ERRATA NO. 2 TO THE FINAL ENVIRONMENTAL IMPACT REPORT

FOR THE CROSSROADS HOLLYWOOD PROJECT

State Clearinghouse No. 2015101073

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Crossroads Hollywood Project Final Environmental Impact Report

A. Introduction

In response to the California Supreme Court decision, *Sierra Club v. County of Fresno* (Friant Ranch, L.P.), S219783 (Friant Ranch decision), this Errata provides a supplemental discussion to the Crossroads Hollywood Final Environmental Impact Report (EIR). The Final EIR ("EIR") is comprised of: (1) the Draft EIR dated April 2017; (2) the comments received on the Draft EIR and the City's responses to them and associated revisions; (3) the August 2018 Errata; (5) this Errata; (6) the Mitigation Monitoring Plan; and (7) a list of persons, organization and public agencies commenting on the draft EIR.

On December 24, 2018, the California Supreme Court (Court) addressed the standard of review for claims challenging the legal sufficiency of an EIR's discussion of environmental impacts. *Sierra Club v. County of Fresno (Friant Ranch, L.P.*) (2018). In affirming in part the Court of Appeal's judgment, the Supreme Court held that the EIR for the Friant Ranch Project—a 942-acre master-planned, mixed-use development with over 2,500 senior residential units, 250,000 square feet of commercial space, and extensive open space/ recreational amenities on former agricultural land in north central Fresno County—was deficient in its informational discussion of the human health impacts of the Project's significant and unavoidable impacts related to air quality in particular.

The Court concluded on page 6 of the decision that an EIR's discussion must: (1) "include sufficient detail to enable those who did not participate in its preparation to understand and to consider meaningfully the issues the proposed project raises" (citing *Laurel Heights Improvement Assn. v. Regents of University of California* (1988) 47 Cal.3d 376, 405 ("*Laurel Heights I*")); and (2) "make[ ] a reasonable effort to substantively connect a project's air quality impacts to likely health consequences." It held that the EIR at issue there did neither, and "should be revised to relate the expected adverse air quality impacts to likely health consequences or explain in meaningful detail why it is not feasible at the time of drafting to provide such an analysis, so that the public may make informed decisions regarding the costs and benefits of the project."

The supplemental information set forth in this Errata does not constitute "significant new information" as that term is defined by CEQA Guidelines, section 15088.5.

Specifically, the supplemental information is not significant because the EIR is not changed in a way that deprives the public of a meaningful opportunity to comment upon a substantial adverse environmental effect of the Project. This supplemental information does not result in or disclose any new significant impacts or a substantial increase in the severity of any impact already identified in the Draft EIR or Final EIR. Thus, none of the conditions in Section 15088.5 of the CEQA Guidelines is met, and recirculation is not required.

B. Project Related Pollutant Emissions

1. Regional Emissions

The Draft EIR for Crossroads Hollywood (Crossroads Draft EIR) concludes that regional emissions would exceed SCAQMD's thresholds of significance even with incorporation of mitigation measures and such impacts were deemed significant and unavoidable. As discussed on page IV.B-56 of the Crossroads Draft EIR, mitigated peak daily regional construction NO_X emissions of 225 pounds per day would exceed the SCAQMD regional significance threshold for NO_X (100 pounds per day). Note that Mitigation Measure B-5 was further strengthened in the Final EIR to include the use of Tier 4 equipment during the grading/excavation/export phase of construction, which reduced peak daily NOx emissions to 193 pounds per day. The duration of this regional impact would be limited to approximately five months of the 48-month construction duration, or to 10 percent of the total construction period.

As shown in Table IV.B-6 on page IV.B-37 of the Crossroads Draft EIR, regional operational emissions associated with Project buildout would result in 101 pounds of VOC and 96 pounds of NO_X, which exceed the SCAQMD regional operational significance thresholds for VOC and NO_X (both thresholds are 55 pounds per day) even with incorporation of mitigation measures. An analysis of daily operational regional emissions of *existing* conditions without the Project versus with the Project was also conducted. The results of these calculations and associated SCAQMD thresholds are presented in Table IV.B-7 on page IV.B-38 of the Crossroads Draft EIR. As shown in Table IV.B-7 of the Crossroads Draft EIR, the net overall operational emissions associated with the Project under existing conditions would be greater in comparison to estimated emissions at Project buildout (2022). This decrease in emissions from 2015 to 2022 simply reflects cleaner newer vehicles in future years and not a change in the intensity of use of the Project. The Project under existing conditions would exceed the established SCAQMD threshold levels for VOC, NO_X and CO. This conclusion assumed that the Project could have been built in 2015, which is not based on reality as it would not have existed in 2015, and, therefore, these actual impacts could not occur. Thus, health effects related to CO for the Project under existing conditions are not discussed further herein.

2. Localized Emissions

The Friant Ranch Project is located within San Joaquin Valley Air Basin and, as such, the Friant Ranch Project EIR's air quality analysis was conducted consistent with the requirements of the San Joaquin Valley Unified Air Pollution Control District (SJVUAPCD). SJVUAPCD does not provide localized significance thresholds related to project-related on-site emissions. The Crossroads Hollywood Project is located within the South Coast Air Basin and, as such, the Crossroads Draft EIR air quality analysis was conducted consistent with the requirements of the South Coast Air Quality Management District (SCAQMD). The SCAQMD provides localized significance thresholds related to on-site emissions.

The Crossroads Draft EIR provides a localized construction air quality analysis which was conducted using the methodology promulgated by the SCAQMD. Look-up tables provided by the SCAQMD were used to determine localized construction emissions thresholds for the Project.[1] LSTs represent the maximum emissions from on-site sources from a project that are not expected to cause or contribute to an exceedance of the most stringent applicable federal or State ambient air quality standard and are based on the most recent background ambient air quality monitoring data (2013–2015) for the Project area presented in Table IV.B-2 on page IV.B-21 of the Crossroads Draft EIR. Although the data shown in Table IV.B-2 demonstrates that ambient air quality is improving in the area, the localized construction emissions analysis did not apply a reduction in background pollutant concentrations for subsequent years of construction (i.e., 2018–2021). By doing so, the analysis was more conservative.

Maximum on-site daily construction emissions for NO_X, CO, PM_{10}, and $PM_{2.5}$ were calculated using CalEEMod and compared to the applicable SCAQMD LSTs for the area (SRA 1). Potential impacts were evaluated at the Larchmont Charter School West immediately east of Development Parcel D. Based on the LST methodology, potential impacts at the adjacent school were evaluated using the 25-meter mass rate LST lookup tables.[2] The assumed 25 meter distance is conservative as the vast majority of construction activities would be located farther away from the Project Site boundary.

The maximum daily localized emissions from Project construction and the LSTs are presented in Table IV.B-5 on page IV.B-35 of the Crossroads Draft EIR. As presented in Table IV.B-5, maximum localized construction emissions for off-site sensitive receptors

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[1] *SCAQMD, LST Methodology Appendix C-Mass Rate LST Look-up Table, revised October 2009.*

[2] *As stated on Page 3-3 of the LST methodology, "[T]he closest receptor distance on the mass rate LST lookup tables is 25 meters. It is possible that a project may have receptors closer than 25 meters. Projects with boundaries located closer than 25 meters to the nearest receptor should use the LSTs for receptors located at 25 meters.*

would not exceed any of the SCAQMD-recommended localized screening thresholds. As a result, the Project would not result in predicted ambient concentrations in the vicinity of the Project Site greater than the most stringent ambient air quality standards for CO,[3] NO_2,[4] and PM_{10} or $PM_{2.5}$.[5] Therefore, localized construction emissions from on-site sources resulting from the Project would result in a less-than-significant air quality impact.

Emissions estimates for criteria air pollutants from operational on-site sources were presented in Table IV.B-8 on page IV.B-39 of the Crossroads Draft EIR. As shown in Table IV.B-8 on page IV.B-39 of the Crossroads Draft EIR, on-site operational emissions would not exceed any of the LSTs. Therefore, localized operational emissions from on-site sources resulting from the Project would result in a less-than-significant air quality impact.

C. Air Quality and Health Effects

A discussion of air pollution and potential health effects was provided on pages IV.B-2 through IV.B-6 of the Crossroads Draft EIR. In addition, the national and state criteria pollutants and the applicable ambient air quality standards were listed in Table IV.B-1 on page IV.B-3 of the Crossroads Draft EIR.

Ambient air pollution is a major public health concern. Excess deaths and increases in illnesses associated with high air pollution levels have been documented in several episodes as early as 1930 in Meuse Valley, Belgium; 1948 in Donora, Pennsylvania; and 1952 in London. Although the levels of pollutants that occurred during these acute episodes are now unlikely in the United States, ambient air pollution continues to be linked to increases in respiratory illness (morbidity) and increases in death rates (mortality).

Air pollution has many effects on the health of both adults and children. Over the past several years, the incidence of a number of diseases has increased greatly. Asthma is perhaps the most important disease with an increasing incidence, but other diseases, such as allergic reactions, bronchitis and respiratory infections, also have been increasing. The cause of these increases may be due at least in part to the effects of air pollution.

The adverse health effects associated with air pollution are diverse and include:

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[3] *20 ppm [23,000 µg/m^3] over a 1-hour period or 9.0 ppm [10,350 µg/m^3] averaged over an 8-hour period.*

[4] *0.18 ppm [339 µg/m^3] over a 1-hour period, 0.1 ppm [188 µg/m^3] over a three-year average of the 98th percentile of the daily maximum 1-hour average, or 0.03 ppm [57 µg/m^3] averaged over an annual period).*

[5] *10.4 µg/m^3 or 1.0 µg/m^3 PM_{10} averaged over an annual period.*

- Increased mortality;
- Increased health care utilization (hospitalization, physician and emergency room visits);
- Increased respiratory illness (symptoms, infections, and asthma exacerbation);
- Decreased lung function (breathing capacity);
- Lung inflammation;
- Potential immunological changes;
- Increased airway reactivity to a known chemical exposure—a method used in laboratories to evaluate the tendency of airways to have an increased possibility of developing an asthmatic response; and
- A decreased tolerance for exercise.

The evidence linking these effects to air pollutants is derived from population based observational and field studies (epidemiological) as well as controlled laboratory studies involving human subjects and animals. There have been an increasing number of studies focusing on the mechanisms (that is, on learning how specific organs, cell types, and biochemicals are involved in the human body's response to air pollution) and specific pollutants responsible for individual effects. Yet the underlying biological pathways for these effects are not always clearly understood.

Although individuals inhale pollutants as a mixture under ambient conditions, the regulatory framework and the control measures developed are mostly pollutant-specific. This is appropriate, in that different pollutants usually differ in their sources, their times and places of occurrence, the kinds of health effects they may cause, and their overall levels of health risk. Different pollutants, from the same or different sources, may sometimes act together to harm health more than they would acting separately. Nevertheless, as a practical matter, health scientists, as well as regulatory officials, usually must deal with one pollutant at a time in determining health effects and in adopting air quality standards. To meet the air quality standards, comprehensive plans are developed such as the Air Quality Management Plan (AQMP) and the Air Toxics Control Plan (ATCP). These plans examine multiple pollutants, cumulative impacts, and transport issues related to attaining healthful air quality. A brief overview of the effects observed and attributed to various air pollutants is presented below.

Certain air pollutants have been recognized to cause notable health problems and consequential damage to the environment either directly or in reaction with other pollutants, due to their presence in elevated concentrations in the atmosphere. Such pollutants have

been identified and regulated as part of the overall endeavor to prevent further deterioration and facilitate improvement in air quality within the Air Basin. The criteria air pollutants for which national and state standards have been promulgated and which are most relevant to current air quality planning and regulation in the Air Basin include ozone (O_3), respirable particulate matter (PM_{10}), fine particulate matter ($PM_{2.5}$), carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and lead. In addition, toxic air contaminants (TAC) and global climate change related greenhouse gases (GHG) are of concern in the Air Basin.

Further discussion of the health effects due to exposure of pollutants exceeding SCAQMD's signficance thresholds (i.e., regional VOC and NO_X) is provided below and is substantially drawn from reviews presented in the SCAQMD's Final 2016 Air Quality Management Plan, Chapter 2 (Air Quality and Health Effects), March 2017.

1. Nitrogen Dioxide (NO_2)

NO_2 is a byproduct of fuel combustion and major sources include power plants, large industrial facilities, and motor vehicles. NO_2 is a gaseous air pollutant that serves as an indicator of gaseous oxides of nitrogen, such as nitric oxide (NO) and other related compounds (NO_X). NO_2 absorbs blue light and results in a brownish-red cast to the atmosphere and reduced visibility. NO_2 also contributes to the formation of PM_{10}. Nitrogen oxides irritate the nose and throat, and increase one's susceptibility to respiratory infections, especially in people with asthma. NO_X is also a precursor to the formation of ozone.

The U.S. EPA in 2010 retained the existing standards of 53 ppb for NO_2 averaged over one year and adopted a new short-term standard of 100 ppb (0.1 ppm) averaged over one hour. The standard was designed to protect against increases in airway reactivity in individuals with asthma based on controlled exposure studies, as well as respiratory symptoms observed in epidemiological studies. The revised standard also requires additional monitoring for NO_2 near roadways.

The adverse effects of ambient nitrogen dioxide air pollution exposure on health were reviewed in the 2008 U.S. EPA Integrated Science Assessment for Oxides of Nitrogen—Health Criteria,[6] and more recently in the 2016 U.S. EPA Integrated Science

[6] *U.S. EPA. (2008). Integrated Science Assessment for Oxides of Nitrogen—Health Criteria (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-08/071. http://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=194645.*

Assessment for Oxides of Nitrogen—Health Criteria.[7] The 2016 U.S. EPA review noted the respiratory effects of NO_2, and evidence suggestive of impacts on cardiovascular health, mortality and cancer.

Experimental studies have found that NO_2 exposures increase responsiveness of airways, pulmonary inflammation, and oxidative stress, and can lead to the development of allergic responses. These biological responses provide evidence of a plausible mechanism for NO_2 to cause asthma. Additionally, results from controlled exposure studies of asthmatics demonstrate an increase in the tendency of airways to contract in response to a chemical stimulus (airway responsiveness) or after inhaled allergens. Animal studies also provide evidence that NO_2 exposures have negative effects on the immune system, and therefore increase the host's susceptibility to respiratory infections. Epidemiological studies showing associations between NO_2 levels and hospital admissions for respiratory infections support such a link, although the studies examining respiratory infections in children are less consistent.

The Children's Health Study in Southern California found associations of NO_2 with respiratory symptoms in asthmatics.[8] Particles and NO_2 were correlated, and it was determined that NO_2 plays a stronger role. Ambient levels of NO_2 were also associated with a decrease in lung function growth in a group of children followed for eight years. In addition to NO_2, the decreased growth was also associated with particulate matter and airborne acids. The study authors postulated that these may be a measure of a package of pollutants from traffic sources.

Results from controlled exposure studies of asthmatics demonstrated an increase in the tendency of airways to contract in response to a chemical stimulus (bronchial reactivity). Effects were observed with an exposure to 0.3 ppm NO_2 for a period ranging from 30 minutes to 3 hours. A similar response is reported in some studies with healthy subjects at higher levels of exposure (1.5 - 2.0 ppm). Mixed results have been reported when people with chronic obstructive lung disease are exposed to low levels of NO_2.

A more detailed discussion of the health effects of NO_2 is provided in Attachment 1 to this memorandum (SCAQMD Final 2016 Air Quality Management Plan, Appendix I: Health Efffects).

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[7] *U.S. EPA. (2016). Integrated Science Assessment for Oxides of Nitrogen—Health Criteria (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-15/068. https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=310879.*

[8] *McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. (2002). "Asthma in exercising children exposed to ozone: a cohort study." Lancet, 359:386-91.*

2. Ozone (O_3)

Ozone is a gas that is formed when volatile organic compounds (VOCs) and NO_X—both byproducts of internal combustion engine exhaust—undergo slow photochemical reactions in the presence of sunlight. Ozone concentrations are generally highest during the summer months when direct sunlight, light wind, and warm temperature conditions are favorable. Ozone is one of the most important air pollutants affecting human health in regions like Southern California. Ozone is a molecule built of three atoms of oxygen linked together in a very energetic combination. When ozone comes into contact with a surface it rapidly releases this extra force in the form of chemical energy. When this happens in biological systems, such as the respiratory tract, this energy can cause damage to sensitive tissues in the upper and lower airways.

The major subgroups of the population considered to be at increased risk from ozone exposure are outdoor exercising individuals including children and people with preexisting respiratory disease(s) such as asthma. The data base identifying the former group as being at increased risk to ozone exposure is much stronger and more quantitative than that for the latter group, probably because of a larger number of studies conducted were with healthy individuals. The adverse effects reported with short-term ozone exposure are greater with increased activity because activity increases the breathing rate and the volume of air reaching the lungs, resulting in an increased amount of ozone reaching the lungs. Children may be a particularly vulnerable population to air pollution effects because they spend more time outdoors, are generally more active, and have a higher ventilation rate than adults.

A number of adverse health effects associated with ambient ozone levels have been identified from laboratory and epidemiological studies. These include increased respiratory symptoms, damage to cells of the respiratory tract, decreases in lung function, increased susceptibility to respiratory infection, and increased risk of hospitalization.

The Children's Health Study, conducted by researchers at the University of Southern California, followed a cohort of children that live in 12 communities in southern California with differing levels of air pollution for several years. A publication from this study found that school absences in fourth graders for respiratory illnesses were associated with ambient ozone levels. An increase of 20 ppb ozone was associated with an 83 percent increase in illness related absence rates (Gilliland, 2001).[9]

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[9] *Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. (2001). "The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illnesses." Epidemiology, 12(1):43-54.*

The number of hospital admissions and emergency room visits for all respiratory causes (infections, respiratory failure, chronic bronchitis, etc.) including asthma show a consistent increase as ambient ozone levels increase in a community. These excess hospital admissions and emergency room visits are observed when hourly ozone concentrations are as low as 0.08 to 0.10 ppm.

Numerous recent studies have found positive associations between increases in ozone levels and excess risk of mortality. These associations persist even when other variables including season and levels of particulate matter are accounted for. This indicates that ozone mortality effects are independent of other pollutants.[10]

Several population-based studies suggest that asthmatics are more adversely affected by ambient ozone levels, as evidenced by increased hospitalizations and emergency room visits. Laboratory studies have attempted to compare the degree of lung function change seen in age and gender-matched healthy individuals versus asthmatics and those with chronic obstructive pulmonary disease. While the degree of change evidenced did not differ significantly, that finding may not accurately reflect the true impact of exposure on these respiration-compromised individuals. Since the respiration-compromised group may have lower lung function to begin with, the same degree of change may represent a substantially greater adverse effect overall.

A publication from the Children's Health Study focused on children and outdoor exercise. In communities with high ozone concentrations, the relative risk of developing asthma in children playing three or more sports was found to be over three times higher than in children playing no sports.[11] These findings indicate that new cases of asthma in children are associated with heavy exercise in communities with high levels of ozone. While it has long been known that air pollution can exacerbate symptoms in individuals with respiratory disease, this is among the first studies that indicate ozone exposure may be causally linked to asthma.

Some lung function responses (volume and airway resistance changes) observed after a single exposure to ozone exhibit attenuation or a reduction in magnitude with repeated exposures. Although it has been argued that the observed shift in response is evidence of a probable adaptation phenomenon, it appears that while functional changes may exhibit adaptation, biochemical and cellular changes which may be associated with

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[10] *Bell ML, McDermott A, Zeger SL, Samet, JM, Dominici, F. (2004). "Ozone and Short-Term Mortality in 95 US Urban Communities, 1987–2000." JAMA 292:2372-2378.*

[11] *McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. (2002). "Asthma in exercising children exposed to ozone: a cohort study." Lancet, 359:386-91.*

episodic and chronic exposure effects may not exhibit similar adaptation. That is, internal damage to the respiratory system may continue with repeated ozone exposures, even if externally observable effects (chest symptoms and reduced lung function) disappear.

In a laboratory, exposure of human subjects to low levels of ozone causes reversible decrease in lung function as assessed by various measures such as respiratory volumes, airway resistance and reactivity, irritative cough and chest discomfort. Lung function changes have been observed with ozone exposure as low as 0.08 to 0.12 ppm for 6-8 hours under moderate exercising conditions. Similar lung volume changes have also been observed in adults and children under ambient exposure conditions (0.10 - 0.15 ppm). The responses reported are indicative of decreased breathing capacity and are reversible.

In laboratory studies, cellular and biochemical changes associated with respiratory tract inflammation have also been consistently reported in the airway lining after low level exposure to ozone. These changes include an increase in specific cell types and in the concentration of biochemical mediators of inflammation and injury such as cytokines and fibronectin. These inflammatory changes can be observed in healthy adults exposed to ozone in the range of 0.08 to 0.10 ppm.

The susceptibility to ozone observed under ambient conditions could be due to the combination of pollutants that coexist in the atmosphere or ozone may actually sensitize these subgroups to the effects of other pollutants. Some animal studies show results that indicate possible chronic effects including functional and structural changes of the lung. These changes indicate that repeated inflammation associated with ozone exposure over a lifetime may result in sufficient damage to respiratory tissue such that individuals later in life may experience a reduced quality of life in terms of respiratory function and activity level achievable. An autopsy study involving Los Angeles County residents provided supportive evidence of lung tissue damage (structural changes) attributable to air pollution. A study of birth outcomes in southern California found an increased risk for birth defects in the aortic and pulmonary arteries associated with ozone exposure in the second month of pregnancy.[12] This is the first study linking ambient air pollutants to birth defects in humans. Confirmation by further studies is needed. In summary, acute adverse effects associated with ozone exposures have been well documented, although the specific causal mechanism is still somewhat unclear. Additional research efforts are required to evaluate the long-term effects of air pollution and to determine the role of ozone in influencing chronic effects.

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[12] *Ritz B, Yu F, Chapa G, Fruin S. (2000). "Effect of Air Pollution on Preterm Birth Among Children Born in Southern California between 1989 and 1993." Epidemiology, 11(5)502-11.*

A more detailed discussion of the health effects of O_3 is provided in Attachment 1 to this memorandum (SCAQMD Final 2016 Air Quality Management Plan, Appendix I: Health Efffects).

D. Adverse Air Quality Impacts and Health Effects

In its Friant Ranch decision, the California Supreme Court conceded that the an explanation of the connection between an individual project's pollutant emissions in excess of thresholds and human health effects may not be possible given the current state of environmental science modeling. However, the California Supreme Court concluded that the Friant Ranch Project EIR itself must explain, in a manner reasonably calculated to inform the public, the scope of what is and is not yet known about the effect of the Project's significant and unavoidable air quality impacts on human health. The specific language provided by the Court is provided below.

> *The EIR fails to provide an adequate discussion of health and safety problems that will be caused by the rise in various pollutants resulting from the Project's development. At this point, we cannot know whether the required additional analysis will disclose that the Project's effects on air quality are less than significant or unavoidable, or whether that analysis will require reassessment of proposed mitigation measures. Absent an analysis that reasonably informs the public how anticipated air quality effects will adversely affect human health, an EIR may still be sufficient if it adequately explains why it is not scientifically feasible at the time of drafting to provide such an analysis.*

The following information is provided to comply with the Court's opinion by adequately explaining why it is not scientifically feasible at the time of drafting the Crossroads EIR to provide an analysis explaining the connection between the Project's regional pollutant emissions and human health. As noted above, the Crossroads EIR does provide a detailed analysis of localized emissions, which was not included as part of the Friant Ranch EIR. SJVUAPCD and SCAQMD have provided amicus briefs explaining the difficulties in providing a correlation between regional pollutant emissions and human health. The complete amicus briefs are included to this memorandum as Attachment 2.

1. SCAQMD Amicus Brief

With regard to the analysis of air quality-related health impacts, the SCAQMD, the air quality authority for the South Coast Air Basin, has stated that "EIRs must generally quantify a project's pollutant emissions, but in some cases it is not feasible to correlate these emssions to specific, quantifiable health impacts (e.g., premature mortality; hospital

admissions)." In such cases, a general description of the adverse health impacts resulting from the pollutants at issue may be sufficient.

The SCAQMD has further stated that from a scientific standpoint, it takes a large amount of additional precursor emissions to cause a modeled increase in ambient ozone levels over an entire region. For example, the SCAQMD's 2012 AQMP showed that reducing NO_X by 432 tons per day and reducing VOC by 187 tons per day would only reduce ozone levels at the SCAQMD's monitor site with the highest levels by only 9 parts per billion.[13] SCAQMD staff does not currently know of a way to accurately quantify ozone-related health impacts caused by NO_X or VOC precursor emissions from relatively small projects.

SCAQMD furher acknowledges that it may be feasible to analyze air quality related health impacts for projects on a regional scale with very high emissions of NO_X and VOCs, where impacts are regional. The example SCAQMD provided was for proposed Rule 1315, which authorized various newly-permitted sources to use offsets from the District's "internal bank" of emission reductions. The CEQA analysis accounted for essentially all of the increases in emisssions due to new or modified sources in the District between 2010 and 2030, or approximately 6,620 pounds per day of NO_X and 89,947 pounds per day of VOC, to expected health outcomes from ozone and parituclate matter (e.g., 20 premature deaths per year and 89,947 school absenses in the year 2030 due to zone).[14]

Based on the above information, at the project level, the Crossroads Hollywood Project would represent a relatively small project, since peak daily construction regional emissions of 93 pounds per day over the SCAQMD's signficance threshold represent approximately 1.4 percent of the emissions analyzed by SCAQMD related to Rule 1315. The Crossroads Hollywood Project's regional operational emissions would result in approximately 46 pounds of VOC and 41 pounds of NO_X over the SCAQMD's significance thresholds, or approximately 0.05 and 0.6 percent of the emissions analyzed by SCAQMD related to Rule 1315, respectively. Furthermore, approximately 95 percent of the Project's construction NO_X emissions and 90 percent of the its operational NO_X emissions would be regional (e.g., emitted by mobile sources distributed across region's roadway network) and different than the identified stationary sources as modeled in SCAQMD's analysis of Rule

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[13] *SCAQMD, Final 2012 AQMP, February 2013, www.aqmd.gov/home/library/clean-air-plans/air-quality-mgt-plan/final-20212-air-quality-management-plan; then follow "Appendix V: Modelling & Attainment Demonstrations" hyperlink, pp. v-4-2, v-7-4, v-7-24.*

[14] *The SCAQMD was able to establish the location of future NO_X and VOC emissions by assuming that new projects would be built in the same locations and proportions as existing stationary sources. This CEQA document was upheld by the Los Angeles County Superior Court in Natural Res. Def. Council v. SCAQMD, Los Angeles Superior Court No. BS110792.*

1315, which would add to the difficulties of modeling Project-related emissions. To provide additional context to the Crossroad pollutant emssions, the SCAQMD's 2016 AQMP provides 162.4 tons per day (324,800 pounds) of VOC and 293.1 tons per day (586,200 pounds) of NO_X emissions basinwide for the baseline year of 2012.[15]

Since SCAQMD staff does not currently know of a way to accurately quantify ozone-related health impacts caused by NO_X or VOC emissions from relatively small projects like the Project, then a general description of the adverse health impacts resulting from the pollutants at issue is all that can be provided at this time. Please see the above description of general adverse health impacts resulting from NO_X and VOC.

2. SJVUAPCD Amicus Brief

The SJVUAPCD amicus brief (see Attachment 2) addresses whether it is scientifically feasible to correlate an individual project's air quality emissions to specific health impacts. Human health impacts associated with criteria pollutants are analyzed and taken into consideration when the EPA sets the national ambient air quality standard (NAAQS) for each criteria pollutant (42 U.S.C. § 7409(b)(1)). The health impact of a particular criteria pollutant is analyzed on a regional, not a facility level, based on how close the area is to complying with (attaining) the NAAQS. As discussed by the SJVUAPCD, it is not feasible to conduct a criteria air pollutant analysis detailing health impacts, as currently available computer modeling tools are not equipped for this task.

In requiring a health risk type analysis for criteria air pollutants, it is important to understand how the relevant criteria pollutants (ozone and particulate matter) are formed, dispersed and regulated. Ground level ozone (smog) is not directly emitted into the air, but is instead formed when precursor pollutants such as NO_X and VOC are emitted into the atmosphere and undergo complex chemical reactions in the process of sunlight. Once formed, ozone can be transported long distances by wind.[16] Because of the complexity of ozone formation, a specific tonnage amount of NO_X or VOCs emitted in a particular area does not equate to a particular concentration of ozone in that area. In fact, even rural areas that have relatively low tonnages of emissions of NO_X or VOC can have high levels of ozone concentrations simply due to wind transport. Conversely, areas that have substantially more NO_X and VOC emissions could experience lower concentrations of

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[15] *SCAQMD, Final 2016 AQMP, Figure 3-1, March 2017, www.aqmd.gov/docs/default-source/clean-air-plans/air-quality-management-plans/2016-air-quality-management-plan/final-2016-aqmp/final2016aqmp.pdf?sfvrsn=15.*

[16] *U.S. EPA, Ground-level Ozone: Basic Information, www.epa.gov/airquality/ozonepollution/basic.html.*

ozone simply because sea breezes disperse the emissions.[17] Secondary particulate matter (PM), like ozone, is formed via complex chemicals such as sulfur dioxides (SO_X) and NO_X.[18] Because of the complexity of secondary PM formation, the tonnage of PM-forming precursor emissions in an area does not necessarily result in an equivalent concentration of secondary PM in that area.

The disconnect between the tonnage of precursor pollutants and the concentration of ozone or PM formed is important because it is not necessarily the tonnage of precursor pollutants that causes human health effects; rather, it is the concentration of resulting ozone or PM that causes these effects. Indeed, the NAAQS, which are statutorily required to be set by USEPA at levels that are requisite to protect the public health, are established as *concentrations of ozone* and not as tonnages of their precursor pollutants. Because the NAAQS are focused on achieving a particular concentration region-wide, the SJVUAPCD's tools and plans for attaining the NAAQS are regional in nature.

The computer models used to simulate and predict an attainment date for ozone are based on regional inventories of precursor pollutants and meteorology within the air basin. At a very basic level, the models simulate future ozone levels based on predicted changes in precursor emissions basin wide. The computer models are not designed to determine whether the emissions generated by an individual development project will affect the date that the air basin attains the NAAQS. Instead, the models help inform regional planning strategies based on the extent all of the emission-generating sources within the air basin must be controlled in order to reach attainment.

In the case of the Crossroads Hollywood Project, regional construction and operational emissions exceed the SCAQMD's recommended daily significance thresholds for NO_X and for NO_X and VOC, respectively. However, this does not mean that one can easily determine the concentration of ozone that will be created at or near the Project Site on a particular day or month of the year, or the specific human health impacts that may occur. Meteorology, the presence of sunlight, and other complex chemical factors all combine to determine the ultimate concentrations and locations of ozone. This is especially true for a project like the Crossroads Hollywood Project, where most of the criteria pollutant emissions derive not from a single "point source," but from area wide sources (consumer products, paint, etc.) or mobile sources (cars and trucks) driving to, from and around the Project Site.

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[17] *SJVUAPCD, 2007 Ozone Plan, Executive Summary p. ES-6. www.valleyair.org/Air_Quality_Plans/AQ_Final_Adopted_Ozone2007.htm.*

[18] *U.S. EPA, Particulate Matter: Basic Information, www.epa.gov/airquality/pariclepollution/basic.html.*

In addition, it would be extremely difficult to model the impact on NAAQS attainment that these over-thresholds emissions from the Crossroads Hollywood Project may have. As discussed above, the currently available tools are equipped to model the impact of all emission sources in the air basin on attainment. According to the most recent EPA-approved SCAQMD basin wide emissions inventory, the VOC inventory is 162.4 tons per day (324,800 pounds) and 293.1 tons per day (586,200 pounds) of NO_X emissions for the baseline year of 2012.[19] Running the photochemical grid model used for predicting ozone attainment with the emissions soley from the Crossroads Hollywood Project (which equates to approximately two-tenths of one percent of the NO_X and VOC in the air basin) is not likely to yield valid information given the relative small scale involved.

E. Air Quality Conclusion

Consistent with the California Supreme Court's Friant Ranch decision, the above information provides additional details regarding the potential health effects from Crossroads Hollywood Project's significant and unavoidable criteria pollutant emissions. It also adequately explains why it is not scientifically feasible at the time of drafting of the EIR to substantively connect this individual Project's air quality impacts to likely health consequences so that the public may make informed decisions regarding the costs and benefits of the project.

F. Noise

With respect to potential noise impacts on human health, the City of Los Angeles (City) currently has not set specific noise limits beyond the City's Noise Regulations. However, the U.S. Occupational Safety and Health Administration (OSHA) and the State's Division of Occupational Safety and Health (also known as Cal/OSHA) have established the permissible noise exposure limits, primarily applicable to workers working in noisy environments. The noise exposure limitation is defined as exposure duration per day (for workers). Per Cal/OSHA, the permissible noise exposure for 8 hours would be 90 dBA (L_{eq}).[20] In addition, the United States Environmental Protection Agency (EPA) provides noise exposure limits for environmental noise with respect to hearing loss. The EPA has identified a noise level With Margin of Safety of 70 dBA (24-hr L_{eq}), based on a 24 hours

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[19] *SCAQMD, Final 2016 AQMP, Figure 3-1, March 2017, www.aqmd.gov/docs/default-source/clean-air-plans/air-quality-management-plans/2016-air-quality-management-plan/final-2016-aqmp/final2016aqmp.pdf?sfvrsn=15.*

[20] *Cal/OSHA, Title 8 Regulations, Subchapter 7. General Industry Safety Orders, Group 15. Occupational Noise, Article 105. Control of Noise Exposure, §5096. Exposure Limits for Noise, Table N-1 Permissible Noise Exposure.*

per day, 365 days per year, over a 40-year period, as exposure levels that produce no more than 5 dB noise-induced hearing damage.[21]

The noise levels generated by the Crossroads Hollywood Project during long-term operation at the off-site noise sensitive receptors would be well below the EPA's limit of 70 dBA (24-hour L_{eq}). Noise levels associated with the Project's construction activities would be short-term and intermittent, as construction equipment would be moving around the Project Site; as such, the EPA's limit would not be relevant. As analyzed in the Draft EIR, the estimated maximum mitigated noise levels due to the Project's construction activities would be 82.3 dBA (L_{eq}). Therefore, with the identified mitigation measures (Draft EIR Table IV.I-25), the Project's construction-related noise levels would be well below the Cal/OSHA permissible noise exposure of 90 dBA (L_{eq}). Moreover, as described in the Draft EIR (Draft EIR Page IV.I-35), the construction impact analysis was based on a worst-case scenarios, which assumes all pieces of construction equipment would be operating simultaneously and located at the construction area nearest the affected receptors. Specifically, the analysis assumes that up to 27 pieces of construction equipment (during the building construction phase) would be operating simultaneously within 150 feet of the affected sensitive receptors (Draft EIR Page IV.I-35), which likely would not occur. Therefore, the estimated construction noise levels at the off-site noise sensitive receptors are likely overstated, and actual noise levels would be lower. As such, Project-related construction noise levels would be unlikely to negatively affect human health as they would be below the noise limits, pursuant to Cal/OSHA noise exposure limit.

Potential human health effects due to vibration includes whole-body exposure to vibration forces. The American Conference of Governmental Industrial Hygienists (ACGIH) published recommendation for prevention of whole-body vibration. The ACGIH sets limit value of 0.9 m/s^2 (8 hour equivalent total value) as impact threshold for whole-body exposure to vibration forces. As analyzed in the Draft EIR, the estimated maximum vibration levels due to the Project's construction activities would be 99 VdB (approximately 0.15 m/s^2) at the nearest off-site receptor (Draft EIR Table IV.I-14), which would be well below the ACGIH limit value of 0.9 m/s^2. Therefore, Project-related vibration levels would be below the available vibration limits, as related to human health.

G. Air Toxics

In response to issues raised by commenters, though it is not required by law and is being provided here for informational purposes only, the City prepared a quantitative Health

[21] *EPA, Noise Effects Handbook: A Desk Reference to Health and Welfare Effects of Noise, July 1981, Table 2-1.*

Risk Assessment (HRA) assessing the potential impacts on human health of Project-related Diesel Particulate Matter (DPM) emissions, attached to this Errata as Attachment 3. The HRA analysis finds that, for carcinogenic exposures, the increase in risk associated with the Project is calculated to be 4.7 in one million, which is less than the applicable threshold of 10 in one million for sensitive receptors in close proximity to the Project Site, resulting in a less than significant impact. For chronic non-carcinogenic exposures, the increase in the respiratory hazard index associated with the Project was estimated to be less than the applicable threshold of one for sensitive receptors in close proximity to the project site, also resulting in a less than significant impact. The HRA thus confirms the conclusion of the Draft EIR that Project-related DPM emissions would not result in significant environmental impacts.

Attachments

Attachment 1

SCAQMD 2016 AQMP Appendix I

SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT

Appendix I

Health Effects

2016 AIR QUALITY MANAGEMENT PLAN

March 2017

FINAL 2016 AQMP
APPENDIX I

HEALTH EFFECTS

MARCH 2017

SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT
GOVERNING BOARD

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Speaker of the Assembly Appointee

Vice Chairman: BEN BENOIT
Mayor Pro Tem, Wildomar
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County of Riverside

JOE BUSCAINO
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County of Orange

DR. CLARK E. PARKER, SR.
Senate Rules Committee Appointee

DWIGHT ROBINSON
Councilmember, Lake Forest
Cities of Orange County

JANICE RUTHERFORD
Supervisor, Second District
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And

In consultation with:
California Air Resources Board

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INTRODUCTION

This document presents a summary of scientific findings on the health effects of ambient air pollutants. The California Health and Safety Code Section 40471(b) requires that the South Coast Air Quality Management District (SCAQMD) prepare a report on the health impacts of particulate matter in the South Coast Air Basin (SCAB) in conjunction with the preparation of the Air Quality Management Plan (AQMP) revisions. This document, which was prepared to satisfy that requirement, also includes sections discussing the health effects of the other major pollutants. The intention of this document is to provide a brief summary of the conclusions of scientific reviews conducted by U.S. EPA and other scientific agencies, with some additional information from more recently published studies.

In addition to the air pollutant health effects summaries, there is an Attachment to this Appendix, which is a list of publications that have resulted from health-related research projects sponsored by SCAQMD over the past several years. Some of these studies are discussed in this Appendix, as appropriate, although there are many other studies referenced here. The studies funded by SCAQMD also help inform the SCAQMD's work in characterizing the air pollution and its effects in our local region and the influences of sources of air pollution in the Basin.

While information on ambient air quality statistics, attainment status, spatial distribution of air pollutants, environmental justice, socioeconomic impacts, control strategies, and cost-effectiveness are important issues that may relate to health effects, these issues are not the focus of this Appendix, and are instead discussed in detail in other chapters and appendices of the AQMP, or in the AQMP Socioeconomic Report.

HEALTH EFFECTS OF AIR POLLUTION

Ambient air pollution is a major public health concern. Excess deaths and increases in illnesses associated with high air pollution levels have been documented in several episodes as early as 1930 in Meuse Valley, Belgium; 1948 in Donora, Pennsylvania; and 1952 in London. Although levels of pollutants that occurred during these acute episodes are now unlikely in the United States, ambient air pollution continues to be linked to increases in illness and other health effects (morbidity) and increases in death rates (mortality).

Adverse health outcomes linked to air pollution include cardiovascular effects, premature mortality, respiratory effects, cancer, reproductive effects, neurological effects, and other health outcomes. The evidence linking these effects to air pollutants is derived from population-based observational and field studies (epidemiological), toxicological studies, as well as controlled laboratory studies involving human subjects and animals. There have been an increasing number of studies focusing on the mechanisms (that is, on learning how specific organs, cell types, and biomarkers are involved in

the human body's response to air pollution). Yet the underlying biological pathways for these effects are not always clearly understood.

Although individuals inhale pollutants as a mixture under ambient conditions, the regulatory framework and the control measures developed are pollutant-specific for six major outdoor pollutants covered under Sections 108 and 109 of the Clean Air Act. This is appropriate, in that different pollutants can differ in their sources, their times and places of occurrence, the kinds of health effects they may cause, and their overall levels of health risk. Different pollutants, from the same or different sources, oftentimes occur together. While the combined effects of multiple air pollutants that occur simultaneously may be important, the air quality standards address each criteria pollutant separately, and thus, this Appendix is divided into sections by pollutant. To meet the air quality standards, comprehensive plans are developed such as the Air Quality Management Plan (AQMP); and to minimize exposure to toxic air contaminants in the South Coast AQMD, a local air toxics control plan is also prepared. These plans examine multiple pollutants, cumulative impacts, and transport issues related to attaining healthful air quality. A brief overview of the effects observed and attributed to various air pollutants is presented in this Appendix. Because the SCAB exceeds the federal standards for ozone and PM2.5, this Appendix focuses more attention in the discussion of these two pollutants, since the health impacts within the SCAB are potentially greater for these two pollutants compared to the health impacts of the other criteria pollutants. For the other pollutants, a brief summary of the associated health effects is provided.

This summary is drawn substantially from reviews presented previously (South Coast Air Quality Management District 1996; South Coast Air Quality Management District 2003; South Coast Air Quality Management District 2007; South Coast Air Quality Management District 2013b), and from the most recent U.S. EPA Integrated Science Assessment (ISA) reviews for Ozone (U.S. EPA 2013b), Carbon Monoxide (U.S. EPA 2010), Particulate Matter (U.S. EPA 2009), Nitrogen Oxides (U.S. EPA 2016), Sulfur Dioxide (U.S. EPA 2008), and Lead (U.S. EPA 2013a). Additional reviews prepared by the California Air Resources Board and the California EPA Office of Environmental Health Hazard Assessment for Particulate Matter (California Air Resources Board and Office of Environmental Health Hazard Assessment 2002), for Ozone (California Air Resources Board and Office of Environmental Health Hazard Assessment 2005) and for Nitrogen Dioxide (California Air Resources Board and Office of Environmental Health Hazard Assessment 2007) were included in the summary. In addition, several large review articles on the health effects of air pollution also helped inform this Appendix (American Thoracic Society 1996a; Brunekreef et al. 2002). More detailed citations and discussions on air pollution health effects can be found in these references.[1] Additionally, a supplemental literature review of mortality and morbidity impacts of PM2.5, ozone, NO_2, and SO_2 was conducted for the AQMP Socioeconomic Evaluation to identify more recent studies (Industrial Economics Inc. 2016b; Industrial Economics Inc. 2016a); this health effects summary also draws upon this literature review to discuss these more recent studies, particularly those published since the

[1] Most of the studies referred to in this Appendix are cited in the above sources. Only specific selected references to provide examples of the types of health effects are cited in this summary.

most recent ISA's. This summary highlights studies that were conducted in the South Coast Air Basin or in Southern California, or alternatively, in California, if few studies from our local region are available on the specific topic. Studies conducted in Southern California give an important "local perspective" in understanding and evaluating the health effects of air pollution. However, studies conducted in other locations also provide critical information that is pertinent to advancing the scientific understanding of the health effects of air pollution, including effects on our local population. As such, this summary also discusses key studies that were conducted in other locations.

Over the decades of national reviews of outdoor air pollution and their health impacts, the U.S. EPA has developed a list of five criteria by which the strength and credibility of data can be judged. This five-tier weight-of-evidence approach provides an objective basis for assessing the breadth, specificity, and consistency of evidence concerning a particular health outcome. Table I-1 shows the five descriptors used by the U.S. EPA for assessing causality, using a weight-of-evidence approach. Within each section discussing a specific pollutant are tables showing summaries of the U.S. EPA conclusions regarding the causality of air pollution health effects, which are the conclusions of their scientific evaluation of the research studies they have reviewed. For the criteria pollutants, the discussion in this Appendix will focus only on those categories of health effects for which the U.S. EPA has determined there is a causal or likely causal relationship with the pollutant, while other health effects may be discussed briefly. In particular, because of the relatively long time gap since the latest U.S. EPA ISA for PM (in 2009), and because the SCAB currently exceeds the federal standards for PM2.5, some additional health endpoints that are emerging as areas of interest with regard to PM exposure are discussed briefly in this Appendix.

It is important to note that the U.S. EPA is tasked with assessing new and emerging air quality science, including health studies, as part of the process of setting the federal air quality standards. In other words, the U.S. EPA's role is to assess the causal relationships between the pollutants and the different types of health endpoints. It is SCAQMD's role to describe the public health impacts of poor air quality in our region, as well as to develop and implement an emission reduction strategy to attain the federal and state ambient air quality standards. Therefore, it is not the intention of this Appendix to assess whether there is or is not an effect of a specific air pollutant on any particular health endpoint, but rather to summarize the health effects and causal determinations as assessed by U.S. EPA and other scientific agencies, to discuss some recent studies published since the latest U.S. EPA reviews, to give some quantitative estimates of the health impacts of particulate matter air pollution in the South Coast Air Basin, and to present a "local perspective" by highlighting studies conducted in the South Coast Air Basin, Southern California, or California.

