APPENDIX F-1

Air Quality Technical Appendix Technical Background This page intentionally left blank.

This technical air quality appendix provides background information used for evaluating the air quality impacts for the proposed Oliveira Dairy Expansion in Merced County.

ENVIRONMENTAL SETTING

Air quality influences public health and welfare, the economy, and quality of life. Air pollutants have the potential to adversely impact public health, the production and quality of agricultural crops, visibility, native vegetation, and buildings and structures.

Because of the great influence of poor air quality on the environment, a far-reaching regulatory structure has been developed to regulate businesses and activities to lessen or moderate the adverse effects of human activities on air quality. Because of the comprehensive nature of federal, state, and regional air quality management, an understanding of the environmental setting of air quality in Merced County and the San Joaquin Valley in general begins with a brief description of the physical environmental factors influencing air quality, followed by a description of the existing regulatory agencies, and their programs and requirements. This is followed by a description of air pollutants.

The San Joaquin Valley is bounded by the Sierra Nevada mountain range to the east, the coastal mountain ranges to the west, the Tehachapi mountains to the south, and San Joaquin County to the north. The Valley is approximately 250 miles long and averages approximately 35 miles in width.

From west to east, elevations in and adjacent to the San Joaquin Valley range from approximately 3,000 feet above mean sea level (msl) along the crest of the coastal mountain ranges, to below sea level in areas of the Valley itself, and above 10,000 feet msl along the crest of the Sierra Nevada mountains. The predominant wind direction in the Valley is from the northwest toward the southeast.

The climate in Merced County is semiarid, characterized by hot, dry summers and cold, moist winters. The warmest month is July with average temperatures in the 90°s Fahrenheit and midday temperatures ranging up to 100° to 110°. The coldest month is January with average low temperatures in the 30°s.

Annual precipitation, mostly rainfall, ranges from 8 to 13 inches in the San Joaquin Valley, 9 to 14 inches in the foothills of the Sierra Nevada, to 13 to 24 inches in the Sierra Nevada. The average length of the frost-free season in Merced County is approximately 250 days per year. Precipitation occurs mainly from November to April; January typically has the highest rainfall. Fog is prevalent in the valley from December to March.

The mountains surrounding the San Joaquin Valley Air Basin (Air Basin) restrict air movement through and out of the basin, and, as a result, impede the dispersion of pollutants from the basin. Inversion layers are formed in the Air Basin throughout the summer and winter. These layers occur when cooler air near the ground surface is overlain by warmer air that prevents the vertical dispersion of pollutants. During the summer, the San Joaquin Valley experiences daytime temperature inversions at elevations from 2,000 to 2,500 feet above the valley floor, and during the winter, inversions occur at elevations from 500 to 1,000 feet above the valley floor.

CRITERIA AND NON-CRITERIA AIR POLLUTANTS

The following section discusses both criteria and non-criteria pollutants. Criteria pollutants are those that are regulated by either the state or federal Clean Air Acts. Non-criteria pollutants are not regulated by these Acts, but are nonetheless of concern for animal confinement facilities because they may be precursors to criteria pollutants, or because of their potential for harm or nuisance. For each pollutant, the following discussion sets forth the major sources of the pollutant; its potential for adverse environmental effects; the trend of the pollutant in the San Joaquin Valley and Merced County in terms of number of violations and concentration in the environment (for nonattainment criteria pollutants only); the amount of the pollutant emitted in the San Joaquin Valley and Merced County (latest available emissions inventory data is for 2012); the role of animal confinement facilities in the emissions; and potential human health effects.

In general, increased emissions could be expected to increase existing levels of chronic lung disease and to increase morbidity¹ and mortality. As shown below, the Air Basin is currently in nonattainment for criteria pollutants. The current levels of air pollution within the basin result in high levels of lung disease, morbidity, and mortality. Emissions in aggregate are expected to increase in the basin between the current time and 2020 from all sources and are expected to result in even higher levels of the health effects noted below. While the ARB is continually refining livestock emission estimates and incorporating this data into its regional air quality models for ozone and particulate matter, there is a lack of commonly accepted epidemiological models to forecast health impacts from dairies and other confined animal facilities (Gaffney 2008; Mitloehner 2007). However, it has been well documented that there are adverse respiratory effects from exposure in agricultural occupations. Harmful air emissions from animal confinement facilities result from feed handling, animal movement, and manure storage and removal; these emissions tend to impact farm workers, who experience higher exposure, more than neighboring residents, who experience lower exposures (Mitloehner 2007).

Criteria Pollutants

Ozone. Ozone (O_3) is not emitted directly into the environment, but is generated from complex chemical reactions between reactive organic gases (ROG), or non-methane hydrocarbons, and oxides of nitrogen that occur in the presence of sunlight. Ozone exposure causes eye irritation and damage to lung tissue in humans. Ozone also harms vegetation, reduces crop yields, and accelerates deterioration of paints, finishes, rubber products, plastics, and fabrics. The human health effects of ozone are discussed in further detail below.

Major ROG and NO_x generators in the San Joaquin Valley include: motor vehicles and farming equipment such as tractors, feed trucks, and pumps; farming operations; and solvent evaporation. Total ROG emissions in 2012 were 33.96 tons per day in Merced County and 357.9 tons per day in San Joaquin Valley. Total NO_x emissions in 2012 were 29.84 tons per day in Merced County compared to total NO_x emissions in the San Joaquin Valley in 2012 of 352.2 tons per day (ARB 2013).

¹ Illness or disease.

With respect to ozone, California and the San Joaquin Valley have some of the most polluted air in the nation. In 2014-2016, all counties in the San Joaquin Valley received an ozone grade of "F" in the State of the Air report (ALA 2018). Table 1 sets forth the average number of days during 2014-2016 in San Joaquin Valley counties where ozone levels were classified as being at unhealthy levels.

Table 1San Joaquin Valley Air Basin – Average Number of High Ozone Days in Unhealthy Ranges by County: 2014-2016				
County	Number of Days Unhealthy for Sensitive Groups	Number of Days Unhealthy	Number of Days Very Unhealthy	Weighted Average
Fresno	195	54	1	92.7
Kern	236	48	1	103.3
Kings	122	8	0	44.7
Madera	114	11	0	43.5
Merced	92	5	0	33.2
San Joaquin	52	3	0	18.8
Stanislaus	87	7	0	32.5
Tulare	229	41	0	96.8

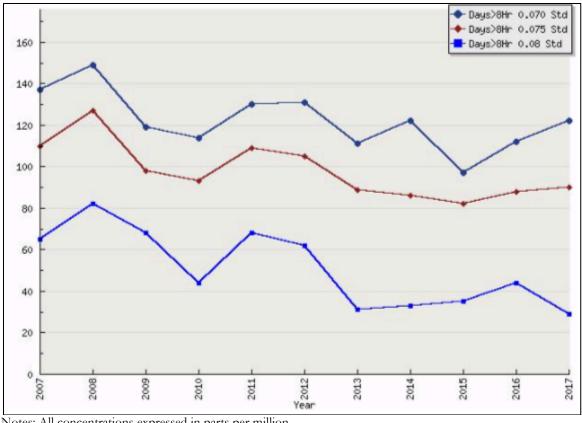
Notes: The number of days it is **Unhealthy For Sensitive Groups** (labeled Orange by the Air Quality Index) are the number of days ozone concentrations range from 71 - 85 ppb; the number of days it is **Unhealthy** (labeled Red by the Air Quality Index) are the number of days ozone concentrations range from 86 - 105 ppb; and the number of days it is **Very Unhealthy** (labeled Purple by the Air Quality Index) are the number of days ozone concentrations range from 106 - 200 ppb.

