one study in particular [39] that was contributing to the majority of the heterogeneity and we could not identify particular methodological features driving the heterogeneity so we classified it as unexplained. Adding in a hierarchical cluster structure here greatly reduced the unexplained heterogeneity (I^2 = 0%, p-value = 0.54), indicating that with this clustered analysis statistical heterogeneity was of minimal concern. Based on this clustered meta-analysis, we found a pooled effect estimate of OR = 2.32 (95% CI 2.15 to 2.51) per 10-μg/m3 increase in $PM_{2.5}$ (Fig 4). Sensitivity analyses here (assuming the added hypothetical study would have a standard error of 0.04, equal to the

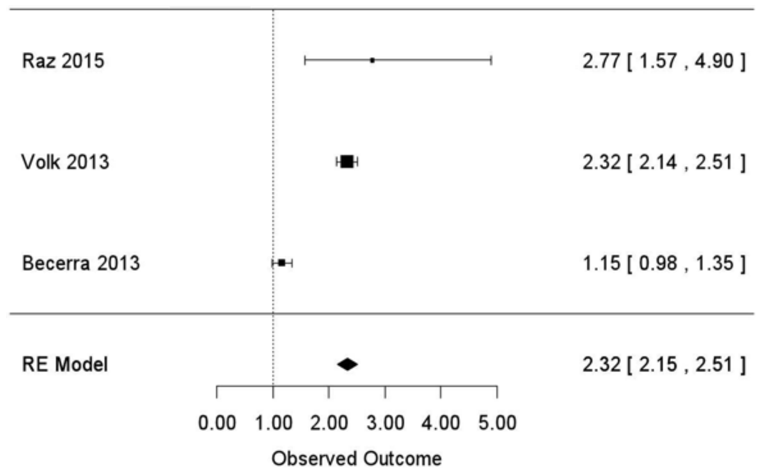

Fig 4. Meta-analysis of human studies; reported effect estimates [95% confidence interval] from individual studies (inverse-variance weighted, represented by size of rectangle) and overall pooled estimate from random effects (RE) model for $PM_{2.5}$ exposure and ASD.

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smallest in our group of studies [39]) found that an added study would need an effect estimate of OR = 1.02 to enlarge the confidence interval to overlap 1, and an OR = 0.14 to shift the overall effect estimate to OR<1. The former OR estimate is outside the range of that reported by existing studies, but not unreasonably so; however, the OR required to shift the overall effect estimate to OR<1 seems quite unlikely.

For all other air pollutant chemicals or classes of chemicals with effect estimates reported for ≥3 studies, we generated scatter plots of reported data (S1 Fig). Odds ratio (OR) or relative risk (RR) estimates were plotted on the log-transformed scale by chemical and separated by categories of "general air pollutants" (including diesel particulate matter, nitrogen dioxide, and ozone), "industrial chemical pollutants" (including benzene, ethylbenzene, methylene chloride, perchloroethylene, styrene, toluene, trichloroethylene, and vinyl chloride), "heavy metal air pollutants" (including arsenic, cadmium, chromium, lead, nickel, manganese, and mercury). We also created a scatterplot for "pesticide air pollutants,", combining all individual pesticides (e.g., acrolein) with grouped categories (e.g., organophosphates). We generally observed a trend towards positive effects (increasing exposure associated with increased autism risk), although there were limited data and confidence intervals commonly overlapped the null. The other air pollutants most consistently reporting statistically significant associations with of ASD included heavy metals with exposures assessed using NATA data. However, for many of these air pollutants, serious risk of bias existed for exposure assessment with one or more studies rated as "high" or "probably high" (Fig 2).

Quality and strength of the overall body of evidence

We rated the quality of the human evidence as "moderate." Our decisions leading to this rating were primarily based on the concern that many of the air pollutant chemicals or classes had exposure assessment methods that were rated "high" or "probably high" for risk of bias for exposure assessment methods (rated as between "0 to -1") (Table 3). There was insufficient number of studies to utilize funnel plot analyses to assess publication bias quantitatively; so we based our decision to not downgrade for publication bias on the fact that we conducted a comprehensive search, found studies from the grey literature, and found studies of variable sizes, designs, and funding sources that had similar findings. We found insufficient evidence to

Table 3. Summary of rating quality and strength of the human evidence.

Category	Downgrades	Rationale
Risk of bias (ROB)	0 to -1	We rated overall risk of bias across all studies between 0 (no downgrade) and -1 (downgrade 1 level). Our rationale was that many studies had probably high or high risk of bias, mostly driven by exposure assessment methods. The lack of speci®city across different pollutant classes was also a concern. Because of the heterogeneity in individual study ratings across all air pollutant contaminants, we found it impossible to assign one overall rating that would be relevant across all studies for all contaminants.
Indirectness	0	Exposures were not directly measured (lacking biomarkers or individual measurement of air pollutants); however this was accounted for in the ROB rating and no other areas of concern existed for indirectness.
Inconsistency	0	Effect estimates across studies were mostly positive (showing increased risk) and small (OR<2) and con®dence intervals overlapped across studies for the majority of estimates.
Imprecision	0	No concern regarding the imprecision in effect estimates across studies.
Publication bias	0	The number of studies included in the meta-analysis were too small (i.e., <10) for a statistical evaluation of potential publication bias. We identi®ed several ®ndings from the grey literature through our comprehensive search, and two studies did ®nd negative ®ndings.
	Upgrades	
Large magnitude of effect	0	All of the studies found null or minimal effects only (i.e., OR<2).
Dose-response	0	Coauthors felt there was some evidence of a dose-response relationship, but not enough to warrant upgrading of the evidence.
Confounding minimizes effect	0	There was no evidence that residual confounding in¯uenced results.
Overall Quality of Evidence	Moderate	Initial rating of ªmoderateº neither downgraded nor upgraded.
Overall Strength of Evidence	Limited	A positive relationship was observed between exposure and outcome where chance, bias, and confounding could not be ruled out with reasonable con®dence. Con®dence in the relationship is constrained by such factors as: the number, size, or quality of individual studies, or inconsistency of ®ndings across individual studies. With more information, the observed effect could change, and this change may be large enough to alter the conclusion.

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upgrade the body of evidence. Ultimately, although there was concern regarding the risk of bias domain, we did not judge this to be sufficient enough to warrant downgrading the evidence and therefore remained at the initial "moderate" rating.

We rated the final overall strength of the evidence as "limited" (Table 2). While the meta-analysis suggested statistically significant effects for PM, these were based on very few studies and sensitivity analysis suggested that even a single added study within the range of ORs reported in the body of research to date could lead to a summary effect evidence that did not attain statistical significance (although an extreme OR estimate would be require to change the direction of the overall effect estimate to below 1). Further the occurrence of heterogeneity that lacked a clear methodological rationale is suggestive of randomness in the available data, was concerning to review authors, and this was influential in our final rating of "limited" overall evidence.

Discussion

We conducted the first systematic review and meta-analysis of the body of human evidence to assess whether early life exposure to ambient air pollutants is associated with ASD. We concluded that there was "limited evidence of toxicity" for the association between early life exposure to air pollution as a whole and diagnosis of ASD. The strongest evidence supported an association between exposure to particulate matter and ASD. We utilized six robust studies (five case-control and one cohort) with minimal risk of bias concerns that represented a total of 9,557 children with autism and 143,997 controls reporting on PM_{10} exposure, and a subset of these that also reported on $PM_{2.5}$. These studies reported effect estimates similar enough to be combined into a meta-analysis we found statistically significant pooled effect estimates for both PM components, with a stronger effect demonstrated for $PM_{2.5}$ than for PM_{10}. We identified statistical heterogeneity in our meta-analysis that could be minimized through incorporating a clustered data analysis structure, but not explained through differences in study design. We determined through sensitivity analysis that future studies comparable to the ones included in our review (i.e., with similar effect estimates and uncertainty) could potentially change the strength of the relationship between PM and ASD estimated from the meta-analysis. In other words, the effect of particulate matter on ASD may be stronger or weaker than these results indicate. However, we found that it would be unlikely that a future study would change the direction of the association between PM and ASD, for example moving air pollution from being a risk factor for ASD to being health protective for ASD. Generally, the other air pollutants that could not be combined into a meta-analysis (such as some of the metal air pollutants) supported positive and statistically significant effects, although the effects were generally small and not consistent. Collectively, these findings led to our conclusion that positive relationships were observed between air pollution exposures generally and ASD, but that chance, bias, and confounding could not be ruled out with reasonable confidence because of the limitations present in the available data. This met our definition of "limited evidence of toxicity" for the association between early life exposure to air pollution generally and ASD.

Our systematic review and meta-analysis had several strengths. This is the first example of applying a systematic approach and combining evidence into a meta-analysis to evaluate the body of evidence for all types of air pollutants collectively. The Navigation Guide approach is based on the GRADE principles [22] for rating the quality and strength of the evidence and incorporates similar criteria and considerations for evaluation, with slight modifications for application to environmental health evidence—in particular, the lack of randomized control human trials. The GRADE approach requires judicious consideration of the contribution of each study to addressing the study question, with general guidance to focus on the high-quality studies—in this case the evidence supporting the link between PM and ASD. However, evidence from all other air pollutant studies were also considered when evaluating the quality and strength of the evidence—for instance, determining whether the studies overall contributed direct evidence to answer the study question or whether the study estimates demonstrated a large magnitude of effect.

Our systematic review results were generally in concert with previous expert-based narrative reviews that addressed the association of ASD and environmental chemical exposures more broadly [18, 65–67]. The only prior review of air pollutants and neuropsychological development that followed a prescribed systematic methodology was limited to published English language studies appearing during or after 2012 and did not include a meta-analysis [68]. The authors of this review rated evidence based on IARC classifications and concluded that there was "sufficient" evidence for an association of ASD with $PM_{2.5.}$ The Navigation Guide classifications are based largely on the IARC classifications. However, our review additionally included a meta-analysis, the results of which further demonstrated an association between air pollution (PM) and ASD, but also revealed some uncertainties in the body of evidence that would not have been apparent except for the meta-analysis. The unexplained heterogeneity in the meta-analysis led us to conclude that the body of evidence fit the definition of "limited toxicity" better than the definition of "sufficient toxicity."

Several newer studies have been published since the end-date of our search, including a European multi-site study combining different scale measurements of ASD symptoms, which reported no association between particulate matter or nitrogen dioxide and autistic traits [69] and two studies from a case-control sample in Pennsylvania, reporting an association between $PM_{2.5}$ and ASD [70] and between some air toxics and ASD [71]. We do not know for certain how these studies would impact our review results without a formal evaluation of their risk of bias and integration of these studies into the overall rating of quality and strength of the evidence. However, we note that the effect estimates reported for PM in these studies generally fall within the range of those reported by the studies included in our meta-analysis.

Together, these findings support the hypothesis that early life exposure to air pollution may contribute to ASD. While some studies have suggested a strong genetic heritability contribution to ASD development, these do not fully explain the recent increases in ASD prevalence and thus environmental risk factors are recognized as playing a strong contribution to the increase in ASD [72–76]. Exposure to chemicals in air pollution may act through several potential pathophysiological mechanisms related to immune function, endocrine disruption, and epigenetic alterations [71]. These mechanisms vary greatly by chemical air pollutant—for instance, particulate pollutants (complex chemical mixtures solely defined by size, such $PM_{2.5}$) are able to penetrate deep into the lungs and can enter blood circulation, where they can induce oxidative stress leading to inflammatory responses that subsequently result in the perturbation of neurodevelopment [77]. In contrast, other specific chemical constituents of air pollution, such as metals, may impact neurodevelopment of the developing fetus through direct exposure, or cause an elevation in inflammatory cytokines in the maternal circulation that subsequently impacts neurodevelopment [66, 78, 79]. While there are many outstanding questions about the nature and extent of the association of air pollution and ASD specifically, there is already strong evidence that certain air pollutants such as PM, lead, and mercury impact brain development [76, 80–82]. Our review identified research gaps that should inform future work on this topic. Further, we provide a concise statement of the strength of the evidence, which decision-makers and policymakers can use to integrate with other factors that are important in setting decisions, such as values and preferences about the outcome, alternatives to avoiding the outcome, and the costs and benefits of action.

Advancing ASD and Air Pollution Research

The limitations identified in this body of evidence reveal gaps in the current scientific literature and point the way for optimizing future research related to the environmental contributors to ASD specifically, and air pollution in general. In particular, future research should account for the following:

1. ***Challenges in assessing exposure to the complex mixture that defines air pollution.*** The available evidence was not readily combinable in a meta-analysis because air pollution exposure was assessed through many different and non-standardized metrics and included data on over 100 different chemical components or surrogate measures of air pollution. While the PM data and several other pollutants were rated as low risk of bias (i.e., ozone, methylene chloride), only the PM data was sufficient for inclusion in a meta-analysis. Many of the other air pollutant exposures were classified as probably high or high risk of bias, with some being based on surrogate measures (i.e., distance to freeway) or lacking accounting for time-activity patterns or spatial accuracy. Studies also varied widely in terms of the methods (monitoring, modeling, etc.) and data source (NATA, TRI, etc.) used to assess exposure. Most studies assigned exposure based on birth address, which implies prenatal exposure, but may not reflect addresses earlier in pregnancy. Notably, some studies improved on this by obtaining a residence history from participants; future studies could potentially utilize tracing services to gather this important information. This is a generic challenge that will likely be present for the majority of air pollution studies, but recent advances in assessment methods such as use of portable personal sensors and exposomic technologies could make a significant impact. To increase the usefulness of study results to decision-making,

investigators need to directly address these challenges and maximize the accuracy and applicability of exposure measurements to the target population.

2. ***Challenges related to study design.*** The included studies varied in the adjusted confounders considered, the timing of exposure or outcome measurement, and the method used for assessing the ASD outcome. Such heterogeneity makes it difficult to combine the results of multiple studies, as there is little empirical basis available to inform how variations in these study characteristics might impact the reported effect estimates. There is a need to establishing basic criteria for environmental studies of ASD based on an improved understanding of how different adjustment factors and timing or method of exposure and outcome in the same population impact effect estimated to maximize the potential for combining studies relevant to the same research question.

3. ***Challenges related to reporting.*** Many effect estimates were reported on different scales or categorized the exposures using different ranges. As such, with the exception of PM, where estimates could fairly easily be standardized, the body of evidence was disparate and largely not combinable. Future studies should increase availability of raw data or broaden reporting of effect estimates using different metrics to ensure statistical combinability in future meta-analyses.

4. ***Challenges related to unexplained heterogeneity.*** In meta-analyses, PM demonstrated statistically significant summary associations with ASD. We observed a larger summary effect estimate for $PM_{2.5}$ (OR = 2.32) than for PM_{10} (OR = 1.20). Because smaller particles have the ability to penetrate into the circulatory system and are thought to be more biologically active [83–86], this pattern of association is not unexpected. Alternatively, the larger effect for $PM_{2.5}$ could be related to the limited available sample of effect estimates as the Volk et al. [52] and Becerra et al. [39] studies, which generated the highest estimates for both PM_{10} and $PM_{2.5,}$ comprised two of three included studies for $PM_{2.5}$ but two of six included studies for $PM_{10.}$ There are possible study design characteristics that differed (for instance, the availability and quality of individual home addresses to estimate individual-level exposures), but it is difficult to assign an exact explanation for the observed heterogeneity in results. Unexplained heterogeneity is troubling but could be resolved with additional studies that could reduce overall variability of estimates and which provide insight into methodological reasons behind the heterogeneity.

5. ***The interplay between genes and the environment.*** One of the included studies reported an elevated risk of ASD only in those with a mutation in the MET tyrosine kinase receptor and those in the highest exposure level category [53]. While interactions between genes and the environment are known to play a key role in ASD, relatively few studies have been able to adequately power such analyses. The Volk et al. [53] study illustrates the importance of considering moderating variables to evaluate heterogeneity in examining ASD etiology. This will be an important consideration in future reviews as more data emerges on this topic.

Synthesizing the Evidence for Decision-Making in Environmental Health

Our case study of applying the Navigation Guide systematic review method to synthesize the science related to ASD and air pollution underscored several key directions for evidence-based decision-making in environmental health.

1. ***Use of novel tools for assessing risk of bias in air pollution studies.*** Due to the complexity of assessing the risk of bias of air pollution exposure assessments, we developed a novel risk of bias tool and piloted its use for this study. This tool is now available [87] and can be

adapted and implemented for evaluating exposure assessment of future systematic reviews and can also serve as a guide to strategically incorporate methods that reduce potential risks of bias in future air pollution studies.

2. ***The need for consistent and complete reporting of research results.*** Without the cooperation of individual study authors, it would have been impossible to complete the meta-analysis. This observation is consistent with our previous systematic reviews, [24, 27, 28, 30], and underscores the need for journal editors to routinely request consistent information from authors when manuscripts are accepted for publication to advance the capacity to conduct robust systematic reviews. To this end, several high-impact journals have already adopted the ARRIVE guidelines for animal studies (http://www.nc3rs.org.uk/ARRIVE/) [88, 89] or MOOSE guidelines for human observational studies [90]. Our experience supports these approaches, and we recommend reporting guidelines be expanded to consider other elements pertinent to high throughput *in vitro* studies and other types of experimental and observational evidence.

3. ***The need to mechanize and automate data synthesis.*** As with our previous systematic reviews, [24, 27, 28, 30], we found efficient ways to sort through a large number of studies captured through a broad search. Keys to this efficiency were implementing explicit predetermined inclusion/exclusion criteria and user-friendly software. We began the review using DRAGON software to perform title and abstract screening of studies but we found DistillerSR to be more flexible and easy to use and discontinued use of DRAGON. The development of increasingly capable software and other tools, including natural language processing and machine reading, [91] will be critical to advancing systematic reviews in clinical and environmental health.

4. ***Time sensitive nature of systematic reviews.*** We had completed our review but had not yet published our results when three new relevant studies were published [69]. This highlights the practice of establishing stopping dates for a review's literature search, which is essential for assessments in regulatory and policy decision-making. The nature of scientific information means knowledge is always evolving, yet it is important to use stopping dates to evaluate what is known in the scientific literature at that time and use the information to make a decision based on this knowledge at hand. In the future we recommend re-running the search immediately before data analysis and planning for a very short time between data analysis and the review team's rating of the quality and strength of the evidence. This will become increasingly feasible as more and more scientists are trained in the method and it becomes more efficient. It also highlights the need to conduct cumulative meta-analyses as more data become available, a common practice in the clinical sciences. A future research project could involve updating the search and investigating how new available studies might change our ratings and decision.

Conclusion

In summary, we conducted the first systematic review and meta-analysis of the literature on the association between ASD and air pollution and concluded that there was "limited evidence of toxicity." We found the strongest available evidence was supporting associations between PM and ASD, which was supported by the results of our meta-analysis. The available body of evidence on air pollution in general and ASD was wide, shallow, and except for PM, limited in their ability to strongly support a relationship if one exists. Our rating of the quality and strength of the evidence for this study question provides insight on the current state of the science but also the research gaps to address in future studies. Accurate measurement of human exposures to air pollutants during developmentally relevant time periods remains a key limitation in this research area, calling for continued work to improve air models, explore biomarkers, and pool and expand study samples to ameliorate effects from exposure measurement error. Identifying the environmental contributors to ASD and neurodevelopment in general is a critical unmet clinical and public health need, as recognized by a recent consensus statement published by leading scientific and medical experts, along with children's health advocates, which identifies the importance of environment as a risk factor for neurodevelopment [92]. Furthermore, the strength of the scientific evidence is but one component of decision-making and other factors such as the co-benefits of reducing air pollution exposures and the severity of potential health outcomes should be taken into consideration when making policy and regulatory decisions. Our research findings and recommendations can support researchers, clinicians, impacted individuals, families, communities, policy-makers, and funding agencies in expediting the scientific discovery in this field as well as advancing evidence-based decision-making on how to take action to prevent future harm.

Supporting Information

S1 Fig. Reported effects estimates scatterplots.
(DOCX)

S1 File. PRISMA Checklist.
(PDF)

S2 File. Instructions for making risk of bias determinations.
(DOCX)

S3 File. List of excluded studies.
(DOCX)

S1 Table. Database-specific search terms.
(DOCX)

S2 Table. Toxicological websites and grey literature databases searched.
(DOCX)

S3 Table. Data extraction fields and description.
(DOCX)

S4 Table. Factors for evaluating the overall quality of a body of evidence.
(DOCX)

S5 Table. Individual study characteristics.
(DOCX)

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Commentary

Pollution from Fossil-Fuel Combustion is the Leading Environmental Threat to Global Pediatric Health and Equity: Solutions Exist

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Abstract: Fossil-fuel combustion by-products are the world's most significant threat to children's health and future and are major contributors to global inequality and environmental injustice. The emissions include a myriad of toxic air pollutants and carbon dioxide (CO_2), which is the most important human-produced climate-altering greenhouse gas. Synergies between air pollution and climate change can magnify the harm to children. Impacts include impairment of cognitive and behavioral development, respiratory illness, and other chronic diseases—all of which may be "seeded" in utero and affect health and functioning immediately and over the life course. By impairing children's health, ability to learn, and potential to contribute to society, pollution and climate change cause children to become less resilient and the communities they live in to become less equitable. The developing fetus and young child are disproportionately affected by these exposures because of their immature defense mechanisms and rapid development, especially those in low- and middle-income countries where poverty and lack of resources compound the effects. No country is spared, however: even high-income countries, especially low-income communities and communities of color within them, are experiencing impacts of fossil fuel-related pollution, climate change and resultant widening inequality and environmental injustice. Global pediatric health is at a tipping point, with catastrophic consequences in the absence of bold action. Fortunately, technologies and interventions are at hand to reduce and prevent pollution and climate change, with large economic benefits documented or predicted. All cultures and communities share a concern for the health and well-being of present and future children: this shared value provides a politically powerful lever for action. The purpose of this commentary is to briefly review the data on the health impacts of fossil-fuel pollution, highlighting the neurodevelopmental impacts, and to briefly describe available means to achieve a low-carbon economy, and some examples of interventions that have benefited health and the economy.

Keywords: children's health; fossil fuel emissions; air pollution; climate change; neurodevelopment; benefits of intervention; policy

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1. Introduction

Children, and especially the poor, bear a disproportionate burden of disease and developmental impairment from both environmental pollution and climate change due to the combustion of coal, oil, gasoline, diesel and natural gas. Assessments of the health and economic costs of the impacts of fossil-fuel combustion by-products on children have typically been fragmented, published in specialized journals and have separately considered air pollution and climate change. This silo effect has precluded a full reckoning of the harm to children that results from a carbon-based economy and has stymied the advancement of properly comprehensive policies to protect this vulnerable group.

This commentary calls for a holistic accounting of the harm from fossil-fuel burning. Such an accounting is needed to spur the required global mitigation and action to reduce disparities between regions and socioeconomic classes and address the growing threat to future generations. Unless we act forcefully right now, our children and theirs will inherit an unsustainable world, lacking the essential ecological resources and social stability to support them. A major theme of this commentary is environmental injustice: the disproportionately heavy health and economic burden that falls on the young, the poor, and certain minorities, especially those in developing countries who are most vulnerable to the impacts of toxic air pollutants as well as CO_2-driven climate change resulting from the combustion of fossil fuel. Alleviating this burden would bring great and lasting benefits to children and their progeny. In his Encyclical, *Laudate Si*, Pope Francis concluded that global capitalism, based on the burning of fossil fuels, has created unsustainable consumption and mounting inequity. This warning was also sounded in the recent report of the Lancet Commission on Health and Climate Change [1]. To paraphrase the Lancet Commission: government policies and other strategies to reduce dependence on fossil fuel and build sustainable communities represent the biggest opportunity of our century to improve public health, redress inequality and increase the resilience of individuals, communities and the broader society.

In a prior commentary [2], I envisioned fossil fuel as the modern day version of the many-headed Hydra in Greek mythology, inflicting multiple forms of health and developmental harm in children through its emissions of toxic pollutants and carbon dioxide (CO_2). Were we to slay the hydra by transitioning to sustainable and renewable energy sources for transport, electricity generation, and industry, we would reap lasting benefits for children. These include significantly fewer cases of babies born preterm or with low birth weight, children with cognitive and behavioral disorders, mental-health problems, asthma and other respiratory illness, and potentially cardiovascular disease and cancer—all of which have been linked to toxic air pollutants. Mitigation of climate change would mean fewer children suffering from heat-related disease, malnutrition, infectious disease, physical and psychological trauma, mental-health problems, and respiratory illness. All of these health benefits would occur immediately and play out over the life course, since exposure-related damage, disease, or impairment in early life can affect long-term health and functioning. Of growing concern are the adverse effects on early brain development, impairing children's ability to learn, hence their future economic productivity and ability to contribute ideas and energy to society. As a result, they, their families and the broader community are less resilient ("able to survive, adapt, and grow in the face of stress and shocks and transform when conditions require") [3]. Society becomes even less fair, since the children who are most affected are the poor and disadvantaged.

This commentary builds upon a prior review by this author that contains more detail and references [2], updating it with more recent or additional scientific and economic data on the current and projected health impacts of fossil-fuel combustion. A major focus here is on the neurodevelopmental impacts of air toxics and climate change and their combination, because heretofore the effects on the developing brain have been less recognized than the other health impacts of these exposures.

2. The Young Are Especially Vulnerable to Air Pollution and Climate Change

The developing fetus and young child are more biologically and psychologically vulnerable than adults to the many adverse effects of toxic air pollutants and climate change from fossil-fuel combustion. This differential susceptibility is due to their rapid growth, dynamic developmental programming, immature detoxification, immune, and thermoregulatory systems, and their dependence on adult caretakers. The complexity of early development conveys vulnerability to disruption by toxic exposures of all kinds, including toxic pollutants and stress. Like the respiratory and immune systems, the brain continues to develop during infancy through childhood; however, the prenatal period is considered to be the most dynamic. For example, between conception and birth the brain undergoes highly synchronized maturation from the initial formation of the neural tube to the proliferation and migration of the neurons, synaptogenesis, apoptosis or "pruning" of synapses, and the early phase of myelination [4]. Most of the more than 86 billion neurons of the mature brain are formed during the prenatal period [5]. Numerous studies demonstrate that the fetal and early childhood stages are especially vulnerable to both genetic damage and epigenetic dysregulation from exposure to xenobiotics and stress; these molecular effects may have lifelong and transgenerational consequence [6–8].

In addition, because children breathe more air per kilogram of body weight than do adults and require three to four times the amount of food on a body-weight basis than adults, they are more exposed to pollutants in air and food [9,10]. Testifying to the differential vulnerability of the young, the World Health Organization (WHO) has estimated that more than 40% of the burden of environmentally related disease and more than 88% of the burden of climate change is borne by children under 5, although that age group constitutes only 10% of the global population [11–13]. The most serious impacts of climate change are occurring in developing countries; however, the entire global population is affected.

3. Fossil-Fuel Combustion is the Major Source of Global Air Pollution and CO_2

Globally, the majority of air pollution is generated by the combustion of fossil fuel (coal, diesel fuel, gasoline, oil, and natural gas) for electricity production, heating, transportation, and industry [14]. Worldwide, in 2011, fossil fuels represented 82% of the total primary energy supply [15]. In the US, oil, natural gas, and coal account for 81% of current fuel use [16]. Energy-related fossil-fuel combustion in high- and middle-income countries and biomass burning in low-income countries accounts for most of the global air pollution, generating 85% of airborne respirable particulate pollution and almost all sulfur dioxide and nitrogen oxide emissions to the atmosphere [17]. Also emitted are black carbon, polycyclic aromatic hydrocarbons (PAH), nitrogen and sulfur dioxides, mercury, and volatile chemicals that form ground-level ozone (O_3). All are associated with multiple adverse health effects in children.

Air pollution affects practically all countries in the world and all parts of society; only one person in 10 lives in a city that complies with the WHO Air quality guidelines [18]. Household air pollution is an important risk factor for an estimated 2.9 billion people worldwide, especially those in low- and middle-income countries where biomass fuels and coal are commonly burned for cooking and heating [19] In total, about 2 billion children live in areas that exceed the WHO annual guideline for fine particles of 10 $\mu g/m^3$ (the concentration of fine particulate matter that constitutes a long-term inhalation hazard) [20]. About 300 million children currently live in areas where outdoor air pollution exceeds international guidelines at least six-fold [21]. These guidelines are likely underestimates, since recent epidemiological studies have reported impacts below these levels.

Globally, although it remains a very important risk, indoor air pollution has been on the decline in recent years due largely to the reduction in use of solid fuels for cooking from around 60% of homes in 1980 to 42% in 2012 [22]. In contrast, ambient air pollution has shown a dramatic rise. Urban ambient air pollution increased by about 8% between 2008 and 2013; and the upward trend is projected to continue [21].

Fossil-fuel combustion is also the major human source of the greenhouse gases and short-lived climate pollutants that drive climate change. As stated in a recent US interagency report: "Many lines of evidence demonstrate that human activities, especially emissions of greenhouse gases, are primarily responsible for the observed climate changes in the industrial era, especially over the last six decades" [23]. Human activities emit about 35 billion metric tons of carbon dioxide into the atmosphere every year, primarily from energy use [24]. The Annual Greenhouse Gas Index, which is used by the National Oceanic and Atmospheric Administration (NOAA) to track the warming influence of long-lived climate-altering greenhouse gases, increased by 40% from 1990 to 2016, with most of that increase attributable to rising CO_2 levels [25]. CO_2 levels in the atmosphere are at their highest in 800,000 years [26]. In the US, coal and natural gas are the largest contributors to carbon pollution (constituting a third of all domestic carbon emissions). Methane released by production of natural gas, oil, and coal is second in importance. Although natural gas emits significantly less toxic air pollution and CO_2 than the other fossil fuels [27], the drilling, extraction, and transportation of natural gas results in the leakage of methane that is 34 times more effective than CO_2 at trapping heat over a 100-year period [28]. Given its increasing share of total fuel use, natural gas is expected to surpass coal as a source of energy-related CO_2 emissions in the US [29].

According to the International Energy Agency, worldwide growth in coal consumption is predicted to decline between 2015 and 2021 as developed countries continue to abandon coal as an energy source and China's consumption plateaus. However, that decline will be offset by growing demand among emerging nations, particularly in India and south-east Asia. Based on the assumption of business as usual, the US Energy Information Administration predicts that coal will remain the second-largest energy source worldwide, following petroleum and other liquid fuels, until 2030; and from 2030 through 2040, it will be the third-largest energy source, surpassed only by liquid fuels and natural gas [30]. In the US and some other countries, carbon emissions from cars and trucks have exceeded carbon emissions from electric power [31].

4. Fossil-Fuel Combustion-Related Air Pollutants and Climate Change are a Major Cause of Environmental Injustice

A major environmental injustice is that children, who are dependent on adults and did not create the problems, bear the brunt of the impacts of air pollution and climate change. It is a further injustice that children in low- and middle-income countries as well as lower-income communities and communities of color in high-income countries like the US are disproportionately affected. Echoing an earlier WHO finding, the recent report of the Lancet Commission on Pollution and Health, on which this author was a commissioner, stated that pollution in all its forms disproportionately affects the poor and marginalized in every country worldwide, with air pollution being the largest contributor to pollution-related deaths, mainly in low- and middle-income countries [32]. Low- and middle-income countries in the WHO South-East Asia and Western Pacific Regions had the greatest air pollution-related burden in 2012, with a total of 2.6 million deaths related to outdoor air pollution and 3.3 million deaths linked to indoor air pollution [33]. However, in contrast to pollution-related deaths, which largely occur among adults over 60, disability-adjusted life years (DALYs) resulting from pollution-related disease are highly concentrated among infants and young children, reflecting the vulnerability of the young and the many years of life lost with each death of a child [34].

Two factors contribute to the socioeconomic disparities in the impacts of air pollution: differential exposure and heightened susceptibility. Globally, there is a notable pattern of disproportionate exposure of the poor and of certain racial/ethnic groups to air pollution. Studies have shown that low-income communities and communities of color in the US experience disproportionately high exposure to particulate air pollution and air pollution from coal-fired power plants [2]. A GIS-based study of over 150,000 children in the US found that the distribution of three stationary and mobile air-pollution sources followed a consistent pattern of racial inequity, with Hispanic and black children facing significantly higher levels of potential exposure than white children [35]. With respect to heightened susceptibility, an analysis of the Mexico City scenario concluded that low socioeconomic status (SES) children in that megacity are not only exposed to high levels of pollution buttend to have inadequate nutrition and deficient schools, and often face domestic, school and street violence . Because these co-factors make children more vulnerable to air toxins, the significant impact of high air pollution is likely affecting predominantly low SES children in Mexico City [36].

The same factors contribute to disparities in the impacts of climate change. It is the poor who are most often forced to live in areas that are especially vulnerable to extreme flooding, drought and other impacts of climate change. Poor children are also less buffered and less resilient in the face of climate change: "a child living in poverty or deprived of adequate water and sanitation before a crisis will be more affected by a flood, drought or storm, less likely to recover quickly and at even greater risk in a subsequent crisis" [37]. Pre-existing inadequate nutrition, lack of adequate social support, and psychosocial stress due to poverty magnify the effects of both climate change and air pollution. Worldwide, the number of children living in poverty is staggering: one billion children, almost half of the 2.2 billion children below 15 years of age, are living in poverty [38]. In the US, the world's most prosperous country, the child poverty rate is a shocking 22%. As will be discussed, our research following parallel birth cohorts in the US and Poland has found that co-exposure of pregnant women to air pollution and social stress or hardship due to poverty significantly increases the adverse effect of air pollution on children's IQ and behavioral problems including attention deficit hyperactivity disorder (ADHD) [39–41]. For example, our research in Poland found evidence of significant interactions between maternal demoralization during pregnancy, itself correlated with material hardship, and prenatal air-pollution exposure on children's behavioral problems [41]. In our NYC research, combined prenatal exposure to PAH and material hardship was associated with a significant reduction in the IQ of children [39].

5. The Health Impacts of Air Pollution in Children Include Mortality and Neurodevelopmental Problems

The Lancet Commission on Pollution and Health [32] noted that air pollution remains one of the great killers of our age, echoing an earlier conclusion by the WHO that: "Air pollution, both ambient (outdoor) and household (indoor), is a public health emergency" and the biggest environmental risk to health. Most of the attention has been on excess mortality in the overall population, with the largest number of deaths in adults [33,42]. However, in children under 5 years' old, 1.7 million deaths are attributed to pollution and environmental risks in general; with air pollution linked to 600,000 of these deaths each year, largely due to pneumonia [37,43]. Because of the increasing trend in outdoor air-pollution levels, according to the Organization for Economic Co-operation and Development (OECD), under-5 mortality could be 50% higher than—or even double—current estimates by 2050 as a result of outdoor air pollution [22]. It is predicted that by 2050 outdoor air pollution will become the leading cause of child death [37]. However, millions more children are affected by chronic illness, including respiratory illness other than pneumonia and effects on physical and cognitive development [37].

A previous commentary by this author reviewed in some detail the documented effects of combustion-related air pollution on multiple outcomes including adverse birth outcomes, respiratory illness, and cancer [2]. Here the focus is on neurodevelopmental impacts, an area that has been under-recognized. Low birth weight and preterm birth will be briefly discussed in the context of child neurodevelopment.

A growing body of evidence indicates that early-life exposure to combustion-related air pollutants adversely affects children's cognitive and behavioral development. There are inconsistencies in the results among studies that can be explained in part by differences in measures used and levels of air pollution, in race/ethnicity, potentially in susceptibility, and in methods to assess children's neurodevelopment.

Particulate matter (PM) and traffic-related pollutants: Many studies have assessed the associations between PM or traffic-related air pollutants and cognitive outcomes in children from cohorts in the US, Europe and Asia [44–54]. The studies relied on estimates of exposure to PM or traffic-related pollutants (black carbon or nitrogen dioxide/NO_2) largely based on land-use regression models, distance of the maternal residence during pregnancy to roadways, or traffic density. Although the results for PM have been mixed, most of the studies on traffic-related pollutant exposure have reported associations with decreased mental and psychomotor development. Several studies found that the two traffic-related pollutants were associated with reductions in children's memory and IQ, after sociodemographic factors were taken into account [52,53]. Exposure to traffic pollution in childhood has also been linked to slower brain maturation [55].

With respect to attention problems, a study in Boston found more such problems with higher exposure to fine, respirable particulate matter having an aerodynamic diameter of less than 2.5 microns ($PM_{2.5}$), during the sensitive periods of gestation, with differences between boys and girls [56]. Studies in South Korea and Japan also reported more attentional problems with higher prenatal exposure to PM or other pollutants [57,58]. The association with prenatal exposure to traffic-related air pollutants was not observed in German cohort studies; but there was a significant association between higher $PM_{2.5}$ levels at the children's current addresses and increased hyperactivity/inattention scores [45].

Air pollution, mainly $PM_{2.5}$ or traffic-related pollution, has been associated in a number of studies with autism spectrum disorder (ASD) [59–63]. In contrast, European studies reported no association with autistic traits [64,65]. Although studies have not all been consistent, there is growing evidence that prenatal exposure to traffic-related air pollutants and $PM_{2.5}$ may be risk factors for ASD [66–68].

There is some evidence using molecular biomarkers and magnetic resonance imaging (MRI) of the brain that chronic exposure to airborne pollutants in the early years may contribute to neurodegenerative disease processes including Alzheimers's disease [69,70]. In Mexico City, researchers have reported distinct brain changes that have been associated with adult neurodegenerative disease in children living in high air-pollution areas [69].

Polycyclic aromatic hydrocarbons (PAH): PAH are a class of neurotoxic air pollutants that my colleagues and I have studied with respect to cognitive and behavioral outcomes and mood disorders in complementary cohort studies in New York City, Krakow, Poland and Chongqing, China [39,71–88] In our NYC cohort, prenatal exposure to PAH measured by personal air-monitoring or biomarkers of PAH exposure in cord or maternal blood was associated with developmental delay, reduced IQ, symptoms of anxiety, depression, and inattention, ADHD, deficient maturation of emotional self–regulation capacity, and poorer social responsiveness in childhood. Significant interactions were observed between prenatal PAH and material hardship due to poverty on child IQ [39] and between prenatal PAH and maternal psychological distress on mood-related problems [2]. More recently, significant combined effects of PAH and material hardship have been observed on ADHD outcomes [40]. MRI brain imaging of a subset of children in the NYC cohort showed significant correlations between measures of prenatal PAH exposure and distinct anatomical changes [80].

An example of the neurodevelopmental benefits of reducing air-pollution levels is provided by our research in Tongliang, China, that compared a cohort born before the closure of a centrally located coal power plant to a cohort conceived after plant closure. The second cohort had more favorable birth and neurodevelopmental outcomes, significantly lower cord blood levels of the biomarker of exposure measured in cord blood (PAH-DNA adducts), higher levels of a protein important in early brain development known as brain-derived neurotrophic factor (BDNF), and longer telomeres, a general marker of health [89,90].

In addition to their immediate toll, preterm birth and low birth weight are known risk factors for a number of neurodevelopmental disorders in children [91]. Recent studies confirm the reproductive effects of air pollution, their socioeconomic and racial disparities and the large cost imposed on affected families, furthering socioeconomic inequality and increasing the risk of neurodevelopmental effects in vulnerable populations. For example, a large prospective study in China found a significant increase in preterm birth with each 5 $\mu g/m^3$ increase in maternal exposure to $PM_{2.5}$ during the pregnancy [92]. A recent analysis found that about 2.7 million premature births per year (18% of preterm births) globally are associated with $PM_{2.5}$ exposure, including from fossil-fuel and biomass burning, mostly in developing countries [93]. A recent multi-country study concluded that, across all study populations, maternal exposure to particulate pollution was associated with low birth weight at term [94]. Racial disparities exist. In the US, preterm birth rates are 7.4% among Non-Hispanic white infants compared to 17.2% for Non-Hispanic black infants. Both social and physical environmental factors contribute to these disparities [95]. These effects are costly to society and individuals. In the US alone, $PM_{2.5}$ caused an estimated 15,000 preterm births in 2010, costing about $5 billion in medical care, special education services and lost economic productivity for that single year's cohort [96].

6. Climate Change is Linked to Serious Health Impacts in Children Including Mortality and Developmental Impairment

As noted in the Introduction, references for this section can be found in [2]. Increased illness, injury, and deaths from heat stress, floods, drought, and increased frequency of forest fires and intense storms are among the direct effects of climate change. The indirect effects include malnutrition and under-nutrition, the spread of infectious-disease vectors, food insecurity, illness due to increased air pollution and aeroallergens, and mental ill health from displacement, social and political instability. Children are especially vulnerable to both the indirect and direct consequences of climate change.

There is broad scientific agreement that climate change has already taken a significant toll on children and that the impacts will increase dramatically unless forceful action is taken. The WHO estimated that climate change since the mid-1970s contributed to about 5 million lost DALYs world-wide in 2000 through malnutrition, diarrhea, and malaria, mostly in children and in developing countries. The toll is expected to rise to 175 million children affected each year in the next several years. These numbers are substantial underestimates since they reflect only a few of the health effects from climate change.

Children bearing the greatest burden of climate-sensitive diseases are those living in regions with the least capacity to adapt to risks—regions that have contributed the least in terms of global emissions of greenhouse gases. Although children in developing countries bear the brunt, the impacts of climate change are increasingly being seen in the US and Europe, especially among populations of low socioeconomic status.

Virtually all of the impacts of climate change can affect children's neurodevelopment, cognitive functioning, behavior, and mental health, either directly or indirectly. Further, as with air pollution, the impacts are likely to be felt over the lifetime, affecting resilience, health and productivity. Climate change impacts the development of children's brains in many ways. Malnutrition during the first 1000 days causes stunting of the brain and body, with associated reduced neurodevelopmental and cognitive function in children and subsequent decreased ability to learn and be economically productive [97]. In 2017, 155 million children under five (1 in 4 children) are stunted due to hunger [98].

Stress from extreme weather events also contributes to neurodevelopmental and mental health problems in children. Although no single extreme weather event, such as floods, droughts, wildfires, or hurricanes and cyclones, can be attributed directly to climate change, human-induced climate change is contributing to the frequency and severity of such events. Over the last several decades, there have also been more intense and frequent heat waves as well as marked regional changes in floods, droughts and wildfires in certain parts of the US and globally [99]. An estimated 66.5 million children world-wide were directly affected by weather-related disasters every year from 1990–2000, of whom 600,000 died. Sea-level rise due to global climate warming has made coastal storms increasingly dangerous for coastal infrastructure and inhabitants, contributing to deaths from drowning. Drowning is a major cause of fatality in children in developing countries. According to a recent study, rates of sea-level rise between 1993 and 2011 exceeded by 60% the highest projections made in 2007 by the Intergovernmental Panel on Climate Change. In 2017, the NOAA estimated that the sea-level rise by the end of this century could reach as high as 6.5 feet, enough to inundate many waterfront cities around the globe [100]. Notable extreme weather events in the last decade include the massive flooding across south-east Asia in 2011 which affected an estimated 9.6 million people, many of them children [101]. In the US, Hurricane Katrina in 2005 forced 1 million people in New Orleans from their homes and left 372,000 children without schools; and Hurricane Sandy in 2012 affected people in 8 countries including 24 states in the US, with particularly severe damage in New Jersey and New York. Children who were affected by Hurricane Katrina were found to have higher rates of anxiety and depression [102]. Most recently, in September 2017 Hurricane Irma devastated islands in the Caribbean; and massive flooding in South Asia placed almost 16 million children in urgent need of life-saving support [103]. While populations of all economic statuses have been impacted, these events have most seriously affected the children in low-income communities.

The psychological and emotional impacts of climate change include the acute, traumatic effects of extreme weather events, mental and emotional distress resulting from direct experience or anxiety about future risks, chronic stress from heat, drought, forced migrations, and climate-related conflicts, and the stress of adjustment in the wake of weather-related disasters. Migration and population displacement as a result of social and political instability due to climate change affects the mental health of children in low-income, developing countries, contributing to the perpetuation of poverty and civil unrest. These countries, in which children less than 18 years' old represent 50% of their population, already bear most of the global burden of poverty and childhood disease.

7. Prenatal or Childhood Exposure to Air Toxics and Climate Change Can Have Long-Term and Synergistic or Combined Health Impacts

By launching a trajectory of adverse effects following the initial physical or developmental impairment, and/or by "seeding" latent disease that only becomes evident in later life, toxic air pollutants can affect health and functioning over the life-course. For example, adverse reproductive outcomes associated with *in utero* environmental exposures are risk factors for neurodevelopmental, respiratory, and other health problems in infancy and childhood, as well as heart disease, chronic obstructive pulmonary disease, and diabetes in adulthood. As noted above, childhood ADHD and ASD have been associated with early-life exposure to air pollution; these disorders may persist into adulthood, affecting professional and personal life and increasing the costs of healthcare for individuals and families. There is empirical evidence that early childhood exposure to air pollution is linked to lower scores on IQ and other intelligence tests, with long-term economic consequences in terms of lifetime earnings.

Similarly, the impacts of climate change can also play out over a child's lifetime. As noted, stunting of children's bodies and brains due to malnutrition during the first 1000 days results in impairment of cognitive functioning and learning. Early adversity and toxic stress are also linked to impairments in learning, behavior, and physical and mental health.

There is an increasing body of evidence that early-life exposures to air pollutants, nutritional deprivation, and stress can result in transgenerational impacts, possibly via the transmission of epigenetic changes. Combustion-related PAH have been shown to alter epigenetic marks in newborns, potentially dysregulating genes involved in disease pathways.

Although such effects have not been adequately documented, there can be harmful synergy or combined effects of toxic air emissions from the burning of fossil fuels and climate change. In California, during the month of November 2017 toxic air pollution from 22 concurrent fires has affected millions of people, adding to the pollution from traffic and stationary sources [104]. As another example, the effect of air-pollution exposure during pregnancy on risk of preterm birth is likely to be magnified by concurrent experience of extreme temperatures, food insecurity and stress due to climate change. The same is true for effects of co-exposure to air toxics, malnutrition and stress on child neurodevelopment. Children affected by malnutrition are likely to be more vulnerable to the neurotoxic effects of air pollution; and children born with low birth weight or preterm due to air pollution will be at greater risk of malnutrition or infectious disease.

8. Economic Benefits of Action are Underestimated but Significant

Lacking in the literature and in the public understanding is a holistic assessment of the economic costs of the many impacts of fossil-fuel combustion on children's health, hence the full economic benefits of action to reduce or prevent these impacts. The numbers below do not capture the full costs to individuals, families and society in terms of direct medical costs, costs to healthcare systems, opportunity costs resulting from lost productivity, and lower economic growth. However, even the limited available estimates of the monetary costs of deaths and morbidity from air pollution or climate change, individually, are staggering. The available data indicate that reduction of dependence on fossil fuel will bring very large economic benefits. They demonstrate the false dichotomy between regulation of air pollution and economic growth. Estimates of the avoided and avoidable economic health costs of air pollution and climate change include the following:

- Avoided health costs attributed to the US Clean Air amendments: ~$2 trillion for the year 2020 [105];
- A prediction of ~$250 billion/year by 2030 ($140 billion to $1050 billion) in avoided health costs from clean energy policies in the US [106];
- An estimated $361 to $886 billion/year in health costs due to US fossil fuel electricity [105,107];
- An estimated cost of ~$187 billion/year due to air pollution from coal combustion in the US [108];
- Gains in lifetime earnings related to a hypothetical 25% reduction in PAH in NYC air: $215 million for each annual NYC birth cohort of Medicaid births [109];
- An estimate of $3.5 trillion/year in costs of ambient air pollution in OECD countries, India and China [1];
- Total annual costs of air pollution currently estimated to be approximately 0.3 per cent of global GDP and expected to increase to approximately 1 per cent of GDP by 2060 [110];
- Reductions in airborne particulate matter between 2001 and 2010 in Taiyuan, Shanxi province, China associated with 2810 fewer premature deaths, 31,810 fewer hospital admissions, 141,457 fewer outpatient visits, 969 fewer emergency department visits, 951 fewer cases of bronchitis and more than 30,000 fewer DALYs attributed to air pollution in Taiyuan in 2010 compared to 2001. The decrease in the estimated cost of premature death due to air pollution: 3.83 billion Yuan, or approximately $621 million USD [111];
- Estimated cost of deaths from air pollution to the global economy: about $225 billion in lost labor income and more than $5 trillion in welfare losses in 2013 (World Bank/Institute for Health Metrics and Evaluation study, cited in WHO, Clear the Air) [112];
- Estimated ~$14 billion cost due to six climate-change-related events in the US between 2002 and 2009 [113];
- The expected benefits of the California climate change program: a $76 billion increase in the state's Gross State Product, a $48 billion increase in real household incomes, and the creation of 403,000 new efficiency- and climate-driven jobs [114];
- Between 1980 and 2017, the cost of 208 extreme weather and climate events in the US: at least $1 billion each, with total damages of more than $1.1 trillion, and a similar increase in these costly events happening around the world [115];
- The estimated global cost of climate change from deaths and diseases such as diarrhea, malnutrition, malaria, and heat stress up to $4 billion per year by 2030 [116];
- Globally, up to $230 billion of avoided external health costs each year by 2030 with an increase to 36% renewables in global energy consumption by 2030 [1].

9. Solutions Exist and Interventions are Being Mounted

Means are at hand, and already at work in many communities, cities and countries to transition from dirty fossil fuels to clean energy. This transition is seen by experts as both the major challenge and the major opportunity of our time [1]. Expert groups have underscored the feasibility of seizing this opportunity, citing policies and initiatives that have been effective. They have also underscored that to be successful and benefit the health and future well-being of children worldwide, the transition from dirty fossil fuels to clean energy must be done equitably and inclusive of all communities, especially those that are disadvantaged [117].

The WHO [42] describes a number of policies in transport, urban planning, power generation and industry that are known to be effective in reducing emissions of fossil fuel-related air toxics and CO_2:

- Clean technologies that reduce industrial smokestack emissions; improved management of urban and agricultural waste, including the capture of methane gas emitted from waste sites as an alternative to incineration (for use as biogas);
- Shifting to clean modes of power generation; prioritizing rapid urban transit, walking and cycling networks in cities as well as rail inter-urban freight and passenger travel; shifting from heavy-duty diesel vehicles to low-emission vehicles and fuels, including fuels with reduced sulfur content;
- Improving the energy efficiency of buildings and making cities more compact, and thus energy efficient;
- Increased use of low-emissions fuels and renewable combustion-free power sources (like solar, wind or hydropower); co-generation of heat and power; and distributed energy generation (e.g., mini-grids and rooftop solar-power generation).

According to the International Energy Agency (IEA), "the technologies for the energy sector to push air pollution levels into a steep decline in all countries exist and are in widespread use today; and they can be applied at great net economic benefit. Such actions can help avoid millions of pollution-related deaths; greenhouse-gas emissions would also be cut and fossil-fuel import bills reduced" [17]. In fact, many countries have lowered CO_2 emissions through fuel-economy standards for auto emissions, limits for power plants, stricter energy-efficiency codes for buildings, and other available methods.

Examples of interventions now being mounted to address climate change and fossil fuel-related pollution include the India Heat Action Plan (HAP) [118]. The HAP has provided an early-warning system to better prepare and protect local communities from deadly heat waves. Along with the immediate benefit in terms of adaptation to the current threat, the increased awareness of climate change is incentivizing the Indian government to move away from coal and other fossil fuel.

Another is the California climate change initiative launched in 2006 as a multi-year program to reduce greenhouse-gas emissions in California, with a 2030 greenhouse-gas emissions reduction target of 40% below 1990 levels. The expected benefits included a $76 billion increase in the state's Gross State Product, a $48 billion increase in real household incomes, the creation of over 400,000 new efficiency- and climate-driven jobs, and more than $8 billion by 2025 in pollution-related health costs avoided [114]. With respect to equity, the program is designed to ensure that the benefits of energy efficiency reach low-income residents as well as middle- and high-income residents.

The regional initiative to reduce air pollution and CO_2 emissions in the north-eastern states (US) known as the Regional Greenhouse Gas Initiative (RGGI) placed a regional limit on the amount of CO_2 that power plants can emit and instituted a cap-and-trade policy. A 2017 analysis found that RGGI created major benefits to public health and productivity including the avoidance of 300–830 early deaths among adults; 39,000–47,000 lost work days; and 35–390 non-fatal heart attacks [119]. The total health-cost savings from RGGI to date are estimated to be $5.7 billion.

Another example is the provincial-level initiative in Taiyuan, Shanxi Province, China. Shanxi Province is the major coal-mining and coal-burning region. Taiyuan, the capital of Shanxi Province, has, in the past, been counted as one of the world's worst cities for air quality. The Columbia Center for Children's Environmental Health (CCCEH) at the Mailman School of Public Health, the Shanxi Medical University, the Center of Disease Control and Prevention of Taiyuan Municipality, and Shanghai Fudan University School of Public Health estimated the health and economic benefits of policies between 2001–2010 to reduce the burden of air pollution in Taiyuan [81]. They include 30,000 fewer DALYs attributed to air pollution in Taiyuan in 2010 compared to 2001 and economic savings in avoided health costs of premature death due to air pollution of 3.83 billion Yuan, or approximately $621 million. The team is now updating these findings, assessing subsequent policy changes from 2010 to 2016, including additional health outcomes in children, and incorporating additional satellite and ground-level monitoring data.

A final example is the Paris Agreement [120]. Unfortunately, alone among the 195 signers of the Treaty, the current US Administration has stated the intention to withdraw from this major international treaty. The US Environmental Protection Agency (EPA) report, Climate Change in the United States: Benefits of Global Action [121], estimated that billions of dollars of avoided damages in the US would result from global efforts to reduce greenhouse-gas emissions. These included a significant portion of the health costs attributed to air pollution and climate change. The Clean Power Plan, now being rolled back by the current administration, was intended to play a key role in meeting the targets set by the Paris Agreement. From a benefit-cost perspective, the EPA estimated that the air pollution co-benefits of the Clean Power Plan were worth $25–$62 billion, far more than the estimated $7–$9 billion in compliance costs [122]. Adding in global climate benefits increased total benefits to $55–$93 billion.

These examples are encouraging but also pose the challenge of more fully documenting their health and economic benefits and the extent to which we promote environmental justice. Fortunately, methods are in place to make advances in that area and efforts are ongoing.

10. Conclusions

Consideration of the full impacts of fossil-fuel pollution and our carbon-based economy shows that unless strong action is taken now, our children and their progeny will inherit an increasingly unsustainable and unfair world in which they, their families and communities will not be able to survive, adapt, grow and transform where needed. The mounting health and economic costs of pollution and climate change from fossil-fuel combustion are already spurring mitigation efforts that can serve as models for other communities, and regional, state and global entities. These provide hope for the future.

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Air pollution exposure during fetal life, brain morphology, and cognitive function in school-age children

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Abstract

Objective. Air pollution exposure during fetal life has been related to impaired child neurodevelopment but it is unclear if brain structural alterations underlie this association. The authors assessed whether air pollution exposure during fetal life alters brain morphology and whether these alterations mediate the association between air pollution exposure during fetal life and cognitive function in school-age children.

Method. We used data from a population-based birth cohort set up in Rotterdam, The Netherlands (2002-2006). Residential levels of air pollution during the entire fetal period were calculated using land-use regression models. Structural neuroimaging and cognitive function were performed at age 6-10 years (n=783). Models were adjusted for several socioeconomic and life-style characteristics.

Results. Mean fine particle levels were 20.2μg/m^3 (range 16.8-28.1). Children exposed to higher particulate matter levels during fetal life had thinner cortex in several brain regions of both hemispheres (e.g. cerebral cortex of the precuneus region in the right hemisphere was 0.045mm thinner (95% Confidence Interval 0.028-0.062) for each 5μg/m^3 increase in fine particles). The reduced cerebral cortex in precuneus and rostral middle frontal regions partially mediated the association between exposure to fine particles and impaired inhibitory control. Air pollution exposure was not associated with global brain volumes.

Conclusions. Exposure to fine particles during fetal life was related to child brain structural alterations of the cerebral cortex and these alterations partially mediated the association between exposure to fine particles during fetal life and impaired child inhibitory control. Such cognitive impairment at early ages could have significant long-term consequences.

Introduction

Air pollution is a global risk factor for various adverse health effects in humans (1–7). There is increasing evidence indicating that air pollution exposure is also related to an impairment of the central nervous system through chronic neuroinflammation and microglia activation which can lead to neuronal damage (8). Since pregnancy and the first years of life are critical windows of developmental vulnerability for the brain, exposure to air pollution during this period could cause permanent changes in the brain even at low levels of exposure (9, 10).

Several epidemiological studies have assessed the association between air pollution exposure during early life and child neurodevelopment (11–16). These studies have found that air pollution exposure during pregnancy or during the first years of life was associated with lower cognitive or psychomotor function and higher behavior problems including autism spectrum disorders. However, they mainly used neuropsychological or clinical instruments to evaluate child neurodevelopment, limiting our understanding of which brain structural and functional alterations underlie these associations. Only few small studies have started using magnetic resonance imaging (MRI) techniques to assess relationships with air pollution (17–20). Three studies found an association between higher exposure to air pollution at home during fetal life or early childhood and white matter abnormalities in children at seven to thirteen years old (17–19). A fourth study in children aged eight to twelve years showed a relationship between air pollution exposure at school and lower functional integration and segregation in key brain networks (20). Despite the fact that prior studies have not found an association between air pollution exposure and cortical thickness, the study of brain morphology is key in providing insights in the underlying neurobiological pathways.

Therefore, the aims of the present study were i) to assess the association between air pollution exposure during fetal life and brain morphology in school-age children and ii) to assess the mediation role of brain morphology on the association between air pollution exposure during fetal life and cognitive function in school-age children. Cognitive function is the result of integration of functions of many different brain regions, and thus there was no *a priori* hypothesis on which specific brain regions would be affected by air pollution exposure during fetal life as no other similar studies have been performed so far. Thus, we used an exploratory approach to examine the association of exposure to air pollutants and brain surface measures.

Methods and Materials

Population and study design

This study was embedded in the Generation R Study, a population-based birth cohort study from fetal life onwards in Rotterdam, the Netherlands (21). A total of 8,879 pregnant women were enrolled and children were born between April 2002 and January 2006. A subgroup of children aged between six and ten years participated in an MRI sub-study (22). Briefly, a total of 1,932 were invited to participate in this sub-study. Children were oversampled based on certain maternal exposures during pregnancy (i.e. cannabis, nicotine, selective serotonin reuptake inhibitors, depressive symptoms, and plasma folate levels) and child behavior problems (i.e. attention deficit hyperactivity disorder, pervasive developmental problems, dysregulation problems, and aggressive problems). Exclusion criteria comprised contradictions for the MRI procedure, severe motor or sensory disorders, neurological disorders, head injuries with loss of consciousness, and claustrophobia. Among those invited, 155 did not answer the invitation call, 447 refused to participate, and 5 could not participate due to contraindications for the MRI procedure. Among the 1,325 that attended the MRI visit, after excluding those with poor MRI data quality and major abnormalities, MRI measurements were available for 1,070 children. Finally, after excluding those without air pollution estimations during fetal life, 783 children were included in the present study. This study was approved by the Medical Ethics Committee of the Erasmus Medical Centre in Rotterdam, The Netherlands. Written informed consent was obtained from parents.

Air pollution exposure

Air pollution levels at mothers' home addresses for the entire fetal period were estimated following a standardized procedure described elsewhere (23–25). Briefly, air pollution monitoring campaigns of three two-week periods of nitrogen dioxide (NO_2) in 80 sites and particulate matter (PM) with aerodynamic diameters <10µm (PM_{10}) and <2.5µm ($PM_{2.5}$ or fine particles), and absorbance of fine particles (a proxy for elemental carbon) in 40 sites were performed in 2009-2010 across The Netherlands and Belgium (26, 27). Coarse particle concentration was calculated as the difference between PM_{10} and $PM_{2.5}$. The three measurements were averaged, adjusting for temporal variation using data from a centrally located background monitoring site with year-round monitoring. Land-use regression models were developed using predictor variables on nearby traffic intensity, population/household density, and land use derived from Geographic Information Systems to explain spatial variation of annual average concentrations (23–25). These models were then used to assign air pollution levels at mothers' home addresses during the entire fetal period using the exact geographical x and y coordinates that corresponded to the addresses reported by each participant. Seven available routine background monitoring network sites were simultaneously used to back-extrapolate to the exact fetal period (6, 25) accounting for the changes of home address during pregnancy (Supplemental Methods S1). This resulted in a single, time-adjusted mean air pollution concentration for each participant for the entire fetal period. Previous research supports stability of measured and modeled spatial contrast in air pollutants for periods up to 18 years (28).

Magnetic Resonance Imaging

Structural MRI scans were obtained on a 3-Tesla scanner (Discovery MR750, GE Healthcare, Milwaukee, USA). Using an 8-channel head coil, a whole-brain high-resolution T1-weighted inversion recovery fast spoiled gradient recalled (IR-FSPGR) sequence was obtained. The scan parameters were the following: repetition time=10.3ms, echo time=4.2ms, inversion time=350ms, flip angle=16^{o}, 186 contiguous slices with a thickness of 0.9mm, and in-plane resolution = 0.9×0.9mm.

To minimize movement children participated in a mock scanning session prior to the actual MRI scanning to introduce them to the scanning environment (22). In the scanner, care was taken that children were comfortable and soft cushions were used to assist with head immobilization. However, it was still possible that children moved in the scanner. Image quality assurance was performed in 2 steps. First, a visual inspection of the image quality of the T1 sequence was done at the scanner. If the image quality was poor or unusable, the scan was repeated with extra instructions for children to lie still. Second, a visual inspection of the surface reconstruction quality was done after the images were processed through the FreeSurfer pipeline. Both steps of quality control had to be passed successfully for data to be included in the analyses.

Cortical reconstruction and volumetric segmentation of global brain measures was performed with the Freesurfer image analysis suite version 5.1.0, (http://surfer.nmr.mgh.harvard.edu/). Briefly, cortical thickness at each vertex was measured by calculating the shortest distance from the white matter to the pial surface. Procedures for the measurement of cortical thickness have been validated against histological analysis and manual measurements (29). Volumetric measures included total brain volume, cortical gray matter volume, cortical white matter volume, subcortical gray matter volumes (i.e., caudate, putamen, pallidum, accumbens, hippocampus, amygdala, and thalamus), and ventricular volume. Freesurfer morphometric procedures have been demonstrated to show good test-retest reliability across scanner manufacturers and across field strengths (30). All Freesurfer output was visually inspected and rated for quality.

Cognitive function

Children's cognitive function was assessed on the day of the scanning or shortly after using an array of subtasks from the Dutch version of the Developmental Neuropsychological Assessment test (NEPSY-II) (31). Detailed description of the test has been published previously (22). Briefly, the subtasks were chosen to tap into specific domains, including: attention and executive functioning, language, memory and learning, sensorimotor function, and visuospatial processing. Children were individually tested in a quiet room by trained investigators.

Potential confounding variables

Potential confounding variables were defined a priori based on direct acyclic graph (DAG) (Supplemental Figure S1) and on previous literature (11, 12, 25). Parental characteristics during pregnancy were collected by questionnaires: parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, maternal parity, family status, and maternal psychological distress (using the Brief Symptom Inventory). Parental weights and heights were measured or self-reported at the first trimester of pregnancy in the research center. Pre-pregnancy body mass index (kg/m^2) was calculated. Child's sex and date of birth were obtained from hospital or national registries. Child genetics ancestry was estimated based on the genome-wide SNP data from whole blood at birth and 4 principal components of ancestry were included to better correct for population stratification (32, 33). Maternal intelligence quotient was assessed at child's age of six years with the Ravens Advanced Progressive Matrices Test, set I. Child's age at scanning was also collected.

Statistical analyses

We performed whole-brain, vertex-wise statistics using the Freesurfer QDEC module (query, design, estimate contrast) for each air pollutant adjusting for child's sex and age. As there are many vertices per hemisphere (~160,000), analyses were corrected for multiple testing using the built-in Monte Carlo null- Z simulations with 10,000 iterations ($p<.01$). Due to limitations in modeling strategy with QDEC (types of variables, number of confounding variables, and inability to impute missingness in confounding variables), subject-level data from the identified regions associated with each air pollutant were imported into STATA (version 14; StataCorporation, College Station, TX, USA) for the following analysis.

Among children with available data on air pollution, neuroimaging, and cognitive function we performed multiple imputation of missing values of potential confounding variables using chained equations to generate 25 complete datasets (34). The percentage of missing values was relatively low and distributions in imputed datasets were similar to those observed (Supplemental Table S1). Children included in the analysis (n=783) were more likely to have mothers from a higher socioeconomic position compared to those that were not included, among children selected for the MRI sub-study (n=1,149) (Supplemental Table S2). This was also the case when we compared our study population to the not included children from the full cohort recruited in pregnancy (n=8,097) (Supplemental Table S3). We used inverse probability weighting to correct for lost to follow-up, i.e. to account for potential selection bias when including only participants with available data as compared to the full cohort recruited at pregnancy (35).

We used linear regression analyses to assess the associations between i) exposure to each air pollutant and global brain measures and ii) exposure to each air pollutant and the cortical thickness of each identified region in the QDEC analysis. Models were adjusted for all potential confounding variables described in the previous section.

Next we selected the tasks that assessed the cognitive function involved with each identified region based on the literature. We assessed whether both air pollution exposure and the cortical thinness of these regions were associated with the selected cognitive functions using adjusted negative binomial or linear regression models depending on the distribution of the outcome. We then applied causal mediation analysis providing estimation of the natural direct effect (NDE), the natural indirect effect (NIE), and the total effect (Supplemental Methods S2) (36). Briefly, we assessed the direct and indirect effects of air pollution exposure during fetal life on cognitive function. We tested whether part of the indirect effect was mediated by cortical thinness (Supplemental Figure S1). We used negative binomial regression for the outcome regression model and linear regression for the mediator regression model. Standard errors were calculated using bootstrapping. All models were adjusted for all potential confounding variables described in the previous section. The total effect results as the product of the natural direct effect (NDE) and natural indirect effect (NIE). We also calculated the proportion mediated as incidence rate ratio $(IRR)^{NDE}(IRR^{NIE} - 1)/(IRR^{NDE}IRR^{NIE} - 1)$.

We performed sensitivity analysis of the association between air pollutants and the cortical thickness of each identified region in the whole-brain analysis: i) we restricted the analysis to those children without attention deficit hyperactivity disorder, pervasive developmental problems, dysregulation problems, and aggressive problems and ii) we restricted the analysis to those children from non-smoking mothers during pregnancy.

Results

Participant characteristics of the study population are shown in Table 1 and Supplemental Table S4. Mean residential air pollution exposure during fetal life was 39.3μg/m^3 for NO_2 (range 25.3-73.3) and 20.2μg/m^3 for fine particles (range 16.8-28.1). Correlation between air pollutants was between 0.43 and 0.79 (Supplemental Table S5). Mothers exposed to higher air pollution levels during fetal life were more likely to have a higher level of education, to have a higher household income, and to be Dutch compared to those exposed to lower levels (Supplemental Table S6-9).

We did not find significant associations between air pollution exposure during fetal life and global brain volume measures (Table 2). Children exposed to higher particulate matter levels during fetal life had thinner cortices in several brain regions in both hemispheres (Figure 1). Sizes of associated brain regions varied between 532 and 2,995mm^2 (Supplemental Table S10). Mean thickness of these brain regions was between 2.31 and 3.17mm^2 (with a minimum thickness of 1.61 to 2.23 mm^2 and a maximum thickness of 3.23 to 3.97 mm^2). After adjusting for potential confounding variables, exposure to particulate matter levels remained strongly associated with thinner cortices of all identified regions (e.g. cerebral cortex of the precuneus region was 0.045mm thinner (95% Confidence Interval (CI) 0.028 to 0.062) for each 5μg/m^3 increase in fine particles) (Table 3). We observed similar results in the different sensitivity analysis (Supplemental Tables S11-12).

Based on the cognitive functions involved with each identified region, we selected the attention and executive functioning tasks for all regions except for the fusiform region where we selected the memory for faces tasks (Supplemental Methods S3). Fine particles exposure during fetal life was associated with a higher number of inhibition errors of the response set task (IRR 1.07; 95% CI 1.01 to 1.14 per each 5μg/m^3 increase in fine particles) (Table 4). No significant associations were observed for the other relationships. A thinner cortex in the precuneus region and the rostral middle frontal region was also associated with a higher number of inhibition errors of that tasks (IRR 1.32; 95% CI 1.00 to 1.77 per each 1mm decrease of the cortex in the precuneus region and IRR 1.69; 95% CI 1.09 to 2.61 per each 1mm decrease of the cortex in the rostral middle frontal region) (Table 5). We finally found that the reduced cortical thickness in the precuneus and rostral middle frontal regions partially mediated the observed association between fine particles exposure during fetal life and the increase number of inhibition errors (natural indirect effect: IRR 1.01; 95% CI 1.00 to 1.02 per each 1mm decrease of the cortex in the precuneus region and in the rostral middle frontal region) (Figure 2). The proportion mediated through the reduced cortical thickness in each of the regions was estimated to be 15%.

Discussion

The present study suggests that particulate matter exposure during fetal life was associated with a thinner cortex in several brain regions and to an impaired inhibitory control in school-age children. The structural alterations in the precuneus and the rostral middle frontal regions partially mediated the association between fine particles exposure and impaired inhibitory control. No association was found between air pollution exposure and global brain volume measures.

Several epidemiological studies have found that air pollution exposure during fetal life was associated with lower cognitive function (11–14). However, very few studies have investigated which brain structural and functional alterations underlie these associations. Child cognitive function is the result of integration of functions of many different brain regions, and thus we did not have *a priori* hypothesis on which specific brain regions could be affected by air pollution exposure during fetal life. In our study we identified that some specific brain regions had thinner cortex in relation to air pollution exposure during fetal life. We do not have a hypothesis why air pollution exposure during fetal life is affecting the grey matter of specific brain regions instead of having a more wide-spread effect. One explanation would be that this is due to the different development of each brain region across adolescence. For example, cortical volume of the frontal lobe showed a relatively stable trajectory in late childhood and an accelerated thinning in adolescence, while decelerating trajectories with increasing age were seen for thickness in the parietal and occipital lobes (37). Further longitudinal studies are warranted to better understand the potential associations at different ages.

To date, only one small study assessed the relationship between air pollution exposure during fetal life and structural brain morphology in 40 children at seven to nine years old from New York City, taking also an exploratory approach as we did in our study (17). Peterson et al. did not find an association between personal polycyclic aromatic hydrocarbons exposure during the third trimester of pregnancy and any measure of cortical thickness. However, they found an association between higher personal polycyclic aromatic hydrocarbons exposure during the third trimester of pregnancy and a lower white matter surface, almost exclusively to the left hemisphere of the brain (17). In contrast with this previous study, we did not find a relationship between exposure to air pollutants during fetal life and white matter volume using a much larger sample of children at a similar age. As there is indication that white matter could be one of the brain structural affected by air pollution exposure during fetal life, future research should focus on white matter microstructure which could uncover deficits that are not apparent with simple white matter volumetric measures.

During pregnancy, the detoxification mechanisms of the developing fetus are still immature and the placenta grants only a partial protection against the entry of environmental toxicants (10, 9). Hence, when the mother is exposed to air pollution, air pollutants might alter the prenatal brain development as a result of oxidative stress and systemic inflammation leading to chronic neuroinflammation, microglia activation, and neuronal migration damage (8). Early disturbances in neuronal path finding, abnormalities in cell proliferation, and differentiation eventually result in a thinner cortex during childhood. Although the prenatal period is considered particularly vulnerable period for brain development, the brain continues to develop until adolescence and postnatal air pollution exposure could also play a role on brain development (8, 11, 12). In the New York City study, they also explored the relationship between postnatal urinary polycyclic aromatic hydrocarbon metabolites and structural brain morphology not finding an association with cortical thickness but showing a lower white matter surface in dorsal prefrontal regions bilaterally (17). Two small studies including around 30 children at six to fourteen years old found that children living in Mexico City had lower white matter volumes and higher rates of subcortical prefrontal white matter hyperintensities compared to those living in a low polluted city of Mexico (18, 19). Again, white matter seems to be influenced by air pollution exposure. Furthermore, in 263 children aged eight to twelve from Barcelona, Spain, higher elemental carbon and NO_2 exposure at school was not associated with brain structure but associated with lower functional integration and segregation in key brain networks relevant to both inner mental processes and stimulus-driven mental operations (20). That study was the first to shown that air pollution exposure might also alter brain functionality which leads to a slower brain maturation. Overall, air pollution exposure to both prenatal and postnatal periods has shown to impair brain development. Further studies are needed to disentangle the specific brain alterations due to prenatal and postnatal air pollution exposure.

Interestingly, our study is the first study showing that fine particles exposure during fetal life was associated with an impaired inhibitory control in school-age children and that thinner cortex in the precuneus and the rostral middle frontal regions partially mediated this association. Inhibitory control, a key component of executive functions, regulates the self-control of resisting temptations and acting impulsively and the selective attention (38). Impaired inhibitory control has been related to several mental health problems such as addictive behaviors (39) or attention deficit hyperactivity disorder (40). The previous study carried out in New York City found that the white matter disruption partially mediated the association between prenatal polycyclic aromatic hydrocarbons exposure and a slower information processing speed in children (17). Therefore, we hypothesize that air pollution exposure during fetal life could lead to brain structural changes and these to specific cognitive delays.

In our study, mean residential NO_2 levels during fetal life were just at the EU limit of 40µg/m^3, with 45% of our population having higher levels. Regarding fine particles, mean residential levels were clearly below the EU limit of 25µg/m^3, with only 0.5% of our population above that limit (41). However, as we observed in our study brain development effects in relationship to fine particles levels below the current EU limit, as well as other studies have found relationships with several health endpoints including natural-cause mortality, cardiovascular and respiratory diseases, cognitive decline, and fetal growth development (1–7), we cannot warrant that this limit is safe. The World Health Organization set a lower limit of 10µg/m^3 for fine particles (42), and in our study we have all our population above this limit. Further health effect research needs to bring more insight into the safety of the current levels of air pollution in our cities.

The strengths of our study are the large number of study participants with imaging data, the prospective and longitudinal nature of the study, the detailed information of air pollution estimations at the individual level during the entire fetal period, and the availability of adjusting the imaging analysis for a large number of socioeconomic and lifestyle factors known to be associated with both air pollution exposure and brain development. Nevertheless, we cannot discard that our results might still be affected by residual confounding due to the unavailability of other relevant potential confounding variables. Another limitation of our study was that children with exposure and outcome data were more likely to have mothers from higher socioeconomic position than those without these data but recruited at the beginning of the cohort in early pregnancy, which could lead to selection bias in our results. To reduce this possible selection bias, we used advanced statistical methods including multiple imputation combined with inverse probability weighting. However, we could have missed variables related to this potential selection bias that that would have a stronger effect in the results. In addition, there is the possibility of chance findings in the observed associations in the current study. The imaging analysis was corrected for multiple testing of the whole-brain, vertex-wise statistics as we have many vertices per hemisphere. However, the causal mediation analysis was hypothesis-driven and we decided not to correct for multiple testing as this could increase type 2 error (43, 44). Instead, our conclusions were based on the general patterns of associations observed in the study. This has been the first study showing that brain structural alterations seem to partially mediate the association between air pollution exposure during fetal life and an impaired cognitive function. Further studies are warranted to replicate these findings and better understand this association.

Conclusions

We showed that fine particles exposure during fetal life was both related to child brain structural alterations of the cerebral cortex and to an impairment of an essential executive function such as inhibitory control. Moreover, the identified structural alterations in two specific regions partially mediated the association between fine particles exposure during fetal life and the impaired inhibitory control. Such cognitive impairment at early ages could have significant long-term consequences including increased risk of mental disorders, low academic achievement, and diminished economic productivity (38), in particular due to the ubiquity of the exposure.

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Figures legend.

Figure 1. Differences in cortical thickness at 6-10 years of age associated with air pollution exposure during fetal life

The colored regions on the surface map represent brain regions that are thinner in relation to higher exposure to air pollution during fetal life in the right and left hemisphere (darker color indicates stronger association). Analyses were adjusted for child's sex and age. All brain regions survived the correction (Monte Carlo null-Z simulation with 10,000 iterations) for multiple comparisons ($p<.01$).

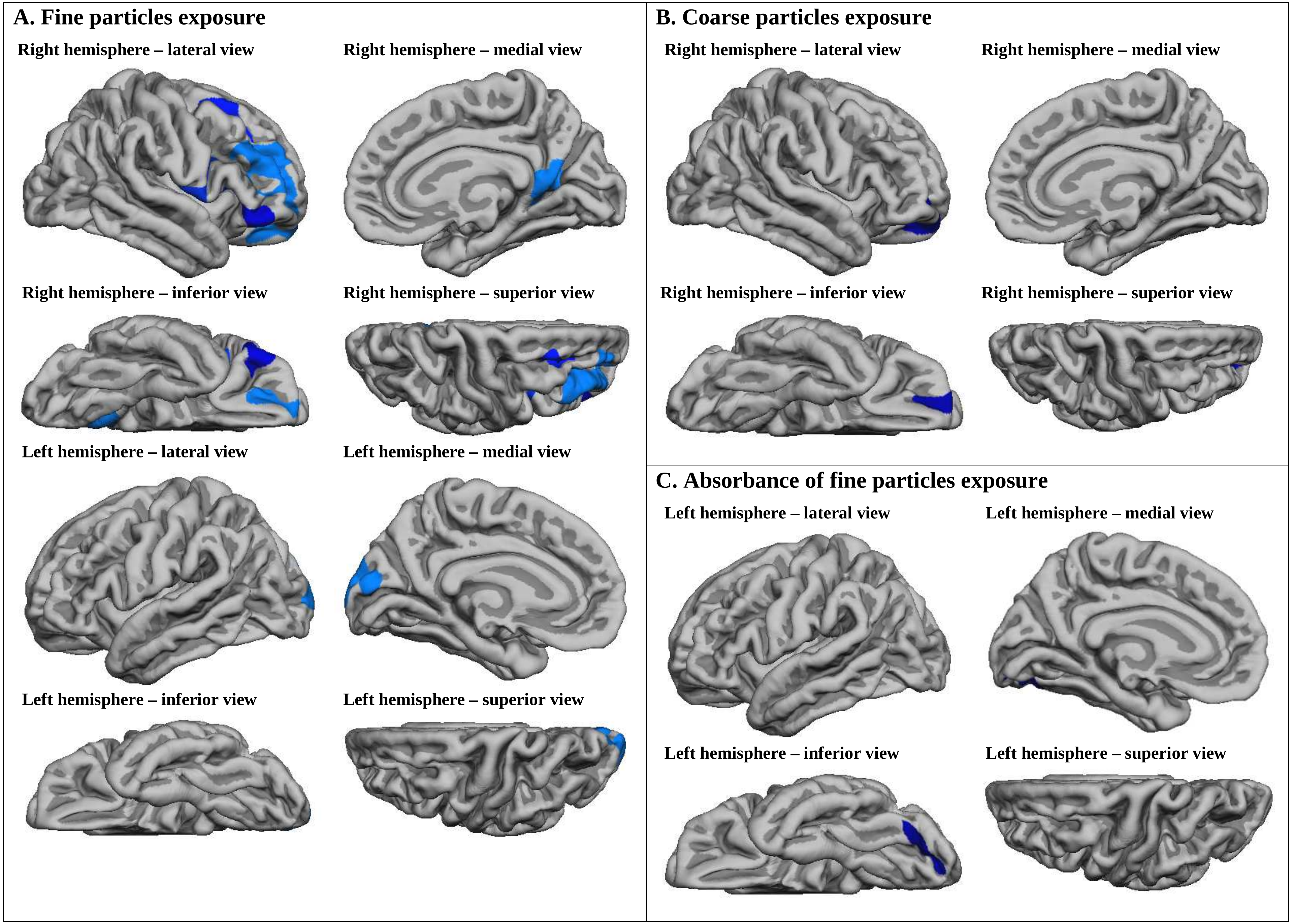
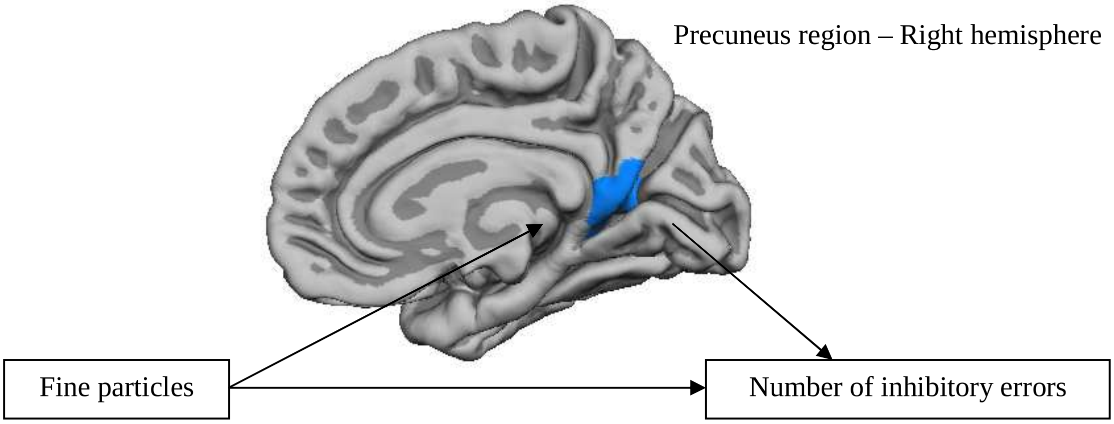
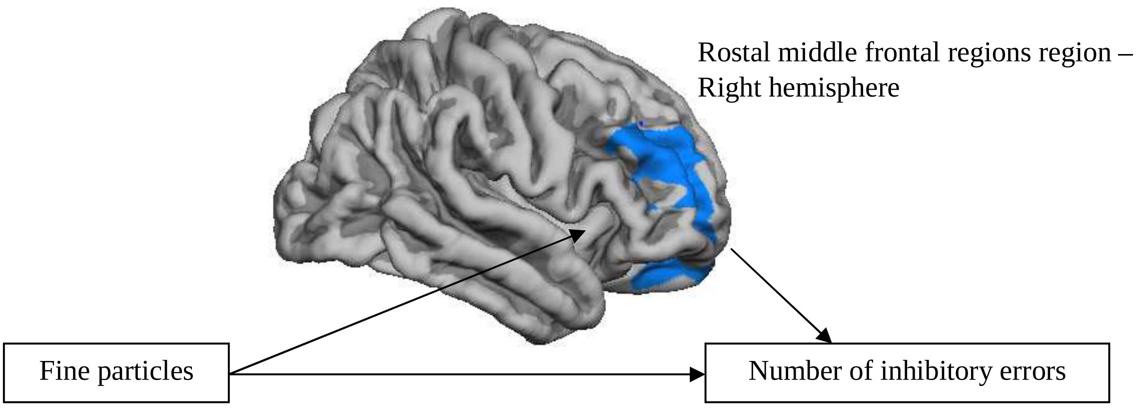

A. Fine particles exposure
Right hemisphere – lateral view
Right hemisphere – medial view
Right hemisphere – inferior view
Right hemisphere – superior view
Left hemisphere – lateral view
Left hemisphere – medial view
Left hemisphere – inferior view
Left hemisphere – superior view
B. Coarse particles exposure
Right hemisphere – lateral view
Right hemisphere – medial view
Right hemisphere – inferior view
Right hemisphere – superior view
C. Absorbance of fine particles exposure
Left hemisphere – lateral view
Left hemisphere – medial view
Left hemisphere – inferior view
Left hemisphere – superior view

Figure 2. Causal mediation analyses between air pollution exposure during fetal life, cortical thickness (in mm) in precuneus and rostral middle frontal regions, and the number of inhibition errors of the response set task at 6-10 years of age

Abbreviations: CI, confidence interval; IRR, incidence risk ratio.

Incidence risk ratio (95% Confidence Interval) from negative binomial regression models adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol consumption, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. As results for both causal mediation analyses were identical rounded to 2 decimal places, only one table is presented.





	IRR	(95% CI)
Natural direct effect	1.06	(1.00 to 1.12)
Natural indirect effect	1.01	(1.00 to 1.02)
Total effect	1.07	(1.00 to 1.13)

Table 1. Participant characteristics and air pollution levels during fetal life

	Distribution		
Participant characteristics	**Percentage**	**Mean**	**(SD)**
Maternal education level			
Primary education	7.0		
Secondary education	44.8		
University education	48.2		
Paternal education level			
Primary education	5.7		
Secondary education	40.9		
University education	53.4		
Monthly household income			
<1,200€	14.1		
1,200€ - 2,000€	17.7		
>2,000€	68.1		
Maternal country of birth			
The Netherlands	65.2		
Cape Verde	4.7		
Morocco	4.7		
Surinam	6.5		
Turkey	4.5		
Other country of birth	14.5		
Paternal country of birth			
The Netherlands	72.7		
Cape Verde	2.6		
Morocco	1.9		
Surinam	5.0		
Turkey	3.4		
Other country of birth	14.4		
Maternal age (years)		30.7	(4.9)
Paternal age (years)		32.9	(5.3)
Family status (mono *vs.* biparental)	13.5		
Maternal parity (multi *vs.* nulliparous)	39.5		

Table 1. (Continuation)

	Distribution		
Participant characteristics	**Percentage**	**Mean**	**(SD)**
Maternal smoking use during pregnancy			
Never	75.8		
Smoking use until pregnancy known	6.5		
Continued smoking use during pregnancy	18.2		
Maternal alcohol use during pregnancy			
Never	37.6		
Alcohol use until pregnancy know	14.3		
Continued alcohol use during pregnancy	48.1		
Maternal pre-pregnancy body mass index (kg/m^2)		24.6	(4.3)
Paternal pre-pregnancy body mass index (kg/m^2)		25.3	(3.3)
Maternal height (cm)		168.6	(7.4)
Paternal height (cm)		182.9	(7.3)
Maternal overall psychological distress		0.3	(0.4)
Maternal intelligence quotient score		98.4	(13.9)
Air pollution levels during fetal life	**Median**	**(Min-Max)**	
NO_2 (µg/m^3)	39.3	(25.3-73.3)	
Fine particles (µg/m^3)	20.2	(16.8-28.1)	
Coarse particles (µg/m^3)	11.8	(9.2-17.8)	
Absorbance of fine particles (10^{-5} m^{-1})	1.9	(1.2-3.6)	

Abbreviations: Max, maximum; Min, minimum; NO_2, nitrogen dioxide, SD, standard deviation.

Table 2. Fully-adjusted association between air pollution exposure during fetal life and global brain volume measures at 6-10 years of age

	Coef.	(95% CI)[a]	P value
NO_2			
Total brain volume	124	(-1118 to 1375)	.84
Cortical gray matter volume	-60	(-853 to 733)	.88
Cortical white matter volume	199	(-287 to 685)	.42
Subcortical gray matter volume	36	(-17 to 89)	.18
Ventricular volume	4	(-57 to 64)	.90
Fine particles			
Total brain volume	-3079	(-7790 to 1632)	.20
Cortical gray matter volume	-2598	(-5583 to 387)	.09
Cortical white matter volume	-268	(-2096 to 1559)	.77
Subcortical gray matter volume	-60	(-258 to 138)	.55
Ventricular volume	-96	(-323 to 131)	.40
Coarse particles			
Total brain volume	-4868	(-10337 to 822)	.09
Cortical gray matter volume	-3542	(-7059 to 8)	.05
Cortical white matter volume	-1129	(-3215 to 1127)	.34
Subcortical gray matter volume	-92	(-325 to 148)	.46
Ventricular volume	-100	(-372 to 168)	.45
Absorbance of fine particles			
Total brain volume	-2861	(-18745 to 24467)	.79
Cortical gray matter volume	-2683	(-16377 to 11012)	.70
Cortical white matter volume	5807	(-2566 to 14180)	.17
Subcortical gray matter volume	418	(-497 to 1334)	.36
Ventricular volume	-64	(-1108 to 979)	.90

Abbreviations: CI, confidence interval; Coef, beta coefficient; NO_2, nitrogen dioxide.
[a]Beta coefficient (95% Confidence Interval) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, marital status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. Coefficients represent the differences in volumes (cm^3) per each increase of $10 \mu g/m^3$ of NO_2, $5 \mu g/m^3$ of fine particles, $5 \mu g/m^3$ of coarse particles, and $10^{-5} m^{-1}$ of absorbance of fine particles.

Table 3. Fully-adjusted association between air pollution exposure during fetal life and cortical thickness (in mm) at 6-10 years of age

	Hemisphere	Size brain region (mm^2)	Coef.	(95% CI)[a]	P value
Fine particles exposure					
Precuneus region	Right	936	-0.045	(-0.062 to -0.028)	<.001
Pars opercularis region	Right	753	-0.024	(-0.033 to -0.014)	<.001
Pars orbitalis region	Right	651	-0.028	(-0.043 to -0.012)	.001
Rostral middle frontal region	Right	2,995	-0.029	(-0.041 to -0.018)	<.001
Superior frontal region	Right	722	-0.029	(-0.043 to -0.016)	<.001
Cuneus region	Left	843	-0.022	(-0.035 to -0.009)	.002
Coarse particles exposure					
Lateral orbitofrontal region	Right	565	-0.037	(-0.059 to -0.016)	.001
Absorbance of fine particles exposure					
Fusiform region	Left	532	-0.105	(-0.160 to -0.049)	<.001

Abbreviations: CI, confidence interval; Coef, beta coefficient.

[a]Beta coefficient (95% Confidence Interval) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. Coefficients represent the differences in thickness (mm) per each increase of $5\mu g/m^3$ of fine particles, $5\mu g/m^3$ of coarse particles, and $10^{-5}m^{-1}$ of absorbance of fine particles.

Table 4. Adjusted association between air pollution levels during fetal life and cognitive function at 6-10 years of age

	IRR	(95% CI)[a]	P value
Fine particles exposure			
Auditory attention task			
Correct responses	1.00	(0.99 to 1.01)	.61
Commission errors	1.00	(0.89 to 1.16)	.95
Omission errors	0.98	(0.92 to 1.03)	.38
Inhibition errors	1.10	(0.63 to 1.93)	.73
Response set task			
Correct responses	1.01	(1.00 to 1.02)	.17
Commission errors	1.00	(0.96 to 1.04)	.79
Omission errors	0.97	(0.94 to 1.00)	.07
Inhibition errors	1.07	(1.01 to 1.14)	.02
Coarse particles exposure			
Auditory attention task			
Correct responses	1.00	(0.99 to 1.01)	.71
Commission errors	0.99	(0.87 to 1.13)	.88
Omission errors	0.98	(0.92 to 1.05)	.63
Inhibition errors	0.98	(0.55 to 1.76)	.95
Response set task			
Correct responses	1.01	(0.99 to 1.02)	.39
Commission errors	0.97	(0.92 to 1.02)	.19
Omission errors	0.98	(0.94 to 1.02)	.28
Inhibition errors	1.04	(0.97 to 1.12)	.24
	Coef.	**(95% CI)[b]**	**P value**
Absorbance of fine particles exposure			
Memory for faces task	0.22	(-0.24 to 0.69)	.34
Memory for faces delayed task	0.29	(-0.23 to 0.81)	.27

Abbreviations: CI, confidence interval; Coef, beta coefficient; IRR, incidence rate ratio.

[a]Incidence rate ratio values (95% Confidence Interval) from negative binomial regression model or [b]beta coefficients (95% Confidence Interval) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry.

