6.3 Human Health Effects

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While other public health impacts, which include human exposures to polluted water or antibiotic resistant microorganisms that may arise from CAFOs, are not being addressed in this chapter, occupational exposures to CAFO environments will be reviewed and discussed because of their relevance to human response to CAFO air emissions.

The lung contains the largest epithelial surface in the body, consisting of more than 100 square meters of surface area in the average adult male (compared with approximately 2 square meters of skin). The average adult male inhales up to 15 kg of air daily, and children inhale proportionately more for their size. Because of this high surface area and high volume of air exchanged, the lung is capable of absorbing vast quantities of inhaled substances. Defense mechanisms of the lung, including the cough reflex, mucociliary transport, and the innate immune system are efficient at combating inhaled particulate matter and microorganisms. Gases, vapors, and aerosols (of "respirable" size, approximately 1-10 microns in diameter) are readily inhaled and absorbed.

Health effects associated with inhalation of toxins and bioaerosols are manifold. Medical problems commonly associated with inhaled agents include respiratory diseases (asthma, hypersensitivity pneumonitis, industrial bronchitis), cardiovascular events (sudden death associated with particulate air pollution), and neuropsychiatric conditions (due to odor as well as delayed effects of toxic inhalations).

Most studies of human exposures to airborne agricultural hazards have focused on occupational exposures in agricultural settings. With the rise of large, industrial CAFOs as the preeminent form of livestock production and their associated higher production of gases, vapors, and fumes, these exposures now have the potential to affect larger numbers of individuals, including members of the neighboring community not involved in agriculture or related industrial livestock production. Few studies have directly examined the health effects of proximity or exposure to CAFOs in the community, thus extrapolations must be considered from well-documented effects of these toxins in laboratory settings and occupational exposure studies. Donham and colleagues (1977) first reported that workers in swine confinement facilities described significantly more respiratory symptoms than non-exposed workers; subsequent studies have confirmed this symptomatology and have also documented increased risks of respiratory infections, progressive declines in pulmonary function, and poisoning from hydrogen sulfide in this occupational group.

For many reasons, standards for community exposures to the toxic agents released from CAFOs must be stricter than that for occupational exposures. First of all, community members may include subgroups of especially susceptible individuals, for example the elderly, children, and those with pre-existing impairments. Secondly, community members may be exposed continuously to released substances rather than for a workshift or less; this is especially true for those who do not work outside the home, and for pre-school children. Moreover, exposed community members may not have chosen to live in proximity to a CAFO, whereas occupationally exposed individuals have some choice in their employment. Thus, ambient exposure levels arising from CAFOs, including ammonia and hydrogen sulfide, must be significantly lower than occupational levels; notwithstanding, many components of the CAFO environment, e.g. bioaerosols including endotoxins and glucans, have no current recommended or mandated occupational exposure limits.

6.3.1 Studies of Adverse Health Effects from Specific Exposures found in CAFO Emissions

It is important to examine the literature regarding adverse health effects arising from individual chemicals and mixtures of chemical compounds, often referred to as odorants, exposures known to be components of emissions from CAFOs. The following is a summary of available, published findings from clinical, experimental and epidemiological observations for several categories of these exposures. The concentration of exposure is not always known or measured, but there have been several studies of individual chemical exposures that have documented both concentrations and durations of exposure, some at very low levels. The vast majority of these observations come from occupational, experimental, and non-CAFO community exposures, many of which were made among selected populations of workers or healthy volunteer subjects. Regulatory agencies have used many of these findings, taking into account uncertainty and susceptibility factors, in making their recommendations regarding exposure limits for exposed communities (See Chapter 8.0).

6.3.1.1. Ammonia

Ammonia is both a component of animal waste and released in waste treatment processes. Well recognized as a human toxin, the current OSHA PEL for ammonia is a TWA of 50 ppm (also its odor threshold), although ACGIH and NIOSH recommend a lower TWA of 25 ppm. Concentrations of greater than 100 ppm have been regularly reported in poultry confinement operations (Mulhausen et al, 1987). The EPA has found that animal agricultural operations are responsible for almost three fourths of ammonia air pollution in the United States (Harris et al, 2001), although numerous other industries are associated with inhalation exposure to ammonia. EPA has recommended as reference concentration for chronic inhalation of ammonia of 1.4 ppm. ATSDR has recommended a long-term MRL of 300 ppb for community exposures (See Chapter 8.0 for detailed discussion).

Water-soluble, ammonia is rapidly absorbed in the upper airways, with the result of damaging upper airway epithelia. Moderate concentrations (50-150 ppm) can lead to severe cough and mucous production; higher concentrations (>150 ppm) may cause scarring of the upper and lower airways (Close et al, 1980; Leduc et al, 1992). A consequence of these inflammatory responses, in some cases, is reactive airways dysfunction syndrome (RADS) and associated persistent airway hyperresponsiveness (Bernstein and Bernstein, 1989; Flury et al, 1983). At higher concentrations, sufficient ammonia may bypass the upper airways to cause lower lung inflammation and pulmonary edema (Close et al, 1980; Sobonya, 1977). Massive exposure to ammonia can be fatal, including in the agricultural sector, a consequence to disruption of tanks of anhydrous ammonia (Sobonya, 1977). These fatalities, as well as the chronic lung disease seen following as little as two minutes of exposure to high concentrations of ammonia gas may result in the development of bronchiolitis obliterans (de la Hoz et al, 1996; Kass et al, 1972; Sobonya, 1977; Walton, 1973), restrictive lung disease (de la Hoz et al, 1996), and bronchiectasis (Leduc et al, 1992).

In addition to pulmonary disease, exposure to ammonia also leads to irritation of the eyes, sinuses, and skin. Exposure to 100 ppm ammonia for short (30 second) duration leads to nasal irritation and increases in nasal airway resistance (McLean et al, 1979). When increasing concentrations of ammonia are delivered by spontaneous respiration, severe nasal irritation develops at 134 ppm after 5 minutes; some individuals report symptoms as low as 32 ppm (Keplinger et al, 1973). Clinical sinusitis has been reported following accidental exposure to ammonia as well (Brautbar, 1998). Chemical burns to the skin and eyes are also commonly seen following high-concentration ammonia exposures (Latenser and Loucktong, 2000).

Although the most serious adverse effects of ammonia inhalation are usually seen with concentrations of ammonia that have been associated with fatal exposures (in the range of 500 ppm), evidence exists that lower concentrations of ammonia can reach the alveoli and may be adsorbed to respirable particulates, as may be seen in complex bioaerosols such as those found in the agricultural setting resulting in a research-recommended occupational exposure limit of 7 ppm (See Section 6.3.2.2). Similar occupational exposures to ammonia (9 ppm) have been studied among soda ash workers (Holness et al, 1989) who reported increased symptoms of coughing, wheezing, nasal complaints, eye irritation, throat irritation, and skin complaints; however, no changes in lung function were observed when measured over a working shift. It was noted that this was a cross-sectional study of a small population and that selection bias may have therefore occurred.

6.3.1.2. Hydrogen Sulfide

Hydrogen sulfide is one of the most important of the gases arising from the storage, handling, and decomposition of animal wastes. Smelling like rotten eggs, this gas that is recognized as both an irritant and an asphyxiant, is a prominent component of odorants released from CAFOs. Current OSHA PEL for H_2S are 10 ppm (with STEL of 15 ppm), while NIOSH has recommended a time weighted average occupational exposure limit of 10 ppm. For community exposures, EPA has recommended a reference concentration for long-term exposure of 7 ppb (See Chapter 8.0 for full discussion).

Levels as high as 1,000 ppm have been reported (Donham and Gustafson, 1982) following the perturbation of manure lagoons, and levels greater than 100 ppm are considered immediately hazardous to life and health. Exposure to these elevated levels of H_2S can cause rapid loss of consciousness, and H_2S has been implicated in a number of deaths when encountered in confined environments in agricultural settings. The primary mode of absorption of H_2S is through inhalation (Bhambhani et al, 1996a).

One particular hazard is that, although the odor threshold is quite low (less than 1 ppm), at levels over 6 ppm the intensity of the smell only modestly increases; above 150 ppm, exposure to hydrogen sulfide may actually reduce the sense of smell, hindering the olfactory detection of high concentrations of the gas and making H_2S monitoring equipment mandatory in occupational settings (van Aalst et al, 2000). The toxic effects of hydrogen sulfide are based on its property as a chemical asphyxiant; it binds to the mitochondrial enzyme cytochrome oxidase, blocking oxidative phosphorylation and ATP production. This leads to anerobic metabolism and the development of lactic acidosis (Nichols and Kim, 1982).

Experimental exposure studies have been carried out examining the effects of inhalation of low levels of H2S on healthy volunteers (Bhambhani et al 1996a, 1996b, 1997). Inhalation of 5 ppm of H_2S by exercising men leads to a significant decrease in the concentration of citrate synthase, a marker of aerobic metabolism, in muscle biopsy tissue, although no increases in lactic acidosis were noted (Bhambani et al, 1996b). Levels of 10 ppm cause no change in physiologic measures of pulmonary function (Bhambani et al, 1996a), but do cause a significant decline in maximal oxygen update (VO2max) and an associated increase in blood lactate in exercising men and women (Bhambani et al, 1997). Jappinen and colleagues (1990) exposed a group of asthmatics (severe asthmatics were eliminated from the study) to 2 ppm of hydrogen sulfide for 30 minutes. Three complained of headache and two were found to have increased airway resistance, but there was no

change in other lung function values or associated symptoms. Members of a Mobile Monoriting Team of the Texas Natural Resource Conservation Commission (TNRCC) evaluated hydrogen sulfide concentrations downwind from an oil refinery and reported 0.09 ppm 30-minute averages over a period of five hours (Texas Natural Resources Conservation Commission, 1998). Six staff members reported eye and throat irritation, headache, and nausea. These experimental studies indicate consistent patterns of adverse health effects after short, low concentrations of exposure to hydrogen sulfide.

Epidemiological studies of workers exposed to hydrogen sulfide exposure include pulp mill workers who reported increased respiratory symptoms (irritation and cough), as well as increased headache and migraine; it was noted that these workers were also exposed to other sulfur compounds including sulfur dioxide and mercaptans (Partti-Pellinen et al, 1996). Jappinen and colleagues (1990) studied pulp mill workers thought to be exposed to hydrogen sulfide levels usually below a maximum permitted concentration of 10 ppm and reported no significant changes in lung function and airway hyperresponsiveness at the end of the workday, compared with control values. Hessel and colleagues (1997) studied oil and gas workers at undefined, but probably moderately high, exposures to hydrogen sulfide (as some of the workers lost consciousness); nearly third of the workers reported symptoms.

Several additional epidemiological studies of community residents exposed to low levels of hydrogen sulfide have been reported. A U.S. Public Health Service study of a general population exposed to levels in excess of 0.3 ppm reported adverse health effects including shortness of breath, eye irritation, nausea, and loss of sleep (United States Public Health Service, 1964). Jaakkola and colleagues (1991) studied chronic community exposure to hydrogen sulfide and TRS (total reduced sulfur) compounds (hydrogen sulfide annual means of 0.006 ppm and daily means of 0.07 ppm) and found that both asthma and chronic bronchitis were slightly more prevalent, that eye and nasal symptoms were found significantly more often, and that these symptoms were dose-related. They concluded that the WHO standard of 0.1 ppm (24 hour average) did not protect against these adverse health effects. Jaakkola and colleagues (1991) also studied the respiratory infection rate among infants exposed to ambient hydrogen sulfide levels of 0.001 ppm, and at half-hour maximal exposures of 0.125 ppm, and reported that exposed infants had higher rates of respiratory infection, but that combined effects of other air pollutants may have been contributing factors. Haahtela and colleagues (1992) studied community residents exposed to peak exposures of hydrogen sulfide of 0.095 ppm (four hour average) and 0.025 and 0.030 ppm over two days of exposure, compared to control days, with four hour exposures ranging between 0.00007 and 0.002 ppm. Cough, throat irritation, and eye symptoms were observed significantly more often during the peak exposure period. The author concluded that the WHO guideline of 0.10 ppm for a 24 hour average did not provide adequate protection from adverse health effects. Rossi and colleagues (1993) studied the occurrence of asthma attacks in relation to air pollution events (hydrogen sulfide levels ranged from the highest 1 hour mean of 0.011 ppm and daily 24 hour means of 0.002 ppm), and reported significant associations between the frequency of asthma attacks at an emergency room and nitrogen sulfides, sulfur dixoxide, total suspended particulates, and hydrogen sulfide. Partti-Pellinin and colleagues (1996) studied a general population exposed to TRS levels of up to 0.1 ppm over a 24-hour period. Based on a self-administered questionnaire, the authors concluded that the exposed community reported more cough, respiratory infections, and headaches than the reference community, and also that headaches, depression, tiredness, and nausea were more often reported on days when the 1 hour or daily mean TRS levels exceeded 0.028 ppm (both communities were exposed to similar levels of sulfur dioxide). These community studies of hydrogen sulfide and TRS

exposures are especially useful because they report measured low levels of exposure and associated adverse health effects. However, as is the case with community exposures to CAFOs, these are invariably mixed exposures to hydrogen sulfide and other chemicals, some of which may contribute to the adverse health effects described in these studies. Campagna and colleagues (2001) studied the effects of ambient hydrogen sulfide and TRS levels on hospital visits for respiratory diseases among children and adults in Dakota City and South Sioux City, Nebraska. While peak levels of hydrogen sulfide were as high as 1,375 mean levels over an entire day were much lower. An increase in asthma hospital visits was seen a day following peak TRS exposures among children and an increase in hospital visits for all respiratory disease was seen following peak exposures for both TRS and hydrogen sulfide.

Finally, Xu and colleagues (1998) has reported a retrospective epidemiological study of spontaneous abortion among a large cohort of female workers in a petrochemical plant in Beijing, China. Among women exposed only to hydrogen sulfide (concentrations were not reported because of the retrospective nature of the study), a rate of spontaneous abortion of 12.3% was observed and a significant association with hydrogen sulfide exposure was reported (OR, 2.3, CI 1.2-4.4).

Chronic low-level exposure is associated with anosmia, the loss of ability to detect odors. At higher levels, hydrogen sulfide exposure causes loss of consciousness, shock, pulmonary edema, coma and death. Survivors of hydrogen sulfide poisoning are reported to commonly have neuropsychiatric defects which may be permanent; a recent study by Kilburn of University of Southern California has demonstrated that even exposure to low concentrations of hydrogen sulfide leads to significant neuropsychologic abnormalities, including impaired balance, visual field performance, color discrimination, hearing, memory, mood, and intellectual function (Kilburn, 1997). These effects may be due to anoxic encephalopathy.

6.3.1.3. Particulates

The air in CAFOs is contaminated with high concentrations of particulates, approximately one quarter of which is protein; about one third of suspended dust is considered respirable (< 10 microns in diameter, PM10). Occupational and environmental studies have demonstrated an average of 2-6 mg/m^3 dust concentrations, and levels up to 20 mg/m^3 may be encountered. National ambient air standards for PM10 are an annual average of 50 mcg/m^3 with a 24-hour average of 150 mcg/m^3. Of these, particles between 4 and 10 microns are deposited in the airways and smaller particles (< 2.5 microns) progress into and may be absorbed by the terminal bronchiols and alveoli. Particles which settle in the upper airways are associated with asthma and bronchitis; smaller particles may be absorbed and have systemic effects including, in studies of urban air pollution, increased rates of cardiac death. In addition to direct inflammatory response to inhaled allergens, dust can also convey inflammatory and/or irritating gases or chemicals (such as ammonia, hydrogen sulfide, or endotoxin) deeper into the lung, thereby enhancing their toxic effects.

Although certain mineral particulates, such as silica dioxide, lead to characteristic pulmonary inflammatory and scarring conditions known as pneumoconioses, even inhalation of seemingly inert dust particles appear to have adverse long-term consequences. In a number of occupational settings, cumulative exposure to dust particles in the respiratory range is one of the most important causes of persistent respiratory symptoms and progressive declines in lung function (Healy et al, 2001; Ulvestad et al, 2001); and this has also been reported in non-occupational settings (Dockery and Pope, 1994; Dockery et al, 1993; Pope et al, 1995, Lippmann et al, 2000).

6.3.1.4. Bioaerosols

An important component of the environment released from CAFOs is microbiologic in origin. Swine manure contains up to 10^8 coliform bacteria/gram, and CAFOs contain these organisms in airborne and respirable particles; total organism load may exceed 10^{10} cfu/m^3 at times. Some of the microorganisms that are present in the CAFO environment are human pathogens, creating a potential risk of infection for those exposed to these agents. Dust in CAFOs and other agricultural settings, contains far more than merely viable organisms. Microbial products of medical importance include antigens, glucans, and endotoxins.

Exposure to protein antigens derived from plants, animals, and microbes are known to cause a variety of medical problems. Inhalation of thermophilic bacteria, commonly found in moldy hay and other damp locations, leads to a condition known as hypersensitivity pneumonitis, a respiratory condition characterized by granulomatous inflammation of the lung, restrictive physiology, and progressive dyspnea. Associated with detection of antibodies to these organisms in the blood, hypersensitivity pneumonitis (also known as "farmer's lung" in agricultural settings), is found among agricultural workers and others occupationally exposed to these agents (Skorska et al, 2000).

Asthma may also be caused or exacerbated by exposure to conditions common in CAFOs. Atopic asthma is caused, in susceptible individuals, by sensitization to and subsequent inhalation of allergens, agents that can lead to asthma in previously non-sensitized individuals. Those with a previous diagnosis of asthma may have their asthma triggered in a non-specific way by exposure to the dust and irritant-inducing agents arising from the CAFO environment. CAFOs contain, among other compounds, high concentrations of grain dust, dust mites, animal dander, pollen grains, molds and fungal spores, and dried fecal particles, each of which may induce or exacerbate asthma. Proximity to CAFOs, and periodic/seasonal agricultural activities (e.g., agriculture chemical and manure applications), are frequently cited by rural asthma patients as exposures resulting in asthma exacerbation making asthma control more difficult.

Endotoxins are lipopolysaccharide complexes that are products of gram-negative bacterial cell walls. Ubiquitous in the environment, they are present in high concentrations in agricultural settings such as grain elevators, feed barns, and CAFOs. Endotoxins are important components of exposures responsible for the adverse health effects following inhalation of organic agricultural dust. Acute effects of endotoxin inhalation include symptoms of cough, chest tightness, and dyspnea and alterations in pulmonary function characterized most typically by a decline in FEV1; over a working shift and overtime; systemic effects include fever, rigors, myalgia, arthralgia, and other "flu-like" symptoms. Although no occupational standards currently exist for endotoxin in the United States, Dutch Expert Committee on Occupational Standards of the National Health Council has proposed a limit of 50 EU/m^3 (4.5 ng/m^3) over an 8-hour exposure period (Heederik and Douwes, 1997).

Kline and colleagues (1999) evaluated the responses of 72 normal, non-smoking, non-atopic, non-asthmatic volunteers who were exposed to graded doses of endotoxin by inhalation in a clinical exposure facility. Each subject first inhaled 0.5 mcg of endotoxin then underwent spirometry prior to inhaling a greater concentration of endotoxin. Cumulative levels of endotoxin inhalation consisted of 0.5, 1.5, 3.5, 6.5, 11.5, 21.5, 41.5 mcg. The protocol was terminated for decline in FEV1 to < 90% of baseline or a total of 41.5 mcg. Among study participants, a wide range of sensitivity to the bronchospastic effects of inhaled endotoxin was found; some individuals demonstrated a 20%

decline in FEV1 following inhalation of as little as 1.5 mcg whereas others were resistant to these effects and did not even decline by 10% following inhalation of over 41.5 mcg. In a separate study, asthmatic individuals were found to have an enhanced degree of symptoms and bronchospasm following inhalation challenges compared with normal control subjects (Kline et al, 2000). Other studies have also found that inhalation exposure to endotoxin and endotoxin-containing grain dust leads to the development of bronchospasm and airway inflammatory responses (Blaski et al, 1996; Jagielo et al, 1996; Michel et al, 1989; Michel et al, 1996; Michel et al, 1997; Schwartz et al, 1995a).

Most of the reports of community, occupational, and ambient effects due to endotoxin exposure are related to inhaled endotoxin; this is clearly different than the case of patients suffering from gram-negative infections, who are typically exposed to endotoxin via the blood stream. The greatest effect of inhaled endotoxin is on airway inflammation and the induction of bronchial hyperresponsiveness, both characteristic of asthma. Interestingly, some recent studies have demonstrated a protective effect of endotoxin exposure relative to the development of allergic disease. Von Mutius and colleagues (2000) recently reported that environmental endotoxin exposure of farmers' children protects them from the development of atopy; Gereda and colleagues (2000), in a study of urban homes, found that home levels of endotoxin inversely correlated with likelihood of allergen sensitization in infants. In a similar vein, Gehring and colleagues (2001) found that environmental exposure to endotoxin protected infants from the development of atopic eczema. These effects of endotoxin on early-life development of allergic responsiveness may be due to the deviation away from a Th2-type response to allergens and towards a Th1-type response, however alternate explanations are possible.

Exposure of adults, however, (and infants and children in some studies) appears to be clearly detrimental with regards to airway function and asthma. In contrast to the studies showing protective effects of endotoxin on the development of disease among infants, Park and colleagues (2001) reported that infants with at least one asthmatic/allergic parent were placed at increased risk of developing wheezing when their home environment contained higher levels of ambient endotoxin. Douwes and colleagues (2000), in a community study of household dust, found that endotoxin content of dust was associated with increased peak flow variability among asthmatic children. Michel and colleagues (1991) reported that asthmatic patients with higher levels of home endotoxin exposure develop more symptoms and require more intensive treatment than those from homes with lower levels of endotoxin. In a separate study, the same group confirmed that asthma severity correlates with endotoxin exposure (Michel et al, 1992). In a study conducted in Brazil, Rizzo and colleagues (Rizzo et al, 1997) found that endotoxin (but not dust mite) content of dust significantly correlated with symptom scores in asthmatic children.

Controlled laboratory studies of endotoxin exposure confirm that inhalation induces airway inflammation and bronchial hyperreactivity. Blaski and colleagues (1996) reported that both normal control subjects and atopic individuals developed airway neutrophilia and reduced airflow following inhalation of 0.4 mcg/kg of endotoxin. Jagielo and colleagues (1996) found that the endotoxin content of grain dust was responsible for its ability to induce inflammation and obstructive airway physiology in normal volunteers. Michel and colleagues (1989) found that endotoxin inhalation by asthmatics resulted in significantly more airflow reduction than in normals. Among asthmatics, the reduction in airflow (Michel et al, 1992) and development of symptoms of chest tightness and dyspnea (Kline et al, 2000) are greater than the difference in development of airway inflammation. Even among non-asthmatics, a significant variability in responsiveness to the effects of inhaled

endotoxin can be seen (Kline et al, 1999); this appears to be explained, at least in part, by genetic factors (Arbour et al, 2000).

Mycotoxins, beta-glucans, and other components of fungal pathogens appear to have a similar range of toxicity to endotoxins, including both inflammatory and immunostimulatory effects. These compounds, however, have been less well studied in human exposures, and their concentration in CAFOS is unknown (American Thoracic Society, 1998).

6.3.1.5. Volatile Organic Compounds

Of the thousands of gases, vapors, particles, and aerosols present in CAFOs, over 24 odorous chemicals, often referred to as odorants, have been identified (Cole et al, 2000). Volatile acids, mercaptans, and amines are particularly odorous even in miniscule concentrations. Ammonia and hydrogen sulfide, as noted above, are also pungently aromatic.

Although long recognized as a neighborhood nuisance, recent studies have suggested that odiferous exposures emitted from CAFOs may well have adverse health effects (Schiffman et al, 2000). Odor appears to play a significant role in the recognition of and concern over symptoms in neighbors of hazardous waste sites (Shusterman, 1992; Shusterman et al, 1999). Schiffman and colleagues (1995) from Duke University have reported that indicators of altered mood, assessed using validated scales, are significantly worse in subjects who live in the vicinity of intensive swine operations compared with control subjects.

Chen and colleagues (1999) have demonstrated, using odor threshold dilution analysis, that odor intensity in swine buildings is reproducible and measurable. Zahn and colleagues (2001) have analyzed malodorous volatile organic compound components of swine production facility air samples, and have demonstrated, using an artificial swine odor solution, that alterations in the concentrations of these components can be detected by study subjects. No odor studies were found that related the quantitative measurement of odor intensity in the downwind air stream from livestock facilities with adverse health effects among community residents. However, there is an extensive literature relating non-CAFO odors and adverse health effects that are relevant to community exposures to CAFO exposures.

Of the hundreds of gases, vapors, particles, and aerosols present in CAFOs, 331 volatile organic compounds (VOCs) and fixed gases were recently characterized by Schiffman and colleagues (2001). These compounds, assessed at the point of emission, included many acids, alcohols, aldehydes, amides, amines, aromatics, esters, ethers, fixed gases, halogenated hydrocarbons, hydrocarbons, ketones, nitriles, other nitrogen-containing compounds, phenols, sulfur-containing compounds, steroids, and other compounds. The authors (Schiffman et al, 2001) further observed that the vast majority of these compounds were found at concentrations below their published irritant or odor thresholds, yet human assessments of the combined odors and their irritant effects were described as "strong" at a distance of 1000 feet.

While CAFO odors have long been recognized as a neighborhood nuisance, recent studies have suggested that odiferous exposures emitted from CAFOs may well have adverse health effects (Schiffman, 1997; Schiffman et al, 1995; Thu et al, 1997; Wing and Wolf, 2000). Direct measurement of odorous or other noxious substances were not made in these studies, therefore, a direct linkage to level of exposure could not be reported. A Duke University workshop summarized by experts in

assessing the potential health effects of odor from animal operations (Schiffman, Walker, Dalton, Lorig, Raymer, Shusterman and Williams) addressed this issue (Schiffman et al, 2001). They observed that health symptoms have been reported with increasing frequency from low level exposures from manures and biosolids; "the most frequently reported health complaints include eye, nose, and throat irritation, headache, nausea, diarrhea, hoarseness, sore throat, cough, chest tightness, nasal congestion, palpitations, shortness of breath, stress, drowsiness, and alteration in mood". They further observed that these symptoms usually occurred briefly at the time of exposure, but that hypersensitive individuals, such as asthmatics, could have their condition exacerbated with persisting symptoms.

Exactly how odors from CAFOs may result in these symptoms is not well understood. The Duke workshop discussed freeways, or paradigms, by which ambient odors may produce health symptoms (Schiffman et al, 2000). In the first paradigm, the symptoms may occur at levels of exposure that would also be expected to cause irritant effects from combinations of irritants that may be additive or synergistic in their effect. In this paradigm, the adverse health effect typically occurs at a higher level than the concentration at which the odor would first be detected.

In the second paradigm, symptoms may occur at odor concentrations below that expected from irritants. The mechanism by which these odorants may cause their adverse effects is not known (Schiffman et al, 2000). Schiffman and colleagues (1995) reported that CAFO odors perceived as unpleasant can impair mood. Shusterman and colleagues (1991) observed increased symptom prevalence and an "odor worry" interaction associated with odor from hazardous waste sites. Schiffman and colleagues (2000) summarized evidence that negative mood, stress, and environmental worry may lead to biochemical and physiological effects with subsequent health outcomes. Other studies suggest that bias concerning odors can alter the response relating to health effects (Dalton et al, 1997). These results provide evidence that both the perceived odor and cognitive expectations about a chemical can significantly affect individual response. Other studies have also demonstrated that ones current cognitive state can bias ancillary characteristics of an odor such as preference or acceptability (Knasko, 1993). Some studies have shown that persons can report experiencing strong odors as an outcome by showing that cognitive factors can lead to reports of odors when none are present (Knasko, 1992; O'Mahoney, 1978). Knasko and colleagues (1990) have also observed that an odorant stimulus is greatly influenced by the environment surrounding the exposure, which can include the social context or the perceiver's mental state. It is also recognized that those working in an odorous environment may adapt to the odor following long term exposure. Dalton and Wysocki (1996) have advocated for the development of laboratory procedures that combine long-term odor exposure in a naturalistic setting with psychological tests.

A third way for paradigm, is when the odorant is a part of a mixture that contains bioactive pollutants such as bioaerosols containing organic dust, endotoxin, glucans, allergens, microorganisms, or other toxins (Schiffman et al, 2000). In this paradigm, the individual is exposed to odors, but the adverse health effect is likely to arise from a non-odorant toxin. Relevant to this paradigm is the study of Reynolds and colleagues (1997) who sampled at 60 meters for hydrogen sulfide, ammonia, endotoxin, and total dust. A reason to sample for dust and ammonia together is that it is now recognized that some ammonia absorbs to respirable dust particles thereby providing a vehicle to transport ammonia and dust-latent toxins, like endotoxin, deep into the lung.

To date there has been relatively little research quantifying odorants. Zahn and colleagues (2001) completed a multi-component analysis of malodorous DOCs found in air samples from 29 swine

production facilities using a 19-component artificial swine odor solution. The results of this study concluded that this approach can be applied toward estimating perceived odor intensity. Schiffman and colleagues (2001) studied six swine operations in North Carolina. In addition to quantifying the DOCs and fixed gases from these facilities, they used six methods for trained human panel members to assess the intensity of odor at varying distances from swine facilities. Scentometer measurements were made at 12 feet, 750 feet, and 1250 feet from the swine facilities and range from a high of 170 D/T (dilutions to threshold) to a low of 2 D/T.

It is recognized that there is great variability between odors arising from CAFOs, and that odorous gases may be transformed through interactions with other gases and particulates between the source and the receptor (Peters and Blackwood, 1977). It is also recognized that there is variability in odor persistence, "persistence factor" defined as the relative time that odorous gases will remain perceptible (Summer, 1971). There is a need to combine quantitative assessments of odors with environmental measurements in well-designed, controlled studies of symptoms and other health outcomes at the community level.

6.3.1.6. Experimental Occupational Exposures among Naïve Subjects

Workers in CAFOS are exposed, on a daily basis, to a wide array of gases, vapors, dusts, and other compounds. Thus, it is challenging to identify, in this occupational setting, which specific components of their exposure is responsible for health outcomes. Experimental occupational exposures among normal volunteers have addressed this issue.

Two clinical epidemiological studies of normal volunteers in swine CAFOs have been reported, both from Canada. Cormier and colleagues (1997) exposed 7 previously non-exposed, normal subjects to a swine building and found significant respiratory symptoms, declines in lung function, and clear evidence of a marked inflammatory response via analysis of bronchoalveolar lavage (BAL) fluid post exposure. Total dust, endotoxins, and ammonia were measured but no individual exposures, rather a mixed exposure, appeared to be responsible for these adverse health effects. Senthilselvan and colleagues (1997) made similar observations among 20 naive subjects, while also showing that treatment of the swine facility with canola oil significantly reduced symptoms, declines in lung function, airway hyperresponsiveness, and mean dust and endotoxin concentrations.

6.3.2. Occupational Health Effects

The first description of health hazards to people working in these CAFO's was in 1977 (Donham et al, 1977). This early study revealed that over 60 percent of veterinarians working in these facilities experienced one or more respiratory health symptoms. This report led to many subsequent studies in the US, Canada, and Europe (Donham, 1993). In addition to respiratory illnesses, other occupational health problems associated with CAFOs have been documented, including traumatic injuries, noise-induced hearing loss, needle sticks, hydrogen sulfide and carbon monoxide poisonings, and infectious diseases (Donham et al, 1982a; Donham et al, 1982b; Donham, 1985).

Workers in confined poultry and dairy operations are also at risk, but most beef operations are in open lots, thus reducing worker respiratory exposures. The increasing industrialization of livestock production will continue to result in more independent producers leaving the industry, or becoming quasi-employees of large-scale producers as contract growers. Furthermore, many minority workers are becoming employees of larger producers, raising potential legal issues of undocumented workers and further need of OSHA regulation of these large operations. In the past, OSHA has been

restricted in agriculture because of a federal law that restricts enforcement on farms with ten or fewer employees. Many of the large industrial CAFOs now employ hundreds of workers and these workers will work full shifts in animal confinement buildings in contrast to smaller, independently owned CAFOs where periods of exposure are typically much shorter. This increase in large, industrial CAFOs will, therefore, likely lead to increased cumulative exposure and thus greater risk to adverse health effects. To date, OSHA has not addressed the CAFO issue, in spite of strong evidence of worker health risks.

The worker health component of this review is assembled to characterize the range of occupational health hazards associated with large-scale livestock production, but concentrates on health effects from air toxics and a brief discussion on measures needed to decrease health risks among workers.

Table 2 lists major categories of hazards and then further classifies diseases or health outcomes within those categories. The order does not necessarily relate to incidence, prevalence, or severity—these are common health risks among all intensive livestock production operations. The vast majority of the research in this area has been with swine production. Therefore, this report will deal largely with swine operations. However, similar exposures and adverse health effect observations have been made among those working in concentrated poultry production (Bar-Sela et al, 1984; Lenhart et al, 1990; Morris et al, 1991).

The principal health risks for CAFO workers result from respiratory exposures to a wide range of toxic, irritant, and inflammatory substances emitted into the air. Ammonia, hydrogen sulfide, carbon monoxide, particulate matter, endotoxin, and other bioaerosols have received the majority of research attention. However, infectious diseases, noise, trauma, fires, explosions, electrocutions, thermal stress, poisonings, and drowning are all also important causes of morbidity and mortality (Randolph and Rhodes, 1993). Often overlooked are emotional stress and chronic musculoskeletal pain that can lead to significant impairment and to disability in this workforce. This report will be limited to air toxics and resultant respiratory diseases.

6.3.2.1. Respiratory Diseases

Respiratory exposures lead to the most common health hazard among swine farmers and CAFO workers. There are both acute illnesses and chronic respiratory diseases among CAFO workers. The most serious acute hazard is hydrogen sulfide poisoning, which results from sudden exposure to high levels (> 500 ppm) of this gas. This is a confined space entry hazard (areas that are not vented and may trap toxic gases) in CAFOs, with hydrogen sulfide the principle hazard (Donham et al, 1982a; Osbern and Crapo, 1981). Acute respiratory distress syndrome (ARDS), or pulmonary edema, can result in CAFO workers from acute or chronic exposure to hydrogen sulfide (H_2S). There have been at least 19 acute deaths in workers resulting from sudden H_2S exposure of above 500 ppm secondary to liquid manure agitation. These people may collapse and stop breathing following only a few breaths at this high exposure (hydrogen sulfide is an asphyxiant). Severe pulmonary edema from the irritant properties of hydrogen sulfide and death may result. Longer-term lower exposure may also lead to ARDS during or following an accumulative or multiple period exposure (Donham et al, 1982a).

Other respiratory illnesses result from less acutely toxic exposures and lead to non-fatal acute lung insults as well as chronic declines in lung function (Bongers et al, 1987; Choudat et al, 1994; Cormier et al, 1997; Crook et al, 1991; Donham et al, 1984). Respiratory problems associated with this

environment are listed in Table 3 by upper respiratory tract, airway, interstitial, and mixed airway and interstitial lung diseases. The pathogenesis of these respiratory diseases is primarily acute and chronic airway inflammation. Classical immunologically mediated asthma and hypersensitivity pneumonitis appear to be uncommon among CAFOs workers (Matson et al, 1983).

Acute bronchitis is the most common complaint among CAFOs workers, affecting as many as 70 percent of those exposed. This is an irritant-induced inflammatory condition of the airways. The symptoms of bronchitis are cough and sputum production. Chronic bronchitis is noted by chronic phlegm for two or more years. This condition affects about 25 percent of CAFO workers. Acute and chronic bronchitis may be accompanied by an asthma-like condition, with symptoms of chest tightness, wheezing, difficulty breathing, and shortness of breath (the symptoms most typically reported).

Frequent upper respiratory tract conditions include sinusitis and rhinitis. Some studies have referred to these collectively as mucus membrane irritation (MMI) (Rylander, 1994; Rylander et al, 1989). MMI may be attributable to the combination of bioaerosol, endotoxin and ammonia and other irritant exposures (Donham, 1986; Donham et al, 1986a).

Sinusitis is often chronic among CAFO workers who may complain of a continual or frequent cold "they just cannot shake," of a stuffy head, difficulty in breathing through the nose, headache, and/or "popping ears." These symptoms are a result of a noninfectious, toxic inflammation and swelling of the mucus membranes of the sinus cavities and the Eustachian tubes leading to the middle ear. This is often accompanied by a chronic irritant rhinitis and pharyngitis.

Allergic rhinitis (also called hay fever) has rarely been attributed to confinement exposures. Such persons may have a specific allergy to some component of the swine environment. These symptoms are similar to irritant rhinitis, except it usually develops after only brief exposure to the environment and may be accompanied by itchy, watery eyes and possibly acute chest tightness (allergic asthma). Workers with pre-existing allergic rhinitis often self-select themselves out of CAFO work which contributes to a selected, or survivor, population of CAFO workers.

An asthma-like syndrome, similar to byssinosis (a condition of workers exposed to cotton and other vegetable textile dusts), has been described among CAFO workers. This condition is characterized by chest tightness, wheezing, and/or cough on return to work after two or more days of work absence, and mild acquired airway hyperresponsiveness. It may occur early in exposure to the CAFO environment and is not an immunologically mediated condition. It was documented in 11 percent of a population-based study of Iowa swine confinement workers (Donham et al, 1990).

Occupational asthma includes periodic airway obstruction, chest tightness, wheezing, and dyspnea, does not occur on first exposure, but may develop after weeks to months of CAFO exposure. CAFO workers with pre-existent asthma typically experience severe asthma upon first exposure to animal confinement facilities and select themselves out of these jobs. Occupational asthma may result from repeated exposure to the work environment. It has two basic mechanisms: 1) immunologically mediated or allergic, or 2) chronic irritation. Rarely have there been documented allergic (IgE) mediated causes for CAFO workers' illnesses. These "susceptible" workers almost always leave the work force early because of severe asthma, and the condition is very difficult to manage among workers who continue to work in the CAFO environment. Non-allergic occupational asthma, asthma-like syndrome, and/or reactive airways disease, has been found to be

common (up to 20 percent) of current CAFO workers. This condition may lead to progressive declines in lung function and chronic obstructive pulmonary disease, which is a chronic irreversible condition (Schwartz et al, 1992; Schwartz et al, 1995b). CAFO exposures, dust concentration, endotoxin concentration, and cross-shift decline in lung function (FEV1) have been found to be significant determinants of progressive decline in lung function over time (Reynolds et al, 1996; Schwartz et al, 1995a; Schwartz et al, 1995b; Vogelzang et al, 1998; Vogelzang et al, 2000).

Occupational asthma is distinct from organic dust toxic syndrome (ODTS). ODTS results in a flu-like spectrum of symptoms including headache, joint and muscle pain, fever, fatigue and weakness, cough, shortness of breath, and irritation of the airways and the cells lining the small sacs of the lung. ODTS may be clinically mistaken for farmer's lung, as they have similar acute symptoms, e.g., the delayed onset of severe influenza-like symptoms, following exposure. However, farmer's lung (hypersensitivity pneumonitis) is seen (now rarely) in mainly dairy farming operations, but has not been documented in swine workers (Rylander, 1994). However, 33 percent (Donham et al, 1990) of swine producers have reported episodes of ODTS, which is an influenza-like illness followed by exposure to a higher than usual dust load, e.g., moving and sorting hogs. A chronic or sub acute condition (a possible variant of ODTS) has been described among swine workers and is characterized by chronic fatigue and possibly persistent mild pulmonary infiltrates (Auger, 1992). However, there are only anecdotal cases observed and no human studies that have been conducted (Donham, 1993); there is some evidence for a persistent pulmonary infiltrate condition from one animal study (Donham and Leininger, 1984).

It is recognized that several of these respiratory conditions may occur in an individual CAFO worker, and they may occur at the same time. It is possible, for instance, for an individual CAFO worker to have signs and symptoms of an asthma-like condition, bronchitis, and episodes of ODTS. This produces an interrelated group of conditions (a syndrome) of illness caused by exposure to the swine building environment (Table 1).

6.3.2.2. Control of the Occupational Environment

CAFO worker health risks can be significantly reduced through a comprehensive program of environmental monitoring and control through the use of management practices, engineering controls, judicial use of personal protective equipment, and health surveillance. However, such programs are exceedingly rare in today's CAFO industry. There is little to no exposure monitoring except for research purposes, and routine health surveillance in this worker population is rare. Engineering controls are generally implemented if they will benefit hog production, but rarely with worker health as the principal motivation. There is some evidence to suggest that healthy swine confinement workers can usually tolerate exposures to total dust (2.5 mg/m^3), respirable dust (0.23 mg/m^3), ammonia (7 ppm), endotoxin (100 EU/m^3), and micro-organisms/m^3 (10^5) without experiencing significant acute respiratory symptoms (Donham et al, 1986a; Donham et al, 1986b; Donham et al, 1990; Reynolds et al, 1996). However, further studies are needed to confirm these findings and to assess the combined effects of common CAFO exposures, including ammonia, endotoxin, and the use of disinfectants, which together appear to influence respiratory disease outcomes (Preller et al, 1995).

It is important to recognize that CAFO workers are a survivor population, meaning that the most severely affected workers have already left the workplace. In addition, there is evidence that workers exposed to inhaled endotoxin develop a tolerance (at least to acute symptoms) to this toxicant.

However, long-term exposure may lead to chronic airway obstruction, even in the absence of acute symptoms. Some previously unexposed individuals in the general community population would be expected to react acutely to lower concentrations of CAFO exposures.

Management practices and engineering controls can significantly reduce exposures to inhaled toxicants (Senthilselvan et al, 1997). These include frequent facility cleaning (frequent power washing from floor to ceiling, at least every three weeks); addition of extra fat and a urease inhibitor, e.g., microaid, to the feed; self-cleaning flooring; and improved lagoon operation (Mutel et al, 1992). The ventilation system, by itself, cannot necessarily assure a healthful environment. Health surveillance and the management procedures, mentioned above, must also be implemented. Also, the ventilation system must be properly engineered and maintained; very often, higher cool weather exchange ventilation rates are needed; and lower animal density (swine mass per unit of barn volume) may be required.

