Chapter 3

HEALTH IMPACTS AND HEALTH RISK REDUCTION STRATEGY

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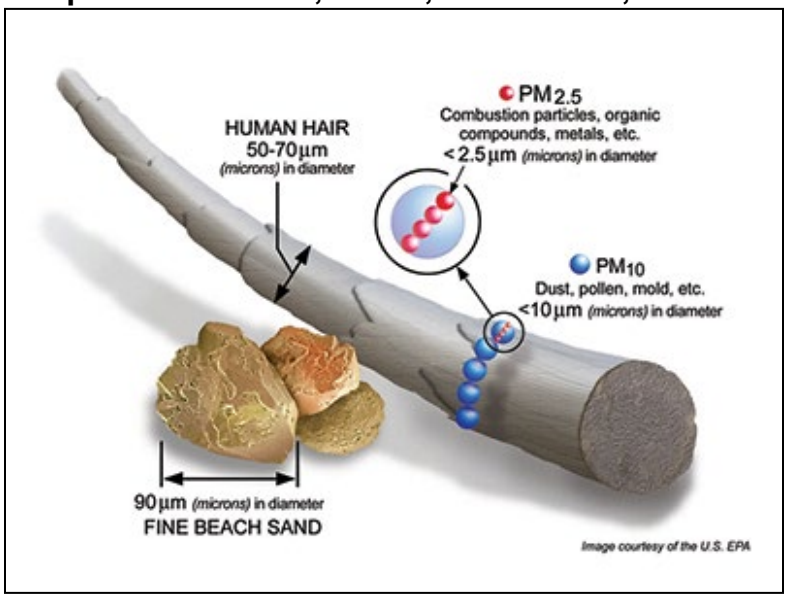
3.1 PM2.5 POLLUTION DEFINED

Particulate matter (PM) is a mixture of solid particles and liquid droplets in the air. PM can be emitted directly into the atmosphere (primary PM), or can form as secondary particulates in the atmosphere through the photochemical reactions of precursors (when precursors are energized by sunlight). Thus, PM is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. PM10 is particulate matter that is 10 microns or less in diameter, and the PM2.5 subset includes smaller particles that are 2.5 microns or less in diameter (Figure 3-1).

Figure 3-1 Comparison of PM10, PM2.5, Human Hair, and Fine Beach Sand



3.1.1 PM2.5 Composition in the Valley

PM2.5 can be emitted directly from an emission source or formed secondarily through chemical reactions in the atmosphere. The nature and formation of PM2.5 in the San Joaquin Valley is highly complex and may consist of components of nitrate, components of sulfur, organic carbon, elemental carbon, soil and more, as summarized in Table 3-1 and shown in Figure 3-2 to Figure 3-5 below. Speciation data collection started in 2000 with the Chemical Speciation Network (CSN), and in 2004, additional speciation monitoring came online with the Interagency Monitoring of Protected Visual Environments (IMPROVE) network.

The analysis of specific elements and compounds from PM2.5 speciation data networks is valuable in understanding the potential effects on monitored air quality from

emissions and activity in the Valley, and how these components have changed in response to air quality improvement strategies implemented in the region.

Speciation data from both the CSN and IMPROVE networks have been instrumental to understanding temporal trends, diurnal trends, and seasonal variations of PM2.5 species throughout the Valley. Seasonal variations of PM2.5 species reflect the importance of using seasonally dependent control strategies like Rule 4901, which mitigates PM2.5 pollution in the San Joaquin Valley due to residential wood burning in the winter. PM2.5 speciation data is also helpful in understanding changes in emissions sources such as windblown dust, vehicle emissions, and burning of biomass, including residential wood burning, open burning, and wildfires.

The complex mixture of PM2.5 is attributable to stationary, mobile, and area-wide sources, as well as naturally occurring emissions. Although the list of components contributing to PM2.5 in the Valley is lengthy, it can be grouped into broader representative categories. Table 3-1 provides summary descriptions of each of these broader categories. Refer to Chapter 2, Section 2.1.1 for more information on the nature and formation of PM2.5.

While not all sites measure all types of components, understanding the different components that make up the total PM2.5 mass is helpful for identifying the sources that contribute to PM2.5 pollution in the Valley. Figure 3-2 to Figure 3-5 show the percentage breakdown of PM2.5 speciation based on the 2020-2022 three-year average, as well as the percentage breakdown of just the highest PM2.5 values (top 10%) measured during that same period at the PM2.5 speciation sites in the Valley.

Understanding the various PM2.5 species, including how each species is formed or emitted, how much each contributes to the Valley's total PM2.5 concentrations, how each is linked to different public health impacts, and emission sources, is of the utmost importance for the development of an effective, health-protecting control strategy.

Table 3-1 Summaries of PM2.5 Components

PM2.5 Components	Description
Organic Carbon	Directly emitted, primarily from combustion sources (e.g., residential wood combustion). In addition, smaller amounts attached to geologic material and road dusts. May also be emitted directly by natural/biogenic sources.
Elemental Carbon	Also called soot or black carbon; formed during incomplete combustion of fuels (e.g., diesel engines).
Soil (Geologic Material)	Road dust and soil dust that are entrained in the air from activity, such as soil disturbance or airflow from traffic.
Trace Metals	Identified as components from soil emissions, or found in other particulates emitted in connection with combustion, engine wear, brake wear, and similar processes. Can also be emitted from fireworks.
Sea Salt	Sodium chloride in sea spray where sea air is transported into the Valley. At this time this component is only measured at the Fresno and Bakersfield sites.
Ammonium Nitrate	Reaction of ammonia and nitric acid, where the nitric acid is formed from nitrogen oxide emissions, creating nitric acid in photochemical processes or nighttime reactions with ozone.
Ammonium Sulfate	Reaction of ammonia and sulfuric acid, where the sulfuric acid is formed primarily from sulfur oxide emissions in photochemical processes, with smaller amounts forming from direct emissions of sulfur.
Combined Water	A water molecule attached to one of the above molecules.