Table 5. Adjusted association between thinner cortical thickness (in mm) and the total number of inhibitory numbers of the response set task at 6-10 years of age

	IRR	(95% CI)[a]	P value
Precuneus region	1.32	(1.00 to 1.77)	0.05
Pars opercularis region	0.83	(0.49 to 1.42)	0.49
Pars orbitalis region	1.16	(0.83 to 1.61)	0.38
Rostral middle frontal region	1.69	(1.09 to 2.61)	0.02
Superior frontal region	1.28	(0.89 to 1.86)	0.18

Abbreviations: CI, confidence interval; IRR, incidence rate ratio.

[a]Incidence rate ratio values (95% Confidence Interval) from negative binomial regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. CI denotes confidence interval, IRR denotes incidence risk ratio.

Air Pollution Exposure During Fetal Life, Brain Morphology, and Cognitive Function in School-Age Children

Supplemental Information

Table of contents

Methods S1. Description of the back-extrapolation methodology of the air pollution levels

We used a back-extrapolation procedure to estimate the levels back in time during each fetal period of each woman in order to assess if fetal period is a relevant exposure period (1, 2). The estimated yearly concentrations ($C_{yearly,i}$) at each home address i were combined with time-specific measurements from seven available routine background monitoring network sites by averaging the daily concentrations during 1) the year corresponding to the LUR yearly concentration (C_{yearly}) and 2) each fetal period p_i considered (C_{p_i}). The ratio C_{p_i}/C_{yearly} constituted the temporal component of the model. For each pollutant, the concentration ($C_{p_i, i}$) estimated at the home address i during the fetal period for woman i was estimated as the product of the temporal (C_{p_i}/C_{yearly}) and spatial ($C_{yearly, i}$) components. In cases when air quality monitoring data from background station was unavailable for a given pollutant, we used measurements for another pollutant during the same time period as a replacement; the choice of that pollutant used to back-extrapolate another pollutant was based on an extensive study of temporal correlations between pollutants simultaneously available (i.e. PM_{10} was used as a proxy for $PM_{2.5}$ and back smoke as a proxy for $PM_{2.5}$absorbance). We accounted for change of home address during the whole fetal period since the date of moving and new address was available.

Methods S2. Description of the causal mediation analysis

The causal mediation analysis provides a better understanding of the causal chain by which an independent variable (X) influences a dependent variable (Y) through a mediator (M). Consistent with its conceptual definition (3), this involves sequential testing of the following: i) the effect of the exposure (X) on the outcome (Y); ii) the effect of the exposure e (X) on the mediator (M); iii) the effect of the mediator (M) on the outcome (Y) controlling for the exposure (X), and iv) the effect of the exposure (X) on the outcome (Y) controlling for the mediator (M). The causal mediation analysis provides estimation of the natural direct effect (NDE), the natural indirect effect (NIE), and the total effect (3). The natural direct effect (NDE) expresses how much the outcome (Y) would change if the exposure (X) is set at a level $a=1$ to level $a=0$ but for each individual the mediator (M) is kept at the level it would have taken in the absence of the exposure. The natural indirect effect (NIE) expresses how much the outcome (Y) would change on average if the exposure (X) is controlled at level $a=1$, but the mediator (M) is changed from the level it would take if $a=0$ to the level it would take if $a=1$. The total effect can be defined as how much the outcome (Y) would change overall for a change in the exposure (X) from level $a=0$ to level $a=1$.

In our study, we applied causal mediation analysis to assess the direct and indirect effects of air pollution exposure during fetal life (X) on cognitive function (Y) where we tested whether part of the indirect effect was mediated by cortical thinness (M) (Figure S1). We used negative binomial regression for the outcome regression model and linear regression for the mediator regression model. Standard errors were calculated using bootstrapping. All models were adjusted for all potential confounding variables described in the section “Potential confounding variables” of the manuscript. The total effect results as the product of the natural direct effect (NDE) and natural indirect effect (NIE). We also calculated the proportion mediated as incidence rate ratio $(IRR)^{NDE}(IRR^{NIE} – 1)/(IRR^{NDE}IRR^{NIE} – 1)$.

Methods S3. Cognitive function tests selected based on the identified regions

In the first analysis, we found that higher particulate matter levels during fetal life were associated with thinner cortices in specific regions of the frontal, parietal and occipital brain regions (Table 3). Post-hoc, we went back to the literature to find out in which cognitive processes these regions were involved. The frontal brain regions and the (pre)cuneus are known to be involved in attention and executive functions (4, 5) while the fusiform gyrus is known to be involved in the face perception, object recognition, and memory (6). Therefore, we selected two specific tasks of the NEPSY-II test for the mediation analysis: the attention and executive functioning task and the memory for faces task.

Attention and executive functioning task

Children were assessed with two different tasks from the attention and executive functioning domain of the NEPSY-II: auditory attention task and response set task (7–9). The auditory attention task was administered first. It is designed to assess selective auditory attention and the ability to sustain it (vigilance). Selective attention refers to the ability to focus on a specific task while suppressing irrelevant stimuli. Sustained attention refers to the ability to attend to a task for a long(er) period of time. In the auditory attention task, the children were presented with recording of a long list of color words and other words and were asked to only respond to the word “red” by touching the red circle on the sheet in front of them. The sheet also contained a blue, black, and yellow circle, but these circles had to be ignored. Touching the red circle within two seconds indicated a correct response.

The response set task was then administered. This task taps into response inhibition and working memory. Inhibition is the ability to suppress (automatic) behavior. Working memory is required to keep information actively in mind for as long as needed to complete a task. In this task, children must respond to the word “red” by touching the yellow circle, respond to “yellow” by touching the red circle, and lastly, respond to the word “blue” by touching the blue circle. All of the other colors or words should be ignored. Touching the correct circle within two seconds indicates a correct response. Touching another color is incorrect, as is having delayed response (not within a 2 seconds interval).

For each task, four scores were calculated: total number of correct responses, total number of commission errors (i.e. the number of times that the child responded erroneously to a non-target), total number of omission errors (i.e. the number of target to which the children failed to respond), and inhibition errors (i.e. the number of times that the child responded to a color word inappropriately; in other words, fails to inhibit an inappropriate response).

Memory for faces task

Children were assessed with two different tasks from the memory and learning domain of the NEPSY-II: memory for faces task and memory for faces delayed task (7–9). The memory for faces test is designed to assess encoding of facial features, as well as face discrimination and recognition. The child was first presented with multiple series of three faces and was asked to look closely at each face (for five seconds). The child was then provided with another set of three faces and was asked which face he or she had seen before. Immediate recall is the skill to retrieve information from memory immediately after learning.

The memory for faces delayed task is designed to assess long-term memory for faces. This task was assessed after a delay period of 15 to 25 minutes and measured the ability to retrieve information after a longer period of time.

For both tasks, all presented faces showed a neutral expression. A total correct score was calculated for both tasks.

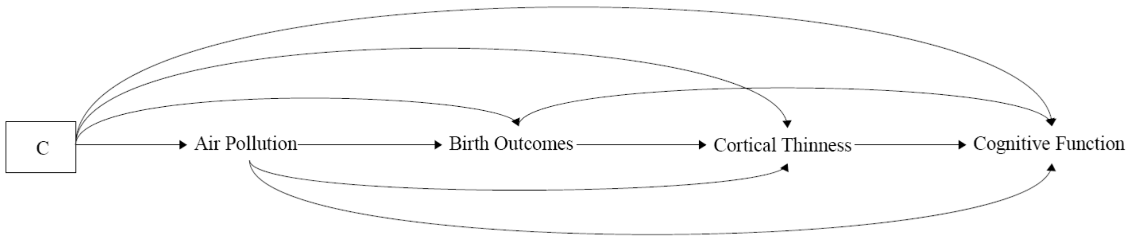


Figure S1. Direct Acyclic Graph

C denotes all the potential confounding variables in the relationship between air pollution exposure in fetal life and cognitive function in childhood, such as SES, parental lifestyle and ethnicity. This theoretical selection of confounders was reflected as completely as the data availability allowed. In our study we included: parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child genetic ancestry. Additionally, the models were adjusted for child's sex and child's age at the MRI session. The box indicates the conditioning on the potential confounders. Solid arrows represent existing pathways indicating thereby the direction of the associations.

Table S1. Distribution of participant characteristics in observed and imputed datasets

	Observed dataset[a]		Imputed datasets[a]		% data imputed
Maternal education level					2.8
Primary education	7.0		7.4		
Secondary education	44.8		45.1		
University education	48.2		47.4		
Paternal education level					26.6
Primary education	5.7		9.2		
Secondary education	40.9		43.9		
University education	53.4		46.9		
Monthly household income					10.6
<1,200€	14.1		15.8		
1,200€ -2,000€	17.7		18.4		
>2,000€	68.1		65.8		
Maternal country of birth					1.4
The Netherlands	65.2		64.7		
Cape Verde	4.7		4.8		
Morocco	4.7		4.8		
Surinam	6.5		6.6		
Turkey	4.5		4.7		
Other country of birth	14.5		14.5		
Paternal country of birth					20.8
The Netherlands	72.7		66.3		
Cape Verde	2.6		4.2		
Morocco	1.9		3.1		
Surinam	5.0		6.7		
Turkey	3.4		4.9		
Other country of birth	14.4		14.8		
Maternal age (years)	---		---		0.0
Paternal age (years)	32.9	(5.3)	32.8	(5.5)	18.3
Family status (mono *vs.* biparental)	13.5		13.9		2.8
Maternal parity (multi *vs.* nulliparous)	39.5		39.4		0.4
Maternal smoking use during pregnancy					7.2
Never	75.4		74.8		
Smoking use until pregnancy known	6.5		6.6		
Continued smoking use during pregnancy	18.1		18.6		
Maternal alcohol use during pregnancy					6.9
Never	37.6		38.0		
Alcohol use until pregnancy known	14.3		14.3		
Continued alcohol use during pregnancy	48.1		47.7		
Maternal pre-pregnancy body mass index (kg/m^2)	---		---		0.0
Paternal pre-pregnancy body mass index (kg/m^2)	25.3	(3.3)	25.3	(3.4)	18.3
Maternal height (cm)	---		---		0.0
Paternal height (cm)	182.9	(7.3)	182.3	(7.5)	18.3
Maternal overall psychological distress	0.3	(0.4)	0.3	(0.4)	12.1
Maternal intelligence quotient score	98.4	(13.9)	98.1	(13.9)	5.4

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables

Table S2. Comparison of participant characteristics between included and not included subjects in the study among the 1,932 subjects selected for the MRI sub-study

	Included[a] (n=783)		Not included[a] (n=1,149)		P value[b]
Maternal education level					<.001
Primary education	7.0		11.0		
Secondary education	44.8		50.5		
University education	48.2		38.5		
Paternal education level					.006
Primary education	5.7		8.5		
Secondary education	40.9		46.4		
University education	53.4		45.1		
Monthly Household income					.001
<1,200€	14.1		19.5		
1,200€ -2,000€	17.7		21.3		
>2,000€	68.1		59.2		
Maternal country of birth					<.001
The Netherlands	65.2		53.9		
Cape Verde	4.7		4.8		
Morocco	4.7		4.8		
Surinam	6.5		7.8		
Turkey	4.5		9.0		
Other country of birth	14.5		19.7		
Paternal country of birth					.02
The Netherlands	72.7		66.2		
Cape Verde	2.6		2.7		
Morocco	1.9		3.5		
Surinam	5.0		6.3		
Turkey	3.4		6.7		
Other country of birth	14.4		14.5		
Maternal age (years)	30.7	(4.9)	29.6	(5.2)	<.001
Paternal age (years)	32.9	(5.3)	32.7	(5.8)	.42
Family status (mono *vs·* biparental)	13.5		15.8		.18
Maternal parity (multi *vs·* nulliparous)	39.5		40.3		.87
Maternal smoking use during pregnancy					.02
Never	75.4		69.1		
Smoking use until pregnancy known	6.5		7.9		
Continued smoking use during pregnancy	18.2		22.9		
Maternal alcohol use during pregnancy					<.001
Never	37.6		46.6		
Alcohol use until pregnancy known	14.3		15.0		
Continued alcohol use during pregnancy	48.1		38.3		
Maternal pre-pregnancy body mass index (kg/m^2)	24.6	(4.3)	24.9	(4.6)	.23
Paternal pre-pregnancy body mass index (kg/m^2)	25.3	(3.3)	25.1	(3.5)	.25
Maternal height (cm)	168.6	(7.4)	167.7	(7.5)	.01
Paternal height (cm)	182.9	(7.3)	181.9	(8.1)	.02
Maternal overall psychological distress	0.3	(0.4)	0.4	(0.5)	<.001
Maternal intelligence quotient score	98.4	(13.9)	94.2	(14.7)	<.001

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and t-student test for continuous variables

Table S3. Comparison of participant characteristics between included and not included subjects in the study among the 8,879 subjects recruited in the full cohort in pregnancy

	Included[a] (n=783)		Not included[a] (n=8,097)		P value[b]
Maternal education level					<.001
Primary education	7.0		12.1		
Secondary education	44.8		46.6		
University education	48.2		41.3		
Paternal education level					.05
Primary education	5.7		8.6		
Secondary education	40.9		41.1		
University education	53.4		50.3		
Monthly Household income					<.001
<1,200€	14.1		21.6		
1,200€ -2,000€	17.7		18.7		
>2,000€	68.1		59.7		
Maternal country of birth					<.001
The Netherlands	65.2		47.8		
Cape Verde	4.7		4.1		
Morocco	4.7		6.9		
Surinam	6.5		9.3		
Turkey	4.5		9.6		
Other country of birth	14.5		22.3		
Paternal country of birth					<.001
The Netherlands	72.7		60.2		
Cape Verde	2.6		2.5		
Morocco	1.9		4.5		
Surinam	5.0		7.0		
Turkey	3.4		7.2		
Other country of birth	14.4		18.5		
Maternal age (years)	30.7	(4.9)	29.5	(5.3)	<.001
Paternal age (years)	32.9	(5.3)	32.7	(5.8)	.30
Family status (mono *vs·* biparental)	13.5		14.9		.30
Maternal parity (multi *vs·* nulliparous)	39.5		44.9		.005
Maternal smoking use during pregnancy					.14
Never	75.4		74.4		
Smoking use until pregnancy known	6.5		8.5		
Continued smoking use during pregnancy	18.1		17.1		
Maternal alcohol use during pregnancy					<.001
Never	37.6		51.2		
Alcohol use until pregnancy known	14.3		13.6		
Continued alcohol use during pregnancy	48.1		35.2		
Maternal pre-pregnancy body mass index (kg/m^2)	24.6	(4.3)	24.9	(4.6)	.08
Paternal pre-pregnancy body mass index (kg/m^2)	25.3	(3.3)	25.3	(3.5)	0.78
Maternal height (cm)	168.6	(7.4)	167	(7.4)	<.001
Paternal height (cm)	182.9	(7.3)	181.4	(8.0)	<.001
Maternal overall psychological distress	0.3	(0.4)	0.3	(0.4)	.24
Maternal intelligence quotient score	98.4	(13.9)	95.3	(15.5)	<.001

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and t-student test for continuous variables

Table S4. Global brain volume measures (in mm) in children at 6-10 years of age

	Mean	(SD)	Minimum	Percentile 25	Median	Percentile 75	Maximum
Total brain volume	1146063	(121154)	709551	1062833	1141372	1227930	1549471
Cortical gray matter volume	551350	(65306)	300283	508679	551737	597360	746402
Cortical white matter volume	381212	(47090)	227365	347471	379598	412231	565693
Subcortical gray matter volume	61961	(4940)	45762	58555	61801	65086	77729
Ventricular volume	11119	(5045)	3752	7700	9871	13240	39891

Table S5. Spearman correlations between air pollution levels during fetal life

	NO_2	Fine particles	Coarse particles	Absorbance of fine particles
NO_2	1.00			
Fine particles	0.43	1.00		
Coarse particles	0.66	0.68	1.00	
Absorbance of fine particles	0.79	0.69	0.75	1.00

Abbreviation: NO_2, nitrogen dioxide

Table S6. Participant characteristics according to NO_2 levels during fetal life

	NO_2 levels ($\mu g/m^3$)			
	Low (<37.1)[a]	Medium (37.1-41.7)[a]	High (>41.7)[a]	P Value[b]
Maternal education level				.24
Primary education	7.8	8.7	4.4	
Secondary education	47.3	43.3	43.8	
University education	44.9	48.0	51.8	
Paternal education level				.02
Primary education	4.1	10.2	3.0	
Secondary education	44.3	36.0	42.2	
University education	51.6	53.8	54.8	
Monthly household income				.13
<1,200€	16.5	16.5	9.4	
1,200€ - 2,000€	16.5	19.0	17.6	
>2,000€	67.0	64.6	73.0	
Maternal country of birth				.24
The Netherlands	69.0	58.5	68.0	
Cape Verde	5.4	6.6	2.0	
Morocco	4.3	6.2	3.5	
Surinam	6.2	6.6	6.6	
Turkey	3.5	4.7	5.5	
Other country of birth	11.6	17.4	14.5	
Paternal country of birth				.55
The Netherlands	76.0	63.4	78.7	
Cape Verde	2.9	3.4	1.4	
Morocco	1.5	3.4	0.9	
Surinam	6.4	6.4	2.4	
Turkey	2.9	3.9	3.3	
Other country of birth	10.3	19.5	13.3	
Maternal age (years)	30.3 (5.0)	30.6 (5.2)	31.3 (4.6)	.04
Paternal age (years)	32.9 (5.3)	32.8 (5.4)	33.1 (5.2)	.89
Family status (mono *vs.* biparental)	13.0	14.4	13.1	.88
Maternal parity (multi *vs.* nulliparous)	39.8	41.2	37.5	.90
Maternal smoking use during pregnancy				.83
Never	77.3	75.3	73.4	
Smoking use until pregnancy known	6.1	5.8	7.6	
Continued smoking use during pregnancy	16.6	18.9	19.0	
Maternal alcohol use during pregnancy				.48
Never	39.3	36.9	36.6	
Alcohol use until pregnancy known	11.3	14.3	17.2	
Continued alcohol use during pregnancy	49.4	48.8	46.2	
Maternal pre-pregnancy BMI (kg/m^2)	24.7 (4.4)	24.7 (4.4)	24.5 (4.1)	.84
Paternal pre-pregnancy BMI (kg/m^2)	25.7 (3.4)	25.3 (3.5)	25.0 (3.2)	.11
Maternal height (cm)	169.4 (6.9)	167.6 (7.4)	168.7 (7.7)	.03
Paternal height (cm)	183.5 (7.2)	182.1 (7.9)	182.9 (6.7)	.12
Maternal overall psychological distress	0.3 (0.3)	0.3 (0.5)	0.3 (0.4)	.16
Maternal intelligence quotient score	98.8 (14.2)	97.6 (13.5)	98.7 (13.8)	.55

Abbreviation: BMI, body mass index; NO2, nitrogen dioxide.
[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and one-way ANOVA test for continuous variables

Table S7. Participant characteristics according to fine particles levels during fetal life

	Fine particles levels (μg/m³)			
	Low (<19.7)[a]	**Medium (19.7-21.0)[a]**	**High (>21.0)[a]**	**P Value[b]**
Maternal education level				.05
Primary education	10.8	5.1	5.1	
Secondary education	41.4	44.4	48.6	
University education	47.8	50.6	46.2	
Paternal education level				.41
Primary education	8.0	3.6	5.7	
Secondary education	38.0	42.0	42.5	
University education	54.0	54.4	51.8	
Monthly household income				.003
<1,200€	20.7	13.0	8.7	
1,200€ - 2,000€	13.8	18.0	21.4	
>2,000€	65.5	69.0	69.9	
Maternal country of birth				.18
The Netherlands	64.5	64.5	66.5	
Cape Verde	7.8	3.5	2.7	
Morocco	5.1	5.0	3.9	
Surinam	7.0	7.3	5.1	
Turkey	3.9	5.4	4.3	
Other country of birth	11.7	14.3	17.5	
Paternal country of birth				.25
The Netherlands	69.9	73.5	74.7	
Cape Verde	6.0	0.5	1.4	
Morocco	1.0	3.0	1.8	
Surinam	6.5	4.4	4.1	
Turkey	4.0	3.4	2.8	
Other country of birth	12.6	15.2	15.2	
Maternal age (years)	30.9 (5.1)	30.8 (4.9)	30.5 (4.8)	.57
Paternal age (years)	33.1 (5.2)	33.0 (5.2)	32.7 (5.5)	.79
Family status (mono *vs.* biparental)	15.6	12.8	12.2	.50
Maternal parity (multi *vs.* nulliparous)	42.4	35.0	40.9	.03
Maternal smoking use during pregnancy				.011
Never	74.6	77.9	73.7	
Smoking use until pregnancy known	10.2	5.8	3.6	
Continued smoking use during pregnancy	15.2	16.3	22.7	
Maternal alcohol use during pregnancy				.15
Never	36.3	38.0	38.4	
Alcohol use until pregnancy known	10.1	15.7	16.8	
Continued alcohol use during pregnancy	53.6	46.3	44.8	
Maternal pre-pregnancy body mass index (kg/m²)	24.5 (4.4)	24.6 (4.3)	24.8 (4.2)	.66
Paternal pre-pregnancy body mass index (kg/m²)	25.8 (3.5)	25.0 (3.2)	25.2 (3.3)	.07
Maternal height (cm)	168.1 (7.2)	168.6 (7.4)	168.9 (7.6)	.45
Paternal height (cm)	182.7 (6.6)	182.9 (7.6)	183.0 (7.6)	.87
Maternal overall psychological distress	0.3 (0.4)	0.3 (0.4)	0.3 (0.4)	.61
Maternal intelligence quotient score	98.4 (15.0)	98.8 (13.5)	97.9 (13.1)	.77

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and one-way ANOVA test for continuous variables

Table S8. Participant characteristics according to coarse particles levels during fetal life

	Coarse particles levels (μg/m³)			
	Low (<11.3)[a]	**Medium (11.3-12.4)[a]**	**High (>12.4)[a]**	**P Value[b]**
Maternal education level				.04
Primary education	10.2	6.3	4.3	
Secondary education	43.9	48.6	41.9	
University education	45.9	45.1	53.8	
Paternal education level				.27
Primary education	8.5	4.9	3.9	
Secondary education	40.2	43.7	38.9	
University education	51.3	51.4	57.2	
Monthly household income				<.001
<1,200€	21.1	16.0	5.5	
1,200€ - 2,000€	17.2	18.2	17.7	
>2,000€	61.6	65.8	76.8	
Maternal country of birth				.007
The Netherlands	65.0	60.3	70.2	
Cape Verde	7.0	5.1	1.9	
Morocco	4.3	7.4	2.3	
Surinam	6.6	8.2	4.7	
Turkey	4.7	5.4	3.5	
Other country of birth	12.5	13.6	17.4	
Paternal country of birth				.008
The Netherlands	69.7	72.6	75.7	
Cape Verde	5.0	2.0	0.9	
Morocco	2.0	3.0	0.9	
Surinam	6.5	7.6	1.4	
Turkey	3.5	4.6	2.3	
Other country of birth	13.3	10.2	18.8	
Maternal age (years)	30.3 (5.1)	30.8 (5.2)	31.0 (4.4)	.28
Paternal age (years)	32.7 (5.6)	33.2 (5.4)	33.0 (5.0)	.67
Family status (mono *vs.* biparental)	16.1	13.9	10.6	.19
Maternal parity (multi *vs.* nulliparous)	39.1	42.5	36.9	.61
Maternal smoking use during pregnancy				.007
Never	72.4	79.2	74.6	
Smoking use until pregnancy known	10.7	5.1	3.6	
Continued smoking use during pregnancy	16.9	15.7	21.8	
Maternal alcohol use during pregnancy				.08
Never	39.3	41.1	32.5	
Alcohol use until pregnancy known	11.9	11.9	18.9	
Continued alcohol use during pregnancy	48.8	47.0	48.6	
Maternal pre-pregnancy body mass index (kg/m²)	24.3 (4.0)	25.1 (4.7)	24.6 (4.1)	.09
Paternal pre-pregnancy body mass index (kg/m²)	25.4 (3.4)	25.2 (3.5)	25.4 (3.1)	.87
Maternal height (cm)	168.7 (7.5)	168.4 (7.0)	168.5 (7.6)	.89
Paternal height (cm)	182.8 (6.9)	183.0 (7.6)	182.9 (7.4)	.96
Maternal overall psychological distress	0.3 (0.4)	0.3 (0.4)	0.3 (0.4)	.39
Maternal intelligence quotient score	98.6 (14.5)	98.0 (14.0)	98.5 (13.2)	.88

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and one-way ANOVA test for continuous variables

Table S9. Participant characteristics according to absorbance of fine particles levels during fetal life

	Absorbance of fine particles levels (μg/m³)			
	Low (<1.8)[a]	Medium (1.8-2.0)[a]	High (>2.0)[a]	P Value[b]
Maternal education level				.16
Primary education	9.1	7.4	4.4	
Secondary education	47.2	41.8	45.4	
University education	43.7	50.8	50.2	
Paternal education level				.40
Primary education	7.9	3.8	5.5	
Secondary education	38.9	44.6	39.2	
University education	53.2	51.6	55.3	
Monthly household income				.08
<1,200€	18.6	14.1	9.8	
1,200€ - 2,000€	18.2	17.9	17.0	
>2,000€	63.2	67.9	73.2	
Maternal country of birth				.08
The Netherlands	63.4	65.3	66.8	
Cape Verde	7.0	3.9	3.1	
Morocco	6.6	4.6	2.7	
Surinam	8.6	5.4	5.5	
Turkey	2.7	5.8	5.1	
Other country of birth	11.7	15.1	16.8	
Paternal country of birth				.31
The Netherlands	70.8	73.1	74.2	
Cape Verde	4.5	1.5	1.8	
Morocco	2.5	2.0	1.4	
Surinam	6.9	5.0	3.2	
Turkey	3.5	2.5	4.2	
Other country of birth	11.8	15.9	15.2	
Maternal age (years)	30.1 (5.1)	31.2 (4.8)	30.8 (4.8)	.04
Paternal age (years)	32.7 (5.1)	33.0 (5.7)	33.1 (5.2)	.75
Family status (mono *vs.* biparental)	14.2	14.1	12.3	.78
Maternal parity (multi *vs.* nulliparous)	40.4	41.9	36.2	.30
Maternal smoking use during pregnancy				.25
Never	77.8	76.1	72.2	
Smoking use until pregnancy known	7.8	5.8	5.8	
Continued smoking use during pregnancy	14.4	18.1	22.0	
Maternal alcohol use during pregnancy				.33
Never	39.5	34.8	38.4	
Alcohol use until pregnancy known	10.7	16.0	16.1	
Continued alcohol use during pregnancy	49.8	49.2	45.5	
Maternal pre-pregnancy body mass index (kg/m²)	24.8 (4.4)	24.7 (4.6)	24.4 (3.9)	.58
Paternal pre-pregnancy body mass index (kg/m²)	25.5 (3.4)	25.0 (3.2)	25.5 (3.4)	.28
Maternal height (cm)	168.5 (7.2)	168.1 (7.0)	169.1 (7.9)	.32
Paternal height (cm)	182.7 (7.1)	183.1 (7.4)	182.8 (7.5)	.84
Maternal overall psychological distress	0.3 (0.5)	0.3 (0.4)	0.3 (0.4)	.12
Maternal intelligence quotient score	98.5 (14.8)	98.3 (13.2)	98.3 (13.6)	.99

[a]Values are percentages for the categorical variables and mean (standard deviation) for the continuous variables.
[b]χ^2 test for categorical variables and one-way ANOVA test for continuous variables

Table S10. Thickness (in mm) of the identified thinner brain regions in relation to higher exposure to air pollution during fetal life

	Hemisphere	Size (mm^2)	Mean	(SD)	Minimum	Percentile 25	Median	Percentile 75	Maximum
Precuneus region	Right	936	3.14	(0.32)	1.61	3.01	3.22	3.36	3.97
Pars opercularis region	Right	753	3.00	(0.19)	2.23	2.88	3.02	3.14	3.46
Pars orbitalis region	Right	651	2.92	(0.30)	2.02	2.73	2.93	3.12	3.71
Rostral middle frontal region	Right	2995	2.73	(0.22)	2.00	2.60	2.76	2.89	3.23
Superior frontal region	Right	722	2.63	(0.27)	1.85	2.43	2.65	2.83	3.31
Cuneus region	Left	843	2.31	(0.25)	1.67	2.14	2.29	2.46	3.28
Lateral orbitofrontal region	Right	565	2.82	(0.33)	1.86	2.59	2.83	3.06	3.88
Fusiform region	Left	532	2.37	(0.24)	1.62	2.21	2.37	2.51	3.42

Abbreviations: SD, standard deviation

Table S11. Adjusted association between air pollution exposure during fetal life and cortical thickness (in mm) at 6-10 years of age restricting to those children without attention deficit hyperactivity, pervasive developmental, dysregulation, and aggressive problems

	Hemisphere	Size brain region (mm^2)	Coef.	(95% CI)[a]	P value
Fine particles exposure					
Precuneus region	Right	936	-0.045	(-0.062 to -0.028)	<.001
Pars opercularis region	Right	753	-0.024	(-0.033 to -0.014)	<.001
Pars orbitalis region	Right	651	-0.028	(-0.043 to -0.012)	.001
Rostral middle frontal region	Right	2,995	-0.029	(-0.041 to -0.018)	<.001
Superior frontal region	Right	722	-0.029	(-0.043 to -0.016)	<.001
Cuneus region	Left	843	-0.022	(-0.035 to -0.009)	.002
Coarse particles exposure					
Lateral orbitofrontal region	Right	565	-0.037	(-0.059 to -0.016)	.001
Absorbance particles exposure					
Fusiform region	Left	532	-0.105	(-0.160 to -0.049)	<.001

Abbreviations: CI, confidence interval; Coef, beta coefficient.
[a]Beta coefficient (95% Confidence Interval) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. Coefficients represent the differences in thickness (mm) per each increase of $5 \mu g/m^3$ of fine particles, $5 \mu g/m^3$ of coarse particles, and $10^{-5} m^{-1}$ of absorbance of fine particles.

Table S12. Adjusted association between air pollution exposure during fetal life and cortical thickness (in mm) at 6-10 years of age restricting to those children from non-smoking mothers during pregnancy

	Hemisphere	Size brain region (mm^2)	Coef.	(95% CI)[a]	P value
Fine particles exposure					
Precuneus region	Right	936	-0.048	(-0.065 to -0.032)	<.001
Pars opercularis region	Right	753	-0.026	(-0.035 to -0.016)	<.001
Pars orbitalis region	Right	651	-0.026	(-0.041 to -0.011)	<.001
Rostral middle frontal region	Right	2,995	-0.028	(-0.040 to -0.017)	<.001
Superior frontal region	Right	722	-0.027	(-0.041 to -0.013)	<.001
Cuneus region	Left	843	-0.016	(-0.029 to -0.003)	.016
Coarse particles exposure					
Lateral orbitofrontal region	Right	565	-0.042	(-0.063 to -0.022)	<.001
Absorbance of fine particles exposure					
Fusiform region	Left	532	-0.082	(-0.136 to -0.029)	.003

Abbreviations: CI, confidence interval; Coef, beta coefficient.
[a]Beta coefficient (95% Confidence Interval) from linear regression model adjusted for parental educational levels, monthly household income, parental countries of birth, parental ages, maternal prenatal smoking, maternal prenatal alcohol use, parental body mass indexes and heights, maternal parity, family status, maternal psychological distress, maternal intelligence quotient, and child sex, age, and genetic ancestry. Coefficients represent the differences in thickness (mm) per each increase of 5$\mu g/m^3$ of fine particles, 5$\mu g/m^3$ of coarse particles, and $10^{-5}m^{-1}$ of absorbance of fine particles.

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The 2016 global and national burden of diabetes mellitus attributable to $PM_{2.5}$ air pollution

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Summary

Background $PM_{2.5}$ air pollution is associated with increased risk of diabetes; however, a knowledge gap exists to further define and quantify the burden of diabetes attributable to $PM_{2.5}$ air pollution. Therefore, we aimed to define the relationship between $PM_{2.5}$ and diabetes. We also aimed to characterise an integrated exposure response function and to provide a quantitative estimate of the global and national burden of diabetes attributable to $PM_{2.5}$.

Methods We did a longitudinal cohort study of the association of $PM_{2.5}$ with diabetes. We built a cohort of US veterans with no previous history of diabetes from various databases. Participants were followed up for a median of 8·5 years, we and used survival models to examine the association between $PM_{2.5}$ and the risk of diabetes. All models were adjusted for sociodemographic and health characteristics. We tested a positive outcome control (ie, risk of all-cause mortality), negative exposure control (ie, ambient air sodium concentrations), and a negative outcome control (ie, risk of lower limb fracture). Data for the models were reported as hazard ratios (HRs) and 95% CIs. Additionally, we reviewed studies of $PM_{2.5}$ and the risk of diabetes, and used the estimates to build a non-linear integrated exposure response function to characterise the relationship across all concentrations of $PM_{2.5}$ exposure. We included studies into the building of the integrated exposure response function if they scored at least a four on the Newcastle-Ottawa Quality Assessment Scale and were only included if the outcome was type 2 diabetes or all types of diabetes. Finally, we used the Global Burden of Disease study data and methodologies to estimate the attributable burden of disease (ABD) and disability-adjusted life-years (DALYs) of diabetes attributable to $PM_{2.5}$ air pollution globally and in 194 countries and territories.

Findings We examined the relationship of $PM_{2.5}$ and the risk of incident diabetes in a longitudinal cohort of 1 729 108 participants followed up for a median of 8·5 years (IQR 8·1–8·8). In adjusted models, a 10 μg/m³ increase in $PM_{2.5}$ was associated with increased risk of diabetes (HR 1·15, 95% CI 1·08–1·22). $PM_{2.5}$ was associated with increased risk of death as the positive outcome control (HR 1·08, 95% CI 1·03–1·13), but not with lower limb fracture as the negative outcome control (1·00, 0·91–1·09). An IQR increase (0·045 μg/m³) in ambient air sodium concentration as the negative exposure control exhibited no significant association with the risk of diabetes (HR 1·00, 95% CI 0·99–1·00). An integrated exposure response function showed that the risk of diabetes increased substantially above 2·4 μg/m³, and then exhibited a more moderate increase at concentrations above 10 μg/m³. Globally, ambient $PM_{2.5}$ contributed to about 3·2 million (95% uncertainty interval [UI] 2·2–3·8) incident cases of diabetes, about 8·2 million (95% UI 5·8–11·0) DALYs caused by diabetes, and 206 105 (95% UI 153 408–259 119) deaths from diabetes attributable to $PM_{2.5}$ exposure. The burden varied substantially among geographies and was more heavily skewed towards low-income and lower-to-middle-income countries.

Interpretation The global toll of diabetes attributable to $PM_{2.5}$ air pollution is significant. Reduction in exposure will yield substantial health benefits.

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Introduction

Air pollution is an important global health problem.[1] $PM_{2.5}$—the most widely studied air pollutant—is associated with increased risk of cardiovascular disease, pulmonary disease, kidney disease, and other non-communicable diseases,[2,3] and contributed to about 4·2 million premature deaths in 2015.[4] A growing body of evidence strongly suggests an association between $PM_{2.5}$ pollution and the risk of diabetes.[5–11]

The *Lancet* Commission[12] on pollution and health published its report in October, 2017, and it provided a comprehensive review of the effect of the so-called pollutome on human health. The Commission outlined a glaring deficiency in evidence and provided a set of recommendations to fill important knowledge gaps. One of the recommendations outlined by the Commission is to “define and quantify the burden of diabetes attributable to $PM_{2.5}$ air pollution”.[12] An assessment of the global and national burden of diabetes attributable to $PM_{2.5}$ would provide a better understanding of the epidemiology of diabetes, identify endemic areas, and further contribute to the global and national discussions

Research in context

Evidence before this study

Previous epidemiological evidence suggests that environmental exposure to $PM_{2·5}$ is associated with risk of diabetes. However, the *Lancet* Commission on pollution and health identified knowledge gaps and outlined several research recommendations including the need to further "define and quantify the burden of diabetes attributable to $PM_{2·5}$ air pollution".

Added value of this study

This study addresses the research recommendation and provides evidence that ambient $PM_{2·5}$ pollution is associated with increased risk of diabetes. We examined the association in a longitudinal cohort of about 1·7 million US veterans, in which we control for relevant individual-level variables and ecological characteristics. We tested a positive control, as well as negative outcome and exposure controls to address concern about spurious causal inference. The study synthesised previous evidence to build an integrated exposure response function to characterise the risk of diabetes across all $PM_{2·5}$ concentrations experienced by humans. The integrated exposure response function was non-linear in that risk increased substantially above $PM_{2·5}$ concentrations of 2·4 μg/m³, and then exhibited a more moderate increase in risk at concentrations above 10 μg/m³. Additionally, the study suggests that in 2016, there were about 3·2 million cases of incident diabetes, and about 8·2 million healthy life years lost due to diabetes attributable to air pollution. The burden varied substantially by geography and was most pronounced in less developed countries.

Implications of all the available evidence

Taken together, the findings address the knowledge gap outlined in the *Lancet* Commission on pollution and health to "define and quantify the burden of diabetes attributable to $PM_{2·5}$ air pollution". Most importantly, the study shows that substantial risk exists at concentrations well below those outlined in the air quality standards of WHO and national and international regulatory agencies. Although the non-linearity of the integrated exposure response function suggests modest reduction in risk unless $PM_{2·5}$ is decreased substantially in high-pollution areas, given the considerable number of people living in heavily polluted geographies, even incremental reductions in $PM_{2·5}$ will ameliorate the burden of diabetes. Finally, we observed that the burden of diabetes attributable to $PM_{2·5}$ exhibited substantial geographical variability, and was more skewed towards regions that are least prepared to cope with the consequences of this excess burden. The results will possibly be helpful to promote the public's awareness about the effect of $PM_{2·5}$ pollution on the risk of diabetes, and serve to inform and guide policy making aimed at addressing health consequences of environmental air pollution.

on the hazardous effect of air pollution on diabetes. Therefore in this study, we aimed to further define the relationship of $PM_{2·5}$ and diabetes, using a longitudinal cohort study design. We also aimed to characterise an integrated exposure response function, using the body of evidence on the relationship of $PM_{2·5}$ pollution and diabetes; and to provide a quantitative estimate of the global and national burden of diabetes attributable to $PM_{2·5}$ in 194 countries and territories, using the Global Burden of Disease (GBD) methodologies.

Methods

Longitudinal cohort study design

We did a longitudinal cohort study of the association of $PM_{2·5}$ with diabetes. A cohort of US veterans with no previous history of diabetes was built by linking the US Department of Veterans Affairs' databases[13–23] with the US Environmental Protection Agency's (EPA) Community Multiscale Air Quality Modeling System of $PM_{2·5}$,[24,25] where time of cohort entry was set as date of last outpatient blood panel between Oct 1, 2003, and Sept 30, 2004. Further details on these datasets and cohort construction are provided in the appendix (pp 2–3). Participants were followed up for a median duration of 8·5 years. The outcome of incident diabetes was defined by International Classification of Diseases-9 code, diabetes medication prescription, or an HbA_{1c} measurement more than 6·4% (>46·4 mmol/mol); and participants were censored at death or end of follow-up (Sept 30, 2012). $PM_{2·5}$ exposure value was assigned on the basis of county of residence at time of cohort entry.

See Online for appendix

Cox proportional hazard models were used to examine the relationship between $PM_{2·5}$ and the risk of diabetes, with censoring at death or end of follow-up. Selection of covariates was informed by previous studies.[15,16–18,26] All models were adjusted for age, race, sex, estimated glomerular filtration rate, systolic blood pressure, hyperlipidaemia, chronic lung disease, cardiovascular disease, cancer, body-mass index, smoking status, use of an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, percentage of people in poverty in each county of residence, population density of county of residence, number of admissions to hospital before beginning of follow-up, and how many times serum creatinine was measured before beginning of follow-up. Further details on data sources, variable definitions, and statistical analyses are included in the appendix (pp 2–11). Missing data were not imputed. In analyses, a 95% CI of a hazard ratio (HR) that does not include unity was considered significant. In all analyses, $p<0·05$ was considered significant.

We additionally curated data from the US County Health Rankings datasets and controlled for US county level characteristics in the following six domains: health outcomes, health behaviours, clinical care, social and economic factors, physical environment, and demographics.[27,28] We also did a restricted cubic spline analysis to characterise the morphology of a non-linear association between $PM_{2·5}$ and the risk of diabetes;[29] assessed exposure in quartiles; assessed exposure in time-varying models, where geographical location was updated as participants moved and average annual exposure was matched to geographical location at any specific time; used the National Aeronautics and Space Administration's (NASA) Socioeconomic Data and Applications Center's Global Annual $PM_{2·5}$ Grids from Moderate Resolution Imaging Spectroradiometer, Multi-angle Imaging Spectroradiometer, and Sea-Viewing Wide Field-of View Sensor's aerosol optical depth remote spaceborne satellite sensing data[30,31] as an alternative data source for exposure; varied the spatial resolution of exposure definition where we assigned exposure levels on the basis of the nearest air monitoring station within 30 miles, 10 miles, and 5 miles; assessed the relationship between $PM_{2·5}$ and risk of all-cause mortality as a positive control;[1,32] assessed the relationship between ambient air sodium concentrations and risk of diabetes as a negative exposure control;[33] assessed the relationship between ambient air sodium concentrations and risk of all-cause mortality; and assessed the relationship between $PM_{2·5}$ and the risk of lower limb fracture. Further details on these sensitivity analyses are provided in the appendix (pp 5–11).

The use of a negative control is a valuable complement to other epidemiological methods and serves to identify and resolve both suspected and unsuspected sources of spurious causal inference including confounding, mismeasurements, and other biases, as well as design or analytic flaws.[34] Ambient air sodium concentration is measured by air monitoring stations; however, there is no biological basis to support an association between sodium concentrations in the air and the risk of diabetes. Therefore, ambient air sodium is an appropriate negative exposure control.[34] The negative outcome control was selected on the basis of the criteria outlined by Lipsitch and colleagues.[34] There is no previous knowledge of and no biological or mechanistic plausibility to explain an association between $PM_{2·5}$ and the risk of lower limb fracture. We therefore considered it a suitable negative outcome control.

Integrated exposure response function

An integrated exposure response function based on GBD methodologies was built to assess the risk of diabetes due to $PM_{2·5}$ across the spectrum of $PM_{2·5}$ exposure concentrations around the world.[4,35,36] A literature review was done, where we evaluated currently available literature on the associations between risk of diabetes and $PM_{2·5}$, passive smoking, and active smoking for the use in building an integrated exposure response function.[5–11,37–60] Passive smoking and active smoking were used as proxy exposures for high concentration of $PM_{2·5}$, because published literature on $PM_{2·5}$ tends to be from developed countries with these values on the lower end of the spectrum, therefore, leaving a scarcity of evidence on the relationship at higher concentrations of exposure.[4,35,36,61] Exposure attribution, as estimated by previous studies,[32,35] is derived from breathing rate (ie, average volume of air breathed per minute), and the $PM_{2·5}$ mass per cigarette, or ambient exposure due to living with someone who smokes.

Studies were included in the building of the integrated exposure response function if they scored at least a four on the Newcastle-Ottawa Quality Assessment Scale[10,62]—a nine-point scale for assessing quality of cohort studies—and were only included if the outcome was type 2 diabetes or all types of diabetes. Active smoking studies were only included if they contained a recorded dose-response of cigarettes per day, which was necessary for assigning a corresponding $PM_{2·5}$ exposure value, and if the reference group consisted of those who had never smoked. Passive smoking studies were included if the reference group had never smoked and were not exposed to passive smoke. Passive smoke was assigned a $PM_{2·5}$ exposure of 35 μg/m³, and active smoking 667 μg/m³ per cigarette per day.[4,35,36] Selected studies, along with the Veterans Affairs longitudinal cohort study presented here, were included in building the integrated exposure response function; details on included studies are presented in the appendix (pp 12, 19–28).

For the **Moderate Resolution Imaging Spectroradiometer** see https://modis.gsfc.nasa.gov/about/

For the **Multi-angle Imaging Spectroradiometer** see https://www-misr.jpl.nasa.gov/

For the **Sea-Viewing Wide Field-of View Sensor** see https://eospso.nasa.gov/missions/sea-viewing-wide-field-view-sensor

The integrated exposure–response function fits available epidemiological data using a Bayesian hierarchical modelling approach, and is based on GBD methodology, which has been described elsewhere in detail.[4,35,36] The theoretical minimum risk exposure level (TMREL) was assigned on the basis of a uniform distribution of $PM_{2·5}$ from 2·4 μg/m³ to 5·9 μg/m³, representing exposure values between the minimum and fifth percentiles of exposure distributions from outdoor air pollution cohort studies.[4,35,63] TMREL by its definition should minimise individual-level and population-level risk and be theoretically possible to achieve, but not necessarily affordable or feasible to achieve.[63] Studies were weighted using the quality effects approach.[64] Results were obtained from 1000 sets of simulated values.[4,35,36] The mean and 95% uncertainty intervals (UIs) are presented.

Estimation of the burden of diabetes due to $PM_{2·5}$

National annual $PM_{2·5}$ exposure estimates, which are population weighted and derived from the integration of satellite data, surface measurements, geographical data, and a chemical transport model, were obtained from GBD 2015.[4,65] Estimates are population weighted. Incident rates, years of life lived with disability (YLD), years of life lost (YLL), and disability-adjusted life-years (DALYs) of diabetes and all causes, and their UIs were obtained from GBD 2016.[66,67] The GBD methodology, explained elsewhere in detail,[36,68] estimates these measures by using data from specific published literature on diabetes and mortality in hierarchical models.[66,68–71] The GBD Population Estimates dataset provided population size.[72] Country income classifications were obtained from the World Bank.[73]

The population attributable fraction (PAF) of diabetes due to $PM_{2·5}$ represents the proportion of diabetes that would be eliminated if the $PM_{2·5}$ exposure was reduced to concentrations equal to or less than the TMREL. The PAF of diabetes due to $PM_{2·5}$ exposure above the TMREL was calculated with a GBD 2016 equation,[71] using risk estimates from the integrated exposure response function. The TMREL was set as a uniform distribution between 2·4 μg/m³ and 5·9 μg/m³, for which levels under the TMREL were treated as contributing no risk.[4] The attributable burden of disease (ABD), defined as the number of incident cases of diabetes per year attributable to $PM_{2·5}$ exceeding the TMREL, was calculated using estimates of diabetes from the GBD 2016 study[68] multiplied by the PAF of diabetes due to $PM_{2·5}$ exceeding the TMREL.

YLD due to diabetes is a measure of the burden placed on a population due to the ill-effects of living with diabetes. YLL due to diabetes is a measure of the burden placed on a population due to dying prematurely from diabetes. The DALY due to diabetes is a summary measure of YLD and YLL, and represents the total years of healthy life lost due to ill-health, disability, or early death due to diabetes.[74] YLD, YLL, and DALYs of diabetes due to $PM_{2·5}$ were estimated by multiplying the diabetes- specific GBD values of the corresponding measure by the PAF of diabetes due to $PM_{2·5}$ exceeding the TMREL.[36,68] Details of these measures are discussed in the appendix (pp 13–15).

Uncertainty in measurements was factored in our estimations through the generation of measures from a distribution of 10 000 estimates, and the median and 95% UIs are reported. Further details on estimation and UIs are presented in the appendix (pp 14, 15). Burden measures are reported as values, rates per 100 000 population, and age-standardised rates per 100 000 population. World maps of age-standardised ABD, YLD, YLL, and DALY rates are presented. Age-standardised DALY rates were additionally analysed by World Bank income classification and the socio-demographic index quintile.

Statistical analysis

We did all analyses in SAS (version 7.1). We generated maps using ArcMap (version 10.5). The study was approved by the Institutional Review Board of the VA Saint Louis Health Care System (Saint Louis, MO, USA).

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

We examined the relationship of $PM_{2·5}$ and the risk of incident diabetes in a longitudinal cohort of 1 729 108 participants followed up for a median of 8·5 years (IQR 8·1–8·8). The demographic and health characteristics of the cohort participants are detailed in the appendix (pp 16–17). $PM_{2·5}$ concentrations obtained from EPA ranged from 5·0 μg/m³ to 22·1 μg/m³. In models adjusted for individual-level sociodemographic and health characteristics, a 10·0 μg/m³ increase in $PM_{2·5}$ exposure was associated with increased risk of diabetes (HR 1·15, 95% CI 1·08–1·22; table 1). Because characteristics of geographies might confound the association between $PM_{2·5}$ and the risk of diabetes,[75] we curated the US County Health Rankings' datasets[27,28] and built analyses additionally controlling for 55 US county-level variables in the six domains aforementioned. Models additionally adjusting for US county characteristics yielded consistent results in that an increase in $PM_{2·5}$ was associated with increased risk of diabetes (HR 1·12, 95% CI 1·02–1·24; table 1). A spline analysis suggested that the relationship between $PM_{2·5}$ concentrations and the risk of incident diabetes increased with increased concentrations of $PM_{2·5}$ and then nearly plateaued at concentrations exceeding 12·0 μg/m³ (figure 1). The results were consistent in analysis considering $PM_{2·5}$ in quartiles; in that compared with quartile 1 (5·0–10·1 μg/m³), the risk was increased in quartile 2 (consisting of $PM_{2·5}$ concentrations of 10·2–11·8 μg/m³; HR 1·08, 95% CI 1·05–1·12) and then nearly plateaued in quartiles 3 and 4 (consisting of $PM_{2·5}$ concentrations ≥11·9 μg/m³; HR 1·13 [95% CI 1·07–1·18] for quartile 3, and 1·14 [1·10–1·19] for quartile 4; table 1). Results were consistent when exposure was treated as time varying (HR 1·18, 95% CI 1·10–1·25), where it was updated as cohort participants moved from one location to another and as $PM_{2·5}$ estimates changed over the duration of follow-up (table 1).

We additionally considered $PM_{2·5}$ estimates derived from NASA's spaceborne satellite sensors as an alternative data source to define ambient $PM_{2·5}$ exposure concentrations. Analyses considering these data yielded results consistent with those shown using exposure data obtained from the EPA ground-based air monitoring stations (HR 1·13, 95% CI 1·11–1·15; table 1). Results were consistent in models where exposure concentrations were assigned on the basis of the nearest air monitoring station within 30 miles, 10 miles, and 5 miles (appendix p 18).

We examined the association of $PM_{2·5}$ and risk of all-cause mortality where a priori observations suggest that an association is expected (ie, the positive outcome control).[1,76] Our results showed a significant association between $PM_{2·5}$ concentrations and the risk of death (HR 1·08, 95% CI 1·03–1·13; table 1). We tested the association between ambient air sodium concentrations and the risk of diabetes (ie, a negative exposure control);

	Exposure (data source)	Outcome	Sample size	Event rate	Incident rate per 100 000 person-years	HR (95% CI)
Primary model	$PM_{2·5}$* (EPA)	Diabetes	1 729 108	397 966 (23·0%)	3414·9	1·15 (1·08–1·22)
Additionally controlled for US county characteristics	$PM_{2·5}$* (EPA)	Diabetes	1 301 070	300 500 (23·1%)	3426·2	1·12 (1·02–1·24)
Exposure as quartiles						
5·0–10·1 μg/m³	$PM_{2·5}$* (EPA)	Diabetes	446 334	94 564 (21·2%)	3087·6	1·00 (Ref)
10·2–11·8 μg/m³	$PM_{2·5}$* (EPA)	Diabetes	442 939	102 456 (23·1%)	3431·4	1·08 (1·05–1·12)
11·9–13·6 μg/m³	$PM_{2·5}$* (EPA)	Diabetes	408 580	98 439 (24·1%)	3604·6	1·13 (1·07–1·18)
13·7–22·1 μg/m³	$PM_{2·5}$* (EPA)	Diabetes	431 255	102 507 (23·8%)	3566·1	1·14 (1·10–1·19)
Time-varying exposure	$PM_{2·5}$* (EPA)	Diabetes	1 729 108	397 966 (23·0%)	3414·9	1·18 (1·10–1·25)
Alternative exposure data source	$PM_{2·5}$* (NASA)	Diabetes	1 670 031	383 894 (23·0%)	3410·9	1·13 (1·11–1·15)
Positive outcome control	$PM_{2·5}$* (EPA)	All-cause mortality	1 729 108	368 387 (21·3%)	2740·7	1·08 (1·03–1·13)
Negative exposure control	Sodium† (EPA)	Diabetes	820 160	191 826 (23·4%)	3484·8	1·00 (0·99–1·00)
Negative exposure control	Sodium† (EPA)	All-cause mortality	820 160	173 240 (21·1%)	2718·8	1·00 (1·00–1·01)
Negative outcome control	$PM_{2·5}$* (EPA)	Lower limb fracture	1 729 108	96 165 (5·6%)	740·0	1·00 (0·91–1·09)

*HRs for every 10 μg/m³ increase in $PM_{2·5}$. †HRs for every IQR increase (0·045 μg/m³) in sodium. HR=hazard ratio. EPA=US Environmental Protection Agency. NASA=National Aeronautics and Space Administration.

Table 1: **Analyses of the Veterans Affairs longitudinal cohort study of the association of $PM_{2·5}$ and diabetes**

the results showed a non-significant association (HR 1·00, 95% CI 0·99–1·00; table 1). There was also no significant association between air sodium concentrations and the risk of all-cause mortality as a negative exposure control (HR 1·00, 95% CI 1·00–1·01) and no significant association between $PM_{2·5}$ and risk of lower limb fracture as a negative outcome control (1·00, 0·91–1·09; table 1).

A summary table listing the studies used in the analysis of synthesising the integrated exposure response function is provided in the appendix (pp 19–28). The integrated exposure response function showed that the risk of diabetes increased substantially for $PM_{2·5}$ concentrations above the lower bound of the TMREL of 2·4 μg/m³ then exhibited a more moderate increase in risk at concentrations above 10 μg/m³ (figure 2).

In 2016, the global burden of incident diabetes attributable to $PM_{2·5}$ was, in 1000s, 3002·9 (95% UI 2208·6–3798·9). Globally, ABD per 100 000 population was 40·62 (95% UI 29·9–51·4), and age-standardised ABD per 100 000 population was 40·4 (29·7–51·1; table 2). Global diabetes DALYs attributable to long-term exposure to $PM_{2·5}$ were 8·2 million (95% UI 5·8–11·0), consisting of 4·1 million (2·4–6·2) YLD and 4·1 million (3·1–5·1) YLL. The 2016 global YLD, YLL, and DALYs of diabetes attributable to $PM_{2·5}$ in 1000s, in rate per 100 000 population, and age-standardised rate per 100 000 population are reported in table 3. Age-standardised DALY rates per 100 000 population increased as World Bank income classification decreased, and also as sociodemographic index decreased (figure 3).

Tables 2 and 3 also report the ABD, YLD, YLL, and DALYs for the ten most populated countries. ABD, YLD,

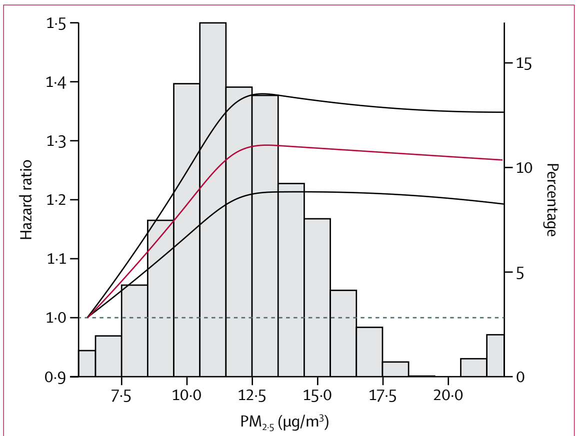


Figure 1: **Spline analysis of $PM_{2·5}$ and the risk of diabetes**
The red line is the hazard ratio. The black lines are the 95% CIs. A histogram of the distribution of $PM_{2·5}$ exposure is presented in the background in grey. The lowest $PM_{2·5}$ value included in the analysis was 6·2 μg/m³ and it served as the reference.

YLL, and DALYs for the 194 countries and territories are provided in the appendix (pp 29–53). Among the ten most populated countries, China had the highest ABD of 600·3 (95% UI 447·2–757·3), followed by India with an ABD of 590·5 (447·0–737·1), and then the USA, with an ABD of 149·5 (85·2–210·3) in 1000s (table 2). Pakistan had an ABD per 100 000 population of 58·8 (95% UI 44·1–74·3), followed by the USA with an ABD per 100 000 population of 46·3 (26·4–65·1), and then India with an ABD per 100 000 population of 44·9 (34·0–56·0). Age-standardised ABD showed that Pakistan had the highest with 72·6 (95% UI 54·4–91·8), followed by India with 48·7 (36·9–60·8), and then Bangladesh with 48·6 (37·2–60·2) incident cases of diabetes per 100 000 population. There was substan-

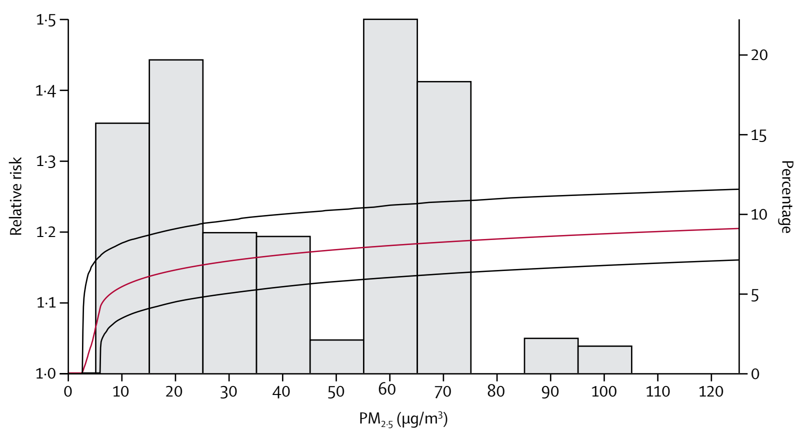


Figure 2: **Integrated exposure response function of the association between $PM_{2·5}$ and diabetes**
A histogram of the distribution of $PM_{2·5}$ exposure among the countries is presented in the background in grey. The red line is the mean estimated relative risk. The black lines are 95% uncertainty intervals.

	$PM_{2·5}$ exposure concentration (µg/m³)	Attributable burden of disease in 1000s (95% UI)	Attributable burden of disease per 100 000 population (95% UI)	Age-standardised attributable burden of disease per 100 000 population (95% UI)
Global	42·3	3002·9 (2208·6–3798·9)	40·6 (29·9–51·4)	40·4 (29·7–51·1)
China	57·2	600·3 (447·2–757·3)	43·9 (32·7–55·4)	37·1 (27·8–46·6)
India	72·6	590·5 (447·0–737·1)	44·9 (34·0–56·0)	48·7 (36·9–60·8)
USA	8·3	149·5 (85·2–210·3)	46·3 (26·4–65·1)	37·4 (21·3–52·5)
Indonesia	15·0	104·8 (69·1–141·0)	40·7 (26·8–54·7)	41·3 (27·3–55·4)
Brazil	11·1	49·8 (31·0–68·6)	23·8 (14·8–32·7)	23·6 (14·7–32·5)
Pakistan	63·0	112·4 (84·2–141·9)	58·8 (44·1–74·3)	72·6 (54·4–91·8)
Nigeria	36·9	24·6 (17·7–31·6)	13·3 (9·6–17·1)	21·1 (15·2–27·1)
Bangladesh	87·0	69·8 (53·4–86·5)	43·1 (33·0–53·5)	48·6 (37·2–60·2)
Russia	15·8	36·17 (23·9–49·0)	24·8 (16·3–33·6)	19·9 (13·2–27·0)
Japan	13·1	34·9 (22·2–48·3)	27·7 (17·7–38·4)	21·8 (14·0–29·7)

UI=uncertainty interval.

Table 2: **Attributable burden of diabetes associated with $PM_{2·5}$ exposure globally and for the top ten most populous countries**

tial geographical heterogeneity in age-standardised ABD per 100 000 population; the burden was high in several geographical regions including Central America, north Africa and the Middle East, southern sub-Saharan Africa, south Asia, Oceania, and several countries in southeast Asia (figure 4A; appendix pp 29–35). By contrast, several countries had low age-standardised ABD per 100 000 population, including Australia, New Zealand, and Greenland, as well as some of those in central Europe and central Asia (figure 4A; appendix pp 29–35).

Among the ten most populated countries, India had the highest DALYs (1625·8, 95% UI 1193·7–2104·8), followed by China (1251·5, 828·5–1753·3), and then Indonesia (400·0, 261·7–544·5), in 1000s (table 3). DALYs per 100 000 population showed Indonesia as the highest with 155·1 DALYs (95% UI 101·5–211·1), followed by India with 123·4 (90·7–159·9), and then the USA with 108·5 (59·3–163·9). Age-standardised DALYs per 100 000 population showed Pakistan as the highest with an age-adjusted DALY rate of 221·7 (95% UI 159·0–291·6), followed by Indonesia with 189·4 (124·4–255·7), and then India with 165·5 (122·5–212·3; table 3).

Mapping of the geographical distribution of age-standardised DALYs across the world showed populations in Central America, north Africa and the Middle East, southern sub-Saharan Africa, south Asia, and several countries in southeast Asia exhibited high age-standardised DALYs (figure 4B; appendix pp 36–51). Canada, Greenland, several countries in central and eastern Europe as well as central Asia, Russia, and Australia and New Zealand had low estimates of age-standardised DALYs (figure 4B; appendix pp 36–51). Finally, our estimates suggest that in 2016 there were 206 105 (95% UI 153 408–259 119) global deaths from diabetes attributable to $PM_{2·5}$ exposure.

Discussion

Our results suggest that there is a significant association between increased $PM_{2·5}$ exposure and the risk of diabetes. Additionally, our integrated exposure response function suggests that risk is significant at concentrations below those recommended by regulatory agencies. Finally, we observed substantial geographical variation in the burden of diabetes attributable to air pollution, for which we estimated that in 2016, there were about 3·2 million cases of incident diabetes and about 8·2 million years of healthy life lost due to diabetes attributable to elevated concentrations of $PM_{2·5}$.

The association of $PM_{2·5}$ pollution and the risk of diabetes is remarkably consistent across a number of studies from different populations; it is consistent when using EPA or NASA data to define exposure, and it passed the scrutiny of application of both positive and negative controls. The application of negative exposure and outcome controls is especially important to identify non-causal associations and serves as an important complement to other epidemiological methods for improving causal inference.[34] The biological mechanism underpinning the association is based on the premise that pollutants enter the bloodstream where they might interact with tissue components to produce pathological effects. This mechanism is now supported by evidence both in experimental models and humans that inhaled nanoparticles, which when sufficiently small can enter the bloodstream and interact with distant organs—including liver tissue—and exhibit affinity to accumulate at sites of vascular inflammation.[77,78] Furthermore, experimental and human evidence suggests that exposure to ambient air pollutants can lead to clinically significant disturbances in the autonomic nervous system, oxidative stress, inflammation, endoplasmic reticulum stress, apoptosis, and broad metabolic derangements in glucose and insulin homoeostasis including glucose intolerance, decreased insulin sensitivity and impaired secretion, and increased blood lipid concentrations, thus providing biological mechanistic plausibility to the association of $PM_{2·5}$ exposure and the risk of diabetes.[79–87]

Our integrated exposure response function suggests that the risk of diabetes increased substantially between the TMREL and the air quality standards recommended by WHO (10 µg/m³) and the EPA (12 µg/m³); there was a more moderate increase in the risk at $PM_{2·5}$ concentrations greater than 10 µg/m³. The findings are consistent with recent data[2,3,88] suggesting that even low concentration of air pollution might be unsafe, in which the unfortunate effect of air pollution becomes obvious at relatively low concentrations below those currently considered safe by regulatory agencies. The morphology of our integrated exposure response function is congruent with observations from other studies that examined the effect of $PM_{2·5}$ and other non-communicable diseases,[4] in which following an initial sharp increase the risk also nearly plateaued and subsequently exhibited minimal increase in risk.[4] The non-linear integrated exposure response function implies that in the most polluted countries a modest reduction in $PM_{2·5}$ will yield small reduction in risk; however, given the large populations living in heavily polluted geographies, even small incremental reductions in $PM_{2·5}$ will yield substantial reduction in the burden of diabetes.

The toll of diabetes attributable to $PM_{2·5}$ pollution is substantial; long-term exposure to $PM_{2·5}$ contributed to about 3·2 million cases of diabetes in 2016, representing 14% of total incident diabetes globally. It contributed to about 8·2 million DALYs representing 14·4% of DALYs due to diabetes and 0·3% of the overall global toll of DALYs due to all diseases. The high toll is driven in part by the fact that more people breathe polluted air than ever before, as average population-weighted $PM_{2·5}$ exposure has increased by 11·2% from 39·7 µg/m³ in 1990 to 44·2 µg/m³ in 2015. Estimates of $PM_{2·5}$ attributable diabetes at the global and national levels reflect the influence not only of the increase in population-weighted $PM_{2·5}$ exposure, but also of demographic expansion and underlying epidemiological trends of increased burden of non-communicable disease in general, and more specifically diabetes.

Our results suggest substantial geographical variation in the burden of diabetes attributable to air pollution and that the burden is more heavily skewed toward regions that are less developed (low-income and lower-to-middle-income countries, and countries with a lower sociodemographic index). As countries develop economically and undergo an epidemiological transition, non-communicable diseases are likely to become even

	YLD			YLL			DALYs		
	YLD in 1000s (95% UI)	YLD per 100 000 population (95% UI)	Age-standardised YLD per 100 000 population (95% UI)	YLL in 1000s (95% UI)	YLL per 100 000 population (95% UI)	Age-standardised YLL per 100 000 population (95% UI)	DALYs in 1000s (95% UI)	DALYs per 100 000 population (95% UI)	Age-standardised DALYs per 100 000 population (95% UI)
Global	4081·6 (2364·6–6158·6)	55·2 (32·0–83·3)	57·0 (33·1–85·8)	4140·9 (3087·1–5136·1)	56·0 (41·8–69·5)	60·0 (44·8–74·5)	8217·4 (5778·1–10971·5)	111·2 (78·2–148·4)	116·9 (82·6–155·4)
China	829·8 (455·0–1274·2)	60·7 (33·3–93·2)	50·1 (27·4–77·1)	423·4 (321·7–519·3)	31·0 (23·5–38·0)	27·1 (20·6–33·2)	1251·5 (828·5–1753·3)	91·6 (60·6–128·3)	77·1 (51·2–107·7)
India	693·3 (396·7–1040·9)	52·7 (30·2–79·1)	63·2 (36·9–94·2)	933·3 (718·8–1140·9)	70·9 (54·6–86·7)	102·3 (78·7–125·1)	1625·8 (1193·7–2104·8)	123·4 (90·7–159·9)	165·5 (122·5–212·3)
USA	216·2 (107·8–354·2)	67·0 (33·4–109·7)	50·0 (25·0–81·8)	134·4 (76·8–188·5)	41·6 (23·8–58·4)	30·7 (17·6–43·1)	350·4 (191·6–529·1)	108·5 (59·3–163·9)	80·7 (43·9–122·2)
Indonesia	124·0 (65·5–198·1)	48·1 (25·4–76·8)	55·0 (29·2–87·7)	275·8 (183·4–363·4)	106·9 (71·1–140·9)	134·0 (89·1–176·6)	400·0 (261·7–544·5)	155·1 (101·5–211·1)	189·4 (124·4–255·7)
Brazil	71·6 (36·8–116·1)	34·1 (17·5–55·4)	34·9 (18·0–56·4)	120·5 (75·6–162·7)	57·4 (36·1–77·5)	63·2 (39·6–85·3)	192·2 (117·9–271·5)	91·6 (56·2–129·4)	98·1 (60·3–138·0)
Pakistan	120·0 (69·2–180·1)	62·9 (36·3–94·3)	93·5 (54·9–139·4)	132·1 (95·2–173·2)	69·2 (49·9–90·7)	127·7 (92·1–167·4)	252·5 (178·4–337·2)	132·2 (93·4–176·6)	221·7 (159·0–291·6)
Nigeria	26·1 (14·9–39·7)	14·1 (8·1–21·5)	27·8 (16·0–42·1)	33·5 (22·3–46·7)	18·1 (12·1–25·3)	45·4 (30·1–63·5)	59·9 (41·1–81·1)	32·4 (22·3–43·9)	73·6 (50·6–99·6)
Bangladesh	78·8 (45·0–118·3)	48·7 (27·8–73·1)	63·3 (36·8–94·2)	86·6 (65·5–108·9)	53·5 (40·4–67·2)	87·5 (66·2–109·9)	165·6 (120·5–215·9)	102·3 (74·4–133·4)	151·0 (111·6–194·4)
Russia	64·6 (35·6–101·4)	44·3 (24·4–69·4)	32·3 (17·8–50·6)	28·2 (15·9–43·6)	19·3 (10·9–29·9)	14·3 (8·2–21·9)	93·2 (56·1–138·1)	63·8 (38·4–94·6)	46·8 (28·1–69·4)
Japan	89·0 (45·7–144·3)	70·8 (36·3–114·8)	41·9 (21·4–68·0)	13·3 (8·7–17·7)	10·6 (6·9–14·1)	5·1 (3·3–6·8)	102·3 (56·2–159·8)	81·4 (44·7–127·1)	46·9 (25·4–74·0)

YLD=years of life lived with disability. UI=uncertainty interval. YLL=years of life lost. DALYs=disability-adjusted life-years.

***Table 3:* YLD, YLL, and DALYs of diabetes associated with $PM_{2·5}$ exposure globally and for the top ten most populous countries**

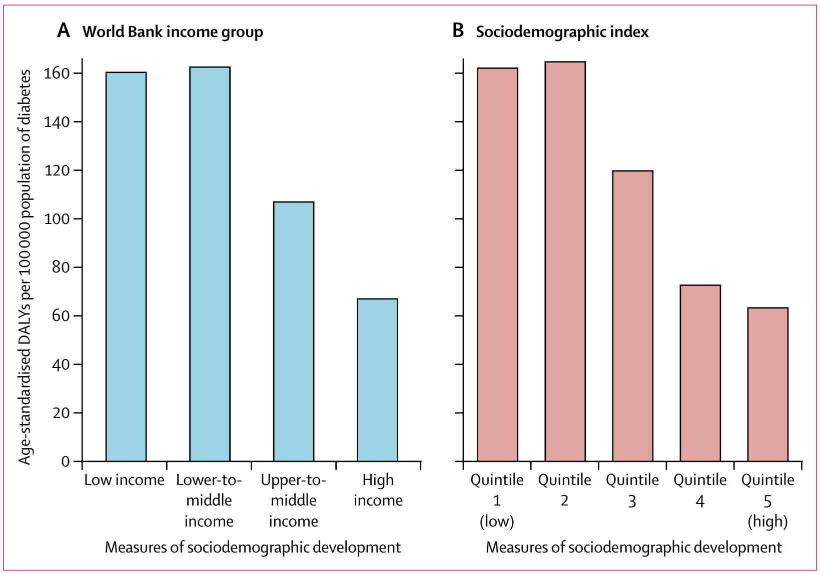


Figure 3: **Age-standardised DALYs per 100 000 population of diabetes by World Bank income group (A) and sociodemographic index quintile (B)**
DALYs=disability-adjusted life-years.

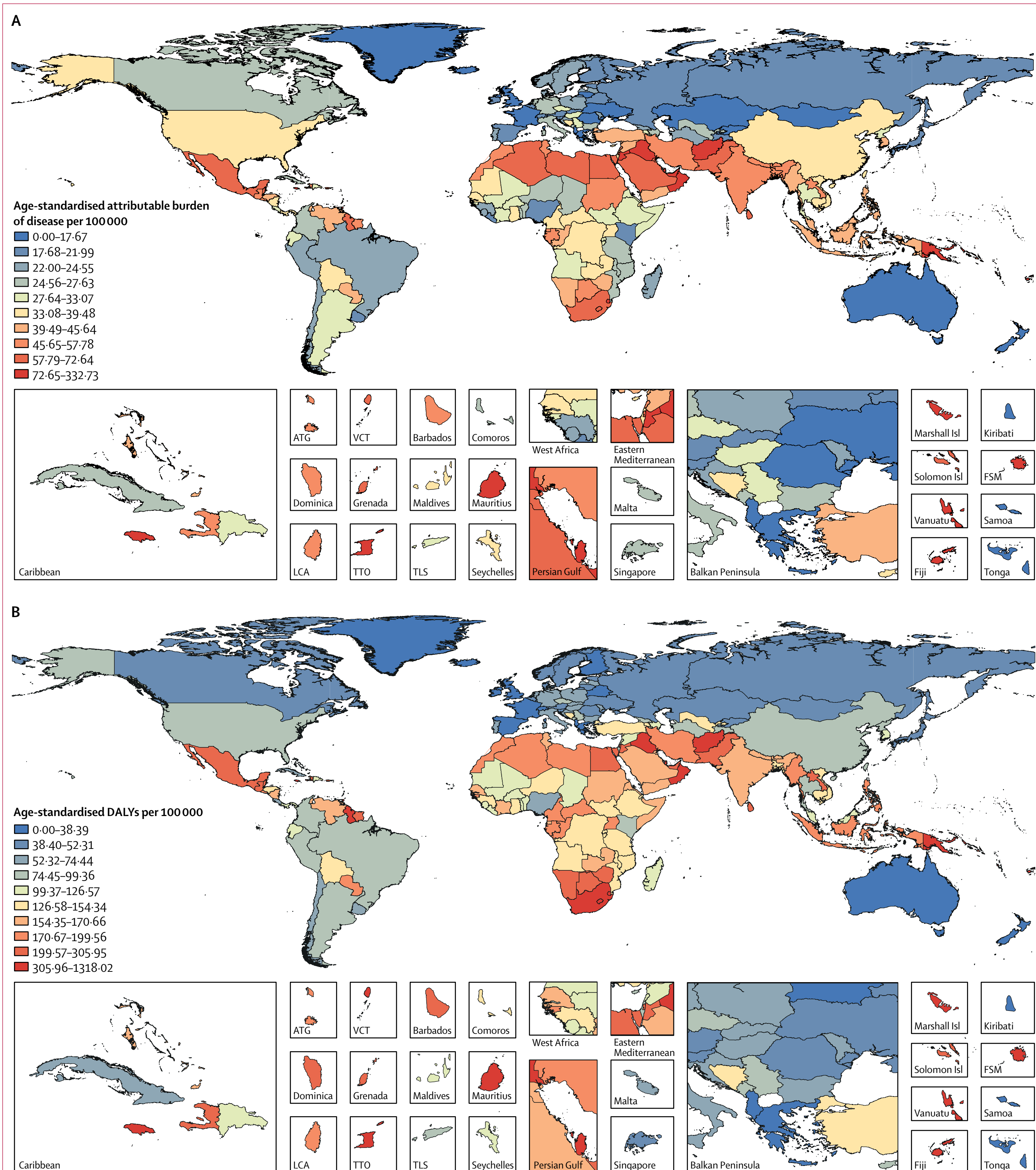
more prominent as major causes of disease and death, and the contribution of air pollution to non-communicable diseases in general, and specifically to diabetes will probably become even more pronounced. The forces of demographic expansion, ageing, epidemiological transition, and rapid industrialisation in low-income and lower-to-middle-income countries will probably increase the burden of health loss and death due to air pollution. The burden of health loss from diabetes attributable to $PM_{2·5}$ pollution is not insignificant in well developed countries and in geographies with relatively lower air pollution. Developing a better understanding of the effect of low concentrations of pollution (those currently considered safe) on health should be also be addressed by funding agencies and the scientific community.[88] Scientific evidence to define concentrations of particulate matter that are safe is needed to inform advocacy and guide policy making.

This study has several limitations. Our analyses neither considered the source of $PM_{2·5}$ nor the chemical composition and toxic content of $PM_{2·5}$, which might vary within and among countries; however, studies have shown that estimates using non-specific $PM_{2·5}$ biomass alone will underestimate the burden of disease attributable to $PM_{2·5}$ pollution.[1,4,65] Our study focused on quantitating the burden of diabetes associated with $PM_{2·5}$ exposure (ie, the *Lancet* Commission on pollution and health research recommendation number two); however, evaluation of the burden of diabetes associated with exposure to other pollutants including carbon monoxide, nitrogen dioxide, and others should be undertaken in future research.[86,89] Although we accounted for several individual-level and county-level health characteristics, used two different data sources to define exposure, and took care to vary the spatial resolution of exposure definition, our analyses do not account for individual-level differences in socioeconomic status, physical activity,[90–92] and $PM_{2·5}$ exposure; however, the successful application of both a negative exposure control and negative outcome control lessens the concern about residual confounding. Our analyses do not provide insight into the subnational level; this level is particularly important because several countries are especially large and there is likely to be substantial national geographical variation in both $PM_{2·5}$ and underlying morbidity and mortality rates related to diabetes (eg, in India and China) that is not captured in our analyses. In this study, we used estimates for incident diabetes generated by the GBD study group, and although these Bayesian estimates are considered robust, they are limited by the quality of the available data.[93] Furthermore, variability and inconsistency of data collection methods and tools across the countries could influence geographical variations.[93] Because data for the relationship of $PM_{2·5}$ and the risk of diabetes was primarily derived from studies done in countries with relatively lower $PM_{2·5}$ air pollution (eg, USA, Canada, and western Europe), we relied on active and passive smoking as proxies for exposure to higher concentrations of $PM_{2·5}$ pollution to build our integrated exposure response function;[35] this analytical strategy is well accepted, widely used, and represents the optimal methodological approach to quantitate the risk of disease associated with $PM_{2·5}$ exposure given the available data.[1,4,35,65,94]

Our study also had key strengths, such as the examination of the relationship between $PM_{2·5}$ and the risk of diabetes in a longitudinal cohort for which we also tested a positive control, negative exposure control, and negative outcome control to resolve concerns about causal inference. We also leveraged the availability of data from GBD 2016, which is the most comprehensive compilation and analysis of global health information available. We use GBD methodologies including the concept of DALYs to comprehensively capture the burden of disease across the world and a measure of uncertainty.

In conclusion, we provided evidence for a relationship between $PM_{2·5}$ and the risk of diabetes, we synthesised available evidence and integrated it to build an exposure response function describing the risk of diabetes at each level of ambient $PM_{2·5}$ exposure, and we quantitated the burden of diabetes including the number of incident cases of diabetes per year, and the years of healthy life lost due to diabetes attributable to $PM_{2·5}$.

Figure 4: **Age-standardised burden of incident diabetes attributable to $PM_{2·5}$ per 100 000 population (A) and age-standardised DALYs due to incident diabetes attributable to $PM_{2·5}$ per 100 000 population (B)**
DALYs=disability-adjusted life-years. ATG=Antigua and Barbuda. VCT=Saint Vincent and the Grenadines. LCA=Saint Lucia. TTO=Trinidad and Tobago. Isl=Island. FSM=Federated States of Micronesia. TLS=Timor-Leste.


A
Age-standardised attributable burden of disease per 100 000
0·00–17·67
17·68–21·99
22·00–24·55
24·56–27·63
27·64–33·07
33·08–39·48
39·49–45·64
45·65–57·78
57·79–72·64
72·65–332·73
Caribbean
ATG
VCT
Barbados
Comoros
West Africa
Eastern Mediterranean
Balkan Peninsula
Marshall Isl
Kiribati
Dominica
Grenada
Maldives
Mauritius
Malta
Solomon Isl
FSM
Vanuatu
Samoa
LCA
TTO
TLS
Seychelles
Persian Gulf
Singapore
Fiji
Tonga
B
Age-standardised DALYs per 100 000
0·00–38·39
38·40–52·31
52·32–74·44
74·45–99·36
99·37–126·57
126·58–154·34
154·35–170·66
170·67–199·56
199·57–305·95
305·96–1318·02
Caribbean
ATG
VCT
Barbados
Comoros
West Africa
Eastern Mediterranean
Balkan Peninsula
Marshall Isl
Kiribati
Dominica
Grenada
Maldives
Mauritius
Malta
Solomon Isl
FSM
Vanuatu
Samoa
LCA
TTO
TLS
Seychelles
Persian Gulf
Singapore
Fiji
Tonga

Contributors
BB, YX, and ZA-A did the background research and study design. BB and YX collected the data. BB, YX, and ZA-A analysed and interpreted the data. BB and ZA-A drafted the manuscript. ZA-A supervised and provided mentorship. Each author contributed important intellectual content during manuscript drafting or revision. All authors accept accountability for the overall work.

Declaration of interests
We declare no competing interests.

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Assessing the Distribution of Air Pollution Health Risks within Cities: A Neighborhood-Scale Analysis Leveraging High-Resolution Data Sets in the Bay Area, California

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Background: Air pollution-attributable disease burdens reported at global, country, state, or county levels mask potential smaller-scale geographic heterogeneity driven by variation in pollution levels and disease rates. Capturing within-city variation in air pollution health impacts is now possible with high-resolution pollutant concentrations.

Objectives: We quantified neighborhood-level variation in air pollution health risks, comparing results from highly spatially resolved pollutant and disease rate data sets available for the Bay Area, California.

Methods: We estimated mortality and morbidity attributable to nitrogen dioxide (NO_2), black carbon (BC), and fine particulate matter [PM $\leq 2.5\ \mu m$ in aerodynamic diameter ($PM_{2.5}$)] using epidemiologically derived health impact functions. We compared geographic distributions of pollution-attributable risk estimates using concentrations from *a*) mobile monitoring of NO_2 and BC; and *b*) models predicting annual NO_2, BC and $PM_{2.5}$ concentrations from land-use variables and satellite observations. We also compared results using county vs. census block group (CBG) disease rates.

Results: Estimated pollution-attributable deaths per 100,000 people at the 100-m grid-cell level ranged across the Bay Area by a factor of 38, 4, and 5 for NO_2 [mean = 30 (95% CI: 9, 50)], BC [mean = 2 (95% CI: 1, 2)], and $PM_{2.5}$, [mean = 49 (95% CI: 33, 64)]. Applying concentrations from mobile monitoring and land-use regression (LUR) models in Oakland neighborhoods yielded similar spatial patterns of estimated grid-cell–level NO_2-attributable mortality rates. Mobile monitoring concentrations captured more heterogeneity [mobile monitoring mean = 64 (95% CI: 19, 107) deaths per 100,000 people; LUR mean = 101 (95% CI: 30, 167)]. Using CBG-level disease rates instead of county-level disease rates resulted in 15% larger attributable mortality rates for both NO_2 and $PM_{2.5}$, with more spatial heterogeneity at the grid-cell–level [NO_2 CBG mean = 41 deaths per 100,000 people (95% CI: 12, 68); NO_2 county mean = 38 (95% CI: 11, 64); $PM_{2.5}$ CBG mean = 59 (95% CI: 40, 77); and $PM_{2.5}$ county mean = 55 (95% CI: 37, 71)].

Discussion: Air pollutant-attributable health burdens varied substantially between neighborhoods, driven by spatial variation in pollutant concentrations and disease rates. https://doi.org/10.1289/EHP7679

Introduction

Air pollution is associated with a large burden of death and disability worldwide, with fine particulate matter [PM $\leq 2.5\ \mu m$ in aerodynamic diameter ($PM_{2.5}$)] estimated to be responsible for 4.9 million deaths globally in 2015 (GBD 2017 Risk Factor Collaborators 2018). Nitrogen dioxide (NO_2), a traffic-related air pollutant, is also linked with adverse health outcomes, although it is often not quantified in pollution-attributable disease burden studies, potentially because coarsely resolved concentration estimates are often unable to capture highly spatially variable patterns in NO_2 (Anenberg et al. 2017). Recent advances in the understanding of the health effects of NO_2, meta-analyses (Atkinson and Butland 2018; U.S. EPA 2016), and published recommendations from a committee of scientists (Atkinson and Butland 2018) provide guidance on evaluating and interpreting NO_2, as a marker of the mixture of traffic air pollution, in health impact assessments.

Much of the air pollution disease burden is concentrated in cities (Anenberg et al. 2019). Cities are home to about half the world's population (United Nations 2019) and 80% of the U.S. population (U.S. Census Bureau 2018). Many cities also experience both high air pollution levels (Krzyzanowski et al. 2014; Marlier et al. 2016) and health inequity challenges (Grant et al. 2017; Kioumourtzoglou et al. 2015; Stephens 2018). However, estimated health impacts from air pollution have typically been reported at the country, state, or county level, masking potential heterogeneity in impacts at fine spatial scales.

Understanding how air pollution-related health risks vary within cities could help inform policies aimed at improving public health and reducing population disparities in exposure and risk in urban areas. Recent efforts have estimated air pollution health impacts at the city level, finding dramatic variation in health risks across cities globally (Achakulwisut et al. 2019; Anenberg et al. 2019). However, only a limited number of studies have assessed air pollution mortality risks at the neighborhood level, and these have focused on individual cities and have generally not compared the advantages and disadvantages of different concentration data sources (Brønnum-Hansen et al. 2018; Kheirbek et al. 2013; Kihal-Talantikite et al. 2018; Martenies et al. 2018; Mueller et al. 2017, 2018, 2020; Pierangeli et al. 2020). In addition, these previous city-scale studies may not have captured the spatial distribution of air pollution-related health risks given that the grid sizes used in those studies can dilute

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hotspots of high concentrations co-located with large populations (Fenech et al. 2018; Korhonen et al. 2019; Li et al. 2016; Punger and West 2013). Beyond horizontal grid size, the resolution of emissions inputs to estimate concentrations can also influence the resulting estimated air pollution-related health impacts. Two studies examining the impacts of both varying horizontal grid and emissions resolution on health burden estimates report mixed results. Paolella et al. (2018) reported a reduced ability of coarse resolution concentration estimates to identify disparities in health impacts, whereas a study by Thompson et al. (2014) found limited difference for $PM_{2.5}$ attributable health impacts with varying emissions and grid resolution (Thompson et al. 2014).

Despite these differences, finer-resolution exposure estimates may decrease the potential for exposure misclassification. Estimating air pollution health impacts at the "hyperlocal" scale (resolving neighborhoods within cities) is now possible with high-resolution pollutant concentrations derived from mobile monitoring and modeling, complemented by satellite remote sensing. Here, we exploit a novel and extremely high-spatial–resolution pollution concentration data set from mobile monitoring of NO_2 and black carbon (BC) using Google Street View (hereafter referred to as Street View) cars throughout the Bay Area, California, from 2015 to 2017. Previously, these measurements have been used to create street-level annual average concentrations of NO_2 and BC, a land-use regression (LUR) model (Messier et al. 2018), and an epidemiological analysis of relationships between long-term exposure to NO_2 and cardiovascular disease (CVD) outcomes (Alexeeff et al. 2018), all for Oakland, California. Jointly, these efforts demonstrated the application of highly resolved concentration data to analyze intra-urban variation in pollutant exposure and the associated health risks. Building upon these efforts, here we use Street View concentrations to assess air pollution health impacts at the neighborhood scale. To our knowledge, our analysis is the first to use air pollution levels from sensor-aided mobile monitoring in a health impact assessment. Given that most cities globally do not have the same availability of highly spatially resolved concentration data as the Bay Area, we compare pollutant-attributable health risks estimated using the Street View concentrations vs. less data- and resource-intensive predictive models. These predictive models use land-use variables and satellite observations of aerosol optical depth (AOD) that can be applied in any city globally to create street-level annual average concentrations of NO_2 and $PM_{2.5}$ (Larkin et al. 2017; van Donkelaar et al. 2016).

Neighborhood-level health risks from air pollution are driven not just by exposure levels but also by baseline disease rates, which themselves vary within cities (e.g., Fann et al. 2012), influencing attributable mortality estimates (Chowdhury and Dey 2016; Hubbell et al. 2009). Prior air pollution morbidity and mortality assessments have typically used baseline disease rates at the state, county, or national level owing to the limited availability of more highly resolved health data (Alotaibi et al. 2019; Caiazzo et al. 2013; Cohen et al. 2017; Fann et al. 2012, 2017; Zhang et al. 2018). Here, in addition to comparing across concentration data sets, we also assess the influence of baseline disease rates with varying spatial resolutions (i.e., county-level vs. census block group (CBG)-level baseline disease rates) on estimated pollution-attributable health risks.

The San Francisco Bay Area of California has a population of >7 million people. This case study for the Bay Area, where high-resolution concentration and disease rate data are available, allows us to explore intra-urban disparities in air pollutant exposure, pollution-attributable health risks, and pollution-attributable disease burdens—three related but distinct metrics that are used in policy contexts. The objectives of our study were to *a*) identify the degree of spatial heterogeneity in air pollution-related health impacts at the neighborhood scale within a city; *b*) compare the spatial patterns of air pollution disease burdens estimated using different concentration and baseline disease rate data sets; and *c*) draw lessons learned for conducting neighborhood-scale air pollution health impacts in cities where highly resolved concentration and baseline disease rate data sets are not available. We anticipate that our results can be used to inform best practices (currently under development) for assessing air pollution-related health risks within cities globally, as well as efforts by policymakers to address disparities in the health impacts of air pollution.

Methods

We used epidemiologically derived health impact functions to estimate mortality and morbidity that may be attributable to NO_2, BC, and $PM_{2.5}$, on a 100-m grid resolution for the Bay Area, using different concentration inputs and varying spatial resolutions for baseline disease rates. We used the Bay Area Air Quality Management District's (BAAQMD) nine-county definition of the Bay Area, which included Alameda, Contra Costa, Marin, Napa, San Francisco, Santa Clara, San Mateo, Solano, and Sonoma counties (Figure 1). Within the Bay Area, we focused on Alameda County, for which we were able to obtain CBG-level disease rates, and within Alameda County, the areas of West, Downtown, and East Oakland, where the Street View cars measured pollution levels (Table 1). Oakland is home to a major container port and has four large interstates (I-880 to the south and west; I-80 and I-580 to the north; and I-980 transecting West and Downtown Oakland), as well as numerous rail yards and rail lines. East and West Oakland have been designated by the California Environmental Protection Agency (EPA) Environmental Justice Task Force as priority communities bearing disproportionate pollution burdens (Environmental Justice Task Force 2017).

