

Cattle Producer's Handbook

Animal Health Section

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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. The change from dry, sparse, poor-quality pasture to lush, rapidly growing forage results in an undesirable fermentation in the rumen and the production of 3-methylindole (3MI), which is rapidly absorbed from the rumen into the blood. As blood carrying 3MI circulates through the lung, a toxic intermediate compound is produced that causes lung damage and results in ABPE. 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.

In this publication we will define ABPE and describe cow management and techniques to reduce the occurrence of this disease. Prevention is the best control for ABPE since no effective treatment for the disease is available.

Cause

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. This bacteria is a gram positive, non-motile, non-spore-forming rod. 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 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 pathogenesis of ABPE is shown in Fig. 1.

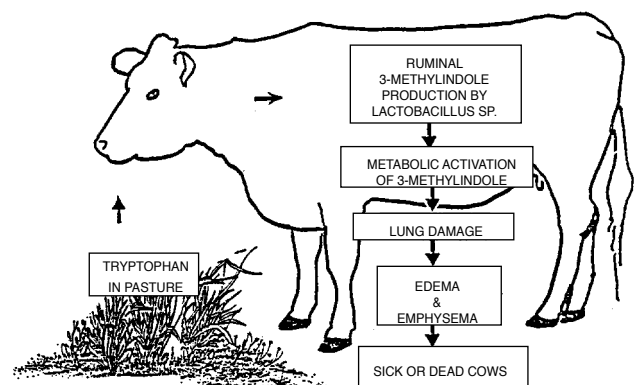


Fig. 1. Summary of pathogenesis of ABPE.