Double muscling – the double-edged sword

Double-muscled animals are highly sought after in some production systems in Europe. A double-muscled animal has about 20% more meat and an excellent dressing percentage of around 50% due to less fat, organs, and bone.

There are several European cattle breeds in which numerous mutations cause homozygous double muscling (carrying two similar double-muscling-causing alleles of the gene), including the Belgian Blue, Maine Anjou, Marchigiana, Piedmontese, Aubrac, and various other breeds. The double-muscled European breeds have also been selected over the years for additional muscling genes. If the detrimental myostatin (MSTN) mutations therefore occur in an unrelated breed, the effect on muscling is not nearly as severe as it is in, for example, the Belgian Blue.

An advertisement for semen of a double-muscled Belgian Blue bull.

In these European production systems, double-muscled animals are kept in small herds, which allows them to receive the special feed and specific care and attention they require due to various health issues associated with the condition. Calves are marketed as veal before weaning. Calving difficulties caused significant issues before caesarean sections became common. Less detrimental variants, such as F94L, were marketed as the 'profit gene' because they cause double muscling, but without the high birthweights.

Thus, double-muscled animals have a place under specific circumstances and production systems, mainly in Europe. However, they are ill-suited to extensive production systems in South Africa, the USA, and Australia, primarily because of the cows' inability to calve naturally and the specialised nutrition and care they require. The question then remains: why did the detrimental mutations nt821 and Q204X spread through beef cattle breeds in South Africa?

A carrier of nt821 (left showing prominent muscle development and a free bull right).

Partial recessiveness

MSTN mutations are partially recessive (showing 'incomplete penetrance'). If an animal inherits a normal myostatin gene from one parent and a detrimental myostatin mutation from the other (heterozygous/carrier), it can display degrees of double muscling or exhibit some traits of double muscling and not others. The reason for this is likely interactions with other unknown genes or the environment.

Carriers of Q204X (left) and nt821 (right), which show good muscle definition.

Carrier bulls display good muscle definition due to more muscle cells and less subcutaneous fat, giving them a selection advantage. As carrier bulls were increasingly selected as breeding bulls, the frequency of the detrimental MSTN mutations also increased in many breeds. As the frequency of the MSTN variants has increased over time, muscling induced by MSTN mutations has also become the norm for a 'good-looking bull' among breeders.

Despite the selection advantage conferred by good muscling, the disadvantages of carriers of detrimental MSTN mutations might not always have been realised. For example, for many years, efforts to control calving difficulty included selection based on breeding values or measurement of pelvic size, with little success.

Disadvantages

The detrimental MSTN mutations cause heavier birthweights, which increases the risk of difficult calvings and calves that need to be pulled. Large calves suffocate because they crush the umbilical cord while moving through the birth canal. The cow takes longer to recover and therefore to become pregnant again (longer calving intervals). This is the most significant disadvantage of detrimental MSTN mutations and is often the reason breeders want to eradicate double muscling from the cow herd. This issue particularly affects heifers that are still growing and developing, and are calving for the first time.

The biggest disadvantage of nt821 and Q204X is the severe calving problems and loss of heifers, cows, and calves caused by higher birthweights.

Less fat leads to poorer reproductive performance and other performance metrics in normal production environments due to lower energy reserves, and also reduces adaptability. Reduced subcutaneous fat and marbling present a major disadvantage for heifers and cows: Fat is not only needed for fertility but also serves as an energy reserve for cows, essential for their own and their calves' survival in extensive areas or during droughts, for example.

Extra muscling and a blueish tint due to less fat is visible in the two nt821-carrier carcasses on both sides of a free carcass.

After calving, cows, particularly heifers, are typically in a negative energy balance, meaning their feed intake is insufficient to meet their energy needs. They need to recover from calving so that, ideally, they can become pregnant again after three months. Furthermore, they have a suckling calf that depends on them for growth. Heifers are genetically predisposed to use energy for their own growth, which places an additional burden on them. Less fat also shortens the carcass's shelf life and reduces the meat's flavour.

Left: Low capacity of a Q204X bull due to an underdeveloped rumen and lungs, causing low growth. Right: Straight hocks of an NT821 calf.

Although direct weaning weight might be positively affected, carrier cows tend to produce significantly less milk, which is again negative, especially in drought conditions. Carrier cows are typically culled sooner than cows free from the MSTN gene, primarily due to a tendency toward calving problems, lower condition, and longer calving intervals.

