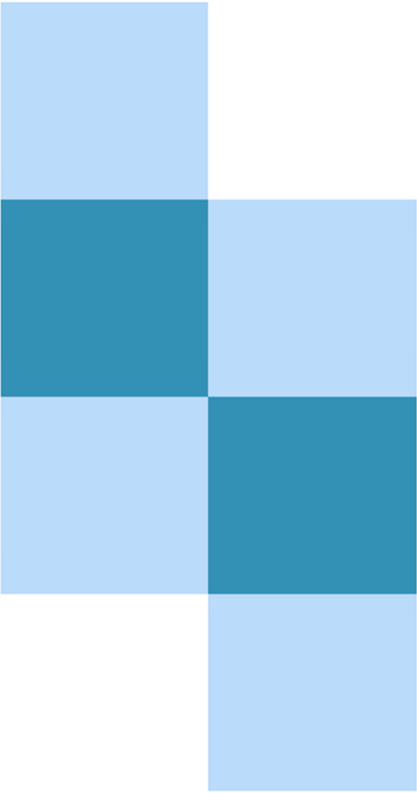




Chapter 2

Risk-Based Strategy



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Chapter 2: Risk-Based Strategy

[Note: The evaluation being conducted to develop this plan is an ongoing work in progress, and will continue to be revised and updated throughout the public process.]

2.1 WHAT IS THE RISK-BASED STRATEGY?

EPA's National Ambient Air Quality Standards (NAAQS) are a primary driving force for new emissions controls, air quality improvements, and resulting 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 to progress a region towards 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 sources 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 CAA framework and EPA standards. There is a growing recognition within the scientific community that the NAAQS alone can be incomplete measures for public exposure to air pollution. Thus, while the CAA's NAAQS-SIP process is motivated by public health, this 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 (Lippman, 2012 HEI Pres).

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 Risk-Based Strategy into the development of the *2012 PM_{2.5} Plan*. The purpose of this chapter is to more fully 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:

- First, as background, the key aspects of particulate pollution that shape human exposure and risk from PM_{2.5} will be briefly reviewed, along with a discussion of how the RBS is reflected in existing District programs.
- The subsequent section discusses in more detail these key health risk elements of PM_{2.5} particles and exposure.
- The third section outlines how these risk elements are employed in the risk characterization methodology that will be utilized in subsequent chapters for evaluating sources of PM_{2.5} and related control or incentive options.

2.2 BACKGROUND FOR THE RISK-BASED STRATEGY

EPA first established a PM_{2.5} NAAQS in 1997 to accompany the existing PM₁₀ NAAQS due to the mounting epidemiological evidence that PM_{2.5} was more harmful than PM₁₀. 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.” However, particulate inhalation studies found that smaller PM_{2.5} particles penetrate more deeply in the lungs, where particles more effectively avoid immune system defenses. Toxicological analyses of PM_{2.5} identified chemical species that acted 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 a multitude of studies that have largely identified which chemical species of PM_{2.5} are most harmful and their sources (2009 ISA). Health researchers have also documented the negative cardiovascular and immune system effects of ultrafine particles, or 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 PM_{2.5} 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 an important 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 end goal is a stronger, more health protective plan that reflects the current trajectory of scientific knowledge towards a more complete understanding of population risk from PM_{2.5} particles.

The NAAQS-SIP process and the RBS are complimentary strategies, and 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 PM_{2.5} while ignoring others. The current mass-based indicator (micrograms per cubic meter of air) will continue to serve as the final yardstick for PM_{2.5} attainment. As required under the CAA, the District is committed to attaining the 2006 PM_{2.5} standards as expeditiously as possible, and the District will not ignore sources of PM_{2.5} under its jurisdiction that could contribute to the Valley's attainment of the PM_{2.5} 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 been reducing harmful species of PM_{2.5} when and where 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 PM_{2.5} 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 by about 13% in Bakersfield and Fresno, resulting in an estimated 59 to 121 avoided cases of annual premature mortality (Lighthall et al, 2009).

