



AGRICULTURAL RESPIRATORY HAZARDS AND DISEASE

A PRIMER FOR WISCONSIN PRACTITIONERS AND HEALTH/SAFETY PROFESSIONALS

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INTRODUCTION

The agriculture workplace has long been known to be associated with respiratory disease. Respiratory disease is among the main chronic health conditions affecting farmers (Brackbill et al, 1994). Those who are at potential risk include farmers and farm families, agricultural workers, abattoir workers, greenhouse and nursery workers, veterinarians, and grain elevator workers. While the massive exposures leading to severe acute disease have decreased, it is postulated that there has been a significant increase in subacute and chronic respiratory disease resulting from increased indoor air exposure (Donham 2000; Von Essen and Donham, 1999). Animal confinement workers or dairy technicians may spend as much time as 40-50 hours or more a week indoors in larger operations, resulting in longer exposures to higher levels of gases and dusts. This is a result from changing animal production techniques with higher animal densities and shift work in animal feeding units (AFOs) and high density concentrated animal feeding operations (CAFOs). Many of these conditions are found in Wisconsin. In fact, some of the early work in establishing the methods in the diagnosis of Farmer's Hypersensitivity Pneumonitis (FHP), formerly known as Farmer's Lung Disease, and Organic Dust Toxic Syndrome (ODTS) was performed in Wisconsin at the Marshfield Clinic. There are a number of common exposures that will lead to respiratory illnesses, often with overlapping clinical signs and symptoms (**See Table 1**). These include organic dusts, molds, bacteria, and gases from fermentation of silage and manure. Other respiratory hazards include inorganic dusts, pesticides, and other agricultural chemicals. There are also infectious respiratory conditions that are not unique to agriculture but may be encountered in the work and living environments associated with farm families See Table 1 for a compendium of agricultural respiratory diseases.

This chapter will provide an overview of the agricultural respiratory hazards that can be commonly encountered in Wisconsin, as well as those respiratory conditions that are more likely to be encountered by agricultural workers, farmers and their families. Rural providers are on the front lines and are in an excellent position to decrease morbidity and disability resulting from acute and chronic exposure to agricultural respiratory toxins. Prevention of unnecessary exposures and the early accurate



diagnosis of respiratory disease resulting from the agricultural environment is best performed initially in the primary care clinic, rather than the tertiary referral center. Unfortunately, it is recognized that rural primary care health care providers often do not have the training to provide this method of preventive health care (Hartye, 1990). The focus of this paper will be to provide some of the tools for rural health care providers to recognize and accurately diagnose and treat respiratory conditions resulting from agricultural exposures utilizing an accurate occupational and environmental history and appropriate differential diagnosis. The occupational and environmental respiratory toxins and sources of exposures, pathophysiology, diagnosis and treatment, and prevention of the of the resulting respiratory clinical conditions will be discussed.

LEARNING OBJECTIVES

At the completion of this chapter, the health care provider will be able to:

Describe five common agricultural respiratory toxins and their sources.

Identify four respiratory conditions associated with agricultural dusts and gases and the differential diagnosis of those conditions, particularly the difference between Farmer's Hypersensitivity Pneumonitis (farmer's lung) and organic dust toxic syndrome.

Describe the basics of clinical evaluation and treatment of the common agricultural respiratory conditions.

Identify three preventive strategies to decrease exposure to agricultural respiratory toxins. Describe which personal respirators are appropriate for agricultural dusts, gases, and immediately dangerous to life and health conditions.

Agricultural Exposures from a Wisconsin Perspective

It is helpful the health professional to have an understanding of the type of agricultural operations that occur in their region in order to anticipate the potential significant respiratory hazards that may occur in the farmers and farm family that present to the clinic. The various agricultural commodities, whether crop or animal, have unique associated respiratory hazards and work practices that affect the exposure. Each county's extension health/safety educator or University of Wisconsin are excellent sources of information in providing this information.

According to the most recent statistics available from 2000, the Wisconsin Agricultural Statistics Service, lists Wisconsin as a leading agricultural producing state ranking 10th nationally in overall production (USDA Agricultural Statistics Services, 2001). Wisconsin ranks in the top five nationally in dairy, corn for silage, cabbage, sweet corn for fresh market (**See Table 2**). Wisconsin is also a leading state in the production of cranberries, turkeys and ducks, forage, ginseng, and livestock. Hog production is not as significant in Wisconsin as other agricultural states but it does occur and ranks #18 nationally. The top five commodities grown in Wisconsin in descending order are dairy, cattle and calves, corn, potatoes, and soybeans. (**See Table 3**) Producing and processing these different



commodities result in varying exposures to the respiratory tract and include organic dusts, gases, microorganisms, infectious disease, and agricultural chemicals, including pesticides. Farm sites and work practices that are associated with respiratory toxins include barns, chicken coops, silos, grain bins, applying pesticides to crops and vegetables, and manure storage pits. If the clinician has an understanding of the type of agricultural commodity that their patient produces or works and lives around, the appropriate screening and diagnostic questions can be asked. This will lead to the correct diagnosis and prevention of recurrence and progression of the disease.

It is important to understand that agricultural work is undergoing significant change as a result of biotechnological advances and respiratory hazards can continue to evolve over time. The size of a farm will also affect the respiratory hazards. Presently the majority of Wisconsin farms ranges in size from 50 to 499 acres and are predominately family owned (USDA Agricultural Statistics Service, 2001). Because of the small size of the farms and the small number of employees, Occupational Health and Safety Administration (OSHA) health and safety regulations are not applied or enforced. As farms become larger and/or corporate owned, such as dairy farms that employ over 11 or more employees with shift work, OSHA regulations may extend to the farm site and help decrease unnecessary exposure to respiratory toxins. Until that time occurs, the rural health care provider can play an important role in the recognition and prevention of agricultural respiratory disease.

AGRICULTURAL RESPIRATORY HAZARDS

DUSTS

Organic Dusts

Due to the nature of Wisconsin farming, organic dusts are the most common cause of agricultural respiratory disease on most Wisconsin farms. Silos, dairy and poultry barns, and grain bins are sources of high levels of organic dusts. Organic dust is a complex mixture of vegetable matter, pollens, animal dander, insect, rodent and bird feces, feathers, microorganisms, bacterial and fungal cell wall toxins, pesticides, and antibiotics and can be thought of as a chemical soup. These components lead to an inflammatory response in the mucous membranes and respiratory tract. The components can lead to simple inflammation or an IgE-mediated immune response to allergens contained in the dust. Allergens include animal products, antibiotics and animal feed additives, pollens, storage mites, fungal and bacterial molds, and protein components of grain dusts. **Bacterial sources**, particularly thermophilic **actinomycetes** such as *Saccharaopolyspora rectivirgula*, and **fungal molds**, particularly members of *Aspergillus* genus, are associated with sensitization leading to hypersensitivity pneumonitis. Allergic conditions can include upper airway allergic symptoms such as rhinitis, as well as asthma.

The levels of molds and bacteria are can be extremely high, particularly in moldy bedding, feed, and silage. High levels of dusts and molds are associated with particular activities such as unloading grain bins, and silo unloading and uncapping in the fall (**See Table 4**). Aerosols that are inhaled while working in these areas contain in the range of 10^4 to 10^7 bacterial colony forming units/ cubic meter (cfu/m^3) and 10^3 to 10^6 fungal cfu/m^3 . The size of these particles is also important and range from less than 0.1 microns to 100 microns. **Respirable dust particles**, or those particles that are 5.0 microns (μ) in diameter or smaller, make up 40% of the organic dust and penetrate deeply into the air exchange



unit consisting of the terminal bronchioles and alveoli. Respirable particles primarily damage the lower airways and terminal alveolar unit while the larger particles that settle out in the upper airways and are associated with upper airway irritation.

A significant component of grain dust associated with inflammation is bacterial **endotoxin**. This consists of a heat-stable lipopolysaccharide (LPS) found in bacterial cell walls, primarily from gram-negative bacteria, and released with bacterial death and cell wall lysis. LPS contains the biologically active lipid A that is considered to be responsible for the inflammatory effects. Endotoxins are found in dusts associated with agricultural operations found in Wisconsin, including animal confinement operations, livestock farming, grain elevators, and potato processing. Routine measurement of endotoxins are not performed on agricultural operations nor are there regulatory levels set at this time for safe exposure to endotoxins. Research has demonstrated a dose-response effect and deterioration of pulmonary functions have been shown at levels over 100 endotoxin units/m³ (EU/m³) and also with organic dust levels (Schenker, 1998; Donham et al, 2000; Reynolds et al, 1996; Schwartz et al, 1995a; Schwartz et al, 1995b). Other microbial products that are probable sources of inflammation include (1,3) beta-d-glucans from fungal species, exotoxins from gram-positive bacteria, phytotoxins from plants, and T-cell-activating superantigens (Schenker, 1998).

Inorganic Dusts

Inorganic dusts are primarily an issue in field activities associated with plowing, tilling, haying, and harvesting. The newer tractors and combines generally provide adequate respiratory protection because of the air filtration in enclosed cabs, but is dependent upon changing the filtration regularly. Grain handling, manual harvesting of tree fruit and grapes, Christmas tree farms, potato harvesting, and small vegetable harvesting by hand can also cause an exposure to inorganic dust that may be higher than OSHA regulatory levels for nuisance dust (Schenker, 1998). Inorganic dust is much less of an issue in Wisconsin as compared to the Great Plains and the major fruit producing areas. **Silicates**, including primarily the **noncrystalline diatomite silica** but also crystalline silica or quartz, make up the bulk of inorganic dust (Neiuwenhuijsen and Schenker, 1999). Burning stubble, particularly rice stubble, can also expose workers to aerosolized inorganic dust (McCurdy et al, 1996). Inorganic dust is not as significant as organic dust or as toxic as industrial sources of quartz dust. Those individuals with underlying chronic obstructive pulmonary disease, including asthma and chronic bronchitis, can experience aggravation of the underlying disease. Persistent and repetitive exposure to high levels could lead to restrictive lung disease but that would be uncommon in Wisconsin. A common work practice in Wisconsin is hiring both retirees and students to sort potatoes on a conveyor belt during harvest which results in exposure to inorganic dust.

