

APPENDIX G

Health Impacts of Local Air Pollution

Stockton CERP

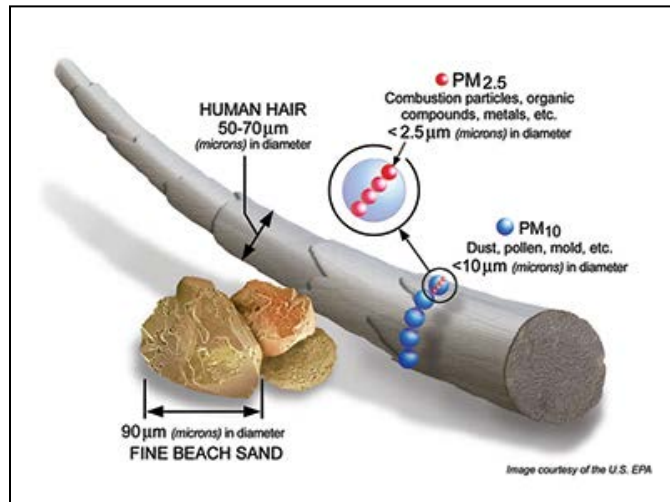
San Joaquin Valley Air Pollution Control District

HEALTH IMPACTS OF LOCAL AIR POLLUTION

PARTICULATE MATTER

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 PM_{2.5} subset includes smaller particles that are 2.5 microns or less in diameter (Figure G-1).

Figure G-1 Visual Comparison of PM₁₀, PM_{2.5}, Human Hair, and Fine Beach Sand



NATURE AND FORMATION OF PM_{2.5}

The nature and formation of PM_{2.5} in the San Joaquin Valley (Valley) is highly complex. Significant differences in regional natural environments and the relative contribution of emissions in various regions across the Valley means that local monitoring and pollution control strategy efforts like AB 617 are important for understanding and reducing pollution in impacted communities like Stockton. Differences within PM_{2.5} itself, directly-emitted PM_{2.5} versus secondary PM_{2.5} forming in the atmosphere through series of chemical reactions, adds to the complexity inherent in emission reduction efforts.

PM_{2.5} can be composed of any material that has a diameter of 2.5 microns or less. PM_{2.5} can be emitted directly as primary PM_{2.5} from various sources or formed secondarily through chemical reactions in the atmosphere. Naturally occurring emissions from biogenic sources, such as plants, can also add to the formation of

PM2.5. The resulting ambient PM2.5 mixture can include aerosols (fine airborne solid particles and liquid droplets) consisting of components of nitrates, sulfates, elemental carbons, organic carbon compounds, acid aerosols, trace metals, geological materials, and more.

PM2.5 SPECIES IN STOCKTON

PM2.5 in Stockton comprised of many species that contribute to the total PM2.5 mass, as summarized in Table G-1, below. This complex mixture is attributable to fmobile, stationary, and area-wide sources, as well as naturally occurring emissions. Although the list of species contributing to PM2.5 in Stockton is lengthy, it can be grouped into larger representative categories. The following is a brief description of how each of these larger species categories are formed and emitted into the atmosphere.

Table G-1 Summaries of PM2.5 Species

PM2.5 Species	Description
Organic carbon	Directly emitted, primarily from combustion sources (e.g. residential wood combustion). Also, 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).
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 having been emitted in connection with combustion from 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.
Secondary organic aerosol	Secondary particulates formed from photochemical reactions of organic carbon.
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.

HEALTH IMPACTS OF PM

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.

The potential health impacts of particle pollution are linked to the size of the particles, with the smaller particles having larger impacts. 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, and premature death. Children, older adults, and individuals with heart or lung diseases are the most likely to be affected by PM2.5. 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.

Understanding various PM2.5 species, including how each species is formed, how much each contributes to the Valley's total PM2.5 concentrations, and how each is linked to different public health impacts, is of the utmost importance for the development of an effective, health-protecting emissions control strategy. For example, ammonium nitrate is estimated to comprise about 40% of the Valley's annual average PM2.5 concentrations, but it is generally regarded as having relatively low toxicity compared to other PM2.5 species, such as organic or elemental carbon.

UNDERSTANDING IMPACTS THROUGH LOCAL HEALTH STUDIES

The District plays an active role in funding leading edge health research focusing on the Valley population. 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.¹ 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, elemental carbon) and health outcomes, including emergency department visits and hospitalizations associated with selected cardiovascular and respiratory conditions.

¹ Capitman, J.A., & 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*. Fresno, CA: Central Valley Health Policy Institute for the San Joaquin Valley Air Pollution Control District.

The District also sponsored a pilot study of PM_{0.1} (ultrafine particles) in Fresno, where UCSF-Fresno investigated the quantity and spatial distribution of PM_{0.1} plumes from motor vehicles, lawn care equipment, wood burning, and restaurants. Following this study, the District then funded a UC Davis research project to develop a model of PM_{0.1} population exposure in the Valley based on previous Valley observational research. PM_{0.1} exposure was correlated with short- and long-term health effects by making use of the large body of Valley epidemiological data that has been generated by the previous studies described above.

In addition, the District sponsored a project with Providence Engineering to conduct a study examining differences in exposure to PM_{2.5} 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. 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. Through the implementation of the CERP and beyond, District will continue to seek out and fund research opportunities that further the understanding air pollution impacts on public health.