TABLE I-1

U.S. EPA's Weight of Evidence Descriptions for Causal Determination of Health Effects

DETERMINATION	WEIGHT OF EVIDENCE
Causal Relationship	Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures. That is, the pollutant has been shown to result in health effects in studies in which chance, bias, and confounding could be ruled out with reasonable confidence. For example: (a) controlled human exposure studies that demonstrate consistent effects; or (b) observational studies that cannot be explained by plausible alternatives or are supported by other lines of evidence (e.g., animal studies or mode of action information). Evidence includes replicated and consistent high-quality studies by multiple investigators.
Likely To Be A Causal Relationship	Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain. That is, the pollutant has been shown to result in health effects in studies in which chance and bias can be ruled out with reasonable confidence but potential issues remain. For example: (a) observational studies show an association, but co-pollutant exposures are difficult to address and/or other lines of evidence (controlled human exposure, animal, or mode of action information) are limited or inconsistent; or (b) animal toxicological evidence from multiple studies from different laboratories that demonstrate effects, but limited or no human data are available. Evidence generally includes replicated and high-quality studies by multiple investigators.
Suggestive Of A Causal Relationship	Evidence is suggestive of a causal relationship with relevant pollutant exposures, but is limited because chance, bias, and confounding cannot be ruled out. For example, at least one high-quality epidemiologic study shows an association with a given health outcome but the results of other studies are inconsistent.
Inadequate To Infer A Causal Relationship	Evidence is inadequate to determine that a causal relationship exists with relevant pollutant exposures. The available studies are of insufficient quantity, quality, consistency or statistical power to permit a conclusion regarding the presence or absence of an effect.
Not Likely To Be A Causal Relationship	Evidence is suggestive of no causal relationship with relevant pollutant exposures. Several adequate studies, covering the full range of levels of exposure that human beings are known to encounter and considering susceptible populations, are mutually consistent in not showing an effect at any level of exposure.

(Adapted from U.S. EPA, 2009)

OZONE

Ozone is a gaseous air pollutant that is a highly reactive compound and a strong oxidizing agent. When ozone comes into contact with the respiratory tract, it can react with tissues and cause damage in the airways. Ozone, or its reaction products, can penetrate into the gas exchange region of the deep lung. Both short-term and long-term exposures to ozone have been linked to respiratory effects. Ozone from man-made sources is formed by photochemical reactions when pollutants such as volatile organic compounds, nitrogen oxides, and carbon monoxide react with sunlight. The main sources of such ozone precursors are discussed in detail in the draft 2016 AQMP Chapter 3. Additionally, a discussion of the spatial distribution of ozone is provided in the draft 2016 AQMP Chapter 2.

In 1997, the U.S. EPA established the first federal standard for ozone averaged over 8 hours, at 0.08 ppm. In 2005, the California Air Resources Board (CARB) established standards of 0.09 ppm averaged over one hour and at 0.070 ppm averaged over eight hours. In 2008, the U.S. EPA lowered the federal standard for ozone to 0.075 ppm averaged over eight hours. On the basis of recent evaluations of ozone health effects, U.S. EPA's Clean Air Scientific Advisory Committee recommended in 2015 that the National Ambient Air Quality Standard (NAAQS) for ozone be reduced and recommended a range in which 0.070 ppm would be the upper limit. In 2015, the U.S. EPA concluded that the current national standard was not adequate to protect public health and lowered the 8-hour ozone standard to 0.070 ppm (U.S. EPA 2015b). While the federal standards must be attained within a specified time frame, the California standards do not have specific defined deadlines, but must be attained by the earliest practicable date.

The table below provides the overall U.S. EPA staff conclusions on the causality of short-term (i.e. hours, days, weeks) and long-term (i.e. months, years) ozone health effects for the health outcomes evaluated (U.S. EPA 2013b).

TABLE I-2

Summary of U.S. EPA's Causal Determinations for Health Effects of Ozone

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory Effects	Causal relationship
Cardiovascular Effects	Likely to be a causal relationship
Central Nervous System Effects	Suggestive of a causal relationship
Effects on Liver and Xenobiotic Metabolism	Inadequate to infer a causal relationship
Effects on Cutaneous and Ocular Tissues	Inadequate to infer a causal relationship
Mortality	Likely to be a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory Effects	Likely to be a causal relationship
Cardiovascular Effects	Suggestive of a causal relationship
Reproductive and Developmental Effects	Suggestive of a causal relationship
Central Nervous System Effects	Suggestive of a causal relationship
Cancer	Inadequate to infer a causal relationship
Mortality	Suggestive of a causal relationship

(From U.S. EPA, 2013a Table 1-1)

Short-Term Exposure Effects of Ozone

The adverse effects reported with short-term ozone exposure are greater with increased activity because activity increases the breathing rate, the depth of the breaths, and the volume of air reaching the lungs, resulting in an increased amount of ozone reaching deeper into the lungs. Children are considered to be a particularly vulnerable population to air pollution effects because their lungs are still growing, they typically spend more time outdoors, are generally more physically active, and have a higher ventilation rate relative to their body weight, compared to adults (U.S. EPA 2013b).

A number of adverse health effects associated with ambient ozone levels have been identified from laboratory and epidemiological studies (American Thoracic Society 1996b; U.S. EPA 2006; U.S. EPA 2013b). These include increased respiratory symptoms, damage to cells of the respiratory tract,

decrease in lung function, increased susceptibility to respiratory infection, an increased risk of hospitalization, and increased risk of mortality. For short-term ozone exposures, the U.S. EPA determined in the most recent ISA that the evidence supports a causal relationship for respiratory effects, and a likely causal relationship for cardiovascular effects and mortality.

In the laboratory, exposure of human subjects to low levels of ozone causes reversible decreases in lung function as assessed by various measures such as respiratory volumes, airway resistance and reactivity, irritative cough and chest discomfort. The results of several studies where human volunteers were exposed to ozone for 6.6 hours at levels between 0.04 and 0.12 ppm were summarized by Brown (Brown et al. 2008). As shown in Figure I-1, there is an increasing response on lung function with increasing exposure levels in moderately exercising subjects. A study published after the analysis by Brown et al. exposed healthy young adults for 6.6 hours under intermittent moderate exercise to each of the following: filtered air, and ozone at 0.06, 0.07, 0.08, and 0.087 ppm (Schelegle et al. 2009). The study found decreases in lung function (forced expiratory volume in 1 second, or FEV1) with each of the different levels of ozone exposure, although the decrease in lung function at 0.06 ppm was not statistically different from exposure to filtered air. Lung function (FEV1) decreases were approximately 5 percent, 7 percent, and 11 percent at ozone exposure levels of 0.07, 0.08, and 0.087 ppm. A more recent study (Kim et al. 2011) exposed young healthy adults to ozone in the range of 0.06 to 0.10 ppm for 6.6 hours while engaging in intermittent moderate exercise, and found that the study participants exhibited an approximately 2 percent reduction in lung function (FEV1) and an increase in pulmonary inflammation after exposure to ozone at the 0.06 ppm concentration.

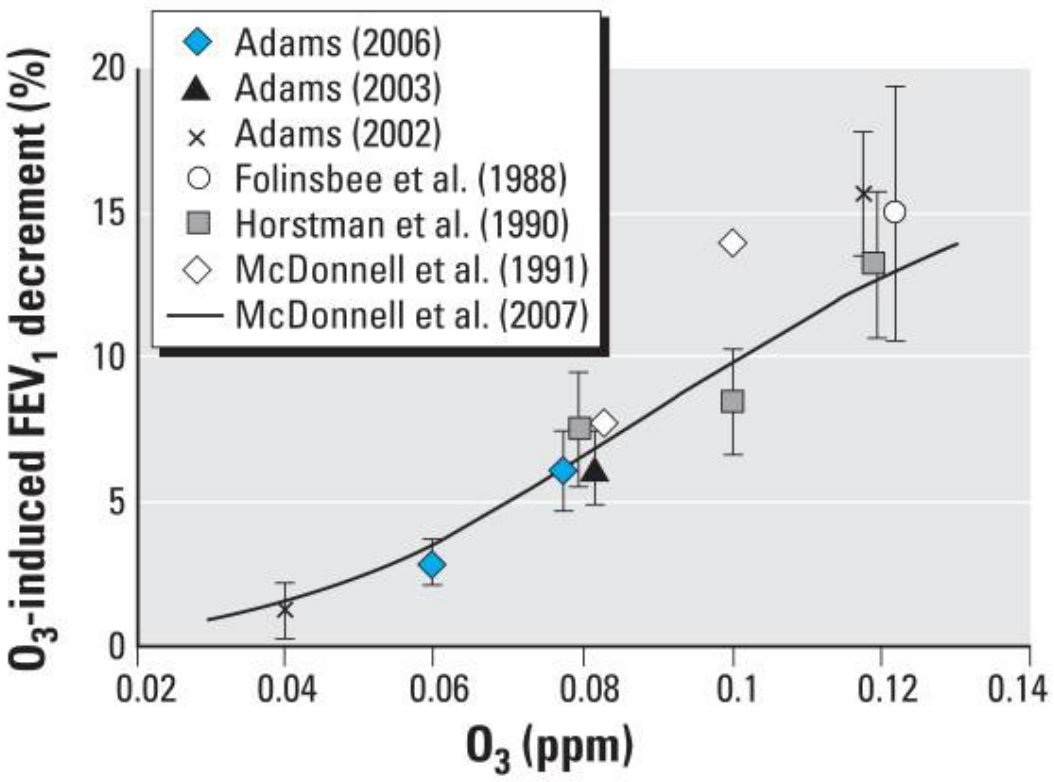


FIGURE I-1

Comparison of mean ozone-induced decrements in lung function following 6.6 hours of ozone exposure. Error bars represent the standard error. McDonnell et al. (2007) was a summary of results from several studies, and is represented by the line in the graph. (From: (Brown et al. 2008))

Some changes in lung function (volume and airway resistance changes) observed after study participants were exposed to ozone only once exhibit attenuated responses or a reduction in magnitude of responses when exposures are repeated, although there were a range of individual human responses observed, including some non-responders (Linn et al. 1988). Although it has been argued that the observed shift in response is evidence of a probable development of tolerance, it appears that while functional changes may exhibit attenuation, biochemical and cellular changes which may be associated with episodic and chronic exposure effects may not exhibit an attenuation. That is, internal damage to the respiratory system may continue with repeated ozone exposures, even if externally observable effects (chest symptoms and reduced lung function) disappear. An additional argument against toleration is that after several days or weeks without ozone exposures, the responsiveness (in terms of lung function as well as symptoms) returns, which is evidence that any tolerance developed is relatively short-lived (U.S. EPA 2013b).

Laboratory studies have also compared the degree of lung function change seen in healthy individuals versus asthmatics and those with chronic obstructive pulmonary disease (COPD). In several

laboratory studies of individuals with COPD, the percent decreases in lung function from short-term ozone exposures ≤0.30ppm among patients with COPD generally did not differ from the lung function decrements experienced by healthy patients (Linn et al. 1982; Solic et al. 1982; Linn et al. 1983; Kehrl et al. 1985). That finding, however, may not accurately reflect the true impact of exposure on these respiration-compromised individuals. Since the respiration-compromised group may have lower lung function to begin with, the same total percent change in lung function may represent a substantially greater relative adverse effect overall. Other studies have found that subjects with asthma are more sensitive to the short-term effects of ozone in terms of lung function and inflammatory response, as evidenced by measuring changes in lung function, increased hospitalizations, and emergency room visits for respiratory conditions (U.S. EPA 2013b). This evidence supports the hypothesis that asthmatics are a particularly sensitive population to the health effects of ozone.

In laboratory studies of animals, cellular and biochemical changes associated with respiratory tract inflammation have also been consistently found in the airway lining after low- level exposure to ozone. These changes include an increase in specific cell types and in the concentration of biochemical mediators of inflammation and injury such as Interleukin-1, Interleukin-6, Interleukin-8, Tumor Necrosis Factor α (TNF-α), and fibronectin (Van Bree et al. 2002; Johnston et al. 2007; U.S. EPA 2013b).

In addition to controlled laboratory conditions, epidemiological studies of individuals exercising outdoors, including children attending summer camp, have shown associations of reduced lung function with ozone exposure. There were wide ranges in responses among individuals. U.S. EPA's 2013 ISA indicated that most studies found reductions in lung function (FEV_1) in the range of approximately <1 to 2 percent when standardized to an increase of 0.04 ppm for a 1-hour maximum, an increase of 0.03 ppm for an 8-hour maximum, and an increase of 0.02 ppm for a 24-hour average (U.S. EPA 2013b). Somewhat greater decrements in lung function (4.9 to 7.3 percent) were found in children with asthma who had respiratory infections or were using corticosteroid medication.

Epidemiologic studies have found that increases in short-term ozone levels are associated with impacts on children's respiratory health, including increases in respiratory symptoms in children with asthma, and increased numbers of absences from school. Studies conducted in various cities in the U.S. and in other countries have reported increased respiratory symptoms among children with asthma, including wheeze, cough, difficulty breathing, and chest symptoms/tightness (U.S. EPA 2013b). The Children's Health Study, conducted by researchers at the University of Southern California, followed for several years a cohort of children that live in 12 communities in Southern California with differing levels of air pollution. A publication from this study reported that school absences in fourth graders for respiratory illnesses were positively associated with short-term increases in ambient ozone levels. An increase of 20 ppb (0.02 ppm) ozone was associated with a 63 percent increase in illness-related absence rates and an 83 percent increase in respiratory illnesses (Gilliland et al. 2001). A small panel study of Hispanic children with asthma living in the Huntington Park neighborhood of Los Angeles, California reported that a 10.8 ppb increase in ozone averaged

over 8 hours nearly doubled the odds of having asthma symptoms that interfered with daily activities (Delfino et al. 2003). Despite these studies, and some others linking ozone exposures with school absences, the U.S. EPA concluded that only limited evidence is currently available linking these ozone exposures to respiratory-related school absences (U.S. EPA 2013b).

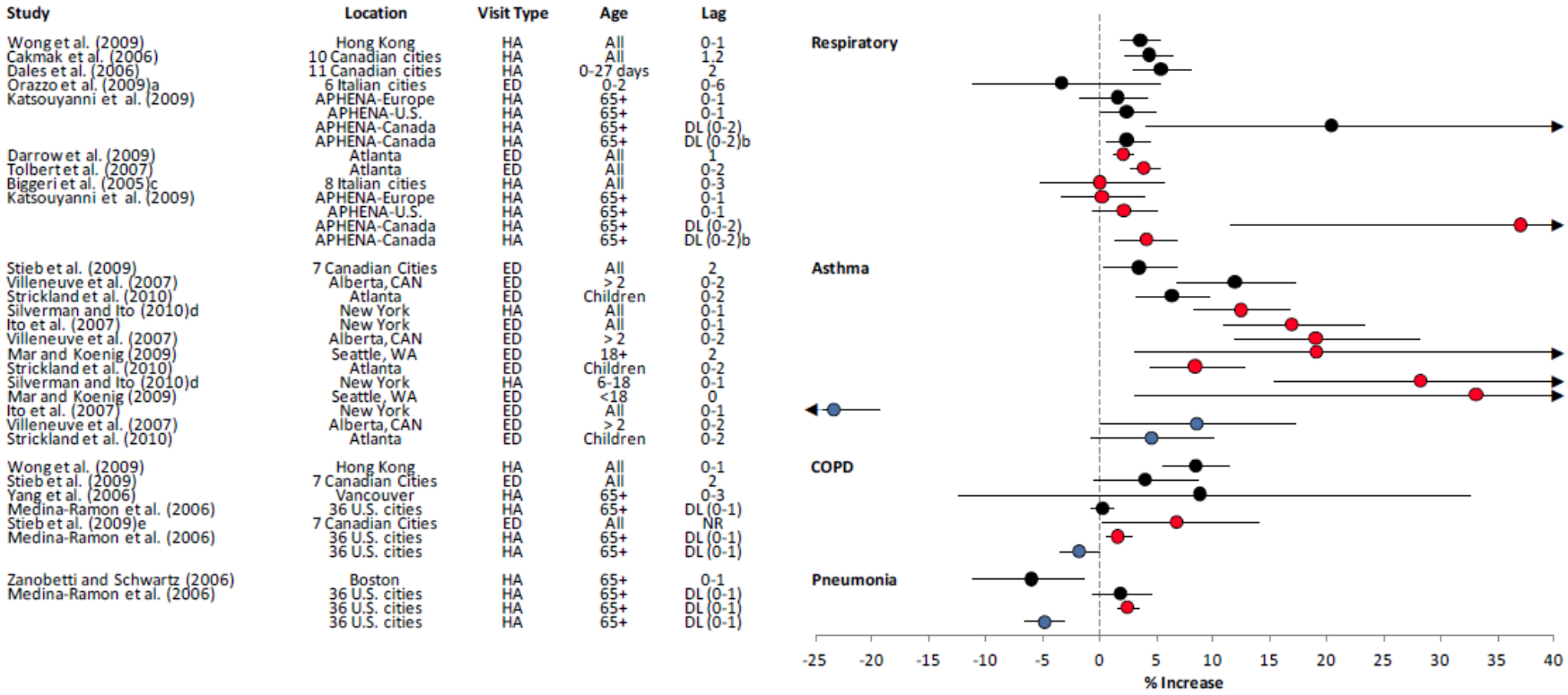
Numerous studies have found associations of short-term ozone levels and hospital admissions and emergency department admissions for respiratory conditions, and the U.S. EPA concluded in the latest ISA that the most recent epidemiological studies conducted in both single cities and multiple cities continue to provide evidence supporting a causal relationship between short-term ozone exposures and respiratory effects (U.S. EPA 2013b). The studies generally found stronger associations for asthma and COPD in the warm season or in the summer months, compared to the cold season, and also provided evidence that children are at greatest risk of ozone-related respiratory health effects. Several of these studies reviewed in the ISA had average ozone concentrations well below 60 ppb averaged over 8 hours and still reported associations with respiratory outcomes. One study of asthma emergency department visits reported ozone effects at concentrations as low as 30 ppb (Strickland et al. 2010). Figure I-2 presents examples of studies regarding all-year and seasonal analysis of ozone exposure and hospital admissions or emergency department visits. This figure illustrates the associations found between ambient ozone exposure and key respiratory outcomes (asthma, COPD and pneumonia), and shows the stronger effects with summertime ozone exposures. Recently, a study in California reported that short-term ozone exposures were associated with emergency department visits for asthma, acute respiratory infections, pneumonia, COPD, and upper respiratory tract infections, with more consistent associations during the warm season (Malig et al. 2016). This California study provides additional supporting evidence for ozone-related respiratory effects.

The potential cardiovascular effects of short-term ozone exposure have been studied in toxicological, human exposure, and epidemiological studies. Controlled human exposure studies have found that ozone exposures produce changes in heart function (as measured by heart rate variability) and increases in biomarkers in the blood for systemic inflammation and oxidative stress. The limited number of toxicological studies on this topic provide evidence of cardiovascular effects. The effects observed include increased heart rate variability, arrhythmias, vascular disease, and inflammation and oxidative stress leading to atherosclerosis, which can lead to tissue damage due to ischemia and reperfusion (i.e. having the blood supply cut off and then restored to the tissues) (U.S. EPA 2013b). The controlled human exposure and toxicological studies provide evidence of cardiovascular effects of ozone, and some plausible mechanisms for these effects. Epidemiological studies, including some recent multi-city studies show relatively consistent associations between short-term ozone exposures and cardiovascular mortality (these studies are discussed further below). However, epidemiological studies do not provide consistent evidence of cardiovascular morbidity with short-term ozone exposures. Studies conducted in the Los Angeles area or in California also do not provide consistent evidence of short-term ozone effects on cardiovascular morbidity. A study of elderly non-smokers in the Los Angeles area with a history of heart disease found no associations between ozone exposure and blood pressure nor ST-segment depression, a measure of cardiac ischemia (Delfino et

al. 2010; Delfino et al. 2011). A Los Angeles-based study of cardiovascular hospital admissions did not find increased risk with ozone exposures (Linn et al. 2000). However, a biomarker study of students at UC Berkeley who spent their summer vacation in either the Los Angeles or San Francisco Bay Area found that ozone exposures over a period of 2 weeks or 1 month were associated with increases in a biomarker of lipid peroxidation, but no association was found for a biomarker of antioxidant capacity (Chen et al. 2007). Lipid peroxidation is an indicator of oxidative stress, which may be triggered by pulmonary inflammation caused by ozone exposure. Given the strong evidence of cardiovascular morbidity from experimental studies and the consistent positive associations reported in epidemiological studies of cardiovascular mortality, but the lack of consistent evidence from epidemiological studies of cardiovascular morbidity, the U.S. EPA determined that there is a likely causal relationship between short-term ozone exposures and cardiovascular effects (U.S. EPA 2013b).

For mortality effects, the U.S. EPA 2013 ISA concluded that there was a likely causal relationship for short-term ozone exposures. This determination is supported by numerous studies have found positive associations between short-term increases in ozone levels and excess risk of mortality from all non-accidental causes, cardiovascular causes, and respiratory causes (Bell et al. 2004; Bell et al. 2005; Huang et al. 2005; Ito et al. 2005; Levy et al. 2005; Bell et al. 2008; Zanobetti et al. 2008). Studies conducted across multiple cities in the U.S. Canada, Europe and Asia reported increased cardiovascular and respiratory mortality risks with increased short-term ozone exposures, and several studies additionally reported increased mortality risk for summer season ozone exposures (Katsouyanni et al. 2009; Samoli et al. 2009; Stafoggia et al. 2010; Wong et al. 2010). Some studies have also demonstrated that these associations persist even when other variables including season and levels of particulate matter are accounted for, indicating that ozone mortality effects may be independent of other pollutants, although there is some variability across studies with regard to the sensitivity of the ozone associations to adjustment for PM (Bell et al. 2004; Huang et al. 2005; Katsouyanni et al. 2009; Stafoggia et al. 2010). With regard to respiratory effects, the substantial evidence supporting a causal relationship between short-term ozone exposures and respiratory morbidity provides strong support for the recent evidence from epidemiological studies linking such exposures to respiratory mortality. For cardiovascular effects, while there is strong evidence linking cardiovascular mortality with short-term ozone exposures, the epidemiological studies of non-fatal outcomes do not provide consistent evidence for a coherent mechanism linking ozone exposures to cardiovascular mortality (U.S. EPA 2013b).

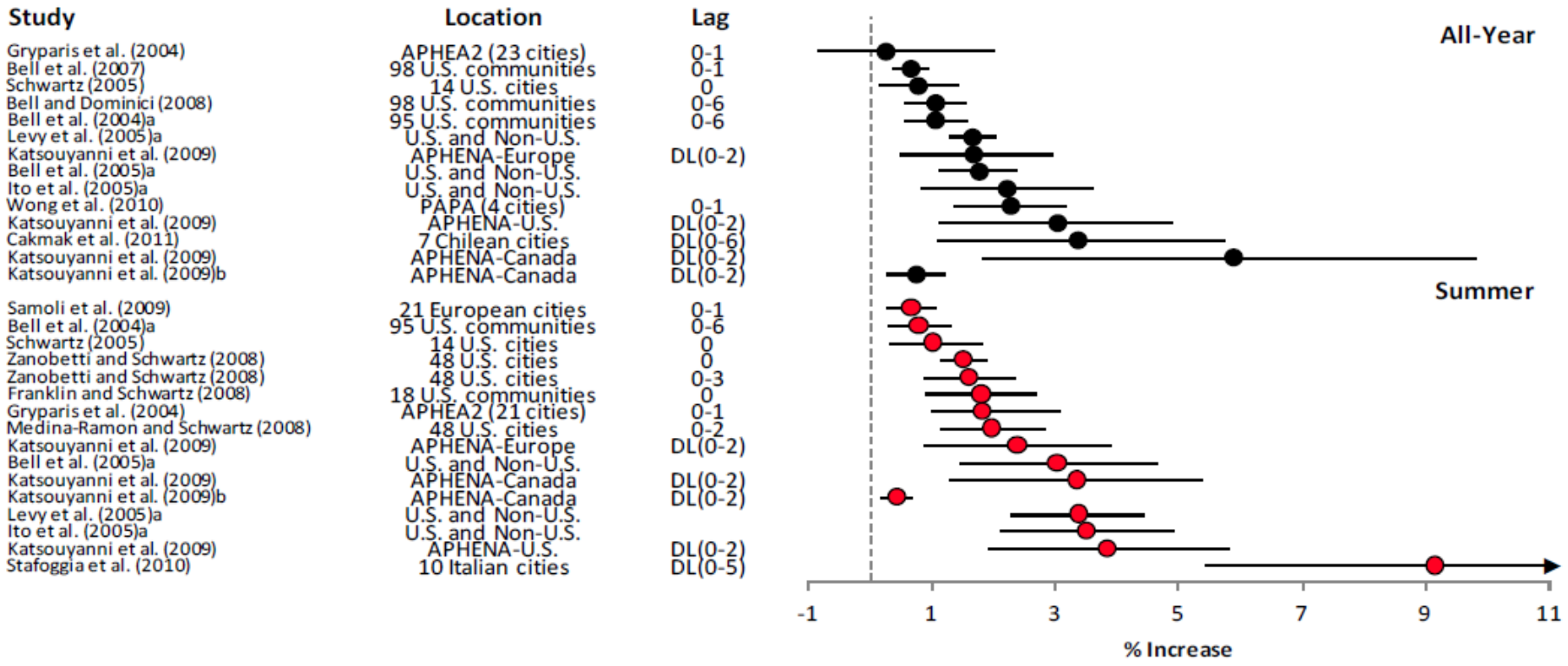
Examples of studies showing the relative change in mortality risks for all-year and summer-only analyses are shown in Figure I-3.



Note: Effect estimates are for a 20 ppb increase in 24-hour; 30 ppb increase in 8-hour max; and 40 ppb increase in 1-hour max O_3 concentrations. HA=hospital admission; ED=emergency department. Black=All-year analysis; Red=Summer only analysis; Blue=Winter only analysis. (From (U.S. EPA 2013b) Figure 6-19)

FIGURE I-2

Change in respiratory-related hospital admission and emergency department visits in studies that presented all-year and/or seasonal results.



Note: Effect estimates are for a 40 ppb increase in 1-hr max, 30 ppb increase in 8-hr max, and 20 ppb increase in 24-hr average O_3 concentrations. (From (U.S. EPA 2013b) Figure 6-27)

FIGURE I-3

Summary of mortality risk estimates for short-term O_3 exposure and all-cause (nonaccidental) mortality.

Long-Term Exposure Effects of Ozone

The U.S. EPA 2013 ISA for Ozone concluded that there was a likely causal relationship between long-term ozone exposure and respiratory effects (U.S. EPA 2013b). Evidence supporting this determination comes from epidemiological and toxicological studies, particularly studies of asthma and related symptoms, asthma-related hospital admissions, lung function, lung inflammation and oxidative stress. Other health effects of long-term ozone exposure were determined to have "suggestive" or "inadequate" evidence of causality, although the few studies of respiratory mortality provide support to the respiratory health effects of ozone.

The Adventist Health and Smog Study (AHSMOG) and Children's Health Study cohorts are two large long-term studies conducted in California that examined several aspects of long-term ozone effects in adults and children, respectively. Several of these studies focused on asthma development and exacerbation. The AHSMOG study included adult, non-smoking, non-Hispanic white Seventh Day Adventists living in California. The 10-year follow-up AHSMOG study reported that a 10 ppb increase in annual mean ozone exposures increased the risk of asthma development in males by three-fold (relative risk 3.12, 95 percent confidence interval: 1.16, 5.85), but no effect was seen among females (relative risk 0.94, 95 percent confidence interval: 0.65, 1.34) (Greer et al. 1993). The 15-year follow-up AHSMOG study used an ozone metric focusing on 8-hour average exposures, and reported that a 10 ppb increase was associated with a 30 percent increased risk of developing asthma in males (relative risk 1.31, 95 percent confidence interval: 1.01, 1.71), and these effects persisted even after accounting for other pollutants (McDonnell et al. 1999). The latter study also found no effect in females, although this may reflect a greater potential for misclassification of air pollution exposure in females compared to males, due to different time-activity patterns resulting in greater time spent outdoors among males (U.S. EPA 2013b). In the Children's Health Study, among children living in 12 Southern California communities with high ozone concentrations, the relative risk of developing asthma in children playing three or more sports was found to be over three times higher than in children playing no sports (McConnell et al. 2002). The high ozone communities had a 4-year mean daytime ozone concentration of 59.6 ppb, compared to 40.0 ppb for the low-ozone communities. These findings indicate that new cases of asthma in children may be associated with performance of heavy exercise in communities with high levels of ozone. While it has long been known that air pollution can exacerbate symptoms in individuals with preexisting respiratory disease, this is among the first studies that indicate ozone exposure may contribute to asthma onset. However, three more recent Southern California studies did not find an association between ozone exposures and childhood asthma incidence, but did report increased risks of asthma onset with higher exposures to particulate matter or NO_2 (Islam et al. 2007; McConnell et al. 2010; Nishimura et al. 2013). These studies did not examine whether genetic factors may have played a role in making some people more susceptible than others to the respiratory effects of ozone exposure. Some analyses from the Children's Health Study identified specific genetic variants that, when combined with ambient ozone exposure, either increase or decrease the risk of developing asthma (Islam et al. 2008; Islam et al. 2009; Salam et al. 2009). These genetic variants are involved with antioxidant and/or anti-

inflammatory pathways, and are likely involved in key elements of asthma development (U.S. EPA 2013b).

Other studies examined the impact of long-term ozone exposures and respiratory symptoms, particularly among asthmatics. Studies have linked long-term ozone exposures to increased risk of having poorly-controlled asthma, increased asthma symptoms, and respiratory-related school absences (Gilliland et al. 2001; Akinbami et al. 2010; Jacquemin et al. 2012). An analysis from the CHS found no association between long-term ozone exposures and chronic lower respiratory tract symptoms, and another found an increased risk of bronchitic symptoms within a community, although the association was reduced when accounting for other pollutants (McConnell et al. 1999; McConnell et al. 2003). However, two studies from the CHS demonstrated gene-environment interactions for genes that are involved in inflammation or antioxidant pathways. One study found that asthmatic children with a particular genetic variant that reduces expression of the cytokine TNF-α (as part of an inflammatory response) had reduced risk of bronchitic symptoms for children in low-ozone communities, but not for children in high-ozone communities (Lee et al. 2009). A second study found that a particular genetic variant reduced the risk of respiratory-related school absences among children living in communities with high levels of ozone (defined in this study as being above the median value of 46.9 ppb) (Wenten et al. 2009).

Results of epidemiologic studies of hospital admissions and emergency department visits support the relationship between ozone exposure and respiratory effects. In a 2007 study conducted in Southern California, an increased risk of having poorly-controlled asthma was associated with living in areas above the 90th percentile ozone level (28.7 ppb, annual average) among men and elderly individuals (Meng et al. 2007). A study in the South Coast Air Basin found that ozone was associated with increased hospital discharges for asthma among children (Moore et al. 2008). Another study in the South Coast Air Basin looked at infants hospitalized for bronchiolitis. This study found a reduced risk of infant bronchiolitis hospitalization with increased ozone exposure, although there was no association for ozone when accounting for the effect of PM2.5, which was positively associated with this respiratory outcome (Karr et al. 2007). A study of people with asthma was conducted in the San Joaquin Valley of California, and found that a 10 ppb increase in ozone exposures averaged over one year increased the odds of asthma-related hospital admissions and emergency department visits by approximately 50 percent, and the odds of asthma symptoms among adults by about 40 percent (Meng et al. 2010). Studies conducted in other locations have also reported increases in asthma hospitalizations (U.S. EPA 2013b).

Some animal studies show results that indicate possible chronic effects including functional and structural changes of the lung. However, morphological, developmental, and immunological differences make it difficult to apply these results to humans experiencing ambient exposures. These changes observed in airway responsiveness provide support for the long-term effects of ozone in asthma development or exacerbation (U.S. EPA 2013b). However, epidemiologic studies examining long-term ozone exposures and lung function deficits have reported mixed results. For example, an analysis of the first CHS cohort found that PM2.5 and NO_2 exposures were associated with decreased

lung function, but did not find an association for ozone (Gauderman et al. 2004). An autopsy study involving Los Angeles County residents who died between ages 14 and 25 years due to violent death, although conducted many years ago when pollutant levels were higher than currently measured, provided supportive evidence of lung tissue damage (structural changes), which the authors suggested were attributable to air pollution (Sherwin 1991), although many uncertainties remain about the extent to which air pollution explains the findings.

Unlike short-term ozone exposures, there is limited evidence linking long-term ozone exposures with mortality. A large study based on the American Cancer Society Cancer Prevention Study II (CPS-II) cohort included 96 metropolitan statistical areas in the U.S., and reported that a 10 ppb increase in daily maximum 1-hour ozone concentrations averaged between April and September (warm season) was associated with a relative risk of 1.040 (95 percent confidence interval: 1.010, 1.067) for respiratory deaths, but no association with cardiovascular deaths (Jerrett et al. 2009). A U.S. study of Medicare enrollees reported increased risk of mortality with higher ozone exposures averaged over the warm season, among patients who had previously been hospitalized for congestive heart failure, myocardial infarction, COPD and diabetes (Zanobetti et al. 2011). A recent large-scale study found increased risk of all-cause, cardiovascular, and respiratory mortality with long-term ozone exposures, even after accounting for the effects of PM2.5 and NO_2, as well as other behavioral and demographic factors, including smoking (Turner et al. 2016). Other studies have found temperature to be an important potential risk factor for mortality, and may confound or modify the associations between air pollution exposure and mortality (Basu et al. 2002; Cheng et al. 2008). The Turner 2016 study examined the role of temperature, and found that the associations between ozone and mortality differed based on average daily maximum temperatures (Turner et al. 2016). While the U.S. EPA determination in the latest ISA was that the evidence was suggestive of long-term ozone exposure causing mortality, the studies of respiratory mortality support the evidence for the respiratory effects of ozone exposure, for which U.S. EPA has concluded there is a causal relationship.

For non-respiratory health endpoints, the U.S. EPA causal determinations were “suggestive of a causal relationship” (for cardiovascular, reproductive and developmental, central nervous system and mortality effects) or “inadequate to infer a causal relationship” (for cancer). Some studies conducted in California have examined reproductive or developmental effects, including birth defects, low birth weight or birth weight reductions, stillbirth and autism (Ritz et al. 2002; Ritz et al. 2007; Morello-Frosch et al. 2010; Becerra et al. 2013; Mobasher et al. 2013; Trasande et al. 2013; Laurent et al. 2014; Green et al. 2015; Symanski et al. 2016). Other recent studies have examined cardiovascular effects (Koken et al. 2003; Ensor et al. 2013; Rodopoulou et al. 2014). While many of these studies have reported associations with ambient ozone levels, the most recent U.S. EPA determination in 2013 was that the evidence was suggestive of a causal determination, but did not yet rise to a higher level.

Sensitive Populations for Ozone-Related Health Effects

A number of population groups are potentially at increased risk for ozone exposure effects. In the most recent ISA for ozone in 2013, the U.S. EPA has identified several populations as having adequate evidence for increased risk from ozone exposures. These include children, older adults, outdoor workers, and individuals with asthma, certain variations in genes related to oxidative metabolism or inflammation, or reduced intake of certain nutrients such as Vitamins C and E (Kreit et al. 1989; Horstman et al. 1995; Sienra-Monge et al. 2004; Romieu et al. 2012; U.S. EPA 2013b; Bell et al. 2014). There is suggestive evidence for other potential factors, such as a person's sex, socioeconomic status, and obesity (U.S. EPA 2013b). Some other factors that could affect sensitivity to ozone have also been studied; however, there was inadequate evidence to conclude whether these were risk factors for ozone sensitivity. The table below summarizes the evidence for factors affecting sensitivity to ozone from the 2013 ISA for ozone.

TABLE I-3

Summary of Evidence for Potential Increased Susceptibility to Ozone-Related Health Effects

Evidence Classification	Potential At Risk Factor
Adequate evidence	Genetic factors Asthma Children Older adults Diet Outdoor worker
Suggestive evidence	Sex SES Obesity
Inadequate evidence	Influenza/infection COPD Cardiovascular disease Diabetes Hyperthyroidism Race/ethnicity Smoking Air conditioning use
Evidence of no effect	--

From (U.S. EPA 2013b) Table 8-6

As previously mentioned, one group that has been recognized as being particularly sensitive to the effects of ozone is young children with asthma, because their lungs are still developing, their potential for increased exposure due to time spent exercising outdoors, and their high ventilation rates relative to body weight (U.S. EPA 2013b). Some factors that may contribute to the increased sensitivity among people with asthma include having an altered innate immune function and factors that decrease their antioxidant defenses (Alexis et al. 2014). Ozone creates secondary oxidation products that are electrophilic, and certain genetic factors influence a person's ability to metabolize

these electrophiles, which can affect respiratory function (U.S. EPA 2013b). Asthma exacerbations are more prevalent and severe in young boys than in girls, but the evidence on whether boys are more susceptible than girls to the effects of air pollution on asthma symptoms is not consistent (Guarnieri et al. 2014).

Summary – Ozone Health Effects

In summary, outdoor ozone exposures have been associated with a range of negative human health effects. The strongest evidence for negative health impacts are on the respiratory system, and are measured by decreased lung function performance and increased cell injury. In addition, the 2013 ISA also concluded that there was a likely causal relationship between short-term ozone exposures and cardiovascular effects (such as changes in heart function, and increased systemic inflammation and oxidative stress) as well as respiratory mortality. Although the specific mechanisms of action for ozone effects on the various health endpoints have not been fully identified, there is evidence of the important roles of oxidation of key enzymes and proteins, inflammatory responses, changes in immune response, and modification and activation of neural reflex pathways (U.S. EPA 2013b).

The previous U.S. EPA review of ozone in the 2006 Air Quality Criteria Document (AQCD) had already concluded that there was clear, consistent evidence that acute ozone exposure is causally associated with respiratory effects (U.S. EPA 2006). Additionally, the 2006 AQCD for ozone concluded that the evidence was highly suggestive of ozone causing mortality, but that there was limited evidence for ozone causing cardiovascular effects. In the 2013 ISA, the U.S. EPA cited that several lines of evidence provide support for the respiratory effects of ozone, including human exposure studies, epidemiology and toxicology, which led to the conclusion that there was a causal relationship with short-term ozone exposures, and a likely causal relationship with long-term ozone exposures. In humans, respiratory effects were detected in laboratory studies at 0.06 ppm ozone concentrations, and in epidemiological studies with average ozone concentrations as low as 0.03 ppm (Strickland et al. 2010; Kim et al. 2011). Some populations are more sensitive to the health effects of ozone than others, including elderly persons, children, outdoor workers and persons with asthma.

PARTICULATE MATTER

Airborne particulates are a complex group of pollutants that vary in physical, chemical, and biological dimensions. Physically, particles can vary by size, surface area and roughness, shape, and mass. Chemically, they vary by chemical composition. Biologically, they can vary by toxicity. In addition, particles vary by source, and can come from anthropogenic (man-made, such as from combustion of fuels, or frictional abrasion) or “natural” (plants – for example, pollens and spores) origins. The composition of particulate matter can vary across sub-regions, and a description of the spatial differences in PM composition can be found in the draft 2016 AQMP Chapter 2 and Appendix II.

The National Ambient Air Quality Standard for particulate matter was established in 1971, and set limits on the ambient level of Total Suspended Particulates (TSP). In 1987, the national particulate matter standards were revised to focus on particles sized 10 µm (micrometers) aerodynamic diameter and smaller. These can be inhaled and deposited throughout the upper and lower

respiratory system, depositing in both airways and gas-exchange areas of the lung. These particles are referred to as PM10. U.S. EPA initially promulgated ambient air quality standards for PM10 of 150 μg/m^3 averaged over a 24-hour period, and 50 μg/m^3 for an annual average. U.S. EPA has since rescinded the annual PM10 standard, but kept the 24-hour standard.

As more health research data has become available, concerns have centered on smaller and smaller particles. Additional focus has been placed on particles having an aerodynamic diameter of 2.5 μm or less (PM2.5). A greater fraction of particles in this size range can penetrate and deposit deep in the lungs. The U.S. EPA established standards for PM2.5 in 1997 and in 2006 lowered the air quality standards for PM2.5 to 35 μg/m^3 for a 24-hour average and reaffirmed 15 μg/m^3 for an annual average standard. There was considerable controversy and debate surrounding the review of particulate matter health effects and the consideration of ambient air quality standards (Kaiser 1997; Vedal 1997) when the U.S. EPA promulgated the initial PM2.5 standards in 1997. In 2002, the California Air Resources Board adopted an air quality standard for PM2.5 at a level of 12 μg/m^3, in the form of an annual average.

Since that time, additional studies have been published and some of the key studies were closely scrutinized and the data reanalyzed by additional investigators. The reanalyses confirmed the original findings, and there are now additional data confirming and extending the range of the adverse health effects of PM2.5 exposures. In 2012, the U.S. EPA revised the PM2.5 annual average standard to 12.0 μg/m^3 (U.S. EPA 2013c). This federal standard is set at same level as the current California PM2.5 annual standard, although the California standard does not have a specified attainment date. In 2014, the U.S. EPA announced it is preparing an ISA as part of the review of the federal PM standards (the process is described briefly in the draft AQMP Chapter 8). The draft AQMP Chapter 2 and Appendix II provide additional information about how PM levels in the South Coast Air Basin compare to the federal and state standards.

There have been several reviews of the health effects of ambient particulate matter (American Thoracic Society 1996a; Brunekreef et al. 2002; U.S. EPA 2004; U.S. EPA 2009; Brook et al. 2010). In addition, the California Air Resources Board (CARB) and the Office of Environmental Health and Hazard Assessment (OEHHA) have reviewed the adequacy of the California Air Quality Standards for Particulate Matter (California Air Resources Board and Office of Environmental Health Hazard Assessment 2002).

The major types of health effects associated with particulate matter include:

- Increased mortality
- Exacerbation of respiratory disease and of cardiovascular disease as evidenced by increases in:
    - Respiratory symptoms, exacerbation of asthma
    - Cardiovascular symptoms, non-fatal myocardial infarction
    - Hospital admissions and emergency room visits

- Physician office visits
- School absences

- Adverse birth outcomes
- Effects on lung function
- Changes in lung morphology

In the 2009 Integrated Science Assessment for Particulate Matter, the U.S. EPA presented conclusions on the particulate matter causal determination of several health effects based on an updated review of scientific studies (U.S. EPA 2009). The conclusions are presented separately for particulates in the size range of 2.5 to 10 micrometers (µm) in aerodynamic diameter (PM10-2.5, often referred to as the coarse fraction) and those ≤2.5 µm (PM2.5, or fine particles). Of note, there is currently no federal or California standard for PM10-2.5, although a PM10 standard remains in effect. These conclusions are depicted in the following tables.

TABLE I-4

Summary of U.S. EPA's Causal Determinations for Health Effects of PM10-2.5

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Suggestive of a causal relationship
Respiratory effects	Suggestive of a causal relationship
Mortality	Suggestive of a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Inadequate to infer a causal relationship
Respiratory effects	Inadequate to infer a causal relationship
Mortality	Inadequate to infer a causal relationship
Reproductive and developmental	Inadequate to infer a causal relationship

(From (U.S. EPA 2009) Table 2-3 and Section 2.3.4)

There are also differences in the composition and sources of particles in the different size ranges that may have implications for health effects. The particles in the coarse fraction (PM10-2.5) are mostly produced by mechanical processes. These include automobile tire wear, industrial processes such as cutting and grinding, and resuspension of particles from the ground or road surfaces by wind and human activities, such as agricultural, mining, and construction operations, which may be particularly important in rural areas.

TABLE I-5

Summary of U.S. EPA's Causal Determinations for Health Effects of PM2.5

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Causal relationship
Respiratory effects	Likely to be a causal relationship
Central nervous system	Inadequate to infer a causal relationship
Mortality	Causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Causal relationship
Respiratory effects	Likely to be a causal relationship
Mortality	Causal relationship
Reproductive and developmental	Suggestive of a causal relationship
Cancer, Mutagenicity, Genotoxicity	Suggestive of a causal relationship

(From (U.S. EPA 2009) Tables 2-1 and 2-2)

In contrast, particles smaller than 2.5 μm are mostly derived from combustion sources, such as automobiles, trucks, and other vehicle exhaust, as well as from stationary combustion sources. The particles are either directly emitted or are formed in the atmosphere from gases that are emitted. Components from material in the earth's crust, such as dust, are also present, with the amount varying in different locations.

Attention to another range of very small particles has been increasing over the last several years. These are generally referred to as "ultrafine" particles, with diameters of 0.1 μm or less. Ultrafine particles are mainly composed of particles from fresh emissions of combustion sources, but are also formed in the atmosphere by condensation of vapors that are emitted or by chemical or photochemical reactions with other contaminants in the air.

Ultrafine particles have relatively short half-lives (minutes to hours) and the particle size rapidly grows through condensation and coagulation processes into particles within the PM2.5 size range. Ultrafine particles are garnering interest since a limited number of epidemiological and some laboratory studies, though not all, indicate that their toxicity may be higher on a mass basis than larger particles. There is also evidence that these small particles, or toxic components carried on their surface, can translocate from the lung to the blood and to other organs of the body, or through the olfactory bulb into the brain (U.S. EPA 2009). Currently, there are no federal or California

standards for ultrafine particles. As such, the health effects of ultrafine particles is discussed in a separate section following the discussion of PM10 and PM2.5.

The current federal and California standards for particulate matter are listed in Table I-6.