The Weighted Average was derived by counting the number of days in each unhealthful range (orange -Unhealthy For Sensitive Groups; red – Unhealthy; purple - Very Unhealthy; maroon - Hazardous) in each year (2014-2016), multiplying the total in each range by the assigned standard weights (i.e., 1 = orange, 1.5 = red, 2.0 = purple, 2.5 = maroon), and calculating the average.

Source: American Lung Association 2018, accessed website January 2018 www.stateoftheair.org/

As shown in Chart 1 and Chart 2, within the San Joaquin Valley, the number of days with ozone levels in violation of the applicable NAAQS and SAAQS has decreased overall from 2007 to 2017.

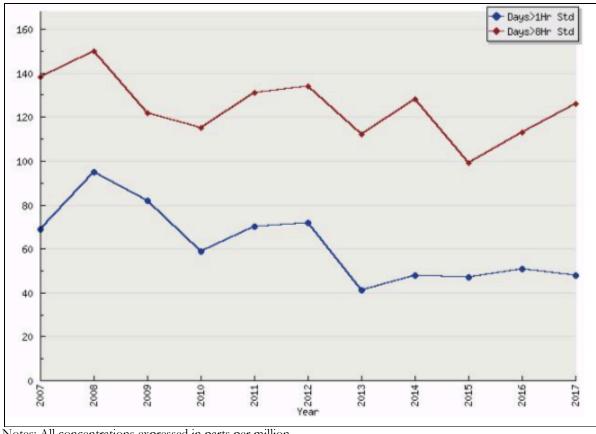
CHART 1: SAN JOAQUIN VALLEY AIR BASIN TREND IN NUMBER OF HIGH OZONE DAYS IN VIOLATION OF NAAQS: 2007-2017



Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

CHART 2: SAN JOAQUIN VALLEY AIR BASIN TREND IN NUMBER OF HIGH OZONE DAYS IN VIOLATION OF SAAQS: 2007-2017

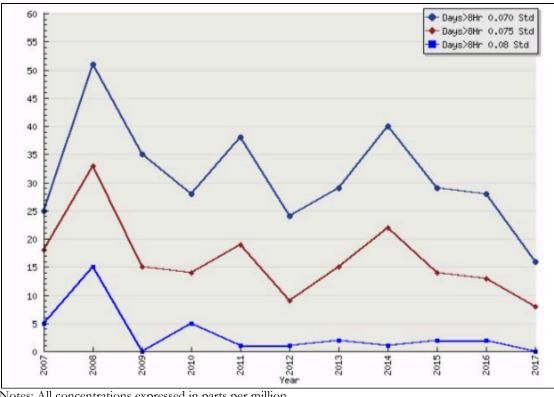


Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Chart 3 and Chart 4 indicate that Merced County saw an overall decrease in days above federal and state ozone standards from 2007-2017.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

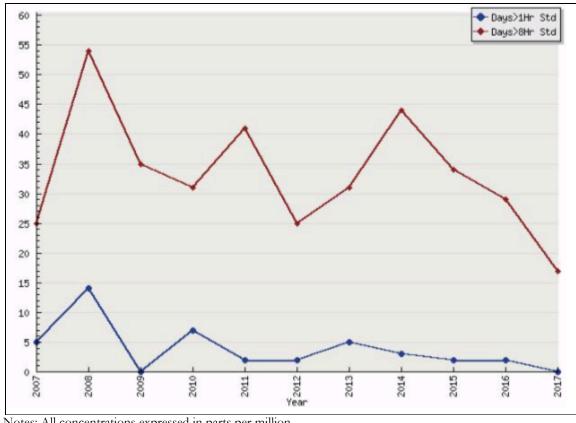
CHART 3: MERCED COUNTY TREND IN NUMBER OF HIGH OZONE DAYS IN VIOLATION OF NAAQS: 2007-2017



Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

CHART 4: MERCED COUNTY TREND IN NUMBER OF HIGH OZONE DAYS IN VIOLATION OF SAAQS: 2007-2017

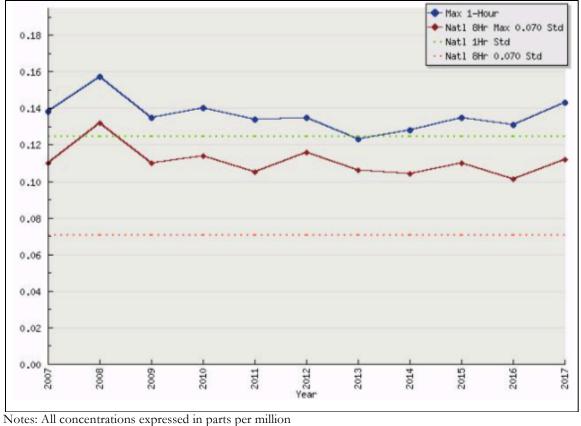


Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

As shown in Chart 5 and Chart 6, within the San Joaquin Valley, peak ozone concentrations have generally been holding steady during the period from 2007-2017.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

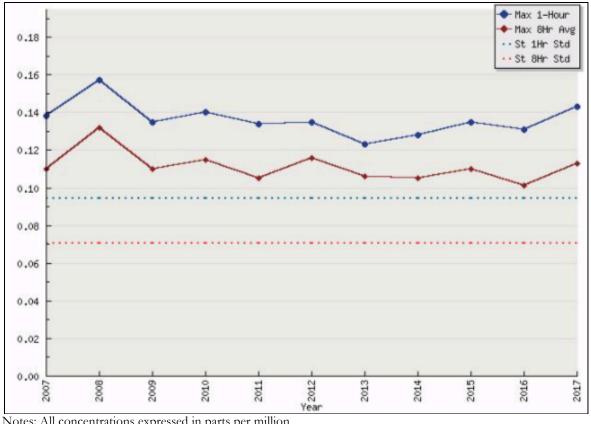
CHART 5: SAN JOAQUIN VALLEY AIR BASIN TREND IN OZONE CONCENTRATIONS (FEDERAL STANDARDS): 2007-2017



Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

CHART 6: SAN JOAQUIN VALLEY AIR BASIN TREND IN OZONE CONCENTRATIONS (STATE STANDARDS): 2007-2017

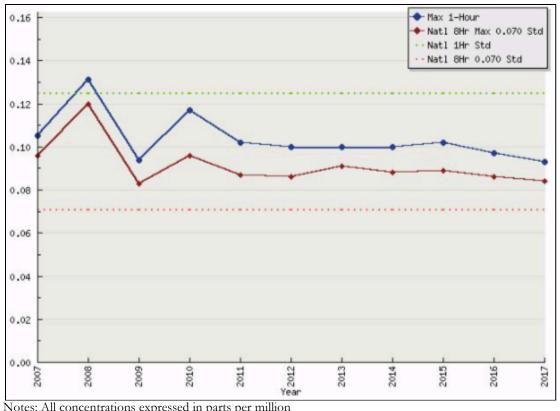


Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

As indicated on Chart 7 and Chart 8, overall, there have been variations in peak ozone concentrations, though they have generally been holding steady.

CHART 7: MERCED COUNTY TREND IN OZONE CONCENTRATIONS (FEDERAL STANDARDS): 2007-2017



Notes: All concentrations expressed in parts per million An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

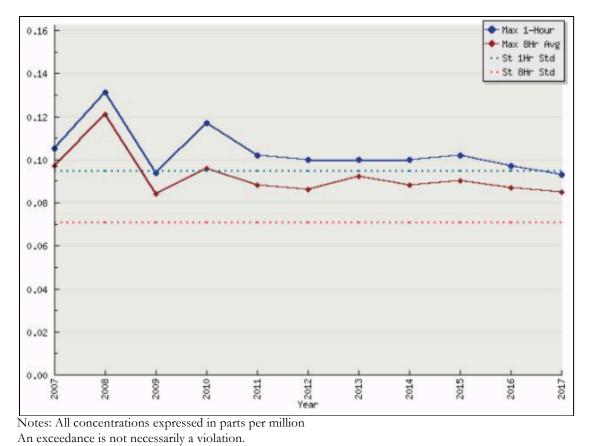


CHART 8: MERCED COUNTY TREND IN OZONE CONCENTRATIONS (STATE STANDARDS): 2007-2017

Emissions from Animal Confinement Facilities. The San Joaquin Valley currently experiences some of the highest surface ozone (O_3) concentrations in the United States even though it has a population density that is an order of magnitude lower than many urban areas with similar ozone problems. Previously unrecognized agricultural emissions may explain why O_3 concentrations in the San Joaquin Valley have not responded to traditional emissions control programs (Howard, et al. 2010). Because ozone is not directly emitted into the atmosphere, but is the result of a reaction between NO_x and ROG (which are directly emitted), emissions from animal confinement facilities will be discussed under those pollutants.