Figure 3-2 Modesto PM2.5 Speciation

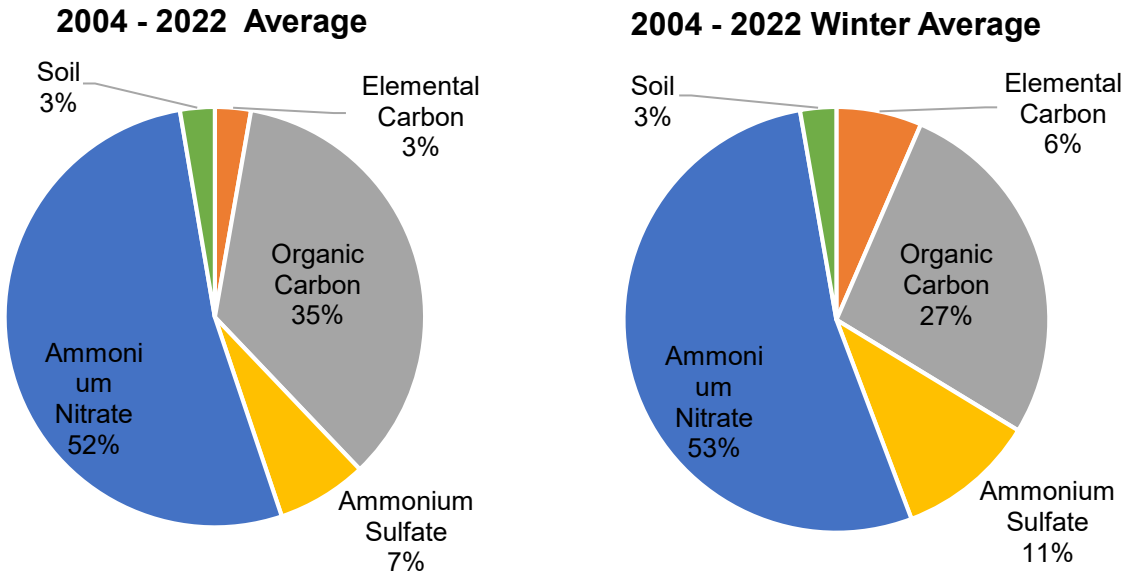


Figure 3-3 Fresno PM2.5 Speciation

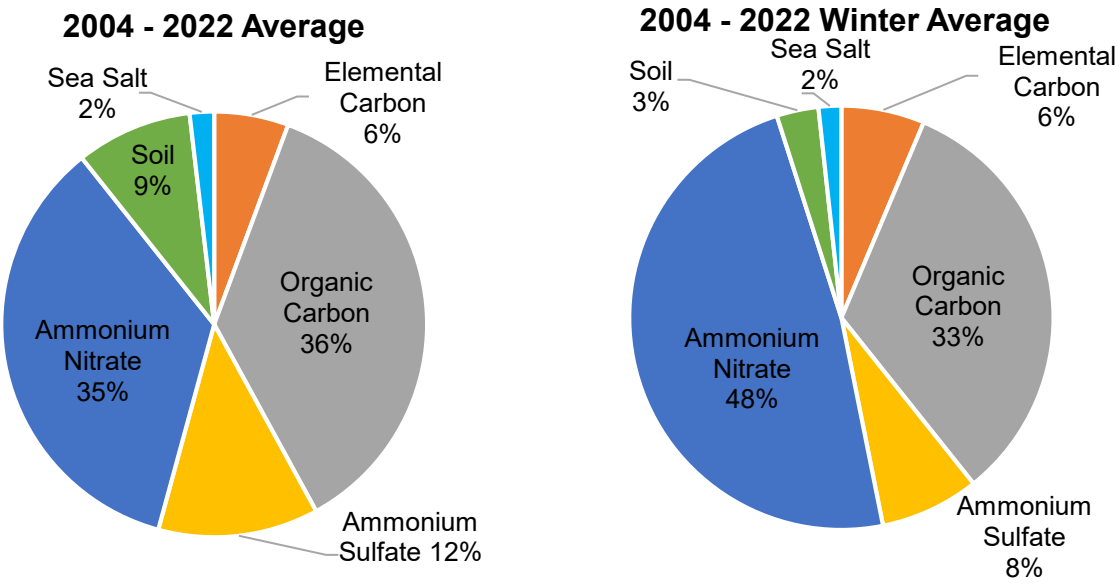


Figure 3-4 Visalia PM2.5 Speciation

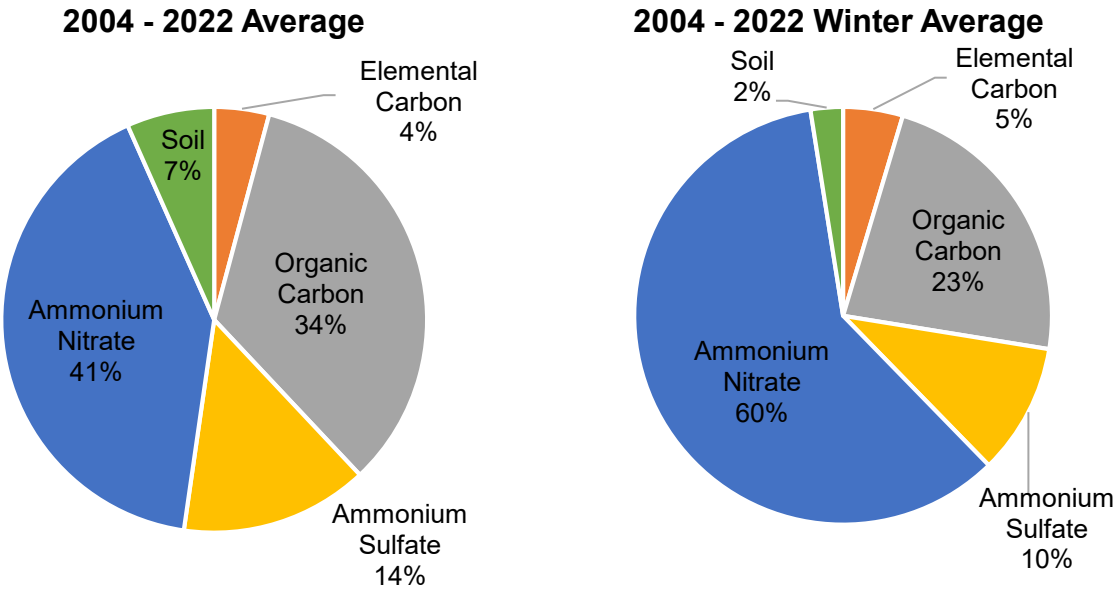
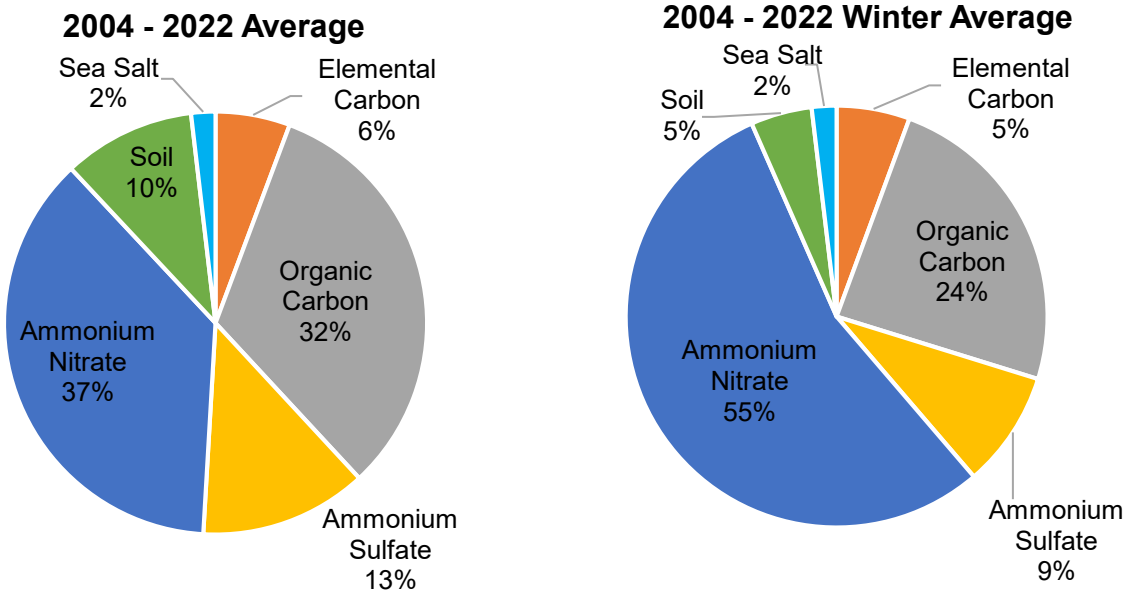


Figure 3-5 Bakersfield PM2.5 Speciation



3.2 HEALTH IMPACTS OF PM2.5

Any particles 10 microns or less are considered respirable, meaning they can be inhaled into the body through the mouth or nose. PM10 can generally pass through the nose and throat and enter the lungs. PM2.5 can be inhaled more deeply into the gas exchange tissues of the lungs, where it can be absorbed into the bloodstream and carried to other parts of the body.[1]

The potential health impacts of particle pollution are linked to the size of the particles, with smaller particles having larger impacts, as smaller particles are able to be absorbed by the alveoli and enter other organs through lung aeration. Numerous studies link PM2.5 to a variety of health problems, including aggravated asthma, increased respiratory symptoms (irritation of the airways, coughing, difficulty breathing), decreased lung function in children, development of chronic bronchitis, irregular heartbeat, non-fatal heart attacks, increased respiratory and cardiovascular hospitalizations, lung cancer, type 2 diabetes, and premature death.[2] Children, older adults, and individuals with heart or lung diseases are the most likely to be affected by PM2.5.[3] Many studies have quantified and documented the health benefits of attaining the U.S. Environmental Protection Agency (EPA) air quality standards for PM. The specific impacts of PM2.5 and supporting research studies are further discussed in the sections below.

In addition to affecting human health, air pollution also affects the health of the natural environment. In cases such as smoke produced from wildfires, PM2.5 can be transported from sources hundreds of miles away to contribute to visibility problems at remote locations, such as the Sierra Nevada mountain range and associated national parks. As fine particulate matter settles out of the air, it can make lakes and streams acidic, change an ecosystem's nutrient balance, and affect ecosystem diversity. PM2.5 can affect vegetation by damaging foliage, disrupting the chemical processes within plants, reducing light adsorption, and disrupting photosynthesis. As the Valley progresses toward attainment of EPA's human-health-based PM2.5 standards, there will also be less harmful impacts to the surrounding natural environment.

3.3 HEALTH RISK REDUCTION STRATEGY

As discussed in Chapter 1, the EPA National Ambient Air Quality Standards (NAAQS or standards) are health-protective air quality standards set by the US EPA Administrator. NAAQS for different pollutants are set by EPA based on technical recommendations from the EPA Clean Air Scientific Advisory Committee (CASAC). CASAC formulates

[1] Xing, Yu-Fei, et al. *The Impact of PM2.5 on the Human Respiratory System*. Journal of Thoracic Disease. January 2016. Retrieved from: www.ncbi.nlm.nih.gov/pmc/articles/PMC4740125/

[2] Cong Liu, Changyuan Yang, Yaohui Zhao, Zongwei Ma, Jun Bi, Yang Liu, Xia Meng, Yafeng Wang, Jing Cai, Haidong Kan, Renjie Chen. *Associations between long-term exposure to ambient particulate air pollution and type 2 diabetes prevalence, blood glucose and glycosylated hemoglobin levels in China*. Environment International, Volumes 92–93. 2016. Pages 416-421, ISSN 0160-4120. Retrieved from: https://doi.org/10.1016/j.envint.2016.03.028

[3] EPA. Research on Health Effects from Air Pollution. Retrieved from: https://www.epa.gov/air-research/research-health-effects-air-pollution

their advice for air quality standards after a comprehensive review of scientific studies on air pollution and health impacts. These standards are the primary driving force for new emissions controls that result in air quality improvements and health benefits to Valley residents. In the conventional planning process for attaining these standards, success in protecting public health is defined by whether the standards are met at all air monitors. In effect, the reduction in PM2.5 mass, which shows progress toward attainment of the standard, serves as the surrogate for population exposure and risk.

