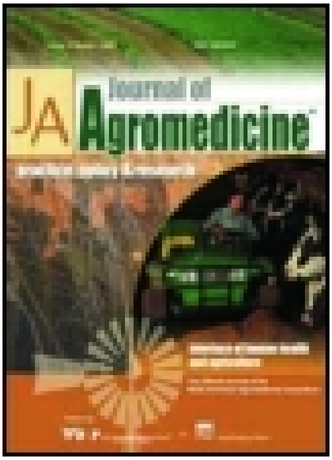

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Potential Health Effects of Odor from Animal Operations, Wastewater Treatment, and Recycling of Byproducts

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ABSTRACT. Complaints of health symptoms from ambient odors have become more frequent in communities with confined animal facilities, wastewater treatment plants, and biosolids recycling operations.

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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 alterations in mood. Typically, these symptoms occur at the time of exposure and remit after a short period of time. However, for sensitive individuals such as asthmatic patients, exposure to odors may induce health symptoms that persist for longer periods of time as well as aggravate existing medical conditions. A workshop was held at Duke University on April 16-17, 1998 cosponsored by Duke University, the Environmental Protection Agency (EPA), and National Institute on Deafness and Other Communication Disorders (NIDCD) to assess the current state of knowledge regarding the health effects of ambient odors. This report summarizes the conclusions from the Workshop regarding the potential mechanisms responsible for health symptoms from ambient odors. Methods for validation of health symptoms, presence of odor, and efficacy of odor management techniques are described as well. *[Article copies available for a fee from The Haworth Document Delivery Service: 1-800-342-9678. E-mail address: <getinfo@haworthpressinc.com> Website: <http://www.HaworthPress.com>]*

KEYWORDS. Health effects, odor, nasal irritation, irritant, confined animal feeding operations (CAFOs), dust, particulates, wastewater treatment, biosolids, composting

EXECUTIVE SUMMARY

A workshop was held at Duke University on April 16-17, 1998 cosponsored by Duke University, the Environmental Protection Agency (EPA), and National Institute on Deafness and Other Communication Disorders (NIDCD) to determine the current state of knowledge regarding the health effects of ambient odors. Special emphasis was placed on potential health issues related to odorous emissions from animal manures and other biosolids. Odors are sensations that occur when a complex mixture of compounds (called odorants) stimulate receptors in the nasal cavity. Most odorants associated with animal manures and biosolids are volatile organic compounds (VOCs) that are generated by bacterial degradation of protein, fat, and carbohydrates in the organic matter. Reactive inorganic gases such as ammonia and hydrogen sulfide are also important odorants that can be emitted from animal manures and biosolids.

People are exposed to odor every day. Most of these odors produce no complaints and may be pleasant. When odors from manures and biosolids rise to the level that complaints are produced, many of these complaints are focused on the unpleasant nature of the odor rather than on health symptoms. However, health symptoms have been reported with increasing frequency to low levels of odor 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 alterations in mood. Typically, these symptoms occur at the time of exposure and remit after a short period of time. However, for sensitive individuals such as asthmatic patients, exposure to odors may induce health symptoms that persist for longer periods of time as well as aggravate existing medical conditions. It is not known at present if there is a cumulative impact of exposure to irritants/odors from agricultural operations and municipal wastewater treatment facilities on neighbors over time as has been documented for workers continuously exposed to odorous air in swine facilities.

Workshop participants discussed three paradigms by which ambient odors may produce health symptoms in communities with odorous manures and biosolids. In the first paradigm, the symptoms are induced by exposure to odorants at levels that also cause irritation (or other toxicological effects). That is, irritation–rather than the odor–is the cause of the symptoms, and odor simply serves as an exposure marker. In this paradigm irritancy (or other toxicity) generally occurs at a concentration somewhat higher (about 3 to 10 times higher) than the concentration at which odor is first detected (odor threshold). While the concentration of each individual compound identified in odorous air from agricultural and municipal wastewater facilities seldom exceeds the concentration that is known to cause irritation, the combined load of the mixture of odorants can exceed the irritation threshold. That is, the irritation induced by the mixture derives from the addition (and sometimes synergism) of individual component VOCs.

In the second paradigm health symptoms occur at odorant concentrations that are not irritating. This typically occurs with exposure to certain odorant classes such as sulfur-containing compounds and organic amines at concentrations that are above odor detection thresholds but far below irritant thresholds. Health symptoms often reported include a stinging sensation, nausea, vomiting, and headaches. The mechanism by which health symptoms are induced by sulfur gases or organic amines for which odorant potency far exceeds the irritant potency is not well understood. Factors such as the degree of unpleasantness of the odor, the exposure history (prior experience with odor), beliefs about the safety of an odor, and emotional status may play a role in inducing health symptoms. Noxious odors that are neither irritating nor toxic can set up a cascade of events such as physiological stress or nutritional problems (caused by altered food intake) that lead to health effects. The genetic basis of aversions to malodors is not well understood, but brain imaging studies suggest that noxious odors stimulate different brain areas than those that process pleasant odors.

In the third paradigm, the odorant is part of a mixture that contains a co-pollutant that is essentially responsible for the reported health symptom. Odorous airborne emissions from confined animal housing, composting facilities, and land application of sludge can contain other components that may be the cause of the symptoms such as bioaerosols consisting of endotoxin, dust from food, airborne manure particulates, glucans, allergens, microorganisms, or toxins. Thus, an individual may encounter odors from swine facilities while simultaneously exposed to dust or gram-negative endotoxin. In this case, the symptoms or health effects are more likely to result from the irritant effects of the dust or from the inflammatory responses to endotoxin exposure rather than from the odor. That is, odor again acts as an exposure marker as in Paradigm 1. Somatic symptoms (i.e., those affecting the body) including altered respiratory behavior to the odor alone can be acquired via Pavlovian conditioning due to association of odorous emissions with a physiological challenge (e.g., dust, endotoxin). Subsequently the odor in the absence of the co-pollutant will produce the symptoms. These odor associations are readily established and robust; while they can be extinguished (unlearned), this process occurs slowly.

There is wide variability among individuals in the odorant concentrations that cause health complaints. To address this issue, levels of odor exposure were defined to clarify the intensities associated with potential health impacts described above in the three paradigms. This set of odor levels in increasing intensity includes the following:

Level	Description
1) odor detection	The level of odor that can first be differentiated from ambient air.
2) odor recognition	The level of odor at which the odor quality can be characterized, e.g., the level at which a person can detect that an odor is apple or manure.
3) odor annoyance	The level at which a person is annoyed by an odor but does not show or perceive a physical reaction. Note: Health symptoms are not expected at these first three levels unless the odor occurs with a co-pollutant such as dust as in Paradigm 3 or the level of annoyance is intense or prolonged.
4) odor intolerance (causing somatic symptoms)	The level at which an individual may show or perceive physical (somatic) symptoms to an odor. Note: This level corresponds to Paradigm 2 in which the odor induces symptoms even though the odorant concentration is lower than that known to cause irritation.
5) perceived irritant	The level at which a person reports irritation or physical symptoms as a result of stimulation of nerve endings in the respiratory tract.
6) somatic irritant	The level at which an odorant (not an odor) results in a negative physical reaction regardless of an individual's predisposition. This can occur when an odorous compound (e.g., chlorine) damages tissue. Note: Perceived and somatic irritation correspond to Paradigm 1.
7) chronic toxicity	The level at which an odorant can result in a long-term health impact.
8) acute toxicity	The level at which an immediate toxic impact is experienced, e.g., a single event may evoke an acute health impact. Note: In the case of chronic or acute toxicity, the compound should not be considered an odorant but rather a compound with toxic effects that happens to have an odor.

The range of odor intensities and odorant concentrations that correspond to these 8 levels varies across individuals.

A majority of the studies reviewed in this report are taken from laboratory experiments where greater control is possible and mostly not from confined animal feeding operations, municipal wastewater or biosolids treatment, or the recycling of these byproducts. By the review of these studies, examples are given that can help elucidate the types of health symptoms that may occur from exposure to odorous volatile compounds and associated particulates from animal feeding and the processing and recycling of animal manures and biosolids. In addition, this review helps establish a basis for future management and research regarding the potential impacts of odor on human health from such operations.

The odor exposures that have received the greatest research attention are those that involve irritation. Physiological responses to irritation in the upper respiratory tract (nose, larynx) and/or lower respiratory tract (trachea, bronchi, deep lung sites) have been documented in both humans and animals. Irritation of the respiratory tract can alter respiratory rate, reduce respiratory volume (the amount of air inhaled), increase duration of expiration, alter spontaneous body movements, contract the larynx and bronchi, increase epinephrine secretion, increase nasal secretions, increase nasal airflow resistance, slow the heart rate, constrict peripheral blood vessels, increase blood pressure, decrease blood flow to the lungs, and cause sneezing, tearing, and hoarseness. Release of the potent hormone epinephrine (also called adrenalin) subsequent to nasal irritation may be a source of feelings of anger and tension that have been reported by persons exposed to odors. Epidemiological studies in communities with animal operations and municipal wastewater facilities have reported increased occurrence of self-reported health symptoms consistent with exposure to irritants.

The odorous emissions that reach neighbors of animal and municipal wastewater facilities and recycling operations are a function of the concentration of volatiles produced at the source as well as their emission rates, dispersion, deposition, and degradation in the downwind plume. Furthermore, numerous sources at a facility can contribute to the total odor and irritation intensity experienced by neighbors. In the case of confined swine operations, for example, odor sources including animal housing, lagoons (or other storage units), and land application of manure all contribute to the sensory impact. VOCs emitted from swine houses probably contribute substantially to irritation downwind if not strongly diluted after emission. Particulates (e.g., dust) may also contribute to the irritation.

Workshop participants concluded that current evidence suggests that the symptom complaints experienced by neighbors of some odorous animal operations and municipal wastewater facilities may constitute health effects. In addition, odorous compounds from these operations together with odor sources other than animal and wastewater byproducts all contribute to odor complaints and air quality in an air shed. However, further research studies in both laboratory and field settings are necessary to quantify the concentration/intensity ranges that cause health complaints in the general population as well as in sensitive (e.g., allergic) individuals. These studies should utilize objective biomarkers of health symptoms to validate health complaints. The workshop participants developed a battery of objective tests to be used for these studies. The research should also be performed in a manner that removes confounding and bias (i.e., belief that odor presents a risk or is toxic). Overall, workshop participants agreed that if health complaints can be documented by objective measures of physical symptoms, then such health symptoms should be considered health effects. The importance attached to such health effects, however, is dependent upon a number of value-laden variables, including exposure and/or symptom prevalence, severity, and perceived degree of impairment in the lives of affected individuals.

Workshop participants also concluded that health impacts from odorous facilities can be minimized using a variety of methods for odor remediation. For agricultural facilities, as an example, feed additives (compounds incorporated into the animal's diet), digestive deodorants (bacteria or enzymes that reduce undesirable odors through biochemical metabolic degradative processes), adsorbents (products with large surface area that adsorb targeted odorants before they are released), and chemical deodorants (strong oxidizing agents or germicides) have been used although the cost effectiveness of these approaches has not yet been proven. The efficacy of these odor remediation techniques in reducing odor itself can be quantified by olfactometry (a measurement technique that uses the human nose as the sensor), gas chromatography (an analytical method that separates the gaseous mixture of chemical compounds into its molecular constituents), and/or an electronic nose (a sensor array that mimics the performance of the human nose).

This report summarizes of current state of knowledge regarding the health effects of ambient odors with special emphasis on odorous emissions from animal manures and other biosolids. The potential mechanisms that are responsible for health symptoms are discussed.

Methods for validation of health symptoms, presence of odor, and efficacy of odor management techniques are also described.

INTRODUCTION

Complaints of adverse health symptoms are reported with increasing frequency in communities near odorous agricultural, industrial, and municipal facilities. Intensive livestock rearing, solid waste processing, composting, storage, disposal, and/or land application sites from agricultural, industrial, and municipal facilities have all been involved in complaint processes. In agriculture, there is a national trend toward increased numbers of animals per livestock facility. In North Carolina, for example, the number of hogs has nearly quadrupled since 1990 from 2.5 million to 9.6 million while the number of hog farms has decreased by almost one half (from 10,000 to 5,800).[1] In many areas, people with residences neighboring intensive livestock operations, especially swine facilities, complain because their once clean air has offensive odors; in addition they say it affects their health.[2] The most common health complaints associated with environmental odors from agricultural sources and biosolids include eye, nose, and throat irritation, headache, nausea, hoarseness, cough, nasal congestion, palpitations, shortness of breath, "stress," drowsiness, and alterations in mood.[3-6] These symptoms attributed to odors are generally acute in onset (occur at the time of exposure) and self-limited in duration (remit after a short period of time). Persons with allergies and asthma often assert that odors exacerbate their symptoms.[7] Persons who report adverse health symptoms from odors usually indicate that they have problems with numerous types of odorous compounds.[6]

Because of the increased number of questions about possible health symptoms from odors, a workshop to address the issue was held at Duke University on April 16-17, 1998 cosponsored by Duke University, the Environmental Protection Agency (EPA), and National Institute on Deafness and Other Communication Disorders (NIDCD). The purpose of this workshop was to determine the current state of knowledge regarding the effects of ambient odors on health and well-being. Special emphasis was placed on potential health issues related to odorous emissions from animal manures and biosolids. The list of Workshop participants is given in Acknowledgements. This paper summarizes the issues raised during the workshop including potential mechanisms by which odorous emissions may give rise to health effects. Methods for evaluation, documentation, and remediation of odors are described as well.

