PENNSTATE

Dusts From Decayed Grain, Hay, And Silage

Summary

A number of farm tasks involve potential exposure to spoiled hay, grain, and silage: breaking open bales of hay, removing the top layer of silage from a silo, shoveling grain orcleaning out grain storage structures. When these activities are performed in a barn, bin, silo, or other enclosure, farmers may inhale significant quantities of bacterial and fungal spores and byproducts. These can induce either of two respiratory reactions: hypersensitivity pneumonitis, referred to here as farmer's lung, and a toxic organic dust syndrome (TODS) similar to that caused by inhalation of grain dusts ("grain fever") and confinement house dusts.

Cases of TODS and acute cases of farmer's lung present in a similar way: as delayed febrile illness of variable severity with cough, dyspnea, myalgia, and malaise, following exposure circumstances similar to those mentioned above, and typically resolving spontaneously within days. However, the two illnesses differ in several significant ways. Farmer's lung is an allergic alveolitis, while TODS is a nonallergic inflammatory response of the alveoli. TODS is relatively common and often occurs simultaneously in a cluster of exposed persons, while farmer's lung is a fairly rare response of sensitized individuals. Only farmer's lung can become chronic, with some cases resulting in progressive irreversible damage to lungs leading to permanent disability or death. Differentiation of acute farmer's lung and TODS may be difficult, and the latter may be misdiagnosed as farmer's lung. Tests helpful in separating the two illnesses include chest radiographs, blood gas measurements, immunoserology, and pulmonary function tests; lung biopsy, bronchoalveolar lavage, and bronchoprovocation may be useful in selected cases.

Farm management and engineering changes that reduce exposure to dusts from spoiled plant material are advisable for all farmers. For farmers suffering from chronic farmer's lung or from repeated attacks of acute illness, such changes are mandatory to prevent possible permanent impairment that may necessitate leaving the farm.

I. DECAYED GRAIN, HAY, AND SILAGE DUSTS ON THE FARM

Animal feed (hay, silage, and grain) that is put into storage with a high moisture content favors the growth of bacteria and fungi. These microorganisms produce spores and byproducts (mycotoxins and endotoxins) of respirable size (less than 10 11 in diameter) which, when released into the air, can be inhaled in large quantities and induce respiratory effects within the airways or alveoli.

More specifically, hay baled while moist heats spontaneously, and proceeds through a natural succession of fungal and bacterial populations The last organisms to grow in the hay are a number of bacterial species termed "thermophilic actinomycetes", which include Micropolyspora faeni and several species of Thermoactinomyces. Silage, which includes chopped oats ("oatlage"), hay ("haylage"), or corn ("cornlage"), is purposefully stored wet in order to undergo the anaerobic ensilage process, which preserves plant material. Tne uppermost layer of silage remains aerobic and spoils. Grain, when stored before being adequately dried, likewise will spoil and harbor quantities of microorganisms. (See Fig. 1) The species of fungi and bacteria that dominate will change with the type of plant material in storage, moisture and heat conditions, and storage conditions, but typically will include the thermophilic actinomycetes and a variety of fungi.

While feed-related microorganisms and their by-products pose a major threat to farm workers, these workers can suffer the same type of response (hypersensitivity pneumonitis) when they are exposed to other agents. These other agents range from fungi and bacteria growing in wood chips to proteins of bird droppings. Some of the many agents found on the farm that are capable of producing hypersensitivity pneumonitis are listed in Table 1. Since these are associated with production or processing of agricultural products, they all could be considered agricultural occupational hazards.

