Chapter 5: Ecosystem services and human well-being

Authors: VR Haden, D Liptzin, TS Rosenstock, J VanDerslice, S Brodt, BL Yeo, R Dahlgren, K Scow, J Riddell, G Feenstra, A Oliver, K Thomas, D Kanter, and TP Tomich

Section 5.3: Clean air

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Main Messages

Nitrogen is a component of, or aids in the formation of, five known air pollutants including NO_x, NH_3, O_3, $PM_{2.5}$ and PM_{10}. Air pollutants have important impacts on the economy, the environment, and human health, and thus are regulated by state and federal agencies.

Major emissions sources include the combustion of fossil fuels in the transportation, energy generation and industrial sectors, as well as agricultural fertilizers and livestock. Higher NO_x concentrations tend to be measured in and around California's urban areas and originate mostly from the transportation and industrial sectors. Concentrations of ground level O_3, which is formed from emissions of NO_x and volatile organic compounds (VOCs), are highest during the summer months in the South Coast, Bay Area and Central Valley regions. The majority of NH_3 emissions come from livestock waste and N fertilizers, thus concentrations of NH_3 tend to be higher in the southern part of California's Central Valley.

Levels of $PM_{2.5}$ and PM_{10} are highest in the South San Joaquin Valley, South Coast and Salton Sea regions. In the San Joaquin Valley, where livestock activities occur, NH_3 is the dominant constituent of secondary particulate matter. In the urban areas of the South Coast, compounds formed from NO_x NO_x make up a larger fraction of the particulate matter.

Air quality regulations and technological innovations have led to significant declines in NO_x, O_3, $PM_{2.5}$ and PM_{10} over the past four decades. However, much of the state still has air quality that fails to meet one or more of the standards set by national and state agencies to protect human health.

There are important racial disparities in exposure to air pollutants. In the South Coast and San Joaquin Valley Air Basins, a larger percentage of the Black and Hispanic populations are exposed to $PM_{2.5}$ concentrations that are above the NAAQS (35 $\mu g/m^3$) relative to White and Other Races.

Air pollutants are associated with many health problems. These include: difficulty breathing, reduced lung function, asthma, respiratory infections, chronic obstructive pulmonary disease, cardiovascular disease, overall deaths, and deaths due to specific respiratory and cardiac causes. In California, over 12,000 premature deaths per year from cardiopulmonary disease and ischemic heart disease are attributed to elevated $PM_{2.5}$ levels. Studies suggest that the health damages in California associated with poor air quality are on the order of tens of billions of dollars per year.

Air pollution, particularly O_3, has adverse effects on crop growth. Yield losses ranging from 1 – 33%, depending on the sensitivity of the crop and level of exposure, can reduce revenues for agricultural producers and increase food costs for consumers. The overall economic impact of O_3 on agricultural production in California is estimated to be on the order of hundreds of millions of dollars per year.

5.3 Clean air

This section examines how nitrogen influences air quality throughout California. While various forms of reactive N such as nitrogen oxides (NO + NO_2 are together referred to as NO_x) and ammonia (NH_3) are naturally occurring components of the earth's atmosphere, anthropogenic activities have significantly increased their ambient concentrations. Moreover, these forms of N in the air have important impacts on environmental quality and human health. For example, emissions of NO_x and NH_3 directly and indirectly influence the formation ozone, and particulate matter which are criteria air pollutants and thus regulated by the US EPA under the US Clean Air Act, the California Air Resources Board, and various regional air districts (Table 5.3.1; USEPA 2012). Because of their high reactivity, NO_x and NH_3 interact with other chemical constituents in the atmosphere to create a range of harmful secondary chemicals; with tropospheric O_3, nitric acid (HNO_3), ammonium particulates (e.g., NH_4NO_3, $(NH_4)_2SO_4$), and peroxyacetyl nitrates (PANs) among the most important (Figure 5.3.1). While these secondary chemicals can all be components of smog, the relative abundance of NH_3 and SO_2 in the air will determine if these constituents are important components of smog in a given location within California. In addition to an examination of the primary and secondary compounds resulting from nitrogen emissions, we also review the available literature on the health and economic impacts of human exposure to these air pollutants, with a particular focus on vulnerable regions and populations in California.

[Table 5.3.1]

[Figure 5.3.1]

5.3.1. Relationship between nitrogen and air pollutants

5.3.1.1 Emissions of NO_x and NH_3

The primary forms of N that influence air quality are NO_x and NH_3. NO_x is a general term used to refer to nitric oxide (NO) and nitrogen dioxide (NO_2). Fossil fuel combustion is the main anthropogenic source

of NO (Figure 5.3.1; Figure 5.3.2). It is also formed and emitted by natural sources such as soils, wildfires, and lightning. When released into the atmosphere NO is rapidly oxidized to NO_2, thus the formation of both gases are jointly referred to as NO_x emissions and the concentrations are reported in units of NO_2. Since high levels of NO_2 can have negative effects on human health and wellbeing, the US EPA has defined NO_2 as a primary criteria pollutant, and set a national ambient air quality standard (NAAQS) of 0.053 ppm, averaged annually (Table 5.3.1; USEPA 2012). The California ambient air quality standard (CAAQS)set by the California Air Resources Board is even more stringent than the US EPA, at an annual average of 0.030 ppm NO_2 (Table 5.3.1; CARB 2012).

[Figure 5.3.2]

Emissions of NH_3 originate from both anthropogenic (e.g., livestock, N fertilizers, fossil fuel combustion) and biogenic sources (e.g., soils and vegetation). In California, agricultural sources account for more than 77% of anthropogenic NH_3 emissions, with approximately 66% attributed to livestock and 12% to N fertilizers (Benjamin 2000; Chapter 4). The N mass balance presented in Chapter 4 of this assessment also indicates that fossil fuel combustion from mobile sources (i.e. from 3-way catalytic converters which reduce NO to NH_3) is responsible for about 13% of total statewide NH_3 emissions and is a dominant source of NH_3 in the air above many urban areas (Bishop et al. 2010; Nowak et al. 2012).). Gaseous NH_3 is in chemical equilibrium with ionized ammonium (NH_4^+), and the amount of NH_3 volatilized into the atmosphere depends on environmental factors such as temperature, pH, and NH_4 concentrations in a given substrate (e.g., soil, water, manure, fertilizer). NH_3 is not considered a direct hazard to the general population at ambient levels in the atmosphere, and thus is not regulated by the US EPA as a primary criteria pollutant under the Clean Air Act. However, NH_3 is an important precursor in the formation of fine particulate matter ($PM_{2.5}$), which is a regulated pollutant (Figure 5.3.1; USEPA 2012). In occupations where workers are at risk of being exposed to localized NH_3 concentrations much

156 higher than ambient levels, the Occupational Safety and Health Administration (OSHA) has established a
157 permissible eight hour exposure limit of 50 ppm (USEPA 1989). This occupational exposure limit applies
158 to industrial facilities and includes concentrated animal feeding operations (e.g., poultry houses, swine
159 facilities, dairies, feedlots) which tend to have high levels of volatilized NH_3 from urine and manure, and
160 various meat packing and food processing plants that use NH_3 for refrigeration (Donham et al. 2000;
161 Donham et al. 2002).
162
163 ***5.3.1.2 Formation, buildup and decay of tropospheric O_3***
164 Tropospheric O_3 is formed from NO_x, carbon monoxide (CO) and volatile organic compounds (VOCs) in
165 sunlight driven reactions (Figure 5.3.1). Oxidized NO produces NO_2, which then undergoes rapid
166 photochemical decay to reform NO and atomic oxygen (O). High concentrations of oxygen gas (O_2) in
167 the troposphere allow atomic O and O_2 gas to rapidly combine to form O_3 (Seinfeld and Pandis 1998). In
168 the absence of VOCs, O_3 will oxidize NO back to NO_2 and thus restart the cycle with no net gain of O_3.
169 However when VOC molecules are present they break down to form hydroxyl and peroxyl radicals that
170 oxidize NO more rapidly than O_3, thus resulting in a buildup of O_3 (Seinfeld and Pandis 1998).
171 Tropospheric O_3 is ultimately broken down by ultraviolet light or through oxidation reactions with plant
172 and animal tissue or other components of the land surface. National and California air quality standards
173 for O_3 are 0.075 and 0.070 ppm respectively, based on the annual fourth-highest daily maximum 8-hr
174 concentration averaged over 3 years (Table 5.3.1; USEPA 2012; CARB 2012).
175
176 ***5.3.1.3 Sources and formation of particulate matter***
177 Particulate matter is one of the least understood components of atmospheric pollution, mainly due to
178 the large variation in the source and chemical composition of aerosolized particles (Solomon et al.

2007). Particulate matter can be made up of ammonium salts (e.g., NH_4NO_3, $(NH_4)_2SO_4$), metals, organic chemicals, dust, soot, smoke and airborne organic material (pollen, mold spores, etc.). National and state regulatory agencies classify particulate matter by the diameter of the particles, with fine particulate matter ($PM_{2.5}$) being 2.5 µm or less, and inhalable coarse particulate matter (PM_{10}) being between 10 µm or less. National and state ambient PM standards are listed in Table 5.3.1 for both a 24-hr and annual average exposure (USEPA 2012; CARB 2012).

While $PM_{2.5}$ can be formed directly from the combustion of fossil fuels and various organic or inorganic materials, secondary chemical reactions occurring in the atmosphere are also an important mechanism of formation (Figure 5.3.1; Figure 5.3.2). In regions heavily impacted by human activities, elevated levels of NH_3, NO_x, VOCs, and sulfur dioxide (SO_2) contribute to the formation of $PM_{2.5}$ through secondary reactions (Krupa 2003). In areas where agricultural activities contribute large amounts of NH_3 to the atmosphere, ammonium salts make up a large fraction of $PM_{2.5}$ through the conversion of gaseous NH_3 to solid NH_4^+ via reaction with atmospheric acids (i.e., H_2SO_4, HNO_3) (Krupa 2003). Atmospheric HNO_3 concentrations are influenced by NO_x levels, thus both NH_3 and NO_x play a contributing role in the formation of fine particulate matter.

The extent to which N plays a role in the formation or chemical structure of various types of PM_{10} can vary widely based on the source. Particles in this size fraction are commonly associated with fugitive dust arising from agricultural and forestry activities, vehicles traveling on paved and unpaved roads, construction activities, and wind erosion (Chow et al. 2003). Other important components of PM_{10} are the ash and smoke from managed burns or wild fires. PM_{10} can also be formed directly from fuel combustion by the industrial and transportation sectors or through secondary chemical reactions occurring in the atmosphere.

5.3.1.4 Acid precipitation and fog

Air pollutants containing N may also impact human health, plant growth and the environment, by increasing the acidification of precipitation in its various forms (e.g. rain, snow, fog). In particular, the occurrence of acidic fog (or the "fog-smog-fog" cycle) in California's urban, rural and natural areas has received a moderate amount attention in the research literature since the acidity (i.e. the H^+ concentration) in fog is typically 10 to 100 times greater than typical acid rain events (Waldman 1982; Brewer et al. 1983; Munger et al. 1983; Temple et al. 1987). Acid precipitation and fog can be caused by the interaction between atmospheric water droplets and various air pollutants including gaseous NO_2 and SO_2, as well as other aerosols and particulates. For instance, when NO_2 molecules are dissolved in water droplets they react with OH to form nitric acid (HNO_3) which results in an overall decrease in pH. The presence of reduced forms of N (e.g. NH_3 and NH_4) in fine particulates can also increase the concentration of various amino compounds in atmospheric water droplets which can have a net acidic, neutral or basic effect depending on the compound (Zhang and Anastasio 2003).

5.3.2 Spatial and temporal trends in air pollutants

While state regulatory agencies measure atmospheric concentrations of NO_x, O_3, and particulate matter using a series of surface monitoring stations distributed throughout the state, NH_3 levels are not systematically monitored. Placement and distribution of monitoring stations tend to be concentrated in regions with high emissions, thus more data are available for air basins near California's major urban centers. Given the cost of surface monitoring and its limited geographic coverage, remote sensing techniques are increasingly being used to fill in known spatial gaps in air quality data. For example, a new collaboration among the National Oceanic and Atmospheric Administration (NOAA), CARB, and the California Energy Commission (CEC), is using heavily instrumented aircraft to periodically measure NO_x,

225 NH_3 and a wide variety of secondary pollutants throughout California (Ryerson et al. 2013). Ground level
226 concentrations of NO_x, NH_3, O_3 and PM are also detectable by satellites, which are being used in
227 conjunction with surface data and meteorological models to give a more complete assessment of spatial
228 trends (Gupta et al. 2006; Hidy et al. 2009; Clarisse et al. 2009; Clarisse et al. 2010). Despite the
229 advantages of better geographic coverage, the main limitation of remotely sensed data (from both
230 aircraft or satellite) is that they generally lack the continuous temporal resolution needed to calculate
231 average concentrations for the time periods required for compliance with national and state ambient air
232 quality standards (Table 5.3.1; Hidy et al. 2009).

233 As primary pollutants, the spatial and temporal trends of NO_x and NH_3 are closely linked to
234 emissions sources. Since transportation and industrial emissions are the main sources of NO_x, higher
235 concentrations of this pollutant tend to be measured in and around urban areas. Spatial data derived
236 from interpolated surface measurements and satellite images both show that concentrations of NO_x
237 (reported in units of NO_2) are highest in the Los Angeles region and to a lesser extent near San Francisco
238 and Sacramento (Figure 5.3.3; Russell et al. 2010; Kar et al. 2010). These data also indicate that NO_x
239 levels in San Diego, the Imperial Valley and several cities in the Central Valley (e.g., Fresno, Bakersfield)
240 are notably higher than less populated regions of the state.

241 [Figure 5.3.3]

242 Despite rising vehicle use and population, levels of NO_x across California have declined at a
243 relatively constant rate over the last several decades; a trend that is well established based on evidence
244 from emissions inventories (Cox et al. 2009; Millstein and Harley 2010; McDonald et al. 2012), surface
245 measurements (Figure 5.3.4; Parrish et al. 2011; Ban-Weiss et al. 2008; Russell et al. 2010; Lafranchi et
246 al. 2011), and satellite observations (Kim et al. 2009; Russell et al. 2010). Using satellite and surface
247 measurements Russell et al. (2010) observed an annual decline in NO_x of 9% y^{-1} between 2005 and 2008

for Los Angeles, San Francisco and Sacramento (Figure 5.3.5). Smaller declines in NO_x levels of approximately 4% y^{-1} over the same period were also observed in the San Joaquin Valley for Fresno and Bakersfield. A combination of regulatory policies (e.g., vehicle emissions standards) and technological innovations (e.g., catalytic converters, cleaner fuels, fuel efficient engines) are largely responsible for the declining levels of NO_x and overall improvements in California's air quality (Parrish et al. 2011, McDonald et al. 2012, Warneke et al. 2012).

[Figure 5.3.4]

[Figure 5.3.5]

Since the majority of anthropogenic NH_3 emissions come from livestock production and fertilizer application, atmospheric NH_3 concentrations tend to be higher in rural areas with intensive agriculture (Figure 5.3.2; Benjamin 2000; Clarisse et al. 2009; Nowak et al. 2012). For example, recent infrared satellite data show that, relative to other parts of California, NH_3 levels are highest in the San Joaquin Valley where the state's dairy and poultry industries are concentrated (Figure 5.3.6; Clarisse et al. 2009; Clarisse et al. 2010; Benjamin 2000). This region also coincides with California's highest levels of groundwater nitrate (Harter and Lund 2012). Furthermore, in examining a full global dataset Clarisse et al. (2009) found that the San Joaquin Valley had the highest annual daily average NH_3 values (> 3 mg m^{-2}) of any agricultural region in the world. While atmospheric concentrations of NH_4 are generally lower in urban areas, the relative amount of NH_3 from vehicle emissions is often larger than from agricultural sources. For example, airborne measurements collected in the South Coast Air Basin during 2010 indicate that automobiles in the air basin emitted 62 metric tons of NH_3 per day, while dairy facilities emitted 33 metric tons of NH_3 per day (Nowak et al. 2012). Other than coarse NH_3 emission inventories conducted at rather infrequent intervals, NH_3 levels are not routinely monitored by the state (CARB 2000). As such, there are few available data upon which to establish temporal trends for NH_3 emissions

271 in California. However, changes in NH_4 levels over time are likely to correspond closely to changes in the
272 cattle and poultry populations as well as shifts in vehicle use and emissions control technology in the
273 transportation sector.
274 [Figure 5.3.6]
275 While NO_x and NH_3 levels are heavily dependent on the source of primary emissions, the
276 formation of secondary pollutants such as O_3 and particulate matter are more closely tied to the
277 topography, meteorological conditions and atmospheric constituents present in a given time and place.
278 High O_3 levels most frequently occur during California's summer "O_3 season" when high solar radiation
279 facilitates the decay of NO_2 and subsequent formation of O_3 (Kaduwela 2007). Concentrations of O_3 also
280 tend to be higher downwind of urban areas because of the time lag between the primary emissions of
281 NO_x and the secondary photochemical reactions that produce O_3 (Pusede and Cohen 2012).
282 Paradoxically, O_3 concentrations can be higher on weekends, especially in urban areas, when NO_x
283 concentrations are lower (Altshuler et al. 1995). The weekend effect is likely a function of the relative
284 concentrations of VOC to NO_x (Murphy et al. 2007a; Murphy et al. 2007b). In California, a distinct
285 weekend effect occurs in the Los Angeles, South Coast and San Francisco air basins (Kaduwela 2007).
286 In the past four decades, 8-h average O_3 levels (3-yr averages of the 4th highest annual maxima)
287 in the Los Angeles area have declined by more than 50%; down from nearly 300 ppb in the 1970s to just
288 over 100 ppb in 2010 (Figure 5.3.4; Parrish et al. 2011). While improvements in O_3 have also been
289 observed in the Central Valley over the same period, O_3 levels for the San Joaquin Valley remain higher
290 than in California's coastal cities (CAPCOA 2012). Recent satellite data indicate that the San Joaquin
291 Valley and South Coast regions also have the highest levels of $PM_{2.5}$ (>than 14 $\mu g/m^3$) in the state (Figure
292 5.3.7; Van Donkelaar et al. 2010). Similar to trends in O_3, $PM_{2.5}$ levels have declined more rapidly in the
293 South Coast Air Basin than in the San Joaquin Valley Air Basin (CAPCOA 2012).