ANIMAL CONFINEMENT GASES

The primary animal confinement gases of human health concern are **hydrogen sulfide (H₂S)** and **ammonia (NH₃)**. Carbon dioxide (CO₂) and methane (CH₄) are also formed and are considered simple asphyxiants and are of secondary concern. CO₂ is produced from animal respiration and is of concern if 5000 ppm or greater. CH₄ may be a risk for explosion at higher concentrations. Bacterial decomposition of animal manure and urine results in the gas production. Hot summer days result in



higher levels of gas production. Under facility manure storage pits and outdoor lagoons contain both high levels of hydrogen sulfide and methane but also oxygen deficient environments at levels immediately dangerous to life and health (IDLH) that are insufficient to support human life. These environments can be toxic to animals and humans. They also are sources of lethal exposures in a farm child's home environment, as well as an occupational exposure. Concentrations of dusts and gases are higher in the winter when ventilation is decreased to save on heating costs.

Hydrogen Sulfide

Hydrogen sulfide (H_2S) is a very toxic **chemical asphyxiant** and has a mechanism similar to cyanide. The primary mechanism is to inhibit the cytochrome oxidase system and interrupt the oxidative phosphorylation process. Paralysis of the respiratory center is the primary lethal toxic effect and results in immediate “knockdown” at high concentrations. H_2S is heavier than air and colorless. It has a very low odor threshold, which results in an unpleasant “rotten egg” odor at 1-3ppm. The toxic effects begin at 100-150 ppm with **paralysis of the olfactory nerve** and inability to detect the smell of H_2S at the higher toxic concentrations. Pulmonary edema can occur after thirty minutes of exposure to 250 ppm. Knockdown occurs at 500+ ppm and respiratory paralysis occurs at 500-1000 ppm. (See **Table 5** for significant levels of H_2S). Generally, the concentrations of H_2S are at low levels and may cause eye or respiratory irritation. Cough, dyspnea, and chest pain may occur at irritant levels. There is significant danger of **lethal levels of hydrogen sulfide when manure is agitated**, which occurs when under barn manure pits are emptied. H_2S , which is normally heavier than air, is carried into the air in gas bubbles and can result in indoor concentrations as high as 1000-1500 ppm. Permanent neurologic damage, including demyelination and basal ganglia damage and ataxia can occur after respiratory arrest if death does not occur. Temporary neurologic effects include hearing, visual, and olfactory loss.

Treatment consists of immediate removal from exposure and removal of contaminated clothes in a manner not to risk other exposures. **Mouth-to-mouth resuscitation is not recommended** (Deng, 2001). Supplemental 100% oxygen and treatment of metabolic acidosis is imperative as is consultation with the Regional Poison Center (See Table 10). Treatment is considered to be similar to cyanide exposures but not as effective. A recommended treatment of **10 ml of a 3% intravenous sodium nitrite infusion injected over 2-4 minutes** is given to induce methemoglobin to scavenge the sulfide (Deng, 2001). Initially amyl nitrate ampoules may be given by inhalation in the Emergency Department if the sodium nitrite infusion is not ready (Deng, 2001; Kerns and Kirk, 1998). Hyperbaric oxygen treatment should be considered in severe poisonings with associated coma and pulmonary edema.

Rescue should be performed only by a person trained in use of a SCBA and with a properly maintained SCBA and a rescue harness and spotters. Family members or co-workers should not enter a manure pit or lagoon to perform a rescue without those precautions or multiple deaths may result. As difficult as it is, the only appropriate course of action is to call 911 and await properly trained and equipped rescue response team. Prevention consists of keeping the manure pit levels below twelve inches of the top of the pit, removal of animals and humans during times of manure agitation and emptying and not entering for 24 hours after emptying. A person should never enter a manure pit or lagoon without a safety harness and line, an additional person readily available, and monitoring of H_2S



and oxygen content if a SCBA is not worn.

Ammonia

Ammonia (NH₃) is a pungent respiratory and mucous membrane irritant with a low odor threshold. It is lighter than air and causes respiratory inflammation. Ammonia is additive with the effects of dusts and can be carried further into the terminal respiratory bronchioles by adsorbing onto respirable dust. It is at least additive and probably synergistic with cigarette smoke. Eye, nose, and throat irritation is common and higher concentrations result in cough and chest pain. Tolerance develops with continued exposure. This will lead to deeper and greater respiratory exposure as deeper breaths will occur when the person adapts to the irritant effects of ammonia. Ammonia does not exist as a single exposure agent but in combination with organic dusts and endotoxin and often results in **sinusitis and chronic bronchitis** after years of exposure. The regulatory Occupational Safety and Health Administration (OSHA) permissible exposure levels (PEL) are 25 ppm but recent research has indicated that respiratory disease can occur after chronic ongoing exposure to concentrations as low as 7 ppm (Donham et al, 2000; Reynolds et al, 1996). Prevention involves proper ventilation and the use of an ammonia specific chemical cartridge respirator and goggles. Gas concentrations can be measured with colorimetric tubes that are available through agricultural safety catalogues.

NONINFECTIOUS RESPIRATORY DISEASES AND SYNDROMES

Many respiratory conditions may initially present as a viral-like syndrome and may be misdiagnosed and treated inappropriately as bacterial sinusitis and bronchitis with antibiotics. **Table 6** lists agriculturally related respiratory conditions that may have an initial presentation similar to viral syndrome or bronchitis. Obtaining a good occupational history and having a high clinical index of suspicion is essential in making the correct diagnosis. The exposure causing the diseases in the following section may be from organic dust or a mixed exposure of dust and animal confinement gases.

Farmer's Hypersensitivity Pneumonitis

"Doc, I think I have Farmer's Lung".

"Well, you farm and have a cough and shortness of breath so you must be right.

You might as well stop farming"

Farmer's Hypersensitivity Pneumonitis (FHP) is what was previously referred to as Farmer's Lung Disease. It is a form of hypersensitivity pneumonitis, or allergic alveolitis, that is specific to sensitization to **thermophilic actinomycetes** (Gram + filamentous bacteria) **or Aspergillus fungal species** found in organic dust in agricultural operations. Occupations at risk include dairy farmers, poultry workers, zoo keepers, and nursery workers. It is more common in the northern temperate regions in the Northeast, North Central, and Great Plains northern states. Typical exposures occur in the early winter through late spring and involve moldy silage, feed, and hay and straw bedding. Due to the existing combination of environmental factors, the predominance of smaller family dairy operations, and the large numbers of dairy cattle found in Wisconsin, FHP is as or more common in Wisconsin than in many of the other agricultural states in the United States. However, it is still not that common and not as common as Organic Dust Toxic Syndrome (ODTS) and is frequently misdiagnosed



due to an inadequate evaluation and incomplete differential diagnosis. It is more likely to be found in dairy operations than hog confinement operations. It is estimated that the prevalence of sensitization to the organism causing FHP is 5-20% based upon seroprevalence. The estimates of the prevalence of actual FHP disease ranges from 1-10% (May and Schenker, 1996). FHP is not as frequently seen as in previous generations due to greater awareness of the disease by farmers and agricultural workers and greater availability of proper respiratory protection.

FHP is considered to be a complex disease characterized by sensitization to the antigenic dust components with elements of both an immunologic cell-mediated and a humoral response. Continued exposure to the antigen will result in both an antigen-antibody Type III immunologic response and a late-phase T-cell mediated response with granuloma formation consistent with a Type IV reaction. Eventually irreversible interstitial fibrosis and restrictive lung disease occurs from the persistent inflammatory response with repeated exposures to the antigen at lower concentrations.. **A common pathway in developing FHP is the repeated exposure to high levels of organic dusts without proper personal respiratory protection during dust high dust producing activities.**

Three stages of FHP have been identified. The **acute stage** is what is most commonly thought of as Farmer's Lung. The symptoms are identical to ODTS (See Table 7 contrasting FHP and ODTS) and consist of delayed viral-like symptoms 4-8 hours after exposure to organic dusts. The symptoms commonly are a spectrum of chills, fever, myalgias (body aches), headache, nausea, pleuritic chest pain, cough, and shortness of breath. It is a self-limited condition and the severe symptoms will resolve in approximately 48 hours if no further exposure occurs while fatigue and cough may persist for 5-7 days. Physical examination will reveal tachycardia, respiratory distress with tachypnea, and bibasilar crackles. Wheezing is much less common unless there is associated underlying asthma. The **subacute stage** may take longer to develop after exposure and is associated with recurrent episodes of acute symptoms and weight loss and fatigue are more commonly seen. Clinical symptoms may continue for 7-10 days and dyspnea on exertion can last for several months. The **chronic stage** can be very difficult to identify as respiratory symptoms are much less common. Fatigue, progressive dyspnea on exertion, and weight loss are the more common nonspecific symptoms. Evidence of cor pulmonale (right-sided heart failure) with associated clubbing and cyanosis can be seen in advanced cases. Progression to respiratory failure and death may occur with inadequate treatment and continued respiratory exposure.

Diagnostic criteria for FHP are listed in **Table 8**. **Diagnostic tests** include leukocytosis (elevated white blood cell count) with a left shift and arterial blood gases with **decreased arterial PaO₂ and oxygen saturation**. **Chest radiographic findings** typically show a **bibasilar interstitial infiltrate** in the acute and subacute stages but may be normal in 10% of those with symptoms. **Pulmonary function testing** plays an essential role and will show **restrictive lung disease in the acute and subacute stages** with a **decreased forced vital capacity (FVC)** on pulmonary function tests with a **decreased diffusing capacity of CO (DLCO)**. In the chronic stages, emphysema and an obstructive pattern (decreased FEV₁) resulting in a mixed obstructive/restrictive pattern may also occur. **Bronchoalveolar lavage (BAL)** with an increased **mononuclear lymphocytosis** of greater than 20% and often greater than 60% lymphocytes, can be very helpful in establishing a diagnosis. High resolution chest CT (HRCCT) will show a ground glass pattern in the acute stage; diffuse micronodules in the subacute stage; and nodules, honeycombing, interstitial disease in the chronic stage. Diagnostic biopsy will show typical granulomas and interstitial lung disease but is generally not indicated. **Serum precipitins or antibody serology is**



often positive, up to 90%, in the acute stage. It is less likely to be positive in the chronic stages. The antigen causing the sensitization may not be present in the standard Farmer's Lung panel of serum precipitins and a false negative test will occur. **Negative antibody testing does not rule out FHP if the other clinical signs and diagnostic tests indicate FHP.** Positive serum precipitins also may indicate sensitization but not active disease if the clinical pattern does not fit FHP.

