Double Muscling in Shorthorns

by Dale Asser

The term "Double Muscle" is coming up more often these days when breeders are describing cattle they have or have seen with a very heavily muscled appearance, especially abnormally large, wide and rounded rear quarters.

Double muscling does not actually mean that an animal has two muscles in place of a normal one. However, due to a genetic condition, these animals have a greater number of muscle fibres which can result in a much larger than normal muscle mass (as much as 20 - 40% larger). The double muscle condition which is common in many mammals comes from a genetic abnormality in the production of "myostatin", which is a protein found in a gene that regulates muscle growth. In normal animals, myostatin tells the muscles to stop growing when they reach a particular size. However, the defective gene lacks the myostatin protein and therefore allows the muscle to keep growing.

The most obvious departure from normality in the double muscled animal is the enlargement of the skeletal muscles which is most apparent in the rump area but also across the shoulders. There are, however, other distinguishing features which may be obvious to varying degrees. They are:

- prominent creases between the muscle groups, especially in the rump
- minimal fat cover, which results in muscle definition appearing more pronounced
- thinner bones, especially in the front legs
- females may have a masculine appearance
- in addition, other less common abnormalities have been reported such as: enlarged tongues in newborns, underdeveloped genitals and increased susceptibility to respiratory disease.

Nine Myostatin mutations (also called variants) of the gene have been identified. Some variants are found to be more prominent in certain cattle breeds. Some of those identified in Canadian breeds are mentioned below. Six of the nine variants are classified as 'disruptive' as they cause the double muscling affect along with, among other traits, larger birth weights and increased calving difficulties. The other three variants are referred to as 'missense' or non-disruptive and while they increase muscularity with reduced external and intramuscular fat, they do not change birth weights.

The Nine Identified Myostatin Variants:

a) Disruptive Variants
   C313Y
   E291X
   nt419 - sometimes found in Shorthorns
   nt821 - most common variant found in Angus
   0204X
   E226X - most common variant found in Shorthorns

b) Non-Disruptive Variants
   D182N
   F94L - most common variant found in Limousin & sometimes Shorthorn
   S105C

DNA tests can be performed to determine if an animal has one or more of the myostatin variants or is free of all variants. Test results will usually be presented as in the following examples:

0 = no variants present
1, E226X = 1 copy of the variant is present
2, E226X = 2 copies of the variant are present
1, E226X; 1, nt419 = 1 copy each of 2 different variants are present

Predictable breeding results from mating sires and dams who do and do not have a Myostatin defect variant present in their DNA can be grouped as follows:

0 x 1,V = 50% have 0 copies; 50% have 1 copy
1,V x 1,V = 25% have 0 copies; 50% have 1 copy; 25% have 2 copies
0 x 2,V = 100% have 1 copy
1,V x 2,V = 50% have 1 copy; 50% have 2 copies
2,V x 2,V = 100% have 2 copies

The myostatin defect is not classified as a simple recessive gene defect, like TH, where a carrier is an animal having no physical affect, but 'carries' the defect (essentially hidden) and can pass it on genetically to offspring. The myostatin defect is not classified as recessive as it affects all that are not '0' for variants. However, that physical affect could be almost physically undetectable all the way to very obviously affected, to the point of looking deformed. Typically, it would be expected that those with one variant would show less increased muscularity than those with two variants.

Undoubtedly you have heard of the Belgian Blue and Piedmontese cattle breeds in particular who consciously breed for the myostatin defect in their cattle in order to produce the extra muscle that results in greater carcass yield. As with many defects there are often associated traits that may be positive or negative. Such is the case with the myostatin defect. While it can be negatively associated with heavier birth weight, calving difficulties, thinner bones and less fat cover, it is also linked to traits that can be seen as positive, such as leaner meat (less intramuscular fat), more tenderness, larger rib eye measurement and higher carcass yield.

For reasons arising from some of the traits listed above, breeders are not always immediately dismissive of the

myostatin defect as they may feel they can breed for the positive effects and manage the negative downsides. As the Shorthorn breed has traditionally been known for and sought after for predominantly its maternal traits, it is important to keep this in mind if we want to attempt to manage this defect in order to increase carcass yield. A common complaint over the past few decades has been the Shorthorns' high birth weights and calving difficulties, and it would seem quite probable that the myostatin defect, unchecked or poorly managed, would quickly turn back the clock on the advances the Shorthorn breed has made in recent years to improve birth weights and calving ease, especially among commercial buyers.

Canadian Shorthorn Association Recommendations:

* Myostatin test your bulls and donor females and any other cows you suspect may carry the defect due to their appearance or known affected animals in their pedigree. Myostain tests can be done by through Delta Genomics with simple hair samples. You can contact the registry to order the test for you, especially if are doing parentage verification or other tests at the same time, or if you already have a sample on file at the lab – most likely that stored sample can also be used to test for Myostatin. The test can also be ordered directly from the lab by accessing the forms on the CSA or Delta websites.

* List ALL of your Myostatin test results in the CSA database where it will be displayed in the online pedigree (send a copy of your results to Belinda at the CSA office.)

* Before you buy a bull, female, semen or embryos, ask the seller if the animal or its parents have been tested; if not, check out their pedigrees to determine if there is a possibility of a variant having been passed on from past generations.

* Resist selling animals who have tested positive for the Myostatin variant, especially bulls (an 'extra meaty' looking bull in your yard may be very alluring to a potential buyer, but if that bull is positive for a Myostatin variant, the calving problems off that bull may lose that buyer and everyone he knows as a customer).

* If you feel you can personally manage the potential downsides of retaining a myostatin positive female (calving difficulties in particular), be sure you are breeding to a myostatin free bull and always test the progeny. ◆