Health Impact Function

For each pollutant–outcome pair, we derived concentration–response factors (CRFs) from relative risk (RR) estimates (Table 1) identified through a literature review using PubMed and Google Scholar (see the Supplemental Material "Literature Review" and Tables S1–S15 and Figures S1–S11). We used epidemiological studies with large geographic areas as opposed to those conducted in single cities, assuming large epidemiological studies more fully account for population variation and confounding factors and have more statistical power. Where available, we used pooled risk estimates from meta-analyses. We applied a log-linear function to all analyses, based on current evidence for $PM_{2.5}$ and, for NO_2, a combination of limited evidence for linear vs. log-linear functions and only small differences between the two at the concentrations in our study. Equation 1 describes the log-linear health impact function used for all pollutant–health end point pairs:

$$y_{h,i,a} = m_{h,i,a} \times P_{i,a} \times (1 - e^{-\beta_{h,a}\Delta x_i}) \tag{1}$$

where $y_{h,i,a}$ represents the number of cases of the health outcome (h) for age group (a) attributable to the pollutant for each grid cell (i); $m_{h,i,a}$ represents the baseline disease rate for each health end point (h), age group (a), and grid cell (i); $P_{i,a}$ represents the population count for each grid cell (i) and age group (a); and $1 - e^{-\beta_{h,a}\Delta x_i}$ represents the attributable fraction, with $\beta_{h,a}$ the natural log of the RR per x concentration above the baseline (Δx) in each grid cell (i), for each health end point (h) and age group (a). We accounted for uncertainty by calculating the attributable cases at the 2.5th and 97.5th percentiles of the RR estimates. All health

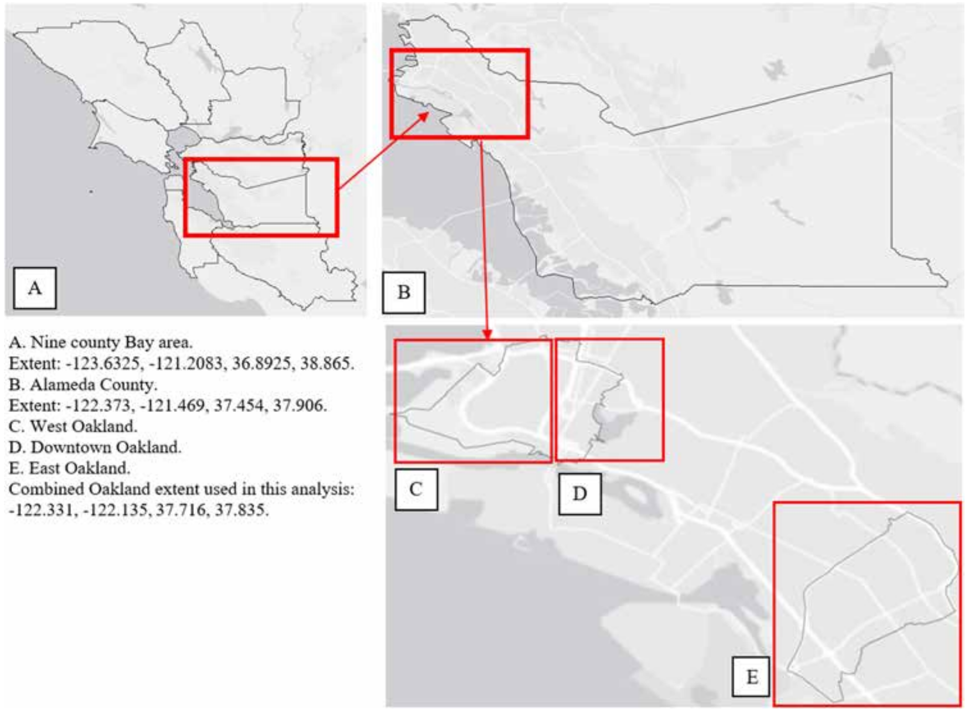

Figure 1. Geographic area of analysis for (A) the Bay Area, California, highlighting (B) Alameda County and (C) West, (D) Downtown, and (E) East Oakland within Alameda County. Base map data from ArcMap (version 10.4; Esri), HERE, Garmin, FAO, NOAA, USGS, © OpenStreetMap contributors, and the GIS User Community.

impact calculations were conducted in R (version 3.5.3; R Development Core Team).

For all pollutants, we assumed no threshold for low concentrations because a recent study identified health impacts at $PM_{2.5}$ concentrations as low as $2\,\mu g/m^3$ (Crouse et al. 2012) and a recent NO_2 epidemiological study included concentrations as low as 2 ppb (Khreis et al. 2017). Given that we applied a log-linear function to both $PM_{2.5}$ and BC, we likewise assumed no threshold for BC. For NO_2, the U.S. EPA (2016) determined that there are causal and likely causal relationships for short-term and long-term exposure and respiratory effects, respectively. Because we were able to obtain baseline disease rates for pediatric asthma emergency room (ER) visits and pediatric asthma incidence, two of which included respiratory outcomes included in the U.S. EPA's "Integrated Science Assessment for Nitrogen Oxides" (U.S. EPA 2016), we included these health end points for short- and long-term exposure to NO_2. Recent meta-analyses have also determined that there is a likely causal relationship between long-term exposure to NO_2 and increased risk of mortality (COMEAP 2018) and potentially for CVD mortality, the most commonly included cause-specific mortality end point among included studies in the meta-analysis (Atkinson et al. 2018). We estimated impacts of NO_2 on all-cause and CVD mortality. Although we examined NO_2, there remains active debate on the independent causal relationship between long-term NO_2 on mortality and other health outcomes. NO_2 is, however, a well-established marker of localized traffic-related air pollution, such as ultrafine particles and polycyclic aromatic hydrocarbons and is used as a proxy to estimate the mortality burden due to highly variable local traffic-related air pollution (Atkinson and Butland 2018) important for urban air pollution policy decision making.

For $PM_{2.5}$, we included health end points determined to be causal or likely to be causal by the U.S. EPA, including all-cause mortality, CVD mortality, CVD hospitalizations among the elderly, and pediatric asthma incidence and ER visits (U.S. EPA 2019). For BC, the U.S. EPA concluded that there is currently insufficient evidence to ascribe any one component of $PM_{2.5}$ as more strongly associated than total $PM_{2.5}$ mass, although some studies found associations between long-term exposure to BC and all-cause and CVD mortality, and between short-term BC exposure and CVD hospitalizations (U.S. EPA 2019). We therefore included all-cause and CVD mortality, as well as CVD hospitalizations for BC. Because applying the log-linear model to individual $PM_{2.5}$ components can distort the risk estimates given nonlinearity at the low end of the curve (Anenberg et al. 2012), we performed a sensitivity analysis in which we assumed the BC contribution to $PM_{2.5}$ mortality was the same as its contribution to $PM_{2.5}$ concentrations.

NO_2, BC, and $PM_{2.5}$ Concentrations

We used multiple pollutant concentration data sets, including mobile monitoring (BC and NO_2) and predictive models for the United States and globally using an LUR model (NO_2), and for the United States (BC and $PM_{2.5}$) and globally ($PM_{2.5}$) using satellite-based models. Maps of concentrations for each pollutant, data set source, and geographical extent are provided in Figures S12–S28.

For the mobile monitoring data set, two Street View cars equipped with fast-response instrumentation [NO_2 via cavity attenuation phase shift spectroscopy (Model T500U, Teledyne Inc.), and BC via photoacoustic absorption spectroscopy (Droplet Measurement Technologies)] repeatedly drove every road in West, Downtown, and East Oakland during daytime hours ($\sim$0900–1800 hours) on weekdays between 28 May 2015 and 21 December 2017, producing >3 million data points (Apte et al. 2017; Aclima et al. 2019). These measurements were aggregated to independent drive pass means, and then medians of the drive pass means were calculated for 30-m road segments, reflecting long-term spatial differences in concentrations (Messier et al.

Table 1. Relative risks (RRs) used for estimating the health impacts (95% CIs in parentheses) and inputs used for calculating each pollutant–health outcome pair.

Pollutant	Concentrations	Health outcome	Baseline disease rates	Age group	Study	RR (95% CI)
BC	Street view for Oakland and van Donkelaar et al. 2019 for Bay Area	All-cause mortality	Alameda County: CBG level Bay Area: county level	Adults	Janssen et al. 2011	1.007 (1.004, 1.009)
		CVD mortality	Alameda County: CBG level Bay Area: county level	Adults	Janssen et al. 2011	1.018 (1.011, 1.031)
		CVD hospitalizations	Bay Area: county level	Elderly	Peng et al. 2009	1.020 (1.008, 1.032)
NO_2	Street View for Oakland, Larkin et al. 2017 (main results) and Bechle et al. 2015 (sensitivity) for Bay Area	All-cause mortality	Alameda County: CBG level Bay Area: county level	Adults	Atkinson and Butland 2018	1.040 (1.011, 1.069)
			Alameda County: CBG level Bay Area: county level	Adults	Crouse et al. 2015	1.1025 (1.082, 1.145)
			Alameda County: CBG level Bay Area: county level	Elderly	Eum et al. 2019	1.023 (1.021, 1.026)
		CVD mortality	Alameda County: CBG level Bay Area: county level	Adults	Atkinson et al. 2018	1.006 (1.004, 1.009)
			Alameda County: CBG level Bay Area: county level	Elderly	Eum et al. 2019	1.103 (1.099, 1.108)
		Asthma incidence	State level for California	Pediatric	Khreis et al. 2017	1.258 (1.098, 1.374)
		Asthma ER visits	Bay Area: ZIP-code level	All ages	Orellano et al. 2017	1.024 (1.005, 1.043)
			Bay Area: ZIP-code level	All ages	Zheng et al. 2015	1.002 (1.001, 1.003)
			Bay Area: ZIP-code level	Pediatric	Orellano et al. 2017	1.040 (1.001, 1.081)
			Bay Area: ZIP-code level	Pediatric	Zheng et al. 2015	1.003 (1.002, 1.004)
$PM_{2.5}$	van Donkelaar et al. 2016 (main results) and Di et al. 2016 (sensitivity) for Bay Area	All-cause mortality	Alameda County: CBG level Bay Area: county level	Adults	Krewski et al. 2009	1.06 (1.04, 1.08)
			Alameda County: CBG level Bay Area: county level	Elderly	Di et al. 2017	1.084 (1.081, 1086)
		CVD mortality	Alameda County: CBG level Bay Area: county level	Adults	Turner et al. 2016	1.12 (1.09, 1.15)
			Alameda County: CBG level Bay Area: county level	Elderly	Thurston et al. 2016	1.100 (1.050, 1.150)
		CVD hospitalizations	Bay Area: county level	Elderly	Bravo et al. 2017	1.008 (1.006, 1.010)
		Asthma incidence	State level for California	Pediatric	Khreis et al. 2017	1.344 (1.105, 1.629)
		Asthma ER visits	Bay Area: ZIP-code level	Pediatric	Lim et al. 2016	1.048 (1.028, 1.067)

Note: RRs are reported per $10\,\mu g/m^3$ for $PM_{2.5}$, per 10 ppb for NO_2, and per $1\,\mu g/m^3$ for BC. RRs for NO_2 reported per $10\,\mu g/m^3$ were converted to RR per 10 ppb assuming ambient air pressure of 1 atmosphere and temperature of 25° C. Adults, 25–99 years of age; BC, black carbon; CBG, census block group; CI, confidence interval; CVD, cardiovascular disease; elderly, 65–99 years of age; ER, emergency room; NO_2, nitrogen dioxide; $PM_{2.5}$, fine particulate matter; pediatric, 0–17 years of age.

2018). The resulting data set indicated substantial spatial variability at fine scales, with median concentrations for road segments within the same city blocks observed to vary by up to a factor of five. Here, we further aggregated the 30-m segment averages to a $100\text{ m} \times 100\text{ m}$ grid resolution using a mean of all the mobile measurement points in each grid cell. This resulted in a concentration data set with an annual average NO_2 concentration range of 3.37 to 45 ppb [mean = 12.7, standard deviation (SD) = 6.6] and annual average BC concentration range of 0.2 (limit of detection) to $2.59\,\mu g/m^3$ (mean = 0.47, SD = 0.35).

For NO_2, LUR models offer full spatial coverage in addition to the very high spatial resolution needed to capture near-roadway concentrations (Hystad et al. 2011). Here, we used a global LUR that estimated annual average NO_2 at $100\text{ m} \times 100\text{ m}$ resolution for 2011 using satellite measurements, numerous land-use predictor variables, and annual measurement data from 5,220 air monitors in 58 countries (Larkin et al. 2017). The resulting NO_2 concentrations for 2011 in the Bay Area ranged from 1 to 37 ppb (mean = 8, SD = 4), and the model explained 54% (adjusted R^2 of 0.54) of the variance in global NO_2 concentrations, with an absolute mean error of 3.7 ppb. This data set has been applied in recent health impact assessments quantifying the global burden of NO_2 on pediatric asthma incidence (Achakulwisut et al. 2019). Because the global LUR model was not calibrated specifically for the United States, we also estimated results using a U.S.-specific LUR (Bechle et al. 2015). Results from the Street View concentrations were not included in the global LUR; therefore, we do not expect spatial distributions in concentrations to match. We reported estimates using the global LUR as the main results to inform best practices for neighborhood-scale health impact assessments in cities globally.

Although $PM_{2.5}$ was not measured by the Street View cars, $PM_{2.5}$ is more spatially homogenous compared with NO_2 and can therefore be estimated using more coarsely resolved predictive models. Therefore, we used surface concentrations derived from satellite observations of AOD from both global (van Donkelaar et al. 2016) and U.S.-specific models (van Donkelaar et al. 2019; Di et al. 2016). The global $PM_{2.5}$ data set [0.01×0.01 ($\sim 1\text{ km}^2$)-degree resolution] combined AOD from three satellite products, Goddard Earth Observing System (GEOS)-Chem chemical transport modeling, and geographically weighted regression to merge surface monitor *in situ* measurements of $PM_{2.5}$. The model accounted for 81% of the variance in $PM_{2.5}$ and resulted in annual average surface $PM_{2.5}$ concentrations ranging from 3 to $18.5\,\mu g/m^3$ (mean = 9, SD = 2.8) across the Bay Area for 2016. The global $PM_{2.5}$ data set was inclusive of BC, although the authors recently developed a North American product, employing similar methods to estimate $PM_{2.5}$ and speciated components of $PM_{2.5}$ also at 0.01×0.01-degree resolution. Although U.S. estimates for BC explained 68% of the total variance in BC, estimates for BC in the U.S. Northwest are considerably lower ($R^2 = 0.29$). For the North American data set in the Bay Area for 2016, BC concentrations for 2016 ranged from 0.1 to $0.7\,\mu g/m^3$ (mean = 0.3, SD = 0.1) and $PM_{2.5}$ was slightly lower than the global model with concentrations ranging from 2.9 to $11\,\mu g/m^3$ (mean = 5.9, SD = 1.5). For $PM_{2.5}$, we compared health burden estimates using global satellite-derived estimates to North American satellite-derived estimates, whereas for BC, our main analysis compared the satellite-derived model to Street View mobile monitoring concentrations. Given that satellite-derived $PM_{2.5}$ concentrations are highly uncertain (Diao et al. 2019), we also estimated results using a more statistically based $PM_{2.5}$ model for the United States (Di et al. 2016, 2017).

Baseline Disease Rates and Demographics

Maps of baseline disease rates for all health end points and spatial resolutions are provided in Figures S29–S35. We obtained all-cause and CVD mortality rates at both the CBG and county levels (Table S16). For the CBG level, we obtained counts and rates for all-cause and CVD mortality [categorized according to the *International Statistical Classification of Diseases, 10th Revision* (ICD-10; WHO 2016) ICD-10 codes I10–I75] from the Alameda County Public Health Department for adults and the elderly. CBG rates were based on 7-y averages of death counts (2011–2017) over average population counts for 2012, 2014, and 2016 (Eayres and Williams 2004) and were age-adjusted using the standard 2000 U.S. Census population (Pickle and White 1995). In addition, CBGs with counts <10 were suppressed to protect confidentiality (Brillinger 1986). Combined, these methods avoid interannual variability for small-area (CBG-level) baseline disease rates and resulted in a conservative mean relative standard error of 15 (range = 7–58, SD = 5) for 1,046 CBGs. For all-cause mortality ages $\geq$25 y, there were 6 (0.5%) missing block groups, and for all-cause mortality ages $\geq$65 y, there were 9 (0.86%) missing block groups. For CVD mortality, there were 37 (3.53%) missing block groups for ages $\geq$25 y and 71 (6.79%) missing block groups for ages $\geq$65 y. To impute missing CBG baseline disease rates, we used an average of the five nearest neighbor rates. We obtained age-adjusted county-level mortality data for 2016 for both all-cause and CVD mortality most closely matching our CBG disease categories (ICD-10 codes I00–I78) from CDC Wonder (CDC 2018). CBG baseline mortality rates show more heterogeneity in the spatial distribution of disease. Annual all-cause mortality for adults ranged from 29 to 331 per 10,000, compared with 21 to 38 per 10,000 using the county rates.

We were unable to obtain baseline disease rates at the CBG level for nonmortality end points. For CVD hospitalizations rates, we used county-level rates from the BenMap-CE 1.4.14 (BenMap) software produced by the U.S. EPA for conducting health impact assessment (Sacks et al. 2018). Rates were available in BenMap for the elderly in 5-y age groups: ages 65–69, 70–74, 75–79, 80–84, and 85–99 y. The BenMap program uses 2010 U.S. Census data as the denominator when pooling age groups into a single rate. We applied the 5-y age group rates to the 10-y age groups (65–74, 75–84, and $\geq$85 y) available from the 2010 U.S. Census and used the U.S. Census data from BenMap as the denominator. We weighted the rates by age group count and created an aggregated rate per county ($n = 9$) for CVD hospitalizations. CVD hospitalization (ICD-9 codes 390–429) rates in 2014 ranged from 296 to 604 per 10,000 for ages 65–99 y, across counties in the Bay Area. For asthma ER visits (ICD-9 code 493/ICD-10 code J45), we used county-level rates and ZIP-code–level rates from the California Department of Public Health for 2016 (CDPH 2017, 2019), and we used county rates to impute data for missing ZIP-code rates (17% of the pediatric population and 10% of the adult population). Across the ZIP codes in the Bay Area, 2016 baseline rates of asthma ER visits among children ranged from 1 to 154 per 10,000, and for adults, from 1 to 175 per 10,000. For pediatric asthma incidence, we applied a California statewide baseline rate for 2008 of 107 per 10,000 persons ($n = 96{,}550$) (Milet et al. 2013) because more recent and finer resolution data were not available. Preprocessing of baseline disease rates was conducted in ArcMap (version 10.4; Esri).

We used nighttime (i.e., estimates of permanent residents) population counts from the LandScan USA data set at 100 m $\times$ 100 m resolution for 2017 given that it most closely aligned with the temporal availability of our pollutant and baseline disease rate data sets (Oak Ridge National Laboratories 2020; Bhaduri et al. 2007). Compared with the daytime population, we considered the nighttime population to be more consistent with the common approach of epidemiological studies to assign exposure based on home address. LandScan USA employed a multidimensional dasymetric modeling technique, spatially redistributing the U.S. Census data to inhabited land-use areas. Because LandScan USA does not include age breakdowns, we calculate the fraction of the total population in different age groups using age-specific counts from the Gridded Population of the World (version 4) for 2010, available at 1-km resolution from the Socioeconomic Data and Applications Center at the Center for International Earth Science Information Network at Columbia University (CIESEN 2019). To ascertain whether using fine-resolution baseline disease rates identifies disparities in pollutant-attributable disease between population subgroups, we estimated the percentage of pollutant-attributable cases in Alameda County in CBGs with $>$50% minority (Black, Asian, Hispanic, Pacific Islander, and American Indian) population, using U.S. Census population counts at the CBG level for 2010, the most recent year available.

Because our intention was to inform best practices for cities around the world to conduct within-city health impact assessments, we compared the spatial distributions of NO_2- and BC-attributable pollutant-attributable disease burdens estimated using Street View vs. globally available modeled concentrations from LUR- (Larkin et al. 2017) and satellite-based models (van Donkelaar et al. 2016). To do this comparison, we used the Getis-Ord local statistic in ArcPro (version 2.7), which provides a Z-score with accompanying p-value indicating whether each area has estimated pollutant-attributable cases that are higher or lower than surrounding grid cells (Ord and Getis 1995). We also compared the influence of CBG vs. county-level disease rates on the spatial patterns of estimated air pollution-related all-cause mortality in Alameda County given that baseline disease rates are not typically available at the CBG scale.

We also conducted several policy-relevant sensitivity analyses to assess the pollutant-attributable health impacts that could be avoided if air pollutant concentrations were reduced to lower levels. We specifically assessed two hypothetical scenarios in which concentrations of each pollutant were reduced to the minimum and median grid-cell–level concentrations of each data set. These scenarios are conceptually similar to pollution reduction targets for West Oakland established in the West Oakland Community Action Plan (BAAQMD and West Oakland Environmental Indicators Project 2019) as part of efforts by the State of California to identify and reduce air pollution among disproportionately exposed California communities, as required by 2017 Assembly Bill 617 (Cal AB 617 2017).

Results

Overall Pollutant-Attributable Disease Burdens in the Bay Area

We first estimated the total burden of NO_2, BC, and $PM_{2.5}$ across the Bay Area using the spatially complete concentration estimates from the global LUR and satellite-based models and county-level disease rates (Table 2–4). We estimated that 2,520 [95% confidence interval (CI): 740, 4,190], 150 (95% CI: 80, 190), and 3,080 (95% CI: 2,100, 4,020) deaths could be attributable to NO_2, BC, and $PM_{2.5}$ annually in the Bay Area, respectively. We also estimated asthma morbidity attributable to NO_2 and $PM_{2.5}$ across the Bay Area. Using a state-level asthma incidence rate and ZIP-code–level asthma ER visits, we estimated 5,210 (95% CI: 2,340, 6,780) and 5,590 (95% CI: 2,120, 8,250) new pediatric asthma cases, and 620–730 (95% CI: 20, 1,400) and 720 (95% CI: 430, 990) asthma ER visits attributable to NO_2 and $PM_{2.5}$ annually in the Bay Area, respectively. For NO_2 and $PM_{2.5}$, for which we had both U.S. and global concentration models, we

Table 2. Health impact model inputs and estimated pollutant-attributable cases of various health outcomes for nitrogen dioxide (NO_2) for the Bay Area.

				Total burden using concentrations from Larkin et al. (2017)		Burden remaining after reduction to median concentrations (11.6 ppb)		Total burden reduced using median concentrations (%)	Burden remaining after reduction to minimum concentrations (1 ppb)		Total burden reduce using minimum concentrations (%)
NO_2	Concentration–response function	Age groups	Baseline disease rate resolution	Attributable cases per 100,000	Attributable cases	Attributable cases per 100,000	Attributable cases		Attributable cases per 100,000	Attributable cases	
All-cause mortality	Atkinson and Butland 2018	Adults	County	50 (10, 80)	2,520 (740, 4,190)	30 (10, 50)	1,380 (400, 2,330)	45	3 (1, 10)	180 (50, 300)	93
	Eum et al. 2019	Elderly	County	110 (100, 120)	1,020 (930, 1,150)	60 (60, 70)	570 (520, 640)	44	10 (10, 10)	70 (70, 80)	93
CVD mortality	Atkinson et al. 2018	Adults	County	20 (10, 30)	1,100 (740, 1,760)	12 (8, 19)	610 (410, 980)	45	2 (1, 2)	80 (50, 130)	93
	Eum et al. 2019	Elderly	County	150 (140, 160)	1,410 (1,360, 1,470)	90 (80, 90)	800 (770, 840)	43	10 (10, 10)	110 (100, 110)	93
Asthma incidence	Khreis et al. 2017	Pediatric	State of California	300 (140, 390)	5,210 (2,340, 6,780)	180 (80, 240)	3,070 (1,310, 4,110)	41	20 (10, 30)	420 (170, 580)	92
Asthma ER visits	Orellano et al. 2017	All ages	ZIP code	30 (10, 60)	2,420 (520, 4,230)	20 (0, 30)	1,270 (270, 2,250)	45	2 (0, 5)	160 (30, 290)	93
		Pediatric	ZIP code	40 (0, 80)	730 (20, 1,400)	20 (0, 50)	400 (10, 780)	47	3 (0, 10)	50 (0, 100)	93
	Zheng et al. 2015	All ages	ZIP code	30 (20, 40)	2,480 (1,540, 3,400)	20 (10, 20)	1,300 (810, 1,800)	45	2 (1, 3)	170 (100, 230)	93
		Pediatric	ZIP code	40 (30, 50)	620 (450, 790)	20 (10, 30)	340 (250, 440)	47	3 (2, 3)	40 (30, 60)	93

Note: Values in parentheses indicate 95% confidence intervals based on the error in the relative risk estimates only. Adults, 25–99 years of age; CVD, cardiovascular disease; elderly, 65–99 years of age; ER, emergency room; pediatric, 0–17 years of age.

Table 3. Health impact model inputs and estimated pollutant-attributable cases of various health outcomes for the Bay Area.

				Total burden using concentrations from van Donkelaar et al. (2019)		Median concentrations ($0.3\ \mu g/m^3$)		Total burden reduced using median concentrations (%)	Minimum concentrations ($0.1\ \mu g/m^3$)		Total burden reduce using minimum concentrations (%)
BC	Concentration–response function	Age groups	Baseline disease rate resolution	Attributable cases per 100,000	Attributable cases	Attributable cases per 100,000	Attributable cases		Attributable cases per 100,000	Attributable cases	
All-cause mortality	Janssen et al. 2011	Adults	County	3 (2, 4)	150 (80, 190)	2 (1, 2)	90 (50, 120)	37	1 (0, 1)	30 (20, 40)	78
CVD mortality	Janssen et al. 2011	Adults	County	1 (0, 1)	50 (30, 60)	1 (0, 1)	30 (20, 40)	38	0 (0, 0)	10 (5, 10)	78
CVD hospitalizations	Peng et al. 2009	Elderly	County	40 (10, 60)	340 (130, 530)	20 (10, 40)	210 (80, 340)	36	10 (0, 10)	70 (30, 120)	78

Note: Values in parentheses indicate 95% confidence intervals based on the error in the relative risk estimates only. Adults, 25–99 years of age; BC, black carbon; CVD, cardiovascular disease; elderly, 65–99 years of age.

Table 4. Health impact model inputs and estimated pollutant-attributable cases of various health outcomes for fine particulate matter ($PM_{2.5}$) for the Bay Area.

$PM_{2.5}$	Concentration–response function	Age groups	Baseline disease rate resolution	Total burden using concentrations from van Donkelaar et al. 2016		Median concentrations ($10\,\mu g/m^3$)		Total burden reduced using median concentrations (%)	Minimum concentrations ($3\,\mu g/m^3$)		Total burden reduce using minimum concentrations (%)
				Attributable cases per 100,000	Attributable cases	Attributable cases per 100,000	Attributable cases		Attributable cases per 100,000	Attributable cases	
All-cause mortality	Krewski et al. 2009	Adults	County	60 (40, 80)	3,080 (2,100, 4,020)	50 (30, 60)	2,500 (1,700, 3,280)	19	20 (10, 20)	790 (530, 1,030)	74
	Di et al. 2017	Elderly	County	310 (300, 310)	2,920 (2,820, 2,980)	250 (240, 260)	2,400 (2,320, 2,450)	18	80 (80, 80)	760 (730, 780)	74
CVD mortality	Turner et al. 2016	Adults	County	40 (30, 40)	1,790 (1,390, 2,170)	30 (20, 40)	1,470 (1,130, 1,790)	18	10 (10, 10)	470 (360, 580)	74
	Thurston et al. 2016	Elderly	County	120 (60, 170)	1,160 (610, 1,650)	100 (50, 140)	950 (500, 1,360)	18	30 (20, 50)	300 (160, 440)	74
CVD hospitalizations	Bravo et al. 2017	Elderly	County	40 (30, 50)	360 (280, 430)	30 (20, 40)	290 (220, 350)	19	10 (10, 10)	90 (70, 110)	75
Asthma incidence	Khreis et al. 2017	Pediatric	State of California	320 (120, 480)	5,590 (2,120, 8,250)	270 (100, 410)	4,640 (1,710, 7,000)	17	90 (30, 150)	1,560 (540, 2,510)	72
Asthma ER visits	Lim et al. 2016	Pediatric	ZIP code	40 (30, 60)	720 (430, 990)	30 (20, 50)	590 (350, 800)	19	10 (10, 20)	180 (110, 250)	75

Note: Values in parentheses indicate 95% confidence intervals based on the error in the relative risk estimates only. Adults, 25–99 years of age; CVD, cardiovascular disease; elderly, 65–99 years of age; ER, emergency room; pediatric, 0–17 years of age; $PM_{2.5}$, particulate matter $\leq 2.5\,\mu m$ in aerodynamic diameter.

estimated 30% and 37% larger annual attributable mortality burdens using the global vs. U.S. concentration data sets (NO_2 global = 2,520; NO_2 U.S. = 1,930; $PM_{2.5}$ global = 3,080; $PM_{2.5}$ U.S. = 1,960 = Excel Tables S1 and S2), consistent with the magnitude difference in concentration estimates.

Estimated 100-m grid-cell–level pollution-attributable mortality rates (annual attributable deaths per 100,000 people) ranged across the Bay Area by a factor 38, 4, and 5 for NO_2, BC, and $PM_{2.5}$ (NO_2 range = 3–113, mean = 30, SD = 13; BC range = 1–4, mean = 2, SD = 1; $PM_{2.5}$ range = 20–95, mean = 49, SD = 13; Excel Tables S1–S3). For NO_2, the largest mortality impact among counties occurred in Alameda County, where it was 14 times larger than in the least impacted county, Napa (Bay Area county range = 40–570 attributable deaths annually, mean = 280, SD = 210; city range = 0–390, mean = 11, SD = 38; Excel Tables S4 and S5). Inter-county and -city variation in BC-attributable mortality was also high, with Santa Clara County having a burden 13 times that of Napa County (county range = 3–40 attributable deaths annually, mean = 16, SD = 12; city range = 0–20, mean = 1, SD = 2; Excel Tables S6 and S7). For $PM_{2.5}$, the most impacted county (Santa Clara) had a $PM_{2.5}$ mortality burden that was 12 times larger than the least impacted county (Napa) (county range = 60–690 attributable deaths annually, mean = 340, SD = 230; city range = 0–395, mean = 14, SD = 40; Excel Tables S8 and S9).

Influence of Varying Pollutant Concentrations Data Sets and Baseline Disease Rates

We next focused in on West, Downtown, and East Oakland ($\sim 30\,km^2$), the part of the Bay Area where Street View measurements of NO_2 and BC are available and can be compared with the application of concentrations from predictive models. Total estimated NO_2-attributable deaths in Oakland approximately doubled when using the LUR [77 annual attributable deaths (95% CI: 23, 127)] compared with the Street View concentrations [39 (95% CI: 12, 66)] (Figure 2). NO_2-attributable mortality rates ranged across 100-m grid cells by a factor of 11 and 26 using LUR and Street View estimates, respectively (LUR range = 32–342 annual attributable deaths per 100,000, mean = 101, SD = 31; Street View range = 15–396, mean = 64, SD = 39).

BC-attributable mortality burdens using concentrations from the predictive model (van Donkelaar et al. 2019) exceeded the estimates based on Street View concentrations in Oakland [Street View = 2 annual attributable deaths (95% CI: 1, 3); satellite-derived: 4 (95% CI: 2, 5)]. For BC and $PM_{2.5}$, pollution-attributable mortality rates ranged across 100-m grid cells by a factor of 39, 12, and 8 when using BC Street View, U.S. BC satellite-derived, and global $PM_{2.5}$ satellite-derived concentrations, respectively (range = 0–39 annual attributable deaths per 100,000, mean = 4, SD = 4; range = 1–12, mean = 5, SD = 1; range = 22–183, mean = 96, SD = 29). In our sensitivity analysis using a proportional approach that assumed that the BC contribution to $PM_{2.5}$ mortality is the same as its contribution to $PM_{2.5}$ concentrations (1.25–7.5% using the satellite-derived concentration estimates), the range of the BC-attributable fraction of mortality across 100-m grid cells was very similar to our core results (0.07–0.5%, applying the log-linear CRF to BC, and 0.06–0.4%, using the proportional approach).

Despite moderate-to-low correlation between concentrations from the Street View monitoring and predictive models, we found similar spatial clusters of NO_2 and BC-attributable fractions using both concentration data sets. For the grid cells in the Oakland area for which we had both Street View and LUR (NO_2) and U.S. satellite-derived (BC) results, a large fraction of grid cells (NO_2: 45%, $n = 1,619$; BC: 37%, $n = 1,334$) were fully concordant using

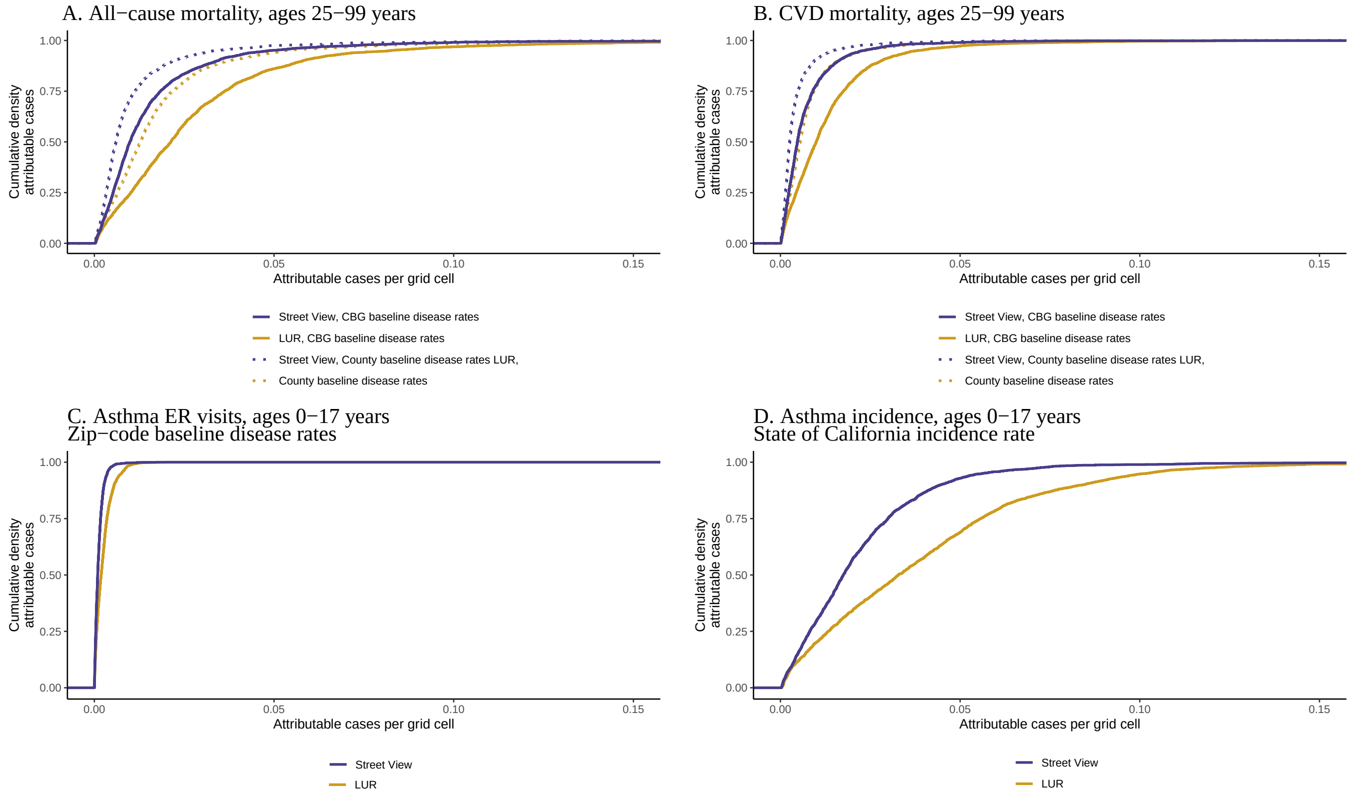

Figure 2. Cumulative density of annual nitrogen dioxide (NO_2)-attributable cases per 100 m × 100 m grid cell in Oakland, California, using different concentration and baseline disease rate data sets. (A) All-cause mortality, ages 25–99 y; (B) cardiovascular disease mortality, ages 25–99 y; (C) asthma ER visits, ages 0–17 y, ZIP-code baseline disease rates; and (D) asthma incidence, ages 0–17 y, State of California incidence rate. Note: ER, emergency room.

the *Z*-score statistic, meaning that both concentration data sets identified clusters of attributable cases that are both significant ($p < 0.05$) and in the same direction (either a higher or lower value cluster) (Figures S36 and S37). Another 37% ($n = 1{,}309$) and 22% ($n = 799$) of grid cells for NO_2 and BC had directional concordance, but concentration data sets identified differing significance in the clusters. We also found that 13% ($n = 452$) and 15% ($n = 546$) of grid cells for NO_2 and BC, respectively, were directionally discordant between the two concentration data sets but had the same significance level. About 5% ($n = 193$) and 25% ($n = 899$) of grid cells for NO_2 and BC were completely discordant. Although both data sets identified similar hotspots, the Street View data set identified a wider range of *Z*-scores (NO_2 range $= -3.98$ to 10.02, mean $= -0.06$, SD $= 2.42$; BC range $= -3.80$ to 12.77, mean $= -0.09$, SD $= 2.20$) as compared with LUR and satellite-derived concentrations (NO_2 range $= -4.07$ to 7.51, mean $= 0.19$, SD $= 2.17$; BC range $= -8.46$ to 2.53, mean $= -0.04$, SD $= 2.06$).

We next assessed the influence of CBG vs. county-level baseline disease rates on estimated pollutant-attributable mortality within Alameda County, where we had baseline all-cause mortality rates for both spatial resolutions. Using the same modeled concentrations of NO_2 (LUR) and $PM_{2.5}$ (satellite-derived) in both cases, CBG baseline disease rates yielded 15% and 13% higher spatially aggregated estimates of pollutant-attributable mortality rates compared with the application of county baseline disease rates (NO_2 CBG = 60 annual attributable deaths per 100,000; NO_2 county = 52; $PM_{2.5}$ CBG = 70; $PM_{2.5}$ county = 61). Differences were even more evident at finer spatial aggregations. For example, for just the Oakland area extent, CBG baseline disease rates yielded 52%, 67%, and 57% higher NO_2, BC, and $PM_{2.5}$-attributable all-cause mortality rates compared with the application of county baseline disease rates (NO_2 CBG = 97 annual attributable deaths per 100,000; NO_2 county = 64; BC CBG = 5; BC county = 3; $PM_{2.5}$ CBG = 88; $PM_{2.5}$ county = 56). Applying CBG baseline disease rates also revealed spatial heterogeneity in estimated pollutant-attributable mortality rates that was masked when using county-level disease rates. Applying CBG baseline disease rates yielded grid-cell–level pollutant-attributable rates that varied by factors of 29, 12, and 14 for NO_2, BC, and $PM_{2.5}$ (NO_2 CBG range = 15–434 annual attributable deaths per 100,000, mean = 41, SD = 19; BC CBG range = 1–12, mean = 2, SD = 1; $PM_{2.5}$ CBG range = 19–267, mean = 59, SD = 17; Excel Tables S9–S12) across Alameda County, whereas applying county baseline disease rates yielded less spatial heterogeneity (6, 4, and 3 times, respectively; NO_2 county range = 19–113 annual attributable deaths per 100,000, mean = 38, SD = 11; BC county range = 1–4, mean = 2, SD = 1; $PM_{2.5}$ county: range = 32–81, mean = 55, SD = 8).

We next estimated the percentage of pollutant-attributable mortality and morbidity cases in CBGs with >50% minority population. We found that using CBG instead of county baseline disease rates resulted in a larger percentage of pollutant-attributable cases for CBGs with >50% minority population in Alameda County (Table S17 and Figures S38–S40). For example, using CBG disease rates, we estimated that 75% of NO_2-attributable mortality occurred in majority minority CBGs, whereas that percentage was 72% when using county-level disease rates. The differences between applications of CBG vs. county-level disease rates were small but generally consistent across pollutants and health end points. Within Oakland, CBGs with the highest percentage of minorities and highest estimated NO_2-attributable mortality rates were located in West Oakland near I-880, a high-traffic–volume truck route, and in Chinatown, in the southeastern part of Downtown Oakland (Figures S41 and S42).

Policy-Relevant Reductions

Using concentrations from the global (NO_2 and $PM_{2.5}$) and U.S. (BC) model data sets for the Bay Area, we found that rolling back concentrations to the median ($NO_2 = 8$ ppb; $BC = 0.3\ \mu g/m^3$; $PM_{2.5} = 10\ \mu g/m^3$, respectively) reduced attributable deaths by 45%, 37%, and 19% [remaining deaths for $NO_2 = 1{,}380$ (95% CI: 400, 2,330); $BC = 90$ (95% CI: 50, 120); $PM_{2.5} = 2{,}500$ (95% CI: 1,700, 3,280); Tables 2–4]. Varying magnitudes of reductions in attributable deaths were due to the distribution of concentrations, with $PM_{2.5}$ concentrations comparatively right skewed. Rolling back to minimum concentrations reduced cases by 93%, 78%, and 74% for NO_2, BC, and $PM_{2.5}$, respectively [177 (95% CI: 50, 300), 32 (95% CI: 20, 40), and 790 (95% CI: 530, 1,030) remaining for 1-ppb, 0.1-$\mu g/m^3$, and 3-$\mu g/m^3$ concentrations)]. Health benefits of the reduction scenarios were more pronounced within Oakland, where pollutant-attributable mortality was reduced by 59%, 40%, and 52% for NO_2, BC, and $PM_{2.5}$, respectively, under a reduction to the median concentration [32 (95% CI: 9, 53), 2 (95% CI: 1, 3), and 56 (95% CI: 38, 74) cases remaining] and by 94%, 80%, and 95% under the reduction to the minimum concentration [4 (95% CI: 1, 7), 1 (95% CI: 0, 1), and 18 (95% CI: 12, 23) cases remaining]. Health benefits were smaller when using Street View concentrations for NO_2, with a 13% and 66% decrease in NO_2-attributable mortality under reductions to the median and minimum concentrations, respectively [34 (95% CI: 20, 57) and 13 (95% CI: 4, 23) cases for 11.59 and 3.37 ppb, respectively]. Estimates for reductions using Street View BC concentrations were not possible owing to few BC-attributable cases in Oakland (<1). The top five CBGs with the highest premature mortality burden within Oakland would have experienced an estimated 27% and 79% reduction in NO_2-attributable mortality if concentrations were reduced to median and minimum concentrations, respectively. These same five CBGs had a greater than 50% population of racial and ethnic minorities, indicating that the policy changes would disproportionately benefit minority populations.

Discussion

We estimated the spatial distribution of NO_2, BC, and $PM_{2.5}$-attributable health impacts at the neighborhood-scale within the Bay Area, California, where high spatial resolution concentrations from mobile monitoring, as well as CBG-level disease rate data sets are available. We found 38-, 4-, and 5-fold variation in mortality attributable to NO_2, BC, and $PM_{2.5}$ across grid cells in the Bay Area, indicating that pollution-attributable risks can vary considerably within individual cities. This variation was observable regardless of whether predictive models or mobile monitoring concentration data sets were used, although the mobile monitoring concentrations revealed more spatial heterogeneity. Spatial heterogeneity in air pollution-attributable health risks was more pronounced when we applied CBG rather than county-level baseline disease rates.

Depending on the concentration and baseline disease data sets used, estimated NO_2-attributable mortality in Oakland at the 100-m grid-cell level varied by a factor of 2–26, BC-attributable mortality (annual deaths per 100,000) varied by a factor of 2–39, and $PM_{2.5}$-attributable mortality varied by a factor of 2–8. We found the least heterogeneity using county baseline disease rates and concentration estimates from a global model, and the greatest variation using Street View concentrations with CBG baseline disease rates. Using concentrations from Street View mobile monitoring and predictive models yielded similar spatial patterns in air pollution-attributable health risks because baseline disease rates also play an important role. For the same reason, CBGs with the highest air pollution-attributable health risks were not necessarily those with the highest pollutant concentrations.

Comparing the influence of baseline disease rates and concentrations on spatial distribution, we found that neighborhoods with the highest air pollution-attributable health risks were not necessarily those with the highest pollutant concentrations. Each additional input to the health impact function changed the spatial distribution of the estimated health burden. For example, calculating the percent of mortality that can be attributed to air pollution incorporates only the CRF and concentrations (Figure 3A). When baseline disease rates were also incorporated to estimate attributable mortality rates (Figure 3B), spatial patterns in risk shifted to different areas within West and Downtown Oakland. Finally, when population was included to estimate pollutant-attributable disease burdens (Figure 3C), spatial patterns shifted yet again. Therefore, considering only concentrations without incorporating baseline disease rates and population distribution may not adequately capture the neighborhoods with greatest pollutant-attributable health risks and burdens. The relative importance of disease rates and concentrations on spatial heterogeneity in risk estimates depended on the data set and its spatial resolution, as well as the risk metric used (attributable fraction, attributable rate, or attributable cases).

Our aggregated estimate of 3,080 (95% CI: 2,100, 4,020) $PM_{2.5}$-attributable annual deaths in the Bay Area was approximately double a previously published estimate of 1,500 deaths attributable to $PM_{2.5}$ in San Francisco (Anenberg et al. 2019) that used satellite-derived $PM_{2.5}$ concentrations from the Global Burden of Disease Study (Shaddick et al. 2018). Our estimate was also higher than the estimate from the BAAQMD (2017) Clean Air Plan of $\sim 2{,}500$ annual deaths attributable to anthropogenic $PM_{2.5}$ emissions, which used county-level baseline disease rates and population estimates, a Community Multiscale Air Quality Modeling model estimate of $PM_{2.5}$ and a mean of 12 different CRFs (BAAQMD 2017). Analysis of various $PM_{2.5}$ concentration estimates have indicated substantial differences in spatial patterns, complicating comparability between risk assessments (Diao et al. 2019). We also used different disease rates, concentration–response functions, and low-concentration thresholds.

Mobile monitoring offers a spatially explicit observational record but has incomplete spatial coverage. Predictive models using land-use variables and satellite remote sensing have the advantage of complete spatial coverage, but estimated concentrations are uncertain. In areas where there were overlapping Street View and predictive model data for the same pollutant (NO_2), we found higher NO_2-attributable deaths when using the LUR [77 annual deaths (95% CI: 23, 137)] compared with the Street View concentrations [39 (95% CI: 12, 66)]. Compared with concentrations, NO_2-attributable mortality rates using Street View and LUR were more correlated owing to the smoothing effect of applying the same baseline disease rates ($R = 0.67$). These results indicated that the Street View data set detects extremes in concentrations and associated health burdens that are not identified by the LUR concentration data set. Comparing these results was challenged by inherent differences in the data sets: First, NO_2 concentrations decreased between 2011, for which we had LUR concentration estimates, and 2015, when the Street View mobile monitoring occurred (Duncan et al. 2016). Second, the Street View data set captured high near-roadway exposures, whereas the LUR model represented broader spatial average concentrations with smaller decay gradients of concentrations as you move away from main thoroughfares and highways, resulting in higher concentrations in residential areas. In addition, Street View measurements were taken during the daytime, which may underestimate daily NO_2 concentrations by 15–20% given that daytime

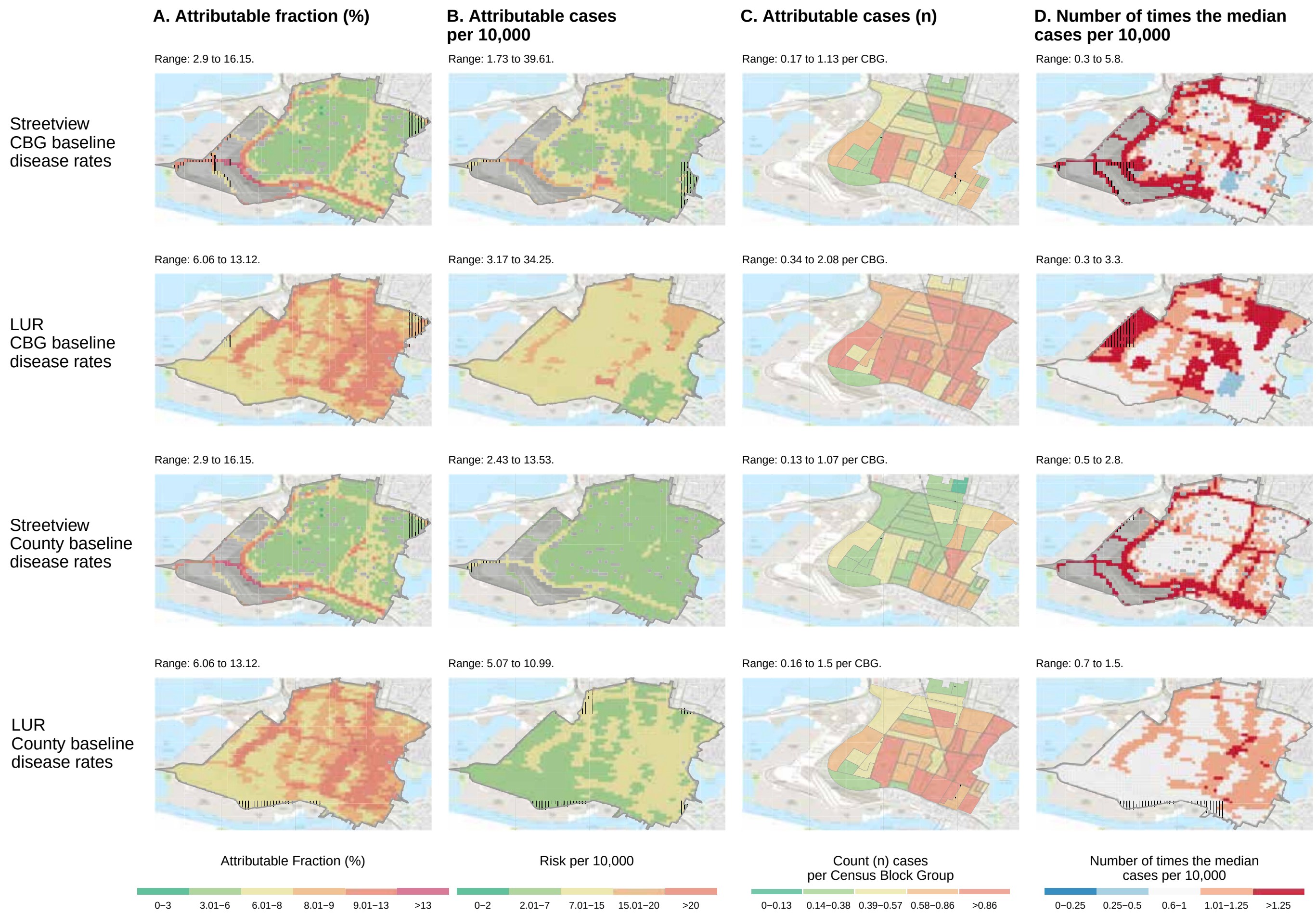

Figure 3. Spatial distribution of nitrogen dioxide (NO_2)-attributable all-cause mortality among adults in the West and Downtown Oakland area. (A) Attributable fraction; (B) attributable cases per 10,000; (C) attributable cases; and (D) number of times the median cases per 10,000. (Extent: −122.253, 477; −122.327, 816; 37.791, 334; 37.832, 312) using the Street View mobile monitoring (rows 1 and 2) and data from Larkin et al. 2017 land-use regression (LUR) (rows 3 and 4) concentration data sets, as well as census block group (CBG) (rows 1 and 3) and county (rows 2 and 4) baseline disease rates. Results depicted are for the central estimates of concentrations and relative risk estimates. Gray areas represent areas where concentration data were not available. Base map and data from OpenStreetMap and OpenStreetMap Foundation.

ambient NO_2 is depressed by photolysis. However, this effect may have been balanced by the Street View data set, which did not account for lower weekend concentrations. Given these differences, NO_2 concentrations from these two data sets were not well correlated ($R = 0.55$), and the LUR concentrations were overall higher with less spatial variability.

Our study was limited in several ways. Although concentration and population data sets are increasingly available at high resolutions, baseline disease rates are still difficult to obtain at urban and intra-urban scales. For example, for asthma incidence, we were only able to apply a statewide incidence rate, although prevalence data for asthma shows spatial heterogeneity of asthma within the Bay Area and California. Expanding disease surveillance and increasing access to highly resolved baseline disease rates in cities around the world would improve health impact assessment estimates and capacity to detect areas within cities that have elevated pollution-attributable health risks. Our analysis of racial disparities in air pollution health risks was also limited because we did not incorporate racial- and ethnicity-specific baseline disease rates or RR estimates. In addition, we applied CRFs from meta-analyses and large, nationwide cohort studies that had high quality and statistical power although their populations may not have matched the population distribution in our analysis, introducing additional unquantifiable uncertainty to our analysis.

Our pollutant-specific estimates cannot be summed together because we applied single-pollutant epidemiological models and, as such, there could be a significant amount of overlap between the deaths estimated to be attributable to each individual pollutant. Some of the relationship between NO_2 and adverse health outcomes may have been accounted for by concurrent $PM_{2.5}$ exposures, resulting in overlap of attributable deaths in our results presented for NO_2 and $PM_{2.5}$, although $PM_{2.5}$ alone does not fully capture the effects of near road traffic pollutants more strongly correlated with NO_2 (Atkinson and Butland 2018). Similarly, estimates presented here for $PM_{2.5}$ are inclusive of BC and other $PM_{2.5}$ species. Therefore, results for BC should be interpreted as a subset of $PM_{2.5}$-attributable health outcomes. We calculated results for both BC and $PM_{2.5}$ because although $PM_{2.5}$ can have multiple sources, BC is a combustion-related particle that represents the impacts of $PM_{2.5}$ traffic-related pollution as opposed to pollution form other regional sources (Janssen et al. 2011). The low magnitude of results precluded us from drawing strong conclusions from this comparison. The high spatial heterogeneity of the Street View BC concentrations resulted in poor correlation between the two data sets ($R = 0.03$), although the smoothing effect of applying the same CBG baseline disease rates to each data set resulted in increased correlation between BC-attributable mortality rates ($R = 0.36$).

The effective use of a two-pollutant CRF in health impact assessment relies upon RR estimates from studies able to meaningfully parse relationships between correlated pollutants (Dominici et al. 2010; Stafoggia et al. 2017), which were not available for most of our pollutant–health outcome pairs. For one sensitivity analysis wherein a two-pollutant model CRF for NO_2 was available, we applied a $PM_{2.5}$-adjusted RR estimate for NO_2 and CVD mortality among the elderly from the study by Eum et al. (2019), which resulted in lower total attributable case estimates (254 attributable all-cause deaths within Alameda County, using the unadjusted CRF, and 121 deaths, using the adjusted CRF), indicating a considerable portion of the burden estimated for NO_2 may be attributable to $PM_{2.5}$.

As demonstrated, pollutant concentrations varied substantially within cities, whereas air pollution cohort studies in the United States have often compared exposure between cities. No long-term North American cohort studies have analyzed within-city variation in RR estimates for $PM_{2.5}$ and all-cause mortality (Vodonos et al. 2018). The meta-analysis we employed for the relationship between NO_2 and all-cause mortality relied upon a mix of studies examining within- (i.e., the ESCAPE cohorts) and between-city (i.e., the Harvard Six Cities cohort) exposure comparisons. As a sensitivity analysis, we estimated results using a within-city RR estimate (Table 1) from a subcohort of the Canadian Census Health and Environment Cohort, which found significant relationships between NO_2 and all-cause for within-city exposure comparisons but not for between-city exposure comparisons (Crouse et al. 2015). This within-city RR estimate was also included in the meta-analysis for our main CRF (Atkinson and Butland 2018). Although the choice of CRF changed the overall magnitude of aggregated air pollution-attributable health impacts for each pollutant, it did not affect our main conclusions about the intra-urban heterogeneity because we applied the CRF uniformly across the domain. Future use of statistical methods able to assess correlated exposures (Stafoggia et al. 2017) will allow for improved application of two-pollutant model estimates health impact assessment and policy-making.

Interpreting and communicating the uncertainties in a health impact assessment is a known challenge (Nethery and Dominici 2019) because, with each input parameter to the health impact function, there is associated uncertainty. We estimated uncertainty using the CIs of the RR estimates, but we were unable to quantify uncertainty in the pollutant concentrations, baseline disease rates, and population estimates. Gridded population estimates are also increasingly available at a fine spatial resolution. Prior to selecting a population data set, we examined use of WorldPop (Tatem 2017) estimates for 2016, which are also available on a $100\ m \times 100\ m$ resolution. Although provided at a high resolution, we found the WorldPop estimates lacked the spatial heterogeneity available in other population data sets. Although population estimates are still a source of uncertainty in our assessment, we believe using a data set specific to the United States that incorporates both census and satellite data reduced part of this uncertainty. Among our pollutant concentration data sets, the Street View data set included only measurements that were taken during the daytime and on weekdays and may not, therefore, have fully captured long-term annual averages. The LUR data set incorporates *in situ* data, although concentrations are ultimately estimations of the sum of all oxidized atmospheric odd-nitrogen species (NO_y) estimations and not observations of actual NO_2 concentrations (Dickerson et al. 2019). Future work can make use of more spatially refined estimates of $PM_{2.5}$ (Di et al. 2019). In addition, NO_2 satellite-derived models have now been developed (Di et al. 2020) that can be compared with results from LUR models.

In addition, we assumed a causal relationship between NO_2 and all-cause mortality, although the putative agent(s) in the traffic-related pollution mixture are unknown, adding to uncertainty in our estimates. Epidemiological studies often use NO_2 as a marker of traffic-related air pollution because it is easily measured and for consistency in characterizing spatial patterns in traffic-related air pollution (Beckerman et al. 2008; Levy et al. 2014). However, none of the studies in the meta-analysis we used to derive RRs for NO_2 and all-cause and CVD mortality adjusted for traffic-related particles or other chemicals, including BC and $PM_{2.5}$, in the traffic pollution mixture (Atkinson and Butland 2018). It therefore remains unclear whether NO_2 itself is associated with mortality or whether NO_2 serves as a proxy for other elements of the traffic-related air pollution mixture. Following COMEAP recommendations, the NO_2 mortality impacts should be interpreted as a metric of the overall mortality burden due to mixture of near field traffic-related air pollution.

Another challenge with hyperlocal air pollution health impact assessment that requires further exploration was capturing pollution exposure accurately for population movement. We believe this limitation was mitigated for two reasons: *a*) air pollution disproportionately affects the very young and very old, who tend to stay closer to home throughout the day (Chambers et al. 2017; Spalt et al. 2016); and *b*) most air pollution epidemiological studies use residential address as the method of assigning exposure, thus accounting for population movement would be inconsistent with the epidemiological studies from which we drew concentration–response relationships. In addition, using only residential address in exposure assessment within epidemiological studies has been found to underestimate health effects of $PM_{2.5}$ by about 10% (Nyhan et al. 2019). Similarly, without information available on the time-varying activity patterns of our population, we were unable to account for time–activity data in our risk assessment; however, exposure misclassification likely contributed less than other variables (e.g., RRs) to uncertainty in our health impact results. Our results indicate population movement out of highly polluted areas may substantially reduce population pollutant-attributable health burden. However, this points to the need for more epidemiological analysis using exposure assessment techniques beyond central site monitors, as well as techniques that account for people's movements rather than assigning exposure at the residential address. This factor should be explored in greater detail to understand how population movement affects actual exposure levels and estimated health impact assessment results. A related limitation is that the high-resolution concentration data sets we used did not match the exposure assessment techniques used in the epidemiological studies from which we derived our CRF, which most frequently used stationary monitors and, increasingly, LUR and satellite-based models, which may be more coarsely resolved than the data sets we used here. Thus, the CRFs we applied may be inconsistent with the exposure estimates we used.

Some recommended best practices in conducting air pollution health impact assessments in cities globally can be derived from the insights from this work. First, we found that applying fine-scale mobile monitoring or satellite LUR-derived air pollution data in health impact assessment reveals large and unequal distributions of the air pollution burden in cities. This indicates that spatial distribution of air pollution impacts could be routinely assessed in city air quality health impact assessments. Second, the distribution of air pollution and its risks and burdens did not follow the same patterns owing to large underlying spatial health disparities (reflected in baseline disease rates) and population distribution. Although most of the research in this area has focused on producing increasingly fine resolution estimates of air pollutant concentrations, similar emphasis has not been given to estimating or measuring spatial patterns of disease. Ignoring health disparities results in underestimating air pollution impacts in areas already burdened by poor health and masks the disproportionate impact faced by disadvantaged communities within cities. This has important environmental justice implications, and local disease rates should be incorporated as a best practice into city air pollution health impact assessments.

Conclusions

We found that air pollution health risks vary considerably within cities and that information on the spatial distribution of pollutant concentrations alone is insufficient to identify areas of elevated risk and burden of disease attributable to air pollution. We anticipate that these findings will apply to other health impact assessments conducted on the local scale given that spatial heterogeneity in disease rates is not unique to the Bay Area. Using pollutant concentrations from predictive models and mobile monitoring measurements identify similar spatial patterns of disease because disparities in baseline disease rates drive a substantial portion of heterogeneity in air pollution-attributable health risks. For areas with limited resources or where intensive mobile monitoring is not feasible, LUR- and satellite-derived models may be sufficient for identifying intra-urban areas of elevated risk, but additional research is needed to determine whether these findings hold in other areas. In addition, LUR- and satellite-derived models typically do not account for the mixture of vehicle types and traffic volume and, therefore, may be improved with the information about roadway concentrations captured in mobile monitoring data sets. Future work may seek to integrate multiple sources of pollutant concentration information to leverage the advantages of each. It is also important to expand reporting of disease rates at subcity scales.

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Climate Change and Health Profile Report
Santa Clara County

CDPH
California Department of
PublicHealth

UCDAVIS
UNIVERSITY OF CALIFORNIA

February 2017

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Disclaimers

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MESSAGE FROM CALIFORNIA DEPARTMENT OF PUBLIC HEALTH DIRECTOR DR. KAREN SMITH

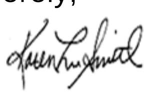
I am pleased to present Climate Change and Health Profile reports for each of the counties in California. The reports provide climate change projections for counties, and identify vulnerabilities and assets to support local adaptation planning for climate change. The Climate Change and Health Profile reports are the first in a series of CalBRACE materials being developed by the CDPH Office of Health Equity to foster mobilization to prevent and reduce injury and disease related to climate change.

As we continue building capacity to address climate change we are also aligning CDPH's work with the Governor's Executive Order B-30-15, which specifically addresses the need for climate adaptation and "actions [that] should protect the state's most vulnerable populations." The mission of the Office of Health Equity is to achieve the highest level of health and mental health for all people, with special attention focused on those who have experienced socioeconomic disadvantage and historical injustice. These reports focus resources on planning to protect those most vulnerable to the health impacts of climate change. We are also working with others in California to achieve emissions reduction targets, in order to slow further climate changes. These steps further our goal of becoming the healthiest state in the nation.

Each California county will experience the health impacts of climate change uniquely. The CDPH Office of Health Equity's CalBRACE project provides tools and resources to counties so that they can prioritize and adopt climate change adaptation and preparation strategies that fit their communities and geographies.

Climate change challenges our commitment to achieve equity in health and wellbeing in California, as it deepens the need to take actions that reduce vulnerabilities and increase resilience to climate change in our communities. Faced with this challenge, we approach climate change planning as an opportunity to improve living conditions and social determinants of health so that we can improve health, equity, and address climate change. These reports are one component of a comprehensive approach to creating healthy equitable communities and building resilience to climate change impacts. I hope that these reports provide you with information you can apply as you join me in this effort to protect our communities from the preventable health impacts of climate change.

Sincerely,



Karen Smith, MD, MPH
CDPH Director and State Public Health Officer

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We also thank the many reviewers from county health departments who provided valuable feedback about local conditions and the challenges and opportunities that exist for them to take action to address health and climate change. The local health departments are the vital link to promote public health prevention and wellness in communities, cities, and neighborhoods.

Our work was informed and enriched by the teams from the staff of the CDC Climate-Ready States and Cities Initiative’s program, Building Resilience Against Climate Effects (BRACE), grantee states and cities, collaboratives, and communities of practice.

Cover photo credits

California Fire Siege 2007, California Department of Forestry and Fire Protection (CAL FIRE); Jeff Poskanzer, California King Tides Project - Open Source; and California Division of Occupational Safety and Health (Cal/OSHA)

INTRODUCTION

Through legislation and Governor's Executive Orders, the State of California has mobilized to meet the challenges and opportunities posed by climate change. The overall strategy is embodied in reducing carbon emissions, promoting readiness for climate impacts, and conducting research to provide the best available science to guide our actions. In the course of this work, technical documents, strategies, and planning guidance have been produced by state agencies, including the California Department of Public Health (CDPH).

The *Climate Change and Health Profile Report* seeks to provide a county-level summary of information on current and projected risks from climate change and potential health impacts. This report represents a synthesis of information on climate change and health for California communities based on recently published reports of state agencies and other public data.

We have compiled and edited this wealth of information from technical documents and created a report accessible to public health professionals and their partners in state, regional, and local government, the private sector, and community-based organizations. We also highlight the public health dimensions of climate change along with its environmental impacts.

The content of this report was guided by a cooperative agreement between CDPH and the CDC Climate-Ready States and Cities Initiative's program *Building Resilience Against Climate Effects* (BRACE). The goals of BRACE are to assist state health departments to build capacity for climate and health adaptation planning. This includes

using the best available climate science to project likely climate impacts, identifying climate-related health risks and populations vulnerable to these impacts, assessing the added burden of disease and injury that climate change may cause, identifying appropriate interventions, planning more resilient communities, and evaluating to improve the planning effort. Communities with economic, environmental, and social disadvantages are likely to bear disproportionate health impacts of climate change.

This *Climate Change and Health Profile Report* is intended to inform, empower, and nurture collaboration that seeks to protect and enhance the health and well-being of all California residents.

This report is part of a suite of tools that is being developed by the California Department of Public Health to support local, regional, and statewide efforts of the public health sector to build healthy, equitable, resilient, and adaptive communities ready to meet the challenges of climate change.

Along with a county-level climate change and health vulnerability assessment and state guidance documents, such as *Preparing California for Extreme Heat: Guidance and Recommendations,* the profile provides a knowledge base for taking informed action to address climate change.

Background

What is climate change?


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Modern life has entailed the burning of coal, natural gas, petroleum, and other fossil fuels in our power plants, factories, businesses, farms, homes, and cars. Key byproducts of energy production and consumption are carbon dioxide, methane, and other pollutants. These gases are called greenhouse or heat trapping gases because as they mix in the atmosphere, they create a barrier that stops heat produced by the sun from escaping the Earth's surface.

The changing climate is evident from observations of increased global average air and ocean temperatures, widespread melting of snow and ice, and rising global average sea level rise.[1] The average carbon dioxide concentration in the atmosphere topped 400 parts per million (ppm) in 2013, which far exceeds the range experienced over the last 650,000 years.[1,2]

An overwhelming consensus of scientists now warns that climate change is due to human activities. This consensus extends to warning that if we do not curb our current carbon emissions, the increase in the planet's temperature will cause significant harm to natural systems and threaten our health and very existence.[1]

Efforts to reduce carbon emissions, called mitigation, are imperative. Because of the longevity of atmospheric carbon dioxide, the increased levels already present will continue to cause climate impacts such as sea level rise and atmospheric warming that cannot be reversed.[3] Adaptation is the term used to describe the measures we take to prepare for and respond to these inevitable climate changes.

> ***An overwhelming consensus of scientists now warns that this climate change is due to human activities.***

How does climate change impact climate and weather?

Changes in atmospheric and ocean temperatures affect the general behavior of Earth's water, including how the atmosphere holds water vapor as it warms. Along with

temperature, the timing, amount, and the manner in which the water circulates (i.e., the hydrologic cycle) or covers the Earth are all part of what defines our climate and weather.

Weather can be thought of as the short-term variability of local daily temperature, precipitation (i.e., rain, snow), wind, and events like storms (hurricanes, tornados, etc.) throughout a year. Climate can be thought of as the general pattern on a larger geographic area and time scale, usually in the span of decades.

California is unique in the United States and has a Mediterranean type of climate with a distinct dry season (May to October) and wet season (November to April) which is modified by proximity to the coast or mountains and variable elevation.

The future amount of carbon emitted into the Earth's atmosphere has two broad drivers:

- the dependence of economic growth on fossil fuels, and
- the growth of the world's population.[1,4]

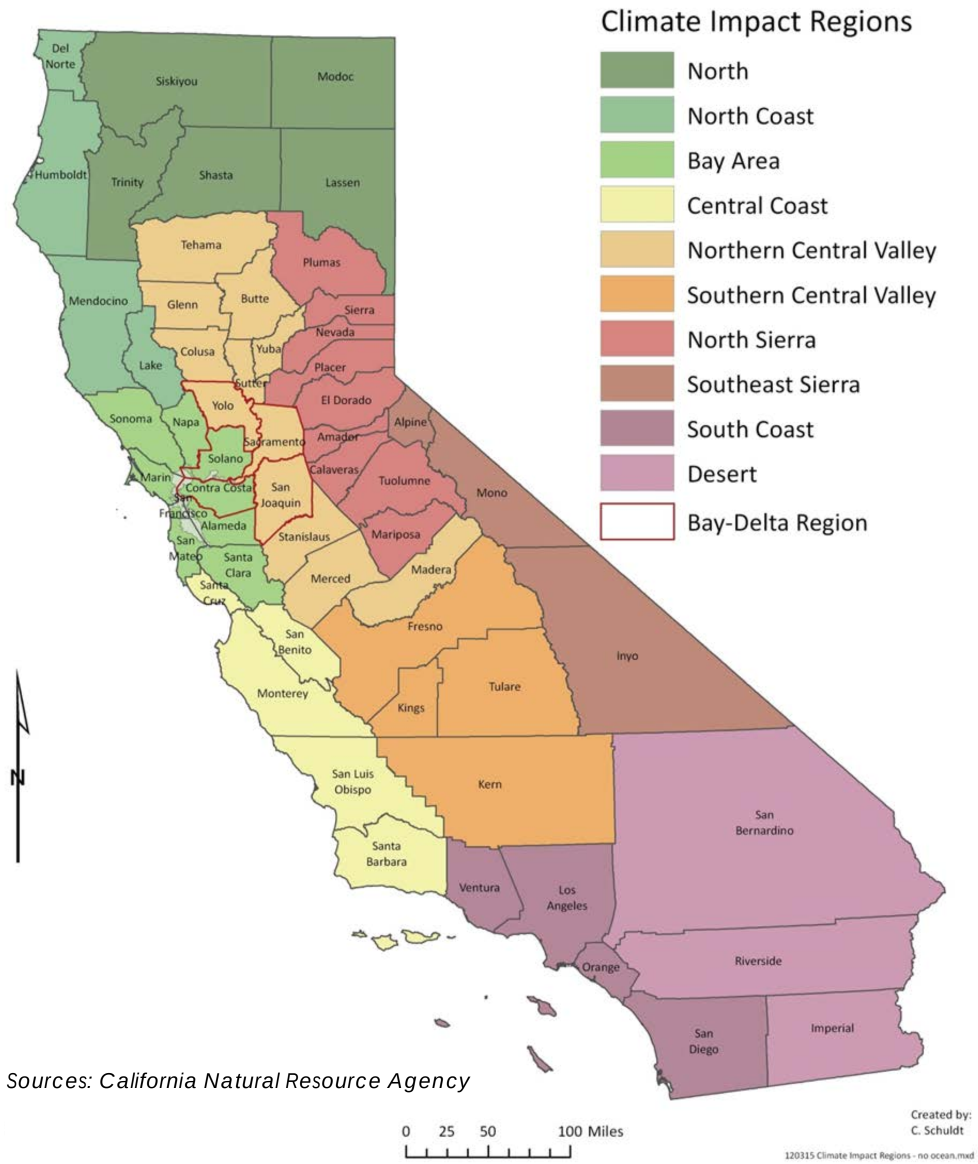
Based on the different combinations of economic development strategies and population growth, scientists have constructed formal scenarios[4] of future carbon emissions during the 21st century and predicted the associated climate impacts compared to a 1990 baseline.[4] In an optimistic scenario, in which world economies become much less dependent on fossil fuels and the world population levels off after 2050[1], the average global temperature is predicted to increase by 1.8°C (3.2° F).

In a pessimistic scenario, in which we continue to emit greenhouse gases on the same current trajectory, climate models predict a 3.4° C (6.1° F) increase. This scenario is based on the assumption that the world continues its path of fossil fuel-intensive economic development and that the world population increases during the 21st century.

On the backdrop of gradually increasing temperatures and sea levels, these climate models also predict an increase in the frequency and intensity of extreme weather events such as hurricanes, floods, and droughts.[1] More information about climate models is described in Appendix 1.

WHAT ARE THE CLIMATE PROJECTIONS FOR THE BAY AREA REGION?

The impact of climate change in California varies across the state due to diversity in biophysical setting, climate, and jurisdictional characteristics. The California Adaptation Planning Guide organized the state into climate impact regions based on county boundaries in combination with projected climate impacts, existing environmental settings, socioeconomic factors, and regional designations and organizations.[5] Figure 1 is a map of climate impact regions.



Sources: California Natural Resource Agency

Figure 1. California Climate Impact Regions designated in the *California Climate Adaptation Planning Guide*[5]

Table 1 summarizes Cal-Adapt projections for the Bay Area Region and is intended to identify the major types of changes projected for the region. Regional projections may differ from county level projections.

Table 1. Summary of Cal-Adapt Climate Projections for the Bay Area Region[5]

	RANGES
Temperature Change, 1990-2100	January: Increase in average temperature of 2°F by 2050 and up to 5°F. July: Increase in average temperature of 4°F by 2050 and up to more than 6°F by 2100. *(Modeled high temperatures – average of all models; high carbon emissions scenario)*
Precipitation	Precipitation varies widely in this region, with annual totals over 40 inches in northern Sonoma County to roughly 15 inches in the eastern portions of Solano and Contra Costa counties. A moderate decline in annual rainfall, 1 to 3 inches by 2050 and 4 to 5 inches by 2090, is projected throughout the region. *(CCSM3 climate model; high carbon emissions scenario)*
Sea Level Rise	By 2100, sea levels may rise up to 66 inches, posing considerable threats to coastal areas and particularly to low-lying areas adjacent to San Francisco bay. The number of acres vulnerable to flooding is expected to increase 20 to 30 percent in most parts of the Bay Area, with some areas projected for increases over 40 percent. Coastal areas are estimated to experience an increase of approximately 15 percent in the acreage.
Heat Wave	Along the coast, particularly to the south, heat wave is defined as five days over 72°F to 77°F; in other areas the threshold is in the mid- to upper 90s. Over most of the region, a limited increase in the number of heat waves is expected by 2050, with only the eastern areas expecting more than one or two more per year. By 2100, between six and 10 more heat waves can be expected per year.
Fire Risk	There is little change in projected fire risk in this region, save for the slight increases expected in western Marin County. *(GFDL model, high carbon emissions scenario)*

Source: Public Interest Energy Research, 2011. Cal-Adapt[6]; OPC 2013. State of California Sea-level Rise Guidance Document[7]

What are the Climate Projections for Santa Clara County?

Projected Temperature Changes in Santa Clara County

Overall, temperatures are expected to rise substantially throughout this century. During the next few decades, scenarios project average temperature to rise between 1°F and 2.3°F in California.[6] The projected temperatureincreases begin to diverge at mid-century so that, by the end of the century, the temperature increases projected in the higher emissions scenario are approximately twice as high as those projected in the lower emissions scenario. Figure 2 shows the projected temperature changes in 2099 scenarios for Santa Clara County.[6]

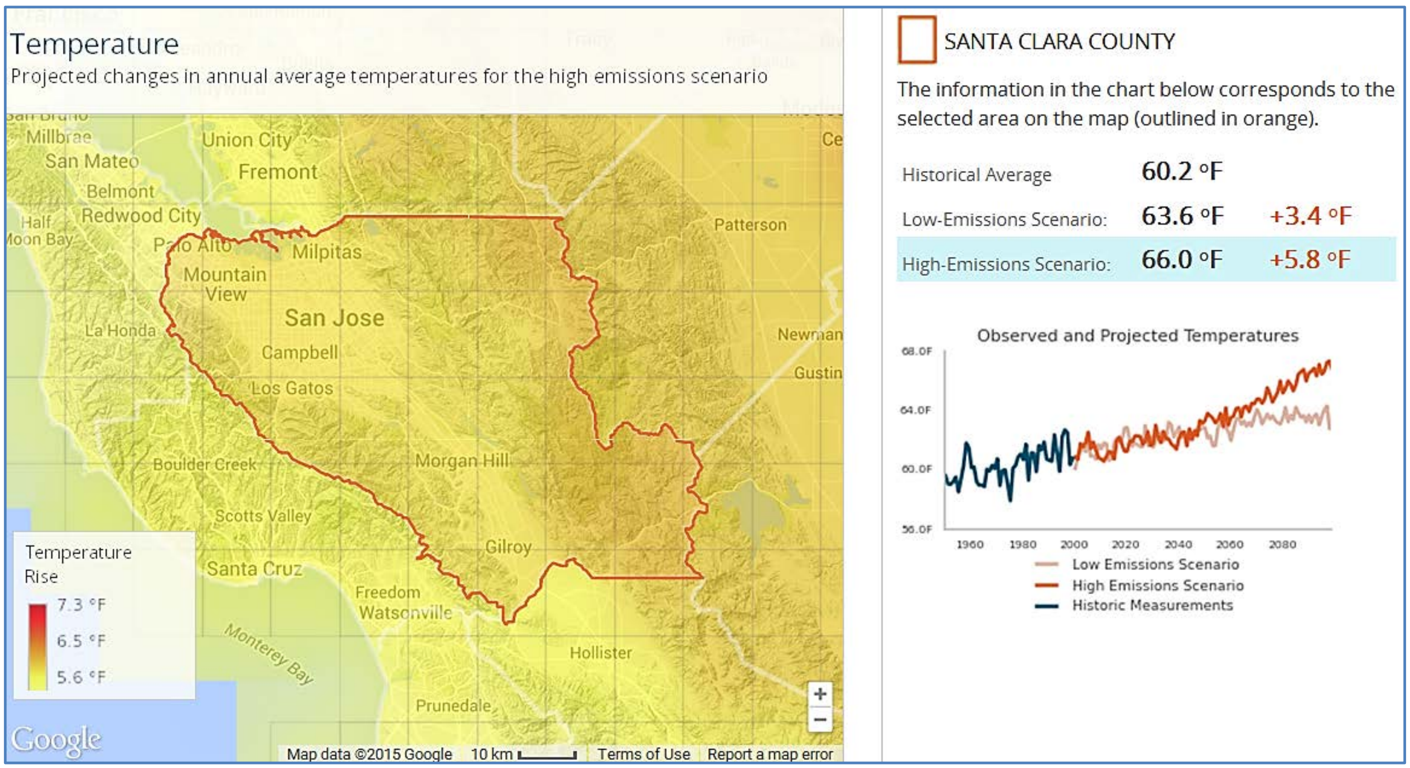


Figure 2. Projected changes in annual average temperature in future carbon emissions scenarios, Santa Clara County, 2099[6]

Current Fire Hazard Severity Zones in Santa Clara County

While all of California is subject to some degree of fire hazard, there are specific features that make some areas more hazardous.[8] Figure 3 visualizes current fire hazard severity zones in Santa Clara County.

Fire Hazard Severity Zones (FHSZ) were developed using a computer model.[8] They predict the physical damage a fire is likely to cause based on the factors that influence fire likelihood and behavior.[8] Many factors are considered such as fire history, existing and potential fuel (natural vegetation), flame length, blowing embers, terrain, and typical weather for the area.[8,9]

Fire Hazard Severity Zones are categorized into three categories:

- **Moderate**

  Wildland areas supporting areas of typically low fire frequency and relatively modest fire behavior, or developed/urbanized areas with a very high density of non-burnable surfaces (including roadways, irrigated lawn/parks, and low total vegetation cover (<30%) that is highly fragmented and low in flammability).[9]

- **High**

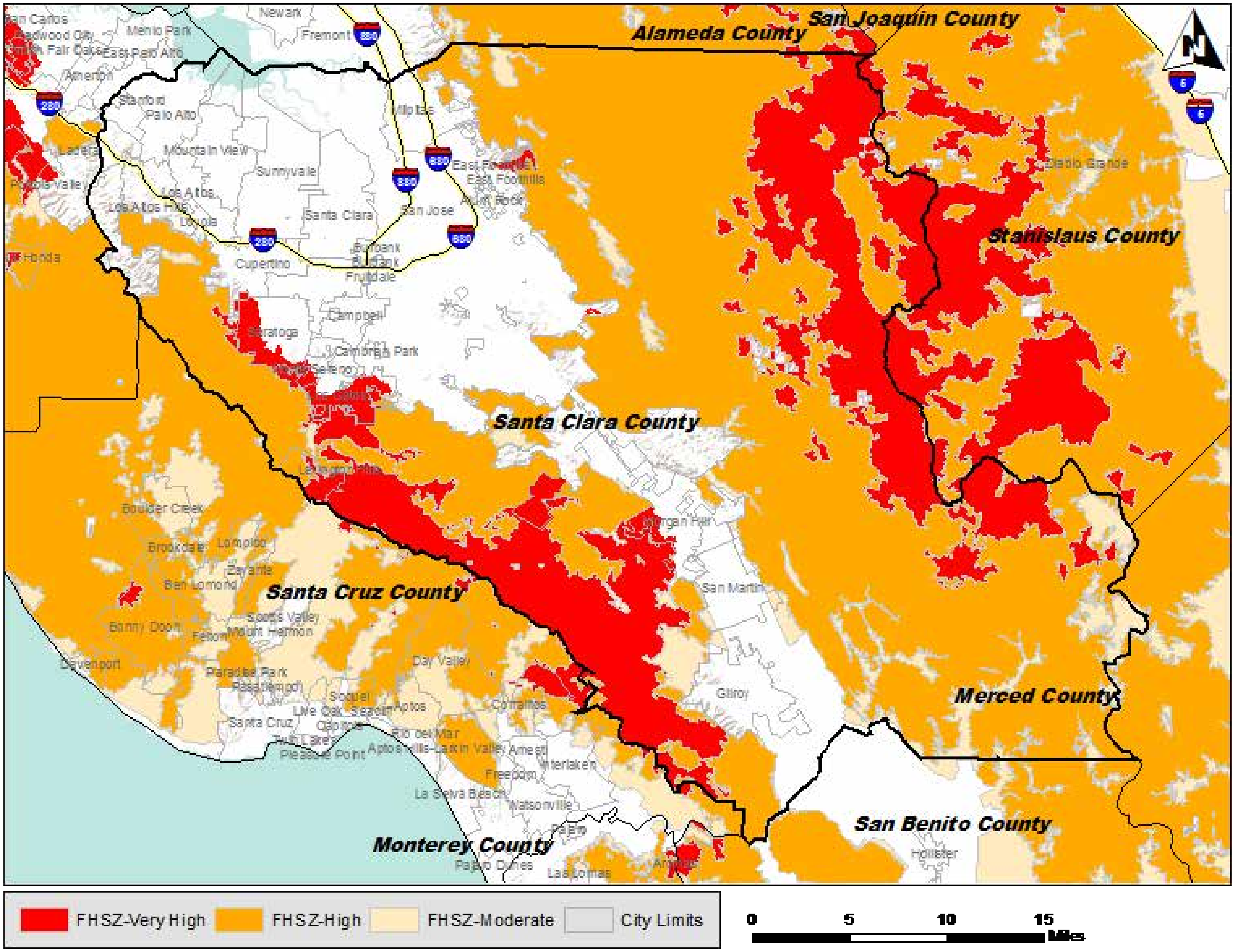
  Wildland areas supporting medium- to high-hazard fire behavior and roughly average burn probabilities, or developed/urbanized areas with moderate vegetation cover and more limited non-burnable cover. Vegetation cover typically ranges from 30-50% and is only partially fragmented.[9]

- **Very High**

  Wildland areas supporting high to extreme fire behavior resulting from climax fuels typified by well-developed surface-fuel profiles (e.g., mature chaparral) or forested systems where crown fire is likely, or developed/urban areas typically with high vegetation density (>70% cover) and associated high fuel continuity. This allows for flames to spread over much of the area impeded only by isolated non-burnable areas.[9]

The FHSZ rating system is more completely described at *http://frap.fire.ca.gov/projects/hazard/fhsz_review_instructionsv1_3b.pdf.*

Figure 3. Current Fire Hazard Severity Zones (FHSZ), Santa Clara County, 2007[9]
Note: Map includes only state and local responsibility areas.



Projected Wildfire Acreage in Santa Clara County

Periodic natural fire is an important ecosystem disturbance. Uncontrolled wildfires, however, can be extremely damaging to communities and ecosystems. Fire can promote vegetation and wildlife diversity, release nutrients into the soil, and eliminate heavy accumulation of underbrush that can fuel catastrophic fires.

The map below (Figure 4) displays the projected increase or decrease in potential area burned based on projections of the Coupled Global Climate Model (version 3) for the high carbon emissions scenario in 2085.[6] The bar graphs to the right of the map in Figure 4 illustrate the projected time trend over the 21st century for both the high and low emissions scenarios. Please note that these data are modeled solely on climate projections and do not take landscape and fuel sources into account. The projections of acreage burned are expressed in terms of the relative increase or decrease (greater or less than 1) from a 2010 baseline for fires that consume at least 490 acres. The 2010 baseline reflects historic data from 1980-1989 and trends through 2010. Data on the number of fires and the acreage burned are described later in this report.

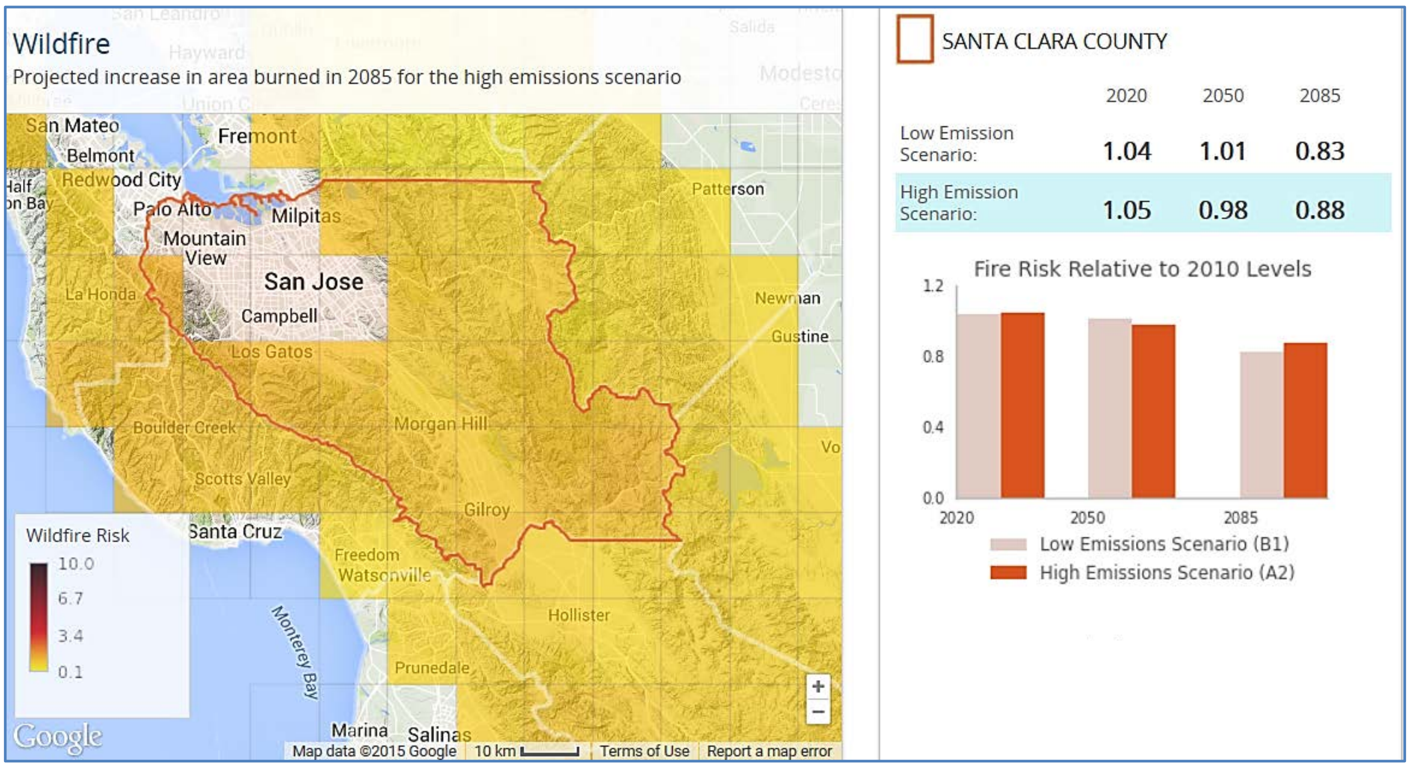


Figure 4. Relative increase in wildfire acreage in future carbon emission scenarios, Santa Clara County[6]

Projected Sea Level Rise in Santa Clara County

California's coastline, which includes more than 2,000 miles of open coast and enclosed bays, is vulnerable to a range of natural hazards, including storms, extreme high tides, and rising sea levels.[6]

Sea level rise increases the threat of coastal flooding.[10] Climate change models indicate that California may see up to a 66 inch (167 cm) rise in sea level within this century.[7]

The map in Figure 5 displays areas that may be inundated during an extreme flood event known as a 100-year flood.[11] The100-year flood is used as a standard for planning, insurance, and environmental regulations. The 100-year flood means that, in any given year, there is a 1% chance a flood risk area will be flooded. "100-year flood" is a technical term and does not imply that floods of this magnitude can only occur once every hundred years.

The blue color indicates areas already threatened today.[11] The red color represents the area projected to be threatened for a 55-inch sea level rise, which is consistent with a high carbon emissions scenario.[11] The map does not take into account protective structures, such as levees, or the effects of wind and waves.[11]

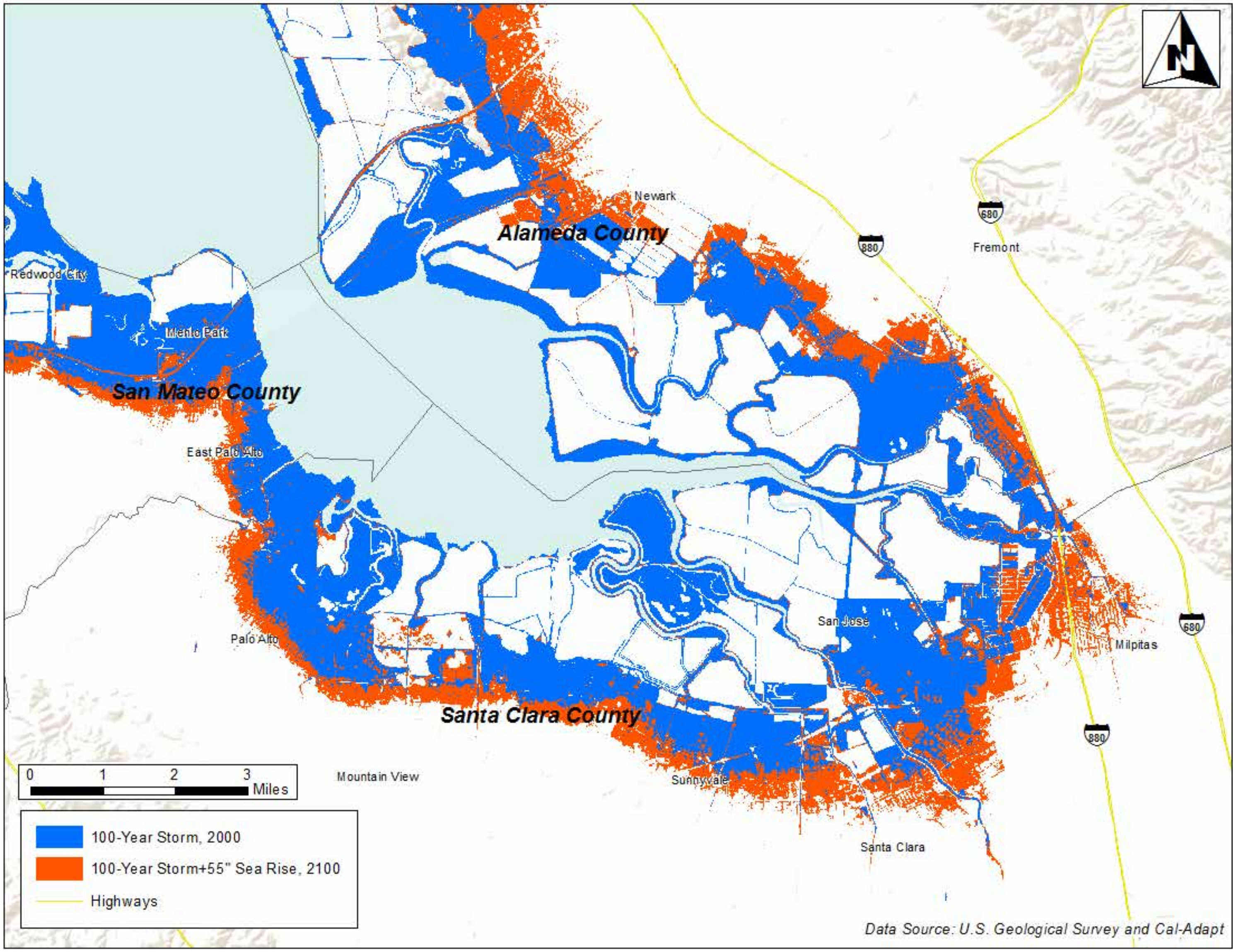
For in depth planning and estimating risks from sea level rise, the California Ocean Protection Council highly recommends integrating shoreline changes, bathtub effect, and 100-year storm flood. The State *Sea Level Rise Policy Guidance* Document is being updated in 2017.