Even though the *2008 PM_{2.5} Plan* was developed per EPA requirements for the 1997 PM_{2.5} standards (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 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 ultrafine particles (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 ultrafine particles that induce 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 to 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, 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 utilizes 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 more informed decisions and plan outdoor activities for times with the best air quality, reducing potential 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. 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 ultrafine particulates in Fresno, partnering with UCSF-Fresno to investigate the quantity and spatial distribution of ultrafine particle plumes from motor vehicles, lawn care equipment, wood burning, and restaurants.

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 will be integrating the RBS into various aspects of the *2012 PM2.5 Plan*. This integration is still in progress, and will be more fully developed in future drafts with public/stakeholder input and with continued analysis. The District expects to incorporate the Risk-based Strategy into this plan with the following:

- **Information regarding health effects of PM2.5:** Chapters 1 and 2 include detailed discussions regarding the different types of PM2.5, and their associated health effects.
- **Ambient data analysis:** In addition to evaluating PM2.5 mass trends per CAA and other EPA guidelines, Chapter 3 and Appendix A discuss PM2.5 species-based trends. This species analysis considered with health research will highlight which PM2.5 and PM2.5 precursor sources might be prioritized under the RBS. The District is also evaluating the timing of higher PM2.5 concentrations, to see if there are certain times of the day or times of the year when PM2.5 reductions might have more public health benefits.
- **Health Research:** The Risk-Based Strategy 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, District will be using EPA's BenMAP benefit estimation model to estimate the economic value of reduced population exposure resulting from the District's proposed attainment strategy.
- **Source-by-source assessment:** The District is conducting a thorough analysis of all potential opportunities to reduce emissions of directly-emitted PM2.5 and significant PM2.5 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/or 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 ultrafines**
5. **Population intake fraction**

This qualitative exposure assessment employed in this SIP is to be distinguished from 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 model in a SIP context is pervasive empirical uncertainty regarding the values of the different elements listed above. In considering the health impacts of a given source, even if the chemical composition of emissions, the geographic pattern and volume of its emissions, and the spatial distribution of the exposed population are known, it is very difficult to isolate and quantify the regional health impacts from that emissions source since 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. Recognizing these limitations, it is nevertheless possible to employ a simple but robust exposure characterization tool for making important qualitative and categorical distinctions regarding the relative contribution of a given source to population exposure and associated risk.

2.4.1 Relevance to Attainment

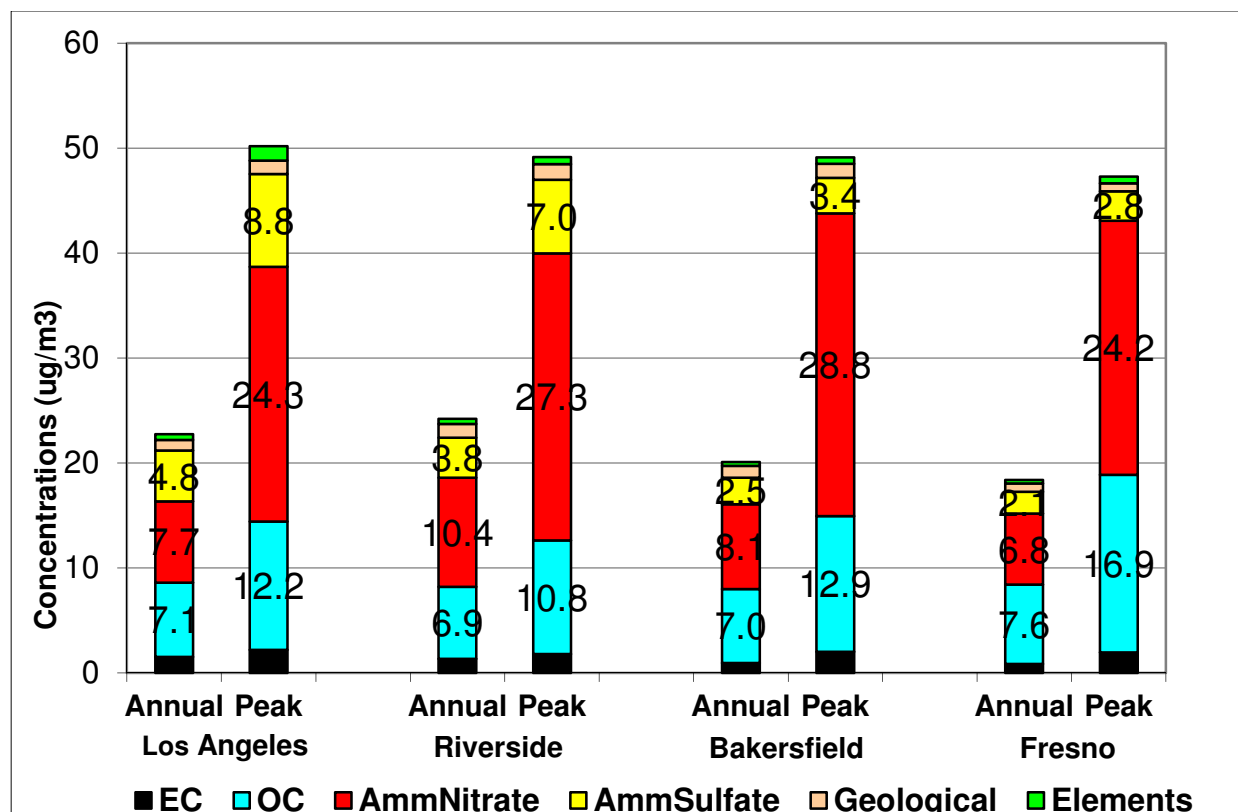
An important element of the Risk-based Strategy is the relevance of the emissions reductions to the Valley's attainment of EPA's health-based standards. This portion of the analysis will consider emissions type (such as PM_{2.5}, NO_x, or SO_x), seasonality of the emissions (since PM_{2.5} exceedance 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