Health Effects. Ozone is a powerful respiratory irritant at the levels frequently found in most of the nation's urban areas during summer months. Symptoms include shortness of breath, chest pain when inhaling deeply, wheezing, and coughing, and increased susceptibility to respiratory infections. Exposure to ozone increases: risk of premature mortality, pulmonary inflammation, the risk of asthma attacks, and the need for medical treatment and for hospitalization of persons with asthma. In studies of animals, ozone exposure has been found to increase susceptibility to bacterial pneumonia infection.

Source: California Air Resources Board, 2018. Ozone Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

One study of 16 Canadian cities over a 10-year period found that air pollution, including ozone, at relatively low concentrations, is associated with excess admissions to the hospital for respiratory diseases (Burnett, et. al. 1997). Ozone levels typically rise during the May through September period when higher temperatures and the increased amount of sunlight combine with the stagnant atmospheric conditions that are associated with ozone air pollution episodes. Studies have shown that long-term, repeated exposures to high levels of ozone can decrease lung function and damage lung tissue (Kunzli, et. al. 1997; Frischer, et. al. 1999; Kinney, et. al. 2000). Ozone exposure also causes eye irritation.

High ozone levels are particularly dangerous for children. Research has tied increases in school absentee rates from school and health problems (sore throats, coughs, asthma attacks, and similar problems) to increases in ozone levels (Gilliland, et. al. 2001; Chen, et. al. 2000). Children with asthma are particularly susceptible to decreased lung function with ozone exposure (Peters, et. al. 1999; Mortimer, et. al. 2000).

Ozone air pollution increases susceptibility to influenza, pneumonia, and other infections, which are especially dangerous for the elderly (Sartor, et. al. 1997). In addition, ozone can significantly worsen the condition of people with chronic bronchitis and emphysema, and since most of these diseases occur in the elderly population, these elderly are at special risk for exposure to ozone. Ozone also has been shown to increase allergic responses in people with asthma or allergies (Jorres, et. al. 1996; Peden, et. al. 1995).

Ozone also harms vegetation, reduces crop yields, and accelerates deterioration of paints, finishes, rubber products, plastics, and fabrics. Monitored air quality data and a health model allow the state to quantify the potential scope of harm to Californians from air pollution each year – from premature death to asthma attacks, as well as the impacts on health care and productivity. A long-term study of six U.S. cities estimated that the U.S. could prevent approximately 34,000 premature deaths a year if the nation could lower annual levels of particle pollution by 1 μ g/m³ (ALA 2018).

Respirable Particulate Matter (PM₁₀). Particulate matter is divided into primary and secondary forms. Primary particulate matter is in the same chemical form in which it was emitted into the atmosphere. According to the National Emissions Trends Inventory (2011 data), approximately 52 percent of PM_{10} emissions are due to dust (EPA 2014). The main sources of fugitive dusts are unpaved roads, construction, and paved roads. Additional sources of PM_{10} include fuel combustion, mobile sources, industrial processes, agriculture, fires, solvents, and miscellaneous sources. The major sources of PM_{10} in Merced County include farming operations, paved and unpaved road dust, food and agriculture industrial processes, managed burning and disposal, heavy-duty diesel trucks, and fugitive windblown dust. Total PM_{10} emissions in 2012 were 1.57 tons per day in Merced County and 281.6 tons per day in the San Joaquin Valley (ARB 2013).

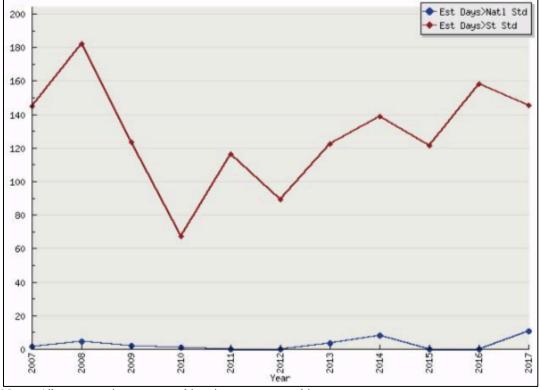
Secondary particulate matter is formed by chemical reactions of free, adsorbed, or dissolved gases in the atmosphere. Secondary aerosol formation depends on a variety of factors including: concentrations of precursors; concentrations of other reactive species (ozone, hydroxyl radical, peroxy radicals, or hydrogen peroxide); atmospheric conditions (solar radiation and relative humidity); and the interactions of precursors and preexisting particles within cloud or fog droplets on or in the liquid film on solid particles. The length of time particles remain in the atmosphere varies depending on their size. Coarse particles can settle within hours and do not normally travel long distances. However, in dust storms these particles may travel further. Smaller particles may

accumulate in the atmosphere where they may remain suspended for days by normal air motions and have very low deposition rates. These particles may travel thousands of miles before being removed from the atmosphere. Accumulation-mode particles may be incorporated into cloud formation processes. These and other particles can be removed from the atmosphere through precipitation.

 PM_{10} is released directly into the atmosphere by stationary and mobile sources, and consists of a wide range of solid and liquid particles which have an aerodynamic diameter less than or equal to a nominal 10 µm. Examples are smoke, dust, aerosols, and metallic oxides. Due to small size, the particles can be inhaled and cause damage to lung tissue in humans. The human health effects of PM_{10} will be discussed in further detail below. Major sources of PM_{10} include vehicles, power generation, industrial processing, wood burning, road dust, construction/farming activities, and fugitive windblown dust.

As shown in Chart 9, within the San Joaquin Valley, the number of days with PM₁₀ levels in violation of the applicable NAAQS remained similar from 2007-2017, ranging from zero to 10 days. Within Merced County, there were no violations of NAAQS within the period of record (see Chart 10). Daily violations of SAAQS in the San Joaquin Valley reached a high point in 2008, followed by an overall decrease from 2008-2013.

Chart 9: Number of Days above PM_{10} Standards for San Joaquin Valley Air Basin: 2007-2017

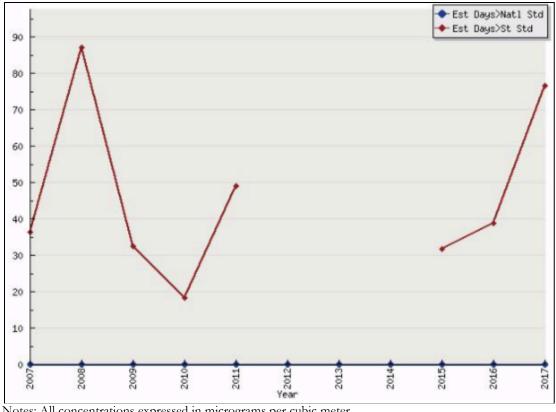


Notes: All concentrations expressed in micrograms per cubic meter. An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. PM₁₀ Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

As shown in Chart 10, daily violations of SAAQS in Merced County recorded at the Merced 2334 M Street station have varied from 2007 to 2017, though Merced County's violations have experienced an overall increase over this time period.





Notes: All concentrations expressed in micrograms per cubic meter. An exceedance is not necessarily a violation.