[Figure 5.3.7]

Throughout California, the pH of precipitation (pH =5.2-6.2) tends to be less acidic than in other US states located in the industrial centers of the Northeast and Midwest (pH=4.3-4.9) (NOAA 2006). However, California's frequent fog events may still pose problems given fog-water's tendency to have higher concentrations of acidity than rain-water. For instance, several studies conducted in the 1980s recorded median pH levels of 3.3 in fog, 3.6 in mist and 4.49 in rain at various urban and rural sites in southern California, with some pH values for fog and mist reaching as low as 2.15 (Waldman 1982; Brewer et al. 1983; Munger et al. 1983). Given the gradual decline in NO_x emissions observed in California and other states over recent decades, the problem of acid precipitation has become less severe (Burns et al. 2011; Parrish et al. 2011).

5.3.3 Patterns of exposure to air pollutants in California

With well-established reductions in NO_x, O_3, $PM_{2.5}$ and PM_{10} documented throughout much of the state, considerable progress has been made to improve California's air quality over the past several decades (Parrish et al. 2011). But while these improvements may highlight the efficacy of certain regulatory policies and technological advances, a number of significant air quality problems persist. For example, the majority of California's counties are still designated as "non-attainment" areas based on California ambient air quality standards for O_3, $PM_{2.5}$ and PM_{10} , thus highlighting the health risks that remain for much of the state's population (Figure 5.3.2; Figure 5.3.8). According to Hall et al. (2008), air pollution levels in the South Coast Air Basin (SoCAB) and the San Joaquin Valley Air Basin (SJVAB) remain among the worst in the US, and during peak periods many other urban areas still reach O_3 and $PM_{2.5}$ concentrations roughly double the acceptable federal limit for vulnerable populations (Hall et al. 2008;

Parrish et al. 2011). PM_{10} levels in the Salton Sea Air Basin also frequently exceed values in other parts of the State (CDPH 2015).

[Figure 5.3.8]

With the exception of Los Angeles, all regions of California are currently in compliance with the state and national ambient air quality standards for NO_2 (Figure 5.3.8; Table 5.3.1; EPA 2010; CARB 2011). In certain high vehicle traffic areas within the SoCAB, NO_2 levels can sometimes exceed air quality standards for short periods (NAAQS = 0.18 ppm for a 1-hour average). For example, in 2009 NO_2 levels over the national standard for the 1-hour average were recorded during 3 days in Los Angeles and during one day in both San Bernardino and Imperial counties (CARB 2011). Los Angeles is the only area of the state that is still designated as being in "non-attainment" according to more stringent California standards (CARB 2011).

In contrast with NO_2 standard attainment in California, the US EPA has classified 31 counties in the South Coast, Central Valley and Bay Area as federal non-attainment areas for O_3, with the South Coast and the San Joaquin Valley Air Basins further designated "extreme non-attainment areas" (CARB 2011; Hall et al. 2008a; Hall et al. 2008b). From 2005 to 2007, O_3 levels for large parts of Kern, Tulare, San Bernardino and Riverside counties exceeded the NAAQS 8-hour maximum standard (>0.075 ppm) more than 50 days a year (see Figure 5.3.9; Hall et al. 2008a). In 2009, eight counties throughout the state had more than 30 days where the 8-hour O_3 level exceeded the national standard (Figure 5.3.10). While Los Angeles County had fewer non-attainment days per year for the national 8-hour O_3 standard than counties in the San Joaquin Valley, due to its high population density it had the highest number of person-days per year (104.97 million) in which people were exposed to unhealthy levels of O_3 (Hall et al. 2008a).

[Figure 5.3.9]

[Figure 5.3.10]

Many areas in the South Coast, Central Valley and Bay Area are also classified as being in nonattainment for $PM_{2.5}$ under both state and nation standards (Figure 5.3.8; USEPA 2010; CARB 2011). Moreover, Hall et al. (2008) found that from 2005 to 2007 100% of the population living in the Madera, Fresno, Kings, Tulare, and Kern counties (a total of 2,142,056 people) were exposed to annual average $PM_{2.5}$ concentrations above the NAAQS (15 $\mu g/m^3$). Over the same period, about 75% of the population living in Los Angeles county (a total of 10,199,229 people) was also exposed to annual average $PM_{2.5}$ concentrations above the NAAQS (Hall et al. 2008). Data also suggest that the majority of people living in the Bay Area and the Sacramento Valley are also exposed to average annual $PM_{2.5}$ levels above the NAAQS (CARB 2011). While progress in reducing $PM_{2.5}$ has been made, annual average $PM_{2.5}$ concentrations in the San Joaquin Valley and South Coast Air Basins must still fall by approximately 30% to meet the federal standards (Hall et al. 2008). As with $PM_{2.5}$, the vast majority of California counties have failed to achieve low enough average annual concentrations of PM_{10} to be classified as being in attainment with state air quality standards (Figure 5.3.8; CARB 2011).

5.3.4 Disparities in exposure to air pollutants

Since low-income and minority neighborhoods, particularly those in urban areas, are more likely to be adjacent to large roads, heavy industry, and other pollution sources, certain socioeconomic classes and ethnic groups are likely to be at greater risk of exposure to various air pollutants (Su et al. 2009; Hall et al. 2006; Hall et al. 2008; Morrello-Frosch and Jesdale 2006; Marshall 2008; Marshall 2006). Epidemiological studies also suggest that racial disparities in exposure to air pollution are more prominent in metropolitan areas with high racial segregation among residents (Morrello-Frosch and Jesdale 2006). In California, no studies have yet examined disparities in exposure to NO_2, however two

recent studies by Hall et al. (2006, 2008) do provide a detailed analysis of exposure to O_3 and $PM_{2.5}$ among ethnic groups residing in the San Joaquin Valley and South Coast Air Basins.

Examining exposure to O_3, Hall et al. (2008) observed that geographic differences in ethnicity among residents within and between different counties in Southern California result in different O_3 exposure frequencies. In particular, they estimated the number of person-days and average number of days residents of the San Joaquin Valley and South Coast Air Basins were exposed to O_3 above the 8-hour NAAQS threshold (0.075 ppm). They found that during the 2005-2007 period Hispanics in the San Joaquin Valley and Whites in the South Coast Air Basin were more frequently exposed than other racial groups to O_3 levels above the 8-hour NAAQS limit (Hall et al. 2006; Hall et al. 2008).

Hall et al. (2008) found even more prominent disparities in exposure to $PM_{2.5}$, with approximately 55% of White, 60% of Other Race, 70% of Hispanic, and 78% of Black residents in the South Coast Air Basin exposed to average annual $PM_{2.5}$ concentrations above the 15 $\mu g/m^3$ NAAQS threshold. Similarly results were also found in the San Joaquin Valley Air Basin, with an estimated 61% of White, 56% of Other Race, 72% of Hispanic, and 66% of Black residents exposed to annual $PM_{2.5}$ concentrations above the average annual $PM_{2.5}$ NAAQS (Figure 5.3.11). While studies examining racial disparities and air pollution are not available for California's other regional air basins, the patterns of exposure among minorities in highly segregated communities are likely to occur elsewhere in the state (Morrello-Frosch and Jesdale 2006).

[Figure 5.3.11]

5.3.5 Human well-being and air quality

The poor air quality caused by high levels of NO_2, O_3, $PM_{2.5}$, PM_{10} is known to have a number of well-established impacts on human health. There is strong evidence linking these pollutants with health

problems such as difficulty breathing, reduced lung function, asthma, respiratory infections, chronic obstructive pulmonary disease (COPD), cardiovascular disease (CVD), deaths due to specific respiratory and cardiac causes, and overall deaths (Table 5.3.2; Figure 5.3.2). Studies also show, albeit with less certainty, that these pollutants are related to other problems such as lung cancer, low birth weight babies or pre-term births (Table 5.3.2; Figure 5.3.2). These pollutants have also had adverse effects on human well-being by increasing the school absences and the number of restricted activity days (RADs) and minor restricted activity days (MRADs) (Hall et al. 2010). Given that these pollutants each have different chemical and physical properties, the mechanisms by which they impact human health can vary widely. Both O_3 and NO_2 are strong oxidants. When they are inhaled, they can injure cell structures, create reactive chemicals that cause other damage, and initiate biochemical processes, such as inflammation. While particulate matter is not a strong oxidant in itself, it can react with fluids lining the airway, and cause similar adverse reactions and inflammation. Below, the epidemiological evidence related to poor air quality is examined with the goal of understanding the degree to which various health problems are affected by each pollutant.

[Table 5.3.2]

5.3.5.1 Interpreting epidemiological evidence

As with many environmental exposures, the results of epidemiologic studies focusing on the effects of air pollutants vary considerably. Some studies show significant associations between airborne contaminants and certain health impacts, while others do not. This can be due to differences in the study design, the population studied, and various interactions among other related factors. The relationships between pollutant exposures and health outcomes all have uncertainty, which is determined based on the consistency of findings across studies. This is particularly true for rigorous

studies that provide a plausible physiologic mechanism for the health impact. In some cases an estimate of the magnitude of the effect is derived from the results of the best studies. Environmental regulations are then based, in part, on these observed effects.

Several hundred epidemiological studies in the United States have been published relating levels of ambient air pollutants to health problems. There have been fewer studies examining the health effects of NO_2 and NH_3 relative to those involving O_3 and particulate matter. As such, there is stronger evidence linking O_3 and particulate matter to adverse health outcomes. In this section we present an overview of the evidence, highlighting the most significant studies, and those focusing on populations in California.

5.3.5.2 Evidence of the impacts of NO_2, O_3 and particulate matter exposure on respiratory health

There is ample evidence that both short-term and long-term exposures to these air pollutants can begin a cascade of biochemical impacts that can lead to a number of health problems. These health problems include respiratory symptoms, increases in respiratory infection, decreased lung functioning, decreased lung growth in children, increases in the severity of asthma, and increases in the number of admissions to hospitals or emergency departments for respiratory problems or asthma attacks. Recent studies also suggest that the incidence of air pollution related health impacts occurs with relatively high frequency in California, with the residents of counties in the South Coast and San Joaquin Air Basins facing high levels of exposure to various pollutants (Table 5.3.3; Hall et al. 2006; Hall et al. 2008)

[Table 5.3.3]

Asthma is one of the leading illnesses among children and adults, affecting 25 million Americans or approximately 8.4% of adults and 9% of children (CDC 2008; CDC 2009). This is nearly a 10% increase

since 2001 (NCHS 2011). Asthma can have a major impact on one's quality of life. This impact is reflected in the fact that in 2008 over 10 million school days and over 14 million work days were missed due to asthma, nationwide (NCHS 2011).

Asthma is a condition where the muscles surrounding the bronchial airways contract, and an inflammatory response leads to the secretion of thick mucus into the airway. This makes it very difficult to breathe (NHLBI 2007). A person experiencing an asthma attack will have trouble breathing, and will wheeze because of the narrowed airways. Such asthma attacks, or exacerbations, are brought on by a trigger, some factor that starts the inflammatory process. It can be cold air, an allergen, or a gas that irritates the airway. Asthmatics have developed a hyperresponsiveness, where the inflammatory process responds immediately to a small trigger, one that does not affect a non-asthmatic.

To better understand the effect of NO_2, O_3 and particulate matter exposure on the development and exacerbation of asthma, studies have examined the relationship between exposure levels to these pollutants and each of the steps of the disease process: increasing the inflammatory response, developing hyper-reactivity, lung function, asthma exacerbations, visits to the hospital or emergency room for asthma, and deaths.

Airway inflammation

When cells lining the trachea experience a physical or chemical insult, it can trigger an inflammatory response, that is, a biological response designed to protect the body from infectious agents. NO_2 and O_3 are strong oxidants. When inhaled, they can react with the membranes of the cells lining the trachea. This activates macrophages, a type of white blood cell, present in the lung tissue. This leads to the release of chemicals that change the membranes of blood vessels along the lung, to allow leukocytes

(white blood cells) into the lung. They release other chemicals (interleukins, proteases and oxidative species) to limit cell injury. This is called an inflammatory response.

A number of studies have examined whether exposure to these pollutants leads to specific aspects of the inflammatory response by observing the levels of specific chemicals in lung fluids. While some studies have shown evidence of inflammation in response to NO_2 exposure, particularly in children, others have not. Two comprehensive reviews, in fact, came to contradictory conclusions (USEPA 2008a; Hesterburg 2009). Many laboratory studies have found evidence of inflammation after exposure to O_3 and particulate matter (Mudway and Kelly 2004; USEPA 2006; Alexis 2010; Dahl et al. 2007; USEPA 2006; USEPA 2009). Exposure to O_3 over several days elicited the greatest inflammatory response. Biochemical markers of inflammation occur after a single exposure to O_3 (USEPA 2006; Alexis et al. 2010). Short-term exposure to $PM_{2.5}$ was related to higher levels of a biomarker of inflammation among asthmatic children (Lui et al. 2009; Delfineo et al. 2006; Mar et al.2005), and older adults (Adamkiewicz 2004; Jansen et al. 2005; Adar et al. 2007). A cohort study of over 2,000 children found higher levels of inflammation with higher annual average $PM_{2.5}$ levels (Dales et al. 2008).

Hyperresponsiveness

Hyperresponsiveness refers to the tendency to initiate a rapid, intense constriction of the smooth muscles around the bronchi in response to an allergen. This response is not common among people who do not have asthma, but is typical of asthmatics. (Crockcroft and Davis 2006). In fact, asthma is diagnosed by exposing a person to such a trigger, and observing the result. The severity of asthma, risk of exacerbations, and impact on lung function are all clearly related to the degree of hyperresponsiveness (Xuan et al. 2001; Murray et al. 1986). Children who demonstrate hyperresponsiveness are at higher risk of developing asthma and not fully developing their lung capacity

and function (Postma and Boezen 2004; Xuan 2001). Exposure to environmental irritants, including NO_2, O_3 and particulate matter and cigarette smoke, may induce inflammation, and as a result, indirectly impact lung function by increasing the lung's responsiveness.

Several studies have investigated whether prior exposure to air pollutants increases the level of severity of an asthma exacerbation (in terms of lung function) resulting from subsequent exposure to a trigger (such as an allergen, cold air, etc.). There has been clear evidence from animal and clinical studies that O_3 increases a person's response to a respiratory stimulus (Jorres et al. 1996; Kehrl et al. 1999; Holz, et al. 2002), but these have been at levels well above air quality standards and greater than almost all observed levels. This increase in airway responsiveness was observed in both asthmatics and those without asthma. Particulate matter, in contrast, has not been associated with increased airway hyperresponsiveness (USEPA 2009). The results from studies examining NO_2 varied, with some of the studies demonstrating an effect, while others did not. However, for those that did show evidence of NO_2 induced hyperresponsiveness, the effects occurred at much lower levels of NO_2 exposure than were associated with inflammation (USEPA 2008b). An analysis of several studies by EPA indicates that exposure to NO_2 at levels found in ambient air is related to airway hyperresponsiveness for mild asthmatics (EPA 2008). This is a possible mechanism that would explain a link between these exposures and the number of hospital admissions for asthma (USEPA 2009).