TABLE I-6

Ambient Air Quality Standards for Particulate Matter

STANDARD	FEDERAL	CALIFORNIA
PM10 24-Hour average	150 μg/m^3	50 μg/m^3
PM10 Annual Average	--	20 μg/m^3
PM2.5 24-Hour Average	35 μg/m^3	--
PM2.5 Annual Average	12 μg/m^3	12 μg/m^3

Short-Term Exposure Effects of PM

Epidemiological studies have provided evidence for most of the effects listed above. In an extensive report focusing on the history of particulate matter research, the U.S. EPA reviewed several well-conducted studies that reported an association between mortality and increased daily or several-day-average concentrations of PM10 (U.S. EPA 2004). In addition, excess mortality and morbidity are reported in many studies involving communities across the U.S. as well as in Europe, Asia, and South America (U.S. EPA 2009; Lu et al. 2015; Shah et al. 2015; Cai et al. 2016), although there are some studies that show no effect for the specific exposures and outcomes evaluated (Milojevic et al. 2014; Wang et al. 2015; Zu et al. 2016). While there were some studies conducted in California, the importance of assessing results from studies from many different locations around the world should not be understated. The repeatability and consistency of results across many locations strengthens the weight of evidence in the determination of causality.

A review and analysis of epidemiological literature for acute adverse effects of particulate matter was published by the American Thoracic Society in 1996, where several adverse effects were listed as associated with daily PM10 exposures (Table I-7). The review also reported that individuals who are elderly or have preexisting lung or heart disease are more susceptible than others to the adverse effects of PM10 (American Thoracic Society 1996a).

TABLE I-7

Combined Effect Estimates of Daily Mean Particulate Pollution (PM10)

	% CHANGE IN HEALTH INDICATOR PER EACH 10 µg/m^3 INCREASE IN PM10
Increase in Daily Mortality	
Total deaths	1.0
Respiratory deaths	3.4
Cardiovascular deaths	1.4
Increase in Hospital Usage (all respiratory diagnoses)	
Admissions	1.4
Emergency department visits	0.9
Exacerbation of Asthma	
Asthmatic attacks	3.0
Bronchodilator use	12.2
Emergency department visits*	3.4
Hospital admissions	1.9
Increase in Respiratory Symptom Reports	
Lower respiratory	3.0
Upper respiratory	0.7
Cough	2.5
Decrease in Lung Function	
Forced expiratory volume	0.15
Peak expiratory flow	0.08

* One study only
(From: (American Thoracic Society 1996a))

Since then, many more recent studies have provided additional evidence that excess mortality and morbidity are associated with short-term exposure to PM10 and PM2.5 (Pope et al. 2006).

Estimates of mortality effects from studies of PM10 exposures range from 0.3 to 1.7 percent increase for a 10 µg/m^3 increase in PM10 levels. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS), a study of 20 of the largest U.S. cities, determined a combined risk estimate of about a 0.5 percent increase in total mortality for a 10 µg/m^3 increase in PM10 (Samet et al. 2000a). This

study also analyzed the effects of gaseous co-pollutants. When the gaseous pollutants were included in the analyses, the estimated associations between PM10 and mortality remained, though they were somewhat reduced. These results suggest that the effects reported in the study are likely due to the particulate exposures; they cannot readily be explained by coexisting weather stresses or other pollutants.

An expansion of the NMMAPS study to 90 U.S. cities also reported association with PM10 levels and mortality (Samet et al. 2000b; Health Effects Institute 2003). After the study was published, it was discovered that some of the study analyses had been performed with incorrect default values. The strong positive association between acute PM10 exposure and mortality remained, both upon reanalysis using revised software and using alternative modeling approaches (Dominici et al. 2002; Health Effects Institute 2003).

Studies of short-term exposures to PM2.5 have also found associations with increases in mortality. The NMMAPS study conducted a national analysis of PM2.5 mortality association for 1999-2000. The risk estimates were 0.29 percent for all-cause mortality and 0.38 percent for cardio-respiratory mortality (Dominici et al. 2007). In its 2009 review, U.S. EPA determined that estimates for PM2.5 generally are in the range of 0.29 to 1.21 percent increase in total deaths per 10 $\mu g/m^3$ increase in 24-hour PM2.5 levels. The estimates for cardiovascular related mortality range from 0.03 to 1.03 percent per 10 $\mu g/m^3$, and for respiratory mortality estimates range from 1.01 to 2.2 percent per 10 $\mu g/m^3$ 24-hour PM2.5 (U.S. EPA 2009). Figure I-4 shows a summary of U.S. and Canadian studies of mortality and short-term PM2.5 exposures, which shows that the most consistent positive associations were seen with cardiovascular and all-cause deaths. Positive associations for respiratory deaths were also seen in several of these studies, although the precision of the estimates for respiratory deaths was lower relative to that of all-cause or cardiovascular deaths.

Several studies have attempted to assess the relative importance of particles smaller than 2.5 µm and those between 2.5 µm and 10 µm (PM10-2.5). While some studies report that PM2.5 levels are better predictors of mortality effects, others suggest that PM10-2.5 is also important. Most of the studies found higher mortality associated with PM2.5 levels than with PM10-2.5. For example, a study of six cities in the U.S. found that particulate matter less than 2.5 µm was associated with increased mortality, but that the larger particles were not. In the U.S. EPA review (U.S. EPA 2009), several studies were presented that found associations of PM10-2.5 and mortality. Some of the studies showed differences by region of the U.S. In one study of 47 U.S. cities that had both PM2.5 and PM10 data available to calculate PM10-2.5 as a difference, overall, the study found a significant association between the computed PM10-2.5 and all-cause, cardiovascular, and respiratory mortality. The study also reported differences by season and climate area (Zanobetti et al. 2009).

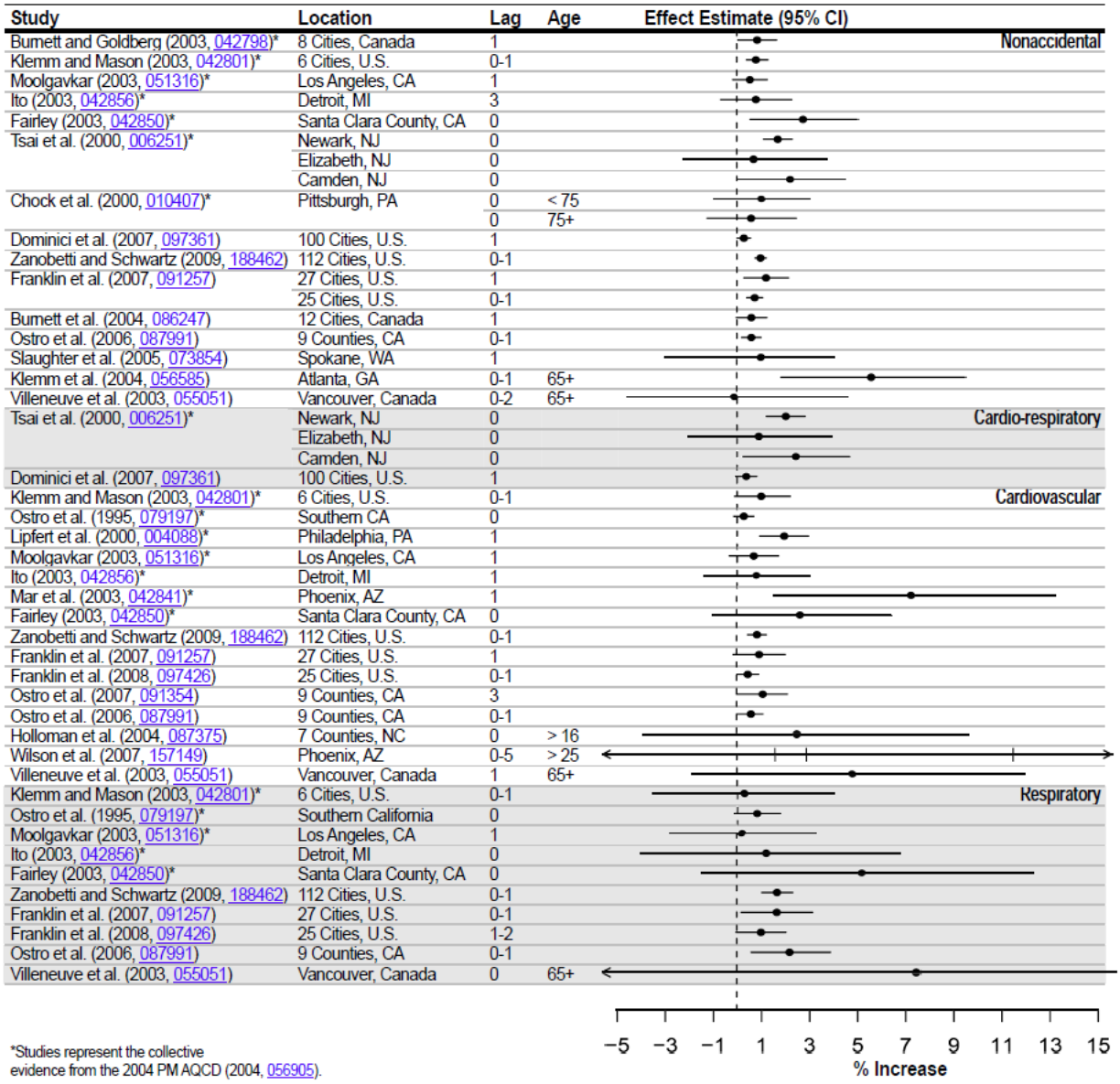


FIGURE I-4

Summary of Non-accidental All-Cause and Cause-Specific Mortality per 10 µg/m3 Increase in PM2.5 Short-term Exposures, for U.S.- and Canadian-based studies (from (U.S. EPA 2009), Figure 6-27). "Lag" indicates the number of days between the exposure and the outcome assessed.

A major knowledge gap in understanding the relative importance of "fine" PM (PM2.5) and "coarse" PM (PM10-2.5) is the relative lack of direct measurements of PM10-2.5. Most estimates are made by subtracting PM2.5 from PM10 measured at co-located samplers, a process that is subject to errors that are inherent in the subtracting of one relatively large number from another. More research is needed to better assess the relative effects of coarse (PM10-2.5) fractions of particulate matter on mortality. A graph from the U.S. EPA review is included in the figure below to demonstrate ranges

of mortality findings associated with coarse particulates. Consistent positive associations are seen, particularly for cardiovascular and nonaccidental all-cause mortality, with varying degrees of precision across the different studies.

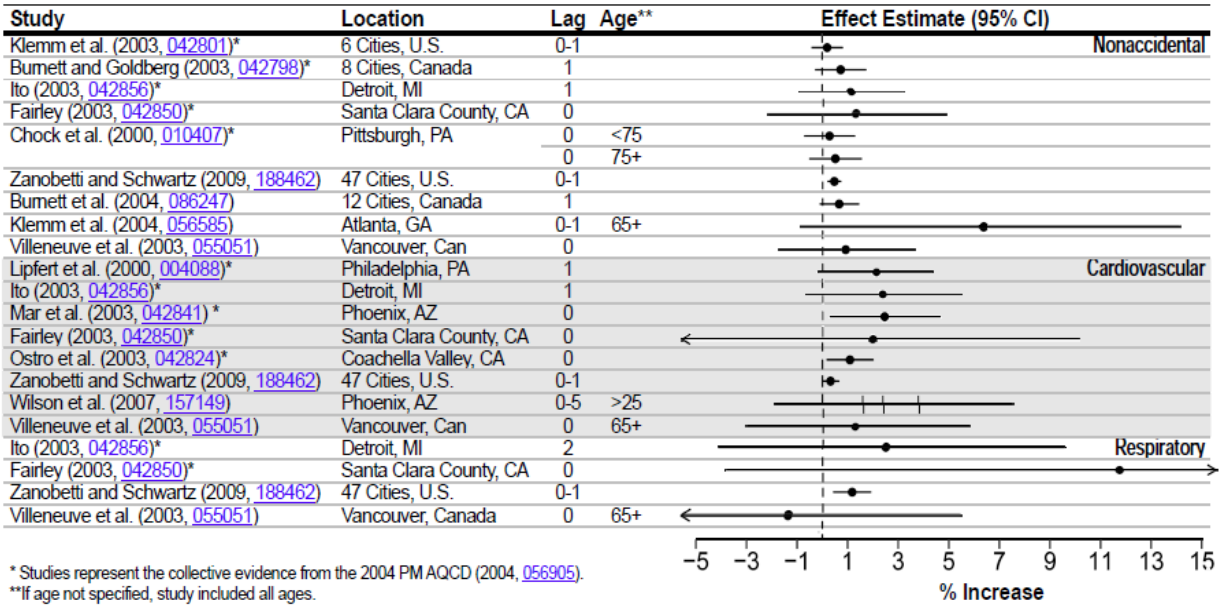


FIGURE I-5

Summary of Percent Increase in Total (Nonaccidental) and Cause-Specific Mortality Per 10 µg/m3 Increase in PM10-2.5 Short-term Exposure (from (U.S. EPA 2009), Figure 6-30). "Lag" indicates the number of days between the exposure and the outcome assessed.

A number of studies have evaluated the association between particulate matter exposure and indices of morbidity such as hospital admissions, emergency room visits or physician office visits for respiratory and cardiovascular diseases. The effect estimates for these various morbidities are generally higher than the estimates for mortality. Observed effects have been associated with PM10, PM2.5 and PM10-2.5.

In the NMMAPS study, hospital admissions for those 65 years or older were assessed in 14 U.S. cities. Several models were compared to estimate associations of hospital admissions for specific disease categories and short-term PM10 levels. Hospital admissions showed an increase ranging from 0.68 – 1.47 percent for cardiovascular diseases, a range of 1.46 – 2.88 percent increase for COPD, and a range of 1.31 – 2.86 percent increase for pneumonia per 10 µg/m^3 increase in PM_{10} (Samet et al. 2000b). In the reanalysis of the study (Health Effects Institute 2003), it was found that when using different models, the pollution coefficients were generally lower. However, the authors note that most of the conclusions of associations with PM10 exposures and hospital admissions held. Two recent Southern California studies evaluated associations between short-term PM2.5 levels and asthma-related hospital or emergency admissions. One study, based in Orange County, reported

increased risk of asthma-related hospital encounters with increased ozone and PM2.5 in the warm seasons, and with CO, NO_x, and PM2.5 in the cool seasons (Delfino et al. 2014). The second study, conducted in Los Angeles County, reported monthly average PM2.5, CO, and NO_2 levels were positively associated with asthma hospitalization rates (Delamater et al. 2012).

Similarly, school absences, lost workdays, and restricted activity days have also been used in some studies as indirect indicators of acute respiratory conditions (Ostro 1987; Ostro 1990; Ransom et al. 1992; Gilliland et al. 2001; Park et al. 2002; Hales et al. 2016). These observations help support the hypotheses that particulate matter exposures increase inflammation in the respiratory tissues and may also increase susceptibility to infection (U.S. EPA 2009).

Some studies have reported that short-term particulate matter exposure is associated with changes in lung function (lung capacity and breathing volume); upper respiratory symptoms (hoarseness and sore throat); and lower respiratory symptoms (increased sputum, chest pain and wheeze). The severity of these effects is widely varied and is dependent on the population studied, such as adults or children with and without asthma. Sensitive individuals, such as those with asthma or pre-existing respiratory disease, may have increased or aggravated symptoms associated with short-term particulate matter exposures. Several studies have followed the number of medical visits associated with pollutant exposures. A range of increases from 1 to 4 percent for medical visits for respiratory illnesses was found corresponding to a 10 $\mu g/m^3$ change in PM10. A number of studies also looked at levels of PM2.5 or PM10-2.5. The findings suggest that both the fine and coarse fractions may have associations with some respiratory symptoms (U.S. EPA 2009). Among the newer health endpoints evaluated in recent studies of short-term effects of PM2.5 is stroke. One recent meta-analysis evaluated 16 studies of short-term PM2.5 exposures and estimated a 5 percent increased risk of stroke for each 10 $\mu g/m^3$ increase in PM2.5 (Shin et al. 2014).

The biological mechanisms by which particulate matter can produce health effects have been investigated in laboratory studies. Brook et al. (Brook et al. 2010) summarized three likely pathways by which PM exerts it effects on cardiovascular health outcomes: (1) PM can activate inflammatory pathways and cause systemic oxidative stress, leading to the production of pro-inflammatory cytokines; (2) PM can disrupt the autonomic nervous system leading to increased blood pressure, increased arrhythmic potential, and decreased heart rate variability; and (3) PM, particularly UFPs or particle constituents such as organic compounds and metals, can enter the bloodstream and cause increased constriction of the blood vessels and increased blood pressure. Each of these pathways may also lead to the formation of reactive oxygenated species (ROS, or free radicals) that can cause DNA oxidation and systemic inflammation. Inflammatory responses in the respiratory system in humans and animals can lead to inflammation in fat tissues and in the liver, which can lead to vascular dysfunction (e.g. atherosclerosis), changes in metabolic function (e.g. insulin resistance), and increased thrombotic potential (Brook et al. 2010). Several reviews discuss mechanistic studies in detail (Brunekreef et al. 2002; Brook et al. 2004; Brook et al. 2010). A study in cells using ambient air samples in communities near railyards in the South Coast Air Basin found that the PM2.5 phase of ambient air pollution contains prooxidant components, primarily metals, which can trigger an

inflammatory response in the cells (Eiguren-Fernandez et al. 2015; Cho 2016). The same study noted that vapor phase pollutants, which contain most of the electrophiles, may trigger a different biological response in the cells, suppressing inflammatory responses and could result in a reduced ability to fight off infections.

Some studies have examined the health effects of short-term exposures to specific PM constituents and sources (Lippmann 2014; Basagana et al. 2015; Atkinson et al. 2016). While there is some evidence suggesting possible links with specific constituents or sources, such as diesel exhaust, sulfates (related to coal combustion), and certain metals, the U.S. EPA determined that there were not enough studies evaluating short-term constituent- or source-specific exposures at the time of the previous Integrated Science Assessment to be able to make a causal determination (U.S. EPA 2009).

Long-Term Exposure Effects of PM

Numerous studies have evaluated the health effects of long-term (months to years) or chronic exposure to particulate matter, with the largest number of studies examining cardiovascular and respiratory health endpoints, as well as mortality. Other health outcomes that have been linked to long-term PM exposures include reproductive effects, cancer outcomes, and, more recently, metabolic syndromes and neurological effects. The U.S. EPA 2009 Integrated Science Assessment for Particulate Matter (ISA for PM) concluded that sufficient evidence is available to support a causal determination for long-term PM2.5 exposures and cardiovascular and mortality effects, and a likely causal relationship for respiratory effects. A summary of the evidence is presented below, focusing on the long-term effects of PM2.5 exposures.

Many research studies, including some recent studies, have evaluated the health effects of exposures to air pollutants from traffic emissions using a variety of exposure modeling techniques (Hart et al. 2014; Harris et al. 2015; Kingsley et al. 2015; Rice et al. 2015; Danysh et al. 2016). In general, these articles are not discussed in detail here, because of the difficulty in attributing the observed effects to a specific pollutant or combination of pollutants. However, these studies do provide supporting evidence that air pollutants from traffic exhaust are linked to health effects in humans.

Long-Term Particulate Matter Exposures and Mortality

Since the initial promulgation by U.S. EPA of the National Ambient Air Quality Standards for PM2.5, controversy has remained over the association of mortality and exposures to PM2.5. Several large, prospective cohort studies conducted in the U.S. and Canada were used to evaluate long-term PM exposures and mortality, including total number of deaths and deaths due to specific causes. The strongest and most consistent evidence of long-term PM2.5 effects are for cardiovascular mortality, particularly ischemic heart disease, and there is evidence that ambient PM2.5 exposure is associated with and lung cancer mortality (Dominici et al. 2006; Krewski et al. 2009; Jerrett et al. 2013; International Agency for Research on Cancer 2015). Below is a brief discussion of the evidence linking

PM and mortality reviewed in the U.S. EPA 2009 ISA along with more recently published studies, with a focus on large prospective studies and studies conducted in California or Southern California.

In the assessment of evidence for mortality outcomes linked to long-term PM exposures, the 2009 U.S. EPA ISA for PM reviewed 15 studies evaluating PM2.5 exposures, 2 studies evaluating PM10-2.5 exposures, and 5 studies evaluating PM10 exposure. The majority of these studies were conducted in the United States, and 3 of the studies of PM2.5 exposures were conducted in California or Southern California. Previous reviews conducted in 1996 and 2004 by U.S. EPA assessed evidence primarily from large prospective cohort studies, such as the Harvard Six Cities Study (Dockery et al. 1993), the American Cancer Society (ACS) Study (Pope et al. 1995; Pope et al. 2002), and the Seventh-Day Adventist Health Air Pollution (AHSMOG) Study (Abbey et al. 1999; McDonnell et al. 2000). The U.S. EPA 2004 PM Air Quality Criteria Document concluded that there was strong evidence linking long-term PM2.5 exposures to all-cause and cardiopulmonary mortality, but not enough evidence for a link with PM10-2.5. The 2009 U.S. EPA ISA for PM similarly concluded that the newer studies provide additional evidence to support a causal determination for long-term PM2.5 exposures and increased mortality risk, but there continues to be insufficient evidence supporting such a link with particles in the coarse fraction. This most recent U.S. EPA review evaluated the additional updated analyses of the previously-established large cohort studies (Harvard Six Cities, ACS, AHSMOG, and Veterans studies), and noted two new major cohorts that provide further evidence linking PM2.5 and mortality: the Women's Health Initiative (WHI) study (Miller et al. 2007) and the Medicare Cohort Studies (Eftim et al. 2008).

The American Cancer Society Cancer Prevention Study II (ACS) is a large, prospective national cohort study of over one million participants in the U.S. recruited from all 50 states, the District of Columbia and Puerto Rico, and followed over many years. Over the past two decades, studies using data from this cohort have reported associations for PM2.5 for both total mortality and cardiorespiratory mortality (Pope et al. 1995; Krewski 2000; Pope et al. 2002; Jerrett et al. 2005; Krewski et al. 2009; Jerrett et al. 2013; Pope et al. 2015). The survey included several measures of smoking and exposure to second-hand smoke, which were included in the statistical models to account for the potential confounding effects of smoking. The original study reported that long-term exposures to fine particulate air pollution were associated with cardiopulmonary and lung cancer mortality (Pope et al. 1995). In a reanalysis of the data (Krewski 2000), mortality rates and PM2.5 levels were analyzed for 50 metropolitan areas of the U.S. Average (median) levels from monitors in each metropolitan area were used to estimate PM2.5 exposures. At these levels of aggregation, regional differences in the association of PM2.5 and mortality were noted, with higher mortality risks in the Northeast and Midwest, and more moderate mortality risks in the West.

Another follow-up study of the American Cancer Society cohort confirmed and extended the findings in the initial study. The researchers estimated that, on average, a 10 $\mu g/m^3$ increase in fine particulates was associated with approximately a 4 percent increase in total mortality, a 6 percent increase in cardiopulmonary mortality, and an 8 percent increase in risk of lung cancer mortality (Pope et al. 2002). In an additional reanalysis and extension of the American Cancer Society cohort

from 1982 to 2000 (Krewski et al. 2009), and including additional metropolitan areas for the most recent years, effects estimates on mortality were similar, though somewhat higher than those reported previously. The extended analyses included an additional 11 years of cohort follow-up compared to the original study. The authors reported positive and significant association between a 10 $\mu g/m^3$ change in PM2.5 level and all-cause, cardiopulmonary disease, and ischemic heart disease deaths. Mortality from ischemic heart disease was associated with the largest risk estimates.

Subsets of the ACS study data have also been evaluated to estimate effects in California and the metropolitan Los Angeles area (Jerrett et al. 2005; Jerrett et al. 2013). These results are discussed further below, along with results of other California or Southern California-based studies.

The Harvard Six Cities Study is a large prospective cohort study of adults in six U.S. cities, and began in the year 1974. The original analysis and a subsequent reanalysis found positive associations between particulate matter and sulfate in relation to mortality, after controlling for potential confounding factors such as smoking status, sex, age, and other factors (Dockery et al. 1993)(Krewski 2000). An extension of the Harvard Six Cities Cohort confirmed the association of mortality with PM2.5 levels, and reported that improvements in PM2.5 levels over the study time period were associated with decreased mortality risk (Laden et al. 2006). An update to this study covering the years 1974 to 2009 found a linear relationship of PM2.5 levels and mortality from all causes, cardiovascular causes, and from lung cancer (Lepeule et al. 2012). According to the authors, the PM2.5 levels decreased over time, but no evidence of a threshold for these effects was found.

AHSMOG is a cohort study of non-Hispanic white Seventh-day Adventists in California, with participants followed starting from the late 1970's. Confounding due to smoking in this study is unlikely due to very low smoking rates in this population; however, the study is limited in its the ability to apply the findings to other population groups. The study has linked long-term PM10 exposures and other air pollutants to deaths from all natural causes and deaths due to lung cancer among males (Abbey et al. 1999), although the authors concluded that these associations were likely due to exposures to fine particles rather than the coarse fraction of PM10 (McDonnell et al. 2000). In a re-analysis of the data, the study found PM2.5 was associated with an increased risk of coronary heart disease mortality among females but not among males (Chen et al. 2005). Similar associations among females only were found for coarse particles and PM10.

Other cohort studies include an analysis of mortality and PM2.5 exposures in a Medicare enrollee population. Zeger et al. (Zeger et al. 2008) assembled a Medicare enrollee cohort by including all Medicare enrollees residing in over 4,500 zip codes with centroids within six miles of a PM2.5 monitor. PM2.5 data was obtained from the monitoring stations, and mean annual levels were calculated for the zip codes within six miles of each monitor. The authors found that long-term exposures to PM2.5 was associated with all-cause mortality for the eastern and central portions of the U.S., and these mortality risk estimates were similar to those previously published in the Six Cities Study and the American Cancer Society cohorts. The authors reported that there were no statistically significant associations between zip code levels of PM2.5 and all-cause mortality rates in the western

region of the U.S. This finding was attributed largely to the higher PM2.5 levels in Los Angeles area counties compared to other western urban areas, but there were not higher mortality rates in the Los Angeles area counties. Several factors could explain this finding. The authors note that the toxicity of the PM mixture may differ by location, e.g. with higher PM2.5 sulfate levels in the eastern region. In addition, the use of ecological data rather than individual-level data for exposure assessment and some confounding factors, and the assessment of all-cause mortality rather than cause-specific mortality may have impacted the results of this study. For example, the authors used county-level COPD risk as an estimate of smoking prevalence, because individual-level measures of smoking were not available. The authors further reported that they found no associations of PM2.5 with all-cause mortality in persons aged 85 years or higher, which may reflect other competing causes of death in this age group not related to air pollution exposures.

The Women's Health Initiative (WHI) Study is a nationwide cohort of post-menopausal women in 36 metropolitan areas of the U.S. who had no history of cardiovascular disease (Miller et al. 2007). The study found that long-term exposure to PM2.5 was associated with a 24 percent increased risk of cardiovascular disease and a 76 percent increased risk of death from cardiovascular causes for each additional 10 $\mu g/m^3$ of PM2.5; these relative risk estimates are larger than those reported in the ACS and Six Cities Studies, but differences in health status, PM composition, and overall mortality risk in these distinct populations may account for such differences in the effect estimates. The WHI study results accounted for the potential confounding effects of several factors, including medical risk factors for cardiovascular disease, measures of socioeconomic status, and cigarette smoking. Another large cohort study focusing on women is the Nurses' Health Study, which found that PM10 exposures were associated with all-cause mortality and fatal coronary heart disease, with exposures 24 months prior to death having the strongest effects (Puett et al. 2008). These results accounted for several potential confounders, including smoking status and history, medical risk factors for cardiovascular disease, and area-level measures of socioeconomic status. This study did not evaluate PM2.5 exposures.

A recent pooled analysis of 22 European cohorts and including over 350,000 participants evaluated long-term air pollution exposures and exposure to PM2.5, PM10, and nitrogen oxides, using land use regression models to estimate exposures (Beelen et al. 2014). The authors reported that a 5 $\mu g/m^3$ increase in PM2.5 was associated with approximately a 7 percent increase in mortality from natural causes.

Estimates of mortality risks associated with long-term PM2.5 levels from recent studies are shown in the figure below. The recent evidence is consistent with past studies, showing increased risk of premature death with increased PM2.5 exposures. For cause-specific mortality, consistent positive associations are seen with cardiovascular mortality endpoints and with lung cancer deaths, but weak associations are seen with overall respiratory mortality.

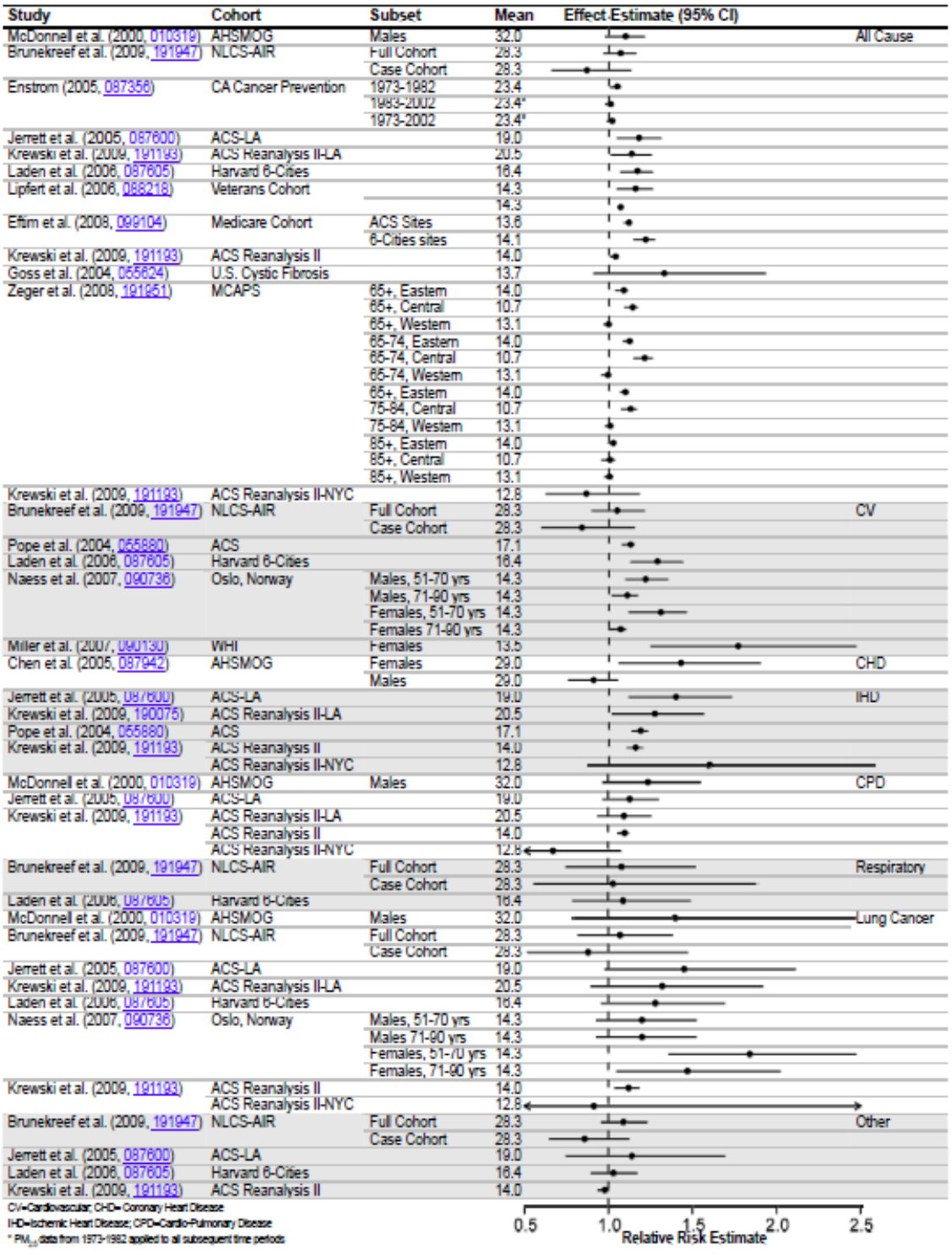


FIGURE I-6

Mortality Risk Estimates, Long-Term Exposure to PM2.5 in Cohort Studies (From (U.S. EPA 2009), Figure 7-7). "Mean"=mean PM2.5 exposure estimates in the study. CV=cardiovascular, CHD=coronary heart disease, IHD=ischemic heart disease, CPD=cardiopulmonary disease.

In addition to the AHSMOG study, other analyses of mortality and PM2.5 levels specific to California have also been reported, including an analysis of a subset of the ACS II data. An analysis of the ACS II study (Jerrett et al. 2013) followed individuals in California from that cohort recruited starting in 1982, with follow-up to 2000. PM2.5 levels at subject residences were estimated using land use regression models. Over 40 potential confounders were included in the statistical models, and included individual-level variables (e.g. smoking, diet, demographic, and other factors) and neighborhood-level variables (e.g. unemployment, poverty, income inequality, racial composition). The authors noted that mortality rates differ in urban areas compared to non-urban areas, and adjusted for urban/rural status in the model to estimate pollution effects on mortality. All-cause mortality, mortality from cardiovascular disease, and mortality from ischemic heart disease were positively associated with PM2.5 levels in single-pollutant models. These associations with PM2.5 remained after additional adjustment for ozone levels. Because of moderate correlations across pollutants, it may not be possible to draw conclusions about which pollutant(s) in this mixture cause the observed effects. Positive associations of all-cause and certain cause-specific mortality rates with estimated NO_2 and ozone levels were also found. The authors concluded that these results indicate that several components of combustion-related pollutant mixture are associated with mortality.

A study analyzed data from the California Teachers Study cohort of over 100,000 active and retired school teachers recruited in 1995, and followed through 2005 (Lipsett et al. 2011). Pollutant exposures at the subject residences were estimated using data from ambient monitors, and extrapolated using a distance-weighted method. The authors reported that a 10 $\mu g/m^3$ increase in PM2.5 was associated with a 20 percent risk increase in mortality from ischemic heart disease, but no associations were found with all-cause, cardiovascular, or lung cancer mortality. A 10 $\mu g/m^3$ increase in PM10 was associated with increased risk of ischemic heart disease and incident stroke. These results accounted for several individual- and neighborhood-level factors, including smoking, second-hand smoke, medical risk factors for cardiovascular disease, and indicators of socioeconomic status.

A more recent analysis of the California Teachers Study cohort from 2001 through 2007 estimated the association between particulate pollutants and all-cause, cardiovascular, ischemic heart disease, and respiratory mortality (Ostro et al. 2015). Exposure data at the residential level were estimated by a chemical transport model that computed pollutant concentrations from over 900 sources in California. Besides particle mass, monthly concentrations of 11 species and 8 sources or primary particles were generated at 4-km grids. The results were reported as finding statistically significant associations of ischemic heart disease mortality with PM2.5 mass and several of its components (Figure I-7). The study also found significant positive associations between ischemic heart disease mortality and ultrafine particle mass as well as several ultrafine particulate components including elemental carbon, organic carbon, copper, metals, meat cooking, and mobile source derived components. An earlier study using data from the same cohort had used monitoring data to estimate mortality risk, and similarly reported increased risk of all-cause, cardiopulmonary, and ischemic heart disease mortality with higher exposures to PM2.5 mass. This study also reported increased ischemic heart disease risk with higher exposures to PM2.5 constituents such as organic carbon, sulfates, and

nitrates (Ostro et al. 2010). Both studies adjusted for several individual- and neighborhood-level covariates, including smoking status and indicators of socioeconomic status.

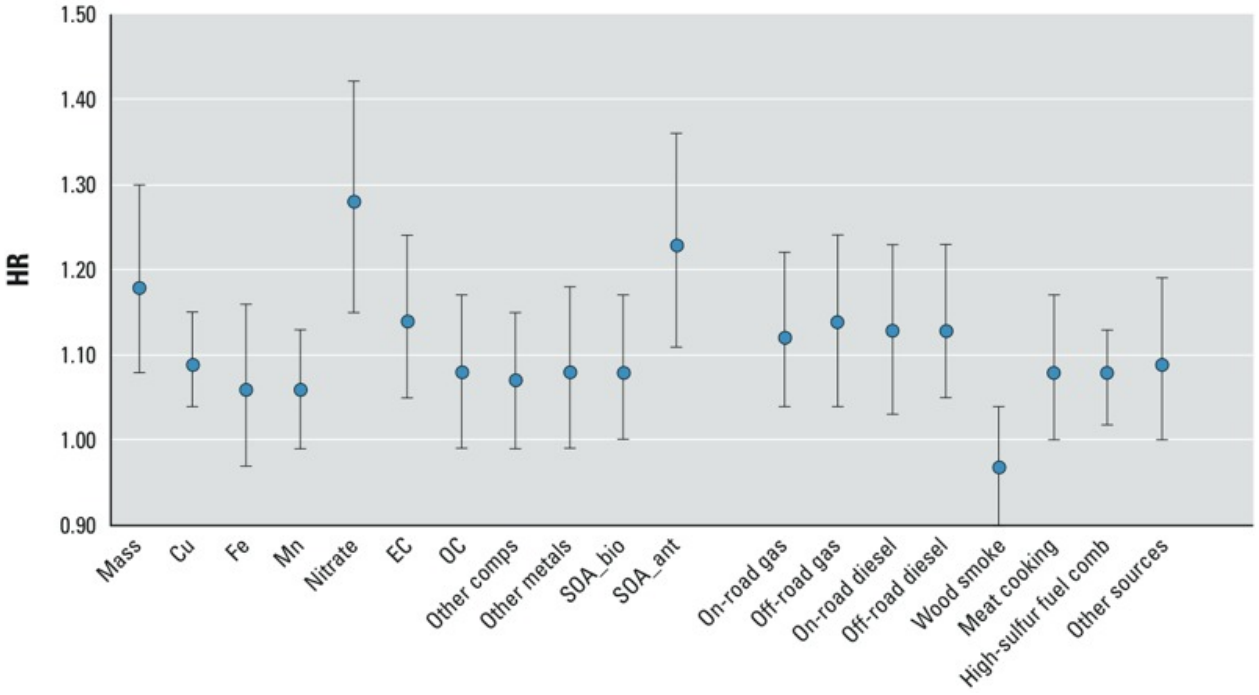


FIGURE I-7

Association of PM2.5 constituents and sources with Ischemic Heart Disease mortality (Hazard Ratios and 95 percent Confidence Intervals) using interquartile range. Abbreviations: comb = combustion; comps = components; SOA_bio= secondary organic aerosols from biogenic sources (derived from long-chain alkanes, xylenes, toluenes, and benzene and their oligomers); SOA_ant=secondary organic aerosols from biogenic sources (derived from isoprenes, monoterpenes, and sesiquiterpenes and their oligomers). (From (Ostro et al. 2015))

A cohort of elderly individuals (average age of 65 years in 1973) recruited from 11 California counties was followed over several years (Enstrom 2005). A positive association for long-term PM2.5 exposure with all-cause deaths was reported from 1973–1982. However, no significant association was found in the later time period of 1983–2002. PM2.5 levels were obtained from measurements made during 1979- 1983 by the EPA as part of the Inhalable Particle Monitoring Network and the cohort was confined to those participants in the American Cancer Society Cancer Prevention Study I who were living in the 11 counties that had one of the monitors. Pollutant levels were estimated using data from these monitors and averaged over each county, which may lead to exposure misclassification and bias toward finding no effect. The study adjusted for several potential confounding factors, including demographic factors, smoking, body mass index, and other factors.

The California Air Resources Board recently conducted a cross-sectional study of long-term PM2.5 exposures in rural and urban areas within California, using ambient monitoring data from 116

stations in the monitoring network, and calculating zip code-level exposure estimates (Garcia et al. 2016). The study observed larger effect sizes for increased PM2.5-related mortality risk in rural compared to urban areas from all causes, cardiovascular disease and cardiopulmonary disease. In urban areas, the study found PM2.5 exposures to be associated with increased risk of cardiovascular disease, ischemic heart disease, and cardiopulmonary disease; however, for all-cause non-accidental mortality risk, only an exposure model restricted to people living within 10 km of a monitoring station in urban areas showed an association with PM2.5. This study did not control for the potential confounding effects of smoking.

A recent study analyzed data from the National Institutes of Health AARP Diet and Health cohort, including about 160,000 participants in California (Thurston et al. 2016). Census tract-level PM2.5 exposures were estimated based on land use regression models. For the California cohort, PM2.5 levels were associated with an approximately 10 percent increase in cardiovascular disease mortality risk for each additional 10 $\mu g/m^3$ of PM2.5. A small but positive effect estimate was found for all-cause mortality in California, and no association was found for respiratory mortality in the California cohort, although the estimates indicated uncertainty in the magnitude and direction of these effects. This study adjusted for several potential confounders, including demographic factors, smoking, and indicators of socioeconomic status.

A few studies have focused on particulate matter exposure and health effects in residents of Southern California. Two analyses of the American Cancer Society II cohort, for example, focused specifically on the Los Angeles Metropolitan area using methods to estimate exposures on a finer geographical scale than previous studies that used geographic scales at the county or metropolitan area. Improved exposure estimation methods reduce potential bias from exposure misclassification. Using data from monitoring stations in the Los Angeles area, one study applied interpolation methods (Jerrett et al. 2005) and another applied land use regression techniques (Krewski et al. 2009) to estimate PM2.5 exposures to the study participants. Significant associations of PM2.5 with mortality from all causes and cardiopulmonary disease were reported, with the magnitude of risks being higher than those from the national studies of the American Cancer Society II cohort. Such improved exposure estimation techniques can reduce misclassification bias in epidemiological studies. It should be noted that various analyses were presented in these as well as other studies to estimate the influence of various individual-level and ecologic variables that might also be related to health effects risks. Including such variables helps control for potential confounding, but generally reduces the estimated association between PM2.5 and all-cause mortality. It may be illustrative to describe some of the estimates from the various calculations as presented by the authors of the Los Angeles area cohort (Krewski et al. 2009). In the descriptions in Table I-9, HR refers to the "hazard ratio" expressed for a 10 $\mu g/m^3$ change in PM2.5 exposure, followed by the 95 percent Confidence Interval. For example, if the hazard ratio is 2, the risk would be twice as high; and, conversely if the hazard ratio is 0.5, the risk would be one-half of that of the reference group. Several of the analyses results follow as excerpted from Krewski, 2009. Table I-8 includes PM2.5, plus various additional individual and ecological variables. Similar effects of covariate adjustment were seen for hazard ratios for

mortality from ischemic heart disease, although effect estimates were stronger for ischemic heart disease mortality compared to those for all-cause mortality.

TABLE I-8

Influence of Adding Confounding Variables on All-Cause Mortality

VARIABLE INCLUDED	HAZARD RATIO per 10 µg/m^3 change in PM2.5 exposure
PM2.5 alone (stratified for age, sex, and race)	1.197 (95% CI, 1.082–1.325);
PM2.5 with 44 individual-level covariates*	1.143 (95% CI, 1.033–1.266)
PM2.5 with 44 individual-level covariates and the ecologic covariate of unemployment	1.127 (95% CI, 1.015–1.252)
PM2.5 with 44 individual-level covariates and social factors extracted from the principal component analysis (which account for 81% of the total variance in the social variables)	1.142 (95% CI, 1.026–1.272).
PM2.5 with 44 individual-level covariates and all ecologic covariates that were individually associated with mortality in bivariate models with PM2.5 exposure	1.115 (95% CI, 1.003–1.239)
PM2.5 parsimonious model that included 44 individual-level covariates and ecologic confounder variables that both reduced the pollution coefficient and had associations with mortality	1.126 (95% CI, 1.014–1.251)

*These covariates included several measures of smoking.
(From Krewski, 2009)

U.S. EPA also released a Regulatory Impact Analysis (U.S. EPA 2012) which looked at the costs and benefits of alternate PM2.5 standard levels. As part of the analysis, U.S. EPA looked at California-specific studies regarding PM2.5 and mortality published in the scientific literature. The U.S. EPA analysis concluded "most of the cohort studies conducted in California report central effect estimates similar to the (nation-wide) all-cause mortality risk estimate we applied from Krewski et al. (2009) and Laden et al. (2006) albeit with wider confidence intervals. A couple of cohort studies conducted in California indicate higher risks than the risk estimates we applied." Thus, in U.S. EPA's judgment, the California-related studies provided estimates of mortality consistent with or higher than those from the national studies.

At the time of the 2009 ISA, few studies had examined long-term exposures to chemical-specific PM constituents or compared source-specific PM effects on mortality (U.S. EPA 2009). The 2009 ISA discussed only two studies that used direct measurements of PM constituents other than sulfates: the Veteran's Cohort (Lipfert et al. 2006) and the Netherlands Cohort Study (Beelen et al. 2008). These studies found mortality associations with long-term exposures to traffic pollutants, nitrates and sulfates.

With measures adopted to control emissions of air pollutants, ambient levels of PM2.5 have been decreasing. These reductions in particulate matter have been associated with reductions in mortality. For example, studies have found that increases in life expectancy are associated with reductions in air pollution levels, and that a portion of this increase can be attributed to reductions in PM2.5 exposures (Correia et al. 2013; Pope et al. 2013).

Long-Term Particulate Matter Exposures and Cardiovascular Effects

Studies of cardiovascular mortality provide the strongest evidence of an association between PM2.5 exposures and cardiovascular effects. The U.S. EPA 2009 ISA review determined that the evidence is sufficient to infer a causal relationship between long-term PM2.5 exposures and cardiovascular effects. In addition to the studies of mortality, other epidemiological studies provide additional evidence of sub-clinical and clinical cardiovascular effects, while toxicological studies suggest a plausible biological mechanism for such effects (Fanning et al. 2009; U.S. EPA 2009).