TOXICITY OF CHEMICAL SPECIES

PM_{2.5} particles vary in their toxicity depending on their chemical composition. PM_{2.5} particles are characterized by a widely diverse combination 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 PM_{2.5} mass exposure.² 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.³

Many emissions sources evaluated in this CERP are sources of direct (primary) PM_{2.5} emissions characterized by a unique combination of chemical species. Other sources emit chemical species such as ammonia and nitrogen oxides (NO_x), precursors that contribute to the formation of secondary PM_{2.5} species. The PM_{2.5} chemical species categories adopted in the exposure characterization model include elemental carbon (black carbon), organic carbon compounds (OC), metals (elements), ammonium nitrate, ammonium sulfate, and geological. PM_{2.5} in Stockton will be regularly speciated throughout the implementation of the CERP and Community Air Monitoring Plan. The following discussion provides an overview of PM_{2.5} species and their associated health impacts.

² Bell, M.L. (2012). *Assessment of the Health Impacts of Particulate Matter Characteristics*. Research Report 161. Boston: MA. Health Effects Institute.

³ Kelly, F.J. (2006). Oxidative Stress: Its Role in Air Pollution and Adverse Health Effects. *Occupational Environmental Medicine*, 60, 612–616.

Organic carbon (OC): OC species found in PM_{2.5} 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.⁴ 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 of PM_{2.5} 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.⁵ In addition, OC and metals have been shown to indirectly stimulate ROS production by macrophages, which are cells responsible for defending the lungs from pathogens and aerosols.

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.⁶ 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 spillover effects on immune system functioning.⁷ 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 PM_{2.5} and was accompanied by decreased lung function.⁸

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

⁴ U.S. Environmental Protection Agency [EPA]. (2004, October). *Air Quality Criteria for Particulate Matter: Final Report*. Washington, D.C.: EPA 600/P-99/002aF-bF.

⁵ 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

⁶ 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

⁷ 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

⁸ 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)..

in macrophage cells, indicating a significantly lower toxicity level relative to OC species.⁹ A recent study of PM 0.1-based exposure of EC in mice found modest cardiovascular effects. Pulmonary inflammation was noted but only at high doses beyond normal ambient concentrations.¹⁰ A recent study in Mexico City found an association between exposure levels of EC and lung function decrements among asthmatic and non-asthmatic children.¹¹

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. Exposure to EC is a PM_{2.5} risk factor, although there is more evidence to date that other chemical species, e.g. metals and OC, found in these particles are the primary drivers of negative health effects.

Metals: A combination of clinical, experimental, and epidemiological studies have implicated several of the metals found in PM_{2.5} 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 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.¹² 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 PM_{2.5} to cardiovascular effects. A national epidemiological study recently found that communities with higher fractions of nickel, vanadium, and EC in their PM_{2.5} also had higher risk of cardiovascular and respiratory hospitalization.¹³ 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 PM_{2.5} 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

⁹ Vogel, C.F., Sciallo, 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

¹⁰ Vesterdal, L.K., Folkmann, J.K., Jacobsen, N.R., Sheykhzade, M., Wallin, H., Loft, S., & Møller, P. (2010). Pulmonary Exposure to Carbon Black Nanoparticles and Vascular Effects. *Particle and Fibre Toxicology* 7:33. doi: 10.1186/1743-8977-7-33

¹¹ Barraza-Villarreal, A., Escamilla-Núñez M.C., Hernández-Cadena L., Texcalac-Sangrador. J.L., Sienra-Monge, J.J., Del Río-Navarro, B., Cortez-Lugo, M., Sly, P.D., & Romieu, I. (2011). Elemental Carbon Exposure and Lung Function in Schoolchildren from Mexico City. *European Respiratory Journal*, 38, 548–552. doi: 10.1183/09031936.00111410

¹² 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

¹³ 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

identified as a principle cardiovascular risk factor.¹⁴ 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.¹⁵

In conclusion, metals found in PM2.5 produced from combustion of diesel fuel, coal, residual oil, and motor oil are recognized as chemical drivers of cardiovascular and 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.¹⁶

Ammonium nitrate: Ammonium nitrate is classified as a secondary inorganic species (not directly emitted) primary source of PM2.5, and it does not contain carbon. Nitrate is 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.

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 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 from statistical associations between exposure variables and health outcomes only. 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.¹⁷ 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

¹⁴ Lippmann, M., Ito, K., Hwang, J-S., Maciejczyk, P., & Chen, L-C. (2006). Cardiovascular Effects of Nickel in Ambient Air. *Environmental Health Perspectives*, 114(11), 1662–1669. doi: 10.1289/ehp.9150

¹⁵ 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

¹⁶ 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.

¹⁷ 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

emphasizing the need to further study the links between pre-term birth and PAH exposure.

One experimental study was found that explicitly looked for toxic mechanisms driven by ammonium nitrate.¹⁸ The study exposed rats to high concentrations of nitrate (70 to 420 $\mu\text{g}/\text{m}^3$) in combination with EC. After exposure, animals were sacrificed and a necropsy was performed, followed by a range of tests for pathological impacts between the control (non-exposed) and exposed groups. The authors did not find abnormalities that could be tied to the experimental exposure to nitrate alone or in combination with EC. This absence of experimental evidence for mechanisms of pathology for inhaled ammonium nitrate is consistent with its low oral toxicity.

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. Mass concentrations of sulfate are significantly lower than for nitrate in the Valley, averaging from 10% to 11% of PM_{2.5} mass on an annual average basis. Fossil fuel combustion is the primary source of sulfate in the Valley, but globally, coal combustion is the primary source. 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. Oral toxicity is low and it is approved as a food additive by the US Food and Drug Administration and the European Union. One study¹⁹ examined the response of 20 non-smoking subjects to four-hour exposure sessions in chambers containing 500 $\mu\text{g}/\text{m}^3$ of sulfate aerosol, a concentration over two orders of magnitude above ambient levels in the Valley. Pulmonary function tests were performed to assess the response of these exposures. No significant changes in pulmonary function or bronchial reactivity were observed immediately after the individual exposures or 24 hours after exposure. In an experimental study that also exposed rats to 500 $\mu\text{g}/\text{m}^3$ of sulfate for four to eight months, modest pulmonary impacts were noted.²⁰ After four months, cellular immunologic responsiveness was not impaired, but physiologic changes were detected, including enlargement of bronchial epithelial (surface) cells and in alveolar size.

