MEMBERSHIP TIPS

by Jerry Cassady
director of member services

M1 announcement

With the recent announcement regarding the carrier status of two very old, heavily used Angus sires, Myostatin nt821 Gene Deletion (M1 genetic condition) has garnished increased awareness and discussion.

History

The first written account of bovine muscular hypertrophy (double muscled) was by a British farmer named George Culley in his stock almanac back in 1807.

He described the muscular phenotype as "double-layered" and "without fat about it." A more modern definition states affected animals are extremely heavily muscled in appearance, including abnormally large, wide, and rounded rump and thighs with prominent creases between muscle groups. There is usually little fat covering, and bones are thin.

In 1972, during collaboration between the American Angus Association and genetic scientists at Kansas State University, the double-muscling trait was included within the genetic defect policy established and approved by the Association. This approved policy included observation, reporting and monitoring of several genetic conditions. More recent science has determined those animals that expressed this bovine muscular hypertrophy within the Angus population have the nt821 (M1) deletion in the myostatin genotype. It is this deletion within the bovine genome that causes the double-muscled phenotype.

Definition

The M1 deletion is caused by a simple recessive mutation. This deletion is known to promote growth of skeletal muscle and cause muscular hypertrophy in various breeds of cattle.

Cattle that are homozygous (inherit both copies of the trait) for the mutated gene will exhibit the M1 double-muscled phenotype.

Cattle that are heterozygous (inherit one copy of the trait) for the mutated gene will appear normal, but will be carriers of the M1 deletion. Because of their normal appearance, carriers of M1 used in breeding programs (registered or commercial) are unknowingly responsible for propagating the recessive mutation within the cattle population.

Double-muscled animals produce very lean meat with greater muscle mass. Some may see this as advantageous; however, the main reasons this phenotype is selected against is due to the reduction in quality grade and increased incidence of dystocia (calving difficulty).

Why now?

The Myostatin nt821 gene deletion was recognized as a strain of double muscling in the summer of 2011. Although, as we just discussed, this was not the first experience with double muscling for the Association. Breeders have spent decades placing selection pressure against this trait, and incidence of this phenotype in the U.S. Angus population is rare.

Recent DNA testing of two very old, high-use Angus bulls revealed their carrier status of the M1 genetic condition. Therefore, although not a new genetic condition for Angus producers, breeders may be seeing an increase in M1 potential carriers within their AAA Login accounts.

Identify M1 carriers

Although testing for the M1 condition is not required for registration, a commercial DNA test is available to determine if an animal carries the M1 mutation.

Members are advised to utilize the carrier management tool available within AAA Login for direction to strategically test older genetics first to clear descendant potential carriers within your herd (see Membership Tips, May 2022).

Summary

Myostatin was initially identified as the factor causing the double-muscling phenotype due to the presence of mutations inactivating the gene, and therefore, leading to the loss of the ability to stop muscle fiber growth.

The carriers have the gene deleted on one of the chromosome pairs; the affected animals have the gene deleted on both chromosomes, and muscle growth is then not properly regulated.

Recent action by the Association Board of Directors requires artificial insemination (AI) sires be tested for the full suite of known commercially available tests for genetic conditions monitored by the Association to be AI permitted. This assured many descendants to be clear of genetic conditions such as in this case of M1.

That action, coupled with decades of selection pressure against the M1 trait and the fact that the Angus breed has superior quality grades and a very low incidence of dystocia, we know the incidence of the double-muscled phenotype within the Angus population has been suppressed. AJ

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Editor's note: For more information regarding the M1 announcement and subsequent steps to test potential carriers, please contact the Member Services Department at (816) 383-5100 or email me directly at jcassady@angus.org.

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Important notice regarding M1 test results for Algoma Bardoliermere 46 and Bovimela Baros 13A:

The American Angus Association has recently become aware that Angus sires Algoma Bardoliermere 46, AAA 3617847, and Bovimela Baros 13A, AAA 7029686, are carriers of the Myostatin nt821 Gene Deletion (M1). M1 is a non-lethal genetic condition caused by a recessive mutation on a single cattle chromosome. M1 was recognized as a strain of double muscling June 20, 2011. There are no restrictions for registration regarding the M1 condition. However, animals may be added to your potential carrier list for M1 based on these results. A testing option is available for the condition. The rules associated with genetic conditions can be found in Part 3 of our *Breeder's Reference Guide*.