NAAQS, as currently established, are essentially *mass-based* standards. In the case of PM2.5, the current standards do not account for particle size distribution, chemical species composition, surface area, and other factors of health risk. In contrast, recent health-science research has substantially deepened knowledge of air pollutant health risk beyond the current Clean Air Act (CAA) framework and EPA standards. There is a growing recognition within the scientific community that the NAAQS alone can be incomplete measures of public exposure to air pollution. Thus, while the CAA, NAAQS, and state implementation plan (SIP) process is motivated by public health, the process alone does not fully address public health impacts of ambient air pollution. To fully address potential public health benefits, an attainment strategy can use a more comprehensive, multidimensional population exposure assessment approach that goes beyond ambient mass measurements.[4]

The District Governing Board adopted the Health Risk Reduction Strategy (HRRS) to prioritize protection of public health by maximizing public health improvements resulting from the District's attainment strategies and related initiatives. The HRRS works in parallel with the District's other strategies to minimize cumulative population exposure to air pollution and the corresponding regional health risk.

3.3.1 Background of the Health Risk Reduction Strategy

As a response to mounting epidemiological evidence that PM2.5 was more harmful than PM10, EPA established a PM2.5 NAAQS in 1997 to accompany the previously established PM10 NAAQS. PM10 occurs at larger mass concentrations than PM2.5, so the shift to PM2.5 somewhat conflicted with the time-tested toxicological precept of "the dose (mass) makes the poison." Particulate inhalation studies have found that the smaller PM2.5 particles penetrate more deeply into the lungs, where particles more effectively avoid immune system defenses. In spite of decreasing levels of air pollution over the last decade, recent epidemiological studies have found correlations between adverse health effects and exposure to air pollution, particularly exposure to PM2.5 pollution and mortality at levels below the current NAAQS.[5] Toxicological analyses of PM2.5 identified chemical species that acted differentially to promote respiratory and

[4] Lippmann M, Chen L-C, Gordon T, Ito K, Thurston GD. (2013). *National Particle Component Toxicity (NPACT) Initiative: Integrated Epidemiologic and Toxicologic Studies of the Health Effects of Particulate Matter Components*. Research Report 177. Health Effects Institute, Boston, MA. Retrieved from: https://www.healtheffects.org/publication/national-particle-component-toxicity-npact-initiative-integrated-epidemiologic-and

[5] Dominici F, Schwartz J, Di Q, Braun D, Choirat C., Zanobetti A. (2019). *Assessing Adverse Health Effects of Long-Term Exposure to Low Levels of Ambient Air Pollution: Phase 1.* Research Report 200. Health Effects Institute, Boston, MA. Retrieved from: https://www.healtheffects.org/system/files/dominici-rr-200-report.pdf

cardiovascular inflammation. While it was unclear at that time which PM2.5 chemicals were the most harmful, the scientific consensus was that the health risks stemmed from the chemicals rather than the particle mass themselves.

In the years since the first PM2.5 NAAQS was established, scientists have conducted numerous studies that have identified which chemical species of PM2.5 are most harmful and have pinpointed their sources.[6] Combustion and non-combustion sources have been found to produce fine particles of different toxicities.[7] PM2.5 exposure may also relate to an increased risk of cardiac arrhythmias in both adults and adolescents.[8] This smaller-is-more-dangerous phenomenon parallels the previous discovery regarding the higher toxicity of PM2.5 particles compared to larger and heavier PM10 particles. In each case, the dose-makes-the-poison assumption governing the NAAQS for carbon monoxide, lead, ozone, and the other criteria pollutants does not apply to particulates.

Addressing the complexity of health risks posed by particulate pollution has been a motivating factor in the development and application of the HRRS. Rather than ignore this growing body of scientific knowledge, the District's HRRS seeks to embrace it to the extent possible within the current CAA to maximize public health benefits. In practice, this knowledge provides the District with the necessary scientific foundation for justifying and prioritizing the pollution control measures that are necessary for demonstrating attainment of federal standards. The outcome is stronger and more health-protective plans that reflect the current trajectory of scientific knowledge toward a more complete understanding of population risk from PM2.5 particles.

The NAAQS-SIP process and the HRRS are complimentary strategies, not an either-or scenario. The HRRS should not be interpreted as a zero-sum tradeoff that emphasizes controls on certain forms and sources of high-risk PM2.5 while ignoring others. The current mass-based indicator (micrograms per cubic meter of air) will continue to serve as the final yardstick for PM2.5 attainment and as a surrogate for achieving significant health benefits. A number of the District programs have been influenced by the underlying principles and goals of the HRRS and provide a model of the success and added potential benefits possible under this strategy.

- **The District's residential wood burning emission reduction strategy** includes wood burning curtailments implemented through District Rule 4901 (Wood Burning Fireplaces and Wood Burning Heaters), in conjunction with the District's incentive grant program for fireplace and woodstove change-outs, and robust public education and outreach efforts. This approach is designed to improve public health by reducing toxic wood smoke emissions in Valley neighborhoods during the peak PM2.5 winter season (November through

[6] EPA. (2009). *Integrated Science Assessment for Particulate Matter*: Final Report. Washington, D.C.: EPA/600/R-08/139F. Retrieved from: https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546
[7] Park, M., Joo, H.S., Lee, K. et al. *Differential toxicities of fine particulate matters from various sources*. Sci. Rep 8, 17007. (2018). Retrieved from: https://doi.org/10.1038/s41598-018-35398-0
[8] Fan He PhD, Jeff D. Yanosky ScD, Julio Fernandez-Mendoza PhD, Vernon M. Chinchilli PhD, Laila Al-Shaar PhD, Alexandros N. Vgontzas MD, Edward O. Bixler PhD, and Duanping Liao MD, PhD. (2022). *Acute Impact of Fine Particulate Air Pollution on Cardiac Arrhythmias in a Population-Based Sample of Adolescents: The Penn State Child Cohort*. thttps://www.ncbi.nlm.nih.gov/pmc/articles/PMC9683666/

February), and has proven to be extremely effective in advancing the District's objectives to attain the PM2.5 federal standards and protect public health. Additionally, through the District's Residential Wood Smoke Reduction Program, which is based on Rule 4901, the District has declared and enforced episodic wood burning curtailments, also called "No Burn" days, since 2003. The District's Residential Wood Smoke Reduction Program and District Rule 4901 reduce harmful species of PM2.5 when and where those reductions are most needed, in impacted urbanized areas when the local weather is forecast to hamper particulate matter dispersion.

Commitments in the District's *2018 Plan for the 1997, 2006, and 2012 PM2.5 Standards (2018 PM2.5 Plan)* included rulemaking for Rule 4901 to further lower wood burning curtailment levels, as well as enhancements to the District's incentive grant funding levels, public outreach and education, enforcement, and air quality forecasting programs. In 2019, the District amended Rule 4901 to lower curtailment thresholds for older, higher-polluting wood burning heaters, open hearth fireplaces, and non-registered wood burning heaters in the Hot Spot counties of Madera, Fresno, and Kern. In the remaining Valley counties, the previous curtailment thresholds remain in place. These more stringent curtailment thresholds established in the Hot Spot counties are coupled with increased Fireplace and Woodstove Change-Out Program incentive amounts to cover nearly the entire cost of replacing high polluting wood burning units with natural gas units. To complement the regulatory and incentives changes, the District has implemented an education and outreach campaign to increase public awareness of the health benefits from residential wood smoke reduction program, along with focused rule enforcement efforts in Hot Spot counties and in areas of concern. The District also continues to investigate and employ the latest air quality modeling tools and techniques to support the air quality forecasting component of the program.

In addition, consistent with the District's *2018 PM2.5 Plan*, the District added a contingency provision to Rule 4901 for the 1997, 2006, and 2012 PM2.5 standards. On May 18, 2023 the District amended Rule 4901 to establish a sequence of increasingly stringent contingency Level One and Level Two episodic wood burning curtailment thresholds for all Valley counties that would be triggered 60 days after the issuance of a final determination by EPA, pursuant to 40 CFR §51.1014(a), that the District has failed to meet one of four required trigger elements for any of the PM2.5 NAAQS. Already the most stringent residential wood combustion control strategy in the nation, these contingency provisions further enhance the stringency of the Rule and the District's residential wood smoke reduction program.

- **District grant programs** reach beyond the current CAA NAAQS-SIP process to reduce emissions in advance of or beyond regulations. For example, through the District's popular Clean Green Yard Machine incentive grant program, the District has awarded over $2.7 million for over 15,000 pieces of electric residential lawn and garden equipment, and through the Zero-Emission Landscaping Equipment

Voucher Program, approximately $2.3 million for the replacement of over 924 pieces of gas- and diesel-powered commercial landscape maintenance equipment.[9] Through the Fireplace & Woodstove Change-Out Program (formerly known as the Burn Cleaner Program), the District has replaced approximately 30,000 high-polluting wood burning devices with cleaner alternatives.[10] These grant programs result in a decrease in urban, localized health risks associated with the use of gas-powered equipment and wood burning devices.