Lung function

Lung function is best measured by spirometry, the use of sophisticated devices which carefully record the amount of air inhaled and exhaled, and the velocity of the air as it is exhaled (Barreiro and Perillo 2004). Inflammation can affect the size of the airway opening, which impacts both how quickly a person can draw in or expel air, and how much air they breathe. Long term exposures to air pollutants or other

499 exposures such as smoke can permanently reduce the capacity of the lung. The impact of exposure to air
500 pollutants is assessed by conducting baseline spirometry, and repeated measures at different levels of
501 exposure. Each of four studies that used spirometry on school children found decreased lung function
502 with increasing ambient NO_2 levels (Moshammer et al. 2006; Hoek and Brunekreef 1994; Linn et al.
503 1996; Timonen 2002). For example, Moshammer et al. (2006) found that, among children, an increase in
504 NO_2 concentration of 20 ppb was associated with a 4% reduction in the total amount of air capacity of
505 the lung (i.e., forced expiratory volume). Two studies of adult populations did find significant
506 associations between NO_2 levels and lung function using spirometry among a population of never-
507 smokers (Schlindler et al. 2001) and patients with COPD (Silkoff et al. 2005).
508 There are a number of clinical studies where relatively small numbers of volunteers were
509 exposed to air pollutants at different concentrations, and their lung function was monitored. While most
510 of these were studies of asthmatics or people with COPD, only some of the studies observed significant
511 impacts of NO_2 or particulate matter on lung function. However studies have consistently found that six
512 hours of exposure to O_3, even at levels found in some cities, can decrease lung function, although in
513 many studies volunteers returned to normal in a matter of hours (USEPA 2006).
514 Over 30 epidemiologic studies have examined the effects of short-term and long-term
515 particulate matter exposures on lung function and many of these have seen lung function adversely
516 affected by exposure. Gauderman (2002) found such a relationship in the Child Health Study (below). A
517 similar number of studies have examined the effects of O_3 exposure both in clinical studies and
518 epidemiologic studies (USEPA 2006). Associations were seen in many studies, particularly among
519 asthmatics, workers outside and in older populations.
520 Normally as the lungs of a child develop, these measures of lung function increase as well. A
521 number of studies have followed children over time (3 to 10 years) with annual exams to see if the

growth in lung function (e.g., the amount of air inhaled) is impacted by long term exposure to NO_2 (Oftedal et al. 2008; Gauderman et al. 2004; Rojas-Martinez et al. 2007). In each of these studies children living in areas with higher NO_2 levels had less lung growth than other children. As other pollutants tended to vary in the same way as NO_2, the studies could not show that the effects were directly associated with NO_2, but only with increases in pollution levels.

Respiratory symptoms

Researchers have investigated air pollutant concentrations in ambient air as well as in the indoor environment, and the prevalence of respiratory symptoms among asthmatics and those without asthma. Assessments of the effects of NO_2 in ambient air include three large studies, each of which focused on children (Schwartz et al. 1994; Mortimer et al. 2002; Schildcrout et al. 2006). In each of these studies children in several cities in the US were followed over time (from 2 months to a year of data collection), and their respiratory symptoms (e.g., coughing, wheezing, and/or shortness of breath), and/or use of asthma rescue medications were recorded. All three studies found significant associations between NO_2 levels and the frequency of respiratory symptoms, and the effects were larger among asthmatics. The strongest associations were found for NO_2 levels averaged over the previous 2 to 6 days. As NO_2 levels tend to vary with other air pollutants that can also induce inflammation and impact respiratory symptoms (e.g., O_3 and $PM_{2.5}$), the researchers used statistical techniques which accounted for the levels of these pollutants. In most cases there was still an effect of NO_2. These studies provide some of the most convincing evidence that NO_2 levels in ambient air are associated with increases in respiratory symptoms, particularly among asthmatics. Combined together, these studies show that a 20 ppb increase in average NO_2 levels is associated with a 14% increase in the risk of experiencing adverse respiratory symptoms (USEPA 2008b).

A number of other studies were similar in design, but used children in only one location. This reduced the variability in NO_2 levels, and in the relationship of NO_2 with other factors that potentially affected respiratory problems. Studies in Paris (Just et al. 2002), Sydney and Perth, Australia (Jalaluden et al. 2004; Rodriguez et al. 2007), the United Kingdom (Ward et al. 2002) and the Netherlands (Boezen et al. 1999) all found associations between ambient NO_2 levels and cough or other respiratory symptoms in children.

A number of recent studies that followed people over time assessed the effects of particulate matter. As pollution levels dropped between 1993 and 2000, Bayer-Ogelsby et al. (2005) observed a drop in the incidence of chronic cough, bronchitis, and colds among children. The incidence of cough, phlegm and wheezing dropped among adults in Switzerland between 1991 and 2000, as PM levels dropped (Schlindler et al. 2009). Several other studies have found similar results. Short-term exposures to particulate matter have not had strong associations. Recently Weinmayer et al. (2010) reviewed 36 studies that looked at asthma symptoms, cough and peak expiratory flow, a measure of lung function. Overall there was good evidence of PM_{10} being associated with these symptoms, and less convincing evidence of associations with NO_2. In many studies acute exposures to O_3 are related to respiratory symptoms among people with asthma, but there are also several studies where there is no effect (USEPA 2006). The effects from multiple days of exposure have been observed to be larger than the effects from a single day (Escamilla-Nunez 2008; Romieu et al. 2006).

Some research has assessed the relationship between indoor levels of NO_2 and respiratory symptoms. The advantages of this study design are that NO_2 levels can be accurately assessed through direct monitoring, and that exposure is almost exclusively to NO_2 as other air pollutants do not tend to occur in the indoor environment. The most convincing study randomly selected schools to replace their un-vented heaters with heaters that were vented (Pilotto et al. 2004). This change led to a substantial

reduction in indoor NO_2 levels. The children in the intervention schools had significantly fewer episodes of asthma attacks, difficulty breathing and tightness of the chest during the day. Two observational studies (Belanger et al 2006; Kattan 2007) found that the risk of poor respiratory symptoms (i.e., wheeze or cough) was 50% greater for increases of 20 ppb of NO_2. McConnell (2006), found respiratory symptoms to be associated with NO_2, but that the effect of NO_2 exposure was greater for children who had a dog. Several other studies, all with a large number of subjects, did not find an association.

Asthma

Directly assessing the relationship between the proportion of a population with asthma (prevalence) or the number of new cases of asthma in a given time period (incidence) with air pollutant levels typically requires a large study population that is followed over time. One of the best studies of this type is the Child Health Survey, a study of children in several communities in southern California. In one part of this study NO_2 levels were measured in children's homes. The mean NO_2 level was associated with a history of asthma and the amount of asthma medications used (Gauderman et al. 2005). Each 20 ppb increase in NO_2 levels was associated with an eight-fold increase in asthma prevalence (Gauderman 2004). Millstein et al (2004) found that while NO_2 was not related to the use of asthma medications, other air pollutants were, and the effect was greater among kids who spent more time outside. New onset asthma was reportedly associated with outside exercise, especially where O_3 levels were high, and with estimated O_3 levels near homes and schools (McConnell et al. 2002; McConnell et al. 2010). O_3 level was also demonstrated to be highly related to new onset asthma among genetically susceptible children (Islam et al. 2008; Islam et al. 2009). Islam et al. (2007) also found that the protective effective of good lung function against new onset asthma was reduced if the child lived in a high $PM_{2.5}$ community.

Many other studies have established strong associations between the onset of asthma and O_3 and $PM_{2.5}$ levels (USEPA 2008b; USEPA 2009; USEPA 2006). For example, a study in the Netherlands among young children (0-4 yrs) found an association between NO_2 and $PM_{2.5}$ levels and ever being diagnosed with asthma (Brauer et al. 2007). A nationwide study including over 30,000 children found that relatively small increases in O_3levels were associated with developing asthma, or having an asthma attack (Akinbamia et al. 2010). Other studies have looked at exposure to traffic (based on distance to major roads) and found associations with asthma, without looking at specific pollutants.

Some investigators have used data about hospital admissions to specifically study asthma admissions. These cases of asthma are usually the most serious cases. Overall the results of these studies are mixed and provide some indication of a relationship between daily mean NO_2 levels and asthma admissions, with stronger evidence for effects on children. Grineski et al. (2010) demonstrated that the risk of being admitted to a hospital for asthma when NO_2 levels were elevated was higher for minority children and children without health insurance.

There have been many studies of particulate matter and asthma admissions using different statistical methods and comparing admissions to particulate matter levels at different times in the past (e.g., the day before, 3 days before etc.). Most of the studies showed some effects, with somewhat more consistent results in studies of older people.

While there have been fewer studies of O_3, this pollutant has been consistently found to be related to asthma admissions, although study results vary. In New York, chronic O_3exposure was significantly associated with asthma severe enough to require hospitalization for children 1 to 6 years of age (Lin et al. 2008). An analysis of six years of intensive care unit (ICU) and non-ICU admissions for asthma found associations with O_3 among children 6-18 years of age (Silverman and Ito 2010). A linear concentration-response relationship was observed, even at levels below the regulatory levels. A recent

study in California found that annual average O_3, $PM_{2.5}$ and PM_{10} levels were each associated with asthma-related hospitalizations or emergency room visits (Meng et al. 2010).

Chronic Obstructive Pulmonary Disease (COPD)

COPD refers to a serious lung condition that makes breathing very difficult. It consists of two diseases: chronic bronchitis and emphysema. It is the result of long-term exposure to agents that impact the lungs such as smoking and air pollutants. These exposures actually affect the physical structure of the lung. COPD is the 4th most common cause of death in the US, with over 12 million diagnosed cases (NHLBI 2010). Most of the evidence indicates that exposure to air pollutants can lead to emergency department visits, hospitalizations and deaths among people with COPD; there is less evidence that exposure actually leads to the development of COPD (Ko et al. 2010).

Only a handful of studies have looked at NO_2 and emergency department or hospital admissions for COPD, and the results were inconclusive. In Los Angeles, NO_2 levels were associated with COPD in older adults (Moolgavkar 2003), as well as among adults over 30 (Linn et al 2000). Large studies in Canada and Finland examined the relationship of several pollutants and emergency department (ED) visits for specific causes. O_3 levels were associated with asthma and COPD (Halonen et al. 2010), while particulate matter was associated with asthma, particularly during the warm season (Stieb et al. 2009). Many studies also have documented associations between particulate matter and COPD, emergency department visits, and hospitalizations (Dominici et al. 2006; Medina-Ramon et al. 2006; Peel et al. 2005; Chen et al. 2004).

Respiratory infections

There is some evidence that short-term exposures to NO_2 can affect the body's natural defenses against viral or bacterial respiratory infections, increasing the risk of respiratory infections. Two studies of groups of children followed over time observed associations between ambient NO_2 and PM exposures and ear, nose and throat infections (Brauer et al. 2007; Brauer et al. 2002) and ear infections (Brauer et al. 2006) in children. Several studies have found associations between NO_2 levels and emergency ED visits and hospitalizations for many respiratory conditions including respiratory infections (Peel 2005). However, a study focused on the relationship between lower respiratory tract infection in children and NO_2 in three European cities (up to 42 ppb) found no association (Sunyer et al. 2004).

There are several possible mechanisms that could lead to increased susceptibility from NO_2 exposure (Chauhan and Johnston 2003). Exposures to higher than ambient levels of NO_2 and O_3 have, in some human and animal studies, been shown to temporarily reduce the action of the cilia that help capture and expel foreign bodies from the airway. However other studies have not observed this effect. In animal studies, exposure to O_3 has led to damage to the cilia. A number of clinical studies have examined the effects of NO_2 exposure on immune and biochemical responses that could account for more severe symptoms from a respiratory infection among those exposed to NO_2. While some studies have found a significant effect, others have not (USEPA 2008b; Hesterberg et al. 2009).

There is even stronger evidence that exposure to NO_2 can worsen the severity of respiratory infections. Studies of children in England (Chauhan et al. 2003) found that those children exposed to high levels of NO_2 (>7.4 ppb week before) had more severe symptoms and decreased lung function as compared to children with low levels of NO_2 exposure. This study also found that children exposed to higher levels of NO_2 were nearly twice as likely to suffer an asthma exacerbation associated with the infection in the week after the infection had started, as compared to children exposed to lower levels

(Linaker et al. 2000). This is considered to be one of the strongest studies in that exposure was carefully measured using detectors pinned to the children's clothing.

There are few studies examining the effects of O_3 or particulate matter on respiratory infection. In one study visits to physicians in a managed care organization in Atlanta for upper and lower respiratory infection were not related to O_3 levels (Sinclair et al. 2010). While there are a number of studies of particulate matter and respiratory infection and/or pneumonia, two multi-city studies in the US both found increases in admissions for respiratory infections or pneumonia (Medina-Ramon et al. 2006; Dominici et al. 2006)

5.3.5.3 Hospital Admissions for respiratory problems

Well over 100 studies have been conducted to assess the relationship of ambient pollutant levels and the number of hospital admissions and/or ED visits for asthma exacerbations, chronic obstructive pulmonary disease (COPD), all respiratory complaints, or other related health problems. There are two types of studies. To look at short-term effects, the numbers of visits are compared to pollutant levels, either maximum levels or 24-hour average concentrations, for the day of the admission or some number of days prior to the admission. The effects of longer-term exposures are measured by using average pollution levels over months or years and subsequent hospital or ED admission rates.

While all these studies share the same general design, there are differences in the population studied, the health endpoints used, the way exposure is measured and the locations. These differences in study design can lead to variability in the observed results. Overall, such studies are limited in that they can only demonstrate an association between days that have a higher number of admissions and days when pollutant levels are higher; they do not collect additional data from each person about the other factors that can lead to respiratory problems, and as such they cannot control for these factors in

the analysis. Nevertheless, these studies show a consistent set of results. In the vast majority of studies, days with higher levels of NO_2, O_3 and/or particulate matter were associated with higher numbers of hospital or ED admissions for all respiratory complaints.

Overall most of the studies showed that a 20 ppb increase in NO_2 was associated with a 1% to 25% increase in the number of admissions. Even though high levels of NO_2 tend to occur on the same days as high levels of other pollutants, controlling for the other air pollutants did not significantly change the estimated effect of NO_2 levels. While some of the studies looked specifically at children or people over 65, the results were about the same.

Katsouyanni et al. (2009) recently conducted a study which combined information from several large multi-city studies of air pollution and respiratory hospital admissions. Daily increases in O_3 resulted in significant increases in admissions; a change of 40 ppb was associated with a 2 – 3 % increase in admissions. Particulate matter levels were not consistently associated with admissions. While very few studies have looked at the independent effects of the different types of particulate matter, Ostro et al. (2009), using hospital records from six counties in California, found that many of the specific components of $PM_{2.5}$, including nitrate, were independently related to respiratory hospitalizations among children; the effect of NO_2 was similar to that of $PM_{2.5}$. In one of the only studies of neonates (birth to 27 days after birth), Dales et al. (2006) found that higher O_3 levels were followed by more hospital admissions.

5.3.5.4 Evidence of the impacts of NO_2, O_3 and particulate matter exposure on cardiovascular disease

Air pollutants can affect the cardiovascular system through several mechanisms, including oxidative stress and inflammation which can lead to the rupture of plaque, effects on the coronary blood vessels, and effects on the autonomic nervous system which controls heart rate.

Possible effects of NO_2 on cardiovascular disease (CVD) have been studied using hospital and emergency department admission data. A large number of studies found that mean 24-hour NO_2 levels or 1-hour maximum levels related to the number of CVD admissions (Metzger et al. 2004; Tolbert et al. 2007; Anderson et al. 2007; Jalaludin et al. 2006). When only cardiac diseases were considered, almost all studies found statistically significant associations (von Klot et al. 2005; Simpson 2005; Chang et al. 2005). Studies examining correlations between NO_2 and stroke were mixed; overall there was little evidence of an effect.

Several toxicological studies provide evidence that O_3 exposure could be related to cardiovascular problems. While the studies linking O_3 exposure and CVD morbidity did not show a linkage, large multi-country studies (Katsouyanni et al. 2009), and a multi-city study in the US (Zanobetti and Schwarts 2008) found that during the warm season, short-term increases in O_3 were followed by increases in deaths due to cardiovascular disease.

Particulate matter is the air pollutant that appears to have the greatest impact on CVD (Wellenius et al. 2012). The US EPA has concluded that both short-term and long-term exposures to $PM_{2.5}$ are causally related to cardiovascular effects, and that "a causal relationship is likely to exist" between cardiovascular disease and larger particles (USEPA 2009). This conclusion was based on the large number of studies, toxicological, clinical and epidemiological, that have observed impacts of particulate matter exposure on many aspects of cardiovascular health. People with existing CVD and the elderly appear to be at higher risk (Brook et al. 2010).

