Genomics and Single Genes

Available for **Beef Cattle**

Jason Reding

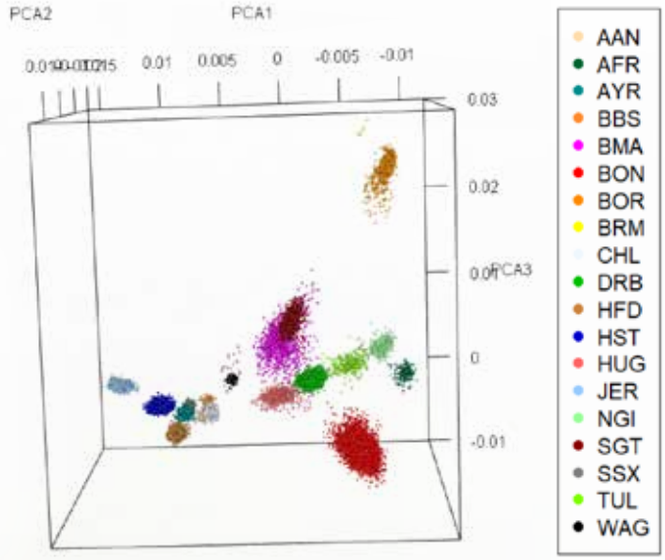
INTRODUCTION

Genomics in livestock has made tremendous advances in recent years. The ability to capture an animal's unique DNA sequence has become a cornerstone of stud breeding in the local and international community. Over 50 000 DNA markers, called single nucleotide polymorphisms (SNPs), are analysed on a genomic chip (genotype) and is sometimes referred to as an "All in One" DNA test.

The use of genomic information in beef cattle has a significant impact on increasing the prediction accuracy of production and fertility traits that are lowly heritable, sex-limited and are only measured later in life (Berry et al., 2014; Van Marle-Köster & Visser, 2018). Although this aspect of genomics may be limited to breed societies with routine genetic evaluations, other vital cases should not be overlooked by commercial and informal livestock breeders.

GENEBOX

A three-dimensional "Genebox" can reveal the true depth of genetic variation between Indicine and Taurine breeds, as well as the corresponding composites. Each dot represents a single animal's genotype, with animals from the same breed



clustering together. A larger cluster indicates a larger amount of genetic variation, while breeds that cluster closer together share ancestry, such as the Sussex, Hereford, and Angus breeds, all of British Taurine origin. The Brahman and Taurine breeds cluster furthest from each other due to the opposing Indicine and Taurine genetics present in these breeds. The Nguni (in green), a Sanga-type breed, clusters closely with the Tuli and Afrikaner Sanga-type breeds (Reding et al., 2021).

SINGLE GENES

Genes have the ability to mutate or change. The common form of a gene is called the original or 'wild type'. Some genes have many different variations, but an animal can only have two of these variants, one copy from the sire and one from the dam. These genes can be the same variant (homozygous) or different variants (heterozygous). If the animal is heterozygous (a carrier), one gene may dominate. (Animals with a polled gene and a gene for horns will be phenotypically polled as the polled gene dominates the horn gene).

Traits are rarely 100% influenced by a single gene; there are usually interactions with other genes and the environment that may (slightly) alter the phenotype. The single gene mutations mentioned in this article are available for further viewing on the Online Mendelian Inheritance in Animals (OMIA) website (Nicholas et al., 1995).

POLLEDNESS

The genetics of polledness has been of major interest to cattle breeders and livestock geneticists. The revolution of genomics, has allowed for the unravelling of the true genetic mechanism that controls the expression of horns or polledness. Referred to as the POLLED gene, this gene is located on Chromosome 1 of the cow genome. The first mutation was identified in Brahman cattle and is called the Celtic variant. This is the main mutation causing polledness in beef and dairy cattle worldwide, and it was confirmed in the Bonsmara and Drakensberger breeds by (Grobler et al., 2018).

A few researchers noticed that some cattle breeds were still phenotypically polled but were homozygous horned (HH) for the Celtic variant. This indicated that a different mutation on the POLLED gene may be responsible. Further research has revealed three other variants within this POLLED gene, such as the Friesian variant in Holsteins, the Mongolian variant in Kazakh breeds, and more recently the Guarani variant in Nellore and Gyr breeds (Nicholas et al., 2023). Currently, all four of these POLLED gene mutations can cause polledness in all cattle breeds.

DOUBLE MUSCLING

The myostatin protein regulates the normal growth and development of muscle in an animal's body. A few mutations can disrupt myostatin's ability to function normally, causing muscles to grow uncontrollably, resulting in an animal being double-muscled. Q204X and nt821 mutations cause a break in the myostatin protein, and affected animals will be double-muscled. The F94L mutation is touted as the "for profit gene" as it does not break the myostatin protein but rather affects its ability to bind to muscles and ensure normal muscling. These mutations behave recessively, meaning it is almost impossible to identify carriers of double-muscling mutations, as they only appear in an affected calf, which is a result of mating two carrier animals (Csürhés et al., 2023).