Source: California Air Resources Board, 2018. PM₁₀ Data Summaries. Accessed at http://www.arb.ca.gov/adam/trends/trends1.php

As discussed in Chapter 5, *Air Quality and Odors*, of this Draft EIR, the San Joaquin Valley has been reclassified as attainment of the federal PM_{10} standard.

Emissions from Animal Confinement Facilities. In animal confinement facility operations, particulates are primarily produced as a result of animal movement on dry manure, soil tillage, harvesting, and vehicle travel on unpaved roads. Also, as discussed below, secondary PM_{10} formation occurs as a result of the reaction of ammonia with nitrous oxides/sulfur oxides to form aerosols. PM_{10} emission rates from confined animals are a subject of great importance in the arid portions of the United States. Estimates of PM_{10} emissions from dairies vary greatly, from 4 lbs/1,000 head/day (Sweeten et al. 2000) to 90.51 lbs/1,000 head/day (Flocchini et al. 2001). In 2006, the SJVAPCD identified emission factors of 5.46 lbs/head/year for milk and dry cows in open corrals, 1.37 for milk and dry cows in freestalls, 10.55 for heifers in open corrals, and 1.37 for calves in pens or open corrals (SJVAPCD 2006). These emission factors will be used in this technical appendix and associated EIR.

Health Effects. Exposure to elevated levels of particulate matter causes irritation of the eyes and respiratory system. The nature of impacts to the respiratory system is related to the size of the individual particles (EPA 2001). Particulate matter having particle diameters greater than 10 μ m tend to deposit in the nasopharyngeal region of the respiratory tract and are not carried into the deeper airways and lungs. Airborne particles of diameter about 2.5 – 10 μ m deposit in the trachea and upper bronchi due to impaction. Those airborne particles of diameter 0.3 – 0.5 μ m undergo the least respiratory tract deposition due to impaction and are carried into the deeper alveolar (i.e., gas exchange) regions of the lung.

Research ties increased hospital admissions and mortality rates to increases in PM_{10} (Samet et al. 2000; Levy et al. 2000) and supports concern for health effects from larger particulate matter as well (Lippman et al. 2000).

The purpose of SJVAPCD Regulation VIII (Fugitive PM₁₀ Prohibitions) is to reduce ambient concentrations of fine particulate matter by requiring actions to prevent, reduce, or mitigate dust emissions. The rules apply to specified outdoor fugitive dust activities, including construction, landfills, government-owned roads, oil production facilities, and off-field agricultural sources (i.e., unpaved roads and equipment storage areas). Both Regulation VIII and Rule 4550 apply to agricultural tilling, land preparation, and harvesting.

Under AB 2588, any facility that emits 10 tons (or more) of PM_{10} per year must submit emissions estimates. Prior to 1998, agricultural and livestock operation were not subject to AB 2588 regulations. Revisions to the California Health and Safety Code Section 44380.1 require agricultural and livestock operations to comply with emission monitoring and reporting mandates. Enforcement of these provisions is under the jurisdiction of the local air pollution control district.

Fine Particulate Matter (PM_{2.5}). PM_{2.5} is atmospheric particulate matter having a particle size less than 2.5 μ m in diameter. There are three primary origins of PM_{2.5}: (1) primary solid particulate matter that is emitted directly in the solid phase; (2) primary condensable particulate matter that can be emitted at high temperature in the gas phase, but condenses into the solid phase upon dilution and cooling; and (3) secondary particulate matter that is formed through atmospheric reactions of gaseous sulfur dioxide (SO₂) and nitrogen oxide (NO_x) precursor emissions (EPA 1999a). These small particles can be inhaled into the lungs and have the potential to cause health-related impacts in sensitive persons.

Primary solid particulate matter results largely from fires, with additional major sources from dust, agriculture, fuel combustion, mobile sources, and industrial processes (EPA 2013).

Primary condensable particulate matter is largely comprised of semivolatile organic compounds that condense at ambient temperatures to form aerosols.

Secondary $PM_{2.5}$ forms through chemical reactions that convert common gaseous pollutants into very small particles. Secondary $PM_{2.5}$ is dominated by sulfur and nitrogen species, but in some locations there can also be significant contributions from secondary organic aerosol (EPA 1999a). Ammonia emissions from dairies are considered to be precursors to $PM_{2.5}$ formation. Atmospheric ammonia is discussed further below.

In the San Joaquin Valley, $PM_{2.5}$ concentrations peak in the fall and winter. The average $PM_{2.5}$ mass measured on episode days in San Joaquin Valley during a 1996 wintertime study were 57 µg/m³ for urban areas and 31 µg/m³ for rural areas (Pun and Seigneur 1998). The contribution to the urban areas was primarily from ammonium nitrate (30 percent), vegetative burning (21 percent) such as wood burning fireplaces, and mobile sources (14 percent). For the rural areas, the contribution was primarily ammonium nitrate (50 percent), with less from vegetative burning (9 percent) and mobile sources (10 percent) (Magliano 1997). During these episodes of high $PM_{2.5}$ mass concentrations, air stagnation is the single most important factor for the accumulation of particulate matter in the San Joaquin Valley (Pun and Seigneur 1998).

As discussed above, the San Joaquin Valley air basin and Merced County have been designated as nonattainment for $PM_{2.5}$. Both the ARB and local air districts in violation of the NAAQS for $PM_{2.5}$ will have to prepare SIPs indicating control strategies that could be used to reduce particulate emissions. The ARB estimates $PM_{2.5}$ emissions for 2012 to be 6.18 tons per day in Merced County and 75.6 tons per day in the San Joaquin Valley (ARB 2013).

Emissions from Animal Confinement Facilities. Ammonia emissions from dairies are considered to be precursors to $PM_{2.5}$ formation. Atmospheric ammonia is discussed further below. In reactions in the atmosphere, gaseous ammonia combines with sulfur oxide (SO_x) and NO_x to form ammonium nitrate and ammonium sulfate $PM_{2.5}$ particles. It most areas of the United States, airborne nitric acid (HNO₃) concentrations (primarily from internal combustion engines) are generally lower than sulfate concentrations (primarily from coal burning). However, nitric acid concentrations can be higher in the San Joaquin Valley (Sheth and Giel 2000).

Formation of $PM_{2.5}$ requires both ammonia and SO_x/NO_x . Dairies are a source of ammonia, but do not necessarily produce $PM_{2.5}$ unless the SO_x/NO_x concentration is sufficiently high. In most rural settings, SO_x/NO_x concentrations limit the formation of $PM_{2.5}$.

Health Effects. Exposure to elevated levels of particulate matter causes irritation of the eyes and respiratory system. The nature of impacts to the respiratory system is related to the size of the individual particles (EPA 2001). Those airborne particles of diameter $0.3 - 0.5 \mu m$ undergo the least respiratory tract deposition due to impaction and are carried into the deeper alveolar (i.e., gas exchange) regions of the lung.

Brief exposures to approximately $100 \ \mu g/m^3$ particulate matter results in increased mucociliary clearance in healthy humans (EPA 2001). Exposures to greater than about $400 \ \mu g/m^3$ particulate matter cause bronchoconstriction in asthmatics. Acute exposures to approximately $1,000 \ \mu g/m^3$ particulate matter did not cause airway inflammation in healthy adults, but may cause bronchiolar hyperreactivity in sensitive subjects. Because of the potential to cause bronchiolar hyperreactivity, brief exposures to high concentrations of particulate matter may result in respiratory distress including asthma attacks in individuals with pre-existing respiratory illness.

Longer-term exposure to particulate matter is associated with chronic respiratory inflammation, rhinitis, asthma, increased susceptibility to respiratory tract infections, and increased mortality (EPA 2001). Relative risk of mortality is highest in the elderly and those individuals with pre-existing respiratory conditions (e.g., asthma). While previous reports of mortality associated with exposure to high levels of particulate matter focused on respiratory effects, more recent data suggests that

cardiac failure may be a more important contributor to particulate matter-related mortality (EPA 2001).