- **The District's information and educational programs, such as the Real-Time Air Advisory Network (RAAN),** also contribute to the HRRS. Users of RAAN are able to subscribe to automated mobile device updates, via email or text, when local ozone or PM2.5 concentrations threaten health, allowing users the ability to make informed decisions about when outdoor activities should be limited.[11] Subscribers can better plan outdoor activities for times with the best air quality, reducing potential air quality health risks.

- **The District tracks and sponsors health and PM2.5 research.** As part of the District's HRRS, the District is playing an active role in supporting leading-edge health research focusing on the Valley population. Previously, the District sponsored the first major epidemiological investigation of health effects of air pollution in the Valley, focusing on the populations of Modesto, Fresno, and Bakersfield.[12] The study found that daily exposure to high PM2.5 concentrations was significantly correlated with increased daily hospital and emergency room admission rates for asthma and other respiratory and cardiovascular diseases. To follow up on this study, the District sponsored another epidemiological study to examine which of the chemical species found in Valley PM2.5 are most highly correlated with hospital admission rates. In more detail, the study explored statistical associations between varying concentrations of PM2.5 components (e.g., ammonium nitrate, ammonium sulfate, organic carbon, and elemental carbon) and health outcomes, including emergency department visits and hospitalizations associated with selected cardiovascular and respiratory conditions.

  The District also sponsored a pilot study of PM0.1 (ultrafine particles) in Fresno, where UCSF-Fresno investigated the quantity and spatial distribution of PM0.1 plumes from motor vehicles, lawn care equipment, wood burning, and restaurants.[13] Following this study, the District funded a UC Davis research project to develop a model of PM0.1 population exposure in the Valley based on

[9] As of March 31, 2024
[10] As of March 20, 2024
[11] SJVAPCD. Using the Real-Time Air Quality Advisory Network. (2019). https://www.valleyair.org/Programs/RAAN/documents/RAAN-Users-Guide.pdf
[12] Capitman, J.A., and Tyner, T.R. (2011). *The Impacts of Short-term Changes in Air Quality on Emergency Room and Hospital Use in California's San Joaquin Valley*. California State University, Fresno, Fresno CA. https://chhs.fresnostate.edu/cvhpi/documents/aqr-web.pdf
[13] Capitman, J.A., and Tyner, T.R. (2011). *The Impacts of Short-term Changes in Air Quality on Emergency Room and Hospital Use in California's San Joaquin Valley*. California State University, Fresno, Fresno CA. https://chhs.fresnostate.edu/cvhpi/documents/aqr-web.pdf

previous Valley observational research.[14] PM0.1 exposure was correlated with short- and long-term health effects by making use of the large body of Valley epidemiological data generated by the previous studies described above.

In addition, the District sponsored a project with Providence Engineering to examine differences in exposure to PM2.5 in residential neighborhoods. In this field project, Providence Engineering deployed approximately 30 passive PM samplers in neighborhoods across the Fresno area to provide a better spatial understanding of concentration variation in the urban area.[15] The samples were analyzed later in a laboratory to provide particle size, mass, and speciation estimates, followed by source apportionment analysis. Overall, the project provided the District with a finer understanding of how the health risk of fine particles varies in different urban locations.

The District continues to seek out opportunities to support research that furthers the understanding of PM-related impacts on public health, while also monitoring ongoing external PM-related research.

3.4 TOXICITY OF CHEMICAL SPECIES

PM2.5 particles vary in their toxicity depending on their chemical composition. Recent research into regional variability in the health response to PM2.5 exposure indicates that health outcomes may be influenced by differential toxicity of different PM species.[16] PM2.5 particles are characterized by diverse combinations of chemicals depending on unique regional combinations of meteorology, topography, and pollution sources. In addition to experimental and clinical research that has identified these toxicity differences, epidemiological studies have found regional differences in health impacts despite comparable regional PM2.5 mass exposure.[17] Beyond the intrinsic toxicity of individual chemicals, the unique combinations of chemicals generated by some sources can actually magnify health risk above and beyond what their mass concentrations would suggest.[18]

Many emissions sources evaluated in this Plan are sources of direct (primary) PM2.5 emissions, characterized by unique combinations of chemical species. Other sources emit chemical species such as ammonia and nitrogen oxides (NOx), precursors that contribute to the formation of secondary PM2.5 species. The PM2.5 chemical species

[14] Qi Ying, Jin Lu, Michael Kleeman. *Modeling air quality during the California Regional PM10/PM2.5 Air Quality Study (CPRAQS) using the UCD/CIT source-oriented air quality model – Part III. Regional source apportionment of secondary and total airborne particulate matter*. Atmospheric Environment, Volume 43, Issue 2. (2009). Pages 419-430, ISSN 1352-2310. Retrieved from: https://doi.org/10.1016/j.atmosenv.2008.08.033

[15] Zeng Y. *A comprehensive particulate matter monitoring system and dosimetry-based ambient particulate matter standards*. J Air Waste Manag Assoc. 2006 Apr;56(4):518-29. doi: 10.1080/10473289.2006.10464528. PMID: 16681216.

[16] Kodros JK, Volckens J, Jathar SH, Pierce JR. *Ambient Particulate Matter Size Distributions Drive Regional and Global Variability in Particle Deposition in the Respiratory Tract*. Geohealth. 2018 Oct 17;2(10):298-312. doi: 10.1029/2018GH000145. PMID: 32159003; PMCID: PMC7007101.

[17] Bell, M.L. (2012). *Assessment of the Health Impacts of Particulate Matter Characteristics*. Research Report 161. Boston: MA. Health Effects Institute.

[18] Kelly, F.J. (2006). *Oxidative Stress: Its Role in Air Pollution and Adverse Health Effects*. Occupational Environmental Medicine, 60, 612–616.

categories adopted in the exposure characterization model include elemental carbon (black carbon), organic carbon compounds (OC), metals (elements), ammonium nitrate, ammonium sulfate, and geological material. PM2.5 is regularly speciated at several Valley monitoring sites. The following discussion provides an overview of PM2.5 species and their associated health impacts.

Organic carbon (OC): OC species found in PM2.5 aerosol are generated as primary organic aerosol (POA), predominantly through the combustion of hydrocarbons. Key POA sources include cooking, industrial processes, mobile source exhaust, prescribed burning, tire wear, and wood burning.[19] Secondary organic aerosols (SOA) are formed from the oxidation of motor vehicle hydrocarbons, prescribed burning, wood burning, solvent use, and industrial processes.

OC is recognized as one of the most biologically reactive PM2.5 chemical species categories, with ample evidence of high toxicity found in experimental, clinical, and epidemiological studies. OC, often in combination with metals such as iron, has been shown to generate reactive oxygen species (ROS) that drive several different mechanisms of pulmonary inflammation, including disruption of normal immune system functioning.[20] Alveolar macrophages and epithelial cells are the first cellular responders to inhaled PM2.5 in the respiratory tract and macrophages clear and process inhaled PM2.5.[21] OC and metals have been shown to indirectly stimulate ROS production by macrophages, which are responsible for defending the lungs from pathogens and aerosols, in a process called respiratory burst. When there is excessive ROS production due to exposure to PM2.5, it can lead to damage of key cellular components, oxidative stress, or cell death.[22]

One of the primary OC species categories is polycyclic aromatic hydrocarbons (PAH). PAH species fall into two categories: a high molecular weight fraction and a low molecular weight fraction. The former is found in diesel exhaust and engine oil, and is a significant risk factor for lung cancer.[23] Low molecular weight PAH is found in other hydrocarbon combustion particles and serves as a precursor to the formation of an important OC species category known as quinones. Formed from atmospheric processing of PAH or within the body (in vivo), quinones have been shown to be one of the most important drivers of pulmonary oxidative stress, resulting in a host of negative

[19] EPA. *Air Quality Criteria for Particulate Matter: Final Report*. Washington, D.C.: EPA 600/P-99/002aF-bF. (October 2004).
[20] Ayres, J.G., Borm, P., Cassee, F.R., Castranova, V., Donaldson, K., Ghio, A. … Froines, J. (2008). *Evaluating the Toxicity of Airborne Particulate Matter and Nanoparticles by Measuring Oxidative Stress Potential—A Workshop Report and Consensus Statement*. Inhalation Toxicology 20, 75–99. doi: 10.1080/08958370701665517
[21] Hiraiwa K, van Eeden SF. *Contribution of lung macrophages to the inflammatory responses induced by exposure to air pollutants*. Mediators Inflamm. 2013;2013:619523. doi: 10.1155/2013/619523. Epub 2013 Aug 22. PMID: 24058272; PMCID: PMC3766602.
[22] Traboulsi H, Guerrina N, Iu M, Maysinger D, Ariya P, Baglole CJ. *Inhaled Pollutants: The Molecular Scene behind Respiratory and Systemic Diseases Associated with Ultrafine Particulate Matter*. International Journal of Molecular Sciences. 2017; 18(2):243. Retrieved from: https://doi.org/10.3390/ijms18020243
[23] Landvik, N.E., Gorria, M., Arlt, V.M., Asare, N., Solhaug, A., Lagadic-Gossmann, D., & Holme, J.A. (2007). *Effects of Nitrated-Polycyclic Aromatic Hydrocarbons and Diesel Exhaust Particle Extracts on Cell Signalling Related to Apoptosis: Possible Implications for their mutagenic and Carcinogenic Effects*. Toxicology, 231, 159–174. doi: 10.1016/J.tox.2006.12.009

spillover effects on immune system functioning.[24] Quinone formation via chemical aging of PAH occurs during multi-day winter stagnation events in the Valley. A District-funded clinical study of asthmatic patients in Fresno found that quinone levels in urine correlated with sustained (multi-day) high ambient concentrations of PM2.5 and was accompanied by decreased lung function.[25]

