

Evidence of genetic variability in cattle health traits: Opportunities for improvement

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Introduction

From the perspective of today's society, animal health is synonymous with animal welfare and any mention of a sick animal conjures up a negative perception in conjunction with factory farm. To those of us actually in the beef industry, animal health is also synonymous with animal welfare and we continually strive to improve the welfare of our cattle.

Improved health reduces costs associated with treatment, reduces mortality losses, and eliminates the reduced levels of performance typically associated with sick cattle. While management decisions can clearly reduce incidence of disease, there are typically costs associated with those management procedures. Historically, management of disease focused on modifying the animals' environment through vaccination, low stress handling, and through the treatment of clinically ill animals with little attention given to the potential for genetic improvement of health-related traits. Much of this is likely due to one of the greatest weaknesses in current national cattle evaluation--a lack of tools upon which to make these selection decisions. In turn, that lack of tools likely springs from difficulty in identifying the economically relevant traits related to animal health. The term "health" includes a vast array of potential traits for selection.

The Challenge

At a rudimentary level, health traits, as related specifically to "disease" fall into three general categories. The first group contains those diseases that are the result of a defect in the individual's genetic composition such as osteopetrosis, Arthrogryposis Multiplex, fawn calf, tibial hemimelia, etc. The second class contains those diseases associated with non-transmittable environmental challenges such as fescue toxicity, facial eczema, or high-altitude (brisket) disease. One could class these traits as more directly related to adaptability or as being environmentally induced. The final class represents those diseases related to some specific disease vector or pathogen whether it be bacterial, viral or parasitic in nature. From this point forward we will refer to these as pathogen-associated. All three of these categories likely offer the opportunity to capitalize on genetic improvement.

As recent experience in the beef industry would show, there is clear opportunity to eliminate genetic-caused disease from populations through the use of gene marker tests. These tests have been very successful in identifying animals carrying a specific deleterious recessive gene. This process alone is evidence of our potential to improve health traits, at least in the first category. The remaining two classifications are more challenging. In field data, there are often issues related to the accuracy of diagnoses, to the utility of data collected across production environments, and to concerns relative to differences in pathogen exposure.

The Process

As with any new trait that becomes a candidate for genetic evaluation and selection, there is a process that must be completed prior to developing selection tools. First the economically relevant traits and potential indicator traits must be identified. For instance, before the theory behind calculating heifer pregnancy EPD was developed, background research showed that

yearling scrotal circumference in bulls was related to age of puberty in their daughters with the logic being younger age of puberty should result in higher heifer conception rates. Subsequently, scrotal circumference was shown to have a genetic component, or put another way, it was shown to have a heritability greater than zero. As with any trait we wish to improve, health traits must be shown to be under some degree of genetic control. Put another way, there must be genetic variability in the population we are selecting from. Without that genetic control there is no opportunity for genetic improvement. Often the difficulty lies in identifying the appropriate phenotype or outcome to collect in order to meet our goals. We believe this is especially true of health traits.

Once the appropriate traits are identified the challenge becomes collecting field data in sufficient quantities to develop a genetic evaluation. Field data would ideally be collected on the economically relevant trait itself, but barring that, highly related indicator traits could be used. This process is similar to the development and use of ultrasound data on breeding animals for prediction of carcass merit in their slaughter progeny. Ideally we would collect carcass data on every animal for use in genetic evaluation, but that is problematic. To overcome this issue, ultrasound was introduced.

In the absence of the adequate field data, an alternative is the development of DNA marker tests that explain significant amounts of variability in the traits of interest. This development often requires extensive research populations of highly phenotyped individuals along with sufficient validation populations, but once successfully developed DNA marker test results can be used to facilitate delivery of EPD to producers for selection.

In summary the process followed in new trait development is to

1. Identify the appropriate economically relevant traits and associated indicator traits
2. Develop methods for sufficient collection of field data to determine if genetic variability exists in the traits (and if alternative measures such as DNA marker tests could be developed)
3. Based on the results of #2 continue to collect appropriate data
4. Use the information collected in #3 to begin to release selection tools for use by breeders in selecting for improved animal health.

With that as the process, let's examine the opportunities for genetic improvement in health traits given current research. The discussion will evaluate health traits for both an environmentally induced disease and a pathogen induced disease.