Epidemiological studies of subclinical effects typically have used subclinical measures of atherosclerosis, which is an underlying disease contributing to many clinical cardiovascular outcomes such as myocardial infarction, sudden cardiac death, stroke, and vascular aneurysms (U.S. EPA 2009). A study in Southern California residents used the carotid intima-media thickness (CIMT) as a measure of subclinical atherosclerosis (Kunzli et al. 2005). The subjects' residential areas were geocoded and a geospatial extrapolation of ambient monitoring data was used to assign annual mean concentrations of ambient PM2.5. The authors report results of an association between atherosclerosis and ambient air pollution as measured by PM2.5. The associations of PM2.5 and CIMT were strongest in women ≥ 60 years of age. The Multi-Ethnic Study of Atherosclerosis (MESA) is a population-based study of people living in 6 U.S. cities or counties, including Los Angeles, CA (Diez Roux et al. 2008). The MESA study reported that 20-year average PM2.5 exposures corresponded to a small increase in CIMT, although the magnitude of the increase was much smaller than the Kunzli 2005 study. The study accounted for the potential influence of sociodemographic factors, lipid status, smoking, diabetes, body mass index, and geographical location. Such differences may be attributable to differences in the study populations. Other sub-clinical outcome measures for atherosclerosis in the MESA study were weakly associated or not associated with PM exposures.

Clinical cardiovascular outcomes have also been examined in several epidemiological studies, including two that were based on prospective cohort studies: the Women's Health Initiative (WHI) Observational Study (Miller et al. 2007) and the Nurses' Health Study (Puett et al. 2008). Both these studies also examined cardiovascular mortality, and found links with long-term particulate matter

exposures. The WHI study included only women who were free of cardiovascular disease at enrollment, and estimated PM2.5 exposures using a nearest monitor approach. The study found PM2.5 exposures to be associated with cardiovascular disease outcomes, including myocardial infarction, revascularization, stroke, coronary heart disease death, and cerebrovascular disease, and accounted for the several potential confounding factors, such as sociodemographic factors, medical risk factors for cardiovascular disease, and cigarette smoking (Miller et al. 2007). An analysis of the Nurses' Health Study included women without a history of myocardial infarction and who lived in certain metropolitan areas in the northeastern U.S. (Puett et al. 2008). Long-term PM10 exposures were estimated using land use regression models as well as air pollution monitoring data, and the results accounted for potential confounding by smoking status and history, medical risk factors for cardiovascular disease, and area-level measures of socioeconomic status. This study found positive associations with the risk of all-cause and coronary heart disease mortality, and the results were suggestive of a link to coronary heart disease events although there was a great deal of uncertainty in this result. Other studies conducted in the U.S. and Europe have examined clinical cardiovascular outcomes with varying results (U.S. EPA 2009).

The U.S. EPA 2009 ISA concluded that epidemiologic studies, along with toxicological evidence linking PM exposures to atherosclerosis and other cardiovascular outcomes, provides evidence linking PM to cardiovascular effects and mortality. While the associations between PM and subclinical and clinical measures have inconsistent results, the consistency of the studies linking PM exposures to cardiovascular mortality and the coherence of the toxicological studies provide support for U.S. EPA's causal determination.

Long-Term Particulate Matter Exposures and Respiratory Effects

The U.S. EPA 2009 ISA review determined that the evidence for long-term particulate matter exposures on respiratory effects is likely to be causal. Several studies, including prospective cohort studies, have assessed the effects of long-term particulate matter exposure on respiratory symptoms and lung function changes. Consistent, positive associations have been found with respiratory symptoms, such as bronchitis, poorly controlled asthma, and decreased lung function in children (U.S. EPA 2009; Guarnieri et al. 2014). Since many of the studies of children included survey measures, these studies typically controlled for the potential confounding effect of tobacco smoking by the child and exposure to second-hand smoke at home, and some studies were also able to account for exposure to maternal smoking *in utero*.

The Southern California Children's Health Study established cohorts of school children from 12 Southern California communities, and followed these participants over time. One of the early studies from this cohort reported positive associations of particulate matter with prevalent bronchitis or phlegm among children with asthma. These effects were also associated with NO_2 and acid vapor levels (McConnell et al. 1999). Another study based on this cohort reported a lower rate of growth in lung function in children living in areas with higher levels of particulate pollution (Gauderman et al. 2000). Decreases in lung function growth were associated with PM10, PM2.5, PM10-2.5, acid

vapor, and NO_2. There was no association with ozone levels. The investigators were not able to identify independent effects of the pollutants but noted that motor vehicle emissions are a major source of the pollutants.

A follow-up study on a second cohort of children confirmed the findings that decreased lung function growth was associated with particulates, nitric oxides, and elemental carbon levels (Gauderman et al. 2002). Elemental carbon is often used as a measure for diesel particulate. Additionally, children who moved to areas with less air pollution were found to show improvement in lung function growth rate, while those who moved to areas with higher PM10 and NO_2 showed declines in lung function growth rates (Avol et al. 2001). By the time the fourth graders graduated from high school, a significant number showed lower lung function. The risk of lower lung function was about four times higher in children with the highest PM2.5 exposure when compared to the lowest exposure communities (Gauderman et al. 2004).

A follow-up report from the Children's Health Study assessed whether improving air quality in Southern California over the past decade has led to beneficial changes in health (Gauderman et al. 2015). It was reported that as the levels of nitrogen oxide and fine particulates were reduced as the result of reductions in air pollution emissions, the deficits in lung function growth were also of a smaller magnitude. Recently, the Children's Health Study cohort data were also used to evaluate associations with bronchitic symptoms in children (Berhane et al. 2016). The study found that reductions in NOx, ozone, and PM10 and PM2.5 were associated with decreases in bronchitic symptoms, with stronger effects observed in children with asthma. These results indicate that improvements in air quality, as measured by fine particulate and nitrogen oxides, are associated with improvements in children's health in Southern California.

A limited number of studies have linked PM exposures to asthma incidence. In an analysis of the Children's Health Study in Southern California, Islam et al. found that while children with better lung function are generally at lower risk of developing asthma, living in an area with long-term average PM2.5 levels ≥13.7 $\mu g/m^3$ offset this protective characteristic; in other words, this study related high PM2.5 levels with new-onset asthma in children (Islam et al. 2007). The U.S. EPA 2009 ISA report also reviewed two European studies that linked PM2.5 with asthma onset in children (Brauer et al. 2007) and adults (Kunzli et al. 2009). Two recent studies were identified in our literature search: the first study used the Sister Study national cohort and found that a 3.6 $\mu g/m^3$ increase in PM2.5 was associated with a 20 percent increased risk of incident asthma and a 14 percent increase in incident wheeze among adult females (Young et al. 2014); the second study was a study of Medicaid-enrolled children in Harris County, Texas, and found PM2.5 was associated with new-onset asthma in single-pollutant models (Wendt et al. 2014). However, accounting for the potential effects of other pollutants added substantial uncertainty in the overall effect estimates for PM2.5, meaning that it is difficult to distinguish in this study whether the effects are due to PM2.5 or other pollutant exposures.

The U.S. EPA 2009 ISA also noted that studies from many different locations, including Mexico City, Sweden, and a national cohort in the U.S. provide additional coherent and consistent evidence of respiratory effects associated with PM exposures.

Long-Term Particulate Matter Exposures and Emerging Areas of Interest

Beyond cardiovascular, respiratory and mortality effects, the U.S. EPA 2009 ISA review concluded that the evidence available at the time was suggestive of a causal relationship between long-term exposures to PM and reproductive/developmental effects, as well as cancer. Since the 2009 ISA, there have been several studies conducted that evaluated these health endpoints in relation to PM exposures, as well as studies of metabolic syndrome and neurological health outcomes. Because of the relatively long time gap since the latest ISA for PM, and because the SCAB exceeds the federal standards for PM2.5, these health endpoints are discussed briefly here, with a focus on studies conducted since the 2009 ISA, and studies conducted in California or in the SCAB.

Cancer

The U.S. EPA 2009 ISA review concluded that existing evidence is suggestive of a link between PM2.5 and cancer, with studies of lung cancer providing the strongest evidence. More recently, the International Agency for Research on Cancer (IARC) recently designated outdoor air pollution and particulate matter as carcinogenic to humans (Group 1 carcinogens), and a meta-analysis provided quantitative evidence for the associations between particulate matter and lung cancer risk (Hamra et al. 2014; International Agency for Research on Cancer 2015). The IARC review included studies evaluating associations between outdoor air pollution and lung cancer, urinary bladder cancer, breast cancer, leukemia and lymphoma, childhood cancers, and total cancers. Among these cancers, the IARC Working Group concluded that outdoor air pollution and particulate matter cause lung cancer, and that positive associations were observed between outdoor air pollution and urinary bladder cancer. The IARC Working Group also noted that associations with childhood leukemia were suggestive of an association, and, while there were some inconsistencies across studies, an association could not be ruled out. To estimate overall lung cancer risk, the meta-analysis included 14 studies reporting on PM2.5 and 9 studies reporting on PM10; the vast majority of these were cohort studies from North America and Europe. The meta-analysis found positive associations for both PM10 and PM2.5 and lung cancer risk, with the PM2.5 results being more consistent. Additionally, the study analyzed whether the association between PM2.5 and lung cancer differed by smoking status, and found positive associations for each smoking status group (current smokers, former smokers, and never-smokers).

A recent study from the Adventist Health and Smog Study-2 (AHSMOG-2) cohort in the U.S. and Canada reported that a 10 ug/m^3 increase in ambient PM2.5 increased the risk of lung cancer incidence by about 40 percent, after accounting for ozone exposures (Gharibvand et al. 2016). Because all participants are non-smokers, with over 80 percent never having smoked, and with the former smokers having an average of 24 years between quitting smoking and being diagnosed with lung cancer, the likelihood of confounding by smoking in this cohort is much lower than in most other

populations. Another recent study conducted in California evaluated air pollution in relation to survival after being diagnosed with lung cancer, and found that patients living in areas with higher NO_2, PM2.5 and PM10 had shorter survival times, particularly for those patients who were diagnosed at earlier stages of lung cancer (Eckel et al. 2016). Few other studies have evaluated air pollution effects on lung cancer survival, so this study represents a relatively newer area of research.

Reproductive Health Outcomes

The U.S. EPA 2009 ISA review concluded that existing evidence is suggestive of a link between PM2.5 and reproductive health effects. Numerous studies report evidence indicating that particulate matter exposure during pregnancy may be associated with adverse birth outcomes, with relatively consistent evidence linking PM2.5 and PM10 exposures to low birth weight or decreases in birth weight (Bobak et al. 1999; Sram et al. 2005; Stieb et al. 2012). Among the studies reviewed in the 2009 U.S. EPA ISA for particulate matter or in the literature search for more recent and/or local studies, several studies of low birth weight (defined as <2,500g or approximately 5.5 pounds at birth) or reductions in birth weight were conducted in California or in the Southern California region (Basu et al. 2004; Parker et al. 2005; Salam et al. 2005; Wilhelm et al. 2005; Morello-Frosch et al. 2010; Wilhelm et al. 2012; Basu et al. 2014; Laurent et al. 2014). Two of these studies were conducted in Los Angeles County and were published since the last AQMP in 2012, and both examined low birth weight among full-term babies ("term low birth weight"). Laurent et al. reported that a 5.82 $\mu g/m^3$ increase in PM2.5 exposures during pregnancy was linked to a 2.5 percent increased risk of term low birth weight (Laurent et al. 2014). The second study evaluated PM2.5 exposures by source, and found increased odds of term low birth weight with increased exposure to PM2.5 from diesel sources, gasoline, geological sources, as well as elemental carbon (Wilhelm et al. 2012). Studies from the U.S., Brazil, Mexico, the Czech Republic, South Korea, Japan, and Taiwan have reported that neonatal and early postnatal exposure to particulate matter may lead to increased infant mortality (U.S. EPA 2009). Among these studies, one was conducted in Southern California, and found increased risks for deaths among infants between one and 12 months old associated with exposures to particulates and other pollutants; however, no effect was seen for neonatal mortality (defined as mortality in the first month after birth) (Ritz et al. 2006). Some newer research has also linked particulate matter exposures to risk of certain birth defects and stillbirth. A California-based study used monitoring station data and traffic density measures to evaluate potential associations with a variety of birth defects in the San Joaquin Valley (Padula et al. 2013a; Padula et al. 2013b; Padula et al. 2013c; Padula et al. 2015). One of these studies reported evidence suggesting that PM10 and PM2.5 may increase the risk of certain congenital heart defects (Padula et al. 2013b). For neural tube defects, increased risks were linked to higher exposures to carbon monoxide and nitrogen oxide (Padula et al. 2013a), but higher risks for spina bifida with PM10 exposures were found only among mothers living in lower socioeconomic status neighborhoods (Padula et al. 2015). An earlier study conducted in Los Angeles County used ambient monitoring data to estimate exposures, and reported increased risk of certain congenital heart defects with higher exposures to carbon monoxide, but not for PM10; PM2.5 was not evaluated in this study (Ritz et al. 2002). A couple of recent studies evaluated PM2.5 exposures during gestation and risk of stillbirth. A recent study conducted in Ohio used monitoring station data

to evaluate stillbirth risk, and found that higher levels of PM2.5 exposure in the third trimester was linked to a 42 percent increased risk of stillbirth (DeFranco et al. 2015). A California-based study similarly found an increased risk of stillbirth with higher PM2.5 exposures averaged over the entire pregnancy, but the association may have been confounded by co-occurring nitrogen dioxide exposures (Green et al. 2015). A third study, conducted in Taiwan, found that higher PM10 and sulfur dioxide exposures in the first trimester were associated with increased risk of stillbirth among babies who were born preterm; PM2.5 was not assessed in this study (Hwang et al. 2011).

In the U.S. EPA review, it was noted that stronger associations with birth weight reductions are observed with PM2.5 compared to PM10, and animal toxicological studies provide supportive evidence, although a specific mechanism is not known (U.S. EPA 2009). These results and many other studies provide evidence that fetuses and infants are subgroups affected by particulate matter exposures.

Neurological Health Outcomes

A 2012 review conducted by a panel of research scientists convened by the National Institute of Environmental Health Sciences identified several studies that reported links between outdoor air pollution and central nervous system effects, such as decreased cognitive function, Alzheimer's disease, Parkinson's disease, and impacts on behavioral testing and development in childhood (Block et al. 2012). Toxicological studies suggest that the damage may be caused through an oxidative stress pathway, and demonstrate that PM can be inhaled into the lungs and translocated to the brain, and that ultrafine particles to reach the brain through the olfactory nerve (Peters et al. 2006). Some more recent studies have evaluated neurological impacts of PM, ranging from studies of older adults to prenatal exposures. The Normative Aging Study evaluated older men in Boston, MA, and reported an association between black carbon (a marker of traffic exhaust) and cognitive function, as measured through cognitive tests (Power et al. 2011). A study conducted in the Los Angeles Basin used monitoring data to evaluate long-term exposures in a middle-aged and older adult population, and reported PM2.5 exposure was associated with decreased verbal learning (Gatto et al. 2014). A study of school children in Spain reported that children attending schools with higher levels of air pollution, as measured by elemental carbon (a marker of diesel exhaust), NO_2, and ultrafine particles, experienced smaller growth in several cognitive measures (Sunyer et al. 2015). Three recent studies reported that PM2.5 exposures during the prenatal period were associated with autism in childhood. One study was conducted in Los Angeles County, and reported that 7 percent increased odds of autism with a 4.68 $\mu g/m^3$ increase in PM2.5; the effect estimate increased to 15 percent when accounting for ozone in the statistical models (Becerra et al. 2013). A California-based study found that an 8.7 $\mu g/m^3$ increase in PM2.5 during the prenatal period or in the first year of life doubled the odds of autism (Volk et al. 2013). The third study was based on the Nurses' Health Study II cohort, and reported an increased risk of autism with prenatal PM2.5 exposures, but not with exposures before pregnancy or after delivery (Raz et al. 2015). These studies provide emerging evidence of health effects of air pollution on neurological health outcomes.

Metabolic Syndrome

Metabolic syndrome, which is the clustering of several known risk factors for cardiovascular disease (Huang 2009), is a relatively new health outcome to be studied in relation to air pollution exposure. The U.S. EPA 2009 ISA reviewed only one epidemiological study and one toxicological study. These studies provided some evidence that particulate matter exposures may be linked to markers of metabolic syndrome, such as insulin resistance, hypertension, high cholesterol, or obesity, or that having a metabolic syndrome may increase susceptibility to the effects of PM10 exposures on cardiovascular outcomes (U.S. EPA 2009). More recently, a Swiss epidemiological study reported that long-term PM10 exposures were associated with increased risk of metabolic syndrome (Eze et al. 2015). Two other human studies found that people with metabolic syndrome exposed to particulate matter air pollution experienced cardiovascular effects and worsening insulin resistance (Devlin et al. 2014; Brook et al. 2016). Some recent animal studies have also reported impacts of PM on the development of obesity and metabolic syndrome, and that animals with pre-existing metabolic syndrome may be more sensitive to the cardiovascular effects of PM exposure (Brocato et al. 2014; Wagner et al. 2014; Wei et al. 2016).

Ultrafine Particles

As noted above, numerous studies have found associations between particulate matter levels and adverse health effects, including mortality, hospital admissions, and respiratory disease symptoms. The vast majority of these studies used particle mass of PM10, PM2.5, or PM10-2.5 as the measure of exposure. Some researchers have postulated, however, that ultrafine particles may be responsible for some of the observed associations of particulate matter and health outcomes (Oberdorster et al. 1995; Seaton et al. 1995). Ultrafine particles are typically defined as particles with aerodynamic diameters of less than 0.1 µm or 100 nm. Ultrafine particles are formed as a result of combustion processes as well as secondary atmospheric transformations. Vehicle emissions, especially diesel exhaust, are major sources of ultrafine particles; therefore, proximity to a major roadway is an important factor that affects an individual's exposure to ultrafine particles (Zhu et al. 2002; HEI Review Panel on Ultrafine Particles 2013). There is currently no federal or California standard for ultrafine particles.

U.S. EPA staff has presented conclusions on causal determination of several health effects of ultrafine PM based on a recent review of the available scientific studies (U.S. EPA 2009). These causal determinations are depicted in Table I-9.

TABLE I-9

Summary of U.S. EPA's Causal Determination of Ultrafine PM by Exposure Duration and Health Outcome

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Suggestive of a causal relationship
Respiratory effects	Suggestive of a causal relationship
Central nervous system	Inadequate to infer a causal relationship
Mortality	Inadequate to infer a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular effects	Inadequate to infer a causal relationship
Respiratory effects	Inadequate to infer a causal relationship
Mortality	Inadequate to infer a causal relationship
Reproductive and developmental	Inadequate to infer a causal relationship
Cancer, Mutagenicity, Genotoxicity	Inadequate to infer a causal relationship

(From (U.S. EPA 2009) Table 2-4 and Chapters 6 and 7)

In 2013, a review of the health effects of ultrafine particles concluded that current available evidence does not support that exposures to ultrafine particles alone account for the adverse health effects that have been associated with other ambient pollutants such as PM2.5, although the report noted several limitations in the exposure data relating to ultrafine particles (HEI Review Panel on Ultrafine Particles 2013). However, a more recent assessment of the studies published since that time suggest that UFP's may be more harmful compared to health compared to PM10 and PM2.5 (Li et al. 2016). Several potential mechanisms have been brought forward to suggest that the ultrafine portion may be important in determining the toxicity of ambient particulates, some of which are discussed below.

Smaller particles can also be inhaled deeper into the lungs, although the relationship between deposition fraction and particle size is complex. The ultrafine particles between 20-30 nm generally have higher fractional deposition in the alveolar region of the lung, where air exchange takes place. Because ultrafine particles are cleared from the lung more slowly compared to larger particles, the ultrafine particles can accumulate in the lung tissue where they can also translocate into the blood and to other organs (HEI Review Panel on Ultrafine Particles 2013). Ultrafine particles can also enter the brain tissues through the olfactory nerve (Peters et al. 2006). For a given mass concentration, ultrafine particles have much higher numbers of particles and surface area compared to larger particles. Particles can act as carriers for other adsorbed agents, such as trace metals and organic

compounds; and the larger surface area may transport more of such toxic agents than larger particles. Combined with the slower clearance of UFP's from the alveolar region of the lung, these small particles can deliver a greater amount of toxics to this part of the lung, causing increased inflammation (Li et al. 2016).

Exposures of laboratory animals to ultrafine particles have found cardiovascular and respiratory effects. Using an animal model of atherosclerotic disease, mice exposed to concentrated ultrafine particles (defined as less than 0.18 µm) near a roadway in Southern California showed larger early atherosclerotic lesions than mice exposed to concentrated PM2.5 or to filtered air (Araujo et al. 2008). In a mouse allergy model, exposures to concentrated ultrafine particles (less than 0.18 µm) resulted in a greater response to antigen challenge to ovalbumin (Li et al. 2010), indicating that vehicular traffic exposure could exacerbate allergic inflammation in already-sensitized animals. More specifically, ambient UFP's with a higher polycyclic aromatic hydrocarbon (PAH) content and higher oxidant potential triggered greater allergic inflammation in mice compared to a mixture of fine and ultrafine particles (Li et al. 2009). A related study identified specific proteins that are up-regulated among the exposed mice, which were proteins involved in allergic airway inflammation and immune system response (Kang et al. 2010). These results suggest that UFP's may play a role in the development or exacerbation of asthma, and point to an oxidative stress pathway. Additionally, some experiments using engineered nanoparticles found that the particle exposure led to a suppressed immune response to infections (Li et al. 2016).

Controlled exposures of human volunteers to ultrafine particles either laboratory-generated or as products of combustion, such as diesel exhaust containing particles, have found physiological changes related to vascular effects. Mills et al., for example found exposure to diesel exhaust particulate at 300 $\mu g/m^3$ attenuated both acetylcholine and sodium-nitroprusside-induced vasorelaxation (Mills et al. 2011). These exposures were higher than typical ambient concentrations, although the authors state that such concentrations can be found regularly in heavy traffic, occupational settings, and in some of the most polluted cities in the world. This study showed that diesel exhaust particulates had impacts on vascular function while carbon nanoparticles did not change vascular function, providing evidence that is complementary to the epidemiological studies linking particulate matter exposure to cardiovascular outcomes. Several other human exposures studies have reported effects of UFP's on inflammatory markers, lung function, heart rate and heart rate variability, including effects on people with asthma, diabetes, or metabolic syndrome (Li et al. 2016).

There is a lack of long-term studies of human population exposure to ultrafine particles, as there is currently no ultrafine monitoring network in the U.S. As noted above, however, a recent study from California estimated exposures to PM2.5 and ultrafine particles among members of the California Teachers Study cohort. Positive, statistically significant associations of ischemic heart disease mortality were observed with modeled PM2.5 and with ultrafine particle mass concentrations derived from chemical transport models using California emissions inventories (Ostro et al. 2015). Other epidemiological studies have reported links between UFP exposures both indoors and

outdoors with decreased microvascular function and increased systemic inflammation in adults (Karottki et al. 2014; Olsen et al. 2014), and with oxidative DNA damage in children (Song et al. 2013).

There have been several cross-sectional epidemiological studies of ultrafine particles, mainly from Europe. Some of these studies found effects on hospital admissions and emergency department visits for respiratory and cardiovascular effects, whereas other studies did not find such effects (U.S. EPA 2009). A recent study conducted in Rochester, NY reported that ambient UFP exposures in the prior week were associated with increased risk of asthma-related medical visits indicative of asthma exacerbation; the study did not find associations with accumulation mode PM, PM2.5, black carbon, or sulfur dioxide (Evans et al. 2014). Concentrations of ultrafine particles can vary geographically, and it is not clear how well the central-site monitors used in these studies reflect actual exposures.

Additional discussion on the sources and health effects of ultrafine particles can be found in Chapter 9 of the 2012 AQMP.

Sensitive Populations for PM-Related Health Effects

Certain populations may be more sensitive to the health effects of particulate air pollution, and evidence to assess susceptibility comes from epidemiological, controlled human exposure, and toxicological studies of PM2.5 and PM10 exposures. The U.S. EPA 2009 ISA for PM concluded that there is evidence supporting increased susceptibility to the effects of PM among children (for respiratory effects) and older adults (for cardiovascular effects), individuals with pre-existing cardiovascular or respiratory conditions, individuals with lower socioeconomic status (sometimes assessed using proxy measures such as educational attainment or residential location), and individuals with certain genetic polymorphisms that control antioxidant response, regulate enzyme activity, or regulate procoagulants (U.S. EPA 2009). In addition, there is some limited evidence that additional factors may increase a person's susceptibility to PM health effects, including chronic inflammatory conditions (e.g. diabetes, obesity) and life stage, with pregnant women and fetuses *in utero* being potentially more susceptible. Table I-10 summarizes the U.S. EPA's 2009 ISA assessment of susceptibility factors for particulate matter.

TABLE I-10

Summary of Evidence for Potential Increased Susceptibility to PM-Related Health Effects

Assessment of Evidence	Potential At Risk Factor
Increased susceptibility to PM	Older Adults (≥65 years) Children (<18 years) Genetic factors Cardiovascular diseases Respiratory illnesses Socioeconomic status (SES) Educational attainment (surrogate of SES) Residential location (surrogate of SES)
Increased susceptibility to PM, but limited studies available	Pregnancy and developmental effects Diabetes Obesity Health status, e.g. nutrition (surrogate of SES)
Did not increase susceptibility to PM	Gender Race/ethnicity
Did not increase susceptibility to PM, but limited studies available	Respiratory contributions to cardiovascular effects

Adapted From (U.S. EPA 2009) Table 8-2

Summary - Particulate Matter Health Effects

A considerable body of scientific evidence from epidemiologic, controlled human exposure and toxicological studies support the causal determinations for particulate matter and several categories of health endpoints, with the strongest evidence supporting a causal relationship for PM2.5 exposures with cardiovascular effects and mortality. Specific cardiovascular effects include cardiovascular deaths, hospital admissions for ischemic heart disease and congestive heart failure, changes in heart rate variability and markers of oxidative stress, and markers of atherosclerosis. The scientific evidence also supported a likely causal relationship for PM2.5 exposure with respiratory effects, such as hospital admissions for COPD or respiratory infections, asthma development, asthma or allergy exacerbation, lung cancer, impacts on lung function, lung inflammation, oxidative stress, and airway hyperresponsiveness. Both short-term and long-term particulate matter exposures are linked to health effects in humans. Young children, older adults, and people with pre-existing respiratory or cardiovascular health conditions are among those who may be more susceptible to the adverse effects of PM.

Estimates of the Health Burden of Particulate Matter in the South Coast Air Basin

In terms of estimating health burdens of air pollution exposure, CARB has conducted analyses in the past estimating exposures and quantitative health effects from exposures to particulate matter as well as other pollutants. A recent assessment focused on premature mortality and PM2.5, and

estimated the deaths associated with exposures above 5.8 µg/m^3, which is an estimate of background PM2.5 (California Air Resources Board 2010a). The analysis used the U.S. EPA's risk assessment methodology for calculating premature mortality and used ambient air quality measurements averaged over a three-year period of 2006-2008. An update to this analysis using ambient air quality data from 2009-2011 indicated that PM2.5-related premature deaths in California due to cardiopulmonary causes as 7,200 deaths per year with an uncertainty range of 5,600 – 8,700. Estimates were also made for the California Air Basins. For the South Coast Air Basin, the estimate was 4,000 cardiopulmonary deaths per year with an uncertainty range of 3,200–4,900. These estimates were calculated using the associations of cardiopulmonary mortality and PM2.5 from the second exposure period from Krewski (Krewski et al. 2009).

Another analysis of health impacts in the South Coast was conducted as part of the Socioeconomic Report for the 2012 AQMP. The analysis estimated the anticipated costs and benefits of adopting the measures in the Final 2012 AQMP, which included the projected public health benefits associated with lower PM2.5 concentrations as a result of the 2012 plan (South Coast Air Quality Management District 2012). Based on that analysis, the projected annual number of averted deaths due to PM2.5 reductions from the 2012 AQMP was 668 deaths in year 2014, and 275 deaths in year 2023. In addition, estimated numbers of health conditions prevented per year due to the 2012 AQMP were shown for several other health endpoints, including respiratory and cardiovascular outcomes. The estimates of cases averted in year 2014 were 597 cases of acute bronchitis, 29 to 261 non-fatal heart attacks, 18,384 person-days for lower and upper respiratory symptoms, 153 respiratory emergency room visits, 151 hospital admissions, 287,447 person-days of minor restricted activity, 48,805 work loss days, and 26,910 person-days of asthma attacks. Importantly, these estimates of prevented mortality and morbidity should not be compared to the estimates of deaths attributable to PM2.5 conducted by CARB, because these analyses are intended to answer different questions. The SCAQMD estimates address the question of "how many cases are averted due to the adoption of the 2012 AQMP?" while the CARB estimates address the question of "how many deaths are attributable to PM2.5 exposures above 5.8 µg/m3?". Both analyses provide important information regarding the health impacts of PM2.5.

NITROGEN DIOXIDE

Nitrogen dioxide (NO_2) is a gaseous air pollutant that serves as an indicator of gaseous oxides of nitrogen, such as nitric oxide (NO) and other related compounds (NO_x). These gases can undergo photochemical reactions to form ground-level ozone, and are important contributors to ozone pollution levels in the SCAB. Evidence of the health effects of NO_2 is derived from human and animal studies, which link NO_2 with respiratory effects such as decreased lung function and increases in airway responsiveness and pulmonary inflammation (U.S. EPA 2016). The U.S. EPA in 2010 retained the existing standards of 53 ppb for NO_2 averaged over one year, and adopted a new short-term standard of 100 ppb (0.1 ppm) averaged over one hour. The standard was designed to protect against increases in airway reactivity in individuals with asthma based on controlled exposure studies, as well as respiratory symptoms observed in epidemiological studies. The revised standard also requires additional monitoring for NO_2 near roadways.

In the current U.S. EPA Integrated Science Assessment for Nitrogen Oxides (U.S. EPA 2016), the staff conclusion for causal relationships between exposures and health effects are shown in the following table.

TABLE I-11

Summary of U.S. EPA's Causal Determination for Health Effects of Nitrogen Dioxide

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory effects	Causal relationship
Cardiovascular and related metabolic effects	Suggestive of a causal relationship
Total mortality	Suggestive of a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory effects	Likely to be a causal relationship
Cardiovascular and related metabolic effects	Suggestive of a causal relationship
Reproductive and developmental effects	Fertility, Reproduction, and Pregnancy: Inadequate to infer a causal relationship Birth Outcomes: Suggestive of a causal relationship Postnatal Development: Inadequate to infer a causal relationship
Total Mortality	Suggestive of a causal relationship
Cancer	Suggestive of a causal relationship

(From (U.S. EPA 2016), Table ES-1)

Since the previous U.S. EPA Integrated Science Assessment (ISA) for Nitrogen Oxides from 2008, the causal determination for short-term and long-term respiratory effects have been updated in the 2016 ISA to reflect the stronger evidence now available pointing to a causal or likely causal relationship. For non-respiratory outcomes, the U.S. EPA also updated their assessment of the weight of evidence to show that the evidence for several short- and long-term outcomes is suggestive, but not sufficient to infer a causal relationship. Evidence for low-level nitrogen dioxide (NO_2) exposure effects is derived from laboratory studies of asthmatics and from epidemiological studies. Additional evidence is derived from animal studies. In the 2016 ISA, the U.S. EPA cited the coherence of the results from a variety of studies, and a plausible biological mechanism (whereby NO_2 reacts with the respiratory lining and forms secondary oxidation products that increase airway responsiveness and allergic

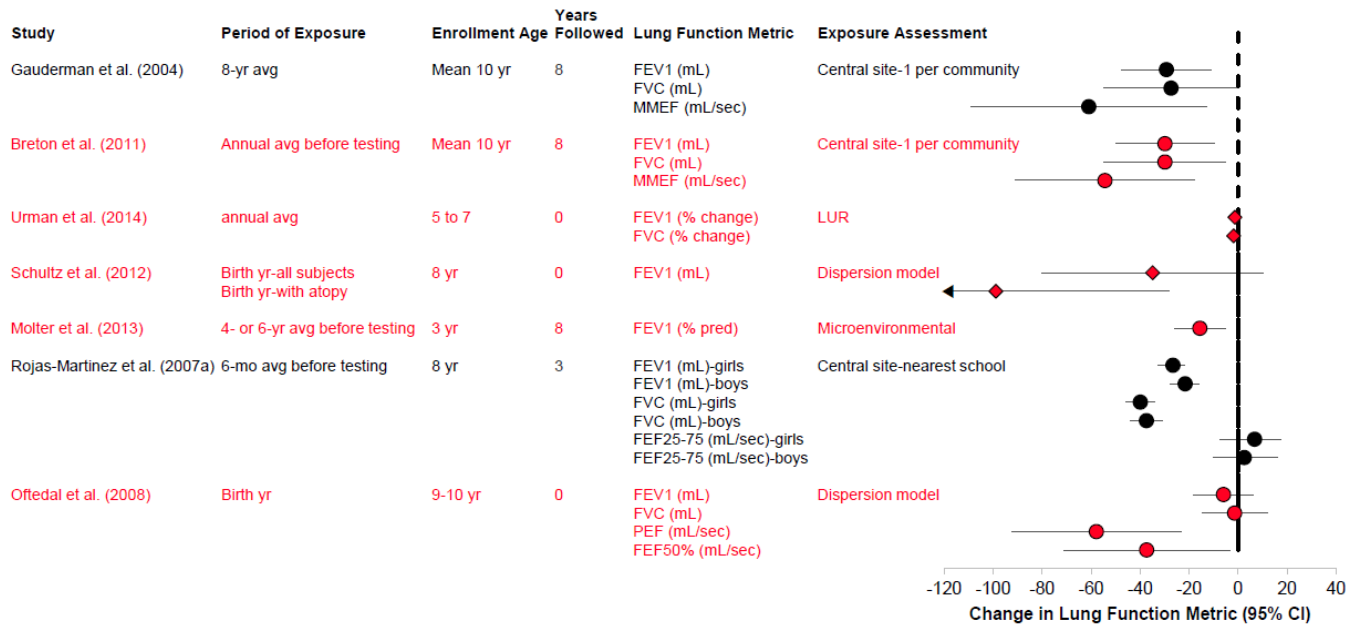
inflammation) to support the determination of a causal relationship between short-term NO_2 exposures and asthma exacerbations ("asthma attacks"). The long-term link with respiratory outcomes was strengthened by recent experimental and epidemiological studies, and the strongest evidence available is from studies of asthma development.

Several studies related to outdoor exposure have found health effects associated with ambient NO_2 levels, including respiratory symptoms, respiratory illness, decreased lung function, pulmonary inflammation, increased emergency room visits for asthma, and cardiopulmonary mortality. However, since traffic exhaust is an important source of NO_2 and several other pollutants, such as particulate matter, exposure generally occurs in the presence of other pollutants, making it more difficult for these studies to distinguish the specific role of NO_2 in causing effects independent of other pollutants. However, studies linking NO_2 to asthma exacerbations and human experimental studies provided support for the U.S. EPA determination that this causal relationship exists for short-term NO_2 exposures independent of other traffic-related pollutants (U.S. EPA 2016). The report also concludes that epidemiological studies do not rule out the possible influence of other traffic-related pollutants on the observed health effects.

The Children's Health Study in Southern California has evaluated a variety of health endpoints in relation to air pollution exposures, including lung function, lung development, school absences, and asthma. The study found associations between long-term exposure to air pollution, including NO_2, PM10, and PM2.5, and respiratory symptoms in asthmatic children (McConnell et al. 1999). Particles and NO_2 levels were correlated, and independent effects of individual pollutants could not be discerned. A subsequent analysis using more refined exposure estimation methods indicated consistent associations between long-term NO_2 exposures and respiratory symptoms in children with asthma (McConnell et al. 2003).

Ambient levels of NO_2 were also associated with a decrease in lung function growth in a group of children followed for eight years, including children with no history of asthma. In addition to NO_2, the decreased growth was also associated with particulate matter and airborne acids. The study authors postulated this may be a result of a package of pollutants from traffic sources (Gauderman et al. 2004).

A number of studies have since reported deficits in lung function associated with nitrogen oxides exposures. Examples are shown in Figure I-8.



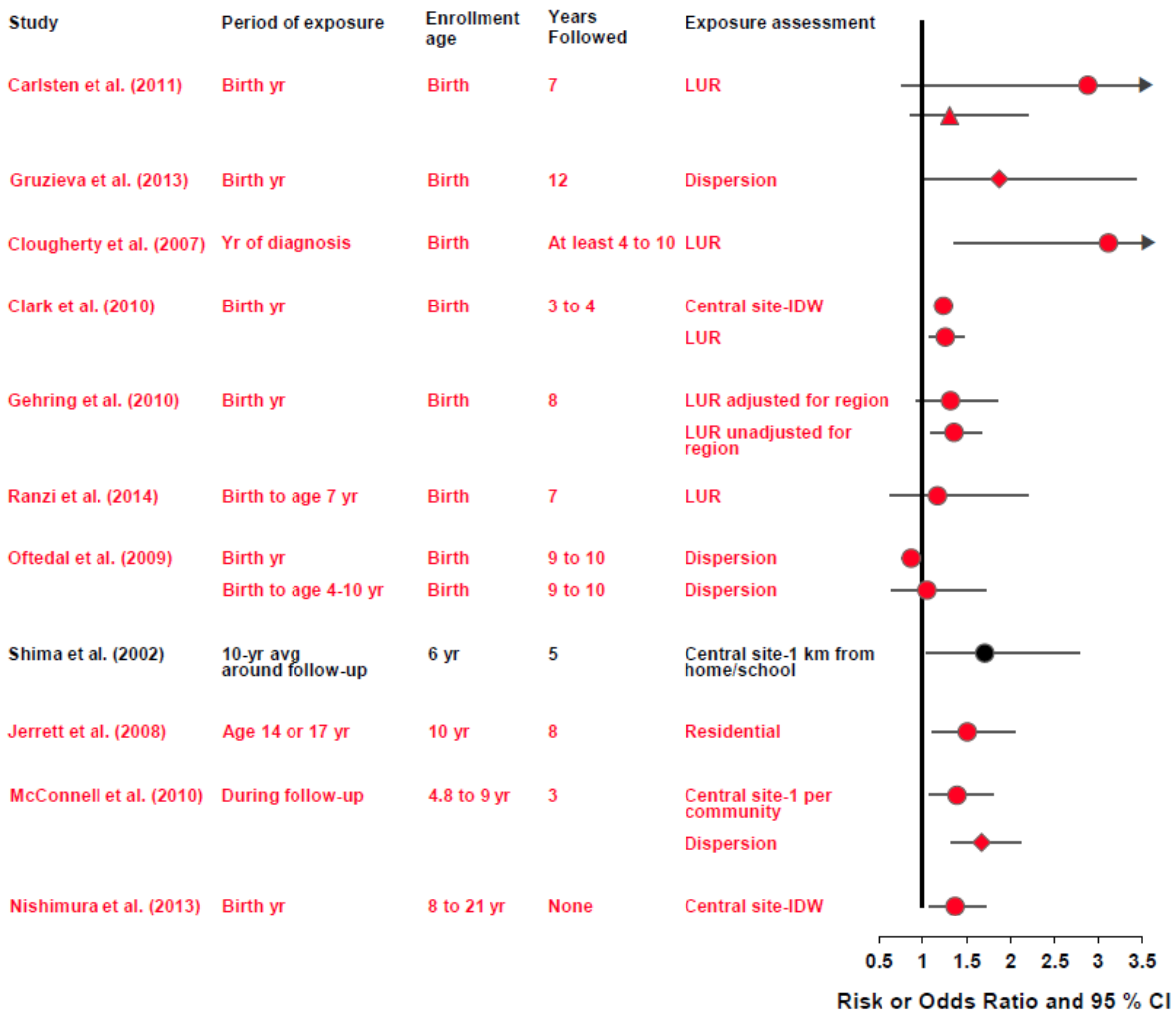
Note: Studies in red are recent studies. Studies in black were included in the 2008 ISA for Oxides of Nitrogen. Circles = NO_2; Diamonds = NO_x. All mean changes in this plot are standardized to a 10-ppb increase in NO_2 and a 20-ppb increase in NO_x concentration. Effect estimates from studies measuring NO_x in $\mu g/m_3$ (Schultz et al., 2012) have not been standardized.

FIGURE I-8

Associations of nitrogen dioxide (NO_2) or the sum of nitric oxide and NO_2 (NO_x) with lung function indices from prospective studies of children (From (U.S. EPA 2016), Figure 6-5).

A follow-up report from the Children's Health Study has assessed whether improving air quality in Southern California over the past several decades has led to beneficial changes in health among children (Gauderman et al. 2015). It was reported that as the levels of nitrogen oxide and fine particulates came down as the result of air pollution emissions reductions, the deficits in lung function growth were also of a smaller magnitude. Such improvements were observed in children with asthma as well as in those without asthma. These results indicate that improvements in air quality are associated with improvements in children's health.

In recent years, the most compelling evidence of long-term effects of NO_2 has been from prospective cohort studies that link NO_2 exposures to the development of asthma, primarily in children. The U.S. EPA included several recent studies in their review, as shown in the Figure I-9. The vast majority of these studies found that higher NO_2 exposures were linked to an increased risk or odds of developing asthma among children.



Effect estimates are standardized to a 10-ppb increase in NO_2, with the exception of Gruzieva et al. (2013) who examined NOx in µg/m3 and Oftedal et al (2009) who did not report increments for the effect estimates for the birth to age 4 years or birth to age 10 years exposure periods. Note: Black symbols = studies evaluated in the 2008 Integrated Science Assessment for Oxides of Nitrogen; Red symbols = recent studies. Circles=NO_2; triangles=NO; diamonds=NOx.

FIGURE I-9

Associations of ambient nitrogen dioxide (NO_2) concentrations with asthma incidence in longitudinal cohort studies of children (From (U.S. EPA 2016), Figure 6-1).

Among the studies of childhood asthma incidence reviewed in the 2016 U.S. EPA ISA for Oxides of Nitrogen, two studies were conducted in Southern California. Both studies were based on the Children's Health Study cohort, but one study used a smaller subset of the cohort and estimated NO_2 exposures using monitors at the children's homes (Jerrett et al. 2008). The second study examined over 2000 children and used data from air monitoring stations as well as modeled NO_2 levels to estimate exposures (McConnell et al. 2010). Both studies found a positive association between NO_2 exposures and the onset of asthma in these children, however, because NO_2 is often strongly

correlated with PM2.5 and other components of traffic-related air pollution, it is possible that the effects observed are due to some other component of traffic exhaust for which NO_2 serves as a proxy measure. The consistency of the effects found linking NO_2 exposure and asthma development in children, the use of prospective longitudinal study designs following children for several years, and the use of several different methods to estimate exposures are noted strengths of such studies. Experimental studies have found that NO_2 exposures increase responsiveness of airways, pulmonary inflammation, and oxidative stress, and can lead to the development of allergic responses. These biological responses provide evidence of a plausible mechanism for NO_2 to cause asthma.

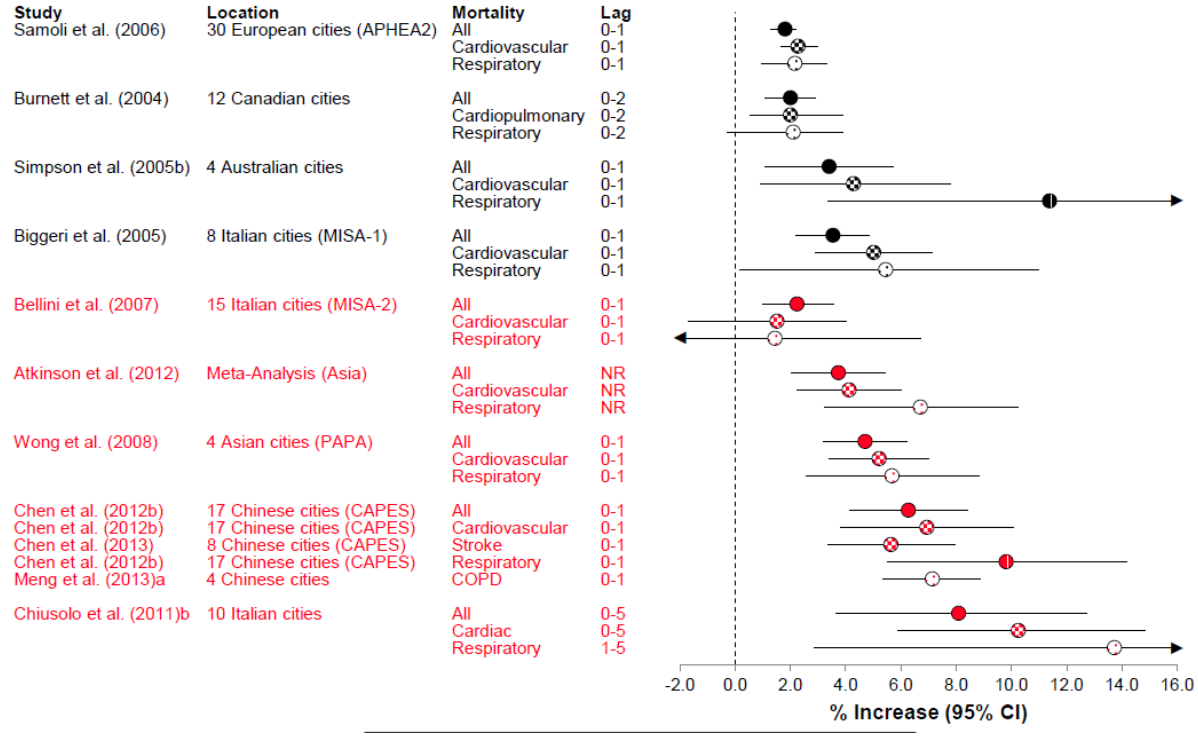
Results from controlled exposure studies of asthmatics demonstrate an increase in the tendency of airways to contract in response to a chemical stimulus (airway responsiveness) or after inhaled allergens (U.S. EPA 2016). Effects were observed among adult volunteers with asthma when exposed to 100 ppb NO_2 for 60 minutes and to 200-300 ppb for 30 minutes, with approximately 70 percent of study participants experiencing an increase in airway responsiveness. A similar response was reported in some studies with healthy subjects at higher levels of exposure (1.5 - 2.0 ppm), although these changes in healthy adults are likely of little or no clinical significance. Increased airway responsiveness among people with asthma can lead to worse symptoms and reduced lung function. Mixed results have been reported from controlled human exposure studies of people with chronic obstructive lung disease, with some studies reporting no change in symptom score while other studies reporting increased symptom scores when participants were exposed to NO_2 while exercising (U.S. EPA 2016).