Physiology of Odor Perception

Health symptoms from odors can potentially result from two sources: the odor (the sensation) or the odorant (the chemical or mixture of chemicals that happens to have an odor). Odor sensations are induced when odorants interact with receptors in the olfactory epithelium in the top of the nasal cavity. Signals from activated receptors are transmitted via the olfactory nerve (first cranial nerve) to the olfactory bulb and ultimately to the brain. Odor sensations are described by adjectives such as floral, fruity, earthy, fishy, fecal, and urinous.[8] Odorant compounds are diverse in molecular structure but most are non-ionic compounds with molecular weights of less than 300. In general odorants are hydrophobic organic compounds that contain a limited number of functional groups although the presence of a functional group is not a prerequisite for odor. Some reactive inorganic gases such as ammonia and H_2S can also be odorants.

Odorants can also stimulate free nerve endings of four other cranial nerves (trigeminal, vagus, chorda tympani, and glossopharyngeal nerves) to induce sensations of irritation. Sensory neurons of the trigeminal nerve innervate the eyes, nose, anterior 2/3 of the tongue, gums, and cheeks.[9,10] The trigeminal nerve responds to five different classes of stimuli: (1) chemical, (2) mechanical (such as dust particles that touch the mucous linings of the nose, eye, or mouth), (3) thermal (temperature), (4) nociceptive (pain), and (5) proprioceptive (movement/position).[11] Trigeminal stimulation by odorous chemicals and dust induces sensations such as irritation, tickling, burning, stinging, scratching, prickling, and itching.[12-14] Examples of odorous compounds found in the home or office which are also irritants include chlorine, gasoline, camphor, menthol, alcohol, vinegar, and various solvents.[15-17] Diesel exhaust is an example of a mixture of odorous compounds found outdoors that is an irritant.[18]

Free nerve endings of the vagus nerve transmit information on irritation in the throat, trachea, and lungs. Free nerve endings of the chorda tympani nerve (along with the trigeminal nerve) mediate irritation on the anterior tongue during mouth breathing; free nerve endings of the glossopharyngeal nerve transmit information about irritation on the posterior tongue. Overall, the same compound can generate sensations of both odor and irritation, but the concentration necessary to elicit irritation is generally higher than that needed for odor. Almost any airborne chemical can, in sufficient concentration, stimulate chemosensory trigeminal receptors in the nose and eyes, damage tissue, or cause toxic effects.[19]

Paradigms by Which Odors Can Affect Health Symptoms

There are at least three paradigms that may explain how odors or odorants could potentially affect human health.[20] In Paradigm 1, the symptoms are induced by exposure to an odorant at levels that also cause irritation (or other toxicological effects). In this case, irritation–rather than the odor–is the cause of the symptoms, and odor simply serves as an exposure marker. For odorants acting under Paradigm 1, the irritancy (or other toxicity) generally occurs at a concentration above–but within an order of magnitude–of the odor threshold. That is, the detection threshold for irritancy (concentration at which irritancy is first detected) is between 3-10 times higher than the concentration at which odor is first detected. (The odor detection threshold is the concentration at which odor is first detected.) Examples include ammonia, chlorine, and formaldehyde (e.g., from building products) as well as acrolein, acetaldehyde, and organic acids (e.g., from cigarettes). At concentrations above the irritant threshold, both odor and irritant sensations can coexist. The sensation of odor is merely coincident with the more relevant irritative process; symptoms are more likely caused by irritation rather than "odor-induced." In this paradigm, odor is a warning of potential health symptoms at elevated concentrations.

In Paradigm 2, by contrast, exposure to odorous compounds at concentrations above the odor threshold but below irritant levels is associated with health symptoms. This typically occurs with exposure to certain odorant classes such as sulfur-containing compounds and organic amines with odor thresholds that are 3-4 orders of magnitude (that is 10^3 and 10^4 times) below the levels that cause classical toxicological or irritant symptoms. Industrial and biological sulfur gases (e.g., hydrogen sulfide, mercaptans, or thiophenes) have odor thresholds in the ppb (parts per billion) or ppt (parts per trillion) range[21,22] but they do not produce objective mucous membrane irritation until they reach a level of 10-20 ppm (parts per million). Nevertheless, health symptoms are often reported from residents of communities exposed to industrial sulfur gases and other malodorous compounds at levels exceeding the odor threshold but below irritant thresholds.[6,23] The mechanism by which health complaints are induced by compounds whose odorant potency far exceeds the irritant potency is not well understood, but perceptual/psychological as well as genetic/physiological factors may play a role.[6,20]

The third paradigm in which odors may be associated with symptoms is one in which the odorant is part of a mixture that contains a co-pollutant that is actually responsible for the reported health symptom. Odorous airborne emissions from confined animal operations, composting facilities, and sludge can contain other components that may be the cause of the symptoms such as bioaerosols consisting of endotoxin, dust from food, airborne manure particulates, glucans, allergens, microorganisms, or toxins. Thus, an individual may encounter odors from swine facilities while simultaneously exposed to dust or gram-negative endotoxin. In this case, the health symptoms are more likely to result from the irritant effects of the dust or from the inflammatory responses to endotoxin exposure than the odor. That is, odor again acts as an exposure marker (as in Paradigm 1).

It should be noted that odor perception is not always an adequate warning of impending toxicity. This situation arises when a compound is toxic or irritating at concentrations below the odor threshold. One example is arsine gas (not found in animal or biosolids operations) which may cause hemolysis and potential renal failure at a concentration at or below its odor threshold.[20] Other examples are methyl isocyanate (the contaminant released at Bhopal, India) and methyl isothiocyanate (breakdown product of the pesticide Metam sodium) for which the odor thresholds are higher than the irritant threshold.[20,24] A few compounds produce irritation almost in the absence of odor; for example, CO_2 is an irritant that produces minimal, if any, odor response in humans.[25]

EVIDENCE THAT ODORS CAN PRODUCE HEALTH SYMPTOMS

There is experimental evidence to support each of the paradigms given above. This evidence is described below in order to elucidate the mechanisms by which odorous emissions can cause health symptoms.

Evidence for Paradigm 1: Irritation Rather Than the Odor Causes the Health Symptoms

There is extensive evidence that odorous volatile compounds can produce irritation in both the upper respiratory tract (nose, larynx) and lower respiratory tract (trachea, bronchi, deep lung sites). This irritation involves both sensory signals (mediated by the trigeminal and vagus nerves) as well as actual inflammation of tissues. Sensory irritation can arise: (1) from a single odorous compound above its irritant threshold,[6,26] (2) from the aggregate effect of low concentrations of odorous chemicals not normally considered to be irritants,[12] or (3) from weak trigeminal stimulation in combination with much higher levels of olfactory stimulation.[14] The fact that mixtures of low concentrations of odorants can induce sensory irritation is due to the fact that the primary mixture constituents can be additive (or, in some cases, even synergistic) in their ability to produce irritation,[19,27,28] i.e., the irritancy of the mixture may, in some cases, be greater than the sum of the individual components. Even subthreshold levels of individual volatile organic compounds (VOCs) can add together when delivered in a mixture to produce noticeable sensory irritation.

Irritation thresholds for specific single compounds vary widely across volatile chemicals; furthermore, the degree of chronic structural damage associated with exposure to irritants is compound specific. For example, the compound ortho-chlorobenzylidene malononitrile (tear gas) has irritant properties as low as 0.05 ppm (parts per million). Inhalation of this compound causes acute effects including irritation, burning, and pain in the upper and lower airways and eyes. Headache and altered breathing also occur acutely. The severity of the symptoms is dependent on the length of exposure and the concentration.[29] However, in persons without respiratory allergies or asthma, the symptoms usually remit within minutes to hours without showing long-term respiratory effects. Other irritants such as chlorine are more chemically reactive and attack tissue. Chlorine is irritating at or below 0.5 ppm;[30] at higher concentrations, it can cause acute respiratory injury and long-term reactive airway dysfunction.[31] Some individual chemicals can be tolerated at concentrations up to 1,000-2,000 ppm and above without irritation.[28,32] Sensory irritation as well as odor can also be produced by a mixture of individual chemicals at subthreshold concentrations.[19,27] This agonistic effect may explain why odorous emissions from swine operations which contain low levels of hundreds of component compounds lead to self-reports of irritant sensations[3,4] even though the concentrations of individual chemical constituents are below known irritant threshold concentrations.[33] It should be noted here that neither ortho-chlorobenzylidene malononitrile (tear gas) nor chlorine are found in manure or biosolids. However, the mixture of volatile compounds emitted from manures and biosolids do have the potential to cause sensory irritation with or without health complaints.

Physiological Symptoms Caused by Sensory Irritation

Administration of irritant compounds to the upper and/or lower airway in laboratory studies produces many systemic responses including: (1) changes in respiratory rate, depending upon the primary level of irritation (upper versus lower), (2) reduced respiratory volume, (3) increased duration of expiration, (4) alterations in spontaneous body movements, (5) contraction of the larynx and bronchi, (6) increased epinephrine secretion, (7) increased nasal secretion, (8) increased nasal airflow resistance, (9) increased bronchial tone, (10) decreased pulmonary ventilation, (11) bradycardia, (12) peripheral vasoconstriction, (13) increased blood pressure, (14) closure of the glottis, (15) sneezing, (16) closure of the nares, (17) decreased pulmonary blood flow, (18) decreased renal blood flow and clearance, and (19) lacrimation or tearing.[9,17,34-42] Irritants can also induce hoarseness of voice[43] and impair mucociliary clearance functioning.[26] These physiological responses suggest that irritant sensations in the upper respiratory tract are a warning that the respiratory system may be at risk from harmful substances. Reflexive breath stoppage (apnea) subsequent to stimulation of the trigeminal nerve in the upper airway is probably a defensive device to prevent inhaling chemicals in the air that might damage the lungs or respiratory tract. This breath stoppage does not occur in isolation as evidenced by a subsequent cascade of physiological symptoms associated with this response. This nasal reflex induces activity in the sympathetic division of the autonomic nervous system (ANS) leading to increases in circulating epinephrine. This causes acceleration of heart rate and peripheral vasoconstriction (leading to an increase in blood pressure). In addition, activity in the sympathetic division of the ANS is often associated with emotional induction of fear or anger. Sustained exposure to irritating solvents can also impact neurobehavioral functioning.[44] These factors along with the unpleasant sensory properties of irritation make strong trigeminal stimulation a memorable event, and one which is likely to be regarded as highly aversive.