Resources

- California Ocean Protection Council 2013; State of California Sea-level Rise Guidance Document
  http://www.opc.ca.gov/webmaster/ftp/pdf/docs/2013_SLR_Guidance_Update_FINAL1.pdf

- For more in depth resources for county-specific values for acres vulnerable to flooding please see:
  Sea Level Rise and Coastal Flood Web Tools Comparison Matrix: California
  http://sealevel.climatecentral.org/matrix/CA.html

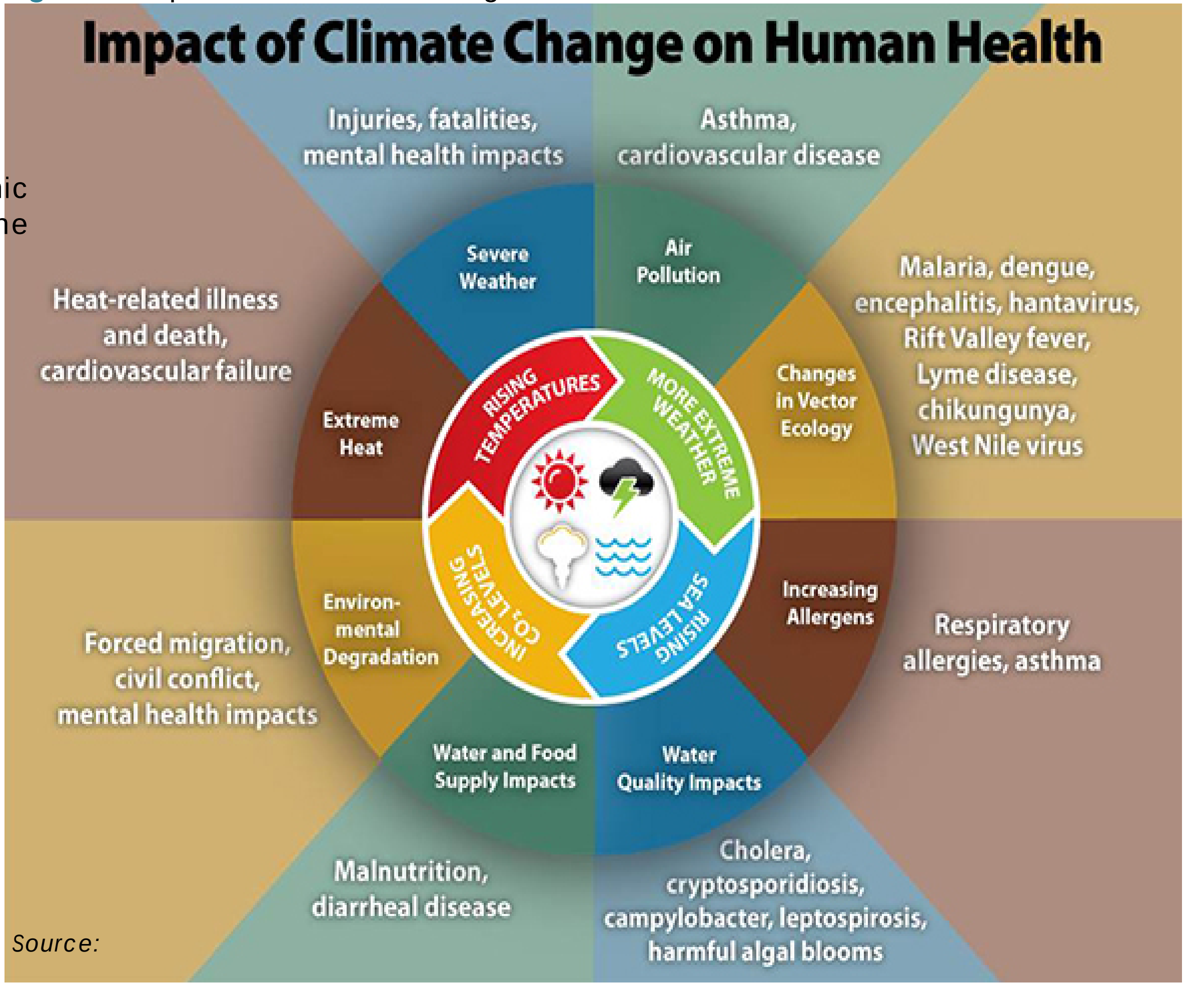
Figure 5. Baseline inundation areas for a 100-year flood (2000) and modeled scenario with additional 55 inches of sea level rise (2100), Santa Clara County[6] Note: Current projections are 66 inches above sea level.[7]



OVERVIEW OF CLIMATE CHANGE AND HEALTH IMPACTS

Researchers have examined the pathways in which increased temperatures and hydrologic extremes can impact health and generally recognize three main pathways: direct exposures, indirect exposures, and socioeconomic disruption (Figure 6).[12] Based on the review of weather-related natural disasters and historical patterns[13,14] and scientific judgment, public health researchers have suggested the nature and direction of health harms or benefits.[12,15]

Figure 6. Impact of Climate Change on Human Health



Source:

Extreme Weather-Related Injury, Displacement, and Mental Health

Extreme weather events (storms, flooding) cause fatal and nonfatal injuries from drowning, being struck by objects, fire, explosions, electrocution, or exposure to toxic materials. A widespread weather-related natural disaster may destroy or ruin housing, schools and businesses and cause temporary or permanent displacement. Individuals and families may experience post-traumatic stress, depression, and increased risk of suicide.[16,17]

Health Impacts of Heat

Increased temperatures manifested as heat waves and sustained high heat days directly harm human health through heat-related illnesses (mild heat stress to fatal heat stroke) and the exacerbation of pre-existing conditions in the medically fragile, chronically ill, and vulnerable.[18,19] Increased heat also intensifies the photochemical reactions that produce smog and ground level ozone and fine particulates ($PM_{2.5}$), which contribute to and exacerbate respiratory disease in children and adults. Increased heat and carbon dioxide enhance the growth of plants that produce pollen, which are associated with allergies. Increased temperatures add to the heat load of buildings in urban areas and exacerbate existing urban heat islands adding to the risk of high ambient temperatures.

Health Impacts of Drought

Lack of moisture, already at a severe level in California due to a current multi-year drought and decades of fuel accumulation from historical forestry and fire suppression practices, increases the risk of wildfires.[20] Devastating wildfires like the Rim Fire of 2013 impact watersheds and increase the risk of landslides or mudslides, and sediment in run-off that reduce water quality. In addition to fire-related injuries, local and regional transport of smoke, ash, and fine particles increases respiratory and cardiovascular risks.

Increasing temperatures and changes in precipitation may lead to intensified drought conditions.[21] Drought decreases the availability and quality of water for humans. This includes reduced water levels to fight wildfires. Drought may increase exposure to health hazards including wildfires, dust storms, extreme heat events, flash flooding, degraded water quality, and reduced water quantity.[22] Dust storms associated with drought conditions have been associated with increased incidents of Valley fever, a fungal pathogen.[22]

Vector-borne Illnesses

Climatic changes alter the range, biogeography, and growth of microbes and the vectors of food, water, and vector-borne illnesses.[22] This includes the changes in aquatic environments that could increase harmful algal blooms and lead to increases in foodborne and waterborne illnesses.[21]

Food Insecurity

Climate change is expected to have global impacts on food production and distribution systems.[22] This can cause food prices to increase, which makes food less affordable and increases food insecurity, obesity, and malnutrition in economically constrained households.[22]

Sea Level Rise, Mold, and Indoor Air Quality

Through sea level rise, salt water may intrude into coastal aquifers thus reducing quality and quantity of water supply. Coastal erosion can contribute to the loss of recreational venues and pose a variety of hazards to infrastructure and public safety. Water intrusion into buildings can result in mold contamination leading to indoor air quality problems.[22]

Socioeconomic Disruption

Widespread social and economic disruption includes damage to the infrastructure for the delivery of health services and for general economic well-being. Health care facilities, water treatment plants, and roads for emergency responders and transportation for health care personnel can be damaged in climate-related extreme weather events. Increased burden of disease and injury will test the surge capacity of health care facilities. Economic disruption can lead to income loss, income insecurity, food insecurity, housing insecurity, and mental health problems, which in turn may increase substance abuse, suicide,[23,24] and other health problems.

Energy production and distribution is also threatened by heat and wildfires through loss of efficiency, generating capacity, and fires disrupting transmission lines. California's ports that provide the gateway to goods for California, national, and international markets are at risk from sea level rise and coastal storms.

WHICH POPULATION SUBGROUPS ARE PARTICULARLY VULNERABLE?

All Californians are vulnerable to the health impacts of climate change. Even if one is fortunate to live, work, study, or play in a place without direct contact with wildfires, flooding, or sea level rise, no one can entirely avoid excessive heat or the indirect effects of extreme weather events. The table in Appendix 2 summarizes the populations more vulnerable to the health impacts of climate change.

Based on medical reviews of individuals who died during heat waves and other extreme weather events, those who are particularly vulnerable to the direct effects of climate change include the very old and very young, individuals who have chronic medical conditions and psychiatric illness, people taking multiple medications, people without means for evacuation (no access to public transit or private cars), people who are socially isolated, medically fragile people, and people living in institutions.[17] Acclimatization to heat may help reduce risks from heat waves in the healthy general population, but may not be sufficient to protect those with underlying medical conditions.

A much larger part of the population is vulnerable to intermediate or socioeconomic factors such as preexisting physical and mental health conditions, cultural or physical isolation, occupations involving outside or high risk work, a precarious socioeconomic status, or lack of social cohesion and collective efficacy. Collective efficacy and local community cohesion may be associated with effective action to plan and coordinate responses to climate threats.[20]

The underlying burden of disease and injury accounted for by the social determinants of health[25] considers the economic, service, and built environments in which people live, work, learn, and play.[26] Climate change magnifies existing health disparities. Disadvantaged populations, such as those with low education, experiencing racial segregation, low social support, poverty, and income inequality face disproportionate climate-related health burden.[27]

Community resilience refers to actions taken by individuals, neighborhoods, organizations, and multiple sectors of government to resist and overcome obstacles and to promptly recover from climate threats. In the short term, this may include traditional elements of public health preparedness and community development. However, in the long term, this may include actions to broadly promote population health and decrease the number of people with physical and mental conditions rooted in the social determinants of health.

Health inequities based on race/ethnicity, income, geography (urban/rural) are widespread today in California.[26] Even without climate change, demographic changes already underway will increase the size of vulnerable populations in California in the coming decades. The population is aging and the share of individuals aged 65 or more years will increase from 13 percent in 2010 to 19 percent in 2050.[28] In many California communities, racial and ethnic minorities constitute the majority of residents.

What are the Health Status, Health Inequities, and Population Vulnerabilities in Santa Clara County?

Climate change impacts the health and well-being of Californians. Estimates for health status, health inequities, and population vulnerabilities are summarized in Figures 7 and 8.

There is a broad range of environmental hazards attributed to climate change including heat waves, wildfires and wildfire smoke, air pollution, sea level rise and inland flooding.

All Californians are at risk from extreme heat. In 2010, approximately 2% (34,030 residents) of the county's population lived in fire hazard zones of moderate to very high severity. From 1980 to 1989 (a pre-climate change baseline), 24 wildfires at least 490 acres in size consumed a total of 112,892 acres in the Bay Area Region.

In addition to heat and wildfire, the county is at risk of hazards posed by sea level rise and coastal flooding. In 2010, approximately 12,532 residents lived on coastal blocks that were at risk of inundation from a 100-year flood. With an additional 55 inches of sea level rise, which is toward the upper end of projections for the year 2100, the inundation zone would potentially include 39,377 residents. This is likely an underestimate as more recent climate change models indicate that California may see up to a 66 inch (167 cm) rise in sea level within this century.[10]

Climate change affects the social and environmental drivers of health outcomes. The effects of climate change can exacerbate existing health conditions and compound the risks of adverse health outcomes. The age-adjusted death rate, which takes into account the effect of the population's age distribution, is a basic indicator of the health status of communities.

In 2010, the age-adjusted death rate in Santa Clara County was lower than the state average. Disparities in death rates among race/ethnicity groups highlight how certain populations disproportionately experience health impacts. Within the county, the highest death rate occurred among American Indians and Pacific Islanders and the lowest death rate occurred among Asians.

In 2012, nearly 40% of adults (545,922) reported one or more chronic health conditions including heart disease, diabetes, asthma, severe mental stress or high blood pressure. In 2012, 15% of adults reported having been diagnosed with asthma. In 2012 approximately 19% of adults were obese (statewide average was 25%). In 2012, nearly 8% of residents aged 5 years and older had a mental or physical disability (statewide average was 10%).

In 2005-2010, there was an annual average of 99 heat-related emergency room visits and an age-adjusted rate of 5.7 emergency room visits per 100,000 persons (the statewide age-adjusted rate was 10 emergency room visits per 100,000 persons).

Among climate-vulnerable groups in 2010 were 124,464 children under the age of 5 years and 196,944 adults aged 65 years and older. In 2010, there were approximately 30,350 people living in nursing homes, dormitories, and other group quarters where institutional authorities would need to provide transportation in the event of emergencies.

Social and demographic factors and inequities affect individual and community vulnerability to the health impacts of climate change. In 2010, 12% of households (73,743) did not have a household member 14 years or older who spoke English proficiently (called linguistically isolated; statewide average was 10%). In 2010, approximately 14% of adults aged 25 years and older had less than a high school education (statewide average was 19%).

In 2010, 9% of the population had incomes below the poverty level (the statewide average was 14%). Eighteen percent of households paid 50% or more of their annual income on rent or a home mortgage (statewide average was 22%). In 2012, approximately 115,000 (34%) of low-income residents reported they did not have reliable access to a sufficient amount of affordable, nutritious food (called food insecurity; statewide average was 42%).

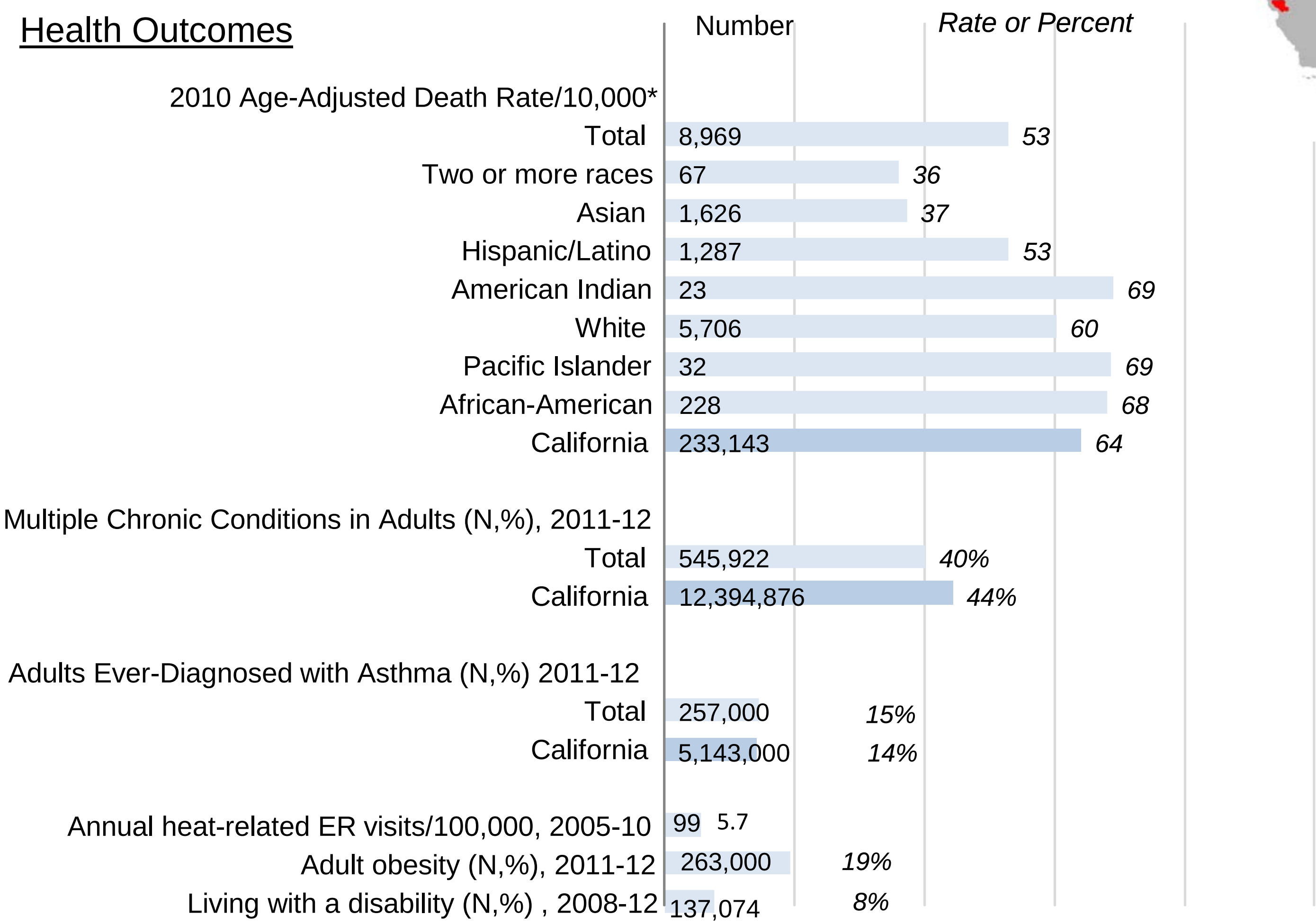
In 2010, Santa Clara County had approximately 39,367 outdoor workers whose occupation increased their risk of heat illness. In 2010, roughly five percent of households did not own a vehicle that could be used for evacuation (statewide average was 8%). In 2012, approximately 35% of residents did not live within a half mile to frequent public transit. In 2009, approximately 35% of households were estimated to lack air conditioning, a strategy to counter adverse effects of heat (statewide average was 36%). In 2011, tree canopy, which provides shade and other environmental benefits, was present on 9% of the county's land area (statewide average was 8%).

Social capital is embedded in social relationships and networks and refers to the existence of trust and mutual aid among the members of society.[29,30] These relationships are important in building resilience when confronted with extreme climates.[30] There is evidence that populations with higher levels of political participation also have greater social capital.[31,32] Sixty-five percent of registered voters voted in the 2010 general election (statewide average was 58%).

Natural disasters worsened by climate change increase the displacement of victims, which in turn increase population densities and tensions over resources.[33,34] Violent crime also increases during heat events.[35] Safe neighborhoods that are free of crime and violence are an integral component of healthy neighborhoods and community resilience. In 2010, Santa Clara County experienced approximately 3 violent crimes per 1,000 residents (statewide rate was 4 per 1,000 residents).

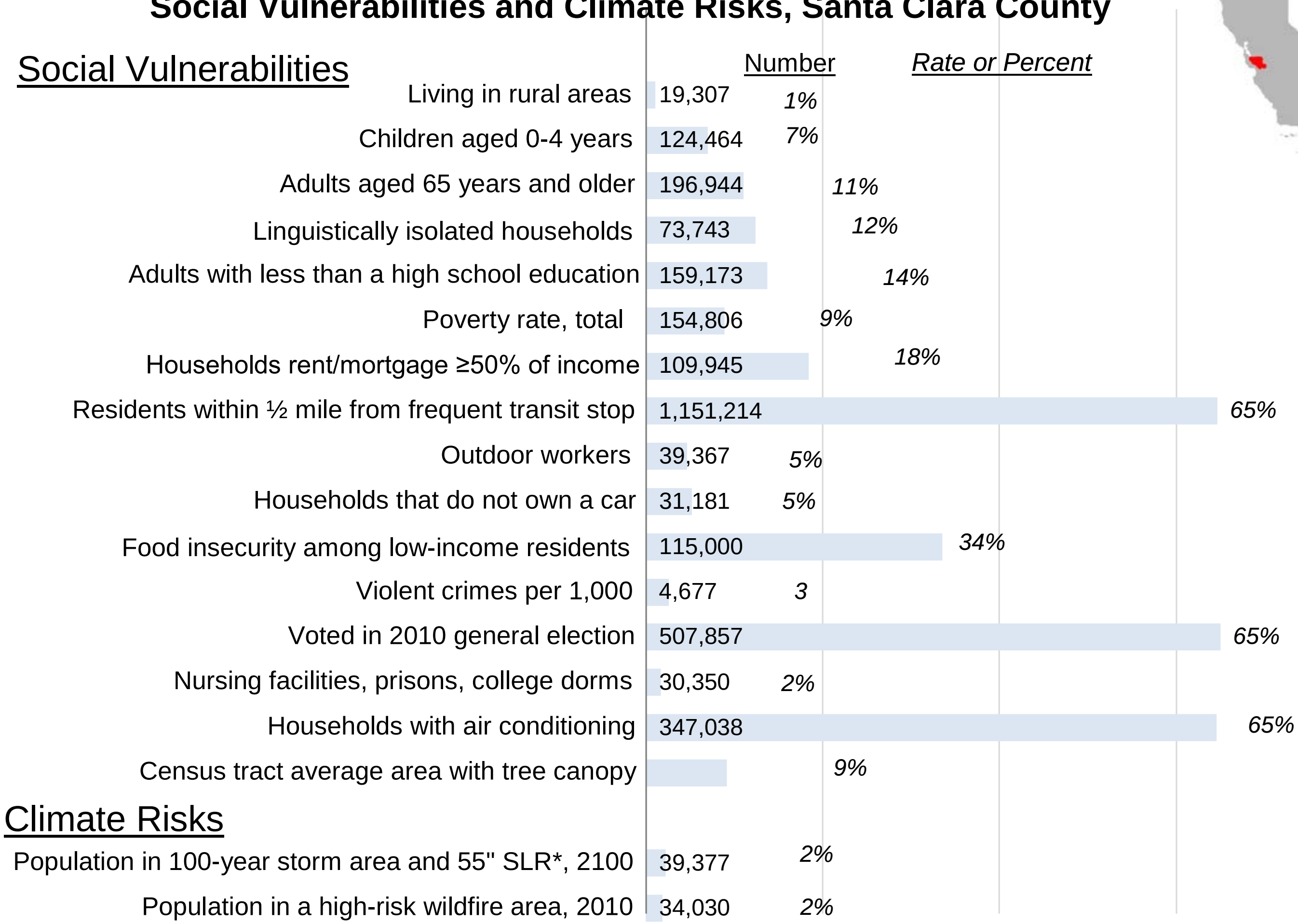
These findings highlight specific populations that are most susceptible to health risks, as well as the social determinants of health and adaptive capacity that contributes to resilience or conversely intensifies the impacts from climate change.

Figure 7. Profile of Health Outcomes and Inequities, Social Vulnerabilities and Climate Risks, Santa Clara County



* Groups with less than 20 observations are not presented

Figure 8. Profile of Health Outcomes and Inequities, Social Vulnerabilities and Climate Risks, Santa Clara County



*Current sea level rise (SLR) projections are up to 66 inches by 2100.[7]

Data sources for indicators in Figures 7 and 8 are described in Appendix 3.

SELECTED PUBLIC HEALTH STRATEGIES AND ACTIONS FOR ADAPTING TO CLIMATE CHANGE

Findings from this report describe climate risks in Santa Clara County and highlight certain populations who are most susceptible to health risks from current and future climate change exposures. Some of the changes due to climate change will occur over the long term, but broad shifts in our weather can be seen now and will result in many direct and indirect health risks.

Coping with a changing climate presents opportunities for local health departments and partners to consider policies, actions, and infrastructure design that will not just protect the public from climate change threats, but also establish health equity, resiliency, and sustainability. A critical step for building resilience is to improve capacity of communities to prepare, respond, and recover from climate-related health risks. Steps need to be taken to ensure that the most vulnerable populations have access to information, services, and resources to prepare and respond to climate risks.

The goal of public health adaptation strategies is to minimize the negative health impacts of climate change. A selection of the near-term and long-term strategies and actions steps for adapting to climate change are outlined in Table 2. These include community education and engagement, public health workforce development, identification of co-benefits, bolstering existing functions of public health preparedness and surveillance, multi-sectorial partnership building, and research.

Table 2. Selected public health strategies and action steps for adapting to climate change[36]

STRATEGIES	NEAR-TERM ACTIONS	LONG-TERM TERM ACTIONS
1. Promote community resilience to climate change to reduce vulnerability	• Promote healthy, built environments • Identify and reduce health vulnerabilities • Improve food security and quality	• Promote food sustainability • Reduce heat islands • Support social and community engagement • Promote increased access to health care
2. Educate, empower and engage California residents, organizations and businesses to reduce vulnerability through mitigation and adaptation	• Educational outreach campaign tying into existing efforts • Specific outreach to vulnerable populations	• Proactive social marketing campaign

3. Identify and promote mitigation and adaptation strategies with public health co-benefits	• Identify and prioritize strategies with public health co-benefits	
4. Establish, improve and maintain mechanisms for robust rapid surveillance of environmental conditions, climate-related illness, vulnerabilities, protective factors and adaptive capacities	• Monitor outcomes (state and local) • Develop existing environmental contaminant biomonitoring • Maintain and upgrade water accessibility information • Improve heat warning systems	• Convert to electronic surveillance systems to improve disease reporting, management and surveillance
5. Improve and sustain public health preparedness and emergency response	• CDPH and local health departments should refine existing preparedness plans and conduct exercises	
6. Work in multi-sectoral partnerships (local, regional, state and federal)	• Expand training and education to build collaborative capacity	
7. Conduct applied research to enable enhanced promotion and protection of human health	• Vulnerability assessments • Research collaboration • Assess local impacts on health	
8. Implement policy changes at local, regional and national levels	• Policy collaboration with stakeholders • Occupational safety standards	• Model policies and training • Public engagement
9. Identify, develop and maintain adequate funding for implementation of public health adaptation strategy	• Identify and develop funding mechanisms	• Develop funding mechanisms/AB32 for education and research

Source: California Natural Resources Agency[36] *(http://resources.ca.gov/docs/climate/Statewide_Adaptation_Strategy.pdf)*

Resources

This report brought together recently published, technical information from state-sponsored research and planning documents, including:

- California Climate Change Adaptation Planning Guide, 2012
  http://resources.ca.gov/climate/safeguarding/adaptation_policy_guide/

- Safeguarding California: Reducing Climate Risk, 2014
  http://resources.ca.gov/docs/climate/Final_Safeguarding_CA_Plan_July_31_2014.pdf

- California Climate Adaptation Strategy, 2009
  http://resources.ca.gov/docs/climate/Statewide_Adaptation_Strategy.pdf

- Cal-Adapt: Exploring California's Climate Change Research
  http://cal-adapt.org/

- Preparing California for Extreme Heat: Guidance and Recommendations
  http://www.climatechange.ca.gov/climate_action_team/reports/Preparing_California_for_Extreme_Heat.pdf

For more information and resources for climate change adaptation and public health planning, please visit:

- CDPH CalBRACE web page
  http://www.cdph.ca.gov/programs/Pages/CalBRACE.aspx

- CDPH Climate Change and Health Team web page
  https://www.cdph.ca.gov/programs/Pages/ClimateChange.aspx

- CDC BRACE web page
  http://www.cdc.gov/climateandhealth/default.htm

- The Impacts of Climate Change on Human Health in the United States: A Scientific Assessment 2016
  https://health2016.globalchange.gov/

- Urban Heat Island Index for California
  http://calepa.ca.gov/UrbanHeat/Maps/default.htm

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APPENDIX 1: HOW ARE FUTURE CHANGES IN CLIMATE PREDICTED?

Scientists use historical weather data and mathematical models to describe historical trends and to predict the impacts of global warming.[3] Historical data show that on average sea levels are already rising, primarily from the expansion of water.[1] Historical data also show that in the past century average temperatures are increasing, polar ice and glaciers are melting at increased rates, and snow pack in mountains is diminishing compared to time periods in which human-generated carbon emissions were relatively small.[1]

Climate models are computer simulation over time of the Earth's atmosphere and oceans taking into account solar radiation, surface reflection, circulating air masses and wind, heat stored in oceans, sea ice, evaporation from land surfaces and green plants, cloud cover, and other factors. A key input to climate projection models is the current and projected amount of carbon dioxide and other greenhouse gases emitted into the atmosphere.

The future amount of carbon emitted into the Earth's atmosphere has two broad drivers:

- the dependence of economic growth on fossil fuels, and
- the growth of the world's population.

Based on the different combinations of economic development strategies and population growth, scientists have constructed formal scenarios[4] of future carbon emissions during the 21st century and predicted their associated climate impacts compared to a 1990 baseline.[6]

The average global temperature is predicted to increase by 1.8°C (3.2° F) for an optimistic scenario called B1 in which world economies become much less dependent on fossil fuels and the world population levels off after 2050.[1] In a pessimistic scenario called A2, climate models predict a 3.4° C (6.1° F) increase, based on the assumption that the world continues its path of fossil fuel intensive economic development and that the world population increases during the 21st century.

On the backdrop of gradually increasing temperatures and sea levels, the climate models also predict an increase in the frequency and intensity of extreme weather events such as hurricanes, floods and droughts[1]. Using these global climate models as a starting point, the Scripps Institution of Oceanography at the University of California, San Diego has further refined climate impacts in California to 12 km grids (7 by 7 miles).[6] This allows California communities to have local data to inform climate adaptation planning.

APPENDIX 2: PUBLIC HEALTH IMPACTS OF CLIMATE CHANGE IN CALIFORNIA

CLIMATE CHANGE EXPOSURES	HEALTH IMPACTS	POPULATIONS MOST AFFECTED
Extreme Heat	• Premature death • Cardiovascular stress and failure • Heat-related illnesses such as heat stroke, heat exhaustion, and kidney stones	• Elderly • Children • Diabetics • Low-income • Urban residents • People with respiratory diseases • Agricultural workers • Those active outdoors
Poor Air Quality/ Air Pollution	• Increased asthma, allergies, chronic obstructive pulmonary disease (COPD), and other cardiovascular and respiratory diseases	• Children • Elderly • People with respiratory diseases • Low income • Those active outdoors
Wildfires	• Injuries and death from burns and smoke inhalation • Eye and respiratory illnesses due to air pollution • Exacerbation of asthma, allergies, chronic obstructive pulmonary disease (COPD), and other cardiovascular and respiratory diseases • Risk from erosion and land slippage after wildfires • Displacement and loss of homes	• People with respiratory diseases

CLIMATE CHANGE EXPOSURES	HEALTH IMPACTS	POPULATIONS MOST AFFECTED
Severe Weather, Extreme Rainfall, Floods, Water Issues	• Population displacement, loss of home and livelihood • Death from drowning • Injuries • Damage to potable water, wastewater, and irrigation systems, resulting in decrease in quality/quantity of water supply and disruption to agriculture • Water- and food-borne diseases from sewage overflow	• Coastal residents, and residents in flood-prone areas • Elderly • Children • Low income
Increased average temperature	• Cardiovascular disease • Increased number and range of: • Vector-borne disease, such as West Nile virus, malaria, Hantavirus, or plague • Water-borne disease, such as cholera and *E. coli* • Food-borne disease, such as *salmonella* poisoning • Harmful algal blooms causing skin disease and poisoning • Allergies caused by pollen, and rashes from plants such as poison ivy or stinging nettle • Vulnerability to wildfires and air pollution	• Children • Elderly • Agricultural workers • Those active outdoors • People with respiratory disease • People with acute allergies
Agricultural Changes	• Changing patterns and yields of crops, pests, and weed species, resulting in higher prices for food and food insecurity, hunger, and malnutrition • Changes in agriculture/forestry, leading to lost or displaced jobs and unemployment	• Agricultural workers • Rural communities • Low income • Elderly • Children

CLIMATE CHANGE EXPOSURES	HEALTH IMPACTS	POPULATIONS MOST AFFECTED
Drought	• Hunger and malnutrition caused by disruption in food and water supply, increased cost and conflict over food and water • Food- and water-borne disease • Emergence of new contagious and vector-borne disease	• Low income • Elderly • Children
All Impacts	Mental Health Disorders: (e.g., depression, anxiety, Post-Traumatic Stress Disorder, substance abuse, and other conditions) caused by: • Disruption, displacement, and migration • Loss of home, lives, and livelihood Health Care Impacts • Increased rates of illness and disease, emergency room use, and related costs borne by employers, health plans, and residents • Damage to health facilities	• All populations • Low income • Health care staff

Sources:

- *Climate Action for Health: Integrating Public Health Into Climate Action Planning. California Department of Public Health*[37]
- *Public Health-Related Impacts of Climate Change in California, A Report From: California Climate Change Center*[38]
- *Global Climate Change Impacts in the United States, Cambridge University Press*[39]
- *Centers for Disease Control and Prevention, Climate and Health Program*[40]

APPENDIX 3: DATA SOURCES

DATA SOURCE	INDICATOR
1. California Department of Forestry and Fire Protection (CalFIRE). Fire and Resource Assessment Program; *http://frap.cdf.ca.gov/*.	• Population in a high-risk wildfire area
2. Center for Health Statistics and Informatics. *Vital Statistics Query System*. Sacramento, CA: California Department of Public Health; 2004. *www.apps.cdph.ca.gov/vsq/default.asp.*	• Age-Adjusted Death Rate / 10,000 with race/ethnicity stratification
3. Environmental Health Tracking Program. Heat-Related Illness Data Query Options. Environmental Health Investigations Branch, California Department of Public Health, Richmond, CA *http://www.ehib.org/page.jsp?page_key=913.*	• Annual heat-related ER visits / 100,000
4. Federal Bureau of Investigation. *Uniform Crime Reports.* DS. Washington, DC: U.S. Department of Justice, Federal Bureau of Investigation; 2011. *https://www.fbi.gov/about-us/cjis/ucr/ucr.*	• Violent crimes per 1,000
5. Heberger M, Cooley H, Herrera P, Gleick P, Moore E. *The Impacts of Sea Level Rise on the California Coast*. Oakland, CA: Pacific Institute; 2009. *http://www.pacinst.org/wp-content/uploads/2013/02/exec_sum11.pdf*.	• Population in 100-year flood prone area (for coastal counties) • Population in 100-year flood area and 55” of sea level rise (for coastal counties)
6. Palmgren C, Stevens N, Goldberg M, Barnes R, Rothkin K. *California Residential Appliance Saturation Survey*. Oakland, CA: KEMA, Inc.; 2009.	• Households with air conditioning
7. Public Interest Energy Research (PIER) Program. *Cal-adapt: Exploring California's Climate Change Research*. Sacramento: California Energy	• Historical record of wildfires and acres

Commission; 2011. *http://cal-adapt.org.*	consumed
8. UCLA Center for Health Policy Research. *California Health Interview Survey.* Los Angeles, CA: UCLA Center for Health Policy Research; 2009. *http://www.chis.ucla.edu/main/default.asp?page=dac* (multiple chronic conditions, asthma, food insecurity (<200% poverty.	• Multiple chronic conditions in adults • Adults ever-diagnosed with asthma • Food insecurity (low-income residents) • Adult obesity
9. U.S. Census Bureau. *American Community Survey or SF1 file (Living with disability, living in rural areas, population aged < 5 years and >65 years living alone, linguistically isolated, poverty rate, housing cost burden, outdoor workers, group quarters)* DS. Washington, DC: U.S. Census Bureau; 2012. *http://factfinder2.census.gov.*	• Living with disability (age 5 and older) • Living in rural areas • Children aged 0-4 years • Adults aged 65 years and older • Linguistically isolated households • Poverty rate, total • Households rent/mortgage ≥50% of income • Outdoor workers • Households that do not own a car • Nursing facilities, prisons, college dorms • Adults with less than a high school education
10. US Environmental Protection Agency. *National Land Cover Data*; *http://www.epa.gov/mrlc/nlcd-2001.html.* Accessed March 24, 2011.	• Census tract average area with tree canopy
11. Southern California Association of Governments (SCAG), Metropolitan Transportation Commission (MTC), Sacramento Council of Governments (SACOG), and population data from the U.S. Census Bureau.	• Residents within ½ mile from frequent transit stop
12. Statewide Database, University of California Berkeley Law, Center for Research, California Secretary of State, Elections Division, Reports of Registration, California Department of Finance, Demographic Unit.	• Registered voters who voted in 2010 general election

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REVIEW

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Synergistic health effects of air pollution, temperature, and pollen exposure: a systematic review of epidemiological evidence

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Abstract

Background: Exposure to heat, air pollution, and pollen are associated with health outcomes, including cardiovascular and respiratory disease. Studies assessing the health impacts of climate change have considered increased exposure to these risk factors separately, though they may be increasing simultaneously for some populations and may act synergistically on health.

Our objective is to systematically review epidemiological evidence for interactive effects of multiple exposures to heat, air pollution, and pollen on human health.

Methods: We systematically searched electronic literature databases (last search, April 29, 2019) for studies reporting quantitative measurements of associations between at least two of the exposures and mortality from any cause and cardiovascular and respiratory morbidity and mortality specifically. Following the Navigation Guide systematic review methodology, we evaluated the risk of bias of individual studies and the overall quality and strength of evidence.

Results: We found 56 studies that met the inclusion criteria. Of these, six measured air pollution, heat, and pollen; 39 measured air pollution and heat; 10 measured air pollution and pollen; and one measured heat and pollen. Nearly all studies were at risk of bias from exposure assessment error. However, consistent exposure-response across studies led us to conclude that there is overall moderate quality and sufficient evidence for synergistic effects of heat and air pollution. We concluded that there is overall low quality and limited evidence for synergistic effects from simultaneous exposure to (1) air pollution, pollen, and heat; and (2) air pollution and pollen. With only one study, we were unable to assess the evidence for synergistic effects of heat and pollen.

Conclusions: If synergistic effects between heat and air pollution are confirmed with additional research, the health impacts from climate change-driven increases in air pollution and heat exposure may be larger than previously estimated in studies that consider these risk factors individually.

Keywords: Air pollution, Temperature, Pollen, Systematic review

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Background

Climate change is expected to increase exposure to environmental health risk factors, including extreme temperatures, air pollution, and aeroallergens [1–5]. These environmental health risk factors are associated with a range of health outcomes, including cardiovascular and respiratory disease [5]. Changes in these risk factors will be spatially heterogeneous, depending on local emission sources, meteorology, vegetation type and distribution, and other factors. As these risk factors do not exist in isolation, populations may experience simultaneous increases in exposure to heat, air pollutants, and pollen. Understanding whether these environmental health risk factors have synergistic effects on health outcomes can inform future climate change health risk assessments. The objective of this paper is therefore to determine whether the current state of the epidemiological evidence supports the presence of synergistic effects between heat, air pollutants, and pollen on human health outcomes.

Both average and extreme temperatures are expected to increase with climate change [5]. These changes may compromise the body's ability to regulate temperature leading to a range of health outcomes, including heat exhaustion, heatstroke, and hyperthermia [6]. Exposure to extreme heat events can worsen cardiovascular and respiratory diseases, as well as other chronic conditions, such as cerebrovascular disease, diabetes, and kidney disease [7, 8]. The mechanisms by which heat exacerbates respiratory disease are not well understood. In respiratory diseases such as asthma and chronic obstructive lung disease, inflammation plays a central role in the pathogenesis and exacerbation of the disease. Heat increases systemic and pulmonary inflammation as a consequence of thermoregulation – the attempt by the body to maintain a temperature within a safe range [9]. A second mechanism by which heat affects chronic lung disease may be related to impairment in breathing patterns meant to compensate for elevations in body temperature [10, 11]. Heat induces cardiovascular disorders through multiple mechanisms including cell damage, inflammation, and blood clotting [12]. For mortality, epidemiological studies have linked even small increases in daily mean or maximum temperatures with increases in premature death. Applying these epidemiological exposure-response relationships to climate model simulations of future temperature, studies have attributed tens of thousands of premature deaths to increasing temperatures in the United States by mid-century [13]. The most vulnerable population subgroups to heat include older adults, children, people working outdoors, and economically disadvantaged communities [7], as well as end stage renal disease patients [14]. While climate adaptation measures can lessen some of the health impacts, climate change-related temperature increases are expected to be an important health risk factor in the U.S. and globally in the future.

Air pollution exposures may also increase with climate change through various pathways, including increased frequency of stagnation events that prohibit atmospheric venting, enhanced photochemical production of secondary pollutants (e.g. tropospheric ozone and some components of fine particulate matter, $PM_{2.5}$), and increasing "natural" gaseous and particulate emissions influenced by warmer and drier conditions (e.g. wildfire smoke, airborne soil dust, and ozone and $PM_{2.5}$ formation from biogenic volatile organic compounds) [2]. As a result, simulations of future air quality under various climate change scenarios indicate a likely "climate penalty" for ozone, making it harder to attain ambient air quality standards even with the same level of anthropogenic emission controls in place [15, 16]. The literature is more mixed for the effects of climate change on $PM_{2.5}$ given the varied and often counteracting effects of climate on $PM_{2.5}$ components and precursor emissions, as well as atmospheric transport and loss. Recent studies suggest a potentially large influence of wildfire smoke and airborne soil dust on $PM_{2.5}$ concentrations [17]. Air pollution exposure can have large implications for human health, particularly heart and lung disease and mortality, through various mechanisms. Exposure to air pollutants, such as $PM_{2.5}$ and ozone, increases oxidative stress leading to pulmonary and systemic inflammation and increased permeability of the lung lining (airway epithelium), increased airway hyperresponsiveness in asthmatics, and decreases in lung function in healthy patients and patients with chronic lung disease [17, 18]. Development and worsening of cardiovascular disease in response to air pollution exposure likely occurs along pathways that include systemic inflammation, alterations in coagulation, dysfunction in the lining of blood vessels (endothelial dysfunction), and progression of atherosclerosis [19]. Following these pathways, air pollution is associated with increased respiratory and cardiovascular mortality. Given the large body of epidemiological literature providing strong evidence for associations between $PM_{2.5}$ and premature mortality from cardiovascular disease, respiratory disease, and lung cancer, and between ozone and respiratory mortality, even small increases in pollution levels in the future can have profound influences on human health outcomes [17, 20].

Climate change is also expected to affect the start, duration, and intensity of the pollen season, with changes differing by region [21]. Climate change and rising greenhouse gas concentrations are correlated with aeroallergens in a number of ways, including increased and faster plant growth, increased pollen production by plants, increased allergenic proteins contained in pollen, earlier start time of plant growth, and longer plant seasons [22]. Meteorological conditions, including precipitation, atmospheric temperature, humidity, and wind speed, can alter the concentrations of plant pollens, which can then influence the occurrence of allergic diseases [23]. Inhalation of pollen grains causes disruption of the immune system within the lungs and increases the susceptibility of individuals to respiratory viral infections [24]. These breakdowns in immune system defenses following exposure to pollen are seen not only in patients with underlying allergies, but also in healthy individuals. In asthmatics, exposure to pollen activates an array of immune cells resulting in bronchoconstriction and increased permeability of airway epithelium [25]. There are few studies that have examined the link between aeroallergen exposure and cardiovascular disease; however, airborne pollen may be a risk factor for myocardial infarction [26]. The mechanism may be related to pollen triggering mast cell activation and histamine release leading to coronary artery spasm or plaque rupture. With the pervasiveness of allergies and allergic asthma among diverse populations throughout the U.S. and the world, climate-related changes in aeroallergen exposure may have widespread impacts on allergic rhinitis and asthma emergency department visits, both of which place a heavy burden on the U.S. healthcare system.

There is substantial literature on respiratory and cardiovascular outcomes related to the isolated exposure to heat, air pollution, or pollen [12, 27–29]. However, fewer studies examine potential synergies or mechanisms behind interactions among these environmental risk factors. There is evidence that air pollutants can bind to pollen grains, precipitating faster release of allergens, increasing allergen absorption in the lungs, and potentiating the allergenicity of pollen, however this is mostly supported in in vitro and animal studies and the clinical significance on a population level is less certain [17, 30, 31]. Prior studies suggest a joint effect of air pollution and heat on health outcomes such as mortality and respiratory morbidity [32]. Many disease states, including heart and lung disease, share a common pathway in which exposure to heat, air pollution, and pollen causes systemic and organ-specific inflammation and cellular damage [9, 17, 28, 33].

Previous studies assessing the potential health impacts of future climate change have considered heat, air pollution, and pollen exposure individually and have not accounted for potential synergistic effects [7, 34–40]. For example, the comprehensive Climate Change Impacts and Risk Analysis project for the U.S. includes estimates of future increases in heat-related mortality, ozone-related mortality, and asthma emergency department visits attributable to aeroallergens, with substantial increases simulated for moderate and severe climate scenarios [4, 41]. Each of these risk factors was considered separately when estimating future health impacts. If there are synergistic effects between these exposures, using single-hazard approaches may underestimate the health impacts of heat, air pollution, and pollen exposures under climate change.

Here, we conduct a systematic literature review of epidemiological studies to determine whether simultaneous exposure to heat, air pollution, and pollen (or a subset of these risk factors) synergistically increases the risk of mortality from any cause and mortality and morbidity of cardiovascular and respiratory disease specifically. We focus on these three risk factors as they share common attributes – they are conditions of the ambient air and have been found to affect respiratory and cardiovascular health. Other risk factors associated with climate change may also affect these health systems, but we consider the body of literature to be too nascent to support a more inclusive systematic review. Results of our review may be useful to more comprehensively characterize future public health disease burdens under climate change scenarios.

Methods

Search strategy, study selection, and data extraction

We conducted a systematic literature review using the Navigation Guide, a methodology for evaluating environmental evidence based on methods used in the clinical sciences [42]. The objective of this systematic review is to assess whether there are interactions between exposure to criteria air pollutants, extreme heat, and pollen, or a subset of these three risk factors, on cardiovascular or respiratory outcomes in human populations. Criteria air pollutants include ground-level ozone (O_3), carbon monoxide (CO), nitrogen dioxide (NO_2), lead, particulate matter (PM), and sulfur dioxide (SO_2).

We define the “Population”, “Exposure”, “Comparator”, and “Outcomes” (PECO) statement as:

- Population: Any human population of any age in any location.
- Exposure: Areas where populations are simultaneously exposed to a) criteria air pollutants and extreme heat; b) criteria air pollutants and pollen; c) pollen and extreme heat; or d) all three risk factors.
- Comparator: Areas where these simultaneous exposures are not occurring.
- Outcome: Cardiovascular and respiratory diseases or mortality.

We searched the databases PubMed, ProQuest, and Scopus with the search terms “air pollution”, “air quality", "air pollutants", "pollen", "aeroallergens", "temperature", "heat", "dust", "NO_2", "SO_2", "particulate matter", "ozone", "multipollutant" for exposures, and the terms "cardiovascular", "respiratory", "mortality", "asthma", and "allergies" for outcomes (Table S1). We conducted a first search on April 22, 2019 and an updated search with more search terms on April 29, 2019. We found additional articles through hand searching the references of fully screened articles.

We included original studies that measured at least two of the exposures (heat, air pollution, and pollen) and at least one of the health outcomes (cardiovascular or respiratory disease or mortality), without limiting by publication date. We excluded studies that were not published in English, did not study a human population, did not measure at least two of the exposures, did not report quantitative results for exposure-response relationships, or did not describe interactions between the exposures. We screened for reference duplicates using Mendeley Desktop. When it was not clear whether studies met the inclusion criteria or not, two reviewers discussed each study and came to a joint decision on inclusion or exclusion.

Data extraction and risk of bias for each included study

Two authors independently extracted data and analyzed risk of bias for each included study. A third author reviewed all studies to resolve discrepancies between the two independent reviewers' risk of bias ratings. We evaluated risk of bias for each of our included studies using the Cochrane Collaboration's "Risk of Bias" tool and the Agency for Healthcare Research and Quality's domains [43]. The domains we evaluated included study design, exposure assessment (air pollution), exposure assessment (temperature), exposure assessment (pollen), detection of outcome, reporting, and conflict of interest. Study design was rated as "low" risk of bias if it was a cohort, case crossover, or time series design. To be rated as "low" risk of bias for air pollution exposure assessment, the study must have measured at least two criteria pollutants and must have measured them in a way that represented individual exposure. To be rated as "low" risk of bias for pollen exposure assessment, the study had to use a method that measured pollen exposure at an individual level. To be rated as "low" risk of bias for temperature, studies had to use data from meteorological surveillance networks; we did not judge a lack of individual exposure measurement to introduce high risk of bias for temperature since temperature is less spatially heterogeneous compared with air pollution. To be rated as "low" risk of bias for detection of health outcome, the study had to use the *International Classification of Diseases* (ICD) to classify the health outcome category. To be rated as "low" risk of bias for reporting, the study had to report all outcomes that were assessed. To be rated as "low" risk of bias for conflict of interest, the study had to acknowledge that there was no conflict of interest. The possible ratings for the studies for each domain were "low", "probably low", "probably high", or "high" risk of bias. We used the "probably low" and "probably high" categories when not enough information was given to definitively assign "low" and "high" ratings.

Quality and strength of evidence across studies

To evaluate the quality and strength of evidence across all studies, we used the Grading of Recommendations Assessment, Development and Evaluation (GRADE) systematic review approach [44]. We stratified papers by the following categories of multiple exposures: 1) heat, air pollution, and pollen; 2) heat and air pollution; 3) air pollution and pollen; and 4) heat and pollen.

To evaluate the quality of the evidence across all studies, we upgraded and downgraded studies according to several criteria. Downgrading factors included serious risk of bias, serious indirectness in the studies such that evidence is not directly comparable to our PECO statement criteria, serious inconsistency in effect estimates across studies, serious imprecision due to small sample size and/or small outcome count, and likely publication bias resulting in an over or underestimate of true effects from exposure. Downgrading for serious risk of bias by − 1 occurred if there were instances of an unclear limitation in the evidence and by − 2 if there were instances of serious limitations or very serious limitations during the assessments. Downgrading for inconsistency by − 1 occurred if there were minimal or no overlap of confidence intervals and by − 2 if there was wide variance of point estimates across studies. Downgrading for indirectness by − 1 was applied if there were large differences in study population and by − 2 if there were large differences and if surrogate outcomes were applied. Downgrading for imprecision by − 1 occurred if there was a small sample size or small outcome count and by − 2 if there was both.

Upgrading factors included large magnitude of effect such that confounding alone could not explain the association, consistent dose-response gradient across studies, all plausible confounding would reduce a demonstrated effect, and all possible confounding would suggest a spurious effect when the actual results show no effect. After considering the upgrading and downgrading factors, the studies were then given a rating of "low quality", "moderate quality", or "high quality." Possible ratings were 0, meaning no change from initial quality rating, − 1 or − 2, meaning downgrades in quality rating, and + 1 and + 2, meaning upgrades in quality rating. Upgrading for large magnitude of effect by + 1 occurred with the effect estimate was large such as a relative risk of 2 or higher and by + 2 if there was a very large effect estimate such as a relative risk of 5 or higher. Upgrading for dose-response by + 1 was applied if there was observation that there was a dose response gradient between increased exposure and increased outcomes and by + 2 if there was a rapid and large absolute increase in outcomes as dose increased. Upgrading for effect of plausible confounding by + 1 was applied if the plausible confounders were adjusted for in the analysis.

We evaluated the strength of evidence across all studies based on quality of the evidence, direction of effect estimates, confidence in effect estimates, and other attributes [45]. To the extent possible, we discuss these ratings according to categories of health outcomes (e.g. all-cause mortality, cardiovascular disease, and respiratory disease). The ratings for strength of the evidence are: "evidence of lack of association" (studies show no adverse effect), "inadequate evidence" (studies permit no conclusion about an effect), "limited evidence" (studies suggest an effect but only in a single or limited number of studies), and "sufficient evidence" (studies indicate a causal relationship between exposure and effect). We followed the more detailed definitions of each strength rating given by Johnson et al. [46].

Results

Our search retrieved 1730 unique records, and we added 16 papers identified through other sources (Fig. 1). We screened 605 papers after removing duplicates and assessed the full text of 406 articles for eligibility. We excluded 350 articles because they did not describe interactions between the exposures or did not describe the outcome measures. Ultimately, we included 56 studies that met our eligibility criteria. Table 1 includes descriptions of each study.

Of these 56 studies, six measured air pollution, heat, and pollen; 39 measured air pollution and heat; 10 measured air pollution and pollen; and one measured heat and pollen. Forty-six studies were a time series design, three were cohort studies, one was a cross sectional design, one was a nested case control design, and five were a case-crossover design. Data collection in these studies ranged from 1987 to 2010 and publication date ranged from 2002 to 2018. The qualifying studies ranged widely

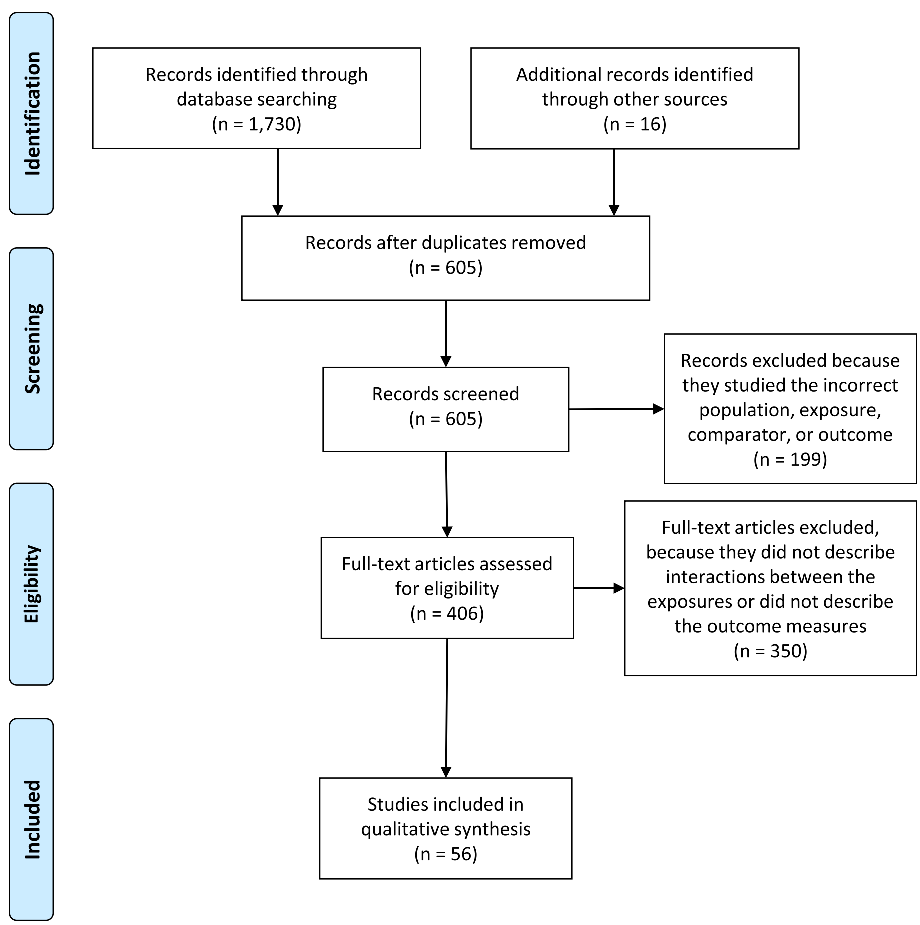

Fig. 1 PRISMA Diagram showing the number of studies included and excluded at each step

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
Air pollution, heat, and pollen ($n = 6$)								
Respiratory								
Hebbern 2015 [47]	Time series	10 Canadian cities	Apr 1994- Mar 2007	Asthma hospital admissions	Not reported	CO, O_3, NO_2, SO_2, PM_{10}, $PM_{2.5}$	Weed, tree, grass	Daily Mean
Makra 2015 [48]	Time series	Szeged, Hungary	1999–2007	Asthma emergency room visits	0–14 years; 15–64 years; 65+ years ($n = 936$ asthma ER visits)	CO, NO, NO_2, SO_2, O_3, PM_{10}	Ambrosia, maple, alder, mugwort, birch, hemp, hornbeam, goosefoot, hazel, ash, walnut, mulberry, pine, plantain, platan, grasses, poplar, oak, dock, willow, yew, linden, elm, nettle	Daily Mean, daily maximum, daily minimum, daily range
							Matyasovszky 2011 [49]	Time series
Szeged, Hungary	1999–2007	Respiratory hospital admissions	All ages; 15–64 years; 65+ years ($n = 133,464$ hospital admission)	CO, NO, NO_2, SO_2, O_3, PM_{10}	Ambrosia, maple, alder, mugwort, birch, hemp, hornbeam, goosefoot, hazel, ash, walnut, mulberry, pine, plantain, platan, grasses, poplar, oak, dock, willow, yew, linden, elm, nettle	Daily mean, maximum, minimum, range		
Mazenq 2017 [50]	Nested case control	Southeastern France	Jan 2013-Dec 2013	Asthma emergency room visits	3–18 years ($n =$ 1182 asthma ER visits)	PM_{10}, $PM_{2.5}$	cypress, birch, ash, grass, urticaceae	Daily average
Mireku 2009 [51]	Retrospective time series	Detroit, MI	Jan 2004- Dec 2005	Asthma emergency room visits	1–18 years ($n =$ 25,401 asthma ER visits)	$PM_{2.5}$, PM_{10}, SO_2, O_3	Total	Daily average
Witonsky 2019 [52]	Retrospective cohort	Bronx, NY	Jan 2001- Dec 2008	Asthma emergency room visits and hospitalizations	All ages ($n = 42$, 065 asthma ER visits; $n = 1664$ asthma-related hospitalizations)	NO_x, O_3, SO_2	Grass, weed, tree,	Daily average
Air pollution and temperature ($n = 39$)								
Multiple health endpoints								
Analitis 2014 [53]	Ecological time series	9 European cities	1990–2004	All natural, cardiovascular, and respiratory mortality	0–64, 65–74, 75–84, and 85+ years (n not reported)	SO_2, PM_{10}, NO_2, O_3, CO		3-h average
Analitis 2018 [54]	Ecological time series	9 European cities	2004–2010	All natural, cardiovascular, and respiratory mortality	All ages; 15–64, 65–74, 75+ years (n not reported)	PM_{10}, O_3, NO_2		Daily mean
Breitner 2014 [55]	Time series	Bavaria, Germany	1990–2006	Non accidental, cardiovascular,	< 85, 85+ years ($n = 338,631$	PM_{10}, O_3		Daily mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
				respiratory mortality	deaths)			
Cheng 2012 [56]	Time series	Shanghai, China	2001–2004	Non-accidental, cardiovascular, respiratory mortality	All ages (n = 173, 911 deaths)	PM_{10}, O_3, SO_2, NO_2		Daily minimum, maximum, mean
Li 2011 [57]	Time Series	Tianjin, China	2007–2009	Cardiovascular, respiratory, cardiopulmonary, stroke and IDH, Non accidental mortality	All ages; < 65, 65+ years (n = 111,087 deaths)	PM_{10}, SO_2, NO_2		Daily mean
Li 2015 [58]	Time Series	Guangzhou, China	2003–2011	Non accidental mortality, cardiovascular mortality, respiratory mortality	< 65, 65+ years (n = 213,737 deaths)	PM_{10}		Daily mean
Lokys 2018 [59]	Time series	28 districts, Germany	2001–2011	Cardiovascular and respiratory hospital admissions	Not reported	NO_2, SO_2, O_3, PM_{10}		Daily mean
All-cause or non-accidental only								
Burkart 2013 [60]	Time Series	Berlin and Lisbon	1998–2010	All cause mortality	Age not reported (n = 698,586 deaths)	PM_{10}, O_3		Hourly mean
Chen 2018a [61]	Time series	8 European cities	1999–2013	Non accidental mortality	0–74, 75+ years (n = 742,526 deaths)	$PM_{2.5}$, PM_{10}, O_3		Daily mean
Chen 2018b [62]	Time Series	8 European cities; 86 US Cities	1999–2013; 1987–2000	Non accidental mortality	All ages (n not reported)	PM_{10}, NO_2, O_3		Daily mean
Dear 2005 [63]	Time series	12 French cities	Aug-03	All cause mortality	All ages (n not reported)	O_3		24 h Minimum, maximum
Filleul 2006 [64]	Time series	9 French cities	Aug-03	All cause mortality	All ages (n not reported)	O_3		Daily maximum
Jhun 2014 [65]	Time series	97 cities	1987–2000	Non accidental mortality	0–99 years (n not reported)	O_3		Daily high
Kim 2015 [66]	Time series	7 South Korean cities	Jan 2000-Dec 2009	Daily non accidental deaths	< 65, 65+ years (n = 828,787 deaths)	PM_{10}		Daily mean
Liu 2016 [67]	Time Series	20 US communities	1987–2000	Non accidental mortality	Not reported	O_3		Daily mean
Meng 2012 [68]	Time series	8 Chinese cities	2001–2008	Non accidental mortality	Not reported	PM_{10}		Daily mean
Moolgavkar 2003 [69]	Time Series	Cook County, IL & LA County, CA	1987–1995	Non accidental mortality	All ages; 65+ years (n not reported)	O_3, SO_2, NO_2, CO, PM		Daily minimum, median, maximum

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
Park 2011 [70]	Time series	Seoul, South Korea	Jun 1999- Dec 2007	Non accidental mortality	All ages; 65–74, 75–84, 85+ years (n = 291,665 deaths)	PM_{10}, NO_2, SO_2, CO, O_3		Daily mean, minimum, maximum
Pattenden 2010 [71]	Time series	15 conurbations in England and Wales	1993–2003	All cause mortality	0–64, 65–74, 75–84, 85+ years (n not reported)	O_3, PM_{10}		Two day Mean
Peng 2013 [72]	Time series	23 European Cities; 12 Canadian Cities; 86 US cities	Canada 1987–1996; Europe 1990–1997; US 1987–1996	Non accidental mortality	All ages; < 75, 75+ years (n not reported)	NO_2, SO_2, O_3, PM_{10}		Daily mean
Rainham 2005 [73]	Time series	Toronto, Canada	1981–1999	Non Trauma mortality	Not reported	CO, NO_2, SO_2, O_3, $PM_{2.5}$		Daily mean
Scortichini 2018 [74]	Time series	25 Italian cities	2006–2010	Mortality from natural causes	35+ years (n = 187,743 deaths)	O_3, PM_{10}		Daily mean
							Shaposhnikov 2014 [75]	Time series
Moscow, Russia	2006–2009, 2010	Non accidental mortality	All ages; < 65, 65+ years (n = 10, 860 deaths)	O_3, PM_{10}		Daily mean		
Stafoggia 2008 [76]	Case crossover	9 Italian cities	1997–2004	Mortality from natural causes	35+ years (n = 321,024 deaths)	PM_{10}		Daily mean, apparent
Sun 2015 [77]	Time Series	Hong Kong	1999–2011	Mortality from natural causes	Age not reported (n = 456,317 deaths)	$PM_{2.5}$, NO_2, SO_2, O_3		Daily mean
Vanos 2015 [78]	Time series	12 Canadian cities	1981–2008	Non accidental mortality	Not reported	O_3, NO_2, $PM_{2.5}$, SO_2		Daily mean
Wilson 2014 [79]	Time Series	95 US cities	1987–2000	Mortality	Not reported	O_3		Daily mean
Zhang 2006 [80]	Time series	Shanghai, China	Jan 2001- Dec 2004	Non accidental mortality	All ages; 0–4, 5–44, 45–64, 65+ years (n = 173,911 deaths)	O_3, PM_{10}, SO_2, NO_2		Daily mean
Respiratory only								
Ding 2017 [81]	Case crossover	Taiwan	2000–2013	COPD mortality	40–64, 65–79, 80+ years (n not reported)	$PM_{2.5}$, O_3, SO_2		Daily mean, maximum, minimum
Jo 2017 [82]	Time series	Busan, South Korea	2007–2010	Hospital admissions for respiratory disease	0–15,16–64, 65+ years (n not reported)	$PM_{2.5}$, PM_{10}		Daily average, minimum, maximum, range
Kunikullaya 2017 [83]	Retrospective ecological time series	Bangalore, India	One year	Asthma-related emergency room visits and hospitalizations	> 18 years (n not reported)	SO_2, NO_2, PM_{10}, $PM_{2.5}$		Daily mean
Lam 2016 [84]	Time series	Hong Kong	2004–2011	Asthma hospitalizations	< 5, 5–14, 15–59, 60+ years (n = 56,	PM_{10}, SO_2, NO_2, O_3		Daily mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
					112 asthma admission)			
Mirabelli 2016 [85]	Retrospective cross sectional	United States	2006–2010	Asthma symptoms	18+ years ($n = 50$, 356 respondents)	$PM_{2.5}$, O_3		Average daily mean
Qiu 2018 [86]	Time series	Chengdu, China	Jan 2015- Dec 2016	COPD hospital admissions	All ages; < 60, 60–70, 70–80, 80+ years ($n = 54$, 966 COPD admission)	PM_{10}, $PM_{2.5}$, NO_2, SO_2, CO, O_3		Daily mean
Winquist 2014 [87]	Time Series	Atlanta, GA	16 years	Asthma emergency department visits	5–17 years (n not reported)	CO, NO_2, SO_2, O_3, $PM_{2.5}$		Daily minimum, maximum,
Cardiovascular only								
Lee 2018 [88]	Case crossover	Seoul, South Korea	2008–2014	Migraine emergency room visits	All ages; < 40, 40–64, 65+ years ($n = 18{,}921$ ER visits)	$PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, CO		Hourly mean
Luo 2017 [89]	Time series	3 Chinese cities	2008–2011	Cardiovascular mortality	All ages; < 65, 65+ years ($n =$ 290,593 deaths)	PM_{10}, NO_2, SO_2		Daily minimum, maximum, mean
Ren 2008 [90]	Time Series	95 US cities	1987–2000	Cardiovascular mortality	< 65, 65–74, 75+ years ($n =$ nearly 4 million cardiovascular deaths)	O_3		Daily maximum
Ren 2009 [91]	Time series	95 US cities	1987–2000	Cardiovascular mortality	< 65, 65–74, 74+ years (n= > 4.3 million cardiovascular deaths)	O_3		Daily maximum
Air pollution and pollen ($n = 10$)								
Respiratory								
Anderson 1998 [92]	Time series	London	Apr 1987- Feb 1992	Asthma emergency admissions	All ages; 0–14, 15–64, 65+ years (n not reported)	O_3, NO_2, Black smoke, SO_2	Birch, Grass, Oak	Mean 24 h
Cakmak 2012 [93]	Time series	11 Canadian cities	Apr 1994-Mar 2007	Asthma hospital admissions	Not reported	CO, $PM_{2.5}$, PM_{10}, NO_2, SO_2	Tree, Weed	Mean 24 h
Chen 2016 [94]	Time-series case-crossover	Adelaide, South Australia	Jul 2003- Jun 2013	Asthma hospital admissions	0–17, 18+ years ($n = 36$, 024admissions)	$PM_{2.5}$, NO_2, PM_{10}	Ash tree, birch, cypress, eucalyptus, fruit tree, olive tree, pinus, plane tree, she-oak, wattle, chenopodia-ceae, compositae, plantain, polygonaceae, salvation jane, grass	Daily average
Cirera 2012 [95]	Time series	Cartagena, Spain	Jan 1995- Dec 1998	COPD and asthma emergency room	Age not reported ($n = 1617$ asthma and 2322 COPD	SO_2, NO_2, TSP, O_3	Poaceae, Urticaceae	Hourly mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
				visits,	ER visits)			
Galan 2003 [96]	Time series	Madrid, Spain	1995–1998	Asthma emergency department visits	Age not reported ($n = 4827$ asthma attacks)	SO_2, PM_{10}, NO_2, O_3, CO	*Olea europaea*, Plantago sp., Poaceae, Urticaceae	Daily mean
Gleason 2014 [97]	Time-stratified case-crossover	New Jersey	April - Sept 2004–2007	Asthma emergency department visits	3–17 years ($n =$ 21,854 asthma ED visits)	O_3, PM	Tree, grass, weed, ragweed	Daily mean
Goodman 2017 [98]	Time series	New York City	1999–2009	Asthma hospital Admissions	< 6, 6–18, 19–49, 50+ years ($n =$ 295,497 asthma admission)	O_3, PM	Tree, weed, total	Daily average, maximum, minimum
Krmpotic 2011 [99]	Time series	Zagreb, Croatia	Jan 2004- Dec 2006	Asthma hospital admissions	> 18 years ($n =$ 4125 asthma admissions)	NO_2, CO, PM_{10}	Alder, Hazel, Birch, Hornbeam, Oak, Grasses, Ragweed	Daily minimum, maximum, mean
Ross 2002 [100]	Prospective Cohort	East Moline, IL	7 months	Peak Expiratory flow rates, respiratory symptoms, frequency of asthma attacks, asthma medication use	5–49 years ($n =$ 59 people)	O_3, PM, SO_2	Grass, Ragweed, Total	Daily mean, Maximum
Cardiovascular								
Stieb 2000 [101]	Time series	Saint John, Canada	Jul 1992- Jun 1994, Jul 1994- Mar 1996	Cardiorespiratory emergency department visits	Age not reported ($n = 19{,}821$)	CO, H_2S, NO_2, O_3, SO_2, TRS	Ascomycetes, basidiomycetes, deuteromycetes, ferns, grass, tree, weed	Daily average
Heat and pollen ($n = 1$)								
Silverberg 2015 [102]	Cohort Study	United States	2006	Pediatric hay fever	0–17 years ($n =$ 91,642)	–	Total	Monthly mean

Study	Study design	Exposure assessment (air pollution)	Exposure assessment (temperature)	Exposure assessment (pollen)	Detection of outcome	Reporting	Conflict of Interest
Air pollution, heat, and pollen							
Hebbern 2015	1	2	1	2	1	1	1
Makra 2015	1	3	2	3	1	1	2
Matyasovszky 2011	1	3	2	3	2	1	2
Mazenq 2017	1	2	1	3	1	1	1
Mireku 2009	1	3	1	3	2	1	2
Witonsky 2019	1	3	1	3	1	1	1
Air polution and heat							
Analitis 2014	1	2	2	n/a	1	1	2
Analitis 2018	1	2	2	n/a	1	1	1
Breitner 2014	1	3	1	n/a	1	1	1
Burkart 2013	1	3	1	n/a	2	1	2
Chen 2018a	1	2	2	n/a	1	1	1
Chen 2018b	1	2	2	n/a	1	1	1
Cheng 2012	1	2	1	n/a	1	1	1
Dear 2005	1	3	2	n/a	2	1	2
Ding 2017	1	2	1	n/a	1	1	1
Filleul 2006	1	2	1	n/a	2	1	1
Jhun 2014	1	3	1	n/a	2	1	2
Jo 2017	1	2	2	n/a	1	1	1
Kim 2015	1	3	1	n/a	1	1	1
Kunikullaya 2017	1	3	2	n/a	1	1	1
Lam 2016	1	2	1	n/a	1	1	1
Lee 2018	1	2	1	n/a	1	1	1
Li 2011	1	2	2	n/a	1	1	2
Li 2015	1	3	1	n/a	1	1	1
Liu 2016	1	3	1	n/a	1	1	1
Lokys 2018	2	3	2	n/a	1	1	2
Luo 2017	1	2	1	n/a	1	1	1
Meng 2012	1	2	2	n/a	1	1	1
Mirabelli 2016	2	3	3	n/a	3	1	1
Moolgavkar 2003	1	3	2	n/a	1	1	2
Park 2011	1	2	1	n/a	1	1	1
Pattenden 2010	1	3	2	n/a	1	1	1
Peng 2013	1	2	2	n/a	2	1	1
Qiu 2018	1	2	2	n/a	1	1	1
Rainham 2005	1	2	2	n/a	1	1	2
Ren 2008	1	3	2	n/a	1	1	1
Ren 2009	1	3	2	n/a	1	1	2
Scortichini 2018	1	2	2	n/a	1	1	1
Shaposhnikov 2014	1	2	1	n/a	1	1	2
Stafoggia 2008	1	3	2	n/a	1	1	1
Sun 2015	1	2	1	n/a	1	1	1
Vanos 2015	1	2	1	n/a	1	1	2
Wilson 2014	1	3	1	n/a	2	1	2
Winquist 2014	1	2	3	n/a	1	1	2
Zhang 2006	1	2	1	n/a	1	1	1
Air pollution and pollen							
Anderson 1998	1	3	n/a	3	1	1	2
Cakmak 2012	1	2	n/a	2	1	1	1
Chen 2016	1	4	n/a	2	1	1	1
Cirera 2012	1	3	n/a	3	2	1	1
Galan 2003	1	3	n/a	2	1	1	2
Gleason 2014	1	2	n/a	3	1	1	1
Goodman 2017	1	2	n/a	4	1	1	2
Krmpotic 2011	1	3	n/a	3	1	1	2
Ross 2002	1	2	n/a	2	3	1	2
Stieb 2000	1	4	n/a	3	2	1	2
Heat and pollen							
Silverberg 2015	1	n/a	2	4	2	1	1

Legend

1 Low risk of bias
2 Probably low risk of bias
3 Probably high risk of bias
4 High risk of bias

Fig. 2 Final risk of bias evaluation for each study in air pollutants and pollen types measured, metrics used for each exposure type (e.g. averaging times, time lags), and health outcomes (including asthma and hay fever symptoms, cardiovascular and respiratory emergency department visits and hospitalizations, cause-specific mortality, and all-cause mortality).

Risk of bias determinations and rationale for each study can be found in Tables S2 through S57. Almost all of the studies were rated as "low" or "probably low" risk of bias for study design, detection of outcome, reporting, and conflict of interest (Fig. 2). Risk of bias for exposure assessment varied across the studies. For air pollution and pollen, we rated many studies as having a "probably high" risk due to a lack of exposure measurement at an individual level, as they used exposure assessment techniques such as central site monitors that are broadly representative of regional air pollution levels but may not represent individual exposure well. Several of these studies only used one central site monitor, which we judged could potentially introduce bias since pollution levels vary spatially within geographic areas such as cities. For temperature, studies were generally rated as having a "low" or "probably low" risk of bias since data were sourced from meteorological monitoring networks and temperature is less spatially heterogeneous compared with air pollution.

We next assessed the quality and strength of the evidence across the studies. We found six studies that examined potential interactive effects between simultaneous exposure to all three risk factors: air pollutants, pollen, and heat (Table 1). The studies were conducted in Canada, France, Hungary, and the U.S. and all focused on respiratory hospitalizations and emergency department visits (all except one focused specifically on asthma). The studies used widely different methods for categorizing temperature exposure, including spatial synoptic classification [47, 48], seasonal analysis [52], and interday temperature change [51]. Generally, the studies were individually rated as low risk of bias for most categories, including study design, detection of outcome, reporting, and conflict of interest. However, we judged some to be at probably high risk of bias for exposure assessment for both air pollutants and pollen. The findings across the studies were inconsistent, with some studies reporting interactive effects of all three or some combination of the exposures [47–49, 52], while others reported independent effects that were unaffected by controlling for the other risk factors [51] or were inconclusive when considering simultaneous exposure to all three risk factors [50].

Overall, we rated the quality of the evidence for synergistic respiratory effects between air pollution, heat, and pollen as "low" since studies were inconsistent in finding significant evidence of interactive effects and studies that reported positive associations of interactions had minimal magnitudes (Table 2). We rated the overall strength of the evidence as "limited" since synergistic effects between heat, air pollution, and pollen were observed in some studies, but these findings were not consistent across studies.

We found 39 articles that examined potential interactive effects between exposure to air pollutants and heat (Table 1). These studies were carried out in Europe, the U.S., Canada, Russia, Taiwan, South Korea, India, Hong Kong, and China. Most were conducted in urban areas. A majority of the studies (29) included health endpoints that were not disease-specific, such as all-cause and non-accidental mortality. A smaller subset of 12 studies considered respiratory disease specifically (some focusing on asthma specifically) and 11 considered cardiovascular disease specifically (we have included migraine in this category as a potential indicator of cardiovascular disease, Adelborg et al. [103]). Most studies included multiple criteria pollutants – most often ozone and PM_{10}, though some only included ozone, and some also included $PM_{2.5}$, $PM_{2.5-10}$, NO_2, SO_2, and CO. The temperature metric differed between studies and included daily mean, minimum and/or maximum.

Of these 39 studies addressing synergistic effects between air pollution and heat, 19 reported interactive effects between heat and air pollution exposure on health outcomes studied. Out of these studies, 15 of 29 studies examined health outcomes that were not disease-specific (e.g. all-cause mortality, hospital admissions) and found synergistic effects [53–55, 57, 58, 60, 61, 66, 68, 71, 73–77], four of 12 studies found synergistic effects for respiratory health outcomes [55, 57, 59, 84], and eight of 11 studies found synergistic effects for cardiovascular health outcomes [54, 55, 57–59, 88, 90, 91]. Here, we are not distinguishing between mortality and morbidity for respiratory and cardiovascular health outcomes. Generally, the studies found synergistic effects from simultaneous exposure to extremely high temperatures and air pollution, with a potentially additional role of relative humidity. A method of weather classification that incorporated humidity used in some of the papers was spatial synoptic classification (SSC), which is described as a "semi-automated statistical approach designed to classify complex daily weather conditions into one of six distinct categories, or a transitional category" and uses values of temperature, dew point, u and v components of wind, cloud cover, and sea level pressure [47, 48, 73, 78]. A strength of this group of studies was the large datasets of pollutant levels and meteorology, including from the National, Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the United States [61, 65, 67, 90, 91] and the Ultrafine Particles and Health Study Group in Europe [61, 62]. Compared with the other categories in our review, air pollution and heat studies covered the

Table 2 Rating of the quality and strength of the evidence for studies assessing interactive effects between heat, air pollution, and pollen ($n = 6$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = "Moderate"			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded because of "probably high" risk of bias for air pollution exposure assessment for four studies and for pollen exposure assessment for five studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in populations for the duration of study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	Some evidence of consistent effects, but the studies were too varied in definitions of risk factors and methods to judge consistency in effect estimates.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies.	0	Studies did not report a consistent relationship between dose and response.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.
Overall Quality of Evidence		Low	The overall quality of the evidence supporting interactive effects is low.
Overall Strength of Evidence		Limited	An association was sometimes observed for synergy between heat, air pollution, and pollen, but the potentially high risk of bias for air pollution exposure could have impacted results and there is a lack of consistently significant findings.

broadest geographic area and included the largest number of people in the studies.

The evidence was strongest for synergistic effects between heat and exposure to either ozone and $PM_{2.5}$. For ozone, 11 of 29 studies reported synergistic effects with heat [53–55, 60, 61, 71, 73, 74, 84, 90, 91]. These effects were found among inter quartile temperature analysis, seasonal analysis, and heatwave analysis in the studies. Effects were found for all-cause mortality, non-accidental mortality, cardiovascular mortality, and morbidity outcomes. High levels of ozone and high temperatures tended to be reported together and the strongest effects on outcomes were found at the highest exposures. We also found evidence for synergistic effects between heat and particulate matter, with 10 of 27 studies reporting synergistic effects [53, 54, 60, 61, 66, 73–76, 88]. These effects were found among inter quartile temperature analysis, seasonal analysis, and heatwave analysis in the studies. Effects were found for all-cause mortality, non-accidental mortality, and morbidity outcomes. A potential interactive effect between heat and particulate matter is further supported by Mazenq et al. [50], who found that temperature and particulate matter were linked but pollen was not.

While most studies assessing synergistic effects between air pollution and temperature focused on heat, several examined effects of cold [55, 56, 58–62, 67, 70, 73, 77, 79, 80, 83, 84, 86–88]. Generally, stronger results were found in warmer seasons when compared to cold seasons. Zhang et al. [80] was the only study in our review that found that synergy between ozone and the cold season was stronger than for the warm season.

We upgraded the overall quality of the evidence of synergistic effects between air pollution and heat because of the relatively consistent finding of significant exposure-response relationships showing interactive effects (Table 3). The consistent findings of interactive effects between air pollutants and heat held for all three

Table 3 Rating of the quality and strength of the evidence for studies assessing interactive effects between heat and air pollution ($n = 39$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = "Moderate"			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded due to "probably high" risk of bias for air pollution exposure assessment for 16 studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in the United States for the duration of the study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	There was not a wide variability in estimates of effects.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies	1	Exposure-response relationship was directionally consistent across 15 of the 34 studies in the category.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect
Overall Quality of Evidence		Moderate	The dose response relationships described in a number of studies did not warrant an upgrade for the overall quality rating.
Overall Strength of Evidence		Sufficient	An association was generally observed for synergistic effects of heat and air pollution exposure, specifically for ozone and PM, but the potentially high risk of bias from the air pollution exposure assessment methods in several studies could have impacted results.

health outcome categories considered: health outcomes that were not disease-specific (e.g. all-cause mortality), respiratory disease, and cardiovascular disease, though more studies found interactive effects for non-cause-specific endpoints and for cardiovascular disease than for respiratory disease. This result may highlight the need for more studies focusing not only on respiratory disease, but also on other diseases. These factors led us to rate the overall quality of the evidence as "Moderate" and the overall strength of the evidence as "Sufficient."

We found 10 studies that assessed potential interactive effects between exposure to air pollution and pollen (Table 1). These studies were conducted in Europe, Canada, Australia, and the U.S. Studies included a variety of pollen types and air pollutants, with little consistency between them. Health outcomes considered were all respiratory morbidity (mostly hospital admissions and emergency department visits), with the exception of one that focused on cardiopulmonary emergency department visits [101].

The studies in this category were inconsistent in their study designs and findings. For example, Anderson et al. [92] concluded that there was no evidence for synergy between air pollutants and pollen, with the exception of SO_2 and grass pollen in children during the warm season. Chen et al. [94] also found little evidence of interactions between air pollutants and pollen but did find that several of the air pollution and pollen exposures were stronger in the cool season than in the warm season. In contrast, Goodman et al. [98] found that, in most populations, adjusting for outdoor pollen generally attenuated relative risk of hospital admissions for both ozone and $PM_{2.5}$. Ross et al. [100] found the association between ozone and asthma medication use was increased after adjusting for aeroallergens. Cakmak et al. [93] found that there were synergistic effects on asthma hospitalization between tree pollen and increasing $PM_{2.5}$, and between weed pollen and PM_{10}.

Given that the 10 studies included inconsistent pollen types and air pollutants, with inconsistent results, we were unable to draw strong conclusions for this category. Overall, we rated the quality of the evidence as “Low” and the strength of the evidence as “Limited.” We did not upgrade the quality of the evidence since the studies reported inconsistent findings, and since studies that did find synergistic effects reported effect estimates that had low magnitudes (Table 4).

Our search only found one study that examined interactions between heat and pollen [102]. This study explored climate factors and pollen count impacts on pediatric hay fever prevalence among 91,642 children across the U.S. Hay fever prevalence was shown to increase with the second, third, and fourth quartile mean annual temperature and mean total pollen counts. This study was particularly strong given the large size and national representation of the included population. However, with only one study, we did not draw conclusions regarding the quality and strength of evidence for interactive effects between heat and pollen.

Discussion

We conducted a systematic literature review of human population health studies to examine the evidence for synergistic effects from simultaneous exposure to air pollution, pollen, and heat, or a subset of these three risk factors. We found limited evidence for synergistic respiratory effects of air pollution, pollen, and heat; sufficient evidence for synergistic all-cause mortality, cardiovascular, and respiratory effects of air pollution and heat (particularly for ozone and particulate matter); and limited evidence for synergistic respiratory effects of air pollution and pollen. We were unable to assess evidence for pollen and heat because only one paper came up in our searches.

Overall, there was a substantially larger body of literature examining interactive effects between air pollution and heat, compared with those that included pollen as an exposure of interest. The evidence for interactive effects between air pollution and heat is further strengthened by

Table 4 Rating of the quality and strength of the evidence for studies assessing interactive effects between air pollution and pollen ($n = 10$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = “Moderate”			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded because of “high” or “probably high” risk of bias for air pollution exposure assessment for six studies and “high” or “probably high” risk of bias for pollen exposure assessment for six studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in the populations for the duration of study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	The studies were inconsistent in pollen types and air pollutants, precluding judgment as to whether reported effect estimates would be consistent or inconsistent.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies	0	Studies did not report a consistent relationship between dose and response.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect
Overall Quality of Evidence		Low	The overall quality of the evidence supporting interactive effects is low.
Overall Strength of Evidence		Limited	An association was shown in a few studies between air pollution and pollen and increased outcomes, however the results were inconsistent and there was a potentially high risk of bias from the exposure assessments in several studies.

large datasets of pollutant levels and meteorological data, including from the National, Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the U.S. and the Ultrafine Particles and Health Study Group in Europe. An additional strength across all categories was that a majority of the studies had a low risk of bias for study design, with many of them using a time series design.

Though there were some strengths in the literature, we also found serious weaknesses that precluded our ability to draw strong conclusions as to the existence of interactive health effects from simultaneous exposure to these risk factors. Limitations included that all of the studies we found were short-term studies that were unable to address effects of long-term exposure. We found no cohort studies that could properly attribute exposure at an individual level and account for health outcomes that may take years to manifest. In addition, exposure measurements and metrics for air pollutants, pollen, and temperature were inconsistent and not standardized between the studies. Judging the potential bias from exposure measurement for air pollution, temperature, and pollen is difficult with only limited information available in the papers. For example, some papers did not report the number of monitoring stations used to assign exposures or the length of time for which the exposure data were collected. Recent studies of air pollution have begun using more sophisticated methods to assign exposure, such as models that use satellite remote sensing or land use variables that provide greater spatial coverage compared with ground monitors such as those run by government monitoring networks [104–106]. For pollen, the studies in this review all used pollen count as the exposure metric, which may not account for pollen potency [23]. Another limitation is that many studies were missing information about confounders that were considered, which could influence the magnitude of the associations they found. Finally, while we restricted our review to studies that looked at interaction between two of the three hazards, several studies may have treated these risk factors as mediators or effect modifiers. Future research should explore the role of these issues. Additional research should also explore effects of these risk factors on additional health outcomes, such as birth outcomes, as well as vulnerable populations, including children, the elderly, pregnant women, and people with genetic predisposition to cardiovascular and respiratory disease.

We included only heat, air pollution, and pollen in this review, as they are all conditions of the ambient air for which we judged there to be enough epidemiological literature to assess. Other important environmental drivers of disease related to the ambient air that we did not include here are occupational exposures; different types of air pollutant mixtures (including from different combustion sources and different composition of particulate matter); and exposure to airborne bacteria, viruses, molds, and fungus. In reality, people are exposed to a complex set of risk factors that remain poorly defined and explored in the literature. In addition, the chronic diseases considered affected by these risk factors are multi-factorial with heavy influence from genetic and lifestyle (e.g. diet, exercise) factors. Our literature review highlights the importance of including environmental factors in epidemiological and risk assessment studies, even if strong conclusions cannot yet be drawn from the current set of available studies.

Conclusions

In this systematic literature review of epidemiological studies, we found evidence for synergistic effects of heat and air pollutants (particularly for ozone and particulate matter), but not for the combination of heat, air pollution, and pollen together or of air pollution and pollen or heat and pollen. Our findings support consideration of combined effects of heat and air pollution in assessing health impacts from these risk factors in the present day and in the future as climate change progresses. However, the literature is too nascent to support inclusion of interactive effects between air pollution and pollen or heat and pollen in risk assessments. Future research should continue to explore potential interactive effects of environmental exposures on human health, as people are often exposed to multiple environmental risk factors simultaneously. This is a rapidly evolving field of study, and our review and conclusions should be updated to include new evidence as it becomes available. If new evidence supports our conclusion that heat and air pollution exposure act synergistically on human health, the health impacts from climate change-driven increases in air pollution and heat exposure may be larger than previously estimated in studies that consider these risk factors individually.

Supplementary Information

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Additional file 1.

Abbreviations

CO: Carbon monoxide; GRADE: Grading of Recommendations Assessment, Development and Evaluation; NO2: Nitrogen dioxide; O3: Ozone; PECO: Population, Exposure, Control, Outcome; PM2.5: Fine particulate matter; PM10: Coarse particulate matter; SO2: Sulfur dioxide

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Authors' contributions

S.C.A. conceived of the study, oversaw the analysis, and was responsible for drafting the manuscript. S.H. conducted the literature review, evaluated risk of bias and strength and quality of the evidence, and wrote much of the manuscript. E.W. evaluated risk of bias. N.N. and P.K. reviewed the analysis and contributed to the manuscript writing. The author(s) read and approved the final manuscript.

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Availability of data and materials
All data are available within the article and supplemental material.

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Not applicable.

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Competing interests
The authors declare that they have no competing interests.

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Environmental Health

REVIEW

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Synergistic health effects of air pollution, temperature, and pollen exposure: a systematic review of epidemiological evidence

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Abstract

Background: Exposure to heat, air pollution, and pollen are associated with health outcomes, including cardiovascular and respiratory disease. Studies assessing the health impacts of climate change have considered increased exposure to these risk factors separately, though they may be increasing simultaneously for some populations and may act synergistically on health.
Our objective is to systematically review epidemiological evidence for interactive effects of multiple exposures to heat, air pollution, and pollen on human health.

Methods: We systematically searched electronic literature databases (last search, April 29, 2019) for studies reporting quantitative measurements of associations between at least two of the exposures and mortality from any cause and cardiovascular and respiratory morbidity and mortality specifically. Following the Navigation Guide systematic review methodology, we evaluated the risk of bias of individual studies and the overall quality and strength of evidence.

Results: We found 56 studies that met the inclusion criteria. Of these, six measured air pollution, heat, and pollen; 39 measured air pollution and heat; 10 measured air pollution and pollen; and one measured heat and pollen. Nearly all studies were at risk of bias from exposure assessment error. However, consistent exposure-response across studies led us to conclude that there is overall moderate quality and sufficient evidence for synergistic effects of heat and air pollution. We concluded that there is overall low quality and limited evidence for synergistic effects from simultaneous exposure to (1) air pollution, pollen, and heat; and (2) air pollution and pollen. With only one study, we were unable to assess the evidence for synergistic effects of heat and pollen.

Conclusions: If synergistic effects between heat and air pollution are confirmed with additional research, the health impacts from climate change-driven increases in air pollution and heat exposure may be larger than previously estimated in studies that consider these risk factors individually.

Keywords: Air pollution, Temperature, Pollen, Systematic review

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Background

Climate change is expected to increase exposure to environmental health risk factors, including extreme temperatures, air pollution, and aeroallergens [1–5]. These environmental health risk factors are associated with a range of health outcomes, including cardiovascular and respiratory disease [5]. Changes in these risk factors will be spatially heterogeneous, depending on local emission sources, meteorology, vegetation type and distribution, and other factors. As these risk factors do not exist in isolation, populations may experience simultaneous increases in exposure to heat, air pollutants, and pollen. Understanding whether these environmental health risk factors have synergistic effects on health outcomes can inform future climate change health risk assessments. The objective of this paper is therefore to determine whether the current state of the epidemiological evidence supports the presence of synergistic effects between heat, air pollutants, and pollen on human health outcomes.

