Dust Pneumonia in Feeder Cattle

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This NebGuide discusses dust pneumonia in feeder cattle and is directed to veterinarians and cattle producers. The publication presents a review of potential causes and discusses the environmental links to the clinical observations of the condition. The paper also highlights the preventative and treatment measures.

Overview

In feeder cattle, dust pneumonia is a sporadic respiratory condition, which along with laryngeal abscesses ("hard breathers") and tracheal edema ("honkers"), does not have a well-defined cause. The veterinary term for feedlot dust pneumonia is acute interstitial pneumonia (AIP). The condition appears to result from a hypersensitivity that can be associated with several causes that result in similar tissue damage and laboratory findings. Typically, the highest incidence of dust pneumonia, hard breathers and honkers occurs in late spring, summer and early fall. The sporadic nature and uncertain list of causes of these diseases in feeder cattle make preventive measures difficult to economically justify and frustrating to institute, but reducing known risks may reduce the incidence.

Acute Interstitial Pneumonia (AIP)

A long list of names has been associated with acute interstitial pneumonia (AIP). These include: dust pneumonia, atypical interstitial pneumonia, pulmonary adenomatosis, acute bovine pulmonary edema & emphysema, acute respiratory distress syndrome, fog fever, lungers, and bovine asthma. An equally long list of potential causes has been associated with the disease. They include tryptophan (an amino acid) metabolism, fibrosing alveolitis (scaring of the air sacks), hypersensitivity, plant toxins, viral and bacterial infections. Additionally, parasitic disease, irritant gases, and metabolic or endotoxic shock have been included as speculated causes in some cases. Feedlot employees refer to cases that develop in late summer as dust pneumonia. When cases of AIP occur an epidemiologic investigation may reveal links to one or more of the potential causes, but frequently a definitive diagnosis is difficult to obtain. A meticulous visual examination of the tissues (necropsy) may provide the best means to link the disease with a cause and differentiate AIP from other causes of death.

The term dust pneumonia has always made a good deal of sense. The feedlot condition occurs most often in the hot, dry, and dusty part of the year. As the sun goes down on hot days and the air begins to cool down the cattle begin to stir. The cooler air is heavier than the warm air and traps the dust stirred by cattle. Often times a type of "dust fog" will appear to develop on a feedyard. Dust particles less than four microns in size can travel to the bottom of an animal's lungs. The fine, talcum powder like dust that settles on surfaces around a feedyard is most likely small enough, which if breathed in would travel to the bottom of the lungs. Remembering that the dust we are discussing contains dried manure, it seems reasonable, that some dust particles may have toxins, specifically bacterial endotoxins, which could damage lung cells and set off a severe respiratory condition. While dust may be involved in many cases of AIP, there are other potential causes that must be considered.

Cattle are very susceptible to heat stress. This heat sensitivity may be the principle reason AIP is principally *(most often)* observed in the late spring through early fall. The rumen provides an internal heat source for cattle, which is wonderful for helping keep them warm when the ambient temperature is cold. For this reason the "shirt-sleeve" temperature for cattle is approximately 20° Fahrenheit (F) cooler than humans. When the ambient temperature reaches 70°F in the spring when cattle still have their winter coats cattle will begin to suffer from heat stress, especially cattle with dark hides. Even after winter coats are shed the upper critical ambient temperature is just over 80°F for most breeds of cattle, including long eared (zebu) breeds. The cattle are not likely to die from heat stress unless the heat is combined with high relative humidity and low winds. Heat stressed cattle will change some behaviors. Most notably will be an increase in respiratory rate (panting) and a change in their eating behavior. High ambient temperatures will almost always be associated with decreased feed intake, a change in eating patterns and an increase in water intake.

A likely association between AIP, elevated ambient temperatures is due to the effect elevated ambient temperatures have on the eating behavior of cattle and subsequent digestive disturbances. Cattle, if left to their own feeding schedule, will tend to eat a larger portion of their daily feed in late evening and night. This is especially true when the daily ambient temperatures have approached or exceeded the animal's upper critical temperature. Erratic feed consumption by an individual animal can be associated with metabolic disturbances, including rumen acidosis. Epidemiologic evidence confirms the likelihood of an AIP case developing in a pen of cattle that have had a digestive dead (greater than 60 days on feed and found dead with no visual tissue damage evidence of other body system disease) to be twice as great as in a pen that has not had a digestive dead.

The amino acid tryptophan, when digested by *Lactobacillus (and other bacteria)* in the rumen, can produce a chemical, indole-3-acetic acid that when converted to 3-methylindole (3MI) forms a very reactive chemical, 3 methyleneindolenine that when absorbed are toxic to the cells in the lung. The level of tryptophan varies but is present in virtually all feedstuffs. Purple mint (*Perilla frutescens*) and moldy sweet potatoes also contain reactive compounds that damage the respiratory epithelium similarly to 3 methyleneindolenine. Purple mint contains chemical (perilla ketone, a substituted furan chemical similar to 4 ipomeanol) that is found in moldy sweet potatoes. These reactive substances are extremely toxic to lung tissue. AIP will develop when these substances are absorbed and travel to the lung via the blood stream. This scenario has been well documented experimentally. Because the reactive chemical noted can be found in particular feedstuffs or found when feedstuffs are changed (most notably cattle grazing dry/dead grass switched to grazing lush green forages), a careful investigation should be done before dismissing the occurrence of AIP as one of those warm weather diseases.