Lower airway irritation usually produces an increase in breathing rate and pulmonary ventilation and little change in heart rate or blood pressure.[34] There are instances, however, in which lower airway irritation can cause decreased respiratory rate (postexpiratory apnea)[45]. Volatile chemical irritants can also cause local redness, edema, pruritis or pain, and eventually altered function.[45] Excessive irritation in the lower airway (as well as upper airway) may lead to tissue damage and, eventually, scarring. Airway irritation is also associated with non-respiratory tract health complaints such as headache and lassitude.[6,12]

Controlled experiments have been performed to assess health impacts of specific airborne irritants. For example, Hudnell et al.[46] at the Environmental Protection Agency evaluated health symptoms in 66 healthy males who were exposed to an odorous mixture of 22 common volatile organic compounds (25 mg/m^3 total concentration which is representative of levels found in new homes and office buildings) for 2.75-hours. Subjects were also exposed to clean air for an equivalent time in a control session. Subjects rated the intensity of perceived odor, irritation, and other variables before and during exposure. During exposure to the VOC mixture, self-reported eye and throat irritation, headache, and drowsiness increased or showed no evidence of adaptation (that is, no reduction in intensity). Thus, irritation did not decrease over the 2.75 hour-long sessions but odor intensity decreased by 30%. Exposure for a longer period (6 hours) in another study did show, however, some significant adaptation in nasal irritation.[47] Hudnell et al.[46] concluded that the health symptoms in the 2.75 hour study were caused by stimulation of free nerve endings in the eyes and respiratory tract by additive or synergistic interactions among subthreshold levels of this particular combination of VOCs. Seventeen of the 22 compounds tested by Hudnell et al.[46] are also found in emissions from swine odor:[33] n-butylacetate, p-xylene, n-butanol, n-decane, ethylbenzene, n-hexanal, n-hexane, 2-butanone, cyclohexane, 3-methyl-2-butanone, 4-methyl-2-pentanone, n-pentanal, isopropanol, n-propylbenzene, trimethylbenzene, n-undecane, and 1-octene. However, it is not yet known if the levels of VOCs experienced by neighbors exposed to emissions from manure and biosolids are comparable to those tested by Hudnell et al.[46]

Two types of nerve fibers in the trigeminal nerve conduct nociceptive (pain) afferent pulses: finely myelinated A-delta fibers and unmyelinated C fibers.[48,49] Dull and burning painful sensations are characteristic of C fibers while sharp, stinging sensations appear after activation of A-delta fibers.[50-52] Activation of trigeminal C fibers by irritants leads to the release of neuropeptides including substance P into the nose. Substance P induces neurogenic inflammation including vasodilation, increased blood flow, increased vascular permeability, increased ocular pressure and pupillary contraction.[40] Substance P release is associated with an increased presence of polymorphonuclear neutrophilic leukocytes (PMNs) in the nasal cavity which indicates the presence of acute inflammation.[53] Exposure to 25 mg/m^3 VOCs for 4 hours led to increased levels of PMNs in nasal lavage fluid.[54] The release of substance P by trigeminal stimuli is also one potential mechanism by which trigeminal irritants may cause head pain.[55] Vasculature in the cranium is supplied by substance P-containing C fibers of the trigeminal nerve. Thus, inhaled irritants in the air may induce headaches and migraines by increasing cortical blood flow via the trigeminovascular system, i.e., via stimulation of a sensory (trigeminal) nerve.

Relationship Between Trigeminal and Olfactory Sensations

There is often a temporal disparity between odor and irritant sensations with odor sensations tending to precede the irritant sensations. This is due in part to the fact that chemical agents must migrate through the mucosa to activate free nerve endings of the trigeminal nerve. This fact coupled with the relatively slow transmission time of the C fibers leads to a slowly responding system in comparison to olfaction. Sensations of odor and irritation also respond differently to continuous chemosensory stimulation. Odor sensations tend to fade quickly (adaptation) upon stimulation while irritancy can grow sharply over a period of time[52,56] though it may ultimately adapt to some degree by six hours of exposure.[47] The growth of irritancy over time may be due in part to the kinetics of overcoming the buffering capacity of nasal mucus or may represent cumulative damage to structural elements. Thus odor is a warning of potential health symptoms from irritation at elevated concentrations. Continuous exposure to compounds such as ammonia or H_2S can lead to odor fatigue and/or tolerance, and this reduced sensitivity may jeopardize health when the warning signal is not adequately perceived.

Sustained occupational exposure to a sensory irritant over months or years can reduce the perceived odor and irritation intensity. For example, acetone-exposed workers required higher concentrations of acetone to detect both its odor and irritating sensory properties.[57] Olfactory perception of other compounds was not necessarily affected.[58,59] There are at least three possible causes of this persistent reduction in perceived irritation and odor intensity with occupational exposure. First, inhaled acetone (and other odorous VOCs) can be absorbed from the lungs into the blood stream to dissolve in fat stores of the body. The VOCs (in this case acetone) are subsequently released slowly from the fat stores back into the blood and lungs to cause continuous adaptation of olfactory receptors as they are exhaled. Odorous VOCs have been found in the blood and brain after three hours of exposure,[60] and olfactory receptors have been shown to respond to blood-borne odorants.[61] Second, there is a possibility that peripheral changes such as down-regulation of receptors could account for the elevated thresholds and reduced responses. A third possibility is that a cognitive factor contributes to a person's perception of odor, and that acetone-exposed workers learn to tune out the smell of acetone at a cognitive level. The reduced sensory perception of acetone by acetone workers reduced the number of health symptoms they reported relative to unexposed controls.[58,59] It is possible that workers who can tolerate the initial exposure to irritants stay on the job while those that cannot, leave for other jobs. (This has been called the "healthy worker phenomenon.")

There is perceptual interaction between the olfactory and trigeminal sensations but the results of studies differ somewhat in their findings. Kendal-Reed et al.[14] found that low to moderate levels of self-reported nasal irritation are attributable not only to trigeminal stimulation but also to relatively weak trigeminal stimulation in combination with much higher levels of olfactory activation. Cain, on the other hand, suggested that strong odors can lead to a perceptual reduction in the irritation produced by the trigeminal stimulus.[62] That is, odors can "mask" trigeminal stimuli and vice versa. While masking does occur, the overall intensity of the experience is rated as more intense as the concentrations of the two stimuli increase. Stimulation of the nose and eye with low levels of odorous VOCs are often either additive or synergistic, leading to responses characteristic of irritants. Walker and colleagues[63] have studied respiratory responses following stimulation of the eye and nose. Using a specially designed olfactometer that provided different channels for the eye and nose, they collected respiration data in human subjects to "nose only" and "eye + nose" trials. Using amyl acetate (a banana-like and relatively pleasant smell at low concentration), they found that breathing flow rate increased at the lower concentration presented to "nose only." At the highest concentration of "nose only" administration, breathing flow was slightly reduced. When the same stimuli were presented to the "eye + nose," subjects responded as if they had been exposed to far more amyl acetate, that is, breathing was significantly reduced as a function of concentration. From these studies, it appears that receptors in the eye interact with those in the nose to alter breathing and initiate respiratory volume reductions at relatively low concentrations of chemical stimulation.

The fact that odor sensations are linked so closely with irritant sensations is due in part to the central projections of the olfactory and trigeminal systems. The trigeminal nerve projects to fibers that overlap with brain areas of olfactory projection such as the mediodorsal nucleus of the thalamus.[64] Additionally, the trigeminal nerve projects to many areas of the brainstem associated with autonomic responses such as nasal secretion, sneezing, and respiration.[36] Silver and Finger[11] emphasized that these physiological reflexes are "among the strongest in the body." The magnitude of these responses underscores the evolutionary importance of olfaction as a warning and response mobilization system.

Methods to Quantify Irritation

A variety of methods have been developed to quantify irritation and specifically to determine the concentration at which volatile compounds activate the trigeminal nerve. Measurement of irritation is generally achieved in one of three ways. First, verbal measures can be obtained in human subjects in psychophysical experiments. Second, electrophysiological (nonverbal) responses to irritants such as nasal mucosal potentials and central event-related potentials can be measured in human subjects. Third, animal models can be used to assess respiratory or neural effects of irritants. Equations using quantitative structure-activity relationships (QSAR) based on solvation energies have also been used to predict nasal irritation thresholds but this is still in the basic research phase.[65,66]

Human Psychophysical Ratings. First, irritant thresholds and intensity ratings can be obtained in normosmic human subjects.[12,67,68] Psychophysical ratings involve verbal reports of perceived odor and irritation. However, in normosmic subjects, ratings of irritation can be affected by the concomitant olfactory sensations. In order to determine the effect of trigeminal input alone, judgments of irritation are frequently obtained in anosmics who lack olfactory sensations but have an intact trigeminal system.[69-71] There is some controversy, however, whether anosmics are an appropriate model since irritation (as well as olfaction) may be blunted in anosmic/hyposmic subjects.[72] Recently, localization of chemosensory nasal stimulants has been used to determine sensitivity to irritants by determining nasal lateralization thresholds in normosmics.[73-75] Irritation, unlike olfaction, can be localized to one nostril or the other. Thus, the lowest concentration at which a vapor can just be lateralized (the nostril receiving the stimulus can be determined), constitutes the true irritant threshold. Several experimental methods have been used to determine lateralization thresholds.[57,76] In addition, Cometto-Muñiz and Cain[77] found that thresholds for eye irritation closely predict nasal irritation thresholds, and can serve as a practical means to assess potency for nasal irritation in normosmics.

Schiffman[78] used the lateralization method to determine if the odorous ambient air inhaled by persons located 1500 feet downwind from a swine facility was an irritant. The odorous air was delivered to one nostril and clean air from a Tedlar® bag was delivered to the other. The four subjects (while blindfolded) were able to correctly identify which nostril received the odor. Furthermore, they rated the sensation in the nostril to which ambient air was directed as irritating. While additional studies must be performed to further investigate this result, it suggests that inhalation of odorous ambient air downwind from swine facilities can stimulate the trigeminal nerve and induce sensory irritation.

Human Electrophysiological Responses to Irritants. Electrophysiological methods for measuring responses to irritation include peripheral negative mucosal potentials (NMPs) and central event-related potentials (ERPs).[79-81] NMPs are recorded by means of an electrode on the septal wall of the nasal cavity along the line between bony and cartilaginous parts of the nose (referenced against the contralateral bridge of the nose). The NMPs are thought to result from activation of both C-fibers and A-delta fibers. Odorants do not tend to produce NMPs at concentrations below the irritation threshold.[80] ERPs are recorded from electrodes on the scalp and respond to both trigeminal and olfactory stimuli. In one study, substances that stimulated the trigeminal nerve were found to produce maximum amplitudes at the vertex, and those that stimulated the olfactory nerve produced maximum amplitudes at parieto-central sites.[82] In addition, trigeminal stimulation involves the right hemisphere more than the left according to Hari et al.[83] ERPs appear to reflect nociceptive information transmitted by A-delta fibers of the trigeminal nerve but not necessarily C fibers.[52] Reflexive changes in nasal blood flow to irritants can be measured using a laser Doppler flow meter.[80] Pneumotachograph measurements indicate that there is a reduction of tidal volume (volume per breath) that begins at the threshold of nasal irritation.[17]

Animal Studies of Irritation. Electrophysiological responses to irritants can be determined in an animal model by recording from the ethmoid nerve (a branch of the ophthalmic division of the trigeminal nerve) which innervates the anterior nasal mucosa[9] or by recording from the nasopalatine nerve (a branch of the maxillary division) which innervates the nasal mucosa in the posterior portion of the nasal cavity.[84] Animal models can also be used to assess respiratory responses to irritants. Reflexively induced decreases in respiratory rate are caused by stimulation of the trigeminal nerves by irritants.[37,85] Mice are placed in a plethysmograph with their heads in an exposure chamber. The respiratory rate is monitored before, during, and after exposure. The dose-response relationship between the maximum percentage decrease in respiratory rate during the exposure period (e.g., 10 minutes) and the logarithm of the concentration of the irritant is plotted. The RD_{50} (50% decrease in respiratory frequency) is calculated from the log concentration-response curve. A computerized version of this test has been developed to quantify breathing patterns in unanesthetized mice exposed to volatile chemicals.[86-91] It should be noted that reflex momentary apnea (interruption of inhalation) in response to irritation can also be recorded in humans. Apnea is reflexive response to irritant stimulation that protects the upper airway.[92] Breathing patterns before, during, and after presentation of various concentrations of a potential irritant can be used to determine the concentration sufficient to elicit the reflex.[12,93] While bioassays of irritation in animals can provide helpful information, current research suggests that humans are more sensitive to irritation than animals.[17]

Evidence for Paradigm 2: Health Symptoms Occur at Odorant Concentrations That Are Not Irritating

Historically, malodor has been considered an indicator of potential health risk.[94-96] However, the mechanism by which unpleasant odors cause health complaints in the absence of irritation or toxicity is poorly understood. Health complaints do occur at levels of VOCs that are below irritant thresholds.[23,97] Factors such as the degree of unpleasantness of the odor, the exposure history (prior experience with odor), beliefs about the safety of an odor, and emotional status may play a role in inducing health symptoms. Both genetics and learning may play a role in health complaints to unpleasant (but nonirritating) odors. There is an extensive animal literature that indicates that airborne chemicals can affect behavior. In humans, airborne chemical signals have even been shown to affect ovulation.[98]

Physiological Responses to an Unpleasant Odor in the Absence of Irritation

In one study, fourteen of 26 workers exposed to presumably safe levels of odorous sewer gases (as measured by gas detection equipment) experienced sore throat, cough, chest tightness, breathlessness, thirst, sweating, irritability, and loss of libido. Severity of symptoms was dose related. Clinical follow up showed deteriorating respiratory symptoms and lung function tests in the most seriously affected.[23] Chemical analysis showed that the workers had been exposed to a mixture of thiols and sulfides. In another study, exposure to the odor of n-propyl mercaptan in an agricultural setting for 6 weeks led to significant exposure effects including headache, diarrhea, runny nose, sore throat, burning/itching eyes, fever, hay fever attacks, and asthma attacks.[99]

The mechanism by which these unpleasant odors induced health symptoms in the absence of irritation or toxicity is not known. However, Gift and Foureman[100] reported that the RD_{50} values (concentration that induces 50% decrease in respiratory rate) for a random sample of unpleasant smelling compounds were much lower than for pleasant smelling compounds. Schiffman[78] found that shallow and irregular breathing patterns were induced by exposure to unpleasant odors (swine odors, rotten fish, sulfides) while deeper stable breathing patterns were characteristic of exposure to pleasant odors (chocolate chip cookies, orange cake). These differences in breathing patterns (whether genetic or learned) may influence health symptoms.