Agriculturally-Related Hypersensitivity Pneumonitis		
Agent	**Exposure**	**Common Name of Disease**
Thermophilic actinomycetes and fungi:		
Micropolyspora faeni,	SPOILED HAY AND OTHER	FARMER'S LUNG
OTHER THERMOPHILIC	FODDER	
ACTINOMYCETES AND FUNGI		
Thermoactinomyces vulgaris	Moldy compost	Mushroom worker's lung
Thermoactinomyces viridis	Moldy compost	Mushroom worker's lung
Alternaria spp.	Moldy wood chips	Wood worker's lung
Pullularia pullulans	Moldy redwood dust	Sequoisis
Aspergillus clavatus	Moldy cheese	Cheese washer's lung
Aspergillus spp.	Moldy malt and barley dust	Malt worker's lung
Cryptostroma corticale	Moldy maple bark	Maple bark stripper's lung
Penicillium caseii	Cheese mold	Cheese worker's lung
Penicillium roqueforti	Cheese mold	Cheese worker's lung
Thermophilic actinomycetes	Vineyards	Vineyard sprayer's lung
Animal proteins:		
Chicken proteins	Chicken products	Feather plucker's lung
Duck proteins	Feathers	Duck fever
Hair proteins	Hairdust	Furrier's lung
Hair serum proteins	Hen droppings	Hen worker's lung
Pigeon serum proteins	Pigeon droppings	Pigeon breeder's lung
Turkey proteins	Turkey products	Turkey handler's lung
Arthropods:		
Sitophilus grainarius	Infested wheat	Wheat weevil lung
Unknown antigens:		
	Cereal grain	Grain measurer's lung
	Tobacco plants	Tobacco

Who is exposed to these dusts, and when?

Almost any farmer, but especially livestock or grain farmers, is likely to be exposed to dust consisting of bacterial and fungal spores and their by-products. This dust will be released any time a farmer is transporting or working with moldy animal feed, but exposures are not likely to be threatening unless the feed and farmer are enclosed in a barn, silo, or other structure.

Section II describes the two respiratory responses to spoiled fodder: the hypersensitivity pneumonitis farmer's lung, and the toxic organic dust syndrome (TODS). Either of these can occur from any exposure to moldy feed. However, farmer's lung is most typical of dairy farmers who are breaking open bales of hay to feed or bed their barn-enclosed cattle. Acute farmer's lung is thus seen most commonly in winter and spring, before cattle are put out to pasture. Farmer's lung is more prevalent in the north temperate zone.

TODS is seen most commonly following preparation of a conventional upright silo for mechanical unloading. After loading a silo, farmers often place a plastic sheet over the silage, and then add another foot or so of silage to hold this sheet in place. This upper silage "cap" becomes grossly contaminated with microorganisms as it dries. Before starting to feed out the silage, a farmer must climb into the silo, pitch this contaminated silage out ot the silo, and lower the mechanical silage unloader into place. High concentrations of microogranisms and their by-products can be inhaled during this task. Farmers are also commonly exposed to clouds of these dusts while shoveling moldy grain in a barn or bin. How common are these dusts and resulting respiratory diseases?

Disturbance of spoiled plant material can produce spore clouds of very high concentration: breaking open bales of hay in a confined space, such as in a barn, has produced clouds of 1.6 x 109 spores/m3 of thermophilic actinomycetes. A person doing light work in this setting may retain 7.5 x 105 spores/minute in the lungs.' Concentrations of 4 x 109 viable spores/m3 have been documented in silo openings (written communication, John J. May, M.D., August 1985).

The prevalence of resulting disease varies from study to study and is difficult to interpret, but prevalence of farmer's lung usually is well below 5% of the farming population (in the United States, although higher in western England, Scotland, and Finland). For example, 3.9% of one surveyed group of Wyoming farmers and dairy producers gave a history typical of farmer's lung.2 A population based survey of over 1400 Wisconsin farmers yielded a 0.42% prevalence of confirmed clinical cases.3 Ten percent of the surveyed population showed serum precipitins to at least one farmer's lung antigen; the highest prevalence was among dairy farmers with the largest farms and largest herds. Some studies demonstrate that a much higher percentage of the exposed population has developed antibodies. Why many farmers with farmer's lung antibodies fail to develop clinical disease is unknown.