Both average and extreme temperatures are expected to increase with climate change [5]. These changes may compromise the body's ability to regulate temperature leading to a range of health outcomes, including heat exhaustion, heatstroke, and hyperthermia [6]. Exposure to extreme heat events can worsen cardiovascular and respiratory diseases, as well as other chronic conditions, such as cerebrovascular disease, diabetes, and kidney disease [7, 8]. The mechanisms by which heat exacerbates respiratory disease are not well understood. In respiratory diseases such as asthma and chronic obstructive lung disease, inflammation plays a central role in the pathogenesis and exacerbation of the disease. Heat increases systemic and pulmonary inflammation as a consequence of thermoregulation – the attempt by the body to maintain a temperature within a safe range [9]. A second mechanism by which heat affects chronic lung disease may be related to impairment in breathing patterns meant to compensate for elevations in body temperature [10, 11]. Heat induces cardiovascular disorders through multiple mechanisms including cell damage, inflammation, and blood clotting [12]. For mortality, epidemiological studies have linked even small increases in daily mean or maximum temperatures with increases in premature death. Applying these epidemiological exposure-response relationships to climate model simulations of future temperature, studies have attributed tens of thousands of premature deaths to increasing temperatures in the United States by mid-century [13]. The most vulnerable population subgroups to heat include older adults, children, people working outdoors, and economically disadvantaged communities [7], as well as end stage renal disease patients [14]. While climate adaptation measures can lessen some of the health impacts, climate change-related temperature increases are expected to be an important health risk factor in the U.S. and globally in the future.

Air pollution exposures may also increase with climate change through various pathways, including increased frequency of stagnation events that prohibit atmospheric venting, enhanced photochemical production of secondary pollutants (e.g. tropospheric ozone and some components of fine particulate matter, $PM_{2.5}$), and increasing "natural" gaseous and particulate emissions influenced by warmer and drier conditions (e.g. wildfire smoke, airborne soil dust, and ozone and $PM_{2.5}$ formation from biogenic volatile organic compounds) [2]. As a result, simulations of future air quality under various climate change scenarios indicate a likely "climate penalty" for ozone, making it harder to attain ambient air quality standards even with the same level of anthropogenic emission controls in place [15, 16]. The literature is more mixed for the effects of climate change on $PM_{2.5}$ given the varied and often counteracting effects of climate on $PM_{2.5}$ components and precursor emissions, as well as atmospheric transport and loss. Recent studies suggest a potentially large influence of wildfire smoke and airborne soil dust on $PM_{2.5}$ concentrations [17]. Air pollution exposure can have large implications for human health, particularly heart and lung disease and mortality, through various mechanisms. Exposure to air pollutants, such as $PM_{2.5}$ and ozone, increases oxidative stress leading to pulmonary and systemic inflammation and increased permeability of the lung lining (airway epithelium), increased airway hyperresponsiveness in asthmatics, and decreases in lung function in healthy patients and patients with chronic lung disease [17, 18]. Development and worsening of cardiovascular disease in response to air pollution exposure likely occurs along pathways that include systemic inflammation, alterations in coagulation, dysfunction in the lining of blood vessels (endothelial dysfunction), and progression of atherosclerosis [19]. Following these pathways, air pollution is associated with increased respiratory and cardiovascular mortality. Given the large body of epidemiological literature providing strong evidence for associations between $PM_{2.5}$ and premature mortality from cardiovascular disease, respiratory disease, and lung cancer, and between ozone and respiratory mortality, even small increases in pollution levels in the future can have profound influences on human health outcomes [17, 20].

Climate change is also expected to affect the start, duration, and intensity of the pollen season, with changes differing by region [21]. Climate change and rising greenhouse gas concentrations are correlated with aeroallergens in a number of ways, including increased and faster plant growth, increased pollen production by plants, increased allergenic proteins contained in pollen, earlier start time of plant growth, and longer plant seasons [22]. Meteorological conditions, including precipitation, atmospheric temperature, humidity, and wind speed, can alter the concentrations of plant pollens, which can then influence the occurrence of allergic diseases [23]. Inhalation of pollen grains causes disruption of the immune system within the lungs and increases the susceptibility of individuals to respiratory viral infections [24]. These breakdowns in immune system defenses following exposure to pollen are seen not only in patients with underlying allergies, but also in healthy individuals. In asthmatics, exposure to pollen activates an array of immune cells resulting in bronchoconstriction and increased permeability of airway epithelium [25]. There are few studies that have examined the link between aeroallergen exposure and cardiovascular disease; however, airborne pollen may be a risk factor for myocardial infarction [26]. The mechanism may be related to pollen triggering mast cell activation and histamine release leading to coronary artery spasm or plaque rupture. With the pervasiveness of allergies and allergic asthma among diverse populations throughout the U.S. and the world, climate-related changes in aeroallergen exposure may have widespread impacts on allergic rhinitis and asthma emergency department visits, both of which place a heavy burden on the U.S. healthcare system.

There is substantial literature on respiratory and cardiovascular outcomes related to the isolated exposure to heat, air pollution, or pollen [12, 27–29]. However, fewer studies examine potential synergies or mechanisms behind interactions among these environmental risk factors. There is evidence that air pollutants can bind to pollen grains, precipitating faster release of allergens, increasing allergen absorption in the lungs, and potentiating the allergenicity of pollen, however this is mostly supported in in vitro and animal studies and the clinical significance on a population level is less certain [17, 30, 31]. Prior studies suggest a joint effect of air pollution and heat on health outcomes such as mortality and respiratory morbidity [32]. Many disease states, including heart and lung disease, share a common pathway in which exposure to heat, air pollution, and pollen causes systemic and organ-specific inflammation and cellular damage [9, 17, 28, 33].

Previous studies assessing the potential health impacts of future climate change have considered heat, air pollution, and pollen exposure individually and have not accounted for potential synergistic effects [7, 34–40]. For example, the comprehensive Climate Change Impacts and Risk Analysis project for the U.S. includes estimates of future increases in heat-related mortality, ozone-related mortality, and asthma emergency department visits attributable to aeroallergens, with substantial increases simulated for moderate and severe climate scenarios [4, 41]. Each of these risk factors was considered separately when estimating future health impacts. If there are synergistic effects between these exposures, using single-hazard approaches may underestimate the health impacts of heat, air pollution, and pollen exposures under climate change.

Here, we conduct a systematic literature review of epidemiological studies to determine whether simultaneous exposure to heat, air pollution, and pollen (or a subset of these risk factors) synergistically increases the risk of mortality from any cause and mortality and morbidity of cardiovascular and respiratory disease specifically. We focus on these three risk factors as they share common attributes – they are conditions of the ambient air and have been found to affect respiratory and cardiovascular health. Other risk factors associated with climate change may also affect these health systems, but we consider the body of literature to be too nascent to support a more inclusive systematic review. Results of our review may be useful to more comprehensively characterize future public health disease burdens under climate change scenarios.

Methods

Search strategy, study selection, and data extraction

We conducted a systematic literature review using the Navigation Guide, a methodology for evaluating environmental evidence based on methods used in the clinical sciences [42]. The objective of this systematic review is to assess whether there are interactions between exposure to criteria air pollutants, extreme heat, and pollen, or a subset of these three risk factors, on cardiovascular or respiratory outcomes in human populations. Criteria air pollutants include ground-level ozone (O_3), carbon monoxide (CO), nitrogen dioxide (NO_2), lead, particulate matter (PM), and sulfur dioxide (SO_2).

We define the “Population”, “Exposure”, “Comparator”, and “Outcomes” (PECO) statement as:

- Population: Any human population of any age in any location.
- Exposure: Areas where populations are simultaneously exposed to a) criteria air pollutants and extreme heat; b) criteria air pollutants and pollen; c) pollen and extreme heat; or d) all three risk factors.
- Comparator: Areas where these simultaneous exposures are not occurring.
- Outcome: Cardiovascular and respiratory diseases or mortality.

We searched the databases PubMed, ProQuest, and Scopus with the search terms “air pollution”, “air quality", "air pollutants", "pollen", "aeroallergens", "temperature", "heat", "dust", "NO_2", "SO_2", "particulate matter", "ozone", "multipollutant" for exposures, and the terms "cardiovascular", "respiratory", "mortality", "asthma", and "allergies" for outcomes (Table S1). We conducted a first search on April 22, 2019 and an updated search with more search terms on April 29, 2019. We found additional articles through hand searching the references of fully screened articles.

We included original studies that measured at least two of the exposures (heat, air pollution, and pollen) and at least one of the health outcomes (cardiovascular or respiratory disease or mortality), without limiting by publication date. We excluded studies that were not published in English, did not study a human population, did not measure at least two of the exposures, did not report quantitative results for exposure-response relationships, or did not describe interactions between the exposures. We screened for reference duplicates using Mendeley Desktop. When it was not clear whether studies met the inclusion criteria or not, two reviewers discussed each study and came to a joint decision on inclusion or exclusion.

Data extraction and risk of bias for each included study

Two authors independently extracted data and analyzed risk of bias for each included study. A third author reviewed all studies to resolve discrepancies between the two independent reviewers' risk of bias ratings. We evaluated risk of bias for each of our included studies using the Cochrane Collaboration's "Risk of Bias" tool and the Agency for Healthcare Research and Quality's domains [43]. The domains we evaluated included study design, exposure assessment (air pollution), exposure assessment (temperature), exposure assessment (pollen), detection of outcome, reporting, and conflict of interest. Study design was rated as "low" risk of bias if it was a cohort, case crossover, or time series design. To be rated as "low" risk of bias for air pollution exposure assessment, the study must have measured at least two criteria pollutants and must have measured them in a way that represented individual exposure. To be rated as "low" risk of bias for pollen exposure assessment, the study had to use a method that measured pollen exposure at an individual level. To be rated as "low" risk of bias for temperature, studies had to use data from meteorological surveillance networks; we did not judge a lack of individual exposure measurement to introduce high risk of bias for temperature since temperature is less spatially heterogeneous compared with air pollution. To be rated as "low" risk of bias for detection of health outcome, the study had to use the *International Classification of Diseases* (ICD) to classify the health outcome category. To be rated as "low" risk of bias for reporting, the study had to report all outcomes that were assessed. To be rated as "low" risk of bias for conflict of interest, the study had to acknowledge that there was no conflict of interest. The possible ratings for the studies for each domain were "low", "probably low", "probably high", or "high" risk of bias. We used the "probably low" and "probably high" categories when not enough information was given to definitively assign "low" and "high" ratings.

Quality and strength of evidence across studies

To evaluate the quality and strength of evidence across all studies, we used the Grading of Recommendations Assessment, Development and Evaluation (GRADE) systematic review approach [44]. We stratified papers by the following categories of multiple exposures: 1) heat, air pollution, and pollen; 2) heat and air pollution; 3) air pollution and pollen; and 4) heat and pollen.

To evaluate the quality of the evidence across all studies, we upgraded and downgraded studies according to several criteria. Downgrading factors included serious risk of bias, serious indirectness in the studies such that evidence is not directly comparable to our PECO statement criteria, serious inconsistency in effect estimates across studies, serious imprecision due to small sample size and/or small outcome count, and likely publication bias resulting in an over or underestimate of true effects from exposure. Downgrading for serious risk of bias by − 1 occurred if there were instances of an unclear limitation in the evidence and by − 2 if there were instances of serious limitations or very serious limitations during the assessments. Downgrading for inconsistency by − 1 occurred if there were minimal or no overlap of confidence intervals and by − 2 if there was wide variance of point estimates across studies. Downgrading for indirectness by − 1 was applied if there were large differences in study population and by − 2 if there were large differences and if surrogate outcomes were applied. Downgrading for imprecision by − 1 occurred if there was a small sample size or small outcome count and by − 2 if there was both.

Upgrading factors included large magnitude of effect such that confounding alone could not explain the association, consistent dose-response gradient across studies, all plausible confounding would reduce a demonstrated effect, and all possible confounding would suggest a spurious effect when the actual results show no effect. After considering the upgrading and downgrading factors, the studies were then given a rating of "low quality", "moderate quality", or "high quality." Possible ratings were 0, meaning no change from initial quality rating, − 1 or − 2, meaning downgrades in quality rating, and + 1 and + 2, meaning upgrades in quality rating. Upgrading for large magnitude of effect by + 1 occurred with the effect estimate was large such as a relative risk of 2 or higher and by + 2 if there was a very large effect estimate such as a relative risk of 5 or higher. Upgrading for dose-response by + 1 was applied if there was observation that there was a dose response gradient between increased exposure and increased outcomes and by + 2 if there was a rapid and large absolute increase in outcomes as dose increased. Upgrading for effect of plausible confounding by + 1 was applied if the plausible confounders were adjusted for in the analysis.

We evaluated the strength of evidence across all studies based on quality of the evidence, direction of effect estimates, confidence in effect estimates, and other attributes [45]. To the extent possible, we discuss these ratings according to categories of health outcomes (e.g. all-cause mortality, cardiovascular disease, and respiratory disease). The ratings for strength of the evidence are: “evidence of lack of association” (studies show no adverse effect), “inadequate evidence” (studies permit no conclusion about an effect), “limited evidence” (studies suggest an effect but only in a single or limited number of studies), and “sufficient evidence” (studies indicate a causal relationship between exposure and effect). We followed the more detailed definitions of each strength rating given by Johnson et al. [46].

Results

Our search retrieved 1730 unique records, and we added 16 papers identified through other sources (Fig. 1). We screened 605 papers after removing duplicates and assessed the full text of 406 articles for eligibility. We excluded 350 articles because they did not describe interactions between the exposures or did not describe the outcome measures. Ultimately, we included 56 studies that met our eligibility criteria. Table 1 includes descriptions of each study.

Of these 56 studies, six measured air pollution, heat, and pollen; 39 measured air pollution and heat; 10 measured air pollution and pollen; and one measured heat and pollen. Forty-six studies were a time series design, three were cohort studies, one was a cross sectional design, one was a nested case control design, and five were a case-crossover design. Data collection in these studies ranged from 1987 to 2010 and publication date ranged from 2002 to 2018. The qualifying studies ranged widely

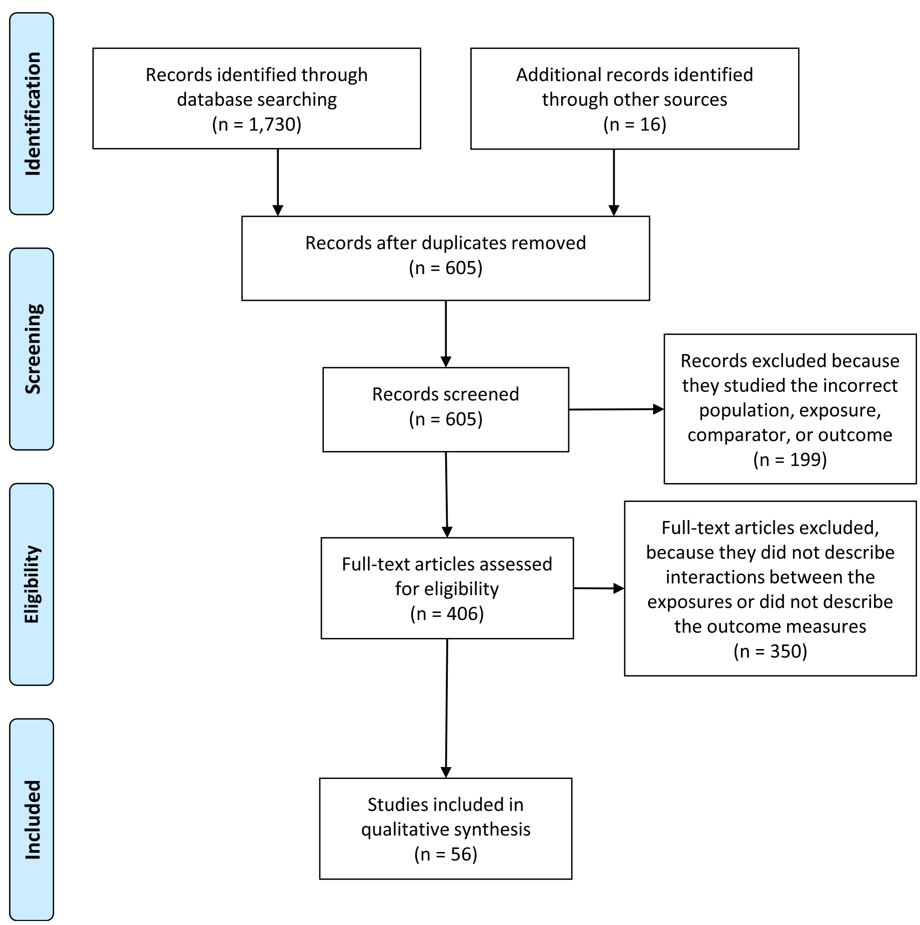

Fig. 1 PRISMA Diagram showing the number of studies included and excluded at each step

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
Air pollution, heat, and pollen ($n = 6$)								
Respiratory								
Hebbern 2015 [47]	Time series	10 Canadian cities	Apr 1994- Mar 2007	Asthma hospital admissions	Not reported	CO, O_3, NO_2, SO_2, PM_{10}, $PM_{2.5}$	Weed, tree, grass	Daily Mean
Makra 2015 [48]	Time series	Szeged, Hungary	1999–2007	Asthma emergency room visits	0–14 years; 15–64 years; 65+ years ($n = 936$ asthma ER visits)	CO, NO, NO_2, SO_2, O_3, PM_{10}	Ambrosia, maple, alder, mugwort, birch, hemp, hornbeam, goosefoot, hazel, ash, walnut, mulberry, pine, plantain, platan, grasses, poplar, oak, dock, willow, yew, linden, elm, nettle	Daily Mean, daily maximum, daily minimum, daily range
							Matyasovszky 2011 [49]	Time series
Szeged, Hungary	1999–2007	Respiratory hospital admissions	All ages; 15–64 years; 65+ years ($n = 133,464$ hospital admission)	CO, NO, NO_2, SO_2, O_3, PM_{10}	Ambrosia, maple, alder, mugwort, birch, hemp, hornbeam, goosefoot, hazel, ash, walnut, mulberry, pine, plantain, platan, grasses, poplar, oak, dock, willow, yew, linden, elm, nettle	Daily mean, maximum, minimum, range		
Mazenq 2017 [50]	Nested case control	Southeastern France	Jan 2013-Dec 2013	Asthma emergency room visits	3–18 years ($n =$ 1182 asthma ER visits)	PM_{10}, $PM_{2.5}$	cypress, birch, ash, grass, urticaceae	Daily average
Mireku 2009 [51]	Retrospective time series	Detroit, MI	Jan 2004- Dec 2005	Asthma emergency room visits	1–18 years ($n =$ 25,401 asthma ER visits)	$PM_{2.5}$, PM_{10}, SO_2, O_3	Total	Daily average
Witonsky 2019 [52]	Retrospective cohort	Bronx, NY	Jan 2001- Dec 2008	Asthma emergency room visits and hospitalizations	All ages ($n = 42,065$ asthma ER visits; $n = 1664$ asthma-related hospitalizations)	NO_x, O_3, SO_2	Grass, weed, tree,	Daily average
Air pollution and temperature ($n = 39$)								
Multiple health endpoints								
Analitis 2014 [53]	Ecological time series	9 European cities	1990–2004	All natural, cardiovascular, and respiratory mortality	0–64, 65–74, 75–84, and 85+ years (n not reported)	SO_2, PM_{10}, NO_2, O_3, CO		3-h average
Analitis 2018 [54]	Ecological time series	9 European cities	2004–2010	All natural, cardiovascular, and respiratory mortality	All ages; 15–64, 65–74, 75+ years (n not reported)	PM_{10}, O_3, NO_2		Daily mean
Breitner 2014 [55]	Time series	Bavaria, Germany	1990–2006	Non accidental, cardiovascular,	< 85, 85+ years ($n = 338,631$	PM_{10}, O_3		Daily mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
				respiratory mortality	deaths)			
Cheng 2012 [56]	Time series	Shanghai, China	2001–2004	Non-accidental, cardiovascular, respiratory mortality	All ages (n = 173, 911 deaths)	PM_{10}, O_3, SO_2, NO_2		Daily minimum, maximum, mean
Li 2011 [57]	Time Series	Tianjin, China	2007–2009	Cardiovascular, respiratory, cardiopulmonary, stroke and IDH, Non accidental mortality	All ages; < 65, 65+ years (n = 111,087 deaths)	PM_{10}, SO_2, NO_2		Daily mean
Li 2015 [58]	Time Series	Guangzhou, China	2003–2011	Non accidental mortality, cardiovascular mortality, respiratory mortality	< 65, 65+ years (n = 213,737 deaths)	PM_{10}		Daily mean
Lokys 2018 [59]	Time series	28 districts, Germany	2001–2011	Cardiovascular and respiratory hospital admissions	Not reported	NO_2, SO_2, O_3, PM_{10}		Daily mean
All-cause or non-accidental only								
Burkart 2013 [60]	Time Series	Berlin and Lisbon	1998–2010	All cause mortality	Age not reported (n = 698,586 deaths)	PM_{10}, O_3		Hourly mean
Chen 2018a [61]	Time series	8 European cities	1999–2013	Non accidental mortality	0–74, 75+ years (n = 742,526 deaths)	$PM_{2.5}$, PM_{10}, O_3		Daily mean
Chen 2018b [62]	Time Series	8 European cities; 86 US Cities	1999–2013; 1987–2000	Non accidental mortality	All ages (n not reported)	PM_{10}, NO_2, O_3		Daily mean
Dear 2005 [63]	Time series	12 French cities	Aug-03	All cause mortality	All ages (n not reported)	O_3		24 h Minimum, maximum
Filleul 2006 [64]	Time series	9 French cities	Aug-03	All cause mortality	All ages (n not reported)	O_3		Daily maximum
Jhun 2014 [65]	Time series	97 cities	1987–2000	Non accidental mortality	0–99 years (n not reported)	O_3		Daily high
Kim 2015 [66]	Time series	7 South Korean cities	Jan 2000-Dec 2009	Daily non accidental deaths	< 65, 65+ years (n = 828,787 deaths)	PM_{10}		Daily mean
Liu 2016 [67]	Time Series	20 US communities	1987–2000	Non accidental mortality	Not reported	O_3		Daily mean
Meng 2012 [68]	Time series	8 Chinese cities	2001–2008	Non accidental mortality	Not reported	PM_{10}		Daily mean
Moolgavkar 2003 [69]	Time Series	Cook County, IL & LA County, CA	1987–1995	Non accidental mortality	All ages; 65+ years (n not reported)	O_3, SO_2, NO_2, CO, PM		Daily minimum, median, maximum

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
Park 2011 [70]	Time series	Seoul, South Korea	Jun 1999- Dec 2007	Non accidental mortality	All ages; 65–74, 75–84, 85+ years (*n* = 291,665 deaths)	PM_{10}, NO_2, SO_2, CO, O_3		Daily mean, minimum, maximum
Pattenden 2010 [71]	Time series	15 conurbations in England and Wales	1993–2003	All cause mortality	0–64, 65–74, 75–84, 85+ years (n not reported)	O_3, PM_{10}		Two day Mean
Peng 2013 [72]	Time series	23 European Cities; 12 Canadian Cities; 86 US cities	Canada 1987–1996; Europe 1990–1997; US 1987–1996	Non accidental mortality	All ages; < 75, 75+ years (n not reported)	NO_2, SO_2, O_3, PM_{10}		Daily mean
Rainham 2005 [73]	Time series	Toronto, Canada	1981–1999	Non Trauma mortality	Not reported	CO, NO_2, SO_2, O_3, $PM_{2.5}$		Daily mean
Scortichini 2018 [74]	Time series	25 Italian cities	2006–2010	Mortality from natural causes	35+ years (*n* = 187,743 deaths)	O_3, PM_{10}		Daily mean
							Shaposhnikov 2014 [75]	Time series
Moscow, Russia	2006–2009, 2010	Non accidental mortality	All ages; < 65, 65+ years (*n* = 10, 860 deaths)	O_3, PM_{10}		Daily mean		
Stafoggia 2008 [76]	Case crossover	9 Italian cities	1997–2004	Mortality from natural causes	35+ years (*n* = 321,024 deaths)	PM_{10}		Daily mean, apparent
Sun 2015 [77]	Time Series	Hong Kong	1999–2011	Mortality from natural causes	Age not reported (*n* = 456,317 deaths)	$PM_{2.5}$, NO_2, SO_2, O_3		Daily mean
Vanos 2015 [78]	Time series	12 Canadian cities	1981–2008	Non accidental mortality	Not reported	O_3, NO_2, $PM_{2.5}$, SO_2		Daily mean
Wilson 2014 [79]	Time Series	95 US cities	1987–2000	Mortality	Not reported	O_3		Daily mean
Zhang 2006 [80]	Time series	Shanghai, China	Jan 2001- Dec 2004	Non accidental mortality	All ages; 0–4, 5–44, 45–64, 65+ years (*n* = 173,911 deaths)	O_3, PM_{10}, SO_2, NO_2		Daily mean
Respiratory only								
Ding 2017 [81]	Case crossover	Taiwan	2000–2013	COPD mortality	40–64, 65–79, 80+ years (n not reported)	$PM_{2.5}$, O_3, SO_2		Daily mean, maximum, minimum
Jo 2017 [82]	Time series	Busan, South Korea	2007–2010	Hospital admissions for respiratory disease	0–15,16–64, 65+ years (n not reported)	$PM_{2.5}$, PM_{10}		Daily average, minimum, maximum, range
Kunikullaya 2017 [83]	Retrospective ecological time series	Bangalore, India	One year	Asthma-related emergency room visits and hospitalizations	> 18 years (n not reported)	SO_2, NO_2, PM_{10}, $PM_{2.5}$		Daily mean
Lam 2016 [84]	Time series	Hong Kong	2004–2011	Asthma hospitalizations	< 5, 5–14, 15–59, 60+ years (*n* = 56,	PM_{10}, SO_2, NO_2, O_3		Daily mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
					112 asthma admission)			
Mirabelli 2016 [85]	Retrospective cross sectional	United States	2006–2010	Asthma symptoms	18+ years ($n = 50$, 356 respondents)	$PM_{2.5}$, O_3		Average daily mean
Qiu 2018 [86]	Time series	Chengdu, China	Jan 2015- Dec 2016	COPD hospital admissions	All ages; < 60, 60–70, 70–80, 80+ years ($n = 54$, 966 COPD admission)	PM_{10}, $PM_{2.5}$, NO_2, SO_2, CO, O_3		Daily mean
Winquist 2014 [87]	Time Series	Atlanta, GA	16 years	Asthma emergency department visits	5–17 years (n not reported)	CO, NO_2, SO_2, O_3, $PM_{2.5}$		Daily minimum, maximum,
Cardiovascular only								
Lee 2018 [88]	Case crossover	Seoul, South Korea	2008–2014	Migraine emergency room visits	All ages; < 40, 40–64, 65+ years ($n = 18,921$ ER visits)	$PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, CO		Hourly mean
Luo 2017 [89]	Time series	3 Chinese cities	2008–2011	Cardiovascular mortality	All ages; < 65, 65+ years ($n =$ 290,593 deaths)	PM_{10}, NO_2, SO_2		Daily minimum, maximum, mean
Ren 2008 [90]	Time Series	95 US cities	1987–2000	Cardiovascular mortality	< 65, 65–74, 75+ years ($n =$ nearly 4 million cardiovascular deaths)	O_3		Daily maximum
Ren 2009 [91]	Time series	95 US cities	1987–2000	Cardiovascular mortality	< 65, 65–74, 74+ years (n= > 4.3 million cardiovascular deaths)	O_3		Daily maximum
Air pollution and pollen ($n = 10$)								
Respiratory								
Anderson 1998 [92]	Time series	London	Apr 1987- Feb 1992	Asthma emergency admissions	All ages; 0–14, 15–64, 65+ years (n not reported)	O_3, NO_2, Black smoke, SO_2	Birch, Grass, Oak	Mean 24 h
Cakmak 2012 [93]	Time series	11 Canadian cities	Apr 1994-Mar 2007	Asthma hospital admissions	Not reported	CO, $PM_{2.5}$, PM_{10}, NO_2, SO_2	Tree, Weed	Mean 24 h
Chen 2016 [94]	Time-series case-crossover	Adelaide, South Australia	Jul 2003- Jun 2013	Asthma hospital admissions	0–17, 18+ years ($n = 36$, 024admissions)	$PM_{2.5}$, NO_2, PM_{10}	Ash tree, birch, cypress, eucalyptus, fruit tree, olive tree, pinus, plane tree, she-oak, wattle, chenopodia-ceae, compositae, plantain, polygonaceae, salvation jane, grass	Daily average
Cirera 2012 [95]	Time series	Cartagena, Spain	Jan 1995- Dec 1998	COPD and asthma emergency room	Age not reported ($n = 1617$ asthma and 2322 COPD	SO_2, NO_2, TSP, O_3	Poaceae, Urticaceae	Hourly mean

Table 1 Descriptive information for all included studies, categorized by the combination of risk factor exposures *(Continued)*

Study	Type	Location	Duration	Outcome	Population	Pollutants Measured	Pollen Measured	Temperature Measurement
				visits,	ER visits)			
Galan 2003 [96]	Time series	Madrid, Spain	1995–1998	Asthma emergency department visits	Age not reported ($n = 4827$ asthma attacks)	SO_2, PM_{10}, NO_2, O_3, CO	*Olea europaea*, Plantago sp., Poaceae, Urticaceae	Daily mean
Gleason 2014 [97]	Time-stratified case-crossover	New Jersey	April - Sept 2004–2007	Asthma emergency department visits	3–17 years ($n =$ 21,854 asthma ED visits)	O_3, PM	Tree, grass, weed, ragweed	Daily mean
Goodman 2017 [98]	Time series	New York City	1999–2009	Asthma hospital Admissions	< 6, 6–18, 19–49, 50+ years ($n =$ 295,497 asthma admission)	O_3, PM	Tree, weed, total	Daily average, maximum, minimum
Krmpotic 2011 [99]	Time series	Zagreb, Croatia	Jan 2004- Dec 2006	Asthma hospital admissions	> 18 years ($n =$ 4125 asthma admissions)	NO_2, CO, PM_{10}	Alder, Hazel, Birch, Hornbeam, Oak, Grasses, Ragweed	Daily minimum, maximum, mean
Ross 2002 [100]	Prospective Cohort	East Moline, IL	7 months	Peak Expiratory flow rates, respiratory symptoms, frequency of asthma attacks, asthma medication use	5–49 years ($n =$ 59 people)	O_3, PM, SO_2	Grass, Ragweed, Total	Daily mean, Maximum
Cardiovascular								
Stieb 2000 [101]	Time series	Saint John, Canada	Jul 1992- Jun 1994, Jul 1994- Mar 1996	Cardiorespiratory emergency department visits	Age not reported ($n = 19{,}821$)	CO, H_2S, NO_2, O_3, SO_2, TRS	Ascomycetes, basidiomycetes, deuteromycetes, ferns, grass, tree, weed	Daily average
Heat and pollen ($n = 1$)								
Silverberg 2015 [102]	Cohort Study	United States	2006	Pediatric hay fever	0–17 years ($n =$ 91,642)	–	Total	Monthly mean

Study	Study design	Exposure assessment (air pollution)	Exposure assessment (temperature)	Exposure assessment (pollen)	Detection of outcome	Reporting	Conflict of Interest
Air pollution, heat, and pollen							
Hebbern 2015	1	2	1	2	1	1	1
Makra 2015	1	3	2	3	1	1	2
Matyasovszky 2011	1	3	2	3	2	1	2
Mazenq 2017	1	2	1	3	1	1	1
Mireku 2009	1	3	1	3	2	1	2
Witonsky 2019	1	3	1	3	1	1	1
Air polution and heat							
Analitis 2014	1	2	2	n/a	1	1	2
Analitis 2018	1	2	2	n/a	1	1	1
Breitner 2014	1	3	1	n/a	1	1	1
Burkart 2013	1	3	1	n/a	2	1	2
Chen 2018a	1	2	2	n/a	1	1	1
Chen 2018b	1	2	2	n/a	1	1	1
Cheng 2012	1	2	1	n/a	1	1	1
Dear 2005	1	3	2	n/a	2	1	2
Ding 2017	1	2	1	n/a	1	1	1
Filleul 2006	1	2	1	n/a	2	1	1
Jhun 2014	1	3	1	n/a	2	1	2
Jo 2017	1	2	2	n/a	1	1	1
Kim 2015	1	3	1	n/a	1	1	1
Kunikullaya 2017	1	3	2	n/a	1	1	1
Lam 2016	1	2	1	n/a	1	1	1
Lee 2018	1	2	1	n/a	1	1	1
Li 2011	1	2	2	n/a	1	1	2
Li 2015	1	3	1	n/a	1	1	1
Liu 2016	1	3	1	n/a	1	1	1
Lokys 2018	2	3	2	n/a	1	1	2
Luo 2017	1	2	1	n/a	1	1	1
Meng 2012	1	2	2	n/a	1	1	1
Mirabelli 2016	2	3	3	n/a	3	1	1
Moolgavkar 2003	1	3	2	n/a	1	1	2
Park 2011	1	2	1	n/a	1	1	1
Pattenden 2010	1	3	2	n/a	1	1	1
Peng 2013	1	2	2	n/a	2	1	1
Qiu 2018	1	2	2	n/a	1	1	1
Rainham 2005	1	2	2	n/a	1	1	2
Ren 2008	1	3	2	n/a	1	1	1
Ren 2009	1	3	2	n/a	1	1	2
Scortichini 2018	1	2	2	n/a	1	1	1
Shaposhnikov 2014	1	2	1	n/a	1	1	2
Stafoggia 2008	1	3	2	n/a	1	1	1
Sun 2015	1	2	1	n/a	1	1	1
Vanos 2015	1	2	1	n/a	1	1	2
Wilson 2014	1	3	1	n/a	2	1	2
Winquist 2014	1	2	3	n/a	1	1	2
Zhang 2006	1	2	1	n/a	1	1	1
Air pollution and pollen							
Anderson 1998	1	3	n/a	3	1	1	2
Cakmak 2012	1	2	n/a	2	1	1	1
Chen 2016	1	4	n/a	2	1	1	1
Cirera 2012	1	3	n/a	3	2	1	1
Galan 2003	1	3	n/a	2	1	1	2
Gleason 2014	1	2	n/a	3	1	1	1
Goodman 2017	1	2	n/a	4	1	1	2
Krmpotic 2011	1	3	n/a	3	1	1	2
Ross 2002	1	2	n/a	2	3	1	2
Stieb 2000	1	4	n/a	3	2	1	2
Heat and pollen							
Silverberg 2015	1	n/a	2	4	2	1	1

Legend

- 1 Low risk of bias
- 2 Probably low risk of bias
- 3 Probably high risk of bias
- 4 High risk of bias

Fig. 2 Final risk of bias evaluation for each study in air pollutants and pollen types measured, metrics used for each exposure type (e.g. averaging times, time lags), and health outcomes (including asthma and hay fever symptoms, cardiovascular and respiratory emergency department visits and hospitalizations, cause-specific mortality, and all-cause mortality).

Risk of bias determinations and rationale for each study can be found in Tables S2 through S57. Almost all of the studies were rated as "low" or "probably low" risk of bias for study design, detection of outcome, reporting, and conflict of interest (Fig. 2). Risk of bias for exposure assessment varied across the studies. For air pollution and pollen, we rated many studies as having a "probably high" risk due to a lack of exposure measurement at an individual level, as they used exposure assessment techniques such as central site monitors that are broadly representative of regional air pollution levels but may not represent individual exposure well. Several of these studies only used one central site monitor, which we judged could potentially introduce bias since pollution levels vary spatially within geographic areas such as cities. For temperature, studies were generally rated as having a "low" or "probably low" risk of bias since data were sourced from meteorological monitoring networks and temperature is less spatially heterogeneous compared with air pollution.

We next assessed the quality and strength of the evidence across the studies. We found six studies that examined potential interactive effects between simultaneous exposure to all three risk factors: air pollutants, pollen, and heat (Table 1). The studies were conducted in Canada, France, Hungary, and the U.S. and all focused on respiratory hospitalizations and emergency department visits (all except one focused specifically on asthma). The studies used widely different methods for categorizing temperature exposure, including spatial synoptic classification [47, 48], seasonal analysis [52], and interday temperature change [51]. Generally, the studies were individually rated as low risk of bias for most categories, including study design, detection of outcome, reporting, and conflict of interest. However, we judged some to be at probably high risk of bias for exposure assessment for both air pollutants and pollen. The findings across the studies were inconsistent, with some studies reporting interactive effects of all three or some combination of the exposures [47–49, 52], while others reported independent effects that were unaffected by controlling for the other risk factors [51] or were inconclusive when considering simultaneous exposure to all three risk factors [50].

Overall, we rated the quality of the evidence for synergistic respiratory effects between air pollution, heat, and pollen as "low" since studies were inconsistent in finding significant evidence of interactive effects and studies that reported positive associations of interactions had minimal magnitudes (Table 2). We rated the overall strength of the evidence as "limited" since synergistic effects between heat, air pollution, and pollen were observed in some studies, but these findings were not consistent across studies.

We found 39 articles that examined potential interactive effects between exposure to air pollutants and heat (Table 1). These studies were carried out in Europe, the U.S., Canada, Russia, Taiwan, South Korea, India, Hong Kong, and China. Most were conducted in urban areas. A majority of the studies (29) included health endpoints that were not disease-specific, such as all-cause and non-accidental mortality. A smaller subset of 12 studies considered respiratory disease specifically (some focusing on asthma specifically) and 11 considered cardiovascular disease specifically (we have included migraine in this category as a potential indicator of cardiovascular disease, Adelborg et al. [103]). Most studies included multiple criteria pollutants – most often ozone and PM_{10}, though some only included ozone, and some also included $PM_{2.5}$, $PM_{2.5-10}$, NO_2, SO_2, and CO. The temperature metric differed between studies and included daily mean, minimum and/or maximum.

Of these 39 studies addressing synergistic effects between air pollution and heat, 19 reported interactive effects between heat and air pollution exposure on health outcomes studied. Out of these studies, 15 of 29 studies examined health outcomes that were not disease-specific (e.g. all-cause mortality, hospital admissions) and found synergistic effects [53–55, 57, 58, 60, 61, 66, 68, 71, 73–77], four of 12 studies found synergistic effects for respiratory health outcomes [55, 57, 59, 84], and eight of 11 studies found synergistic effects for cardiovascular health outcomes [54, 55, 57–59, 88, 90, 91]. Here, we are not distinguishing between mortality and morbidity for respiratory and cardiovascular health outcomes. Generally, the studies found synergistic effects from simultaneous exposure to extremely high temperatures and air pollution, with a potentially additional role of relative humidity. A method of weather classification that incorporated humidity used in some of the papers was spatial synoptic classification (SSC), which is described as a "semi-automated statistical approach designed to classify complex daily weather conditions into one of six distinct categories, or a transitional category" and uses values of temperature, dew point, u and v components of wind, cloud cover, and sea level pressure [47, 48, 73, 78]. A strength of this group of studies was the large datasets of pollutant levels and meteorology, including from the National, Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the United States [61, 65, 67, 90, 91] and the Ultrafine Particles and Health Study Group in Europe [61, 62]. Compared with the other categories in our review, air pollution and heat studies covered the

Table 2 Rating of the quality and strength of the evidence for studies assessing interactive effects between heat, air pollution, and pollen ($n = 6$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = "Moderate"			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded because of "probably high" risk of bias for air pollution exposure assessment for four studies and for pollen exposure assessment for five studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in populations for the duration of study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	Some evidence of consistent effects, but the studies were too varied in definitions of risk factors and methods to judge consistency in effect estimates.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies.	0	Studies did not report a consistent relationship between dose and response.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.
Overall Quality of Evidence		Low	The overall quality of the evidence supporting interactive effects is low.
Overall Strength of Evidence		Limited	An association was sometimes observed for synergy between heat, air pollution, and pollen, but the potentially high risk of bias for air pollution exposure could have impacted results and there is a lack of consistently significant findings.

broadest geographic area and included the largest number of people in the studies.

The evidence was strongest for synergistic effects between heat and exposure to either ozone and $PM_{2.5}$. For ozone, 11 of 29 studies reported synergistic effects with heat [53–55, 60, 61, 71, 73, 74, 84, 90, 91]. These effects were found among inter quartile temperature analysis, seasonal analysis, and heatwave analysis in the studies. Effects were found for all-cause mortality, non-accidental mortality, cardiovascular mortality, and morbidity outcomes. High levels of ozone and high temperatures tended to be reported together and the strongest effects on outcomes were found at the highest exposures. We also found evidence for synergistic effects between heat and particulate matter, with 10 of 27 studies reporting synergistic effects [53, 54, 60, 61, 66, 73–76, 88]. These effects were found among inter quartile temperature analysis, seasonal analysis, and heatwave analysis in the studies. Effects were found for all-cause mortality, non-accidental mortality, and morbidity outcomes. A potential interactive effect between heat and particulate matter is further supported by Mazenq et al. [50], who found that temperature and particulate matter were linked but pollen was not.

While most studies assessing synergistic effects between air pollution and temperature focused on heat, several examined effects of cold [55, 56, 58–62, 67, 70, 73, 77, 79, 80, 83, 84, 86–88]. Generally, stronger results were found in warmer seasons when compared to cold seasons. Zhang et al. [80] was the only study in our review that found that synergy between ozone and the cold season was stronger than for the warm season.

We upgraded the overall quality of the evidence of synergistic effects between air pollution and heat because of the relatively consistent finding of significant exposure-response relationships showing interactive effects (Table 3). The consistent findings of interactive effects between air pollutants and heat held for all three

Table 3 Rating of the quality and strength of the evidence for studies assessing interactive effects between heat and air pollution ($n = 39$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = "Moderate"			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded due to "probably high" risk of bias for air pollution exposure assessment for 16 studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in the United States for the duration of the study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	There was not a wide variability in estimates of effects.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies	1	Exposure-response relationship was directionally consistent across 15 of the 34 studies in the category.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect
Overall Quality of Evidence		Moderate	The dose response relationships described in a number of studies did not warrant an upgrade for the overall quality rating.
Overall Strength of Evidence		Sufficient	An association was generally observed for synergistic effects of heat and air pollution exposure, specifically for ozone and PM, but the potentially high risk of bias from the air pollution exposure assessment methods in several studies could have impacted results.

health outcome categories considered: health outcomes that were not disease-specific (e.g. all-cause mortality), respiratory disease, and cardiovascular disease, though more studies found interactive effects for non-cause-specific endpoints and for cardiovascular disease than for respiratory disease. This result may highlight the need for more studies focusing not only on respiratory disease, but also on other diseases. These factors led us to rate the overall quality of the evidence as "Moderate" and the overall strength of the evidence as "Sufficient."

We found 10 studies that assessed potential interactive effects between exposure to air pollution and pollen (Table 1). These studies were conducted in Europe, Canada, Australia, and the U.S. Studies included a variety of pollen types and air pollutants, with little consistency between them. Health outcomes considered were all respiratory morbidity (mostly hospital admissions and emergency department visits), with the exception of one that focused on cardiopulmonary emergency department visits [101].

The studies in this category were inconsistent in their study designs and findings. For example, Anderson et al. [92] concluded that there was no evidence for synergy between air pollutants and pollen, with the exception of SO_2 and grass pollen in children during the warm season. Chen et al. [94] also found little evidence of interactions between air pollutants and pollen but did find that several of the air pollution and pollen exposures were stronger in the cool season than in the warm season. In contrast, Goodman et al. [98] found that, in most populations, adjusting for outdoor pollen generally attenuated relative risk of hospital admissions for both ozone and $PM_{2.5}$. Ross et al. [100] found the association between ozone and asthma medication use was increased after adjusting for aeroallergens. Cakmak et al. [93] found that there were synergistic effects on asthma hospitalization between tree pollen and increasing $PM_{2.5}$, and between weed pollen and PM_{10}.

Given that the 10 studies included inconsistent pollen types and air pollutants, with inconsistent results, we were unable to draw strong conclusions for this category. Overall, we rated the quality of the evidence as "Low" and the strength of the evidence as "Limited." We did not upgrade the quality of the evidence since the studies reported inconsistent findings, and since studies that did find synergistic effects reported effect estimates that had low magnitudes (Table 4).

Our search only found one study that examined interactions between heat and pollen [102]. This study explored climate factors and pollen count impacts on pediatric hay fever prevalence among 91,642 children across the U.S. Hay fever prevalence was shown to increase with the second, third, and fourth quartile mean annual temperature and mean total pollen counts. This study was particularly strong given the large size and national representation of the included population. However, with only one study, we did not draw conclusions regarding the quality and strength of evidence for interactive effects between heat and pollen.

Discussion

We conducted a systematic literature review of human population health studies to examine the evidence for synergistic effects from simultaneous exposure to air pollution, pollen, and heat, or a subset of these three risk factors. We found limited evidence for synergistic respiratory effects of air pollution, pollen, and heat; sufficient evidence for synergistic all-cause mortality, cardiovascular, and respiratory effects of air pollution and heat (particularly for ozone and particulate matter); and limited evidence for synergistic respiratory effects of air pollution and pollen. We were unable to assess evidence for pollen and heat because only one paper came up in our searches.

Overall, there was a substantially larger body of literature examining interactive effects between air pollution and heat, compared with those that included pollen as an exposure of interest. The evidence for interactive effects between air pollution and heat is further strengthened by

Table 4 Rating of the quality and strength of the evidence for studies assessing interactive effects between air pollution and pollen ($n = 10$)

Category	Summary of Criteria	Downgrades	Rationale
Initial Rating of Human Evidence = "Moderate"			
Risk of Bias	Study limitations- a substantial risk of bias across body of evidence.	-1	Downgraded because of "high" or "probably high" risk of bias for air pollution exposure assessment for six studies and "high" or "probably high" risk of bias for pollen exposure assessment for six studies.
Indirectness	Evidence was not directly comparable to the chosen population, exposure, comparator, and outcome.	0	Measured outcomes were assessed for humans in the populations for the duration of study periods, as outlined in the PECO statement.
Inconsistency	Wide variability in estimates of effect in similar populations.	0	The studies were inconsistent in pollen types and air pollutants, precluding judgment as to whether reported effect estimates would be consistent or inconsistent.
Imprecision	Studies had a small sample size and small outcome count.	0	The studies had large sample sizes with adequate samples for outcomes during study periods.
Publication Bias	Studies missing for body of evidence, resulting in an over or underestimate of true effects from exposure.	0	The studies were large studies that varied in year, data sources, and methods of statistical analysis that appeared to report outcomes found regardless of results.
Category	**Summary of Criteria**	**Upgrades**	**Rationale**
Large magnitude of effects	Study found confounding alone unlikely to explain association with large effect estimate as judged by reviewers.	0	Studies that reported positive associations of interactions reported effect estimates with low magnitudes.
Dose-response	Consistent relationship between dose and response in one or multiple studies, and/or exposure response across studies	0	Studies did not report a consistent relationship between dose and response.
Confounding minimizes effect	Upgraded if consideration of all plausible residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect.	0	No evidence that residual confounders or biases would underestimate the effect or suggest a spurious effect when results show no effect
Overall Quality of Evidence		Low	The overall quality of the evidence supporting interactive effects is low.
Overall Strength of Evidence		Limited	An association was shown in a few studies between air pollution and pollen and increased outcomes, however the results were inconsistent and there was a potentially high risk of bias from the exposure assessments in several studies.

large datasets of pollutant levels and meteorological data, including from the National, Morbidity, Mortality, and Air Pollution Study (NMMAPS) in the U.S. and the Ultrafine Particles and Health Study Group in Europe. An additional strength across all categories was that a majority of the studies had a low risk of bias for study design, with many of them using a time series design.

Though there were some strengths in the literature, we also found serious weaknesses that precluded our ability to draw strong conclusions as to the existence of interactive health effects from simultaneous exposure to these risk factors. Limitations included that all of the studies we found were short-term studies that were unable to address effects of long-term exposure. We found no cohort studies that could properly attribute exposure at an individual level and account for health outcomes that may take years to manifest. In addition, exposure measurements and metrics for air pollutants, pollen, and temperature were inconsistent and not standardized between the studies. Judging the potential bias from exposure measurement for air pollution, temperature, and pollen is difficult with only limited information available in the papers. For example, some papers did not report the number of monitoring stations used to assign exposures or the length of time for which the exposure data were collected. Recent studies of air pollution have begun using more sophisticated methods to assign exposure, such as models that use satellite remote sensing or land use variables that provide greater spatial coverage compared with ground monitors such as those run by government monitoring networks [104–106]. For pollen, the studies in this review all used pollen count as the exposure metric, which may not account for pollen potency [23]. Another limitation is that many studies were missing information about confounders that were considered, which could influence the magnitude of the associations they found. Finally, while we restricted our review to studies that looked at interaction between two of the three hazards, several studies may have treated these risk factors as mediators or effect modifiers. Future research should explore the role of these issues. Additional research should also explore effects of these risk factors on additional health outcomes, such as birth outcomes, as well as vulnerable populations, including children, the elderly, pregnant women, and people with genetic predisposition to cardiovascular and respiratory disease.

We included only heat, air pollution, and pollen in this review, as they are all conditions of the ambient air for which we judged there to be enough epidemiological literature to assess. Other important environmental drivers of disease related to the ambient air that we did not include here are occupational exposures; different types of air pollutant mixtures (including from different combustion sources and different composition of particulate matter); and exposure to airborne bacteria, viruses, molds, and fungus. In reality, people are exposed to a complex set of risk factors that remain poorly defined and explored in the literature. In addition, the chronic diseases considered affected by these risk factors are multi-factorial with heavy influence from genetic and lifestyle (e.g. diet, exercise) factors. Our literature review highlights the importance of including environmental factors in epidemiological and risk assessment studies, even if strong conclusions cannot yet be drawn from the current set of available studies.

Conclusions

In this systematic literature review of epidemiological studies, we found evidence for synergistic effects of heat and air pollutants (particularly for ozone and particulate matter), but not for the combination of heat, air pollution, and pollen together or of air pollution and pollen or heat and pollen. Our findings support consideration of combined effects of heat and air pollution in assessing health impacts from these risk factors in the present day and in the future as climate change progresses. However, the literature is too nascent to support inclusion of interactive effects between air pollution and pollen or heat and pollen in risk assessments. Future research should continue to explore potential interactive effects of environmental exposures on human health, as people are often exposed to multiple environmental risk factors simultaneously. This is a rapidly evolving field of study, and our review and conclusions should be updated to include new evidence as it becomes available. If new evidence supports our conclusion that heat and air pollution exposure act synergistically on human health, the health impacts from climate change-driven increases in air pollution and heat exposure may be larger than previously estimated in studies that consider these risk factors individually.

Supplementary Information

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Additional file 1.

Abbreviations

CO: Carbon monoxide; GRADE: Grading of Recommendations Assessment, Development and Evaluation; NO2: Nitrogen dioxide; O3: Ozone; PECO: Population, Exposure, Control, Outcome; PM2.5: Fine particulate matter; PM10: Coarse particulate matter; SO2: Sulfur dioxide

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Authors' contributions

S.C.A. conceived of the study, oversaw the analysis, and was responsible for drafting the manuscript. S.H. conducted the literature review, evaluated risk of bias and strength and quality of the evidence, and wrote much of the manuscript. E.W. evaluated risk of bias. N.N. and P.K. reviewed the analysis and contributed to the manuscript writing. The author(s) read and approved the final manuscript.

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Availability of data and materials
All data are available within the article and supplemental material.

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Not applicable.

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Competing interests
The authors declare that they have no competing interests.

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Budget and Policy Post

March 8, 2017

Residential Water Use Trends and Implications for Conservation Policy

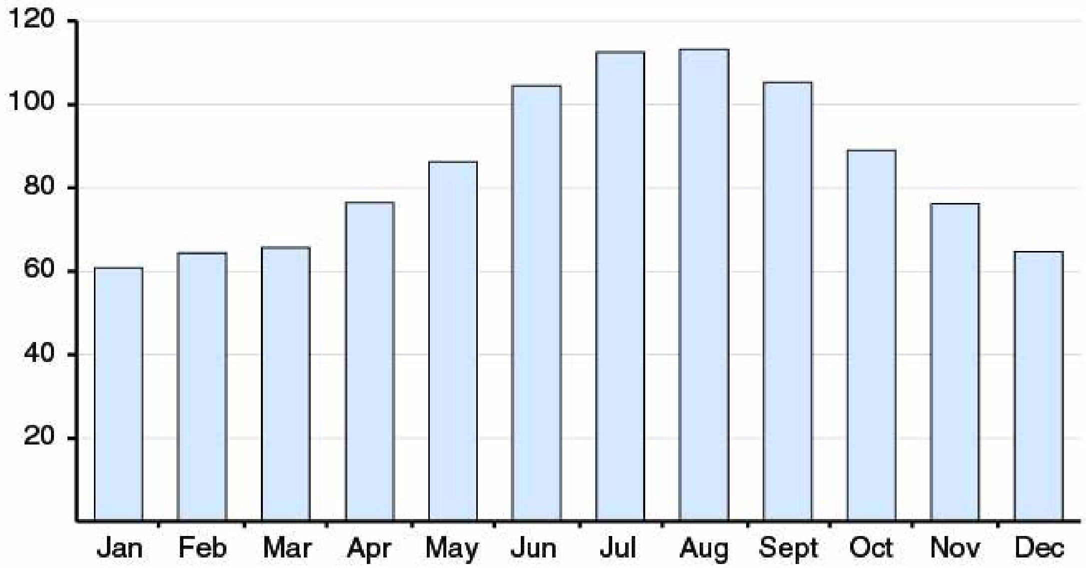
For the past two years, urban water agencies in California have been submitting monthly data on residential water use to the State Water Resources Control Board (SWRCB). The monthly update covering December 2016 was recently published on the board's website <http://www.waterboards.ca.gov/water_issues/programs/conservation_portal/conservation_reporting.shtml> , providing a full year of data for 2016. This web post discusses trends in residential water use and what these data imply for policymakers in the coming year.

Average Residential Water Use in 2016: 85 Gallons Per Person Per Day. The reported data shows that on average Californians used 85 gallons of water per person per day in 2016. As shown in Figure 1, water use was highest in the summer months of June through September, where it averaged 109 gallons per person per day. By comparison, during the cooler and wetter months of January through March of 2016, average per capita water use was only 64 gallons per person per day.

Figure 1

Urban Water Use Highest in Summer

Average Per Capita Use Per Day in Gallons, 2016

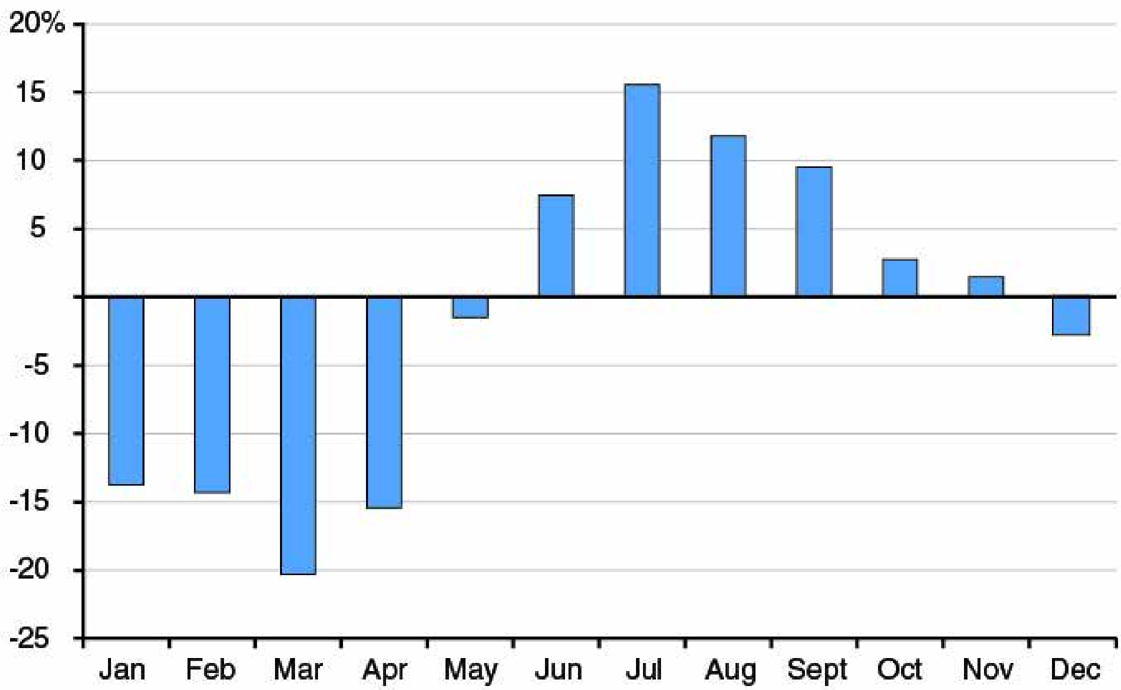


Water Use Higher in Last Half of 2016 Compared to 2015 . . . One notable trend highlighted in the data is the increase in per capita residential water use in the second half of 2016—particularly the summer months—compared to the same months in 2015. In the first four months of 2016, per capita water use was an average of 15 percent lower than the same months in 2015. However, as shown in Figure 2, in the summer months of 2016, per capita water use was an average of 11 percent higher than the same months the prior year. As discussed above, these months are when residential water use tends to be highest. A likely explanation for this shift is that in May 2016 the SWRCB eased mandatory water restrictions <http://www.waterboards.ca.gov/water_issues/programs/conservation_portal/docs/2017feb/fs020817_emergency_reg.pdf> that had been in place for urban water agencies since June 2015. These restrictions were eased (and replaced with a self-certification process, which was recently extended) because of improved water conditions. More recently, over the final three months of 2016, residential water use was similar to the amounts used in the same months of 2015, probably due to significant precipitation in October and December.

Figure 2

Residential Water Use Increased After Mandatory Restrictions Eased[a]

Percent Change in 2016 Compared to 2015

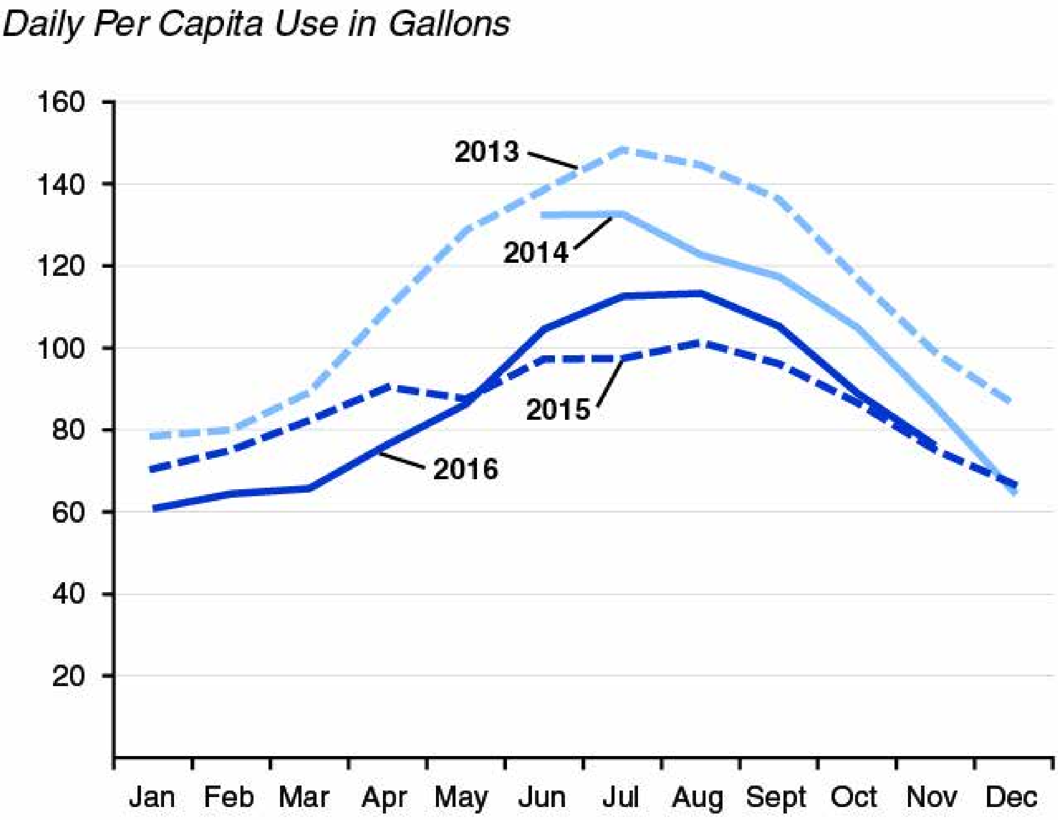


[a] Restrictions were eased by the State Water Resources Control Board in May 2016.

. . . But Significantly Below Usage in 2013 and 2014. Importantly, even though statewide use was higher in last year's summer months compared to 2015, it was significantly lower than in prior years. For example, residential usage in the summer of months of 2016 was 30 percent lower than in the same months of 2013 and 13 percent lower than in the same months of 2014. The fact that summer usage was lower in 2016 than in those earlier years probably reflects several factors, including (1) the ongoing effects of permanent conservation efforts (such as turf replacements); (2) continuation of some local agencies' conservation efforts (such as limiting the number of days people can water lawns); and (3) continuation of behavioral changes by Californians (such as taking shorter showers). Figure 3 shows statewide residential per capita water use by month for most of the past four years. (Only partial year data is available for 2014.)

Figure 3

Residential Water Use Remains Lower Than in 2013 and 2014[a]



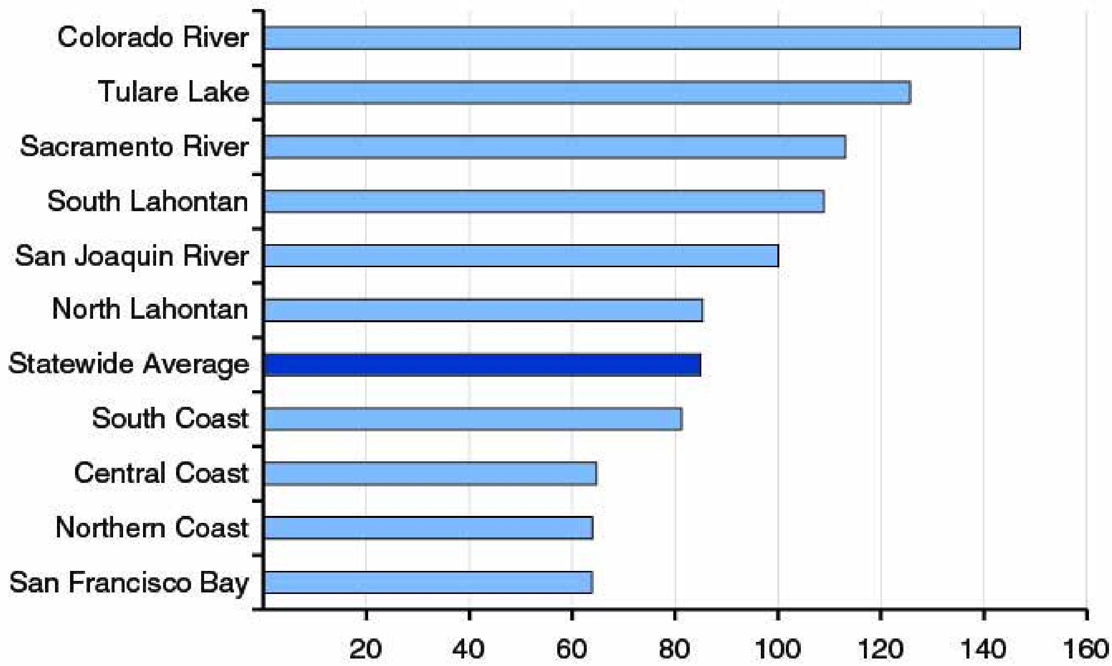
[a] Only partial year data available for 2014.

Use Varies Significantly Across State. California is divided into ten hydrologic regions <http://www.ppic.org/main/mapdetail.asp?i=1576> . Historically, residential water use in these areas has varied, and this remained true in 2016. As shown in Figure 4, average residential water use in 2016 ranged from a low of 64 gallons per day in the San Francisco Bay region to 147 gallons per day in the Colorado River region.

Figure 4

Average Residential Water Use Varies by Region

Daily Per Capita Residential Use in Gallons in 2016



Importantly, all ten regions saw increases in residential water use since SWRCB lifted the mandatory restrictions, but the amount of increase has varied by region. In particular, the Colorado River, Sacramento River, North Lahontan, and Tulare Lake regions all saw per capita water use increase by at least 15 percent during 2016's summer months compared to the same months in 2015. (We note, however, that the Sacramento River and North Lahontan regions saw use reductions in the fall of 2016 compared to the same months in 2015.)

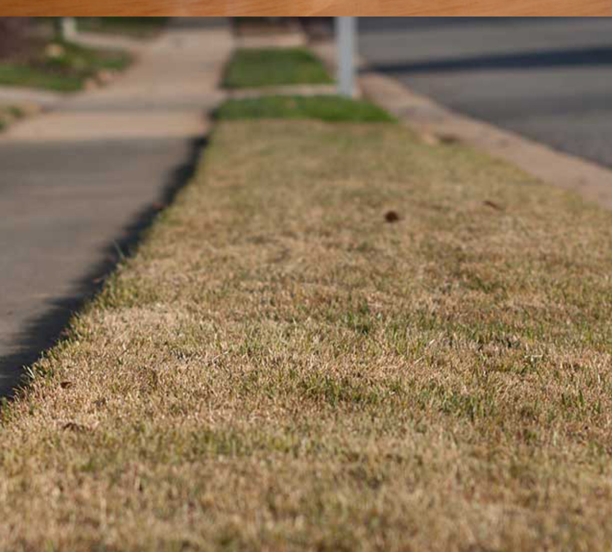
Precipitation Data Suggests 2017 Will Be a Very Good Water Year for California. Fortunately, recent data suggest that the 2017 water year (October 2016 through September 2017) is very likely to be significantly better than recent years. Precipitation has been far above average throughout much of the state. As of mid-February 2017, many of the state's largest reservoirs <http://cdec.water.ca.gov/cgi-progs/products/rescond.pdf> were filled well above historical averages for this time of the year. In addition, snowpack measurements in early March 2017 found that average snowpack in the state was 185 percent of historical averages for that time of the year.

Drought Resiliency Likely to Still Be Part of Policy Conversation. Despite these positive signs for 2017, there are several reasons why it will be important for policymakers and program managers to continue to monitor conditions and discuss what, if any, conservation and other measures should be implemented to protect the state's water supplies for urban, agricultural, and environmental uses. First, as of the beginning of 2017, there continue to be some areas of the state that face ongoing negative effects that have accumulated due to multiple years of low precipitation. This includes dry and contaminated wells that reduce people's access to clean drinking water, as well as irreversible land subsidence caused by overdrafting of groundwater during the drought. Second, it will be important to assess the short- and longer-term effectiveness of water conservation and other activities on water use patterns by residential—as well as agricultural—users. While drought conditions have been mostly alleviated in 2017, such data would be valuable in helping the state respond to the next drought. Third, it will be important to track the health of sensitive ecosystems and species. In recent years, for example, the drought has negatively impacted endangered fish species and contributed to increased tree mortality throughout the state. In many cases, it is too early to tell how quickly affected habitats and species will recover following multiple years of drought.

In November 2016, the administration released a draft report <http://www.water.ca.gov/wateruseefficiency/conservation/docs/EO_B-37-16_Report.pdf> identifying additional steps it recommends taking to increase long-term conservation efforts around the state. These include a range of proposed actions that could require new legislation, additional funding, and coordination of state and local agencies. The administration believes that these actions would increase the state's resilience and ability to respond to droughts in the future. More recently, the Governor's 2017-18 budget proposed an additional $178 million in one-time funding for resources and other agencies—primarily from the General Fund—for continued drought-response activities. As we discuss in our review of the Governor's budget proposals <http://www.lao.ca.gov/reports/2017/3558/resources-environment-budget-021517.pdf> for resources and environmental protection agencies, we recommend approving some of the requested funding—and consider providing some of it on an ongoing basis—to address current conditions and increase the state's resilience in future droughts.

AUG 2022

CALIFORNIA'S WATER SUPPLY STRATEGY
Adapting to a Hotter, Drier Future



CALIFORNIA FOR ALL

CALIFORNIA NATURAL RESOURCES AGENCY

CALIFORNIA Water Boards

CalEPA California Environmental Protection Agency

cdfa CALIFORNIA DEPARTMENT OF FOOD & AGRICULTURE

Introduction

Our climate has changed. We are experiencing extreme, sustained drought conditions in California and across the American West caused by hotter, drier weather. Our warming climate means that a greater share of the rain and snowfall we receive will be absorbed by dry soils, consumed by thirsty plants, and evaporated into the air. This leaves less water to meet our needs.

This is our new climate reality, and we must adapt.

During his first months in office, Governor Newsom issued an **executive order** calling on State Agencies to create a comprehensive **Water Resilience Portfolio**. The Portfolio prioritized key actions to secure California's water future. *Over the last two years we've **made major progress** that includes*: working to bring our groundwater basins into balance; updating infrastructure to move water throughout the state; restoring river systems, including the nation's largest dam removal effort on the Klamath River; and improving water management through new voluntary agreements and technology investments.

California is investing billions of dollars into these actions to secure the future of California's water supply.

Over the last three years, **state leaders have earmarked more than $8 billion to modernize water infrastructure and management**. The historic three-year, $5.2 billion investment in California water systems enacted in 2021-22 has enabled emergency drought response, improved water conservation to stretch water supplies, and enabled scores of projects by local water suppliers to become more resilient to current and future droughts. The 2022-23 budget includes an *additional* $2.8 billion for drought relief to hard-hit communities, water conservation, environmental protection for fish and wildlife, and long-term projects to permanently strengthen drought resilience.

Over the last two years, scientists and water managers have been alarmed by the accelerating impacts of the warming climate on our water supply. **We now know that hotter and drier weather could diminish our existing water supply by up to 10% by 2040**. So we are ***taking action***.

We have invested billions in securing the future of California's water supply and this focused *Water Supply Strategy* updates state priorities based on new data and accelerating climate change.

To ensure California has the water needed for generations to come, this Strategy includes:

- **Create storage space for up to 4 million acre-feet of water**, allowing us to capitalize on big storms when they do occur and store water for dry periods

- **Recycle and reuse at least 800,000 acre-feet of water per year by 2030**, enabling better and safer use of wastewater currently discharged to the ocean

- **Free up 500,000 acre-feet of water** through more efficient water use and conservation, helping make up for water lost due to climate change

- Make new water available for use by **capturing stormwater and desalinating ocean water and salty water in groundwater basins**, diversifying supplies and making the most of high flows during storm events

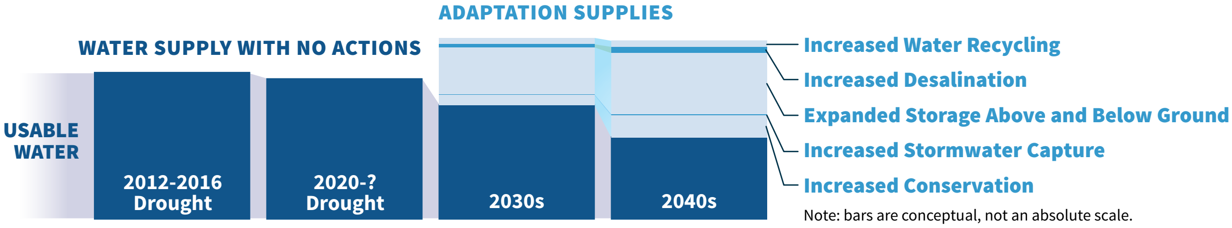
To match the pace of climate change, California must move smarter and faster to update our water systems. **The modernization of our water systems will help replenish the water California will lose due to hotter, drier weather, and generate enough water for more than 8.4 million households.**

CALIFORNIA'S WATER SUPPLY STRATEGY
Adapting to a Hotter, Drier Future

This document outlines California's strategy and priority actions to adapt and protect water supplies in an era of rising temperatures.

Over the next 20 years, California could lose 10 percent[1] of its water supplies.

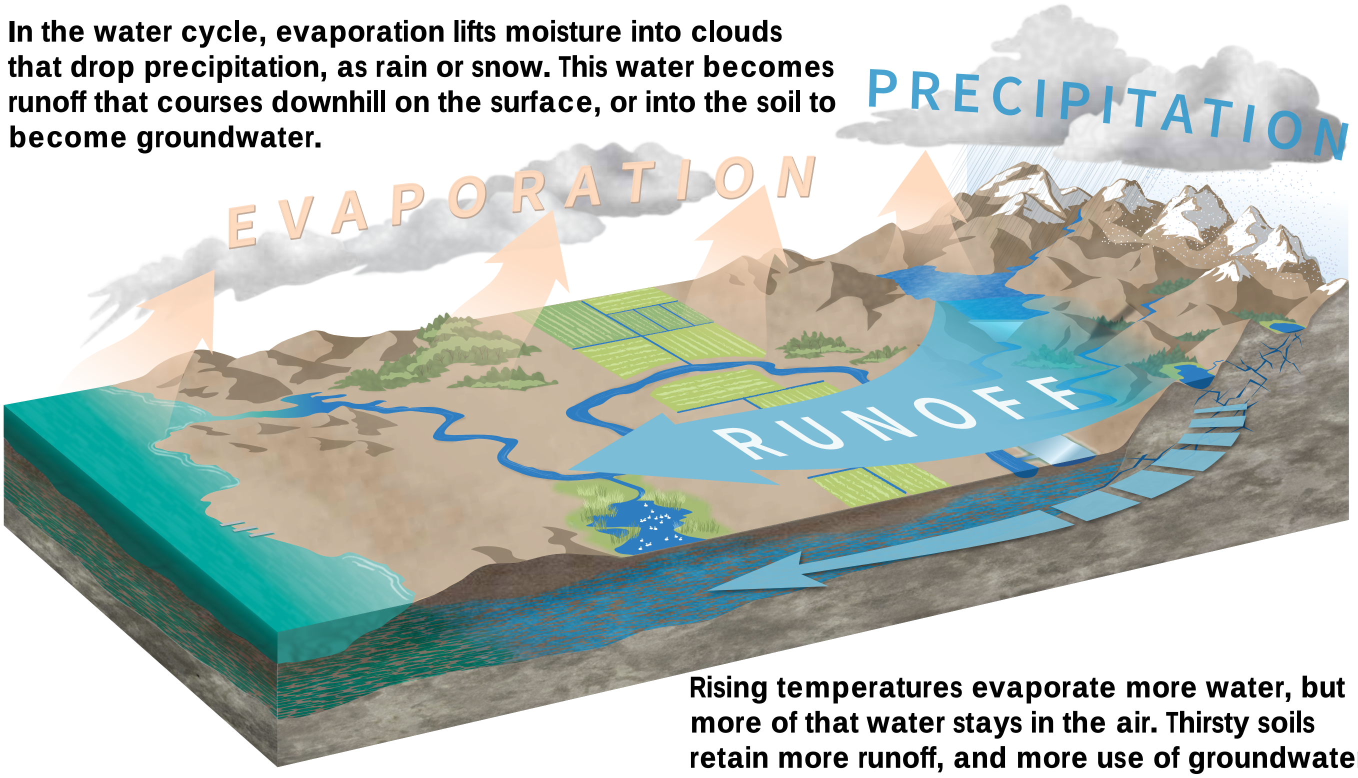
Our climate has changed, and the West continues to get hotter and drier. As it does, we will see on average less snowfall, more evaporation, and greater consumption of water by vegetation, soil, and the atmosphere itself.



In previous droughts the ratio of precipitation to evaporation to runoff has been similar. However, as temperatures rise, evaporation increases, with the consequence of a fall in runoff. As average temperatures continue to increase, the increase in evaporation will continue, with a concurrent drop in runoff.

The coming water cycle: the air claims more

In the water cycle, evaporation lifts moisture into clouds that drop precipitation, as rain or snow. This water becomes runoff that courses downhill on the surface, or into the soil to become groundwater.



Rising temperatures evaporate more water, but more of that water stays in the air. Thirsty soils retain more runoff, and more use of groundwater requires more water for recharging watertables.

[1] DWR estimates a 10% reduction in water supply by 2040. This is a planning scenario that considers increased temperatures and decreased runoff due to a thirstier atmosphere, plants, and soils. According to the California Water Plan Update, California's managed water supply ranges from 60-90 MAF per year so the effect of a drier climate results in a disappearance of about 6-9 MAF of water supply.

California's precipitation always has swung between drought and flood. Those swings are becoming more severe. Regardless of drought or flood, in this changed climate there will be less water available for people to use than there would have been in a cooler climate because of the way plants, soils, and the atmosphere use water as temperatures rise.

The volume of water used by people in California for agriculture, urban, and environmental purposes ranges from 60 million acre-feet per year to 90 million acre-feet per year. A loss of 10 percent of that volume to hotter, drier conditions could mean the disappearance of about six million acre-feet to nine million acre-feet of water supply. For comparison's sake, California's largest reservoir – Shasta – holds 4.5 million acre-feet.

Water underpins much of what we care about as Californians. To thrive and grow as a state, we will have to make up for a loss of supply. We must innovate, conserve, store, reuse, and repurpose water.

This document outlines four sets of actions the State will pursue to prepare California for its new climate reality.

These targeted actions aim to secure supplies for people, so that homes, schools, and businesses do not suffer disruptions, and the state's agricultural economy continues to thrive.

In concert with these actions, the State is working to protect fish and wildlife populations by removing stream barriers, restoring aquatic habitat, bolstering stream flows at ecologically important times, and expanding floodplains and wetlands.

The State also continues to make progress extending clean, safe drinking water to all Californians; in the last three years, the number of people impacted by failing water systems has fallen from 1.6 million to 934,000, and the state has delivered emergency drinking water assistance to 9,456 households and 150 water systems in this drought.

The actions in this strategy aim primarily to support the urban and suburban water systems that serve most Californians and to stabilize water supplies for agriculture. But benefits from these actions will extend to environmental protection and fulfillment of the right of every Californian to safe drinking water, and the State continues to advance those efforts apart from this strategy.

How California is taking action to protect community water supplies

The Water Resilience Portfolio has guided State water policy since July 2020 and will continue to do so. It is a comprehensive suite of actions that support local water resilience. However, the record-breaking temperatures and aridity of the 2012-16 drought, followed so closely by another stretch of similar conditions beginning in the winter of 2020-21, send a strong climate signal that we must heed. These new, more extreme conditions make clear that to secure water supplies, we must double down on a set of actions within the Water Resilience Portfolio, with haste.

Executing this strategy will require coordination with local, tribal, and federal partners to:

1) Develop new water through recycling and desalination.

2) Capture and save more stormwater, above ground and below ground.

3) Reduce use of water in cities and on farms.

4) Improve all water management actions with better data, forecasting, conveyance, and administration of water rights.

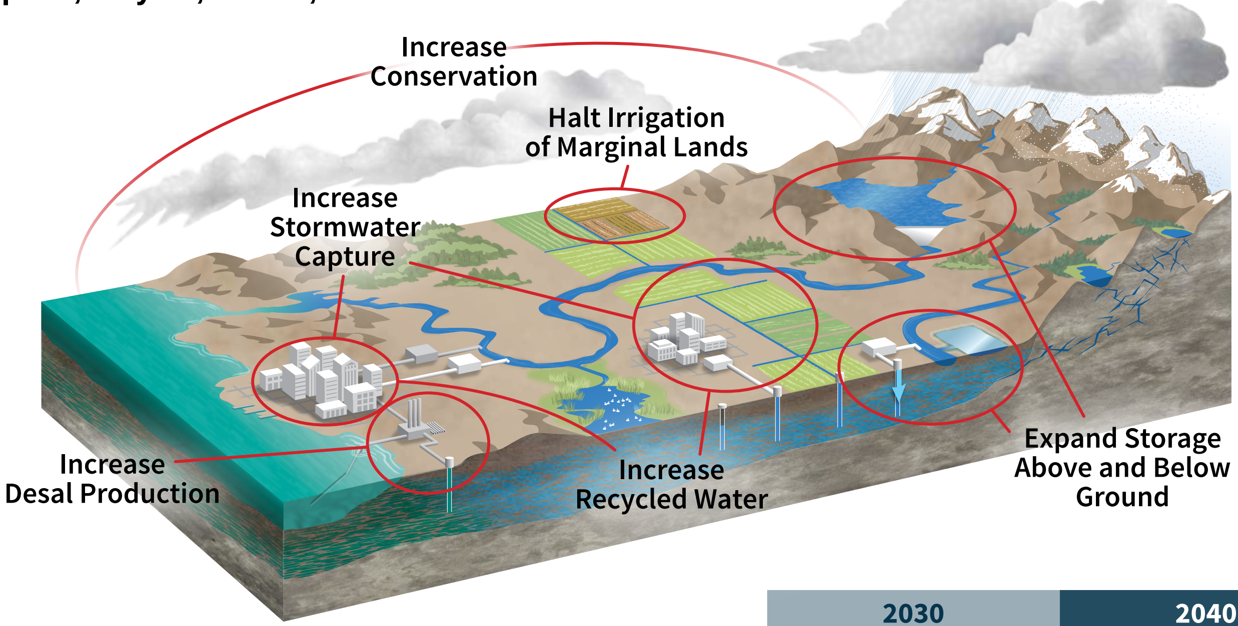
1. Develop New Water Supplies

Investments in wastewater recycling and desalination technology can help drought-proof communities.

1.1 Reuse at least 800,000 acre-feet of water per year by 2030 and 1.8 million acre-feet by 2040, with most of that additional recycling involving direct wastewater discharges that are now going to the ocean.

Closing the evaporative gap

To offset increased evaporation tied to warmer average temperatures, California must capture, recycle, de-salt, and conserve more water.



	2030		2040	
Increase Recycled Water	.8 MAF	About 5 MAF	1.8 MAF	About 7 MAF
Increase Desal Production	28,000 AF		84,000 AF	
Increase Stormwater Capture	.25 MAF		.5 MAF	
Increase Conservation	.5 MAF		.5 MAF	
SUBTOTAL FOR RECYCLED, DESAL, STORMWATER AND CONSERVATION	1.6 MAF		2.9 MAF	
Expand Storage Above and Below Ground*	3.7 MAF		4 MAF	
Total	4.8 MAF		6.9 MAF	

*Additional storage capacity does not equate to a similar volume of new water supply. MAF – million acre-feet.

Currently, recycled water offsets about nine percent of the state's water demand, about 728,000 acre-feet per year. The State Water Resources Control Board (State Water Board) has invested a total of $1.8 billion in recycled water projects statewide over the last five years that are in various stages of development. Once completed, those projects will generate an additional 124,000 acre-feet of new water supply.

Approximately 1.5 million acre-feet per year of treated wastewater is currently discharged to California's ocean waters. Not all of this can be recycled, as some water is needed to discharge brine, and wastewater in some places provides critical streamflow for fish and wildlife. But in many places, communities can tap this resource to build water supply resilience.

Current regulations enable communities to use recycled water for drinking via a reservoir or aquifer, and in 2023, the State Water Board will establish direct potable reuse regulations that allow suppliers to distribute recycled water without first putting it into a reservoir or aquifer.

Implementation Steps:

- The State will consider greater investments and leverage federal dollars where possible to build on the $3.2 billion in financing for water recycling projects that the State Water Board has provided to 94 projects since 2012. At roughly $15,000 an acre-foot, it would require a state, local, and federal investment of approximately $10 billion to achieve the 2030 goal and $27 billion to achieve the 2040 goal of recycling an additional 1.8 million acre-feet of water.
- By January 1, 2024, the State Water Board will work with local water and sanitation agencies to identify recycled water projects that hold the potential to be operational by 2030 and by no later than 2040.
- The State Water Board will formalize a process currently underway by convening a strike team to identify and resolve permitting and funding obstacles.
- The State Water Board will track the permitting and funding status of recycled water projects with a public, digital dashboard.
- The State will support local water sustainability plans that include water recycling, including (but not limited to):
  - Operation NEXT/Hyperion 2035 (City of Los Angeles)
  - Pure Water San Diego (City of San Diego)
  - Integrated Water Resources Plan and Climate Action Plan (Metropolitan Water District of Southern California)
  - Water Supply Management Program 2040 (East Bay Municipal Utility District)
- The State Water Board will act on direct potable reuse regulations by December 2023.

1.2 Expand brackish groundwater desalination production by 28,000 acre-feet per year by 2030 and 84,000 acre-feet per year by 2040 and help guide location of seawater desalination projects where they are cost effective and environmentally appropriate.

There are 14 seawater desalination plants across the state, with a combined production capacity of approximately 89,000 acre-feet per year. Some are not operating at full capacity and could be positioned to generate additional water supplies in drought, much as "peaker" power plants operate in short bursts to support electricity reliability at times of peak demand. Another 23 brackish groundwater desalination plants have a combined production capacity of 139,627 acre-feet per year. Brackish groundwater requires significantly less energy to treat than seawater.

Proposals to build desalination projects along the coast must be approved under the Coastal Act, in addition to other regulatory requirements. As California becomes hotter and drier, we must become more resourceful with the strategic opportunity that 840 miles of ocean coastline offer to build water resilience.

Implementation Steps:

- By January 1, 2024, the Department of Water Resources (DWR) and the State Water Board, in coordination with local agencies, will identify the brackish desalination projects that have the potential to be operational by 2030 and by no later than 2040. The State will consider investing in grants to local agencies for planning and building desalination projects.
- By January 1, 2024, the State Water Board will review groundwater basins impaired by salts and nutrients and determine the volume of water available for brackish groundwater desalination.
- As the State's representative on the U.S. Department of Energy's five-year, $100 million desalination innovation hub, DWR will continue to guide research investments towards technological breakthroughs that solve California desalination challenges.
- The State will help streamline and expedite permitting to provide better clarity and certainty to further desalination projects. To this end, by June 30, 2023, the State Water Board, Coastal Commission, DWR and other State entities (e.g. State Lands Commission) will develop criteria for siting of desalination facilities along the coast and recommend new standards to facilitate approval.
- Within the following year, these agencies will identify potential available mitigation sites to facilitate the expedited approval of desalination facilities. The State Water Board will consider amendments to the Desalination Policy in its Ocean Plan to streamline permits that meet the recommended siting and design standards for projects located in the identified priority areas.

2. Expand Water Storage Capacity Above and Below Ground by Four Million Acre-Feet.

While creating more space to store water in reservoirs and aquifers does not create more precipitation, and though having enough rain and snowfall to fill storage space is out of our control, we need diversion infrastructure, more places to park runoff, and the conveyance to eventually move the water to where it is needed to take advantage of fast-moving storms. Expanding storage capacity improves the ability to capture runoff when diversions cause the least harm to the environment. Furthermore, apart from a hotter and drier climate, capturing water runoff is needed to help correct decades of over-pumping of groundwater basins.

2.1 Expand average annual groundwater recharge by at least 500,000 acre-feet.

Vast capacity to store water exists underground in California. Intentional, directed recharge of groundwater is one of the fastest, most economical, and widely available ways to harness the bounty of wet years to cope with dry years. It has the additional advantage of helping to halt or prevent land surface collapse due to over-pumping, which can damage roads, canals, and bridges. Expanding groundwater recharge requires adherence to laws, so that the environment and water users upstream and downstream are not harmed when streamflow is directed underground. With the multi-faceted suite of actions below, the State intends to help local water agencies to accelerate the pace and scale of groundwater recharge. These actions center on helping local agencies understand the best locations for recharge, analyze the impact of their recharge proposals on the environment and other water users, and expeditiously permit their projects.

Local agencies are developing groundwater recharge projects around the state. By the end of next year, the State cumulatively will have invested $350 million in local assistance for recharge projects. In planning documents, local agencies have proposed more than 340 new recharge projects that, if built, could result in as much as 2.2 million acre-feet of additional stored water in a single wet year by 2030. Until those projects are permitted, it is unclear how much water those projects will have the capacity to divert to underground storage; multiple proposals may rely on the same sources of unappropriated water. But an additional 500,000 acre-feet is a reasonable estimate of the additional average annual recharge volume that may be obtained after these projects are vetted, permitted, and constructed.

California must be ready to respond to future wet winters. Fortunately, several processes already are in place that could be used to divert water from high-flow events to underground storage. Additional outreach, education, and technical assistance will be critical for preparing diverters for a potentially wet winter so that permits can be put in place before the start of the rainy season.

Should local actions become too fragmented or inefficient to maximize recharge opportunities, the state should consider a coordinated, state-level approach to provide for orderly, efficient disbursement of rights to high winter flows.

Implementation Steps: To help achieve this target, DWR and the State Water Board will continue to provide regulatory and technical assistance to local agencies that have received State funds to ensure that groundwater recharge project proponents can successfully navigate the regulatory processes. The State will weigh the following actions. Some would require additional investments and, possibly, regulatory changes.

- **Outreach:**
  - DWR and the State Water Board will conduct a series of outreach activities to highlight temporary permitting pathways in advance of winter, to assess the status of

proposed recharge projects, and to better align state and local agencies to advance groundwater recharge. The outreach would focus on the use of an existing 180-day temporary permit process and would note that permit applications should be received no later than October 1 to be ready for diversions in January.

- By December 2022, DWR will evaluate a process whereby it files for 180-day temporary permits in certain watersheds on behalf of local agencies, in order to advance the development of the permit terms and conditions. DWR also would pay the filing fee, which could help facilitate local willingness to participate.

Technical Assistance:

- DWR will provide outreach and assistance to help connect potential diverters with State Water Board permitting staff to answer specific questions and provide information that enables effective permit applications.
- By October 2022, the State Water Board water right permitting staff will prioritize groundwater recharge permits.

- **Incentives:**
  - The State will weigh immediate and long-term incentives for recharge project applicants to pursue the State Water Board's streamlined recharge permitting pathway. Incentives could include:
  - Waiving of application costs partially or fully for a two-year period.
  - Connecting infrastructure funding to applications that use the State Water Board's streamlined underground storage permitting approach.
  - Prioritization of State funding for groundwater recharge projects that target high-flow events, which raise fewer concerns about the environment and other water right holders than projects that seek to capture water in "shoulder" seasons of spring, summer, and fall.
  - DWR will expand its watershed modeling tools to better assess water available for recharge on a watershed basis.
- **Regulatory Streamlining:**
  - The State will streamline water right permits for recharge projects receiving DWR grants or conducted under DWR's Flood-Managed Aquifer Recharge Program.
  - The State Water Board will develop permanent regulations for water availability analyses that specify methodologies, data, and alternatives for conducting such analyses.
  - The Administration will pursue legislation to revise the water right application process to deliver decisions more quickly.
- **State Administration of Potential Recharge Flows**:
  - DWR and the State Water Board will develop a mechanism to create a more consistent, economical, and equitable approach for allocation of water rights for groundwater recharge. The initial proposal would focus on the State securing all reasonably available future flood flows in the Central Valley, allowing the State to

then allocate the available water in an orderly, holistic, equitable, and integrated approach. The process would:

- Level the playing field for local agencies, especially those that lack the resources to navigate the water right process.
- Set clear water availability metrics for every potential applicant, allowing for fair comparisons among applicants.
- Address equity concerns, including, for example, the need to protect domestic wells or abate subsidence.
- Leverage other funding opportunities.
- Spur tight coordination between the State Water Board and DWR in the allocation of water rights.

2.2 Work with local proponents to complete the seven Proposition 1-supported storage projects and consider funding other viable surface storage projects.

Seven locally-driven projects are underway to increase the state's overall capacity to store water by 2.77 million acre-feet – nearly three times the capacity of Folsom Lake. The seven projects are on track to receive a combined $2.7 billion in state funding from Proposition 1, the 2014 water bond, once they meet the requirements imposed in the bond law. Four of the projects involve groundwater storage and three involve creation of a new or expanded reservoir. Two of these seven projects are likely to begin construction next year. Project proponents are working now to obtain permits, arrange financing, finalize environmental documents, and negotiate contracts with state agencies for the delivery of public benefits from the projects, including environmental flows.

