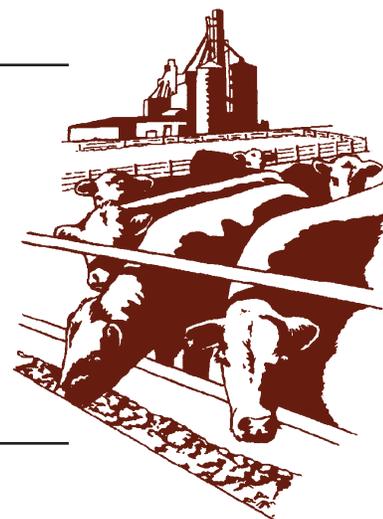


# Beef Cattle Handbook



BCH-3100

Product of Extension Beef Cattle Resource Committee  
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## Acute Bovine Pulmonary Edema and Emphysema in Beef Cattle: Causes and Prevention

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Acute bovine pulmonary edema and emphysema (ABPE) is a naturally occurring illness in cattle. ABPE is caused by an abrupt change in pasture conditions. Cows develop clinical signs of lung disease 1 to 14 days after the pasture change, and death often follows within 2 to 4 days after the appearance of clinical signs.

### Cause of ABPE

ABPE occurs most frequently after cows are abruptly switched from pastures containing sparse, dry, low-quality forage to pastures containing lush, rapidly-growing forage. The abrupt change results in the formation of an undesirable toxic end-product of ruminal fermentation, 3-methylindole (3MI). Ruminal formation of 3MI is a two-step process. Tryptophan, a normal amino acid found in protein, is first changed to indoleacetic acid (IAA) by ruminal bacteria. A *Lactobacillus* species of bacteria has been isolated from the rumen that converts IAA to 3MI. 3MI is produced only from IAA and not directly from tryptophan.

Excess 3MI that is formed during the first few days after a pasture change is absorbed from the rumen, transported to the lung in the blood, and further metabolized to a toxic compound. Metabolism of 3MI occurs

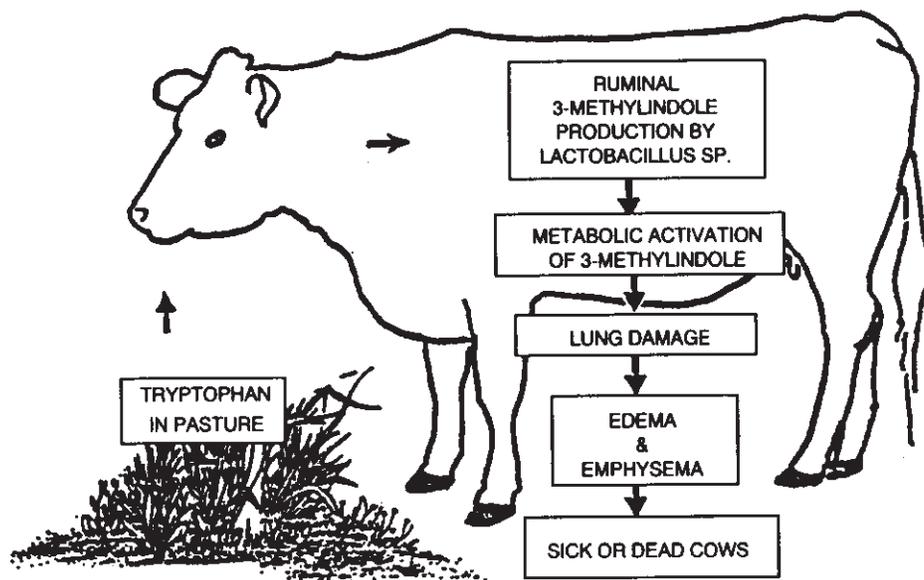


Figure 1. Summary of pathogenesis of ABPE.

in the mixed function oxidase system of the lung, resulting in the formation of a highly reactive intermediate that causes selective injury to lung cells. This causes lung damage and the development of ABPE. A summary of the pathogenesis of ABPE is shown in Fig. 1.

### Signs of ABPE

Clinical signs of ABPE usually occur 1 to 14 days after an abrupt change to lush green pasture and death may fol-

low in 2 to 4 days. Clinical signs associated with ABPE are progressive. Animals may exhibit labored, shallow breathing, panting, an "expiratory grunt," open frothy mouth, and extended neck and head. These are all signs of an animal with impaired breathing. Inhalation and exhalation are difficult because of decreased elasticity of the lung. At necropsy, pathological changes are limited to the lungs, which are inflated, two to three times heavier than normal, firm and rubbery, and dark red. The airways are filled with frothy edema fluid, and the lungs have a glistening appearance and contain large interlobular gas bullae. Histologically, the alveoli and small airways contain edema, hyaline membranes, and cellular infiltration. In advanced stages, alveolar cell hyperplasia is present.