For each of these studies, the modest health impacts observed at very high exposure levels are consistent with the low intrinsic toxicity of sulfate. This is consistent with results of a review of the epidemiological and toxicological research on sulfate.²¹ Researchers found that PM sulfate was a weaker indicator of health risk than PM_{2.5}

¹⁸ Cassee, F., Arts, J.H., Fokkens, P.H., Spoor, S.M., Boere, A.J., van Bree, L., & Dormans, J.A. (2002). Pulmonary Effects of Ultrafine and Fine Ammonium Salts Aerosols in Healthy and Monocrotaline-Treated Rats Following Short-Term Exposure. *Inhalation Toxicology*, 14(12), 1215–1229. doi: 10.1080/08958370290084872

¹⁹ Kulle, T.J., Sauder, L.R., Shanty, F., Kerr, H.D., Ferrell, B.P., Miller, W.R., & Milman, J.H. (1984). Sulfur Dioxide and Ammonium Sulfate Effects on Pulmonary Function and Bronchial Reactivity in Human Subjects. *American Industrial Hygiene Association Journal*, 45(3), 156–161. ISSN:1542-8125. doi: 10.1080/15298668491399569

²⁰ Smith, L.G., Busch, R.H., Buschbom, R.L., Cannon, W.C., Loscutoff, S.M., & Morris, J.E. (1989). Effects of Sulfur Dioxide or Ammonium Sulfate Exposure, Alone or Combined, for 4 or 8 Months on Normal and Elastase-Impaired Rats. *Environmental Research* 49(1), 60-78. doi: 10.1016/S0013-9351(89)80022-2

²¹ Reiss, R., Anderson, E.L., Cross, C.E., Hidy, G., Hoel, D., McClellan, R., Moolgavkar, S. (2007). Evidence of Health Impacts of Sulfate-and Nitrate-Containing Particles in Ambient Air. *Inhalation Toxicology*, 19(5), 419-449. doi: 10.1080/08958370601174941

mass. Because sulfate is correlated with PM_{2.5} mass, this result is inconsistent with sulfate having a strong health influence. The study concluded that the epidemiologic and toxicologic evidence provide little or no support for a causal association of sulfate and health risk at ambient concentrations.

Geological: Winter season and annual average PM_{2.5} 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. This coarse fraction—PM 2.5-10—contains a much higher fraction, as do particles beyond the PM₁₀ size category. 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, and agricultural processes of the area. Geological material typically consists of 5% to 15% PM particles.

Other researchers examined the respiratory inflammation potential of PM_{2.5} soil dust from windblown dust and vehicle-generated particles from unpaved roads, taken from nine different sites in the western U.S.²² None of the sites were located in the Valley. Cultured human epithelial cells were exposed and then were assessed for their release of cytokines known to be triggered by oxidative stress. PM_{2.5} from five of the sites was found to be benign, three of the sites demonstrated measurable cytokine response, and PM_{2.5} from one site was found to be highly reactive. Endotoxin, a potentially reactive bio-aerosol that is often found in PM, was not found to be a contributing factor to the variations in inflammatory potential.

Although not technically a geologic species, respirable road dust (RRD) has been recognized and analyzed as a separate form of PM_{2.5} that has relevance to exposure characterization. In this context, RRD is defined as PM less than 2.5 microns in diameter that is deposited along paved roadways as a result of roadway breakdown, tire wear, brake wear, deposition of exhaust-related particles, and other anthropogenic sources. Speciation analysis²³ of RRD in southern California identified over 100 organic compounds including n-alkanes, n-alkanoic acids, n-alkenoic acids, n-alkanals, n-alkanols, benzoic acids, benzaldehydes, polyalkylene glycol ethers, PAH, oxy-PAH, steranes, hopanes, natural resins, and other compound classes. This relatively toxic mix of OC species is coincident with a range of metals associated with motor vehicle exhaust and component wear. RRD particles are re-suspended by passing traffic, leaf blowers, and other sources for possible inhalation by individuals in or near the roadway.

To conclude, the geologic fraction of PM_{2.5} found in the Valley makes a relatively small contribution to overall PM_{2.5} mass and, by itself, has relatively low toxicity. RRD, while not of geologic origins, has been reviewed here because of its relevance to subsequent exposure characterization of sources.

²² Veranth, J., Rielly, C.A., Veranth, M.M., Moss, T.A., Langelier, C.R., Lanza, D.L., & Yost, G.S. (2004). Inflammatory Cytokines and Cell Death in BEAS-2B Lung Cells Treated with Soil Dust, Lipopolysaccharide, and Surface-Modified Particles. *Toxicological Science* 82(1), 88–96. doi: 10.1093/toxsci/kfh24

²³ Rogge, W. F., Hildemann, L. M., Mazurek, M. A., Cass, G. R. and Simoneit, B. R. T. (1993). Sources of Fine Organic Aerosol—3. Road Dust, Tire Debris, and Organometallic Brake Lining Dust—Roads As Sources and Sinks. *Environmental Science & Technology* 27(9), 1892-1904.

