



Chapter 2

Risk-Based Strategy



This page intentionally blank.

Chapter 2: Risk-Based Strategy

2.1 WHAT IS THE RISK-BASED STRATEGY?

The U.S. Environmental Protection Agency's (EPA) National Ambient Air Quality Standards (NAAQS) 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 PM_{2.5} mass, which shows progress toward attainment of the standard, serves as the surrogate for population exposure and risk.

NAAQS are indeed health-based, and attaining these standards will result in clear and significant health benefits. However, NAAQS, as currently established, are essentially *mass-based* standards. In the case of PM_{2.5}, the current standards do not account for particle size distribution, chemical species composition, surface area, and other factors of health risk. There is inherent complexity in documenting the health risks associated with exposure to particles (which have a wide range of characteristics) as compared to pollutants like ozone (which has more consistency between molecules).

In contrast, recent health-science research has substantially deepened our 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's 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.¹

EPA policy directly acknowledges the importance of a Risk-based Strategy to maximize public health benefits within a region's efforts to attain the National Ambient Air Quality Standards (NAAQS). EPA's March, 2012, PM_{2.5} implementation guidance memo states, "...it is likely that SIPs for the 2006 24-hour PM_{2.5} NAAQS may need to include *greater emphasis on reducing emissions from local sources* [emphasis added] as compared to plans to attain the 1997 PM_{2.5} NAAQS."² EPA's memo further encourages that states consider evidence from published literature indicating that reductions of direct PM_{2.5} have a greater health benefit per ton than reductions of other criteria

¹ Lippman, M. (2012, April 16). Presentation: Results from National Particle Component Toxicity (NPACT) Program and NYU: Toxicology Findings, Integration, and implications. Presented at the Annual Meeting of the Health Effects Institute (HEI) in Chicago, IL, April 15–17, 2012. Presentation retrieved from <http://www.healtheffects.org/Slides/AnnConf2012/Lippmann-MonPM.pdf>

² U.S. Environmental Protection Agency (2012, March 2). Memorandum from the Office of Air Quality Planning and Standards: Implementation Guidance for the 2006 24-Hour Fine Particle (PM_{2.5}) National Ambient Air Quality Standards (NAAQS). Retrieved from http://www.epa.gov/ttn/naaqs/pm/pdfs/20120302_implementation_guidance_24-hr_pm2.5_naaqs.pdf

pollutants, such as SO₂ and NO_x,³ and that providing methods local air quality plans can use to maximize health benefits and minimize risk inequality.⁴

In September 2010, the District Governing Board adopted a research-driven Risk-based Strategy (RBS) designed to maximize public health improvements resulting from the District's attainment strategies and related initiatives. The overall goal of the RBS is to minimize cumulative population exposure to air pollution and corresponding health risk in the region. This risk reduction goal is being pursued through the integration of emerging scientific knowledge into the District's control strategies, incentive programs, public communication, and enforcement actions.

The District is integrating the RBS into the development of the *2012 PM_{2.5} Plan*. The purpose of this chapter is to document the scientific foundation for the RBS and how it is being applied to the District's PM_{2.5} SIP. This chapter includes the following elements:

- The key aspects of particulate pollution that shape human exposure and risk from PM_{2.5}, along with a discussion of how the RBS is reflected in existing District programs;
- Further details about key health risk elements of PM_{2.5} particles and exposure; and
- An outline of how these risk elements are used to characterize the associated risk when evaluating sources of PM_{2.5} and related control or incentive options in subsequent chapters.

2.2 BACKGROUND FOR THE RISK-BASED STRATEGY

As a response to mounting epidemiological evidence that PM_{2.5} was more harmful than PM₁₀, EPA established a PM_{2.5} NAAQS in 1997 to accompany the previously established PM₁₀ NAAQS. PM₁₀ occurs at larger mass concentrations than PM_{2.5}, so the shift to PM_{2.5} somewhat conflicted with the time-tested toxicological precept of “the dose (mass) makes the poison.” Particulate inhalation studies found that the smaller PM_{2.5} particles penetrate more deeply into the lungs, where particles more effectively avoid immune system defenses. Toxicological analyses of PM_{2.5} identified chemical species that acted differentially to promote respiratory and cardiovascular inflammation. While it was unclear at that time which PM_{2.5} chemicals were the most harmful, the scientific consensus was that the health risks stemmed from the chemicals rather than the particles themselves.

In the 15 years since the first PM_{2.5} NAAQS was established, scientists have conducted many studies that have identified which chemical species of PM_{2.5} are most

³ Fann, N., Fulcher, C.M., & Hubbell, B.J. (2009). The Influence of Location, Source, and Emission Type in Estimates of the Human Health Benefits of Reducing a Ton of Air Pollution. *Air Quality, Atmosphere & Health*, 2(3), 169–176. doi: 10.1007/s11869-009-0044-0

⁴ Fann, N., Roman, H.A., Fulcher, C.M., Gentile, M.A., Hubbell, B.J., Wesson, K., & Levy, J.I. (2011). Maximizing Health Benefits and Minimizing Inequality: Incorporating Local-Scale Data in the Design and Evaluation of Air Quality Policies. *Risk Analysis*, 31(6), 908–922. doi: 10.1111/j.1539-6924.2011.01629.x

harmful and have pinpointed their sources.⁵ Health researchers have also documented the negative cardiovascular and immune system effects of ultrafine particles, or particles that are 0.1 microns or smaller (PM 0.1), based on these particles' ability to penetrate the alveolar region of the lungs and deliver chemicals into the bloodstream. 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 RBS. Rather than ignore this growing body of scientific knowledge in the development of this SIP, the District's RBS 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 in this plan. The outcome is a stronger, more health-protective plan that reflects the current trajectory of scientific knowledge toward a more complete understanding of population risk from PM2.5 particles.