Short-term controlled studies of rats exposed to NO_2 over a period of several hours indicate cellular changes associated with allergic and inflammatory responses that can lead to liver damage and reduced hepatic function. Rodent models exposed to NO_2 repeatedly for 4 to 14 days demonstrated increased airway responsiveness with high levels of exposure (4000 ppb). Animal studies also provide evidence that NO_2 exposures have negative effects on the immune system, and therefore increase the host's susceptibility to respiratory infections. Epidemiological studies showing associations between NO_2 levels and hospital admissions for respiratory infections also support such a link (U.S. EPA 2016).

Several epidemiological studies conducted in California have examined associations between NO_2 exposures and other health effects, including some recent studies evaluating cardiovascular effects (Coogan et al. 2012; Bartell et al. 2013; Wittkopp et al. 2013), mortality (Lipsett et al. 2011; Bartell et al. 2013; Jerrett et al. 2013), birth outcomes (Ghosh et al. 2012; Laurent et al. 2014; Padula et al. 2014; Ritz et al. 2014; Green et al. 2015), and cancer (Ghosh et al. 2013). Many studies conducted in other geographic areas have also found links with these health outcomes, and the latest assessment by U.S. EPA is that the existing studies are suggestive of a causal relationship for some of these endpoints or inadequate to infer a causal relationship for other endpoints (U.S. EPA 2016). In addition, some of the newer outcomes evaluated in relation to NO_2 exposures include neurological outcomes such as Parkinson's disease (Ritz et al. 2016), Alzheimer's disease (Oudin et al. 2016), and autism (Becerra et al. 2013; Volk et al. 2013), as well as metabolic diseases such as diabetes and obesity (Coogan et al. 2012; Robledo et al. 2015; White et al. 2016). However, many of these studies

use NO_2 exposures as a proxy measure for traffic-related air pollutants, and do not aim to identify a specific pollutant within the mix of pollutants from this source. Thus, there is uncertainty on whether NO_2 exposure has independent relationships with non-respiratory related health effects, or whether NO_2 is simply a marker of near-road air pollution exposure, which includes a mixture of air pollutants, including some air toxics.

Examples of studies reporting an association of mortality with short-term NO_2 exposures are shown in the figure below.



Note: Black symbols = multicity studies evaluated in the 2008 Integrated Science Assessment for Oxides of Nitrogen; Red symbols = recent studies. Filled circle = total mortality; Crosshatch = cardiovascular mortality; Vertical lines = respiratory mortality.

FIGURE I-10

Percentage increase in total, cardiovascular, and respiratory mortality from multi-city studies for a 20-ppb increase in 24-hour average or 30-ppb increase in one-hour maximum nitrogen dioxide concentrations (From (U.S. EPA 2016), Figure 5-23).

SULFUR DIOXIDE

Sulfur dioxide (SO_2) is a gaseous air pollutant that has been linked to a variety of respiratory effects, such as decreased lung function and increased airway resistance. Controlled laboratory studies involving human volunteers have clearly identified asthmatics as a very sensitive group to the effects of ambient sulfur dioxide (SO_2) exposures. Healthy subjects have failed to demonstrate any short-term respiratory functional changes at exposure levels up to 1.0 ppm over 1-3 hours. In exercising asthmatics, brief exposure (5-10 minutes) to SO_2 at levels between 0.2-0.6 ppm can result in increases in airway resistance and decreases in breathing capacity. The response to SO_2 inhalation is

observable within two minutes of exposure, increases further with continuing exposure up to five minutes, then remains relatively steady as exposure continues. SO_2 exposure is generally not associated with any delayed reactions or repetitive asthmatic attacks (U.S. EPA 2008). In 2010, the U.S. EPA SO_2 air quality standard was set at 75 ppb (0.075 ppm) averaged over one hour to protect against acute asthma attacks in sensitive individuals.

The EPA assessment based on the 2008 Integrated Science Assessment for Sulfur Oxides is shown in the table below (U.S. EPA 2008). The U.S. EPA recently released a draft of the revised ISA for SO2 (U.S. EPA 2015a) which evaluates recent evidence assessing links to mortality and cardiovascular, respiratory, carcinogenic, and reproductive effects (Brunekreef et al. 2009; Hart et al. 2011; Pascal et al. 2013; Chen et al. 2014; Gianicolo et al. 2014; Milojevic et al. 2014; Moridi et al. 2014; Stingone et al. 2014; Straney et al. 2014; Wang et al. 2014; Winquist et al. 2014; Yang et al. 2014; Ancona et al. 2015; Green et al. 2015; Rich et al. 2015; Shah et al. 2015; Yorifuji et al. 2015).

TABLE I-12

Summary of U.S. EPA's Causal Determinations for Health Effects of Sulfur Oxides

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory morbidity	Causal relationship
Cardiovascular morbidity	Inadequate to infer a causal relationship
Mortality	Suggestive of a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Respiratory morbidity	Inadequate to infer a causal relationship
Carcinogenic effects	Inadequate to infer a causal relationship
Prenatal and neonatal outcomes	Inadequate to infer a causal relationship
Mortality	Inadequate to infer a causal relationship

(From (U.S. EPA 2008) Chapter 3)

In epidemiologic studies of children and adults, associations of short-term variations in SO_2 levels with increases in respiratory symptoms, emergency department visits, and hospital admissions for respiratory-related causes have been reported. There is uncertainty as to whether SO_2 is associated with the effects or whether other co-occurring pollutants may explain the observed effects, although some studies indicated that the SO_2 effects remained even after accounting for the effects of other pollutants, including PM2.5. Coupled with the human clinical studies, these data suggest that SO_2 can trigger asthmatic episodes in individuals with pre-existing asthma (U.S. EPA 2008).

Animal studies have shown SO_2 effects on pulmonary inflammation with acute exposure at concentrations consistent with ambient SO_2 levels. Toxicological studies using animals found that repeated exposures to concentrations of SO_2 as low as 0.1 ppm promoted allergic sensitization and airway inflammation. Such evidence, combined with human clinical studies and epidemiological studies in people with asthma support the U.S. EPA determination of a causal relationship between short-term SO_2 exposure and respiratory morbidity. One of these studies was conducted in the Los Angeles area, and found that higher ambient SO_2 levels were associated with increased odds of asthma symptoms among Hispanic children with asthma (Delfino et al. 2003).

Some epidemiological studies indicate that the cardiovascular mortality effects associated with short-term exposures to ambient SO_2 were generally reduced when accounting for other pollutants, although the evidence is still suggestive of a causal relationship. Few epidemiological studies are available to assess the potential confounding effects of other co-occurring pollutants in studies of long-term effects. For example, there is some evidence that sulfates, which are formed when SO_2 oxidizes rapidly in the atmosphere, may be associated with lung function changes, although the evidence is not consistent (Reiss et al. 2007). Sulfates are positively correlated with SO_2 levels, so it is difficult to distinguish the effect of one individual pollutant. Based on a level determined necessary to protect the most sensitive individuals, the California Air Resources Board (CARB) in 1976 adopted a standard of 25 $\mu g/m^3$ (24-hour average) for sulfates. There is no federal air quality standard for sulfates.

CARBON MONOXIDE

Carbon monoxide (CO) is a gaseous air pollutant that has a high affinity to bond with oxygen-carrying proteins (hemoglobin and myoglobin). The resulting reduction in oxygen supply in the bloodstream is responsible for the toxic effects of CO, which are typically manifested in the oxygen-sensitive organ systems. The effects have been studied in controlled laboratory environments involving exposure of humans and animals to CO, as well as in population-based studies of ambient CO exposure effects. People with deficient blood supply to the heart (ischemic heart disease) are known to be susceptible to the effects of CO. Protection of this group is the basis of the existing National Ambient Air Quality Standards for CO at 35 ppm for one hour and 9 ppm averaged over eight hours. The health effects of ambient CO have been recently reviewed by U.S. EPA, with the strongest evidence supporting a likely causal link between short-term CO exposures and cardiovascular outcomes, although studies have linked both short-term and long-term CO exposures to several other health outcomes (Table I-13) (U.S. EPA 2010).

TABLE I-13

Summary of U.S. EPA's Causal Determinations for Health Effects of Carbon Monoxide

SHORT-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular morbidity	Likely to be a causal relationship
Central nervous system	Suggestive of a causal relationship
Respiratory morbidity	Suggestive of a causal relationship
Mortality	Suggestive of a causal relationship
LONG-TERM EXPOSURES	
Health Outcome	**Causality Determination**
Cardiovascular morbidity	Inadequate to infer a causal relationship
Central nervous system	Suggestive of a causal relationship
Birth outcomes and developmental effects	Suggestive of a causal relationship
Respiratory morbidity	Inadequate to infer a causal relationship
Mortality	Not likely to be a causal relationship

(From (U.S. EPA 2010) Table 2-1)

Inhaled CO has no known direct toxic effect on lungs but rather exerts its effects by interfering with oxygen transport—through the formation of carboxyhemoglobin (COHb, a chemical complex of CO and hemoglobin)), which reduces the amount of oxygen the blood can carry to the tissues. Exposure to CO is often evaluated in terms of COHb levels in blood, measured as percentage of total hemoglobin bound to CO. Endogenous COHb is estimated to be <1 percent in healthy individuals, but COHb levels are sensitive to health status and metabolic state, with higher levels among smokers and persons with inflammatory diseases. Estimates based on a large prospective study of adults conducted in the 1970s showed a dose-response relationship between the average number of cigarettes smoked per day and the COHb concentrations (never smokers: 1.59±1.72 percent, former smokers: 1.96±1.87 percent, 1-5 cigarettes/day: 2.31±1.94 percent, 6–14 cigarettes/day: 4.39±2.48 percent, 15–24 cigarettes/day: 5.68±2.64 percent, >=25 cigarettes/day: 6.02±2.86 percent) (Hart et al. 2006).

Under controlled laboratory conditions, healthy subjects exposed to CO sufficient to result in 5 percent COHb levels exhibited reduced duration of maximal exercise performance due to the inability to deliver sufficient oxygen to the heart and other muscles. Studies involving subjects with coronary artery disease who engaged in exercise during CO exposures have shown that COHb levels as low as

2.4 percent can lead to earlier onset of electrocardiograph changes indicative of deficiency of oxygen supply to the heart. Other effects of inadequate oxygen delivery to the body tissues include earlier onset of chest pain, increase in the duration of chest pain, headache, confusion and drowsiness (U.S. EPA 2000).

A number of epidemiological studies have found associations between short-term ambient CO levels and increased hospital admissions and emergency department visits for ischemic heart disease, including myocardial infarction (U.S. EPA 2010). In studies reporting results stratified by age and sex, larger effects were generally observed among older adults and among males. Examples of such studies, including information on number of days of lag time between exposure and hospital admissions for key cardiovascular outcomes, are shown in the figure below.

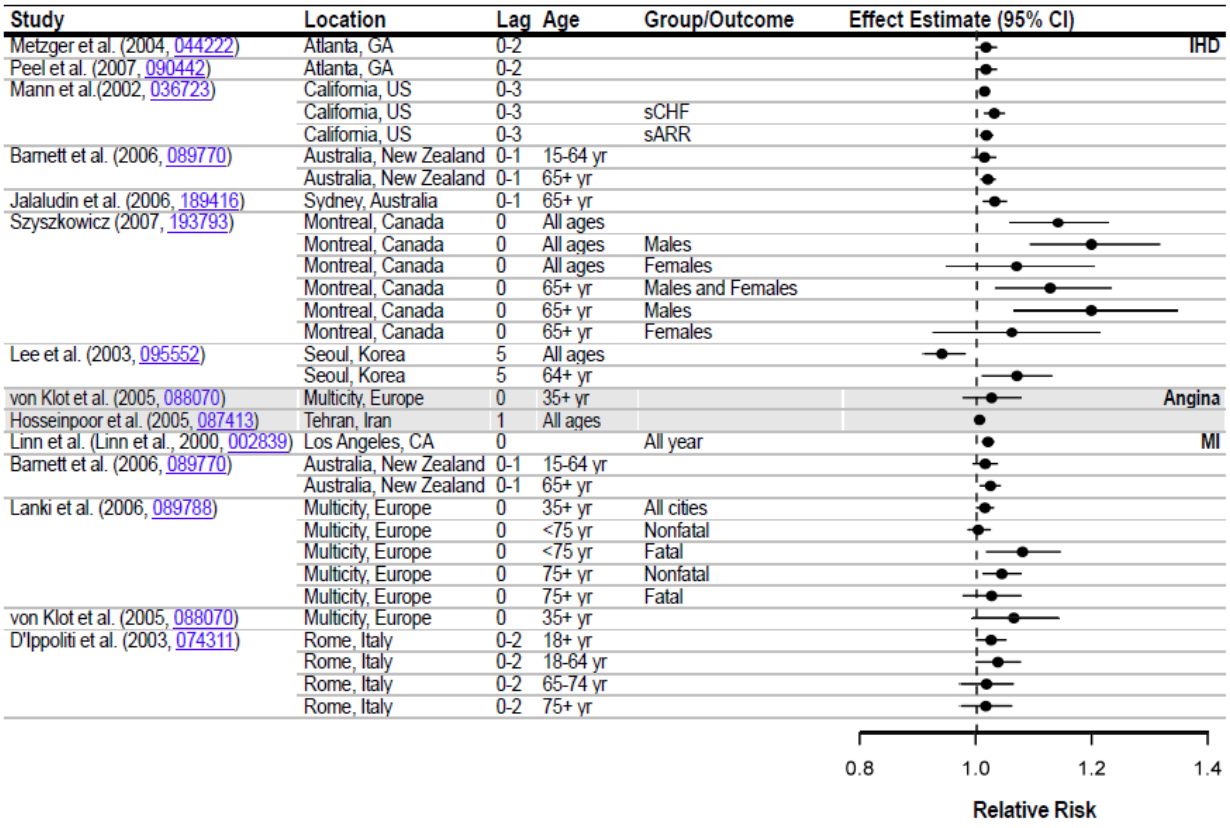


FIGURE I-11

Effect estimates (95 percent confidence intervals) associated with hospital admissions for various forms of heart disease. Effect estimates have been standardized to a 1 ppm increase in ambient CO for 1-h max CO concentrations, 0.75 ppm for 8-h max CO concentrations, and 0.5 ppm for 24-h average CO concentrations (From (U.S. EPA 2010), Figure 5-2). Lag time is the time between the exposure and the outcome measured. The closed circle on the diagram indicates the effect estimate, while the bar indicates the 95 percent confidence interval.

Research studies have also evaluated ambient CO exposures in relation to reproductive health outcomes. Epidemiological studies conducted in Southern California have reported an association

between with CO exposure during pregnancy and increases in pre-term births (Ritz et al. 2000; Wilhelm et al. 2005; Ritz et al. 2007). The increases in the pre-term births were also associated with PM10 or PM2.5 levels. There are very few studies examining CO exposure and birth defects, but one Southern California study found increased risks for cardiac-related birth defects with carbon monoxide exposure in the second month of pregnancy (Ritz et al. 2002). Toxicological studies in laboratory animals with higher than ambient levels of CO have also reported decrements in birth weight and prenatal growth, as well as impaired neurobehavior in the offspring of exposed animals (U.S. EPA 2010). The U.S. EPA concluded in their most recent review that the evidence linking long-term CO exposures with reproductive health outcomes was suggestive of a causal relationship.

LEAD

Lead (Pb) is a toxic air contaminant that is recognized to exert an array of deleterious effects on multiple organ systems. There are a number of potential public health effects at low level exposures, and there is no recognized lower threshold for health effects (U.S. EPA 2013a). The health implications are generally indexed by blood lead levels which are related to lead exposures both from inhalation as well as from ingestion. Effects include impacts on population IQ as well as heart disease and kidney disease. The initial air quality standard for lead was established by U.S. EPA in 1978 at a level of 1.5 µg /m^3 averaged over a calendar quarter. U.S. EPA revised the NAAQS for lead in 2008 to a level of 0.15 µg/m^3 averaged over a rolling three-month period to protect against lead toxicity. The SCAB's attainment status for lead is described in the draft 2016 AQMP Chapter 2.

The U.S. EPA has recently reviewed the health effects of ambient lead exposures in conjunction with an Integrated Science Assessment and a review of the NAAQS for lead (U.S. EPA 2013a; U.S. EPA 2015c). Lead can accumulate and be stored in the bone, and this lead in bone can be released into the blood when the bone is metabolized, which happens naturally and continuously. Blood lead is the most common measure of lead exposure, and it represents recent exposure and may be an indicator of total body burden of lead (U.S. EPA 2013a). The following table gives the summary of causality conclusions from the U.S. EPA review, which illustrates the wide range of health effects associated with lead exposure.

TABLE I-14

Summary of U.S. EPA's Causal Determinations for Health Effects of Lead

HEALTH OUTCOME	CAUSALITY DETERMINATION
Children - Nervous System Effects	
Cognitive Function Decrements	Causal relationship
Externalizing Behaviors: Attention, Impulsivity and Hyperactivity	Causal relationship
Externalizing Behaviors: Conduct Disorders in Children and Young Adults	Likely to be a causal relationship
Internalizing Behaviors	Likely to be a causal relationship
Auditory Function Decrements	Likely to be a causal relationship
Visual Function Decrements	Inadequate to infer a causal relationship
Motor Function Deficits	Likely to be a causal relationship
Adults – Nervous System Effects	
Cognitive Function Decrements	Likely to be a causal relationship
Psychopathological Effects	Likely to be a causal relationship
Cardiovascular effects	
Hypertension	Causal relationship
Subclinical Atherosclerosis	Suggestive of a causal relationship
Coronary Heart Disease	Causal relationship
Cerebrovascular Disease	Inadequate to infer a causal relationship
Renal Effects	
Reduced Kidney Function	Suggestive of a causal relationship
Immune System Effects	
Atopic and Inflammatory Response	Likely to be a causal relationship
Decreased Host Resistance	Likely to be a causal relationship
Autoimmunity	Inadequate to infer a causal relationship
Hemotologic Effects	
Decreased Red Blood Cell Survival and Function	Causal relationship
Altered Heme Synthesis	Causal relationship
Reproductive and Developmental Effects	
Development	Causal relationship
Birth Outcomes (low birth weight, spontaneous abortion)	Suggestive of a causal relationship
Male Reproductive Function	Causal relationship
Female Reproductive Function	Suggestive of a causal relationship
Cancer	
Cancer	Likely to be a causal relationship

(From (U.S. EPA 2013a) Table ES-1)

Children appear to be sensitive to the neurological toxicity of lead, with effects observed at blood lead concentration ranges of 2–8 µg/dL. No clear threshold has been established for such effects. According to the U.S. EPA review, the most important effects observed are neurotoxic effects in children and cardiovascular effects in adults. The effects in children include impacts on intellectual

attainment and school performance. Figure I-12 provides a summary of the lowest levels of blood lead that have been associated with certain neurological, hematological and immune effects in children.

Lowest Observed Effect Blood Lead Level	Neurological Effects	Hematological Effects	Immune Effects
30 µg/dL		Increased urinary δ-aminolevulinic acid	
15 µg/dL	Behavioral disturbances (e.g., inattention, delinquency) Altered electrophysiological responses	Erythrocyte protoporphyrin (EP) elevation	
10 µg/dL	Effects on neuromotor function CNS cognitive effects (e.g., IQ deficits)	Inhibition of δ-aminolevulinic acid dehydratase (ALAD) Pyrimidine-5′-nuclotidase (Py5N) activity inhibition	Effects on humoral (↑ serum IgE) and cell-mediated (↓ T-cell abundance) immunity
5 µg/dL	(???)	(???)	
0 µg/dL			

Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-17 of U.S. Environmental Protection Agency (1986a).

FIGURE I-12

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children (From (U.S. EPA 2007), Table 3-1)

Figures I-12 and I-13, taken from the U.S. EPA review (U.S. EPA 2007), depict the health effects of lead in relation to blood levels. In the figure, the question marks indicate that there are no demonstrated threshold blood lead levels for health effects. The Centers for Disease Control (CDC) has recently revised their lead hazard information and replaced their level of concern for adverse effects of 10 µg/dL blood lead level with a childhood blood lead level reference value of 5 µg/dL to identify children and environments associated with lead-exposure hazards (Centers for Disease Control and Prevention 2016).

Figure I-13 provides a summary of the lowest levels of blood lead that have been associated with key health effects in adults. For adults, evidence supports a causal relationship between lead and increased blood pressure and hypertension, as well as coronary heart disease (myocardial infarction, ischemic heart disease, and heart rate variability). Other health effects among adults are also relatively high on the causal scale, including neurological, hematological, and renal effects.

Lowest Observed Effect Blood Lead Level	Neurological Effects	Hematological Effects	Cardiovascular Effects	Renal Effects
30 µg/dL	Peripheral sensory nerve impairment	Erythrocyte protoporphyrin (EP) elevation in males		Impaired Renal Tubular Function
20 µg/dL	Cognitive impairment			
15 µg/dL	Postural sway	Erythrocyte protoporphyrin (EP) elevation in females		
		Increased urinary δ-aminolevulinic acid		
10 µg/dL		Inhibition of δ-aminolevulinic acid dehydratase (ALAD)	Elevated blood pressure ↓	
5 µg/dL			(???)	Elevated serum creatine (↓ creatine clearance)
0 µg/dL				

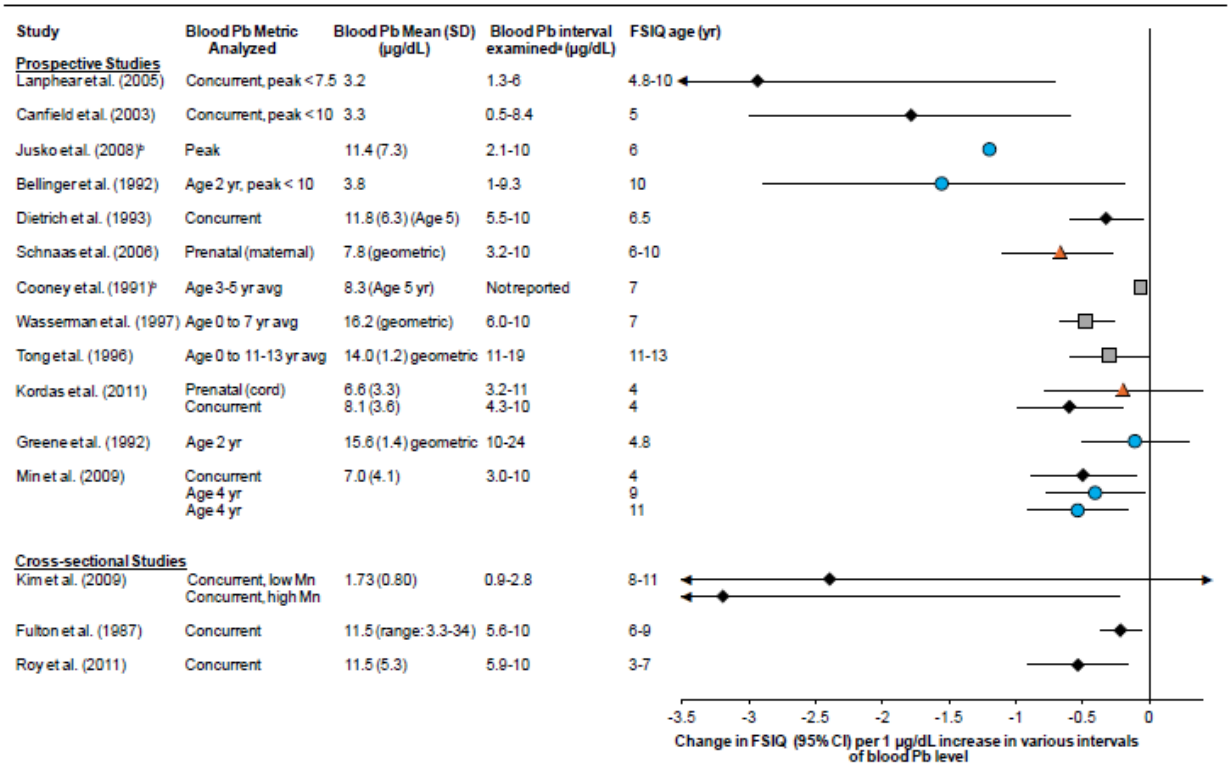
Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-16 of U.S. Environmental Protection Agency (1986a).

FIGURE I-13

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults (From (U.S. EPA 2007), Table 3-2)

In its most recent review of lead health effects, the U.S. EPA confirmed its previous conclusion regarding the cognitive decline in children as the most sensitive adverse effect associated with lead exposures. The effects as measured by a reduction in IQ from a number of studies are shown in the following figure. According to the review, the currently available evidence supports a median estimate of -1.75 IQ points for a change of 1 µg/dL blood lead to describe the neurocognitive impacts on young children (U.S. EPA 2015c).



[a]See Table 4-3 for explanation of the blood Pb level interval examined. Effect estimates were calculated for the lowest range examined in the study or the 10th percentile of blood Pb level to a blood Pb level of 10 µg/dL.
[b]Sufficient data were not available to calculate 95% CI.

Note: Mn = manganese. Results are presented for most of the cohorts examined in the literature and generally are grouped according to strength of study design, representativeness of the study population characteristics and blood Pb levels examined, and extent of consideration for potential confounding. There is not necessarily a continuum of decreasing strength across studies. Results usually are presented for the oldest age examined in cohorts. Multiple results from a cohort are grouped together. To facilitate comparisons among effect estimates across studies with different distributions of blood Pb levels and model structures (e.g., linear, log-linear), effect estimates are standardized to a 1 µg/dL increase for the lowest range of blood Pb levels examined in the study or the interval from the 10th percentile of blood Pb level to 10 µg/dL. For populations with 10th percentiles near or above 10 µg/dL, the effect estimate was calculated for the 10th to 90th percentile of blood Pb level. The percentiles are estimated using various methods and are only approximate values. Effect estimates are assumed to be linear within the blood Pb level interval evaluated. The various tests used to measure FSIQ are scored on a similar scale (approximately 40-160 FSIQ points). Black diamonds, blue circles, orange triangles, and gray squares represent effect estimates for concurrent, earlier childhood, prenatal, and lifetime average blood Pb levels, respectively. The horizontal lines associated with point estimates represent 95% confidence intervals (CI).

FIGURE I-14

Associations of Blood Pb Levels with Full-Scale IQ (FSIQ) in Children (From (U.S. EPA 2013a), Figure 4-2)

TOXIC AIR CONTAMINANTS

Toxic air contaminants are pollutants for which there generally are no ambient air quality standards. The Toxic Air Contaminant Identification and Control Act (AB 1807, Tanner, 1983) created California's first program to reduce exposures to air toxics by requiring CARB to adopt Air Toxics Control measures. Air Districts must either enforce these measures or adopt their own equally or more stringent measures. The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, Connelly,

1987) supplements the earlier program by requiring air toxics inventories for certain facilities, notification of people's exposure to significant health risks, and facility plans to reduce these risks. Under California's Air Toxics Program, the Office of Environmental Health Hazard Assessment (OEHHA) assesses the health effects of substances that may pose a risk of adverse health effects, and CARB assesses the potential for humans to be exposed to these substances. These effects are usually an increased risk for cancer, adverse birth outcomes, or respiratory effects. After review by the state Scientific Review Panel, CARB holds a public hearing on whether to formally list substances that may pose a significant risk to public health as a Toxic Air Contaminant. Chapter 9 of the draft 2016 AQMP describes the Air Toxics Control Plan for the SCAQMD.

Air toxics include many different types of chemicals, and the discussion here will not address all air toxics in a comprehensive manner. However, this section will discuss very briefly diesel particulate matter and volatile organic compounds (VOC's), because diesel particulate matter is the most significant contributor to cancer risk in the South Coast Air Basin, and because some VOC's are air toxics, and are part of the control measures proposed in the current Air Quality Management Plan.

Diesel Particulate Matter

The California Air Resources Board listed diesel particulate matter as a Toxic Air Contaminant in 1998, based on the determination that it was a human carcinogen (California Air Resources Board 2010b). The International Agency for Research on Cancer, an arm of the World Health Organization, classified diesel exhaust as probably carcinogenic to humans in 1989 (International Agency for Research on Cancer 1989). More recently, IARC convened an international panel of scientists to review the published literature since the initial classification regarding the carcinogenicity of diesel combustion emissions. The panel concluded that diesel exhaust is a substance that causes lung cancer in humans (International Agency for Research on Cancer 2012b).

OEHHA also establishes potency factors for air toxics that are carcinogenic. The potency factors can be used to estimate the additional cancer risk from ambient levels of toxics. This estimate represents the chance of contracting cancer in an individual over a lifetime exposure to a given level of an air toxic and is usually expressed in terms of additional cancer cases per million people exposed.

SCAQMD conducted studies on the ambient concentrations and estimated the potential health risks from air toxics (South Coast Air Quality Management District 2000; South Coast Air Quality Management District 2008; South Coast Air Quality Management District 2015). In the latest SCAQMD Multiple Air Toxics Exposure Study, MATES IV, a one-year monitoring program was undertaken at 10 sites throughout the SCAB over the time period July 2012 – June 2013 (South Coast Air Quality Management District 2015). Over 30 substances were measured, which included the toxics that contributed the most to health risks in the Basin. The results showed that the overall lifetime risk for excess cancer from a 70-year lifetime exposure to the levels of air toxics calculated from the regional model was 367 in a million. This reflects a greater than 50 percent reduction in exposures and risks compared to the MATES III Study that was conducted from 2004 -2006. The largest contributor to this risk was diesel particulate matter, accounting for 68 percent of the air

toxics risk. The average measured levels were also compared to the non-cancer chronic Reference Exposure Levels (RELs), and found to be below the established RELs for the over 30 substances measured.

In 2015, OEHHA updated the calculation procedure to estimate cancer risks from air toxics exposures (Dodge et al. 2015). The revisions to the calculation methodology included accounting for higher risks attributable to early life exposures (up to age 16 years), updates to the population distribution of breathing rates by age, and a reduction in the time of household residence. In combination, these changes resulted in risk estimates in the MATES IV study to be about 2.5 times higher than the previous methodology employed in the MATES studies. The average lifetime risk for excess cancer cases is estimated to be 897 per million using the updated procedure (South Coast Air Quality Management District 2015). However, it is important to note that results from the MATES IV study still represent approximately a 50 percent reduction in air toxics levels and cancer risk compared to MATES III. In addition to the maps in the MATES IV final report (South Coast Air Quality Management District 2015), an interactive map of the MATES IV cancer risks from air toxics calculated using the 2015 OEHHA guidelines is available through this website: http://www.aqmd.gov/home/tools/public.

In 2009, the Advanced Collaborative Emissions Study (ACES) reported that newer diesel engine technologies are very effective in reducing the amount of emissions from diesel trucks, as required by recent regulations (Khalek et al. 2009). In a long-term exposure study published in 2015, rats breathing the lower emissions did not develop cancer, while the rats breathing the higher emissions from older diesel engines (in previous studies) did develop cancer (McDonald et al. 2015). However, the 2015 study did not evaluate whether the PM from the newer engines was any more or less toxic compared to the older engines on a gram per gram basis; the study was not designed to determine such differences. Therefore, without any additional data on the toxicity of PM from the newer diesel engines, the analysis done in the MATES IV study used the same risk factor for both, applied to the mass of PM. For example, whether a person is exposed to 10 ug/m^3 of particulate matter from a single old diesel engine or several new diesel engines, the cancer risk would be the same because it is calculated based on 10 ug/m^3 of exposure.

In the Particulate Matter section of this Appendix, the vast majority of the studies described evaluated the health effects of total PM2.5 exposures by mass, regardless of whether they were from newer diesel engines, older diesel engines, or other sources. While this new diesel technology is very effective in terms of reducing the amount of emissions from diesel trucks, what people are being exposed to is a total concentration of PM from many sources. Health studies generally use this total concentration to analyze whether or not there is an effect on the specific health outcomes evaluated. In addition, it is important to note that direct PM2.5 emissions from diesel engines represent a small portion of overall PM2.5 exposure. NO_x emissions from diesel engines that eventually lead to PM2.5 formation in the atmosphere, however, represent a larger component of PM2.5 exposure (South Coast Air Quality Management District 2013a; Harley 2014).

Volatile Organic Compounds

VOC's are a class of air pollutants that undergo photochemical reactions in the air to form ozone. It should be noted that there are no state or national ambient air quality standards for VOCs because they are not classified as criteria pollutants. VOCs are regulated, however, because limiting VOC emissions reduces the rate of photochemical reactions that contribute to the formation of ozone.

VOCs are also transformed into organic aerosols in the atmosphere, contributing to higher PM and lower visibility levels. In addition, VOC's that have toxic properties are also regulated as air toxics. Chapter 3 of the draft 2016 AQMP presents data on VOC sources and emissions in the South Coast Air Basin.

Some examples of VOC's that are known to cause health effects include benzene, toluene, ethylbenzene and xylenes (abbreviated BTEX), 1,3-butadiene, formaldehyde, and perchloroethylene. Several of these VOC's are carcinogenic. Based on the MATES IV analysis, benzene, 1,3-butadiene, and carbonyls (formaldehyde and acetaldehyde) together account for approximately 21 percent of the total cancer risk from air toxics in the SCAB. Not all carcinogenic VOC's are known to cause the same types of cancers, although several are associated with blood cancers. For example, the cancers most closely associated with long-term benzene exposure are leukemias. Formaldehyde is linked to nasopharyngeal cancer and leukemias, while 1,3-butadiene causes cancers in both the blood and lymphatic systems (International Agency for Research on Cancer 2012a).

Many VOC's can also cause non-cancer health effects. For these types of health outcomes, OEHHA has developed acute and chronic Reference Exposure Levels (RELs). RELs are concentrations in the air below which adverse health effects are not likely to occur. Acute RELs refer to short-term exposures, generally of one-hour duration. Chronic RELs refer to long-term exposures of several years. OEHHA has also established eight-hour RELs for several substances. The ratio of ambient concentration to the appropriate REL can be used to calculate a Hazard Index. A Hazard Index of less than one would not be expected to result in adverse effects (Dodge et al. 2015).

In the MATES IV assessment of chronic non-cancer health risks, the monitored air toxics levels were found to be below the chronic RELs. In other words, the general levels of air toxics in the SCAB are not expected to cause adverse non-cancer health effects. Importantly, the MATES IV monitoring network was designed to characterize the air toxics exposures in the basin overall. Given that ambient monitoring is necessarily conducted at a limited number of locations, and modeling is limited to a spatial resolution of 2km, there may be higher exposures not captured by the fixed-site monitoring. To address this limitation, particularly in some communities with environmental justice concerns, the MATES IV study also included local-scale studies in 3 communities very close to known industrial sources or large mobile source facilities, with a focus on ultrafine particles and diesel PM emissions. Details of these study results can be found in the MATES IV final report (South Coast Air Quality Management District 2015).

ODORS

Environmental odors are recognized as having the potential to cause health effects and/or quality of life impacts. The theory of "miasma" dates back to Hippocrates in ancient Greek times, and related bad odors to disease. The health effects of environmental odors can vary widely, and depend on the compound causing the odor, the level of the compound, as well as the sensitivity and physiological responses of the person detecting the odor.

Different levels of odor exposure can cause a range of responses and health effects, and the science of odor as a potential health issue was summarized previously by Schiffman and Williams (Schiffman et al. 2005b). There are two key nerves in the nasal cavity involved in odor effects: the olfactory nerve provides the sense of smell, while the trigeminal nerve provides the sense of irritation. At very low levels, an odor can be detected (i.e. odor threshold), and at slightly higher levels, an odor can be recognized and identified. At levels higher than detection or recognition levels, an odor can cause annoyance or intolerance, and at even higher levels, an odor can cause irritation or possible toxicity, if the odor is caused by a compound that is also an air toxic (Schiffman et al. 2005b).

Schiffman and Williams proposed three mechanisms of action for odor symptoms (Schiffman et al. 2005b). In the first mechanism, an odor substance can be at the level that can produce irritation, which triggers the trigeminal nerve. This mechanism is considered a toxic effect because symptoms appear when the chemical concentration is at or above the irritation level; here, the odor serves only as the marker of the toxic effect. In the second mechanism, the odor compound is below the irritation level but above odor detection thresholds, which can result in odor annoyance. This mechanism is relatively common among environmental odors, and has been studied in communities exposed to odors from landfills, hazardous waste sites or concentrated animal feeding operations (CAFO's) (Shusterman et al. 1991; Schiffman et al. 2005a; Heaney et al. 2011; Schinasi et al. 2011; Blanes-Vidal et al. 2012; Hooiveld et al. 2015). In this mechanism, the health effect is not a toxicological effect, and the dose does not necessarily correlate well with the effect in these instances. Genetic factors, previous exposure ("learning"), and beliefs about the safety of the odor may play important roles in these odors causing health symptoms (Shusterman 2001). The third proposed mechanism is when an odor substance is present along with a co-pollutant or endotoxin that is capable of producing health effects. In this mechanism, the effect is also a toxic effect, but the odor serves as a marker of the presence of a mixture that includes a toxic compound; if the co-pollutant were not present, no health effect would be expected in this scenario.

Individual characteristics can play important roles in altering an individual's response to an odor. Factors that can influence odor perception include age, genetics, gender, medical history (including mental health, neurological conditions, and other health conditions), health-related behaviors (tobacco, alcohol), and occupational and environmental factors (Greenberg et al. 2013; Wilson et al. 2014; Agency for Toxic Substances and Disease Registry 2016). Additionally, an individual's cognitive associations with the odor prior to an exposure can result in increased reporting of health-related symptoms after exposure (Shusterman et al. 1991; Shusterman 2001; Greenberg et al. 2013). Common symptoms associated with environmental odor exposures include headache, nasal

congestion, eye, nose and throat irritation, hoarseness or sore throat, cough, chest tightness, shortness of breath, wheezing heart palpitations, nausea, drowsiness, and mental depression (Agency for Toxic Substances and Disease Registry 2016). If the concentrations of the odor compound are below irritation levels, then the symptoms are not expected to persist once the person is no longer exposed; however, being exposed to odor levels at or above irritation levels for longer periods of time may cause symptoms that persist after moving out of the exposure area (Agency for Toxic Substances and Disease Registry 2016).

CONCLUSIONS

A large body of scientific evidence shows that the adverse impacts of air pollution on human and animal health are clear. A considerable number of population-based and laboratory studies have established a link between air pollution and increased morbidity and, in some instances, premature mortality. Importantly, the health effects of air pollution extend beyond respiratory effects, and there is substantial evidence that air pollution (including particulate matter and ozone) exposures cause cardiovascular morbidity and mortality. Some air pollutants, such as diesel PM, lead, and several other air toxics, have been linked to increased cancer risk. Health studies have also identified populations who may be more susceptible to the adverse effects of air pollution, such as children, older adults, low SES communities, people with certain pre-existing health conditions, and people with certain genetic factors. Understanding the impacts of air pollution on these more susceptible populations can help inform policies that better protect public health, for example, in setting standards for criteria air pollutants, and in the development of methods to evaluate air toxics health risks. Continued research on the effects of specific PM constituents and ultrafine particles will be important in furthering the understanding of how these pollutants affect human health.

As the scientific methods for the study of air pollution health effects have progressed over the past decades, adverse effects have been shown to occur at lower levels of exposure. For some pollutants, no clear thresholds for effects have been demonstrated. The new findings have, in turn, led to the revision and lowering of National Ambient Air Quality Standards (NAAQS) which, in the judgment of the Administrator of the U.S. EPA, are necessary to protect public health. Chapter 8 of the draft 2016 AQMP provides an overview of the extensive, multi-year, public process involved in setting federal air quality standards. Assessments of the scientific evidence from health studies is an important part of the process, and has helped inform revisions to the federal air pollution standards. Figures I-15 and I-16 are meant to convey some of the historical context to recent revisions to the NAAQS for ozone and for particulate matter, with regard to key developments in the understanding of the health effects of these pollutants.

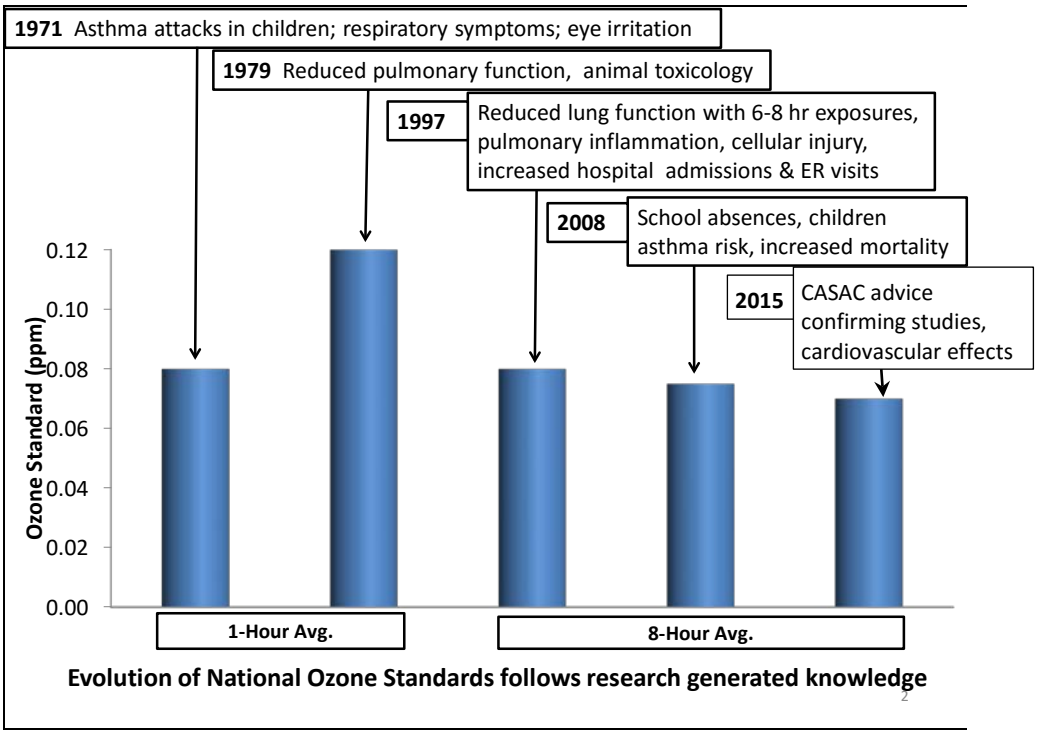


FIGURE I-15

Historical Context to Revisions of NAAQS for Ozone

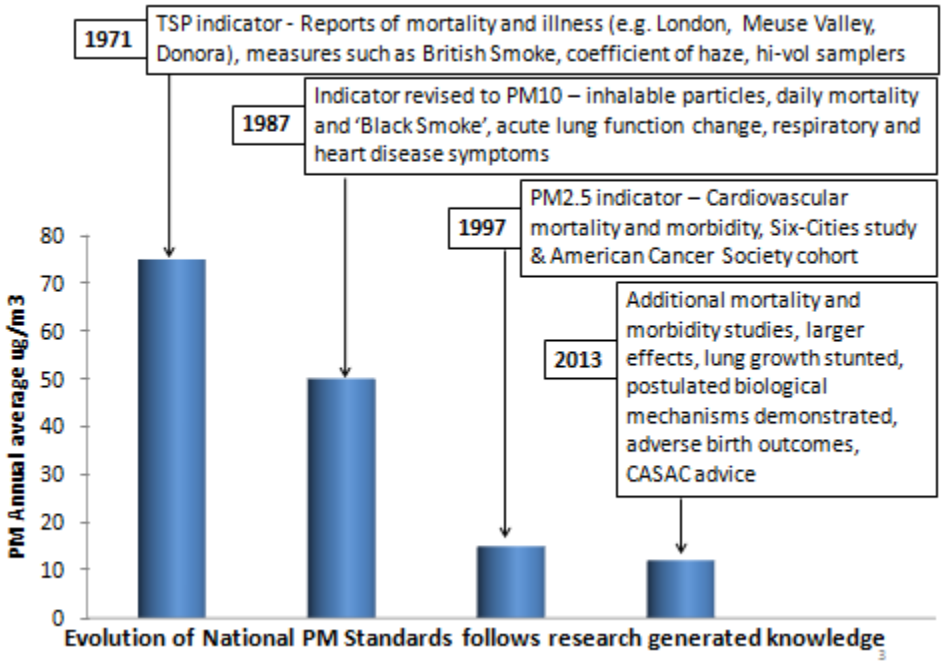


FIGURE I- 16

Historical Context to Revisions of NAAQS for PM

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Attachment 2

Sierra Club v. County of Fresno Amicus Briefs

CASE NO. S219783

IN THE SUPREME COURT OF CALIFORNIA

SIERRA CLUB, REVIVE THE SAN JOAQUIN, and
LEAGUE OF WOMEN VOTERS OF FRESNO,
Plaintiffs and Appellants

v.