723 Over 20 studies have studied the effects of particulate matter exposure on heart rate variability
724 (HRV, a risk factor for arrhythmias and heart attacks) and most have found exposure to reduce HRV
725 (Brook 2008). Some studies have found effects of particulate matter, O_3 and NO_2 on arrhythmia, rapid
726 heart rate (tachycardia), ECG abnormalities, increased blood pressure, thrombosis, inflammation
727 markers, and coagulation factors (Brook 2008). For these outcomes the results have been inconsistent,
728 with some studies showing an effect of particulate matter exposure (USEPA 2009). Several studies have
729 shown a relationship to atherosclerosis (Brook 2010; Brook et al. 2010). A number of studies have also
730 examined the relationship between long-term particulate matter exposure (1-5 years) and CVD, with
731 most observing positive associations (Miller et al. 2007, Baccarelli et al. 2008, Zanobetti and Schwartz
732 2007). For example, Miller et al. (2007) observed significant effects on stroke, cerebrovascular disease,
733 and all CVD using a prospective study. Likewise, Baccarelli et al. (2008) found significant effects on deep
734 vein thrombosis (DVT), while Zanobetti and Schwartz reported higher rates of hospitalization for heart
735 attacks among survivors of an initial heart attack in 21 US cities.
736 Examining the temporal relationship between short-term particulate matter levels and
737 subsequent emergency department visits or hospital admissions is the most common study design used
738 to look at the risk of particulate matter exposure. There are many of these studies, and recent studies
739 include large populations from multiple cities and countries. Almost every study looking at all CVDs
740 observed a significant association with recent particulate matter levels (Brook 2007; USEPA 2009).
741 Several studies established associations with hospital/ED admissions for specific conditions, including
742 heart attacks, ischemic heart disease (lack of blood flow to the heart muscle), and congestive heart
743 failure. Authors of a review of 49 studies concluded that there was evidence that exposure to particulate
744 matter for less than a day can lead to ischemia and heart attacks, especially among the elderly and those
745 who already have heart disease (Burgan 2010). A few studies have tried to determine the form of the

746 concentration-response relationship between particulate matter and CVD. As with many of these
747 studies, Zanobetti and Schwartz (2005) found an almost linear relationship with no threshold, that is,
748 even very low levels of PM_{10} were associated with an increase in the number of hospital admissions for
749 heart attacks.
750

751 ***5.3.5.5 Evidence of the impacts of NO_2, O_3 and particulate matter exposure on cancer***

752 Exposure to NO_2 is hypothesized to lead to the formation of nitrosamines in the lung. Nitrosamines
753 resulting from ingesting nitrate or nitrite have been shown to be carcinogenic (IARC 2010). Only two
754 studies have examined the effect of NO_2 exposure on the risk of developing lung cancer, however both
755 found significant relationships (Nafstad et al. 2003; Nyberg et al. 2000). O_3 and particulate matter are
756 also thought to possibly be involved in the carcinogenic process, possibly through their effects on cells
757 or DNA. One recent, large study looked at O_3 and overall cancer deaths, finding no association (Krewski
758 2009). This same study did see effects of $PM_{2.5}$ and lung cancer mortality. Many other studies did not
759 produce significant results.
760

761 ***5.3.5.6 Evidence of the impacts of NO_2, O_3 and particulate matter exposure on birth outcomes***

762 Over 20 studies have examined the prevalence of low birth weight (LBW) delivery (<2,500 gm or 5.5
763 lbs.), pre-term delivery (PTB, < 37 weeks) or babies that are small for the number of weeks of gestation
764 (SGA) with ambient air pollutant levels. Almost none of the studies looking at O_3 saw any evidence of an
765 association (Shaw and Balkhair 2010; Stilerman et al. 2008), the notable exceptions being two studies in
766 California which showed effects on birth weight. NO_x exposure was associated with LBW, PTB or SGA in
767 a small number of studies; no effect was seen in most of the studies (USEPA 2008b; Shaw and Balkhari

2010). A California-wide study observed a small but statistically significant increase in LBW with increasing NO_2.

There are several studies which have shown reproductive impacts associated with exposure to $PM_{2.5}$. A study in southern California, limited to women who lived close to an air monitor, found a significant increase in the risk of a LBW baby and a PTB (Wilhelm and Ritz 2000). Data from the Child Health Study in Southern California did not indicate an impact on birth weight by PM_{10}, but a clear association exists with O_3 levels, even when accounting for the other air pollutants (Salam et al. 2005). A California-wide study of births, limited to those at 40 weeks gestation and residence near a monitoring station, did find reductions in birth weight (Parker et al. 2005). A 2007 study in Southern California interviewed the mothers to obtain information on other risk factors for PTB; their results demonstrated a strong effect of $PM_{2.5}$ exposure (Ritz et al. 2007).

While there are relatively few studies of air pollutants and stillbirth or infant death, two studies in California found recent increases in PM_{10} levels to be associated with an increased risk of childhood deaths (Ritz et al. 2006; Woodruff et al 2006; Schwartz 2005). Other studies have not found significant effects. There are few studies and little evidence of any effect of O_3 or NO_x on infant mortality or stillbirth. There are too few studies of the effects of air pollution on birth defects to draw any conclusions.

5.3.5.7 Evidence of the impacts of NO_2, O_3 and particulate matter exposure on mortality

The association of NO_2 exposures and mortality (death) rates represents the overall impact of daily and long-term exposures. The best of these studies include populations from several cities. Five out of six of these studies found some association with either all deaths or deaths just due to cardiovascular or

790 respiratory causes (Brook et al. 2007; Samoli et al. 2006; Simpson et al. 2005; Hoek et al. 2002; Stieb et
791 al. 2003; Burnett et al. 2004; Dominici et al. 2003).

792 At least 15 studies over the last 10 years have examined the relationship of short-term O_3 levels
793 and all non-accidental deaths; every study documented such effects, with the largest effects occurring
794 during the warm months. Many of these were large, multi-city studies. Most of these studies also looked
795 specifically at deaths due to respiratory and cardiovascular causes; deaths due to these causes were also
796 related to O_3 levels (Brook et al. 2010; McClellan et al. 2009). Two studies (Bell et al. 2006; Katsouyanni
797 et al. 2009) examined the concentration-response relationship and found that excess deaths occurred at
798 O_3 levels below the regulatory limits for the US. Bell et al. (2006) found that even low levels of O_3 are
799 associated with increased risk of premature mortality and that the risk of mortality is statistically
800 significant with daily average O_3 concentration above 80 $\mu g/m^3$.

801 Many studies of particulate matter and mortality, including large numbers (thousands to
802 millions) of people across cities in the US and abroad, provide consistent evidence of the effects of
803 short-term and long-term exposure to particulate matter (Dockery et al. 1993; Brook et al. 2010; EPA
804 2009). A study of 8011 adults from six US cities (Topeka KS, Stubenville OH, St. Louis MS, Harriman TN,
805 Watertown MA, and Portage WS), which controlled for smoking and other personal factors, found that
806 fine particulate matter was positively associated with death from COPD and lung cancer but not with
807 death from other causes combined (Dockery et al. 1993). In the Brook et al. (2010) and EPA (2009)
808 studies, particulate matter was associated with all causes of death (non-accidental), and was related to
809 greater increases in deaths due to ischemic heart disease, COPD, and cardiovascular disease.
810

811 ***5.3.5.8 Summary of health impacts of NO_2, O_3 and particulate matter***

812 Hundreds of studies of the health impacts of NO_2, O_3 and particulate matter have been conducted since
813 the 1960s, and there is strong overall evidence that exposure to these pollutants leads to respiratory
814 symptoms, reduced lung growth, asthma exacerbations, and respiratory infections (Table 5.3.2). In
815 addition, such exposures are associated with visits to emergency rooms and hospital admissions for
816 respiratory complaints, asthma, COPD, and cardiac problems. Increases in mortality, in general and
817 specifically for respiratory and cardiovascular causes, occur after high ambient pollutant levels. Children,
818 the elderly and people with existing COPD or CVD are more likely to be affected.

819 Many of these epidemiological studies are limited by the way that exposure is measured.
820 Typically when some pollutant levels are high, so are the other air pollutant levels. As such, when there
821 is an association between, say, NO_2 levels and a health outcome, the effect may actually be partly due
822 to exposure to the other pollutants in the air as well. Some studies try to take the other pollutant
823 measures into account, which increases the chance that the observed association is due to the specific
824 pollutant under study. Further, many of the studies simply use observed or predicted ambient pollutant
825 levels near each case's house, and do not account for where they actually were or how long they were
826 indoors.

827 Given the known and suspected relationships of NO_2, particulate matter and O_3 with many
828 health problems, the overall public health impact of these exposures is potentially very high. The
829 California Air Resources Board estimates that each year exposure to $PM_{2.5}$ results in 7,300 excess deaths
830 from cardiopulmonary diseases and 5,500 from ischemic heart disease (for exposures greater than 5.8
831 µg/m3) (CARB 2010). If O_3 was decreased to California's standard, it is estimated that each year 630
832 deaths, 4200 hospital admissions for respiratory diseases, 660 ER visits for asthma, and 4.7 million days
833 of missed school among children would be averted (Ostro et al. 2006).

834

5.3.5.9 Research Needs

As this review has shown, there are many health impacts that result from exposure to NO_2, O_3 and particulate matter in the air. Certainly more studies are needed to determine the extent to which these air pollutants might be related to other diseases, such as birth defects and stillbirth, lung cancer, and respiratory infection. Better information is needed about the actual levels of exposure among the population, and differences in exposure by location, income, race and ethnicity. For almost all of the health impacts associated with air pollutants, much better data are needed describing the relationship between the concentration in the air and the number of people affected. This would help to determine the public health improvements that would come about from changes in air quality standards and air contaminant levels. If the precise, total health effects of nitrogen emissions is desired, then more studies are needed to better quantify the proportion of PM_{10} and $PM_{2.5}$ that are principally made up of nitrogen containing compounds, and the regions and conditions under which such particles are formed. Such data would also be useful if combined with studies estimating the health care costs resulting from health impacts of nitrogen derived air pollutants.

5.3.6 Economics of air quality

Muller and Mendelsohn (2007) estimated air pollution damages of NO_x, NH_3, PM_{10}, $PM_{2.5}$, for the US as a whole from over 10,000 point and aggregated nonpoint sources in the contiguous United States using the Air Pollution Emission Experiments and Policy (APEEP) analysis model. Based on their estimates, damage costs due to mortality account for about 71% ($53 billion) of the total costs and morbidity account for about 23% ($17 billion) of the total costs (Muller and Mendelsohn 2007). Overall, damage costs due to human health account for 94% of total damages (Muller and Mendelsohn 2007). The remaining 6% of this total cost damage include visibility impairment ($2.7 billion), reduction in

agricultural yield ($1.2 billion), reduction in timber production ($80 million), depreciation of manmade materials ($100 million), and diminished forest health (Muller and Mendelsohn 2007). These different types of damage costs are not available specifically for California.

The APEEP model was also used to estimate county-level damage costs associated with mobile, point, and non-point sources based on data from the US EPA's 2002 National Emissions Inventory (Muller and Mendelsohn 2009). Marginal damages are calculated by first calculating the county-level air pollution concentration, exposures, physical effects, and dollar damages with the 2002 baseline emissions level (Muller and Mendelsohn 2007). The damages are recomputed by adding one ton of a pollutant to one source. The resulting difference between the damages from an additional ton of a pollutant and the baseline damages is the marginal damage of the pollutant.

Figure 5.3.12 shows that the highest marginal damage costs (in \$ ton^{-1} of pollutant) due to NO_x in California occurred in San Joaquin, Sacramento, Stanislaus, Yolo, Solano, Napa, Sonoma, Merced, Fresno, and Marin (Muller and Mendelsohn 2009). The marginal damages from NO_x emissions is rather difficult to estimate and hence the spatial pattern of damages is more dispersed and complex. This is due to several important factors. First, since particulate matter formation is limited by the availability of NH_3, NO_x may not contribute to $PM_{2.5}$ if there are little ambient NH_3 available. In such, cases NO_x damages will be minor. Second, NO_x contributes to the formation of O_3 which can reduce crop yield and timber production in rural areas. However, since the formation of O_3 is a non-linear function, characterizing the spatial distribution of both NO_x and O_3 and their impacts on non-health related goods and services remains challenging for researchers (personal communication with author, June 2012).

[Figure 5.3.12]

Damages due to NH_3 and $PM_{2.5}$ had a notably different geographic distribution with Los Angeles, San Francisco, Contra Costa, Orange, Alameda, Santa Cruz, Santa Clara, Marin, and San Diego

881 having the highest marginal damage costs (Figure 5.3.12). Since NH_3 and $PM_{2.5}$ tend to have more
882 serious impacts on human health than on crop and timber production, the marginal damages due to
883 these pollutants tend to be larger (on a per ton basis) in urban areas with large and dense populations.
884 Considerable uncertainty exists in virtually any estimate of the economic damages associated
885 with air pollution. Muller (2011) conducted a statistical analysis of the uncertainty in air pollution
886 damages from 565 electric generating units in the US and found that the largest sources of uncertainty
887 were due to high variance in adult mortality dose-response relationships, mortality valuation and the
888 methods of air quality modeling. Muller (2011) also found that the estimated marginal damage (per ton
889 damage) distributions are positively skewed and are more variable in urban rather than in rural areas.
890 Likewise, the European Nitrogen Assessment (2011) suggested that variation in the estimation of
891 economic damage costs associated with the dose-response function and a lack of comparability among
892 "willingness to pay (WTP) studies" are key sources of uncertainty and thus pose important constraints
893 for economic research.
894
895 ***5.3.6.1 Economic costs of air pollution on human health***
896 The economic impact of air pollution on the health of Californians has received considerable attention in
897 the literature. Indeed some of the earliest research on the subject is based in California (Hall et al. 1992).
898 Economic valuation of the health outcomes resulting from air pollution is typically based on three pieces
899 of information: (1) dose-response relationships; (2) levels of exposure; (3) demographic information (e.g
900 population by age and other characteristics); and (4) economic valuation of health outcomes (e.g.,
901 mortality). First, data from epidemiological studies is developed into dose-response functions that
902 estimate the severity of the outcome for a given level of pollutant. Second, models of population density
903 and mobility are coupled with air quality data and then used to estimate exposure by accounting for

changes in the distribution of the population over space and time. Finally, the dose-response relations and estimated exposures are used to calculate the costs, in this case US dollars (USD), associated with hospital visits, treatment and or mortality for various health outcomes.

It is well established that the estimated health damage costs of O_3 and $PM_{2.5}$ in California are considerable. Romley et al. (2010) examined private and public insurer spending on hospital admissions and emergency room visits related to illnesses with respiratory, cardiovascular, asthma causes. They found that poor air quality which fails to meet NAAQS and CAAQS for O_3 and $PM_{2.5}$ has likely contributed to nearly 30,000 hospital admissions and emergency room visits, and cost Californians more than $193 million between 2005 and 2007 (Romley et al. 2010). Three-quarters of these incidents are attributable to high $PM_{2.5}$ while the rest were due to high O_3 levels.

Much of the economic research in California focuses on health outcomes related to O_3 and $PM_{2.5}$ exposure in the South Coast and San Joaquin Valley Air Basins due to the intensity of the concerns in these locations (Hall et al. 2003). For example, Romley et al (2010) found that the majority of air pollution related hospital visits in California occurred in the SJVAB and SoCAB. They also examined how health and cost effects of air pollution differentiate across the state by income and ethnic group, and found that failing to meet NAAQS had the greatest effect on Hispanic and African Americans communities, with most of the patients poor. Consequently, they suggest a disproportionate share of the cost burden falls on public insurers, such as Medicare and Medi-Cal (Romley et al. 2010). Hall et al. (2010) estimated the economic costs in both areas and found that the incidence of O_3 and $PM_{2.5}$ combined is estimated to cause nearly 6 billion \$ yr^{-1} (2007 \$) worth of negative health effects in SJVAB and 19 billion dollars/year (2007\$) in the SoCAB (Table 5.3.4), of which 98% results from $PM_{2.5}$ exposure basin-wide. The authors note that these values likely underestimate actual cost because of the inability to catalog or quantify all adverse effects suggested to result from exposure in economic terms.

927 [Table 5.3.4]

928 Rather than estimating the costs, Ostro et al. (2006) examined the economic health benefits
929 from a decrease in the incidence of premature morality, hospital admissions, emergency room visits,
930 lost school days, minor reduced activity days of attaining the national and the more restrictive 8-hour O_3
931 levels throughout California. The authors found that this would result in benefits equaling 2.8 billion
932 dollars per annum (2000 $) and 4.5 billion dollars for obtaining the NAAQS and CAAQS, respectively.