PARTICLE SIZE AND DEPOSITION

Particle size has a significant bearing on bodily deposition, net exposure, and corresponding health risk, even within the PM_{2.5} size fraction. 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.²⁴ Within the PM_{2.5} size range, particles less than 0.1 microns (PM 0.1) and greater than 10 microns are least likely to be exhaled, and thus have higher deposition fractions.²⁵

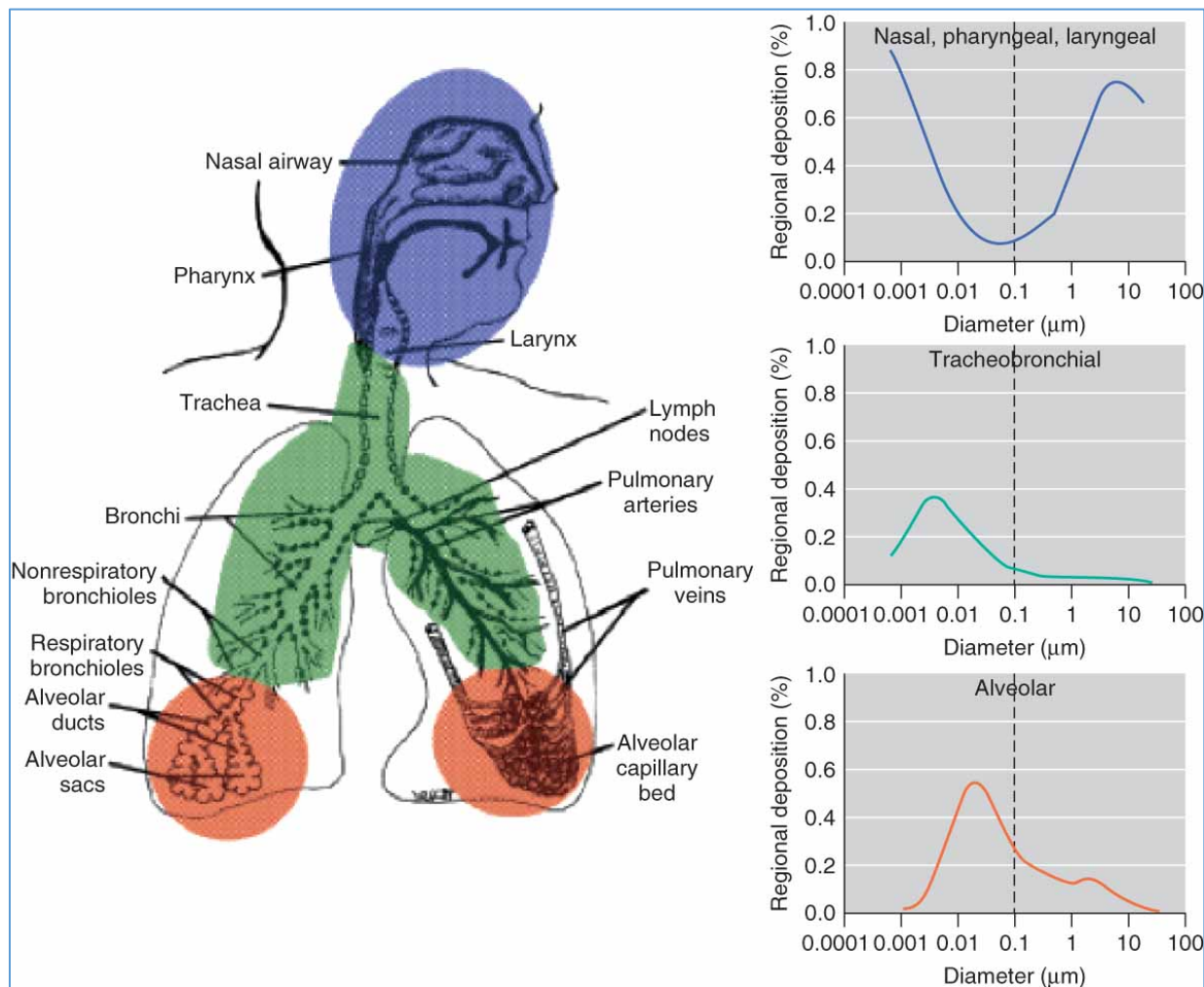
The relationship between particle size, zone of deposition, and deposition fraction are depicted in Figure G-2 and is summarized as follows:

- **Nasal, pharyngeal, laryngeal:** The uppermost segment of the respiratory tract is the primary zone of deposition for the smallest and largest particles. Approximately 80% of extremely small particles of one nanometer (0.001 micron) diameter or less are retained here with a comparable deposition fraction in the 10 micron diameter.
- **Tracheobronchial:** The deposition fraction in this zone peaks at nearly 40% for particles with diameters between 1 and 10 nanometers. Almost 100% of the particles above the PM 0.1 size cut are either deposited in the other two deposition zones or exhaled.
- **Alveolar:** Deposition in the gas exchange zone of the lungs peaks in the 10 nanometer size with a gradual dissipation of deposition beyond the PM 0.1 size.

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

²⁵ U.S. Environmental Protection Agency [EPA]. (2004, October). *Air Quality Criteria for Particulate Matter*. Final Report. Washington, D.C.: EPA 600/P-99/002aF-bF.

Figure G-2 Relationships between Particle Size Distribution and Respiratory Deposition Zones



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.²⁶ These chemicals can trigger heart attacks and premature death among individuals with pre-existing heart conditions.²⁷ Extremely small particles can also be absorbed into the brain via the nasal tract, bypassing the protection provided by the blood-brain barrier.²⁸ The effects of particles deposited primarily in the tracheobronchial region center on respiratory function.²⁹

²⁶ 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.

