Beef Logic

by R.A. "Bob" Long

Myostatin and muscling — Part 1

Cattle breeders soon will add a new word, *myostatin*, to their vocabulary. In 1997 scientists at Johns Hopkins University, working with a group of proteins (hormonelike material) that regulate cell growth and differentiation, discovered the gene that encodes myostatin.

Myostatin is one of a group of growth factors and apparently inhibits the growth of muscle fibers in the developing fetus. In an experiment the researchers "knocked out" the myostatin gene in mice and observed two to three times more muscle in the transgenic mice than in normal mice with the gene intact. Further, histological examination of the muscles revealed those from the "knockout" mice showed an increase in both number of muscle fibers and in size of fibers.

Discovery of these double-muscled mice led to examination of two breeds of double-muscled cattle — Belgian Blue and Piedmontese. These studies revealed mutations in the gene that codes for myostatin, establishing that myostatin performs a similar function in both mice and cattle. Additional work by these and other researchers has identified the gene in pigs, chickens and humans.

These findings have resulted in extensive research around the world in hopes of developing ways to use this mechanism to control human diseases, such as muscular dystrophy, AIDS and cancer. Even regeneration and repair of both skeletal and heart muscle may be possible in the future.

Of particular interest to cattlemen is extensive work being conducted at the Roman L. Hruska U.S. Meat Animal Research Center (MARC) at Clay Center, Neb. Both the Belgian Blue and Piedmontese breeds are being studied in various crosses with cattle having the myostatin gene intact.

Both breeds are homozygous for the double-muscle gene. A single copy of the gene (the heterozygote) improves muscling and reduces fat.

For example, researchers at Clay Center estimate a 7% increase in muscle and a 14% decrease in total carcass fat (including marbling) will result from mating a double-muscled bull with a female carrying normal, active myostatin genes.

Logically, this crossbreeding plan is being considered by commercial breeders as a way to meet market demands for muscular, low-fat, tender carcasses. However, there are disadvantages to this program that come to light on review of what is known about double muscling.

Double muscling (muscular hypertrophy) of cattle is a condition characterized by extremely heavy muscle development, reduced fat deposition and thin skin. The affected animals are not actually double-muscled. They have exactly the same number of muscles as normal cattle, but each muscle is proportionately larger.

At least 10% of double-muscled females never mature sexually, and those that do exhibit late puberty, low fertility, difficult calving (often requiring a cesarean) and poor milk production. Likewise, double-muscled bulls frequently exhibit decreased scrotal circumference, small testicles, and reduced semen volume and quality.

Carcasses from double-muscled cattle are thick and have large ribeyes, as would be expected. These carcasses are lean and have considerably less subcutaneous, seam, kidney and mesenteric fat than those of normal cattle.

Likewise, marbling is practically devoid in double-muscled carcasses, and they rarely grade better than USDA Standard regardless of how long the cattle have been fed. These lean, heavily muscled carcasses yield an extremely high percentage of edible portions (high cutability), and the meat is more tender than normal beef as measured by shear force.

The absence of outside fat, however, allows extremely high shrinkage from moisture evaporation during processing and shipment. The absence of marbling also results in a cooked product that is dry, lacks flavor, and is usually rejected by retailers and consumers.

The double-muscled gene cannot be tolerated in breeding herds because of the negative effect on maternal traits. This effect requires the sale and slaughter of all heifers and a separate breeding program for replacement females.

Further, since the F_1 carcasses (which carry one copy of the gene) lack the marbling to make USDA Choice, and in many cases USDA Select, they must be sold as branded beef and require extensive promotion as a health food to realize a profit. Further, any oversupply would take a beating in the commodity market due to the low quality grade.

The myostatin gene and its elimination or replacement as a tool for changing carcass composition is a complex procedure.

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