COUNTY OF FRESNO,
Defendant and Respondent

FRIANT RANCH, L.P.,
Real Party in Interest and Respondent

After a Decision by the Court of Appeal, filed May 27, 2014
Fifth Appellate District Case No. F066798

Appeal from the Superior Court of California, County of Fresno
Case No. 11CECG00726

APPLICATION FOR LEAVE TO FILE AMICUS CURIAE BRIEF OF SAN JOAQUIN VALLEY UNIFIED AIR POLLUTION CONTROL DISTRICT IN SUPPORT OF DEFENDANT AND RESPONDENT, COUNTY OF FRESNO AND REAL PARTY IN INTEREST AND RESPONDENT, FRIANT RANCH, L.P.

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APPLICATION

Pursuant to California Rules of Court 8.520(f)(1), proposed Amicus Curiae San Joaquin Valley Unified Air Pollution Control District hereby requests permission from the Chief Justice to file an amicus brief in support of Defendant and Respondent, County of Fresno, and Defendant and Real Parties in Interest Friant Ranch, L.P. Pursuant to Rule 8.520(f)(5) of the California Rules of Court, the proposed amicus curiae brief is combined with this Application. The brief addresses the following issue certified by this Court for review:

> Is an EIR adequate when it identifies the health impacts of air pollution and quantifies a project's expected emissions, or does CEQA further require the EIR to *correlate* a project's air quality emissions to specific health impacts?

As of the date of this filing, the deadline for the final reply brief on the merits was March 5, 2015. Accordingly, under Rule 8.520(f)(2), this application and brief are timely.

1. Background and Interest of San Joaquin Valley Unified Air Pollution Control District

The San Joaquin Valley Unified Air Pollution Control District ("Air District") regulates air quality in the eight counties comprising the San Joaquin Valley ("Central Valley"): Kern, Tulare, Madera, Fresno, Merced, San Joaquin, Stanislaus, and Kings, and is primarily responsible for attaining air quality standards within its jurisdiction. After billions of dollars of investment by Central Valley businesses, pioneering air quality regulations, and consistent efforts by residents, the Central Valley air basin has made historic improvements in air quality.

The Central Valley's geographical, topographical and meteorological features create exceptionally challenging air quality

conditions. For example, it receives air pollution transported from the San Francisco Bay Area and northern Central Valley communities, and the southern portion of the Central Valley includes three mountain ranges (Sierra, Tehachapi, and Coastal) that, under some meteorological conditions, effectively trap air pollution. Central Valley air pollution is only a fraction of what the Bay Area and Los Angeles produce, but these natural conditions result in air quality conditions that are only marginally better than Los Angeles, even though about ten times more pollution is emitted in the Los Angeles region. Bay Area air quality is much better than the Central Valley's, even though the Bay Area produces about six times more pollution. The Central Valley also receives air pollution transported from the Bay Area and northern counties in the Central Valley, including Sacramento, and transboundary anthropogenic ozone from as far away as China.

Notwithstanding these challenges, the Central Valley has reduced emissions at the same or better rate than other areas in California and has achieved unparalleled milestones in protecting public health and the environment:

- In the last decade, the Central Valley became the first air basin classified by the federal government under the Clean Air Act as a "serious nonattainment" area to come into attainment of health-based National Ambient Air Quality Standard ("NAAQS") for coarse particulate matter (PM10), an achievement made even more notable given the Valley's extensive agricultural sector. Unhealthy levels of particulate matter can cause and exacerbate a range of chronic and acute illnesses.
- In 2013, the Central Valley became the first air basin in the country to improve from a federal designation of "extreme" nonattainment to

actually attain (and quality for an attainment designation) of the 1-hour ozone NAAQS; ozone creates "smog" and, like PM10, causes adverse health impacts.

- The Central Valley also is in full attainment of federal standards for lead, nitrogen dioxide, sulfur dioxide, and carbon monoxide.
- The Central Valley continues to make progress toward compliance with its last two attainment standards, with the number of exceedences for the 8-hour ozone NAAQS reduced by 74% (for the 1997 standard) and 38% (for the 2008 standard) since 1991, and for the small particulate matter (PM2.5) NAAQS reduced by 85% (for the 1997 standard) and 61% (for the 2006 standard).

Sustained improvement in Central Valley air quality requires a rigorous and comprehensive regulatory framework that includes prohibitions (e.g., on wood-burning fireplaces in new residences), mandates (e.g., requiring the installation of best available pollution reduction technologies on new and modified equipment and industrial operations), innovations (e.g., fees assessed against residential development to fund pollution reduction actions to "offset" vehicular emissions associated with new residences), incentive programs (e.g., funding replacements of older, more polluting heavy duty trucks and school buses)[1], ongoing planning for continued air quality improvements, and enforcement of Air District permits and regulations.

The Air District is also an expert air quality agency for the eight counties and cities in the San Joaquin Valley. In that capacity, the Air District has developed air quality emission guidelines for use by the Central

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[1] San Joaquin's incentive program has been so successful that through 2012, it has awarded over $ 432 million in incentive funds and has achieved 93,349 tons of lifetime emissions reductions. *See* SAN JOAQUIN VALLEY AIR POLLUTION CONTROL DISTRICT, 2012 PM2.5 PLAN, 6-6 (2012) *available at* http://www.valleyair.org/Workshops/postings/2012/12-20-12PM25/FinalVersion/06%20Chapter%206%20Incentives.pdf.

Valley counties and cities that implement the California Environment Quality Act (CEQA).[2] In its guidance, the Air District has distinguished between toxic air contaminants and criteria air pollutants.[3] Recognizing this distinction, the Air District's CEQA Guidance has adopted distinct thresholds of significance for *criteria* pollutants (i.e., ozone, PM2.5 and their respective precursor pollutants) based upon scientific and factual data which demonstrates the level that can be accommodated on a cumulative basis in the San Joaquin Valley without affecting the attainment of the applicable NAAQS.[4] For *toxic air* pollutants, the District has adopted different thresholds of significance which scientific and factual data demonstrates has the potential to expose sensitive receptors (i.e., children, the elderly) to levels which may result in localized health impacts.[5]

The Air District's CEQA Guidance was followed by the County of Fresno in its environment review of the Friant Ranch project, for which the Air District also served as a commenting agency. The Court of Appeal's holding, however, requiring correlation between the project's criteria

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[2] *See, e.g.*, SAN JOAQUIN VALLEY AIR POLLUTION CONTROL DISTRICT, PLANNING DIVISION, GUIDE FOR ASSESSING AND MITIGATING AIR QUALITY IMPACTS (2015), *available at* http://www.valleyair.org/transportation/GAMAQI_3-19-15.pdf ("CEQA Guidance").

[3] Toxic air contaminants, also known as hazardous air pollutants, are those pollutants that are known or suspected to cause cancer or other serious health effects, such as birth defects. There are currently 189 toxic air contaminants regulated by the United States Environmental Protection Agency ("EPA") and the states pursuant to the Clean Air Act. 42 U.S.C. § 7412. Common TACs include benzene, perchloroethylene and asbestos. *Id.* at 7412(b).

In contrast, there are only six (6) criteria air pollutants: ozone, particulate matter, carbon monoxide, nitrogen oxides, sulfur dioxide and lead. Although criteria air pollutants can also be harmful to human health, they are distinguishable from toxic air contaminants and are regulated separately. For instance, while criteria pollutants are regulated by numerous sections throughout Title I of the Clean Air Act, the regulation of toxic air contaminants occurs solely under section 112 of the Act. Compare 42 U.S.C. §§ 7407 – 7411 & 7501 – 7515 with 42 U.S.C. § 7411.

[4] See, e.g., CEQA Guidance at http://www.valleyair.org/transportation/GAMAQI_3-19-15.pdf, pp. 64-66, 80.

[5] See, e.g., CEQA Guidance at http://www.valleyair.org/transportation/GAMAQI_3-19-15.pdf, pp. 66, 99-101.

pollutants and local health impacts, departs from the Air District's Guidance and approved methodology for assessing criteria pollutants. A close reading of the administrative record that gave rise to this issue demonstrates that the Court's holding is based on a misunderstanding of the distinction between toxic air contaminants (for which a local health risk assessment is feasible and routinely performed) and criteria air pollutants (for which a local health risk assessment is not feasible and would result in speculative results).[6] The Air District has a direct interest in ensuring the lawfulness and consistent application of its CEQA Guidance, and will explain how the Court of Appeal departed from the Air District's long-standing CEQA Guidance in addressing criteria pollutants and toxic air contaminants in this amicus brief.

2. How the Proposed Amicus Curiae Brief Will Assist the Court

As counsel for the proposed amicus curiae, we have reviewed the briefs filed in this action. In addition to serving as a "commentary agency" for CEQA purposes over the Friant Ranch project, the Air District has a strong interest in assuring that CEQA is used for its intended purpose, and believes that this Court would benefit from additional briefing explaining the distinction between criteria pollutants and toxic air contaminants and the different methodologies employed by local air pollution control agencies such as the Air District to analyze these two categories of air pollutants under CEQA. The Air District will also explain how the Court of Appeal's opinion is based upon a fundamental misunderstanding of these two different approaches by requiring the County of Fresno to correlate the project's *criteria* pollution emissions with *local* health impacts. In doing

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[6] CEQA does not require speculation. *See, e.g., Laurel Heights Improvement Ass'n v. Regents of Univ. of Cal.*, 6 Cal. 4th 1112, 1137 (1993) (upholding EIR that failed to evaluate cumulative toxic air emission increases given absence of any acceptable means for doing so).

so, the Air District will provide helpful analysis to support its position that at least insofar as criteria pollutants are concerned, CEQA does not require an EIR to correlate a project's air quality emissions to specific health impacts, because such an analysis is not reasonably feasible.

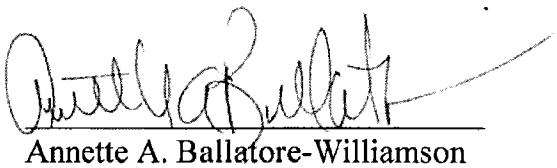
Rule 8.520 Disclosure

Pursuant to Cal. R. 8.520(f)(4), neither the Plaintiffs nor the Defendant or Real Party In Interest or their respective counsel authored this brief in whole or in part. Neither the Plaintiffs nor the Defendant or Real Party in Interest or their respective counsel made any monetary contribution towards or in support of the preparation of this brief.

CONCLUSION

On behalf of the San Joaquin Valley Unified Air Pollution Control District, we respectfully request that this Court accept the filing of the attached brief.

Dated: April 2, 2015



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SAN JOAQUIN VALLEY UNIFIED
AIR POLLUTION CONTROL
DISTRICT

CASE NO. S219783

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I. INTRODUCTION.

The San Joaquin Valley Unified Air Pollution Control District ("Air District") respectfully submits that the Court of Appeal erred when it held that the air quality analysis contained in the Environmental Impact Report ("EIR") for the Friant Ranch development project was inadequate under the California Environmental Quality Act ("CEQA") because it did not include an analysis of the correlation between the project's criteria air pollutants and the potential adverse human health impacts. A close reading of the portion of the administrative record that gave rise to this issue demonstrates that the Court's holding is based on a misunderstanding of the distinction between toxic air contaminants and criteria air pollutants.

Toxic air contaminants, also known as hazardous air pollutants, are those pollutants that are known or suspected to cause cancer or other serious health effects, such as birth defects. There are currently 189 toxic air contaminants (hereinafter referred to as "TACs") regulated by the United States Environmental Protection Agency ("EPA") and the states pursuant to the Clean Air Act. 42 U.S.C. § 7412. Common TACs include benzene, perchloroethylene and asbestos. *Id.* at 7412(b).

In contrast, there are only six (6) criteria air pollutants: ozone, particulate matter, carbon monoxide, nitrogen oxides, sulfur dioxide and lead. Although criteria air pollutants can also be harmful to human health,

they are distinguishable from TACs and are regulated separately. For instance, while criteria pollutants are regulated by numerous sections throughout Title I of the Clean Air Act, the regulation of TACs occurs solely under section 112 of the Act. *Compare* 42 U.S.C. §§ 7407 – 7411 & 7501 – 7515 *with* 42 U.S.C. § 7411.

The most relevant difference between criteria pollutants and TACs for purposes of this case is the manner in which human health impacts are accounted for. While it is common practice to analyze the correlation between an individual facility's TAC emissions and the expected localized human health impacts, such is not the case for criteria pollutants. Instead, the human health impacts associated with criteria air pollutants are analyzed and taken into consideration when EPA sets the national ambient air quality standard ("NAAQS") for each criteria pollutant. 42 U.S.C. § 7409(b)(1). The health impact of a particular criteria pollutant is analyzed on a regional and not a facility level based on how close the area is to complying with (attaining) the NAAQS. Accordingly, while the type of individual facility / health impact analysis that the Court of Appeal has required is a customary practice for TACs, it is not feasible to conduct a similar analysis for criteria air pollutants because currently available computer modeling tools are not equipped for this task.

It is clear from a reading of both the administrative record and the Court of Appeal's decision that the Court did not have the expertise to fully

appreciate the difference between TACs and criteria air pollutants. As a result, the Court has ordered the County of Fresno to conduct an analysis that is not practicable and not likely yield valid information. The Air District respectfully requests that this portion of the Court of Appeal's decision be reversed.

II. THE COURT OF APPEAL ERRED IN FINDING THE FRIANT RANCH EIR INADEQUATE FOR FAILING TO ANALYZE THE SPECIFIC HUMAN HEALTH IMPACTS ASSOCIATED CRITERIA AIR POLLUTANTS.

Although the Air District does not take lightly the amount of air emissions at issue in this case, it submits that the Court of Appeal got it wrong when it required Fresno County to revise the Friant Ranch EIR to include an analysis correlating the criteria air pollutant emissions associated with the project with specific, localized health-impacts. The type of analysis the Court of Appeal has required will not yield reliable information because currently available modeling tools are not well suited for this task. Further, in reviewing this issue de novo, the Court of Appeal failed to appreciate that it lacked the scientific expertise to appreciate the significant differences between a health risk assessment commonly performed for toxic air contaminants and a similar type of analysis it felt should have been conducted for criteria air pollutants.

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A. Currently Available Modeling Tools are not Equipped to Provide a Meaningful Analysis of the Correlation between an Individual Development Project's Air Emissions and Specific Human Health Impacts.

In order to appreciate the problematic nature of the Court of Appeals' decision requiring a health risk type analysis for criteria air pollutants, it is important to understand how the relevant criteria pollutants (ozone and particulate matter) are formed, dispersed and regulated.

Ground level ozone (smog) is not directly emitted into the air, but is formed when precursor pollutants such as oxides of nitrogen (NOx) and volatile organic compounds (VOCs) are emitted into the atmosphere and undergo complex chemical reactions in the process of sunlight.[1] Once formed, ozone can be transported long distances by wind.[2] Because of the complexity of ozone formation, a specific tonnage amount of NOx or VOCs emitted in a particular area does not equate to a particular concentration of ozone in that area. In fact, even rural areas that have relatively low tonnages of emissions of NOx or VOCs can have high levels of ozone concentration simply due to wind transport.[3] Conversely, the San Francisco Bay Area has six times more NOx and VOC emissions per square mile than the San Joaquin Valley, but experiences lower

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[1] *See* United States Environmental Protection Agency, *Ground-level Ozone: Basic Information*, available at: http://www.epa.gov/airquality/ozonepollution/basic.html (visited March 10, 2015).
[2] *Id.*
[3] *Id.*

concentrations of ozone (and better air quality) simply because sea breezes disperse the emissions.[4]

Particulate matter ("PM") can be divided into two categories: directly emitted PM and secondary PM.[5] While directly emitted PM can have a localized impact, the tonnage emitted does not always equate to the local PM concentration because it can be transported long distances by wind.[6] Secondary PM, like ozone, is formed via complex chemical reactions in the atmosphere between precursor chemicals such as sulfur dioxides (SOx) and NOx.[7] Because of the complexity of secondary PM formation, the tonnage of PM-forming precursor emissions in an area does not necessarily result in an equivalent concentration of secondary PM in that area.

The disconnect between the *tonnage* of precursor pollutants (NOx, SOx and VOCs) and the *concentration* of ozone or PM formed is important because it is not necessarily the tonnage of precursor pollutants that causes human health effects, but the concentration of resulting ozone or PM. Indeed, the national ambient air quality standards ("NAAQS"), which are statutorily required to be set by the United States Environmental Protection

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[4] *San Joaquin Valley Air Pollution Control District 2007 Ozone Plan*, Executive Summary p. ES-6, available at: http://www.valleyair.org/Air_Quality_Plans/docs/AQ_Ozone_2007_Adopted/03%20Executive%20Summary.pdf (visited March 10, 2015).

[5] United States Environmental Protection Agency, *Particulate Matter: Basic Information*, available at: http://www.epa.gov/airquality/particlepollution/basic.html (visited March 10, 2015).

[6] *Id.*

[7] *Id.*

Agency ("EPA") at levels that are "requisite to protect the public health," 42 U.S.C. § 7409(b)(1), are established as concentrations of ozone or particulate matter and not as tonnages of their precursor pollutants.[8]

Attainment of a particular NAAQS occurs when the concentration of the relevant pollutant remains below a set threshold on a consistent basis throughout a particular region. For example, the San Joaquin Valley attained the 1-hour ozone NAAQS when ozone concentrations remained at or below 0.124 parts per million Valley-wide on 3 or fewer days over a 3-year period.[9] Because the NAAQS are focused on achieving a particular concentration of pollution region-wide, the Air District's tools and plans for attaining the NAAQS are regional in nature.

For instance, the computer models used to simulate and predict an attainment date for the ozone or particulate matter NAAQS in the San Joaquin Valley are based on regional inputs, such as regional inventories of precursor pollutants (NOx, SOx and VOCs) and the atmospheric chemistry and meteorology of the Valley.[10] At a very basic level, the models simulate future ozone or PM levels based on predicted changes in precursor

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[8] See, e.g., United States Environmental Protection Agency, *Table of National Ambient Air Quality Standards*, available at: http://www.epa.gov/air/criteria.html#3 (visited March 10, 2015).

[9] *San Joaquin Valley Unified Air Pollution Control District 2013 Plan for the Revoked 1-Hour Ozone Standard*, Ch. 2 p. 2-16, available at: http://www.valleyair.org/Air_Quality_Plans/OzoneOneHourPlan2013/02Chapter2ScienceTrendsModeling.pdf (visited March 10, 2015).

[10] *Id.* at Ch. 2 p. 2-19 (visited March 12, 2015); *San Joaquin Valley Unified Air Pollution Control District 2008 PM2.5 Plan*, Appendix F, pp. F-2 – F-5, available at: http://www.valleyair.org/Air_Quality_Plans/docs/AQ_Final_Adopted_PM2.5/20%20Appendix%20F.pdf (visited March 19, 2015).

emissions Valley wide.[11] Because the NAAQS are set levels necessary to protect human health, the closer a region is to attaining a particular NAAQS, the lower the human health impact is from that pollutant.

The goal of these modeling exercises is not to determine whether the emissions generated by a particular factory or development project will affect the date that the Valley attains the NAAQS. Rather, the Air District's modeling and planning strategy is regional in nature and based on the extent to which *all* of the emission-generating sources in the Valley (current and future) must be controlled in order to reach attainment.[12]

Accordingly, the Air District has based its thresholds of significance for CEQA purposes on the levels that scientific and factual data demonstrate that the Valley can accommodate without affecting the attainment date for the NAAQS.[13] The Air District has tied its CEQA significance thresholds to the level at which stationary pollution sources permitted by the Air District must "offset" their emissions.[14] This "offset"

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[11] *Id.*

[12] Although the Air District does have a dispersion modeling tool used during its air permitting process that is used to predict whether a particular project's directly emitted PM will either cause an exceedance of the PM NAAQS or contribute to an existing exceedance, this model bases the prediction on a worst case scenario of emissions and meteorology and has no provision for predicting any associated human health impacts. Further, this analysis is only performed for stationary sources (factories, oil refineries, etc.) that are required to obtain a New Source Review permit from the Air District and not for development projects such as Friant Ranch over which the Air District has no preconstruction permitting authority. *See* San Joaquin Valley Unified Air Pollution Control District Rule 2201 §§ 2.0; 3.3.9; 4.14.1, available at: http://www.valleyair.org/rules/currntrules/Rule2201041l.pdf (visited March 19, 2015).

[13] *San Joaquin Valley Unified Air Pollution Control District Guide to Assessing and Mitigating Air Quality Impacts*, (March 19, 2015) p. 22, available at: http://www.valleyair.org/transportation/CEQA%20Rules/GAMAQI%20Jan%202002%20Rev.pdf (visited March 30, 2015).

[14] *Id.* at pp. 22, 25.

level allows for growth while keeping the cumulative effects of all new sources at a level that will not impede attainment of the NAAQS.[15] In the Valley, these thresholds are 15 tons per year of PM, and 10 tons of NOx or VOC per year. *Sierra Club*, *supra*, 172 Cal.Rptr.3d at 303; AR 4554. Thus, the CEQA air quality analysis for criteria pollutants is not really a localized, project-level impact analysis but one of regional, "cumulative impacts."

Accordingly, the significance thresholds applied in the Friant Ranch EIR (15 tons per year of PM and 10 tons of NOx or VOCs) are not intended to be indicative of any localized human health impact that the project may have. While the health effects of air pollution are of primary concern to the Air District (indeed, the NAAQS are established to protect human health), the Air District is simply not equipped to analyze whether and to what extent the criteria pollutant emissions of an individual CEQA project directly impact human health in a particular area. This is true even for projects with relatively high levels of emissions of criteria pollutant precursor emissions.

For instance, according to the EIR, the Friant Ranch project is estimated to emit 109.52 tons per year of ROG (VOC), 102.19 tons per year of NOx, and 117.38 tons per year of PM. Although these levels well

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[15] [15] *San Joaquin Valley Unified Air Pollution Control District Environmental Review Guidelines* (Aug. 2000) p. 4-11, available at: http://www.valleyair.org/transportation/CEQA%20Rules/ERG%20Adopted%20_August%202000_.pdf (visited March 12, 2015).

exceed the Air District's CEQA significance thresholds, this does not mean that one can easily determine the concentration of ozone or PM that will be created at or near the Friant Ranch site on a particular day or month of the year, or what specific health impacts will occur. Meteorology, the presence of sunlight, and other complex chemical factors all combine to determine the ultimate concentration and location of ozone or PM. This is especially true for a project like Friant Ranch where most of the criteria pollutant emissions derive not from a single "point source," but from area wide sources (consumer products, paint, etc.) or mobile sources (cars and trucks) driving to, from and around the site.

In addition, it would be extremely difficult to model the impact on NAAQS attainment that the emissions from the Friant Ranch project may have. As discussed above, the currently available modeling tools are equipped to model the impact of *all* emission sources in the Valley on attainment. According to the most recent EPA-approved emission inventory, the NOx inventory for the Valley is for the year 2014 is 458.2 tons per day, or 167,243 tons per year and the VOC (or ROG) inventory is 361.7 tons per day, or 132,020.5 tons per year.[16] Running the photochemical grid model used for predicting ozone attainment with the

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[16] *San Joaquin Valley Unified Air Pollution Control District 2007 Ozone Plan*, Appendix B pp. B-6, B-9,
available at:
http://www.valleyair.org/Air_Quality_Plans/docs/AQ_Ozone_2007_Adopted/19%20Appendix%20B%20April%202007.pdf (visited March 12, 2015).

emissions solely from the Friant Ranch project (which equate to less than one-tenth of one percent of the total NOx and VOC in the Valley) is not likely to yield valid information given the relative scale involved.

Finally, even once a model is developed to accurately ascertain local increases in concentrations of photochemical pollutants like ozone and some particulates, it remains impossible, using today's models, to correlate that increase in concentration to a specific health impact. The reason is the same: such models are designed to determine regional, population-wide health impacts, and simply are not accurate when applied at the local level.

For these reasons, it is not the norm for CEQA practitioners, including the Air District, to conduct an analysis of the localized health impacts associated with a project's criteria air pollutant emissions as part of the EIR process. When the accepted scientific method precludes a certain type of analysis, "the court cannot impose a legal standard to the contrary." *Kings County Farm Bureau v. City of Hanford* (1990) 221 Cal.App.3d 692, 717 n. 8. However, that is exactly what the Court of Appeal has done in this case. Its decision upends the way CEQA air quality analysis of criteria pollutants occurs and should be reversed.

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B. The Court of Appeal Improperly Extrapolated a Request for a Health Risk Assessment for Toxic Air Contaminants into a Requirement that the EIR contain an Analysis of Localized Health Impacts Associated with Criteria Air Pollutants.

The Court of Appeal's error in requiring the new health impact analysis for criteria air pollutants clearly stems from a misunderstanding of terms of art commonly used in the air pollution field. More specifically, the Court of Appeal (and Appellants Sierra Club et al.) appear to have confused the health risk analysis ("HRA") performed to determine the health impacts associated with a project's toxic air contaminants ("TACs"), with an analysis correlating a project's criteria air pollutants (ozone, PM and the like) with specific localized health impacts.

The first type of analysis, the HRA, is commonly performed during the Air District's stationary source permitting process for projects that emit TACs and is, thus, incorporated into the CEQA review process. An HRA is a comprehensive analysis to evaluate and predict the dispersion of TACs emitted by a project and the potential for exposure of human populations. It also assesses and quantifies both the individual and population-wide health risks associated with those levels of exposure. There is no similar analysis conducted for criteria air pollutants. Thus, the second type of analysis (required by the Court of Appeal), is not currently part of the Air District's process because, as outlined above, the health risks associated

with exposure to criteria pollutants are evaluated on a regional level based on the region's attainment of the NAAQS.

The root of this confusion between the types of analyses conducted for TACs versus criteria air pollutants appears to stem from a comment that was presented to Fresno County by the City of Fresno during the administrative process.

In its comments on the draft EIR, the City of Fresno (the only party to raise this issue) stated:

> [t]he EIR must disclose the human health related effects of the Project's air pollution impacts. (CEQA Guidelines section 15126.2(a).) The EIR fails completely in this area. The EIR should be revised to disclose and determine the significance of TAC impacts, and of human health risks due to exposure to Project-related air emissions.

(AR 4602.)

In determining that the issue regarding the correlation between the Friant Ranch project's criteria air pollutants and adverse health impacts was adequately exhausted at the administrative level, the Court of Appeal improperly read the first two sentences of the City of Fresno's comment in isolation rather than in the context of the entire comment. *See Sierra Club v. County of Fresno* (2014) 172 Cal.Rptr.3d 271, 306. Although the comment first speaks generally in terms of "human health related effects" and "air pollution," it requests only that the EIR be revised to disclose "the significance of TACs" and the "human health risks due to exposure."

The language of this request in the third sentence of the comment is significant because, to an air pollution practitioner, the language would only have indicated only that a HRA for TACs was requested, and not a separate analysis of the health impacts associated with the project's criteria air pollutants. Fresno County clearly read the comment as a request to perform an HRA for TACs and limited its response accordingly. (AR 4602.)[17] The Air District submits that it would have read the City's comment in the same manner as the County because the City's use of the terms "human health risks" and "TACs" signal that an HRA for TACs is being requested. Indeed, the Air District was also concerned that an HRA be conducted, but understood that it was not possible to conduct such an analysis until the project entered the phase where detailed site specific information, such as the types of emission sources and the proximity of the sources to sensitive receptors became available. (AR 4553.)[18] The City of Fresno was apparently satisfied with the County's discussion of human health risks, as it did not raise the issue again when it commented on the final EIR. (AR 8944 – 8960.)

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[17] Appellants do not challenge the manner in which the County addressed TACs in the EIR. (Appellants' Answer Brief p. 28 fn. 7.)

[18] Appellants rely on the testimony of Air District employee, Dan Barber, as support for their position that the County should have conducted an analysis correlating the project's criteria air pollutant emissions with localized health impacts. (Appellants Answer Brief pp. 10-11; 28.) However, Mr. Barber's testimony simply reinforces the Air District's concern that a risk assessment (HRA) be conducted once the actual details of the project become available. (AR 8863.) As to criteria air pollutants, Mr. Barber's comments are aimed at the Air District's concern about the amount of emissions and the fact that the emissions will make it "more difficult for Fresno County and the Valley to reach attainment which means that the health of Valley residents maybe [sic] adversely impacted." Mr. Barber says nothing about conducting a separate analysis of the localized health impacts the project's emissions may have.

The Court of Appeal's holding, which incorrectly extrapolates a request for an HRA for TACs into a new analysis of the localized health impacts of the project's criteria air pollutants, highlights two additional errors in the Court's decision.

First, the Court of Appeal's holding illustrates why the Court should have applied the deferential substantial evidence standard of review to the issue of whether the EIR's air quality analysis was sufficient. The regulation of air pollution is a technical and complex field and the Court of Appeal lacked the expertise to fully appreciate the difference between TACs and criteria air pollutants and tools available for analyzing each type of pollutant.

Second, it illustrates that the Court likely got it wrong when it held that the issue regarding the criteria pollutant / localized health impact analysis was properly exhausted during the administrative process. In order to preserve an issue for the court, '[t]he "exact issue" must have been presented to the administrative agency....' [Citation.] *Citizens for Responsible Equitable Environmental Development v. City of San Diego*, (2011) 196 Cal.App.4th 515, 527 129 Cal.Rptr.3d 512, 521; *Sierra Club v. City of Orange* (2008) 163 Cal.App.4th 523, 535, 78 Cal.Rptr.3d 1, 13. "'[T]he objections must be sufficiently specific so that the agency has the

opportunity to evaluate and respond to them.' [Citation.]" *Sierra Club v. City of Orange*,163 Cal.App.4th at 536.[19]

As discussed above, the City's comment, while specific enough to request a commonly performed HRA for TACs, provided the County with no notice that it should perform a new type of analysis correlating criteria pollutant tonnages to specific human health effects. Although the parties have not directly addressed the issue of failure to exhaust administrative remedies in their briefs, the Air District submits that the Court should consider how it affects the issues briefed by the parties since "[e]xhaustion of administrative remedies is a jurisdictional prerequisite to maintenance of a CEQA action." *Bakersfield Citizens for Local Control v. City of Bakersfield* (2004) 124 Cal.App.4th 1184, 1199, 22 Cal.Rptr.3d 203.

III. CONCLUSION

For all of the foregoing reasons, the Air District respectfully requests that the portion of the Court of Appeal's decision requiring an analysis correlating the localized human health impacts associated with an individual project's criteria air pollutant emissions be reversed.

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[19] *Sierra Club v. City of Orange*, is illustrative here. In that case, the plaintiffs challenged an EIR approved for a large planned community on the basis that the EIR improperly broke up the various environmental impacts by separate project components or "piecemealed" the analysis in violation of CEQA. In evaluating the defense that the plaintiffs had failed to adequately raise the issue at the administrative level, the Court held that comments such as *"the use of a single document for both a project-level and a program-level EIR [is] 'confusing',"* and *"[t]he lead agency should identify any potential adverse air quality impacts that could occur from all phases of the project and all air pollutant sources related to the project,"* were too vague to fairly raise the argument of piecemealing before the agency. *Sierra Club v. City of Orange*, 163 Cal.App.4th at 537.

correlating the localized human health impacts associated with an individual project's criteria air pollutant emissions be reversed.

Respectfully submitted,

Dated: April 2, 2015

Catherine T. Redmond
Attorney for Proposed Amicus Curiae

SAN JOAQUIN VALLEY
UNIFIED
AIR POLLUTION CONTROL
DISTRICT

CERTIFICATE OF WORD COUNT

Pursuant to Rule 8.204 of the California Rules of Court, I hereby certify that this document, based on the Word County feature of the Microsoft Word software program used to compose and print this document, contains, exclusive of caption, tables, certificate of word count, signature block and certificate of service, 3806 words.

Dated: April 2, 2015

Annette A. Ballatore-Williamson
District Counsel (SBN 192176)

Sierra Club et al, v. County of Fresno, et al
Supreme Court of California Case No.: S219783
Fifth District Court of Appeal Case No.: F066798
Fresno County Superior Court Case No.: 11CECG00726

PROOF OF SERVICE

I am over the age of 18 years and not a p[arty to the above-captioned action; that my business address is San Joaquin Valley Unified Air Pollution Control District located at 1990 E. Gettysburg Avenue, Fresno, California 93726.

On April 2, 2015, I served the document described below:

APPLICATION FOR LEAVE TO FILE AMICUS CURIAE BRIEF OF SAN JOAQUIN VALLEY UNIFIED AIR POLLUTION CONTROL DISTRICT IN SUPPORT OF DEFENDANT AND RESPONDENT, COUNTY OF FRESNO

On all parties to this action at the following addresses and in the following manner:

PLEASE SEE ATTACHED SERVICE LIST

(XX) **(BY MAIL)** I caused a true copy of each document(s) to be laced in a sealed envelope with first-class postage affixed and placed the envelope for collection. Mail is collected daily at my office and placed in a United State Postal Service collection box for pick-up and delivery that same day.

( ) **(BY ELECTRONIC MAIL)** I caused a true and correct scanned image (.PDF file) copy to be transmitted via electronic mail transfer system in place at the San Joaquin Valley Unified Air Pollution Control District ("District"), originating from the undersigned at 1990 E. Gettysburg Avenue, Fresno, CA, to the address(es) indicated below.

( ) **(BY OVERNIGHT MAIL)** I caused a true and correct copy to be delivered via Federal Express to the following person(s) or their representative at the address(es) listed below.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct and that I executed this document on April 2, 2015, at Fresno, California.

Esthela Soto

SERVICE LIST

Sierra Club et al, v. County of Fresno, et al
Supreme Court of California Case No.: S219783
Fifth District Court of Appeal Case No.: F066798
Fresno County Superior Court Case No.: 11CECG00726

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S219783

IN THE SUPREME COURT OF C ALIFORNIA

SIERRA CLUB, REVIVE THE SAN JOAQUIN, and
LEAGUE OF WOMEN VOTERS OF FRESNO,

Plaintiffs and Appellants,

v.

COUNTY OF FRESNO,

Defendant and Respondent,

and,

FRIANT RANCH, L.P.,

Real Party in Interest and Respondent.

After a Published Decision by the Court of Appeal, filed May 27, 2014
Fifth Appellate District Case No. F066798

Appeal from the Superior Court of California, County of Fresno
Case No. 11CECG00726
Honorable Rosendo A. Pena, Jr.

APPLICATION OF THE SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT FOR LEAVE TO FILE BRIEF OF *AMICUS CURIAE* IN SUPPORT OF NEITHER PARTY AND *[PROPOSED]* BRIEF OF *AMICUS CURIAE*

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TO THE HONORABLE CHIEF JUSTICE AND JUSTICES OF THE SUPREME COURT:

APPLICATION FOR LEAVE TO FILE *AMICUS CURIAE* BRIEF

Pursuant to Rule 8.520(f) of the California Rules of Court, the South Coast Air Quality Management District (SCAQMD) respectfully requests leave to file the attached *amicus curiae* brief. Because SCAQMD's position differs from that of either party, we request leave to submit this amicus brief in support of neither party.

HOW THIS BRIEF WILL ASSIST THE COURT

SCAQMD's proposed amicus brief takes a position on two of the issues in this case. In both instances, its position differs from that of either party. The issues are:

1) Does the California Environmental Quality Act (CEQA) require an environmental impact report (EIR) to correlate a project's air pollution emissions with specific levels of health impacts?
2) What is the proper standard of review for determining whether an EIR provides sufficient information on the health impacts caused by a project's emission of air pollutants?

This brief will assist the Court by discussing the practical realities of correlating identified air quality impacts with specific health outcomes. In short, CEQA requires agencies to provide detailed information about a project's air quality impacts that is sufficient for the public and decisionmakers to adequately evaluate the project and meaningfully understand its impacts. However, the level of analysis is governed by a rule of reason; CEQA only requires agencies to conduct analysis if it is reasonably feasible to do so.

With regard to health-related air quality impacts, an analysis that correlates a project's air pollution emissions with specific levels of health impacts will be feasible in some cases but not others. Whether it is feasible depends on a variety of factors, including the nature of the project and the nature of the analysis under consideration. The feasibility of analysis may also change over time as air districts and others develop new tools for measuring projects' air quality related health impacts. Because SCAQMD has among the most sophisticated air quality modeling and health impact evaluation capability of any of the air districts in the State, it is uniquely situated to express an opinion on the extent to which the Court should hold that CEQA requires lead agencies to correlate air quality impacts with specific health outcomes.

SCAQMD can also offer a unique perspective on the question of the appropriate standard of review. SCAQMD submits that the proper standard of review for determining whether an EIR is sufficient as an informational document is more nuanced than argued by either party. In our view, this is a mixed question of fact and law. It includes determining whether additional analysis is feasible, which is primarily a factual question that should be reviewed under the substantial evidence standard. However, it also involves determining whether the omission of a particular analysis renders an EIR insufficient to serve CEQA's purpose as a meaningful, informational document. If a lead agency has not determined that a requested analysis is infeasible, it is the court's role to determine whether the EIR nevertheless meets CEQA's purposes, and courts should not defer to the lead agency's conclusions regarding the legal sufficiency of an EIR's analysis. The ultimate question of whether an EIR's analysis is "sufficient" to serve CEQA's informational purposes is predominately a question of law that courts should review de novo.

This brief will explain the rationale for these arguments and may assist the Court in reaching a conclusion that accords proper respect to a lead agency's factual conclusions while maintaining judicial authority over the ultimate question of what level of analysis CEQA requires.

STATEMENT OF INTEREST OF *AMICUS CURIAE*

The SCAQMD is the regional agency primarily responsible for air pollution control in the South Coast Air Basin, which consists of all of Orange County and the non-desert portions of the Los Angeles, Riverside, and San Bernardino Counties. (Health & Saf. Code § 40410; Cal. Code Regs., tit. 17, § 60104.) The SCAQMD participates in the CEQA process in several ways. Sometimes it acts as a lead agency that prepares CEQA documents for projects. Other times it acts as a responsible agency when it has permit authority over some part of a project that is undergoing CEQA review by a different lead agency. Finally, SCAQMD also acts as a commenting agency for CEQA documents that it receives because it is a public agency with jurisdiction by law over natural resources affected by the project.

In all of these capacities, SCAQMD will be affected by the decision in this case. SCAQMD sometimes submits comments requesting that a lead agency perform an additional type of air quality or health impacts analysis. On the other hand, SCAQMD sometimes determines that a particular type of health impact analysis is not feasible or would not produce reliable and informative results. Thus, SCAQMD will be affected by the Court's resolution of the extent to which CEQA requires EIRs to correlate emissions and health impacts, and its resolution of the proper standard of review.

CERTIFICATION REGARDING AUTHORSHIP AND FUNDING

No party or counsel in the pending case authored the proposed amicus curiae brief in whole or in part, or made any monetary contribution intended to fund the preparation or submission of the brief. No person or entity other than the proposed *Amicus Curiae* made any monetary contribution intended to fund the preparation or submission of the brief.

Respectfully submitted,

DATED: April 3, 2015

SOUTH COAST AIR QUALITY
MANAGEMENT DISTRICT
KURT R. WIESE, GENERAL COUNSEL
BARBARA BAIRD, CHIEF DEPUTY COUNSEL

By: Barbara Baird
Barbara Baird
Attorneys for [proposed] Amicus Curiae
SOUTH COAST AIR QUALITY
MANAGEMENT DISTICT

BRIEF OF AMICUS CURIAE

SUMMARY OF ARGUMENT

The South Coast Air Quality Management District (SCAQMD) submits that this Court should not try to establish a hard-and-fast rule concerning whether lead agencies are required to correlate emissions of air pollutants with specific health consequences in their environmental impact reports (EIR). The level of detail required in EIRs is governed by a few, core CEQA (California Environmental Quality Act) principles. As this Court has stated, "[a]n EIR must include detail sufficient to enable those who did not participate in its preparation to understand and to consider meaningfully the issues raised by the proposed project." (*Laurel Heights Improvement Assn. v. Regents of the Univ of Cal.* (1988) 47 Cal.3d 376, 405 ["*Laurel Heights I*"]) Accordingly, "an agency must use its best efforts to find out and disclose all that it reasonably can." (*Vineyard Area Citizens for Responsible Growth, Inc. v. City of Rancho Cordova* (2007) 40 Cal.4th 412, 428 (quoting CEQA Guidelines § 15144)[1].). However, "[a]nalysis of environmental effects need not be exhaustive, but will be judged in light of what is reasonably feasible." (*Association of Irritated Residents v. County of Madera* (2003) 107 Cal.App.4th 1383, 1390; CEQA Guidelines §§ 15151, 15204(a).)

With regard to analysis of air quality related health impacts, EIRs must generally quantify a project's pollutant emissions, but in some cases it is not feasible to correlate these emissions to specific, quantifiable health impacts (e.g., premature mortality; hospital admissions). In such cases, a general description of the adverse health impacts resulting from the pollutants at issue may be sufficient. In other cases, due to the magnitude

[1] The CEQA Guidelines are found at Cal. Code Regs., tit. 14 §§ 15000, *et seq.*

or nature of the pollution emissions, as well as the specificity of the project involved, it may be feasible to quantify health impacts. Or there may be a less exacting, but still meaningful analysis of health impacts that can feasibly be performed. In these instances, agencies should disclose those impacts.

SCAQMD also submits that whether or not an EIR complies with CEQA's informational mandates by providing sufficient, feasible analysis is a mixed question of fact and law. Pertinent here, the question of whether an EIR's discussion of health impacts from air pollution is sufficient to allow the public to understand and consider meaningfully the issues involves two inquiries: (1) Is it feasible to provide the information or analysis that a commenter is requesting or a petitioner is arguing should be required?; and (2) Even if it is feasible, is the agency relying on other policy or legal considerations to justify not preparing the requested analysis? The first question of whether an analysis is feasible is primarily a question of fact that should be judged by the substantial evidence standard. The second inquiry involves evaluating CEQA's information disclosure purposes against the asserted reasons to not perform the requested analysis. For example, an agency might believe that its EIR meets CEQA's informational disclosure standards even without a particular analysis, and therefore choose not to conduct that analysis. SCAQMD submits that this is more of a legal question, which should be reviewed de novo as a question of law.

ARGUMENT

I. RELEVANT FACTUAL AND LEGAL FRAMEWORK.

A. Air Quality Regulatory Background

The South Coast Air Quality Management District (SCAQMD) is one of the local and regional air pollution control districts and air quality

management districts in California. The SCAQMD is the regional air pollution agency for the South Coast Air Basin, which consists of all of Orange County and the non-desert portions of Los Angeles, Riverside, and San Bernardino Counties. (Health & Saf. Code § 40410, 17 Cal. Code Reg. § 60104.) The SCAQMD also includes the Coachella Valley in Riverside County (Palm Springs area to the Salton Sea). (SCAQMD, *Final 2012 AQMP (Feb. 2013)*, http://www.aqmd.gov/home/library/clean-air-plans/air-quality-mgt-plan/final-2012-air-quality-management-plan; then follow "chapter 7" hyperlink; pp 7-1, 7-3 (last visited Apr. 1, 2015).) The SCAQMD's jurisdiction includes over 16 million residents and has the worst or nearly the worst air pollution levels in the country for ozone and fine particulate matter. (SCAQMD, *Final 2012 AQMP (Feb. 2013)*, http://www.aqmd.gov/home/library/clean-air-plans/air-quality-mgt-plan/final-2012-air-quality-management-plan; then follow "Executive Summary" hyperlink p. ES-1 (last visited Apr. 1, 2015).)

Under California law, the local and regional districts are primarily responsible for controlling air pollution from all sources except motor vehicles. (Health & Saf. Code § 40000.) The California Air Resources Board (CARB), part of the California Environmental Protection Agency, is primarily responsible for controlling pollution from motor vehicles. (*Id.*) The air districts must adopt rules to achieve and maintain the state and federal ambient air quality standards within their jurisdictions. (Health & Saf. Code § 40001.)