Environmentally Induced Disease

What are the opportunities for genetic improvement of environmentally induced disease using the above process? To examine this we will use high-altitude disease commonly referred to as brisket disease as the template. Historically this disease manifested itself in cattle in environments above 5500 feet of elevation. The disease manifests itself with a swollen brisket area, reduced appetite, reduced thriftiness (poor doing) and eventual death. Physiologically, the disease is the result of lower concentrations of oxygen at higher elevations. In that low oxygen environment, the heart responds vigorously by forcing blood through the pulmonary system in turn forcing fluid out of the circulatory system resulting in the swollen brisket.

Here, the economically relevant trait would be resistance to brisket disease or survival at higher altitudes. In extensive range environments typical at these elevations, precise identification of afflicted animals is often problematic and collection of data is difficult. In the absence of that data, an indicator trait for brisket disease was developed—pulmonary artery pressure (PAP) based on evidence that animals diagnosed with brisket had elevated PAP. Subsequent research showed that PAP was heritable (.46; Enns et al., 1992) and should respond to selection. In 1992, the first expected progeny differences for PAP were calculated and used in selection of breeding stock at the Tybar Ranch near Carbondale, CO. The trend in PAP since 1992 has been consistently downward (favorable) since that time (Figure 1) resulting in a reduction in lost performance and mortalities.

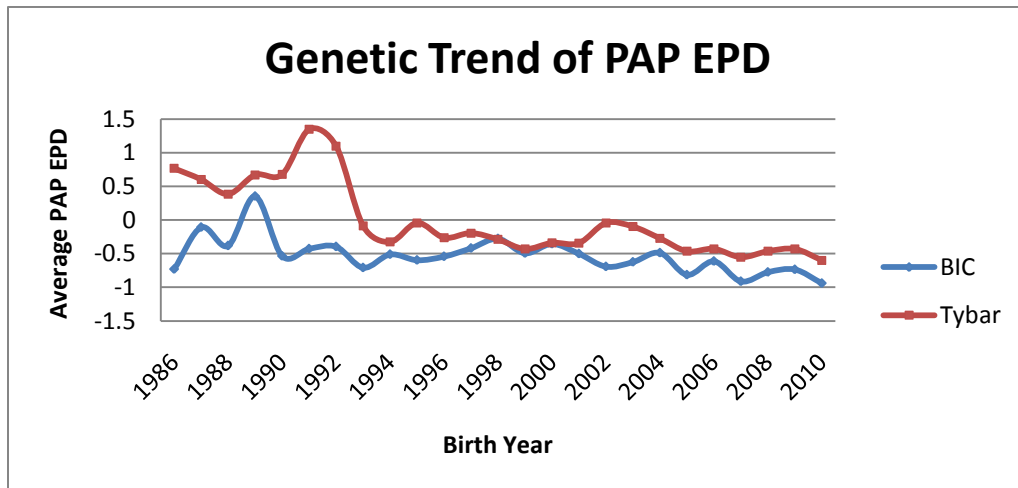


Figure 1. Genetic trend in pulmonary artery pressure at the Tybar Ranch (Tybar) and the CSU John E. Rouse Beef Improvement Center (BIC) since selection with EPD began in 1992 (Tybar) and 2002 (BIC).

The Colorado State University Beef Improvement Center near Saratoga, WY has used EPDs in their selection program since 2006 with a similar favorable response, albeit slower. The reduced rate of progress is a result of the use of the facility as a test herd for evaluating for sires from elevations below 5500 feet.

The limitation of the PAP test is that animals are required to reside above 5500 feet for at least 30 days before the test is performed. This requirement limits the quantity of data that can ultimately be collected and costs other than the cost of data collection itself often preclude testing of animals whose native environment was less than 5500 feet of elevation. This limitation is not uncommon as one of the difficulties often associated with environmental health challenges—often, as in this case, the animals must be in that environment to determine susceptibility to the specific environmentally induced disease. This limitation illustrates the need to develop appropriate indicator traits genetically correlated to the traits of interest and/or to develop genetic marker panels explaining sufficient genetic differences in susceptibility. DNA marker tests would allow for screening of animals from lower elevations to identify those most likely to produce progeny adapted to high elevations.

Pathogen-Associated Disease

The class of animal health traits associated with pathogens pose similar difficulties for genetic improvement. The challenge lies in identifying the economically relevant traits, associated

indicator traits, available data, and DNA marker tests to enable the implementation of genetic evaluation.