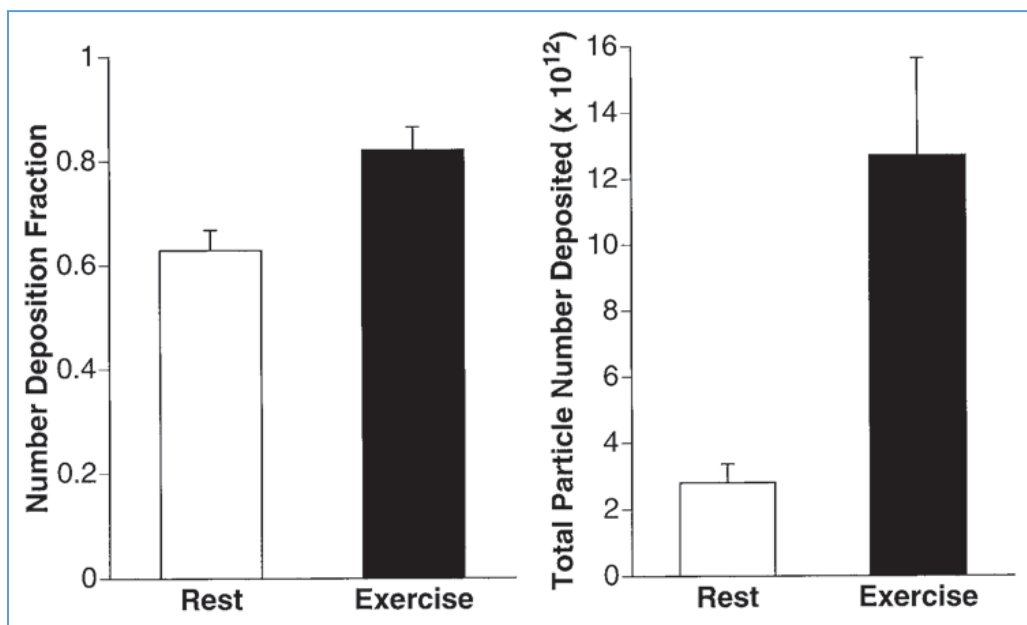
²⁷ Nel A. (2005). Air Pollution-Related Illness: Effects of Particles. *Science*, 308(5723), 804–806. doi: 10.1126/science.1108752

²⁸ 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

²⁹ U.S. Environmental Protection Agency [EPA]. (2009). *Integrated Science Assessment for Particulate Matter*. Final Report. Washington, D.C.: EPA/600/R-08/139F.

As depicted in Figure G-3, particle deposition and associated health risk is magnified by exercise in several ways. First, the amount of inhaled air per minute rises substantially when breathing faster and more deeply. Second, breathing harder means that particles, especially PM 0.1, are more likely to penetrate the alveolar region of the lungs where absorption into the bloodstream occurs. A 2003 study³⁰ found that during moderate exercise 80% of inhaled PM 0.1 was deposited in the lungs, compared with 60% lung retention while at rest (see left panel in Figure G-3). However, because the volume of air exchanged per minute increases substantially during exercise, overall PM 0.1 deposition increased by 450% (right panel). This phenomenon underscores the health risk posed to individuals who work or exercise in areas where sources of hydrocarbon combustion result in very high PM 0.1 particle concentrations.

Figure G-3 Particle Number Deposition Fraction (DF) and Total Particle Deposition of PM 0.1 at Rest and Exercise



EXPOSURE TO ULTRAFINE PARTICLES (PM 0.1)

Elevated exposure to freshly emitted PM 0.1 is a critical health risk factor that often does not correspond to ambient PM_{2.5} concentrations at local monitors. PM 0.1 are formed through nucleation and gas-to-particle reactions and grow (or shrink) through a number of mechanisms including condensation, coagulation, and volatilization.³¹ High concentrations of primary (directly emitted) PM 0.1 are typically found near fresh sources of hydrocarbon combustion, including coal plants, charbroiled meat, diesel and gasoline vehicles, wood combustion, and lawn care equipment. These combustion

³⁰ Daigle, C., Chalupa, D.C., Gibb, F.R., Morrow, P.E., Oberdörster, G., Utell, M.J., & Frampton, M.W. (2003). Ultrafine Particle Deposition in Humans during Rest and Exercise. *Inhalation Toxicology*, 15(6), 539–552. doi: 10.1080/08958370304468

³¹ Solomon, P. (2012). An Overview of Ultrafine Particles in Ambient Air. *EM: Journal of the Air & Waste Management Association*, May, 18–26.

particles start out very small, grow larger over time and space, and evolve chemically at the same time. Secondary PM 0.1 typically is formed via particle nucleation from gas or liquids and is characterized by larger geographic scales and more uniform population exposure.

Despite being extremely small, PM 0.1 has an extremely high surface area, as seen in Figure G-4. Compared to an equal mass of particles of two microns (PM 2.0) in diameter, ultrafine particles that are 1,000 times smaller (20 nanometers or PM 0.02) nonetheless have 125 times the surface area.³² In addition, PM 0.1 produced by hydrocarbon combustion typically contain a rich mixture of chemicals with potential health effects, including nickel, iron, vanadium, PAH, and others.³³ Chemical potency, very high surface area, and alveolar deposition are signal characteristics of PM 0.1 from hydrocarbon combustion that result in significant health risks from chronic exposure.

Figure G-4 Electron Micrograph of an Ultrafine Particle³⁴

Sub-populations who live or work near sources of primary PM 0.1 from hydrocarbon combustion are particularly at risk. Health scientists have generated an overwhelming body of epidemiological (statistical) evidence that individuals near freeways (less than 300 meters) are being harmed via chronic inhalation of PM 0.1 from vehicles.³⁵ Similarly, a 2011 study of residential wood burning in Cambria, California found very high neighborhood concentrations of PM 0.1 from wood smoke even though concentrations of PM2.5 at the nearby ambient monitor met the federal health

³² Donaldson, K., Stone, V., Clouter, A., Renwick, L., & MacNee W. (2001). Ultrafine Particles. *Occupational Environmental Medicine* 58, 211–216. doi: 10.1136/oem.58.3.21

