

March 4, 2010

**A LITTLE BIT COUNTRY
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Understanding Genetic Defects in Beef Cattle

Recently there has been a surge of inquiries relating to genetic defects in beef cattle.

Most of us were either not around or were too young to have personal experience with the most famous genetic defect in the 20th century—**snorter dwarfism**. It became an issue in Angus and Hereford cattle during the 1940's and 1950's. There have been all kinds of stories about its impact on the industry. Some lead us to believe the industry was about to collapse but over a short period of time, cattle breeders identified the carriers of the defect and were able to develop breeding programs to eliminate its occurrence.

Other genetic defects have since emerged. The two which are receiving the most attention are Curly calf syndrome (Arthrogyrosis Multiplex), also referred to as AM and Ostropetrosis (Marble Bone) or OS.

Most genetic defects are inherited as simple recessives much like the color inheritance of red and black in cattle. Red is recessive gene and black is dominate. For red to occur both parents must have the red gene. Such is the case with AM and OS. Both parents must carry the recessive gene for an animal to be afflicted with the defect.

With AM, the curly calf refers to the shape of the spine when the calf is born. This condition had been reported to the American Angus Association who then solicited the help of Dr.David Steffan of the University of Nebraska to determine the cause.

Osteopetrosis (OS) calves are usually born dead two to four weeks early. Their bones are solid and do not contain marrow, making them very brittle and easily broken. The calves possess a lower short lower jaw and impacted molars. It too is inherited as simple recessive trait. These two defects and others have been identified in most beef breeds. Fortunately university and USDA scientists have developed DNA test procedures to identify carriers of AM and OS even though the animals may appear normal.

Judging from sale catalogues and promotional material, cattle breeders seem to be taking a very proactive stance in using the scientific techniques to identify potential defect carriers in their herds even though they have not experienced animals with any of the defects. Breeders very willingly allow their respective associations access to the test information. At least one breed association acknowledges animals which have tested free (F) or as carriers (C). In this case, I have noticed the list of free animals is much larger than the carrier list. This scientific ability to identify carrier animals will intensify the industry's ability to eradicate genetic defects. However, since mutations of the DNA structure can occur in every generation, genetic defects will never be completely eradicated.

For more on genetic defects I direct you to the Bovine Connection proceedings report of Dr. David Buchanan, Professor of Animal Science at North Dakota State University found on our website: www.ag.ndsu.edu.williamscountyextension

The message I prefer to leave is both the cow and bull must be carriers for either of the two defects to express themselves. Also, an animal that has one undesirable gene may have thousands of very desirable genes. With today's technology, it is possible to use that superior animal and identify superior offspring which are free from the defect and then market the carriers as food product.