The NAAQS-SIP process and the RBS are complimentary strategies, not an either-or scenario. The RBS 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. As required under the CAA, the District is committed to attaining the 2006 PM2.5 standard as expeditiously as possible, and the District will not ignore sources of PM2.5 under its jurisdiction that could contribute to the Valley's attainment of the PM2.5 NAAQS.

A number of the District's programs have been influenced by the underlying principles and goals of the Risk-based Strategy and provide a model of the success and added potential benefits possible under this strategy.

- **District Rule 4901 (Wood Burning Fireplaces and Wood Burning Heaters) and the District's corresponding Check-Before-You-Burn program** have both been reducing harmful species of PM2.5 where and when those reductions are most needed—in impacted urbanized areas when the local weather is forecast to hamper PM dispersion. By decreasing emissions from residential wood burning, Rule 4901 decreases directly emitted PM2.5, as well as carbon monoxide, formaldehyde, sulfur dioxide, irritant gases, and known and suspected carcinogens, such as polycyclic aromatic hydrocarbons (PAH). In 2008, the Central Valley Health Policy Institute found that District wood burning curtailments on days with high PM concentrations reduced annual PM exposure

⁵ U.S. Environmental Protection Agency [EPA]. (2009). Integrated Science Assessment for Particulate Matter: Final Report. Washington, D.C.: EPA/600/R-08/139F. Available at <http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=216546>

by about 13% in Bakersfield and Fresno, resulting in an estimated 59 to 121 avoided cases of annual premature mortality.⁶

Even though the *2008 PM_{2.5} Plan* was developed per EPA requirements for the 1997 PM_{2.5} standard (with a 24-hour standard of 65 µg/m³), the 2008 plan included a commitment to amend Rule 4901 in 2009 (with implementation in 2010) to align the wood-burning curtailment threshold with the newer 2006 PM_{2.5} standard (with a 24-hour standard of 35 µg/m³). Then, based on research reiterating the effectiveness of Rule 4901 in protecting public health, as well as public support for a stronger rule, the District amended and implemented Rule 4901 in 2008—one year ahead of the scheduled rule development and two years ahead of scheduled implementation. The amended rule also set the curtailment level lower than initially planned (to 30 µg/m³) to provide an extra margin of safety and to address air quality forecast uncertainties.

The significant increase in the number of curtailment days resulting from the lower threshold has resulted in a parallel reduction in nighttime neighborhood exposure to PM 0.1, including exposure that has been shown to occur as a result of indoor infiltration. This aspect of Rule 4901, i.e. reducing the frequency of elevated exposure to PM_{0.1} that induces immune system sensitization and cardiovascular inflammation, has been carried forward into the Risk-based Strategy. The District's prioritization of Rule 4901 is one of the best examples of a District policy aimed at maximizing public health benefits based on a rigorous assessment of population exposure and risk.

- **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 grant program, the District has replaced over 2,000 high-polluting gas-powered lawn mowers with clean electric mowers, thus decreasing the urban, localized health risks associated with the use of gas-powered equipment. As described in Appendix C of this plan, the District is now expanding its lawn care emissions reductions programs to the commercial sector. In conjunction, District-funded field measurements of PM 0.1 by UCSF-Fresno found very high concentrations in and around lawn care activities, indicating a very high intake fraction for lawn care workers and concomitant cardiovascular risk.
- **The District's information and educational programs, such as the Real-Time Air Quality Advisory Network (RAAN)**, also contribute to the Risk-based Strategy. RAAN uses real-time data from air monitoring stations throughout the Valley to provide hour-by-hour air quality updates to schools and other subscribers. Subscribers can use this information to make informed decisions and plan outdoor activities for times with the best air quality, reducing potential

⁶ Lighthall, D., Nunes, D., & Tyner, T.R. (2009). Environmental Health Evaluation of Rule 4901: Domestic Wood Burning. Fresno, CA: Central Valley Health Policy Institute for the San Joaquin Valley Air Pollution Control District. Retrieved from <http://www.fresnostate.edu/chhs/cvhipi/documents/wood-burning-report.pdf>

air quality health risks. Reflecting the latest science on PM_{2.5} exposure risk for sensitive individuals, ambient concentrations of PM_{2.5} that are used to trigger RAAN health risk warnings are more health protective than those used in the EPA's Air Quality Index.

