

Myostatin and muscling – Part 2

My last column discussed the fact that the gene pair that encodes myostatin (often referred to as the "myostatin gene") is involved in double muscling. Myostatin, one of the growth factors, apparently inhibits the number and size of muscle fibers in the developing fetus. Therefore, if this gene pair is altered by either natural mutation or by laboratory manipulation, as in the "knockout" mice I mentioned last month, the amount of myostatin produced is reduced or eliminated. The resulting animal is more muscular than normal and is said to be double-muscled.

There is considerable disagreement among researchers as to whether the gene for double muscling is dominant, recessive or neither. The phenotypic expression (visual appearance) of the condition is variable, with affected individuals showing wide differences in muscle development. In attempts to explain this variation, geneticists hypothesize incomplete dominance, incomplete penetrance and various gene modifiers. However, logic would suggest a simpler explanation.

It is my opinion that a single pair of genes controls double muscling in the bovine. Normal animals for this trait are homozygous for the normal gene and are symbolized as *DD*. Double-muscled individuals are homozygous for the doublemuscled gene and are symbolized as *dd*. The heterozygous animal results from crossing a normal individual with a double-muscled one and is symbolized as *Dd*. The offspring tends to be intermediate to its parents in muscular development.

**The total muscular development** of an individual animal is controlled by a large number of gene pairs — the number and chromosomal locations of which are

unknown. This package of genetic material is unique for each individual and should be considered its genetic base for muscling. When two animals are mated, each offspring receives a random half of each parent's genetic base. This explains the wide variation in degree of muscling among cattle.

The genetic base for muscling in a herd or breed of normal animals determines the increase in muscling when the doublemuscled gene is introduced. For example, introducing the double-muscled gene into a population of thinly muscled dairy cattle, such as Holstein, results in less increase in muscling than if the gene were added to a population of heavily muscled cattle, such as Charolais.

The percentage increase in muscle is the same, but the genetic base for muscling is greater in the Charolais than in the Holstein population. Hence, there is a more extreme phenotypic expression of double muscling in cattle carrying a genetic base for heavy muscling.

Because of the negative effects of the double-muscled gene on reproductive efficiency, it must be avoided in commercial breeding females. Likewise, the gene cannot be tolerated in the homozygous condition because of negative effects on carcass quality. However, the gene can be used in the heterozygous form to improve cutability in a sophisticated crossbreeding program.

Many commercial breeders have used a crossbreeding program that employs strains of females superior in maternal traits mated with a terminal-cross bull excelling in growth and carcass characteristics. It is quite logical, then, to use a bull homozygous for the double-muscled gene as a terminal-cross sire. While doing so, the breeder should remember that  $F_1$  females should not be retained as herd replacements.

The breeder of terminal sires must be willing to accept the reduced reproductive efficiency of a double-muscled herd and be certain the terminal sires (or semen) offered the commercial breeder are homozygous for the double-muscled gene.

**The F<sub>1</sub> carcasses lack** sufficient marbling to meet current commodity-market standards for quality (marbling). Further, even though more tender as indicated by shear-force test, the meat lacks flavor and juiciness and requires more-careful preparation. The carcasses require special promotional programs and niche marketing.

Herein lies the major problem. As in any branded beef program with a product deficient in marbling, the supply must meet the demand exactly; otherwise, trouble results. Production of such unique carcasses must be planned three or four years before marketing.

Assume a successful advertising program and strong demand for cuts from certain  $F_1$ carcasses. If supply is less than demand, the customers are unhappy, and the retailer drops the product. If supply exceeds demand, the excess carcasses must go into the commodity market, and because of deficient marbling, sell at a low price.

This explains one advantage of the branded program managed by Certified Angus Beef LLC (CAB). Here, if carcasses fail to grade average-Choice or better, they still qualify for a good price in the regular beef market because the Angus breed's average for marbling is high.

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