The federal Clean Air Act (CAA) requires the United States Environmental Protection Agency (EPA) to identify pollutants that are widely distributed and pose a threat to human health, developing a so-called "criteria" document. (42 U.S.C. § 7408; CAA § 108.) These pollutants are frequently called "criteria pollutants." EPA must then establish "national ambient air quality standards" at levels "requisite to protect public health",

allowing "an adequate margin of safety." (42 U.S.C. § 7409; CAA § 109.) EPA has set standards for six identified pollutants: ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, particulate matter (PM), and lead. (U.S. EPA, National Ambient Air Quality Standards (NAAQS), http://www.epa.gov/air/criteria.html (last updated Oct. 21, 2014).)[2]

Under the Clean Air Act, EPA sets emission standards for motor vehicles and "nonroad engines" (mobile farm and construction equipment, marine vessels, locomotives, aircraft, etc.). (42 U.S.C. §§ 7521, 7547; CAA §§ 202, 213.) California is the only state allowed to establish emission standards for motor vehicles and most nonroad sources; however, it may only do so with EPA's approval. (42 U.S.C. §§ 7543(b), 7543(e); CAA §§ 209(b), 209(c).) Sources such as manufacturing facilities, power plants and refineries that are not mobile are often referred to as "stationary sources." The Clean Air Act charges state and local agencies with the primary responsibility to attain the national ambient air quality standards. (42 U.S.C. § 7401(a)(3); CAA § 101(a)(3).) Each state must adopt and implement a plan including enforceable measures to achieve and maintain the national ambient air quality standards. (42 U.S.C. § 7410; CAA § 110.) The SCAQMD and CARB jointly prepare portion of the plan for the South Coast Air Basin and submit it for approval by EPA. (Health & Saf. Code §§ 40460, et seq.)

The Clean Air Act also requires state and local agencies to adopt a permit program requiring, among other things, that new or modified "major" stationary sources use technology to achieve the "lowest achievable emission rate," and to control minor stationary sources as

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[2] Particulate matter (PM) is further divided into two categories: fine particulate or $PM_{2.5}$ (particles with a diameter of less than or equal to 2.5 microns) and coarse particulate (PM_{10}) (particles with a diameter of 10 microns or less). (U.S. EPA, Particulate Matter (PM), http://www.epa.gov/airquality/particlepollution/ (last visited Apr. 1, 2015).)

needed to help attain the standards. (42 U.S.C. §§ 7502(c)(5), 7503(a)(2), 7410(a)(2)(C); CAA §§ 172(c)(5), 173(a)(2), 110(a)(2)(C).) The air districts implement these permit programs in California. (Health & Saf. Code §§ 42300, et seq.)

The Clean Air Act also sets out a regulatory structure for over 100 so-called "hazardous air pollutants" calling for EPA to establish "maximum achievable control technology" (MACT) for sources of these pollutants. (42 U.S.C. § 7412(d)(2); CAA § 112(d)(2).) California refers to these pollutants as "toxic air contaminants" (TACs) which are subject to two state-required programs. The first program requires "air toxics control measures" for specific categories of sources. (Health & Saf. Code § 39666.) The other program requires larger stationary sources and sources identified by air districts to prepare "health risk assessments" for impacts of toxic air contaminants. (Health & Saf. Code §§ 44320(b), 44322, 44360.) If the health risk exceeds levels identified by the district as "significant," the facility must implement a "risk reduction plan" to bring its risk levels below "significant" levels. Air districts may adopt additional more stringent requirements than those required by state law, including requirements for toxic air contaminants. (Health & Saf. Code § 41508; *Western Oil & Gas Assn. v. Monterey Bay Unified APCD* (1989) 49 Cal.3d 408, 414.) For example, SCAQMD has adopted a rule requiring new or modified sources to keep their risks below specified levels and use best available control technology (BACT) for toxics. (SCAQMD, *Rule 1401-New Source Review of Toxic Air Contaminants*, http://www.aqmd.gov/home/regulations/rules/scaqmd-rule-book/regulation-xiv; then follow "Rule 1401" hyperlink (last visited Apr. 1, 2015).)

B. The SCAQMD's Role Under CEQA

The California Environmental Quality Act (CEQA) requires public agencies to perform an environmental review and appropriate analysis for projects that they implement or approve. (Pub. Resources Code § 21080(a).) The agency with primary approval authority for a particular project is generally the "lead agency" that prepares the appropriate CEQA document. (CEQA Guidelines §§ 15050, 15051.) Other agencies having a subsequent approval authority over all or part of a project are called "responsible" agencies that must determine whether the CEQA document is adequate for their use. (CEQA Guidelines §§ 15096(c), 15381.) Lead agencies must also consult with and circulate their environmental impact reports to "trustee agencies" and agencies "with jurisdiction by law" including "authority over resources which may be affected by the project." (Pub. Resources Code §§ 21104(a), 21153; CEQA Guidelines §§ 15086(a)(3), 15073(c).) The SCAQMD has a role in all these aspects of CEQA.

Fulfilling its responsibilities to implement its air quality plan and adopt rules to attain the national ambient air quality standards, SCAQMD adopts a dozen or more rules each year to require pollution reductions from a wide variety of sources. The SCAQMD staff evaluates each rule for any adverse environmental impact and prepares the appropriate CEQA document. Although most rules reduce air emissions, they may have secondary environmental impacts such as use of water or energy or disposal of waste—e.g., spent catalyst from control equipment.[3]

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[3] The SCAQMD's CEQA program for its rules is a "Certified Regulatory Program" under which it prepares a "functionally equivalent" document in lieu of a negative declaration or EIR. (Pub. Resources Code § 21080.5, CEQA Guidelines § 15251(l).)

The SCAQMD also approves a large number of permits every year to construct new, modified, or replacement facilities that emit regulated air pollutants. The majority of these air pollutant sources have already been included in an earlier CEQA evaluation for a larger project, are currently being evaluated by a local government as lead agency, or qualify for an exemption. However, the SCAQMD sometimes acts as lead agency for major projects where the local government does not have a discretionary approval. In such cases, SCAQMD prepares and certifies a negative declaration or environmental impact report (EIR) as appropriate.[4] SCAQMD evaluates perhaps a dozen such permit projects under CEQA each year. SCAQMD is often also a "responsible agency" for many projects since it must issue a permit for part of the projects (e.g., a boiler used to provide heat in a commercial building). For permit projects evaluated by another lead agency under CEQA, SCAQMD has the right to determine that the CEQA document is inadequate for its purposes as a responsible agency, but it may not do so because its permit program already requires all permitted sources to use the best available air pollution control technology. (SCAQMD, *Rule 1303(a)(1) – Requirements*, http://www.aqmd.gov/home/regulations/rules/scaqmd-rule-book/regulation-xiii; then follow "Rule 1303" hyperlink (last visited Apr. 1, 2015).)

Finally, SCAQMD receives as many as 60 or more CEQA documents each month (around 500 per year) in its role as commenting agency or an agency with "jurisdiction by law" over air quality—a natural resource affected by the project. (Pub. Resources Code §§ 21104(a), 21153; CEQA Guidelines § 15366(a)(3).) The SCAQMD staff provides comments on as many as 25 or 30 such documents each month.

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[4] The SCAQMD's permit projects are not included in its Certified Regulatory Program, and are evaluated under the traditional local government CEQA analysis. (Pub. Resources Code §§ 21150-21154.)

(SCAQMD Governing Board Agenda, Apr. 3, 2015, Agenda Item 16, Attachment A, http://www.aqmd.gov/home/library/meeting-agendas-minutes/agenda?title=governing-board-meeting-agenda-april-3-2015; then follow "16. Lead Agency Projects and Environmental Documents Received by SCAQMD" hyperlink (last visited Apr. 1, 2015).) Of course, SCAQMD focuses its commenting efforts on the more significant projects.

Typically, SCAQMD comments on the adequacy of air quality analysis, appropriateness of assumptions and methodology, and completeness of the recommended air quality mitigation measures. Staff may comment on the need to prepare a health risk assessment detailing the projected cancer and noncancer risks from toxic air contaminants resulting from the project, particularly the impacts of diesel particulate matter, which CARB has identified as a toxic air contaminant based on its carcinogenic effects. (California Air Resources Board, Resolution 98-35, Aug. 27, 1998, http://www.arb.ca.gov/regact/diesltac/diesltac.htm; then follow Resolution 98-35 hyperlink (last visited Apr. 1, 2015).) Because SCAQMD already requires new or modified stationary sources of toxic air contaminants to use the best available control technology for toxics and to keep their risks below specified levels, (SCAQMD Rule 1401, supra, note 15), the greatest opportunity to further mitigate toxic impacts through the CEQA process is by reducing emissions—particularly diesel emissions—from vehicles.

II. THIS COURT SHOULD NOT SET A HARD-AND-FAST RULE CONCERNING THE EXTENT TO WHICH AN EIR MUST CORRELATE A PROJECT'S EMISSION OF POLLUTANTS WITH RESULTING HEALTH IMPACTS.

Numerous cases hold that courts do not review the correctness of an EIR's conclusions but rather its sufficiency as an informative document. (*Laurel Heights 1, supra*, 47 Cal.3d at p. 392; *Citizens of Goleta Valley v.*

Bd. of Supervisors (1990) 52 Cal.3d 553, 569; *Bakersfield Citizens for Local Control v. City of Bakersfield* (2004) 124 Cal.App.4th 1184, 1197.)

As stated by the Court of Appeal in this case, where an EIR has addressed a topic, but the petitioner claims that the information provided about that topic is insufficient, courts must "draw[] a line that divides *sufficient* discussions from those that are *insufficient*." (*Sierra Club v. County of Fresno* (2014) 226 Cal.App.4th 704 (superseded by grant of review) 172 Cal.Rptr.3d 271, 290.) The Court of Appeal readily admitted that "[t]he terms themselves – sufficient and insufficient – provide little, if any, guidance as to where the line should be drawn. They are simply labels applied once the court has completed its analysis." (*Id.*)

The CEQA Guidelines, however, provide guidance regarding what constitutes a sufficient discussion of impacts. Section 15151 states that "the sufficiency of an EIR is to be reviewed in light of what is reasonably feasible." Case law reflects this: "Analysis of environmental effects need not be exhaustive, but will be judged in light of what was reasonably feasible." (*Association of Irritated Residents v. County of Madera, supra,* 107 Cal.App.4th at p. 1390; see also CEQA Guidelines § 15204(a).)

Applying this test, this Court cannot realistically establish a hard-and-fast rule that an analysis correlating air pollution impacts of a project to quantified resulting health impacts is always required, or indeed that it is never required. Simply put, in some cases such an analysis will be "feasible"; in some cases it will not.

For example, air pollution control districts often require a proposed new source of toxic air contaminants to prepare a "health risk assessment" before issuing a permit to construct. District rules often limit the allowable cancer risk the new source may cause to the "maximally exposed individual" (worker and residence exposures). (*See, e.g.*, SCAQMD Rule 1401(c)(8); 1401(d)(1), *supra* note 15.) In order to perform this analysis, it

is necessary to have data regarding the sources and types of air toxic contaminants, location of emission points, velocity of emissions, the meteorology and topography of the area, and the location of receptors (worker and residence). (SCAQMD, *Supplemental Guidelines for Preparing Risk Assessments for the Air Toxics "Hot Spots" Information and Assessment Act (AB2588), pp. 11-16*; (last visited Apr. 1, 2015) http://www.aqmd.gov/home/library/documents-support-material; "Guidelines" hyperlink; AB2588; then follow AB2588 Risk Assessment Guidelines hyperlink.)

Thus, it is feasible to determine the health risk posed by a new gas station locating at an intersection in a mixed use area, where receptor locations are known. On the other hand, it may not be feasible to perform a health risk assessment for airborne toxics that will be emitted by a generic industrial building that was built on "speculation" (i.e., without knowing the future tenant(s)). Even where a health risk assessment can be prepared, however, the resulting maximum health risk value is only a calculation of risk—it does not necessarily mean anyone will contract cancer as a result of the project.

In order to find the "cancer burden" or expected additional cases of cancer resulting from the project, it is also necessary to know the numbers and location of individuals living within the "zone of impact" of the project: i.e., those living in areas where the projected cancer risk from the project exceeds one in a million. (SCAQMD, Health Risk Assessment Summary form, http://www.aqmd.gov/home/forms ; filter by "AB2588" category; then "Health Risk Assessment" hyperlink (last visited Apr. 1, 2015).) The affected population is divided into bands of those exposed to at least 1 in a million risk, those exposed to at least 10 in a million risk, etc. up to those exposed at the highest levels. (*Id.*) This data allows agencies to calculate an approximate number of additional cancer cases expected from

the project. However, it is not possible to predict which particular individuals will be affected.

For the so-called criteria pollutants[5], such as ozone, it may be more difficult to quantify health impacts. Ozone is formed in the atmosphere from the chemical reaction of the nitrogen oxides (NO_x) and volatile organic compounds (VOC) in the presence of sunlight. (U.S. EPA, Ground Level Ozone, http://www.epa.gov/airquality/ozonepollution/ (last updated Mar. 25, 2015).) It takes time and the influence of meteorological conditions for these reactions to occur, so ozone may be formed at a distance downwind from the sources. (U.S. EPA, *Guideline on Ozone Monitoring Site Selection* (Aug. 1998) EPA-454/R-98-002 § 5.1.2, http://www.epa.gov/ttnamti1/archive/cpreldoc.html (last visited Apr. 1, 2015).) NO_x and VOC are known as "precursors" of ozone.

Scientifically, health effects from ozone are correlated with increases in the ambient level of ozone in the air a person breathes. (U.S. EPA, *Health Effects of Ozone in the General Population,* Figure 9, http://www.epa.gov/apti/ozonehealth/population.html#levels (last visited Apr. 1, 2015).) However, it takes a large amount of additional precursor emissions to cause a modeled increase in ambient ozone levels over an entire region. For example, the SCAQMD's 2012 AQMP showed that reducing NO_x by 432 tons per day (157,680 tons/year) and reducing VOC by 187 tons per day (68,255 tons/year) would reduce ozone levels at the SCAQMD's monitor site with the highest levels by only 9 parts per billion. (South Coast Air Quality Management District, *Final 2012 AQMP (February 2013)*, http://www.aqmd.gov/home/library/clean-air-plans/air-quality-mgt-plan/final-2012-air-quality-management-plan; then follow "Appendix V: Modeling & Attainment Demonstrations" hyperlink,

[5] See discussion of types of pollutants, supra, Part I.A.

pp. v-4-2, v-7-4, v-7-24.) SCAQMD staff does not currently know of a way to accurately quantify ozone-related health impacts caused by NO_x or VOC emissions from relatively small projects.

On the other hand, this type of analysis may be feasible for projects on a regional scale with very high emissions of NO_x and VOCs, where impacts are regional. For example, in 2011 the SCAQMD performed a health impact analysis in its CEQA document for proposed Rule 1315, which authorized various newly-permitted sources to use offsets from the districts "internal bank" of emission reductions. This CEQA analysis accounted for essentially *all* the increases in emissions due to new or modified sources in the District between 2010 and 2030.[6] The SCAQMD was able to correlate this very large emissions increase (e.g., 6,620 pounds per day NO_x (1,208 tons per year), 89,180 pounds per day VOC (16,275 tons per year)) to expected health outcomes from ozone and particulate matter (e.g., 20 premature deaths per year and 89,947 school absences in the year 2030 due to ozone).[7] (SCAQMD Governing Board Agenda, February 4, 2011, Agenda Item 26, *Assessment for: Re-adoption of Proposed Rule 1315 – Federal New Source Review Tracking System* (see hyperlink in fn 6*)* at p. 4.1-35, Table 4.1-29.)

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[6] (SCAQMD Governing Board Agenda, February 4, 2011, Agenda Item 26, Attachment G, *Assessment for: Re-adoption of Proposed Rule 1315 – Federal New Source Review Tracking System, Vol. 1, p.4.0-6,* http://www.aqmd.gov/home/library/meeting-agendas-minutes/agenda?title=governing-board-meeting-agenda-february-4-2011; the follow "26. Adopt Proposed Rule 1315 – Federal New Source Review Tracking System" (last visited April 1, 2015).)

[7] The SCAQMD was able to establish the location of future NO_x and VOC emissions by assuming that new projects would be built in the same locations and proportions as existing stationary sources. This CEQA document was upheld by the Los Angeles County Superior Court in *Natural Res. Def. Council v SCAQMD*, Los Angeles Superior Court No. BS110792).

However, a project emitting only 10 tons per year of NO_x or VOC is small enough that its regional impact on ambient ozone levels may not be detected in the regional air quality models that are currently used to determine ozone levels. Thus, in this case it would not be feasible to directly correlate project emissions of VOC or NO_x with specific health impacts from ozone. This is in part because ozone formation is not linearly related to emissions. Ozone impacts vary depending on the location of the emissions, the location of other precursor emissions, meteorology and seasonal impacts, and because ozone is formed some time later and downwind from the actual emission. (EPA Guideline on Ozone Monitoring Site Selection (Aug. 1998) EPA-454/R-98-002, § 5.1.2; https://www.epa.gov/ttnamti1/archive/cpreldoc.html; then search "Guideline on Ozone Monitoring Site Selection" click on pdf) (last viewed Apr. 1, 2015).)

SCAQMD has set its CEQA "significance" threshold for NO_x and VOC at 10 tons per year (expressed as 55 lb/day). (SCAQMD, *Air Quality Analysis Handbook*, http://www.aqmd.gov/home/regulations/ceqa/air-quality-analysis-handbook; then follow "SCAQMD Air Quality Significance Thresholds" hyperlink (last visited Apr. 1, 2015).) This is because the federal Clean Air Act defines a "major" stationary source for "extreme" ozone nonattainment areas such as SCAQMD as one emitting 10 tons/year. (42 U.S.C. §§ 7511a(e), 7511a(f); CAA §§ 182(e), 182(f).) Under the Clean Air Act, such sources are subject to enhanced control requirements (42 U.S.C. §§ 7502(c)(5), 7503; CAA §§ 172(c)(5), 173), so SCAQMD decided this was an appropriate threshold for making a CEQA "significance" finding and requiring feasible mitigation. Essentially, SCAQMD takes the position that a source that emits 10 tons/year of NO_x or VOC would contribute cumulatively to ozone formation. Therefore, lead agencies that use SCAQMD's thresholds of significance may determine

that many projects have "significant" air quality impacts and must apply all feasible mitigation measures, yet will not be able to precisely correlate the project to quantifiable health impacts, unless the emissions are sufficiently high to use a regional modeling program.

In the case of particulate matter ($PM_{2.5}$)[8], another "criteria" pollutant, SCAQMD staff is aware of two possible methods of analysis. SCAQMD used regional modeling to predict expected health impacts from its proposed Rule 1315, as mentioned above. Also, the California Air Resources Board (CARB) has developed a methodology that can predict expected mortality (premature deaths) from large amounts of $PM_{2.5}$. (California Air Resources Board, *Health Impacts Analysis: PM Premature Death Relationship*, http://www.arb.ca.gov/research/health/pm-mort/pm-mort_arch.htm (last reviewed Jan. 19, 2012).) SCAQMD used the CARB methodology to predict impacts from three very large power plants (e.g., 731-1837 lbs/day). (Final Environmental Assessment for Rule 1315, *supra*, pp 4.0-12, 4.1-13, 4.1-37 (e.g., 125 premature deaths in the entire SCAQMD in 2030), 4.1-39 (0.05 to 1.77 annual premature deaths from power plants.) Again, this project involved large amounts of additional $PM_{2.5}$ in the District, up to 2.82 tons/day (5,650 lbs/day of $PM_{2.5}$, or, or 1029 tons/year. (*Id.* at table 4.1-4, p. 4.1-10.)

However, the primary author of the CARB methodology has reported that this $PM_{2.5}$ health impact methodology is not suited for small projects and may yield unreliable results due to various uncertainties. [9] (SCAQMD, *Final Subsequent Mitigated Negative Declaration for: Warren*

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[8] SCAQMD has not attained the latest annual or 24-hour national ambient air quality standards for "$PM_{2.5}$" or particulate matter less than 2.5 microns in diameter.

[9] Among these uncertainties are the representativeness of the population used in the methodology, and the specific source of PM and the corresponding health impacts. (*Id.* at p. 2-24.)

E&P, Inc. WTU Central Facility, New Equipment Project (certified July 19, 2011), http://www.aqmd.gov/home/library/documents-support-material/lead-agency-permit-projects/permit-project-documents---year-2011; then follow "Final Subsequent Mitigated Negative Declaration for Warren E&P Inc. WTU Central Facility, New Equipment Project" hyperlink, pp. 2-22, 2-23 (last visited Apr. 1, 2015).) Therefore, when SCAQMD prepared a CEQA document for the expansion of an existing oil production facility, with very small $PM_{2.5}$ increases (3.8 lb/day) and a very small affected population, staff elected not to use the CARB methodology for using estimated $PM_{2.5}$ emissions to derive a projected premature mortality number and explained why it would be inappropriate to do so. (*Id.* at pp 2-22 to 2-24.) SCAQMD staff concluded that use of this methodology for such a small source could result in unreliable findings and would not provide meaningful information. (*Id.* at pp. 2-23, 2-25.) This CEQA document was not challenged in court.

In the above case, while it may have been technically possible to plug the data into the methodology, the results would not have been reliable or meaningful. SCAQMD believes that an agency should not be required to perform analyses that do not produce reliable or meaningful results. This Court has already held that an agency may decline to use even the "normal" "existing conditions" CEQA baseline where to do so would be misleading or without informational value. (*Neighbors for Smart Rail v. Exposition Metro Line* (2013) 57 Cal.4th 439, 448, 457.) The same should be true for a decision that a particular study or analysis would not provide reliable or meaningful results.[10]

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[10] Whether a particular study would result in "informational value" is a part of deciding whether it is "feasible." CEQA defines "feasible" as "capable of being accomplished in a successful manner within a reasonable period of time, taking into account economic, environmental, social, and

Therefore, it is not possible to set a hard-and-fast rule on whether a correlation of air quality impacts with specific quantifiable health impacts is required in all cases. Instead, the result turns on whether such an analysis is reasonably feasible in the particular case.[11] Moreover, what is reasonably feasible may change over time as scientists and regulatory agencies continually seek to improve their ability to predict health impacts. For example, CARB staff has been directed by its Governing Board to reassess and improve the methodology for estimating premature deaths. (California Air Resources Board, *Health Impacts Analysis: PM Mortality Relationship*, http://www.arb.ca.gov/research/health/pm-mort/pm-mort.htm (last reviewed Dec. 29, 2010).) This factor also counsels against setting any hard-and-fast rule in this case.

III. THE QUESTION OF WHETHER AN EIR CONTAINS SUFFICIENT ANALYSIS TO MEET CEQA'S REQUIREMENTS IS A MIXED QUESTION OF FACT AND LAW GOVERNED BY TWO DIFFERENT STANDARDS OF REVIEW.

A. Standard of Review for Feasibility Determination and Sufficiency as an Informative Document

A second issue in this case is whether courts should review an EIR's informational sufficiency under the "substantial evidence" test as argued by Friant Ranch or the "independent judgment" test as argued by Sierra Club.

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technological factors." (Pub. Resources Code § 21061.1.) A study cannot be "accomplished in a *successful* manner" if it produces unreliable or misleading results.

[11] In this case, the lead agency did not have an opportunity to determine whether the requested analysis was feasible because the comment was non-specific. Therefore, SCAQMD suggests that this Court, after resolving the legal issues in the case, direct the Court of Appeal to remand the case to the lead agency for a determination of whether the requested analysis is feasible. Because Fresno County, the lead agency, did not seek review in this Court, it seems likely that the County has concluded that at least some level of correlation of air pollution with health impacts is feasible.

As this Court has explained, "a reviewing court must adjust its scrutiny to the nature of the alleged defect, depending on whether the claim is predominantly one of improper procedure or a dispute over the facts." (*Vineyard Area Citizens v. City of Rancho Cordova, supra,* 40 Cal.4th at 435.) For questions regarding compliance with proper procedure or other legal questions, courts review an agency's action de novo under the "independent judgment" test. (*Id.*) On the other hand, courts review factual disputes only for substantial evidence, thereby "accord[ing] greater deference to the agency's substantive factual conclusions." (*Id.*)

Here, Friant Ranch and Sierra Club agree that the case involves the question of whether an EIR includes sufficient information regarding a project's impacts. However, they disagree on the proper standard of review for answering this question: Sierra Club contends that courts use the independent judgment standard to determine whether an EIR's analysis is sufficient to meet CEQA's informational purposes,[12] while Friant Ranch contends that the substantial evidence standard applies to this question.

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[12] Sierra Club acknowledges that courts use the substantial evidence standard when reviewing predicate factual issues, but argues that courts ultimately decide as a matter of law what CEQA requires. (Answering Brief, pp. 14, 23.)

SCAQMD submits that the issue is more nuanced than either party contends. We submit that, whether a CEQA document includes sufficient analysis to satisfy CEQA's informational mandates is a mixed question of fact and law,[13] containing two levels of inquiry that should be judged by different standards.[14]

The state CEQA Guidelines set forth standards for the adequacy of environmental analysis. Guidelines Section 15151 states:

> An EIR should be prepared with a sufficient degree of analysis to provide decision makers with information which enables them to make a decision which intelligently takes account of environmental consequences. An evaluation of the environmental effects of a proposed project need not be exhaustive, but the sufficiency of an EIR is to be reviewed in light of what is reasonably feasible. Disagreement among experts does not make an EIR inadequate, but the EIR should summarize the main points of disagreement among the experts. The courts have looked not for perfection, but for adequacy, completeness, and a good-faith effort at full disclosure.

In this case, the basic question is whether the underlying analysis of air quality impacts made the EIR "sufficient" as an informative document. However, whether the EIR's analysis was sufficient is judged in light of what was reasonably feasible. This represents a mixed question of fact and law that is governed by two different standards of review.

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[13] Friant Ranch actually states that the claim that an EIR lacks sufficient relevant information is, "most properly thought of as raising mixed questions of fact and law." (Opening Brief, p. 27.) However, the remainder of its argument claims that the court should apply the substantial evidence standard of review to all aspects of the issue.

[14] Mixed questions of fact and law issues may implicate predominantly factual subordinate questions that are reviewed under the substantial evidence test even though the ultimate question may be reviewed by the independent judgment test. *Crocker National Bank v. City and County of San Francisco* (1989) 49 Cal.3d 881, 888-889.

SCAQMD submits that an EIR's sufficiency as an informational document is ultimately a legal question that courts should determine using their independent judgment. This Court's language in *Laurel Heights I* supports this position. As this Court explained: "The court does not pass upon the correctness of the EIR's environmental conclusions, but only upon its *sufficiency as an informative document.*" (*Laurel Heights I, supra,* 47 Cal.3d at 392-393) (emphasis added.) As described above, the Court in *Vineyard Area Citizens v. City of Rancho Cordova, supra,* 40 Cal.4th at 431, also used its independent judgment to determine what level of analysis CEQA requires for water supply impacts. The Court did not defer to the lead agency's opinion regarding the law's requirements; rather, it determined for itself what level of analysis was necessary to meet "[t]he law's informational demands." (*Id.* at p. 432.) Further, existing case law also holds that where an agency fails to comply with CEQA's information disclosure requirements, the agency has "failed to proceed in the manner required by law." (*Save Our Peninsula Comm. v. Monterey County Bd. of Supervisors* (2001) 87 Cal.App.4th 99, 118.)

However, whether an EIR satisfies CEQA's requirements depends in part on whether it was reasonably feasible for an agency to conduct additional or more thorough analysis. EIRs must contain "a detailed statement" of a project's impacts (Pub. Res. Code § 21061), and an agency must "use its best efforts to find out and disclose all that it reasonably can." (CEQA Guidelines § 15144.) Nevertheless, "the sufficiency of an EIR is to be reviewed in light of what is reasonably feasible." (CEQA Guidelines § 15151.)

SCAQMD submits that the question of whether additional analysis or a particular study suggested by a commenter is "feasible" is generally a question of fact. Courts have already held that whether a particular alternative is "feasible" is reviewed by the substantial evidence test.

(*Uphold Our Heritage v. Town of Woodside* (2007) 147 Cal.App.4th 587, 598-99; *Center for Biological Diversity v. County of San Bernardino* (2010) 185 Cal.App.4th 866, 883.) Thus, if a lead agency determines that a particular study or analysis is infeasible, that decision should generally be judged by the substantial evidence standard. However, SCAQMD urges this Court to hold that lead agencies must explain the basis of any determination that a particular analysis is infeasible in the EIR itself. An EIR must discuss information, including issues related to the feasibility of particular analyses "in sufficient detail to enable meaningful participation and criticism by the public. '[W]hatever is required to be considered in an EIR must be in that formal report; what any official might have known from other writings or oral presentations cannot supply what is lacking in the report.'" (*Laurel Heights I, supra*, 47 Cal.3d at p. 405 (quoting *Santiago County Water District v. County of Orange* (1981) 118 Cal.App.3d 818, 831) (discussing analysis of alternatives).) The evidence on which the determination is based should also be summarized in the EIR itself, with appropriate citations to reference materials if necessary. Otherwise commenting agencies such as SCAQMD would be forced to guess where the lead agency's evidence might be located, thus thwarting effective public participation.

Moreover, if a lead agency determines that a particular study or analysis would not result in reliable or useful information and for that reason is not feasible, that determination should be judged by the substantial evidence test. (See *Neighbors for Smart Rail v. Exposition Metro Line Construction Authority*, *supra*, 57 Cal.4th 439, 448, 457:

whether "existing conditions" baseline would be misleading or uninformative judged by substantial evidence standard.[15])

If the lead agency's determination that a particular analysis or study is not feasible is supported by substantial evidence, then the agency has not violated CEQA's information disclosure provisions, since it would be infeasible to provide additional information. This Court's decisions provide precedent for such a result. For example, this Court determined that the issue of whether the EIR should have included a more detailed discussion of future herbicide use was resolved because substantial evidence supported the agency's finding that "the precise parameters of future herbicide use could not be predicted." *Ebbetts Pass Forest Watch v. California Dept. of Forestry & Fire Protection* (2008) 43 Cal.4th 936, 955.

Of course, SCAQMD expects that courts will continue to hold lead agencies to their obligations to consult with, and not to ignore or misrepresent, the views of sister agencies having special expertise in the area of air quality. (*Berkeley Keep Jets Over the Bay v. Board of Port Commissioners* (2007) 91 Cal.App.4th 1344, 1364 n.11.) In some cases, information provided by such expert agencies may establish that the purported evidence relied on by the lead agency is not in fact "substantial". (*Id.* at pp. 1369-1371.)

In sum, courts retain ultimate responsibility to determine what CEQA requires. However, the law does not require exhaustive analysis, but only what is reasonably feasible. Agencies deserve deference for their factual determinations regarding what type of analysis is reasonably feasible. On the other hand, if a commenter requests more information, and the lead agency declines to provide it but does *not* determine that the

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[15] The substantial evidence standard recognizes that the courts "have neither the resources nor the scientific expertise" to weigh conflicting evidence on technical issues. (*Laurel Heights I, supra,* 47 Cal.3d 376, 393.)

requested study or analysis would be infeasible, misleading or uninformative, the question becomes whether the omission of that analysis renders the EIR inadequate to satisfy CEQA's informational purposes. (*Id.* at pp. 1370-71.) Again, this is predominantly a question of law and should be judged by the de novo or independent judgment standard of review. Of course, this Court has recognized that a "project opponent or reviewing court can always imagine some additional study or analysis that might provide helpful information. It is not for them to design the EIR. That further study...might be helpful does not make it necessary." (*Laurel Heights I, supra,* 47 Cal.3d 376, 415 – see also CEQA Guidelines § 15204(a) [CEQA "does not require a lead agency to conduct every test. . . recommended or demanded by commenters."].) Courts, then, must adjudicate whether an omission of particular information renders an EIR inadequate to serve CEQA's informational purposes.[16]

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[16] We recognize that there is case law stating that the substantial evidence standard applies to "challenges to the scope of an EIR's analysis of a topic" as well as the methodology used and the accuracy of the data relied on in the document "because these types of challenges involve factual questions." (*Bakersfield Citizens for Local Control v. City of Bakersfield, supra,* 124 Cal.App.4th 1184, 1198, and cases relied on therein.) However, we interpret this language to refer to situations where the question of the scope of the analysis really is factual—that is, where it involves whether further analysis is feasible, as discussed above. This interpretation is supported by the fact that the *Bakersfield* court expressly rejected an argument that a claimed "omission of information from the EIR should be treated as inquiries whether there is substantial evidence supporting the decision approving the project. " *Bakersfield, supra,* 124 Cal.App.4th at p. 1208. And the *Bakersfield* court ultimately decided that the lead agency must analyze the connection between the identified air pollution impacts and resulting health impacts, even though the EIR already included some discussion of air-pollution-related respiratory illnesses. *Bakersfield, supra,* 124 Cal.App.4th at p. 1220. Therefore, the court must not have interpreted this question as one of the "scope of the analysis" to be judged by the substantial evidence standard.

B. Friant Ranch's Rationale for Rejecting the Independent Judgment Standard of Review is Unsupported by Case Law.

In its brief, Friant Ranch makes a distinction between cases where a required CEQA topic is not discussed at all (to be reviewed by independent judgment as a failure to proceed in the manner required by law) and cases where a topic is discussed, but the commenter claims the information provided is insufficient (to be judged by the substantial evidence test). (Opening Brief, pp. 13-17.) The Court of Appeal recognized these two types of cases, but concluded that both raised questions of law. (*Sierra Club v. County of Fresno* (2014) 226 Cal.App.4th 704 (superseded by grant of review) 172 Cal.Rptr.3d 271, 290.) We believe the distinction drawn by Friant Ranch is unduly narrow, and inconsistent with cases which have concluded that CEQA documents are insufficient. In many instances, CEQA's requirements are stated broadly, and the courts must interpret the law to determine what level of analysis satisfies CEQA's mandate for providing meaningful information, even though the EIR discusses the issue to some extent.

For example, the CEQA Guidelines require discussion of the existing environmental baseline. In *County of Amador v. El Dorado County Water Agency* (1999) 76 Cal.App.4th 931, 954-955, the lead agency had discussed the environmental baseline by describing historic month-end water levels in the affected lakes. However, the court held that this was not an adequate baseline discussion because it failed to discuss the timing and amounts of past actual water releases, to allow comparison with the proposed project. The court evidently applied the independent judgment test to its decision, even though the agency discussed the issue to some extent.

Likewise, in *Vineyard Area Citizens* (2007) 40 Cal.4th 412, this Court addressed the question of whether an EIR's analysis of water supply impacts complied with CEQA. The parties agreed that the EIR was required to analyze the effects of providing water to the development project, "and that in order to do so the EIR had, in some manner, to identify the planned sources of that water." (*Vineyard Area Citizens, supra,* at p. 428.) However, the parties disagreed as to the level of detail required for this analysis and "what level of uncertainty regarding the availability of water supplies can be tolerated in an EIR . . . ." (*Id.*) In other words, the EIR had analyzed water supply impacts for the project, but the petitioner claimed that the analysis was insufficient.

This Court noted that neither CEQA's statutory language or the CEQA Guidelines specifically addressed the question of how precisely an EIR must discuss water supply impacts. (*Id.*) However, it explained that CEQA "states that '[w]hile foreseeing the unforeseeable is not possible, an agency must use its best efforts to find out and disclose all that it reasonably can.'" (*Id.*, [Guidelines § 15144].) The Court used this general principle, along with prior precedent, to elucidate four "principles for analytical adequacy" that are necessary in order to satisfy "CEQA's informational purposes." (*Vineyard Area Citizens, supra,* at p. 430.) The Court did not defer to the agency's determination that the EIR's analysis of water supply impacts was sufficient. Rather, this Court used its independent judgment to determine for itself the level of analysis required to satisfy CEQA's fundamental purposes. (*Vineyard Area Citizens, supra*, at p. 441: an EIR does not serve its purposes where it neglects to explain likely sources of water and "... leaves long term water supply considerations to later stages of the project.")

Similarly, the CEQA Guidelines require an analysis of noise impacts of the project. (Appendix G, "Environmental Checklist Form."[17]) In *Gray v. County of Madera* (2008) 167 Cal.App.4th 1099, 1123, the court held that the lead agency's noise impact analysis was inadequate even though it had addressed the issue and concluded that the increase would not be noticeable. If the court had been using the substantial evidence standard, it likely would have upheld this discussion.

Therefore, we do not agree that the issue can be resolved on the basis suggested by Friant Ranch, which would apply the substantial evidence standard to *every* challenge to an analysis that addresses a required CEQA topic. This interpretation would subvert the courts' proper role in interpreting CEQA and determining what the law requires.

Nor do we agree that the Court of Appeal in this case violated CEQA's prohibition on courts interpreting its provisions "in a manner which imposes procedural or substantive requirements beyond those explicitly stated in this division or in the state guidelines." (Pub. Resources Code § 21083.1.) CEQA requires an EIR to describe *all* significant impacts of the project on the environment. (Pub. Resources Code § 21100(b)(2); *Vineyard Area Citizens, supra,* at p. 428.) Human beings are part of the environment, so CEQA requires EIRs to discuss a project's significant impacts on human health. However, except in certain particular circumstances,[18] neither the CEQA statute nor Guidelines specify the precise level of analysis that agencies must undertake to satisfy the law's requirements. (see, e.g., CEQA Guidelines § 15126.2(a) [EIRs must describe "health and safety problems caused by {a project's} physical changes"].) Accordingly, courts must interpret CEQA as a whole to

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[17] Association of Environmental Professionals, 2015 CEQA Statute and Guidelines (2015) p.287.

[18] E.g., Pub. Resources Code § 21151.8(C)(3)(B)(iii) (requiring specific type of health risk analysis for siting schools).

determine whether a particular EIR is sufficient as an informational document. A court determining whether an EIR's discussion of human health impacts is legally sufficient does not constitute imposing a new substantive requirement.[19] Under Friant Ranch's theory, the above-referenced cases holding a CEQA analysis inadequate would have violated the law. This is not a reasonable interpretation.

IV. COURTS MUST SCRUPULOUSLY ENFORCE THE REQUIREMENTS THAT LEAD AGENCIES CONSULT WITH AND OBTAIN COMMENTS FROM AIR DISTRICTS

Courts must "scrupulously enforce" CEQA's legislatively mandated requirements. (*Vineyard Area Citizens, supra*, 40 Cal.4th 412, 435.) Case law has firmly established that lead agencies must consult with the relevant air pollution control district before conducting an initial study, and must provide the districts with notice of the intention to adopt a negative declaration (or EIR). (*Schenck v. County of Sonoma* (2011) 198 Cal.App.4th 949, 958.) As *Schenck* held, neither publishing the notice nor providing it to the State Clearinghouse was a sufficient substitute for sending notice directly to the air district. (*Id.*) Rather, courts "must be satisfied that [administrative] agencies have fully complied with the procedural requirements of CEQA, since only in this way can the important public purposes of CEQA be protected from subversion." *Schenck,* 198 Cal.App.4th at p. 959 (citations omitted).[20]

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[19] We submit that Public Resources Code Section 21083.1 was intended to prevent courts from, for example, holding that an agency must analyze economic impacts of a project where there are no resulting environmental impacts (see CEQA Guidelines § 15131) , or imposing new procedural requirements, such as imposing additional public notice requirements not set forth in CEQA or the Guidelines.

[20] Lead agencies must consult air districts, as public agencies with jurisdiction by law over resources affected by the project, *before* releasing an EIR. (Pub. Resources Code §§ 21104(a); 21153.) Moreover, air

Lead agencies should be aware, therefore, that failure to properly seek and consider input from the relevant air district constitutes legal error which may jeopardize their project approvals. For example, the court in *Fall River Wild Trout Foundation v. County of Shasta,* (1999) 70 Cal.App.4th 482, 492 held that the failure to give notice to a trustee agency (Department of Fish and Game) was prejudicial error requiring reversal. The court explained that the lack of notice prevented the Department from providing any response to the CEQA document. (*Id.* at p. 492.) It therefore prevented relevant information from being presented to the lead agency, which was prejudicial error because it precluded informed decision-making. (*Id.*)[21]

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districts should be considered "state agencies" for purposes of the requirement to consult with "trustee agencies" as set forth in Public Resources Code § 20180.3(a). This Court has long ago held that the districts are not mere "local agencies" whose regulations are superseded by those of a state agency regarding matters of statewide concern, but rather have concurrent jurisdiction over such issues. (*Orange County Air Pollution Control District v. Public Util. Com.* (1971) 4 Cal.3d 945, 951, 954.) Since air pollution is a matter of statewide concern, *Id* at 952, air districts should be entitled to trustee agency status in order to ensure that this vital concern is adequately protected during the CEQA process.

[21] In *Schenck,* the court concluded that failure to give notice to the air district was not prejudicial, but this was partly because the trial court had already corrected the error before the case arrived at the Court of Appeal. The trial court issued a writ of mandate requiring the lead agency to give notice to the air district. The air district responded by concurring with the lead agency that air impacts were not significant. (*Schenck,* 198 Cal.App.4th 949, 960.) We disagree with the *Schenck* court that the failure to give notice to the air district would not have been prejudicial (even in the absence of the trial court writ) merely because the lead agency purported to follow the air district's published CEQA guidelines for significance. (*Id.*, 198 Cal.App.4th at p. 960.) In the first place, absent notice to the air district, it is uncertain whether the lead agency properly followed those guidelines. Moreover, it is not realistic to expect that an air district's published guidelines would necessarily fully address all possible air-quality related issues that can arise with a CEQA project, or that those

Similarly, lead agencies must obtain additional information requested by expert agencies, including those with jurisdiction by law, if that information is necessary to determine a project's impacts. (*Sierra Club v. State Bd. Of Forestry* (1994) 7 Cal.4th 1215, 1236-37.) Approving a project without obtaining that information constitutes a failure to proceed in the manner prescribed by CEQA. (*Id.* at p. 1236.)

Moreover, a lead agency can save significant time and money by consulting with the air district early in the process. For example, the lead agency can learn what the air district recommends as an appropriate analysis on the facts of its case, including what kinds of health impacts analysis may be available, and what models are appropriate for use. This saves the lead agency from the need to do its analysis all over again and possibly needing to recirculate the document after errors are corrected, if new significant impacts are identified. (CEQA Guidelines § 15088.5(a).) At the same time, the air district's expert input can help the lead agency properly determine whether another commenter's request for additional analysis or studies is reasonable or feasible. Finally, the air district can provide input on what mitigation measures would be feasible and effective.

Therefore, we suggest that this Court provide guidance to lead agencies reminding them of the importance of consulting with the relevant air districts regarding these issues. Otherwise, their feasibility decisions may be vulnerable to air district evidence that establishes that there is no substantial evidence to support the lead agency decision not to provide specific analysis. (*See Berkeley Keep Jets Over the Bay, supra,* 91 Cal.App.4th 1344, 1369-1371.)

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guidelines would necessarily be continually modified to reflect new developments. Therefore we believe that, had the trial court not already ordered the lead agency to obtain the air district's views, the failure to give notice would have been prejudicial, as in *Fall River, supra,* 70 Cal.App.4th 482, 492.

CONCLUSION

The SCAQMD respectfully requests this Court *not* to establish a hard-and-fast rule concerning whether CEQA requires a lead agency to correlate identified air quality impacts of a project with resulting health outcomes. Moreover, the question of whether an EIR is "sufficient as an informational document" is a mixed question of fact and law containing two levels of inquiry. Whether a particular proposed analysis is feasible is predominantly a question of fact to be judged by the substantial evidence standard of review. Where the requested analysis is feasible, but the lead agency relies on legal or policy reasons not to provide it, the question of whether the EIR is nevertheless sufficient as an informational document is predominantly a question of law to be judged by the independent judgment standard of review.

Respectfully submitted,

DATED: April 3, 2015

SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT
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CERTIFICATE OF WORD COUNT

Pursuant to Rule 8.520(c)(1) of the California Rules of Court, I hereby certify that this brief contains 8,476 words, including footnotes, but excluding the Application, Table of Contents, Table of Authorities, Certificate of Service, this Certificate of Word Count, and signature blocks. I have relied on the word count of the Microsoft Word Vista program used to prepare this Certificate.

DATED: April 3, 2015 Respectfully submitted,

Barbara Baird

Barbara Baird

PROOF OF SERVICE

I am employed in the County of Los Angeles, California. I am over the age of 18 years and not a party to the within action. My business address is 21865 Copley Drive, Diamond Bar, California 91765.

On April 3, 2015 I served true copies of the following document(s) described as **APPLICATION OF THE SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT FOR LEAVE TO FILE BRIEF OF *AMICUS CURIAE* IN SUPPORT OF NEITHER PARTY AND *[PROPOSED]* BRIEF OF *AMICUS CURIAE*** by placing a true copy of the foregoing document(s) in a sealed envelope addressed as set forth on the attached service list as follows:

BY MAIL: I enclosed the document(s) in a sealed envelope or package addressed to the persons at the addresses listed in the Service List and placed the envelope for collection and mailing following our ordinary business practices. I am readily familiar with this District's practice for collection and processing of correspondence for mailing. Under that practice, the correspondence would be deposited with the United States Postal Service, with postage thereon fully prepaid at Diamond Bar, California, in the ordinary course of business. I am aware that on motion of the party served, service is presumed invalid if postal cancellation date or postage meter date is more than one day after date of deposit for mailing in affidavit.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Executed on April 3, 2015 at Diamond Bar, California.

Patricia Anderson

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Attachment 3

Construction HRA

HEALTH RISK ASSESSMENT

Crossroads Hollywood Project

Prepared by:

Eyestone Environmental, LLC

September 2018

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1.0 Executive Summary

1.1 Findings

This report provides an analysis of potential health risk impacts related to the proposed construction of the Crossroads Hollywood Project (Project) in the City of Los Angeles, California. The analysis evaluated the incremental change in health risk concentration exposure from diesel exhaust/diesel particulate matter (DPM) emitted by heavy-duty construction equipment during Project construction. The findings of the analysis are as follows:

- For carcinogenic exposures, the increase in risk is calculated to be 4.7 in one million, which is less than the applicable threshold of 10 in one million for sensitive receptors in close proximity to the Project Site, resulting in a less than significant impact.