- **The District tracks and sponsors health research.** The District has sponsored several Valley-based health research projects in recent years. In 2010–2011, the District sponsored a first-of-its-kind epidemiological investigation of health effects of air pollution in Modesto, Fresno, and Bakersfield.⁷ The study found that high PM and ozone concentrations clearly correlate to increased hospital and ER admission rates, especially for those 19 and younger. During 2011 and 2012, the District is sponsoring a follow-up epidemiological study to examine which of the chemicals found in Valley PM_{2.5} are most highly correlated with elevated ER and hospital admission rates. The District is also sponsoring a pilot study of PM 0.1 in Fresno, partnering with UCSF-Fresno, to investigate the quantity and spatial distribution of PM 0.1 plumes from motor vehicles, lawn care equipment, wood burning, and restaurants. The District will continue to seek out and fund research opportunities that further the understanding of PM_{2.5} and ozone impacts on public health.

2.3 HOW IS THE RISK-BASED STRATEGY BEING INCORPORATED INTO THIS PLAN?

Though there are several existing District programs that readily fit into this strategy, this *2012 PM_{2.5} Plan* is the District's first formal effort to synthesize research, population-exposure analysis, and comprehensive emissions reductions efforts into a cohesive Risk-based Strategy. This is also the District's first opportunity to demonstrate how the RBS fits within and effectively supplements EPA's current CAA framework.

The District is integrating the RBS into various aspects of the *2012 PM_{2.5} Plan*. The District expects to incorporate the RBS into this plan with the following:

- **Information regarding health effects of PM_{2.5}:** Chapters 1 and 2 include detailed discussions regarding the different types of PM_{2.5} and their associated health effects.
- **Ambient data analysis:** In addition to evaluating PM_{2.5} mass trends per CAA and EPA guidelines, Chapter 3 and Appendix A discuss PM_{2.5} species-based trends. This species analysis, when considered with health research, will highlight which PM_{2.5} and PM_{2.5} precursor sources might be prioritized under the RBS. The District is also evaluating the timing of higher PM_{2.5}

⁷ 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. Retrieved from <http://www.fresnostate.edu/chhs/cvhipi/publications/index.html>

concentrations to see if there are certain times of the day or times of the year when PM_{2.5} reductions might have more public health benefits.

- **Health Research:** The RBS is driven by strong science and research, and the District will continue to evaluate existing research and assist in promoting new research relevant to the Valley.
- **Analysis of health benefits under the attainment strategy:** Building on regional SIP modeling provided by ARB, the District used EPA's BenMAP benefit estimation model to estimate the health benefits in the Valley resulting from the District's attainment strategy. (See section 2.5 and Appendix E for additional details)
- **Source-by-source assessment:** The District is conducting a thorough analysis of all potential opportunities to reduce emissions of directly emitted PM_{2.5} and significant PM_{2.5} precursors in the Valley. The qualitative exposure assessment described below will be used to assist in evaluating the potential health benefit of reducing emissions from these various sources. This more comprehensive assessment will help establish the strongest scientific justification for new source control strategies and incentive program investments.
- **Control measure/strategy prioritization:** Based on the above source-by-source assessment, and other evaluation being conducted as part of developing this plan, priority will be given to regulatory control measures, incentive programs, technology advancement efforts, policy initiatives, and other strategies that maximize public health.

2.4 FIVE-FACTOR EXPOSURE ASSESSMENT METHODOLOGY

To qualitatively evaluate the potential risk reduction benefits from various sources, this plan will employ a scientifically based exposure characterization methodology that draws on the latest scientific understanding about health risk from PM_{2.5} exposure.

The District will use a five-factor exposure assessment methodology under the RBS:

1. **Relevance to attainment**
2. **Toxicity of chemical species**
3. **Particle size and deposition**
4. **Proximity to PM 0.1**
5. **Population intake fraction**

The qualitative exposure assessment employed in this SIP is different than a formal risk assessment. Risk assessment requires the quantification of key elements relating to emission levels, particle or chemical toxicity, dose-response relationships, and total population exposure. The primary drawback for formal risk assessment models in a SIP context is pervasive empirical uncertainty regarding the values of the different elements

listed above. Even if the chemical composition, geographic pattern and volume, and spatial distribution of emissions from a given source are known, it is very difficult to isolate and quantify the regional health impacts of emissions from that source because many other sources are also contributing to PM_{2.5} exposure. In addition, PM_{2.5} aerosols undergo photochemical aging over time and space, often resulting in new secondary organic and inorganic species generated by variable regional source loads and meteorological conditions. Despite these limitations, it is possible to use a simple but robust exposure characterization tool for making important qualitative and categorical distinctions regarding the relative contribution and associated of a given source to population exposure.

2.4.1 Relevance to Attainment

An important element of the RBS is the relevance of the emissions reductions to the Valley's attainment of EPA's health-based standards. This portion of the analysis will consider emissions type (such as PM_{2.5}, NO_x, or SO_x), seasonality of the emissions (since PM_{2.5} exceedance days occur during the winter months), and the percent contribution of that source's emissions relative to the Valley's total emissions inventory. For example, NO_x is the limiting factor for ammonium nitrate and therefore reductions of NO_x emissions in the Valley will provide a greater impact to achieving attainment than reductions of ammonia emissions.