Carbon Monoxide. Carbon monoxide (CO) is a product of inefficient combustion, principally from automobiles and other mobile sources of pollution. Industrial sources of CO emissions generally contribute less than 10 percent of ambient CO levels. Peak CO levels occur typically during winter months because of a combination of seasonal contributions from home heating devices and stagnant weather conditions. CO interferes with the blood's ability to carry oxygen to the body's tissues and results in numerous adverse health effects.

The 2012 CO emission inventory for the Air Basin indicated that on-road motor vehicles and other mobile sources such as aircraft, trains, boats, and farm equipment were the leading sources of CO (ARB 2013). The SJVAPCD is currently unclassified (in attainment) with federal and state CO standards. Because rural areas of the San Joaquin Valley and Merced County are classified as attainment for CO, and animal confinement facilities and activities associated with them are very minor sources of CO, this pollutant will not be discussed further.

Nitrogen Oxides. Nitrogen Oxides react photochemically with hydrocarbons in the presence of sunlight to form ozone. Nitrogen oxides are major contributors to smog formation and acid deposition. The 2012 NO_x emission inventory for the Air Basin indicated that on-road motor vehicles; other mobile sources such as aircraft, trains, boats, and farm equipment; and stationary sources of fuel combustion such as oil and gas production and industrial facilities were the leading sources of NO_x (ARB 2013). The ARB estimates the releases of NO_x in 2012 as 29.84 tons per day from Merced County and 325.2 tons per day from San Joaquin Valley (ARB 2013).

Nitrogen Dioxide (NO₂) is formed in the atmosphere as a result of the reaction of nitric oxide (NO) and oxygen or ozone (O₃). NO is a by-product of high temperature combustion processes, and results from the combination of nitrogen and oxygen. Although NO is much less harmful than NO₂, it can be converted to NO₂ as a result of atmospheric processes within a short period of time. NO₂ can react with oxidants to form HNO₃. HNO₃ can then react with ammonia to form ammonium nitrate.

 NO_2 is a criteria pollutant for which state and federal air quality standards have been set. Currently, the SJVAPCD is in attainment for the state and federal NO_2 standards. This means that concentrations of NO_2 fall below the threshold levels established by the EPA and the State of California.

Emissions from Animal Confinement Facilities. In agriculture, nitrous oxides are released from the nitrification of ammonia in livestock waste, but more is released directly from soil (Eckard 2007).

Health Effects. NO_x are colored gases formed by the combustion of nitrogen-containing fossil fuels, and thermal or biological fixation of nitrogen. NO_x may also be an important indoor air pollutant where unventilated gas stoves or kerosene heaters are used (Klassen 2001).

 NO_2 is a deep lung irritant and may cause pulmonary edema when inhaled in sufficient quantities. It is an oxidant gas, similar to ozone, and deposits in the lung in an area only slightly proximal to the site of ozone deposition. However, it is a less potent irritant than ozone. Levels of NO_2 needed to produce effects are in general far greater than levels that occur in ambient air (Klassen 2001).

Chronic exposures to NO_2 may cause pulmonary damage, decreased pulmonary function, and increased susceptibility to respiratory infection. Such exposures can be a practical problem for farmers, as sufficient amounts can be released by ensilage to produce the symptoms of pulmonary damage known as 'silo-fillers' disease. Studies in laboratory animals simulating the twice-a-day spike in NO_x levels related to automobile pollution have demonstrated significant decreases in end-expiratory volume and vital capacity. Other studies have shown that short-term or long-term exposures to NO_x can increase susceptibility to respiratory infection by bacterial pneumonia or influenza virus. Asthmatics are not more sensitive than normal subjects to NO_2 (Klassen 2001).

Nitrogen oxides have not been associated with carcinogenic health effects in laboratory animals or humans and are not classified as carcinogens by the EPA.

The federal Occupational Safety and Health Administration (OSHA) has established an 8-hour Time Weighted Average (TWA) Threshold Limit Value (TLV) of $9,000 \ \mu g/m^3$ for NO₂ vapors in the workplace. This occupational standard is also applicable to NO.

A dairy may generate minor sources of NO combustion emissions from project operations.

Lead. Sources of lead resulting in concentrations in the air include industrial sources and crustal weathering of soils followed by fugitive dust emissions. Health effects from exposure to lead include brain damage, kidney damage, and learning disabilities.

The Air Basin is currently unclassified (in attainment) with state standards for lead, and no designation has been given in relation to federal attainment status. Because the San Joaquin Valley and Merced County are classified as attainment for lead, and animal confinement facilities and activities associated with them are very minor sources of lead, this pollutant will not be discussed further.

Sulfur Dioxide. Sulfur dioxide is produced when any sulfur-containing fuel is burned. It is also emitted by chemical plants that treat or refine sulfur or sulfur containing chemicals. Because of the complexity of the chemical reactions that convert SO_2 to other compounds (such as sulfates), peak concentrations of SO_2 occur at different times of the year in different parts of the state depending on local fuel characteristics, weather, and topography. SO_2 and other sulfur oxides contribute to acid deposition problems.

The SJVAPCD is currently unclassified (in attainment) with the federal and state SO₂ standards for sulfur dioxide. Because the San Joaquin Valley and Merced County are classified as attainment for sulfur dioxide, and animal confinement facilities and activities associated with them are very minor sources of sulfur dioxide, this pollutant will not be discussed further.

Sulfates. Sulfates are the product of further oxidation of sulfur dioxide, which is produced when any sulfur-containing fuel is burned, or by chemical plants that treat or refine sulfur or sulfur containing chemicals. Sulfates contribute to acid deposition problems, and form aerosols, which contribute to $PM_{2.5}$.

Sulfates are listed as a California criteria air pollutant; a SAAQS has been developed, but a NAAQS has not. The Air Basin is currently in attainment with the state sulfates standards. Because the San Joaquin Valley and Merced County are classified as attainment for sulfates, and animal confinement facilities and activities associated with them are very minor sources of sulfates, this pollutant will not be discussed further.

Hydrogen Sulfide. Hydrogen sulfide is a colorless, flammable, poisonous gas that is used in industrial processes and is generated during anaerobic decomposition of manure. The San Joaquin Valley is currently listed as unclassified in regards to attainment with the SAAQS. At this time, hydrogen sulfide measurements are limited to industrial centers in Southern and Northern California.

Hydrogen sulfide is listed as a California criteria air pollutant and is listed in the state Air Toxics "Hot Spots" Information and Assessment Act (AB 2588) as a substance for which emissions must be estimated for facilities that exceed certain thresholds including facilities that emit 10 or more tons of PM₁₀ annually. Prior to December 1998, agricultural and livestock operations were exempted from AB 2588. However, California Health and Safety Code Section 44380.1 has been revised and agricultural and livestock operations are now only exempt from paying fees associated with AB 2588, but not from complying with the remainder of the Act. Enforcement of AB 2588 requirements is the responsibility of local air quality control districts.

Emissions from Animal Confinement Facilities. Hydrogen sulfide is often linked to anaerobic digestion of organic materials. Data from a beef cattle operation in Minnesota suggests that elevated hydrogen sulfide concentrations have been observed near cattle operations.

Health Effects. Hydrogen sulfide is a colorless, flammable gas with a highly offensive odor of rotten eggs. It is widely utilized as a reagent in chemical synthesis and in the manufacture of deuterium. Major occupational exposures to hydrogen sulfide occur in the petroleum industry, natural gas, soil, sewer gas, and as a chemical by-product.