The **differential diagnosis** includes allergic bronchopulmonary aspergillosis. Eosinophilia and solid brown plugs of phlegm with fungal elements characterize this form of pulmonary infection. Acute asthma would involve more of an obstructive pattern and eosinophils in the BAL fluid. Psittacosis is considered when there is close contact with poultry and high titers to *Chlamydia psittaci* Group B. Barn allergy is associated with rhinitis and allergic asthma with an obstructive pulmonary pattern. The treatment of FHP includes intravenous corticosteroids and nebulized bronchodilators in moderate to severe disease to decrease acute inflammation and shorten the course of disease for the acute forms. In new onset acute disease, removal from exposure is adequate. Steroids are not recommended once irreversible interstitial disease has occurred in the chronic stage. Several treatment regimens of prednisone have been recommended including 30-60 mg/day for 1-2 months and then taper, 0.5-1.0 mg/kg for 1-2 months, or 40 mg/day and taper over one month (Gudmundsson and Wilson, 1999; Sharma, 2002). Substitution of inhaled corticosteroids is acceptable when there is clinical improvement. The avoidance of the sensitizing antigen is essential. Antibiotics are not necessary. Specific diagnostic guidelines in the diagnosis of FHP have been published with four essential elements which include: (a) the history and physical findings and pulmonary function tests indicate interstitial disease, (b) the chest radiograph is consistent with interstitial disease, (c) there is an exposure to a recognized cause of FHP, (d) there is antibody to the antigen. If these criteria are not met, then biopsy is recommended. (Richerson et al, 1989). **Consultation with a pulmonologist experienced in the treatment of hypersensitivity pneumonitis is recommended prior to biopsy as the history, CXR, PFTs, BAL findings, and HRCCT may be sufficient to avoid biopsy** (Patel et al, 2000). The American Thoracic Society has listed several very important take home points of FHP including the fact that FHP may be progressive and fatal if not recognized and adequately treated, chronic irreversible lung disease, including emphysema, is a result of untreated FHP, and that the pattern of recurrence of disease will indicate the long-term clinical outcome (Schenker, 1998).

Prevention includes adequate drying of grain and hay before storage to **avoid excessive mold contamination and wearing respiratory protection around high dust-producing activities.** Persons with diagnosed FHP have been able to work around agricultural operations without recurrent symptoms if adequate respiratory protection is worn (Ohtsuka, 1995) and by avoiding obviously mold contaminated feed or bedding. (See Prevention section later in chapter). **The minimum respiratory protection is a NIOSH approved 2-strap air purifying respirator.** Those with more severe disease will need increased protection with a helmet powered respirator. **Ongoing monitoring with PFTs and oxygen saturation** is essential to ensure that progressive deterioration from pulmonary fibrosis and restrictive lung disease is not occurring. **Those with FHP that have not progressed to the chronic or advanced stage can continue to work in agricultural operations if the respiratory antigen is avoided or decreased by changes in work practices and the use of effective personal respiratory protection, and monitoring does not indicate respiratory deterioration.**



Organic Dust Toxic Syndrome

Organic Dust Toxic Syndrome (ODTS) is identical to acute FHP in the initial presenting clinical symptom complex and occurs in similar environments of high dust producing activities (See Table 7). **An essential difference is that the toxic inflammatory pulmonary reaction causing ODTS is caused by exposure to massive doses of organic dust with associated molds and bacteria but immunologic sensitization does not occur as it does in FHP.** Endotoxin is considered to be a probable chief cause of the inflammatory response but grain dust, particularly of grain sorghum and soybeans, can be inflammatory in itself (Von Essen et al, 1995). Symptoms of ODTS have been identified in over a third of farmers and are particularly common in hog confinement workers (Von Essen and Donham, 1999; Von Essen et al, 1999). Unlike FHP, there are no abnormalities of the arterial blood gases or pulmonary infiltrates. ODTS is self limited and removal from the high levels of dust is adequate. Symptoms are the most severe for 24-72 hours then improve over another 2-7 days. Corticosteroids or antibiotics are not recommended. Prevention recommendations for ODTS are similar for FHP other than powered air purifying respirators are not necessary. There is debate whether chronic ODTS can occur but it is more likely that recurrent exposure to high levels of organic dust can lead to immunologic sensitization leading to either FHP or occupational asthma. Many farmers experience repeated symptoms with recurrent exposures but ODTS is not thought to progress as occurs in recurrent attacks of FHP.

Asthma and Occupational Asthma

Asthma is a classic IgE antigen-antibody mediated sensitization to an environmental antigen and is **defined as a chronic inflammatory pulmonary disorder with reversible obstruction of the lungs as a result of exposure to variable stimuli.** The obstruction may reverse either spontaneously or with treatment. The clinical hallmarks are wheezing, cough, and dyspnea (air hunger). The most common cause is from environmental allergens. Generally, farmers and agricultural workers have a lower prevalence of asthma than the general population. This may be because of the healthy worker effect in which those who do not tolerate the dusty work conditions leave that occupation. There is a recent body of literature from Europe and Australia that suggests that children growing up on farms have a lower prevalence of asthma, hay fever, respiratory, and allergic, or atopic, diseases compared to children not raised on farms (Downs et al, 2001; Ernst and Cormier, 1999; Klintberg et al, 2001; Leynaert et al, 2001; Reidler et al, 2002; Von Ehrenstrein et al, 2000). It is hypothesized that early exposure to antigens in **traditional agricultural operations** provides life-long protection against the development of allergy. Occupational asthma (OA) is a form of asthma that occurs to an antigen that is unique or present at higher concentrations in the work place. OA is not generally common, usually 5% or less of the total workers. Animal confinement workers, including dairy, swine, and poultry workers and grain elevator workers are at increased risk. Continued exposure to dusts leads to recurrent and progressive symptoms of wheezing and shortness of breath with exposure to gradually lower levels of occupational or environmental antigen. **PFTs show an obstructive pulmonary pattern of a decreased FEV₁ and FEV₁/FVC ratio.** The Chest x-ray may show hyperinflation with chronic disease but usually is unremarkable. Diagnosis may also be made by a twenty per cent (20%) decrease in the peak flow readings while in the work place compared to baseline readings established away from work or a 20% improvement from an occupational baseline when away from the work exposure from 2-6 weeks. A twelve per cent or greater improvement in the post-bronchodilator FEV₁ also indicates reversible



airway disease that is compatible with a diagnosis of asthma. Methacholine challenge tests are also used as a nonspecific indicator of reactive airways and can be used to diagnose asthma when combined with a suggestive occupational history. Pre-existing asthma can be aggravated by dusty work conditions and workers in hog confinement operations or grain elevators may not tolerate the occupational conditions for very long (Donham, 2000). Treatment follows the step approach for asthma and avoidance of respiratory antigens and dust. Prevention is the use of appropriate personal respirators and decreasing dust levels by engineering controls.

Asthma-like Syndrome

Asthma-like syndrome is a recently described non-IgE mediated reversible airway disease identified in up to 25% of swine confinement workers (Donham, 2000; Von Essen and Donham, 1999). It is identical in clinical presentation to asthma (cough, chest tightness, wheeze, dyspnea) except it may have a lesser decrement in FEV₁ than asthma and more transient. Unlike asthma, eosinophils are not present in BAL fluid. **A hallmark is that symptoms are more pronounced upon return to work after being away for a period of time or even after a weekend off (Monday morning response)** (Donham, 2000). The diagnosis is difficult to make and requires cross-shift testing, which consists of spirometry before work and immediately after work, preferably on site. The FEV₁ is lower after work but generally less than the 12% decrease seen in asthma (American Thoracic Society, 1998). In true asthma, the decreased FEV₁ is greater than 12% and more persistent. Protection requires adequate personal respiratory protection and decreased dust and gas levels in confined animal operations.

Chronic Bronchitis

Chronic bronchitis defined as a daily productive cough for three months a year for at least two years. Chronic bronchitis is estimated to have a prevalence of 25-50% in animal and grain production workers, and grain elevator workers (Schenker, 1988; Melbostad et al, 1997; Von Essen and Donham, 1999; Zjeda et al, 1994). Swine confinement workers have the highest prevalence. Cigarette smoking by itself is a significant risk in developing chronic bronchitis but it also additive and probably synergistic with the agricultural exposures, particularly endotoxin (Dalphin et al, 1998; Melbostad et al, 1997). Prevention involves adequate respiratory protection, decreasing levels of dusts and gases in agricultural operations, and smoking cessation.

Sinus Conditions

Sinusitis is common and occurs in up to 25% of swine confinement workers (Von Essen and Donham, 1999). Rhinitis symptoms are reported to occur in 20-50% of animal confinement workers. A recently reported syndrome, mucous membrane inflammation syndrome consisting of eye, nasal, and throat symptoms, has been recently described (Schenker, 1998). This complex of symptoms is an irritant reaction and not IgE mediated and is the most commonly reported syndrome in animal confinement workers. Differential diagnoses include bacterial sinusitis and allergic rhinitis. Sinusitis secondary to aspergillus is considered if there is associated purulent sinus drainage. Evaluation may include sinus x-rays or CT scan, nasal scrapings for eosinophils, and allergy testing. Treatment is symptomatic and involves decreasing the exposure to dusts and gases. Antibiotics do not play a major role and recognition of the recurrent exposure is the key to reduction of symptoms.



OTHER TOXIC GASES, FUMES, AND CHEMICALS

Nitrogen Oxides

Nitrogen dioxide (NO₂) is a severe respiratory irritant and is associated with **Silo filler's disease (SFD)**. Silos can be hazardous confined spaces during the fall when being filled with grain (generally corn but also oats) silage and haylage. Haylage and oatage are harvested and blown into silos in the summer and corn silage is generally harvested in the early fall. The grain is stored in upright silos, forage bags, or covered bunkers to allow bacterial fermentation which makes for a more palatable food for cattle. Nitrogen oxide gases (NO_x) begin to form within hours of filling the silos and nitrogen dioxide becomes the predominant gas. Corn harvested after drought or if heavy nitrogen fertilization has occurred will result in greater production of NO₂. The concentration rises quickly reaching a peak within 5-7 days with levels ranging from several hundred to several thousand ppm. The recommended safe level for eight hours of exposure is 3 ppm. NO₂ is heavier than air and yellowish-orange in appearance with bleach-like odor at high concentrations. High levels of NO₂ can also be present when opening the white plastic forage bags that are replacing silos for storage of haylage and can also result in SFD (Pavelchuk et al, 1999).

When combined with water in the lungs, nitrous and nitric acid is formed causing inflammation of the lungs. NO₂ can cause rapid loss of consciousness at high concentrations and lead to permanent pulmonary fibrosis (scarring). Lower concentrations of up to 50-100 parts per million (ppm) can lead to milder symptoms of eye irritation, cough, nausea, fatigue, and laryngo/bronchospasm. Continued exposure can lead to worsening of symptoms and progress over a course of 6-12 hours with pleuritic chest pain, dyspnea, and pulmonary edema. High concentrations (over 200 ppm) can cause immediate loss of consciousness and the development of pulmonary edema in 12-24 hours. Delayed symptoms or relapse can occur in 2-6 weeks and consist of fever, chills, and respiratory symptoms. Brief exposure to high concentrations or prolonged exposure to lower concentrations can lead to pulmonary edema and eventually **bronchiolitis obliterans** (irreversible pulmonary fibrosis) which may develop within weeks to months. Another radiographic presentation seen in subacute disease is small opacities that may be mistaken for miliary tuberculosis.