TODS resulting from exposure to spoiled plant material has only recently been recognized as a response that is distinctive from farmer's lung; much remains to be learned about this syndrome. However, it probably is far more common than farmer's lung or other illnesses associated with feed storage (such as silo filler's disease). In fact, many cases previously diagnosed as farmer's lung may have been TODS. In a study of New York dairy farmers, 14 of 26 feed-related episodes of respiratory illness were identified as **TODS.**

II. RESPIRATORY PROBLEMS FROM DECAYED GRAIN, HAY, AND SILAGE DUSTS

Inhalation of organic dusts from spoiled plant material is thought to produce two symptomatically similar, but pathologically distinctive, respiratory responses: hypersensitivity pneumonitis and TODS. Hypersensitivity pneumonitis, also called extrinsic allergic alveolitis, goes by a number of agent-specific descriptive names (see Table 1), including farmer's lung when resulting from occupational exposure to spoiled plant material dusts and occurring in farmers. TODS is sometimes called atypical farmer's lung, silo unloader's disease, or pulmonary mycotoxicosis. Since these two responses result from similar exposures and present in similar fashion, they are often confused. Indeed, much work still needs to be done to define the exact agents inducing each biological response, and the specific mechanisms involved in each type of response.

Farmer's lung is an allergic response of the alveoli, which has variable presentation depending on host factors and specific circumstances of exposure to spoiled plant material. Illness covers a continuum from acute reversible to chronic debilitating disease. Symptoms of acute illness are observed four to eight hours following exposure, with cough, dyspnea, fever and chills, myalgia, and malaise.The acute illness subsides in two to five days, and respiratory impairment resolves completely.

Continuous, low-level exposure can cause chronic subacute illness with weight loss, fatigue, and insidious onset of cough and dyspnea. Lung impairment usually resolves completely if exposure ceases. However, multiple acute attacks or chronic low-level exposure can lead to irreversible, progressive lung damage that can decrease total lung capacity and diffusion capacity. End stages are similar to those of any chronic interstitial pulmonary fibrosis, with death typically resulting from respiratory insufficiency or corpulmonale.

Characteristic physical findings include fine, crepitant rales in the lower two-thirds of both lungs among many patients. Patients may be cyanotic or hypoxemic. Laboratory findings in acute cases may include leukocytosis, sometimes with eosinophilia. Pulmonary function tests of acutely ill patients reveal decreased lung volumes, small airways obstruction, and decreased carbon monoxide diffusing capacity; PO2 may be decreased. Chest films may show a finely nodular infiltrate in the lower two-thirds of peripheral lung fields, but may also be clear. (See Fig. 3) Chronic cases show a spectrum of abnormalities, including pneumonitis, fibrosis, hyperexpansion, or honeycombing of lungs. Serum precipitins to thermophilic actinomycetes are characteristic of farmer's lung patients.

TODS is thought to be a nonallergic, inflammatory reaction of small airways and the alveoli possibly due to mycotoxins or endotoxins. Clinically, acute cases present very much like

acute farmer's lung, with cough, fever and chills, fatigue, myalgia, and anorexia occurring four to eight hours following exposure to spoiled plant material. Severity varies from a mild, influenzalike illness to profound illness with severe dyspnea. Symptoms subside in two to five days, and resolve completely within ten days. Chronic illness and presumably permanent lung damage do not appear to occur. Multiple exposures simply produce repeated acute illness. No deaths have been known to result from this syndrome.

Table 2 Differentiation of Farmer's Lung and TODS

Farmer's Lung TODS (toxic organic dust syndrome)

Population affected Undefined subset of population; Anyone exposed; clustering of cases

individual cases

Exposure to dusts High or low Must be massive from decayed hay,
grain, or silage

Chest film Characteristically finely nodular Characteristically clear

density, lower lung fields

Blood gas Decreased PO2 Normal

measurements

Serum precipitins Positive to thermophilic actin- Usually negative

omycete battery

Pulmonary function Mild to marked restriction with Normal to mild restriction

tests possible obstructive com-

ponent

Bronchoalveolar Lymphocytes Polymorphonuclear neutrophils

lavage

A number of features differentiate acute farmer's lung and TODS. These are summarized on Table 2 and discussed below. Farmer's lung occurs in only a small subset of any exposed population. Although predisposing factors must exist, these have not yet been defined. Among sensitized farmers, even a small exposure to aerosolized mold and bacteria can elicit an attack. TODS, in contrast, can affect any exposed individual; thus cases often are clustered, with several individuals in a given work situation being affected simultaneously. However, exposure to decayed plant dusts must be massive.