Hydrogen sulfide has a strong odor; the threshold for detection by humans is on the order of 0.025 ppm (0.035 mg/m³). Levels in the range of 3 - 5 ppm (4 - 7 mg/m³) cause an offensive odor. The most common symptoms of low-level hydrogen sulfide exposure in humans are nervousness, cough, nausea, headache, and insomnia (Poda 1966). Hydrogen sulfide concentrations in the range of 50 - 250 ppm (70 - 350 mg/m³) may cause eye and mucous membrane irritation, pulmonary edema, and pneumonia. At concentrations of 500 - 1,000 ppm, hydrogen sulfide acts as a systemic poison, causing sudden fatigue, headache, dizziness, intense anxiety, loss of olfactory function, nausea, convulsions, pulmonary edema, respiratory arrest, unconsciousness, cardiac failure, and death.

Chronic effects in humans do not generally occur due to the strong odor of this chemical. Subchronic exposure of laboratory mice to concentrations of 80 ppm (110 mg/m³) resulted in inflammation of the nasal mucosa and respiratory epithelium, reductions in body weight gain, and decreased organ weights. This study served as the basis for a chronic reference concentration (RfC) developed by the EPA for hydrogen sulfide (IRIS 2006).

There is currently no evidence to indicate that hydrogen sulfide is genotoxic. In addition, there are no reported instances of hydrogen sulfide-induced carcinogenicity in humans or animals, and the EPA does not currently classify this chemical as a carcinogen.

The United States Labor Code cites a Permissible Exposure Limit (PEL) of 10 ppm (14 mg/m^3) for hydrogen sulfide vapors in the workplace (Labor Code Section 142.3 and 144.6). An acceptable ceiling concentration of 50 ppm (28 mg/m^3) for occupational exposures to hydrogen sulfide has been established by federal OSHA, and an 8-hour time-weighted average of 15 ppm (21 mg/m^3).

A NAAQS has not been developed for hydrogen sulfide for public health exposures. However, the California Office of Environmental Health Hazard Assessment (OEHHA) has established an Acute Reference Exposure Level (REL) for hydrogen sulfide of 0.042 mg/m³ based on mild respiratory irritation effects. The California SAAQS for hydrogen sulfide has also been set at 0.042 mg/m³, averaged over one hour. A Chronic REL of 0.010 mg/m³ has been established by OEHHA based on respiratory system effects. For purposes of human health risk assessment, the EPA has established a chronic inhalation reference concentration (RfC) for hydrogen sulfide of 0.042 mg/m³ in mice, divided by an uncertainty factor of 1,000.

Non-Criteria Pollutants

Reactive Organic Gases. Reactive organic gases are a subset of total organic gases (TOG) that form ozone. ROG are created by the incomplete combustion of fuels or other carbon sources. ROGs are composed of hydrocarbons that react with nitrogen oxides and form ozone. Not all hydrocarbons contribute equally to ozone formation.

Their reactivity depends on their chemical structure and the atmospheric conditions to which they are subjected. The current ARB definition of ROGs is any compound of carbon, excluding carbon monoxide, carbon dioxide, carbonic acid, metallic carbides or carbonates, ammonium carbonate, methane, methylene chloride, 1,1,1-trichloroethane, freons, methylated siloxanes, and certain perflourocarbons. The EPA has also exempted the following low-reactive organic compounds: acetone, ethane, methyl acetate, perchloroethylene, and parachlorobenzotrifluoride. California's air pollution control districts report TOG to the ARB. The ARB derives a value for ROG by multiplying TOG by a fraction based on the source of the TOG.

ROG reacts with NO_x in the presence of sunlight to form ozone, a criteria pollutant for which both federal and state standards have been established. ROG emissions in 2008 in the San Joaquin Valley were 134,758 tons, and in Merced County were 10,822 tons (ARB 2009).

The principle sources of ROG in both the San Joaquin Valley and Merced County are miscellaneous industrial processes, motor vehicles, and solvent evaporation (ARB 2013). Trends in ROG emissions within the San Joaquin Valley and Merced County are similar to those identified for ozone above.

The Air Basin has been designated as a nonattainment area for both federal and state ozone standards. Thus, mitigation efforts are ongoing to regulate the emissions of the ozone precursors.

Emissions from Animal Confinement Facilities. Animal confinement facilities, including dairies, are currently believed to be a major component of ROG emissions in Merced County and the San Joaquin Valley.

Health Effects. The toxicity of organics depends on their structure. Most hydrocarbons are nontoxic at low concentrations. Some low-molecular-weight aldehydes are carcinogens and some monocyclic and polycyclic aromatic hydrocarbons (PAH) are suspected or known carcinogens. As described above, ozone causes respiratory irritation and long-term respiratory problems.

Ammonia. Ammonia (NH_3) is a pungent, colorless gaseous compound that is composed of nitrogen and hydrogen. The individual components react in the atmosphere with nitric acid, the product of NO_x , and an oxidant, such as ozone, to form ammonium nitrate. Ammonium nitrate is

considered to be a significant precursor to $PM_{2.5}$ (fine particulate), a criteria pollutant for which NAAQS have been established. State and federal air quality standards have not been set for ammonia.

Ammonia gas can also react with sulfur dioxide or atmospheric sulfates to form ammonium sulfate. These reactions, especially ammonium nitrate, represent a significant percentage of wintertime $PM_{2.5}$ in San Joaquin Valley (Roe and Mansell 2001). The limiting factor in wintertime ammonium nitrate formation in San Joaquin Valley is atmospheric nitrate (Pun and Seigneur 1998). As a result, ammonia emissions are also being studied as a potential source of particulate, which could be classified as $PM_{2.5}$.

There have been a few studies investigating the contribution of ammonium nitrate to particulate matter concentrations in the San Joaquin Valley. A study using data from 1988 and 1989 indicated that the contribution of secondary ammonium nitrate to total PM_{10} was 14.5 percent and 16 percent for Fresno and Bakersfield, respectively (Chow et al. 1992). A study of winter 1996 data indicated that secondary ammonium nitrate contributed to 34 percent and 32 percent of total $PM_{2.5}$ for Fresno and Bakersfield, respectively. Rural areas in this study had a lower total $PM_{2.5}$ concentration, but the contribution from secondary ammonium nitrate averaged 50 percent (Magliano 1997).

Major sources of ammonia include the anaerobic decomposition of manure, soil, and wastewater treatment facilities. Ammonia emissions result from bacterial catalysis of animal digestive wastes, especially urea. In addition to urea, recent studies indicate that approximately 35 percent of total organic nitrogen found in manure is rapidly converted to ammonia, and that additional conversion takes place when manure is stored over a longer period of time (Sweeten et al. 2000). Reducing protein content of feed can significantly reduce ammonia emissions and total nitrogen in dairy manure (James et al. 2000).

Ammonia emissions from livestock operations are generated as a consequence of bacterial activity involving organic nitrogen substrates, such as urea, and organic nitrogen compounds in feces. Kinetically rapid processes initially convert approximately 35 percent of the total organic nitrogen in manure to ammonia and, over longer time periods, primarily during storage, approximately 50-70 percent of the total organic nitrogen can be converted to ammonia (USDA 2000).

Based on a 2003 ARB study, ammonia emissions from dairy cattle in the San Joaquin Valley were approximately 78,997 tons in 2000 (ARB 2003). These emissions comprised 58.7 percent of ammonia emissions within the area (ARB 2003). Presumably emissions in rural areas are increasing as the populations of confined animals increase, though this has not been confirmed by measurement.

Federal and state standards have not been developed for ammonia, but it is listed in AB 2588 as a substance for which emissions must be estimated for facilities that exceed certain thresholds. These thresholds include facilities that emit 10 or more tons of PM_{10} annually.