Treatment of lower exposures (50-100 ppm) with associated mild symptoms consists of removal from the exposure, baseline chest x-ray, and prednisone at a dosage of 40-60 mg for 2 weeks. Exposure to higher concentrations (over 200 ppm) with more severe symptoms requires treatment with 100% humidified oxygen, nebulized bronchodilators, and intravenous corticosteroids and monitoring of arterial oxygen saturation (Sullivan et al, 2001). More aggressive respiratory support such as intubation and mechanical ventilation may be required if acute respiratory distress syndrome (ARDS) occurs. Hypoxemia and metabolic acidosis may occur. Methemoglobinemia has been reported and obtaining a methemoglobin in a victim that does not respond to oxygenation is recommended. Methylene blue is administered in the case of severe methemoglobinemia. Pulmonary or toxicology consultation with the Regional Poison Center is recommended for any significant case of SFD.

Anyone who has exposure to NO₂ and has respiratory symptoms of hypoxia and dyspnea (air hunger) and altered loss of consciousness should be hospitalized for 24 hours. The use of steroids prevents the



development of both relapse and bronchiolitis obliterans and may need to be continued for 6-12 months if bronchiolitis obliterans occurs (Douglas et al, 1989). Once the victim is stable, serial pulmonary functions is recommended to monitor progression of pulmonary disease to bronchiolitis obliterans. Follow-up evaluations should occur at 1 week, 1 month, and 3 months and also include chest radiographs (Rasmussen and Bascom, 2002).

Prevention consists of not entering silos for 10-14 days after filling with silage or haylage. If entry is absolutely necessary within that time, the blowers should be run for 30 minutes before entering. Depressions in the silage may still contain NO_2 after running the blowers as the gas is heavier than air and toxic exposures could result. There should be a spotter available and a harness with a rescue line should be attached to the person entering. Ideally the concentration of both oxygen and nitrogen oxides should be continually monitored with a gas monitor when entering during this time period. A person on site equipped and trained in use of a self-contained breathing apparatus (SCBA) should be present on stand-by for rescue purposes. If a person collapses in a silo during this time period, others should not enter without running the blowers for 30 minutes or they may succumb to lethal concentrations of NO_2 also. Emergency response to summon rescuers with proper protective equipment should occur immediately. The movement away from silos to the use of bunkers or forage bags has decreased the exposure risk, but exposure to toxic concentrations can still occur within the 2 week time period when the coverings are opened. The usual 2-strap dust respirators are not adequate protection against NO_2 .

Anhydrous Ammonia

Anhydrous ammonia (NH_3) is a commonly used nitrogen fertilizer. It is a liquid under pressure but a gas under atmospheric conditions. It is injected into the soil under pressure. Exposure can occur when an injection port is plugged and the vapor is released into the face of a person unplugging the obstruction or from a leaking hose. The toxic property of concern is the extremely high **hygroscopic** property, or extreme affinity for water. Anhydrous ammonia avidly draws out water from tissues and causes a severe caustic burn, freezing and dehydration of tissue, particularly of mucous membranes, including the eyes, sinuses, nose, and upper respiratory tract. An extremely pungent odor is very noticeable. **Corneal burns and laryngeal edema** can result. A sensation that the air is immediately “sucked out” and it is impossible to breath has been personally reported to the author by those with acute respiratory exposure to anhydrous ammonia. A death of a 78 year old male from anhydrous ammonia inhalation occurred in the upper Midwest in 2000. Intubation may be necessary if acute respiratory distress syndrome (ARDS) develops. Cardiopulmonary arrest may occur. Later development of **bronchiolitis obliterans** or reactive airway dysfunction syndrome (RADS), a non-immunologic asthma-like syndrome can result. A recent rural health issue is also the theft of anhydrous ammonia from farm sites and used in the illegal production of methamphetamine.

First aid consists of immediate flushing of the eyes with water for at least 15 minutes during transport to the health care facility if the person is not in respiratory distress. Further flushing of the eyes at the health care facility for at least another 15 minutes should occur. A baseline chest x-ray, oxygen saturation, and observation for respiratory distress for 12-24 hours are recommended. Eye cups and topical ocular anesthetics are indicated for patient comfort. An ophthalmological consult is indicated. Decontamination by removing contaminated clothing should also occur. Prevention consists



of wearing a full face-shield respirator when filling anhydrous tanks or handling ammonia. The minimum protection should be tight fitting non-vented goggles. It is mandatory that all applicator tanks carry at least five gallons of clean water and changed regularly. A 6-8 ounce squirt bottle should be also available and accessible for immediate use when handling anhydrous ammonia.

Carbon Monoxide

Carbon monoxide (CO) is a **toxic odorless and colorless gas that kills**. CO produced by internal combustion engines. Agricultural exposures can occur from kerosene heaters, gasoline-powered pressure washers in animal confinement operations, and running engines in shops or barns. Extremely toxic concentrations can rapidly accumulate in poorly ventilated buildings, as quickly as within 3-5 minutes (NIOSH, 1993). Fetuses of pregnant women and those with ischemic heart disease and angina are at particular risk for toxic effects at lower levels than healthy adults. Symptoms may initially consist of headache, fatigue, difficulty concentrating and dizziness progressing to chest pain, shortness of breath, visual abnormalities and eventually confusion, weakness, and coma at higher levels or prolonged exposure. Loss of consciousness can rapidly develop without warning signs in environments with high concentrations. Pulmonary edema and respiratory arrest may occur. Delayed neurotoxicity can occur after significant poisoning but cannot be predicted by the initial presentation or carboxyhemoglobin level (Seger and Welch, 2001).

Treatment consists of providing 100% oxygen and cardiopulmonary support. Seizures may occur and should be treated appropriately with anticonvulsants. An **immediate baseline venous or arterial carboxyhemoglobin (COHb)** should be drawn and repeated every 4 hours to monitor response. Arterial blood oxygen will be normal as CO is a chemical asphyxiant but metabolic acidosis may occur. Treatment with hyperbaric oxygen in a compression chamber is recommended if there is neurologic impairment not responding to six hours of 100% O₂, cardiovascular involvement, and pregnant women with significant symptoms and significant COHb levels (Kirk and Holstege, 1998; Seger and Welch, 2001). Consultation with the Regional Poison Center should be obtained immediately. To prevent CO poisoning, internal combustion engines should not be used indoors unless there is very good active mechanical ventilation. Only SCBA respirators would give adequate protection from the toxic effects.

Welding

Many farmers do their own welding repairs and machining. Welding releases metal fumes, particularly zinc oxide fumes from galvanized steel. Clinical symptoms of metal fume fever include a viral like syndrome consisting of chills, fever, myalgias, pleuritic chest pain, shortness of breath and cough occurring several hours after exposure. It is a self-limited condition and resolves in 24-48 hours. No specific treatment is necessary. Prevention of metal fume fever consists of use of a welding respirator and adequate ventilation.

Pesticides

Acute exposure to **organophosphates** or carbamates resulting in poisoning can result in pulmonary symptoms. This can occur in applicators or in field workers entering a field before the safe re-entry interval guidelines. A concern could be in ginseng production due to the canopy covering the plants and reported high use of pesticides. **Excessive bronchial secretions and bronchoconstriction** can



cause acute respiratory distress, wheezing, chest pain, cough and hypoxia. Hemoptysis and pulmonary edema may occur. The treatment consists protecting the airway, adequate oxygenation, and administration of large doses of atropine to reverse the muscarinic effects of the pesticides. Cardiorespiratory arrest is the usual cause of death in acute poisoning (Reigert and Roberts, 1999). The other characteristics of organophosphate poisoning are beyond the scope of this chapter.

Paraquat is a dipyridyl herbicide that causes irreversible **pulmonary fibrosis**. The target organ of the toxic effects is the lung regardless of the mechanism of exposure. Increased oxygen will actually enhance the toxic effects. However, pulmonary effects are generally a result of acute intentional or accidental ingestion and not from low level inhalation. Pulmonary effects occur 7-14 days after ingestion and can result in respiratory failure (Reigert and Roberts, 1999).

Fumigants are gases or liquids under pressure used in interiors to kill pests in stored grain and injected into the soil in potato and other grain production. This class of pesticides is rapidly absorbed across the pulmonary membranes. Some may also penetrate skin and rubber and neoprene personal protective equipment. These include methyl bromide, ethylene oxide, and phosphine. The interior environment that is treated by fumigants is extremely dangerous. Inhalation toxic levels of fumigants are associated with respiratory irritation leading to pulmonary edema and cardiogenic shock. Initial symptoms are nonspecific and include headache, nausea, fatigue, dizziness, and cough.

Disinfectants

Exposure to high concentrations of disinfectants such as **chlorine gas, quaternary ammonium compounds, or mixing bleach with ammonia** in poorly ventilated indoor settings may cause acute pulmonary irritation. If the concentration is high enough to cause acute pleuritic chest pain and significant shortness of breath or dyspnea, **reactive airway dysfunction syndrome (RADS)**, a condition clinically identical similar to asthma, may occur. This condition is characterized by a non-immunologic reactive airways response and may last six months or even cause permanent wheezing. It is provoked by subsequent exposure to lower level respiratory irritants such as chemicals, dust, and smoke and even cold and exercise. It does not respond as well to inhaled bronchodilators or corticosteroids as true asthma. The diagnosis can be made from a history of an acute respiratory exposure causing shortness of breath and wheezing with an associated obstructive lung disease pattern on spirometry or abnormal methacholine challenge test.

Drowning and Suffocation

Suffocation and drowning leading to respiratory arrest are consequences of falling in manure lagoons, engulfment in flowing grain, and collapse of trenches. Dung lung, a polymicrobial pneumonia that may result after aspiration of manure following recovery from near drowning in manure pits. Flowing grain is particularly dangerous for children sitting on the edge of emptying grain bins. Entrapment can occur in several seconds and complete engulfment in ten seconds leading to suffocation. Bridging of moldy or wet grain over air pockets in a grain bin can collapse when a person walks over a seemingly secure surface. The collapsing grain will quickly engulf and suffocate the person. Deaths from grain suffocation occur every fall in every grain producing state.