2.4.2 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.⁸ 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.⁹

Many emissions sources evaluated in this plan 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 (carbon black), organic carbon compounds (OC), metals (elements), ammonium nitrate,

⁸ Bell, M.L. (2012). *Assessment of the Health Impacts of Particulate Matter Characteristics*. Research Report 161. Boston: MA. Health Effects Institute. Retrieved from <http://pubs.healtheffects.org/getfile.php?u=685>

⁹ Kelly, F.J. (2006). Oxidative Stress: Its Role in Air Pollution and Adverse Health Effects. *Occupational Environmental Medicine*, 60, 612–616. Retrieved from <http://oem.bmj.com/content/60/8/612.full> doi: 10.1136/oem.60.8.612

ammonium sulfate, and geological (see Appendix A, Section A.4.9). PM_{2.5} is regularly speciated at several Valley monitoring sites (see Chapter 3, Figure 3-7; and Appendix A). The following discussion provides an overview of PM_{2.5} species and their associated health impacts.

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.¹⁰ 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.¹¹ 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.¹² 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.¹³ 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

¹⁰ 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. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903>

¹¹ 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. Retrieved from <http://faculty.unlv.edu/buckb/scanned%20pdf/Ayres%20et%20al%202008.pdf>

¹² 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

¹³ 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

correlated with sustained (multi-day) high ambient concentrations of PM_{2.5} and was accompanied by decreased lung function.¹⁴

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.¹⁵ 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.¹⁶ A recent study in Mexico City found an association between exposure levels of EC and lung function decrements among asthmatic and non-asthmatic children.¹⁷

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.¹⁸ 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.

¹⁴ 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).

¹⁵ 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.

¹⁶ 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. Retrieved from <http://www.particleandfibretoxicology.com/content/7/1/33> doi: 10.1186/1743-8977-7-33

¹⁷ 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.

¹⁸ 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

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.¹⁹ Specifically, cardiovascular hospitalizations were 26% higher in counties with a nickel fraction in the 75th percentile versus counties with nickel in the 25th 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 identified as a principle cardiovascular risk factor.²⁰ In a related study, rats exposed to PM_{2.5} 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.²¹

In conclusion, metals found in PM_{2.5} produced from combustion of coal, residual oil, diesel fuel, 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 PM_{2.5} concentrations are likely to be caused by regional differences in coal combustion and resultant exposure to metals and OC.²²

Ammonium nitrate: Ammonium nitrate (nitrate) is classified as a secondary inorganic species (not directly emitted) primary source of PM_{2.5}, 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 (NO_x), largely from fuel combustion under meteorological conditions found during Valley winters, particularly during multiday stagnation events. As seen in Figure 2-1, nitrate is significant because it can contribute up to 50% or more to PM_{2.5} mass during peak days during winter seasons. The percentage contribution of nitrate to PM_{2.5} mass is substantially reduced in summer, with the 2000–2006 Valley average for June ranging from 13 to 18%, with mass levels at or below 2 µg/m³.²³

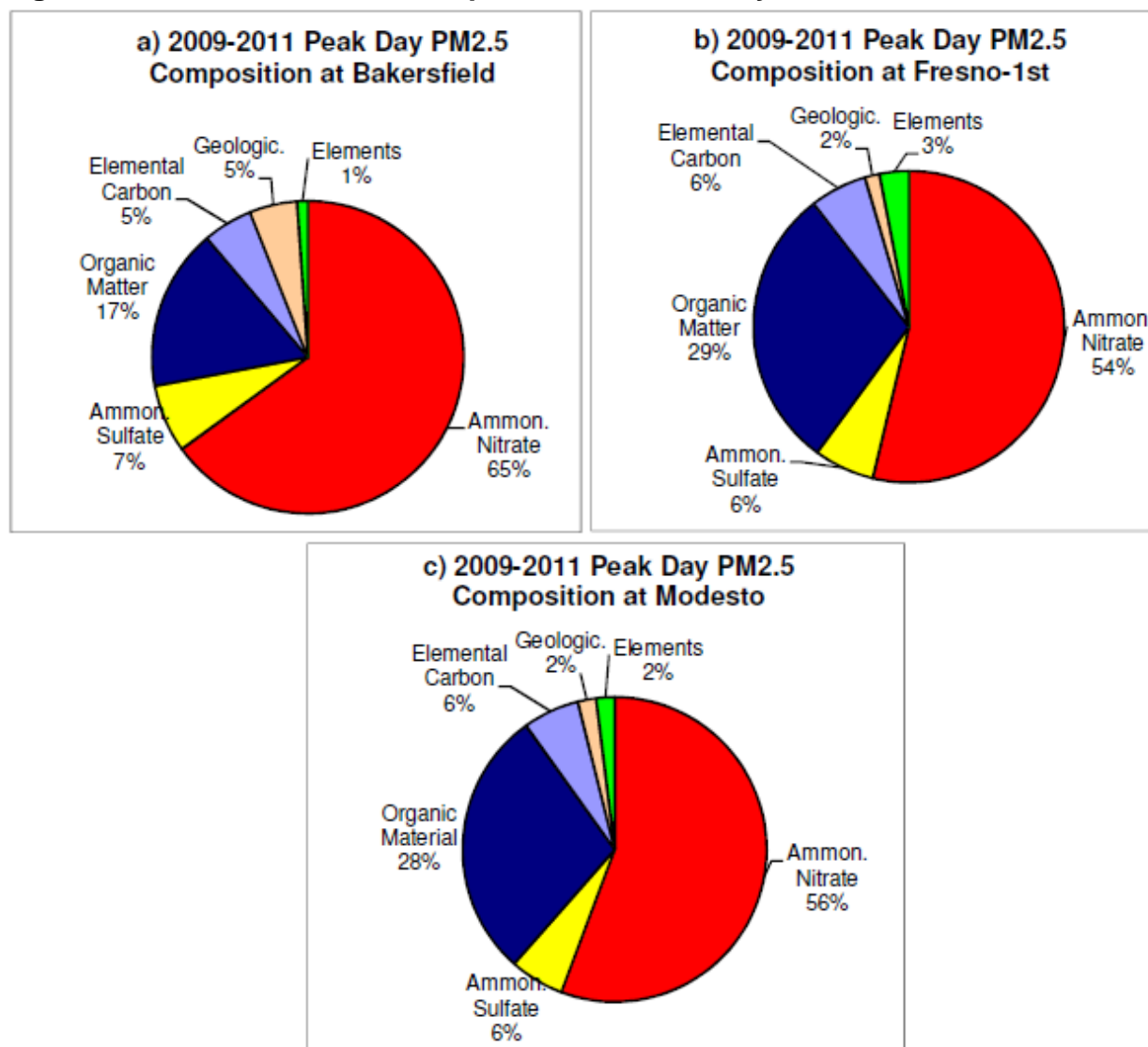
¹⁹ Bell, M.L., Ebisu, K., Peng, R.D., Samet, J.M. & Dominici, F. (2009). Hospital Admissions and Chemical Composition of Fine Particle Air Pollution. *American Journal of Respiratory Critical Care*, 179, 1115–1120. Retrieved from <http://ajrcm.atsjournals.org/content/179/12/1115.full.pdf+html>