PM2.5 particles vary in their toxicity depending on their chemical composition. PM2.5 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 noted these toxicity differences, epidemiological studies have found regional differences in health impacts despite comparable regional PM2.5 mass exposure (Bell, 2012). 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 (Pinkerton—OC and transition metals).

Many emissions sources evaluated in this plan are sources of direct (primary) PM2.5 emissions characterized by a unique combination of chemical species. Other sources emit chemical species such as ammonia and nitrogen oxides (NOx), precursors that contribute to the formation of secondary PM2.5 species. The PM2.5 chemical species categories adopted in the exposure characterization model include elemental carbon (carbon black), organic carbon compounds (OC), metals (elements), ammonium nitrate, ammonium sulfate, and geological (see Figure 2.1). PM2.5 is regularly speciated at several Valley monitoring sites (see Appendix A). The following discussion provides an overview of PM2.5 species and their associated health impacts.

Figure 2.1: Comparison of 2004-2006 Average Annual and Peak Day PM_{2.5} Chemical Composition (based on ARB data).



Organic carbon (OC): Organic carbon species found in PM_{2.5} aerosol are generated as primary organic aerosol (POA), predominantly via combustion of hydrocarbons. Key POA sources include cooking, industrial processes, mobile source exhaust, prescribed burning, tire wear, and wood burning (EPA, 2004 056905, ISA Chapter 3). 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, as well as 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 (Jiang et al., 2008, 156609, ISA 5-1). In addition, OC and metals have been shown to indirectly stimulate ROS production by macrophages, 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 (Landvik et al., 2007). 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 as well as within the body (in vivo), quinones have been shown to be one of the most important drivers of pulmonary oxidative stress, resulting a host of negative spillover effects on immune system functioning (Bolton et al., 2000). 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 (Ikeda et al, 2012).

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, carbon black 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 (Vogel et al, 2005). 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 (Vesterdal, et al., 2010). A recent study in Mexico City found an association between exposure levels of elemental carbon and lung function decrements among asthmatic and non-asthmatic children (Barraza-Villarreal et al., 2011).

Characterization of health effects of elemental carbon from human exposure studies is complicated by the high correlation between carbon black, OC, and metals emitted by diesel exhaust. To conclude, exposure to elemental carbon 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 (Premasekharan et al., 2011). 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.

There is a larger and even more definitive body of research relating exposure to metals in PM_{2.5} to cardiovascular effects, particularly nickel and vanadium. In respect to epidemiological research, a national study recently found that communities with higher

fractions of nickel, vanadium, and elemental carbon in their PM_{2.5} also had higher risk of cardiovascular and respiratory hospitalization (Bell et al., 2009). Specifically, cardiovascular hospitalizations were 26% higher in counties with a nickel fraction in the 75th percentile vs. 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 ships were identified as a principle cardiovascular risk factor (Lippmann et al., 2006). 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 (Chen and Lippmann, 2009).

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 (Lippmann 2012 HEI Presentation).

Ammonium nitrate: Ammonium nitrate (aka nitrate) is classified as a secondary inorganic species, i.e. it is not a directly emitted, primary source of PM_{2.5}, and it does not contain carbon. Ammonium 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 Fig. 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 as well as to average annual daily concentrations. The percentage contribution of nitrate to PM_{2.5} mass is substantially reduced in summer, with the 2000-06 Valley average for June ranging from 13 to 18%, with mass levels at or below 2 µg/m³ (CARB speciation data).

A recent cold season ammonia emissions inventory for the San Joaquin Valley estimated that motor vehicles and livestock production each contribute approximately 40% to total seasonal ammonia (Battye and Aneja, 2003). 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 a LD 50 (dose causing death for 50% of the exposed subjects) reported to be two thirds that of table salt. This raises the question as 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, as well as 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 via experimental and/or 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 preterm birth (Wilhelm et al., 2011). The authors point to other experimental studies that identified very high oxidative stress potential resulting from PAHs, metals, and other OC species collected from LA 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. Cassee et al. (2002) conducted an experimental study that exposed rats to high concentrations of nitrate (70 to 420 $\mu\text{g}/\text{m}^3$) and in combination with elemental carbon. 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 elemental carbon. 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 1, mass concentrations of ammonium sulfate are significantly lower than for nitrate in the Valley, averaging from 4 to 7% of PM_{2.5} mass during winter peaks and approximately 12% of annual averages. Fossil fuel combustion is the primary source of sulfate in this region but globally, coal combustion is the primary source. Unlike ammonium nitrate, mass concentrations of sulfate are not appreciably different in cold and hot seasons.

Research findings regarding the toxicity of ammonium sulfate are comparable to that of ammonium nitrate. Oral toxicity is low and it is approved as a food additive by the US Food and Drug Administration and the EU. Kulle et al. (1984) examined the response of 20 non-smoking subjects to 4 hr. 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 San Joaquin 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 4 to 8 months, modest pulmonary impacts were noted (Smith et al., 1989). After 4 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 was consistent with results of a review of the epidemiological and toxicological research on sulfate conducted by Riess et al. (2007). They 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: As seen in Figure 2.1, winter season and annual average PM_{2.5} found in the San Joaquin Valley contains a very small fraction of species that are termed “crustal”, i.e. having their origins in the earth’s crust. The so-called coarse fraction—PM_{2.5-10}--contains a much higher fraction, as do particles beyond the PM₁₀ size category. Suspended dusts consist mainly of oxides of aluminum, silicon, calcium, titanium, iron, and other metal oxides. The precise combination of these components depends on the geology and industrial/agricultural processes of the area. Geological material typically consists of 5 to 15 percent PM particles, a lower fraction than is reported in Figure 1.

Veranth et al. (2004) examined the respiratory inflammation potential of soil dust PM_{2.5} taken from 9 different sites in the western US taken from windblown dust and vehicle-generated particles from unpaved roads. None of the sites were located in the San Joaquin 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 5 of the sites was found to be benign, 3 of the sites demonstrated measurable cytokine response, and PM_{2.5} from 1 site was found to be highly reactive. Endotoxin, a potentially reactive bioaerosol 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 (RDD) has been recognized and analyzed as a separate form of PM_{2.5} that has relevance to exposure characterization of sources in this SIP. In this context, RDD 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. Rogge et al. (1993) conducted a speciation analysis of RDD in southern California that 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. RDD particles are resuspended by the 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 San Joaquin Valley makes a relatively small contribution to overall PM_{2.5} mass and, by itself, has relatively low toxicity. Respirable road dust, while not of geologic origins, has been reviewed here because of its relevance to subsequent exposure characterization of sources in subsequent chapters.