³³ Morawska, L., Ristovski, Z., & Jayaratne, E.R. (2008). Ambient Nano and Ultrafine Particles from Motor Vehicle Emissions: Characteristics, Ambient Processing and Implications on Human Exposure. *Atmospheric Environment*, 42(35), 8113–8138. doi: 10.1016/j.atmosenv.2008.07.050

³⁴ Nel A. (2005). Air Pollution-Related Illness: Effects of Particles. *Science*, 308(5723), 804–806. doi: 10.1126/science.1108752

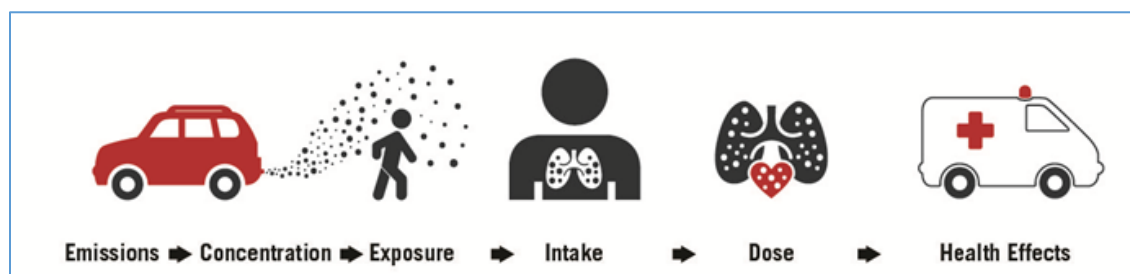
³⁵ Gauderman, W., Vora, H., McConnell, R., Berhane, K., Gilliland, F., Thomas, ... Peters, J. (2007). Effect of Exposure to Traffic on Lung Development from 10 to 18 Years of Age: A Cohort Study. *The Lancet* 369(9561), 571–577.

standard.³⁶ The health risk from fresh sources of PM 0.1 has important environmental justice implications to the extent that elevated exposure to near-source PM 0.1 is concentrated in communities that already face sources of risk related to race or socioeconomic status.³⁷ Chronic exposure to near-source PM 0.1 commonly occurs in locations where local monitors are in attainment for PM2.5 standards and during seasons when ambient PM2.5 concentrations are below the annual daily standard.

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 contribution to the regional PM2.5 emissions inventory and its toxicity. 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 G-5). Known as the intake fraction, this measure of population exposure is defined empirically as the pollutant mass inhaled divided by the mass emitted.³⁸ 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 G-5 Simplified Intake Fraction Model



The relevance of the intake fraction concept can be seen in a recent study of neighborhood variability in wood smoke concentrations in Cambria, California.³⁹ The winter study found very high concentrations of PM 0.1 on a neighborhood scale that were often not reflected in PM2.5 concentrations measured by local air quality monitors.

³⁶ Thatcher, T. & Kirchstetter, T. (2011). *Assessing Near-Field Exposures from Distributed Residential Wood Smoke Combustion Sources*. Report prepared for the California Air Resources Board.

³⁷ London, J., Huang, G., & Zagofsky, T. (2011). *Land of Risk, Land of Opportunity: Cumulative Environmental Vulnerabilities in California's San Joaquin Valley*. Davis, CA: University of California, Davis, Center for Regional Change.

³⁸ 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.

³⁹ Thatcher, T. & Kirchstetter, T. (2011). *Assessing Near-Field Exposures from Distributed Residential Wood Smoke Combustion Sources*. Report prepared for the California Air Resources Board.

In effect, a single wood-burning household had the effect of enveloping the adjacent and downwind homes with a PM 0.1 plume. Furthermore, the study also found that wood smoke PM 0.1 was infiltrating adjacent homes that were not burning, with an average indoor concentration found to be 74% as high as immediately outside the homes. Taking into consideration the length of PM 0.1 inhalation during sleeping hours, the relatively high concentration of PM 0.1 found in the plume, and the number affected of individuals in an urban neighborhood, the intake fraction resulting from the source of the wood smoke would be very high. Assuming that this nightly exposure occurred over the course of a season, the cumulative health risk to the neighborhood would be considerable and would almost certainly exceed the risk indicated by daily concentrations of PM2.5 measured by ambient monitors.

HEALTH IMPACTS OF AIR TOXICS

According to section 39655 of the California Health and Safety Code, a toxic air contaminant (TAC) is "an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health." Toxic air contaminants, otherwise known as "air toxics", are emitted from mobile sources (i.e., cars, trucks, buses, tractors, etc.), which are primarily regulated by the State and U.S.EPA; area sources (i.e., consumer products, dry cleaners), which are regulated the State, U.S.EPA, and the District; and from stationary sources regulated primarily by the District.

Adverse health effects of air toxics can vary by person based on a variety of factors. The EPA, California Office of Environmental Health Hazard Assessment (OEHHA), and CARB have assessed the health effects of variety of air toxics based on scientific studies that estimate the increased risk of illness from a specific human exposure to a toxic air contaminant. This information is then used to understand the risk associated with exposure to these air toxics in the community based on four key factors⁴⁰:

1. **Hazard identification:** describes the illnesses caused by a toxic air pollutant and the amount of evidence for those illnesses.
2. **Exposure assessment:** The size of the increased health risks depends on the exposure level and duration, as well as the number of people exposed. These are estimated as part of the exposure assessment.
3. **Dose-response:** The dose-response assessment estimates the dose-response relationship, which mathematically shows the change in the likelihood of health effects with changes in the levels of exposure to a toxic air pollutant.
4. **Risk characterization:** The risk characterization uses the above assessments to describe the type and size of any increased risk expected as a result of exposure to the air pollutant. It also includes a discussion of the uncertainties associated with the risk estimates.