933 Children (aged 5-18) are generally more vulnerable than adults when exposed to poor air quality
934 and have been identified to be one of the groups most sensitive to high levels of O_3 (Hall et al. 2003).
935 Utilizing advances in health research on the association between O_3 and school absences by Gilliland et
936 al. (2001), Hall et al. (2003) estimated the economic benefit of a reduction in school absences due to
937 improvements in O_3 levels for children aged 5-18 in the SoCAB. The authors estimated the number of
938 children who would have been exposed to specific O_3 concentrations above the California air quality
939 standard and found that the estimated exposure is on a downward trend from the 1990-99 period and
940 has subsequently led to a decrease in O_3-related illness school absences in the SoCAB (Table 5.3.5). The
941 total economic value (benefit), expressed in 2000 $, of differences in annual eight-hour O_3-related all-
942 illness school absences for the 1998 population aged 5-18 from the 1990-92 to 1997-99 period was $245
943 million (with a range of $156 million to almost $344 million annually) in the SoCAB (Hall et al. 2003)
944 (Table 5.3.5; Table 5.3.6). This is a benefit of $75 on a per capita basis for the entire SoCAB (Table 5.3.6).
945 Los Angeles ($147, 689,000) and San Bernardino ($45,666,000) counties would benefit the most from a
946 reduction in school absences (Table 5.3.6). On a per-capita basis, Riverside ($91) and San Bernardino
947 ($114) counties would benefit the most (Table 5.3.6).

948 [Table 5.3.5]

949 [Table 5.3.6]

Due to limited availability of data on medical treatments associated with school-related absences, the cost estimates of the economic benefit of a reduction in school absences by Hall et al. (2003) only included indirect costs (i.e., the value of the caregiver's time). Brandt et al. (2012), on the other hand, considered both the direct and indirect healthcare costs of childhood asthma (including bronchitis symptoms) as a result of exposure to traffic-related pollution (TRP) (e.g. ,NO_2 and O_3) in Riverside and Long Beach in Southern California. These two communities were selected for their study because the primary source of high levels of regional air pollution is due to traffic and residential neighborhoods are in close proximity to high volume traffic corridors. The direct healthcare costs include the costs of bronchitis episodes, ED visits, and hospitalization whereas the indirect costs include the value of time loss (e.g., forgone income) by the parents/caregivers as a result of the child's asthma-specific office visits and school absences due to air pollution (i.e., 8-year mean concentrations of NO_2 and O_3 (1996-2004)). The value of time loss is calculated based on the median household income and the average number of hours worked per year in Riverside and Long Beach respectively (Brandt et al. 2012).

They estimated that the annual cost for a case of childhood asthma-related due to TRP is $4,008 (e.g. 7% of the median household income) in Riverside and $3,819 (8% of the median household income) in Long Beach; with indirect costs due to the child's school absences making up the largest share of the cost (34% for Riverside and 32% for Long Beach) (See Table 5.3.7). The total annual costs of the TRP-related asthma cases were estimated to be $2,808,300 and $6,120,000 (in 2010$) in Riverside and Long Beach respectively (Brandt et al. 2012). Since the populations in these two communities only constitute about 7% of the total population in California, applying the same methodology to calculate the statewide healthcare cost due to NO_2 and O_3 will yield a substantially larger figure than what has been presented in this study (Brandt et al. 2012).

[Table 5.3.7]

Historical comparisons of the economic benefits of controlling air pollution must be made with caution. Scientific advances in understanding concentration-response functions, population growth and mobility (exposure), and economic valuation can make assessments of change dubious. Hall et al. (2010) conducted one of the only existing rigorous comparisons of a 1989 and a 2008 air quality benefit assessment of the economic outcomes and health benefits of O_3 and $PM_{2.5}$ reductions for MRADs and premature deaths, respectively for the SoCAB. Their results show that human exposure to O_3 and $PM_{2.5}$ concentrations between 1989 and 2008 has declined dramatically (Hall et al. 2010). Furthermore, while the health literature is constantly evolving the real economic unit values estimated for adverse health outcomes associated with exposure to concentrations these air pollution has stayed relatively constant (Hall et al. 2010).

5.3.6.2 Economic costs of air pollution on crop production

Air pollution can also have important impacts on agricultural crops and native vegetation. In this section, we focus on the agricultural impacts of N-related air pollutants, namely O_3 and acid precipitation, because of the subsequent implications for the economic wellbeing of California's agricultural community. The impact of N-related air pollutants on native vegetation is dealt with later in Section 5.5., which addresses how N pollution affects biodiversity in native terrestrial and aquatic ecosystems.

Effects of O_3 and acid precipitation on crops

With several recent reviews now available in the scientific literature, the damaging effects of O_3 on agricultural crops are well established (Sandermann 1996; Benton et al. 2000; Fuhrer and Booker 2003; Booker et al. 2009; Emberson et al. 2009). Plant injury occurs when O_3 enters the plant leaf cells via the

stomata, where it can oxidize and degrade cell membranes, pigments and proteins. Common visible symptoms include early leaf senescence, leaf chlorosis, and lower root mass fractions (Thomas 1951; Grulke et al. 1998; Felzer et al. 2007; Wang and Taub 2010). Leaf tissue damaged by O_3 exposure tends to have lower CO_2 assimilation capabilities, caused by a decrease in Rubisco activity (Felzer et al. 2007; Grantz et al. 2006; Booker et al. 2009). Exposure to O_3 can also reduce drought tolerance (Feng et al. 2008), increase disease vulnerability (Calvo et al. 2007), and decrease the yield of many grain and vegetable crops (Bender et al. 1999; Hassan et al. 1999; Holmes and Schultheis 2003; Feng and Kobayashi 2009).

In California, the high levels of O_3 occurring in regions of high agricultural productivity suggest that California's agricultural economy is particularly vulnerable to O_3 (Muller and Mendelsohn 2009). For example, O_3 pollution in the San Joaquin Valley is most acute in the summer months during the peak agricultural season. Important California crops that are known to be vulnerable to high O_3 concentrations include alfalfa (Mutters and Soret 1998), almonds (Retzlaff et al. 1990), apples (Retzlaff et al. 1992), beans (Mutters and Soret 1998), cantaloupe (Mutters and Soret 1998), citrus (e.g., oranges, lemons; Mutters and Soret 1998), cotton (Grantz 2003), grapes (Mutters and Soret 1998), lettuce (Heck et al. 1982), onions (Mills et al. 2007), rice (Sawada and Kohno 2009), stone fruit (e.g., apricots, plums, prunes; Retzlaff et al. 1992) , pears (Retzlaff et al. 1992), potatoes (Vorne 2002), tomatoes (Calvo et al. 2007),and wheat (Feng et al. 2008). In general, the regions of California with a combination of severe O_3 pollution and high agricultural production value (e.g., San Joaquin Valley) have received the greatest attention among researchers (Rowe and Chestnut 1985; Grantz and Shrestha 2005). However, since toxicity can occur on sensitive species at relatively low O_3 concentrations, damages due to minor yield losses in other regions are thought to be widespread (Grantz 2005).

In California, the extent of yield reduction directly attributable to O_3 is highly uncertain for most crops, but estimates suggest that O_3-related yield losses in excess of 10% are not uncommon for particularly sensitive crops (Table 5.3.8; Murphy et al. 1999; Mutters and Soret 1998). Yield reductions for each crop varied widely, depending on species tolerance, severity of exposure, and location with the greatest estimated losses predicted in the San Joaquin and South Coast Air Basins. For example, yields of cantaloupe and table grapes were reduced by approximately 33 and 30% respectively, while fresh market tomatoes were less than 1% (Table 5.3.8; Mutters and Soret 1998). These differences in crop sensitivity add considerable uncertainty to econometric efforts to model losses in crop yield and revenue.

[Table 5.3.8]

Several early literature reviews conducted by Irving (1983) and Jacobson (1984) examining the effects of acid precipitation on plants concluded that the level of acidity in precipitation and fog is seldom sufficient to cause acute injury to crops and natural vegetation. That said, several studies carried out in California indicate that crops may suffer injury from exposure to acidic fog if the pH of water droplets is low enough. For instance, Bytnerowicz et al. (1985) observed symptoms of foliar injury when pinto bean plants were exposed to simulated acidic fog (containing both nitric acid and sulfuric acid) with a pH of 2.4 or less for 8 hours. These results were similar to Granett and Musselman (1984) who observed increasing severity of plant injury when potted lettuce was exposed to a single exposure to acidic fog at a pH of 2.5 and below. In field grown alfalfa, Temple et al. (1987) found symptoms of foliar injury following repeated exposure to simulated fog with a pH of 2.0, and significant reductions in crop growth at pH levels as high as 3.2. These findings are also similar to Takemoto et al. (1987) who measured large declines in vegetative growth (25% reduction in stem dry weight; 23% reduction leaf dry weight) and fresh fruit weight (58% reduction) for field-grown green pepper following repeated

exposure to fog-water with a pH of 1.68 relative to a neutral control (pH = 7.24). Thus, while acidic fog (and to a lesser extent rain pose) a possible threat to crop growth, the risks are likely to be relatively small under ambient field conditions.

Effects of O_3 on costs to agricultural producers and consumers

The economic impact of increasing concentrations of O_3 has most often been assessed in terms of lost revenue to agricultural producers due to lower yields (Howitt et al. 1984; Howitt and Goodman 1989; Kim et al. 1998). To estimate economic impacts, biophysical dose-response models calibrated with crop-specific data from empirical experiments are coupled with economic models. Using the economic model developed by Howitt et al. (1984), Rowe and Chestnut (1985) estimated that between $42 and $117 million in economic benefits from improved crop yield in the San Joaquin Valley could be achieved if California's state air quality standards for O_3 were attained. A study by Howitt and Goodman (1989) that used a similar modeling approach suggested that various policy response scenarios to control O_3 concentrations in California could yield between $50 and $333 million yr^{-1} in economic benefits, with approximately half of those benefits going to agricultural producers.

Since higher food prices are also an outcome of lower crop production, the negative consequences of O_3 exposure on crops are not just limited to producers (Howitt et al. 1984). While California producers carry the majority of the O_3-related costs due to crop losses, consumers are thought to bear between 25% and 50% of the costs (Kim et al. 1998; Howitt and Goodman 1989). Results from Kim et al. (1998) suggest that the benefits of reducing O_3 pollution for crop producers and consumers will likely to range from $50 to $400 million yr^{-1} depending on the level of O_3 reduction achieved (Figure 5.3.13).

[Figure 5.3.13]

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1821 **Figure 5.3.1. Source and chemical links between ozone and PM formation.** Major precursors
1822 are shown in boxes with thick sides. Secondary particle components are shown in boxes with
1823 thin solid sides. Mobile sources (cars, trucks, and off-road vehicles) and plants are major
1824 sources of VOCs, and mobile sources and electricity-generating units are dominant sources of
1825 NO_x, but myriad smaller sources also contribute. Trace species, such as OH, are crucial to the
1826 formation of ozone, sulfate, nitrate, and organic-carbon particulate matter. Ozone also leads to
1827 the oxidation of SO_2 and NO_2. Biologic activity and fertilizer use dominate ammonia (NH_3)
1828 emissions. Source: NRCNA 2008. [Return to text]

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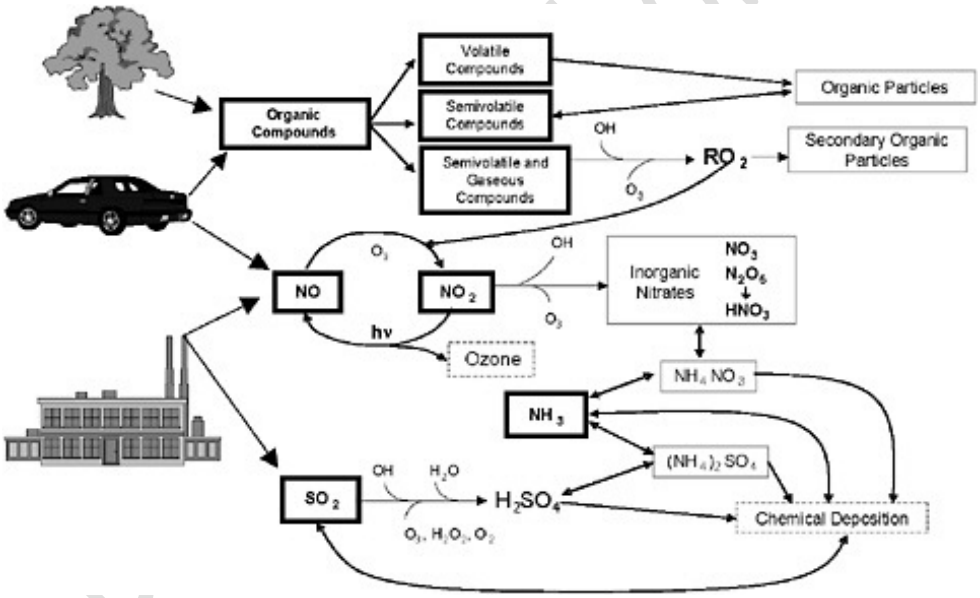

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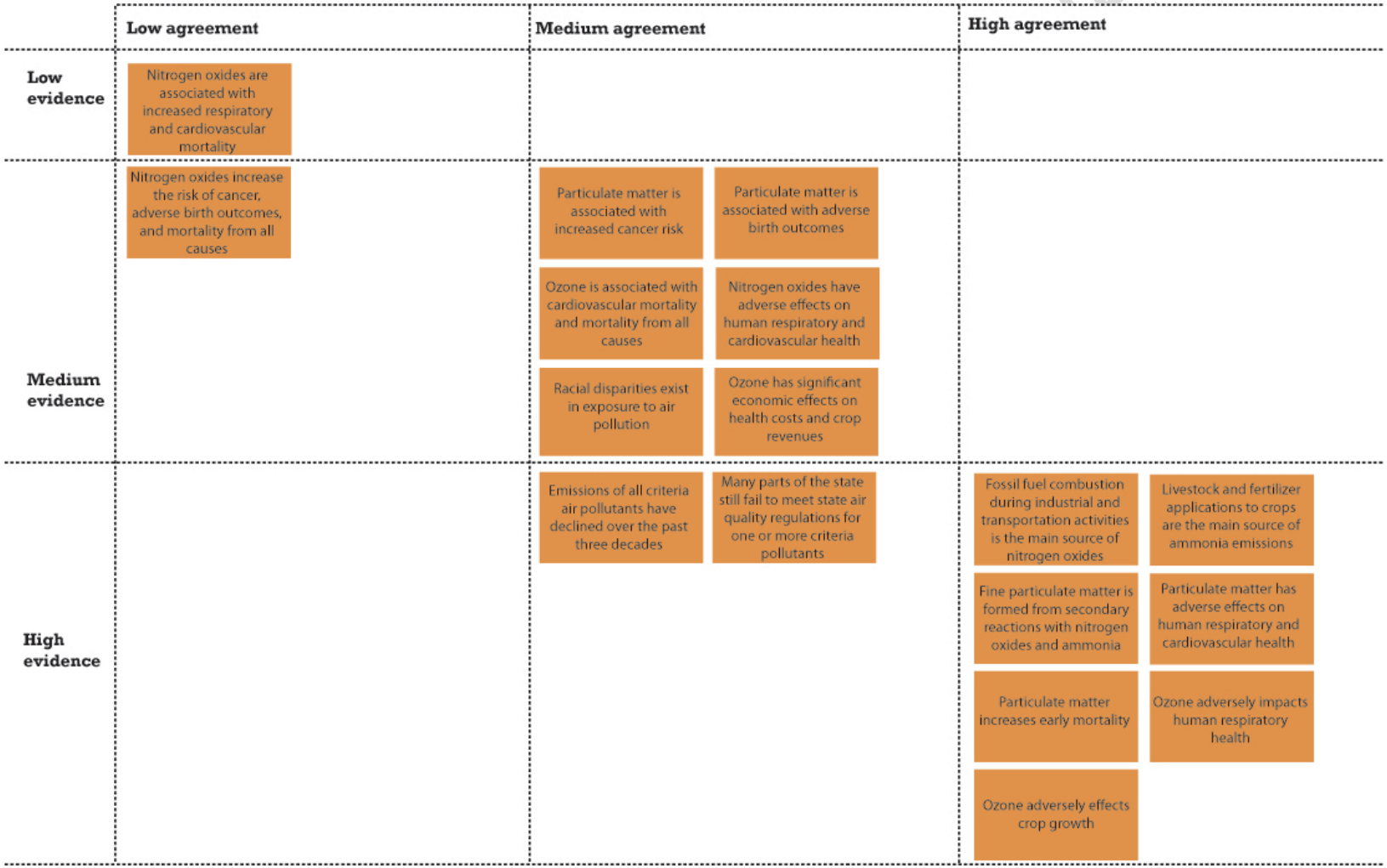
1834 **Figure 5.3.2. Types of uncertainty in nitrogen's impact on air quality and human health in California**. This figure reflects the