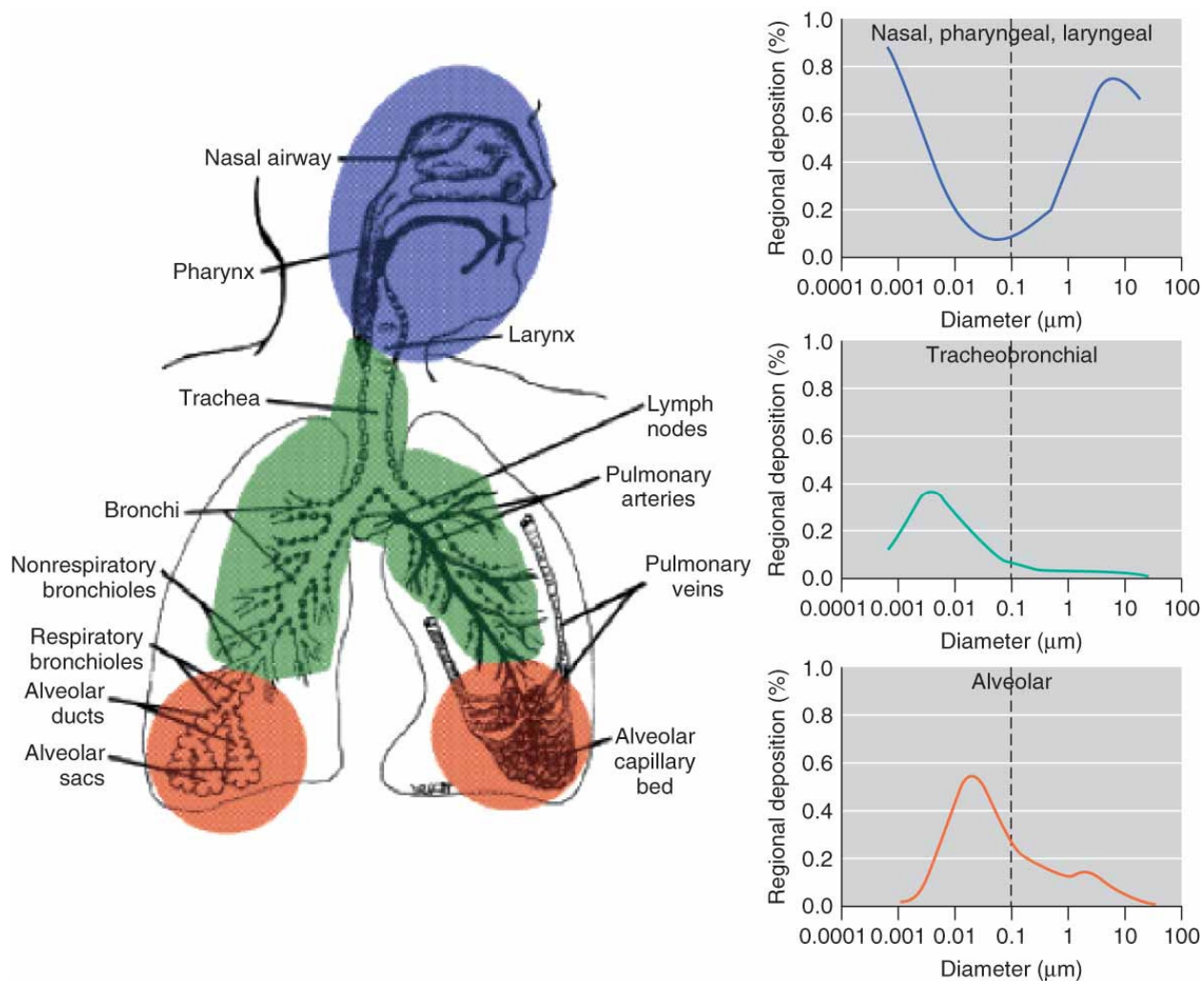
2.4.3 Particle Size and Deposition

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 (1) the percentage of inhaled particles that remain deposited and not exhaled (known as the deposition fraction) and (2) where particles are deposited within the body (ICRP, 1995). Within the PM_{2.5} size range, particles less than PM 0.1, known as ultrafine particles, and those greater than PM_{1.0} are least likely to be exhaled, and thus have higher deposition fractions (EPA 2004).

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

- A. Nasal, pharyngeal, laryngeal: As shown in the graph at the upper right, the uppermost segment of the respiratory tract is the primary zone of deposition for the smallest as well as the largest particles. Approximately 80% of extremely small particles of 1 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.
- 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.