⁴⁰ https://www3.epa.gov/airtoxics/3_90_024.html

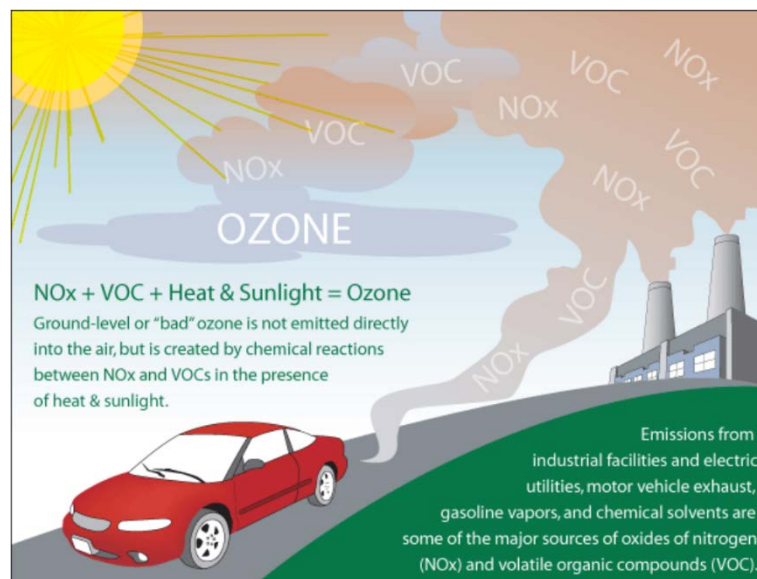
Air toxics can cause health problems by interfering with normal body functions. Commonly, air toxics can change chemical reactions within individual cells, which can kill cells, impair cell function, or re-direct cell activity. The results can be damaged organs, birth defects when the cells of an unborn child are damaged, or cancer that develops when cells begin to grow at an uncontrolled rate. Air toxics can also cause other serious non-cancer health effects. These health effects vary by compound and can include immune system, neurological, reproductive, developmental, and respiratory damage.

In addition to certain types of PM_{2.5} as discussed in this appendix, air toxics can be volatile organic chemicals (VOC's), pesticides, or other compounds that, based on scientific studies of exposure to humans and other mammals, have the propensity to cause these short or long-term health effects.

Chapter 4 of this CERP details how AB 2588, California's *Air Toxics "Hot Spots" Information and Assessment Act*, required the District to quantify emissions of air toxics, determine the health risk caused by those emissions, report emissions and any significant risks through written public reports and neighborhood public meetings, and take steps to reduce such risks. Appendix E outlines the health risk assessment status for the facilities within the Stockton Community.

HEALTH IMPACTS OF OZONE

Ground-level ozone, a gas of three oxygen atoms (O₃), is the main component of summertime smog. It is not directly emitted into the atmosphere, but produced by photochemical reactions between oxides of nitrogen (NO_x) and volatile organic compounds (VOCs) in the presence of sunlight. The Stockton community generally experiences the highest ozone concentrations on hot, sunny summer days with prolonged periods of stagnation. Figure G-6 below further illustrates ozone formation.

Figure G-6 Ozone Formation

Breathing ozone can trigger a variety of health problems, including chest pain, coughing, throat irritation, and congestion. It can reduce lung function and inflame the linings of the lungs. Repeated exposure may permanently scar lung tissue. Children are at a greater risk of experiencing negative health impacts because their lungs are still developing and they are more likely to be active outdoors when ozone levels are high, thus increasing their exposure. Studies have linked rising hospital admissions and emergency room visits to higher ozone levels. Although ozone is formed and transported on a regional scale and not directly emitted from local sources, reducing local VOC and NO_x emissions within the community will have positive health impacts on residents within and around Stockton.

AIR POLLUTION-RELATED HEALTH INDICATORS IN STOCKTON COMMUNITY

As described above, local air pollution studies have helped identify and track the effects of air pollution on short and long-term negative health impacts of Valley residents. In addition, statewide studies led by OEHHA (Office of Environmental Health Hazard Assessment) and CalEPA have looked at a variety of health indicators on a census-tract level to help understand the geographical, economical, and social differences between communities and identify the most disadvantaged and impacted areas in the State, culminating in the State's CalEnviroScreen (CES) 3.0 tool. Using an understanding of the relationship between poor air quality and certain health indicators (cardiovascular disease, asthma, and low birth weight), the District used CES 3.0 data to highlight air pollution-linked health indicators specifically in the Stockton community. As shown in Figures G-6-G-8 below, many areas in the Stockton community are above the 95 percentile, or top 5%, for all three health indicators. Through the District's valley-wide efforts and community action through AB 617, the ultimate goal is to contribute to significantly improving public health in Stockton by improving air quality.

The District will continue to work with the Steering Committee, OEHHA, CARB, and health researchers to track and support local research efforts to understand the public health impacts of local and regional emissions reduction efforts.