Furthermore, unpleasant odors induce different patterns of electrical brain activity and activate different areas of the brain than pleasant odors. Electro-olfactograms (EOGs) and electroencephalograms (EEGs) have unique and distinct patterns that differ according to the hedonic properties of odors.[101-104] Studies using neuroimaging techniques also indicate that there are specific physiological neural markers for olfactory hedonics. Zald and Pardo[105] measured regional cortical blood flow (rCBF) using positron emission tomography and found that highly aversive (sulfides) and pleasant smells (fruits, flowers, and spices) activated different brain regions. The fact that olfactory hedonics differentially affects brain activity may have genetic and/or learned components. Electroencephalography (EEG) and functional magnetic resonance imaging (fMRI) studies have even shown that odorants and airborne chemicals can affect the nervous system without being consciously detected.[106-108] Further research is necessary to determine if the areas of the brain stimulated by odors that differ hedonically is affected by experience and/or national origin. It has not yet been determined whether the patterns of brain activity induced by unpleasant odors contribute to health complaints.

Mood Impairment and Stress Induced by an Unpleasant Odor

Odors perceived to be unpleasant can impair mood[5,109] and increase reactivity to startling stimuli.[110] Schiffman et al.,[5] for example, studied the effect of odorous emissions emanating from large-scale hog operations on the mood of nearby residents. Scores on the Profile of Mood States (POMS) indicated that nearby residents experienced an acute impairment of mood when odor from the swine operations was present. This included increased levels of tension, depression, anger, fatigue, and confusion.

Negative mood, stress, and environmental worry can potentially lead to a number of physiological and biochemical changes with subsequent health consequences.[111,112] These include elevations in blood pressure, both in normotensives and in patients with hypertension,[113-115] immune impairment,[116] increased levels of peripheral catecholamines,[117] increased glucocorticoids,[118] increased secretion of adrenocorticotropic hormone (ACTH) from the pituitary,[117] decreased gastric motility,[119] increased scalp muscle tension in patients with muscle tension headaches,[120,121] and even hippocampal damage.[122] Chronic stress has been associated with development of coronary artery disease, chronic hypertension, and structural changes of the heart in some studies.[123-125] Thus, if odorous stimuli are sufficiently stressful, this could potentially elevate the catecholamines epinephrine and norepinephrine to levels that produce adverse cardiovascular effects including increased heart rate and blood pressure and increased tendency of blood to clot.[126] However, further research is necessary to determine if odors from animal and municipal wastewater facilities do cause these types of stress-related health problems in susceptible individuals.

Several studies have shown a relationship between odors and stress effects. Cardiovascular effects have been reported to numerous odorous stimuli including fresh diluted sidestream cigarette smoke.[127] Several changes in blood lipid measurements were observed in both male and female subjects after exposure for 7.33 hours. In male subjects, there was a 15% increase in triglyceride levels and a 4.8% decrease in high density lipoprotein (HDL) levels. Smith and Scott[128] noted that these lipoprotein changes are consistent with stress-related epinephrine-induced mobilization of free fatty acids and a concomitant decrease in HDL.[129] Steinheider et al.[130,131] found an association between urinary cortisol levels and odor exposure at a fertilizer manufacturing facility. The elevated cortisol levels associated with malodor and irritation can potentially induce stress-related immune dysfunction.

Learned Associations and Health Symptoms

Conditioned or learned associations can play a role in perceptions and health symptoms induced by odors.[132-135] For example, the odor may have been previously associated with a maladaptive physiological response. Abnormal respiration can be produced by an odor if it was previously associated with a respiratory challenge such as an irritant.[136] Histamine can also be released as a learned response to presentation of an odor.[137] Aversive conditioning appears to occur to a broad range of odorous compounds including solvents, aldehydes, acid vapors, and phosphine gas.[132,133,138-142] Odors can also prompt retrieval of emotionally laden memories.[143,144] Odors can modify synaptic plasticity in the hippocampus and piriform cortex (parts of the limbic system) which are associated with learning and emotion.[145] The animal literature indicates that odor aversions are readily established and robust;[146,147] while they can be extinguished, the process occurs slowly. Health symptoms in humans can sometimes be unlearned (extinguished) using a technique called systematic desensitization.[136,148]

Odor-conditioned panic attacks or panic disorder have been reported after exposure to odors in the workplace.[142] Whether these learned responses should be deemed ‘‘health effects’’ from odors, however, is controversial because the term ‘‘health’’ has multiple meanings in scientific, regulatory, and legal settings. According to the World Health Organization (WHO), the definition of ‘‘health’’ is ‘‘. . . a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity.’’ Thus, a symptom that diminishes physical, mental, or social well-being would be a ‘‘health effect’’ according to WHO. The majority of the participants at the Health Effects of Odors workshop considered it appropriate to explore health effects of odors within the WHO definition of health. Participants at a subsequent workshop sponsored by the Centers for Disease Control also agreed the potential health effects associated with exposure to confined animal feeding operations (CAFOs) should be viewed according to the WHO definition of health.[149] Frist[150] emphasized that reactions to odors such as nausea, headache, loss of sleep, and loss of appetite clearly represent a matter for public-health concern and attention under the WHO definition of health. Using a broad definition of health that includes quality of life and social and mental well-being, Mitchell et al.[151] concluded that malodorous air in an urban environment causes adverse health effects. Other types of sensory overload such as noise pollution can also contribute to ill-health using the WHO definition, which includes positive mental and social well-being.[152] Attendees at the workshop also agreed, however, that more experimental data are required to substantiate the opinion that intermittent odors from industrial, agricultural, and municipal facilities adversely affect the health of persons off-site such as residential neighbors. The intensity, duration, and frequency of health symptoms must be carefully evaluated before drawing the conclusion that such symptoms constitute a health effect.

Beliefs About Safety and Health Symptoms

Malodorous compounds frequently engender concerns for safety; in a typical air pollution control district in California, roughly 70-80% of citizen-initiated calls are concerned with environmental odors.[153] In a meta-analysis of three epidemiological surveys conducted near hazardous waste sites, Shusterman and colleagues[112] found that the frequency of perceived environmental odors and degree of self-reported "environmental worry" synergistically predicted such symptoms as headaches, nausea, eye, nose, and throat irritation. Dalton and colleagues[154-156] subsequently showed in an experimental setting that beliefs about the safety of an odor can have an effect on sensory ratings as well as health complaints. In one study, three groups of subjects were given different information (positive, neutral, and negative) about the same odor. The "positive" group, was told that the odor was a natural extract used by aromatherapists while the "negative" group was told that the odorant was an industrial chemical that purportedly caused health effects after long exposure. The negative bias group rated the odors as more irritating and had the greatest number and intensity of self-reported health symptoms including nose, throat, and eye irritation as well as lightheadedness. Smeets and Dalton[156] reported that persons with a tendency to worry in general or to have a negative emotional orientation to life reported more symptoms of sensory irritation to the odor of rubbing alcohol.

Morgan[157] emphasized that unpleasant sensory properties of odorous compounds are not necessarily a good predictor of safety. For example, certain ripe goat cheeses may emit unpleasant odors while being perfectly safe to eat while wild mushrooms may have pleasant odor but are poisonous to eat. The smell of rotten meat is certainly an indication of danger but only if the meat were to be consumed. Hence, unpleasant smells may not be harmful from a toxicological point of view beyond their unpleasant nature; yet, physical reactions to unpleasant odors do occur.

Individual Differences in Physiological Responses to Odors

Bell and colleagues[158-162] have found a subset of the population that appears to have an intolerance to low-level chemical odors from sources such as car exhaust, pesticides, paint, new carpet, and perfume. This intolerance presumably can occur at levels both below and above irritant thresholds. Odor intolerance has been associated with increased cardiopulmonary risk[163] including increased sympathetic tone in the cardiovascular system at rest,[160] different EEG alpha rhythms,[164] lower rapid-eye-movement (REM) sleep,[165] and greater prevalence of chronic cough, phlegm, wheeze, chest tightness, exertional dyspnea, acute respiratory illnesses, hay fever, child respiratory trouble, and physician confirmed asthma.[161] The reasons for these biological responses in odor-intolerant individuals are not known but Bell et al.[162] suggested that sensitized dysfunction of the limbic and mesolimbic systems could account in part for many of the cognitive, affective, and somatic symptoms. Many of these responses may also be learned odor aversions.[166]

Evidence to date suggests that individuals with intolerance to low level chemical odors do not have lower perceptual thresholds, despite their augmented subjective responsiveness to suprathreshold stimuli.[167,168] However, it is noteworthy that intermittent exposure to the odor of androstenone (a boar taint odor) in humans[169] and animals[170] has been found to induce a highly significant increase in odor sensitivity to androstenone in previously insensitive individuals. Elevated sensitivity to isovaleric acid (a component of swine odor) after intermittent exposure to isovaleric acid has also been induced in animals.[170] Wysocki et al.[169] and Wang et al.[170] suggested that the increase in sensitivity to androstenone and isovaleric acid from intermittent exposure may be due to clonal expansion of olfactory receptors with high affinity for these compounds.

Karol[171] suggested that inhalation of airborne chemicals can augment allergic sensitization with episodic pulmonary reactions occurring on subsequent exposures. These reactions could involve the upper respiratory tract (rhinitis), lower respiratory tract (wheeze, bronchospasm), or systemic immune involvement (febrile response). While the mechanisms of sensitization are not well understood, mediators of immunity are definitely involved.

Evidence for Paradigm 3: A Co-Pollutant in an Odorous Mixture Is Responsible for the Reported Health Symptom

In agricultural settings, odorant mixtures typically contain co-pollutants such as particulates, endotoxin, and pesticides. Particulates can arise from confinement building exhausts, dry feedlots, composting facilities, lagoons, and land application sprays. Particulates from intensive animal housing consist mainly of manure, dander (hair and skin cells), molds, pollen, grains, insect parts, mineral ash, feathers, endotoxin, and feed dust.[172] Airborne dust particles can concentrate odorants such as organic acids and ammonia on their surfaces;[173,174] this contributes to odor potential and exacerbates irritancy induced by dust in the respiratory tract. Experimental studies have found a strong link between odor/irritation intensity and levels of particulates.[172] Particulates associated with fecal waste are also known to carry bacteria.[175] Thus, it is likely that some of the health complaints ascribed to odor may, in fact, be caused by particulate matter (liquid or solid) suspended in air or by a synergistic effect between odorants and particulates. A synergistic effect of ammonia and dust exposure has been reported in a study of 200 poultry facilities. The adverse health effects of ammonia and particulates in combination was greater than the additive effect of ammonia and particulates by a factor of 1.5 to 2.0.[176]

Both fine and coarse particles in an odorous plume enter the nasal cavity and can induce nasal irritation. However, these particles differ in the degree to which they traverse the respiratory tract. Fine particles include particulate matter with sizes less that 2.5 μM ($PM_{2.5}$). These particles are more likely than coarse particles to cause respiratory health effects because they reach the gas-exchange region of the lung. Ultrafine particles (i.e., those with a diameter 0.1 μM or less) may be even more toxic than larger sized particles producing severe pulmonary inflammation and damage and even affecting mortality.[177-182] Fine particles remain suspended in the atmosphere for days and can be transported thousands of miles. Particles with sizes from 2.5 μM to 10 μM ($PM_{2.5-10}$) are coarse particles that enter the thorax and may also induce health effects. There is an overlap of fine and coarse mode particles in the intermodal region of 1 to 3 μM.[26,183] Coarse particles are usually mechanically generated. Sources of coarse particles near confined animal operations and other locations of biosolids include windblown dust from soil, feed, manure, unpaved roads, pollen, mold spores, parts of plants and insects, and evaporation of aqueous sprays. Coarse particles tend to settle rapidly from the atmosphere within hours and usually travel short distances (except in dust storms). Coarse particles in outdoor air are less likely to infiltrate indoor air than fine particles.