²⁰ 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. Retrieved from <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1665439/>

²¹ Chen, L.C., & Lippmann, M. (2009). Effects of Metals within Ambient Air Particulate Matter (PM) on Human Health. *Inhalation Toxicology*, 21(1), 1–31. Retrieved from <http://faculty.unlv.edu/buckb/scanned%20pdf/Chen%20and%20Lippmann%202009.pdf>

²² Lippman, M. (2012, April 16). Presentation: Results from National Particle Component Toxicity (NPACT) Program and NYU: Toxicology Findings, Integration, and implications. Presented at the Annual Meeting of the Health Effects Institute (HEI) in Chicago, IL, April 15–17, 2012. Presentation retrieved from <http://www.healtheffects.org/Slides/AnnConf2012/Lippmann-MonPM.pdf>

²³ California Air Resources Board [CARB]. (2011). CARB speciation data: District Staff Analysis of the 2000-2006 Sampler Data for the Four Speciation Samplers in the San Joaquin Valley. Available at <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm>

Figure 2-1 PM_{2.5} Chemical Composition on Peak Days

A recent cold-season ammonia emissions inventory for the Valley estimated that motor vehicles and livestock production each contribute approximately 40% to total seasonal ammonia.²⁴ Green waste composting and nitrogen fertilizer emissions from soil are also major sources. As temperatures increase, the proportional contribution of livestock to the regional inventory increases, peaking at nearly 75% of the total in the hot season.

The relative toxicity of ammonium nitrate is an important issue given its substantial mass contribution to regional PM_{2.5}. 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.

²⁴ Battye, W., Anaja, V.P., & Roelle, P.A. (2003). Evaluation and Improvement of Ammonia Emissions Inventories. *Atmospheric Environment*, 37, 3873–3883.

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.²⁵ 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.

One experimental study was found that explicitly looked for toxic mechanisms driven by ammonium nitrate.²⁶ 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. As seen in Figure 2-1, mass concentrations of sulfate are significantly lower than for nitrate in the Valley, averaging from 6% to 7% of PM_{2.5} mass during winter peaks. 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 study²⁷ 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

²⁵ 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. Available at <http://www.ehjournal.net/content/10/1/89/> doi: 10.1186/1476-069X-10-89

²⁶ 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

²⁷ 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

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.²⁸ 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.²⁹ Researchers found that PM sulfate was a weaker indicator of health risk than PM_{2.5} 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 taken from nine different sites in the western U.S. taken from windblown dust and vehicle-generated particles from unpaved roads.³⁰ 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

²⁸ 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

²⁹ 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

³⁰ 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. Retrieved from <http://toxsci.oxfordjournals.org/content/82/1/88.full.pdf+html> doi: 10.1093/toxsci/kfh24

characterization of sources in this plan. 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 analysis³¹ 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 in subsequent chapters.

2.4.3 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.³² 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.³³

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

- A. Nasal, pharyngeal, laryngeal: As shown in Figure 2-2 at the upper right, 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.
- B. 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.

