High Altitude Disease – An example of genetic variation for adaptability

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High altitude disease, also known as brisket disease, dropsy or big brisket affects some cattle living 5,000 ft or more above sea level. The disease was observed by Spanish conquistadors in South America more than 500 years ago, although its relationship to pulmonary hypertension caused by high altitude was not proven until the 1950s. The disease results from thickening and increased restriction to blood flow in small arteries in the lungs as a result of the reduction in blood O_2 saturation at high elevations. The pulmonary system compensates by increasing blood pressure that in turn increases leakage of fluids into the chest cavity and brisket. The heart may also increase in size, enlarging so much that the valves no longer meet, leading to back-flow of blood at each contraction.

The disease affects both sexes and all breeds of cattle (but not sheep or elk) to varying extents. Cattle subjected to previous selection at high altitude may suffer losses of no more than a few percent attributable to this disease. In contrast, cattle bred at low altitude with no history of natural or artificial selection for high altitude performance may sometimes suffer losses up to 40-50%. Moving cattle to low altitude usually leads to prompt recovery.

The pulmonary artery delivers O₂-depleted blood from the right ventricle to the lungs so the first symptom of the developing disease is an increase in pulmonary arterial pressure (PAP). A saline-filled plastic tube attached to a heart monitor can display the PAP when the tube is passed through a needle into the jugular vein and from there into the upper right side of the heart, through a valve, into the lower right side, through another valve and into the pulmonary artery. This procedure has been used for more than 30 years to generate PAP scores as an indicator trait to assist selection to reduce high altitude disease. Testing is more reliable at higher altitude and only after an acclimatization period of at least three weeks. Elevated PAP can reflect any respiratory or pulmonary disease. Low apparent PAP scores can result from an incorrectly inserted catheter. In order for selection to be effective it must be undertaken by a veterinarian that is competent at this technique. Cattle that are wellsuited to high altitude have pressure scores below 35 mmHg whereas ill-adapted cattle exceed 45 mmHg.

The PAP scores are heritable and repeatable when undertaken by a reliable operator. Heritability estimates range from 0.32 to 0.7 (Enns et al., 1992) with no evidence of maternal genetic or maternal permanent environmental effects. Tybar Angus ranch in Carbondale Colorado have, since 1984, tested almost every animal in their Angus stud. Proven, fashionable Angus sires with moderate growth and high marbling EPDs have been progeny tested for PAP at Tybar. Sire EPDs for PAP have ranged from -5 to +5 mmHg. Sires with favorable (low) PAP EPDs based on their progeny test have been used more widely to produce performance-tested sale bulls with "genes that fit" the high altitude environment.

Cattle can vary in their adaptability to various environments in terms of their ability to withstand stress. Important factors that can cause genotypeenvironment interactions include nutritional, climatic and disease stress. An indicator of the most serious form of genotype-environment interaction is a reranking of sires when progeny tested in different environments. Accordingly, an interesting issue in relation to adaptability to high altitude environments is the comparison of sire productivity based on offspring performance at Tybar ranch and the productivity of the same sires assessed using the American Angus Association performance data obtained across a range of environments over the nation, principally at low altitude.

Growth trait EPDs (birthweight, BWT; weaning weight direct WWD and maternal WWM; and yearling weight, YWT) were computed using a multi-trait animal model applied to Tybar data alone. The correlations were computed between the 132 sire EPDs from Tybar records and the corresponding EPDs from the American Angus Association. The correlations were 0.61, 0.42, 0.39 and 0.33 for BWT, WWD, WWM, YWT, respectively.

The correlations between sire EPDs in independent datasets provide an estimate of the genetic correlation between the two sets of circumstances, provided the evaluations are highly accurate in both datasets. In practice, the accuracy of the EPDs are less than perfect and the estimated correlation between EPDs will be biased downwards relative to the genetic correlation. Monte Carlo procedures can be used to construct the distribution of the expected correlation. This procedure can be adapted for independent or for datasets that demonstrate a part-whole relationship as is the situation in this case where the Tybar records contribute to the American Angus evaluation. Under the null hypothesis that the genetic correlation is unity between growth performance in Tybar productive circumstances and national average circumstances, the 5% critical values were 0.63, 0.65, 0.37 and 0.60 for the four traits respectively.

These results suggest that there is little evidence that BWT and WWM are different traits at Tybar compared to national circumstances. In contrast, the observed correlation in EPDs for WWD and YWT are much too low to have resulted from a unit genetic correlation. Treating the two datasets as independent, the genetic correlation between Tybar and national environments would have to be less than 0.74 for WWD and less than 0.66 for YWT for the observed correlations to be non significant at the 5% level.

There was no evidence that the re-ranking of sires for WWD and YWT was associated with their PAP EPDs. It might have been expected that the bulls with elevated PAP EPDs would have produced ill-adapted offspring for high altitude whose growth was inferior to the offspring of the same sires used as low altitude. Other aspects of the environment must have been responsible for ranking changes.

Enns, R.M. J.S. Brinks, R.M. Bourdon and T.G. Field. 1992. Heritability of pulmonary arterial pressures in Angus cattle. Proc. West. Sect. Amer. Soc. Animal Sci. 43:111-112.