Another AIP scenario that is common in feeder cattle may be associated with ovulation (estrus) in feeder heifers. Research has demonstrated that heifers are more commonly affected by AIP than steers. Additionally, several reports have noted ovarian activity consistent with a functional estrus cycle noted at necropsy of some heifers that die from AIP. Feeder heifers are commonly fed MGA (melengestrol acetate) supplement to suppress estrus activity. Hot weather is known to affect feeding behavior and might cause a sufficient decrease in feed intake to lower the level of MGA below what would be needed to keep heifers from cycling. If a heifer begins to cycle, the additional stress of estrus and the associated riding behavior during hot weather could be related to other digestive and / or metabolic alterations, thus making them more susceptible to 3MI insults when feed intake may resume and increase following standing heat. It is important to note there does not seem to be an association between AIP and estrogenic or TBA implants.

A less likely, yet possible scenario is associated with previous respiratory infection. AIP generally occurs in 0.5 to 1.5 percent of cattle on feed during the months when cattle are at risk for the disease (April through September). However, the rate of AIP during these months in cattle that have been previously treated for respiratory disease is five to eight percent. The risk for an animal developing AIP is 5 to 10 times higher if they have been treated previously for pneumonia than if never treated for respiratory disease. AIP and previous respiratory disease might be associated due to an incomplete healing of the earlier pneumonia in concert with ambient temperatures at or above the animal's upper critical temperature. There is one in-depth study that reported 30 percent of AIP cases also have concurrent *Mycoplasma bovis* respiratory infection.

Some cattle affected with bacterial pneumonia fail to completely eliminate the infection. The animal's immune system walls off the remaining bacteria in small pockets. Although the animal may appear clinically normal, the bacteria *(may*) escape from their walled off encapsulation. Because the animal's immune system has been continuously stimulated by the encapsulated bacteria, should the bacteria break out and enter surrounding tissues the animal's immune system may have a massive reaction (a type of hypersensitivity). The surrounding lung tissue and spaces between the lung tissues (interstitial space) will fill with fluid and reactive white blood cells. The animal's immune response would then be responsible for the acute severe life threatening symptoms.

When cattle get hot their respiratory rate increases (panting) in an attempt to help cool themselves. Should an animal have bacteria entrapped and encapsulated from an earlier pneumonia the increased depth and rate of respiration, caused by heat stress, may cause the capsule surrounding the bacteria to lose its integrity. This would allow the bacteria to escape into surrounding tissues and cause an acute manifestation of a chronic low-grade bacterial infection. This scenario makes sense, but diagnostic laboratory confirmation of this proposed sequence of events is lacking.

Bovine Respiratory Syncytial Virus (BRSV) is commonly accused of being the cause of AIP in feeder cattle. BRSV has been associated with outbreaks of AIP in weanling calves. These explosive episodes of AIP generally have noticeable watery eyes and seldom are associated with a higher incidence in heifers. Extensive diagnostic testing has failed to link the virus to AIP in feeder cattle. Additionally, epidemiologic studies have failed to demonstrate any association between AIP and the use of BRSV vaccine.

AIP Treatment

This is a very difficult disease to treat. It is extremely important to identify AIP affected cattle in the early stages of the disease. (*Care must be exerted in handling these cases quietly and slowly as exertion further compounds their respiratory distress).* Supportive therapy with anti-inflammatory medications (steroids and antihistamines) and prolonged broad-spectrum antibiotic coverage is *(are*) frequently used. The outcome of treatment is often very disappointing. *Therefore, avoidance of medications with slaughter withdrawal requirements and* Salvage slaughter may be the best alternative in uncomplicated cases. It is critical that all medication withdrawal times be met before slaughter. USDA-FSIS will not allow packers to process cattle unless the animal's temperature is below 105°F.

AIP Prevention

- Proper prevention and treatment of the initial respiratory insult is important to avoid chronic low-grade respiratory infections.
- Control dust by frequent pen scraping to remove the loose "sponge" manure layer *and water application to pens, alleys, etc*..
- Review ration *(and bunk*) management, especially in long fed heifers during the hot months of the year.
- Increase feed levels of Rumensin and MGA as needed to ensure proper daily intake of the compounds during times when elevated ambient temperatures are causing erratic consumptions.
- Increasing the Rumensin level up to the upper limit of the recommended feeding rate of 360 mg/head/day to help encourage cattle to eat smaller, more frequent meals.
- Ration mixing and ration intake must be reevaluated if MGA is being fed and riding activity in heifer pens is evident. This indicates the heifers are not getting the proper MGA dose.
- Select feedstuffs that do not favor 3MI production.
- Vitamin E supplementation has been suggested in an attempt to block the conversion of the toxic 3MI reactive intermediates associated with AIP development.

Summary

AIP may be a specific hypersensitivity in cattle that is caused by several causes which results in similar visual tissue damage and diagnostic laboratory findings. While the precise cause of AIP remains unknown, there seems to be a link between rumen function and respiratory health. There is likely an accumulation of insults and is generally the greatest problem in long fed heifers during the hottest months of the year. Managing to minimize 3MI production seems important, especially during hot weather. This includes minimizing fluctuations in feed intake (particularly in heifers), and doing what can be done to control dust. Reducing the known risks may result in significantly less disease occurrence.