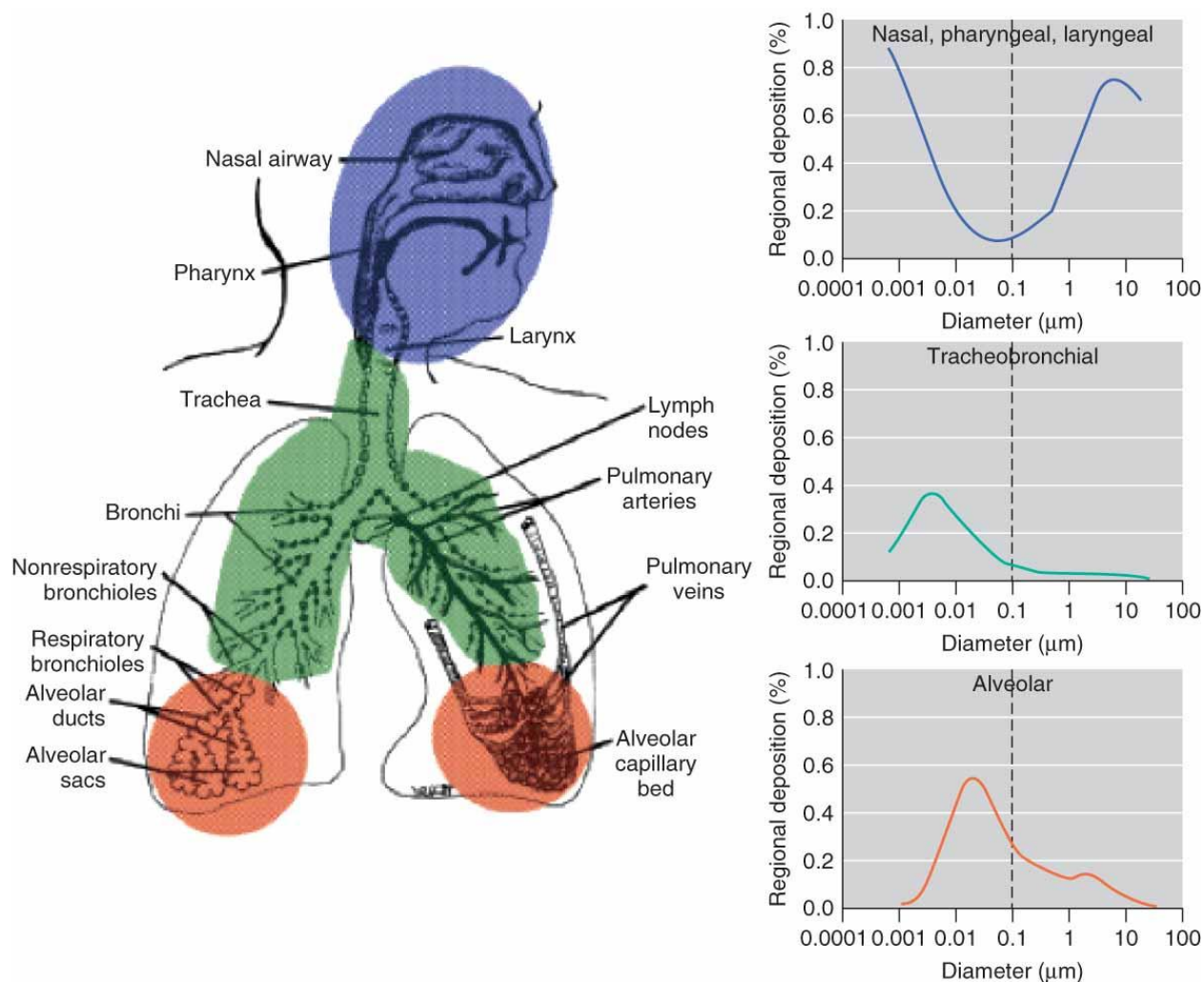
³¹ Rogge, W. F., Hildemann, L. M., Mazurek, M. A., Cass, G. R. and Simoneit, B. R. T. (1993). Sources of Fine Organic Aerosol—3. Road Dust, Tire Debris, and Organometallic Brake Lining Dust—Roads As Sources and Sinks. *Environmental Science & Technology* 27(9), 1892-1904.

³² International Commission on Radiological Protection [ICRP]. (1995). Human Respiratory Tract Model for Radiological Protection. ICRP Publication 66.. *Annals of the ICRP* 24, 1-3.

³³ 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. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903>

C. 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.

Figure 2-2 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.³⁴ These chemicals can trigger heart attacks and premature death among individuals with pre-existing heart conditions.³⁵ Extremely small particles can also be absorbed into the brain via the nasal tract,