Emissions from Animal Confinement Facilities. Overall ammonia emissions from dairy cows have been estimated at 74 lbs/head/year in San Joaquin Valley dairies (Flocchini et al. 2001; ENVIRON 2001). Ongoing studies are evaluating the reliability of these ammonia emissions factors. Based on an emission rate 74 lbs/head/year, and 585,000 dairy cows in Merced County in 2008 and 3,421,000 cows in San Joaquin Valley, ammonia emissions emitted from dairy cows in Merced

County were 21,650 tons and in San Joaquin Valley were 126,577 tons annually (Planning Partners 2008 cumulative herd forecast for Merced County and San Joaquin Valley).

The portion of the dairy (corrals, ponds, crop fields, etc.) that constitutes the source of the ammonia emissions has not yet been determined with certainty. Certainly the corrals, settling basins, ponds, and crop fields that receive effluent all contribute to ammonia emissions. Few studies of particular sources are available. One study evaluated ammonia volatilization from an anaerobic pond in cattle and swine facilities (Sommer et al. 1993). Another study of ammonia emissions from dairy ponds in Washington measured a flux rate of 30 to $75 \,\mu\text{g/m}^2/\text{sec}$, for a 390-cow dairy (Mount et al. 2001). The pond area from which these flux measurements were taken was approximately 1 acre, which may translate into an emission rate from approximately 21 to 53 lbs/head/year or approximately one quarter to three quarters of the total ammonia emission rate of 74 lbs/head/year (Flocchini et al. 2001). The average emission factor for conventional fertilizer applications to cropped fields statewide was calculated to be 2.38 percent of ammonia in the fertilizer (Krauter and Potter undated).

Ammonia concentrations have been measured at 0.36 to 0.98 mg/m³ at dairy corrals (Sweeten et al. 1999), 3.0 to 4.8 mg/m³ at a dairy freestall area (Oorlhoek and Kroodsma 1991), and 0.54 mg/m³ at a corral in the Chino, California area (Luebs et al. 1974). Ammonia concentrations approximately 0.25 miles downwind of a 600-cow dairy in Chino have been measured at 0.018 mg/m³ (Luebs et al. 1974).

Health Effects. Ammonia is a colorless gas or liquid with a strong irritating odor. Exposure to ammonia as a liquid, gas, or in solutions produces irritation to the skin, eyes, and respiratory tract.

Acute accidental exposure to high concentrations of ammonia may result in epidermal blistering, severe irritation, and edema to the nasalpharyngeal passages and pulmonary tissues. Airway obstruction induced by ammonia toxicity results in reduced ventilation, pulmonary oxygen transfer, and fluid accumulation (White 1971). High concentrations of ammonia produce irritation and damage to the eye ranging from irritation to acute corneal injury. Gaseous and/or liquid ammonia can cause burns to the cornea and iris, death to the corneal epithelium and lens, and, in some cases, blindness.

Ammonia exposure to experimental animals in acute and subchronic exposure scenarios resulted in increased respiratory rates and mild to severe irritation of the respiratory tract (Mayan and Merilan 1972). Rats, guinea pigs, rabbits, dogs, and primates exposed to repeated 8 hour per day, 5 days per week exposure for 6 weeks showed symptoms ranging from mild irritation to eye irritation at doses of 125 mg/m³ to death at doses of 470 mg/m³. Death resulted from related respiratory problems (Coon et al. 1970).

Experimental ammonia exposure to human volunteers resulted in development of olfactory thresholds between 10 and 20 ppm and irritation tolerances of between 100 and 150 ppm (Fergusen et al. 1977). In a paired exposure experiment involving knowledgeable and lay subjects, there were significant differences in threshold responses to ammonia vapors, although all subjects expressed eye and nasal irritation, general discomfort, and headache. All volunteers terminated their exposures at a level of 140 ppm (Verberk 1977).

Workers have been occupationally exposed to ammonia gas in large concentrations often through accidental, uncontrolled releases from storage tanks, refrigeration systems, or ruptured lines (White

1971; Hatton et al. 1979; Montague and MacNiel 1980). Accidental exposures in such cases have resulted in occasional fatalities.

There is currently no evidence to indicate that ammonia creates problems in reproduction in laboratory animals or humans. In addition, there are no reported instances of ammonia-induced carcinogenicity in humans or animals. However, there has been speculation about possible carcinogenicity in certain industrial settings, where there are mixtures of coal gases, ammonia, and isobutyl oils (Bittersohl 1971).

The United States OSHA has established an 8-hour TWA PEL of 35 mg/m³ for ammonia vapors in the workplace (29 CFR - 1910.1000, Subpart Z).

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REFERENCES

- American Lung Association (ALA), 2018. State of the Air 2018. Accessed on January 23, 2019 at < https://www.lung.org/our-initiatives/healthy-air/sota/ >.
- Bittersohl, G., 1971. Epidemiologische Untersuchgen uber Krebserkankungen in der chemischen Industrie. Arc. Geschwulstforsch 34:192-209.
- Burnett, R.T., Brook, J.R., Yung, W.T., Dales, R.E., and Krewski, D., 1997. Association between Ozone and Hospitalization for Respiratory Diseases in 16 Canadian Cities. Environmental Research 72, 24-31. 1997.
- California, State of, Air Resources Board (ARB), 2014. Ozone and PM₁₀ Data Summaries (2004-2013). Accessed on September 17, 2014 at
- , 2013. 2012 Estimated Annual Average Emissions. Emission Inventory Data by Air Basin and by County. Published in 2013. Page last reviewed January 26, 2017. Accessed on January 24, 2019 at http://www.arb.ca.gov/ei/emissiondata.htm
 - _, 2003. California Emission Inventory and Reporting System (CEIDARS).
- Chen, L., B.L. Jennison, W. Yang, and S.T. Omaye. 2000. Elementary School Absenteeism and Air Pollution. Inhalation Toxicology 12:997-1016. November 2000.
- Chow, J.C. and R.T. Egami. 1997. San Joaquin Valley 1995 Integrated Monitoring Study: Documentation, Evaluation, and Descriptive Data Analysis of PM₁₀, PM₂₅ and Precursor Gas Measurements. Final Report. DRI Document No. 5460.1F1. Prepared for the California Air Resources Board. Sacramento, CA.
- Coon, R.A., R.A. Jones, L.J. Jenkins, Jr., and J. Siegel. 1970. Animal inhalation studies on ammonia, ethylene glycol, formaldehyde, dimethylamine and ethanol. Toxicol. Appl. Pharmacol. 16:646-655.
- Eckard, R. 2007. "The abatement challenge for Australian Agriculture." The University of Melbourne and Department of Primary Industries, Victoria.
- EPA. See United States, Environmental Protection Agency.
- Fergusen, W.S., W.C. Koch, L.B. Webster, and J.R. Gould. 1977. Human Physiological Response and Adaptation to Ammonia. J. Occup Med 19:319-326.
- Flocchini, R.G., T.A. James, L.L. Ashbaugh, M.S. Brown, O.F. Carvacho, B.A. Holmen, R.T. Matsumura, K. Trzepla-Nabaglo, and C. Tsubamoto. 2001. Interim Report. Sources and Sinks of PM₁₀ in the San Joaquin Valley. August 2001.
- Frischer, T., M. Studnicka, C. Gartner, E. Tauber, F. Horak, A. Veiter, J. Spengler, J. Kuhr, R. Urbanek. 1999. Lung Function Growth and Ambient Ozone: A Three-Year Population Study in School Children. Am. J. Respir Crit Care Med, August 1999; 160:390-396.