OC in PM2.5 can also act as a promoter of tumor formation. After OC is inhaled into the respiratory tract, it undergoes metabolic activation and produces electrophile epoxides, which then causes DNA damage and chromosomal abnormalities, increasing the risk of cancer.[26]

Elemental carbon (EC): Elemental carbon is found in combustion-based aerosols produced by mobile exhaust (mainly diesel), wood burning, and cooking (especially charbroiling). Compared to OC species, there is limited evidence of comparable impacts on ROS production, pulmonary inflammation, and immune system disruption. For example, EC appears not to be a significant agent for the induction of inflammation in macrophage cells, indicating a significantly lower toxicity level relative to OC species.[27]

Characterization of health effects of elemental carbon from human exposure studies is complicated by the high correlation between EC, OC, and metals emitted by diesel exhaust. Ambient EC concentrations have been associated with an increase in systolic pressure and heart rate variability. In one study, hypertensive rats were exposed to urban-industrial aerosol.[28] EC was found to consistently have the strongest association of any PM2.5 component with changes in heart rate and heart rate variability amongst the hypertensive rats. Blood pressure changes were found to be more influenced by variations in EC than in OC, with the EC1 fraction associated with acute cardiovascular responses.

Metals: A combination of clinical, experimental, and epidemiological studies have implicated several of the metals found in PM2.5 with negative respiratory or cardiovascular outcomes, sometimes in conjunction with the action of OC species. One of the most important is iron because of its ability to catalyze the production of hydrogen peroxide, leading to highly reactive hydroxyl radicals (OH). In turn, these highly reactive chemicals stimulate the production and action of cytokines by macrophages. Cytokines

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[24] Bolton, J., Trush, M.A., Penning, T.M., Dryhurst, G., & Monks, T.J. (2000). *Role of Quinones in Toxicology*. Chemical Research in Toxicology, *13(3)*, 135–160. doi: 10.1021/tx99

[25] Ikeda, A., Vu, K.K.-T., Lim, D., Tyner, T.R., Krishnan, V.V., & Hasson, A.L. (2012). *An Investigation of the Use of Urinary Quinones as Environmental Biomarkers for Exposure to Ambient Particle-Borne Pollutants*. Science of the Total Environment (submitted).

[26] Yifan Wang, Siyao Xiao, Yuhan Zhang, Howard Chang, Randall V. Martin, Aaron Van Donkelaar, Audrey Gaskins, Yang Liu, Pengfei Liu, Liuhua Shi. *Long-term exposure to PM2.5 major components and mortality in the southeastern United States*. Environment International, Volume 158, 2022, 106969, ISSN 0160-4120. Retrieved from: https://doi.org/10.1016/j.envint.2021.106969

[27] Vogel, C.F., Sciullo, E., Wong, P., Kuzmicky, P., Kado, N. & Matsumura, F. (2005). *Induction of Proinflammatory Cytokines and C-Reactive Protein in Human Macrophage Cell Line U937 Exposed to Air Pollution Particulates*. Environmental Health Perspectives 113(11), 1536–1541. doi: 10.1289/ehp.8094

[28] Wagner, J.G., Kamal, A.S., Morishita, M. *et al. PM2.5-induced cardiovascular dysregulation in rats is associated with elemental carbon and temperature-resolved carbon subfractions*. Part Fibre Toxicol 11, 25 (2014). Retrieved from: https://doi.org/10.1186/1743-8977-11-25

are cell-signaling molecules that are critical to normal functioning of the immune system. A recent experimental study examined the impact of iron in silica particles in triggering respiratory toxicity.[29] Compared to silica particles with no iron, silica particles with iron were found to have a significantly greater effect on oxidative stress via hydrogen peroxide production with subsequent stimulus of cytokines by macrophages.

Extensive research relates exposure in metals (particularly nickel and vanadium) in PM2.5 to cardiovascular effects. A national epidemiological study recently found that communities with higher fractions of nickel, vanadium, and EC in their PM2.5 also had higher risk of cardiovascular and respiratory hospitalization.[30] Specifically, cardiovascular hospitalizations were 26% higher in counties with a nickel fraction in the 75th percentile versus counties with nickel in the 25% percentile. In an investigation of the relatively higher association between PM2.5 daily concentrations and daily rates of cardiovascular mortality in New York City, the exceptionally high level of nickel and vanadium resulting from residual oil fly ash used for heating and as fuel for ships were identified as a principle cardiovascular risk factor.[31] In a related study, rats exposed to PM2.5 with high fractions of chromium, iron, and nickel fractions responded with significantly reduced heart rate variability and increased heart rates, each being an indicator of cardiovascular disruption and risk.[32]

Chromium, another metallic component of PM2.5, is typically emitted from combustion processes, metal industries, cement-manufacturing plants, tobacco smoke, and chromium-based automotive catalytic converter pollution. Chromium exposure has been associated with various health conditions, such as nasal septum atrophy and cancer.[33] Chromium can be cancerous to both children and adults.[34] Hexavalent chromium (CrVI), one of the valence states of elemental chromium, is an endocrine disruptor, capable of mimicking or interfering with the body's hormones, or endocrine system.[35]

In conclusion, metals found in PM2.5 produced from combustion of coal, residual oil, diesel fuel, and motor oil are recognized as chemical drivers of cardiovascular and

[29] Premasekharan, G., Nguyen, K., Contreras, J., Ramon, V., Leppert, V.J. & Forman, H.J. (2011). *Iron-Mediated Lipid Peroxidation and Lipid Raft Disruption in Low-Dose Silica-Induced Macrophage Cytokine Production*. Free Radical Biology and Medicine, 51(6), 1184–1194. doi: 10.1016/j.freeradbiomed.2011.06.018

[30] Bell, M.L., Ebisu, K., Peng, R.D., Samet, J.M. & Dominici, F. (2009). *Hospital Admissions and Chemical Composition of Fine Particle Air Pollution*. American Journal of Respiratory Critical Care, 179, 1115–1120. doi: 10.1164/rccm.200808-1240OC

[31] Lippmann, M., Ito, K., Hwang, J-S., Maciejczyk, P., & Chen, L-C. (2006). Car*diovascular Effects of Nickel in Ambient Air*. Environmental Health Perspectives, 114(11), 1662–1669. doi: 10.1289/ehp.9150

[32] Chen, L.C., & Lippmann, M. (2009). *Effects of Metals within Ambient Air Particulate Matter (PM) on Human Health*. Inhalation Toxicology, 21(1), 1–31. doi: 10.1080/08958370802105405

[33] Olivia S. Ryder, Jennifer L. DeWinter, Steven G. Brown, Keith Hoffman, Betsy Frey, Ali Mirzakhalili. *Assessment of particulate toxic metals at an Environmental Justice community*. Atmospheric Environment: X, Volume 6, 2020, 100070, ISSN 2590-1621. Retrieved from: https://doi.org/10.1016/j.aeaoa.2020.100070

[34] Cheng, X., Huang, Y., Zhang, S.P., Ni, S.J. and Long, Z.J. (2018). Charac*teristics, Sources, and Health Risk Assessment of Trace Elements in PM10 at an Urban Site in Chengdu, Southwest China*. Aerosol Air Qual. Res. 18: 357-370. Retrieved from: https://doi.org/10.4209/aaqr.2017.03.0112