Fine particles may be formed in the atmosphere from gases through the processes of nucleation and growth.[26,183-188] Nucleation entails formation of very small particles from gases. Substances with low saturation vapor pressures are formed in the gas phase through chemical reactions in the atmosphere or by high-temperature vaporization. These substances grow into particles by coagulation (in which smaller particles coalesce to form larger particles) and condensation (in which gases condense onto existing particles). The resultant particles tend to accumulate in the size range from 0.1 to 1 μM. One example is the oxidation of the gas SO_2 to SO_3 and to sulfuric acid (H_2SO_4) with subsequent formation of fine particles either by nucleation followed by coagulation or by condensation on existing particles. Another example is the oxidation of NO_2 to nitric acid (HNO_3) which reacts with ammonia (NH_3) to form fine particles of ammonium nitrate. Ammonia salts that exist as fine aerosols can be transported long-range in the atmosphere.[186] Third, photochemical reactions generate ozone and OH^-, and these react with organic gases (such as odorous compounds) to form materials with low vapor pressure that can nucleate or condense on existing particles. These processes may occur in the troposphere from precursors emitted into the atmosphere from agricultural facilities such as lagoons on swine farms. They are more likely to occur in the warmer months as a result of atmospheric reactions.

Epidemiologic studies of exposure to particulates have reported statistical associations between daily changes in health outcomes such as mortality and daily variations in the concentrations of different sizes of ambient particulate matter.[183] There is considerable epidemiological evidence predominantly from urban settings that exposure to increased levels of particulates is associated with increased mortality risk, especially among the elderly and individuals with preexisting cardiopulmonary diseases, such as chronic obstructive pulmonary disease (COPD), pneumonia, and chronic heart disease.[26] There is also epidemiological evidence that particulate exposure can increase the risk of respiratory and cardiovascular morbidity such as increased hospital admissions or emergency room visits for asthma or other respiratory problems, increased incidence of respiratory symptoms, or alterations in pulmonary function. This can begin to occur when ambient particles smaller than 10 μM fall between 30 to 150 $\mu g/m^3$ according to the Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society.[189] Daily fluctuations in these levels are related to acute respiratory hospital admissions in children, to school and kindergarten absences, to decrements in peak expiratory flow rates in normal children, and to increased medication use in children and adults with asthma.[189]

The concentration of total particles as well as respirable particles inside confined animal operations far exceeds the 30 to 150 μg/m^3 level at which symptoms can purportedly begin according to the Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society.[189] An overview of the literature suggests that typical total particulate levels inside swine confinement houses are 5 mg/m^3. Total dust levels have even been reported to reach from 15 mg/m^3 up to 52 mg/m^3 in some houses.[190-192] Respirable dust comprises 5 to 50% of the total dust.[174] While levels of dust in the livestock houses are high, the levels at neighboring properties are difficult to determine for several reasons. First, time-averaged sampling of dust downwind gives lower values than the peak dust levels because the samplers are usually in the plume for only a short period of time due to shifts in the wind direction. Second, the geographical location where the plume reaches the level of potential perception (e.g., a neighbor's nose) may be a small physical area that is difficult to locate for measurement purposes in real time. Third, particulates from the swine confinement houses and particulates from the lagoon may both contribute to the exposure but may or may not occur simultaneously.

Bacterial exposures may also be responsible for some health complaints from exposure to odorous emissions from agricultural operations.[193] Bacteria are ubiquitous in swine houses; furthermore, aerosols formed over lagoons may allow the transfer of bacteria from the water into the air with transfer downwind in aerosol droplets.[194] Endotoxin, a heat-stable toxin associated with the outer membranes of certain gram-negative bacteria, can reach levels as high as 2,410 ng/m^3 to 78,600 ng/m^3 in swine facilities.[195] The American Conference of Governmental Industrial Hygienists' Threshold Limit Value-Time Weighted Average (ACGIH TLV-TWA) for endotoxin is 10 ng/m^3; this is the time-weighted average concentration for a conventional 8-hour workday and 40-hour workweek, to which nearly all workers may be repeatedly exposed daily without adverse effects. Endotoxins cause an inflammatory response of the respiratory tract. Atopic asthmatic individuals have elevated sensitivity to respirable endotoxin which results in a variety of immune responses including increased eosinophils in the airways.[196] Furthermore, exposure to allergens in atopic asthmatic individuals augments subsequent endotoxin-induced nasal inflammation.[197]

Studies that trace the transport of odorous VOCs within olfactory and trigeminal nerves may also be helpful in understanding health effects of odors. Both small and large molecules can be transported to the brain in the olfactory and trigeminal nerves.[198-204] Thus odorous VOCs or co-pollutants such as viruses that enter the nose can potentially reach the central nervous system by neuron to neuron transmission. For example, herpes simplex virus can infect the trigeminal nerve and ultimately enter the CNS.[205] Viruses can also infect olfactory receptor neurons.[206] However, far more research is needed to determine if any health effects from exposure to odorous emissions from agricultural facilities or biosolids are due to transport of VOCs or viruses in nasal sensory nerves.

Further research is also required to determine if the levels of dust, endotoxin, or other co-pollutants (such as flying insects) transported in odorous plumes are high enough to cause health symptoms in neighbors of agricultural or municipal operations. Flying insects are attracted to odors from urine, feces and gut mucus[207] and often follow odor plumes to find resources.[208] Flying insects have the potential to carry disease.

Vulnerable Populations

Two segments of the population appear to be especially vulnerable to respiratory effects from odorous environmental exposures: persons with asthma and persons with high occupational exposure to odor and dust.

Asthma and Allergies

Odors have been reported to exacerbate symptoms of asthma[7,209-214] but it is not clear whether the main cause of this worsening is due to direct irritation of mucous membranes by the odorant, to sensory stimulation of the olfactory and/or trigeminal nerve, or to prior conditioning. Asthma is characterized by bronchial hyperresponsiveness and mucosal airway inflammation; it is the leading chronic illness among adults and children.[215] Epithelial damage and epithelial shedding occur in the airway passages in asthma[216,217] as well as other respiratory disorders including nasal allergy[218] and infantile wheeze.[216] Even healthy individuals exposed to a polluted environment (e.g., ozone) can experience epithelial shedding which can last up to 2 weeks or more.[219] Nerve endings are exposed by epithelial shedding;[220] this allows VOCs and particulates access to free nerve endings which augments irritation from inhaled pollutants. Irritants can then set up a low grade neurogenic inflammation with leukocyte recruitment that aggravates asthma and allergy.[221-224] It has been suggested that even anaphylaxis can be triggered by chemical odors.[225]

Occupational and Environmental Exposure

There are health risks associated with prolonged exposure to highly odorous ambient air in the work or home environment.[226,227] Persistent asthma-like symptoms can result from a single excessively high environmental or occupational exposure to odorous/irritant substances such as paint, floor sealant, ammonia, chlorine, acetic acid, and hydrogen sulfide from manure.[228-232] This syndrome was termed RADS (reactive airways dysfunction syndrome) by Brooks et al.[228] The duration of the single exposure can be as short as a few minutes to as long as 12 hours. RADS, by definition, occurs in persons with no evidence of preexisting pulmonary disease. Another defining characteristic is that symptoms can persist after termination of the exposure for at least three months; but in fact they may persist for one year or more. Bronchial biopsies suggest respiratory epithelial injury, but the mechanisms operative in the syndrome appear to be nonimmunological. Persons with RADS were generally aware of an odor that was present during the irritant exposure.[228,229] For example, one man who developed RADS after exposure to a sealant containing several aromatic hydrocarbons (including decane, ethylbenzene, toluene, and xylol) noted a "glue" or "varnish" odor at the time of the exposure. A woman who developed RADS after her apartment was fumigated noted a background odor like "insect exterminating solution."

Agricultural workers have also been reported to suffer respiratory symptoms from exposure to highly odorous and dusty environments. Donham et al.[233] were the first to suggest that there are occupational health risks related to working in highly odorous intensive swine housing facilities. Since that time, other studies have confirmed occupational health risks to swine workers.[226,234-243] Documented irritant/odorant exposures include hydrogen sulfide, ammonia, and dust. In an overview of recent studies, Donham[234] reported that at least 60% of swine confinement workers have acute or sub-acute respiratory symptoms that include dry cough, chest tightness and wheezing on exposure to the work environment. Other frequent symptoms include irritation of the nose, eyes and throat, stuffy nose and head. Furthermore, at least 25% of pig farmers suffer from organic dust toxic syndrome which is characterized by periodic, acute febrile episodes with fever, headache, muscle aches and pains, chest tightness and cough.[234,243] Chronic bronchitis, occupational (non-allergic) asthma, and non-infectious chronic sinusitis are also prevalent among pig farmers.[234,240] These symptoms can be induced by odorous and irritant VOCs as well as dust and endotoxin. There appears to be a synergistic effect between volatile compounds and dust exposure in producing these symptoms.[176] Symptoms appear to be progressive with an annual decline in lung function.[241,242]

Health symptoms can also occur acutely and reversibly with even brief exposure to odorous and dusty agricultural environments. Jolie et al.[244] reported that adverse health symptoms were experienced by 103 of 142 veterinary students (72.5%) who worked with pigs on a swine farm for three hours. Respiratory symptoms including cough, nasal and throat irritation, and sinus trouble were reported by 94/103 (91%) of the students. Other frequent symptoms experienced by the students included eye irritation, headache and tiredness. Students with pre-existing allergies were the most likely to develop respiratory symptoms.

Quantification of Health Symptoms

Workshop participants concluded that current evidence suggests that the symptom complaints experienced by neighbors of some odorous animal operations and other sources of biosolids may constitute health effects. However, further research studies in both laboratory and field settings are necessary to quantify the concentration/intensity ranges that cause health complaints in the general population as well as in sensitive (e.g., allergic) individuals. These studies should utilize objective biomarkers of health symptoms to validate health complaints. A set of potential study tools and biomarkers were proposed at the workshop to validate odor-related symptoms in clinical, epidemiologic, and research studies. These are given in Table 1. Workshop participants stressed the need to relate these health measures to levels of exposure.

TABLE 1. Potential study tools and biomarkers for the validation of odor-related symptoms in clinical (C), epidemiologic (E), and research (R) studies

Symptom	Potential study tool and reference	C	E	R
Eye irritation	Slit lamp examination[245,246]	X	X	
	Blink rate[247]			X
	Tear film stability[248]			X
	Lissamine green staining of conjunctiva[249]			X
	Corneal CO_2 threshold[250]			X
Headache	Electromyography (EMG) for tension headache[251,252]		X	X
	Functional imaging for vascular headache[253]			X
Nasal congestion	Longitudinal study of nasal peak flow[254,255]	X	X	
	Rhinomanometry[255]			X
	Acoustic rhinometry[255]			X
	Rhinostereometry[255]			X
Nasal Irritation,	Physical exam	X		
Burning	Nasal lavage[256-259]		X	X
	Nasal cytology[258]		X	
	Negative mucosal potential[80]			X
	Nasal mucosal blood flow by Laser-Doppler velocimetry[260]			X
Epistaxis (nosebleed)	Physical examination	X	X	X
Throat irritation	Physical examination (insensitive)			
Nausea	None			
Hoarseness/globus	Rhinolaryngoscopy[261]	X		
	Acoustic analysis: noise-to-signal (N/S) ratio[262]			X
Palpitations	Physical exam (heart rate)	X	X	X
	Electrocardiogram/rhythm strip[263,264]	X	X	X
	Ambulatory rhythm monitoring (Holter)[265,266]	X	X	
	Telemetry[267]			X
Sensory alterations	Psychophysical tests[268]	X	X	X
(Taste and smell)	Biopsy of chemosensory tissue[227]	X		X
Shortness-of-breath	Physical exam	X	X	X
(wheezing)	Peak flow[269-271]	X	X	X
	Spirometry/Pulmonary function tests (PFTs)[272,273]	X	X	X
	Methacholine challenge[272,273]	X	X	X
Shortness-of-breath	Physical exam (respiratory rate)	X	X	X
(air hunger)	Arterial blood gas (ABG)/transcutaneous CO_2 (TC-CO_2)[274]	X	X	X
	End tidal CO_2[275-277]	X	X	X
Blood rheology	Altered plasma viscosity caused by inflammatory processes in the lung[278]			X

Symptom	Potential study tool and reference	C	E	R
Stress	Physical exam (Affect, tremor, skin moistness)	X		
	Serum, urine, and salivary cortisol[279-282]		X	X
	Natural killer (NK) cell count[283,284]		X	X
	Salivary IgA[285]		X	X
	Galvanic skin response[286,287]		X	X
	Urinary catecholamines[288,289]		X	X

METHODS TO QUANTIFY LEVEL OF EXPOSURE TO ODORS

Accurate methods to quantify odorous emissions are necessary to determine the relation between potential health symptoms and odors. However, no United States governmental agency has developed standard test methods that can serve as an indicator of odor potential or verify objectionable odor which can be used to relate to potential health symptoms. Furthermore, there is wide variability among individuals in the odor intensities and odorant concentrations that cause health complaints. To address this issue, levels of odor exposure were defined to clarify the intensities associated with potential health impacts described in three paradigms above.[160,290] This set of odor levels in increasing intensity includes the following:

Level	Description
1) odor detection	The level of odor that can first be differentiated from ambient air
2) odor recognition	The level of odor at which the odor quality can be characterized, e.g., the level at which a person can detect that an odor is apple or manure.
3) odor annoyance	The level at which a person is annoyed by an odor but does not show or perceive a physical reaction. Note: Health symptoms are not expected at these first three levels unless the odor occurs with a co-pollutant such as dust as in Paradigm 3 or the level of annoyance is intense or prolonged.
4) odor intolerance (causing somatic symptoms)	The level at which an individual may show or perceive physical (somatic) symptoms to an odor. Note: This level corresponds to Paradigm 2 in which the odor induces symptoms even though the odorant concentration is lower than that known to cause irritation.