Implementation Steps:

- To formalize, streamline and continue existing efforts, the California Natural Resources Agency and the California Environmental Protection Agency will establish an interagency strike team to facilitate state permitting and support completion of these projects.
- Water Commission staff will continue to monitor development of the seven Proposition 1 projects closely.
- Permit teams from the California Department of Fish and Wildlife (CDFW) and the State Water Board will continue working with applicants and with other state agencies to inform and advance the development of contracts for administration of public benefits.
- Water Commission, DWR, CDFW, and State Water Board teams will continue robust coordination. and working with applicants to draft and execute contracts for administration of public benefits.

2.3 Expand San Luis Reservoir by 135,000 acre-feet.

The federal government is proposing to expand San Luis Reservoir in Merced County to capture more winter storm runoff. In extremely wet years like 2017, San Luis fills and California misses an opportunity to capture and store even more water for use during subsequent dry years. The project would expand the capacity of the two-million acre-foot reservoir by 130,000 acre-feet -- enough to supply nearly 400,000 homes a year. DWR is working with the U.S. Bureau of Reclamation (Reclamation) on this proposed project and sees it as an important part of a set of inter-related joint projects to benefit the Central Valley Project and State Water Project, which include upgrading the San Luis Reservoir dam for earthquake safety, modernizing conveyance of water through the Sacramento-San Joaquin Delta, and restoring capacity lost due to subsidence at major Central Valley canals.

Implementation Steps:

- In December 2019, Reclamation and DWR announced a partnership to move forward on the seismic upgrade. Reclamation and DWR celebrated the groundbreaking of the project in June 2022. Construction is expected to finish in 2028. DWR will continue to work with Reclamation to complete the seismic upgrade and expansion.

2.4 Rehabilitate dams to regain storage capacity.

As of May, 112 California dams are rated “less than satisfactory” by State dam inspectors, and the reservoirs behind 41 of those dams cannot be filled beyond a certain level in order to protect public safety. The loss of storage is about 350,000 acre-feet per year. Accelerating dam safety repairs would help local water districts regain lost storage capacity and improve public safety. While this has historically been a federal or local obligation, the Legislature and Administration enacted additional funding to support dam owners faced with costly repairs.

Implementation Steps:

- DWR will administer the $100 million in the 2022-23 budget for local dam safety projects and flood management.

2.5 Support local stormwater capture projects in cities and towns with the goal to increase annual supply capacity by at least 250,000 acre-feet by 2030 and 500,000 acre-feet by 2040.

Over the last 30 years, an average of approximately 324,000 acre-feet of stormwater a year has been captured and recharged in communities in the South Coast alone. While this value varies from year to year, during the exceptionally wet winter of 2004-05 over 900,000 acre-feet of runoff was captured and infiltrated into the local groundwater basins.

The size, cost, and feasibility of stormwater capture projects vary greatly by location. It is extremely difficult for stormwater agencies to accurately measure stormwater capture volume and to predict potential due to uncertainties with annual precipitation.

Implementation Steps:

- Through permitting and funding, the State will incentivize local agencies to develop stormwater capture projects and help offset the cost of completing these projects, including through stormwater crediting systems to encourage public-private partnerships.

- The State Water Board will hire a contractor to provide an estimate of current stormwater capture and use statewide and then re-evaluate every five years progress towards the 2030 and 2040 goals.

3. Reduce Demand

3.1 Build upon the conservation achievements of the last two decades to reduce annual water demand in towns and cities by at least half a million acre-feet by 2030.

During the 2012-2016 drought, Californians did their part to conserve water, with many taking permanent actions that continue to yield benefits; per capita residential water use statewide declined 21 percent between the years 2013 and 2016 and has remained on average 16 percent below 2013 levels as of 2020. Californians need to step up again in this current drought. The State set a target of 15 percent for statewide conservation. Californians have made progress toward that goal in the summer of 2022, but more is needed to cope with the intense drought at hand and for the long term.

California enacted laws in 2018 to set new efficiency standards for how people use water in homes and businesses in ways that make sense in each region. These standards will drive fully-efficient water use in communities and eliminate water waste, even as communities continue to grow. The 2018 legislation calls for these standards to be met by 2030. The State Water Board is on track to set those new standards, informed by extensive data collection and analysis and recommendations from DWR. The recommended standards for indoor and outdoor water use for residential, commercial, industrial, and institutional water use could save 450,000 acre-feet per year starting in 2030. This amount of water would support 1.35 million homes, and the savings would prevent urban water use from rising as much as it would otherwise as population grows and more housing is built. These new standards would not apply to individual Californians, but local water suppliers must ensure the standards are met.

Given the acute need to conserve water in a potentially fourth dry year, the State Water Board will develop emergency conservation measures that would expedite implementation of conservation in a way that is already mandated through the 2018 laws. If drought conditions persist, the new short-term requirements could take effect no later than spring 2023. The new requirements would consider the relative efficiency of each supplier. These new efficiency targets would therefore work as a bridge to take California from voluntary measures to efficiency-based, water-use budgets that account for differences in climate zones, landscape area, population, and other factors.

In addition, the Administration sponsors a robust campaign to motivate urban Californians to save water and is working to accelerate the transition of turf to landscapes that use less water. To this end, the State will partner with local agencies to convert 500 million square feet of ornamental turf by 2030, with corresponding investments in programs and policies that incentivize turf conversion. Removal of 500 million square feet of turf could generate 66,000 acre-feet of water savings each year at an estimated cost of $1 billion.

Implementation Steps:

- The State Water Board will develop short-term efficiency-based conservation targets for every urban retail water supplier based on their unique characteristics like climate zone, water demand, residential landscape area, and population. The Board will compare water suppliers' actual use to their estimated efficient use target and assign them a percent reduction, with a higher reduction target for suppliers whose actual use is further from their efficient use target.
- DWR and the State Water Board will target grants to help local water districts achieve efficiency targets, using funding recently approved by the Legislature.
- The State-run Save Our Water campaign will continue to educate Californians about the severity of the current drought and the need to make water conservation a permanent, daily practice.
- DWR will establish a grant program to support local efforts to replace ornamental turf with drought-tolerant landscaping and—where schools and parks require turf—to make turf irrigation and maintenance more efficient, with a focus on disadvantaged communities.
- The State Water Board will advance adoption of new long-term water use efficiency standards, per existing statute (2018).
  - Once DWR provides its formal recommendations, the State Water Board will begin the process for enacting the regulation to ensure the rule will be in effect by January 1, 2024.

3.2 Help stabilize groundwater supplies for all groundwater users, including a more drought-resilient agricultural economy.

California irrigated agricultural acreage declined by 1 million acres between 2002 and 2017. The approximately eight million acres of irrigated farm and ranchland will shrink by at least an estimated additional 500,000 acres to one million acres between now and 2040 as local agencies transition to groundwater use that is sustainable over coming decades. The conserved water should support a more drought-resilient agricultural economy that retains its vitality.

Implementation Steps:

The State will:

- Continue to implement the Sustainable Groundwater Management Act (SGMA) to protect communities, agriculture, and the environment against prolonged dry periods and climate change, preserving water supplies for existing and potential beneficial use.
- Support local water demand management that includes changes to cropping patterns and fallowing by building upon this year's investment of $40 million in grants to regional organizations working to reduce groundwater reliance and create local environmental and economic opportunities through land-use changes.
- Continue to support conservation and water efficiency practices by agricultural producers.
- Support flexibility in local land use decisions to protect beneficial uses and users.

- Continue direct investment and technical assistance in drought relief for agriculture with dedicated funding to assist socially disadvantaged and underserved populations.

4. Improve Forecasting, Data, and Management, including Water Rights Modernization

Crucial to achieving the water supply actions described here is a common, readily-available set of facts about water supply and use, better forecasting, and integrated use of data and technology. Water rights modernization and reform are critical to ensuring we can efficiently and effectively adapt to a changing climate.

4.1 Improve data collection and modernize forecasts for a changed climate.

Sierra snowpack provides about a third of the water people use in California, yet the existing approach to forecasting snowmelt runoff dates to the 1950s.

To account for climate change, we must simulate the physics of interactions among the atmosphere, water as rain or snow, and the land surface – and we need to do this for individual watersheds, incorporating site-specific features like slope orientation and depth of soil. This requires timely data collection.

Implementation Steps:

The State will:

- Continue to invest in the human and technical resources needed to improve predictions and forecasting for water supply planning.
- Advance a multi-agency effort to install 430 new stream gages and upgrade or re-activate 200 more across the state. These gages provide real-time surface water data for enhanced drought management and flood response.
- Work with the U.S. Army Corps of Engineers leadership to accelerate the pace at which the manuals guiding reservoir operations are updated to reflect a changed climate.

4.2 Improve the flexibility of current water systems to move water throughout the state.

California depends upon aging, damaged, or increasingly risk-prone infrastructure to transport water between different areas of the state. Modern infrastructure and tighter coordination between the state's two major water projects would expand capacity to move water when it is available.

The state and federal water projects are fed by levee-lined channels in the Sacramento-San Joaquin Delta. This Delta infrastructure faces serious threat of failure due to storm surge, sea level rise, and earthquakes that could collapse levees. Loss of this water supply for any amount of time poses significant risk to farms, businesses, and most California homes. South of the Delta, major canals have been damaged by subsidence caused by the over-pumping of groundwater, restricting the capacity to move water when it is available.

DWR proposes to modernize State Water Project (SWP) conveyance in the Delta. Had the proposed project been operational in 2021, the project could have captured and moved an additional 236,000 acre-feet of water into San Luis Reservoir during that winter's few large storms.

Administrative hurdles also limit flexibility to move water. Every year for the last 10 years, the federal and state water projects have applied to the State Water Board for temporary flexibility in the locations where water diverted by either project may be used. These "consolidations of the authorized places of use" of the SWP and the Central Valley Project last only a year and require repetitive work by all parties involved. A permanent change to allow for consolidated place of use among the projects would make water transfers easier and lay the groundwork for discussions about future operation of the two projects.

Implementation Steps:

- DWR will advance the design of and the draft environmental impact report for the proposed Delta conveyance project, which would construct new intakes along the Sacramento River and a tunnel under the Delta to safeguard SWP deliveries and ensure that the SWP can make the most of big but infrequent storm events.
- DWR will disburse $100 million included in the 2022-23 state budget to support costs of repairing four major San Joaquin Valley canals damaged by subsidence.
- DWR and the State Water Board will chart a work plan to address the resources needed for preparation, submittal, and consideration of a joint place of use petition from the federal and state water projects.

4.3 Modernize water rights administration for equity, access, flexibility, and transparency.

The foundation of how California manages water rights dates to the Gold Rush and has not evolved in step with changing public values and management needs. The State Water Board is challenged to provide timely, useful, and meaningful information to guide state and local water management decisions, which are especially vital during periods of drought.

Other western states including Washington, Oregon, Nevada, and Idaho manage water diversions much more nimbly than California, which puts them in better position to adjust to what many call "aridification" – the transition to a drier climate. The ability to adjust diversions quickly also is crucial to protecting fish and wildlife, other water right holders, and public health. To make a century-old water right system work in this new era, the State Water Board needs accurate and timely data, modern data infrastructure, and increased capacity to halt water diversions when the flows in streams diminish. These improvements are a necessary predicate to modernize our water rights system in a manner that respects water right priorities and aligns with current public values and needs.

Implementation Steps:

The State Water Board will:

- Continue to build upon efforts started last summer with the investment of $30 million to digitize existing paper records and rebuild the state's water right data management system.

- Develop pilot projects in two or three watersheds over the next five years to collect real-time diversion data and integrate the data into the State Water Board's water rights data system, with lessons learned and outcomes used to inform statewide tools needed for administering an efficient and effective water rights system.
- Develop data and analytical tools for implementing the water right priority system for an estimated 10 to 15 watersheds.
- Support modeling staff to develop more robust supply/demand models for the Delta watershed.
- Consider adopting regulations that would allow for curtailments of water rights in years when there is not a declared drought emergency. The State currently lacks the authority in most years to implement the priority water rights system without a declared drought emergency.
- Support enforcement staff to help address illegal and unauthorized diversions during dry conditions.
- Consider regulations, legislation, and pursuing resources needed to streamline and modernize the water right system, clarify senior water rights, and establish more equitable fees.

Why target these actions?

The last three years of record-breaking drought made painfully real the hotter, drier pressures on water systems. These four major sets of actions would put to use water that would otherwise be unusable, stretch supplies with efficiency, and expand our capacity to bank water from big storms for dry times. They are designed, in other words, for a climate prone to weather whiplash.

These actions alone will not eliminate local water supply risk. The variability of rain and snow is too great, as is the uncertainty about which projects local agencies will implement. These actions aim to spur local agency adaptation to a new reality and change the way the State does business in order to better support local and regional water management efforts.

Who will carry out this strategy?

The state and federal governments each operate large water delivery systems in California, but local water districts and counties have primary responsibility for getting supplies to homes and businesses. Thousands of local and regional entities play a role in water management. Implementation of this strategy will require decisive state action. It will also require partnerships, as local agency leaders, federal partners, farmers, other business owners, and individual Californians are essential actors in carrying out this plan. To ensure successful implementation in such a decentralized system, the State must lead, set goals, provide incentives, and be prepared to exert greater authority when necessary.

The State will prioritize its funds and human resources to support local projects that satisfy state planning and permitting requirements to protect natural resources and help us collectively reach the targets outlined above. The State will invest in forecasting and data and water right administration – including real-time tracking of water use – to improve all water management actions by state, local, federal, and private entities. The State will also ensure that California's response advances equity and takes into account communities that are most at risk from climate change and that have experienced environmental injustices.

Water affordability is key to ensuring the human right to water – established in California law -- in the face of a hotter, drier state. The State has made strides in promoting affordability through provision of low-interest loans and grants to support infrastructure and planning for water systems, and by addressing pandemic-related water debt. However, the increased investments in infrastructure necessary to meet our future water supply needs will put additional pressure on affordability. The State will identify how best to support low-income households and address community affordability of water systems. Electric and communication utilities have programs to ease cost burdens on low-income members of the community, and it is important to address this in the water utility sector in a way that is workable and sustainable from a state budget perspective.

Where local agencies fail to build water resilience, the State will exert greater regulatory authority or work with the Legislature to gain authority to do so.

Moving Smarter and Faster

Climate change uniquely affects California's regions. This document articulates statewide targets for certain water management strategies, but achieving those overarching goals requires solutions at the local level, where the opportunities and challenges of each watershed vary tremendously. To encourage collaboration across watersheds that leads to greater statewide water resilience, the State will work with stakeholders and the Legislature to create:

- A funding program that incentivizes water users to develop regional targets for recycling, desalination, storage, efficiency, and other water management strategies.
- An expedited permitting path for water projects that help regions achieve those targets.

In order to deliver the pace and scale of projects necessary to meet this unprecedented climate challenge, we must modernize regulatory structures and expand staff capacity so that State agencies can assess, permit, fund and implement projects at the pace this climate emergency warrants.

The Administration will work with the Legislature and stakeholders to pursue the following:

- A more expeditious process for completing, reviewing and finalizing California Environmental Quality Act (CEQA) reviews and Water Code proceedings for critical water infrastructure projects to build drought and flood resilience.
- A voluntary permitting process for water infrastructure projects administered by the Governor's Office of Planning and Research (OPR). State agencies would retain authority to review, identify, and address environmental impacts, but the OPR would expedite the collective permitting process. This proposed process would not be an

option for water projects already under environmental review. The Administration would work with the Legislature to determine eligibility criteria for this voluntary process.

- Legislation, where appropriate, and regulations that would allow for curtailments of water rights in years when there is not a declared drought emergency. The State currently lacks the authority in most years to implement the priority water rights system without a declared drought emergency.

The Administration will:

- Develop water availability analysis guidelines for water right applications that account for high-flow periods on fully appropriated streams and the way climate change is shifting the seasonality and intensity of runoff. Develop permanent State Water Board regulations that specify the data and methodologies to be used for conducting such analyses in order to remove the current ambiguity about regulatory requirements.
- Establish a State Water Board, DWR and the California Department of Food and Agriculture "Groundwater Recharge Coordinating Committee" to jointly implement the groundwater recharge initiatives.
- Establish programmatic permitting for projects of a similar nature (such as water recycling or habitat restoration) in order to lower costs, simplify process, and speed permit approval.
- Institutionalize early alignment and regular internal coordination across state agencies on the permitting of water supply adaptation projects.

Conclusion

The world is getting hotter. The increased heat will intensify the natural swings in California's climate and shrink water supplies. Targeted state funds and focus will support local efforts to conserve, capture, recycle, and de-salt enough water to allow California communities to prosper in a hotter, drier climate.

Valley Water

2020

Urban Water Management Plan

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Santa Clara Valley Water District

2020 Urban Water Management Plan

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Valley Water

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CHAPTER 1 – INTRODUCTION AND OVERVIEW

OVERVIEW

Every five years, urban water suppliers in California are required by State law to prepare an Urban Water Management Plan (UWMP). The plan is a water agency's long-term water resource planning document to ensure that adequate water supplies are available to meet existing and future water needs within its service area. The UWMP provides an overall picture of a water agency's current and future water conditions and management over the next 25 years.

Santa Clara Valley Water District (Valley Water) is a special district that provides water resources management for Santa Clara County. Valley Water manages 10 dams and surface water reservoirs, three water treatment plants, an advanced recycled water purification center, nearly 276 acres of groundwater recharge ponds, and more than 275 miles of streams. Santa Clara County's population is expected to increase from nearly 2 million in 2020 to nearly 2.7 million in 2045, which drives future water demands.

Understanding water demands and how they may change over time allows Valley Water to manage the county's water supply and appropriately plan infrastructure investments. Due to expected population increases and job growth, county-wide demands are projected to increase from 306,000 acre-feet per year (AFY) in 2020 to approximately 345,000 AFY in 2045. The 2045 demand, while higher than present demand, is still down from a peak in the 90s and 2000s owing to significant conservation efforts from Valley Water and the State.

Valley Water maintains diverse water supply sources to meet countywide demands, including local surface water and groundwater, imported water, and recycled water. Water conservation is also an important part of the water supply mix, helping to keep water rates lower while improving water supply reliability. Valley Water is considering investing in projects to help mitigate potential future supply reductions from climate change and new regulations. Valley Water's Water Supply Master Plan 2040 (WSMP) provides a strategy for meeting future water demands, and Valley Water continuously uses an annual Monitoring and Assessment Program (MAP) to track WSMP strategy implementation. With the phased implementation of planned future projects, Valley Water's available supplies are projected to increase over time. Valley Water's many supply sources are subject to hydrologic variability and additional constraints including regulatory constraints, climate change, and water quality variations.

Based on Valley Water's existing and planned sources of supply, Valley Water will be able to meet countywide demands through 2045 under normal, a single dry, and five consecutive dry year conditions. If a five-year drought were to occur in the next five years, Valley Water would employ a range of response actions, including water conservation, bringing back water stored in the Semitropic Groundwater Storage Bank in Kern County, imported water transfers and exchanges, and calling for short-term water use reduction.

As part of the UWMP, Valley Water developed a Water Shortage Contingency Plan (WSCP) to establish actions and procedures for managing water supplies and demands during water shortages due to droughts and other emergencies. Valley Water uses projected countywide end-of-year groundwater storage as an indicator of potential water shortages and a trigger for WSCP actions. In the event of prolonged droughts or other emergency situations, Valley Water considers all available tools for managing available water supplies, including public education and community outreach, coordinating response among the County's municipalities and retailers, augmenting supplies by investing in supplemental supply sources, calling for short-term water use reductions, and balancing demands for treatment plants and recharge facilities, to maximize the use of available supplies in order to meet potential shortage. The WSCP also summarizes planning for natural disaster, drought-related revenue impacts, and Valley Water's legal authority to respond to water shortages.

Valley Water continues to be a leader in water conservation and has implemented a wide range of Demand Management Measures (DMMs) that help reduce water use. Valley Water's conservation programs include metering, public education and outreach, rebates for residential and commercial users, landscape rebates for lawn conversion, free water use audits and consultation, and many more. Collectively, conservation and stormwater capture accounted for about 75,000 AFY in 2020 in water savings over a 1992 baseline. Valley Water has a target to increase these savings to 110,000 AFY by 2040.

Valley Water's 2020 UWMP was adopted by its Board of Directors on June 9, 2021.

1.1 URBAN WATER MANAGEMENT PLAN

The Urban Water Management Planning Act (UWMP Act) (Division 6 Part 2.6 of California Water Code §10610 - 10656) requires all wholesale and retail urban water suppliers (those that directly or indirectly serve more than 3,000 customers or 3,000 acre-feet annually) to prepare an UWMP every five years. Since enacted in 1983, the UWMP Act has been amended numerous times by the State Legislature in response to droughts, water shortages, and the State's view on water supply reliability. The 2020 UWMP includes many additional requirements compared to the 2015 UWMP that were passed by the State Legislature, including:

- Five-year drought water reliability assessment
- Drought risk assessment
- Expanded Water Shortage Contingency Plan
- Reduced reliance on the Sacramento-San Joaquin Delta (Delta)
- Climate change impacts

The statutory deadline to submit the 2020 UWMP to the California Department of Water Resources (DWR) is July 1, 2021.

Valley Water meets the definition of an urban water wholesaler and has prepared UWMPs since 1985. Valley Water's 2020 UWMP documents current and projected water supplies and demands over the next 25 years during normal and drought years, as well as water shortage contingency planning and conservation efforts. The plan provides an overall picture of past, current, and future water conditions and management in Santa Clara County. The UWMP complements Valley Water's other planning efforts, including planning for annual operations, sustainable groundwater management, recycled water, integrated water resource management, and integrated regional water management. The 2020 UWMP updates and supersedes all previous UWMPs.

1.2 REPORT ORGANIZATION

Valley Water's 2020 UWMP was prepared in compliance with the requirements of the current UWMP Act and under the guidance provided by DWR. The UWMP follows the organization recommended by DWR:

- Chapter 1 – Introduction and Overview.

- Chapter 2 – Plan Preparation: Provides information on the process for developing the UWMP, including coordination and outreach efforts.

- Chapter 3 – System Description: Describes Valley Water's water system, including organizational structure and history, major infrastructure, and service area characterization.
- Chapter 4 – Water Demands: Describes and quantifies current and projected water demands in Santa Clara County.
- Chapter 5 – SBX7-7 Baseline, Targets, and 2020 Compliance: Describes Valley Water's efforts to support retailer efforts to achieve 2020 water use targets.
- Chapter 6 – System Supplies: Describes and quantifies Valley Water's current and projected sources of water.
- Chapter 7 – Water Service Reliability and Drought Risk Assessment: Evaluates the reliability of the water supply over the next 25 years for normal, single dry, and five consecutive dry years. Assesses risk associated with a five-year drought.
- Chapter 8 – Water Shortage Contingency Plan: Describes the development, actions, and implementation of Valley Water's WSCP.
- Chapter 9 – Demand Management Measures: Describes Valley Water's efforts to promote water conservation and reduce demand.
- Chapter 10 – Plan Adoption, Submittal, and Implementation: Describes the steps taken to adopt and submit the UWMP and make it publicly available, the plan to implement the UWMP, and DWR's Checklist.

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CHAPTER 2 – PLAN PREPARATION

Valley Water used DWR'S 2020 UWMP Guidebook as the basis for developing its UWMP. The UWMP was prepared in coordination with retailers, cities, the County, and other regional agencies. The UWMP was posted for public review and Valley Water incorporated inputs, as appropriate, that were received through internal and external review processes.

2.1 BASIS FOR PREPARING THE UWMP

Valley Water meets the definition of an urban water wholesaler under the UWMP Act and therefore is required to prepare an UWMP every five years. Valley Water's designated planning team prepared its 2020 UWMP following the guidance provided by DWR's 2020 UWMP Guidebook.

The UWMP Act requires use and submittal of standardized tables. DWR has developed standard tables for inclusion in the 2020 UWMP, and Valley Water used the standard tables for wholesale water suppliers throughout the plan. Valley Water is also the groundwater management agency for Santa Clara County and utilizes a conjunctive use strategy. This UWMP reflects the comprehensive nature of Valley Water's water management program. The tables in the main body of this plan reflect all the supplies and demands in Valley Water's service area, not all of which are managed by Valley Water. All the DWR-required tables are included in Appendix A.

2.2 INDIVIDUAL REPORTING

Valley Water actively engages in regional water supply planning and coordinates with regional partners such as San Francisco Bay Area Integrated Regional Water Management (IRWM), Pajaro IRWM, and Bay Area Regional Reliability (BARR) programs. However, this is an individual UWMP that reports on water demands and supplies in Santa Clara County (Table 2-1).

Table 2-1. Plan Identification

Select Only One	Type of Plan	
☑	**Individual UWMP**	
	☐	Water Supplier is also a member of a RUWMP
	☐	Water Supplier is also a member of Regional Alliance
☐	**Regional Urban Water Management Plan (RUWMP)**	

Similarly, Valley Water chose to report as an individual supplier for the purpose of determining and reporting its compliance with urban water use SBX7-7 baselines and targets as described in Chapter 5.

CHAPTER 2 – PLAN PREPARATION

2.3 REPORTING YEAR AND UNITS OF MEASURE

All information in this plan, unless otherwise noted, is reported on a calendar year basis. Water volumes are expressed in acre-feet (AF) (Table 2-2).

Table 2-2. Supplier Identification

Type of Supplier	
☑	Supplier is a wholesaler
☐	Supplier is a retailer
Fiscal or Calendar Year	
☑	UWMP Tables are in calendar years
☐	UWMP Tables are in fiscal years
Units of Measure Used in UWMP	
Unit	Acre Feet (AF)

2.4 COORDINATON AND OUTREACH

This UWMP was prepared in coordination with the 13 major water retailers in Santa Clara County, the cities in Santa Clara County, the County of Santa Clara (County), the San Francisco Public Utilities Commission (SFPUC), and the Bay Area Water Supply and Conservation Agency (BAWSCA). Valley Water's 13 retailers in Santa Clara County are: California Water Service Company, City of Gilroy, Great Oaks Water Company, City of Milpitas, City of Morgan Hill, City of Mountain View, City of Palo Alto, Purissima Hills Water District, San José Municipal Water System, San Jose Water Company, City of Santa Clara, Stanford University, and City of Sunnyvale.

Valley Water notified the land use agencies and water retailers of the updates of its UWMP by both email and letter dated December 14, 2020 (consistent with CWC 10621(b)). On March 22, 2021, Valley Water emailed its preliminary reliability analysis to its water retailers. Supplies were projected in five-year increments from 2025 through 2045 for normal, single dry, and five consecutive dry years (consistent with CWC 10631). On March 29, 2021, Valley Water provided the retailers with the draft UWMP and WSCP for review. Valley Water notified water retailers and the cities and County on May 17, 2021 of the time and date of the public hearing on the 2020 UWMP and provided information on how to review the UWMP and WSCP. Documentation of these efforts is included in Appendix B. In addition to these required coordination efforts, Valley Water had numerous group and individual communications with retailers and other agencies on issues related to demand and supply projections, reduced reliance on the Delta, reliability analyses, and the WSCP. Valley Water also provided regular updates at various committee meetings throughout the plan development.

CHAPTER 2 – PLAN PREPARATION

Prior to posting the UWMP and WSCP for public review, Valley Water incorporated input, as appropriate, that was received from agencies and retailers. The draft UWMP and WSCP were posted on Valley Water's website and made available for public review on May 17, 2021. The public hearing notice was published on ***San José Mercury News*** on May 18, 2021 and May 25, 2021 and ***Metro*** on May 19, 2021, in accordance with California Government Code 6066. A copy of the public notice is included in Appendix B. The public hearing was held on June 8 and June 9, 2021. Comments were received from several retailers and other parties as part of the public hearing. Additional information on the adoption of the UWMP and WSCP is provided in Chapter 10.

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CHAPTER 3 – SYSTEM DESCRIPTION

Valley Water is a special district that provides water resources management for all of Santa Clara County. Valley Water's water system includes local water from reservoirs, groundwater, imported water, and recycled water. These supplies are used to recharge local groundwater subbasins, treated at drinking water treatment plants, released to local creeks to meet environmental needs, or sent directly to water users. Climate change, new regulatory requirements, and population growth could affect county-wide water supply and demand in the future.

3.1 VALLEY WATER OVERVIEW

Valley Water is an independent special district that provides wholesale water supply, groundwater management, flood protection and stream stewardship in Santa Clara County. Valley Water's service area includes the entirety of Santa Clara County, which is located at the southern end of San Francisco Bay (Figure 3-1). The county encompasses approximately 1,300 square miles and includes 15 cities from Palo Alto in the north to Gilroy in the south. Most water use occurs on the valley floor between the Santa Cruz Mountains to the west and the Diablo Range to the east. Northern Santa Clara County is home to Silicon Valley and the valley floor is highly urbanized. Southern Santa Clara County has some urban development, but much of the land use is rural and agricultural.

Valley Water was formed in 1929 as the Santa Clara Valley Water Conservation District in response to groundwater overdraft and land subsidence. In 1954, Valley Water annexed the Central Santa Clara Valley Water District. In 1968, it merged with the countywide flood control district to form one agency to manage the water supply and flood programs for most of the county. The Gavilan Water District in southern Santa Clara County was annexed in 1987 and since then Valley Water has provided services for the entire county.

Valley Water is the Groundwater Sustainability Agency (GSA) for the Santa Clara and Llagas Subbasins, which are both identified as high priority basins by DWR. Valley Water sustainably manages local groundwater basins to support beneficial use by water retailers, private well users, and the environment. Valley Water is also the GSA for the small portion of the North San Benito Subbasin within Santa Clara County.

Valley Water is governed by an elected seven member Board of Directors following the District Act (https://www.valleywater.org/how-we-operate/about-valley-water/district-act) and its own Board Governance Policies (https://www.valleywater.org/how-we-operate/board-governance-policies).

CHAPTER 3 – SYSTEM DESCRIPTION

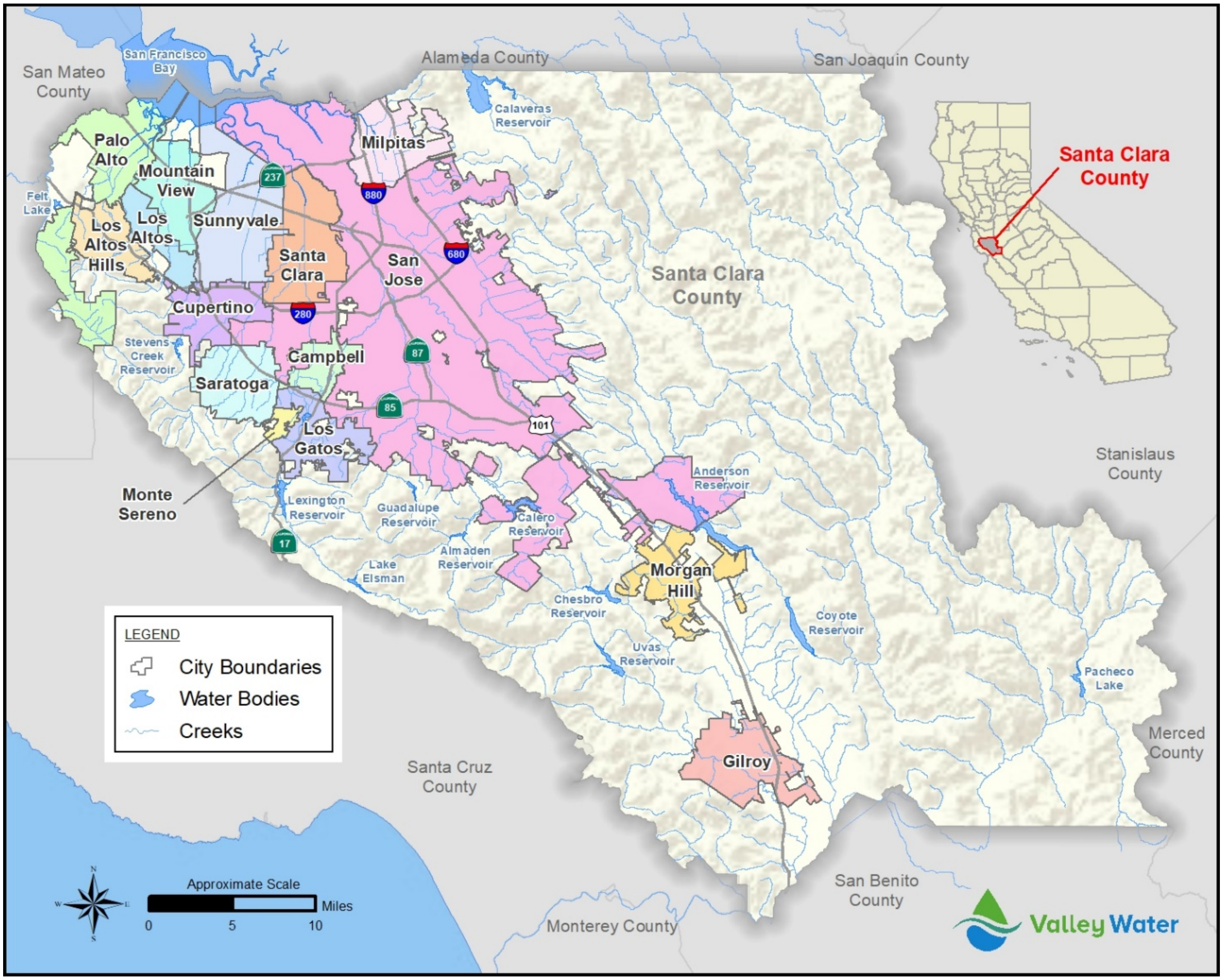

Figure 3-1. Santa Clara County

CHAPTER 3 – SYSTEM DESCRIPTION

3.2 VALLEY WATER'S WATER SUPPLY SYSTEM

Valley Water manages an integrated water resources system to provide supply of clean, safe water, flood protection and stewardship of streams on behalf of Santa Clara County's nearly two million residents. Valley Water manages 10 dams and surface water reservoirs, three water treatment plants, an advanced recycled water purification center, a state-of-the-art water quality laboratory, 142 miles of raw and treated water pipelines, 101 groundwater recharge ponds covering 276 acres, and more than 275 miles of jurisdictional streams, including 91 miles suitable for in-stream recharge (Figure 3-2). Water supplies include local surface water and groundwater, imported water, and recycled water. Water conservation is also an important part of the of the water supply mix, which helps reduce water demands and improve reliability during droughts.

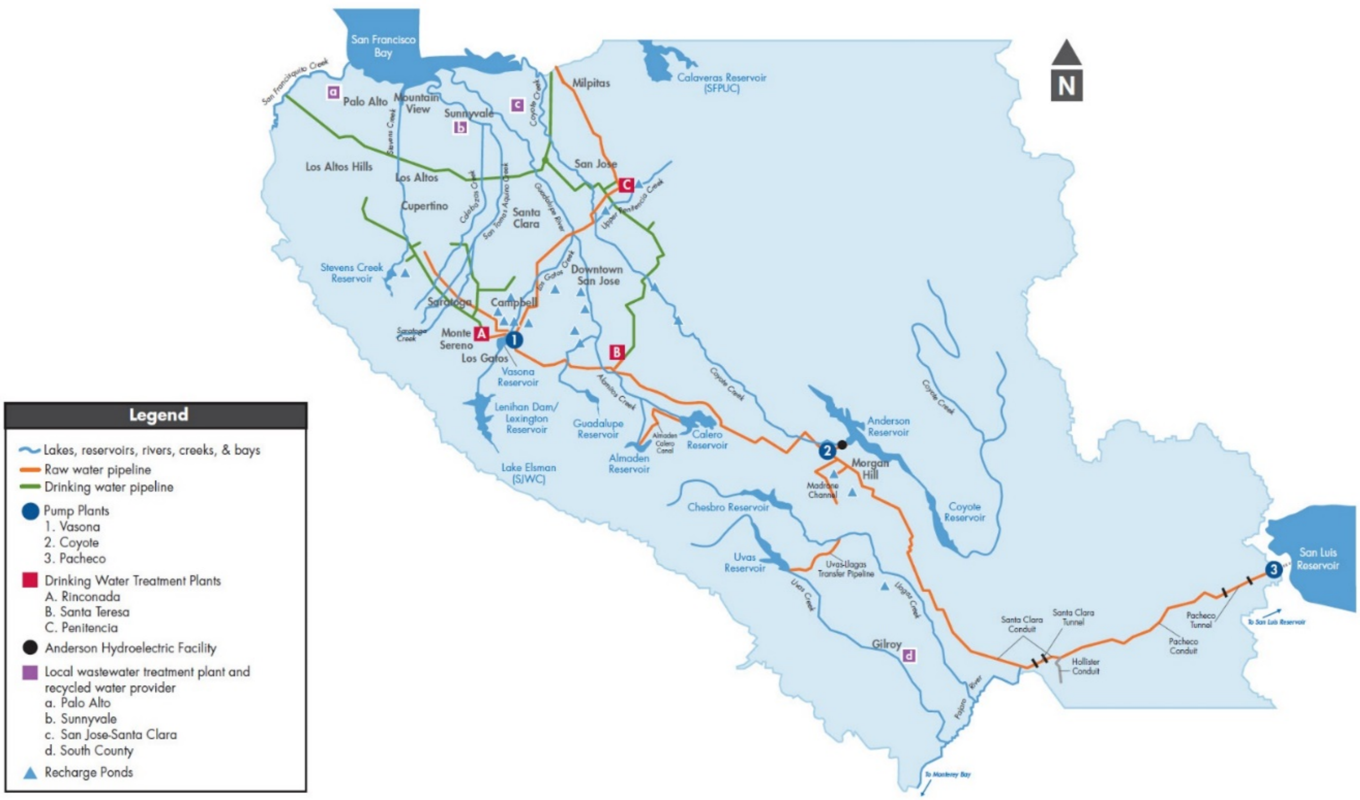

Figure 3-2. Water Supply System

Local water supplies make up about half of the county's water supply. Local sources include natural groundwater recharge and surface water supplies, including surface water rights held by Valley Water, San José Water Company, and Stanford University. A small but growing portion of local water supply is recycled water used for non-potable purposes. Imported water from the State Water Project (SWP), Central Valley Project (CVP), and supplies delivered by the San Francisco Public Utilities Commission (SFPUC) make up about another half of the county's supply. Valley Water's diverse supplies are used to recharge local groundwater subbasins, treated at drinking water treatment plants, released to local creeks to meet environmental needs, or sent directly to water users.

Valley Water has been a leader in conjunctive use in California for decades, utilizing imported and local surface water to supplement groundwater and to maintain reliability in dry years. Conjunctive use helps protect local subbasins from overdraft, land subsidence, and saltwater intrusion and provides critical groundwater storage reserves for use during droughts or outages. After it was formed to address declining groundwater levels and land subsidence, Valley Water constructed reservoirs to capture local water. However, local supplies became insufficient to meet the needs of the county's growing population around the middle of the last century. In response, Valley Water began importing water from the Delta via the SWP in 1965 and from the CVP in 1987. These investments, along with investments in water recycling and conservation, have resulted in sustainable groundwater subbasins and reliable water supplies for the County. Figure 3-3 shows how Valley Water's conjunctive water management strategy has dramatically contributed to a sustainable water supply.

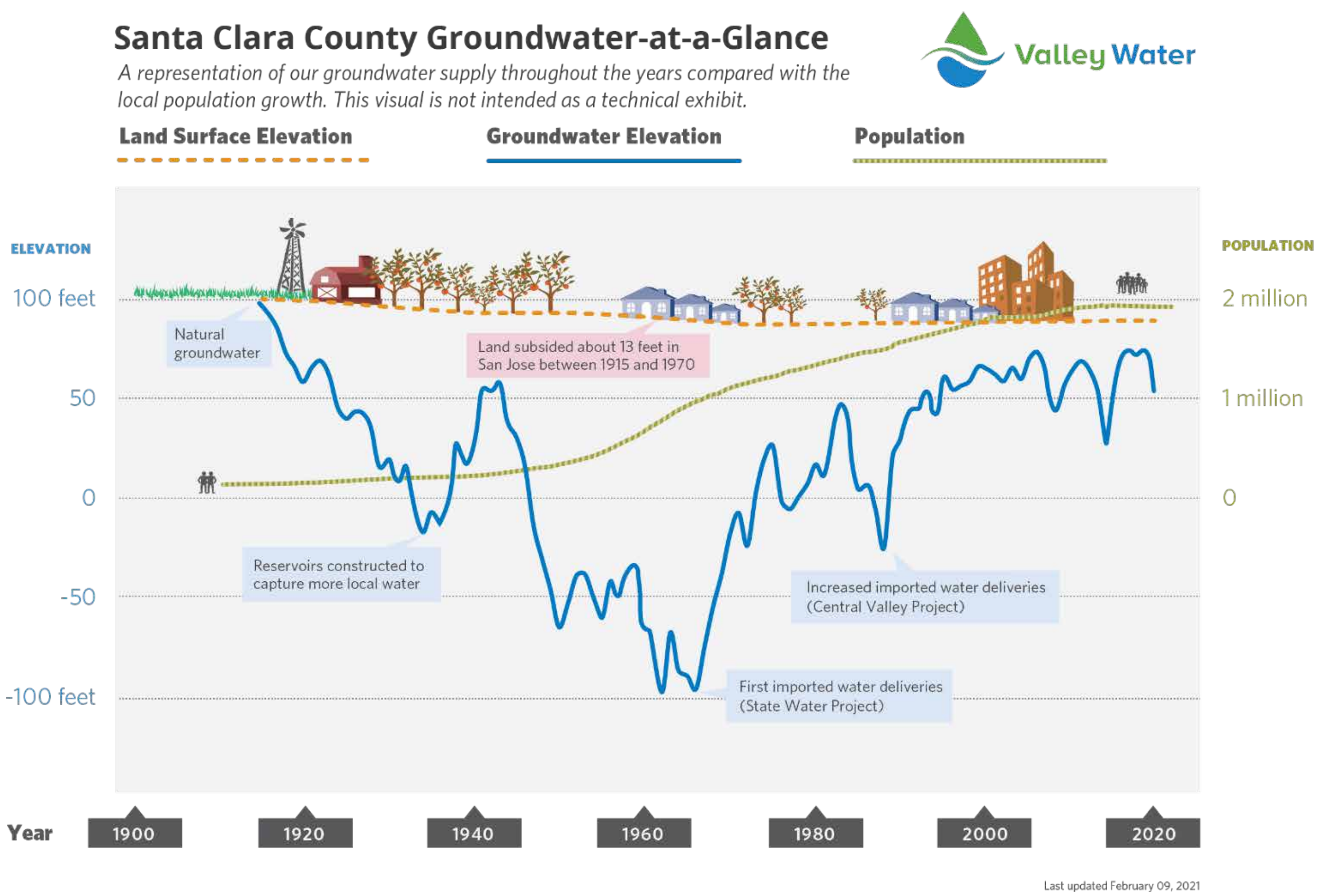

Figure 3-3. Historic Groundwater Conditions

3.3 SERVICE AREA CLIMATE

3.3.1 Historic Climate Data

Santa Clara County has a semi-arid, Mediterranean climate, with warm and dry weather lasting from late spring through early fall. Average annual precipitation ranges from about 15 inches on the valley floor to about 45 inches along the crest of the Santa Cruz Mountains. The average annual precipitation in Santa Clara County was 23.2 inches from 1950-2020, with most precipitation occurring between the months of November and April. The county's temperature is generally moderate. Maximum daily temperatures averaged by month in the County range from 55.7°F to 83.4°F. The average annual evapotranspiration (ETo) is 49.6 inches (Table 3-1).

Table 3-1. Average Climate Data

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Annual
Rainfall (inches)	4.7	4	3.6	1.8	0.6	0.1	0	0.1	0.2	1.1	2.7	4.2	**23.2**
Max. Daily Temp. (°F)	55.7	58.3	61.2	65.6	71.7	78.4	83.4	83.1	80.8	73.8	62.8	55.9	**69.2**
Min. Daily Temp. (°F)	37.9	39.7	40.9	42.8	47.4	52.2	56.6	56.4	54.5	49.2	42.3	37.9	**46.5**
Average ETo (inches)	1.5	1.9	3.5	5	6	6.8	7	6.3	4.8	3.5	1.9	1.4	**49.6**
NOTES: Rainfall and temperature from NOAA climate mapping for Santa Clara County. The 2015 plan used Valley Water Station 86 which represents rainfall in City of San José. ETo from California Irrigation Management Information System (Archived San Jose Station).													

Rainfall in Santa Clara County exhibits great interannual variability (Figure 3-4). Historical rainfall in Santa Clara County has ranged from 5.1 inches in 2013 to 46.7 inches in 1983. Valley Water's conjunctive water management strategy helps maintain groundwater levels and manage supply variability by capturing runoff in wet periods and using this water to recharge the groundwater subbasins for storage and use later.

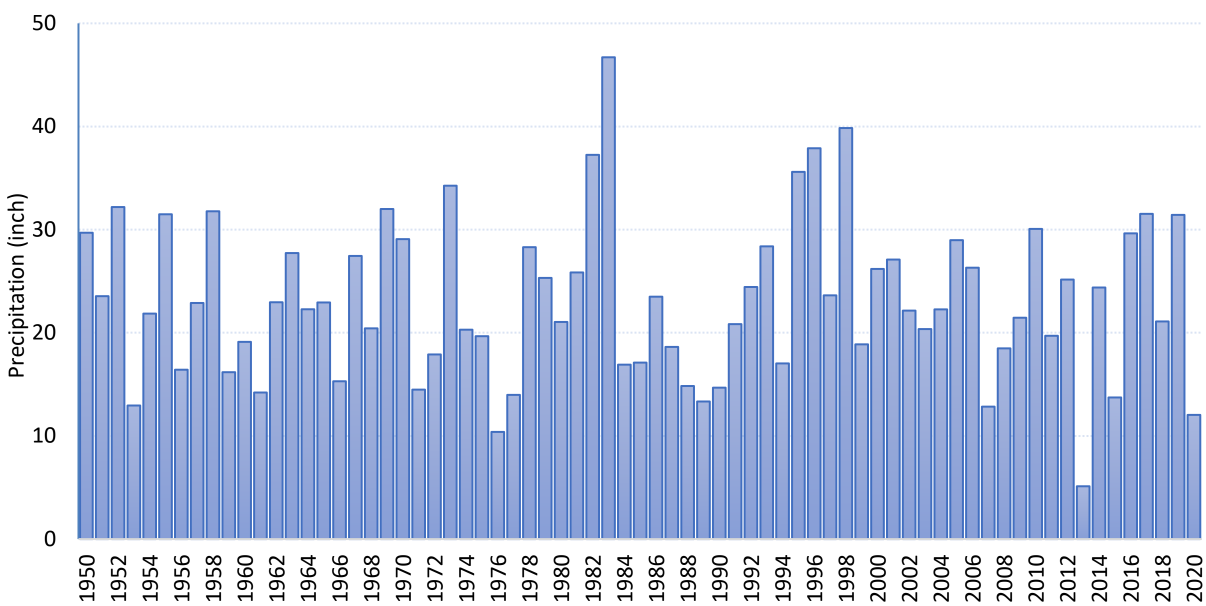

Figure 3-4. Historical Annual Rainfall in Santa Clara County

3.3.2 Climate Change

Climate change impacts such as warming temperatures, shrinking snowpack, increasing weather extremes, and prolonged droughts pose significant challenges in water resources management. Already, climate change impacts are being observed across California and in the San Francisco Bay Area, and climate modeling projections indicate that these impacts will continue or become more extreme. Locally, Santa Clara County is expected to see increasing temperatures, which could result in more extreme heat and drought events and increased demands. While future projections of precipitation are not consistent, some studies indicate that Valley Water could see altered hydrologic patterns and an increase in rainfall averages. Extreme weather events are projected to increase in intensity and droughts could be extended over historic conditions. More severe storms could result in increased flood risk and changes in surface runoff patterns that could challenge local water supply operations. Sea level rise increases the potential for flooding and could significantly affect imported water supplies.

Statewide and local changes in precipitation and temperature could impact Valley Water’s water supplies and operations, the effectiveness of potential water supply investments, and water demand patterns. Recognizing the importance of managing climate change-related vulnerabilities and risks to fulfill its mission, Valley Water is developing a Climate Change Action Plan (CCAP), which will include a wide array of goals and strategies to adapt to climate change. Strategies for water supply adaptation will include promoting and increasing investments in recycled water and water conservation; increasing system flexibility; optimizing the use of existing supplies and infrastructure; and incorporating understanding of projected hydrology and sea level rise into project management and planning. In addition, Valley Water is developing a climate study to assess climate change impacts to its future water supply reliability. The study is expected to be completed in the next two years.

3.4 SERVICE AREA POPULATION AND DEMOGRAPHICS

Santa Clara County currently has a population of nearly 2 million. According to the Plan Bay Area projections in 2017, Santa Clara County’s population is expected to increase by about 36% between 2020 and 2045, up to nearly 2.7 million in 2045 (Table 3-2). Projected population and job growth rates for Santa Clara County are higher than the nine-county Bay Area average. Total jobs are projected to increase an estimated 21% in the same period. However, job growth is not projected to be equal in all sectors. Agricultural jobs are projected to decrease, manufacturing jobs are projected to be stable, and other job sectors are projected to increase. The greatest projected increase is in health and education services.

Table 3-2. Santa Clara County Demographics from Plan Bay Area 2017 Projections

	2020	2025	2030	2035	2040	2045[1]
Population	1,986,340	2,098,695	2,217,750	2,387,165	2,538,320	2,699,046
Single-Family	409,395	409,280	411,725	418,715	422,960	427,248
Multi-Family	297,170	326,965	356,025	411,305	458,695	511,545
Households	679,425	718,565	757,690	815,980	860,810	908,103
Persons per Household	2.87	2.87	2.88	2.88	2.90	2.92
Total Jobs	1,120,420	1,159,110	1,198,370	1,231,000	1,289,870	1,351,555

[1]2045 values are calculated by increasing the 2040 values by the same rate of increase as 2035 to 2040 values.

CHAPTER 3 – SYSTEM DESCRIPTION

Santa Clara County has a racially diverse population that is Asian (37.6%), White (30.4%), Hispanic or Latino (25%), Black or African American (2.4 %), American Indian or Alaska Native (0.2%), Native Hawaiian or Other Pacific Islander (0.4%), with the remainder more than one race or some other race (U.S. Census Bureau, 2019). Santa Clara County is an economic center for high technology and home to Silicon Valley. It has a median household income of $116,178 (2018 dollars) and a poverty level of 7.3%.

3.5 SERVICE AREA LAND USES

Current land uses in Santa Clara County are depicted in Figure 3-5. Land use was derived and categorized from Santa Clara County's parcel data. Approximately half of the land area is open space (52%). Agriculture accounts for 25% of the land area, followed by residential land use (15%). The remaining area consists of commercial, industrial, and institutional use (7%) and transportation and utility use (1%).

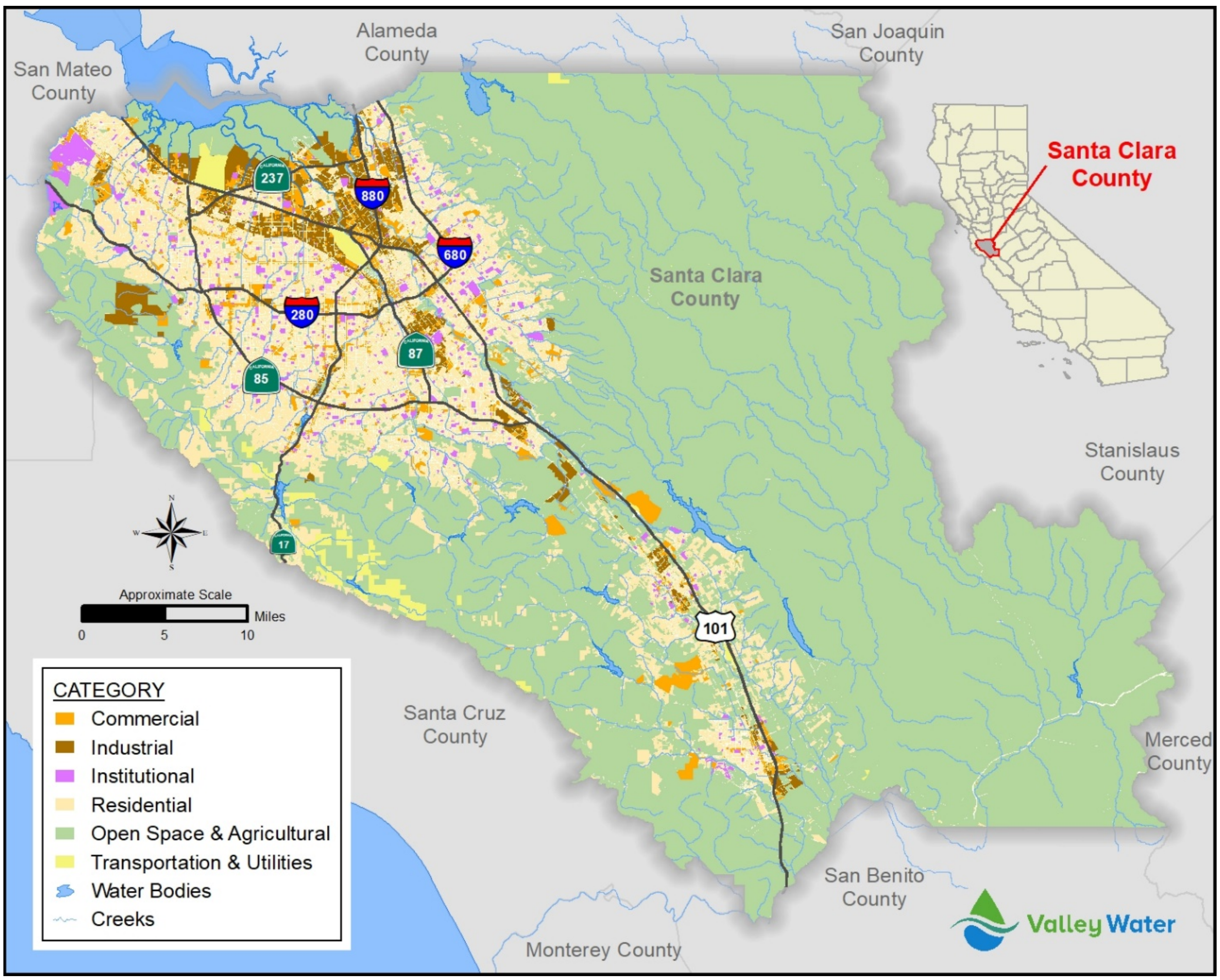

Figure 3-5. Santa Clara County Land Use

Housing growth is a major factor that impacts Valley Water's water demand projections. According to the most recent finalized plan, Plan Bay Area 2040, the number of households will increase in Santa Clara County by 38% between 2015 and 2040. San José, the largest city in the Bay Area, is projected to have the greatest proportion of household growth. The growth in the number of households in Santa Clara County represents the fastest projected growth rate of all counties in the Bay Area. The growth projected in the northern portion of the county is expected to be primarily in the multi-family sector and include redevelopment of built parcels, resulting in greater housing density. The growth projected in the southern portion of the county is expected to be primarily in the single family sector, and thus is expected to have lower housing density than in the northern portion of the County.

To address the County's expected growth in development, Valley Water joined a task force in 2015 to develop an ordinance to support water conservation in new development (Model Water Efficiency New Development Ordinance or MWENDO). The task force consisted of representatives from Santa Clara County, cities, and other stakeholders such as non-profit organizations. The MWENDO was completed in 2017 for consideration by Santa Clara County cities and towns. Valley Water is encouraging municipalities to adopt the ordinance to ensure new and retrofitted development meets strong water efficiency standards.

Plan Bay Area 2050 is a long-range plan currently being developed by the Association of Bay Area Governments (ABAG) and the Metropolitan Transportation Commission (MTC). It is expected to be completed in 2021. One of its main goals is to address the severe, longstanding housing crisis in the Bay Area with the development of strategies to spur housing production, as well as create new jobs. Because of this focus, the 2050 plan differs from previous ones, as it makes more assumptions about the adoption of new policies and strategies to promote housing. According to the draft plan, if the strategies in Plan Bay Area 2050 were implemented, the number of households in the County will increase 73% between 2015 and 2050, and jobs would increase by 46% during this period. Santa Clara County would have the greatest share of regional growth (33%) amongst the nine counties in the Bay Area. Similarly, Santa Clara County would experience the region's greatest share of job growth at 36%. These household and job projections are significantly higher than the projections from previous plans. Valley Water is closely following Plan Bay Area 2050 development to determine whether and how to use its projections to model future water demand.

CHAPTER 4 – WATER DEMANDS

Understanding water demands and the factors that influence them over time is an important first step for water resources planning and assessment. This chapter describes and quantifies Santa Clara County's current water use and water demand projections through the year 2045. Accurately tracking and reporting current water demands allows Valley Water to properly analyze the use of the county's water resources and conduct effective resource planning. Estimating future demand allows Valley Water to manage the county's water supply and appropriately plan infrastructure investments. Assessments of future growth and related water demand, done in coordination with local planning agencies and retailers, provide essential information for developing demand projections. Demand estimates were estimated by Valley Water's Demand Model that was developed based on local planning assumptions and growth projections. Valley Water coordinated with the water retailers and the local planning agencies on demand projections to the extent practicable.

4.1 HISTORICAL AND CURRENT WATER USE

Water use in Santa Clara County includes domestic, municipal, industrial, and agricultural use. Current countywide average annual water use was approximately 310,000 AFY. Actual water use changes from year to year and is influenced by a number of factors such as population growth, hydrology, water conservation, drought, and economic conditions (Figure 4-1). The countywide water use represents the total use of Valley Water supply, SFPUC Supply, and San José Water Company and Stanford University water rights. As a result of Valley Water's investments in water conservation since 1992, overall water use in the county has decreased for the past 15 years despite a 25% increase in population over the same period. The various significant decreases in water use are associated with the extended droughts of 1987-1992, 2007-2010, and 2012-2016. The 2007-2010 drought occurred during an economic recession, which can also depress water use. Currently, Valley Water's water use is still low and there is not likely to be a rebound to pre-drought water use.

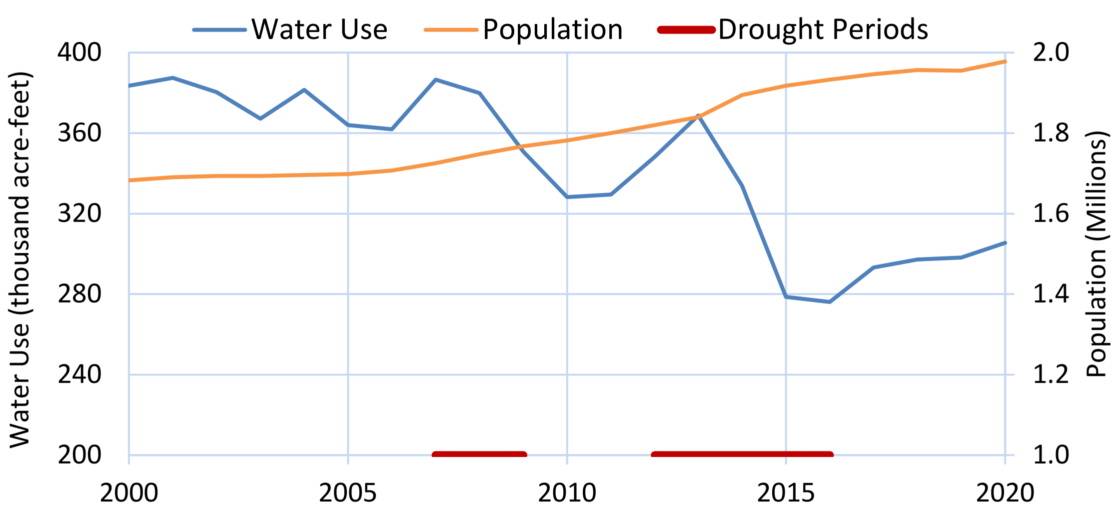

Figure 4-1. Historic Water Use and Population

Water use data from retailer billing information from 2018 were used to determine the approximate distribution of water use by sector (Figure 4-2). The chart represents data from only the retailers that track water use in these sectors. Since not all retailers track their use in all of these sectors, the chart does not represent the full countywide use. Nevertheless, it is still considered a relatively good picture of average water use distribution. Overall, more than half of water use is for residential, and CII sector

(Commercial, Industrial, and Institutional) represents 41% of use. Since agriculture is supported nearly entirely by independent groundwater pumping, that use (which is significant in South County) is not reflected in this figure.

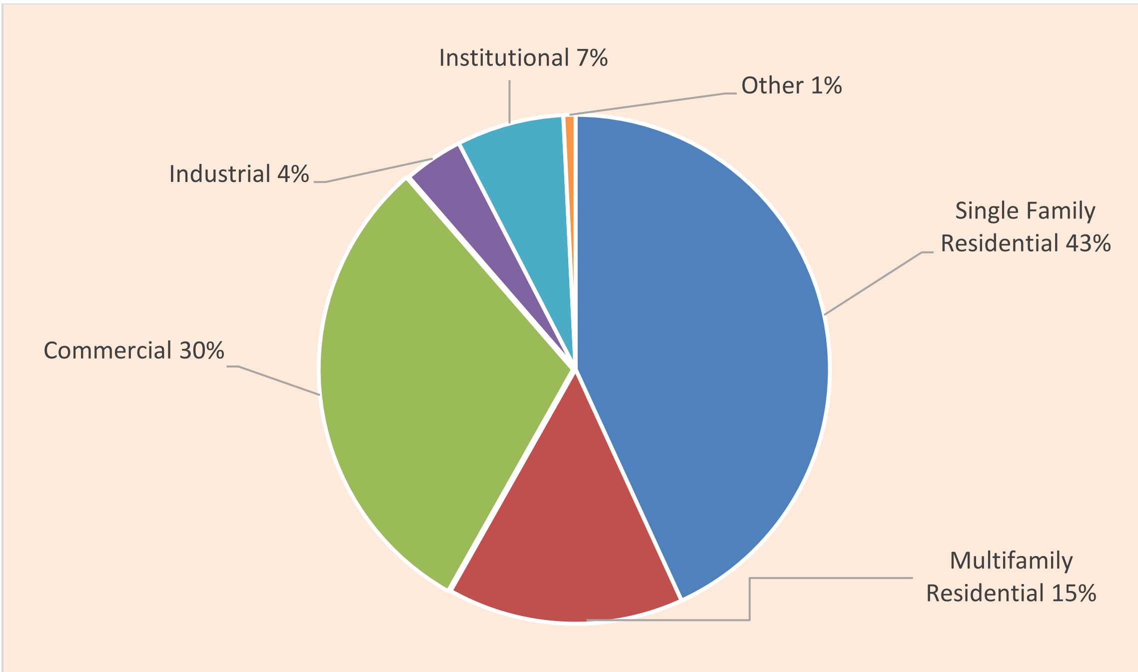

Figure 4-2. Water Use by Sector (2018)

Actual water use of Valley Water's supply in 2020 is estimated at approximately 306,000 AFY (Table 4-1). The current water use indicates a small rebound from the last drought of 2012-2016, indicating that Santa Clara County is "making conservation a way of life" and Valley Water's continued investment in water conservation measures may have contributed to potential overall decline in water use.

CHAPTER 4 – WATER DEMANDS

Table 4-1. Actual Water Use in 2020

Use Type	Water Use[1] (AF)
Treated Water and Groundwater	246,000
Agricultural Irrigation	25,000
Independent Groundwater Pumping	13,000
Recycled Water	17,000
Untreated Surface Water	2,000
Losses	3,000
Total	**306,000**

[1]All numbers are rounded to the nearest 1000 AF. The total water use represents countywide demand which is partially served by the SFPUC and surface water rights held by San Jose Water Company and Stanford University. SFPUC, San Jose Water Company, and Stanford supplies are accounted for in the Retailer Water Use.

4.2 PROJECTED WATER DEMANDS

Valley Water's long-term water supply level of service goal is to meet 100% of annual water demand during non-drought years and at least 80% of annual water demand in drought years. A reliable water demand forecast is critical in determining the level of investment necessary to meet this level of service goal. To meet its planning need for robust demand projections, Valley Water developed a statistical based Demand Model (Model) in 2020 (https://www.valleywater.org/your-water/water-supply-planning/water-demand-study) using recently available water use data and new housing and economic development forecasts. The Model provides forecasted demands in Santa Clara County out to 2045 and is described below.

4.2.1 Retailer Water Demands

Retailer demands represent the majority of countywide demands. The Model uses a statistically based analytical framework that is commonly referred to as an econometric approach to project future retailer demands. The Model is essentially a multivariate regression that defines the relationship between water use and forecasting variables. The forecasting variables used include housing information, median income, economic information, water rates, and weather. The Model is composed of sub-models (or regression equations) for differentiating rates of water use by retail agency, time of year, and water use sector to characterize temporal, geographical, and sectoral variations of water demands. Water use sectors included in the model are single family, multi-family, and commercial, industrial, and institutional (CII).

Historic data were collected to support model development from Valley Water and its water retailers, the US Census, Federal Reserve, and California Department of Finance (CDOF) (Table 4-2). Monthly sectoral water use data from local water retailers for 2000-2019 (although certain water retailers did not have data back to 2000) were used as observed data for model fitting.

Table 4-2. Forecasting Variables Used in the Demand Model

Forecasting Variable	Source
Water rates (by retailer and groundwater area, inflation adjusted)	Valley Water
Drought severity	Valley Water and retailers
Median income	US Census
Economic indices (e.g., unemployment)	Federal Reserve, Economic Cycle Research Institute
Housing density	Derived from US Census and CDOF
Persons per household	Derived from US Census and CDOF
Housing Units	ABAG
Sectoral employment	ABAG
Temperature and precipitation	PRISM (Parameter-elevation Relationships on Independent Slopes Model)

The Model was then used to forecast future demands using the projected forecasting variables with information from the Association of Bay Area Governments (ABAG), CDOF, and PRISM (Table 4-2). An important modeling assumption in forecasting water demand is related to defining a drought rebound. Historically after a drought, water use returns to pre-drought levels within a few years of the drought's end; this is the 'drought rebound'. Valley Water experienced a small rebound in 2017 and then demands remained relatively stable through 2018 and 2019. Therefore, the rebound has been relatively muted for Valley Water, and similar trends were also observed by most peer agencies. Historically demand rebounds have occurred (Figure 4-1), so Valley Water is conservatively assuming there will be a 50% drought rebound by 2025. This assumption will be reevaluated as more water use data become available in the next few years. The aggregated countywide demand projections for retailers are provided in Table 4-3.

Overall, the Model reflects Valley Water's current understanding of expected drought rebound and integrates water use data and sectoral growth forecasts. The Model can be used to evaluate potential future scenarios by adjusting the forecasting variables to help understand the uncertainty related to water demand forecasts. Valley Water will use the Model to support its long-range planning efforts including water supply master planning and the annual MAP.

4.2.2 Independent Groundwater Pumping

Independent groundwater pumping includes groundwater pumping by individual domestic well owners, small and mutual water companies, businesses, non-agricultural irrigation, and environmental cleanup. It includes all non-retailer groundwater pumping in the Municipal and Industrial (M&I) and domestic categories. Statistical analysis of historic data suggests that water use in this aggregated sector is mostly affected by drought, price, weather, and number of wells. Therefore, similar to retailer demand, the Model for this sector was developed that defines the relationship between water use and these factors (drought, price, weather, and number of wells). Historical regression fits for independent groundwater pumpers were performed on annual water use. The Model was fit based on the historic data and used to project future independent groundwater pumping out to 2045 (Table 4-3).

4.2.3 Agricultural Groundwater Pumping

Agricultural water use from groundwater pumping in Santa Clara County has been generally constant over the last twenty years at approximately 25,000 AFY (Figure 4-3). Historically, there is evidence that significant reductions in harvested acres and in agricultural water use had occurred prior to 2000s, and declines in the number of harvested acreage over time were the result of both increasing urban development and higher productivity. Current local land use plans and agricultural reports indicate that the amount of harvested acreage is likely in a stable state, with only minor declines due to increased urban development in the future. Given this, it is therefore assumed that average water use from the last 20 years would be an appropriate and conservative representation of future agricultural water use, which is held constant into the planning horizon as the projected agricultural pumping (Figure 4-3).

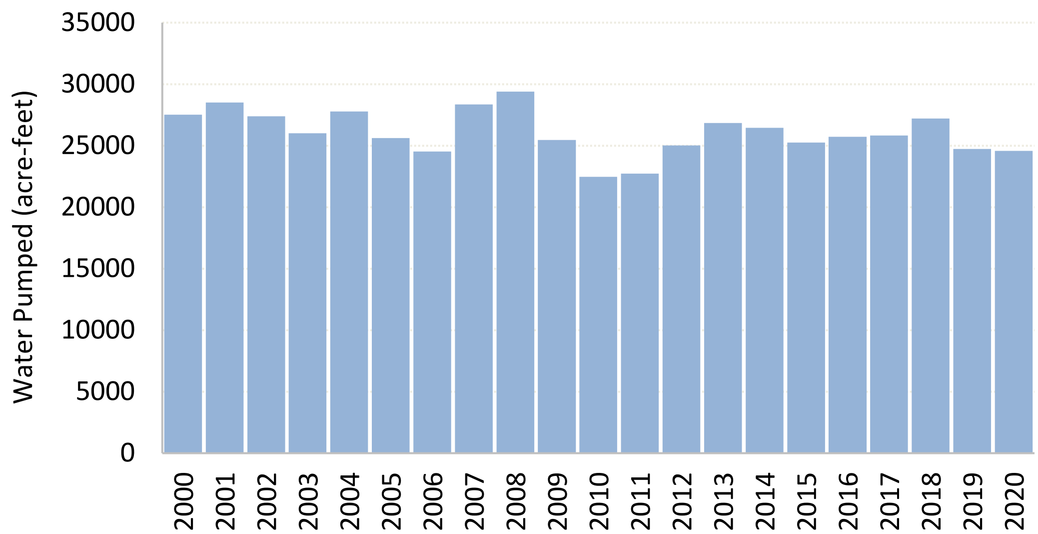

Figure 4-3. Historic Agricultural Groundwater Pumping

4.2.4 Untreated Surface Water

Untreated surface water is available to a limited number of surface water customers under Valley Water's Untreated Surface Water Program (Program). The Program was established in 1974 per Board adopted Resolution 74-28 and the Rules and Regulations for the Service of Surface Water (Rules). The water use is granted through Valley Water issued surface water permits and per the existing Rules, and can be used for landscaping, agricultural irrigation, and municipal and industrial uses. Untreated surface water service is interruptible and supply is not guaranteed. For future demands in this sector, the average of historic use was used and held at a constant rate for the planning horizon (Table 4-3). Valley Water is currently working on developing recommendations to update the Program and its Rules.

4.2.5 Distribution System Water Losses

Distribution system water losses (also known as "real losses") are the physical water losses from Valley Water's distribution system. As required by DWR, Valley Water quantified its treated water distribution system losses using the DWR Water Audit Method. The system losses are projected to increase slightly for the planning horizon with the increase of demand (Table 4-3). A copy of Valley Water's Fiscal Year 2020 Water Loss Audit is provided in Appendix C.

4.2.6 Estimated Future Water Savings

Valley Water, through a unique cooperative partnership with its retailers, has made significant investments in a variety of water conservation programs to permanently reduce water use in Santa Clara County. By taking the lead on implementing many demand management measures, Valley Water currently saves approximately 75,000 AFY from a 1992 baseline. Furthermore, modeling of Valley Water's current programs and implementation of existing regulations (referred to as passive water conservation measures) indicates Valley Water should achieve 99,000 AF water saving by 2030 from the baseline. An additional 10,000 AF of water conservation is forecasted to occur between 2030 and 2040 for a total of 109,000 AF by 2040. These planned water savings were deducted when calculating total countywide water use for the planning period. Valley Water's draft Water Conservation Strategic Plan estimates that passive savings will account for nearly 74,000 AF (75%) and more than 91,000 AF (84%) of all savings by 2030 and 2040, respectively. The adopted codes, plans, and other policies or laws that influence the calculated passive savings include the Energy Policy Act of 1992 and of 2005, the Water Research Foundation's 2016 "Residential End Uses of Water", California Assembly Bill 715 (AB 715), the California Plumbing Code, and the California Green Building Code.

4.2.7 Total Water Use

The countywide total water use is provided in Table 4-3. Overall, future water use is projected to increase over time with population growth, but will be well within the range of historic data (Figure 4-4).

Table 4-3. Projected Countywide Demand

Use Type	Projected Water Use (AF)				
	2025	**2030**	**2035**	**2040**	**2045**
Retailer Demand	288,000	280,000	285,000	290,000	299,000
Agricultural Irrigation	25,000	25,000	25,000	25,000	25,000
Independent Groundwater Pumping	14,000	14,000	14,000	14,000	14,000
Untreated Surface Water	2,000	2,000	2,000	2,000	2,000
Losses	3,000	3,000	3,000	3,000	3,000
TOTAL	**330,000**	**325,000**	**330,000**	**335,000**	**345,000**
NOTES: Total numbers are rounded to the nearest 5000 AF, and all other numbers are rounded to the nearest 1000 AF. The numbers represent countywide demands, which are partially served by the SFPUC, recycled water, and surface water rights held by San Jose Water Company and Stanford University.					

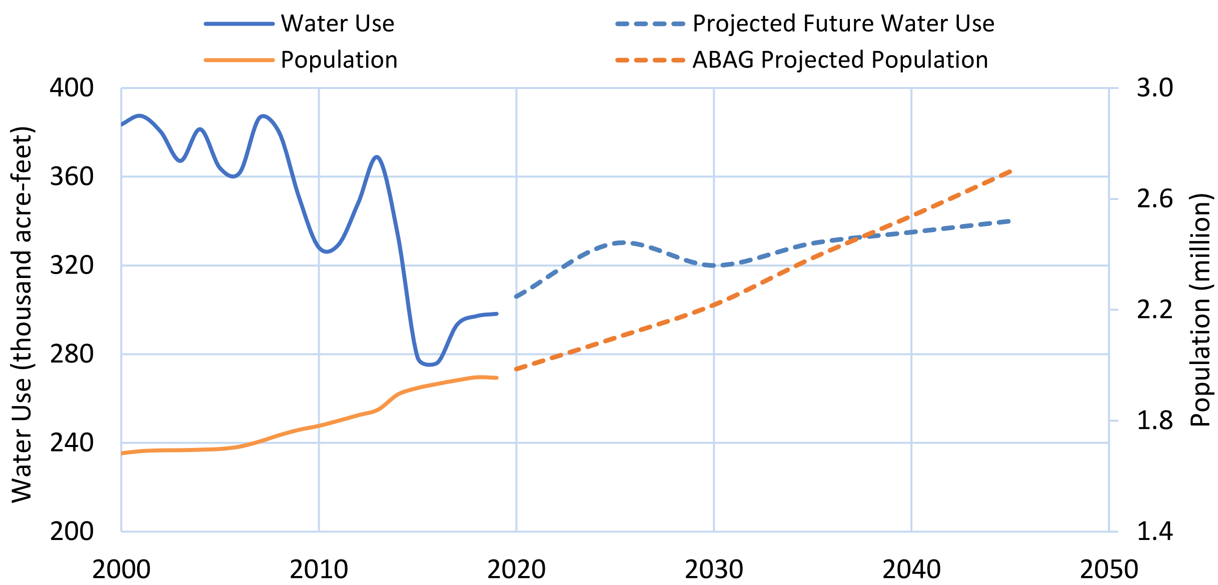

Figure 4-4. Historic and Project Future Water Use

4.3 COORDINATION WITH RETAILERS AND LAND USE PLANNING AGENCIES

Land use planning fundamentally influences future water use patterns and consequently water supply reliability. Therefore, it is in the best interest of Valley Water, land use agencies and the community that land use planning and water management be coordinated. The UWMP provides one such opportunity. Development of the water demand projections for this UWMP included coordination with water utilities and land use agencies, where feasible and appropriate. Valley Water's efforts in coordinating with retailers and planning agencies to understand data and assumptions for water demand projections are described below.

4.3.1 Water Retailer Coordination

Valley Water provides water to 13 water supply retailers of various sizes within its service area, which represent the majority of water use in Santa Clara County. Per UWMP requirements, 11 of Valley Water's retailers are required to prepare an UWMP, and therefore developed their own water demand forecasts to support their plan development. To understand retailer demands, Valley Water collected the information on retailers' water use projections for their service areas, growth assumptions, projection methodology, and associated planning documents. The review of retailer's demand methodologies showed that retailers used different land use planning assumptions in their demand models. Many derive their growth projections directly from population projections in city land use plans while others use ABAG projections, and some use a combination of local plans and known or historic growth considerations. Very few retailers showed their projected demands by water use sectors. The review was useful for providing Valley Water with more understanding about the differences between Valley Water's and retailers' underlying assumptions, which help explain the difference between their respective demand projections. Readers can review water retailer's UWMPs for their most recent demand projections, modeling efforts and assumptions.

The aggregated retailers' water use projections were compared to the countywide demands estimated by Valley Water's Model (Figure 4-5). The comparison shows that the two demand projections are within 1% (2025) to 10% (2045) of each other, with the difference increasing further into the future (Figure 4-5). There are many reasons that the demands may differ, including differences in base years, models, assumptions on growth, conservation factors, etc. Nevertheless, given the many ways models can differ and the general challenge of forecasting demands further into the future, the two projections within 5% of each other for all demand years except 2045. It indicates that the growth scenarios considered in the regional planning document, Plan Bay Area from ABAG, and those considered by the individual retailers have overall alignment in the countywide demand projection.

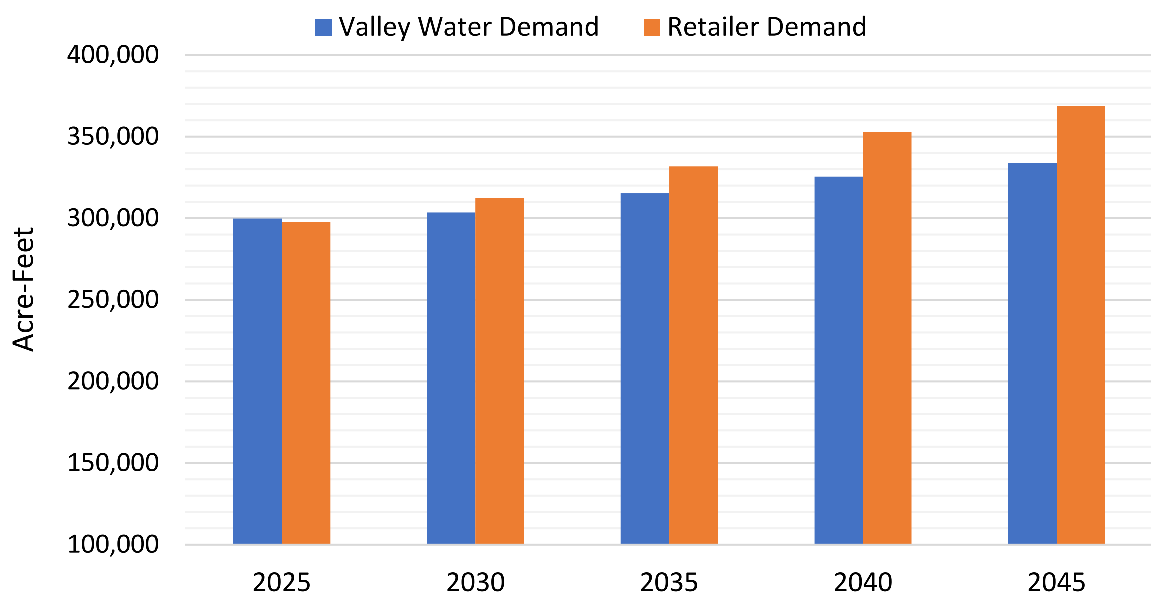

Figure 4-5. Comparison of Total Retailer and Valley Water Projected Demands

Coordination efforts also included collaboration and review of Valley Water's water use projections. In supporting the development of Valley Water's Model, retailers provided Valley Water with their historic water uses by sectors, which were used as observed data to determine the relationship between water use and forecasting variables. During Model development, Valley Water met individually with the retailers to discuss model inputs and assumptions and presented model development updates and results at Board committee and retailer sub-committee meetings. After the Model development was completed, Valley Water provided opportunities for retailers to review and understand the Model-forecasted demands.

While retailer demand forecasts are useful and have been used to inform Valley Water's prior UWMPs, including the most recent 2015 plan, Valley Water is using its custom demand model for planning and for this UWMP to ensure a consistent approach and set of assumptions are used across retailers. To ensure Valley Water continues to meet countywide water demands into the future, Valley Water will continue monitoring water uses in the county and coordinate with the water retailers and regional and statewide efforts to better understand water use patterns. As new information becomes available, Valley Water will adjust its demand projections as needed through the annual MAP process.

4.3.2 Regional Land Use Planning Coordination

Demand projections for this UWMP were based on ABAG's 2017 Plan Bay Area, a long-range regional transportation and land-use blueprint. ABAG's Plan Bay Area incorporates local and regional planning assumptions for population, housing, jobs, and transportation. Therefore, by using the ABAG growth data, regional and local planning and growth were factored into Valley Water's demand projections. In addition, Valley Water also conferred with South County land use planning documents, local County agricultural documents and the Santa Clara County Agricultural Commissioner in developing water use demand assumptions for the agricultural water use sector.

Valley Water has a history of coordinating with land use entities such as cities, ABAG, and MTC on various planning efforts (notably on Urban Water Management Plans every five years), water conservation programs, water supply projects, and the Model Water Efficient New Development Ordinance. Valley Water actively coordinates with land use entities to obtain current and projected development data to quantitatively model long-term water supply and demand projections to support long range water supply planning. Valley Water also participates in Santa Clara County Association of Planning Officials (SCCAPO) meetings to share and exchange information. Valley Water plans to continue building strong relationships with the various planning departments in Santa Clara County. Valley Water also plans to support legislative efforts to promote coordination, and Valley Water's 2021 Legislative Policy Proposals and Guiding Principles include "Support legislative efforts that improve integration of water agencies in land use decision-making processes."

4.4 CLIMATE CHANGE IMPACTS ON WATER DEMAND

Climate change impacts such as warming temperatures, shrinking snowpack, increasing weather extremes, and prolonged droughts pose significant challenges to water resources management. Already, climate change impacts are being observed across California and in the San Francisco Bay Area, and climate modeling projections indicate that these impacts will continue or become more extreme. Climate change has become an important factor in water resources planning in the State and region, although the extent and precise effects of climate change remain uncertain.

Climate change is expected to affect future water demands. While the effects of climate change on demand are not certain, it is anticipated that warmer temperatures and altered rainfall patterns associated with climate change could lead to greater water demands. According to a climate study conducted by Valley Water, average annual maximum temperature within Santa Clara County could increase by 2.0˚F by 2050 under the business as usual scenario, while precipitation in the county will continue to exhibit high year-to-year variability with very wet and very dry years. Projected future increases in temperature can lead to: 1) increased irrigation demands for outdoor landscape or agricultural; 2) increased water use in cooling towers; and 3) increased drought severity and/or length, which could increase the need to request drought-related water use reductions. Valley Water's Demand Model includes temperature and precipitation as forecasting variables and can simulate various climate change scenarios. Valley Water will continue to monitor the science of climate change and revise and update its planning assumptions as more climate studies and data and information become available.

CHAPTER 5 – SBX7-7 BASELINE, TARGETS, AND 2020 COMPLIANCE

The State set a goal of reducing urban water use by 20% with the adoption of the Water Conservation Act of 2009, also known as SB X7-7. Each retail urban water supplier was required to calculate baseline water use for their baseline period and develop water use targets for the years 2015 and 2020 in order to help the State achieve the 20% reduction. Valley Water was very involved in this effort, including participating on the Urban Stakeholder Committee, meeting directly with local water retailers, offering technical and regional alliance support, and reviewing and proposing policies that support ways to meet the targets.

In response to the recent historic drought in California, Governor Brown issued an executive order titled “Making Water Conservation a California Way of Life.” In 2018, Senate Bill (SB) 606 and Assembly Bill (AB) 1668 passed and state-wide implementation will follow in the next decade. Valley Water will continue to be involved with water conservation efforts, supporting local water retailers, and continuing to offer water conservation programs.

5.1 SUPPORT TO RETAILIERS

Under SB X7-7, wholesale water suppliers such as Valley Water are not required to establish and meet baseline and targets for daily per capita water use. However, wholesale agencies are required to provide an assessment of their present and proposed future measures, programs and policies that will help the retail water suppliers in their wholesale service area achieve their SB X7-7 water use reduction targets. This chapter describes the various ways Valley Water is involved and supportive.

5.1.1 Water Conservation Programs

Valley Water has been and continues to be a leader in water conservation with innovative, effective, and comprehensive-in-scope programs. This is consistent with Board Ends Policy E-2 that states Valley Water will “Maximize water use efficiency, water conservation and demand management opportunities.” As one of the initial signatories to the California Urban Water Conservation Council’s (CUWCC) 1991 Memorandum of Understanding Regarding Urban Water Conservation Best Management Practices (MOU), Valley Water is firmly committed to the implementation of the Best Management Practices (BMPs) and Demand Management Measures (DMMs). Valley Water and its major water retailers enjoy a special cooperative partnership in the regional implementation of a variety of water conservation programs, in an effort to permanently reduce water use in Santa Clara County. Chapter 9 of this plan details how Valley Water supports the water retailers with these programs.

5.1.2 Water Retailer Assistance

Valley Water meets regularly with the local water retailers to provide information, offer technical support, and assist with development of regional alliances.

5.1.3 Water Conservation Strategic Plan Development

Valley Water is in process of developing a Water Conservation Strategic Plan (Strategic Plan), which is intended to provide a blueprint for meeting Valley Water’s established conservation policy objectives and targets. The Strategic Plan will evaluate and recommend water conservation measures and programs to meet policy objectives and targets for long-term water conservation and water shortage response; develop schedules for implementation; estimate costs; and identify protocols for monitoring and evaluating program performance over time. The Strategic Plan will also help local water retailers in achieving their water use reduction targets.

The Strategic Plan is also intended to be a tool and reference document to inform and support Valley Water's future conservation program marketing and design. Included in the Strategic Plan will be insights from a retail agency survey, historical participation trends analysis, geospatial participation density analysis, and participation trends by retail agency.

To achieve Valley Water's long-term conservation targets, the Strategic Plan will evaluate and estimate the necessary level of program implementation, an anticipated program schedule that considers device saturation and lifetimes, estimated costs of proposed programs with an emphasis on the most cost-effective programs, and compliance with State of California regulations.

5.1.4 Alternative Methods to Meet Targets

A small but growing source of water for Santa Clara County is recycled water. Using recycled water helps reduce potable water demands; provides a dependable, drought-resilient, locally-controlled water supply; and reduces reliance on imported water. Recycled water is currently about 5% of the county's supply and is distributed for non-potable uses such as landscape and agricultural irrigation, industrial cooling, and dual-plumbed facilities.

Valley Water has recently completed a countywide recycled water master plan, which outlines an approach to achieving its target for recycled water, including both non-potable and potable reuse, to make up 10% of the county's water supply by 2025. Refer to Chapter 6 for more information on Valley Water's recycled water efforts.

CHAPTER 6– SYSTEM SUPPLIES

Valley Water maintains diverse water supply sources to meet countywide demands. Major sources of supply for Valley Water include natural groundwater recharge, local surface water, imported water from the SWP and CVP, and recycled and purified water. In addition, the San Francisco Public Utilities Commission delivers water to eight retailers in the northern part of Santa Clara County, San José Water Company and Stanford have local surface water rights, and several retailers deliver recycled water to customers throughout the county. Potable reuse through groundwater augmentation is a planned future water supply for Valley Water. The projected water supply yields are based on implementing Valley Water's Water Supply Master Plan 2040 (WSMP) in Appendix D.

6.1 IMPORTED WATER

Much of Valley Water's current water supply comes from hundreds of miles away from natural runoff and releases from statewide reservoirs. This imported water is pumped out of the Delta and brought into the county through the complex infrastructure of the SWP and CVP. Valley Water holds contracts for 100,000 AFY from the SWP and for 152,500 AFY from the CVP. The actual amount of water delivered is typically less than these contractual amounts and depends on hydrology, conveyance limitations, and environmental regulations. In addition, supplemental imported water is acquired through transfers and exchanges as needed and available. The imported supplies are sent to Valley Water's three drinking water treatment plants, used for managed groundwater recharge, or stored in local and State and Federal reservoirs for use in subsequent years. Valley Water also stores some of its imported water in the Semitropic Groundwater Bank in the Central Valley for withdrawal during dry periods or as otherwise needed.

Eight retailers in the county have contracts with SFPUC to receive water from the SFPUC Regional Water System. The eight retailers, considered to be wholesale customers of SFPUC, are the cities of Palo Alto, Mountain View, Sunnyvale, Santa Clara, San José, and Milpitas; Purissima Hills Water District; and Stanford University. In addition, NASA-Ames is considered a retail customer of SFPUC. Valley Water does not control or administer SFPUC supplies in the county, but their supply meets some of the countywide demand.

6.1.1 Imported Water Supply Projections

Future SWP allocations are based on the State Water Project Delivery Capability Report (DCR), a biennial report that DWR issues to assist SWP contractors and local planners in assessing the near and long-term availability of supplies from the SWP. DWR issued its most recent update, the draft 2019 DWR State Water Project DCR in August 2020. In this update, DWR provides SWP supply estimates for SWP contractors to use in their planning efforts, including for use in their 2020 UWMPs. The 2019 DCR includes DWR's estimates of SWP water supply availability under both existing (2020) and future conditions (2040).

DWR's estimates of SWP deliveries are based on a computer model that simulates monthly operations of the SWP and CVP systems. Key inputs to the model include the facilities included in the system, hydrologic inflows to the system, regulatory and operational constraints on system operations, and contractor demands for SWP water. In conducting its model studies, DWR must make assumptions regarding each of these key inputs.

In the 2019 DCR for its model study under existing conditions, DWR assumed: existing facilities, hydrologic inflows to the model based on 82 years of historical inflows (1922 through 2003), current regulatory and operational constraints including 2018 Coordinated Operation Agreement Amendment, 2019 biological opinions and 2020 Incidental Take Permit, and contractor demands at maximum

Table A amounts. The long-term average allocation reported in the 2019 DCR for the existing conditions study provide appropriate estimate of the SWP water supply availability under current conditions, which Valley Water used as estimated imported supply for 2025.

To evaluate SWP supply availability under future conditions, the 2019 DCR included a model study representing hydrologic and sea level rise conditions at 2040. The future condition study used all of the same model assumptions as the study under existing conditions, but reflected changes expected to occur from climate change, specifically, projected temperature and precipitation changes centered around 2035 (2020 to 2049) and a 45 cm sea level rise. This future scenario did not include any projected changes to regulations and therefore, may overestimate future SWP and CVP deliveries. However, for the long-term planning purposes of this UWMP, the long-term average allocations reported in the future condition study from 2019 DCR is the only dataset currently available to estimate future SWP water supply availability. This future condition scenario was used to estimate future SWP and CVP supply availability to Valley Water from 2030 to 2045.

Consistent with Executive Order N-10-19, in early 2019, the state announced a new single tunnel project, which proposed a set of new diversion intakes along the Sacramento River in the north Delta for the SWP. In 2019, DWR initiated planning and environmental review for a single tunnel Delta Conveyance Project (DCP) to protect the reliability of SWP supplies from the effects of climate change and seismic events, among other risks. DWR's current schedule for the DCP environmental planning and permitting extends through the end of 2024. DCP will potentially be operational in 2040 following extensive planning, permitting and construction. Since the DCP is still in its early planning phase and costs and yields have not been determined, it is not included as a water supply in this UWMP.