### **Animals Affected**

ABPE occurs almost exclusively in beef cattle, with the highest incidence in mature cows. The largest animals with the highest feed intake capacity most often succumb to the disease. Although the incidence in younger animals and bulls is low, these animals can also be affected. ABPE can occur in all breeds (including dairy cattle), and there is no evidence that prior history of the disease renders an animal more susceptible. Clinical signs and death may be observed in up to half of the herd, but generally only 30 percent of the herd or less will be affected. ABPE seems to occur more often on particular ranches in localized areas, and the absence of ABPE on nearby ranches cannot always be easily explained.

### **Geographical Occurrences of ABPE**

ABPE has been reported in many geographical locations. In Great Britain the disease is called "Fog Fever" because of its association with cattle grazing lush regrowth referred to as foggage. The disease has been reported in parts of Europe, Canada, and throughout the United States, particularly in the western states. In the west, ABPE is often associated with pasture change in the late summer or fall from dry mountain ranges to lush meadows with regrowth following the removal of hay. In general, ABPE may be found anywhere cattle are intensely managed and where abrupt change occurs.

### **Pasture Type Associated with ABPE**

Outbreaks of ABPE are possible during periods of active plant regrowth in the spring and summer, but the disease is most prevalent in early fall. Occurrence usually coincides with cattle being moved from one pasture to another. The disease occurs on many types of forages including rape, kale, alfalfa, turnip tops, small cereal grain forage, rye grass, bermuda grass, and mixed meadow grasses. Any lush forage apparently can result in the formation of 3MI when the rumen is not adapted to the new forage. Grazing management rather than type of forage is the determining factor in the onset of ABPE.

### **Control and Prevention of ABPE**

Cow management is the central theme, and preventing ABPE is of utmost importance because there is no effec-

tive treatment for animals stricken with ABPE. Besides direct financial loss from cow death due to ABPE, additional economic losses result because of orphaned calves, reduced productivity, and reduced forage utilization.

### **Animal Management**

Ranchers should regularly observe pasture grazing conditions and be prepared to move animals to better pasture before cows exhaust their current feed supply. Cattle that have been allowed to remain on dry, overgrazed range land are prime candidates for contracting ABPE. These animals will be hungry. When given access to lush forage, they will tend to overeat. Research has demonstrated that after 2 or 3 weeks of poor quality forage (crude protein less than 6.5 percent and acid detergent fiber greater than 50 percent), the ruminal conditions become optimal for elevated 3MI production. Whether or not cows have been on overgrazed pasture, provide the cows with hay or other feed before a grazing transition.

The goal of preventative management is to provide a more gradual adaptation to the lush forage in the new pasture. Besides feeding hay, limiting cow access to the new pasture for the first few days will aid the adaptation process. This may mean daily moving cows onto and off the pasture. For example, allow cows that are full of hay 1 hour to graze the lush pasture. On the second and subsequent days, increase grazing time by 1 hour over the previous day for the first 7 days. The adaptation period gives the rumen microorganisms time to adapt to the new forage and reduces 3MI formation. Feeding cows restricted quantities of mechanically harvested green forage (green chop) in addition to hay would also adapt the rumen to lush forage.

Additional practices that can reduce losses from ABPE include the use of prophylactic antibiotics (monensin) and/or cutting the pasture before turning in cows. Experimentally, 200 mg monensin per head per day in at least 1 pound of grain for 1 or 2 days before the pasture change and for 7 to 10 days after the change has proven to reduce 3MI formation. A combination of pasture management and dietary monensin will further reduce 3MI formation. Cows fed monensin and given access to pasture containing mowed, partially-dried forage had lower ruminal 3MI concentrations than cows fed supplemental monensin alone. A potential problem with monensin is that some cows may refuse to eat supplements containing monensin or will not consume the required daily supplement. Problems with daily consumption are encountered more often with salt and mineral supplements than protein or energy supplements.

Monensin has FDA clearance for use in mature reproducing cattle. At the time of this publication, other ionophores including Lasalocid do not have FDA approval for use in mature cattle on pasture.

### **Handling Cattle Afflicted with ABPE**

If an outbreak of ABPE occurs, all animals should be removed from the lush pasture and fed good quality hay. Movement of sick animals should be minimal and done

with care. Sick animals with ABPE have a breathing problem and should not be handled in a manner that will increase their need for oxygen. Although antihistamines, corticosteroids, and epinephrine have been used to treat sick cows with ABPE, there is no experimental evidence that the survival of sick animals can be improved by using these drugs.

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