Level	Description
5) perceived irritant	The level at which a person reports irritation or physical symptoms as a result of stimulation of nerve endings in the respiratory tract
6) somatic irritant	The level at which an odorant (not an odor) results in a negative physical reaction regardless of an individual's predisposition. This can occur when an odorous compound (e.g., chlorine) damages tissue. Note: Perceived and somatic irritation correspond to Paradigm 1.
7) chronic toxicity	The level at which an odorant can result in a long-term health impact.
8) acute toxicity	The level at which an immediate toxic impact is experienced, e.g., a single event may evoke an acute health impact. Note: In the case of chronic or acute toxicity, the compound should not be considered an odorant but rather a compound with toxic effects that happens to have an odor.

The range of odor intensities and odorant concentrations that correspond to these 8 levels varies across individuals.

A variety of measurement methods can be used to obtain quantitative data that correspond to each of these 8 levels including: (1) olfactometry, (2) gas chromatography, and (3) the electronic nose. Olfactometry is a measurement technique that uses the human nose as the sensor. It is the most precise approach to quantify odors because the human nose can detect compounds at concentrations that cannot be detected by current real-time analytic methods. Gas chromatography is an analytical method that separates the gaseous mixture of chemical compounds into its molecular constituents. Gas chromatography can be used to obtain quantitative data on the concentrations of individual compounds in an odorant mixture that correspond to the 8 levels above. An electronic nose is an instrumentation system that uses the pattern of response across an array of gas sensors to identify an odor. It holds promise for simulating human responses as the technology improves. New analytic methods will most likely be developed in the future to detect levels and identity of odorous volatile compounds in real time. Each of the three current methods used to quantify odor (olfactometry, gas chromatography, and the electronic nose) are described in more detail below.

Olfactometry

Human assessment of odors is performed by dynamic olfactometry or by static olfactometry. In dynamic olfactometry, an odorous stream of air is delivered continuously toward the nose by an olfactometer, a device that dilutes the odor vapor with odorless gas. In static olfactometry, odorous samples (such as lagoon water or pieces of cotton that have adsorbed odorants) are presented to the nose in an enclosed volume such as a sniff bottle.

Dynamic olfactometry is used to evaluate gaseous samples that are collected in Tedlar® bags or canisters. For example, samples may be obtained from inside swine houses or at the exhaust fans.[68] The dynamic olfactometer produces an odorous airstream that can be diluted to its detection threshold. The detection threshold for a given air sample is the concentration at which an odor is first detected. Dilution to threshold (D/T) measurements are used to measure detectability. The odor concentration at the detection limit is defined to be 1.0 odor unit/m^3 (OU). At about 4 OU (4 dilutions required to reach threshold), complaints about objectionable odors tend to escalate.[78] At each serial dilution above detection threshold, the human panelist may also be asked to rate the odor on standard descriptive scales for odor quality (a measure of odor character), odor intensity (a measure of odor strength), and irritation intensity (a measure of irritation strength). The same types of ratings can be obtained using static olfactometry. Another method to quantify intensity is to match each concentration to a series of n-butanol standards according to ASTM E544-75.[291]

Odor quality or character is usually evaluated on a series of descriptive (adjective) scales. A standard series of 146 adjective descriptors was developed by the American Society for Testing and Materials.[8,291,292] A subset of these descriptors most frequently used by panelists to describe odors from swine operations include: animal, fecal (like manure), sickening, musk-like, stale, sweaty, sewer-odor, ammonia, sour/acid/vinegar, chemical, burnt, smoky, yeasty, cheesy, etherish, anesthetic, like blood, raw meat, turpentine (pine oil), like ammonia, sharp, pungent, acid, camphor-like, wet wool, wet dog, sewer odor, black pepper-like, bean-like, cooked vegetables, urine-like, rancid, seminal, sperm-like, sulphidic, putrid, foul, and decayed.[68]

Each odor dilution can also be evaluated for its acceptability or offensiveness. For example, ratings can be made along the following 9-point scale (extremely pleasant, very pleasant, moderately pleasant, slightly pleasant, neither pleasant nor unpleasant, slightly unpleasant, moderately unpleasant, very unpleasant, extremely unpleasant). While the acceptability of the odor of some VOCs depends on learned or cultural factors (experience), odors of other compounds such as H_2S, mercaptans, amines, and nitrogenous heterocylic compounds are considered offensive by most individuals.

Measurements of odor thresholds off-site of an odor source can sometimes be obtained using a portable olfactometer. Sweeten[293] used a Barnebey-Cheney Scentometer[294] to determine thresholds downwind from swine farms. He found the number of dilutions to threshold (D/T) could be as high as 170 at 3,000 feet and as high as 31 at 1 mile.[293] However, the average odor strength at 3,000 feet was about 10 odor units. Scentometer readings are generally interpreted as follows: 2 (a noticeable odor), 7 (an odor most people would find objectionable), 15 (most would declare it a nuisance), and 31 (extremely nauseating).

Quantification of odor off-site is often difficult to achieve, however, due to shifts in the odor plume, fluctuations in wind speed, and potential background odors. Because odor plumes are moving targets, tracer gases such as SF_6 (sulfur hexafluoride) or helium balloons are potentially helpful in monitoring dispersion of odorants.[295,296] Estimates of odor concentrations off-site are usually predicted from source data (e.g., livestock house or lagoon) using dispersion modeling.

Gas Chromatography/Mass Spectrometry

The constituents in odor mixtures can be separated and identified by gas chromatography (GC) and mass spectrometry (MS), respectively. Volatile compounds identified in livestock manure include sulfides, disulfides, volatile organic acids, alcohols, aldehydes, amines, fixed gases, nitrogen heterocycles, mercaptans, carbonyls, and esters.[33] The concentrations of individual components of the odor mixture are generally in the parts per billion (ppb) or even parts per trillion (ppt) range. For this reason, GC/MS generally requires some form of preconcentration to obtain enough mass for analysis. Thus, quantification of the constituents of an odor mixture cannot be performed in real time. Over 400 compounds have been found in volatile emissions from swine facilities.[33]

One limitation with using GC/MS to quantify odor is that the individual odorous compounds may not smell unpleasant at the concentrations in the mixture, yet the mixture (or combination of odorous compounds) may smell bad. Furthermore, the concentration of individual component compounds (or even concentration of total volatile organics) may not predict the level of odor potential.

Electronic Nose

A device called an electronic nose (E-nose) has recently been developed that holds promise for quantifying odor. The purpose of the device is to mimic the operation of the human nose. The electronic nose consists of three functional components.[297] The key component is an array of gas sensors that respond to volatile organic compounds (VOCs). Various types of sensors have been used in E-noses including metal oxide, conducting polymer, quartz crystal microbalance (QCM), surface acoustic wave (SAW), MOSFETs, and optical sensors. Next is the sample handler, a unit that transports the odorant from a sample collection device to the sensor array. Last, the signal processing system accepts the sensor array response waveforms for analysis. Signal processing may involve pattern recognition using artificial neural networks (ANN), principal component analysis (PCA), cluster analysis, and discriminant function analysis (DFA). The output of the electronic nose can be the identity of the odorant, an estimate of the concentration of the odorant, or the characteristic properties of the odor as might be perceived by a human sniffing the odorant. A drawback to current E-nose models, however, is that they are sensitive only in the high ppb or ppm range while the human nose has exquisite sensitivity in the ppt range.

Other Methods for Assessing Odorous Emissions

Measurements of the number of particulates (as well as their odor quality) before, during, and after treatments can also be obtained in order to evaluate the amount of odor carried on particles (dust) compared to that carried in gaseous form. Dust can be collected simultaneously on the farmer's property and on the neighbor's property using Andersen Non-Viable Eight-Stage Impactor Kits or other such devices. These dust samples can be dissolved in water or other diluent (e.g., just as dust dissolves in mucus) and evaluated for odor by the trained panel using static olfactometry. Any odors from dust on the farmer's property may be compared to odors from dust on the neighboring property to determine if they come from the same source. There are numerous designs for particle samplers including High Volume samplers (HiVol) which collect all the fine particles but only part of the coarse particles and the Wide Area Aerosol Classifier (WRAC) which collects the entire coarse mode.[183] Light scattering techniques (e.g., integrating nephelometer) are also used to sample fine particles.[26]

Levels of marker compounds such ammonia and hydrogen sulfide can also be obtained at the houses, lagoon, property line, and at the neighbor's home. However, correlations between odor intensity and levels of hydrogen sulfide or ammonia have been inconsistent.[78]

MANAGEMENT OF ODOR EMISSIONS

Workshop participants determined that many health complaints associated with odorous emissions could be reduced or eliminated by use of odor remediation techniques. Odorous emissions, regardless of the source, often involve a complex set of biological and physical parameters. Research has shown, however, that it is possible to manage or mitigate odor emissions by a variety of approaches. Management practices at the odor source can often control odor to acceptable levels. In addition, various technology applications are available that can reduce the concentration of odor and/or improve its hedonic tone or "acceptability." However, no odor abatement system, regardless of how advanced the technology, will operate efficiently without proper maintenance and management. Methods and technologies to control odor emissions include facility planning and siting of odor emitting operations.

There is currently much focus on odor emissions from animal operations. Odors generally originate from three points in an animal operation:

1. The production facility itself: When manure is allowed to collect on confinement floors, anaerobic conditions soon predominate and decomposition soon begins.

2. The waste treatment system: Anaerobic lagoons, even under the best of management, will produce some amounts of organic acids and reduced sulfur compounds and will be a source of odor.
3. Land application operations: Final disposal of the treated liquid involves application of the liquid to crop land. Whether this is by surface application or spray irrigation, the result is often release of offensive odors.

Specific technology applications to mitigate odor from animal operations include dietary manipulations, windbreak walls, wet scrubber walls, biofilters, solids separators, anaerobic treatment systems, aerated lagoons, aerobic upflow biofilters, activated sludge systems, sequencing batch reactors, ozonation, and various product additives that can be incorporated into waste treatment, handling or storage systems.[298] Each of these applications has advantages and disadvantages depending on technical, economic, social, and political issues that also influence odor mitigation approaches.

In addition to animal operations, compost facilities are under increasing pressure to address odor emissions. Organic materials composted at such facilities include wastewater treatment residuals (biosolids/sludge), yard waste (grass, leaves, and brush), pre-consumer food wastes (restaurant and grocery store vegetables and fruits), food processing wastes (fruits, vegetables, sludges), animal wastes (manures and carcasses), municipal solid wastes (separated or unseparated), and industrial organics. Odor emissions have been a factor in closure of several expensive compost facilities and are a significant obstacle to implementation of composting as a waste management option in a number of locations.

Central to addressing odor emission issues will be requirements for (1) objective science-based defining of the health and environmental effects of odors emissions, (2) the development of national standardized protocols for measuring odor, (3) the development of portable, and durable technologies/methodologies for rapid odor measurement that highly correlates to sensory perception, (4) establishment of science-based and achievable performance standards relative to odor emissions, and (5) development of cost-effective technologies that enable odor emitting industries (animal operations and others) to meet these performance standards.