Retailers with SFPUC contracts currently use less than their Individual Supply Guarantees and are projected to increase their use of this source of supply. The SFPUC normal year supply projection in Table 6-1 is based on projections by SFPUC wholesale customers. These projections do not account for potential decreases in supply allocations by the SFPUC during dry years. The total supply projection increases modestly through the planning horizon and remains below the sum of Individual Supply Guarantees for the county. If SFPUC supplies available to its wholesale customers are cut back significantly, the retailers with SFPUC contracts may request increase their use of Valley Water supplies or increase groundwater pumping.

Table 6-1. Projected SFPUC Normal Year Supplies (AF)

Service Area	2025	2030	2035	2040	2045
Milpitas	8,800	9,000	9,600	10,000	10,300
Mountain View	10,200	10,600	11,200	11,700	12,300
Palo Alto	11,300	11,400	11,500	11,800	12,100
Purissima Hills	1,900	1,800	1,800	1,700	1,700
San José Muni	5,000	5,000	5,000	5,000	5,000
Santa Clara	5,000	5,000	5,000	5,000	5,000
Stanford	2,300	2,400	2,600	2,800	3,000
Sunnyvale	10,300	10,400	12,000	12,800	13,600
Total	**55,000**	**56,000**	**59,000**	**61,000**	**63,000**

NOTES: Total numbers are rounded to the nearest 1000 AF. All other numbers are rounded to the nearest 100 AF.

6.1.2 Constraints on Imported Water Supplies

Imported water supplies are subject to hydrologic variability. Local and out-of-county storage can help mitigate the impacts of hydrologic variability.

Valley Water's SWP and CVP water supplies are also subject to a number of additional constraints including regulatory requirements to protect fisheries and water quality in the Delta, and conveyance limitations. Delta-conveyed supplies are also at risk from Delta levee failures due to seismic threats and flooding, sea level rise and climate change, declining populations of protected fish species, and water quality variations (including algal blooms). Many water quality variations are addressed, by blending sources and/or switching sources to the drinking water treatment plants. Algae and disinfection byproduct precursors have been especially challenging during recent drought conditions. To address at least some of these constraints, Valley Water continues to evaluate the costs and benefits of participating in the Delta Conveyance Project relative to other water supply options such as developing additional local supplies, securing and optimizing Valley Water's existing water system, and expanding water conservation.

The Cities of San José and Santa Clara are temporary and interruptible customers of the SFPUC. The SFPUC is scheduled to decide whether to make them permanent customers by December 2028. If San José and Santa Clara SFPUC supplies are interrupted, the cities may need to increase their use of Valley Water supplies.

6.2 GROUNDWATER

Valley Water manages the Santa Clara and Llagas subbasins for the benefit of its groundwater customers and the county at large. Since the 1930s, Valley Water's water supply strategy has been to maximize conjunctive use of surface water and groundwater supplies to enhance water supply reliability and avoid land subsidence. Local groundwater resources make up the foundation of the county's water supply, but they need to be augmented by Valley Water's comprehensive water management activities to reliably meet the needs of county residents, businesses, agriculture, and the environment. These activities include managed recharge of imported and local supplies and in-lieu groundwater recharge through the provision of treated surface water and raw water, acquisition of supplemental water supplies, and water conservation and recycling. Valley Water does not directly deliver groundwater to customers but does have some limited emergency groundwater pumping capacity. Valley Water is the designated Groundwater Sustainability Agency (GSA) for the Santa Clara and Llagas groundwater subbasins under California's 2014 Sustainable Groundwater Management Act (SGMA) and has a DWR-approved Alternative to a Groundwater Sustainability Plan (GSP) in place for sustainably managing these subbasins. Valley Water is also the GSA for the small portion of the North San Benito Subbasin within Santa Clara County (most of the subbasin is in San Benito County). Valley Water is supporting efforts by the San Benito County Water District to develop a GSP for the entire North San Benito Subbasin. The area of the North San Benito Subbasin within Santa Clara County is very small and not addressed further in this UWMP.

6.2.1 Groundwater Basin Description

Santa Clara County includes portions of two groundwater basins as defined by DWR : the Santa Clara Valley Basin (Basin 2-009) and the Gilroy-Hollister Valley Basin (Basin 3-003). The two groundwater subbasins within Santa Clara County managed by Valley Water are the Santa Clara Subbasin (Subbasin 2-009.02) and the Llagas Subbasin (Subbasin 3-003.01), which cover a combined surface area of approximately 385 square miles. Due to different land use and management characteristics, Valley Water further delineates the Santa Clara Subbasin into two groundwater management areas: the Santa

Clara Plain and Coyote Valley. The groundwater subbasins are shown in Figure 6-1. The estimated operational storage capacity of the groundwater subbasins is up to 548,000 AF. Valley Water's managed recharge capacity is up to about 144,000 AFY.

The groundwater subbasins provide multiple benefits to residents, businesses, and the environment in Santa Clara County. Groundwater is pumped from the subbasins by retail water suppliers, agricultural users, and private well owners to support municipal, industrial, agricultural, and domestic uses. Although most of the groundwater pumped is a result of Valley Water managed recharge programs, the subbasins are also recharged by the infiltration of rainfall and natural seepage through local creeks and streams. In addition, the groundwater subbasins serve as an extensive conveyance network, allowing water to move from the recharge areas to individual groundwater wells. The groundwater subbasins provide some natural filtration of surface water as it flows through the soil and rock. Unlike surface water, most groundwater in the county can be served by water retailers without additional treatment beyond disinfection. Lastly, the groundwater subbasins provide water storage, allowing water to be carried over from the wet season to the dry season and even from wet years to dry years.

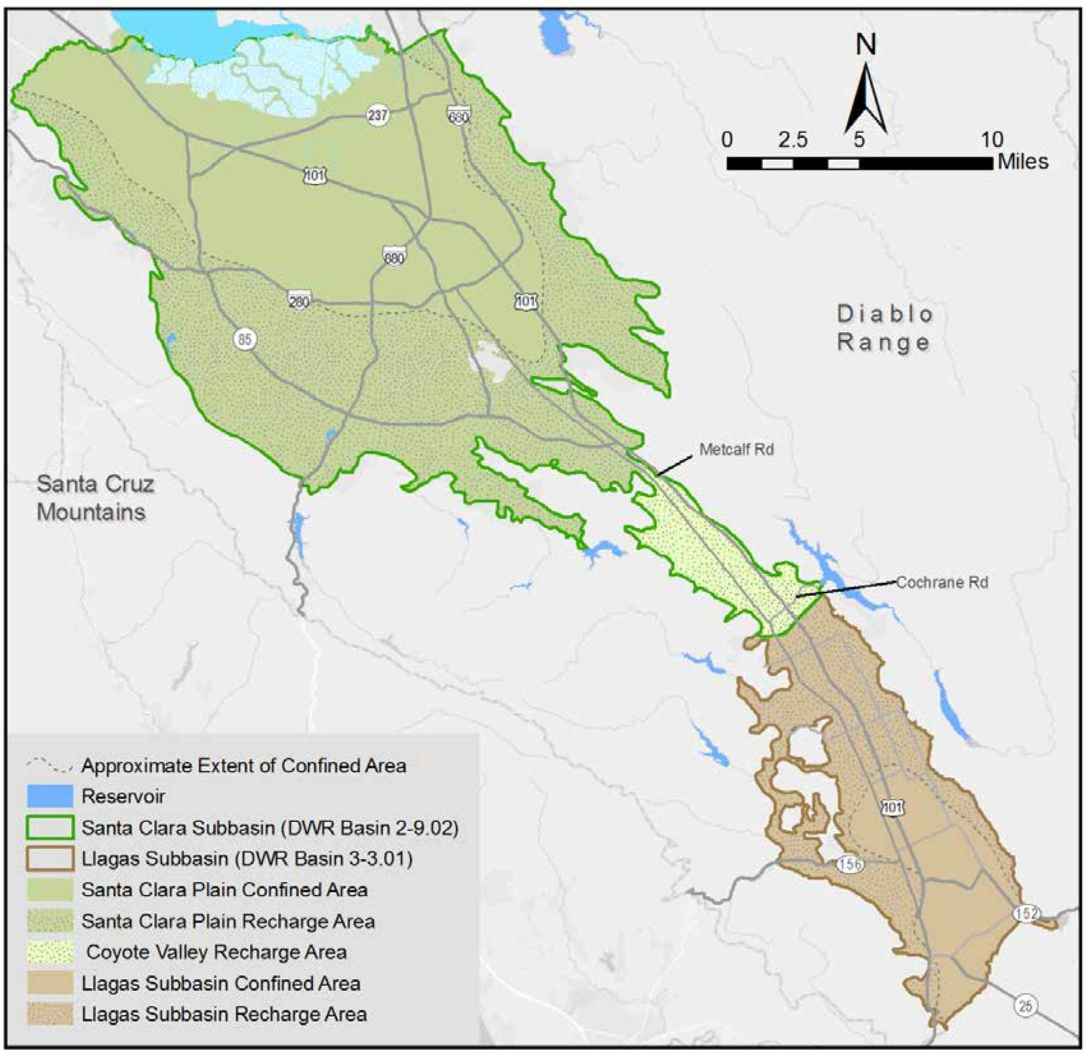

Figure 6-1. Santa Clara County Groundwater

6.2.2 Groundwater Management Plan

DWR has approved Valley Water's Groundwater Management Plan[1] as an Alternative to a GSP for SGMA compliance. The Groundwater Management Plan (included in Appendix E) identifies the following two basin management objectives (BMO):

- BMO 1: Groundwater supplies are managed to optimize water supply reliability and minimize land subsidence.
- BMO 2: Groundwater is protected from existing and potential contamination, including salt water intrusion.

These BMOs describe the overall goals of Valley Water's groundwater management program. Basin management strategies were developed to meet the BMOs. Many of these strategies have overlapping benefits to groundwater resources, acting to improve water supply reliability, minimize subsidence, and protect or improve groundwater quality. The strategies are listed below.

1. Manage groundwater in conjunction with surface water through direct and in-lieu recharge programs to sustain groundwater supplies and to minimize salt water intrusion and land subsidence.
2. Implement programs to protect or promote groundwater quality to support beneficial uses.
3. Maintain and develop adequate groundwater models and monitoring systems.
4. Work with regulatory and land use agencies to protect recharge areas, promote natural recharge, and prevent groundwater contamination.

Valley Water has implemented numerous programs to protect groundwater resources, including comprehensive monitoring programs related to groundwater levels, land subsidence, groundwater quality, recharge water quality, and surface water flow. In addition, Valley Water has developed the following outcome measures in the Groundwater Management Plan to gauge performance in meeting the basin management objectives:

1. Projected end of year groundwater storage is greater than 278,000 AF in the Santa Clara Plain, 5,000 AF in Coyote Valley, and 17,000 AF in the Llagas Subbasin.
2. Groundwater levels are above subsidence thresholds at the subsidence index wells.
3. At least 95% of countywide water supply wells meet primary drinking water standards and at least 90% of South County wells meet Basin Plan agricultural objectives.
4. At least 90% of wells have stable or decreasing concentrations of nitrate, chloride, and total dissolved solids.

Valley Water will update its Groundwater Management Plan in 2021 and submit it to DWR by January 2022 to meet SGMA requirements for five-year updates.

[1] https://www.valleywater.org/your-water/where-your-water-comes-from/groundwater/sustainable-groundwater-management

6.2.3 Current Conditions

DWR has identified both the Santa Clara and Llagas subbasins as high priority subbasins based on criteria that include overlying population, projected growth, number of wells, irrigation acreage, groundwater reliance, and groundwater impacts. Neither subbasin has been identified by DWR as being critically overdrafted.

Groundwater conditions throughout the county are sustainable, with managed and in-lieu recharge programs maintaining adequate storage to meet annual water supply needs and provide a buffer against drought or other shortages. Although groundwater levels declined during the recent (2012-2016) statewide drought, groundwater levels in the Santa Clara and Llagas subbasins quickly recovered after the drought due largely to Valley Water's proactive response and comprehensive water management activities. Valley Water monitors water levels and water quality at wells throughout the county. In addition, it evaluates data from local water suppliers to assess regional groundwater quality and identify potential threats so they can be appropriately addressed. Valley Water also monitors the quality of water used for groundwater recharge to ensure groundwater resources are protected.

Most wells in Santa Clara County produce high-quality water that meets drinking water standards without the need for treatment beyond disinfection. The primary exception is nitrate, which is elevated in many South County wells (primarily domestic wells) and continues to be a groundwater quality challenge. Cleanup is ongoing at a number of sites with industrial contaminants in groundwater, and elevated levels of perchlorate are still observed in a few South County wells. Though not currently regulated in drinking water, per- and polyfluoroalkyl substances (PFAS) are causing increased interest and prompting concern nationwide. Regional testing does not indicate widespread PFAS presence in groundwater, but one water retailer has removed ten wells from service out of an abundance of caution. Valley Water will continue to evaluate the threat posed by PFAS and other contaminants, and to work with other agencies, basin stakeholders, and the public to address these issues and ensure groundwater quality remains high.

Valley Water produces comprehensive groundwater monitoring reports that are available at https://www.valleywater.org/your-water/where-your-water-comes-from/groundwater/groundwater-monitoring.

6.2.4 Natural Groundwater Recharge Supply Projection

Valley Water includes natural groundwater recharge as a source of supply for long-term water supply planning purposes, because it contributes to the available groundwater supply. Natural recharge includes all uncontrolled recharge, including the deep percolation of rainfall, septic system and/or irrigation return flows, and natural seepage through creeks. Based on estimates from Valley Water's groundwater flow and Water Evaluation and Planning (WEAP) models, future average natural groundwater recharge is projected to be fairly constant over the planning horizon.

6.2.5 Constraints on Groundwater Supply

Groundwater supply is largely constrained by hydrologic variability and the estimated 548,000 AF of operational storage capacity within the subbasins. The inflows to the groundwater subbasins are constrained by Valley Water's managed aquifer recharge program and natural recharge. Valley Water has about 144,000 AFY of managed recharge capacity, including more than 90 miles of in-stream recharge and 102 off-stream recharge ponds. Maintaining Valley Water's managed recharge program requires ongoing operational planning for the distribution of local and imported water to recharge facilities; maintenance and operation of reservoirs, diversion facilities, distribution systems, and recharge ponds; and the maintenance of water supply contracts, water rights, and relevant environmental clearance. Valley Water's managed recharge program is critical to maintaining groundwater supply, because natural recharge is insufficient to meet groundwater demands. However, protecting natural recharge capacity is also important. Valley Water's District Act and Board policy help preserve open space that supports agriculture and natural recharge capacity.

Groundwater quality can also be a constraint on groundwater supply. In general, the Santa Clara and Llagas Subbasins have high-quality groundwater, except for nitrate, which is elevated in some wells in the Coyote Valley and Llagas Subbasin from historic and ongoing sources including fertilizers, septic systems, and animal waste. However, nitrate concentrations are generally stable or declining and Valley Water has many programs to protect groundwater quality, including several targeted to improve nitrate in groundwater. Additional details about constraints on groundwater supply and quality and Valley Water's comprehensive groundwater management strategies are described in the 2016 Groundwater Management Plan: https://www.valleywater.org/your-water/where-your-water-comes-from/groundwater/sustainable-groundwater-management.

6.3 LOCAL SURFACE WATER

Valley Water currently has 20 appropriative water rights licenses and one filed water right permit with the State Water Resources Control Board totaling over 227,300 AFY. In addition, two of Valley Water's retailers, San José Water Company and Stanford University, have their own surface water rights that contribute to local surface water availability to their in-county customers. Local runoff is captured in Valley Water's 10 reservoirs, with a total storage capacity of about 166,000 acre-feet, though several are operating at restricted capacity due to seismic stability concerns (Table 6-2). Most of the reservoirs are sized for annual operations, storing water in winter for use in summer and fall. The exception is the Anderson-Coyote reservoir system, which provides valuable carryover of supplies from year to year. Supplies captured in local reservoirs are sent to drinking water treatment plants or diverted downstream for groundwater recharge and to maintain aquatic habitats.

Table 6-2. Existing Reservoir Capacities, Restrictions, and Water Supply Impacts from Restrictions

Reservoir	Reservoir Capacity (AF)	Restricted Capacity (AF)	Restricted Capacity (%)
Almaden	1,555	1,443	93%
Anderson	89,278	2,820	3% (deadpool)
Calero	9,738	4,414	45%
Coyote	22,541	11,843	53%
Guadalupe	3,320	2,134	64%
Stevens Creek	3,056	No restriction	–
Lexington	18,534	No restriction	–
Chesbro	7,967	No restriction	–
Uvas	9, 688	No restriction	–
Vasona	463	No restriction	–
TOTAL	**166,140**	**62,362**	–

6.3.1 Local Surface Water Supply Projection

Valley Water regularly exercises its water rights to ensure the availability of this resource into the future. Future average use of local surface water supply is projected to increase over the planning horizon as Valley Water's dams are seismically retrofitted, allowing operating capacity restrictions to be lifted. To increase the seismic stability of Anderson Dam, Valley Water drained Anderson Reservoir to deadpool (3% of capacity) in October 2020, the lowest level that can be reached through the existing outlet tunnel, to prepare for the reconstruction of the existing earthen Anderson Dam. The reconstruction is expected to last about 10 years and will allow Anderson Reservoir to return to its full operating capacity once completed.

Currently, four other reservoirs are also operating in restricted capacity (Table 6-2). The seismic retrofit is planned for three of them (Almaden, Calero, Guadalupe) to be completed around 2030 to 2035. These reservoirs will reassume their full operating capacity by that time.

6.3.2 Constraints on Local Water Supplies

Local surface water supplies are vulnerable to hydrologic variability, with most reservoirs sized for annual operations. In wetter years, Valley Water is challenged to capture all available supply due to reservoir capacity constraints and flood protection needs. In drier years, Valley Water is challenged to maintain its groundwater recharge program due to reduced storage in local reservoirs, reduced imported water allocations, and regulations and permit conditions that require Valley Water to maintain environmental stream flows.

Other factors can also impact Valley Water's reservoir operations and its use of surface water rights, including meeting reservoir operating rules designed to provide incidental flood protection, maintaining storage levels for environmental or recreation purposes, and dam safety requirements. In 1996, a water rights complaint was filed at the State Water Resources Control Board (SWRCB) challenging that Valley Water's annual operations on Coyote Creek, Guadalupe River, and Stevens Creek impact steelhead trout and Chinook salmon. In 1997, the Central California Coast Steelhead was listed as a threatened species under the Federal Endangered Species Act (ESA). To address the complaint and ESA issues, Valley Water, Guadalupe-Coyote Resource Conservation District (GCRCD), Trout Unlimited, the California Department of Fish and Wildlife (CDFW), U. S. Fish and Wildlife Service (USFWS), and National Marine Fisheries Service (NMFS), established the Fisheries and Aquatic Habitat Collaborative Effort (FAHCE) to develop a Settlement Agreement. The Settlement Agreement was initialed in 2003. Although GCRCD withdrew as a party to the Settlement Agreement, Valley Water continues with its implementation. In addition to several fish habitat improvements already completed as early FAHCE implementation, a key Settlement Agreement provision requires the development of a Fish Habitat Restoration Plan (Restoration Plan), which will implement changes in reservoir releases to continue its support of instream flow needs for salmonids, provide channel enhancements, and include monitoring and adaptive management.

6.4 STORMWATER

Valley Water's managed recharge program includes capturing local runoff in reservoirs and releasing it to groundwater recharge facilities or drinking water treatment plants. On average, about 50,000 AFY of local runoff is recharged through existing recharge facilities. Through its water supply master planning, Valley Water plans to increase stormwater capture and reuse capacity as part of its 'ensure sustainability strategy'. Valley Water's stormwater projects for the next 20 years are summarized below.

- **Green Stormwater Infrastructure (GSI)**. As part of its conservation program, Valley Water initiated a rebate program to incentivize the installation of rain barrels and cisterns, and the construction of rain gardens in residential and commercial landscapes.

- **Flood-Managed Aquifer Recharge (Flood-MAR)**. Valley Water is currently completing phase one of a feasibility study for capturing and recharging stormwater on open space in Santa Clara County, a process referred to as Flood-MAR. Phase one will determine if and where there may be potential areas suitable for Flood-MAR in Santa Clara County, identify technical approaches and related institutional requirements (e.g., permits, water rights, etc.) for installing Flood-MAR projects on lands identified as suitable, and determine potential program incentives. The first phase of the feasibility study is scheduled for completion in 2022.

- **Centralized Stormwater Capture Projects**. Centralized stormwater capture projects capture stormwater from multiple parcels for recharge in a single location and/or are municipal projects, including "green streets" projects. The Santa Clara Basin Storm Water Resources Plan completed in December 2018 identified potential projects throughout northern Santa Clara County. These projects would likely be partnerships with other jurisdictions and require outside funding, so their schedules are yet to be determined. Valley Water will continue to track project opportunities through its participation in the Santa Clara Valley Urban Runoff Pollution Prevention Program. In addition, Valley Water's WSMP includes the Upper Penitencia Creek flood protection project, which could include stormwater retention components.

- **South County Stormwater Resource Plan**. Valley Water developed a Stormwater Resource Plan (SWRP) in collaboration with stormwater permittees in South County (Gilroy, Morgan Hill, and County of Santa Clara) to identify and prioritize GSI opportunities that could be eligible for funding. The SWRP is a planning document that uses a map-based approach to identify and prioritize local and regional GSI projects that can be implemented to improve local surface-water quality through enhanced stormwater management. GSI reduces the quantity and improves the quality of water flowing into our creeks, while also providing other possible benefits, including groundwater infiltration, flood attenuation, aesthetics, reduction in heat islands, and other community benefits. The South County Stormwater Resource Plan is available here.

The greatest risk for implementing stormwater projects is finding willing partners for projects that are cost-effective for Valley Water's water supply program. This risk is somewhat mitigated by regulatory requirements for stormwater management and the availability of grants and other funding sources in support of green infrastructure projects that provide water supply benefits.

6.5 RECYCLED AND PURIFIED WATER

A growing source of water supply for Santa Clara County is recycled and purified water. Recycled water is wastewater that is cleaned through multiple levels of treatment. Purified water is highly treated water of wastewater origin that has passed through proven multistage, multibarrier processes (such as microfiltration, reverse osmosis, and ultraviolet disinfection) to produce water at the quality fit to supplement or provide supply for potable (drinking) water purposes, as verified through monitoring for its safety and as regulated by the State Water Resources Control Board Division of Drinking Water. Using recycled and purified water can help augment drinking water and groundwater supplies through in-lieu recharge; provides a reliable, drought-resilient, locally controlled water supply; and reduces reliance on imported water. Over the past decade, Valley Water has advanced water reuse in the County by leading water reuse planning efforts for the County, developing wholesale recycled water programs, and constructing new infrastructure. Currently, recycled water is about 5% (17,000 AFY, CY 2020) of the county's water supply that is distributed for non-potable uses such as landscape irrigation, industrial cooling, and dual plumbed facilities. This recycled water is produced at the four wastewater treatment plants in the county - Palo Alto Regional Water Quality Control Plant, City of Sunnyvale Water Pollution Control Plant, San José -Santa Clara Regional Wastewater Facility, and South County Regional Wastewater Authority (SCRWA).

Valley Water constructed the Silicon Valley Advanced Water Purification Center (SVAWPC) as a demonstration facility to develop purified water. The SVAWPC can produce up to 8 million gallons of purified water per day, which is currently blended with tertiary treated water to improve the quality for non-potable use for use by a wider variety of customers. Since March 2014, the SVAWPC has demonstrated the effectiveness of advanced treatment technologies (microfiltration, reverse osmosis, ultraviolet light, and advanced oxidation) to produce purified water suitable for potable uses, and set the stage for Valley Water to begin a potable reuse program. Potable reuse will involve using advanced purified water to augment groundwater or surface water supplies.

Valley Water is working with the cities of Palo Alto and Mountain View on additional recycled water options within those cities. In December 2019, Valley Water executed an agreement with the cities of Palo Alto and Mountain View that defined cost-sharing and supply commitments related to future water reuse. A key provision of this agreement is construction of a local Salt Removal Facility in Palo Alto for enhanced Non-potable Reuse (NPR+), which is recycled water for non-potable reuse that has been blended with purified water to reduce concentration of salts and other dissolved solids to enable broader application of recycled water for non-potable end uses and to protect groundwater quality. Other key provisions of this agreement include a minimum commitment of approximately 11,000 AFY of wastewater effluent to Valley Water for purified water production at a future regional Advanced Water Purification Facility, and a water supply option for the cities of Palo Alto and Mountain View to request additional supply if needed.

Valley Water is also working with the cities of San José, Santa Clara, and Palo Alto on a location for a regional Advanced Water Purification Facility at the SVAWPC. The regional facility, to be located in either San José or Palo Alto, would produce up to 11,000 AFY of potable reuse supply by 2028 to replenish groundwater.

Valley Water completed a Countywide Water Reuse Master Plan (CoRe Plan) in 2021 to identify feasible opportunities to expand water reuse, improve water supply reliability, and increase regional self-reliance. The CoRe Plan outlines Valley Water's opportunities and strategies toward achieving up to 24,000 AFY for potable water reuse. The draft CoRe Plan is available at https://fta.valleywater.org/fl/XNyG7Fja6T#folder-link/.

6.5.1 Non-Potable Reuse

The City of San José operates the South Bay Water Recycling (SBWR) system and distributes recycled water generated by the San José/Santa Clara Regional Wastewater Facility. Treated wastewater is supplied to Valley Water's adjacent SVAWPC to be purified with advanced technologies. The SBWR Strategic and Master Plan, which discusses non-potable and potable reuse opportunities, is available at https://s3.us-west-2.amazonaws.com/assets.valleywater.org/South%20Bay%20Water%20Recycling%20-%20Final%20Report%202015.pdf.

Valley Water is a partner with the City of Sunnyvale on the Wolfe Road Recycled Water Facilities Project, which was completed in 2019 and supplies the Apple Park Headquarters with non-potable recycled water for landscape irrigation, toilet flushing, and industrial cooling. By mutual agreement, Valley Water owns while Sunnyvale operates and maintains the Wolfe Road pipeline. Recycled water conveyed through this pipeline is owned by Valley Water and may be resold to other Sunnyvale customers fed from the pipeline, regardless of their location.

In South Santa Clara County, Valley Water partners with the SCRWA, City of Gilroy, and City of Morgan Hill on recycled water programs. Consistent with existing agreements, SCRWA is the recycled water producer, Valley Water is the recycled water wholesaler, and Gilroy and Morgan Hill are the retailers (although recycled water is not currently delivered to Morgan Hill). The 2015 South County Recycled Water Master Plan Update provides a blueprint to expand non-potable reuse infrastructure and is available at https://www.valleywater.org/your-water/recycled-and-purified-water.

6.5.2 Potable Reuse

Valley Water's WSMP includes developing 24,000 AFY of potable reuse capacity. The current plan is that treated wastewater would be purified at a new or expanded purification center in North County and then used to recharge the groundwater at Valley Water's Los Gatos Recharge ponds. Through the agreement with the cities of Palo Alto and Mountain View, Valley Water is evaluating an expanded and expedited potable reuse program that initially would include at least 11,000 AFY of potable reuse production capacity with the potential to increase to 14,000 AFY by 2028, depending on wastewater availability. Valley Water is still evaluating other approaches for securing wastewater supplies to develop a total of 24,000 AFY of potable reuse supplies.

6.5.3 Recycled Water Supply Projection

Valley Water's non-potable recycled water use in CY 2020 was approximately 17,000 AF and projected future use is summarized in Table 6-3. These projections are based on recycled water use estimates provided by the water retailers and increases over the planning horizon.

Valley Water's baseline potable reuse program goal of 11,000 AFY of production capacity for groundwater recharge is scheduled to be on-line by 2028. Based on water supply system modeling, the program will operate at full capacity in dry years, but not necessarily in wetter years or when groundwater levels are high. Average use will increase over time as demands on the groundwater subbasin increase. Additional capacity may be developed in future phases depending on water supply needs, new regulations provided for direct potable reuse, and reverse osmosis concentrate disposal capacity.

Table 6-3. Non-Potable Recycled Water Supply Projection (AF)

Service Area	2025	2030	2035	2040	2045
South Bay Water Recycling					
Milpitas	1200	1200	1200	1200	1200
San José Municipal Water System	4,800	5,500	6,300	7,400	7,400
San Jose Water Company	2,700	3,100	3,600	3,700	3,600
Santa Clara	4,600	5,500	6,600	7,900	9,500

Service Area	2025	2030	2035	2040	2045
Sunnyvale Water Pollution Control Plant					
California Water Service Company	100	100	100	100	100
Sunnyvale	1,000	1,000	1,100	1,200	1,600
Palo Alto Regional Water Quality Control Plant					
Mountain View	500	500	500	500	500
Palo Alto	300	300	300	300	300
South County Regional Wastewater Authority					
Morgan Hill	0	700	1,500	2,200	2,900
Gilroy	1,000	1,200	1,200	1,200	1,200
Total	**16,000**	**19,000**	**22,000**	**26,000**	**28,000**
NOTE: Total numbers are rounded to the nearest 1000 AF. All other numbers are rounded to the nearest 100 AF.					

6.5.4 Constraints on Recycled Water Supplies

The constraints on future SBWR deliveries to retailers include infrastructure capacity and the availability of recycled water (depending on the amount of potable reuse capacity Valley Water develops). Valley Water is including all retailer projections in this analysis because the amount of influent to the SBWR system is sufficient to meet the combined non-potable retailer demands (current and projected) and the assumed potable reuse demand. The CoRe Plan estimates higher use of NPR than Table 6-3, i.e., 39,100 to 42,000 AFY of recycled water by 2040 depending on Morgan Hill's conceptual buildout demands per the 2015 South County Recycled Water Master Plan Update.

Some of the potential constraints on the development of potable reuse include RO concentrate disposal, source water availability, public acceptance, permitting, hydrogeologic conditions, and cost. Once the program is implemented, the largest challenge will be maximizing the use of the available supply during wetter years when storage is full and/or other lower cost supplies are competing for use. These constraints are being addressed as part of the Purified Water Program.

6.6 DESALINATION

Valley Water is a partner in the Bay Area Regional Desalination Project (BARDP), which is evaluating purifying brackish water from Mallard Slough using Contra Costa Water District water rights. Partners include San Francisco Public Utility Commission, Zone 7 Water Agency, and Contra Costa Water District. Partners built a pilot plant in October 2008 and completed the pilot study in April 2009, which showed the project is feasible. Since the pilot study, the 2012-2016 drought showed that the water rights that would be exercised to divert flows to the plant may not be fully available during droughts. Partners are evaluating the water rights to determine how much water can be reliably produced by a desalination facility. In addition, partners are evaluating approaches for conveying project water to each partner agency.

While each of the partner agencies continues to evaluate its need for the project, the agencies are collectively embarking on a study to look more broadly at all the available opportunities to optimize the sharing of water resources across the region, referred to as the Bay Area Regional Reliability Project (BARR Project). BARR agencies include the BARDP partners, East Bay Municipal Utility District, and Alameda County Water District. Through BARR, the agencies will consider the use of existing supplies as well as new supplies through desalination. By taking a more holistic and regional approach to water supply planning, the agencies hope to make the best use of scarce resources to serve the future needs of the Bay Area. More information on the regional desalination project is available at https://regionaldesal.squarespace.com/. Currently, Valley Water is not including desalination in its projected water supplies.

6.7 WATER EXCHANGE AND TRANSFERS

Valley Water conducts short-term water transfers and exchanges as a part of its routine imported water operations. As a reference, Valley Water was able to secure over 13,400 AF of transfer supply in 2020. While Valley Water considers water exchange and transfers as one of the potential options to secure additional water during critical dry years through long-term agreements, there are considerable uncertainties with long term costs and ability to make transfers in critical dry years, during which water quality challenges, regulatory requirements, and pumping restrictions may affect the ability to convey transfer supplies across the Delta. Consequently, Valley Water is not including water transfers and exchanges in its projected water supplies in this plan, except in the Drought Risk Analysis.

6.8 RESERVES

Reserves include local groundwater storage and out of County storage in Semitropic Groundwater Bank and San Luis reservoir. Valley Water puts water into reserves when other supplies exceed demands and takes water out of reserves when other supplies are less than demands. Within a single year, Valley Water may put water into and take water out of reserves, depending on conditions at the time of year. Reserves play a critical role in Valley Water’s overall water management strategy and its ability to meet demands during droughts.

One constraint on using local groundwater storage reserves is the need to maintain adequate groundwater levels to avoid land subsidence, maintaining adequate water levels to support pumping for beneficial use, and providing a reserve in case the subsequent year is dry. The use of local water carryover can be constrained by regulations governing instream flow requirements. Another constraint on reserves is finite storage capacity. In very wet years, Valley Water is challenged to find a place to use or store all its available supplies.

6.9 FUTURE WATER SUPPLY PROJECTS

Valley Water’s WSMP identifies several projects and programs that will increase local water supply to meet future countywide demand. These projects are in the various stages of planning, design, and construction. These projects are summarized in Table 6-4 and their estimated yields are included in the supply projections in Table 6-5. The expansion of Pacheco Reservoir in southern Santa Clara County is one of the proposed future projects identified in the WSMP. Pacheco Reservoir would act as a surface bank for Valley Water’s existing supplies and diversify its reserve storage by increasing the volume of locally banked reserves. In addition, by increasing locally available storage, Valley Water may be better positioned to respond to future water supply emergencies.

Table 6-4. Future Water Supply Projects

Project	Planned Operating Year	Expected Increase in Water Supply (AFY)
Dam Improvements/Seismic Retrofits		
Almaden	2035	17,440
Anderson	2030	
Calero	2035	
Guadalupe	2035	
Delta Conveyance Project	2040	TBD
Pacheco Reservoir Expansion	2035	–
Potable Reuse Program	2028	9,000[1]
Transfer Bethany Pipeline	2025	1,000
[1]Based on an 11,000 AF production capacity with 80% efficiency.		

6.10 SUMMARY OF EXISTING AND PROJECTED WATER SUPPLIES

Valley Water's existing and projected average water supplies are presented in Table 6-5. To forecast average available supplies, Valley Water uses its Water Evaluation and Planning (WEAP) model. The WEAP model simulates future demands, investments, and certain expected future regulations (e.g., local minimum stream flows) using the historic hydrology from 1922-2015 (94 years). Valley Water presents the average supply over 94-years of hydrology (1922 – 2015) in Table 6-5 for groundwater, local surface water, and CVP and SWP allocations, since their availability varies greatly from year to year with hydrologic cycles and there is not a single representative year. Available recycled water and potable reuse supplies are summed and do not significantly vary year to year in the WEAP model. A complete summary of assumptions used in the modeling for this plan is provided in Appendix F.

Actual availability of each supply during any given year depends on hydrology, groundwater recharge operations and conditions, and other factors. Groundwater storage shown assumes groundwater can be drawn down to the severe stage of the Water Shortage Contingency Plan. This does not represent a sustainable long-term groundwater condition, but these supplies represent water that may be needed to get through a prolonged drought. Imported water allocations are provided by DWR in their DCR 2019, which does not include any projected changes to future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. However, through Valley Water's Monitoring and Assessment Program (MAP), Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future. Projects included in the supply projections include transfer Bethany pipeline (2025); Anderson dam seismic retrofit and potable reuse (2030); Guadalupe, Calero, and Almaden dam seismic retrofits and Pacheco Reservoir Expansion (2035); and an additional 35,000 AF of conservation (to reach Valley Water's goal of 109,000 AF by 2040 with a 1992 baseline).

In WEAP, SFPUC supplies decrease in some dry years and droughts, but in most years the amount available is the same as listed in Table 6-5. However, depending on the voluntary agreements for the Bay Delta Plan Phase 1 on the Tuolumne, SFPUC supplies may be significantly decreased in the future. At present, there is not sufficient understanding of how the Phase 1 will impact SFPUC supplies that is provided to Valley Water's retailers, so the potential impact of the voluntary agreements is not yet included in Valley Water's supply analysis. However, Valley Water is tracking this regulatory development and proposing a conservative investment approach for the future to be able to respond to supply uncertainties such as this.

Table 6-5. Projected Average Water Supplies (AF)

Water Supply	2025	2030	2035	2040	2045
Surface water	30,000	70,000	185,000	185,000	185,000
Imported water	130,000	134,000	136,000	139,000	142,000
SFPUC Supply	55,000	56,000	59,000	61,000	63,000
Local groundwater storage	140,000	164,000	163,000	162,000	162,000
Out of County Storage	75,000	75,000	75,000	70,000	70,000
Recycled water (non-potable)	16,000	19,000	22,000	26,000	28,000
Total	**446,000**	**518,000**	**640,000**	**643,000**	**650,000**

NOTES: Recycled water, SFPUC supply, and groundwater storage are rounded to the nearest 1,000 AF. All other supplies are rounded to the nearest 5,000 AF. Supplies shown are based on modeled estimates of available supplies. Actual availability during any given year depends on hydrology, groundwater recharge operations and conditions, regulatory requirements, and other factors. Groundwater storage shown assumes groundwater can be drawn down to the severe stage of the Water Shortage Contingency Plan. This does not represent a sustainable long-term groundwater condition, but these supplies represent water that may be needed to get through a prolonged drought. Imported water allocations are provided by DWR in their Delivery Capability Report (DCR) 2019, which does not include any projected changes to future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. However, through Valley Water's Monitoring and Assessment Program, Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future. Projects included in the supply projections include transfer Bethany pipeline (2025); Anderson dam seismic retrofit and potable reuse (2030); Guadalupe, Calero, and Almaden dam seismic retrofits and Pacheco Reservoir Expansion (2035); and an additional 35,000 AF of conservation (to reach Valley Water's goal of 109,000 AF by 2040 with a 1992 baseline).

6.11 CLIMATE CHANGE IMPACTS TO WATER SUPPLY

Climate change impacts such as warming temperatures, shrinking snowpack, increasing weather extremes, and prolonged droughts pose significant challenges in water resources management, potentially including Valley Water's operational flexibility and water supply availability. Already, climate change impacts are being observed across California and in the San Francisco Bay Area, and climate modeling projections indicate that these impacts will continue or become more extreme. Historic data show that average annual maximum temperatures in Santa Clara County have increased by 2.5°F since 1950. According to *California's Fourth Climate Change Assessment*, sea level has risen over 8 inches in the last 100 years, and the 2012-2016 drought led to a 1-in-500 year low in Sierra snowpack and $2.1 billion in economic losses statewide. The Bay Area will likely see a significant temperature increase by mid-century. Precipitation will continue to exhibit high year-to year variability, with very wet and very dry years. Average Sierra Nevada snowpack is projected to decline, up to 60% in mid-century under a business-as-usual greenhouse gas emissions scenario. Future increases in temperature will likely cause longer and deeper droughts.

CHAPTER 6 – SYSTEM SUPPLIES

Statewide and local changes in precipitation and temperature could significantly impact Valley Water's water supplies and operations, the effectiveness of potential water supply investments, and water demand patterns. Specifically, Valley Water's water supply vulnerabilities to climate change include:

- **Decreases in the quantity of imported water supplies**. Currently the Sierra snowpack acts as a reservoir that captures precipitation as snow in the winter and releases it as runoff through the spring and summer where it is captured by reservoirs in the SWP and CVP system. More precipitation falling as rain and earlier snowmelt in the Sierra may exceed the storage capabilities of the existing SWP and CVP reservoirs meaning much of this runoff would be lost as a water supply. Increases in temperature and evapotranspiration may also lead to a higher intensity of droughts, which can decrease imported water allocations. Rising air temperatures will also increase water temperatures in reservoirs and the Delta, which can lead to increased evaporation rates, a higher risk of harmful algal blooms, and negative impacts to fish and wildlife, all of which can impact the availability of imported water supplies for Valley Water. Sea level rise will also have negative impacts on imported water supplies, largely because of saltwater intrusion into the Delta. Saltwater intrusion can impact water supply allocations, as more fresh water will be needed to flow through the Delta and into San Francisco Bay to hold back saltwater, making it unavailable for CVP and SWP use.

- **Decreases in the ability to utilize local surface water supplies**. Shifts in the timing and intensity of rainfall and runoff could affect Valley Water's ability to capture and use local surface water supplies. It is difficult to capture rainfall when it comes in a few intense storms because reservoirs are more likely to fill and spill, or additional releases will be needed to make room for the storm flows. When it is wet, there are typically lower demands for water, so storm flows and releases to provide additional storage capacity are difficult to put to immediate use. Thus, even if average annual rainfall stays the same, the ability to utilize local supplies may decrease.

- **Increases in irrigation and cooling water demands**. Higher temperatures will increase irrigation demands for agricultural, residential, and commercial/institutional uses, which account for about 40% of water use in the county. Also, the county has several energy plants, multiple data centers, and facilities with cooling towers. Higher temperatures may also increase demands by these users.

- **Decreases in water quality**. Higher temperatures, wildfire, and changes in flow patterns could result in more algal blooms, and increased turbidity in imported and local surface water supplies. Sea level rise could also contribute to increased salinity in Delta conveyed supplies. At a minimum, changes in water quality require additional monitoring. Often, degrading in water quality requires changes to treatment processes, and sometimes, can result in the interruption of supplies from the CVP or SWP.

- **Increases in the severity and duration of droughts**. Droughts are already Valley Water's greatest water supply challenge. With increases in demands and potential reductions in supplies from climate change, this challenge will only grow. Without additional supplies and demand management measures, Valley Water would need to call for more frequent and severe water use reductions. These actions affect the economic and social well-being of the county. More severe and longer droughts will also affect the environmental well-being of the county. Valley Water needs to implement a water supply strategy that will adapt well to future climate change by managing demands, providing drought-resilient supplies, and increasing system flexibility in managing supplies and water quality.

CHAPTER 6 – SYSTEM SUPPLIES

Recognizing the challenges posed by climate change to water supply reliability, Valley Water has embarked on a number of efforts to understand and develop mitigation actions for climate change impacts. Through its annual MAP update and using the WEAP model, Valley Water is analyzing climate impacts to quantify the effect on Valley Water's existing and future local supply. In addition, since imported water represents a significant source in Valley Water's portfolio, Valley Water is in the process of developing a climate study that will use CalSim3 to quantify potential climate change and regulatory impacts to Valley Water's CVP and SWP allocations. Valley Water relies on its long-term master planning efforts to continually develop and improve resilient and adaptable water supplies and strategies. Valley Water's WSMP is reviewed annually and updated every five years to adapt to changing conditions. The most recent update was completed in 2019. The WSMP will continue to develop elements that adapt to future climate changes.

Furthermore, to address climate change impacts to ensure it can continue to provide a clean, reliable water supply, natural flood protection, and water resources stewardship in the future, Valley Water developed a Climate Change Action Plan (CCAP). The CCAP provides goals, strategies, and actions for each of Valley Water's mission areas, including water supply reliability, flood risk reduction, and water resources stewardship, as well as for emergency response. The goals and strategies developed through the CCAP planning process will guide the implementation of specific actions to address climate change. More information about the CCAP can be found at https://www.valleywater.org/your-water/water-supply-planning/climate-change-action-plan.

For the implementation actions, Valley Water is actively promoting water conservation and reuse to increase resilience and mitigate climate change impacts. Valley Water's long-term and comprehensive water conservation and demand management efforts are described in Chapter 9, while existing and future water reuse is detailed in Section 6.4.

6.12 ENERGY INTENSITY

Valley Water has a large network of water infrastructure that includes three water treatment plants, an advanced recycled water purification center, three pump stations, recharge facilities, and 142 miles of pipeline. This water infrastructure is used to pump, treat, convey, and deliver water to end users, and energy is consumed at each stage of the water supply process. Valley Water has an Energy Optimization Plan that guides its efforts to promote energy efficiency in all its operations by establishing Energy Optimization Measures (EOMs). EOMs call for replacement of outdated and inefficient equipment, retrofitting of facilities, and reliability improvements to ensure that Valley Water uses energy as efficiently as possible.

6.12.1 Raw Water Energy Intensity

Valley Water's energy consumption for each water supply process stage is shown in Table O-1C in Appendix A and described further below.

Extract and Divert

Valley Water has the option to use the Coyote Pumping Plant (CPP) to supplement water deliveries by pumping water from Anderson Reservoir to its treatment plants. When pumping from Anderson Reservoir, the energy is recorded by a separate utility meter rather than the CVP project-use meter. Since reservoir levels were lowered drastically in 2020 to prepare for the upcoming Anderson Dam Seismic Retrofit Project, the Extract and Divert energy consumption will likely be higher in 2020 than in future years.

Conveyance

Valley Water has operational control of the Pacheco Pumping Plant (PPP) and CPP. However, the two pumping plants are owned by the U.S. Bureau of Reclamation to provide CVP water deliveries to Santa Clara County. Since approximately 20.8% of the total water pumped at PPP is diverted to San Benito County, the energy data and water deliveries in Table O-1C excludes San Benito's share of the water deliveries and corresponding energy for the PPP. Valley Water used 26,369,000 Kilowatt-hour (kWH) in 2020 to convey water. In addition to Valley Water's share for pumping at the PPP, this total includes in-county pumping at CPP (approximately 405,000 kWH), and approximately 60,600 kWH related to SWP water deliveries. The energy intensity for Conveyance is 219 kWH/AF.

Treatment

Valley Water used 10,658,000 kWH to treat water in 2020 with an intensity of 104 kWH/AF. Energy to treat water is metered at Valley Water's three water treatment plants and includes metered utility data for offsite processes (i.e., drying beds) where applicable. Water pumped within each treatment plant to its treated water reservoir or to adjacent retailer reservoir is included in the Treatment process metered energy data and has been omitted from the Distribution process energy approximation. The Treatment water volume data is for water production which is slightly lower than the total raw water delivered to the water treatment plants (filter to waste, etc.). The estimated total treated water is 103,000 AF in 2020.

Distribution

Energy data to distribute treated water is not readily available. The distribution process energy data is approximated based on the total utility metered energy data for all Valley Water remote electrical services. To improve the approximation, easily identified raw water and safe/clean water sites (dams, South County turnouts, oxygenation trailers, small office buildings) were omitted from the provided Distribution energy data. However, some raw water sites are likely still included in the approximation. Valley Water also shares a treated water intertie facility with SFPUC that accounted for some supplemental treated water deliveries during planned treatment plant outages in 2020. The total energy consumed for Distribution is 215,600 kWH with an intensity of 2.1 kWH/AF.

Treated water is typically gravity-fed from the treatment plants to the retailers (although some power is used for controls, valving, monitoring, etc.). Some retailers have treated water reservoirs adjacent to Valley Water property. For the cases where water is pumped from within the treatment plant to a Valley Water reservoir (ready for distribution) or to an adjacent retailer reservoir, the energy was captured in the Treatment section of the table and not counted in Distribution to avoid double counting.

6.12.2 Recycled Water Energy Intensity

Valley Water manages the SVAWPC, the largest advanced water purification plant in Northern California, with a production capacity of 8 million gallons per day (8,960 AFY). The SVAWPC is creating a locally developed, new supply of water and produces about 4,200 AFY recycled water on average. The highly purified water is a drought-resilient source that can help ensure Silicon Valley has safe, sustainable water now and into the future. The energy consumption by the SVAWPC is 5,985,700 kWH (1230 kWH/AF) and provided in Table O-2 in Appendix A.

CHAPTER 7 – WATER SERVICE RELIABILITY AND DROUGHT RISK ASSESSMENT

Valley Water's diverse water supply portfolio can meet the County's future demands in normal, single dry, and five consecutive dry years, based on the projected demands and existing and planned supplies. The future supplies reflect the planned and phased implementation of WSMP projects over time. During a five-year drought under existing condition, the Drought Risk Analysis (DRA) indicates that Valley Water will be able to meet countywide demands with a combination of local and imported surface water, groundwater, supply from storage, and supplemental sources such as water transfers.

7.1 DEMAND AND SUPPLY ASSESSMENT

A reliable water demand forecast is critical in assessing Valley Water's water service reliability. In 2020, Valley Water developed a new statistically based Demand Model to meet its planning needs. The model provides forecasted Santa Clara County demands out to 2045. The Demand Model and forecasted demands are described in detail in chapter 4.

Valley Water maintains diverse water supply sources to meet countywide demands. Major sources of supply for Valley Water include natural groundwater recharge, local surface water, imported water from the State and Federal projects, and recycled and purified water. In addition, the SFPUC delivers water to eight retailers in the northern part of Santa Clara County. Chapter 6 describes in detail Valley Water's existing and projected sources of supplies and their constraints. Below is a summary of the constraints to Valley Water's water supply sources that are covered in Chapter 6.

- **Imported water –** Valley Water's SWP and CVP water supplies are subject to hydrologic variability and a number of additional constraints including regulatory constraints, seismic threats to the Delta levee system, sea level rise, climate change, and water quality variations.

- **Groundwater –** The groundwater supply is largely constrained by hydrologic variability and the estimated operational storage capacity within the subbasins. The inflows to the groundwater subbasins are constrained by Valley Water's managed aquifer recharge program and natural recharge. Groundwater quality can also be a constraint on groundwater supply.

- **Local surface water –** Local surface water supplies are vulnerable to hydrologic variability and constrained by environmental regulations and permit requirements. Climate change may cause decreases in the ability to utilize local surface water supplies.

- **Recycled and Purified Water –** The constraints on future recycled water deliveries include infrastructure capacity and the availability of recycled water. Some of the potential constraints on the development of potable reuse include reverse osmosis concentrate disposal, public acceptance, permitting, hydrogeologic conditions, wastewater availability from other agencies (Valley Water is not a wastewater agency), and cost.

7.2 SERVICE WATER RELIABILITY

Valley Water's water service reliability was assessed by comparing supplies and demands under three hydrologic conditions – an average year, a single dry year, and five consecutive dry years. The basis and data for these water years are provided in Table 7-1. Valley Water uses the WEAP model to evaluate water supply reliability under these conditions. Developed by the Stockholm Environment Institute, WEAP is a deterministic, integrated water resources management model that uses water demand and supply information and accounts for multiple and competing uses and priorities. Valley Water uses the WEAP model as a tool to support its long-term water supply planning. The WEAP model simulates Valley Water's water supply system comprised of facilities to recharge the county's groundwater subbasins, operation of reservoirs and creeks, treatment and distribution facilities, and raw water conveyance facilities. The model also accounts for non-Valley Water sources and distribution, such as supplies from the SFPUC, non-potable recycled water, and local water developed by other agencies, such as the San Jose Water Company. In essence, the model was formulated to simulate the management of the current and future water resources within the county.

Table 7-1. Basis of Water Year Data for Reliability Assessment

Year Type	Base Year	Volume Available (AF)[1]	% of Average Supply
Average Year	1922-2015	444,600	100%
Single Dry Year	1977	354,700	80%
Consecutive Dry Years 1st Year	1988	344,900	78%
Consecutive Dry Years 2nd Year	1989	369,500	83%
Consecutive Dry Years 3rd Year	1990	340,200	77%
Consecutive Dry Years 4th Year	1991	347,100	78%
Consecutive Dry Years 5th Year	1992	341,400	77%
[1]Volumes available are based on supplies anticipated to be available in 2025. Supply availability will vary with the demand year.			

Valley Water's WEAP water supply planning model operates on a monthly time-step that simulates the water supply and demand over 94 years, using the historic hydrologic sequence of 1922 through 2015. The model tracks water resources throughout the county and delivery of water to meet demands according to availability and priority. Average water supply reliability for average year, a single dry year, and five consecutive dry year sequence was based on the model results of the corresponding years in the 94-year model period. A complete summary of assumptions used in the modeling for this plan is provided in Appendix F.

7.2.1 Average Year Supply Reliability

Valley Water uses the average annual supply over the 94 modeled years to represent the average year condition. Under average conditions, Valley Water's projected water supplies exceed projected demand through 2045 in all demand years (Table 7-2). The increasing supplies reflect the planned and phased implementation of WSMP projects over time, which exceed Valley Water's level of service goal to be prudent given future uncertainties with demands and supplies.

For this analysis, imported water allocation is provided by DWR DCR 2019, which includes projected climate change impacts but does not include any projected changes to future regulations in the Delta watershed. The DWR DCR 2019 dataset was not available when Valley Water completed its WSMP 2040. Because the long-term operations of the SWP and CVP were undergoing re-consultation under the federal and State Endangered Species Acts (ESA and CESA) at the time Valley Water was conducting its WSMP 2040, in order to be conservative for planning purposes, the dataset used at that time included anticipated regulatory constraints with significantly reduced imported water supplies in comparison (25% less imported supplies). For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. Those regulatory constraints did not materialize in the 2019 and 2020 ESA and CESA permits, and thus the DWR DCR 2019 dataset does not include any projected changes to future regulations.

The analysis also assumes that groundwater storage can be drawn down to the severe stage of the Water Shortage Contingency Plan. This does not represent a sustainable long-term groundwater condition, but these supplies represent water that may be needed to get through a prolonged drought.

Table 7-2. Average Year Supplies and Demands

Water Supply	2025	2030	2035	2040	2045
Supply Total	446,000	518,000	640,000	643,000	650,000
Demand Total	330,000	325,000	330,000	335,000	345,000
Difference	116,000	193,000	310,000	308,000	305,000

NOTES: Supplies are rounded to the nearest 1,000 AF and demand numbers to the nearest 5,000 AF. Supplies shown are based on modeled estimates of available supplies. Actual availability during any given year depends on hydrology, groundwater recharge operations and conditions, and other factors. Groundwater storage shown assumes groundwater can be drawn down to the severe stage of the Water Shortage Contingency Plan. This does not represent a sustainable long-term groundwater condition, but these supplies represent water that may be needed to get through a prolonged drought. Imported water allocations are provided by DWR in their Delivery Capability Report (DCR) 2019, which does not include projected future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. However, through Valley Water's Monitoring and Assessment Program, Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future. Projects included in the supply projections include transfer Bethany pipeline (2025); Anderson dam seismic retrofit and potable reuse (2030); Guadalupe, Calero, and Almaden dam seismic retrofits and Pacheco Reservoir Expansion (2035); and an additional 35,000 AF of conservation (to reach Valley Water's goal of 109,000 AF by 2040 with a 1992 baseline).

7.2.2 Single Dry Year Supply Reliability

The single driest year in the 94 model years occurred in 1977, based on the historic hydrological record. Table 7-3 shows estimated supplies and demands for years 2025 through 2045. Supplies appear to be sufficient to meet demands during a single dry year through 2045. This assumes that reserves are at healthy levels at the beginning of the year and that the projects and programs identified in the WSMP are implemented. Supplies available for this single year drought represent water needed not only for that single drought year, but also water that may be needed for a prolonged drought. Valley Water would manage the supplies reported in the table assuming the drought may continue beyond a single year, and thus not all supplies are expected to be used by retailers during the single year drought.

Table 7-3. Single Dry Year Supplies and Demands (AF)

Water Supply	2025	2030	2035	2040	2045
Supply Total	355,000	373,000	497,000	503,000	505,000
Demand Total	330,000	325,000	330,000	335,000	345,000
Difference	25,000	48,000	167,000	168,000	160,000
NOTES: Supply numbers are rounded to the nearest 1,000 AF and demand numbers to the nearest 5,000 AF. The available groundwater is based on modeled estimates if the 1977 hydrology was repeated in the future. Supplies available for the single year drought represent water needed not only for that single drought year, but also water that may be needed for a prolonged drought. Valley Water would manage the supplies reported in the table assuming the drought may continue beyond a single year, and thus not all supplies are expected to be used by retailers during the single year drought. Imported water allocations are provided by DWR in their DCR 2019, which does not include projected future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. However, through Valley Water's Monitoring and Assessment Program, Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future. Projects included in the supply projections include transfer Bethany pipeline (2025); Anderson dam seismic retrofit and potable reuse (2030); Guadalupe, Calero, and Almaden dam seismic retrofits and Pacheco Reservoir Expansion (2035); and an additional 35,000 AF of conservation.					

7.2.3 Five Dry Year Supply Reliability

The greatest challenge to Valley Water's water supply reliability is multiple dry years, such as those that occurred in 1988 through 1992 and in 2012 through 2016. The five dry-year period used in this analysis is 1988 to 1992, which was an extended drought within historic record and WEAP modeling period. The most recent 2012-2016 drought is more severe, but imported water allocations are not available from DWR DCR 2019 for the analysis. Estimated supplies and demands for the period, under different demand years, are shown in Table 7-4. The analysis indicates that with existing and planned projects' supplies, Valley Water's diverse water supplies are sufficient to meet demands throughout the full five-year drought in all demand years without having to call for short-term water use reductions.

Valley Water's basic water supply strategy to compensate for supply variability is to store excess wet year supplies in the groundwater basin, local reservoirs, San Luis Reservoir, and/or Semitropic Groundwater Bank, and draw on these reserve supplies during dry years to help meet demands. These reserves, along with existing and planned future projects in the WSMP, help Valley Water meet demands during a prolonged drought. Valley Water's Board updated its long-term water supply reliability level of service goal in January 2019. The goal is to develop supplies to meet 100% of annual water demand during non-drought years and at least 80% of annual water demand in drought years. Future projects and programs recommended in the WSMP, including additional long-term water conservation savings, water reuse, recharge capacity, storm water capture and reuse, and banking and storage, were developed in accordance with this policy to minimize the need to call for water use reductions greater than 20%. The WSMP's recommended projects exceeded Valley Water's level of service goal to be prudent given future uncertainties with demands and supplies, but also because these projects were developed with a significant higher (approximately 14%) demand projection. As part of the on-going master planning process to address future uncertainties with demands, existing supplies, and proposed projects, Valley Water now conducts annual evaluation of WSMP projects through the MAP process to determine which projects should continue to be invested in to meet the level of service goal and potentially for other benefits such as operational flexibility, supply diversification, and resiliency to future uncertainties.

Table 7-4. Multiple Dry Years Supplies and Demands (AF)

		2025	2030	2035	2040	2045
First Year	Supply Totals	345,000	349,000	491,000	483,000	487,000
	Demand Totals	330,000	325,000	330,000	335,000	345,000
	Difference	15,000	24,000	161,000	148,000	142,000
Second Year	Supply Totals	370,000	376,000	477,000	482,000	501,000
	Demand Totals	330,000	325,000	330,000	335,000	345,000
	Difference	40,000	51,000	147,000	147,000	156,000
Third Year	Supply Totals	340,000	349,000	443,000	450,000	448,000
	Demand Totals	330,000	325,000	330,000	335,000	345,000
	Difference	10,000	24,000	113,000	115,000	103,000
Fourth Year	Supply Totals	347,000	341,000	416,000	421,000	429,000
	Demand Totals	330,000	325,000	330,000	335,000	345,000
	Difference	17,000	16,000	86,000	86,000	84,000
Fifth Year	Supply Totals	341,000	365,000	430,000	440,000	444,000
	Demand Totals	330,000	325,000	330,000	335,000	345,000
	Difference	11,000	40,000	100,000	105,000	99,000

NOTES: Supply numbers are rounded to the nearest 1,000 AF and demand numbers to the nearest 5,000 AF. Supplies shown are based on modeled estimates for hydrologic years 1988-1992. Imported water allocations are provided by DWR in their DCR 2019, which does not include projected future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. However, through Valley Water's Monitoring and Assessment Program, Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future. Projects included in the supply projections include transfer Bethany pipeline (2025); Anderson dam seismic retrofit and potable reuse (2030); Guadalupe, Calero, and Almaden dam seismic retrofits and Pacheco Reservoir Expansion (2035); and an additional 35,000 AF of conservation.

7.3 DROUGHT RISK ASSESSMENT

Droughts, particularly prolonged droughts, remain the greatest challenge to Valley Water's water supply reliability. Assessing and understanding water service reliability and risk during an extended drought are critically important to Valley Water's short-term and long-term water management decisions. This section describes Valley Water's Drought Risk Analysis (DRA) under a drought period lasting for the next five consecutive years.

7.3.1 Data and Method for Drought Risk Analysis

7.3.1.1 DRA Data

Drought Risk Analysis involves comparing total water supply sources available to projected water use for a drought period starting in 2021 that lasts five consecutive years. The expected gross water use (unconstrained demand) for the next five years is based on the interpolation between estimated 2021 water use and projected 2025 water use. A conservative estimate of approximately 320,000 AF was used for 2021, and the projected demand for 2025 is 330,000 AF.

For the sources of supply, the DRA considers all of Valley Water's water supply sources, including imported water (SWP and CVP contract water deliveries, banked supplies in Semitropic, sales, transfers, and carryover in San Luis Reservoir, and SFPUC deliveries), local surface water storage, recycled water, and local groundwater. The supply data for 2021 are from Valley Water's annual water supply and demand assessment, which plans water supply operations for the upcoming year and considers a dry water year scenario. For 2022-2025, WEAP model-estimated supplies under the 1989-1992 hydrologic conditions were used.

7.3.1.2 Basis for Water Shortage Condition

Valley Water's supply comes from a variety of sources. As such, many factors and events affect water supply availability in any given year. Valley Water manages its supplies each year to maintain sustainable groundwater conditions in local sub-basins. If analysis indicates that supplies available that year may not be able to maintain Valley Water's groundwater in the "normal stage," then Valley Water may augment supplies with imported water supply purchases and/or call on the community to make water use reductions. Valley Water has determined that projected end-of-year groundwater storage serves as a useful indicator to determine whether there may be a need to call for a water shortage contingency plan action. Currently, Valley Water uses five stages to categorize its water supply shortage, based on the end-of-year groundwater storage projections for the Santa Clara Subbasin, as summarized in Table 7-5. A crosswalk between Valley Water's shortage stages and DWR's standard stages is provided in Chapter 8.

Table 7-5. Valley Water's Water Shortage Stages and Water Use Reductions

Stage	Stage Title	Projected End-of-Year Groundwater Storage	Recommended Short-Term Water Use Reduction
Stage 1	Normal	Above 300,000 AF	None
Stage 2	Alert	250,000 – 300,000 AF	0 – 10%
Stage 3	Severe	200,000 – 250,000 AF	10 – 20%
Stage 4	Critical	150,000 – 200,000 AF	20 – 40%
Stage 5	Emergency	Below 150,000 AF	>40%

7.3.1.3 DRA Method

The DRA follows Valley Water's annual water supply and demand assessment procedure, which is a water balance approach to estimate supply available based on the previous year end-of-year groundwater storage, carryover supplies stored in San Luis reservoir, storage in local reservoir, non-potable recycled water production, and expected SWP and CVP contract allocations. For 2021, estimated supplies were developed using a combination of Valley Water's annual operations model and groundwater model. Estimated supplies were developed assuming critically dry local conditions (90% exceedance) and CVP and SWP allocation estimates from the USBR and DWR, respectively. For 2022-2025, WEAP model output from model years 1989-1992 was used to estimate supplies that may be available. Demands for the next five years is based on the interpolation between estimated 2021 water use and projected 2025 water use. A conservative estimate of approximately 320,000 AF was used for 2021, and the projected demand for 2025 is 330,000 AF.

7.3.2 Total Water Supply and Demand Comparison

The DRA indicates that Valley Water will be able to meet countywide demands with a combination of local and imported surface water, groundwater, banked supplies in Semitropic storage, and imported water transfer purchases (Table 7-6). In anticipating a potential drought and because Valley Water is required to maintain Anderson Reservoir at deadpool, Valley Water expects to secure at least 6,500 AF of water transfers for 2021. In case it is entering a drought, Valley Water wants to maintain local groundwater as high as possible, especially since Anderson Reservoir cannot be used. In the subsequent dry years, Valley Water will continue to use supplies stored in the local groundwater, local reservoirs, and Semitropic groundwater bank.

Table 7-6. Five-Year Drought Risk Assessment

2021	Total
Total Water Use	320,000
Total Supplies	343,500
Surplus/Shortfall w/o WSCP Action	23,500
Planned WSCP Actions (use reduction and supply augmentation)	
WSCP - supply augmentation benefit	6500
WSCP - use reduction savings benefit	0
Revised Surplus/(shortfall)	30,000
Resulting % Use Reduction from WSCP action	0%
2022	
Total Water Use	322,000
Total Supplies	362,000
Surplus/Shortfall w/o WSCP Action	40,000
Planned WSCP Actions (use reduction and supply augmentation)	
WSCP - supply augmentation benefit	0
WSCP - use reduction savings benefit	0
Revised Surplus/(shortfall)	40,000
Resulting % Use Reduction from WSCP action	0%
2023	
Total Water Use	325,000
Total Supplies	335,000
Surplus/Shortfall w/o WSCP Action	10,000

Planned WSCP Actions (use reduction and supply augmentation)	
WSCP - supply augmentation benefit	0
WSCP - use reduction savings benefit	0
Revised Surplus/(shortfall)	10,000
Resulting % Use Reduction from WSCP action	0%
2024	
Total Water Use	327,000
Total Supplies	344,000
Surplus/Shortfall w/o WSCP Action	17,000
Planned WSCP Actions (use reduction and supply augmentation)	
WSCP - supply augmentation benefit	0
WSCP - use reduction savings benefit	0
Revised Surplus/(shortfall)	17,000
Resulting % Use Reduction from WSCP action	0%
2025	
Total Water Use	330,000
Total Supplies	341,000
Surplus/Shortfall w/o WSCP Action	11,000
Planned WSCP Actions (use reduction and supply augmentation)	
WSCP - supply augmentation benefit	0
WSCP - use reduction savings benefit	0
Revised Surplus/(shortfall)	11,000
Resulting % Use Reduction from WSCP action	0%
NOTE: WEAP model output for hydrologic years 1989-1992 was used to represent years 2 through 5 of the drought. Imported water allocations are provided by DWR in their DCR 2019, which does not include projected future regulations nor the hydrologic sequence for the most recent 2012-2016 drought. For comparison, the lowest total annual imported delivery during the 1987-1992 drought in the DCR 2019 dataset is 83,200AF, while the actual lowest annual imported delivery during the 2012-2016 drought was 60,320 AF. However, through Valley Water's Monitoring and Assessment Program, Valley Water is conservatively planning for investments by considering severe droughts, such as the 2012-2016 drought, will occur in the future.	

CHAPTER 8 – WATER SHORTAGE CONTINGENCY PLAN

This chapter describes the development, actions, and implementation of Valley Water's Water Shortage Contingency Plan (WSCP). The WSCP defines specific triggers, based on groundwater storage levels, when Valley Water will call on the public to reduce water demand, and what actions it will take in these circumstances. Water use reductions may be needed in response to drought conditions or an emergency that affects water supply. The WSCP also summarizes planning for natural disaster, drought-related revenue impacts, and Valley Water's legal authority to respond to water shortages. Where applicable, Valley Water's actions during the recent five-year drought are summarized for reference.

The WSCP was developed consistent with Valley Water's water shortage management objectives:

- To minimize the economic, social, and environmental hardship to the community caused by water shortages.
- To establish water use reduction targets and work closely with retailers and cities in developing efficient and effective demand reduction measures during water shortages that concentrate on eliminating non-essential uses first.
- To maintain and safeguard essential water supplies for public health and safety needs, including during acute catastrophic events.

8.1 WATER SUPPLY RELIABILITY ANALYSIS

Valley Water's basic water supply strategy to compensate for supply variability is to store excess wet year supplies in the groundwater subbasins, local reservoirs, San Luis Reservoir, and Semitropic Groundwater Bank, then to draw on these stored supplies during dry years to help meet demands. Based on projected demands, and Valley Water's existing and planned sources of supply, Valley Water will be able to meet countywide demands through 2045 under normal, a single dry, and five consecutive dry year conditions.

The DRA indicates that if a five-year drought were to occur under existing conditions, Valley Water will need to employ a range of response actions, including using supplies stored in the local groundwater, local reservoirs, and Semitropic groundwater bank, as well as augmenting supplies with supplemental sources such as water transfers and exchanges, to meet potential shortage. See Chapter 7 of the Urban Water Management Plan for details of Valley Water's supplies and demands in various years types.

8.2 ANNUAL WATER SUPPLY AND DEMAND ASSESSMENT PROCEDURES

Valley Water projects available water supplies on an annual basis. Water supply operations planning considers all of Valley Water's water supply system and sources, including imported water (deliveries, banking, sales, transfers, exchanges, carry-over), local surface water reservoirs, local water supply diversions, groundwater recharge systems, groundwater subbasins, and water treatment plants. A summary of available water resources is provided in Table 8-1. Because Valley Water's supply comes from a variety of sources, many factors and events affect water supply availability in any given year. Through its long- term practice, Valley Water has determined that projected end-of-year groundwater storage serves as the best indicator of potential water shortages and early warning signal, and therefore uses it to determine a potential water supply shortage.

CHAPTER 8 – WATER SHORTAGE CONTINGENCY PLAN

Table 8-1. Water Supply Sources

Source	Description
Central Valley Project (CVP)	Contract for 152,500 acre-feet per year (AFY). Actual allocations are based on availability of water supplies after meeting regulations to protect the environment, water quality, and other factors.
State Water Project (SWP)	Contract for 100,000 AFY. Actual allocations are based on availability of water supplies after meeting regulations to protect the environment, water quality, and other factors.
Natural Groundwater Recharge	Approximately 61,000 AF of precipitation and other natural flows are directly recharged to the groundwater basins in an average year. This amount varies with hydrologic conditions. Natural recharge differs from Valley Water's managed recharge program where precipitation is captured in reservoirs and purposely released for recharge in ponds and along managed creeks.
Local Surface Water	Valley Water has water rights to capture and use 223,000 AFY at the reservoirs and diversion structures. Currently, 53,000 AFY are used for water supply on average. This is expected to increase to an average of 83,000 AFY by 2040. The total volume used depends on rainfall and demands.
Recycled Water	Recycled water is produced by the County's four wastewater treatment plants for various non-drinking water (non-potable) purposes. In addition, Valley Water provides advanced treated purified water to South Bay Water Recycling to improve the quality of the non-potable supply. Recycled water use is projected to increase from approximately 17,000 AFY in 2025 to approximately 28,000 AFY by 2045.
San Francisco Public Utilities Commission (SFPUC)	SFPUC's Regional Water System provides water to some cities in the northern part of the County. On average about 55,000 AF is delivered to the County each year.
Semitropic Groundwater Bank	Valley Water has invested in 350,000 AF of out-of-county water storage capacity. Water is delivered to the groundwater bank when surplus supplies are available and withdrawn when supplies are limited. Valley Water's contract allows the withdraw between approximately 31,000 to 78,000 AF of banked water per year, depending on SWP allocation (larger withdrawals are permitted during larger allocation years).
Local Reservoirs	Valley Water operates 10 reservoirs in the County with a total capacity of approximately 166,000 AF that capture runoff from the watershed for release through the year to recharge the groundwater basins. Water captured at Anderson, Coyote, Almaden, and Calero Reservoirs can also be sent to the water treatment plants. Anderson Dam is currently undergoing a seismic retrofit and is not able to provide water storage. Valley Water expects the retrofit project to be completed in 2030.
Groundwater Storage	As the County's Groundwater Sustainability Agency, Valley Water manages the Santa Clara and Llagas subbasins and strives to maintain adequate storage in wet and average years to ensure water supply reliability during dry periods or shortages. The estimated operational storage capacity of the groundwater subbasins is up to 548,000 AF.

CHAPTER 8 – WATER SHORTAGE CONTINGENCY PLAN

Annual water supply operations planning begins each September for the upcoming year and considers water year scenarios that span from wet to very dry. This annual water supply planning considers current groundwater storage, treated water contracts, local water rights and storage, environmental restrictions, source water quality, planned facility maintenance, imported water carryover, imported water contract terms, stored water in carryover and the Semitropic Bank, and potential water transfers. Operations planning serves as the basis for daily operational decisions consistent with the annual strategy to manage water supplies and reserves. The process for annual water supply operations planning is depicted in Figure 8-1.

Figure 8-1. Annual Water Operations Planning

Water supply operations planning is dynamic, and rainfall data, imported water allocations, water supply projections, availability of supplemental supplies, and facility capacities are updated at least monthly to reflect current conditions. The projection of water supplies through the end of the year is based on assumed dry conditions (90% exceedance), median conditions (50% exceedance), and in some cases, critically dry conditions (99% exceedance). As assumptions and projections are updated through the year, Valley Water continues to update its end-of-year groundwater storage projections.

The state of the groundwater basins is reported monthly through a Groundwater Conditions Report (https://www.valleywater.org/your-water/where-your-water-comes-from/groundwater/groundwater-monitoring) and Water Tracker (https://www.valleywater.org/your-water/water-supply-planning/monthly-water-tracker). The Groundwater Conditions Report and Water Tracker contain a description and quantification of available water supplies including local reservoirs, imported water, treated water, recycled water, conserved water, and groundwater data, such as recent managed recharge, pumping, and storage trends. The annual water supply operations planning process together with the monthly reports are how Valley Water tracks and reports its annual water supply, demands, and overall water supply reliability. Projected end-of-year groundwater supply is the indicator for determining a potential water supply shortage and is based on current hydrology and demands, infrastructure capabilities and constraints, and assumed dry and median hydrology scenarios through the rest of the year.

Staff provides the Valley Water Board of Directors (Board) updates on available water supplies, groundwater storage projections, and water demand projections as needed throughout the year and at least once a year, typically in April. Starting in 2022, this update will take the form of an Annual Water Supply and Demand Assessment (Annual Assessment). The Annual Assessment will provide an update on projected demands through the current year, a description and quantification of available water supplies considering dry hydrological and regulatory conditions through the remainder of the current year, and existing infrastructure capabilities and plausible constraints. The Assessment will also include a forecast of water supplies conditions assuming the following year is dry. The forecast for the following year is provided as an informational piece and to provide context of Valley Water's water supply reliability.

The Annual Assessment will include a recommendation if WSCP actions are needed. The recommendation on WSCP actions will be based on the end-of-current-year groundwater storage projection and the water storage stages presented in Section 8.3. The Annual Assessment will be brought to the Board for their information as part of the regular water supply update. If WSCP actions are recommended based on the WSCP, the Board will be asked to decide on taking water shortage actions. The Annual Assessment will be provided to the state annually by July 1. The steps and timeline of the WSCP Annual Assessment Procedure are summarized in Table 8-2.

Table 8-2. WCSC Annual Assessment Procedure

Action	Description	Lead	Timeline
Start water supply operations planning	The water supply operations planning process sets targets for use of available water supplies for the coming year.	Raw Water Operations Unit	September
Initial end-of-Year Groundwater Storage Projection	Modeling / calculation of initial projected groundwater storage for coming year.	Groundwater Management Unit	September
Updates to water supply operations planning	Water supply operations planning projections are updated at least monthly to reflect actual conditions, operations, and updated groundwater storage projections. Projections are made for both dry and median hydrology for the remainder of the year.	Raw Water Operations Unit Groundwater Management Unit	Monthly
Preparation of WSCP Annual Assessment	Based on updates to water supply operations planning and groundwater storage projections, the WSCP Annual Assessment is prepared to document available water supplies, projected demands, and projected end-of-current-year groundwater storage. Recommendations for WSCP actions are based on assumed dry conditions the	Water Supply Planning and Conservation Unit	March

Action	Description	Lead	Timeline
	remainder of the year. The Annual Assessment will also provide a forecast of water supply reliability should the following year be dry.		
Board review of WSCP Annual Assessment / Determination of WSCP shortage actions	The Assessment is presented to the Board for their information and discussion. If shortage actions are recommended by the WSCP, the Board will be asked to take water shortage actions.	Board of Directors	April (Calls for WSCP shortage actions can be brought to the Board at any time during extended droughts and emergencies.)
Finalization of WSCP Annual Assessment	The Annual Assessment is finalized based on feedback provided by the Board.	Water Supply Planning and Conservation Unit Chief Operating Officer	May
Submittal of WSCP Annual Assessment	The Annual Assessment is submitted to DWR.	Water Supply Planning and Conservation Unit	June

8.3 SIX STANDARD WATER SHORTAGE STAGES

Valley Water uses five stages to categorize its water supply shortage. The stages are based on projected countywide end-of-year groundwater storage and include a normal stage and four progressive levels of water shortage (Table 8-3), as described below.

Table 8-3. Valley Water's Shortage Stages and Recommended Water Use Reductions

Stage	Stage Title	Projected End-of-Year Groundwater Storage	Recommended Short-Term Water Use Reduction
Stage 1	Normal	Above 300,000 AF	None
Stage 2	Alert	250,000 – 300,000 AF	0 – 10%
Stage 3	Severe	200,000 – 250,000 AF	10 – 20%
Stage 4	Critical	150,000 – 200,000 AF	20 – 40%
Stage 5	Emergency	Below 150,000 AF	>40%

- Stage 1 is normal water supply availability when groundwater storage is substantially full and no water shortage actions are necessary.
- Stage 2 is the alert stage that is meant to warn the public that current water use is tapping groundwater reserves. This stage is triggered when groundwater storage is projected to drop below 300,000 AF and the Board may request the public and retailers reduce water use by up to 10%.

- Stage 3 is the severe stage. Shortage conditions are worsening, requiring close coordination with retailers and cities to enact ordinances and water use restrictions. This stage is triggered when groundwater storage falls below 250,000 AF. The Board may pass a resolution that requests the public and retailers to reduce water use by 20%.

- Stage 4 represents critical conditions. This is typically the most severe stage in a multi-year drought. This stage is triggered when groundwater storage is projected to fall below 200,000 AF. The Board may increase the demand reduction request up to 40%.

- Stage 5 is for emergency situations. It is meant to address an immediate crisis such as a major infrastructure failure when water supply may only be available to meet health and safety needs. Stage 5 can also be triggered in a deep drought when groundwater levels are projected to fall below 150,000 AF. Water reduction may need to exceed 40%.

Per the UWMP guidebook, Table 8-4 shows a crosswalk between Valley Water's five (5) stages and the standard six (6) stages as defined by the DWR.

Table 8-4. Crosswalk Between Valley Water's Stages and Standard Stages

Stage	Title	Projected Countywide End of Year Groundwater Storage (AF)	Suggested short-term reduction in water use	Stage	Standard water shortage levels
1	**Normal**	>300,000	None		
2	**Alert**	300,000 - 250,000	0-10%	1	Up to 10%
3	**Severe**	250,000 - 200,000	10-20%	2	10 to 20%
4	**Critical**	200,000 - 150,000	20-40%	3	20 to 30%
				4	30 to 40%
5	**Emergency**	<150,000	Over 40%	5	40 to 50%
				6	> 50%

8.4 SHORTAGE RESPONSE ACTIONS

Water supply shortages can occur for a variety of reasons including droughts; loss in ability to capture, divert, store, or utilize local supplies; and/or facility outages. As a wholesale agency, Valley Water does not have direct authority over retail rates and generally does not employ staff to enforce water restrictions. Therefore, Valley Water's water shortage response actions are focused mainly on public education and coordination with municipalities and retailers in the County. During droughts or shortages, Valley Water considers all available tools, including balancing demands for treatment plants and recharge facilities, incentives or requests for retailers to use either groundwater or treated water, and community outreach to maximize the use of available supplies. The collective response actions between Valley Water, residents, businesses, large landscapers, agriculture, municipalities, and retailers preceding and during a water supply shortage are described below (Table 8-5).

Table 8-5. Water Shortage Response Actions

Stage	Requested Short-Term Water Use Reduction	Actions
Stage 1 **Normal**	None	Valley Water continues ongoing outreach strategies aimed toward achieving long-term water conservation targets. Messages in this stage focus on services and rebate programs Valley Water provides to facilitate water use efficiency for residents, agriculture, and business. While other stages are more urgent, successful outcomes in Stage 1 are vital to long-term water supply reliability.
Stage 2 **Alert**	0 – 10%	This stage is meant to warn water users that current water use is tapping groundwater reserves. Work begins to coordinate ordinances with the County, cities and retailers to prepare for Stage 3. Additional communication tools are employed to augment Stage 1 efforts, promote immediate behavioral changes, and set the tone for the onset of shortages. Specific implementation plans are developed in preparation of a drought deepening such as identifying supplemental funding to augment budgeted efforts and initiation of discussions with local, state, and federal agencies to call on previously negotiated options, transfers, and exchanges.
Stage 3 **Severe**	10 – 20%	Shortage conditions are worsening, requiring close coordination with the County, cities, retailers, large landscapers, and agricultural users to implement ordinances and water use restrictions. Significant behavioral change is requested of water users. The intensity of communication efforts increases with the severity of the shortage. Messages are modified to reflect more dire circumstances. Water supplies are augmented through the implementation of options, transfers, exchanges, and withdrawals from groundwater banks.
Stage 4 **Critical**	20 – 40%	This is generally the most severe stage in a multi-year drought. Stage 3 activities are expanded, and Valley Water will encourage the County, cities, and retailers to increase enforcement of their water shortage contingency plans, which could include fines for repeated violations; and all water users to significantly reduce water use.
Stage 5 **Emergency**	40 to 50%	Stage 5 is meant to address an immediate crisis such as a major infrastructure failure but may also be needed in exceptional multi-year drought. Water supply may only be available to meet health and safety needs. Valley Water will encourage all water users to significantly reduce water use, activates its Emergency Operations Center, coordinates closely with municipalities and retailers, and provides daily updates on conditions.

8.4.1 Water Conservation

When the Board calls for short-term water use reductions, cities and water retailers consider implementing their WSCP actions to achieve the necessary water use reductions. Actions to achieve the desired shortage response may be different for each city/water retailer depending on their service area composition (commercial, industrial, residential) and source of water supplies. However, effort is made to make actions common to as many cities/water retailers as possible to provide for more consistent implementation and messaging.

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Reducing water consumption during a water shortage is generally achieved through increased education leading to behavioral changes (e.g., shutting off the water while brushing one's teeth) and water use restrictions (e.g., yard irrigation limited to specific days of the week). These water savings are considered short-term water use reductions and are distinct from long-term and on-going conservation programs described in Chapter 9.

The response to the 2012 to 2016 drought illustrates how Valley Water, cities, the County, and retailers coordinate to reduce water use during water shortages. On February 25, 2014, the Board approved a resolution setting a countywide water use reduction target equal to 20% of 2013 water use through December 31, 2014, and recommended that retail water agencies, cities, and the County implement mandatory measures as needed to achieve the 20% water use reduction target. The water use reduction achieved for calendar year 2014 was 13% compared to 2013 water use.

On March 24, 2015, as the drought continued, the Board called for 30% water use reductions and recommended that retail water agencies, cities and the County implement mandatory measures as needed to accomplish that target, including a two day per week outdoor irrigation schedule. To assist the retailers, cities, and the County achieve the water use reduction targets, Valley Water:

- Increased rebates for water-efficient landscape conversions, irrigation hardware upgrades, graywater laundry to landscape systems, and certain commercial fixtures.
- Created a Water Waste Reporting and Inspection Program.
- Increased staffing to support a water conservation call center.
- Developed several multimedia water conservation outreach campaigns, including “Brown is the New Green” and “Fight the Drought, Inside and Out”.
- Hosted dozens of panels, forums, and presentations.
- Encouraged participation in conservation programs through direct mail letters.
- Reduced the amount of treated water that it supplied to retailers.

Valley Water and retailers coordinated very closely during the drought, with regular meetings and information exchange on water supply conditions, operations, and actions/messaging to achieve water use reduction. All retailers took actions to implement water use reduction requirements and many adopted a coordinated maximum two day per week watering schedule. Together these actions achieved a 28% demand reduction in 2015 over 2013 levels. In 2015, Valley Water held two summits, one with retailers and another with elected officials, to facilitate increased water use reductions and increase coordination to meet the 30% reduction target. A common theme of the summits was that messaging and policy development should be consistent and coordinated throughout the County to reduce confusion among residents, increase ease of implementation, and make compliance and enforcement easier.

The WSCP was developed in accordance with 2020 Urban Water Management Plan guidebook. It provides general guidance on recommended actions to address water supply shortages. Valley Water continuously seeks to improve its water shortage planning efforts, which may be reflected in future refinements to this WSCP. Under extraordinary circumstances and/or rapidly changing water supply conditions, Valley Water may need to undertake water conservation measures that are stricter than those set forth in this WSCP.

8.4.2 Water Supply Augmentation

Valley Water works to maintain high groundwater storage in normal and wet years through a comprehensive managed recharge program and by providing treated water in lieu of groundwater pumping. Excess supplies are stored in the Semitropic Groundwater Bank, and Valley Water can carryover imported supplies in some years in San Luis Reservoir. During a dry year, Valley Water can use these stored supplies without having to call on the public to reduce demands. During an extended drought, Valley Water would need to pursue additional water shortage actions. Valley Water uses a combination of options to bring in additional water supplies to support local demands, including:

- Recovery and import of Valley Water's supplies stored in groundwater banking and exchange programs.
- Use of existing multi-year agreements between Valley Water and other water agencies that provide options to call on pre-negotiated transfer/exchange water.
- Collaboration with water agencies that have available resources to develop and implement agreements for the transfer/exchange of water to Valley Water.
- Participation in pooled water transfer programs with other SWP and CVP contractors.

The quantities of water available through these options are variable and depend on hydrology, pumping capacity, environmental restrictions, and demands from other agencies. These supplemental supplies help Valley Water mitigate the impact of a drought. For example, in 2015 Valley Water secured approximately 69,000 acre-feet in supplemental supplies through transfers, exchanges, public health and safety allocations, and Semitropic banking withdrawals, which helped mitigate the impact of the severely low imported water allocation that year.

8.4.3 Catastrophic Interruption Planning

This section describes planning that Valley Water has undertaken to prepare for catastrophic interruption of water supplies during a disaster.

Infrastructure Reliability Plan

Valley Water completed its first Infrastructure Reliability Plan (IRP) in 2005 and updated it in 2016. The IRP analyzes several outage scenarios for Valley Water's system, including an earthquake, extreme storm, Delta outage, and power outage. Valley Water and retailers agreed on a reliability target during an emergency that Valley Water should be able to restore treated water deliveries to meet the equivalent of a winter month's demand (i.e., February) within 30 days after a major disaster event. Modeling and analyses estimated service restoration time of Valley Water's existing system for minimum winter demands in each of the outage scenarios.

The worst-case outage scenario was a magnitude 7.9 earthquake on the San Andreas fault, which would result in an estimated 30-day outage time before Valley Water can provide minimum treated water demands to retailers. In the Delta outage scenario, modeling demonstrated Valley Water can continue limited service (at an assumed 20% demand reduction) for a 24-month period with no imported water supplies if it occurred in a normal hydrologic year and started with normal groundwater supplies. In a regional power outage, Valley Water can operate facilities on backup fuel storage for an estimated 3 to 10 days, or longer given regular external fuel deliveries.

The 2016 IRP recommends efficient and targeted opportunities to improve system reliability and performance by either shortening Valley Water system outage time following an event or strengthening retailer capability to withstand Valley Water system outages. Important concepts that were incorporated into the identification of project opportunities and the analysis methodology are:

1. *Incorporate recent operational knowledge*: Planned and unplanned maintenance outages of Valley Water pipelines and treatment plants have allowed retailers to learn how to operate their systems without Valley Water treated water supplies. Retailers have operated with Valley Water treated water supply interruptions for up to eight weeks in some cases.

2. *Account for backup supply redundancy*: Most retailer service areas have adequate groundwater pumping capacity to serve as a backup to treated water deliveries and may not require large investments in additional reliability.

3. *Consider raw water and treated water system interdependencies*: Strengthening Valley Water's treated water pipeline system alone may not dramatically improve reliability in scenarios where raw water pipelines fail. The opposite also applies, as strengthening Valley Water's raw water pipeline system alone may not dramatically improve reliability in cases where treated water pipelines fail. Strengthening key portions of both the raw and treated water pipeline systems is needed to provide improved reliability. These improvements are being planned and recommended through the development of a distribution system master plan.

4. *Leverage existing investments*: Where possible and beneficial, leveraging existing assets is preferred, as Valley Water, retailers, and SFPUC have made significant investments in increasing system reliability and operational flexibility since the 2005 IRP.

5. *Favor frequently used assets*: Assets, particularly groundwater wells, which can be used more frequently to enhance daily operations or periodic maintenance operations, are preferred over assets that would be designated as standby for infrequent use only during major emergencies.

6. *Address specific vulnerable areas*: There are specific retailer service areas that are more vulnerable to outages of Valley Water treated water or managed recharge. Focusing on localized solutions to improve reliability in these specific areas may be more effective, with lower costs, than major infrastructure improvements.

Ultimately, Valley Water and retailers determined that targeting specific vulnerable areas for improvement will effectively address identified reliability needs. A total of 20 projects are identified in the 2016 IRP to improve reliability in these specific areas. Some projects were identified for retailer implementation, some for Valley Water implementation, and others for joint implementation. Valley Water has been working to complete the identified projects since 2016.

Local Hazard Mitigation Plan

Valley Water's 2017 Local Hazard Mitigation Plan (2017 LHMP) identifies capabilities, resources, information, and strategies for building resilience and reducing physical and social vulnerabilities to disasters. It also coordinates mitigation actions, providing essential guidance for Valley Water to reduce its vulnerability to disasters. Valley Water developed the 2017 LHMP to be consistent with current legislation, conditions, and best available science. This ensures that hazards are accurately profiled;

policies are consistent with current Valley Water standards and relevant federal, state, or regional regulations; and Valley Water has an updated LHMP consistent with Federal Emergency Management Agency (FEMA) Emergency Response Plan (ERP) requirements. The 2017 LHMP also includes strategies to reduce vulnerability to disaster through education and outreach programs, foster the development of partnerships, and implement risk reduction activities.

Emergency Operations Center

Valley Water's Emergency Services and Security Unit (ESSU) coordinates emergency response and recovery for Valley Water. During any emergency, Valley Water continues the primary missions of providing clean, safe water and flood protection to the people of Santa Clara County. ESSU maintains a full-time professional emergency management staff trained and equipped to respond quickly to support Valley Water's Emergency Operations Center (EOC) and field responders. The ESSU ensures that critical services are maintained, and emergency response is centralized.

The EOC is connected to other agencies and jurisdictions by an array of telecommunications, two-way radio, satellite telephone, and wireless messaging systems. In addition, two response vehicles with many of the same communications capabilities of the EOC enable staff to establish mobile emergency command posts just about anywhere field operations may require. Office of Emergency Services (OES) maintains communications with local, state, and national emergency management organizations and allied disaster preparedness and response agencies.

Milpitas Intertie

During an emergency, in addition to retailers relying on groundwater and their own supplies, Valley Water has a 40-million gallon per day intertie with the SFPUC located in the City of Milpitas, which allows the SFPUC and the East Pipeline systems to exchange water during emergencies and planned maintenance.

8.4.4 Delta-Conveyed Supply Interruption

A strategy was developed by DWR, the Army Corps of Engineers (Corps), Bureau of Reclamation, California Office of Emergency Services (Cal OES), and the State Water Contractors to provide water supply protections that would enable resumption of at least partial deliveries from the Delta in less than six months in the event of an outage.

Valley Water analyzed the impacts of a six-month Delta outage to determine the effect on service. The analysis assumed that all local infrastructure remains intact, as an earthquake or flood in the Delta is unlikely to badly damage local infrastructure. The analysis also assumed normal hydrologic conditions and starting storage conditions, rather than stacking disaster upon disaster (i.e., earthquake plus drought, etc.), access to SFPUC supplies, and implementation of water use reductions of 20%. The impacts of such an outage are largely operational as retailers would be required to use groundwater instead of their usual treated water supplies and Valley Water would actively manage the groundwater recharge program to meet countywide needs. Even with increased pumping, groundwater storage is estimated to remain in the normal (Stage 1) range. Thus, the impacts of a six-month Delta outage are manageable assuming a normal starting position. Valley Water would potentially need to call for more aggressive water use reductions if a Delta outage were to occur during or immediately following a drought.