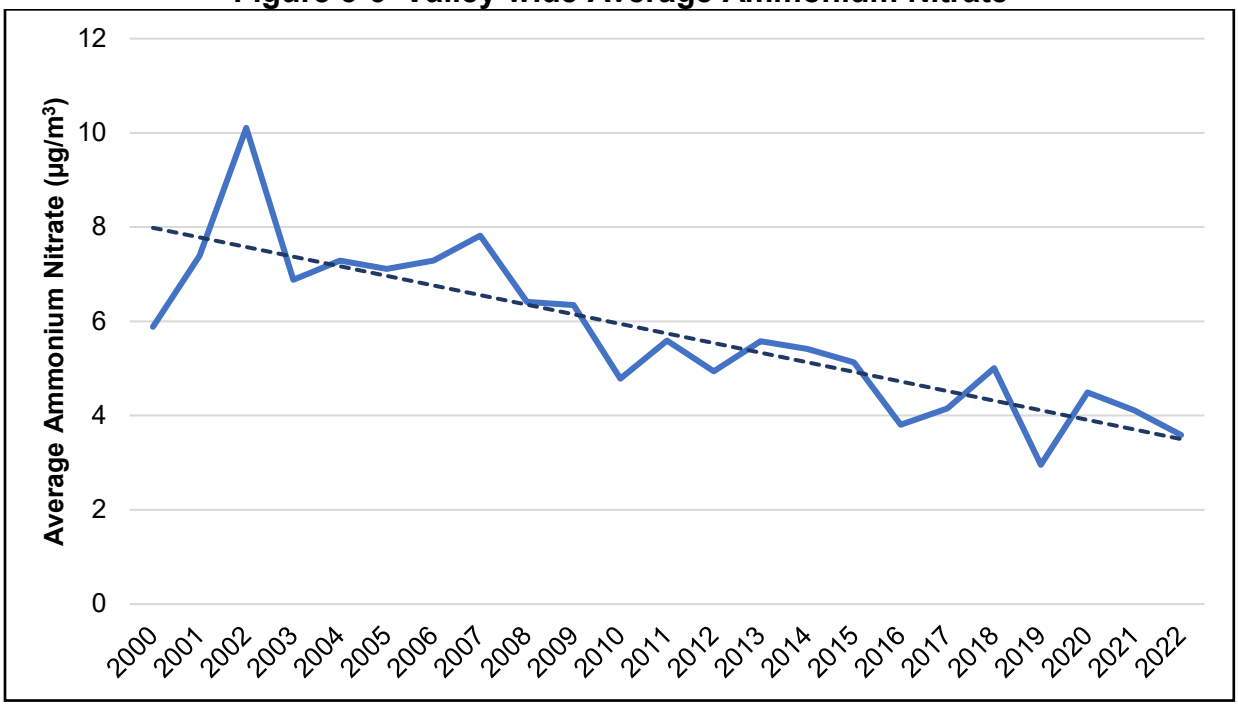
[35] Sakhila K Banu, Jone A Stanley, Kirthiram K Sivakumar, Joe A Arosh, Robert J Taylor, Robert C Burghardt. *Chromium VI − Induced developmental toxicity of placenta is mediated through spatiotemporal dysregulation of cell survival and apoptotic proteins*. Reproductive Toxicology, Volume 68, 2017, Pages 171-190, ISSN 0890-6238. Retrieved from: https://doi.org/10.1016/j.reprotox.2016.07.006

respiratory morbidity and mortality. This has led some researchers to conclude that regional differences in U.S. cardiovascular mortality that cannot be explained by differences in average daily PM2.5 concentrations are likely to be caused by regional differences in coal combustion and resultant exposure to metals and OC.[36] Children are the most vulnerable to toxic metallic elements in the natural environment, and exposure can lead to learning disabilities, memory and attention deficits, and/or psychiatric complications.[37]

Ammonium nitrate: Ammonium nitrate is classified as a secondary inorganic species of PM2.5, formed by atmospheric reactions between two precursors: ammonia and nitric acid. Prior to this reaction, nitric acid generally originates from the chemical processing of nitrogen oxides (NOx), largely from fuel combustion during multiday stagnation events.

Figure 3-6 shows the Valley-wide average ammonium nitrate trend between 2002 and 2022. Overall, average ammonium nitrate concentrations have decreased over the past 20 plus years, largely as a result of the District's comprehensive emissions reduction strategy.

Figure 3-6 Valley-wide Average Ammonium Nitrate



The relative toxicity of ammonium nitrate is an important issue given its substantial mass contribution to regional PM2.5. The oral toxicity of nitrate is very low, with an

[36] Lippmann M, Chen L-C, Gordon T, Ito K, Thurston GD. (2013). *National Particle Component Toxicity (NPACT) Initiative: Integrated Epidemiologic and Toxicologic Studies of the Health Effects of Particulate Matter Components*. Research Report 177. *Health Effects Institute*, Boston, MA.
[37] Lin, Y.C., Li, Y.C., Amesho, K.T.T., Chou, F.C. and Cheng, P.C. (2020). *Filterable $PM_{2.5}$, Metallic Elements, and Organic Carbon Emissions from the Exhausts of Diesel Vehicles*. Aerosol Air Qual. Res. 20: 1319–1328. Retrieved from: https://doi.org/10.4209/aaqr.2020.02.0081

LD50 (dose causing death for 50% of the exposed subjects) reported to be two thirds that of table salt. This raises the question as to whether other factors intrinsic to inhalation could lead to health effects at considerably lower exposure concentrations. As seen in the case of OC species, the most compelling evidence of species toxicity is built on a foundation of experimental, clinical, and epidemiological research. In particular, epidemiological studies draw their inferences only from statistical associations between exposure variables and health outcomes. Uncovering the actual mechanisms of harm, therefore, requires further isolation of mechanisms through experimental and clinical research.

In the case of ammonium nitrate, evidence of toxicity is largely limited to epidemiological research alone. For example, a recent epidemiological study of traffic air toxics and pre-term birth in Los Angeles found statistical associations between nitrate mass, PAH, and several other air pollutants and the increased likelihood of pre-term birth.[38] The authors point to other experimental studies that identified very high oxidative stress potential resulting from PAHs, metals, and other OC species collected from Los Angeles traffic sources as being the likely mechanism for pre-term birth. They conclude by emphasizing the need to further study the links between pre-term birth and PAH exposure.

In another study, PM2.5 from ammonium nitrate and ammonium sulfate was found to be associated with increased prevalence of myocardial infarctions and coronary artery disease. The study found that ammonium nitrate was associated with 15% increased odds of coronary artery disease and 36% increased odds of myocardial infarctions.[39]

Ammonium sulfate: Ammonium sulfate (sulfate) is also classified as a secondary inorganic species. It is formed when sulfuric acid, itself a product of oxidation of sulfur, reacts with ammonia. Fossil fuel combustion is the primary source of sulfate in the Valley, but unlike nitrate, mass concentrations of sulfate are not appreciably different in cold and hot seasons.

Research findings regarding the toxicity of sulfate are comparable to that of nitrate; however, some studies suggest that sulfate may be more harmful than nitrate.[40] The acidity of sulfur compounds in PM2.5 is more harmful because the acidity makes transition metals in particles more bioavailable, leading to oxidative stress when inhaled

[38] Wilhelm, M., Ghosh, J.K., Su, J., Cockburn, M., Jerrett, M. & Ritz, B. (2011). *Traffic-Related Air Toxics and Preterm Birth: A Population-Based Case-Control Study in Los Angeles County, California*. Environmental Health 10: 89. doi: 10.1186/1476-069X-10-89

[39] Slawsky E, Ward-Caviness CK, Neas L, Devlin RB, Cascio WE, Russell AG, Huang R, Kraus WE, Hauser E, Diaz-Sanchez D, Weaver AM. *Evaluation of $PM_{2.5}$ air pollution sources and cardiovascular health*. Environ Epidemiol. 2021 May 20;5(3):e157. doi: 10.1097/EE9.0000000000000157. PMID: 34131618; PMCID: PMC8196100.

[40] Masselot P, Sera F, Schneider R, Kan H, Lavigne É, Stafoggia M, Tobias A, Chen H, Burnett RT, Schwartz J, Zanobetti A, Bell ML, Chen BY, Guo YL, Ragettli MS, Vicedo-Cabrera AM, Åström C, Forsberg B, Íñiguez C, Garland RM, Scovronick N, Madureira J, Nunes B, De la Cruz Valencia C, Hurtado Diaz M, Honda Y, Hashizume M, Ng CFC, Samoli E, Katsouyanni K, Schneider A, Breitner S, Ryti NRI, Jaakkola JJK, Maasikmets M, Orru H, Guo Y, Valdés Ortega N, Matus Correa P, Tong S, Gasparrini A. *Differential Mortality Risks Associated With PM2.5 Components: A Multi-Country, Multi-City Study*. Epidemiology. 2022 Mar 1;33(2):167-175. doi: 10.1097/EDE.0000000000001455. PMID: 34907973; PMCID: PMC7612311.

and systemic health effects in the human body.[41] Simply put, when sulfate is inhaled deep into the lungs, it creates an acidic environment that promotes the absorption of metallic elements of PM2.5.[42] Effects of ammonium sulfate exposure can increase asthmatic and inflammatory responses, and inhalation of ammonium nitrate and ammonium sulfate may induce adverse effects on sperm motility and motion, thus affecting male fertility.[43]

Geological: Winter season and annual average PM2.5 found in the Valley contains a very small fraction of species that are termed *crustal*, i.e., having their origins in the earth's crust. Suspended dust consists mainly of oxides of aluminum, silicon, calcium, titanium, iron, and other metal oxides. The precise combination of these components depends on the geology, industrial processes, and agricultural processes of the area. The geologic fraction of PM2.5 found in the Valley makes a relatively small contribution to overall PM2.5 mass and, by itself, has relatively low toxicity. Silica, a component of geological material, can be deposited in the lungs and destroy respiratory function, whereas heavy metals, which are also found in geological material, can accumulate in the blood and bones and cause damage to the nervous system.[44]