A number of laboratory tests distinguish the two illnesses. Chest radiographs of farmer's lung patients characteristically reveal a finely nodular density in the lower lung fields, while chest radiographs of TODS patients characteristically are clear (although occasionally are abnormal). Blood gas measurements often show decreased PO2 for farmer's lung, but usually no decrease for TODS. Immunoserology of farmer's lung patients is positive, while TODS patients do not typically have antibodies to thermophilic actinomycete antigens. (Note, however, that TODS patients may have previously developed these antibodies, and thus may demonstrate serum precipitins to the thermophilic actinomycete antigens.) Pulmonary function tests, although usually showing marked restriction with farmer's lung, show mild or no restriction with TODS. And, finally, bronchoalveolar lavage (which is done infrequently, and primarily on an investigational basis) yields fluids rich in lymphocytes with farmer's lung, but dominated by leucocytes with TODS, indicative of their respective pathologies.

Readers also should note that a symptomatically similar toxic syndrome can be caused among agricultural workers by exposure to moldy plant material, grain dusts that are not necessarily mold-laden (See Unit 3), cotton dust, and confinement house dusts (See Unit 4). These similar responses, referred to collectively as TODS, may or may not be pathologically identical .

Diagnosis

Because presentation of farmer's lung is highly variable, no single factor is diagnostic. Farmer's lung should be suspected in any farmer with an influenza-like pneumonitis or active interstitial lung disease. Normally, the following combination of factors is sufficient for diagnosis: a typical presentation (symptoms of cough, fever, and dyspnea, and possible basal crepitant rales), following a history of exposure to decayed plant material dusts, supported by positive serology to any of the 15 or so fungal or thermophilic antinomycete antigens, an abnormal chest radiograph revealing lung infiltrates, and abnormal pulmonary function tests including restrictive changes and impaired diffusing capacity. However, caution is required for several reasons. Although the presentation and history of exposure alone may be sufficient in acute cases, these are not so helpful in subacute or chronic cases, where continuous low level exposure may be difficult to identify and onset of disease is insidious. And, although farmer's lung patients demonstrate a positive serology, 10% or more of the farming population may possess farmer's lung antibodies and only a small number of these experience clinical illness. Also, care must be taken to use an appropriate battery of farmer's lung antigens. Both chest radiographs and pulmonary function tests may be highly variable, and in some cases either or both may be normal.

Lung biopsy, lung lavage, and bronchoprovocation are not normally required or advised, but may be useful in an exceptional case when a specific diagnosis is needed (for example for workman's compensation), or with a difficult differential diagnosis. Lung biopsy in farmer's lung reveals a characteristic granulomatous interstitial pneumonitis. (See Fig. 4) Gross thickening of the alveolarcapillary membranes results from mononuclear infiltration into interstitial tissues, resulting in obliteration of the alveoli. Mononuclear cells often form noncaseating granulomata that may occlude bronchioles. Multinucleated Langerhan's giant cells and foreign body type cells that may be birefringent or nonrefringent are common in areas of inflammation. Spores of the causative molds usually are not recognized in tissues.

Lung lavage, usually regarded as experimental, may be helpful in ambiguous cases of interstitial lung disease, and demonstrate an increase of Iymphocytes, an increase in T to B cell ratios (as compared to peripheral blood), and an increase in IgG and IgM (as compared to albumin).

Bronchoprovocation by farmer's lung antigens has been proposed as a definitive diagnostic test, but can involve significant risk and discomfort to the patient and must be done with care in the hands of experienced physicians.

Acute farmer's lung can be misdiagnosed as influenza, a bad cold, infectious pneumonia, or asthma. An occupational history and the recurrent nature of farmer's lung are helpful in differentiating it from these more common illnesses. Differentiation of most cases of farmer's lung from asthma can be based on lack of wheezing, presence of rales, an abnormal chest radiograph, and pulmonary functions with decreased vital capacity, compliance, and diffusing capacity (rather that reversible obstruction).

Acute farmer's lung may easily be confused with TODS resulting from the same exposure. Although presentation of acute farmer's lung and TODS patients is nearly identical, the two illnesses usually can be separated by considering those distinctions outlined in Table 2: cases of TODS must follow massive exposures, cases often are clustered, and most patients will not have serum precipitins to farmer's lung antigens. There is no evidence that TODS will progress to chronic disease.

