

Brisket Disease in Cattle**Situation:**

High altitude or brisket disease is a condition that routinely occurs in cattle raised at elevations above 6,000 feet. Mortality may run about 0.5 to 5 percent among cattle native to high country; however, in lowland cattle brought to higher altitudes or in offspring from untested sires losses can be as high as 30 to 40 percent. Ranchers in the western U.S. have 3 to 5 percent of their herds affected by brisket disease each year resulting in a financial loss of about \$60 million. Producers in the Wyoming-Colorado region routinely test breeding stock (bulls in particular) to determine their predisposition to high altitude disease. The procedure to determine susceptibility, prior to development of overt symptoms, involves direct measurement of blood pressure in the pulmonary artery (PAP test). The PAP test possesses drawbacks: it is invasive, time-consuming, relatively expensive and can only be employed on animals exposed to high altitude for several months. Development of a simpler test, one based on an understanding of the molecular basis of brisket disease and which could be applied to all cattle regardless of elevation, is desirable.

Based on molecular features of human pulmonary arterial hypertension, it is known that bone morphogenic protein receptor 2 (BMPR2) plays a significant role in the development of the condition in humans. Assuming that the human disorder would share similarities with the bovine condition, we investigated alterations in BMPR2 and its cellular targets in a herd of 40 half-sibling steers that had a high incidence of brisket disease. A survey of the protein expression of BMPR2 in the lungs of these cattle indicated that BMPR2 is decreased in the lung tissue of cattle exhibiting high PAP scores. However, no mutations in the BMPR2 gene at the genetic level were detected. Cytoskeletal protein expression of alpha and beta actin increased in lung tissue concomitant with a decrease in BMPR2, reflecting the lungs' attempt to adjust to lower amounts of oxygen. As in the human model of pulmonary hypertension, changes in BMPR2 alone do not cause the disease, and we hypothesize alterations in other genes and/or environmental factors could be important in the development of the disease.

Impact:

Continued work with cattle affected by brisket disease will provide new insight into the causes and inheritance of both the bovine and human forms of the disease. With a solid understanding of molecular and genetic mechanisms underlying the condition, it will be possible to screen and identify all cattle that would be affected by high altitude and provide the producer with quicker, less expensive access to all altitude-resistant seed stock. This would mitigate brisket disease losses and allow for genetic improvement in the Wyoming cattle herd.

Richard J. McCormick
Department of Animal Science
rmccrmck@uwyo.edu