1835 amount of evidence and level of scientific agreement for the effects of various nitrogen-related air pollutants. [Return to text]



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1837 **Figure 5.3.3. Trophospheric NO_2 columns for the months of June (2005-2008) along**

1838 **the West Coast of the USA.** Satellite observations were made using a scanning imaging

1839 absorption spectrometer. Source: Adapted from Kar et al. 2010. [Return to text]

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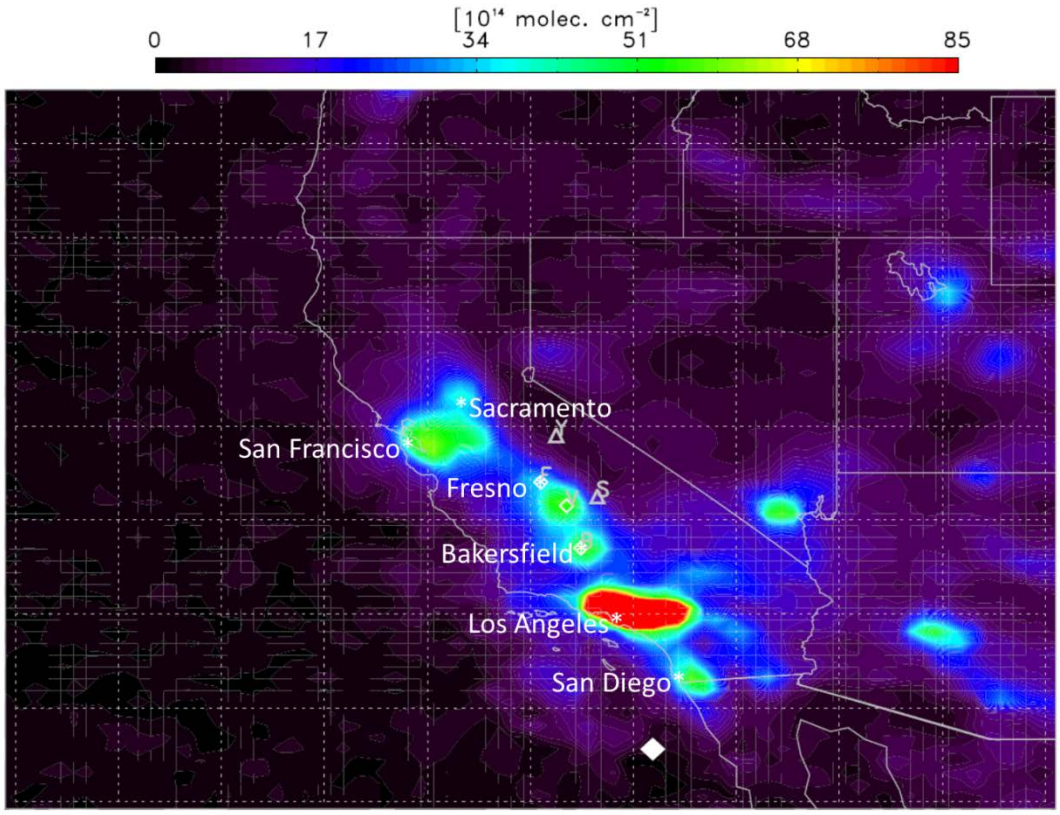

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Figure 5.3.4. Air quality trends in the Los Angeles urban area of California. As per national standards, the O_3 data (8-h average) are 3-yr averages of the 4th highest annual maxima, the CO data (8-h average) are annual maxima, the NO_2 data are annual averages, and the $PM_{2.5}$ data (24-h average) are annual 98th percentiles. Data are derived from monitoring stations in the Southern California Air Basin region. Source: Parrish et al. 2011. [Return to text]

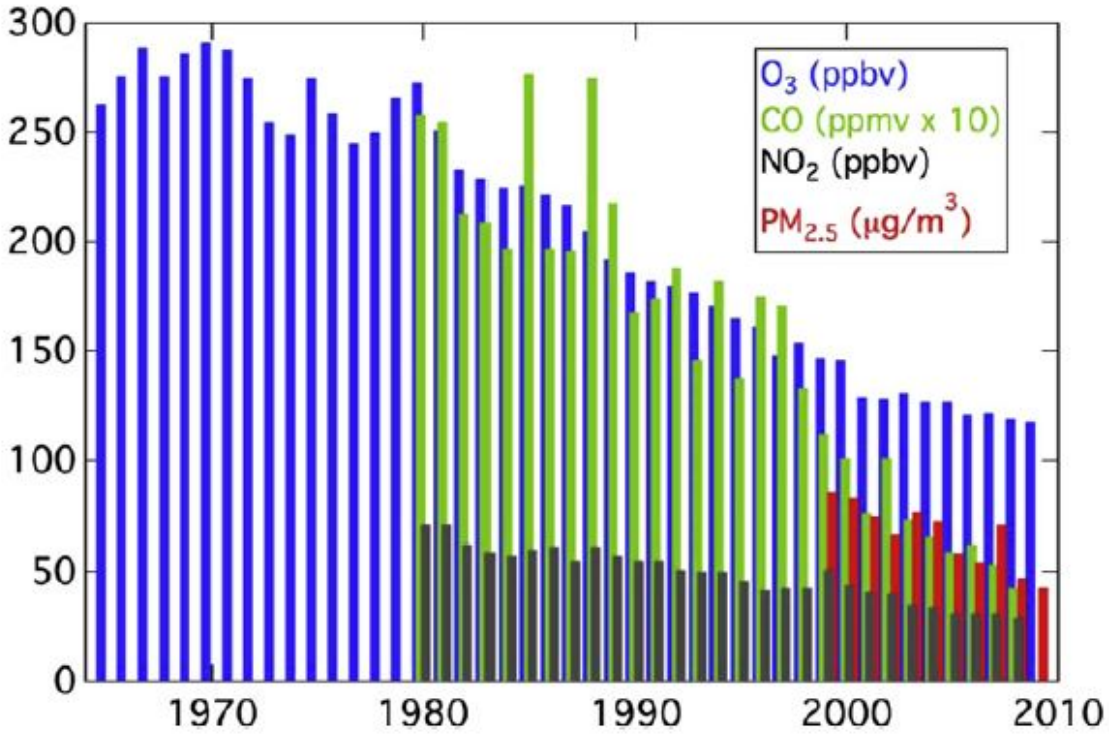

Figure 5.3.5. Trends in NO_2 concentrations through California (2005-2008). Average tropospheric NO_2 column concentrations (molecules/cm^2) from a satellite O_3 monitoring instrument (OMI) (solid) and surface measurements from the CARB (dashed) for weekdays (dark) and weekends (light). Study areas include (a) Sacramento County, (b) the San Francisco Bay Area, (c) the San Joaquin Valley, and (d) the South Coast regions of California. The summer trends are shown by the solid black lines. Note that figures are on different scales to make seasonal cycles visible. Source: Russell et al. 2010. [Return to text]

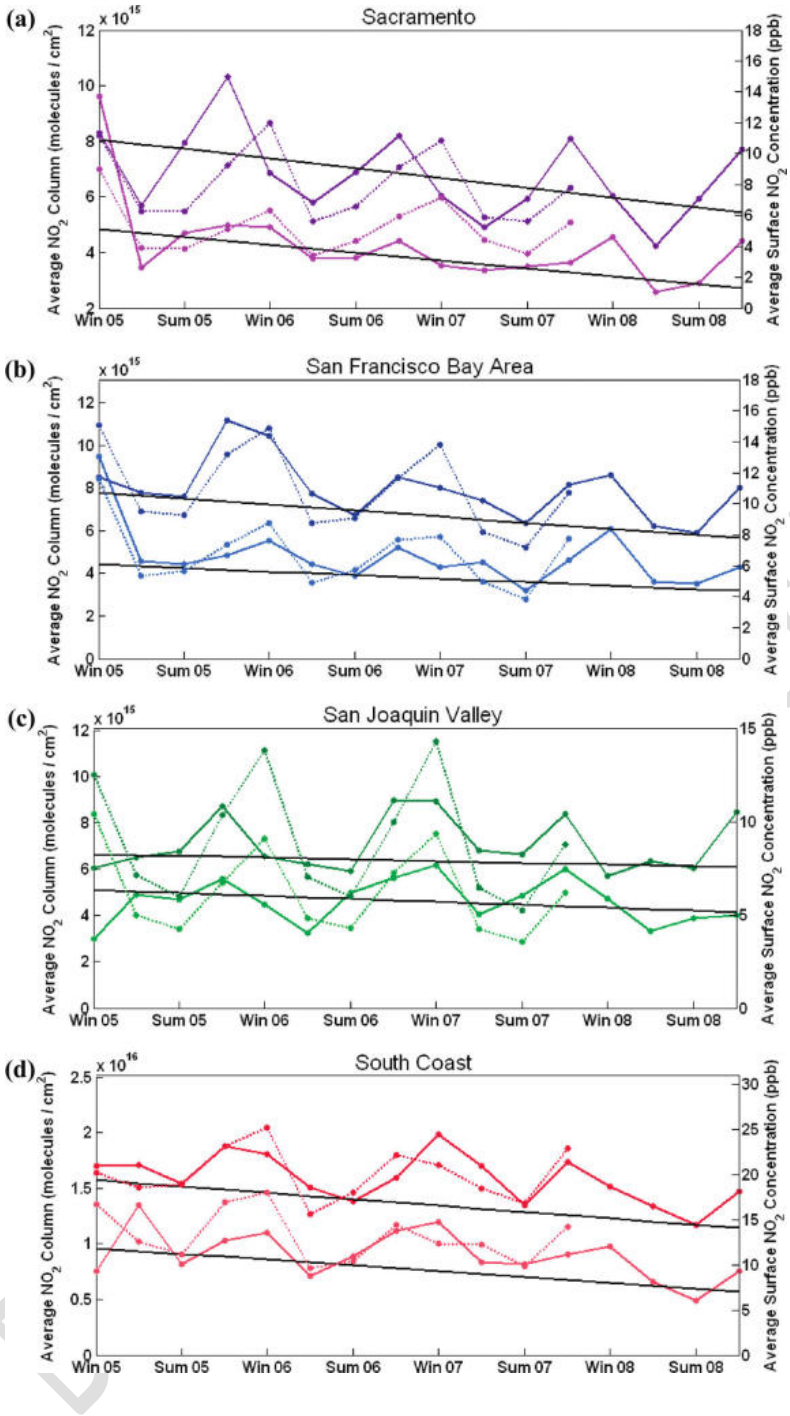

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Figure 5.3.6. Annual averaged NH_3 columns (mg m^{-2}) over the San Joaquin Valley in 2008. Data are derived from satellite observations retrieved using an infrared atmospheric sounding interferometer (IASI). Source: Adapted from Clarisse et al. 2009.

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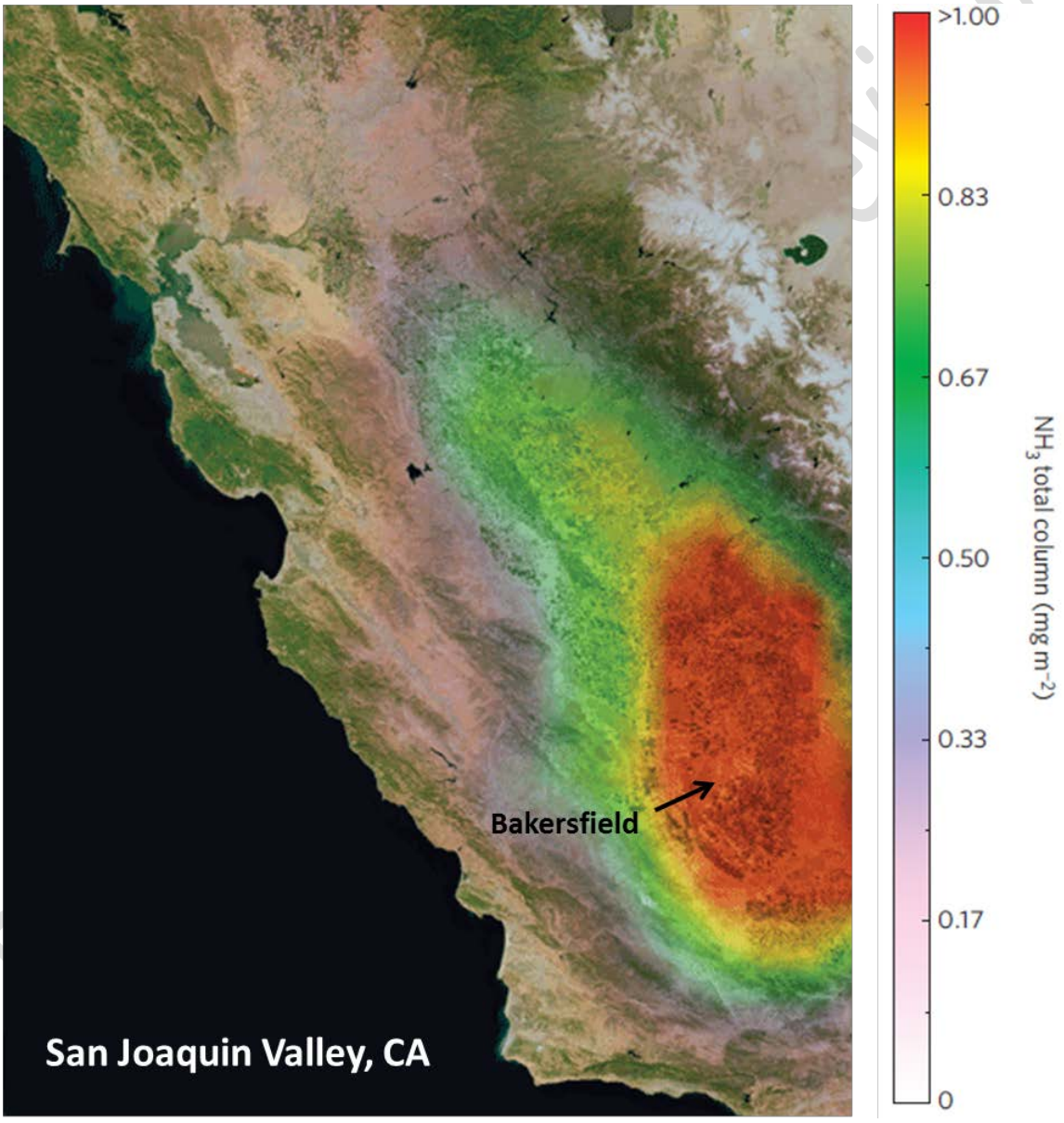

1877 **Figure 5.3.7. Satellite-derived $PM_{2.5}$ and comparison with surface measurements.** (A)
1878 Mean satellite-derived $PM_{2.5}$ between 2001 and 2006 calculated using data retrieved
1879 from a moderate resolution imaging spectroradiometer (MODIS) and multi-angle
1880 imaging spectroradiometer (MISR); white space denotes water or < 50 AOD
1881 measurements. (B) Average coincident values of both measured and satellite-estimated
1882 $PM_{2.5}$. The solid black line denotes unity; thin dotted lines show uncertainty of ± (1
1883 $\mu g/m^3$ + 15%); and the dashed line represents the best fit (Hirsh and Gilroy 1984). (C)
1884 Positions and mean values of coincidently measured surface sites. Source: Van
1885 Donkelaar et al. 2010. [Return to text]