- Gaffney, Patrick. 2008. Staff Air Pollution Specialist, California Air Resources Board. Personal communication with Valerie Rosenkrantz, Planning Partners, regarding air quality modeling and evaluation of human health effects. March 20, 2008.
- Gilliland, F.D., K. Berhane, E.B. Rappaport, D.C. Thomas, E. Avol, W.J. Gauderman, S.J. London, H.G. Margolis, R. McConnell, K. T. Islam, and J.M. Peters. 2001. The Effects of Ambient Air Pollution on School Absenteeism Due to Respiratory Illnesses. Epidemiology 2001, 12:43-54.
- Hatton DV, Leach CS, Beaudet AL, et al. 1979. Collagen breakdown and ammonia inhalation. Arch Environ Health 34:83- 86.
- Howard, Cody J., et al, 2010. "Reactive Organic Gas Emissions from Livestock Feed Contribute Significantly to Ozone Production in Central California." *Environmental Science & Technology*, no. 44 (2010): 2309-2314.
- IRIS. 2006. Hydrogen sulfide.
- James, T., D. Meyer, E. Esparza, E.J. DePeters, and H. Perez-Monti. 1999. Effects of Dietary Nitrogen Manipulation on Ammonia Volatilization from Manure from Holstein Heifers. Journal of Dairy Science, 82:2430-2439.
- Jorres, R., D. Nowak, and H. Magnussen. 1996. The Effect of Ozone Exposure on Allergen Responsiveness in Subjects with Asthma or Rhinitis. Am J Respir Crit Care Med 1996, 153:56-64.
- Kinney, P.L. and M. Lippmann, 2000. Respiratory Effects of Seasonal Exposures to Ozone and Particles. Archives of Environmental Health 55 (3):210-6. 2000.
- Klaassen, C.D.(editor). 2001. Casarett and Doull's Toxicology The Basic Science of Poisons. Sixth Edition. McGraw-Hill.
- Krauter, C. and C. Potter. undated. Ammonia Emissions and Fertilizer Applications in California's Central Valley. Undated.
- Kunzli, N., F. Lurmann, M. Segal, L. Ngo, J. Balmes, and I.B. Tager. 1997. Association between Lifetime Ambient Ozone Exposure and Pulmonary Function in College Freshmen— Results of a Pilot Study. Environmental Research 72, 8-23. 1997. LACWV. See Los Angeles County West Vector & Vector-Borne Disease Control District.
- Levy, J.I., J.K. Hammitt, and J.D. Spengler. 2000. Estimating the Mortality Impacts of Particulate Matter: What can be Learned from Between-Study Variability? Environ. Health Perspect. 108:109-117.
- Lippman M., Ito K. Nadas, and A. Burnett. 2000. Association of particulate matter components with daily mortality and morbidity in urban populations, Res Rep Health Eff Inst (2000) 95: 5–72 discussion 73–82.

- Luebs, R.E., K. Davis, and A. Laag. 1974. Diurnal Fluctuation and Movement of Atmospheric Ammonia and Related Gases from Dairies. Journal of Environmental Quality 3(3): 265269.
- Magliano, K.L., 1997. Chemical Mass Balance Modeling of Data from the 1995 Integrated Monitoring Study. Draft Final Report. California Regional PM₁₀/PM₂₅ Air Quality Study.
- Mayan, M.H., and C.P. Merilan, 1972. Effects of Ammonia Inhalation on Respiration Rates in Rabbits. J. Animal Sci 34:449-452.
- Mitloehner, Frank M. and Marc B. Schenker, 2007. Environmental Exposure and Health Effects from Concentrated Animal Feeding Operations. *Epidemiology*. 2007; 18: 309-311.
- Montague, T.J., and A.R. MacNiel. 1980. Ammonia inhalation. Chest 77:496-498.
- Mortimer, K.M., I.B. Tager, D.W. Dockery, L.M. Neas, S. Redline. 2000. The Effect of Ozone on Inner-City Children with Asthma. Am J. Respir Crit Care Med 2000; 162:1838-1845.
- Mount, George, B. Rumburg, B. Lamb, J. Havig, and H. Westberg. 2001. DOAS Measurement of Atmospheric Ammonia Emissions at a Dairy. 10th International Emission Inventory Conference. Emission Factor and Inventory Group, U.S. EPA.
- Oorlhoek J. Kroodsma W. and Hoeksma P. 1991. Ammonia emission from dairy and pig housing systems. In Odow and Ammonia Emissions/rom Lioerrock Farming (edited by Nielsen V. C. Voarburg J. H. and L'Hermit; P,). pp. 31-41, Elrevier Applied Science. London.
- Peden, D., R.W. Setzer, Jr., and R.B. Devlin. 1995. Ozone Exposure Has Both a Priming Effect on Allergen-induced Responses and an Intrinsic Inflammatory Action in the Nasal Airways of Perennially Allergic Asthmatics. Am J Respir Crit Care Med 1995; 151:1336-45.
- Peters, J.M., E. Avol, W.J. Gauderman, W.S. Linn, W. Navidi, S.J. London, H. Margolis, E. Rappaport, H. Vora, H. Gong, and D.C. Thomas. 1999. A Study of Twelve Southern California Communities with Differing Levels and Types of Air Pollution. Am J Respir Crit Care Med 1999; 159:768-775.
- Poda, G.A. 1966. Hydrogen Sulfide can be handled safely. arch. Environ. Health. 12:795-800.
- Pun, B. and C. Seigneur. 1998. Conceptual Model of Particulate Matter Pollution in the California San Joaquin Valley. Prepared for Technical and Ecological Services, Pacific Gas and Electric Company. September 1998.
- Roe, Stephen M. and G. E. Mansell. 2001. Next Generation Ammonia Inventory for the San Joaquin Valley of California. 10th International Emission Inventory Conference. Emission Factor and Inventory Group, U.S. EPA.
- Samet, J.M., F. Domnici, F.C. Curriero, I. Coursac, and S.L. Zeger. 2000. Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994. New England Journal of Medicine, 343(24): 1742-1749.

- San Joaquin Valley Air Pollution Control District. 2006. "Dairy and Feedlot PM₁₀ Emission Factors." Office Memo. April 12, 2006.
- Sartor, F., C. Demuth, R. Snacken, and D. Walckiers. 1997. Mortality in the Elderly and Ambient Ozone Concentration during the Hot Summer, 1994, in Belgium. Environmental Research 72, 109-117. 1997.
- Sheth and Giel. 2000. Understanding the PM-2.5 Problem. Pollution Engineering Online. March 2000.
- Sommer, S.G., B.T. Christensen, N.E. Nielsen, and J.K. Schjorring. 1993. Ammonia volatilization during storage of cattle and pig slurry—Effect of surface cover. J. Agric. Sci. 121:63–71.
- Sweeten, J.M., L. Erickson, P. Woodford, C. Parnell, K. Thu, T. Coleman, C. Reeder, J. Master, W. Hambleton, G. Bluhm, and D. Triasto. 2000. Air Quality Research and Technology Transfer White Paper and Recommendation for Concentrated Animal Feeding Operations. Adopted by USDA Agricultural Air Quality Task Force, Washington, DC. July 19, 2000.
- United States, Department of Agriculture Agriculture Research Service (USDA-ARS). 2000. Manure and Biproduct Utilization National Program.
- United States, Environmental Protection Agency (EPA), 2013. The National Emissions Inventory, 2011 National Emissions Inventory Data. Last updated on June 13, 2014. Accessed on June 13, 2014 at http://www.epa.gov/air/emissions/index.htm
- _____, 2011. PM2.5 Objectives and History. Accessed on June 13, 2014 at http://www.epa.gov/pmdesignations/faq.htm
- _____, 2001. Air Quality Criteria for Particulate Matter, Volumes I and II, United States Environmental Protection Agency, Office of Research and Development. EPA 600/P-99/002bB. March 2001.
- _____, 1999a. EIIP Volume IX: Chapter 1: Getting Started: Emission Inventory Methods for PM_{2.5}. United States Environmental Protection Agency, Emissions Inventory Improvement Program. July 1999.
- Verbeck, M.M., 1977. Effects of ammonia in volunteers. Int. Arch. Occup. Environ. Health. 39:73-81.
- White, E.S., 1971. Case report. A case of near fatal ammonia gas poisoning. J. Occup. Med 13:549-550.