Myostatin (MSTN)

What is it?

Myostatin (MSTN) is a hormone produced by muscle cells that inhibits muscle differentiation and growth. This influences the production of proteins which controls muscle development. When an animal is identified to be affected for the mutation, they have inactive genes to control muscle growth as effectively which leads to increased muscle mass.

MTSN Affected animals

Animals that inherit the condition express an extreme muscling appearance including abnormally large, wide and rounded rump and thighs with prominent creases between muscle groups. Which is often referred to as "double muscling". While double muscled animals typically have increased retail beef yield, they are also typically leaner (less fat/marbling). Six myostatin variants have been associated with "loss of function", which means they can result in high levels of calving difficulty, lowered fertility and longevity, and may also have heavier birth weights (Bellinge, Liberles, Iaschi, O'Brien, & Tay, 2005). Three variants have been reported to cause an increase in muscle fibre and have been associated with more tender meat but have no impact on calving difficultly, fertility or longevity (Bellinge, Liberles, Iaschi, O'Brien, & Tay, 2005).

Carriers (only carry one allele) for myostatin are likely to be better muscled than unaffected animals (no copies of the mutation), but less so than affected animals. Affected animals (double muscled) can occur when two carriers are mated. Animals that inherit no alleles for the mutant are unaffected and are unable to pass the mutation onto their offspring. In most breeds homozygotes for disruptive mutations have significantly higher phenotypic expression (Miranda, et al., 2002)

However the mutation does not have to be present in the homozygous condition in order for an effect to be detected. A study by Casaset al.(1998) found that animals inheriting a single copy of the mutant allele from a crossbred Belgian Blue, or crossbred Piedmontese sire had increased longissimus muscle area and retail yield, and reduced external and intramuscular fat deposition compared with animals receiving no copies of the mutant (Bellinge, Liberles, Iaschi, O'Brien, & Tay, 2005). Below in table 1 displays the probability of mating outcomes based on animal's genotype.

Table 1 - The table below shows the chances of inheriting depending on the status of the parents:

2 Homozygous Parents	→	100% chance of Homozygous offspring
1 Homozygous Parent 1 Heterozygous Parent	→	50% chance of Homozygous offspring 50% chance of Heterozygous offspring
1 Homozygous Parent 1 Non-carrier Parent	→	100% chance of Heterozygous offspring
2 Heterozygous Parents	→	25% chance of Homozygous offspring 50% chance of Heterozygous offspring 25% chance of non-carrying offspring
1 Heterozygous Parent 1 Non-carrier Parent	→	50% chance of Heterozygous offspring 50% chance of non-carrying offspring
2 Non-carrier Parents	→	100% chance of non-carrying offspring

NOTE: Non-carrier parent (unaffected animal), Homozygous Parent (affected animal, carries two alleles for the mutation), Heterozygote (animal is a carrier, only carries one allele)

Testing cattle for myostatin

Neogen Australasia offers testing for 9 myostatin mutations that have been identified in beef cattle. It is best to contact your breed society to determine which MTSN variant is relevant to your breed.

References

Bellinge, R., Liberles, D., Iaschi, A., O'Brien, P., & Tay, G. (2005). Myostatin and its implications on animal breeding: a review. *Animal Genetics, 36*, 1-6.

Miranda, M., Ménissier, F., Cañon, J., Vallejo, M., Boscher, M., & Dunner, S. (2002). MYOSTATIN GENE POLYMORPHISM AND DOUBLE MUSCLING EXPRESSION IN CATTLE BREEDS : PRELIMINARY RESULTS. *Exploitation of molecular information in animal breeding*, 19-23.

More information

OMIA 000683-9913 : Muscular hypertrophy (double muscling) in Bos taurus: https://omia.org/OMIA000683/9913/

Myostatin genes explained at Aberdeen-Angus AGM: https://www.aberdeen-angus.co.uk/news-and-features/myostatin-genes-explained-at-aberdeen-angus-agm/