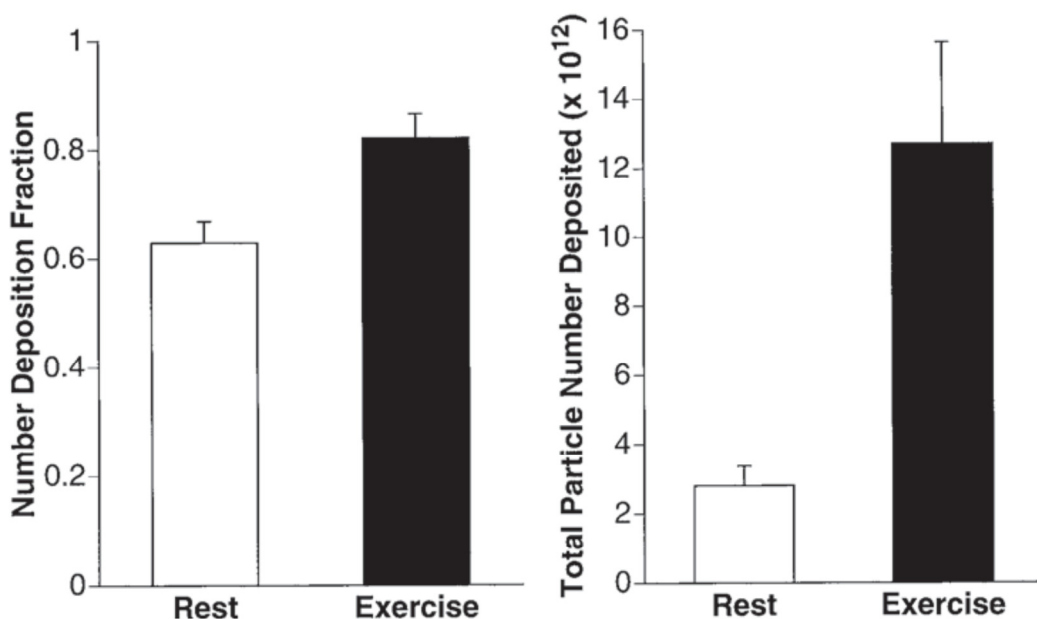
Figure 2-2: Relationships between particle size distribution and respiratory deposition zones



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 (Delfino, Li, Mohamed et al, 2012). At worst, these chemicals can trigger heart attacks and premature death among individuals with pre-existing heart conditions (cite). Extremely small particles can also be absorbed into the brain via the nasal tract, bypassing the protection provided by the blood-brain barrier (Oberdorster et al, 2004). The effects of particles deposited primarily in the tracheobronchial region center on respiratory function (2009 ISA, Dosimetry chapter).

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 study found that during moderate exercise, 80% of inhaled ultrafine particles were deposited in the lungs, compared with 60% lung retention while at rest (see left panel in Fig. 2-3). However, because the volume of air exchanged per minute increases substantially during exercise, overall PM 0.1 particle 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. (Source: Daigle et al., 2003)