Personal protective equipment should not be considered an effective alternative to good management practices and engineering controls. Without a properly supervised respirator program, it is very difficult to assure that exposed personnel will wear the right respirator and that it fits properly, functions properly, and is worn at the appropriate time. Respirators are not well tolerated, especially for strenuous work in a hot, humid environment. The Occupational Safety and Health Administration (OSHA) requires that if respirators are worn to protect workers, they must be worn at all times, and be fit, maintained, and stored properly through an appropriately supervised respirator program. Respirators are an adjunct to management practices, engineering controls, and health surveillance, especially for specific tasks that result in higher-than-normal exposures or for workers in need of increased protection.

Special attention should be given to pregnant women who work in swine confinement facilities. The unborn fetus is susceptible to carbon monoxide and hormonal drugs used in swine production (e.g., oxytocin and prostaglandins). Pregnant women may be at increased risk for spontaneous abortion if they work in swine barns (Donham and Gustafson, 1982).

6.3.2.3. Relationships Between Indoor and External Air Environments

One cannot directly extrapolate occupational health risks observed inside CAFOs to community health risks outside swine production. Although there is discharge of airborne particulates and gases/vapors from the swine barns to the exterior environment, the aerosols differ considerably in composition and in the concentration of specific agents. As aerosols and gases/vapors emanate from a point source travel downwind, the aerosols disperse, become less concentrated and adsorbed gases/vapors may be stripped from particles. There may also be photochemical reactions and ground deposition. Volatile organics present in the outdoor air in the vicinity of a swine production facility may arise from outdoor manure storage facilities and manure application, in addition to particulate and gases in air discharged from the confinement facilities.

Although there is theoretically a definable dose-response relationship for respiratory diseases by individual compounds, the exposures inside CAFOs are always a complex, mixed exposures and differ in many ways from those outside. Perhaps equally important is the fact that the CAFO and community populations are quite different in terms of susceptibility factors. Some members of the general population, including susceptible children, the elderly, asthmatics, and other susceptible individuals, would be expected to develop responses at much lower doses than healthy workers.

Furthermore, individuals living in the vicinity of CAFOs and who may have their quality of life and social and economic conditions affected and feel stress because they have no control over their living conditions.

6.3.2.4. Conclusion

The scientific literature is quite clear that workers in swine or poultry CAFOs are at risk to acute and chronic respiratory diseases from concentrated emissions inside CAFOs. There is, however, adequate information to protect workers, if the industry and regulators take steps to do so--including monitoring engineering, administrative, and personal protective equipment. The swine and poultry industry needs to develop and manage exposures to their workers, and OSHA should take action to protect the health and safety of workers under their jurisdiction.

6.3.3. Community-Based Studies

Community exposures to environmental contamination, most of which has arisen from industrial and agricultural technology over the last 100 years, are now well-recognized public health problems. Exposures include a vast array of chemicals, noise, and ionizing radiation. Other sources of environmental contamination have arisen from the products of armed conflicts, including the some 250,000 American veterans and their families who were exposed to ionizing radiation during the above ground atomic bomb testing program from 1945 to 1962 (Ellis et al, 1992), those exposed to a variety of environmental agents, in addition to a hostile environment, in the Persian Gulf War (Schwartz et al, 1997) and community residents living in proximity to industrial sites in California (Shusterman et al, 1991). These examples, like community exposures to CAFOs, involve environmental exposures under circumstances in which there is little or no environmental control by the affected community.

Ellis has defined community environmental contamination "as a stress that is unique in terms of: 1) its physical characteristics and resultant adaptational dilemmas, 2) the agent or cause of the injury, and 3) the institutional responses to the contamination" (Ellis et al, 1992). Asked by the Centers of Disease Control to assess any adverse health effects of Iowans who served in the Persian Gulf War, The University of Iowa Persian Gulf War Study Group assessed a number of specific and non-specific health outcomes and a number of environmental exposures as well as global exposure to the Persian Gulf War theater among a sample (n=3695) of active and reserve military personnel who served in the war theater and elsewhere during the study period (Schwartz et al, 1997). Significantly higher prevalence of symptoms of depression, posttraumatic stress disorder, chronic fatigue, cognitive dysfunction, bronchitis, asthma, fibromyalgia, alcohol abuse, anxiety, and sexual discomfort were observed. Assessment of health-related quality of life demonstrated diminished mental and physical differences among the PGW as compared with non-PGW military personnel. While significant associations were observed with a number of self-reported environmental exposures during this time period, the exposures and constellation of symptoms did not fit well into an established category of disease or syndrome, but were similar to previous reports of veterans from previous wars thought to arise from the stresses of war. The specific environmental causes of the increased adverse health effects could not ascertained (nor could they be ruled out) from this study and recall bias, to which any such survey is subject, could also not be ruled out as a contributing factor to these associations. Shusterman and colleagues (1991) studied both "environmental worry" and self-perceived environmental odors (especially petrochemical) among 2000 Californians living in proximity to three industrial sites, as well as control sites. Observations found that both "environmental worry" and perceptions regarding odor were associated both

independently and interactively with symptom reporting. Recall bias was recognized as a potential confounder for some of these findings. These methodological approaches are relevant to studies of other community environmental exposures, such as those that arise from CAFOs that include both specific environmental agent exposures and more global (odors/mixed exposures) community exposures, arising from a given source(s) of environmental exposures.

6.3.3.1. Community Studies of Concentrated Livestock Exposures

Schiffman and colleagues (1995) studied North Carolina residents who lived in the vicinity of intensive swine operations (n=44), and compared with matched control subjects who did not live near such operations (n=44). Using a validated Profile of Mood States (Schiffman et al, 1995) they found more negative mood states among those living in proximity to swine operations. The factors affected included tension, depression, anger, reduced vigor, fatigue, and confusion. Greater total mood disturbance was also reported by those living near swine operations. These authors suggested that a variety of factors may have affected the mood of those exposed to odors and living in proximity to swine facilities.

Thu and colleagues (1997) found no difference in the clinical levels of depression or anxiety between Iowans (n=18) living within two miles of a 4,000 sow CAFO and a random sample of demographically similar rural residents (n=18) living near minimal livestock production. However, higher rates of four clusters of symptoms common among CAFO workers and associated with toxic air exposures were observed: (Cluster 1: sputum, cough, shortness of breath, chest tightness, wheezing, p=.02; Cluster 2: nausea, dizziness, weakness, fainting, p=.04; Cluster 3: headaches, plugged ears, p=.06; Cluster 4: runny nose, scratchy throat, burning eyes, p=.12), whereas other symptoms including muscle aches, hearing problems, skin rash, and fever did not differ between the two groups. The authors drew attention to the similarities between the pattern of symptoms among these community residents and CAFO workers and suggested that a larger population-based study was needed.

Wing and Wolf (2000) conducted a population-based study of three rural North Carolina communities, one of which was in the vicinity of a 6,000-head hog operation, one in the vicinity of two intensive cattle operations, and a third area without "liquid waste" livestock operations. A standardized questionnaire was administered by trained interviewers to ascertain health symptoms and indicators of quality of life during the previous 6 months. 155 interviews were completed with a participation rate of 86%. Those living in proximity to the swine operation reported increased rates of headaches, runny nose, sore throat, excessive coughing, diarrhea, and burning eyes compared to rural residents with no livestock operation. Quality of life measures among those living in the vicinity of the swine operation were greatly reduced. The authors were aware of potential recall bias and, therefore, presented the study as a "rural health" study which did not include any questions about hogs, livestock, or odors. They also pointed out that eight symptoms in the miscellaneous category did not differ between the hog and control communities, thereby minimizing the likelihood of significant recall bias.