Options for Addressing Odor Emissions from Animal Operations

Dietary Manipulations

The reduction of nutrients in animal excreta or alteration of the microbial population in an animal's digestive tract as a result of manipulation of the diet or from adding specific odor-reducing materials to the diet may have a positive impact on odor management.[299,300] Nutrients such as nitrogen[301,302] as well as copper and zinc[303] can be reduced through dietary manipulation without impacting the growth performance and health of the animal. This alone is a positive impact on environmental parameters. Odor control through dietary manipulation holds much promise and may revolutionize animal feeding practices within the next few years. Experimental data suggest that dietary manipulations may reduce odor intensity by up to 16%, irritation intensity up to 31%, and improve odor quality by up to 14%.[304]

Windbreak Walls

Walls erected downwind from the fans that exhaust air from livestock buildings provide some blockage of the fan airflow in the horizontal direction and reduce the forward momentum of airflow from the fans. This process may reduce the amount of odorous dust that is transported off the farm, but primarily affects odorous plumes by enhancing dispersion.[305] That is, the airflow from the fans is dispersed upward by windbreaks so that the odorous airflow becomes more dilute when leaving the farmstead and downwind. Objective measures suggest that windbreak walls may reduce irritation leeward of the walls by up to 92%.[305] Several researchers believe that measurement of the impact of windbreak walls on airflow and the dust and odor levels in the airflow at the wall location should be incorporated into dispersion models to predict the downwind impacts of those emissions. Operating cost of structurally sound windbreak walls is relatively low. Installation of windbreak walls is estimated to cost as little as $1.00 per finishing pig space in a building. Windbreaks have been installed downwind of tunnel-ventilated swine and/or poultry buildings in North Carolina, Georgia, Missouri, North Dakota, China, and Taiwan for odor and dust control. The success of windbreak walls in some parts of the world along with the relatively low operating cost of windbreak walls are expected to stimulate further experimentation with airflow deflection devices.

Washing Walls or Wet Scrubbers

Using water to scrub odorous dust and ammonia from the airflow from animal building ventilation fans can be an effective method of controlling odor. A wet scrubber design using an evaporative cooling pad installed in an indoor wall has been tested in North Carolina.[306] Measurements show that the system removes more than 60 percent of the dust at low (cool weather) ventilation rates but less than 20% of the dust at medium to high ventilation rates. This produced a 17% reduction in odor, an 18% reduction irritation, and an 8% improvement of odor quality at high ventilation rates.[306] As expected, the dust was found to carry odorous compounds; therefore, dust removal should reduce odors downwind. The system also reduced ammonia levels in the ventilation airflow by 50 percent at a low ventilation rate.

Wet scrubber wall installation costs were approximately $5.70 per finishing pig space for an 880-head finishing building. The main operating cost was the 1 hp water pump, which will have an annual cost of about $600. Most of the water is recycled, so water usage is very low. The system is beneficial in that it provides some removal of odorous dust and ammonia without imposing a significant airflow restriction on the building fans, unlike industrial air filters and scrubbers. However, higher cleaning efficiencies will presumably be needed for effective odor and dust control in warm weather.

Biofilters

Biofilters may also be used to treat ventilation airflow moving through and out of animal buildings.[307] Biofilters provide a medium for the growth of bacteria or other microbes that convert odorous compounds in the air to more benign products such as water, carbon dioxide and minerals. Air is forced through a biofilter at a slow enough rate that the odorous molecules are absorbed into the media on which the microbes are growing, and the microbes then metabolize the carbon substrate. Substances such as moist compost and wood chips serve as media in biofilters. Periodic moistening of the media is essential. Although they are widely regarded as an effective, low-cost method of cleaning industrial airflows, biofilters are considered an expensive odor control method for animal operations in some parts of the U.S. For example, biofilters properly sized for high summer ventilation rates (required in the Southeast) would be extremely expensive. Since biofilters work best with very odorous air (rather than the more dilute odorous air typical of high summer ventilation rates), biofilters can be used as a cool weather system, with a different system for treating odorous air in warm weather. In one study, biofilters reduced odor by 95%.[307]

Covers for Manure Collection and Treatment Structures

The storage structures that waste management systems use to collect and hold manure can be an odor source. These structures may be used for temporary storage of manure and wastewater until the contents can be spread on land or processed further. In North Carolina, the predominant collection and holding structure is the earthen lagoon, which is designed for biological treatment and sometimes for biogas collection. Covering such structures can reduce odor and gas releases as well as reduce wind-induced volatilization of gases and odor. In one study, Cheng[308] reported that covers reduced odor intensity and irritation by 71% and 91%, respectively.

Covers may be geomembranes such as high-density polyethylene or reinforced polypropylene materials. Such covers may float on the liquid surface or they can be supported above the liquid, which requires extensive structural installation. Geomembranes are costly, especially when supported above the liquid. Covers can be advantageous from a rainwater exclusion standpoint, but floating covers must have a reliable means of removing the rainwater from the cover or else the cover can sink below the wastewater level. Membranes exposed to the sun's ultraviolet rays tend to deteriorate and become brittle after a few years. Covers less than 20 mils thick have generally been unsuccessful because of sunlight blistering, which produces holes in the cover, or because of gas pockets under the cover, which can lead to wind-induced ruptures and tears. Covers today generally have a thickness in the range of 40-60 mils. Geomembrane covers are sometimes used on larger surface area treatment lagoons to capture biogas, which is then used as an alternative fuel. Because of the large surface area of treatment lagoons, such covers are considered costly.

Biocovers, floating layers of slowly biodegradable materials, may also be used to cover manure storage structures and may be made of chopped barley, wheat, flax, brome straw, corn stalks or peat moss. Such covers serve to either limit the volatilization of gases and odors from the surface of the stored contents or to filter these gases, reducing their odor levels. However, the cover materials tend to become waterlogged and sink to the bottom of the storage tank and must be replaced every 4-6 months. When these materials sink, the rate of solids build-up tends to be increased, and it is much more difficult to pump manure solids and sludge from the structure.

Anaerobic Digesters

Anaerobic digesters are generally in the form of enclosures designed to be operated in the mesophilic temperature range (20 to 44°C) or in the thermophilic range (45 to 60°C). Some digesters systems operate at ambient temperature and may be comprised of a covered anaerobic earthen lagoon. An example of such a system is the EPA AgStar System.[308] Within each system, organic material is stabilized, and gaseous by-products, primarily methane and carbon dioxide, are formed. Considerable research has been devoted to recovery and reuse of biogas generated by anaerobic digesters as well as to the odor abatement potential of these systems; however, economics, equipment maintenance costs, erratic biogas production and increased managerial skill requirements have limited the adoption of this technology for manure utilization.

Solids Separation

The separation of the solid and liquid portions of the waste stream from animal housing buildings, known as solids separation, can reduce odor from lagoons by decreasing the organic load being treated by the lagoon. In the past, solid-liquid separation has been used to improve manure handling characteristics and for generation of solids for various purposes but has recently been investigated as a means of odor reduction and nutrient management.[309] Separation of the manure into solid and liquid fractions not only produces a nutrient-rich solid material suitable for composting or land application, but also allows lower organic loading to subsequent treatment systems. Solid-liquid separation can typically remove 50-80% of the suspended matter in manure streams. If the solid material is properly handled and not allowed to undergo anaerobic degradation, offensive odor can be avoided. If the same treatment practices are applied to raw and liquid manure, the lower organic load of the liquid manure produces lower levels of odor-causing compounds.[309] However, the goals of odor reduction and nutrient management may not always be met by the same process. Liquid manure from a solid-liquid separation operation will have a reduced organic load. Aerobic treatment in this case may result in significant nitrate without sufficient carbon to support denitrification. The two processes must be carefully designed to balance all goals of the system. Solids separation was reported to reduce odor by 20-30% in one study.[309]

Composting

Composting is the biological decomposition of organic material under controlled, aerobic, thermophilic conditions into a humus-like stable end product. The composting process has long been used on farms and nonagricultural industries to manage wastes such as municipal wastewater treatment plant biosolids. However, even though it is an aerobic process, a composting operation can generate significant odor. Crawford[310] classified the odors at compost facilities as related to the original substrates, as produced during the composting process, and as produced during the final processing steps. Inorganic compounds of concern include ammonia and hydrogen sulfide, and organic compounds are typically low-molecular weight organic acids, mercaptans, and amines. A comprehensive review of odor compounds associated with composting has been published by Miller.[311]

Odors at compost facilities arise from a variety of sources and locations around the site. In open-air facilities, these sources could be considered as area sources due to the size of the facilities. With enclosed facilities, it is possible to create one or more point sources, depending on the method of odor control. The strength and character of the compost-generated odors is a function of input feedstocks, method of composting (windrow versus aerated static pile), temperature of the compost pile (temperatures above 60°C generate a particularly malodorous smell), age of the compost pile, and C:N ratio of the pile.

Walker[312] described approaches to controlling compost odors by operational techniques. In general, these approaches are summarized as follows:

- Maintain good housekeeping practices
- Mix materials as rapidly and thoroughly as possible
- Maintain aerobic conditions in piles
- Keep temperatures below 60°C
- Avoid ponding of water on-site
- Avoid stockpiling of large amounts of material
- Maintain odor control devices

Croteau et al.[313] evaluated the changes in odor generation at a biosolids and yard waste compost facility in Washington State. This facility was undergoing severe odor problems and switched from a windrow to a static pile method of composting. In this case, a 63 percent reduction in odor generation was achieved by operational changes.

Cerenzio[314] described how a compost facility in New Jersey overcame their odor problems by increasing aeration, enclosing the facility, maintaining better temperature control, and improving compost mix. Alix[315] discussed modifications made to a facility in western Massachusetts, which also overcame odor problems and gained public acceptance by covering their compost operation and scrubbing the off-gases through a biofilter.

Williams[316] described the main methods utilized to scrub compost odors from point sources. The two most widely utilized methods include chemical wet scrubbers and biofilters. In general, odors from compost facilities are difficult to treat because they exhibit the following characteristics:

- 100 percent relatively humidity
- Low energy value
- A complex mixture of nitrogen and sulfur compounds
- Above 40°C
- High levels of ammonia

To overcome the problems inherent in treating compost facilities off-gases, a number of improvements in standard scrubbing treatment methods have been made. Ostojic and O'Brien,[317] Van Durme et al.,[318] Hentz et al.,[319] Thompson et al.,[320] and Muirhead et al.,[321] all describe modifications made to wet scrubbers to treat compost off-gases. Table 2 illustrates the odor removal efficiency experienced at compost facilities that utilize wet chemical scrubber systems. Dunson[322] gives a good description of the control of odors by physical-chemical approaches.

Amirhor and Kuter,[323] Wheeler,[324] E&A Environmental Consultants,[325] Giggey et al.,[326] Ostojic and O'Brien,[317] Kuter et al.,[327] and Boyette,[328] to cite just a few, report on the performance of biofilters in controlling compost odors. Biofilters, described above, work by absorbing the odorous compounds in a water film surrounding organic matter and having the compounds biologically degraded, in contrast to chemical scrubbers, which utilize chemical reactions to neutralize and, thereby, remove odorous compounds. The challenges of biofiltration in treating compost off-gases include removal of excess ammonia, which can interfere with the biological processes, cooling the input air to below 40°C, and reducing the size of the biofilters. Table 3 shows the odor removal efficiency of biofilters at compost facilities. It should be noted here that complaints from nearby residents can still occur with 99% removal of VOCs. That is, 99% removal may not be adequate to eliminate odor complaints.

TABLE 2. Data on odor removal from several wet scrubber installations at composting facilities.[316]

Facility and reference	Date of Test	D/T Inlet		D/T Outlet		Odor Removal %		TRS Removal %	VOC Removal %
		Range	Avg.	Range	Avg.	Range	Avg.		
Akron, OH[317]	3/93, 8/93	53-338	180	12-85	47	55-85	74	–	–
Hamilton, OH[317]	9/91	158-289	223	84-158	127	0-47	31	–	–
Hampton Roads, VA[318]	6/90	–	1,700	–	200	–	88	–	–
Lancaster, PA[317]	9/88	130-380	–	60-140	–	55-67	–	–	–
Montgomery Co, MD[317]	1/92	175-315	230	52-94	63	67-76	72	–	–
Montgomery Co, MD[319]	n/a	–	–	–	–	80-90	–	–	–
Montgomery Co, MD[320]	10/93-1/94	–	–	–	–	–	–	87	90
Schenectady, NY[317]	7/90	480-860	660	110-200	150	70-83	77	–	–
Schenectady, NY[321]	9/90	–	558	–	21	–	96	–	–

D/T = Dilutions to Threshold n/a = Not Available TRS = Total Reduced Sulfur
VOC = Total Volatile Organic Compounds

TABLE 3. Data on odor and VOC removal from several biofilter installations at composting facilities.[316]

Facility and reference	Date of Test	D/T Inlet		D/T Outlet		Odor Removal		TRS Removal %	VOC Removal %
		Range	Avg	Range	Avg	Range	Avg		
Dartmouth, MA[323]	5/93, 12/93	–	–	–	–	76-97	86	81	-
Hamilton, OH[324]	9/91-3/92	180-1,200	635	5-25	19	–	97	99	99
Williamstown, MA[325]	9/93	–	–	–	–	–	95	99	52
Lewiston-Auburn, ME[317,326]	9/93	71-158	115	7-11	8	90-94	93	–	-
Plymouth, NH[327]	4/92	170-318	227	<10-35	23	79-96	90	–	-
Sevier, TN[325]	11/93	–	1,020	–	22	–	99	93	82
Yarmouth, MA[326]	4/93	143-262	214	4-26	12	88-98	95	>90	-

D/T = Dilutions to Threshold TRS = Total Reduced Sulfur VOC = Total Volatile Organic Compounds

Other methods utilized at compost facilities include use of neutralizing sprays at the periphery of the site. These sprays are claimed to neutralize the malodorous compounds or mask them with a pleasant scent, such as pine or citrus, but have had very limited successes, depending on the complexity of the odors produced, the strength of the odors, and the proximity of neighbors. Most applications have not found neutralizing sprays to be effective. The use of carbon filters and thermal oxidizers has not been particularly successful due to the high moisture content of the off-gases and the low heat value.