MICROORGANISM RELATED RESPIRATORY ILLNESSES AND DISEASES

Illnesses Related to Environmental Exposures

Storage Mites

Storage mites, including, *Acarus siro*, *Lepidoglyphus destructor*, and *Gypcyphagus domesticus*, are found in barns and grain and are antigenetically different from house mites. If work clothes that are used in the barn are brought into the home, exposures can result from both home and work, and result in continual exposures. This can lead to barn allergy, a Type I allergic reaction (Terho et al, 1985). Symptoms can include asthma as well as allergic rhinitis. Skin testing can be performed to determine if an immunologic sensitivity has developed but it must be passed on to the allergist or dermatologist that storage mite as well as house mite sensitivity should be evaluated.

Hantavirus

Hantaviruses are members of the single stranded RNA bunyavirus family. Sin Nombre Virus (SNV), is the hantavirus that is considered to be causative organism resulting in hantavirus pulmonary syndrome (HPS). The Center for Disease Control (CDC) defines HPS as febrile illness characterized by bilateral interstitial pulmonary infiltrates and respiratory compromise usually requiring supplemental oxygen and resembling acute respiratory disease syndrome (ARDS). Exposure occurs from typical agricultural activities such as cleaning animal sheds and grain bins, and seasonally closed buildings such as lake cabins (Zeitz et al, 1995). The greatest risk occurs in buildings with increased rodent populations. Agricultural occupations at potential risk include grain farmers, and feedlot workers. The primary vectors are rodents, including deer mice, *Peromyscus maniculatus* and white-footed mice, *P. leucopus*. which are found in enclosed structures in Wisconsin. It is most common in the southwest United States but can occur through the continental U.S. Only one case has been isolated in Wisconsin but the rodent host is common in the state.

Initial symptoms include a short febrile 3-5 day prodromal syndrome consisting of fever, headache, and myalgias. On day seven, cough, and later arthralgias and shortness of breath develop. The condition progresses moderately to rapidly and results in a bilateral interstitial infiltrate, leukocytosis, and elevated liver transaminases, ALT and AST. This often progresses to **pulmonary edema, acute respiratory distress syndrome (ARDS), respiratory arrest and death**. Severe cases also involve disseminated intravascular coagulation (DIC) and myocardial depression. A high index of clinical suspicion is necessary for early diagnosis as the initial prodromal symptoms are nonspecific. Diagnosis is suggested by the clinical history of a typical exposure and clinical findings and confirmed by a positive acute hantavirus IgM for SNV and rising titers of SNV IgG of greater than four-fold increase from baseline. Treatment consists of supportive care, including adequate oxygenation. Prevention includes wearing a N-100 personal respirator, personal protective clothing, including coveralls, goggles, headcover, and shoe coverings or rubber boots when cleaning or spending time in buildings that are potentially rodent infested.

An excellent resource is the CDC *All about hantavirus* website at <http://www.cdc.gov/ncidod/diseases/hanta/hps>.



Blastomycosis

Blastomycosis is caused by a fungal microorganism, *Blastomyces dermatitidis*. It is endemic in Wisconsin, particularly along the Mississippi River Valley watershed and other areas near moist soil with decomposing vegetation. The disease is acquired by inhaling fungal spores that are found in moist soil. Blastomycosis is not specifically an agricultural disease but it is found in those spending time outdoors digging in moist soil and can include foresters, loggers, farmers, and trappers. Northern and central Wisconsin and Minnesota, Manitoba, and Ontario are among the most heavily endemic areas in the world. Blastomycosis is generally more common in men between 25-50 years of age who either work in or visit outdoor areas. A clinical clue can be a history of a pet dog treated for the illness (Bradsher, 1997). Community outbreaks can also occur and affect women and children. The best data on blastomycosis prevalence comes from Wisconsin information. During the period from 1986-1995, a total of 670 cases were reported in Wisconsin. The case fatality rate was 4.39 (29 fatalities) (CDC, 1996). In those patients that are **immunocompromised, the disease is much more aggressive** with a significantly higher mortality rate reported as high as 30% in one case series (Wheat, 1995). If the practitioner does not have a high clinical index of suspicion, the correct diagnosis will not be made and increased morbidity and even death may result.

Any clinician practicing in Wisconsin should consider this as part of the differential diagnosis in a patient that presents with a **flu-like illness associated with cough, dyspnea and pleuritic chest pain progressing to pneumonia** and an outdoor environmental exposure. Acute illness includes the spectrum of symptoms of self-limited flu-like illnesses to febrile conditions resembling bacterial pneumonia. Subacute and chronic illness may present with presentations similar to tuberculosis and fulminant infections resulting from disseminated disease (Davies and Sarosi, 1997). Myalgias, arthralgias, weight loss, and erythema nodosum can also occur. **Other common non-pulmonary presentations are a result of disseminated blastomycosis (in 50% of patients) and include skin lesions and bone osteolytic lesions.** Less commonly, but often enough to be of clinical concern, is the development of central nervous system (CNS) conditions such as meningitis, brain lesions, and epidural abscesses. The chest x-ray presentation is often similar to community-acquired pneumonia and usually affects the upper lobes. **Common findings include a chest x-ray with an alveolar or mass-like infiltrate, typical in 80-90% of patients (Bradsher, 1997). Upper lung focal opacities that are often nodular in appearance are common.** Cavitation, hilar adenopathy, and pleural effusions are relatively uncommon. The masses and nodules can be mistaken for *Mycobacterium tuberculosis* or bronchoalveolar carcinoma. The disease is less common in children, but in those infected, the lower lobes are more commonly involved.

Diagnosis includes obtaining the proper environmental/occupational exposure history, **fungal cultures and 10% KOH stains of sputum, bronchoscopy specimens, or cerebrospinal (CSF) fluids, as well as skin and subcutaneous aspirates.** Serodiagnosis using complement fixation, immunodiffusion, or enzyme immuno-assay (EIA) for the A antigen are helpful when positive (Areno et al, 1997).

Diagnostics specific for blastomycosis should be considered if a person with a significant environmental exposure is not responding to therapy for community-acquired pneumonia. Treatment of pulmonary disease consists of antifungal medications including oral itraconazole or amphotericin B in serious and disseminated disease or in immunocompromised patients (Chao et al, 1997; Davies and



Sarosi, 1997). An excellent reference, which includes radiographic illustrations, is found at *Blastomycosis, thoracic* on emedicine at: <http://www.emedicine.com/radio/topic82.htm>. (Ahrameed).

Zoonotic Respiratory Diseases

Besides the bacterial source of inflammatory endotoxins, bacteria are also associated with infectious disease. (See Table 10) According to the World Health Organization (WHO), **zoonoses** are those diseases naturally occurring between vertebrate animals and humans. It is critical for the rural practitioner to know where to obtain information about potential zoonotic diseases in his or her service area. This review is limited to those agricultural zoonotic diseases that have a significant respiratory component. Sources of information include the hospital epidemiologist or infection control practitioner, extension education specialists assigned to the county or at the university level, the State Veterinarian, State public health department, State Department of Agriculture officials, and the Regional Emergency Animal Disease Eradication Organization(See Table 11 for Wisconsin contacts).

Inhalational Anthrax

Inhalational anthrax is a disease that has been rooted in agricultural and occupational exposures but has been transformed into a primary disease of bioterrorism. It is important for rural practitioners to understand how to diagnose this illness. At this time, even one case of inhalational anthrax is considered to be from a bioterrorism source and is considered a Category A bioterrorism agent (See Table 12). Anthrax is caused by the ingestion, inhalation, or cutaneous inoculation of infective spores of the bacterium *Bacillus anthracis*, a Gram + soil organism. The spores vegetate and grow in the host causing illness. Toxins, including lethal factor, protective factor, and edema factor, are produced and are responsible for the pathophysiological manifestations of the three disease forms. Susceptible animals, including cattle, sheep, goats, and horses, contract the illness from grazing in infected areas. Exposures include skin inoculation or inhalation of spores by wool and tannery workers, goat mill workers, and laboratory workers, and ingestion of contaminated meat. Of the three clinical forms of anthrax, cutaneous, inhalational, and gastrointestinal; **inhalational is the most deadly manifestation** but the cutaneous form is the most common. Anthrax is no longer common in the United States due to an aggressive state domestic animal vaccination and quarantine programs. In the United States, there had been only 224 reported cases of cutaneous disease from 1944-1994 and 18 cases of inhalational anthrax from 1900-1976. There were no reported inhalational cases after 1976 until the 11 cases resulting from the post September, 11, 2001 bioterrorism postal exposures(Swartz, 2001).

Inhalational anthrax begins as a nonspecific viral-like prodrome of 3-5 days with symptoms including low-grade fever, chills, headache and myalgias, nausea/vomiting, cough, and pleuritic pain. Progression to a second stage occurs with associated rapid febrile response, dyspnea, diaphoresis, and shock. Hemorrhagic meningitis with delirium, meningismus, and coma was observed in 50% of cases in the Soviet Union. A pathognomonic feature of inhalational anthrax is mediastinal widening from lymphadenopathy. This was found in all eleven cases of the bioterrorism victims in 2001(Jernigan, 2001). Infiltrates and pleural effusions also occur. *B. anthracis* may be identified in the peripheral smears of blood. Blood cultures will be positive later in disease and become positive after 6-24 hours of growth. It is crucial to culture before starting antibiotics as one or two doses of antibiotics will sterilize the blood cultures. Sputum cultures, nasal swabs, or serodiagnostic tests are not helpful or



indicated in acute illnesses but are useful in epidemiological investigations (Henchel et al, 2001).

The average interval between diagnosis and death is three days unless appropriate antibiotics are quickly instituted at the beginning of clinical suspicion and are life-saving. **According to recommendations by the CDC as of July 2002, treatment** consists of intravenous ciprofloxacin or doxycycline until afebrile and clinically stable and the switching to oral ciprofloxacin or doxycycline for 60 days. Post-exposure prophylaxis should be administered to those with close contacts with inhalational anthrax victims and probable aerosol exposure, including health care workers (Inglesby et al, 2002). This includes vaccination with anthrax vaccine adsorbed (AVA) series of six vaccinations and 60 days of oral doxycycline or ciprofloxacin at the same dosage given for treatment of active disease. Person-to-person transmission is not known to occur but standard precautions are recommended. Specimens are handled under biosafety level 2 conditions and the hospital laboratory should be notified of the suspicions of anthrax (Martin and Marty, 2001). Standard disinfectants are satisfactory to clean surfaces. Cremation is recommended for infected humans and animals. The state Public Health Department, However, current CDC recommendations and notification of the State Public Health Department should be followed as recommendations may change in the future.