Hodne and her University of Iowa colleagues are currently testing the relative power of aspects of medical models and bio-psychosocial models to assess the mental health consequences of CAFO community exposures. For example, they report greater traumatic cognitions associated with post-traumatic stress disorder among residents of rural areas with many CAFOs and areas with traditional livestock production than among rural residents in areas with very little livestock (Hodne, 2001).

They are also exploring the types of stress responses in CAFO neighbors that may mediate the relationship between air emissions and odors and physical and mental health outcomes.

The three published, peer reviewed studies of community residents exposed to CAFO emissions are limited and should be interpreted with caution because of the relatively small numbers of participants, because they did not report environmental exposure data and likely contain some recall bias. However, they are notable because they were all well designed, controlled studies and because the two of the three that examined respiratory and other symptoms common among CAFO workers found similar symptom patterns (while not as prevalent or severe) as those observed among CAFO workers. Two of the three studies also reported indicators associated with diminished a quality of life among those living in proximity to livestock facilities as compare to community controls.

6.3.4. Conclusion

Numerous occupational studies have documented significant increases in respiratory disease and other respiratory adverse health effects, including CAFO-related deaths, acute and chronic respiratory diseases and associated symptoms and acute losses in exposure-related lung function and progressive respiratory impairment, among those who work in CAFOs. However, it is recognized that the CAFO workforce is generally healthy, while those in the general community, including children, the elderly, those with chronic impairments such as pre-existing asthma or chronic obstructive pulmonary disease, are expected to be much more susceptible to CAFO exposures. There is experimental and epidemiological evidence that very low levels of exposures to ammonia, hydrogen sulfide, known to be ambient air toxic gases arising from CAFOs, may result in adverse health effects among healthy volunteers and community residents. While limited in number and scope, the currently published, peer reviewed, community-based studies of adverse health affects associated with CAFO exposures find an increased prevalence of similar symptom patterns, especially respiratory symptoms, and similar indicators of reduced quality of life. Taken together with other experimental and epidemiological observations of adverse health effects observed with low levels of exposures to chemical components (ammonia, hydrogen sulfide) of CAFO emissions, these findings support a conclusion that CAFO air emissions constitute a public health hazard, deserving of public health precautions as well as larger, well controlled, population-based studies to more fully ascertain adverse health outcomes and their impact on community health services.

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TABLE 1
Volatile Compounds Associated with Pig Wastes

Methanol	Methanal	
Ethanol	Ethanal	Ammonia
1-Propanol	Propanal	Methylamine
2-Propanol	Butanal	Ethylamine
1-Butanol	Pentanal	Trimethylamine
2-Butanol	Hexanal	Triethylamine
2-Methyl-1-propanol	Heptanal	Carbonsulphide
3-Methyl-1-butanol	Octanal	Hydrogen sulphide
2-Ethoxy-1-propanol	Decanal	Methanethiol
2-Methyl-2-pentanol	2-Methyl-1-propanal	Dimethylsulphide
2,3-Butanediol	Ethylacetate	Dimethyldisulphide
	Methanaoic acid	Dimethyltrisulphide
	Ethanaoic acid	Diethyldisulphide
3-Hydroxy-2-butanone	Propanoic acid	Propanethiol
Propanone	Butanoic acid	Butanethiol
2-Butanone	2-Methylpropanoic acid	Dipropldisulphide
3-Pentanone	Pentanoic acid	2-Methylthiophene
Cyclopentane	3-Methylbutanoic acid	Propylprop-1-enyldisulphide
1-Octanone	Hexanoic acid	2,4-Dimethylthiophene
2,3-Butanidione	4-Methylpentanoic acid	2-Methylfuran
	Heptanoic acid	
	Octanoic acid	
Phenol	Nonanoic acid	
4-Methylphenol	Phenylacetic acid	
4-Ethylphenol	2-Phenylpropanoic acid	
Toluene		
Xylene		
Indone		
Benzaldehyde		
Benzanoic acid		
Methylphthalene		
Indole		
Skatole		
Acetphenone		
o-Aminoacetophenone		
Aneline		

Source: 1995. Proceedings, "Understanding the Impacts of Large-Scale Swine Production," June 29-30, Des Moines, IA. The University of Iowa Printing Service, Iowa City, IA, pg 51.

TABLE 2
Major Hazard Categories in Swine Production.

Hazards	Subcategories	Examples
Chemical Hazards	Asphyxiation	Carbon monoxide
	lung injury	Nitrogen oxides, ammonia
	contact dermatitis	Allergic, irritant
	Poisonings	Pesticides, fuels, cleaning agents
	Intoxication	Solvents, silo gas, substance abuse
	Immunomodulation	Adjuvants: biocides, phytotoxins
		Immunosuppressants: pesticides
Biological Hazards	Microorganisms	Pathogenic
		Non-pathogenic
	organic dust	Bacterial toxins: endotoxins, exotoxins, enterotoxins
		Fungal toxins: mycotoxins, glucans
		Phytotoxins
		Inflammatory agents
	Aeroallergens	Arachnid detritus
		Animal proteins
		Allergenic fungi
Infectious Hazards	Zoonotic	Systemic
	non-zoonotic	Lung
	antibiotic resistance	Skin
	emerging pathogens	Ocular conjunctivitis
Biomechanical Stress	Trauma	Animal bites
		Falls
		Needle sticks
		Punctures, lacerations, abrasions, burns
		Crushing injuries
		Repetitive trauma
	Noise	Noise-induced hearing loss
		Reduced safety from impaired hearing
Thermal Stress	heat stress	
	cold stress	
Emotional Stress	Occupational	Suicide
	Marital	Depression
	Financial	Anxiety
Drowning		Lagoons
		Pits
		Farm ponds
Fires/explosions	Chemical	Methane in pits
	Electrical	Ignited building materials or feed
	Welding	Ignited building materials or feed
	organic material	Grain, grain dust, compost, hay
Electrocution		Faulty wiring
		Water associated
Chronic pain	Biomechanical stress	Arthralgia
	Arthritis	Myalgia
Fatigue	sleep deprivation	Planting, harvesting
	chronic fatigue syndrome	Chronic endotoxin exposure

Source: 1995. Proceedings, "Understanding the Impacts of Large-Scale Swine Production," June 29-30, Des Moines, IA. The University of Iowa Printing Service, Iowa City, IA, pg 156.

TABLE 3: Respiratory Diseases Associated with Swine Production

Upper Airway Disease
Sinusitis
Irritant Rhinitis
Allergic Rhinitis
Pharyngitis
Lower Airway Disease
Organic Dust Toxic Syndrome (ODTS)
Occupational Asthma
Nonallergic asthma, hyperresponsive airways disease, or reactive airways disease syndrome (RADS)
Allergic asthma (IgE mediated)
Acute or Subacute Bronchitis
Chronic Bronchitis
Chronic Obstructive Pulmonary Disease (COPD)
Interstitial Disease
Alveolitis
Chronic Interstitial Infiltrate
Pulmonary Edema

Source: 1995. Proceedings, "Understanding the Impacts of Large-Scale Swine Production," June 29-30, Des Moines, IA. The University of Iowa Printing Service, Iowa City, IA, pg 158