Because acute farmer's lung and TODS may result from exposure within a silo, either may be confused as silo filler's disease resulting from exposure to nitrous oxides (See Unit 5) However, this latter disease can be traced to silos filled within the previous two weeks with fresh silage.

Chronic farmer's lung can be misdiagnosed as depression, chronic bronchitis, or any chronic interstitial lung disease. Pulmonary sarcoid may prove an especially difficult differential because of histopathologic and other similarities to farmer's lung. Occupational history and other history of exposure to spoiled plant material are critical in establishing a diagnosis of chronic farmer's lung. Lung lavage or bronchoprovocation may be helpful in exceptional cases.

Treatment

There is no specific treatment for farmer's lung or TODS. Removal from the causative environment is usually self-imposed. Since both TODS and acute farmer's lung are self-limiting, with severe symptoms resolving in two to five days and complete resolution occurring within 10 to 60 days, a physician's help is often not solicited by afflicted persons. In severe cases of acute farmer's lung, with extended duration or extreme hypoxemia, supportive therapy (including oxygen and rehydration) may be needed. Use of corticosteroids is thought to reverse the acute course and shorten the duration of illness. Desensitization is not effective; antibiotics, bronchodilators, and antihistamines are ineffective.

Because a small concentration of antigen can provoke illness in highly sensitive individuals, and continued exposure can lead to permanent impairment, avoidance of spoiled plant material is imperative. Early diagnosis and avoidance are most important in preventing irreversible lung damage.

III. PREVENTION

Prevention of exposure to spoiled plant materials is advisable for all farmers; it is imperative for persons sensitized to farmer's lung antigens. This may be accomplished by reducing mold growth in feedstuffs. Capping silage with a plastic sheet held in place by rocks or a heavy chain (rather than additional plant material) reduces the mold and dust in the top layers of silage. Switching to glass-lined, airtight silos (realizing that these silos pose the health risk of asphyxiation) also will reduce mold growth, but may be economically impossible for many farmers. Grain and hay always should be stored when fully dried.

However, elimination of microorganisms from stored fodder is impossible, and thus techniques to prevent aerosolization and inhalation of these particles should be adopted whenever possible. When silo caps are removed, the top layer of silage can be wetted down to prevent spore aerosolization; however, farmers often do not bother to do so. Persons with a history of farmer's lung should never uncap a silo or perform other tasks with a high probability of exposure to massive quantities of plant dusts. Workers uncapping a silo, shoveling grain, or working with feed, especially in any enclosed space, should always wear a certified dust respirator. This respirator should prevent the inhalation of massive amounts of dusts from decayed plant material necessary to cause TODS, and some evidence suggests that spore inhalation is reduced sufficiently to prevent acute farmer's lung in sensitized individuals. The respirator must fit properly and must be properly maintained. In some cases, highly sensitive individuals may need to wear a powered air purifying respirator.(See Unit 9) Handling dusty fodder mechanically in a manner that keeps the farmer distant from the fodder is a desirable work practice, especially when the fodder is in an enclosed space; the widespread acceptance of large round bales that are transported by a tractor (instead of small, square, hand-carried bales) has probably decreased exposure to moldy hay.

Some dairy farmers with a history of farmer's lung have successfully managed their illness by wearing a respirator regularly and by assigning jobs with the potential of exposure to mold to other individuals. Other such farmers have undertaken more dramatic steps to eliminate exposure to decayed plant materials, including use of glass-lined, airtight silos, completely mechanizing cattle feeding operations, and installing large ventilation systems in the barn (oral communication, James Marx PhD, Feb 1985). Although very expensive, these latter measures have allowed sensitized farmers to stay on the farm.

Monitoring of patients with chronic or repeated acute attacks of farmer's lung should focus on regular testing of pulmonary function and chest radiographs, as well as physical examination and history regarding dyspnea following exposure and on exertion. Measurement of blood gases and exercise tolerance is helpful in assessing impairment. If management and environmental control measures do not prevent the recurrence of farmer's lung, and if pulmonary evaluations indicate progressive respiratory impairment, a farmer may have to leave the farm to prevent permanent impairment.