The detrimental MSTN mutations can also cause an underdeveloped reproductive system, leading to lower fertility and longer calving intervals. Heifers develop more slowly – breeders often identify potential carriers by underdeveloped external reproductive organs and udders during inspections, and such heifers are then not incorporated into the herd.

The testes (observed as small in circumference) may also be underdeveloped. Straight hocks in bulls may compromise breeding ability. Animals with underdeveloped lungs may be prone to pneumonia, while an underdeveloped rumen may stunt growth due to an inability to consume enough feed. These animals have a typical 'pencil gut' appearance.

In summary

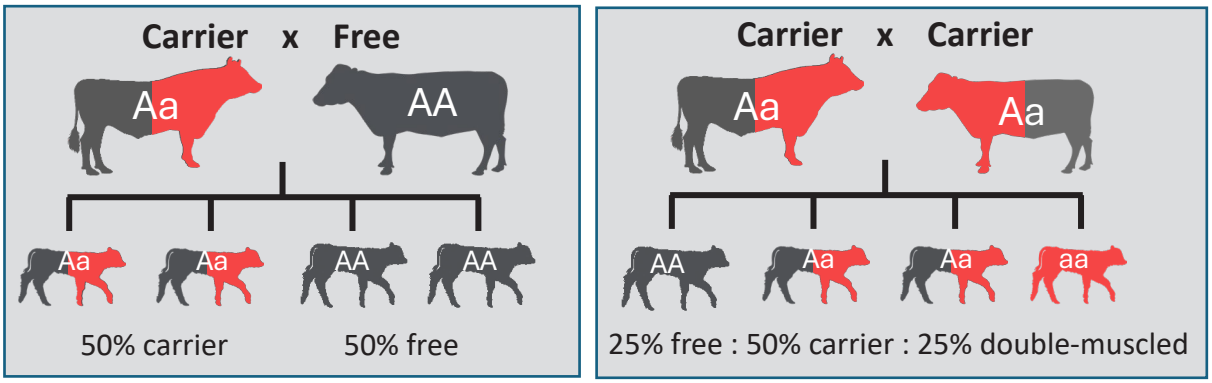
A double-muscled animal is characterised by excessive muscling with minimal subcutaneous fat, a finer bone structure and lower capacity, while fertility is also affected. This is caused by a detrimental mutation in the MSTN gene, with nt821 and Q204X being the most prevalent in South Africa. Double muscling is inherited as a single-gene recessive trait with partial penetrance, meaning carrier animals (heterozygotes) may exhibit some or all characteristics to varying degrees.

A Q204X carrier cow and her double-muscled calf.

Although double-muscled animals are not suitable for extensive production conditions, carrier bulls might have a selection advantage due to their good muscling and heavier weaning calves, which has resulted in the unintentional increase of these mutations in some South African beef cattle breeds, likely introduced through upgrading and/or imported semen.

The biggest disadvantage of carrier animals is an increased risk of calving problems, especially in heifers, due to heavier birthweights. Body fat is essential for the cow herd in the field after calving or during droughts, while milk production and fertility can also be negatively affected in carrier cows.

Figure 1: Inheritance of double-muscling mutations.



Inheritance of double-muscling mutations

The inheritance pattern is easy to remember:

- In all matings with a carrier animal, there is a 50% chance that the offspring will be carriers. If a carrier bull is put among free cows, half of his calves will be carriers.
- If there are already carrier cows in the herd, there is still a 50% chance that the offspring will be carriers, a 25% chance for double-muscled and 25% chance for free calves.
- If one of the parents is free, there is no chance* of a double-muscled calf.
- If both parents are free, there is no chance* of carriers or double-muscled calves.
- A double-muscled calf is only possible if both parents are carriers, and then only with a one in four chance, thus relatively rare. The chance of carriers is, however, 50%. This is also why the gene can 'hide' for generations, until there are many carriers and carrier x carrier matings occur regularly. This leads to more calving difficulty and double-muscled calves being born.

**No chance: Theoretically, there is a very small chance (~1.2 x 10-8 per SNP per generation) that the mutation has spontaneously occurred again. If a carrier calf comes from 'free' parents, the likelihood is much greater that one of the parents is indeed a carrier; that parentage is incorrectly assigned, or that the DNA samples got mixed up. Both parents and the calf must then be tested genomically (not with satellite DNA). – Dr Helena Theron*
(References available from author)