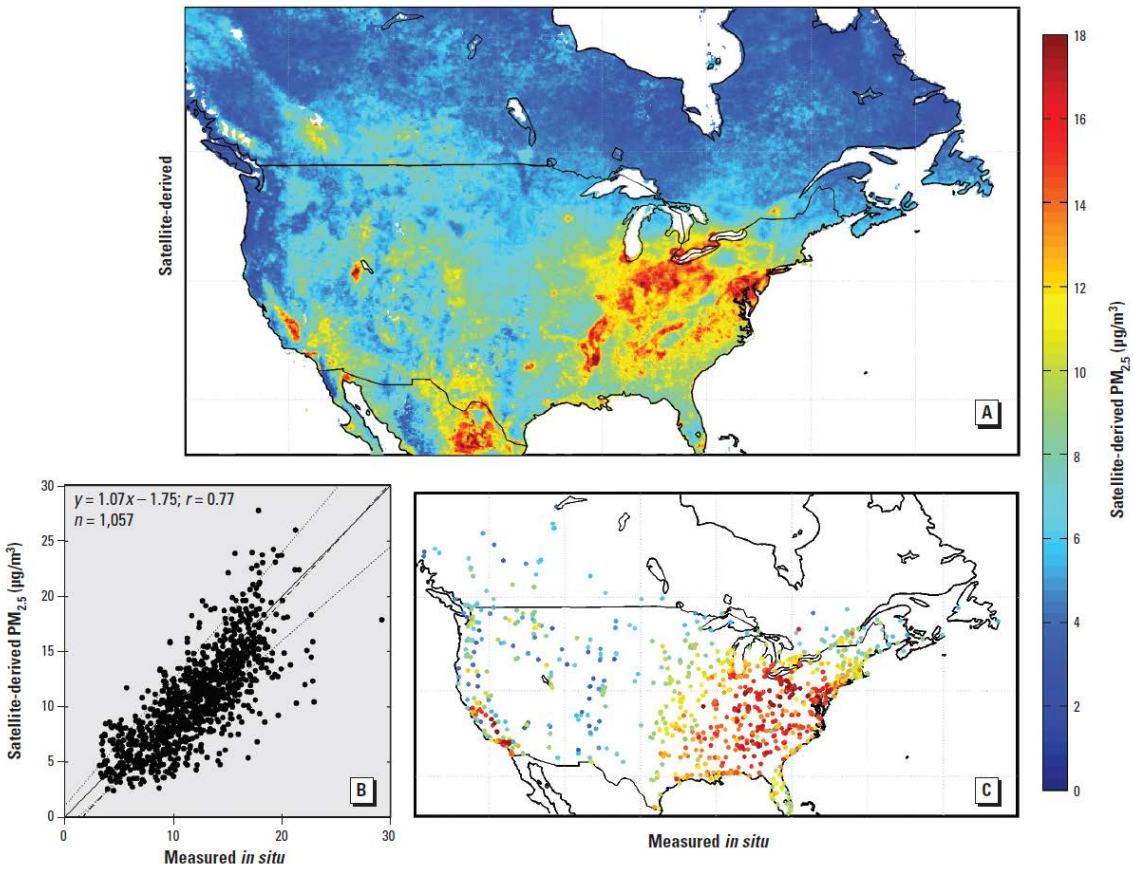

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Figure 5.3.8. Air quality attainment status in California for NO_2, O_3, PM_{10} and $PM_{2.5}$ (2007-2009). Attainment is based on California Ambient Air Quality Standards (CAAQS). Source: CARB 2011.

[Return to text]

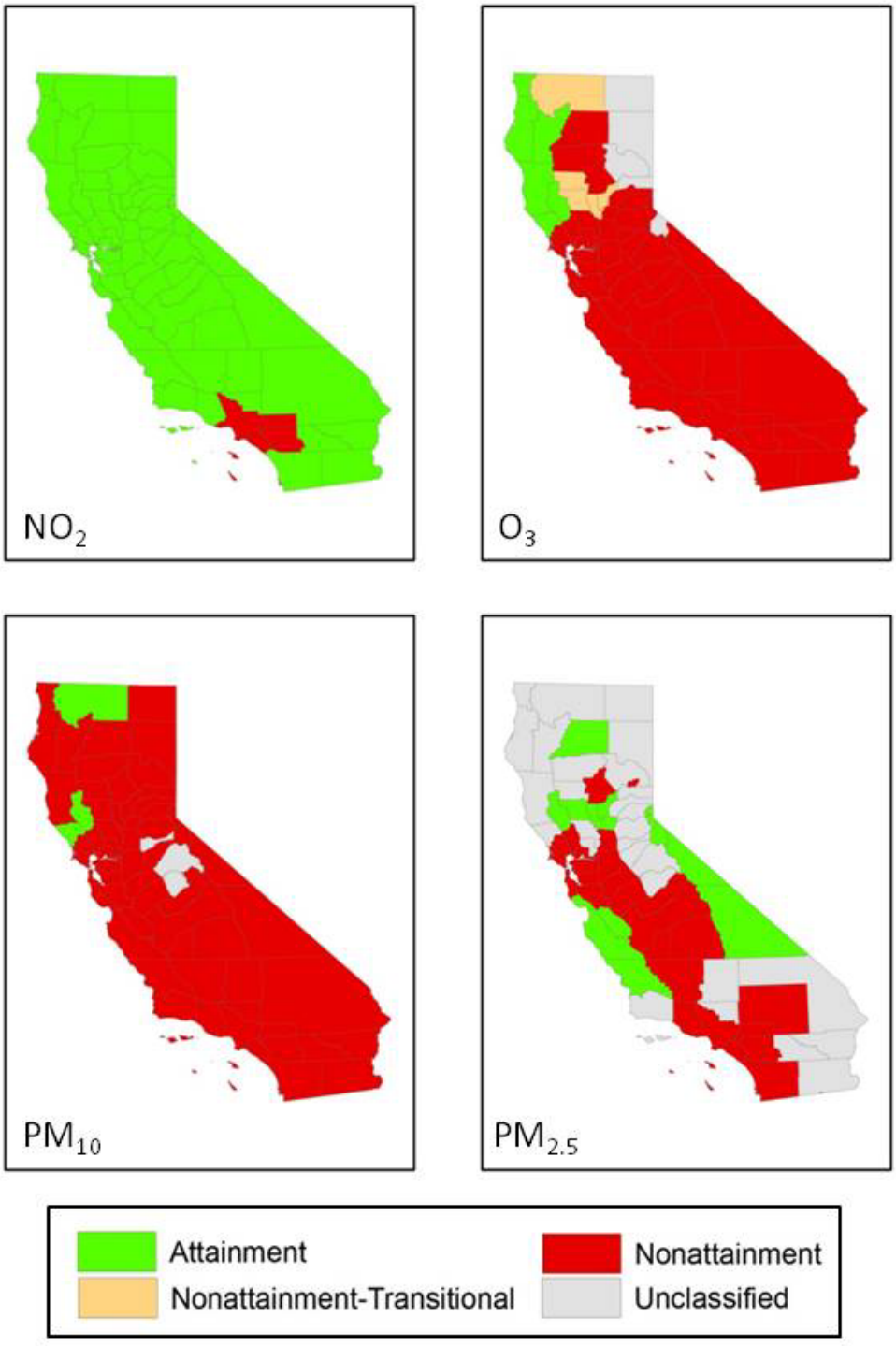
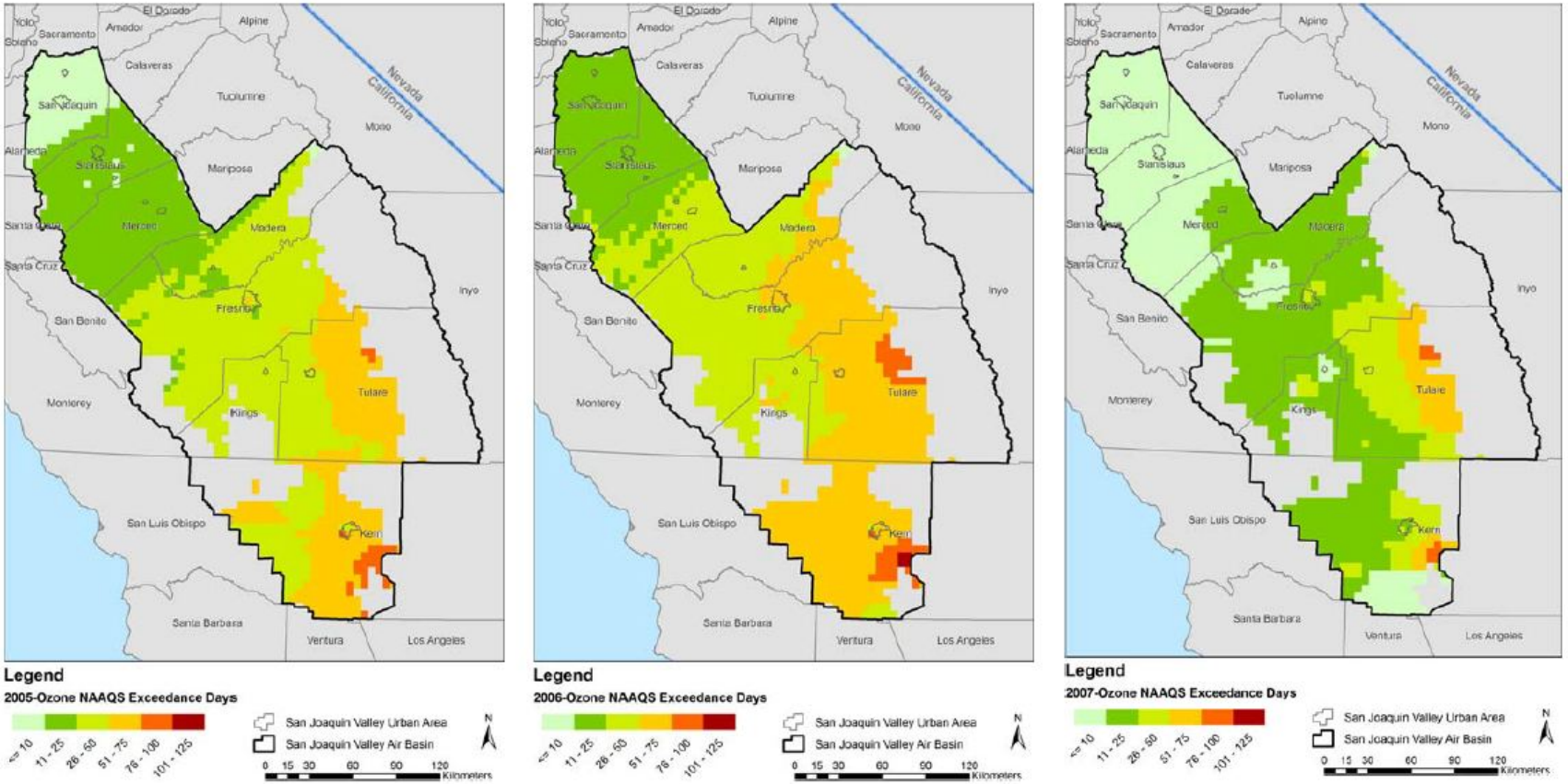


Figure 5.3.9. Spatial distribution of days exceeding the California air quality standard for O_3 in the San Joaquin Valley Air Basin (2005–2007). Maps indicate the number of days per year that the 8-hr daily maximum ozone exceeded 75 ppb in the San Joaquin Valley Air Basin in 2005 (left), 2006 (middle), and 2007 (right). Source: Hall et al. 2008. [Return to text]


Legend
2005-Ozone NAAQS Exceedance Days
<= 10
11 - 25
26 - 50
51 - 75
76 - 100
101 - 125
San Joaquin Valley Urban Area
San Joaquin Valley Air Basin
0 15 30 60 90 120
Kilometers
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2006-Ozone NAAQS Exceedance Days
<= 10
11 - 25
26 - 50
51 - 75
76 - 100
101 - 125
San Joaquin Valley Urban Area
San Joaquin Valley Air Basin
0 15 30 60 90 120
Kilometers
N
Legend
2007-Ozone NAAQS Exceedance Days
<= 10
11 - 25
26 - 50
51 - 75
76 - 100
101 - 125
San Joaquin Valley Urban Area
San Joaquin Valley Air Basin
0 15 30 60 90 120
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1899 **Figure 5.3.10. Days with O_3 levels in California above the 8-hour national ambient air quality**
1900 **standard (NAAQS) in 2009.** The color of the counties and the monitoring stations (depicted by
1901 the dots) both correspond to the number of days above the national O_3 standard.
1902 Source: EPA Air Quality System Data Mart. [Return to text]
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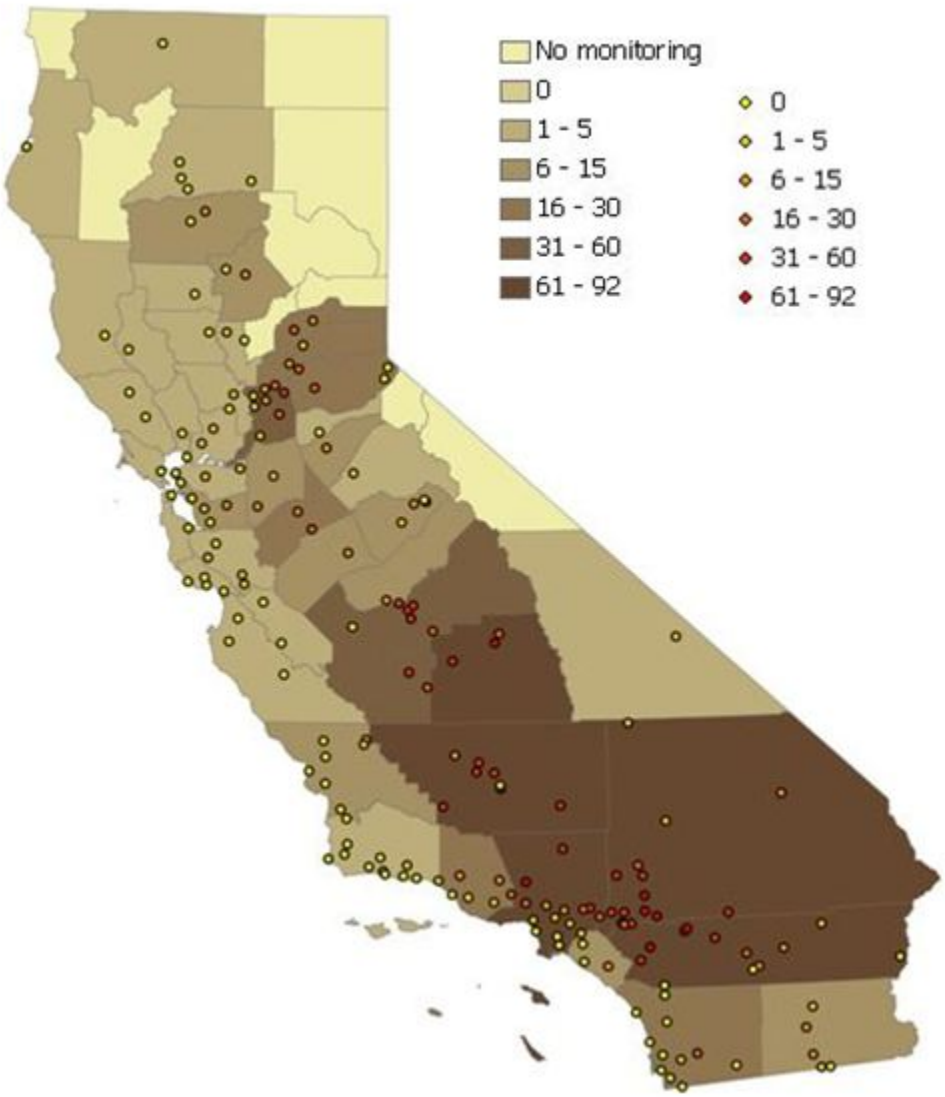

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1906 **Figure 5.3.11. Percent of ethnic populations (White, Other Race, Hispanic, Black) exposed to**
1907 **average annual $PM_{2.5}$ in excess of the NAAQS (>15 µg/m^3).** Data are from the San Joaquin and
1908 South Coast Air Basins Source: Adapted from data by Hall et al. 2008. [Return to text]
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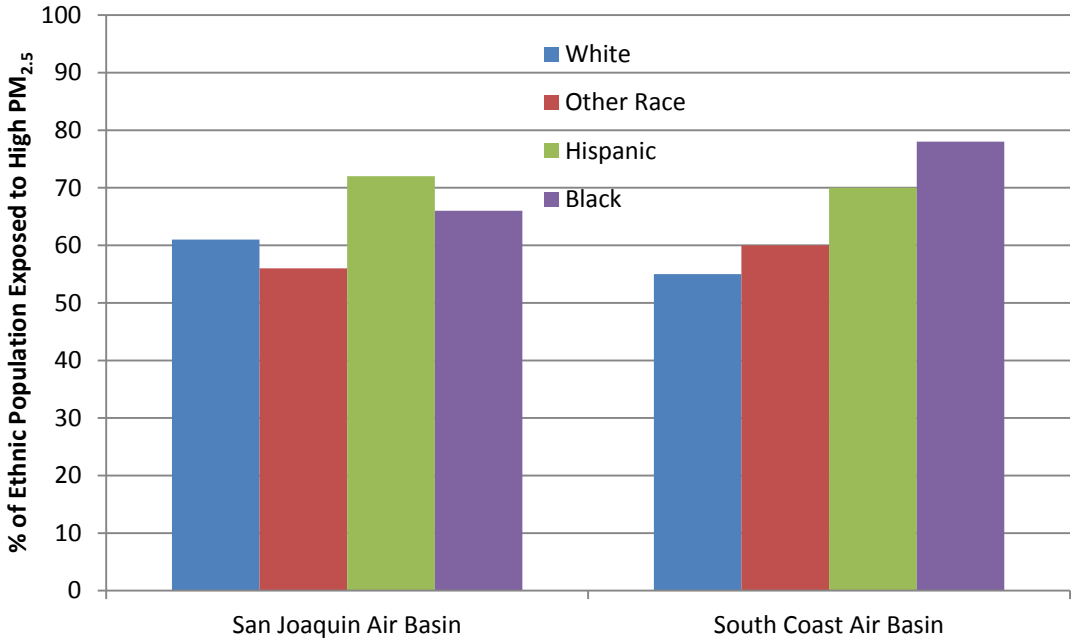