Collection of field data for pathogen-associated diseases is especially problematic as there are typically concerns with whether animals were equally exposed to the disease causing pathogen and therefore had the opportunity to fully express genetic differences. Additionally, questions often arise as to whether animals were correctly diagnosed, the severity of the disease, and appropriate causative pathogens identified.

Given these difficulties, initial selection of disease traits for development of genetic predictions should address those of highest economic importance. Estimates suggest prevention and treatment of disease in the feedlot costs the industry in excess of \$3 billion (Griffin, 1997). More specifically, with Bovine Respiratory Disease Complex has been shown to be increasing in prevalence and is responsible for a large portion of these costs (Loneragan, et al. 2001; Callan and Garry, 2008). Besides these costs associated with its prevention and treatment, BRDC has also been associated with decreased feedlot and carcass performance (e.g. Schneider et al., 2009; Snowden et al., 2006 and 2007). As such, BRDC, has become a high priority for development of tools for genetic improvement

Given the economic importance of BRDC to the cattle industry, Colorado State University in conjunction with Pfizer Animal Genetics and JBS Five River's Cattle Feeding developed a research project to identify selection tools aimed at reducing the incidence of this disease. This project illustrates both the potential for genetic improvement of pathogen-associated disease and the difficulties associated with collecting field data on disease traits.

The project was conducted over 2 years with 1551 steers fed in year 1 and 1319 fed in year two of the study. Animals exhibiting clinical signs of BRDC as determined by commercial feedlot personnel were treated following feedlot protocols and classed as positive for BRDC. Treatments for other feedlot diseases such as pinkeye and bloat were also recorded. Sires of calves were identified with DNA markers and that parentage information was subsequently used to estimate heritability.

BRDC treatment rates were 45% and 7.1%, in year 1 and 2, respectively illustrating the difficulties associated with collection of field data given variable rates in incidence across contemporary groups.

Even with the contemporary group differences present in this study, the probability that animals were treated for BRDC was 17% heritable based on BRDC treatment records in this population and the probability that an individual was treated for **any** health related problem as 24% heritable. "Any" treatment would include treatments for foot rot, pink eye, bloat, etc. To put these values in perspective, the heritability of heifer pregnancy is often in this range as is the heritability of milk production in a number of beef cattle breeds. While not high, both would indicate that there is genetic variability associated with pathogen-associated disease traits.

Collection of treatment data on sire-identified feedlot cattle on a large scale would likely be problematic so our approach has been a two-pronged effort by both evaluating potential indicator traits and determining if DNA marker tests could be developed to predict susceptibility to BRDC.

Conclusion

In each of the three categories of health-related traits there is evidence for genetic control. In two of the three categories, selection tools have been successfully developed and marked genetic progress made. In the category of pathogen-associated disease, while genetic variability exists,

developing data collection systems and DNA marker tests will be critical to the delivery of selection tools to the beef industry.

References

Callan, R. J. and F. B. Garry. 2002. Biosecurity and bovine respiratory disease. *Vet. Clin. North Am. Food Anim. Pract.* 18:57-77.

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. Am. Soc. An. Sci.* 43: 111-112.

Griffin, D. 1997. Economic impact associated with respiratory disease in beef cattle. *Vet. Clin. North Am. Food Anim. Pract.* 3:367-377.

Loneragan, G. H., D. A. Dargatz, P. S. Morley, and M. A. Smith. 2001. Trends in mortality ratios among cattle in US feedlots. *J. Am. Vet. Med. Assoc.* 219: 1122-1127.

Schneider, M. J., R. G. Tait, Jr., W. D. Busby, and J. M. Reecy. 2009. An evaluation of bovine respiratory disease complex in feedlot cattle: Impact on performance and carcass traits using treatment records and lung lesion scores. *J. Anim. Sci.* 87:1821-1827.

Snowder, G. D., L. D. Van Vleck, L. V. Cundiff, and G. L. Bennett. 2006. Bovine respiratory disease in feedlot cattle: Environmental, genetic, and economic factors. *J. Anim. Sci.* 84:1999-2008.

Snowder, G. D., L. D. Van Vleck, L. V. Cundiff, G. L. Bennett, M. Koohmaraie, and M. E. Dikeman. 2006. Bovine respiratory disease in feedlot cattle: Phenotypic, environmental, and genetic correlations with growth, carcass, and longissimus muscle palatability traits. *J. Anim. Sci.* 85:1885-1892.