Ornithosis (Psittacosis)

Infections resulting from *Chlamydia psittacosis* lead to ornithoses, which include psittacosis, and are associated with **poultry production, particularly ducks and turkeys and birds of the psittacidae family**, particularly parrots, parakeets, and canaries, being the most familiar members. Veterinarians, pet shop workers, and zoo workers are at risk as well as poultry workers. The disease is acquired from inhaling organisms from aerosolized dried avian excreta or respiratory secretions from sick birds. The incubation period ranges most frequently from 5-14 days. Approximately 200 cases a year are reported. Symptoms are most commonly a respiratory tract infection with fever, chills, non-productive cough, dyspnea, sore throat and mild pharyngitis, headache and photophobia. Severity ranges from mild viral-like illnesses to severe pneumonia. Central nervous system symptoms are common but associated meningitis, encephalitis, and seizures are rare. The cerebrospinal fluid is usually normal. The clinical presentations that may occur begin with flu-like syndromes without radiographic abnormalities progressing to mild-to-moderate pneumonia, severe pneumonia, and finally acute respiratory failure with associated sepsis and septic shock (Arjomand, 2002). Other complications include hemolytic anemia, disseminated intravascular coagulation, acute glomerulonephritis, rash, and splenomegaly. Chest radiographic findings are typically unilateral with lower lobe infiltration. Another presentation is a bilateral, military, nodular, interstitial pattern. Pleural effusion is rare. Diagnosis is made from occupational or environmental history of typical avian exposure, chest x-ray, and paired acute and convalescent titers with a four-fold rise in *C. psittaci* titers. Cultures are avoided as this can cause disease in laboratory personnel. **Treatment of choice is tetracycline or doxycycline** for 2-3 weeks to prevent relapse. Serious infections require intravenous antibiotics. Mortality with adequate antibiotic therapy is less than 1% but can be as high as 15% without proper treatment. Prevention is the use of personal respiratory protection when handling sick birds and awareness of the correlation between poultry and psittacidae family members and respiratory illness.

Q Fever

Q fever is caused by *Coxiella burnetti*, a member of the Rickettsiaceae family found in soil and water.



Infections are transmitted to humans from infected sheep, goats, and cattle, and cats. It can also be found in pigs, dogs, and poultry as well as the natural reservoirs of small rodents and rabbits. Packing plants, dairies, stockyard facilities, and sheep farmers are at risk. *C. burnetti* can be found in very high numbers in amniotic fluid, placenta, and fetal membranes of sheep and goats (Weber and Rutala, 1999). The organism is very hardy and remains resistant to desiccation and infectious when windblown at distances up to several miles. Serologic evidence of infection in sheep farmers has been found to be higher than expected (Guo et al, 1998). The symptoms of Q fever are primarily a self-limited, mild flu-like syndrome. Those infected can also develop pneumonia, hepatitis, fever of unknown origin, as well as endocarditis in the chronic form. Treatment of choice of the acute form consists of tetracycline or doxycycline within the three days of the onset of symptoms for 14-21 days. Flouroquinolones may also be considered. The chronic form requires treatment for up to three to four years.

Tularemia

The Gram (-) coccobacilli, *Francisella tularensis*, both Types A and B, is the causative organism of tularemia, or “rabbit fever”. It is considered to be both under recognized and under reported. Exposure routes include ingestion, inoculation by bites, inhalation, and tick- borne exposures. Occupations and persons at risk include hunters, butchers, farmers, and fur handlers who had contact with infected animals and birds. The disease can occur in all states but is most common in the south central and western United States. **It is very infectious with as few as ten organisms considered potentially infectious** (Acha and Szyres, 2001).

The incubation period generally is from 2-5 days, with a range as wide as 1-21 days. Symptoms are similar to community-acquired pneumonia. There is no person-to-person transmission. Untreated individuals may develop symptoms that last for weeks to months with progressive weakness and debility. Other presentations that can occur include hemoptysis (rare) and skin rash. The various presentations include ulceroglandular (the most common), oculoglandular, glandular, pneumonic, typhoidal, and septic. The skin lesion of the ulceroglandular form can be mistaken for cutaneous anthrax. The typhoidal presentation consists of pneumonia and systemic symptoms without cutaneous, mucosal membrane lesions, or regional lymphadenitis and can be rapidly fatal. Complications may include meningitis, sepsis, and secondary pleuropneumonia. The chest radiograph may demonstrate bilateral patchy pneumonia with hilar adenopathy (Dennis et al, 2001).

Diagnosis is made by Gram stain of secretions, culture of pharyngeal washings, sputum, or fasting gastric isolates. If the lab is not notified of the clinical suspicion so the specimens are plated on the proper culture media, cultures are less likely to isolate the organism (Martin and Marty, 2001). Serologic testing by ELISA with paired acute and convalescent titers to *F. tularensis* is useful for retrospective diagnosis. **Streptomycin is the treatment of choice.** Gentamycin and ciprofloxacin are both considered acceptable alternatives. Prevention consists of using gloves when skinning or butchering animals and personal respirators when handling animal parts.

Influenza

The first human case of Swine influenza was first identified in Wisconsin in 1976. A recent study in Wisconsin identified higher seroprevalence evidence of swine influenza infection was found to be associated with being a farmer or farm family member, or entering the barn greater than four days a



week compared to nonfarmers (Olsen et al, 2002). Swine can be a source of zoonotic transmission of swine influenza (most commonly classic swine virus of the H1N1 strain) to humans. Avian influenza A (strains H5N1 and H9N2) can be transmitted to humans but is rare. The future risk is the potential of poultry being source for reassortment of mammalian viruses and resulting in human pandemics of new and virulent strains not included in routine immunizations (Wilson et al, 2001).

Tuberculosis

Mycobacterium bovis can result in a pulmonary form of tuberculosis in veterinarians, farm workers, abattoir workers, and zookeepers but has become uncommon. Infection occurs through ingestion of contaminated raw milk or milk products and inhalation. It is rare in the United States since the introduction of mandatory milk pasteurization and animal infection control surveillance by the use of tuberculin tests. The animal form of the disease is most common in cattle but is also rarely found in swine. *M. bovis* is more common in deer in zoos and particularly in deer farms, and potentially can both infect humans and be reintroduced to countries free of the disease (Acha and Szyfres, 2001). The pulmonary infection of *M. bovis* in humans is identical to *Mycobacterium tuberculosis* in both symptoms and radiographic findings and is treated identically, with the exception of pyrazinamide (PZA). Infected humans can transmit the disease to cattle.

Mycobacterium tuberculosis, the main cause of tuberculosis in humans, is not a zoonotic disease but should be mentioned as there is an increased prevalence found in migrant and seasonal agricultural workers. The highest rates for both latent tuberculosis infection and tuberculosis disease are found in Mexican and Central American workers in U.S.-Mexican border communities (Lobala and Cegielski, 2001). The migrant agricultural workforce stream does come to Wisconsin, as it does to many other states. Clinical suspicion of symptoms consisting of productive cough of over two weeks, chills and fever, weight loss, anorexia, and hemoptysis in individuals of susceptible populations living in substandard housing with lack of access to health care services should include the possibility of *M. tuberculosis* infection.

TREATMENT AND DIAGNOSIS ISSUES

Diagnosis

The importance of an occupational and environmental history in making an accurate diagnosis cannot be stressed too much. The acronym **WHACS**, developed by the Agromedicine Program of the Medical University of South Carolina Family Practice Department is very helpful for a quick initial screening (See Table 12). This may trigger clinical suspicion of the possibility of a clinical presentation consistent with agricultural respiratory disease and then a more detailed occupational and environmental history will be taken.

Spirometry, chest radiograph, and oxygen saturation are essential diagnostic tools in acute illness. Spirometry is useful in mild to moderate disease to determine if obstructive disease consistent with asthma or restrictive disease that may be seen in FHP is present. In acutely ill and toxic patients, spirometry is generally not performed due to the patient's inability to perform the test. A chest radiograph is recommended to rule out pulmonary infiltrates indicating FHP, pulmonary edema, or zoonotic infectious process. Sinus films or single view sinus CT scan is helpful in chronic rhinitis and



sinus conditions. This may serve as a baseline in nitrogen oxide or hydrogen sulfide exposures. Measuring arterial oxygen levels with oxygen saturation by pulse oximetry or Pa O₂ by arterial blood gases are indicated in acute respiratory distress. Arterial blood gases will also assess the acid-base status in ARDS and severe H₂S, and NO₂ exposures. Carboxyhemoglobin is the test of choice for possible carbon monoxide poisoning and may be done on venous or arterial blood. Blood tests are generally nonspecific and many conditions (ODTS, FHP, zoonotic diseases) have elevated white blood counts (WBC) in the 10,000 to 18,000 range. Higher counts may indicate a more severe infectious process. Serum precipitins or antibodies are useful in diagnosing acute or subacute FHP. Some zoonotic diseases (HPS, Psittacosis, Q fever, Tularemia) are diagnosed in retrospect by a four-fold rise in paired acute/convalescent titers. Bronchoalveolar lavage (BAL) is helpful in distinguishing ODTS from FHP and is indicated if the clinical history and presentation is suggestive of a dust acquired respiratory condition.

Blood cultures are indicated if an infectious microorganism is a consideration without a clear cut occupational history suggesting an acute dust or gas exposure. They are particularly helpful in inhalational anthrax and to a lesser degree in tularemia. Blood cultures should be drawn before antibiotics are started. Sputum cultures and stains are helpful in blastomycosis, tularemia, and bovine tuberculosis. Chest CT, specifically high resolution chest CT (HRCCT) is helpful in assessing restrictive pulmonary disease and possible interstitial disease, particularly chronic FHP. It is more sensitive than chest radiographs and will show early disease such as ground glass pattern in FHP and assessing granulomas. See **Table 13** for an overview of diagnostic features.

Treatment

The first priority of treatment is to remove the person from continued exposure to the toxic agent. The ABCs of airway support is indicated in any person presenting in pulmonary distress or coma. Adequate oxygenation and respiratory support is essential. This is particularly critical in ARDS resulting from H₂S, NO₂, or CO exposures and severe exacerbation of asthma. Intravenous corticosteroids are recommended for acute exposure and respiratory distress from nitrogen oxides in silos, severe FHP, and severe exacerbations of asthma. Milder presentations may be treated by oral prednisone. Steroids are not indicated in ODTS. A tapering course over 4-6 weeks in severe cases and several weeks in milder cases is generally recommended. Inhaled bronchodilators and corticosteroids by nebulization and later metered dose inhalers (MDIs) are generally recommended in obstructive disease such as occupational asthma. Steroids should not be used to substitute for removal of exposure to the causal mechanism of disease but to treat symptoms while determination of how to safely of return to work is considered.

