Carey Coombs

Beef Shorthorn Cattle Society

**DIRECTOR**

# Double muscling, myostatin and the Beef Shorthorn

**All cattle breeders will be aware of double muscled cattle, particularly the Belgian Blue. This breed has been selectively bred for what is commonly known as double-muscling. This is the manifestation of a condition known as myostatin gene mutation. There are nine known myostatin mutations (also known as variants). Many breeds exhibit phenotypic trait expression that is a direct result of the presence of one or more of these variants, including the Limousin, the Angus and the South Devon.**

The Beef Shorthorn Cattle Society directors are also aware that there are examples of cattle within the Beef Shorthorn breed that appear to be exhibiting characteristics associated with a myostatin mutation. This is not a surprise given that the first documented case of double muscling was 200 years ago in Durham cattle.

The presence of the highly muscled phenotype in the current population is of concern to the board of directors, whose role is to safeguard the integrity and function of the breed and its role in the British farming industry. They have therefore commissioned Weatherby's Scientific, who already carry out the Society's DNA profiling, to undertake a comprehensive survey of sires – and potential sires – in order to ascertain the variety and prevalence of myostatin variants within the breed.

Results to date indicate a significant number of cattle carry one copy of the variant E226X and that a smaller number carry a double copy. In addition, a small number have been found to carry single copy of variants nt419, F94L, and nt821. An explanation of the significance of these results is outlined below.

The double muscle condition arises from a genetic abnormality in the production of 'myostatin', which is a protein that acts on muscle cells' autocrine function to inhibit myogenesis: muscle cell growth and differentiation. It is encoded by the MSTN gene. An animal with the defective gene lacks the myostatin protein and hence muscle growth is unregulated.

The most obvious departure from normal in the phenotype of a double muscled animal is the enlargement of musculature, particularly in the rump and shoulder areas. There are however other traits which may or may not be significant, more particularly where the syndrome is fully expressed:

- *Prominent creases between muscle groups*
- *Minimal fat cover and modified fat composition*
- *Shorter, thinner and less dense bones*
- *Delays in puberty, reduced fertility and reduced milk production*
- *Increased likelihood of dystocia*
- *Enlarged tongues in new born calves*
- *Poorly developed genitals*
- *Increased susceptibility to respiratory disease probably due to increased demands on aerobic metabolic activity*
- *Increased meat tenderness and yield*

There are nine known variants of the myostatin mutation:

- *C313Y common in the Piedmontese*
- *E291X common in the Marchigiana*
- *nt419 sometimes found in Beef Shorthorns and occurs in the Maine Anjou*

- *nt821 Belgian Blue, Blonde, Limousin and South Devon – the most common variant in the Angus*
- *Q204X Charolais, Limousin*
- *E226X – the most common variant found in the Beef Shorthorn and the Maine Anjou*
- *D182N*
- *F94L found in Limousin and sometimes the Beef Shorthorn*
- *S105C*

At present there is little research into the variations of phenotype arising from any of these variants in the Beef Shorthorn. In breeds like the Beef Shorthorn where selection has historically been 'balanced', the effects of the mutation may be less pronounced than in other breeds. In addition, the mutations do not operate in isolation, but interact with other genes in ways that are, as yet, poorly understood. It is often observed that one copy of a variant does not necessarily give rise to a deleterious phenotype. There may, for example, be an increase in musculature without obvious signs of any disadvantageous side effects.

It is therefore inappropriate to take a simplistic approach and disqualify all cattle carrying a variant from the herd book. In addition, not all well-muscled Beef Shorthorns will carry any of the mutations.

Test results are presented as having no copy present (0), one copy (1) or two copies (2).

The results of crossing between sires and dams, who do, and do not, have a mutation can be grouped as follows:

| | |
|---|---|
| **0 x 1** | **50% no copy 50% one copy** |
| **1 x 1** | **25% no copy 50% one copy 25% two copies** |
| **0 x 2** | **100% have one copy** |
| **1 x 2** | **50% have one copy 50% have two copies** |
| **2 x 2** | **100% have two copies** |

The primary role of the Beef Shorthorn is that of breeding replacement females in the beef suckler herd. In recent years, it has become increasingly common for purchasers to remark on higher than expected birthweights and increased number of calving difficulties. There is the potential for an increased incidence of myostatin mutations to hinder the attempts of breeders to counter these trends. Short term gain in the show ring or at bull sales may not make for long term advantage in a competitive market.

In addition, the prime purpose of breeding pedigree cattle is to fix a 'type.' This gives buyers confidence that the breed selected will perform in the environment in which it was chosen to perform, and commercial buyers the ability to exploit the phenomena of hybrid vigour by crossing with another breed of contrasting 'type'. Hence a breed, whilst expressing some small within-breed variation, will for the most part express a consistency of phenotype. Maintenance of variation in the breed of a genetic trait as fundamental and potentially significant as myostatin, removes this predictability.

For example, a commercial buyer uses a Beef Shorthorn bull to breed replacement females. Unknown to him this sire carries one copy of a myostatin variant. When crossed with another breed, in a first or second cross, which also carries the same or a different variant, a very unpredictable outcome arises, and gives rise to the potential for significant problems and subsequent losses.

On the basis of current knowledge, the Beef Shorthorn Cattle Society recommends:

- *that breeders continue to select for 'balanced' cattle that exhibit traits that the breed has historically been known for, and is increasingly sought after for, using traditional and modern techniques.*

In addition, breeders and their customers should be aware of the implications of using bulls or cows that carry the mutation. Breeders may wish to consider testing as many bulls or cows that they suspect may carry the mutation (single or double copy). They may also wish to check the status of any animal they buy.

If no test result is available, then checking the pedigree may determine if there is a possibility of a variant being passed on. If a breeder feels they can manage the potential downsides of retaining a myostatin positive (single or double copy) female, we would encourage the use of a myostatin free bull and that the progeny be tested.

At present the regulations for all bull calves registered with the Society require that a hair sample is submitted for sire verification and myostatin testing. The myostatin test is carried out at the Society's cost and the results will be published on the database.

All stock sires of calves registered since 1 January 2018 will be tested for myostatin at the Society's cost. If insufficient DNA is available from a previously submitted hair sample, breeders will be requested to resubmit samples.

For clarity, any animal carrying single copies of two different myostatin variants will be eligible for sale and will be registered. This will be kept under review however.

All cattle imported in to the UK Herd Book and any progeny resulting from embryo transfer will also be myostatin tested as part of the registration process.

It should be noted that the Society is required by law to make all genetic testing information freely available. The Society is obliged therefore to publish the results on the ABRI database and to include new information as it becomes available. This applies whether testing is carried out by the Society or by an individual breeder.

The Society will continue to monitor the test results and the effect on breed type. Any changes to regulations or advice to breeders will be communicated to members. It is the clear wish of the Society not to inhibit any particular breeding programme and to give ample time for breeders to adjust to any recommendation or directive.

It is hoped that breeders will take into account the Society's recommendations and that any deleterious impacts of myostatin mutations will be self-regulating.

**Carey Coombs,** *Society Director*