The Delta Flood Emergency Management Plan (DWR, 2018) provides strategies for responses to Delta levee failures, including earthquake-induced numerous levee failures during dry conditions with multiple flooded islands and extensive saltwater intrusion, resulting in curtailment of export operations. Under these severe conditions, an emergency freshwater pathway would be established from the central Delta along Middle River and Victoria Canal to the export pumps in the south Delta. The plan includes the pre-positioning of emergency construction materials at stockpile and warehouse sites in the Delta, and development of tactical modeling tools (DWR Emergency Response Tool) to predict levee repair logistics, timelines of levee repair, and suitable water quality to restore exports. Using pre-positioned materials, multiple earthquake-generated levee breaches and levee slumping along the freshwater pathway can be repaired in less than six months. Significant improvements to the central and south Delta levee systems along the emergency freshwater pathway began in 2010 and are continuing. Continued efforts under analysis strive to mitigate not only flood and earthquake risk but also meet future sea-level rise risk.

8.5 COMMUNICATION PROTOCOLS

When the Board calls for short-term water use reduction actions under the WSCP, the cities and water retailers consider implementation of their WSCP actions to achieve the necessary shortage response. Clear, consistent, and effective communication with the cities, retailers, and the public is essential to achieving desired results. Internally, Valley Water works with its Office of Government Relations to coordinate with external stakeholders.

Communication strategies include:

- Clear explanations to the public of the WSCP stages and triggers.
- Early and continuous coordination with retailers regarding response actions, use of groundwater versus treated water, and treated water quality concerns.
- Hosting drought summits with retailers and elected officials to develop coordinated response actions such as drought ordinances and enforcement of a day per week irrigation schedules.
- Providing a straightforward methodology for water use reporting by retailers.
- Allowing treated water contracts with retailers to be adjusted to avoid penalizing retailers for not taking contract amounts and support the use of groundwater and SFPUC for blending, if necessary.
- Working with the agricultural community on water conservation methods.

Valley Water provides communications to stakeholders with various frequencies depending on the WSCP stages. Valley Water's communication protocol are summarized in Table 8-6.

Table 8-6. Valley Water's Communication Protocol Under the WSCP Stages

Stage	Short-Term Water Use Reduction	Communication Strategies
Stage 1 Normal	None	• Ongoing public outreach aimed toward achieving long-term water conservation targets. • Quarterly meetings with retailers to discuss water supply issues.

Stage	Short-Term Water Use Reduction	Communication Strategies
		• Monthly Groundwater Conditions Report and Water Tracker posted on website. • Annual Assessment to Board.
Stage 2 Alert	0 – 10%	• All Stage 1 communications. • Coordinate potential ordinances with cities, County, and retailers to prepare for the potential of a worsening drought. • Additional public outreach to augment Stage 1 efforts, promote immediate behavioral changes, and set the tone for the onset of shortages. • Updates to the Board, Committees, and retailers on preparation for drought messaging.
Stage 3 Severe	10 – 20%	• All communications above. • Monthly coordination with cities, County, and retailers to discuss conditions and to enact and review the progress of ordinances and water use restrictions. • Extensive public outreach to specific user groups such as residential, businesses, large landscapers, and agriculture to support behavioral change of water users. • Work with the press to disseminate drought messages.
Stage 4 Critical	20 – 40%	• All communications above. • Outreach expanded further to support significant behavioral change of water users. • More frequent meetings with cities, County, and retailers to discuss conditions and update enforcement of ordinances and water use restrictions. • Workshops and summits with retailers, elected officials, and other water user groups. • Monthly updates to the Board on water supplies and demands. • Coordination with local, state, and federal emergency agencies.
Stage 5 Emergency	40 – 50%	• All communications above. • Activation of the Emergency Operations Center. • Weekly meetings with cities and retailers on shortage conditions and response. • Expanded coordination with local, state, and federal emergency agencies. • Weekly updates to the press and public on conditions.

8.6 COMPLIANCE AND ENFORCEMENT

In general, most water shortage compliance and enforcement actions occur at the retailer level and with city or County enforcement staff or law enforcement. Valley Water does not issue citations, fines, or surcharges to the public regarding water waste. Instead, Valley Water works with cities and retailers that have a direct relationship with water customers to develop ordinances and water use restrictions to prevent water waste and reduce demand during droughts.

8.7 LEGAL AUTHORITIES

This section describes Valley Water's Legal Authorities as required by Water Code § 10632(a)(7). Valley Water will coordinate with any city or the County to which it provides water supply services for the possible proclamation of a local emergency under California Government Code, California Emergency Services Act (Article 2, Section 8558).

8.7.1 Statutory Authority

Water Code Sections 350 and 375 et seq.

Sections 375 *et seq.* and 350 *et seq.* of the Water Code authorize Valley Water, a wholesale urban water supplier and special district, to, upon appropriate factual findings, implement a water conservation program and/or declare a water shortage emergency by resolution or ordinance and adopt and enforce related conservation measures. If appropriate, Valley Water can reduce the amount of treated water it supplies to local retailers and/or impose water reduction measures.

Santa Clara Valley Water District Act

One source of statutory authority that empowers Valley Water to implement or enforce water shortage response actions is its District Act (The Santa Clara Valley Water District Act, Chapter 1405 of Statutes 1951 of the State of California, Water Code Appendix, Chapter 60). Sections 4 and 5 of the District Act grant Valley Water power to conserve waters within its jurisdiction (as well as import and distribute water).

Specifically, section 4(c) of the District Act authorizes Valley Water to:

- provide for the conservation and management of floodwater, stormwater, or recycled water, or other water from any sources within or outside the watershed in which the district is located for beneficial and useful purposes.
- protect, save, store, recycle, distribute, transfer, exchange, manage, and conserve in any manner any of the waters.
- prevent the waste or diminution of the water supply in the district.
- retain, protect ... drainage, stormwater, floodwater, or treated wastewater, or other water from any sources, within or outside the watershed ....

(District Act, §4 [Objects and purposes], subd. (c)(3), (4), (5) & (6).)

District Act Section 5, paragraph 5, similarly provides that Valley Water may:

... store water in surface or underground reservoirs within or outside of the district ... conserve, reclaim, recycle, distribute, store, and manage water for present and future use within the district; [and]

appropriate and acquire water and water rights, and import water into the district and to conserve within or outside the district, water for any purpose useful to the district ....

Valley Water does, though, charge a groundwater extraction fee on reported extractions and requires all significant groundwater extraction facilities to be metered and extractions reported on either a monthly, semi-annual, or annual basis.

Statutory Groundwater Sustainability Agency Powers

In addition to the District Act and Water Code §§ 350 and 375 et seq., Valley Water is the designated Groundwater Sustainability Agency (GSA) for the Santa Clara and Llagas groundwater subbasins under the Sustainable Groundwater Management Act (SGMA) and has a DWR-approved Alternative to a Groundwater Sustainability Plan (GSP) in place for managing these subbasins. Although Valley Water does not currently restrict groundwater pumping or impose extraction allocations upon owners or operators of groundwater extraction facilities and is not planning to do so, it has the power to do this, if necessary (See Water Code § 10726.4(a)). In February 2018, the Board adopted Resolution 18-04, which describes the fundamental approach to respond to worsening basin conditions, including the steps that Valley Water would take in coordination with stakeholders prior to implementing SGMA authorities to regulate pumping.

8.7.2 Past Valley Water Resolutions

In response to past droughts, Valley Water's Board has passed resolutions calling for reductions in water use. Valley Water passed water conservation related resolutions in April 1988, March 1989, April 1990, March 1992, June 2007, March 2009, July 2010, September 2010, January 2014, February 2014, November 2014, March 2015, November 2015, and June 2016.

Resolutions prior to 2014 generally called for either "voluntary" or "mandatory" reductions in water use. Since 2014, Valley Water has generally avoided use of these words and instead passed resolutions with language identical to, or substantially similar to, the following:

> NOW, THEREFORE, BE IT RESOLVED by the Board of Directors of the Santa Clara Valley Water District that a water use reduction target equal to [e.g., 20] percent of [e.g., 2013] water use is called for through _______________ [future date], and it is further recommended that retail water agencies, local municipalities and the County of Santa Clara implement mandatory measures as needed to achieve the [e.g., 20] percent water use reduction target.

Some Valley Water resolutions have also included language calling for "*a restriction on outdoor watering of ornamental landscapes or lawns with potable water to* [e.g., 4] *days a week through* _________ [future date]."

8.7.3 Contractual Authority

In addition to its statutory authority, Valley Water has contractual authority to reduce the amount of potable, treated water it provides to its retail customers.

Article C, Section 4(c) of Valley Water's standard-form treated water contract with its retailers provides that, if the Board passes a resolution providing for a reduction in water use by more than 10%, it shall reduce the amount of potable treated water it provides to retailers by this same amount, minus 10%. Thus, if the Board calls for a 30% reduction in water use during a drought, it will reduce treated water supplies to its retailers by 20% (a call for a 20% reduction would result in a 10% reduction in treated water supplies, etc.).

Additionally, under Valley Water's standard-form treated water contract, retailers may purchase "non-contract" water above their monthly purchase commitment when additional supplies are available. In the past, in conjunction with a resolution reducing contractual treated water supplies, while Valley Water has continued to allow retailers to purchase "non-contract" treated water, it has increased the price of such "non-contract" water to discourage overuse. Valley Water has the authority to eliminate non-contract water sales altogether if necessary. Although Valley Water's contractual authority relates only to treated water deliveries, not to groundwater extraction, the contracts give Valley Water the ability to adjust treated water pricing to incentivize the use of either treated water or groundwater by its water retailer customers, depending on what best supports local water supply needs and operations.

8.8 FINANCIAL CONSEQUENCES OF WSCP

Under a water shortage scenario, Valley Water expenses are anticipated to increase due to actions to augment water supply and encourage demand reduction. At the same time, revenue would decrease because of a reduction in water sales. Table 8-7 outlines financial consequences and anticipated mitigation actions for each shortage response action stage.

Table 8-7. Valley Water's Financial Consequences and Anticipated Mitigation Actions for Each WSCP Stage

Stage	Stage Title	Requested Short-Term Water Use Reduction	Financial Consequences	Anticipated Mitigation Actions
Stage 1	Normal	None	None	Funding provided for supplemental water supply reserve.
Stage 2	Alert	0 – 10%	Potential increase in operating and maintenance (O&M) expenses and mild to moderate reduction in revenue.	Identify supplemental funding options and reductions to O&M expenses.
Stage 3	Severe	10 – 20%	Moderate to significant increase to O&M expenses and decrease in revenue.	Stage 2 actions plus identify supplement(s) to water rate revenue (likely incremental rate adjustment) and deferral of capital expenditures; use of reserves if necessary.
Stage 4	Critical	20 – 40%	Significant increases to O&M expenses, including supplemental water purchases, and decreases in revenue.	Stage 3 plus short- and long-term O&M budget reductions.
Stage 5	Emergency	40 to 50%	Likely a greater degree of Stage 4.	Stage 4 plus operations limited to core business only.

During past droughts, increased expenses and reduced revenue put upward pressure on future water rates. Valley Water incurred significant costs from actions taken in response to the previous drought (2012 to 2016) and water charges were increased to cover those costs. To minimize rate impacts, Valley Water maintains supplemental funds in its financial reserves to help pay for increased expenditures to remedy shortages. The FY 2021 budget for the supplemental water supply reserve is $15.5M and is projected to grow to roughly $19.5M by FY 2031. The minimum for this reserve is 20% of the annual water purchase budget. The Board may adjust its adopted groundwater production charges midway through the fiscal year, which provides an opportunity to react to unanticipated changes in expenditures or revenue in a timely fashion. Following a drought and the use of the supplemental water supply reserve, funds need to be replenished in subsequent years through groundwater production and treated water charges.

8.9 MONITORING AND REPORTING

This section does not apply to wholesale agencies such as Valley Water.

8.10 WSCP REFINEMENT PROCEDURES

To ensure timely actions are taken per the WSCP during shortages, Valley Water increases its monitoring of its supplies and demands including:

- Groundwater subbasin and land subsidence conditions
- Reservoir storage
- Monthly and season-to-date rainfall at four rainfall stations within the County
- Monthly recycled water deliveries
- Monthly and year-to-date water use for each water retailer in the County
- Current retailer water use compared to a desired decrease in use

Not all water use data is available monthly and there is a time-lag from when water is used and reported. A small percentage of all water use, primarily private well owners, is unmetered and is estimated based on standard tables. Finally, Valley Water does not have access to individual water use account data for water users that purchase water from Valley Water's water retailers. Therefore, Valley Water cannot determine reductions by customer class or by customer unit (per household, for example). This data is only available to the direct retailers.

After a water shortage event is over, Valley Water will conduct an internal 'lessons learned' to determine if the WSCP performed as desired and identify changes to the WSCP if needed. Valley Water holds quarterly meetings with its retailers that allow an opportunity for any retailer to raise issues with the WSCP. In addition, Valley Water develops its MAP report annually as part of its WSMP. The MAP allows the public to raise water supply planning concerns such as the WSCP. Any changes to the WSCP would be prepared by staff and brought to the Board for adoption and amendment to the WSCP.

This WSCP is an independent document separate from the UWMP and may be updated separately at any time. Amendments to this WSCP outside of the UWMP five-year update process will be brought to the Water Conservation and Demand Management Board Committee meetings for public review and comment and to the Board for a public hearing and approval. Valley Water will provide notice to its retailers, cities, and County, and publish this notice on its website, at the start of an amendment process to provide stakeholders an opportunity to participate. Notice will also be provided to retailers, cities, and County at least 30 days prior to a public hearing before the Board that includes the time and place of the hearing.

Amendments approved by the Board will be provided to retailers, cities, County, DWR, and the California State Library within 30 days of approval.

8.11 SPECIAL WATER FEATURE DISTINCTION

Since Valley Water is not a water retailer, it does not make distinctions for special water features such as pools, spas, and decorative water features.

8.12 PLAN ADOPTION, SUBMITTAL, AND AVAILABILITY

Valley Water's Board of Directors set the time and place for the WSCP public hearing for June 8, 2021 at an online meeting. Valley Water notified the water retailers and the cities and County of Santa Clara on May 17, 2021 of the time and date of the public hearing. The draft WSCP was posted on Valley Water's web site (https://www.valleywater.org/your-water/water-supply-planning/urban-water-management-plan) and made available for public review on May 17, 2021. The public hearing notice was published in ***San José Mercury News*** on May 18, 2021 and May 25, 2021 and ***Metro*** on May 19, 2021. Documentation of noticing of the public hearing is included in Appendix B.

Valley Water's Board held the public hearing on June 8 and June 9, 2021 and adopted the WSCP on June 9, 2021. A copy of the conformed Board agenda package for both public hearing and adoption, including the adoption resolution, is in Appendix G. The final WSCP will be posted on Valley Water's website within 30 days of adoption. Paper copies will be made available at the same time the WSCP is posted on the web site for public review during normal business hours.

Within 30 days of Board adoption and prior to July 1, 2021, the adopted WSCP will be submitted electronically to DWR via its Water Use Efficiency data online submittal portal (WUEdata). Electronic copies of the WSCP will also be provided to the cities and County within 30 days of adoption.

CHAPTER 9 – DEMAND MANAGEMENT MEASURES

Valley Water has made significant investments to manage demands for water, and water savings from conservation and stormwater capture were about 75,000 acre-feet per year (AFY) in 2020. Valley Water's Water Supply Master Plan 2040 establishes the target to increase these savings to 99,000 AFY by 2030 and to 110,000 AFY by 2040.

Valley Water develops water supplies and infrastructure to meet the County's water needs and achieve Valley Water Board's Ends Policies for water supply reliability, water conservation, and water recycling. These policies, in conjunction with Valley Water's Water Supply Master Plan 2040, establish broad water supply objectives:

- There is a reliable, clean water supply for current and future generations.
- Water supplies meet at least 100 percent (%) of average annual water demands in non-drought years and water use reductions greater than 20% are not called for during drought years.

9.1 DEMAND MANAGEMENT MEASURES FOR WHOLESALE AGENCIES

This section describes Valley Water's implementation of required Demand Management Measures (DMMs) for wholesale agencies – metering, public education and outreach, water conservation program coordination and staffing, other demand management measures, and asset management. The other measures that Valley Water implements to reduce demands and assist retailers are described in Section 9.2.

9.1.1 Metering

On a monthly basis, Valley Water meters and bills all its retail agency potable water supply deliveries by volume of use. All municipal and industrial water users in the county are currently metered. Valley Water operates an aggressive water measurement program for both treated water deliveries and groundwater users. The current water measurement system measures 100% of all treated water deliveries, 95% of untreated surface water deliveries, and 95% of all groundwater pumping. The remaining 5% of untreated surface water may be used for landscaping, agricultural irrigation, and municipal and industrial uses through Valley Water-issued surface water permits. The remaining 5% (by volume) of groundwater pumping is done by small water users such as residential well owners. Although these residential wells are not metered, an estimate of water pumping or usage is made to determine groundwater production charges. Meters have not been installed on these wells because the cost of installing and reading the meters exceeds the revenue generated by these wells.

Valley Water launched an AMI (Advanced Metering Infrastructure) meter cost-sharing program in 2019. This cost sharing program, for residential and commercial meters, is offered to water retailers in Valley Water's service area. To encourage the installation of these meters, Valley Water will cost share up to $70 per installed AMI meter, and will fund 50% of the cost of the software linked to AMI, when combined with water use reports. Since launched, Valley Water has entered into an agreement with the City of Morgan Hill to share costs for funding the installation of nearly 15,000 AMI meters, as well as home water use reports associated with those meters. Valley Water is in the process of working with other retailers to continue sharing costs for AMI meter installation.

In addition, Valley Water offers rebates for the installation of submeters (since 2008) as well as switching from a mixed-use meter to a dedicated landscape meter (since 2012). The submeter rebate program provides $150 per submeter installed at multi-family housing complexes, such as mobile home parks and condominium complexes. In 2015, the program was expanded to include individual well owners and homes on a shared well. Valley Water plans to continue these programs to meet the region's long-term water conservation goals. Additional program details are in Section 9.2.2.5.

9.1.2 Public Outreach and School Education Programs

9.1.2.1 Public Outreach Programs

Valley Water participates in outreach activities which include multi-media marketing campaigns directed at the diverse county population, website development and maintenance, social media, publications, public meetings, staff participation at community events, interagency partnerships, corporate environmental fairs, professional trade shows, water conservation workshops and seminars, and a speaker's bureau. Outreach efforts focus on supporting customers and key stakeholders to minimize adverse impacts resulting from drought conditions, as well as advancing community knowledge, awareness, and understanding of the conservation and water supply services provided by Valley Water.

Valley Water implemented broad-based advertising programs, participated in community events, collaborated with water retailers to develop their own outreach materials, and reached non-English speaking residents to ensure they were informed about water issues. Valley Water's multi-ethnic outreach expanded beyond translating existing outreach materials to targeting media stories, coverage, and paid advertisements specifically to their communities.

Valley Water's public outreach efforts also include social media and updates to its water conservation program website (www.watersavings.org). The website is updated throughout the year to include the latest program information, new reports/studies, and updates on our workshops. In addition, Valley Water produced and distributed collateral material, including program flyers, free shower timers and other conservation devices, posters, yard and garden signs, restaurant signs for only serving water upon request, and hotel signs encouraging the occupant to reuse their linens.

The most recent outreach campaign that Valley Water promoted ("Yards Have Evolved") focused on encouraging residents to take out their high-water using plants and replace them with low-water using plants. This campaign, which was developed in 2019, featured ads in English, Spanish, Vietnamese and Chinese and included print, online/mobile, social media and radio ads.

In the spring of 2018, Valley Water embarked on an effort to establish a Community-Based Social Marketing strategy to supplement the Conservation campaign. Community-Based Social Marketing, or CBSM for short, is a strategy designed by behavioral scientists (sociologists, psychologists, etc.) to obtain behavior change by removing barriers and establishing social norms. CBSM was initially designed to enhance sustainable and environmentally conscious behaviors. Valley Water's Conservation CBSM Campaign had two objectives: to increase the number of participants in the Landscape Rebate Program and specifically increase lawn conversions; and to increase the number of Graywater Rebate Program participants. Valley Water employed a variety of outreach methods. An evaluation of these methods is expected to be completed in 2021.

Landscape Summit

Starting in 2016, Valley Water has annually held the Landscape Summit, an event developed through Valley Water's Landscape Committee as a forum for landscape professionals to learn about water issues in the county and California as a whole, and how water relates to the landscaping industry. It is also an opportunity for Valley Water to get valuable feedback from landscape professionals, and for attendees to collaborate and exchange ideas. The 6th Annual Landscape Summit was held virtually on February 25, 2021.

Nursery Program

To increase the public's awareness of water-efficient gardening techniques, Valley Water developed the Nursery Program in 1995. This program distributes, at least quarterly, a series of educational materials to nurseries, irrigation supply stores, and box store retailers throughout the county. To display the materials, the program includes literature racks offering free informational materials about water-wise gardening, efficient irrigation techniques, drought-resistant plants, drip irrigation, and Valley Water's water conservation programs. In future program years, the literature racks may ultimately be replaced or supplemented with digital resources that would not need to be replenished as regularly. The Nursery Program literature is currently being distributed to and displayed at more than 30 participating nurseries and vendors. The display, however, has been placed on a temporary hold due to COVID-19 restrictions.

Watershed Approach to Landscaping

Valley Water is partnering with a vendor to develop a comprehensive sustainable landscaping guide, *Watershed Approach to Landscaping*, that is targeted toward residential audiences, landscapers, and irrigation professionals new to sustainable landscape practices. This guide will be ready in early 2021 and will cover how-to and best practice information on building a healthy living soil, selecting local, climate-appropriate, water-wise plants, upgrading to high-efficiency irrigation equipment, capturing rainwater, and reusing graywater.

Demonstration Gardens

Demonstration gardens can inspire community members to incorporate sustainable, ecological, or water-wise plants and techniques into their landscaping. Valley Water has maintained a list of water-wise and California-native plant demonstration gardens to help guide community members in converting their own gardens to be more water-efficient. In 2017, Valley Water created an interactive map that is regularly maintained. This map allows anyone to find demonstration gardens near their home or work by entering an address.

In 2013, Valley Water converted all rotors and sprinklers to in-line drip as part of an on-site demonstration garden on Valley Water's campus. This garden includes plant signs informing the public of the species name and water requirements of the plants on campus. An interactive map, which geotags the labeled plants, was also created for Valley Water's demonstration garden. Visitors can use the interactive map while doing a self-guided walking tour of Valley Water's campus. In the future, Valley Water plans to launch an upgrade of its current demonstration garden to emphasize water-wise, California-native plants and rainwater capture techniques, in addition to efficient irrigation on site.

Workshops

Over the last five years, Valley Water promoted water conservation through workshops and trainings throughout the community. Examples of these include Graywater Laundry to Landscape workshops and presentations to schools, local universities, industry association gatherings, nursery staff, community gardens, native plant society members, corporate events, local Master Gardeners, PG&E's Water Conservation Showcase, and many more. On average, Valley Water conservation staff give about thirty presentations each year.

Because so many sustainable landscaping events take place throughout Santa Clara County and are sponsored by multiple agencies, Valley Water was instrumental in developing and administering the *South Bay Green Gardens* website (www.southbaygreengardens.org). This site was started as a place where all of the public agencies and organizations in the county could promote their events, workshops, etc. The page has become a one-stop shop for information not just on these events, but on all aspects of sustainable landscaping such as pest management, rainwater management, soils and composting, and much more. Valley Water helps fund this site and co-chairs the committee which manages it. The committee includes information about multiple benefits in the site, such as pesticide reduction, water conservation, waste reduction through composting, and stormwater management, in order to show integration of these issues. Additionally, Valley Water staff update the site and make sure the events pages are current.

Bay Area Qualified Water Efficient Landscaper Trainings
In 2019, Valley Water joined with a number of other Bay Area water agencies and the California Water Efficiency Partnership (CalWEP) to create the Bay Area Qualified Water Efficient Landscaper Training (BayQWEL). This regional effort is a professional certification program designed for landscape designers, landscape supervisors, maintenance and irrigation technicians, and park maintenance staff with a focus on water-saving sustainable landscaping techniques. The trainings were initially offered in-person from 2019 to early 2020 in English and Spanish, then adapted to an online curriculum following COVID-19 Shelter-in-Place restrictions later in 2020. Those who become QWEL certified by passing the exam and completing the irrigation audit will be listed as an industry pro on the QWEL website. A total of four online trainings have been offered in 2020, with two more scheduled for early 2021. Additional classes will be scheduled throughout 2021, including the first online Spanish version in March.

Going Native Garden Tour
To showcase exemplary native plant gardens, Valley Water has been a sponsor of the Going Native Garden Tour every spring since 2003. Each year, thousands of participants visit upwards of 60 gardens. These native plant gardens demonstrate the beauty and efficiency of well-maintained native gardens to residents of Santa Clara and San Mateo counties. In addition to showcasing native plants, at least one garden offers native plants for sale each year. In 2020, the tour went completely online, with live garden tours which subsequently were posted as videos online.

Community Events
Each year, Valley Water staffs education booths and activities at public events, libraries and STEAM (Science, Technology, Engineering, the Arts and Mathematics) fairs, providing water education to over 12,800 members of the public. During 2020, Valley Water's Education Outreach program developed a series of virtual presentations and transformed 10 hands-on programs into distance-learning presentations. This has enabled Valley Water to continue to engage with public audiences and deliver water education during the COVID-19 pandemic.

9.1.2.2 School Education Program

Valley Water's Education Outreach program was established in 1995 and has a team of two full-time and 4 part-time staff and student interns that develop and implement water education programs. Education Outreach (EO) provides free grade-level appropriate classroom presentations, puppet shows, and tours of Valley Water facilities to schools, visitor groups and residents within Santa Clara County. The objective is to educate pre-school through college students and residents about water with a focus on water conservation, water supply, watershed stewardship, pollution reduction, flood preparedness, and careers in the water field. EO also provides free education materials to educators, including workbooks and videos, as well as providing hands-on water education training. These educator trainings include both Project WET (Water Education for Teachers) and EO programs that enable educators to lead their own classroom activities to inform their students on water-related topics.

Over the last five years, Valley Water's EO program has reached an average of 15,000 students per year, engaging a total of 75,698 students between 2016 – 2020. EO has supported over 2,900 educators through classroom presentations and tours and provided 20 educator trainings that focus on hands-on water-based science. Students from over 2,300 classrooms have participated in hands-on, Next Generation Science Standards-aligned programs and tours of Valley Water's Outdoor Classrooms and facilities. Examples include lessons using puppet shows and storytelling for pre-K and early elementary students and using hands-on science activities and career development information for middle school, high school, and college students.

9.1.3 Conservation Coordinator

Valley Water established the position of Water Conservation Coordinator in 1990. The current Water Conservation Coordinator is:

Name:	Metra Angelica Richert
Title:	Unit Manager – Water Supply Planning and Conservation Unit
Address:	5750 Almaden Expressway, San Jose, CA 95118
Phone:	(408) 630-2978
Email:	mrichert@valleywater.org

There are four full-time staff members in the Water Conservation Program, typically four part-time temporary staff, and up to six student interns (number varies depending on season and program needs). Staff includes one senior water conservation specialist and three water conservation specialists. The FY 21 water conservation budget is $6.1 million, with funding from water charges, cost-share agreements, and grants.

9.1.4 Other Demand Management Issues

In 2012, voters in Santa Clara County approved the Safe, Clean Water and Natural Flood Protection Program. This enables Valley Water to provide up to $1,000,000 in grant funding for a Water Conservation Innovative Research Grant Program (Grant Program). The goal of the Grant Program was to identify new, innovative technologies that could potentially be incorporated into Valley Water's long-term conservation programs. To date, Valley Water has awarded 14 grants through this program, including grants for several AMI pilots and other water-conserving technologies. In 2020, voters approved a renewal of the Safe, Clean Water and Natural Flood Protection Program, with funds for additional water conservation programs, which will start in FY 21-22.

9.1.5 Asset Management

Valley Water initiated its Asset Management Program in 2002 to ensure continued, reliable services at the level its customers require, at the lowest possible cost. The program includes maintaining an asset registry and a formal, ongoing condition assessment program that monitors risks and maintains a long-term funding model to identify when future asset investments are expected. Valley Water uses this information to develop annual maintenance work plans and make renewal and replacement decisions for its $7.5 billion in water utility assets. In the short term, Valley Water's Asset Management Program seeks to reduce unplanned asset failures or service outages, and the economic, social, or environmental consequences of these failures. For the long-term, the program seeks to minimize operating and capital costs of owning these assets and improve financial planning. Valley Water's Asset Management Planning Model is illustrated in Figure 9-1. Master plans, such as this one, must work hand in hand with Asset Management plans and reports. Typically, the Asset Management Program is focused on meeting existing levels of service. Master plans may propose an increased level of service and/or infrastructure improvements to meet future demands. Recommendations from master plans can influence alternative management strategies and their respective costs in future Asset Management Plans.

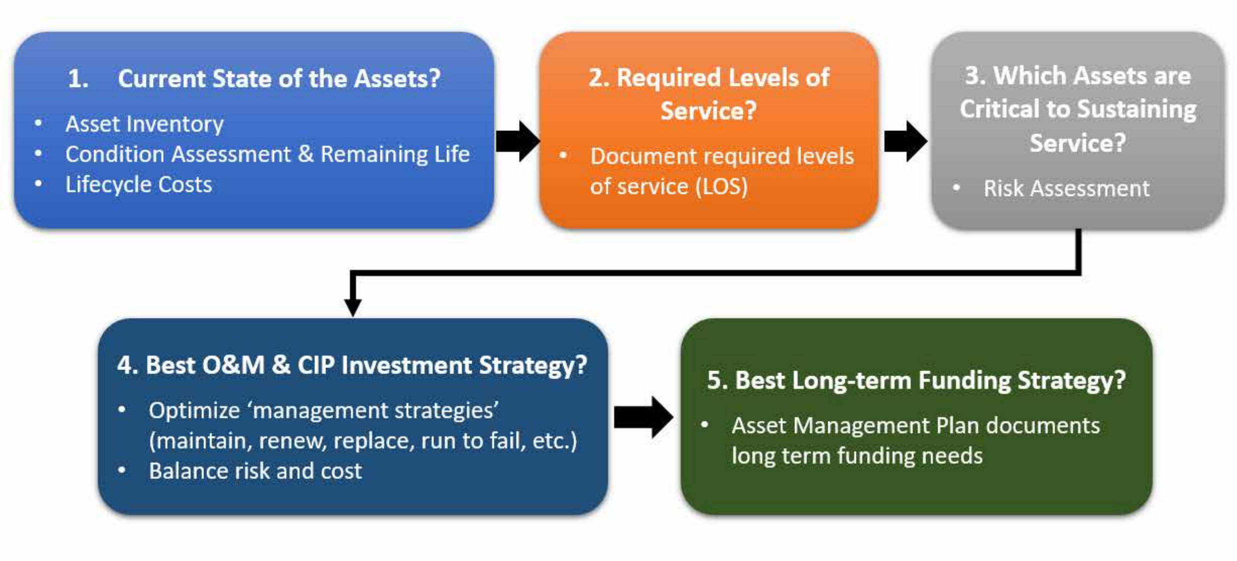

Figure 9-1. Asset Management Planning Model

9.2 PROGRAMMATIC DMMs

Valley Water and its major water retailers enjoy a special cooperative partnership in the regional implementation of a variety of water conservation programs. As the water wholesaler for Santa Clara County, Valley Water is responsible for the implementation of the DMMs.

Participation in all programs listed below is tracked on a monthly basis and by retailer. Furthermore, many water retailers participate in cost sharing agreements maintained by Valley Water. These cost sharing agreements benefit all parties through economies of scale. In FY21, Valley Water is involved in roughly $3.3 million in cost-sharing agreements with the local cities, water retailers, and nonprofit organizations for a variety of water conservation programs.

Additionally, Valley Water regularly sends out customer surveys to determine overall satisfaction with programs and how programs may be improved. Valley Water will continue to work with its water retailers to implement the programs that best meet the public's needs while achieving local, regional, and statewide goals.

9.2.1 Water Waste Prevention Ordinances

Valley Water collaborates with local agencies to develop model water use restrictions that will assist the water retailers and cities in the development of their water waste ordinances. For instance, Valley Water collaborated with local cities and water retailers to develop a model Drought Response and Water Waste Ordinance in 2009 and to develop a model Drought Contingency Plan in 2010. During the last drought, Valley Water collaborated with the water retailers to adopt a consistent two-day per week watering restriction for the majority of the county. After the last drought, Valley Water has continued a 20% voluntary call for conservation with a recommended 3-day per week watering restriction.

CHAPTER 9 – DEMAND MANAGEMENT MEASURES

In 2014, as part of Valley Water's response to the last drought from 2012 to 2016, Valley Water initiated a Water Waste Inspector Program (Water Waste Program). The Water Waste Program facilitates and responds to reports of water waste and violations of local water use restrictions. It also provides an opportunity to educate homeowners and businesses on water conservation as well as the various rebate and technical assistance programs Valley Water offers. In the first two years of the Water Waste Program, nearly 10,000 water waste reports were received and responded to. As of 2020, Valley Water has responded to 11,746 water waste reports.

To facilitate the community's ability to report water waste, four reporting options were developed: email, a water waste hotline, a portal on Valley Water's website, and a mobile application developed for iPhone and Android users. In 2021, the mobile application will be updated as part of a new customer relationship management software. It is expected this tool will improve the effectiveness and efficiency of responding to water waste reports. It will also improve how Valley Water collaborates with local cities and water retailers in resolving water waste countywide.

9.2.2 Residential Programs

9.2.2.1 Water Use Reports

Water use reports have been shown to be effective at encouraging residents to save water and when combined with Advanced Metering Infrastructure, can inform residents about water leaks quickly. In Fiscal Year 2013-14, Valley Water started a program to share costs with the local water retailers City of Palo Alto Utilities Department, City of Santa Clara Water Department, City of Morgan Hill, Gilroy Community Services Department, and San José Municipal Water System on home water use reports. Since the start of this cost sharing program, over 620,000 sites have received water use reports. Valley Water plans to continue to share in the cost of various programs that benefit customers.

9.2.2.2 Residential Surveys

Water Wise House Call Program

As the administrator of this program, Valley Water developed and implemented a strategy to target and market water-use surveys to single-family and multi-family residential customers throughout Santa Clara County, except for the service area of San Jose Water Company (SJWC) as they administer their own program. Between 1998 and 2017, Valley Water performed more than 46,456 residential audits through the Water Wise House Call Program (Table 9-1).

Table 9-1. Residential Program Participation Between FY 2015-16 and FY 2019-20

Residential Programs	Last 5 Years	To-Date
Water Wise House Call Program	5,956	46,465
Water Wise Outdoor Surveys	658	658
DIY Water Wise Survey Kits	1,439	1,439
Fixture Distribution	35,125	375,448
High-Efficiency Clothes Washers	8,924	177,202
High-Efficiency Toilets (HETs)	1,190	26,414
Home Water Use Reports[1]	620,956	620,956

Residential Programs	Last 5 Years	To-Date
Submeters	889	7,172
Graywater	111	124
Total Participation	**675,248**	**1,255,878**
[1]Includes number of sites that receive Home Water Use Reports.		

Valley Water's program included educating the customer on how to read a water meter; checking flow rates of showerheads, faucet aerators, and toilets; installing low-flow showerheads, faucet aerators and toilet flappers if necessary; checking for leaks; checking the irrigation system for efficiency (including leaks); measuring landscaped area; developing an efficient irrigation schedule for different seasons; and providing the customer with evaluation results, water savings recommendations, and other educational materials. In 2004, Valley Water began programming a homeowner's controllers as well (i.e., if allowed by the homeowner, the surveyor will input the recommended schedules into the controller). Valley Water increased program efficiency and participation by using landscape measurements from this program as an initial qualifying step for the Landscape Rebate Program (Sections 9.2.4.3 and 9.2.4.4 for those who chose to participate in both programs

Valley Water's largest retailer, SJWC, offers free water audits to its customers. The audits are performed at customer request, typically in response to a high-water bill concern and/or in response to SJWC or Valley Water marketing efforts. Audits are performed for both residential and commercial customers. Valley Water supports SJWC's water audit program by providing free water conservation supplies, such as showerheads and faucet aerators. SJWC began performing water audits at the end of 1991 and completes about 2,300 per year with approximately 1,700 completed in 2020.

In 2017, Valley Water's free water audit program was replaced by a two-part program, the Water Wise Survey Program. The two-part program offers in-person Water Wise Outdoor Surveys and Do-It-Yourself Water Wise Indoor Surveys, as described below.

Water Wise Survey Program

The outdoor portion of the Water Wise Survey Program is similar in concept to the Water Wise House Call Program's outdoor water audit. Water Wise Outdoor Survey Program offers a free, comprehensive consultation from a trained irrigation professional to single-family and small multi-family sites (under ½ acre of landscape area) in Santa Clara County with a working irrigation system (excluding SJWC customers). The consultation includes evaluating the irrigation system, flagging issues onsite, identifying rebate programs for which participants may also qualify, and creating a custom report detailing the survey findings. Since it launched in 2017, Valley Water performed more than 650 residential Water Wise Outdoor Surveys (Table 9-1).

The Do-It-Yourself (DIY) Water Wise Indoor Surveys Program offers free showerheads, aerators, and toilet flappers to anyone who completes a companion survey form. A physical kit is available in English, Spanish, Chinese, and Vietnamese; additionally, a virtual kit is available. Companion videos are offered to guide customers through the DIY survey steps. Customers must first share their current fixtures that are high water use before Valley Water sends them a free low-flow device. Due to low response rates, Valley Water may cease this requirement to encourage greater participation in this program. The DIY kits are available to single-family and multi-family residential properties throughout Santa Clara County. More than 1,430 kits have been distributed since 2017 (Table 9-1).

Fixture Distribution

Valley Water also distributes high-quality, low-flow showerheads and faucet aerators to community members through water retailers and public events. Since program inception in 1992, more than 375,000 low-flow showerheads and aerators have been distributed throughout the county, including over 35,000 in the last 5 years (Table 9-1).

Valley Water plans to continue offering free showerheads and aerators through its DIY Water Wise Indoor Surveys, its water retailers, and various outreach events to meet the region's long-term water conservation goals.

9.2.2.3 High-Efficiency Clothes Washers

Valley Water offered a residential high-efficiency washer rebate between July 1995 and December 2016. In October 2001, Valley Water began participating in the regional Bay Area Water Utility Clothes Washer Rebate Program, which has been successfully partnering with PG&E between January 2008 and December 2016. To address concerns for local water quality, washers that utilized silver-ion technology did not qualify for this program regardless of their efficiency. In mid-2014, a multi-tiered combined rebate was implemented to transition program participants to more stringent fixture standards:

- Purchasing Energy Star Most Efficient (ESME) washers resulted in the combined rebate increasing to $200 ($125 of which was from Valley Water).
- Purchasing the Consortium for Energy Efficiency's (CEE's) Tier 3 washers received a reduced Valley Water contribution of only $50 with the goal of promoting washers that qualify for the more efficient standard.

In January 2015, qualifying standards were adjusted to streamline requirements to only rebate for qualifying ESME washers at a combined rebate of $150 ($100 of which was from Valley Water) until the program ended on December 31, 2016.

Valley Water approved more than 177,000 rebates during the program's history. In the final 18 months of the program, nearly 9,000 rebates were approved. The program ended in response to the vast improvement of federal Energy Star program's efficiency standards over the years. By the end of the program, Valley Water's Water Conservation Savings Model estimated nearly 60% of all single-family homes had efficient clothes washers within its service area.

9.2.2.4 High-Efficiency Toilets

Valley Water had provided incentives for the retrofit of approximately 244,000 residential toilets from 1992 through June 2003. In 2004, Valley Water shifted to a high-efficiency toilet (HET) program, and between 2004 (the first year of the program) and 2013, Valley Water rebated approximately 16,000 HETs. In response to the State of California's new requirement that all toilets sold or installed in the state flush at 1.28 gallons per flush (gpf) or less, January 2014 marked the beginning of Valley Water's strictest standard yet for HETs to qualify for the rebate program - only Premium HETs would qualify for the $125 rebate. Premium HETs save nearly 15% more water than the state standard of 1.28 gpf by using only 1.1 gpf with superior flush performance (at least 600 grams per flush as evaluated by an independent group under standardized conditions).

Between 2004 and 2016, Valley Water issued over 26,400 HET rebates in total since this iteration of Valley Water's high-efficiency toilet rebate began in 2004 (Table 9-1). The program was phased out in 2016 to reprioritize funds to other programs with greater opportunities for water savings.

9.2.2.5 Submeter Rebate Program

Beginning as a pilot in 2001 and extended in 2008, this program provides a rebate (in FY16, the rebate amount increased from $100 to $150) for every submeter installed at multi-family housing complexes, such as mobile home parks and condominium complexes. Individual well owners and homes on a shared well also qualify.

Water use records from participating mobile home parks showed an average water savings of 23% per mobile home in a pilot study. This program has issued over 7,170 rebates to date (Table 9-1). Valley Water plans to continue to offer this program in the future to reach the region's long-term water conservation goals.

9.2.2.6 Graywater Laundry to Landscape Programs

In the last 5 years, Valley Water issued 40 graywater rebates (launched in 2014) and funded the direct installation of 71 graywater systems (launched in 2019). Since the program launched, 124 total graywater systems have been installed (Table 9-1).

Valley Water's Graywater Laundry to Landscape (L2L) Rebate Program rebate amount started at $100 in 2014, and in response to the drought, increased to $200 a few months later. The Cities of Cupertino and Morgan Hill and San José Municipal Water cost-share with this program to increase the rebate to $400 total. In addition to providing a rebate for properly connecting a clothes washer to a laundry-to-landscape system, the graywater program also provides information, resources, and workshops on graywater. Resources include maintenance steps, detergent information, finding contractors, increasing awareness of local nonprofit organizations that specialize in graywater, and educating constituents on important factors to consider with more complicated graywater systems (e.g., branched-drain graywater and whole house graywater systems) even though rebates for those options are not currently offered.

Graywater use in irrigated landscapes decreases potable water use by approximately 17 gallons per person per day or 14,565 gallons per household (on average), depending on the site and system design. California Plumbing Code (CPC) does not require a permit for installing an L2L system. However, the CPC is specific as to how L2L systems can be installed, and Valley Water's rebate's eligibility requirements are framed to meet those specifications. Additionally, to protect public health and safety, prior to giving project approval, Valley Water checks each applicant's property's depth to groundwater. At post inspections, applicants must demonstrate adherence to the CPC's specifications to help ensure graywater does not pool or drain to their neighbors' properties.

In 2019, Valley Water in partnership with a local non-profit organization, Ecology Action, launched a training program for landscape professionals and a Graywater Direct Installation Program for underserved community members, including low-income individuals, people 60 years or older, U.S. veterans, and people with disabilities. The Green Gardener Graywater Installer Certification Program trained 20 professionals to install L2L graywater systems. Between June 2019 and June 2020, the direct installation service assessed 307 properties and installed 71 L2L graywater systems. Over 31,660 square feet of medium- and high-water use landscapes were converted from potable irrigation to graywater.

9.2.3 Commercial Programs

9.2.3.1 Water Efficient Technology Rebate Program

The Water Efficient Technology Rebate (WET Rebate or WET Program; formerly known as the Custom/Measured Rebate Program) provides rebates for process, technology, and equipment retrofits that save water. To encourage all commercial and industrial businesses to implement permanent water reduction measures, unique projects that meet program requirements are eligible for a rebate of either $4 per hundred cubic feet (CCF) of water saved or 50% of equipment costs excluding taxes and labor, whichever is less, up to $50,000. Projects must save at least 100 cubic feet of water annually. Examples of such projects are generally unique to specific industries such as ozone laundry systems or technologies to reduce potable water use when maintaining ice rinks, with myriad other examples. In January 2014, these rebates were temporarily increased to $8 per CCF to promote participation during the drought before returning to $4 per CCF. Cost sharing agreements increase the rate and maximum rebate in some areas.

To date, Valley Water has funded 110 projects, saving approximately 680,663 CCF/year (1,563 AFY) (Table 9-2). Since 2015, the WET Rebate has helped save over 28,440 CCF per year from 12 completed projects. In 2021, Valley Water will adjust the program so that the rebate will be based on either the lesser of $4 per CCF or up to 100% of equipment costs excluding taxes and labor, up to $100,000. This doubles the potential proportion of equipment costs covered by the rebate in addition to doubling the maximum rebate. The WET Rebate continues to be one of Valley Water’s most cost-effective programs in meeting the region’s long-term water conservation goals.

Table 9-2. Commercial Program Participation Between FY 2015-16 to FY 2019-20

Commercial Programs	Last 5 Years	To-Date
WET Rebates (CCF/Year)[1]	28,440	680,663
WET Rebates	12	110
High-Efficiency Toilet Rebates & Direct Installation (MFD & CII)	7,321	35,052
Urinal Rebates & Direct Installation	464	2,581
Commercial Washer Program	266	4,913
Faucet Aerator Distribution	18,143	26,793
Pre-Rinse Spray Valves	360	4,949
Total Participation	**26,566**	**74,398**

[1]Excludes CCF/Year from WET Rebates.

9.2.3.2 Commercial Toilet and Urinal Programs

Valley Water has been replacing inefficient toilets in commercial, industrial, and institutional (CII) sites since 1994. The CII toilet rebate programs have frequently been offered in tandem with various iterations of high-efficiency urinal (HEU) programs, HET and HEU direct install programs, and retrofit programs for urinal valve installation. Since July 2015, over 7,300 HETs were installed or rebated (Table 9-3). Additionally, since 2005, Valley Water has had a program to replace urinal flush valves of old, inefficient 1.0 gpf or more urinals with a flush valve that uses only a 0.5 gallon per flush. Since the program was started, approximately 2,580 urinals had been retrofitted or rebated, with 464 installed in the last five years (Table 9-3).

In order to increase efficiency and cost effectiveness, Valley Water created a successful pilot program in 2020 which replaced fifty-nine (59) 1.6 gpf toilets with 0.8 gpf toilets in a low-income apartment complex. This pilot will serve as the basis for a new Fixture Replacement Program to launch in 2021 to replace or retrofit toilets, urinals, and more for multi-family residences and commercial, industrial, and institutional properties.

9.2.3.3 Commercial Faucet Aerator Program

Since 2010, Valley Water has offered free 0.5 gallon per minute faucet aerators to qualifying businesses and schools. Nearly 26,800 faucet aerators have been distributed through this program, with 18,143 being distributed during the last five years. Much of the recent distribution is due to a direct distribution program called WaterLink, which was administered by a local non-profit organization, Ecology Action, and focused on water and energy efficiency direct installation measures (see 9.2.3.6).

9.2.3.4 Pre-Rinse Spray Valve Program

Pre-rinse spray valves are designed to remove food waste from dishes prior to dishwashing, and are often used in commercial kitchens. In previous years, Valley Water partnered with other agencies to offer a direct installation program for high-efficiency pre-rinse spray valves (PRSVs). In 2010 Valley Water purchased a quantity of PRSVs with a flow rate of 1.15 gallons per minute for distribution to commercial sites, especially those identified through Valley Water's previous CII Water Survey Program. Since July 2015, nearly 360 pre-rinse spray valves were retrofitted, and nearly 4,950 have been installed since Valley Water began promoting these devices in 2003 (Table 9-3). Valley Water plans to continue distributing these devices to meet the region's long-term water conservation goals.

9.2.3.5 WaterLink Program

In collaboration with Ecology Action, Valley Water funded a program called WaterLink, a water/energy savings program that provided turnkey water/energy upgrades to residents, businesses, schools, and public agencies throughout Santa Clara County. Efforts were focused within Disadvantaged Community Census tracts (defined by scoring 76% and above using California Environmental Screening Tools version 2.0). To achieve significant water and energy savings, the WaterLink program delivered a suite of direct installation projects that produced persistent water/energy savings and tangible economic benefits by reducing utility bills. Direct installation equipment included efficient showerheads and aerators, clothes washers, pre-rinse spray valves, and ozone laundry systems. Additionally, the program included replacing turfgrass with low-water using landscape. The WaterLink program has concluded and totals for these programs are included in Table 9-3.

9.2.4 Landscape Programs

9.2.4.1 Large Landscape Surveys

Analogous to Water Wise Outdoor Surveys offered through the landscape portion of the Water Wise Survey Program, Valley Water has offered and provided large landscape water surveys in the county since 1994. Landscape managers have been provided water-use analyses, scheduling information, in-depth irrigation evaluation, a site-specific water budget, and recommendations for affordable irrigation upgrades. Each site received a detailed report upon completion of the survey. An annual report is produced to recap the previous year's efforts. Previously a stand-alone program, starting in 2015 the program was offered through the Large Landscape Program (described below).

This highly successful and well-received program has conducted nearly 1,820 surveys through 2020. Participants from this program are encouraged to participate in the Landscape Rebate Program (see 9.2.4.3). Valley Water plans to continue to offer and expand this program in the future to reach the region's long-term water conservation goals.

9.2.4.2 Large Landscape Program

The Large Landscape Program (formerly known as the Landscape Water Use Evaluation Program or LWUEP) launched in May 2014. All sites enrolled in the program receive a monthly water usage report. The reports provide an objective evaluation of a site's water use at a glance for every billing period. Various data inputs, including irrigated area, vegetation types, type of irrigation system, and daily weather (evapotranspiration minus effective rainfall) are included in a detailed calculation to develop the water budgets. Sites are encouraged to share the monthly reports with everyone involved in landscape decision making at the site, including the bill payer, site manager, landscape contractor and board members. Sites are also eligible to receive a complimentary on-site landscape field survey by an irrigation expert and receive a thorough investigation of the site's irrigation issues.

A total of 557 sites were enrolled in the program at its outset from the following water retailer service areas: Cities of Gilroy, Mountain View, Palo Alto, Sunnyvale, and Santa Clara. By the end of mid-2015, 1,050 sites were active in this program. In 2020, there are 3,000 active sites that include both potable and recycled water landscapes (Table 9-3). Representing 91% of Valley Water's service area, the full list of participating water retailers includes the original five service areas mentioned above as well as the Cities of Milpitas and Morgan Hill, San José Municipal Water, and San José Water Company. Nearly 122,000 water-use reports and monthly budgets have been distributed. Valley Water's vendor works closely with participating water retailers to market and leverage the services offered through this program for participating sites.

Table 9-3. Commercial Program Participation Between FY 2015-16 to FY 2019-20

Landscape Programs	Last 5 Years	To-Date
Large Landscape Surveys	162	1,816
Large Landscape Program[1]	2,213	3,000
Turf Conversion (square feet)[2]	8,629,926	12,975,063
Irrigation Equipment[3]	313,010	362,160
WBICs[4]	3,960	6,726
In-Line Drip Conversion (square feet)	166,461	166,461

Landscape Programs	Last 5 Years	To-Date
Rain Barrels (number of units)	110	110
Cisterns (gallons)	32,745	32,745
Rain Gardens (square footage of roof area diverted)	12,389	12,389
Landscape Maintenance Program	715	715
Total Participation[5]	**320,170**	**374,527**
[1]Represents total active sites in program. The "Last 5 Years" shows the number of sites added, and "To Date" shows total active sites. [2]Includes pilot programs and partnership with Our City Forest; square footage estimated up to 2011. [3]Excludes WBICs. [4]Includes pilot programs and participation from residential and CII sites. [5]Total excludes square footage from Turf Conversion, In-Line Drip Conversion, Rain Gardens and total gallons from cisterns.		

As of the end of 2019, the sites enrolled in Valley Water program were saving 31% on irrigation usage compared to 2013 usage. Valley Water will continue to offer and expand this program in the future to reach the region's long-term water conservation goals, particularly with regards to opportunities for this program to assist compliance with elements of AB 1668/SB 606.

9.2.4.3 Landscape Rebate Program

Conversion Rebates

Valley Water began to focus on water efficient landscapes by launching a version of the program in early 2005. The original program offered rebates to residential and commercial sites for the replacement of approved high-water using landscape with low-water use plants, mulch and permeable hardscape. Participants could receive up to $0.75 per square foot of irrigated turf grass with a maximum rebate of $1,000 and $10,000 for residential and commercial sites respectively. In an effort to expedite program participation, Valley Water's Board approved doubling the maximum rebate from $1,000 to $2,000 for residents and from $10,000 to $20,000 for commercial sites in March 2009. The rebate cap for commercial, institutional, and multi-family (5 or more units) sites was then increased to $50,000 on January 1, 2020. Cost sharing agreements increase the rate per square foot and rebate cap in some areas.

Currently, any qualified property in Santa Clara County with qualifying high-water using landscape can receive rebates for converting to qualifying low water using landscape with a minimum of 50% qualifying plant coverage; 2 to 3 inches of mulch; and a conversion from overhead irrigation to drip, micro spray, bubbler, or no irrigation. In January 2014, the Landscape Conversion rebate was increased from $0.75 per square foot (sq ft.) to $1.00 per sq ft. However, in April of 2014 in direct response to the drought, Valley Water's Board approved adding funding to the program to support a rebate of $2.00 per sq. ft. with no maximum rebate. On July 1, 2016, the rebate rate returned to $1/sq ft and the rebate caps were reinstituted.

Valley Water continued to experience unprecedented increases in terms of rebate amounts as well as participation and interest from the community through the end of the drought and into FY2020. From July 2015 to June 2020, over $14.3 million dollars was rebated for approximately 8.3 million square feet of conversion. Through June 2020, Valley Water has rebated for over 12.7 million square feet of landscape conversion (Table 9-4). Valley Water plans to continue to offer this rebate in the future in order to reach the region's long-term water conservation goals.

In January of 2019, Valley Water added Rainwater Capture Rebates to the Landscape Rebate Program. Customers now have the opportunity to receive rebates for the installation of rain barrels, cisterns, and rain gardens. Since the start of the Rainwater Capture Rebates, rainwater has been diverted from nearly 20,000 square feet of roof area into qualifying rain gardens, 165 rain barrels have been installed, and cisterns with a total combined capacity of over 33,000 gallons have received a rebate.

Lawn Busters Program

In September 2015, Valley Water executed an Agreement with Our City Forest (OCF), a local non-profit organization, to provide $340,000 to fund OCF's Lawn Conversion Program (Lawn Busters Program). Lawn Busters Program is designed to provide a low cost, expedient option for low-income, elderly, disabled or veteran homeowners and institutions within disadvantaged communities throughout Santa Clara County who wish to convert their lawns to low-water using landscape. In targeting these hard-to-reach sectors, the Lawn Busters Program is intended to help Valley Water meet its short-term drought response goals as well as its long-term water conservation goals. By partnering with OCF, Valley Water combines resources and implements the program more cost-effectively than would be possible otherwise.

Since the start of the Lawn Busters Program, Valley Water added $110,000 to the contract, for a total of $450,000, and OCF has converted roughly 200,000 square feet of lawn to low-water using landscape.

9.2.4.4 Landscape Rebate Program – Irrigation Equipment Rebates

Valley Water provides rebates for the following pieces of irrigation equipment as summarized in Table 9-4.

Table 9-4. Landscape Rebate Program Irrigation Equipment Rebates

Qualifying Hardware and Rainwater Capture Projects	Maximum Rebate Amount per Unit
Rain Sensors	$50
High-Efficiency Nozzles	$5
Rotor Sprinklers or Spray Bodies equipped with Pressure Regulation or Check Valves	$20
Dedicated Landscape Meters, Flow Sensors, or Hydrometers	$1,000
WBICs, 1-12 Stations	$300
WBICs, 13-24 Stations	$1,000
WBICs, 25+ Stations	$2,000
In-Line Drip Irrigation[1] (converting from sprinklers in existing shrub, perennial, or annual planting beds)	$0.25 per square foot
Rain Barrel (40-199 gallons)	$35 per barrel
Cisterns (200 gallons or more)	$0.50 per gallon
Rain Gardens	$1 per square foot of roof area converted[2]

[1]Converts sprinklers in existing shrub, perennial, or annual planting beds.
[2]Up to $300 per site.

Similar to landscape conversion, Valley Water's Board of Directors approved adding funding to the program during the drought to support higher rebate amounts for many of the items listed above. Due to these higher rebate amounts as well as the effects of the drought, Valley Water experienced unprecedented increases in interest and participation from the community over the last few years. While participation rates have slowed compared to the height of the drought years, FY19 and FY20 combined still show over 48,000 irrigation equipment pieces upgraded compared to pre-drought FY12 and FY13 combined numbers of 8,236, a more than 500% increase.

Additionally, nearly 4,000 Weather-Based Irrigation Controllers (WBICs) have been installed during FY15-FY20. Sometimes referred to as "smart controllers", WBICs utilize the principles of evapotranspiration or "ET" to automatically calculate a site-specific irrigation schedule based on several factors, including plants and soil type. The controller then adjusts the irrigation schedule as local weather changes to regulate unnecessary irrigation, saving up to 20% of irrigation water use when used properly. Valley Water plans to continue to offer rebates for WBICs in the future in order to reach the region's long-term water conservation goals.

9.2.4.5 Landscape Maintenance Consultation Program

The Landscape Maintenance Consultation Program, started in May of 2018, was developed based on recommendations from Valley Water's Landscape Committee as a way to help Landscape Rebate Program participants learn how to properly maintain their newly converted low water use gardens. To date, 715 residential rebate customers have participated in the program (Table 9-3). During the free, one-hour consultation, the customer has an opportunity to walk through their garden with a landscape professional, reviewing site specific recommendations for plant maintenance and pruning, soil health, pest management, and irrigation scheduling and maintenance. The Landscape Maintenance Consultation Program will continue to be offered to new rebate program participants whose gardens are at least one year established.

9.3 CONCLUSION

Valley Water, through a unique cooperative partnership with its retailers, offers regional implementation of a variety of water conservation programs in an effort to permanently reduce water use in Santa Clara County. While Valley Water is only responsible for implementation of the DMMs, it continues to collaborate with its water retailers to implement various water conservation programs on a regional basis. By taking the lead in implementing many of the various DMM components, Valley Water is ensuring its long-term water supply reliability goals are met as well as assisting its water retailers in meeting their goals. The goal of Valley Water's DMM components is to save approximately 99,000 AFY by the year 2030 and 110,000 AFY by 2040, using 1992 as a base year. In 2021, Valley Water will update its Water Conservation Strategic Plan to identify new or improved strategies to reach our savings goals as well as future Water Use Objectives required by AB 1668 and SB 606.

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

This section describes the adoption, submittal, and implementation of this 2020 UWMP. A checklist is also provided to facilitate DWR's review of the 2020 UWMP.

10.1 PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Valley Water's Board of Directors set the time and place for the 2020 UWMP public hearing for June 8, 2021. Valley Water notified the water retailers and the cities and County of Santa Clara on May 17, 2021 of the time and date of the public hearing. The draft UWMP was posted on Valley Water's web site (https://www.valleywater.org/your-water/water-supply-planning/urban-water-management-plan) and made available for public review on May 17, 2021. The public hearing notice was published on ***San José Mercury News*** on May 18, 2021 and May 25, 2021 and ***Metro*** on May 19, 2021. Documentation of noticing of the public hearing is included in Appendix B.

Valley Water's Board held the public hearing on June 8 and June 9, 2021 and adopted the UWMP on June 9, 2021. A copy of the conformed Board agenda package for both public hearing and adoption, including the adoption resolution, is in Appendix G. The final 2020 UWMP will be posted on Valley Water's website within 30 days of adoption. Paper copies will be made available at the same time the 2020 UWMP is posted on the web site for public review during normal business hours.

Within 30 days of Board adoption and prior to July 1, 2021, the adopted 2020 UWMP will be submitted electronically to DWR via its Water Use Efficiency data online submittal portal (WUEdata). Electronic copies of the 2020 UWMP will also be provided to the cities and County within 30 days of adoption. Valley Water will implement this adopted 2020 UWMP in accordance with the California Urban Water Management Planning Act.

Following adoption, Valley Water will continue to implement water supply planning programs and projects identified in this 2020 UWMP to meet its level of service goal. As part of the on-going master planning process to address future uncertainties with demands, existing supplies, and proposed projects, Valley Water conducts annual evaluation of water supply projects through the MAP process to maintain a diverse water supply portfolio to provide safe and clean water for current and future generations in Santa Clara County.

10.2 UWMP CHECKLIST

The following checklist is provided to facilitate DWR's review of the completeness of this document and is organized by subject matter. In addition, a complete set of standardized tables prescribed by DWR is provided in Appendix A.

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10615	A plan shall describe and evaluate sources of supply, reasonable and practical efficient uses, reclamation and demand management activities.	Overview of Chapter 1
10630.5	Each plan shall include a simple description of the supplier's plan including water availability, future requirements, a strategy for meeting needs, and other pertinent information. Additionally, a supplier may also choose to include a simple description at the beginning of each chapter.	Chapter 1

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10620(b)	Every person that becomes an urban water supplier shall adopt an urban water management plan within one year after it has become an urban water supplier.	Section 2.1
10620(d)(2)	Coordinate the preparation of its plan with other appropriate agencies in the area, including other water suppliers that share a common source, water management agencies, and relevant public agencies, to the extent practicable.	Section 2.4, 4.4 and Appendix B
10642	Provide supporting documentation that the water supplier has encouraged active involvement of diverse social, cultural, and economic elements of the population within the service area prior to and during the preparation of the plan and contingency plan.	Section 2.4, 10.1 and Appendix B
10631(h)	Retail suppliers will include documentation that they have provided their wholesale supplier(s) - if any - with water use projections from that source.	Not applicable to Valley Water as a Wholesale Supplier.
10631(h)	Wholesale suppliers will include documentation that they have provided their urban water suppliers with identification and quantification of the existing and planned sources of water available from the wholesale to the urban supplier during various water year types.	Section 2.4 and Appendix B
10631(a)	Describe the water supplier service area.	Section 3.1
10631(a)	Describe the climate of the service area of the supplier.	Section 3.3
10631(a)	Provide population projections for 2025, 2030, 2035, 2040 and optionally 2045.	Section 3.4
10631(a)	Describe other social, economic, and demographic factors affecting the supplier's water management planning.	Section 3.4
10631(a)	Indicate the current population of the service area.	Section 3.4
10631(a)	Describe the land uses within the service area.	Section 3.5
10631(d)(1)	Quantify past, current, and projected water use, identifying the uses among water use sectors.	Section 4.2
10631(d)(3)(C)	Retail suppliers shall provide data to show the distribution loss standards were met.	Section 4.2.5 and Appendix C
10631(d)(4)(A)	In projected water use, include estimates of water savings from adopted codes, plans, and other policies or laws.	Section 4.2.6
10631(d)(4)(B)	Provide citations of codes, standards, ordinances, or plans used to make water use projections.	Section 4.2.6

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10631(d)(3)(A)	Report the distribution system water loss for each of the 5 years preceding the plan update.	Optional - Not provided.
10631.1(a)	Include projected water use needed for lower income housing projected in the service area of the supplier.	Does not apply to Valley Water as a Wholesale Supplier.
10635(b)	Demands under climate change considerations must be included as part of the drought risk assessment.	Sections 4.1, 4.4 and 7.3
10608.20(e)	Retail suppliers shall provide baseline daily per capita water use, urban water use target, interim urban water use target, and compliance daily per capita water use, along with the bases for determining those estimates, including references to supporting data.	Does not apply to Valley Water as a Wholesale Supplier.
10608.24(a)	Retail suppliers shall meet their water use target by December 31, 2020.	Does not apply to Valley Water as a Wholesale Supplier.
10608.36	Wholesale suppliers shall include an assessment of present and proposed future measures, programs, and policies to help their retail water suppliers achieve targeted water use reductions.	Section 5.1
10608.24(d)(2)	If the retail supplier adjusts its compliance GPCD using weather normalization, economic adjustment, or extraordinary events, it shall provide the basis for, and data supporting the adjustment.	Does not apply to Valley Water as a Wholesale Supplier.
10608.22	Retail suppliers' per capita daily water use reduction shall be no less than 5 percent of base daily per capita water use of the 5-year baseline. This does not apply if the suppliers base GPCD is at or below 100.	Does not apply to Valley Water as a Wholesale Supplier.
10608.4	Retail suppliers shall report on their compliance in meeting their water use targets. The data shall be reported using a standardized form in the SBX7-7 2020 Compliance Form.	Does not apply to Valley Water as a Wholesale Supplier.
10631(b)(1)	Provide a discussion of anticipated supply availability under a normal, single dry year, and a drought lasting five years, as well as more frequent and severe periods of drought.	Sections 6.10 and 7.2
10631(b)(1)	Provide a discussion of anticipated supply availability under a normal, single dry year, and a drought lasting five years, as well as more frequent and severe periods of drought, *including changes in supply due to climate change.*	Section 6.11
10631(b)(2)	When multiple sources of water supply are identified, describe the management of each supply in relationship to other identified supplies.	Chapter 6
10631(b)(3)	Describe measures taken to acquire and develop planned sources of water.	Sections 6.1 - 6.10

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10631(b)	Identify and quantify the existing and planned sources of water available for 2020, 2025, 2030, 2035, 2040 and optionally 2045.	Section 6.10
10631(b)	Indicate whether groundwater is an existing or planned source of water available to the supplier.	Section 6.2
10631(b)(4)(A)	Indicate whether a groundwater sustainability plan or groundwater management plan has been adopted by the water supplier or if there is any other specific authorization for groundwater management. Include a copy of the plan or authorization.	Section 6.2
10631(b)(4)(B)	Describe the groundwater basin.	Section 6.2
10631(b)(4)(B)	Indicate if the basin has been adjudicated and include a copy of the court order or decree and a description of the amount of water the supplier has the legal right to pump.	Section 6.2
10631(b)(4)(B)	For unadjudicated basins, indicate whether or not the department has identified the basin as a high or medium priority. Describe efforts by the supplier to coordinate with sustainability or groundwater agencies to achieve sustainable groundwater conditions.	Valley Water serves as the Groundwater Sustainability Agency.
10631(b)(4)(C)	Provide a detailed description and analysis of the location, amount, and sufficiency of groundwater pumped by the urban water supplier for the past five years	Section 6.2
10631(b)(4)(D)	Provide a detailed description and analysis of the amount and location of groundwater that is projected to be pumped.	Section 6.2
10631(c)	Describe the opportunities for exchanges or transfers of water on a short-term or long- term basis.	Section 6.7
10633(b)	Describe the quantity of treated wastewater that meets recycled water standards, is being discharged, and is otherwise available for use in a recycled water project.	Not applicable. Valley Water does not treat wastewater.
10633(c)	Describe the recycled water currently being used in the supplier's service area.	Section 6.5
10633(d)	Describe and quantify the potential uses of recycled water and provide a determination of the technical and economic feasibility of those uses.	Section 6.5
10633(e)	Describe the projected use of recycled water within the supplier's service area at the end of 5, 10, 15, and 20 years, and a description of the actual use of recycled water in comparison to uses previously projected.	Section 6.5

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10633(f)	Describe the actions which may be taken to encourage the use of recycled water and the projected results of these actions in terms of acre-feet of recycled water used per year.	Section 6.5
10633(g)	Provide a plan for optimizing the use of recycled water in the supplier's service area.	Section 6.5
10631(g)	Describe desalinated water project opportunities for long-term supply.	Section 6.6
10633(a)	Describe the wastewater collection and treatment systems in the supplier’s service area with quantified amount of collection and treatment and the disposal methods.	Not applicable. Valley Water does not treat wastewater.
10631(f)	Describe the expected future water supply projects and programs that may be undertaken by the water supplier to address water supply reliability in average, single-dry, and for a period of drought lasting 5 consecutive water years.	Section 6.9 and Chapter 7
10631.2(a)	The UWMP must include energy information, as stated in the code, that a supplier can readily obtain.	Section 6.12 and Tables O
10634	Provide information on the quality of existing sources of water available to the supplier and the manner in which water quality affects water management strategies and supply reliability	Chapter 7
10620(f)	Describe water management tools and options to maximize resources and minimize the need to import water from other regions.	Sections 7.1, 7.3 and 8.2, and Chapter 9
10635(a)	Service Reliability Assessment: Assess the water supply reliability during normal, dry, and a drought lasting five consecutive water years by comparing the total water supply sources available to the water supplier with the total projected water use over the next 20 years.	Section 7.2
10635(b)	Provide a drought risk assessment as part of information considered in developing the demand management measures and water supply projects.	Section 7.3
10635(b)(1)	Include a description of the data, methodology, and basis for one or more supply shortage conditions that are necessary to conduct a drought risk assessment for a drought period that lasts 5 consecutive years.	Section 7.3
10635(b)(2)	Include a determination of the reliability of each source of supply under a variety of water shortage conditions.	Section 7.1
10635(b)(3)	Include a comparison of the total water supply sources available to the water supplier with the total projected water use for the drought period.	Section 7.3

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10635(b)(4)	Include considerations of the historical drought hydrology, plausible changes on projected supplies and demands under climate change conditions, anticipated regulatory changes, and other locally applicable criteria.	Sections 7.1 - 7.3
10632(a)	Provide a water shortage contingency plan (WSCP) with specified elements below.	Chapter 8
10632(a)(1)	Provide the analysis of water supply reliability (from Chapter 7 of Guidebook) in the WSCP	Section 8.1 and Chapter 7
10632(a)(10)	Describe reevaluation and improvement procedures for monitoring and evaluation the water shortage contingency plan to ensure risk tolerance is adequate and appropriate water shortage mitigation strategies are implemented.	Section 8.10
10632(a)(2)(A)	Provide the written decision- making process and other methods that the supplier will use each year to determine its water reliability.	Section 8.2
10632(a)(2)(B)	Provide data and methodology to evaluate the supplier’s water reliability for the current year and one dry year pursuant to factors in the code.	Section 8.2
10632(a)(3)(A)	Define six standard water shortage levels of 10, 20, 30, 40, 50 percent shortage and greater than 50 percent shortage. These levels shall be based on supply conditions, including percent reductions in supply, changes in groundwater levels, changes in surface elevation, or other conditions. The shortage levels shall also apply to a catastrophic interruption of supply.	Section 8.3
10632(a)(3)(B)	Suppliers with an existing water shortage contingency plan that uses different water shortage levels must cross reference their categories with the six standard categories.	Section 8.3
10632(a)(4)(A)	Suppliers with water shortage contingency plans that align with the defined shortage levels must specify locally appropriate supply augmentation actions.	Section 8.4
10632(a)(4)(B)	Specify locally appropriate demand reduction actions to adequately respond to shortages.	Section 8.4
10632(a)(4)(C)	Specify locally appropriate operational changes.	Section 8.4
10632(a)(4)(D)	Specify additional mandatory prohibitions against specific water use practices that are in addition to state-mandated prohibitions are appropriate to local conditions.	As a Wholesale Supplier, we cannot set 'mandatory prohibitions'.
10632(a)(4)(E)	Estimate the extent to which the gap between supplies and demand will be reduced by implementation of the action.	As a Wholesale Supplier, we cannot set 'mandatory prohibitions'.

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10632.5	The plan shall include a seismic risk assessment and mitigation plan.	Sections 8.4.3
10632(a)(5)(A)	Suppliers must describe that they will inform customers, the public and others regarding any current or predicted water shortages.	Sections 8.4 and 8.5
10632(a)(5)(B) 10632(a)(5)(C)	Suppliers must describe that they will inform customers, the public and others regarding any shortage response actions triggered or anticipated to be triggered and other relevant communications.	Sections 8.4 and 8.5
10632(a)(6)	Retail supplier must describe how it will ensure compliance with and enforce provisions of the WSCP.	Does not apply to Valley Water as a Wholesale Supplier.
10632(a)(7)(A)	Describe the legal authority that empowers the supplier to enforce shortage response actions.	Section 8.7
10632(a)(7)(B)	Provide a statement that the supplier will declare a water shortage emergency Water Code Chapter 3.	Section 8.7
10632(a)(7)(C)	Provide a statement that the supplier will coordinate with any city or county within which it provides water for the possible proclamation of a local emergency.	Section 8.7
10632(a)(8)(A)	Describe the potential revenue reductions and expense increases associated with activated shortage response actions.	Section 8.8
10632(a)(8)(B)	Provide a description of mitigation actions needed to address revenue reductions and expense increases associated with activated shortage response actions.	Section 8.8
10632(a)(8)(C)	Retail suppliers must describe the cost of compliance with Water Code Chapter 3.3: Excessive Residential Water Use During Drought.	Not applicable to Valley Water as a Wholesale Supplier.
10632(a)(9)	Retail suppliers must describe the monitoring and reporting requirements and procedures that ensure appropriate data is collected, tracked, and analyzed for purposes of monitoring customer compliance.	Not applicable to Valley Water as a Wholesale Supplier.
10632(b)	Analyze and define water features that are artificially supplied with water, including ponds, lakes, waterfalls, and fountains, separately from swimming pools and spas.	Not applicable to Valley Water as a Wholesale Supplier.
10635(c)	Provide supporting documentation that Water Shortage Contingency Plan has been, or will be, provided to any city or county within which it provides water, no later than 30 days after the submission of the plan to DWR.	Sections 8.12, Chapter 10 and Appendix B
10632(c)	Make available the Water Shortage Contingency Plan to customers and any city or county where it provides water within 30 after adopted the plan.	Section 10.1

CHAPTER 10 – PLAN ADOPTION, SUBMITTAL, AND IMPLEMENTATION

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10631(e)(2)	Wholesale suppliers shall describe specific demand management measures listed in code, their distribution system asset management program, and supplier assistance program.	Sections 9.1 and 9.2
10631(e)(1)	Retail suppliers shall provide a description of the nature and extent of each demand management measure implemented over the past five years. The description will address specific measures listed in code.	Not applicable to Valley Water as a Wholesale Supplier.
10608.26(a)	Retail suppliers shall conduct a public hearing to discuss adoption, implementation, and economic impact of water use targets (recommended to discuss compliance).	Not applicable to Valley Water as a Wholesale Supplier.
10621(b)	Notify, at least 60 days prior to the public hearing, any city or county within which the supplier provides water that the urban water supplier will be reviewing the plan and considering amendments or changes to the plan. Reported in Table 10-1.	Section 10.1
10621(f)	Each urban water supplier shall update and submit its 2020 plan to the department by July 1, 2021.	Section 10.1
10642	Provide supporting documentation that the urban water supplier made the plan and contingency plan available for public inspection, published notice of the public hearing, and held a public hearing about the plan and contingency plan.	Section 10.1 and Appendices B and G
10642	The water supplier is to provide the time and place of the hearing to any city or county within which the supplier provides water.	Section 10.1
10642	Provide supporting documentation that the plan and contingency plan has been adopted as prepared or modified.	Section 10.1 and Appendix G
10644(a)	Provide supporting documentation that the urban water supplier has submitted this UWMP to the California State Library.	Section 10.1 and Appendix G
10644(a)(1)	Provide supporting documentation that the urban water supplier has submitted this UWMP to any city or county within which the supplier provides water no later than 30 days after adoption.	Section 10.1 and Appendix G
10644(a)(2)	The plan, or amendments to the plan, submitted to the department shall be submitted electronically.	Section 10.1
10645(a)	Provide supporting documentation that, not later than 30 days after filing a copy of its plan with the department, the supplier has or will make the plan available for public review during normal business hours.	Section 10.1 and Appendix G

Water Code Section	Summary as Applies to UWMP	2020 UWMP Location (Optional column for Agency Review use)
10645(b)	Provide supporting documentation that, not later than 30 days after filing a copy of its water shortage contingency plan with the department, the supplier has or will make the plan available for public review during normal business hours.	Section 10.1
10621(c)	If supplier is regulated by the Public Utilities Commission, include its plan and contingency plan as part of its general rate case filings.	Not applicable to Valley Water.
10644(b)	If revised, submit a copy of the water shortage contingency plan to DWR within 30 days of adoption.	Section 8.10

Improving Health and Wellness through Access to Nature

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Key Words: Environmental Health

Related APHA Policy Statements

APHA Policy Statement 20079 – Building a Public Health Infrastructure for Physical Activity Promotion. In: American Public Health Association
APHA Policy Statement 200712 – Toward a Healthy, Sustainable Food System. In: American Public Health Association
APHA Policy Statement 200619 – Urgent Call for a Nationwide Public Health Infrastructure and Action to Reverse the Obesity Epidemic
APHA Policy Statement 2005-8 – Supporting the WHO Global Strategy on Diet, Physical Activity and Health
APHA Policy Statement 2004-04 – Creating Policies on Land Use and Transportation Systems that Promote Public Health
APHA Policy Statement 2004-02 – Reducing Health Disparities in People with Disabilities through Improved Environmental Programmatic and Service Access.
APHA Policy Statement 2000-13 – Resolution on Overweight in Childhood. In: American Public Health Association
APHA Policy Statement 7228 – Land Use Policy Statement
APHA Policy Statement 7629 – Environmental Health Planning

Abstract

People of all ages and abilities enjoy higher levels of health and well-being when they have nature nearby in parks, gardens, greenways, naturalized schoolyards and playgrounds, and natural landscaping around homes and workplaces. Access to nature has been related to lower levels of mortality and illness, higher levels of outdoor physical activity, restoration from stress, a greater sense of well-being, and greater social capital. Natural elements that promote well-being include trees, diverse vegetation, local biodiversity, water features, parks, natural playscapes, and community and school gardens. The integration of nature into towns and cities has secondary benefits that contribute to better health and more sustainable societies. Trees and vegetation capture carbon dioxide and mitigate global warming. They buffer noise, offer shade, reduce the effect of heat islands, and trap particulates and other airborne pollutants. Parks and other natural areas filter groundwater, reduce stormwater runoff, and prevent combined sewer overflows, improving the functioning of both public and private water systems. In order to promote people-nature contact across American communities, public health practitioners and policymakers should form alliances with parks departments, planning and design departments, housing agencies, greening and garden organizations, cooperative extension services, school districts, and nature centers to prioritize access to natural areas, productive landscapes, and other green spaces for people of all ages, income levels, and abilities. Moreover, public health officials, physicians, nurse practitioners, and other health professionals should advise patients and the public at large about the benefits of green exercise, personal and community gardening, and nature-based play and recreation.

Problem Statement

This policy statement describes connections between nearby green spaces and cognitive, emotional, and physical health outcomes. As listed above, several APHA policy statements have addressed obesity, physical activity, and diet-related diseases, as well as land use and health connections. This policy statement strongly reaffirms APHA's commitment to those policies and thus does not aim to restate their content. It focuses attention on evidence that access to safe, natural settings has a positive influence on health and well-being, increasing the likelihood of walking and other forms of physical activity, fostering social connections, and reducing stress and illness.[1–8] Many studies also show that direct experiences of nature form a foundation for a sense of stewardship and active care for the environment, which is vital for the protection of a life-sustaining planet now and in the future.[9] People evolved in natural environments, but urbanization, the industrialization of agriculture, and a shift to sedentary indoor lifestyles have distanced many people from nature, depriving them of the positive health benefits associated with natural light, green views, local biodiversity, natural landscapes, and gardens and parks near their homes, schools, and workplaces. Low-income and ethnic communities are most likely to lack these resources.[10] A rapidly growing body of evidence establishes that protecting and restoring access to nature in different spheres of people's lives, among those of all ages, social groups, and abilities, can alleviate some of the most important problems in public health, including obesity, stress, social isolation, injury, and violence.

Proposed Recommendations Statement

People of all ages and abilities enjoy higher levels of health and well-being when they have nature nearby in parks, gardens, greenways, naturalized schoolyards and playgrounds, and natural landscaping around homes and workplaces.[11] Access to nature has been related to lower levels of mortality and illness, higher levels of outdoor physical activity, restoration from stress, a greater sense of well-being, and greater social capital. Natural elements that promote well-being include trees, diverse vegetation, local biodiversity, water features, parks, natural playscapes, community gardens, and school gardens. Given the importance of contact with nature for well-being, the American Public Health Association supports the protection and restoration of nature in the environments where people live, work, and play, at every scale from building sites to large regional park systems and ecologically sustainable rural areas.

In addition to having a direct positive impact on well-being, the integration of nature into towns and cities and the protection of biodiversity in rural areas have many secondary benefits that contribute to better health and more sustainable societies. Trees and vegetation capture carbon dioxide and mitigate global warming. They buffer noise, offer shade, reduce the effects of heat islands, and trap particulates and other airborne pollutants. Parks and other natural areas filter groundwater, reduce stormwater runoff, and prevent combined sewer overflows, improving the functioning of both public and private water systems. Areas planted with trees and vegetation absorb water and control erosion, reducing the frequency and severity of floods and mitigating desertification. Natural areas create habitats for biodiversity, providing not only a reservoir of resources for human use but also places where people can feel a sense of wonder and connection with the larger web of life.

The American Public Health Association has been active on environmental issues that affect the public's health, including chemical, radiation, and biological agents. APHA has also been active on issues related to the built environment and the important role of land use planning in shaping health. There has been a strong emphasis on injury prevention, including violence and its interplay with transportation and community environments. This policy statement adds to this body of work by reviewing the mounting scientific evidence connecting nature to social and physical well-being and identifying specific recommendations to influence future policy and related actions at multiple levels of government.

The nature-health connection: People of all ages are more likely to use open spaces with trees, increasing opportunities for social interaction and for children's supervised play.[12] Teenagers value natural areas as places for adventurous play and hanging out with friends, younger children value them for exploration and creative social play, and older adults value these areas for walking, enjoying scenery, and meeting friends.[8,13] Among public housing residents, having green views predicts a stronger sense of community and more social ties with neighbors; also, greener surroundings are associated with a greater sense of safety as well as fewer reported crimes.[14,15] Participation in community gardens is associated with reduced social isolation, a sense of collective efficacy, and increased social networks, social involvement, and neighborhood attachment.[16–19]

In large epidemiological studies that control for income and other potential confounding factors, living in green areas or in walking distance of green spaces is associated with lower levels of mortality and morbidity.[20–24] Benefits include lower rates of heart disease, stroke, obesity, stress, and depression and better coping with stressful events. Specifically, contact with nature contributes to the regulation of the hypothalamic pituitary adrenal system,[25] and walking in nature is associated with better immune system functioning in the form of increased numbers of natural killer (NK) cells, increased NK cell activity, and increased levels of intracellular anti-cancer proteins.[26] Living in a greener environment is positively related to better perceived health in addition to fewer health problems.[27]

Although physical activity promotes health whether people engage in it indoors or outdoors, a number of studies connect "green exercise" outdoors in nature with greater feelings of enjoyment, energy, vitality, restoration, and self-esteem.[22,28,29] The presence of nature has this effect independent of levels of physical activity and social interaction. Several factors affect park use and appreciation, including distance, size, attractiveness, and level of biodiversity.[6] Across socioeconomic levels, people are more likely to walk and be physically active if they live near parks.[6,30] Well-designed greenways and trails encourage walking, active recreation, and active commuting to work. Finally, research has shown a 2-fold increase in fruit and vegetable consumption among people who participate in community gardens relative to those who do not garden.[17]

When people have trees and other vegetation around their homes, they report a greater sense of well-being and greater satisfaction with where they live.[31,32] Many studies associate access to nature through trees, water features, neighborhood parks, or forested areas with reduced levels of stress, whether stress is measured physiologically or by self-report.[5,8,33,34] When people have green views or spend time outdoors in nature, they perform better on tasks that require focused attention.[5,7] When residents of public housing have views of trees rather than entirely built surroundings, they show greater capacity to cope with stress, better conflict management, and lower levels of family aggression.[8,35,36] In addition, measures derived from a mobile electroencephalographic (EEG) headset indicate that when people move from built urban streets into urban green space, they experience real-time drops in frustration, engagement, and excitement and an increase in meditative calm.[37]

Nature-health connections across the life span and social groups: Research indicates that unstructured outdoor activities in natural areas may improve children's health by increasing physical activity, reducing stress, and reducing symptoms of attention disorders.[38,39] Children show higher levels of physical activity outdoors versus indoors, whether outdoor play areas are built or natural; however, children with access to safe green spaces, park playgrounds, and recreational facilities are more likely to be physically active and have a healthy weight than those who lack these resources.[40] Among low-income children, higher levels of neighborhood greenness are associated with a more stable body mass index.[41] On naturalized school grounds, children are reported to be more physically active.[42] Play in natural areas in childhood is associated with seeking natural environments for restoration and recreation in adulthood and with stewardship behaviors to protect the environment.[9,43] Several studies—spanning the preschool years, middle childhood, and adolescence—associate green views or activity in green spaces with more focused attention, better coping with stressful life events, better moods, higher academic achievement,[44–49] and, among children diagnosed with attention deficit hyperactivity disorder, reduced symptoms.[31,38,39,43] Children have lower levels of asthma when they live in neighborhoods with more trees.[50]

Elderly residents report stronger feelings of well-being when they have a garden view from their apartments.[51] When elders have access to gardening or time to rest in a garden, they show a reduced risk of developing dementia and improved mental functioning.[52,53] Alzheimer's patients who can go into a garden at different times of the day show improved group interaction, reduced agitation, and less wandering.[54] Older adults who spend longer durations of time in a park or perceive health-related benefits from their park activity show reduced blood pressure.[55] Walkable green space is associated with greater longevity in older people.[24]

Patients who have views of nature from their hospital windows have been found to recover from surgery more quickly and need less pain medication.[56] Gardens in hospitals provide patients and their families with the stress-reducing benefits of exposure to nature and spaces for social interaction and exercise.[57] In addition, prisoners with views of nature report less illness.[58] The more nature contact that employees describe in their workplace, the less they report stress and health complaints.[59]

Research demonstrates that contact with nature benefits people of all ages, income groups, and abilities; according to some studies, however, low-income groups show greater health benefits from living near green spaces than high-income groups—perhaps because they are more dependent on local environmental resources.[23] Therefore, ensuring access to green spaces in low-income neighborhoods and for people of all abilities appears a promising approach to reducing health inequalities, increasing longevity, and improving health behaviors.[60,61] In urban areas, higher levels of nearby vegetation and vacant lot greening are associated with fewer aggressive acts against partners and lower rates of crime, including gun assaults, robbery, and burglary.[14,36,62,63] The distribution of green spaces, however, is inequitable, with low-income communities and communities of color often lacking safe, well-landscaped, and well-maintained neighborhood parks.[10,56] For example, schools in low-income minority neighborhoods are less likely to have school gardens, and in particular large, well-resourced gardens, than schools in high-income neighborhoods.[64] In places where people have fewer resources, there is limited access to safe and open green spaces where people can walk, jog, or play with children, and thus there are fewer opportunities to meet daily recommended levels of physical activity.[10,65] More than half of adults with disabilities (impairments and/or activity limitations) do not engage in any leisure-time physical activity, in part because of barriers related to the built and natural environment.[66]

For many indigenous peoples, connection to ancestral lands is essential for well-being. Recognizing the importance of this connection, the United Nations Declaration on the Rights of Indigenous Peoples (www.un.org/esa/socdev/unpfii/documents/DRIPS_en.pdf) specifies their rights to maintain their spiritual relationship with their traditional lands and waters; conserve their medicinal plants, animals, and minerals; and incorporate these practices into their self-determination of their health programs.

Opposing Arguments/Evidence

The research cited above addresses alternative points of view related to the value of green spaces and natural landscaping for health. It might be objected that people with higher levels of income can afford greener neighborhoods with more green resources, higher quality outlets for healthy food and health care, and more opportunities for exercise. To avoid self-selection and the potential confounding effect of income, pioneering research on the relationship between contact with nature and health investigated residents of Chicago public housing who were similar socially and economically and who were assigned to their housing unit by housing authority staff in a process that was effectively random assignment.[12,14,35,67] The only factor that varied was whether the area around their identical buildings was landscaped or covered in asphalt. A similar strategy was used in biomarker research conducted in a public housing area in Scotland.[25] In hospital and prison studies, people with similar health conditions have been assigned by chance to rooms with or without green views.[56,58] Other studies cited here have controlled for the effects of income or other potential confounding factors by comparing access to nature in homogeneous populations or through statistical controls.

A second alternative explanation might be that what matters in these studies is people's level of exercise, regardless of where it occurs. Studies have addressed this alternative point of view in two ways. Several studies that have experimentally compared the effects of exercise indoors or in outdoor built settings with the effects of green exercise in natural areas have shown that people usually report a greater sense of well-being or exhibit greater concentration or restoration after green exercise.[28,29,37] Many studies involve passive exposure to nature through window views or simulated exposure via videos or still images, without the element of physical activity.[5,32,45,49] When people are experimentally exposed to scenes of nature versus built environments, they show decreased stress and report greater well-being. Although images of nature have been found to have positive effects, experiments that manipulate exposure to simulated nature versus actual natural landscapes demonstrate greater recovery from stress and greater feelings of energy after contact with actual nature.[29,68]

The presence of parks may not guarantee their use. Activities in parks may be harmful to safety and well-being if the parks are not heavily used, attract illicit activity, and/or are poorly maintained. Research shows that park programming and relationships with park staff may encourage park use.[69] In addition, understanding neighborhood social contexts where people live is critical to promoting safe outdoor play and positive and lasting engagement with natural environments.[70]

It might be claimed that it is impractical to provide access to nature as a routine component of people's lives, in part because of resource constraints facing municipalities nationwide. Although it may be unrealistic to introduce large park systems where they are lacking or to give everyone wilderness experiences, research shows that a number of affordable actions can make a significant difference for people's health and well-being: planting trees; greening vacant lots and alleys;[62] creating greenways for pedestrians and cyclists; maintaining existing parks; cultivating gardens in communities, schools, hospitals, and group homes; and bringing potted plants indoors.[71]

Action Steps

For all of the above reasons, access to nature represents an important approach to promote healthy and active lifestyles across the life span. Therefore, APHA urges public health practitioners, policymakers, community-based and environmental justice groups, and researchers to advance policy and planning activities that incorporate or address the following objectives in relevant legislation, land-use planning guidance, or public health priority-setting regulations:

1. Land use decisions should prioritize access to natural areas, productive landscapes, and other green spaces for people of all ages, income levels, and abilities.

2. Public health officials, physicians, nurse practitioners, and other health professionals should advise patients and the public at large about the benefits of green exercise, personal and community gardening, and nature-based play and recreation and form alliances with parks departments, departments of planning and design, area aging agencies, greening and garden organizations, cooperative extension services, school districts, and nature centers to increase access to green spaces where people live, work, and play and to raise awareness about their value.

3. Access to green spaces as part of livable communities and urban design and planning should include coordinated and cooperative strategies with the partners described

above and should include universal design that serves the needs of all users seamlessly and as invisibly as possible.

4. Partnerships to increase access to natural settings should include efforts to clean up and green vacant lots and contaminated areas in order to transform areas associated with danger and crime into natural and safe environments.

5. Park and recreation funding, through public agencies or public-private partnerships, should be allocated for the construction of parks, gardens, and other outdoor active environments, as well as safety features (e.g., safe playgrounds) in areas that currently lack these resources, and funds for the maintenance of existing parks should be protected or enhanced. In addition, there should be an emphasis on safe walkable and bikable areas.

6. Tree planting and natural landscaping should be promoted around homes, schools, workplaces, hospitals, prisons, and other institutions. Citizens should be encouraged to participate in creating and sustaining these environmental changes.

7. Schoolyards and the play areas of child care centers should include natural landscaping and school gardens, and contact with nature should be promoted through place-based education and adequate periods for safe outdoor recess.

8. Community gardens should be considered as a primary and permanent open space option as part of master planning efforts; gardens should be developed as part of land planning processes rather than as an afterthought in neighborhood redevelopment projects.

9. Trails and greenways should be networked to provide for safe active commuting to work, school, and services as well as recreation areas, with a particular emphasis on safe walking and biking.

10. Hospitals, assisted living facilities, nursing homes, and institutions that care for people with mental illness should provide access to healing gardens and other natural settings.

11. Education and training programs for health professionals, community leaders, students, educators, community residents, and others should include information on the importance of promoting nature contact through green surroundings and active programs and investing in safety to prevent violence, unintentional injuries, and falls.

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