Due to the direct effect on heavier birth weights, dams with double-muscled calves may experience calving difficulties. Affected animals will have less fat deposition, leading to lower lifetime production, fertility, and longevity. It is recommended to ensure that sire bulls are "Free" from these mutations to minimise and potentially eradicate incidences of double muscling in your herd.

MEAT TENDERNESS

A recent study in South African beef cattle breeds revealed that a few mutations on the Calpastatin and Calpain genes affect meat tenderness during the conversion of muscle to meat in the post-slaughter process (Basson et al., 2022). Certain mutation combinations result in an animal being genetically predisposed to having more tender meat. These mutations can be combined to reveal a tenderness score, which ranges from 1 (Average) to 6 (Most Tender).

MILK-RELATED MUTATIONS

The genes significantly associated with milk traits in dairy cattle breeds are also reported to affect these traits in beef cattle breeds. The combined effect of these single genes and other quantitative genes will influence the quantity and quality of the milk the dam produces and can alter the weaning weight of the calf. The ABCG2 is known to increase milk volume while decreasing milk fat kg and milk fat percentages. DGAT1 is a major gene in beef cattle with a relatively large additive effect. It mainly increases fat in milk, intramuscular fat, and carcass fat.

Three genes affect the milk-protein level of the milk, with Beta-Casein, also known as A2 milk, increasing milk volume and milk-protein production. The Kappa-Casein A-variant increases milk production, while the B-variant reduces milk-protein percentages. Beta-Lactoglobulin Haplotype A will increase milk volume, while Haplotype B increases milk fat and milk-protein composition.

DETRIMENTAL MUTATIONS

Genetic disorders such as Crooked Tail, Mule Foot and Ehlers-Danlos Syndrome (connective tissue disorder) cause phenotype deformations that are easily identified. Animals can be mistaken as weak or poor calves, when in fact, they may have a genetic disorder. Glycogen Storage Diseases (GSD and Pompes) are caused by mutations that prevent the storage of glycogen as an energy source and are known to segregate in breeds with Brahman ancestry. Mucopolysaccharides (NAGLU) is a lysosomal storage disease where affected animals may appear drunk due to a staggering gait.

Other detrimental mutations that are observed genomically, mainly for the dairy breeds, include Arachnomelia, Bovine Leukocyte Adhesion Deficiency (BLAD), Bovine Spinal Dysmyelination (BSD), Bulldog Dwarfism, Citrullinemia, Complex Vertebral Malformation, Deficiency of Uridine Monophosphate Synthase (DUMPS), Idiopathic Male Sub-Fertility (IDMS), Mule Foot (Syndactyly), Spinal Muscular Atrophy and Weaver Syndrome. By ensuring your sire bulls are free from these detrimental mutations, you can minimise the risk of these disorders and ensure a future free from genetic disorders in your herd.

FUTURE APPLICATIONS

As research constantly innovates, genomics can assist in revealing the true underlying genetic mechanisms that influence adaptability and fertility traits. Infertility haplotypes, genes which cause embryo or foetal re-absorption, are a widely known problem in the dairy cattle industry, with recent studies revealing similar haplotypes in Limousin and Simmental beef cattle populations.

Genomics will allow for the detection of gene regions that influence adaptation and resistance to certain diseases, assisting farmers with selecting and breeding animals suitable for seasonal changes in environment and disease presence. A common misconception widely stated is that “Genomics is the future”, when genomics is now available in South Africa with current and relevant applications.

REFERENCES

Basson, A., Strydom, P. E., van Marle-Köster, E., Webb, E. C., & Frylinck, L. 2022. Sustained Effects of Muscle Calpain System Genotypes on Tenderness Phenotypes of South African Beef Bulls during Ageing up to 20 Days. Animals 12 https://doi.org/10.3390/ani12060686.

Berry, D. P., Wall, E., & Pryce, J. E. 2014. Genetics and genomics of reproductive performance in dairy and beef cattle. Animal 8, 105–121.

Csürhés, T., Szabó, F., Holló, G., Mikó, E., Török, M., & Bene, S. 2023. Relationship between Some Myostatin Variants and Meat Production Related Calving, Weaning and Muscularity Traits in Charolais Cattle. Animals 13 https://doi.org/10.3390/ani13121895.

Grobler, R., Visser, C., Capitan, A., & van Marle-Köster, E. 2018. Validation of the POLLED Celtic variant in South African Bonsmara and Drakensberger beef cattle breeds. Livest Sci 217, 136–139.

Nicholas, F. W., Tammen, I., & Sydney Informatics Hub. 1995. Online Mendelian Inheritance in Animals (OMIA). https://doi.org/https://doi.org/10.25910/2AMR-PV70.

Nicholas, J. E., Tammen, I., & Sydney Informatics Hub. 2023. OMIA:0004839913: Online Mendelian Inheritance of Animals (OMIA).

Reding, J. J., Theron, H. E., & van der Westhuizen, R. R. 2021. Genebox: A three-dimensional tool to visualise cattle breed differences through principal component clustering. Page: 54 in 52nd Annual SASAS Congress. Pretoria.

Van Marle-Köster, E., & Visser, C. 2018. Genomics for the advancement of livestock production: A South African perspective. S Afr J Anim Sci 48 https://doi.org/10.4314/sajas.v48i5.2.