A research team in southern Alberta has recently completed a two year field study that further characterizes the nature of AIP in southern Alberta feedlot cattle (6). Of the 38 total cases clinically diagnosed as AIP, only 81% were histologically confirmed to be AIP. The incidence of AIP was highest in the summer and occurred in a clustered fashion. In our study, AIP developed exclusively in heifers on finishing diets containing over 80% barley grain, and near the end of the finishing period, usually about a month prior to slaughter. Producers and clinicians have also confirmed that the incidence of AIP is far higher in heifers than steers.

Although *Lactobacillus* spp. in the rumen have been shown to convert the amino acid, L-tryptophan to 3-methylindole (3MI, a possible causative agent of AIP) we observed no significant increase in the numbers of lactobacilli in the rumen of AIP-afflicted cattle. These results suggest that AIP is not simply a result of acidosis, but appears to related to other factors such as sex and(or) physiological maturity of the animal, feed-borne pneumotoxins or dustborne allergens.

Lesions in the lung tissue of the study animals were grossly and histologically similar to those previously described for feedlot AIP. Tests showed that metabolites of 3MI were higher in heifers exhibiting AIP than those that were suffering from some other form of respiratory disease. Significantly lower levels of the 3MI metabolites in the urine of AIP-positive heifers as compared to negative controls suggests that urinary elimination of this metabolite may be impaired in animals suffering from AIP. However, we did not find higher levels of these metabolites in the lung tissues of heifers with AIP.

Although others (7) have concluded that BRSV was a sensitizing factor in the induction of feedlot AIP, we found no evidence of BRSV in any of the animals examined. It is becoming increasingly apparent that development of AIP in the southern Alberta feedlots arises from an exceedingly complex interplay of feed intake, feed composition, individual animal physiology and possible environmental triggers. To our knowledge, this study is the first to document a role for 3MI in the etiology of feedlot AIP and provides a target to implement.
dietary or management practices that may provide heifers with a measure of protection from the disease. For example, increasing the level of cysteine in the diet has been shown to reduce the severity of lung lesions in goats infused with 3MI. It is possible that addition of protein sources such as feather meal or elevating sulfur levels in the diet may reduce the insult of 3MI metabolites on lung tissue in heifers. If AIP results from a greater sensitivity of lung tissue to 3MI metabolites due to dustborne allergens, dust control measures such as sprinkling of the feedlot may also reduce the incidence of the disease. Additionally, there are additives that have been identified that are capable of inhibiting the biochemical pathway responsible for 3MI metabolism. Only through the continued critical analysis of feedlot AIP under field conditions will a potential cure for this extremely costly disease be developed.

References