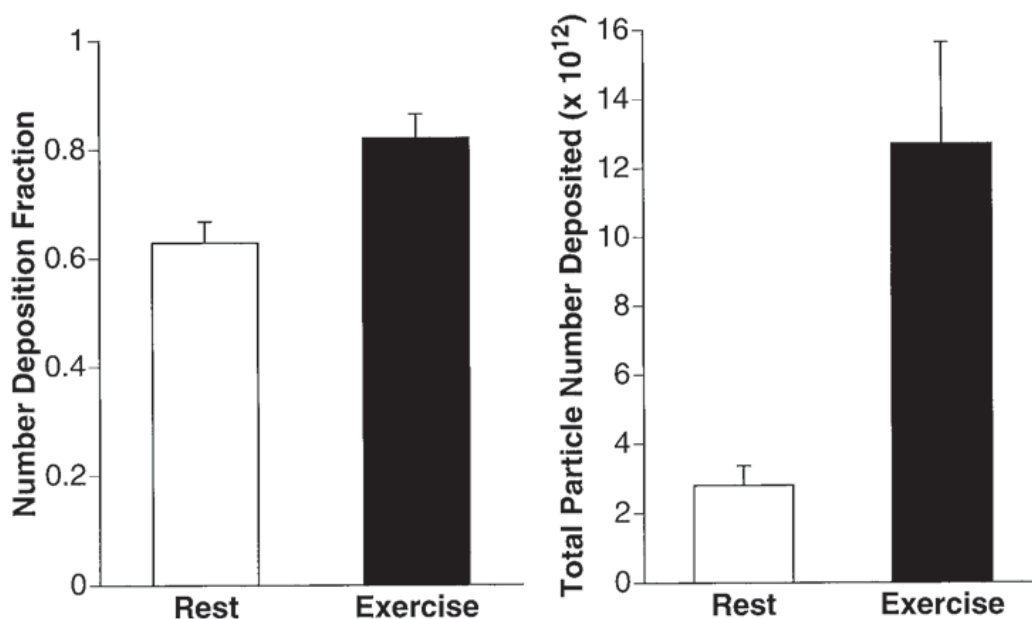
³⁴ Delfino, R.J., Sioutas, C., & Malik, S. (2005). Potential Role of Ultrafine Particles in Associations between Airborne Particle Mass and Cardiovascular Health. *Environmental Health Perspectives* 113(8), 934–946. Retrieved from <http://ehp03.niehs.nih.gov/article/submitArticle.action?articleURL=info%3Adoi%2F10.1289%2Fehp.7938>

³⁵ Nel A. (2005). Air Pollution-Related Illness: Effects of Particles. *Science*, 308(5723), 804–806. doi: 10.1126/science.11108752

bypassing the protection provided by the blood-brain barrier.³⁶ The effects of particles deposited primarily in the tracheobronchial region center on respiratory function.³⁷

As depicted in Figure 2-3, 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 study³⁸ 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 2-3). However, because the volume of air exchanged per minute increases substantially during exercise, overall PM 0.1 deposition increased by 450% (right panel). Discussed further below, 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 2-3 Particle Number Deposition Fraction (DF) and Total Particle Deposition of PM 0.1 at Rest and Exercise



³⁶ 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.

³⁷ U.S. Environmental Protection Agency [EPA]. (2009). *Integrated Science Assessment for Particulate Matter: Final Report*. Washington, D.C.: EPA/600/R-08/139F. Available at <http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=216546>

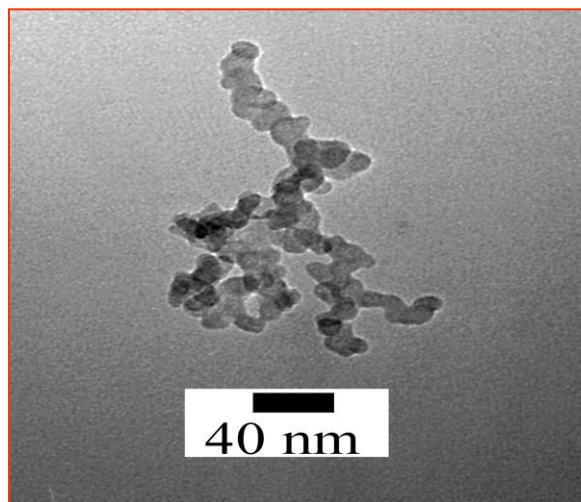
³⁸ Daigle, C., Chalupa, D.C., Gibb, F.R., Morrow, P.E., Oberdörster, G., Utell, M.J., & Frampton, M.W. (2003). Ultrafine Particle Deposition in Humans during Rest and Exercise. *Inhalation Toxicology*, 15(6), 539–552. doi: 10.1080/08958370304468

2.4.4 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.³⁹ 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 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 are 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 2-4. 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.⁴⁰ 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.⁴¹ 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 2-4 Electron Micrograph of an Ultrafine Particle⁴²



³⁹ Solomon, P. (2012). An Overview of Ultrafine Particles in Ambient Air. *EM: Journal of the Air & Waste Management Association*, May, 18–26.

⁴⁰ Donaldson, K., Stone, V., Clouter, A., Renwick, L., & MacNee W. (2001). Ultrafine Particles. *Occupational Environmental Medicine* 58, 211–216. Retrieved from <http://oem.bmj.com/content/58/3/211.short> doi: 10.1136/oem.58.3.21

⁴¹ 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

⁴² Nel A. (2005). Air Pollution-Related Illness: Effects of Particles. *Science*, 308(5723), 804–806. doi: 10.1126/science.1108752

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.⁴³ Similarly, a recent 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 standard.⁴⁴ 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.⁴⁵ 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 of 15 $\mu\text{m}/\text{m}^3$.

2.4.5 Population Proximity and Intake Fraction

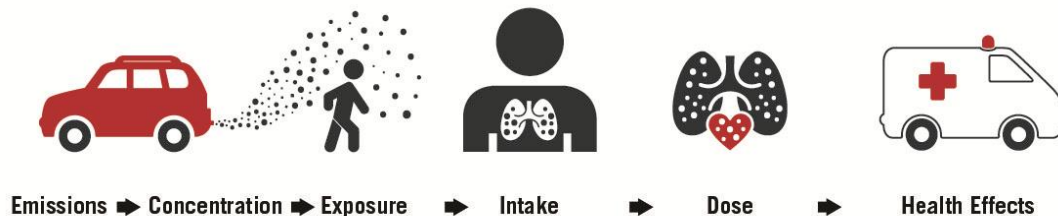
Estimating total exposure and net health risk from a given source of PM2.5 requires that population proximity and population density be considered in addition to the source's contribution to the regional PM2.5 emissions inventory and its toxicity. In addition to factors governing net deposition of inhaled 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 2-5). Known as the intake fraction, this measure of population exposure is defined empirically as the pollutant mass inhaled divided by the mass emitted.⁴⁶ 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.