3.5 PARTICLE SIZE AND DEPOSITION

Particle size has a significant bearing on bodily deposition, net exposure, and corresponding health risk, even within the PM2.5 size fraction. Inhaled PM2.5 deposits deep within pulmonary tissues, interacts and activates local cells, and modifies endogenous structures.[45] Key metrics for deposition assessment include the percentage of inhaled particles that remain deposited and not exhaled (known as the deposition fraction) and the location where particles are deposited within the body.[46] Within the PM2.5 size range, particles less than 0.1 microns (PM0.1) and greater than 10 microns are least likely to be exhaled, and thus have higher deposition fractions.[47]

The biological pathways by which PM2.5 promotes cardiovascular disease are summarized below in Figure 3-7. PM2.5 components, specifically metals and organic

[41] Maciejczyk P, Chen L-C, Thurston G. The Role of Fossil Fuel Combustion Metals in $PM_{2.5}$ Air Pollution Health Associations. *Atmosphere*. 2021; 12(9):1086. https://doi.org/10.3390/atmos12091086

[42] Yifan Wang, Siyao Xiao, Yuhan Zhang, Howard Chang, Randall V. Martin, Aaron Van Donkelaar, Audrey Gaskins, Yang Liu, Pengfei Liu, Liuhua Shi. *Long-term exposure to PM2.5 major components and mortality in the southeastern United States*. Environment International, Volume 158, 2022, 106969, ISSN 0160-4120. Retrieved from: https://doi.org/10.1016/j.envint.2021.106969

[43] Jeong-Won Bae, Hong Ju Kwon, So-Hye Kim, Lei Ma, Hobin Im, Eungyung Kim, Myoung Ok Kim, Woo-Sung Kwon. *Inhalation of ammonium sulfate and ammonium nitrate adversely affect sperm function*, Reproductive Toxicology, Volume 96, 2020, Pages 424-431, ISSN 0890-6238. Retrieved from: https://doi.org/10.1016/j.reprotox.2020.08.009

[44] Yifan Wang, Siyao Xiao, Yuhan Zhang, Howard Chang, Randall V. Martin, Aaron Van Donkelaar, Audrey Gaskins, Yang Liu, Pengfei Liu, Liuhua Shi. *Long-term exposure to PM2.5 major components and mortality in the southeastern United States*. Environment International, Volume 158, 2022, 106969, ISSN 0160-4120. Retrieved from: https://doi.org/10.1016/j.envint.2021.106969

[45] Sanjay Rajagopalan, Sadeer G. Al-Kindi, Robert D. Brook. *Air Pollution and Cardiovascular Disease: JACC State-of-the-Art Review*. Journal of the American College of Cardiology, Volume 72, Issue 17, 2018, Pages 2054-2070, ISSN 0735-1097. Retrieved from: https://doi.org/10.1016/j.jacc.2018.07.099

[46] International Commission on Radiological Protection [ICRP]. (1995). *Human Respiratory Tract Model for Radiological Protection*. ICRP Publication 66. *Annals of the ICRP 24,* 1–3.

[47] EPA. *Air Quality Criteria for Particulate Matter*: Final Report. (2004, October). Washington, D.C.: EPA 600/P-99/002aF-bF.

species, are mediators of oxidative stress, which instigates a local inflammatory response, even when exposure to PM2.5 may be short-term.[48] This is indicative of heightened impact from long-term exposure to PM2.5.

Deposition of very small particles in the alveolar region of the lungs results in the delivery of their chemicals into the bloodstream where they promote cardiovascular disruption and immune system sensitization.[49] These chemicals can trigger heart attacks and premature death among individuals with pre-existing heart conditions.[50] Extremely small particles can also be absorbed into the brain via the nasal tract, bypassing the protection provided by the blood-brain barrier.[51] The effects of particles deposited primarily in the tracheobronchial region center on respiratory function.[52]

Particle deposition and associated health risk is magnified by exercise in several ways. First, during exercise, higher minute ventilation occurs, leading to higher inhalation of pollutants.[53] Second, breathing harder means that particles are more likely to penetrate the alveolar region of the lungs where absorption into the bloodstream occurs; PM2.5 was observed to primarily deposit in the head, pulmonary and tracheobronchial regions.[54] A 2018 study found that lung function improved with physical activity, but this beneficial effect decreased when air pollution concentrations were higher.[55] Another study from 2021 found that during moderate to vigorous activity, individuals increase their risk of inhaling harmful pollutants, resulting in reduced lung function.[56] Although physical exercise is recommended to improve health, air pollution levels should be considered, as there are several adverse health effects from PM inhalation.[57]

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[48] Sanjay Rajagopalan, Sadeer G. Al-Kindi, Robert D. Brook. *Air Pollution and Cardiovascular Disease: JACC State-of-the-Art Review*. Journal of the American College of Cardiology, Volume 72, Issue 17, 2018, Pages 2054-2070, ISSN 0735-1097. Retrieved from: https://doi.org/10.1016/j.jacc.2018.07.099

[49] Delfino, R.J., Sioutas, C., & Malik, S. (2005). *Potential Role of Ultrafine Particles in Associations between Airborne Particle Mass and Cardiovascular Health*. Environmental Health Perspectives 113(8), 934–946.

[50] Nel A. (2005). *Air Pollution-Related Illness: Effects of Particles*. Science, 308(5723), 804–806. doi: 10.1126/science.1108752

[51] Oberdorster, G., Sharp, Z., Atudorei, V., Elder, A., Gelein, R., Kreyling, W., & Cox, C. (2004). *Translocation of Inhaled Ultrafine Particles to the Brain*. Inhalation Toxicology, 16(6-7), 437–445. doi: 10.1080/08958370490439597

[52] EPA. *Integrated Science Assessment for Particulate Matter*: Final Report. (2009). Washington, D.C.: EPA/600/R-08/139F.

[53] Pasqua LA, Damasceno MV, Cruz R, Matsuda M, Garcia Martins M, Lima-Silva AE, Marquezini M, Saldiva PHN, Bertuzzi R. *Exercising in Air Pollution: The Cleanest versus Dirtiest Cities Challenge*. Int J Environ Res Public Health. 2018 Jul 17;15(7):1502. doi: 10.3390/ijerph15071502. PMID: 30018189; PMCID: PMC6069042.

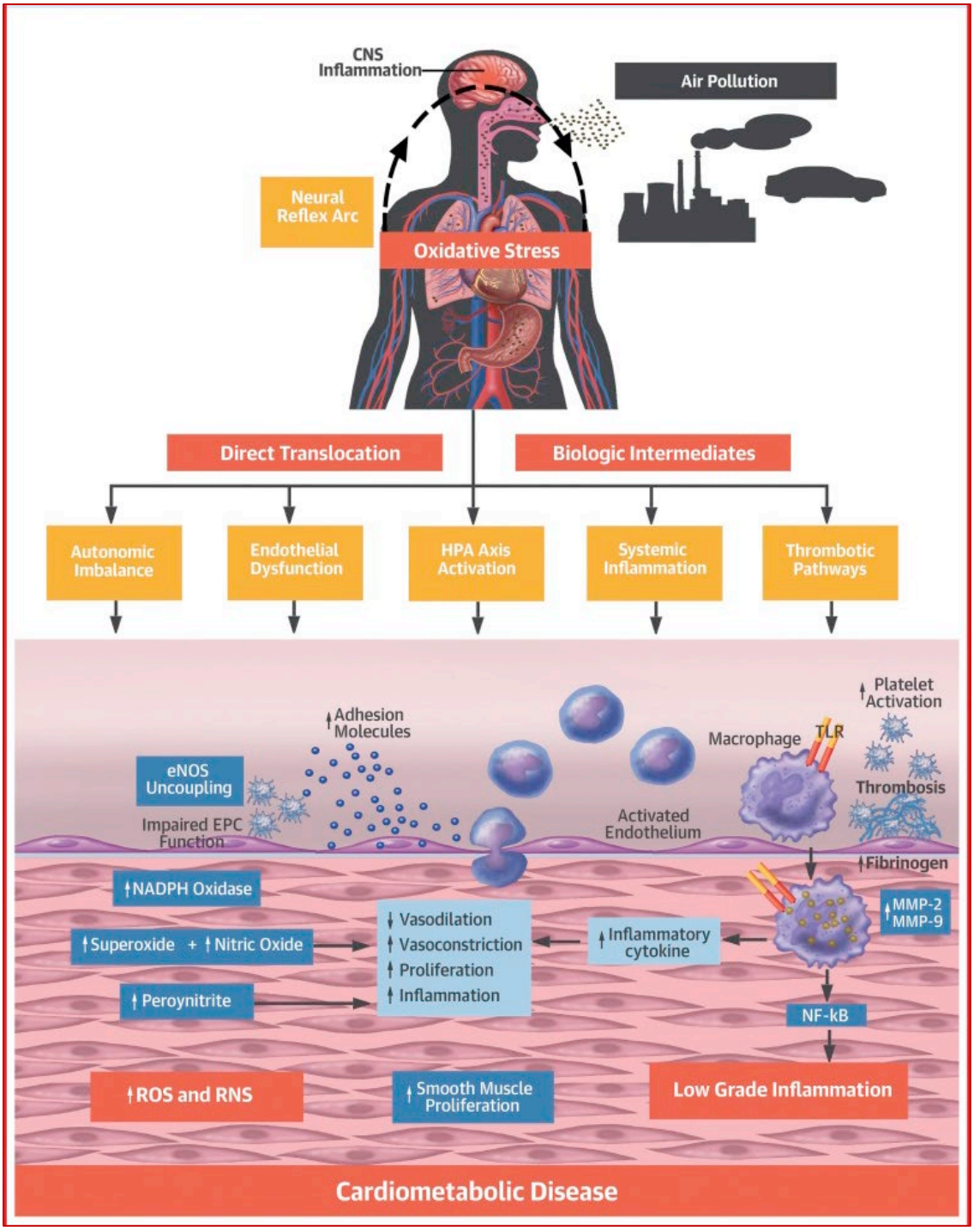
[54] N. Manojkumar, B. Srimuruganandam, S.M. Shiva Nagendra. *Application of multiple-path particle dosimetry model for quantifying age specified deposition of particulate matter in human airway*. Ecotoxicology and Environmental Safety, Volume 168, 2019, Pages 241-248, ISSN 0147-6513. Retrieved from: https://doi.org/10.1016/j.ecoenv.2018.10.091