In severe respiratory distress resulting from exposures to high levels of CO, H₂S, and NO₂, intubation and treatment with hyperbaric oxygen should be considered. Consultation with a poison center and experienced pulmonologist is recommended.

Treatment of zoonotic diseases is specific to the organism and is discussed in the chapter. Consultation with an infectious disease specialist and the state public health department is recommended. Many of the organic dust related respiratory conditions, including mucous membrane inflammation syndrome, asthma-like syndrome, FHP and ODTS are often inappropriately treated with antibiotics.

**PREVENTION**

The agricultural workplace is dangerous and dusty. **Confined spaces**, defined as a spaces not designed for continuous occupancy with restricted means of exit or entrance, and potential exposures to toxic hazards or an oxygen deficient environment. These environments are considered to be potentially **immediately dangerous to life and health (IDLH)**. Examples of confined spaces on farms and agricultural operations resulting toxic levels of chemicals or oxygen deficient conditions include: silos, grain storage bins, manure pits and lagoons, deep trenches, and controlled atmosphere fruit storage. Potential hazards include poisoning, drowning, and suffocation. Prevention involves both the use of personal respiratory protection but the best method of prevention is **engineering out the hazardous exposure**.

Engineering Solutions

Engineering includes improving and increasing mechanical ventilation and animal confinement operations and decreasing the production of dusts and gases. Dust reduction includes adding oil to the animal feed, keeping the feed covered except at feeding time, and substituting sand for straw for animal bedding. Adding a quart of water to a bale of straw or hay before chopping it up for bedding reduces the dust level by 90%. The use of a mist containing canola oil to suppress dust in swine confinement operations decreases dust and endotoxin levels and adverse pulmonary effects in agricultural workers (Senthilselvan et al, 1997; Zhang, 1997). The use of forage bags and bunkers in storing silage and haylage decreases exposure to confined space hazards found in upright silos. Measuring gas concentrations of oxygen, hydrogen sulfide, nitrogen dioxide, and ammonia before entering confined spaces will decrease inadvertent exposure to toxic levels of gases. One time use, single gas measuring devices, such as Draeger tubes using colorimetric methods, are inexpensive means to measure gas levels of ammonia or carbon dioxide in animal confinement operations. IDLH confined spaces such as recently filled silos or structures containing manure that are undergoing physical agitation should be continuously monitored with a direct reading electronic gas meter before entering and while in the confined space.

Respirators

If an engineering solution is not feasible or practical and entry into an area with respiratory toxins is necessary, use of a **personal respirator is necessary to either purify the air by filtering (mechanical) or to supply uncontaminated air**. Tables 14 and 15 summarize the common occupational conditions and recommended respirators. NIOSH-approved 2-strap N-95 respirator, which filters out 95% or particles 0.3 micron in size or greater, is adequate for dust and mist conditions. If a person has a beard or moustache, there is not adequate protection due to an inadequate seal between the face and respirator and he should either shave or use a powered air purifying respirator. If there are oil mists, a R or P rating, which indicates oil resistant or oil proof respirator should be used. Welding requires a specific welding respirator to filter out metal fumes. Mechanical filters are used for dusts, mists and fumes, such as ammonia, require a specific color-coded gas cartridge respirator that contains activated charcoal. Higher concentrations of chemical gases or mists require gas masks, air-supplied respirators, or SCBA. **Pesticide mists and vapors will require a chemical cartridge respirator.**



Labels on the chemicals used in agricultural operations will indicate the proper personal respiratory protection and should be followed. Infectious agents such as *B. anthracis* or *Sin Nombre virus* require the use of a N-100 (previously referred to as HEPA) respirator which filter out 99-99.97% of particles 0.3 microns in size or greater. Either the CDC or the State Public Health Department should be contacted for the current U. S. Public Health recommendations. IDLH conditions with high concentrations toxic gases or oxygen deficient require the use of a self-contained breathing apparatus (SCBA). This would include a manure lagoon that is both oxygen deficient and contains high levels of H₂S, a building with potential carbon monoxide, or a newly filled silo with potential high levels of NO₂. The person should have gone through training in use of a SCBA, be medically cleared, and maintain the SCBA properly. (See Table 16)

Before using the respirator, the person should always perform a fit check to make sure there is no leaking around the edges. Generally, a medium size fits most men and small fits most women. Children generally use a small. Respirators also come in large. Ideally, an individual should be fit tested for the type of respirator they will be using and have a medical evaluation. This is required for industries covered by OSHA mandated respiratory protection programs. A respirator should be stored in a zip lock bag or similar container to keep it free from contamination. Disposable respirators can be used until it is difficult to breath or a chemical can be tasted or smelled. Disposable respirators last about eight hours, less if very dusty conditions. Pre-filters will prolong the life of chemical cartridges. Check valves make it easier to exhale and less likely to fog glasses. Respirators make the work of breathing more difficult. **Those individuals with significant respiratory or cardiac disease, uncontrolled hypertension, claustrophobia, or the possibility of loss of consciousness from diabetes or seizures, facial abnormalities should have a medical evaluation even if they do not fall under OSHA requirements.** Respirators can be obtained from Farm and Fleet stores, feed stores, Agricultural Co-Ops, Agricultural health centers such as the National Farm Medicine Center at Marshfield or the University of Iowa Center for Agriculture Safety and Health (I-CASH), or farm safety and health catalogues such as Gempler's. University extension offices can provide further information about where to obtain respirators.

CONCLUSION

Respiratory disease associated with agricultural exposures will not be correctly identified if not considered as part of the differential diagnosis. If an occupational history is obtained as a standard part of the evaluation, the appropriate diagnosis is more likely to be made and correct treatment can be initiated earlier in the course of the disease. Besides the occupational history, pulmonary function testing, oxygen saturation, and chest x-rays are important components of the evaluation. The presenting symptoms are often vague and nonspecific and can be mistaken for bacterial bronchitis or viral illnesses. The primary care practitioner can play an important role in preventing disability from agricultural respiratory exposures by becoming knowledgeable about the type of exposures that occur in his or her patient population.



Table 1. Agricultural Respiratory Hazards and Diseases

Hazard	Source	Diseases/Illnesses
DUSTS		
Organic Dust Molds, bacterial endotoxin Grain	Animal confinement, silos, grain production/storage	ODTS, FHP, Chronic bronchitis, sinus conditions, asthma,
Inorganic dust	Plowing, tilling, harvest, picking fruit	Aggravation of underlying respiratory conditions, bronchitis
GASES		
Animal confinement gases: Ammonia, hydrogen sulfide	Manure decomposition in hog, cattle, and poultry animal confinement	ODTS, chronic bronchitis, sinus conditions, mucous membrane inflammation, Pulmonary edema respiratory arrest
Nitrogen dioxide	Fermented grains in silos	Silo-filler's disease Bronchiolitis obliterans
Anhydrous ammonia	Liquid fertilizer	Corneal/laryngeal burns Bronchiolitis obliterans
Carbon monoxide	Pressure washers, kerosene heaters, gas engines indoors	Respiratory arrest, coma, neurologic damage pulmonary edema
Zinc oxide, metal fumes	Welding	Metal fume fever
CHEMICALS		
Pesticides (organophosphates, paraquat)	Pesticide application, intentional or accidental ingestion	Bronchospasm, pulmonary secretions, respiratory arrest, pulmonary fibrosis
Disinfectants, chlorine, solvents	Dairy operations	Respiratory irritants, Reactive airway dysfunction syndrome (RADS)
INFECTIOUS MICROORGANISMS		
Anthrax, Q fever, psittacosis, tularemia, blastomycosis, Hantavirus pulmonary syndrome	Infected soil and water, domestic swine, cattle, goats, sheep, rodents	Viral-like illnesses, pneumonia, meningitis, inhalational anthrax

Adapted from: Kirkhorn and Garry; 2000;
Von Essen and Donham, 1999
Schenker, 1997; Schenker, 1996;
Zjeda and Dosman, 1993;



Table 2. Wisconsin Agricultural Production Rankings 2001

Commodity	National Ranking
Corn for silage, snap beans, cranberries, ginseng, cabbage for kraut, milk pelts, cheese(total)	#1
Milk cows, milk production, butter	#2
Oats, potatoes, carrots, sweet corn and green peas for processing	#3
Tart cherries	#4
Cattle, calves (total), honey	#9
Soybeans	#13
Hogs and pigs (total)	#18

Source: Wisconsin Agricultural Statistics Services, Wisconsin Farm Bureau

Table 3. Top 10 Wisconsin Agricultural products by dollar value

(In descending order)

Milk, cattle & calves, corn, potatoes, soybeans, cranberries, greenhouse and nursery, hogs, broilers, hay

Source: Wisconsin Farm Bureau

Table 4. Activities associated with high organic dust levels

Uncapping silos	Pressure washing animal confinement facilities
Chopping bedding	Cleaning up old moldy feed and bedding
Cleaning out old poultry buildings	Caging and handling poultry,
Loading and unloading grain	

Table 5. Significant levels of Hydrogen Sulfide

Concentration	Physiologic effect*	Exposure level guidelines and standards	Agency
0.01-0.03 ppm	Odor threshold	10 ppm: 8 hour TLV ¹	ACGIH ² -guideline NIOSH ³ REL ⁴
3-10 ppm	Strong odor	15 ppm: 15 minute short term exposure limit (STEL) PEL ⁵	OSHA ⁶ mandatory standard
20-30 ppm	Conjunctival irritation	50 ppm-evacuation from agricultural operations/community	OSHA PEL maximum for 10 minutes
50-100 ppm	Respiratory irritation		
100-150 ppm	Olfactory paralysis		
250 ppm	Pulmonary edema		
500 + ppm	“Knockdown”		
500-1000 ppm	Respiratory paralysis		

*Adapted from Guidotti, 1994; Reffenstein 1992

1-Threshold limit value

2-American Council of Government Industrial Hygienists

3-National Institute of Occupational Safety and Health

4-Recommended exposure level

5-Permissible exposure level

6-Occupational Safety and Health Administration



Table 6. Agricultural Respiratory Diseases with initial common flu-like presentations