Figure G-7 Low-Birth Weight CES Percentile Score in Stockton

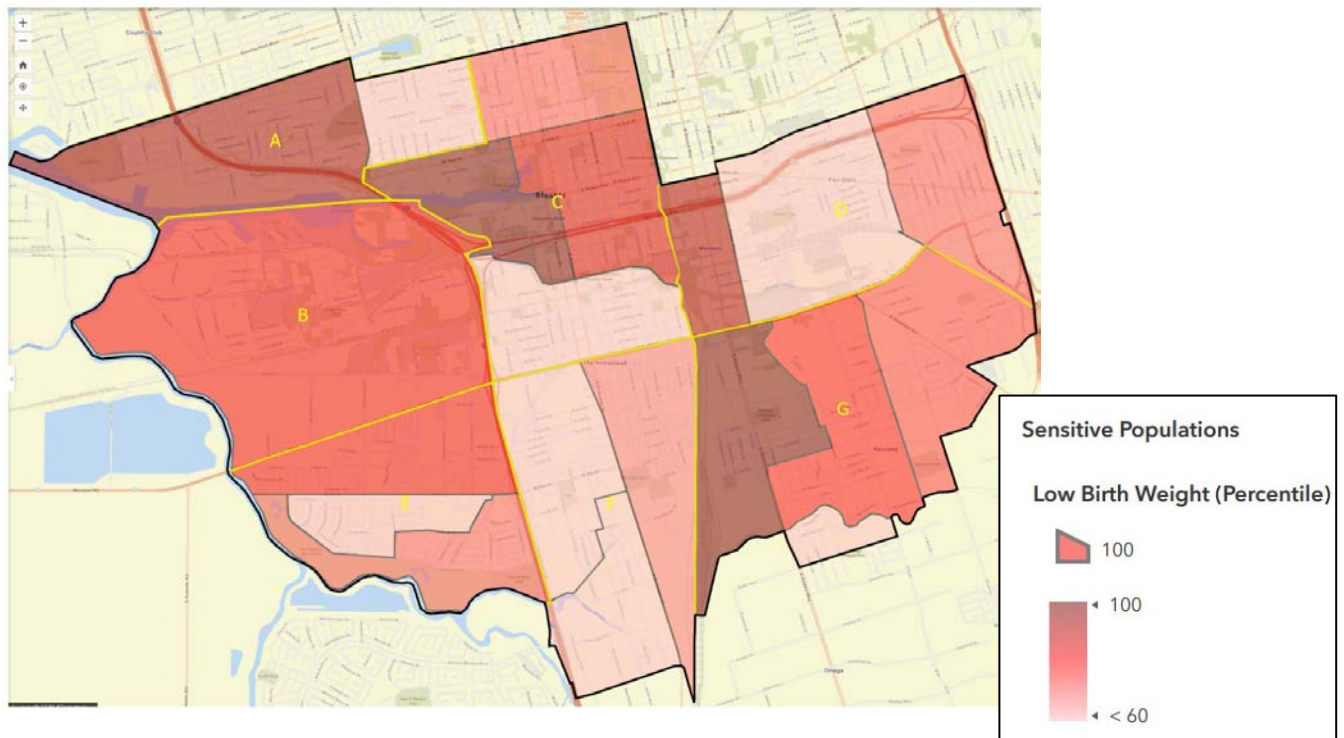


Figure G-8 Cardiovascular Disease CES Percentile Score in Stockton

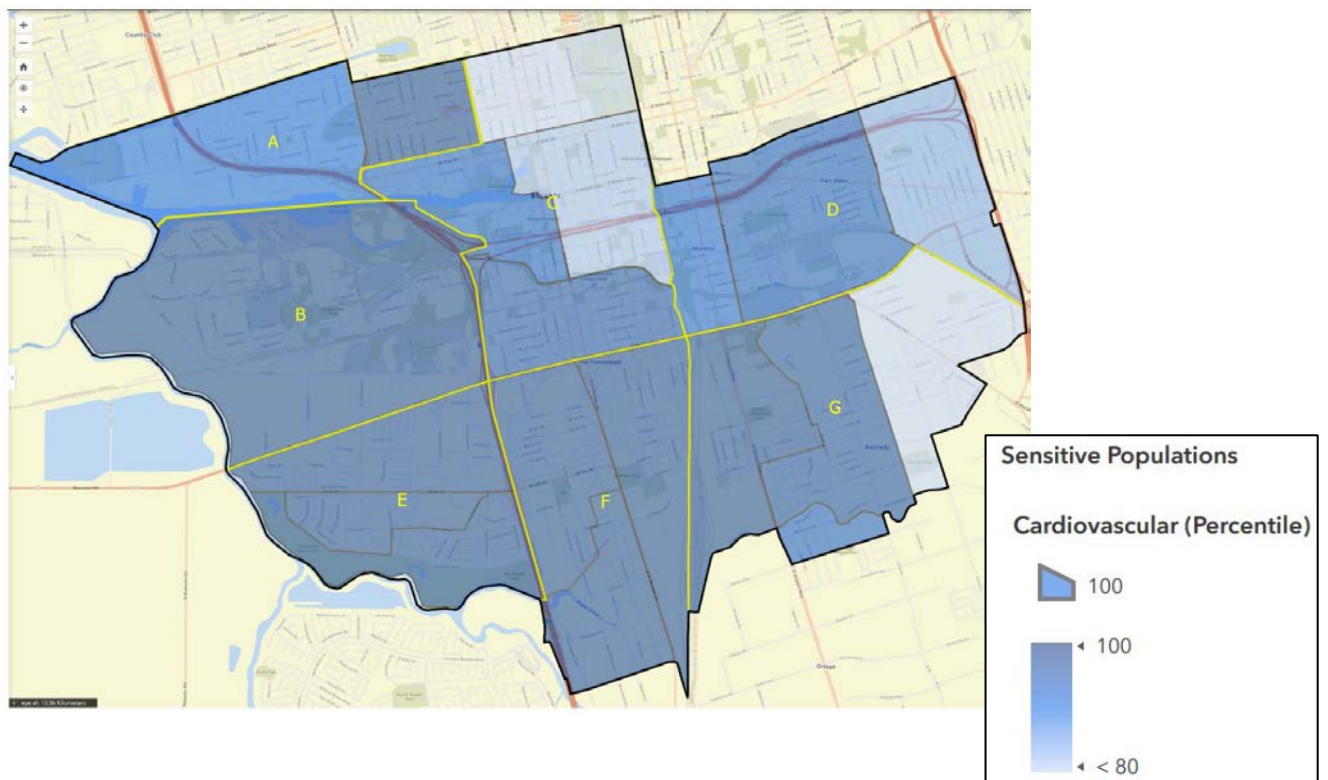


Figure G-9 Asthma CES Percentile Score in Stockton