- For chronic non-carcinogenic exposures, the increase in the respiratory hazard index was estimated to be less than the applicable threshold of one for sensitive receptors in close proximity to the project site, resulting in a less than significant impact.

2.0 Introduction

The Office of Environmental Health Hazard Assessment (OEHHA) adopted a new version of the Air Toxics Hot Spots Program Guidance Manual for the Preparation of Risk Assessments (Guidance Manual) in March of 2015.[1] The Guidance Manual was developed by OEHHA, in conjunction with the California Air Resources Board (CARB), for use in implementing the Air Toxics "Hot Spots" Program (Health and Safety Code Section 44360 et. seq.). The Air Toxics "Hot Spots" Program requires stationary sources to report the types and quantities of certain substances routinely released into the air. The goals of the Air Toxics "Hot Spots" Program are to collect emission data, to identify facilities having localized impacts, to ascertain health risks, to notify nearby residents of significant risks, and to reduce those significant risks to acceptable levels. CARB acknowledges that the Guidance Manual does not include guidance for CEQA and that it would be "handled by individual [Air Pollution Control] Districts."[2]

The intent in developing the Guidance Manual was to provide health risk assessment (HRA) procedures for use in the Air Toxics Hot Spots Program or for the permitting of new or modified stationary sources. Air districts are to determine which facilities will prepare an HRA based on a prioritization process. The Guidance Manual provides recommendations related to cancer risk evaluation of short-term projects. As discussed in Section 8.2.10 of the Guidance Manual, "[t]he local air pollution control districts sometimes use the risk assessment guidelines for the Hot Spots program in permitting decisions for short-term projects such as construction or waste site remediation." Short-term projects that would require a permitting decision by South Coast Air Quality Management District (SCAQMD) typically would be limited to site remediation (e.g., stationary soil vapor extractors) and would not be applicable to the Project. The new Guidance Manual does not provide specific recommendations for evaluation of short-term use of mobile sources (e.g., heavy-duty diesel construction equipment).

Eyestone Environmental, LLC (Eyestone) coordinated with the SCAQMD to determine whether the SCAQMD had any available guidance on use of the new Guidance

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[1] *Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology, Adoption of Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. March 6, 2015, www.oehha.ca.gov/air/hot_spots/hotspots2015.html.*

[2] *CARB, Risk Management Guidance for Stationary Sources of Air Toxics, July 23, 2015, p. 19, www.arb.ca.gov/toxics/rma/rmgssat.pdf.*

Manual. According to Li Jin Sun, SCAQMD CEQA Program Supervisor, SCAQMD is currently evaluating the new Guidance Manual and they have not developed any recommendations on its use for CEQA analyses for potential construction impacts.[3]

Per SCAQMD's recommendation, consultation with the lead agency was conducted. The *L.A. City CEQA Thresholds Guide* (Thresholds Guide) states that "impacts from toxic air contaminants can occur during either the construction or operational phases of a project. During certain construction activities, potential releases of toxic air contaminants could occur during site remediation activities or during building demolition. Toxic air contaminants may also be released during industrial or manufacturing processes, or other activities that involve the use, storage, processing, or disposal of toxic materials."[4] The Thresholds Guide does not specifically recommend an HRA for short-term DPM emissions from construction activities. The Thresholds Guide also sets forth the following factors for consideration on a case-by-case basis in making a determination of significance with regard to toxic air contaminants: the regulatory framework for the toxic material(s) and process(es) involved; the proximity of the toxic air contaminants to sensitive receptors; the quantity, volume, and toxicity of the contaminants expected to be emitted; the likelihood and potential level of exposure; and the degree to which project design will reduce the risk of exposure. Based on this information, the methodology utilized in the Draft EIR remains consistent with City of Los Angeles guidance for preparation of HRAs.

OEHHA's new Guidance Manual provides Age Sensitivity Factors (ASFs) to account for potential increased sensitivity of early-in-life exposure to carcinogens. A review of relevant guidance was conducted to determine applicability of the use of early life exposure adjustments to identified carcinogens. For risk assessments conducted under the auspices of The Air Toxics "Hot Spots" Information and Assessment Act (AB 2588, Connelly, Statutes of 1987; Health and Safety Code Section 44300 et seq.) a weighting factor is applied to all carcinogens regardless of purported mechanism of action. The use of these factors would not be applicable to this HRA as neither the Lead Agency nor SCAQMD have developed recommendations on whether these factors should be used for CEQA analyses of potential DPM construction impacts. For this assessment, the HRA relied upon United States Environmental Protection Agency (USEPA) guidance relating to the use of early life exposure adjustment factors (Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens, EPA/630/R-003F) whereby adjustment factors are only considered when carcinogens act "through the mutagenic mode of action." The USEPA has identified 19 compounds that elicit a mutagenic mode of action for carcinogenesis. For DPM, polycyclic aromatic hydrocarbons (PAHs) and their derivatives,

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[3] *Lijin Sun., SCAQMD CEQA Program Supervisor, Personal Communication via email, May 16, 2018.*

[4] *City of Los Angeles, CEQA Thresholds Guide, 2006, p. B.3-2.*

which are known to exhibit a mutagenic mode of action, comprise less than one percent of the exhaust particulate mass. To date, the USEPA reports that whole diesel engine exhaust has not been shown to elicit a mutagenic mode of action. Therefore, early life exposure adjustments were not considered in this HRA.

Although a construction HRA is not required per the Thresholds Guide, for informational purposes only, an HRA has been prepared in accordance with current SCAQMD Guidance in response to public comments and to provide the City with additional supporting evidence that the Project would result in a less than significant health risk impact from construction of the Project.

3.0 Health Risk Assessment

This section of the HRA includes a discussion of the assessment process, source identification and characterization, identification of chemicals of concern, risk characterization, and conclusions. As discussed above in Section 2.0, the HRA was conducted in accordance with SCAQMD Guidance and Final-Localized Significance Threshold Methodology (LST Guidelines). [5,6] DPM modeled concentrations were used to calculate cancer risk and chronic hazard index at each relevant receptor. The acute hazard index was not quantified since an inhalation Reference Exposure Level (REL) has not been determined by the OEHHA for DPM.

3.1 The Assessment Process

The risk assessment process is typically described as consisting of four basic steps: (1) hazard identification; (2) exposure assessment; (3) dose-response assessment; and (4) risk characterization. In the first step, hazard identification involves determining the potential health effect which may be associated with emitted pollutants. The purpose is to identify qualitatively whether a pollutant is a potential human carcinogen or is associated with other types of adverse health effects. Depending on the chemical, these health effects may include short-term ailments or chronic diseases. The dose-response assessment is designed to characterize the relationship between the amount or dose of a chemical and its toxicological effect on the human body. Responses to toxic chemicals will vary depending on the amount and length of exposure. For example, short-term exposure to low concentrations of chemicals may produce no noticeable effect, but continued exposure to the same levels of chemicals over a long period of time may eventually cause harm. The purpose of the exposure assessment is to estimate the extent of exposure to each substance for which risk will be evaluated. This involves emission quantification, modeling of environmental transport, identification of chemicals of concern, identification of exposure routes, identification of exposed populations, and estimation of long-term exposure levels. Risk characterization is an integration of the health effects and public exposure information

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[5] *SCAQMD, Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Idling Emissions for CEQA Air Quality Analysis, 2003, www.aqmd.gov/home/regulations/ceqa/air-quality-analysis-handbook/mobile-source-toxics-analysis.*

[6] *SCAQMD, Final-Localized Significance Threshold Methodology, 2008.*

developed for emitted pollutants to provide a quantitative probability of adverse health effects.

3.2 Source Identification and Characterization

3.2.1 Source Identification

As indicated above, the primary source of potential air toxics associated with proposed Project construction is DPM from on-site heavy-duty construction equipment. The SCAQMD recommends that an HRA be conducted for substantial sources of long-term DPM operational sources (e.g., truck stops and warehouse distribution facilities) and has provided guidance for analyzing mobile source diesel emissions.[7] While Project construction would not represent a long-term source of DPM emissions[8], the SCAQMD Guidance was used for purposes of modeling parameters and assumptions.

3.2.2 Source Characterization

As described in detail in Section II, Project Description, of the Draft EIR, Project construction would commence with demolition of the existing buildings (excluding Crossroads of the World) and surface parking lots, followed by grading and excavation for the subterranean parking garages. Building foundations would then be placed, followed by building construction, paving/concrete installation, and landscape installation. Project construction is anticipated to occur over approximately 48 months and be completed in 2022. It is estimated that approximately 643,753 cubic yards (cy) of soil would be hauled from the Project Site during the grading and excavation phase, as well as an additional 1,490 cy during off-site improvements to the existing sanitary sewer system related to the realignment of Las Palmas Avenue.

Total DPM emissions over the duration of Project construction were calculated using the SCAQMD recommended California Emissions Estimator Model (CalEEMod) and consistent with the methodology for calculating criteria pollutant emissions provided in Section IV.C, Air Quality, of the Draft EIR. The calculations of the emissions generated during Project construction activities reflect the types and quantities of construction equipment and haul trucks that would be used to complete the proposed construction activities. As the assumptions used in the air quality analysis were developed to

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[7] *SCAQMD, Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Emissions, August 2003.*

[8] *Project construction is short term—four years. Moreover, the Project is residential, commercial, and open spaces uses, none of which are associated with heavy-duty truck use or significant DPM emissions.*

characterize a worst-case peak day of construction by phase, equipment usage assumptions were modified to reflect average daily use. As an example, the heavy-duty construction equipment mix provided in the air quality analysis for the foundation phase reflects all equipment needed for the largest concrete pour day. Thus, average daily DPM emissions from building foundation would be substantially less since maximum pour days would not occur every day during that phase.

The calculation of DPM emissions was based on the Crossroads Project Construction Annual CalEEMod output file provided in Appendix D, Greenhouse Gas Emissions, of the Draft EIR. An adjustment was made to the regional emissions inventory to reflect on-site activity (diesel off-road equipment and diesel truck travel and idling time. The updated modeling is provided in Appendix A of this HRA.

3.3 Identification of Chemicals of Concern

DPM was evaluated for potential health effects in two categories, carcinogenic and non-carcinogenic. Most regulatory agencies consider carcinogens to pose a risk of cancer at all exposure levels (i.e., a “no-threshold” assumption); that is, any increase in dose is assumed to be associated with an increase in the probability of developing cancer. In contrast, non-carcinogens generally are thought to produce adverse health effects only when some minimum exposure level is reached (i.e., a threshold).

3.4 Exposure Quantification

Consistent with SCAQMD’s Localized Significance Threshold (LST) Methodology, this HRA used USEPA’s Regulatory Model AERMOD to assess the downwind extent of DPM concentrations from proposed construction activities. AERMOD accounts for a variety of refined, site-specific conditions that facilitate an accurate assessment of Project impacts. AERMOD’s air dispersion algorithms are based upon a planetary boundary layer turbulence structure and scaling concepts, including the treatment of surface and elevated sources in simple and complex terrain.

Exhaust emissions from construction equipment were treated as a set of side-by-side elevated volume sources. The release height was assumed to be five meters. This represents the mid-range of the expected plume rise from frequently used construction equipment during daytime atmospheric conditions. All construction exhaust emissions were assumed to take place over a four year duration on weekdays between 7 A.M. to 5 P.M. (12-hour period).

Air dispersion models require additional input parameters including local meteorology and receptors. Due to the sensitivity to individual meteorological parameters

such as wind speed and direction, the USEPA recommends that meteorological data used as input into dispersion models be selected on the basis of relative spatial and temporal conditions that exist in the area of concern. In response to this recommendation, meteorological data from the SCAQMD Downtown Los Angeles monitoring station (Source Receptor Area 1) were used to represent local weather conditions and prevailing winds.

Cartesian receptor grids were used to represent adjacent and nearby sensitive land uses. The Cartesian receptor grids were placed at each sensitive use with a built in 10 meter spacing. All receptors were placed within the breathing zone at ground level, which is recommended by SCAQMD for AERMOD modeling. Elevations for both sources and receptors were provided by the U.S. Geological Survey (USGS) and included using the AERMOD terrain processor AERMAP.

A graphical representation of the source-receptor grid network is presented in Appendix C.

3.5 Risk Characterization

3.5.1 Carcinogenic Chemical Risk

As discussed above, carcinogenic compounds are not considered to have threshold levels (i.e., dose levels below which there are no risks). Any exposure, therefore, will have some associated risk. Health risks associated with exposure to carcinogenic compounds at sensitive land uses in close proximity to the proposed Project can be defined in terms of the probability of developing cancer as a result of exposure to a chemical at a given concentration. Under a deterministic approach (i.e., point estimate methodology), the cancer risk probability is determined by multiplying the chemical's annual concentration by its unit risk factor (URF). The URF is a measure of the carcinogenic potential of a chemical when a dose is received through the inhalation pathway. It represents an upper bound estimate of the probability of contracting cancer as a result of continuous exposure to an ambient concentration of one microgram per cubic meter ($\mu g/m^3$) over a 70-year lifetime.

The equation used to calculate the potential excess cancer risk is:

$$Risk_i = C_i \times CP_i \times DBR \times EVF$$

Where:

$Risk_i$ = Lifetime Excess Cancer Risk from exposure to $chemical_i$
C_i = Representative Air Concentration for $chemical_i$ ($\mu g/m^3$)
CP_i = Cancer $Potency_i$ $(mg/kg\text{-}day)^{-1}$

DBR = Daily Breathing Rate (L/kg body weight-day)
EVF = Exposure Value Factor (unitless)

An estimate of an individual's incremental excess cancer risk from exposure to Project construction DPM emissions is calculated by summing the chemical-specific excess cancer risks.

3.5.2 Non-Carcinogenic Chemical Risk

The potential for chronic non-carcinogenic health effects is evaluated by calculating the total hazard index (HI) for the Project construction DPM emissions. This HI represents the sum of the hazard quotients (HQs) developed for each individual project-related chemical, where a HQ is the ratio of the representative air concentration of the chemical to the chemical specific non-cancer REL. The non-cancer RELs represent the daily average exposure concentration at (or below) which no adverse health effects are anticipated. The equations used to calculate the chemical-specific HQs and HIs are:

$HQ_i = C_i/REL_i$
$HI = \Sigma HQi$

Where:

HQ_i = Hazard Quotient for $chemical_i$
C_i = Average Daily Air Concentration for $chemical_i$ ($\mu g/m^3$)
REL_i = Noncancer Reference Exposure Level for $chemical_i$ ($\mu g/m^3$)
HI = Hazard Index

3.6 Conclusions

The results from the health risk calculations provide an estimate of the potential risks and hazards to individuals through inhalation of Project construction DPM emissions over a four year duration. The estimated risks and hazards include: lifetime excess cancer risk estimates, and cumulative chronic HI estimates for the receptor locations of concern.

As shown in Appendix B, the results of the HRA yields a maximum off-site individual cancer risk of 4.7 in a million at the residences located south-east of the Project site. The maximum chronic risk of 0.041 occurs within this same residential area. Cancer risk at the school receptor would be 2.0 in a million. Chronic risk at the school receptor would be 0.025. As the Project would not emit carcinogenic or toxic air contaminants that result in impacts which exceed the maximum individual cancer risk of ten in one million or the chronic index of 1.0, Project-related toxic emission impacts would be less than significant.

4.0 Uncertainty Assessment

Evaluating carcinogenic pollutant concentrations based on OEHHA methodology and SCAQMD Guidance has an implied uncertainty. These methodologies were developed to provide a conservative health risk estimate. The conservative nature of this methodology relies on a number of inputs designed to prevent an underestimation of risk. The following discusses the conservative nature of the risk assessment analysis assumptions utilized in this analysis.

The cancer risk from DPM occurs mainly through inhalation. Output from the dispersion analysis was used to estimate the DPM concentrations. The cancer risk estimate is then calculated based on those estimated DPM concentrations using the risk methodology promulgated by OEHHA. The risk assessment guidelines established by SCAQMD and included in the analysis are designed to produce conservative (high) estimates of the risk posed by DPM, due to the following factors:

- As a conservative measure, the SCAQMD does not recognize indoor adjustments for residential uses. However, studies have shown that the typical person spends approximately 87 percent of their time indoors, 5 percent of their time outdoors, and 7 percent of their time in vehicles. A DPM exposure assessment showed that an average indoor concentration was 2.0 $\mu g/m^3$, compared with an outdoor concentration of 3.0 $\mu g/m^3$.[9]

- OEHHA has a toxicity database that lists TACs and their URFs. A URF describes the cancer potency of a particular TAC and is used to estimate cancer risk.[4] Most of these URFs are extrapolated from animal studies based on continuous exposure to particular toxin. This method can have some significant uncertainties. For example, a chemical that is carcinogenic by one route of exposure is considered to be carcinogenic for all routes of exposure at its maximum potency. Also, it is not realistic for a receptor to be exposed to a continuous concentration of TACs over time. In reality, receptors are exposed to constantly changing concentration levels that would expose receptors to lower levels of TACs over time than analyzed in this analysis.

- The use of the SCAQMD meteorological data set and conservative exposure assumptions (e.g., assumes receptor would be located outside in the same

[9] *South Coast Air Quality Management District (SCAQMD), Health Risk Assessment Guidance for Analyzing Cancer Risks from Mobile Source Diesel Emissions, 2002.*

location 24 hours per day for the entire construction duration) amongst others, likely also lead to overestimated risks.

As such, uncertainty in the health risk analysis is conservative in nature and is designed to prevent any undisclosed impacts to human health. Concentrations reported in this report represent a worst-case scenario that is likely an over estimation of actual pollutant concentrations.

Appendix A

Emissions Calculations

Crossroads Project Construction (Onsite DPM Exhaust Emissions)

Los Angeles-South Coast County, Annual

1.0 Project Characteristics

1.1 Land Usage

Land Uses	Size	Metric	Lot Acreage	Floor Surface Area	Population
General Office Building	95.00	1000sqft	2.18	95,000.00	0
Enclosed Parking with Elevator	2,494.00	Space	22.45	1,223,700.00	0
High Turnover (Sit Down Restaurant)	41.60	1000sqft	0.96	41,600.00	0
Hotel	308.00	Room	10.27	348,500.00	0
Quality Restaurant	41.60	1000sqft	0.96	41,600.00	0
Apartments High Rise	760.00	Dwelling Unit	12.26	643,200.00	2174
Condo/Townhouse High Rise	190.00	Dwelling Unit	2.97	160,800.00	543
Strip Mall	61.80	1000sqft	1.42	61,800.00	0
Supermarket	40.00	1000sqft	0.92	40,000.00	0

1.2 Other Project Characteristics

Urbanization	Urban	**Wind Speed (m/s)**	2.2	**Precipitation Freq (Days)**	33
Climate Zone	11			**Operational Year**	2022
Utility Company	Los Angeles Department of Water & Power				
CO2 Intensity (lb/MWhr)	1227.89	**CH4 Intensity (lb/MWhr)**	0.029	**N2O Intensity (lb/MWhr)**	0.006

1.3 User Entered Comments & Non-Default Data

Project Characteristics -

Land Use - Site Specific

Construction Phase - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Off-road Equipment - Site Specific

Trips and VMT - Equivalent to 5 minutes of idle time (5mph or 0.41667 miles per trip) on Project Site multiplied by total trips from Appendix D of the DEIR.

Demolition -

Grading -

Architectural Coating - Low VOC Consistent with SCAQMD Rule 1301.

Vehicle Trips - Project Specific Traffic Study

Vechicle Emission Factors -

Vechicle Emission Factors -

Vechicle Emission Factors -

Woodstoves -

Energy Use -

Construction Off-road Equipment Mitigation - Mitigation Measure B-5

Mobile Land Use Mitigation -

Area Mitigation -

Energy Mitigation -

Water Mitigation -

Waste Mitigation -

On-road Fugitive Dust - Idle Time

Table Name	Column Name	Default Value	New Value
tblArchitecturalCoating	ConstArea_Nonresidential_Exterior	926,100.00	892,808.00
tblArchitecturalCoating	ConstArea_Nonresidential_Interior	2,778,300.00	2,678,424.00
tblArchitecturalCoating	ConstArea_Residential_Exterior	542,700.00	641,250.00
tblArchitecturalCoating	ConstArea_Residential_Interior	1,628,100.00	1,923,750.00
tblArchitecturalCoating	EF_Nonresidential_Exterior	250.00	100.00
tblArchitecturalCoating	EF_Nonresidential_Interior	250.00	50.00
tblAreaCoating	Area_Nonresidential_Interior	2778300	2678424
tblAreaCoating	Area_Residential_Exterior	542700	641250
tblAreaCoating	Area_Residential_Interior	1628100	1923750
tblConstEquipMitigation	NumberOfEquipmentMitigated	0.00	2.00
tblConstEquipMitigation	NumberOfEquipmentMitigated	0.00	3.00
tblConstEquipMitigation	NumberOfEquipmentMitigated	0.00	6.00
tblConstEquipMitigation	NumberOfEquipmentMitigated	0.00	4.00
tblConstEquipMitigation	Tier	No Change	Tier 3
tblConstEquipMitigation	Tier	No Change	Tier 3
tblConstEquipMitigation	Tier	No Change	Tier 3
tblConstEquipMitigation	Tier	No Change	Tier 3
tblConstructionPhase	NumDays	75.00	349.00
tblConstructionPhase	NumDays	1,110.00	718.00
tblConstructionPhase	NumDays	70.00	23.00
tblConstructionPhase	NumDays	110.00	107.00
tblConstructionPhase	NumDays	75.00	66.00
tblConstructionPhase	NumDays	40.00	131.00
tblConstructionPhase	PhaseEndDate	2/1/2023	9/30/2021
tblConstructionPhase	PhaseEndDate	6/29/2018	6/30/2018
tblConstructionPhase	PhaseStartDate	10/1/2021	6/1/2020
tblGrading	MaterialExported	0.00	645,243.00
tblLandUse	LandUseSquareFeet	997,600.00	1,223,700.00
tblLandUse	LandUseSquareFeet	447,216.00	348,500.00
tblLandUse	LandUseSquareFeet	760,000.00	643,200.00
tblLandUse	LandUseSquareFeet	190,000.00	160,800.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	3.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	3.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	3.00	2.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	4.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	3.00	4.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	1.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	1.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	0.00

tblOffRoadEquipment	OffRoadEquipmentUnitAmount	3.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	3.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	2.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	4.00	0.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	1.00	3.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	3.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	2.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	1.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	2.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	4.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	2.00
tblOffRoadEquipment	OffRoadEquipmentUnitAmount	0.00	1.00
tblOffRoadEquipment	PhaseName		Demolition
tblOffRoadEquipment	PhaseName		Grading
tblOffRoadEquipment	PhaseName		Demolition
tblOffRoadEquipment	PhaseName		Grading
tblOffRoadEquipment	PhaseName		Grading
tblOffRoadEquipment	PhaseName		Demolition
tblOffRoadEquipment	PhaseName		Demolition
tblOffRoadEquipment	UsageHours	7.00	8.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblOnRoadDust	MeanVehicleSpeed	40.00	5.00
tblProjectCharacteristics	OperationalYear	2014	2022
tblTripsAndVMT	HaulingTripLength	20.00	275.00
tblTripsAndVMT	HaulingTripLength	20.00	19,209.00
tblTripsAndVMT	HaulingTripNumber	1,870.00	1.00
tblTripsAndVMT	HaulingTripNumber	80,655.00	1.00
tblTripsAndVMT	VendorTripLength	6.90	8.33
tblTripsAndVMT	VendorTripLength	6.90	20.83
tblTripsAndVMT	VendorTripLength	6.90	31.25
tblTripsAndVMT	VendorTripLength	6.90	8.33
tblTripsAndVMT	VendorTripNumber	0.00	1.00
tblTripsAndVMT	VendorTripNumber	0.00	1.00
tblTripsAndVMT	VendorTripNumber	405.00	1.00
tblTripsAndVMT	VendorTripNumber	0.00	1.00
tblTripsAndVMT	WorkerTripNumber	30.00	0.00
tblTripsAndVMT	WorkerTripNumber	30.00	0.00
tblTripsAndVMT	WorkerTripNumber	30.00	0.00
tblTripsAndVMT	WorkerTripNumber	1,442.00	0.00
tblTripsAndVMT	WorkerTripNumber	288.00	0.00
tblTripsAndVMT	WorkerTripNumber	23.00	0.00

2.0 Emissions Summary

2.1 Overall Construction

Unmitigated Construction

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Year	tons/yr										MT/yr					
2018						0.2923										
2019						0.4257										
2020						0.3726										
2021						0.2641										
Total						**1.3547**										

Mitigated Construction

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Year	tons/yr										MT/yr					
2018						0.2635										
2019						0.4000										
2020						0.3563										
2021						0.2586										
Total						**1.2784**										

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N20	CO2e
Percent Reduction	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**5.63**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**

3.0 Construction Detail

Construction Phase

Phase Number	Phase Name	Phase Type	Start Date	End Date	Num Days Week	Num Days	Phase Description
1	Demolition	Demolition	1/1/2018	1/31/2018	5	23	
2	Grading	Grading	2/1/2018	6/30/2018	5	107	
3	Foundation	Site Preparation	7/1/2018	12/31/2018	5	131	
4	Building Construction	Building Construction	1/1/2019	9/30/2021	5	718	
5	Architectural Coating	Architectural Coating	6/1/2020	9/30/2021	5	349	
6	Paving	Paving	10/1/2021	12/31/2021	5	66	

Acres of Grading (Site Preparation Phase): 0

Acres of Grading (Grading Phase): 0

Acres of Paving: 0

Residential Indoor: 1,923,750; Residential Outdoor: 641,250; Non-Residential Indoor: 2,678,424; Non-Residential Outdoor: 892,808

OffRoad Equipment

Phase Name	Offroad Equipment Type	Amount	Usage Hours	Horse Power	Load Factor

Demolition	Air Compressors	3	8.00	78	0.48
Demolition	Concrete/Industrial Saws	3	8.00	81	0.73
Demolition	Cranes	1	8.00	226	0.29
Demolition	Excavators	2	8.00	162	0.38
Demolition	Rubber Tired Dozers	0	8.00	255	0.40
Demolition	Rubber Tired Loaders	2	8.00	199	0.36
Demolition	Welders	1	8.00	46	0.45
Grading	Bore/Drill Rigs	2	8.00	205	0.50
Grading	Cranes	2	8.00	226	0.29
Grading	Excavators	4	8.00	162	0.38
Grading	Graders	0	8.00	174	0.41
Grading	Pumps	4	8.00	84	0.74
Grading	Rubber Tired Dozers	0	8.00	255	0.40
Grading	Scrapers	0	8.00	361	0.48
Grading	Tractors/Loaders/Backhoes	0	8.00	97	0.37
Foundation	Cement and Mortar Mixers	2	8.00	9	0.56
Foundation	Concrete/Industrial Saws	2	8.00	81	0.73
Foundation	Cranes	2	8.00	226	0.29
Foundation	Plate Compactors	2	8.00	8	0.43
Foundation	Pumps	2	8.00	84	0.74
Foundation	Rubber Tired Dozers	0	8.00	255	0.40
Foundation	Tractors/Loaders/Backhoes	0	8.00	97	0.37
Foundation	Welders	2	8.00	46	0.45
Building Construction	Aerial Lifts	4	8.00	62	0.31
Building Construction	Air Compressors	4	8.00	78	0.48
Building Construction	Cement and Mortar Mixers	3	8.00	9	0.56
Building Construction	Concrete/Industrial Saws	1	8.00	81	0.73
Building Construction	Cranes	3	8.00	226	0.29
Building Construction	Forklifts	4	8.00	89	0.20
Building Construction	Generator Sets	0	8.00	84	0.74
Building Construction	Plate Compactors	3	8.00	8	0.43
Building Construction	Pumps	3	8.00	84	0.74
Building Construction	Tractors/Loaders/Backhoes	0	7.00	97	0.37
Building Construction	Welders	3	8.00	46	0.45
Architectural Coating	Air Compressors	0	6.00	78	0.48
Paving	Cement and Mortar Mixers	2	8.00	9	0.56
Paving	Pavers	0	8.00	125	0.42
Paving	Paving Equipment	1	8.00	130	0.36
Paving	Pumps	1	8.00	84	0.74
Paving	Rollers	1	8.00	80	0.38
Paving	Skid Steer Loaders	2	8.00	64	0.37
Paving	Tractors/Loaders/Backhoes	2	8.00	97	0.37

Trips and VMT

Phase Name	Offroad Equipment Count	Worker Trip Number	Vendor Trip Number	Hauling Trip Number	Worker Trip Length	Vendor Trip Length	Hauling Trip Length	Worker Vehicle Class	Vendor Vehicle Class	Hauling Vehicle Class
Demolition	12	0.00	0.00	1.00	14.70	6.90	275.00	LD_Mix	HDT_Mix	HHDT
Grading	12	0.00	1.00	1.00	14.70	8.33	19,209.00	LD_Mix	HDT_Mix	HHDT
Foundation	12	0.00	1.00	0.00	14.70	20.83	20.00	LD_Mix	HDT_Mix	HHDT
Building Construction	28	0.00	1.00	0.00	14.70	31.25	20.00	LD_Mix	HDT_Mix	HHDT

Architectural Coating	0	0.00	0.00	0.00	14.70	6.90	20.00	LD_Mix	HDT_Mix	HHDT
Paving	9	0.00	1.00	0.00	14.70	8.33	20.00	LD_Mix	HDT_Mix	HHDT

3.1 Mitigation Measures Construction

Use Cleaner Engines for Construction Equipment

Water Exposed Area

Reduce Vehicle Speed on Unpaved Roads

Clean Paved Roads

3.2 Demolition - 2018

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Fugitive Dust						0.0000										
Off-Road						0.0284										
Total						**0.0284**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						3.0000e-005										
Vendor						0.0000										
Worker						0.0000										
Total						**3.0000e-005**										

3.3 Grading - 2018

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Fugitive Dust						0.0000										
Off-Road						0.1347										
Total						**0.1347**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						1.8000e-003										
Vendor						7.0000e-005										
Worker						0.0000										
Total						**1.8700e-003**										

Mitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Fugitive Dust					0.0142	0.0000	0.0142									
Off-Road						0.1200	0.1200									
Total					**0.0142**	**0.1200**	**0.1343**									

Mitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						1.8000e-003										
Vendor						7.0000e-005										
Worker						0.0000										
Total						**1.8700e-003**										

3.4 Foundation - 2018

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Fugitive Dust					0.0000	0.0000	0.0000									
Off-Road						0.1271	0.1271									
Total					**0.0000**	**0.1271**	**0.1271**									

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling					0.0000	0.0000	0.0000									
Vendor					1.2100e-003	2.2000e-004	1.4300e-003									
Worker					0.0000	0.0000	0.0000									
Total					**1.2100e-003**	**2.2000e-004**	**1.4300e-003**									

3.5 Building Construction - 2019

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Off-Road						0.4251										
Total						**0.4251**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling					0.0000	0.0000	0.0000									
Vendor					3.6200e-003	6.3000e-004	4.2400e-003									
Worker					0.0000	0.0000	0.0000									
Total					**3.6200e-003**	**6.3000e-004**	**4.2400e-003**									

3.5 Building Construction - 2020

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Off-Road						0.3720										
Total						**0.3720**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						0.0000										
Vendor						5.8000e-004										
Worker						0.0000										
Total						**5.8000e-004**										

3.5 Building Construction - 2021

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Off-Road						0.2399										
Total						**0.2399**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling					0.0000	0.0000	0.0000									
Vendor					2.7000e-003	3.9000e-004	3.0900e-003									
Worker					0.0000	0.0000	0.0000									
Total					**2.7000e-003**	**3.9000e-004**	**3.0900e-003**									

3.6 Architectural Coating - 2020

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					

Archit. Coating						0.0000										
Off-Road						0.0000										
Total						**0.0000**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						0.0000										
Vendor						0.0000										
Worker						0.0000										
Total						**0.0000**										

3.6 Architectural Coating - 2021

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Archit. Coating						0.0000										
Off-Road						0.0000										
Total						**0.0000**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						0.0000										
Vendor						0.0000										
Worker						0.0000										
Total						**0.0000**										

3.7 Paving - 2021

Unmitigated Construction On-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e

Category	tons/yr										MT/yr					
Off-Road						0.0238										
Paving						0.0000										
Total						**0.0238**										

Unmitigated Construction Off-Site

	ROG	NOx	CO	SO2	Fugitive PM10	Exhaust PM10	PM10 Total	Fugitive PM2.5	Exhaust PM2.5	PM2.5 Total	Bio- CO2	NBio-CO2	Total CO2	CH4	N2O	CO2e
Category	tons/yr										MT/yr					
Hauling						0.0000										
Vendor						4.0000e-005										
Worker						0.0000										
Total						**4.0000e-005**										

Crossroads

Health Risk Assessment - Construction Emissions

CalEEMod Outputs - UNMITIGATED

Year	Exhaust PM10 tons/yr	Scalar	Average Exhaust PM10 tons/yr	lbs/yr	Note:
2018 (Off-Road)	0.2902	0.8	0.23216		
2018 (On-Road)	0.00212	1	0.00212		
2018 (Total)	**0.29232**		**0.23428**	**468.56**	**(Combined Demo, Grading, and Foundation)**
2019 (Off-Road)	0.4251	0.8	0.34008		
2019 (On-Road)	0.00063	0.8	0.000504		
2019 (Total)	**0.42573**		**0.340584**	**681.168**	**(Building Construction)**
2020 (Off-Road)	0.372	0.8	0.2976		
2020 (On-Road)	0.00058	0.8	0.000464		
2020 (Total)	**0.37258**		**0.298064**	**596.128**	**(Building Construction)**
2021 (Off-Road)	0.2637	0.8	0.21096		
2021 (On-Road)	0.00043	0.8	0.000344		
2021 (Total)	**0.26413**		**0.211304**	**422.608**	**(Building Construction and Paving)**

CalEEMod Outputs - MITIGATED

Year	Exhaust PM10 tons/yr	Scalar	Average Exhaust PM10 tons/yr	lbs/yr	
2018	0.2755	0.8	0.2204		
2018	0.00212	1	0.00212		
2018 (Total)	**0.27762**		**0.22252**	**445.04**	**(Combined Demo, Grading, and Foundation)**
2019 (Off-Road)	0.4251	0.8	0.34008		
2019 (On-Road)	0.00063	0.8	0.000504		
2019 (Total)	**0.42573**		**0.340584**	**681.168**	**(Building Construction)**
2020 (Off-Road)	0.372	0.8	0.2976		
2020 (On-Road)	0.00058	0.8	0.000464		
2020 (Total)	**0.37258**		**0.298064**	**596.128**	**(Building Construction)**
2021 (Off-Road)	0.2637	0.8	0.21096		
2021 (On-Road)	0.00043	0.8	0.000344		
2021 (Total)	**0.26413**		**0.211304**	**422.608**	**(Building Construction and Paving)**

Annual diesel particulate matter was calculated as follows:

(1) CalEEMod modeling file (Appendix D-Greenhouse Gas Emissions (Crossroads Project Construction Annual)

(2) Off-Road Equipment (Equipment in the model reflects peak daily activitiy. Therefore, a scalar of 80% was applied to the peak-daily activity to represent average daily activity.

(3) On-road activity from diesel vehicles onsite (5 minutes of idle time per vehicle which is equivalent to a vehicle traveling 0.4167 miles @ 5 mph).

				Equivalent Vehicle Miles Travelled On-Site	
Phase:	**Vendor Trips**	**Haul Trips**	**Trip Distance**	**Vendor**	**Haul**
Demolition:	0	660	0.4167	0.00	275.00
Grading:	20	46100	0.4167	8.33	19208.49
Foundation:	50	0	0.4167	20.83	0.00
Building Construction:	75	0	0.4167	31.25	0.00
Paving:	20	0	0.4167	8.33	0.00

Appendix B

Carcinogenic and Non-Carcinogenic Risk Calculations

Crossroads

Health Risk Calculations - Project Construction

Diesel Particulate Matter Emission Rate Calculation / Scaler

Emission Rate (lbs/year)	536.2
Hours per Day	8
Seconds per Year	10,512,000
Average Annual Emission Rate (g/s)	0.0231
Scaler Concentration (ug/m3)[a]	8.88
Diesel Particulate Concentration (ug/m3)	2.05E-01

[a] Scaler concentration based on a 1 g/s emission rate

Scaler Health Risk Calculations (1 ug/m3)

Source	*Mass GLC*				*Weight Fraction*	*Contaminant*	*Carcinogenic Hazard*		
	(µg/m3)	*DPM Emissions*	*Adjusted Concentration*				*URF*	*CPF*	*RISK*
(a)	*(1 g/s)*	*(g/s)*	*(µg/m3)*	*(mg/m3)*	*(d)*	*(e)*	*(µg/m3)* $^{-1}$ *(f)*	*(mg/kg/day)* $^{-1}$ *(g)*	*(h)*
Construction DPM (Construction Duration)	1.00	1.00000	0.20545	2.1E-04	1.00E+00	Diesel Exhaust Particulate	3.0E-04	1.1E+00	4.65E-06
Total									**4.65E-06**
						DPM Total			**4.65** in a million

Note: Exposure factors used to calculate contaminant intake

exposure frequency (days/year)	350
exposure duration (years)	4.0
inhalation rate (m3/day)	0.393
average body weight (kg)	
averaging time(cancer) (days)	25550
averaging time(noncancer) (days)	14600

Chronic Risk Calculations - DPM

Receptor	**Annual Concentration (ug/m3)**	**Chronic Inhalation REL (ug/m3)**	**Chronic Risk (HI)**
Residential Uses	2.1E-01	5	**4.1E-02**

Crossroads

Health Risk Calculations - Project Construction

Diesel Particulate Matter Emission Rate Calculation / Scaler

Emission Rate (lbs/year)	536.2
Hours per Day	8
Seconds per Year	10,512,000
Average Annual Emission Rate (g/s)	0.0231
Scaler Concentration (ug/m3)[a]	5.43
Diesel Particulate Concentration (ug/m3)	1.26E-01

[a] Scaler concentration based on a 1 g/s emission rate

Scaler Health Risk Calculations (1 ug/m3)

Source	*Mass GLC*				*Weight Fraction*	*Contaminant*	*Carcinogenic Hazard*		
	(µg/m3) (1 g/s)	*DPM Emissions (g/s)*	*Adjusted Concentration (µg/m3)*	*(mg/m3)*			*URF (µg/m3)$^{-1}$*	*CPF (mg/kg/day)$^{-1}$*	*RISK*
(a)					*(d)*	*(e)*	*(f)*	*(g)*	*(h)*
Construction DPM (Construction Duration)	1.00	1.00000	0.12563	1.3E-04	1.00E+00	Diesel Exhaust Particulate	3.0E-04	1.1E+00	2.03E-06
Total									**2.03E-06**
						DPM Total			**2.03** in a million

Note: Exposure factors used to calculate contaminant intake

exposure frequency (days/year)	250
exposure duration (years)	4.0
inhalation rate (m3/day)	0.393
average body weight (kg)	
averaging time(cancer) (days)	25550
averaging time(noncancer) (days)	14600

Chronic Risk Calculations - DPM

Receptor	**Annual Concentration (ug/m3)**	**Chronic Inhalation REL (ug/m3)**	**Chronic Risk (HI)**
School	1.3E-01	5	2.5E-02

Everest Yan

From: Lijin Sun [LSun@aqmd.gov]
Sent: Wednesday, May 16, 2018 3:58 PM
To: Everest Yan
Subject: RE: CalEEMod Default Vehicle Fleet Mix

No new updates on the construction HRA analysis at this time. We have been short-staffed. Therefore, we have not kicked off the public process to develop the recommendations. In the meantime, please continue to consult with the Lead Agency to ensure that the analysis and intended methodologies are sufficient as substantial evidence for the CEQA document. As you know, we use the revised new OEHHA methodology for determining operational health impacts for permitting applications and also for all CEQA projects where we are the Lead Agency.

Thank you,
Lijin

Lijin Sun, J.D.
Program Supervisor, CEQA IGR
South Coast Air Quality Management District
21865 Copley Drive, Diamond Bar, CA 91765
Direct: (909) 396-3308
Fax: (909) 396-3324

From: Everest Yan [mailto:e.yan@eyestoneeir.com]
Sent: Wednesday, May 16, 2018 3:50 PM
To: Lijin Sun <LSun@aqmd.gov>
Subject: RE: CalEEMod Default Vehicle Fleet Mix

Thanks Lijin,

On a different topic, has there been any new guidance from the SCAQMD regarding the use of new OEHHA health risk guidelines for construction analyses? Thanks again.

Everest

From: Lijin Sun [mailto:LSun@aqmd.gov]
Sent: Wednesday, May 16, 2018 11:20 AM
To: Everest Yan
Cc: Barbara Radlein
Subject: RE: CalEEMod Default Vehicle Fleet Mix

Hi Everest,

My colleague, Barbara Radlein, Program Supervisor, CEQA/Rule Development, may help answer your CalEEMod questions. Barbara is copied on this email.

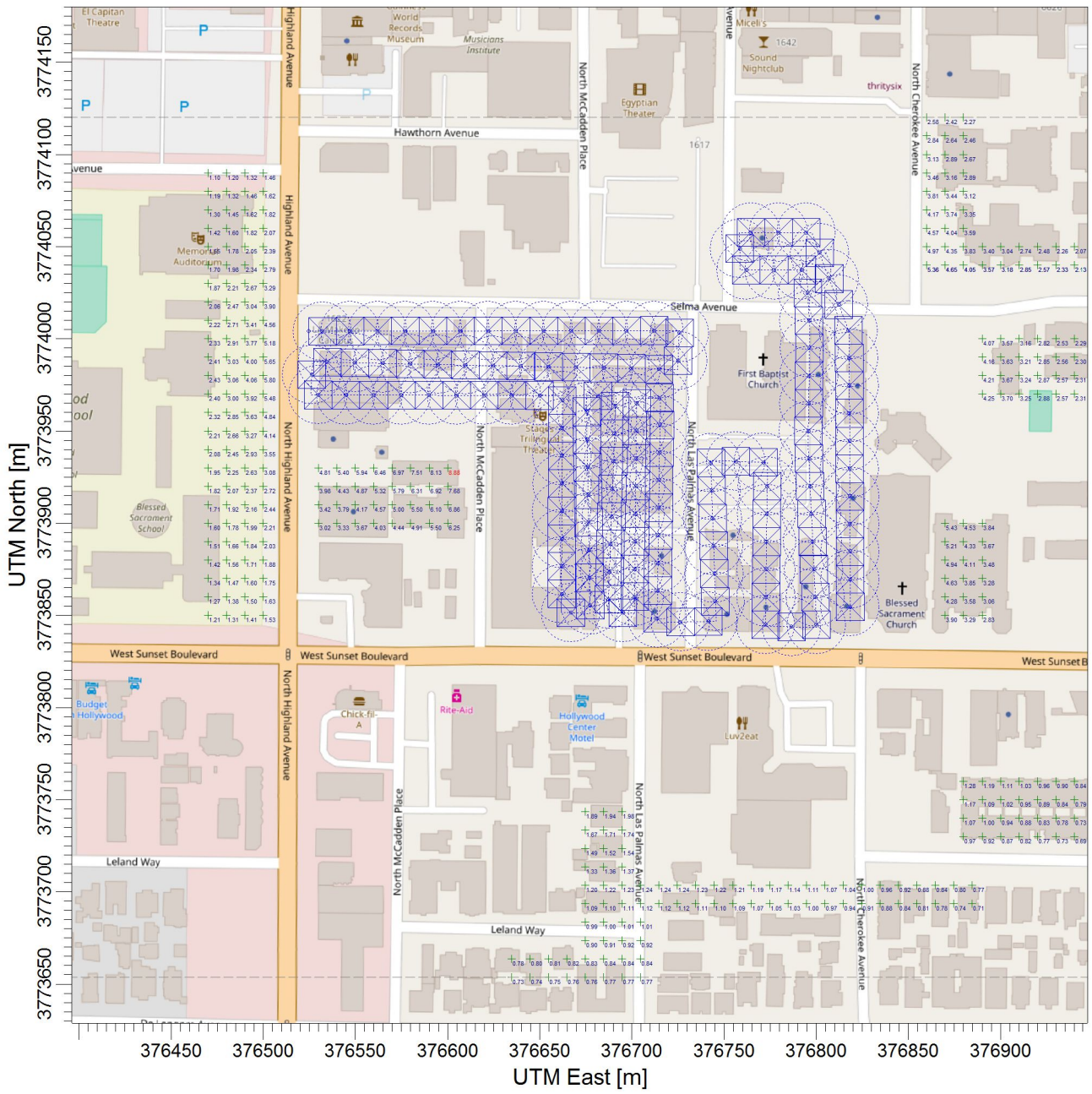
In any event, any assumptions different from the CalEEMod default that you are going to use for the analysis should be well documented in the CEQA document and justified as to why the assumptions are more reasonable to use than the default, if applicable.

Appendix C

AERMOD Source Receptor Configuration

PROJECT TITLE:

C:\AERMOD\Crossroads_HRA2\Crossroads_HRA2.isc



COMMENTS: Crossroads Construction HRA 1 g/s, 8 hrs/day	SOURCES: **1**	COMPANY NAME:	
	RECEPTORS: **365**	MODELER:	
	OUTPUT TYPE: **Concentration**	SCALE: 1:3,467 0 — 0.1 km	
	MAX: **8.88 ug/m^3**	DATE: **9/11/2018**	PROJECT NO.: