High Mountain Disease in Cattle

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Introduction

High-mountain disease (also known as brisket disease) is a debilitating, often fatal condition, affecting some cattle raised at elevations over 6,000 feet. Mortality may run as high as 5% 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 30–40%.

The physiological reaction of susceptible cattle to reduced oxygen at high-altitude is expansion of the arterial walls in the lung resulting in decreased internal diameter, decreased blood flow, and high blood pressure (pulmonary hypertension). Ultimately, these changes cause impairment of heart function. As a result, cattle susceptible to brisket disease exhibit progressive limitation in movement, labored breathing, fatigue, slow growth and weight gain, diarrhea, and edema of the brisket.

To date, direct measurement of pulmonary arterial pressure (known as a PAP test [Figure 1]) of cattle at altitude is the only method available to determine if an animal is susceptible to brisket disease. Efforts to understand and recognize inheritance patterns or develop blood-based markers that would identify susceptible animals have proven inconclusive. PAP testing has been successful in identifying resistant animals, and their use as seedstock has reduced the overall incidence of brisket disease in high-country herds; despite these efforts, the incidence of brisket disease is on the rise, probably because cattle are increasingly bred for size.

Figure 1. Veterinarian Tim Holt performs PAP testing at the Laramie Research and Extension Center.
Objectives

In the first phase of this project, we explored a candidate gene approach to bovine brisket disease based on studies in humans. The follow-on phase is to correlate economically important beef cattle production traits with PAP scores in a population of animals that show no clinical signs of brisket disease.

Materials and Methods

We recently purchased the necessary instrumentation to perform PAP tests at the Laramie Research and Extension Center (LREC) and the James C. Hageman Sustainable Agriculture Research and Extension Center (SAREC) near Lingle. We have a long-standing collaboration with Colorado State University veterinarian Tim Holt, who is instructing personnel in PAP testing procedures and data interpretation.

Results and Discussion

Interestingly, human patients with an inherited form of pulmonary hypertension similar to brisket disease carry mutations in the gene BMPR2. BMPR2 suppresses arterial wall expansion in the lungs, which is consistent with our hypothesis that the BMPR2 signaling pathway is defective in cattle suffering from brisket disease.

We sequenced BMPR cDNAs from 40 half-sibling steers and did not find any mutations in the bovine BMPR2 DNA sequence that correlate with elevated PAP score. However, we have found evidence that BMPR2 gene expression in cattle is reduced in lungs of animals with elevated PAP scores. In addition, lungs from affected animals showed changes in expression of secondary genes, which are regulated by BMPR2. However, these observations do not prove that BMPR2 is causally involved in brisket disease. In the next step, we propose to create a population of steers, a subset of which will be predisposed to the development of the disease. These animals will not develop brisket disease, however, because they will be raised at about 4,100 feet at SAREC and finished under feedlot conditions. Growth rate and feed efficiency will be recorded.

At the completion of the finishing period, animals will be PAP tested and slaughtered at the University of Wyoming Meat Lab, and measures of carcass quality, carcass composition, and meat quality will be taken. Parameters of growth, carcass composition, and quality will be correlated with PAP score to determine if potential for development of brisket disease in the absence of clinical signs influences beef production traits.

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Key words: high-mountain disease, brisket disease, cattle