[55] Laeremans, Michelle & Dons, Evi & Avila-Palencia, Ione & Carrasco-Turigas, Glòria & Orjuela, Juan & Anaya Boig, Esther & Cole-Hunter, Tom & Nazelle, Audrey & Nieuwenhuijsen, Mark & Standaert, Arnout & Van Poppel, Martine & De Boever, Patrick & Int Panis, Luc. (2018). *Black Carbon Reduces the Beneficial Effect of Physical Activity on Lung Function*. Medicine & Science in Sports & Exercise. 50. 1. 10.1249/MSS.0000000000001632.

[56] Lovinsky-Desir S, Jung KH, Montilla M, Quinn J, Cahill J, Sheehan D, Perera F, Chillrud SN, Goldsmith J, Perzanowski M, Rundle A, Miller R. *Locations of Adolescent Physical Activity in an Urban Environment and Their Associations with Air Pollution and Lung Function*. Ann Am Thorac Soc. 2021 Jan;18(1):84-92. doi: 10.1513/AnnalsATS.201910-792OC. PMID: 32813558; PMCID: PMC7780976.

[57] Pasqua LA, Damasceno MV, Cruz R, Matsuda M, Garcia Martins M, Lima-Silva AE, Marquezini M, Saldiva PHN, Bertuzzi R. E*xercising in Air Pollution: The Cleanest versus Dirtiest Cities Challenge*. Int J Environ Res Public Health. 2018 Jul 17;15(7):1502. doi: 10.3390/ijerph15071502. PMID: 30018189; PMCID: PMC6069042.

Figure 3-7 Biological Pathways whereby PM2.5 Promotes Cardiovascular Events[58]

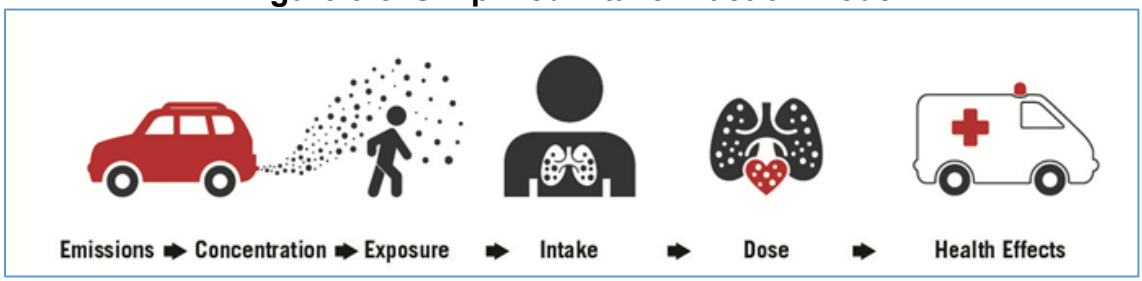


[58] Sanjay Rajagopalan, Sadeer G. Al-Kindi, Robert D. Brook. *Air Pollution and Cardiovascular Disease: JACC State-of-the-Art Review*. Journal of the American College of Cardiology, Volume 72, Issue 17, 2018, Pages 2054-2070, ISSN 0735-1097. Retrieved from: https://doi.org/10.1016/j.jacc.2018.07.099

3.6 POPULATION PROXIMITY AND INTAKE FRACTION

Estimating total exposure and net health risk from a given source of PM2.5 requires that population proximity and population density be considered in addition to the source's toxicity and contribution to the regional PM2.5 emissions inventory. In addition to factors governing net deposition of inhaled particles reviewed above, net population exposure from the source in question is also shaped by the number of exposed individuals who inhale the emissions, and the duration of exposure in conjunction with aerosol concentration levels (see Figure 3-8). Known as the intake fraction, this measure of population exposure is defined empirically as the pollutant mass inhaled divided by the mass emitted.[59] Intake fraction is useful in connecting emissions to health risk because the mass inhaled is a better indicator of health risk than the mass emitted or airborne concentration. Two different pollutant sources with very comparable emission rates of the same pollutant can nonetheless have significantly different intake fractions depending on the surrounding population density. For example, sources of PM2.5 located in rural areas may have an intake fraction that is 10 to 100 times smaller than a comparable source located within a densely populated city.

Figure 3-8 Simplified Intake Fraction Model



The relevance of the intake fraction concept can be seen in a 2018 study on transportation-related air pollution impacts on disadvantaged communities in southern California.[60] The study investigated how spatially targeted emission-reductions could reduce environmental injustice by examining how reducing emissions of a known carcinogen (PM2.5 from diesel engines) would affect total exposure, exposure efficiency, exposure inequality, and exposure injustice. This study used intake fraction as a measurement for exposure efficiency. Furthermore, this study also examined two types of emission reduction strategies that are in use in hundreds of cities worldwide: low-emission zones and truck re-routing. The study found that targeting emission reductions in certain locations can yield disproportionately large advantages for impact, efficiency, equity, and justice.

In a similar study from 2017, intake fraction was used to quantify how emissions from different regions proportionally contribute to human exposure of both primary and secondary particulate matter species over four seasons for twenty-five regions in the United States. It was found that sulfate inhalation occurs over larger distances than

[59] Marshall, J.D., & Nazaroff, W.W. (2004, October). *Using Intake Fraction to Guide CARB Policy Choices: The Case of Particulate Matter*. Unpublished California Air Resources Board Report.
[60] Nam P Nguyen and Julian D Marshall. (2018). *Impact, efficiency, inequality, and injustice of urban air pollution: variability by emission location*. Environmental Resource Letters, 13 024002 DOI 10.1088/1748-9326/aa9cb5.

other particulate matter species, regardless of the season. The study also found that approximately 75% of inhalation occurs within 50km for all seasons and pollutants, increasing in distance by approximately 20% in the wintertime, demonstrating that emission reductions during the winter will have large impacts on health improvement.[61]

In another study, intake fraction was used to examine emissions-to-exposure for wildfires. This study found that not only were localized communities near large fires exposed to wildfire smoke, but exposure to wildfire smoke can extend long distances and affect distant larger urban areas.[62] PM2.5 concentrations are influenced by weather conditions, smoke dispersion, fuel type, and fire behavior; therefore, smoke impacts will be different for each wildfire. This study highlighted the importance of quantifying air quality impacts from wildfires to develop strategies to protect public health with the expected increase of fire season in the Western United States due to climate change.

3.7 SUMMARY OF HEALTH IMPACTS AND HEALTH RISK REDUCTION STRATEGY

Understanding the results of any health risk reduction strategy is critical to assessing the overall value and success of that strategy. As the District continues to develop air quality attainment plans in future years to address the increasingly more stringent NAAQS, the District will evaluate health benefits for Valley residents resulting from adopted air quality attainment plans.

[61] Carmen Lamancusa, Fatema Parvez, Kristina Wagstrom. *Spatially resolved intake fraction estimates for primary and secondary particulate matter in the United States*. Atmospheric Environment, Volume 150, 2017, Pages 229-237, ISSN 1352-2310. Retrieved from: https://doi.org/10.1016/j.atmosenv.2016.11.010

[62] Kathleen M. Navarro, Ricardo Cisneros, Susan M. O'Neill, Don Schweizer, Narasimhan K. Larkin, and John R. Balmes. *Air-Quality Impacts and Intake Fraction of PM2.5 during the 2013 Rim Megafire*. Environmental Science & Technology 2016 *50* (21), 11965-11973. September 21, 2016. DOI: 10.1021/acs.est.6b02252