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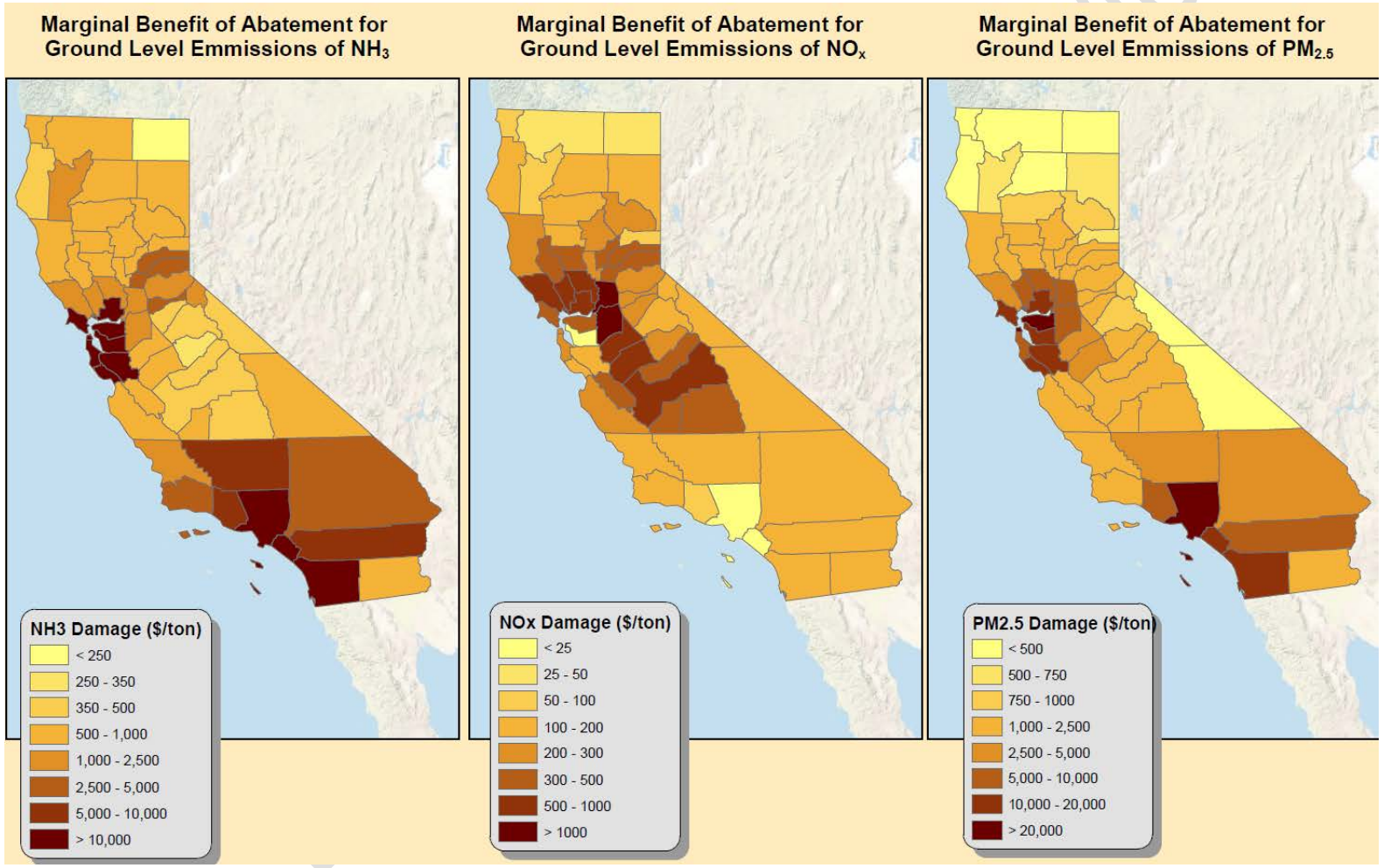
1916 **Figure 5.3.12. Marginal benefits of abatement for ground level NH_3, NO_3 and $PM_{2.5}$ emissions in California.** Damage costs are

1917 expressed in \$ ton^{-1} of each pollutant. (Source: Adapted from Muller and Mendelsohn 2009) [Return to text]



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1919 **Figure. 5.3.13. Annual agricultural benefits for consumers and producers for O_3 control in the San Joaquin Valley, 1998**. Source:

1920 Kim et al. 1998. [Return to text]

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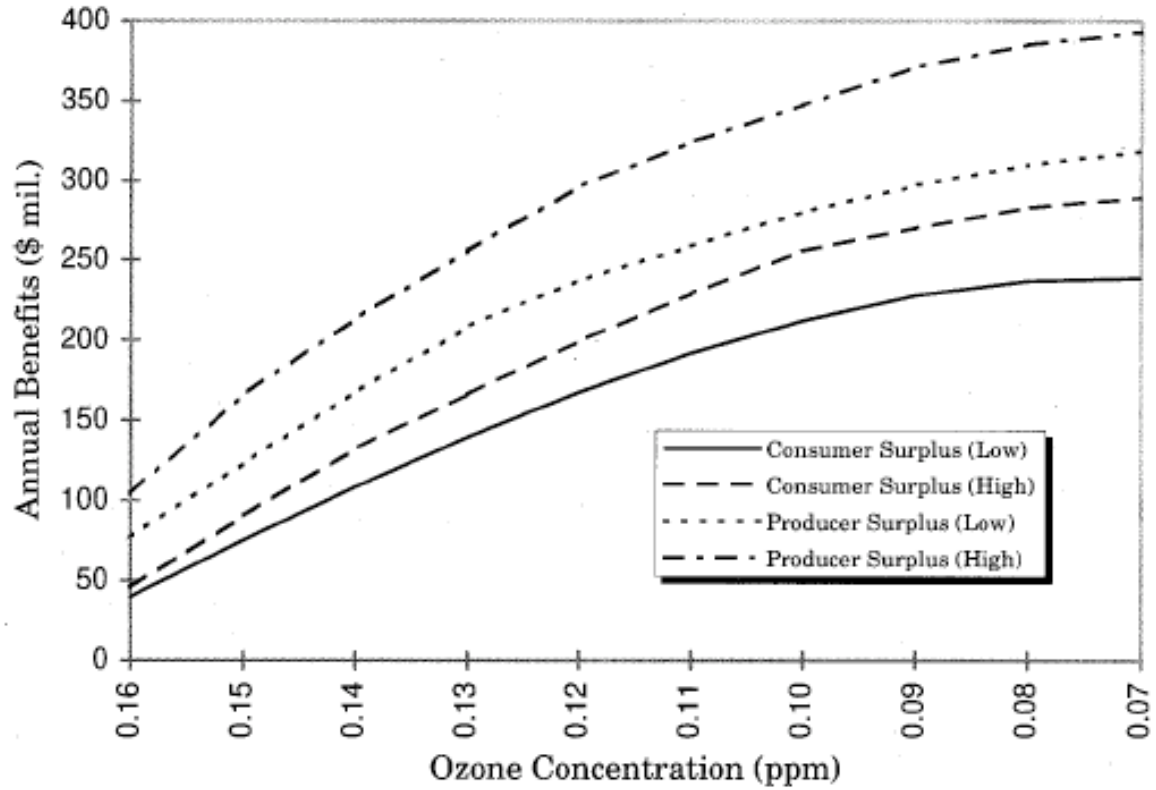

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Table 5.3.1: National and California Ambient Air Quality Standards (AAQS). Source: UEPA 2010; CARB 2011. [Return to text]

Pollutant	Primary/Secondary	Averaging Time	National AAQS	California AAQS	Form
Nitrogen Dioxide (NO_2)	Primary	1-hour	0.100 ppm	0.18 ppm	98th percentile, averaged over 3 years
	Primary and secondary	Annual	0.053 ppm	0.030 ppm	Annual mean
Ozone (O_3)	Primary and secondary	8-hour	0.075 ppm	0.070 ppm	Annual fourth-highest daily maximum 8-hr concentration, averaged over 3 years
		1-hour	See below*	0.09ppm	N/A
Fine Particulate Matter ($PM_{2.5}$)	Primary and secondary	Annual	15 µg/m^3	12 µg/m^3	Annual mean, averaged over 3 years
		24-hour	35µg/m^3	See below**	98th percentile, averaged over 3 years
Respirable Particulate Matter (PM_{10})	Primary and secondary	Annual	See below***	20 µg/m^3	N/A
		24-hour	150 µg/m^3	50 µg/m^3	Not to be exceeded more than once per year on average over 3 years

^ The "Form" column is taken from the US EPA National Ambient Air Quality Standards (NAAQS)

*There is no separate 1-hour O_3 national standard.

** There is no separate 24-hour $PM_{2.5}$ standard in California, though the US EPA promulgated a 24-hour $PM_{2.5}$ ambient air quality standard of 35µg/m^3.

***There is no separate annual PM_{10} national standard.

Table 5.3.2 Strength of evidence relating exposure to air pollutants to specific health problems. [Return to text]

Health Outcome	NOx	O_3	PM
Respiratory symptoms	++	+++	+++
Lung function	++	+++	+++
Asthma	++	+++	+++
COPD	+	++	+++
Respiratory infections	++	+	++
Respiratory mortality	@	+++	+++
Cardiovascular disease	++	+	+++
Cardiovascular mortality	@	++	+++
Birth outcomes	+	+	++
Cancer	+	@	++
Mortality, all causes	+	++	+++

+++ = well-established

++ = provisionally agreed by most

+ = speculative

@ = suggested but unproven

1939 **Table 5.3.3 Estimated incidences of O_3 and $PM_{2.5}$ related adverse health effects in the San**

1940 **Joaquin Valley and South Coast Air Basins by county in 2008.** Source: Hall et al. 2008.

1941 [Return to text]

Ozone-Related Adverse Health Effects By County

	Respiratory Hospital Admissions (All ages)	Asthma Attacks Asthmatic population	Emergency Room Visits	Days of School Absences	Minor Restricted Activity Days	Mortality
San Joaquin Valley Air Basin						
Fresno	46	5,670	17	43,980	42,970	3
Kern	41	4,640	13	37,810	34,620	3
Kings	5	890	3	6,050	7,580	0
Madera	6	780	2	5,500	6,320	0
Merced	8	1,090	3	8,530	8,070	0
San Joaquin	17	2,290	7	13,100	17,170	0
Stanislaus	16	2,100	7	13,500	15,190	1
Tulare	24	2,940	8	23,040	21,830	2
South Coast Air Basin						
Los Angeles	380	59,100	150	653,300	483,840	12
Orange	87	17,010	45	184,500	142,380	3
Riverside	185	22,480	55	125,840	164,470	15
San Bernardino	173	22,380	55	144,690	170,720	11

$PM_{2.5}$-Related Adverse Health Effects By County

	Premature & Post-Neo Natal Mortality	Respiratory Symptoms & Bronchitis	Non-Fatal Heart Attacks	Respiratory & Cardio Hospital Admissions	Children's Asthma ER Visits	Minor Restricted Activity Days	Work Loss Days
San Joaquin Valley Air Basin							
Fresno	212	104,215	156	80	119	103,770	18,500
Kern	183	81,228	119	53	93	80,170	14,280
Kings	29	15,207	27	10	17	18,770	3,340
Madera	33	14,235	24	13	16	16,020	2,850
Merced	38	24,269	33	14	28	21,840	3,880
San Joaquin	110	46,908	78	43	54	49,360	8,740
Stanislaus	99	43,814	70	39	50	45,660	8,120
Tulare	110	54,678	77	37	63	50,750	9,030
South Coast Air Basin							
Los Angeles	1,727	1,000,440	1,960	903	1,175	1,224,600	241,690
Orange	411	233,310	485	175	275	300,010	59,100
Riverside	461	217,570	370	220	255	224,780	44,500
San Bernardino	412	260,480	415	187	305	266,830	52,850

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Table 5.3.4 Health outcomes and economic values related to premature deaths from particulate matter (PM_{10} and $PM_{2.5}$) and minor restricted activity days (MRADs) in the South Coast Air Basin, California – 1989 and 2008. Source: Adapted from Hall et al. 2010.

[Return to text]

Particulates	**1989 Study** (1988 $)	**2008 Study** (1988 $)[a]	**2008 Study** (2007 $)	**1989 Study** (2007 $)
Premature deaths	1617		3000	
VSL	$3.7 million	$3.54 million	$6.63 million	$6.93 million
Total value	$5.98 billion		$19.88 billion	
Ozone				
MRADs	$17.65 million		$961,400	
Unit value	$34.95[b]	$35.08	$65.60	$65.46
Total value	3.79 million		63.16 million	

[a] Adjusted for price level (CPI) and income changes.

[b] Commonly used Tolley et al. (1986) value (our value was 21.50)

Table 5.3.5 Differences in O_3-related all-illness school absences in the SoCAB over time for the 1998 population aged 5-18. Source: Hall et al. 2003. [Return to text]

Time Period	1990-92 to 1997-99	1991-93 to 1997-99	1992-94 to 1997-99	1993-95 to 1997-99	1994-96 to 1997-99	1995-97 to 1997-99	1996-97 to 1997-99
	millions of annual absences						
Decrease in all-illness absences	3.19	2.84	2.47	1.70	1.43	0.984	0.480

Table 5.3.6 Economic value of differences ($) in annual 8-hour O_3-related all-illness school absences from 1990-92 to 1997-99 in the 1998 population aged 5-18. Source: Hall et al. 2003. [Return to text]

School absences	SoCAB	Los Angeles County	Orange County	Riverside County	San Bernardino County
	---------- millions of annual absences ----------				
Total all illness	245,048,000	147,689,000	21,584,000	30,109,000	45,666,000
Per capita all illness (2010 $)	75	74	39	91	114

1972 **Table 5.3.7 Cost of health outcomes attributable to air pollution exposure (NO_2, O_3) for children in Riverside and Long Beach.**

1973 Source: Brandt et al. 2012. [Return to text]

	Riverside		Long Beach		
	Count	**Cost per outcome ($)**	**Count**	**Cost per outcome ($)**	**Total cost per year ($)**
Attributable asthma cases	690 (630-750)	4,008	1,600 (1500-1800)	3,819	8,875,920
NO_2 – Attributable exacerbations of other-cause asthma					
Emergency room visits	40 (5-70)	956	150 (19-280)	944	179,840
Inpatient hospitalizations	8 (6-10)	13,282	27 (22, 32)	13,227	463,385
Clinic office visits	190 (38-340)	158	440 (80-780)	153	97,340
Bronchitis episodes	1500 (440-2300)	975	3,100 (1000-4,400)	918	4,308,300
O_3 – Attributable exacerbations of other-cause asthma					
Emergency room visits	230 (150-310)	956			219,880
Inpatient hospitalizations	12 (9-15)	13,282			159,384
Clinic office-visits	190 (25-360)	161			30,020
Bronchitis episodes	2,900 (160-3,900)	975			2,827,500
School days absent	2,966 (2,223-	230	626 (43-1,114)	205	810,510
Total Annual Cost					**17,972,079**

The 95% confidence intervals are reported in parentheses. All Costs are rounded to the nearest US$ 2010 rate.

Table 5.3.8 Estimated statewide yield loss due to ground-level O_3 in 1993 using 7-hour (27.2 ppb) and 12-hour (25 ppb) mean O_3 exposure crop-loss models. Yield losses are relative to clean background air. Source: Adapted from Grantz and Shrestha 2005. [Return to text]

Crop	Yield loss (%)
	Mutters and Soret
Study	(1998)
Cantaloupe	32.8
Grape, table	29.9
Grape, raisin	26.2
Cotton, upland	23.3
Grape, wine	22.8
Bean, dry	17.5
Orange	14
Onion	10.6
Alfalfa	9.5
Lemon	8.4
Tomato, processing	6.8
Wheat	6.7
Rice	3.9
Corn, field	1.2
Tomato, fresh-market	0.6