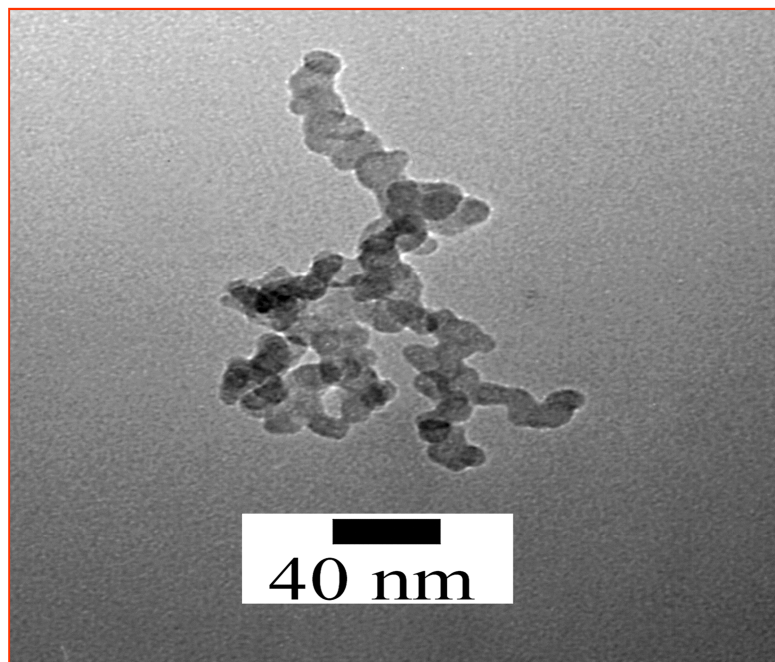


2.4.4 Exposure to Ultrafine Particles (PM 0.1)

Elevated exposure to freshly-emitted ultrafine particles is a critical health risk factor that often does not correspond to ambient PM_{2.5} concentrations at local monitors. Ultrafine particles are formed through nucleation and gas-to-particle reactions and grow (and shrink) through a number of mechanisms including condensation, coagulation, and volatilization (Solomon, 2012). High concentrations of primary (directly emitted) PM 0.1 are typically found near fresh sources of hydrocarbon combustion, including coal, charbroiled meat, diesel and gasoline vehicles, wood, meat charbroiling, 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 are 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 2 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 (Donaldson, 2001). 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 (Morawska et al., 2008). Chemical potency, very high surface area, and alveolar deposition are signal characteristics of ultrafine particles from hydrocarbon combustion that result in significant health risks from chronic exposure.

Figure 2-4: Electron micrograph of ultrafine particle. (Source: Nel et al., 2005)

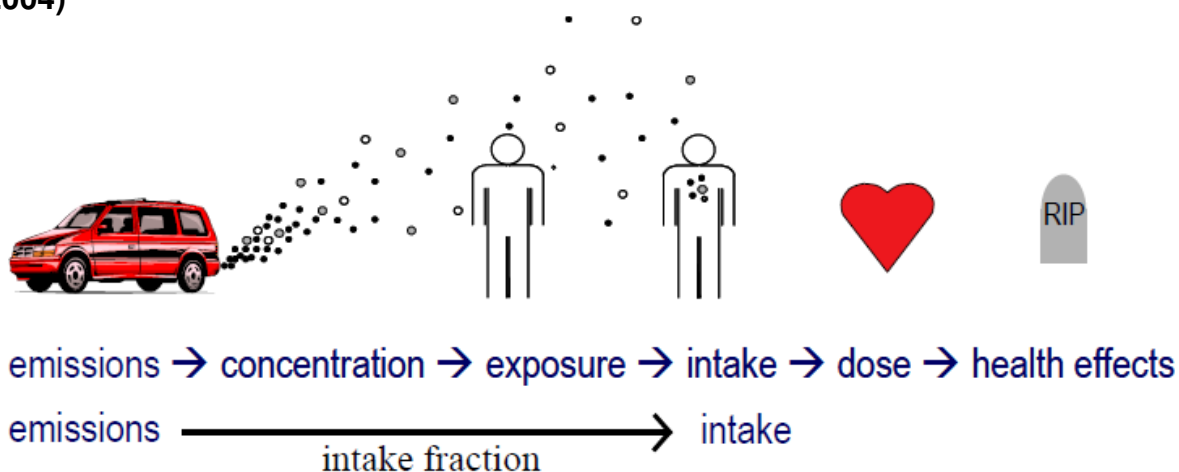


Sub-populations who live or work near sources of primary PM 0.1 from hydrocarbon combustion are particularly at risk. Health scientists have generated an overwhelming body of epidemiological (statistical) evidence that individuals near freeways, e.g. less than 300 meters, are being harmed via chronic inhalation of PM 0.1 from vehicles (e.g. Gauderman et al., 2007). Similarly, a recent study of residential wood burning in Cambria often 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 (Thatcher and Kirchsetter, 2011). The health risk from fresh sources of PM 0.1 PM 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 and/or socioeconomic status (London et al., 2011). 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 (Marshall and Nazaroff, 2004). 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 2-5: Simplified Intake Fraction Model (Source: Marshall and Nazaroff, 2004)



The relevance of the intake fraction concept can be seen in a recent study of neighborhood variability in wood smoke concentrations in Cambria California (Thatcher and Kirchsetter, 2011). 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 PM 2.5 measured by ambient monitors.