Respiratory toxin	Source	Disease Entity
Organic Dust, thermophilic organisms	Dairy, animal confinement	Farmer's Hypersensitivity Pneumonitis
Organic dust, grain dust, endotoxin	Silos, grain elevators	Organic Dust Toxic Syndrome
Organophosphates	Dairy, animal confinement	Organophosphate poisoning
Zinc oxides	Crop production, animal pest control	Metal fume fever
Nitrogen dioxide	Welding	Mild silo-filler's disease
Blastomycosis	Silo fermentation	Early blastomycosis infection
Sin Nombre Virus	Moist soil in north central Wisconsin	Early hantavirus pulmonary syndrome
Zoonotic bacteria	Rodent infected buildings	Early forms of inhalational anthrax, psittacosis, tularemia, Q fever
	Infected soil and domestic animals	

Table 7. Comparison of Organic Dust Toxic Syndrome (ODTS) vs. Acute Farmer's Hypersensitivity Pneumonitis (FHP)

ODTS	FHP
*"Mini-epidemics" 30-40% involved if high concentrations, no sensitization	*Fewer affected 2-10% affected sensitization hallmark of disease
*Symptom delay of 4-8 hours after exposure	*Symptom delay of 4-8 hours after exposure
*Flu -like symptoms for 1-3 days	*Flu -like symptoms for 2-7 days (longer if recurrent)
*Arterial PaO ₂ normal	*Arterial PaO ₂ decreased
*Elevated white blood cells PMNs	*Elevated white blood cells Mononuclear cells
*Chest x-ray normal	*Chest x-ray: Infiltrates lower lobes
*BAL-neutrophils	*BAL-mononuclear cells



Table 8. Diagnostic Criteria for Farmer’s Hypersensitivity Pneumonitis (FHP)

Major-4 needed

1. Symptoms compatible with Hypersensitivity Pneumonitis (HP)
2. Evidence of exposure to antigen:
 - a. Appropriate occupational or environmental exposure
 - b. Serum antibodies
 - c. Basilar infiltrates
3. Radiographic findings of HP
4. Bronchoalveolar lavage (BAL) lymphocytosis
5. Histologic findings compatible with hypersensitivity pneumonitis
 - *HRCCT can substitute for biopsy if 1-3 are met

Minor-2 needed

1. Bibasilar rales
2. Decreased carbon monoxide (DLCO)
3. Arterial hypoxemia

Adapted from Richerson et al, 1989.
Schuyler and Cormier, 1997.

Table 9. Respiratory Diseases Associated with Microorganisms that may be found in Wisconsin

Organism	Environment/host	Disease
<i>Bacillus anthracis</i>	Wet soil, cattle, goats, sheep, hogs	Inhalational anthrax
<i>Blastomyces dermatitidis</i>	Moist soil	Blastomycosis
<i>Chlamydia psittacosis</i>	Poultry production (ducks, turkeys), parrots, parakeets	Psittacosis, pneumonia
<i>Coxiella burnetii</i>	Sheep, goats, cattle Birth products, urine, dust	Q fever
<i>Francisella tularensis</i>	Rabbits, sheep, rodents, rabbits, ticks,	Tularemia, pneumonia
<i>Influenzavirus A (swine and avian)</i>	Swine, poultry	Swine influenza Avian influenza A (AIV)
<i>Mycobacterium bovis</i>	Cattle, swine, deer, sheep	Bovine pulmonary tuberculosis
<i>Mycobacterium tuberculosis</i>	Migrant and seasonal workers	Pulmonary tuberculosis
<i>Sin nombre virus</i>	Rodents, rodent infected buildings	Hantapulmonary syndrome



Table 10. Selected Wisconsin Agencies

Poison Centers:

Children’s Hospital of Wisconsin Poison Center	1-800-222-1222
University of Wisconsin Poison Hospital and Clinics Poison Prevention Education Center	1-608-262-7537

To report infectious animal disease or inquire about zoonotic or agricultural bioterrorism disease

Contact the Wisconsin Department of Agriculture, Trade and Consumer Protection: Division of Animal Health

State Veterinarian	1-608-837-9108
USDA-APHIS, Area Veterinarian in Charge	1-608-274-6746

District Veterinarian

<http://datcp.state.wi.us/ah/agriculture/animals/disease/reporting-disease/veterinarians.html>

Wisconsin County Extension

<http://www1.uwex.edu/ces/cty>

Wisconsin State Laboratory of Hygiene

Public Health and Environmental Laboratory <http://www.slh.wisc.edu/index.shtml>

Public Health Department

Contact local County Public Health Department

Table 11. Categories of Bioterrorism Organisms

Category A

Easily transmitted
High mortality rates
High priority

Bacillus anthracis (anthrax)
Francisella tularensis (tularemia)
Yersinia pestis (plague)
Variola majora (smallpox)
Clostridium botulinum (botulism)
Viral hemorrhagic fevers (e.g. ebola virus)

Category B

Moderately easy to transmit
Low morbidity,
Moderate mortality

Brucella spp. (brucellosis)
Coxiella burnetti (Q fever)
Burkholderia mallei (glanders)
Cryptosporidium parvum
Salmonella spp.

Adapted from J. Bender, 2002



Table 12. WHACS Screening Occupational History

- W**-what do you do?
- H**-how do you do it?
- A**-are co-workers or family members affected?
- C**-are you concerned about the exposure?
- S**-are you satisfied with your job?

Table 13. Diagnosis of Agricultural Respiratory Conditions

Condition	Presentation	Spirometry	Culture	CXR	PaO2	Serology	Other
FHP	Flu-like basilar crackles	Restrictive	NA	Basilar infiltrates HRCCT-ground glass	Decreased (Dec)	(+) acute	BAL>20% lymphocytes Dec DLCO
ODTS	Flu-like Normal exam	Normal(N)	NA	Negative	N	(-)	BAL-neutrophils
Occupational Asthma	Wheeze cough	Obstructive	NA	Hyper-inflation	N to Dec	(-)	
Silo filler's Disease	Chest pain, rales Laryngo/broncho spasm , ARDS Coma	Not specific (NS)	NA	Pulmonary edema (PE) Miliary infiltrate	Dec	NA	Bronchiolitis obliterans Methemoglobin
Barn gas (H ₂)	Knock-down Coma	NA	NA	PE	N to Dec	NA	Neurologic
Anhydrous Ammonia	Laryngo/broncho-spasm , eye	Obstructive	NA	PE	N to Dec	NA	
Carbon Monoxide	ARDS Coma	NS	NA	N or PE	N to Dec	NA	Inc COHb Neurologic
Organo-phosphates	Wheeze Bronchial secretions	Obstructive	NA	PE	Dec	NA	Decreased cholinesterase
Psittacosis	Flu-like pneumonia	NS	NA	Patchy infiltrate	NS	+	
Q fever	Flu-like Pneumonia	NS	NA	N to infiltrates Nonspecific infiltrates Granulomas in healed	NS	+	Inc LFTs Endocarditis
Anthrax	Flu-like to ARDS	NA	+ BC +Gram	PE, Widened mediastinum	Dec	NA	Hemorrhagic meningitis
Tularemia	Flu-like FUO Adenopathy,	NS	Usually (-)	Patchy infiltrates Granulomas	N to Dec	+	PCR, ELISA
HPS	Initial flu-like to ARDS, cardio-Respiratory arrest	NA	Neg	PE	Dec	+	DIC
Blastomycosis	Flu-like	NS	Fungal culture of sputum, KOH stain	Upper lobe infiltrates, masses TB-like, cancer	NS	+	Skin, osteolytic lesions Meningitis



Table 14. Respiratory Protection Devices

Respiratory Hazard	Recommended Personal Respirator Device
Organic Dust (barns, uncapping silos, grain elevators) general protection	2 strap NIOSH N-95 dust mist respirator
Organic Dust and FHP or asthma or very high levels or dusts/molds	Powered air purifying respirator (PAPR)
Confined animal operations and lower level for ammonia	Ammonia chemical cartridge respirator with pre-filter (green ammonia concentration)
Pesticide application, mists, sprays, and liquids	Pesticide, organic vapor, R or P chemical cartridge respirator
Welding	2-strap NIOSH Welding, fume respirator
Higher levels of ammonia, gases, fumes, vapors	Gas mask or canister for specific gas
Hydrogen sulfide in manure lagoon or emptying manure pit, rescue operation	Self-contained breathing apparatus (SCBA)
Recently filled silo, emergency entrance	SCBA
Confined space with possible low oxygen or high levels of gases and no air monitoring available	SCBA
Fruit controlled storage (high CO ₂ , low O ₂)	SCBA
Carbon monoxide	SCBA
Hantavirus, anthrax	N-100 filter (HEPA), preferably with PAPR, SCBA
Fumigants soil	Chemical cartridge, organic vapor
Fumigants, grain storage buildings	Air-supplied respirator or SCBA

Adapted from: Legault and Ayers Murphy and LaCross



Table 15. Medical Examinations for Respi

1. OSHA Respirator Medical Evaluation Questionnaire-mandatory for employees
2. Limited examination(HEENT, cardiorespiratory, clubbing, cyanosis) if respiratory, cardiac, neurologic, insulin dependent diabetes medical conditions. Also if a smoker, history of claustrophobia, or difficulty wearing a respirator.
3. Spirometry indicated if abnormal exam, smoker, or cardiorespiratory history, including asthma, cough, shortness of breath, or angina and chest pain, or difficulty using respirator.
4. Chest x-ray indicated only if abnormal examination or significantly abnormal spirometry.
5. FVC, FEV₁, acceptable if over 70% with no significant symptoms and normally able to do physical requirements of job.
6. Contraindications to respirator use:
 - a. Moderate to severe pulmonary disease (asthma, emphysema, chronic bronchitis)
 - b. Angina, recent myocardial infarction, or congestive heart failure
 - c. Periodic loss of consciousness (seizure disorder not controlled, hypoglycemic insulin reactions)
 - d. Significant claustrophobia
 - e. Unable to obtain adequate fit test (facial deformities, dentures, beard)

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General References

Text

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Web-based

1. Farm Safety & Health Information Clearinghouse. Good links and source of information on practices and safety issues <http://www.bae.umn.edu/~fs>.
2. National Ag Safety Database(NASD) Extension and NIOSH based information <http://www.cdc.gov.nasd>.
3. Ready to Respond: Agro- and bioterrorism issues from the University of Minnesota dealing with zoonotic diseases and food safety. Available at: <http://www.extension.umn.edu/administrative/disasterresponse/terror2.html>.

On-line sources of agricultural personal protective equipment

1. Gempler's Inc. Respirators, monitors, safety/health guides, personal protective clothing, safety equipment <http://gemplers.com>.
2. Industrial Safety Company. Personal protective equipment, safety equipment <http://www.indlsafety.com/catalog/default.asp>.
3. Northern Safety. As above <http://www.northernsafety.com>.