⁴³ 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. Retrieved from [http://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(07\)60037-3/fulltext#article_upsell](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(07)60037-3/fulltext#article_upsell)

⁴⁴ Thatcher, T. & Kirchstetter, T. (2011). *Assessing Near-Field Exposures from Distributed Residential Wood Smoke Combustion Sources*. Report prepared for the California Air Resources Board. Retrieved from <http://www.arb.ca.gov/research/rsc/10-28-11/item2dfr07-308.pdf>

⁴⁵ 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. Retrieved from http://regionalchange.ucdavis.edu/publications/Report_Land_of_Risk_Land_of_Opportunity.pdf

⁴⁶ Marshall, J.D., & Nazaroff, W.W. (2004, October). *Using Intake Fraction to Guide ARB Policy Choices: The Case of Particulate Matter*. Unpublished California Air Resources Board Report.

Figure 2-5 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.⁴⁷ As described above, 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. 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.

⁴⁷ Thatcher, T. & Kirchstetter, T. (2011). *Assessing Near-Field Exposures from Distributed Residential Wood Smoke Combustion Sources*. Report prepared for the California Air Resources Board. Retrieved from <http://www.arb.ca.gov/research/rsc/10-28-11/item2dfr07-308.pdf>

2.5 HEALTH BENEFITS ACHIEVED THROUGH 2012 PM_{2.5} PLAN

Understanding the results of any strategy is critical to assessing the overall value and success of that strategy. As part of integrating the Risk-Based Strategy into this plan, the District has estimated the fundamental metric associated with implementation of the 2012 PM_{2.5} Plan – the health benefits resulting from implementation of this strategy. As presented below and described in more detail in Appendix E, an EPA-developed model called BenMAP has been employed in order to estimate the annual reductions in morbidity (disease) and mortality (premature death) attributable to improved air quality due to the 2012 PM_{2.5} Plan in the attainment year of 2019.

BenMAP is a sophisticated computer software model developed by the U.S. EPA that is well-suited for estimating the health benefits from the 2012 PM_{2.5} Plan. Over the course of the past decade, ongoing progress in the fields of epidemiology and geographic information systems (GIS) have resulted in the development of computer models that are capable of estimating the health benefits of improved air quality with reasonable accuracy when properly applied. These models estimate the number of avoided cases of certain diseases and other health impairment categories, known as health endpoints, which result from a specified reduction in exposure to criteria air pollutants, in this case, reduced exposure to PM_{2.5} concentrations.

Through implementation of the comprehensive control strategy included in this plan and the resulting reductions in PM_{2.5} concentrations throughout the Valley, the District estimates the following health benefits using this model:

Table 2-1: Health Benefits Achieved Through Implementation of the Plan

Health Impact	Health Benefit (reduction in health impact)
Premature Death	671
Acute Myocardial Infarction, Hospital Admissions	93
Asthma Age 0-19, Hospital Admissions	131
Cardiovascular, Hospital Admissions	175
Asthma Age 20-99, Hospital Admissions	246
Asthma Age 20-99, Emergency Room Visits	407
Asthma Age 0-19, Emergency Room Visits	699
Acute Bronchitis	1,498
Upper Respiratory Symptoms	15,523
Lower Respiratory Symptoms	19,011
Asthma Exacerbation	114,376
Work Loss Days	125,138

In addition to quantifying reductions in disease and death based on improvements in county-level PM_{2.5} concentrations, BenMAP can also be used as a tool to quantify the economic benefits associated with quantified health benefits. While assigning economic

values to health impacts is difficult given the tremendous social values associated with these impacts, BenMAP attempts to assign values using an existing body of literature that connects impacts to hard costs such as lost wages, or, in the case of premature death, a social value of \$7.99 million per incidence. Using this model, the District estimates that implementation of the plan will achieve an annual Valley-wide savings of \$102 million in health costs in 2019, as outlined in the following table. Additionally, and more significantly, a social benefit of \$5.36 billion is estimated for the 671 avoided premature deaths.

Table 2-2: Economic Costs Associated with Quantified Health Benefits

Health Impact	Health Benefit (reduction in health impact)
Hospital Admissions, Respiratory	\$386,955
Asthma, Emergency Room Visits	\$429,496
Acute Bronchitis	\$717,759
Acute Myocardial Infarction	\$3,081,686
Upper Respiratory Symptoms	\$3,677,083
Lower Respiratory Symptoms	\$4,502,994
Asthma, Hospital Admissions	\$6,279,033
Cardiovascular, Hospital Admissions	\$6,767,955
Asthma Exacerbation	\$10,660,442
Work Loss Days	\$19,539,004
Acute Respiratory Symptoms	\$46,109,557
Total	\$102,151,964

This page intentionally blank.