Aeration

Waste treatment systems that utilize aerobic conditions can be effective in controlling odors. Although the energy cost of aerobic treatment is often cited as a deterrent to its use, these costs must be weighed against costs of providing some other treatment if a particular farm or facility is under pressure to reduce odors.

Complete aerobic treatment not only stabilizes the organic carbon of the waste stream but it also converts organic nitrogen compounds to ammonium and then to nitrite and nitrate. Sulfur compounds are converted to odorless sulfate instead of odorous sulfide and mercaptan compounds.[329] The recommended aeration capacity for such a system is twice the daily biochemical oxygen demand (BOD) load.[329] However, providing the oxygen necessary to maintain this level of aerobic activity can be expensive with current aeration equipment. This has led to research into partial aeration of various schemes to lower the cost of the system while providing some level of odor control.

Partial aeration has been studied by several investigators.[330-332] This research showed that supplying oxygen such that the oxidation-reduction potential (ORP) is controlled between 100 and 200 mV E_h, where dissolved oxygen cannot be detected, can still provide significant odor reduction during treatment. Volatile fatty acids and other odor-causing compounds were not released from these treatment systems. However, some level of odor returned if wastes were applied to land or stored without aeration after only this minimal treatment. If the organic matter is not stabilized when aeration ceases, anaerobic degradation will occur and odorous compounds will be produced and released.[329] Partial aeration can also be used to provide more complete treatment of wastewater, including nitrogen and phosphorus removal. Westerman and Zhang[329] found that aeration could reduce irritation by up to 55%.

Aerobic Upflow Biofilters/Activated Sludge, Extended Aeration

As noted above, aeration is an effective method of reducing odor from manure or wastewater. Aerobic treatment of manure reduces or prevents the accumulation of volatile fatty acids and various other odorous compounds. Supplying oxygen to waste substrates generally requires considerable energy and is, therefore, expensive. If complete stabilization of the waste is desired, then the oxygenation capacity should be twice the total daily biochemical oxygen demand (BOD) of the waste with a hydraulic retention time of several days. Using a swine facility as an example and electrical energy cost of $0.07 per kilowatt hour, the power cost for running an aeration system to treat the liquid manure continuously is about $11 per year per finishing pig space (each space will grow approximately 2.6 pigs per year). Westerman and colleagues[333] found that the odor and irritation intensities were reduced by up to 75% and 86%, respectively. If partial odor control is desired, then the oxygen supplied could be less than twice the total daily BOD loading. For example, some odor reduction can be accomplished by supplying about a third of the BOD loading. This would cost about $1.80 per year per finishing pig space. However, aeration to supply only partial BOD removal could result in promoting ammonia volatilization, which may be an undesirable tradeoff. If nitrification/denitrification is also desired for reducing nitrogen (by releasing nitrogen gas to the air), then additional aeration above twice the BOD may be required.

Besides different methods to supply oxygen to the wastewater, there are various methods to promote retention of the bacteria responsible for waste treatment. Generally, these methods may be described as suspended media or fixed media. Examples of these two methods are an activated sludge treatment using recycled solids as a suspended media and a biofilter using fixed media to retain bacteria.

The activated sludge system has typically been used for municipal waste for complete stabilization, and thus would tend to have high energy costs for supplying twice the BOD loading. The biofilter system could be designed to satisfy all of the BOD or only part of the BOD, depending on the objectives. The operating costs and the odor of the effluent would depend on what degree of treatment is desired, and the energy costs would probably fall between the $1.80 and $11 per year per finishing pig space depending on degree of treatment (using the assumed energy cost of $0.07 per kilowatt hour). It should be noted that either system would likely require screening or removing the larger solids in the manure before the aeration treatment and would also produce biosolids from the treatment system. Both of these byproducts would tend to have more odor than the liquid discharged from the treatment system and would likely require more treatment, such as lime stabilization to reduce odor.

Sequencing Batch Reactors (SBR)

Sequencing batch reactors (SBRs) have the potential to stabilize organic matter and reduce nitrogen from swine production effluent effectively and inexpensively.[334] The sequence of batch operations in these reactors can be adjusted to suit the needs of the type of wastewater being treated. As applied to swine wastewater, the cycles include fill, react, settle, decant, and idle. The react cycle is the time during which waste is stabilized and nutrients are transformed and consumed. Nitrogen and phosphorus removal is accomplished by cycling the reactor between aerated and anoxic states during this period. Several researchers have investigated this system for swine wastewater treatment with good results.[335,336,337] There is considerable variability in the reduction of odor (35-89%) and irritation (39-99%) that has been reported.

Ozonation

Ozone, a triatomic allotrope of oxygen has a large oxidation potential and has been widely investigated for its potential to improve air quality. Ozone has also been used as a disinfectant and deodorizing agent. Laboratory and field evaluations of ozone treatments to reduce livestock odors have been conducted or are ongoing.[338,339] However, due to the toxic nature of ozone, there is some concern regarding its use to treat indoor air spaces. Several professional groups including the Occupational Safety and Health Administration and the American Lung Association have expressed concern that the levels of ozone required to effectively deodorize polluted indoor air often exceed recommended or permissible exposure limits for humans. There do not appear to be major objections to ozonating lagoon water from a human health standpoint, but health concerns with indoor ozone are likely to cause health and safety regulators to address lagoon ozonation as well. Nevertheless, the relatively high indoor odorant levels in some livestock buildings and the potential for ozone to be rapidly depleted, thus minimizing ozone emissions to outdoor air, continue to make ozonation of indoor air an attractive but somewhat controversial possibility.

Product Additives

Product additives are generally described as compounds that can be added directly to freshly excreted or stored manure for purposes of odor abatement. There are hundreds of chemical and biological additives, masking agents and other commercial products that are being marketed to animal producers for odor management. In addition to odor management, many of these products are marketed as having other beneficial effects, including management of ammonia and hydrogen sulfide volatilization from stored manure; improved fertilizer value of the manure; fly control; improved animal health and feed conversion; and promotion of manure solids breakdown to enhance manure management and handling. Regarding odor abatement, these products can generally be grouped into several categories based on their mechanism of action.

- Masking Agents. These are mixtures of aromatic oils that have a strong characteristic odor of their own. They are designed to cover up, or mask, the targeted undesirable odor with a more desirable one;
- Counteractants. These are mixtures of aromatic oils that cancel or neutralize the targeted odor such that the intensity of the mixture is less than that of the constituents;
- Digestive Deodorants. These contain bacteria or enzymes that reduce undesirable odors through biochemical metabolic degradative processes;
- Adsorbents. These products have a large surface area that may be used to adsorb targeted odors before they are released, or volatilized, to the environment;
- Feed Additives. These are compounds incorporated into the animal's diet to improve animal performance and reduce targeted odors;
- Chemical Deodorants. These are strong oxidizing agents or germicides that alter or eliminate microbial action responsible for odor production or chemically oxidize compounds that make up the undesirable odor mixture.

During the past 2 years, approximately 2 dozen of these product types have been evaluated by the NC State University Animal and Poultry Waste Management Center.[340] In general, only a few of the products significantly improved odor parameters under the conditions tested.

Far more peer-reviewed research on management of odor emissions is necessary before conclusions about the efficacy of odor interventions can be made with certainty.

FINAL COMMENTS

Our current state of knowledge clearly suggests that it is possible for odorous emissions from animal operations, wastewater treatment, and recycling of biosolids to have an impact on physical health. The most frequently reported symptoms attributed to odors include eye, nose, and throat irritation, headache, nausea, hoarseness, cough, nasal congestion, palpitations, shortness of breath, stress, drowsiness, and alterations in mood. Many of these symptoms (especially irritation, headache, hoarseness, cough, nasal congestion, and shortness of breath) can be caused by stimulation of the trigeminal nerve in the nose at elevated levels of odorous VOCs. Co-pollutants in an odorous plume may also play a role. A genetic basis for some odor aversions may be the basis for complaints from unpleasant but nonirritating odors; unpleasant odors have been shown to activate different brain areas than pleasant ones.

Most published studies indicate that there are occupational health risks to workers in intensive livestock units who are exposed continuously to high concentrations of odorous VOCs, particulates, and microbes. However, more scientific data are necessary to quantify health symptoms from the types of exposures experienced by neighbors downwind of livestock or wastewater operations (e.g., continuous exposure to the lower levels of odorous emissions or intermittent exposures to high levels from temporary discharges). Objective scientific data must be obtained that relate specific concentrations of VOCs, particulates (including ammonium aerosols), and microorganisms alone and in combination to objective measures of health symptoms.

There are many potential study tools and biomarkers for the validation of odor-related health symptoms in clinical, epidemiologic, and research studies (see Table 1). These tools and biomarkers will be helpful in distinguishing between direct health effects (e.g., sensory irritation) and indirect effects (e.g., stress). Objective measures of health effects must then be related to the concentrations of odorous emissions as well as frequency and duration of exposure. A variety of methods are available to quantify odorous emissions including olfactometry, gas chromatography, and the electronic nose. However, there is still a need to develop portable, reliable, and sensitive sensors for field measurement of odorous emissions in real time.

Future studies will help establish minimal risk levels (MRLs) for odorous emissions analogous to those utilized by the Agency for Toxic Substances and Disease Registry (ATSDR), that is, substance-specific minimal risk levels (MRLs) to evaluate health effects. MRLs are defined as "estimates of daily human exposure to a chemical that are likely to be without an appreciable risk of adverse noncancer health effects over a specified duration of exposure." In addition, knowledge of MRLs for odorous emission will assist in the development and implementation of cost-effective odor-abatement techniques that will enable operators of livestock and wastewater operations to meet performance standards.

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The workshop participants included experts in diverse fields including medicine, organic chemistry, biochemistry, psychology, engineering, biology, and other disciplines. Academic institutions, regulatory agencies, environmental groups, the swine industry, consulting firms, and odor remediation companies were represented. During the two day conference, formal presentations were made on a variety of topics including odor issues in North Carolina (Ron Levine, MD, Deputy Secretary, North Carolina Department of Health and Human Services), basic olfactory physiology (Donald Leopold, MD, Department of Otolaryngology, University of Nebraska Medical School), health symptoms from odors (Dennis Shusterman, MD, Division of Occupational Medicine, University of California, San Francisco), changes in the olfactory epithelium from VOCs (Kevin Morgan, PhD, Glaxo-Wellcome), effect of odors on the brain (Tyler Lorig, PhD, Washington & Lee University), psychoimmune aspects of asthma (Maria Boccia, PhD, University of North Carolina), psychological effects of odors (Pam Dalton, PhD, Monell Chemical Senses Center), and health effects of dust (Kelley Donham, DVM, Institute of Agricultural Medicine & Occupational Health, University of Iowa). The participants then assembled into five working groups devoted to the following topics: medical issues, irritation, dust, toxicology, odor measurement, psychology, odor management, and legal issues. The remainder of the workshop was spent in these working groups in which participants discussed and integrated information on each topic. The purpose of this paper is to provide a summary of some of the issues that were addressed at the workshop.

The workshop participants were Dr. Joel Alpert, E & A Environmental Consultants, Inc.; Dr. Carol M. Baldwin, Veterans Affairs Medical Center, Tucson, AZ; Dr. Andy Baumert, National Pork Producers Council; Dr. Maria L. Boccia, University of North Carolina; Dr. Robert W. Bottcher, North Carolina State University; Dr. C. E. Buckley, III, Duke University Medical Center; Dr. Dwaine S. Bundy, Iowa State University; Mr. M. Steve Cavanaugh, Jr., Cavanaugh & Associates, Inc.; Dr. John Classen, North Carolina State University; Dr. Bill

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The leaders of the breakout groups were Dr. Tyler Lorig, Dr. Pamela Dalton, Dr. C. M. Williams, Dr. Dennis Shusterman, and Dr. Jim Raymer.

The organizers of the workshop were Dr. Susan S. Schiffman, Duke

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