SORTING GATE

Saving lives: Reducing the risk of high-altitude disease

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For cattle producers ranching at high altitudes, the risk of losing cattle due to this oxygen-deprived, hypoxic environment is far too real. High-altitude disease (HAD) has been a problem for producers grazing cattle at elevations greater than 5,000 feet (ft.) for more than 100 years.

The disease is more commonly known as "brisket" disease due to the recognized edema, or swelling, resulting in that particular region of the body. However, other symptoms can include heavy breathing, lethargy, runny nose and diarrhea, causing cattle exhibiting these symptoms to be misdiagnosed with respiratory or pneumonia issues.

Postmortem inspection reveals a malformation of the right ventricle of the heart and other abnormalities in the pulmonary artery and lungs.

Underlying cause

This malformation is due to a process called shunting. All species undergo pulmonary vascular shunting when under hypoxic conditions, but shunting happens at a much greater rate in cattle compared to any other species. In the lungs, the response to hypoxia is vasoconstriction, or the narrowing of blood vessels. This narrowing of the blood vessels redistributes blood away from poorly oxygenated areas of the lung to oxygen-rich areas, which then can be pumped to the rest of the body.

If hypoxic exposure is limited, cattle can be removed from the oxygen-deprived environment and lungs regain nearly full function. However, under chronic hypoxic exposure, like in the case of grazing beef cattle, permanent hypertrophy and thickening of the medial layers of the pulmonary arterioles takes place.

This vascular remodeling increases pulmonary resistance leading to pulmonary hypertension (high blood pressure), eventual right heart failure and death. The incidence of this disease appears to be relatively low in native-born cattle (<5%); however, disease incidence in non-native cattle that are taken to altitude to live has been documented at nearly 40%.

Reducing incidence

Pulmonary hypertension is classified as the cause of HAD. In order to reduce its incidence, a method to capture mean pulmonary arterial pressures (PAP) to use as indicators of HAD susceptibility has been developed. Mean PAP is estimated from the collection of systolic and diastolic pressures measured within the pulmonary artery and measured in millimeters of Mercury (mmHg).

The PAP measurement reflects the resistance to blood flow experienced by the right ventricle. PAP records have been utilized for decades to decrease the incidence of HAD. Research in the area reports PAP score is moderately heritable; therefore, genetic selection for the trait can take place.

In response to this research, the American Angus Association and Angus Genetics Inc. (AGI) started to investigate a genetic evaluation for PAP. This evaluation considers measurements taken at 5,500 ft. or greater to be high-elevation measurements; whereas, measurements taken from 4,000 to 5,500 ft. are considered to be moderate elevation.

Heritability estimates for high-elevation yearling PAP scores (h^2=0.39) were larger when compared to moderate-elevation yearling PAP scores (h^2=0.20) with the genetic correlation between the two elevation groups reported around 0.75.

In addition, high-altitude weaning PAP scores are included in the Association's genetic evaluation to account for any pre-selection bias that may occur as a result of selection pressure at weaning time. Only weaning PAP scores taken at elevations greater than 5,500 ft. are utilized in the evaluation. The heritability of high-elevation weaning PAP (h^2=0.25) is similar to that of moderate-elevation yearling PAP.

With this research in hand, the Association released research expected progeny differences (EPD) for high-elevation yearling PAP, earlier this year. The EPD predicts the genetic differences in PAP score for yearling-age cattle at elevations greater than 5,500 ft., with lower EPDs being more favorable. This tool, like any EPD, should be used to select parents for the next generation and must be used to compare two animals.

A lower PAP EPD predicts a sire should produce progeny with lower mean pressures, decreasing the risk of contracting HAD. For example, if Sire A has a +2.0 PAP EPD and Sire B has a -1.00 PAP EPD, this would mean Sire B's calves, on average, should have PAP scores 3 mmHg lower. Therefore if Sire A's calves have an average PAP score of 41 mmHg, then Sire B's calves would be predicted to have an average PAP score of 38 mmHg.

It is important to remember the PAP EPD is not a replacement for taking scores on cattle living at elevation. Animals may have good genetics to pass on to the next generation, but due to a life event [bovine respiratory disease (BRD) for instance], their respiratory system may be damaged, causing them to lack the viability to survive at high altitudes.

Think of PAP in terms of scrotal size. Bulls may have the genetic potential to pass along larger scrotal size genetics, but scrotal (SC) EPDs are not a replacement for breeding soundness exams (sometimes referred to as BSE). The EPD can be valuable to select parents for the next generation with less risk. However, if a sire is to be taken to higher elevations to live, they should be tested themselves before doing so.

In total, more than 13,000 high-elevation yearling, moderate-elevation yearling, and high-elevation weaning PAP scores were utilized to predict these PAP EPDs. Both age and elevation are accounted for in the model. Only artificial insemination (AI) sires with accuracy values of greater than 0.40 are published in this research report, which can be found at *http://bit.ly/PAP-EPD*.

PAP EPD remains a tool to increase the environmental adaptability of cattle living at altitude. While research exists and is ongoing to understand the possible link between bovine congestive heart failure (BCHF) experienced at high altitude vs. lower altitude, no definitive evidence of a direct link exists at this time. PAP EPD is not considered a solution to avoiding BCHF in the feedlot.

For more information or questions on PAP EPD, please contact the American Angus Association office at 816-383-5100.

Editor's note: "Sorting Gate" is a regular *Angus Beef Bulletin* column featuring herd improvement topics for commercial producers using Angus genetics. Authored by staff of Angus Genetics Inc. (AGI), regular contributors include Dan Moser, president; Stephen Miller, director of genetic research; and Kelli Retallick, director of genetic service. For additional information on performance programs available through the American Angus Association and AGI, *visit www.angus.org* and select topics under the "Management" tab.