# Some guidelines should you find a defective calf

By Jim Cotton
Editor

First. . . .genetic defects surface in all breeds. No known breed is exempt. Strenuous efforts by both breeders and breed associations have been undertaken to search the causes ofcongenital defects and to eliminate them from the population. Progress in this area has been substantial.

However, there still exists the possibility of a defect appearing across the hundreds of thousands of calves born each year.

In its effort to deal more effectively with the problem of genetic defects, the American Angus Assn. supports studies and contributes financially to breeders who will follow the reporting program outlined

**1.** The breeder must make a reasonable effort to determine that the defective calf is affected by a genetic problem. This may require consultation with a veterinarian or other knowledgeable sources. The veterinarian must immediately file a report with the American Angus Assn.

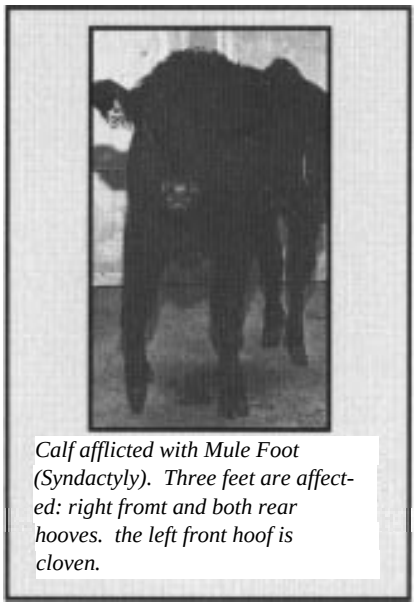
(Not all deformities have a genetic origin. Viral infections have been established as causing congenital defects. There are those uncommon instances pro voked by some abnormality in the uterus and/or the cow's feed or forage. Cases of severe mineral deficiencies, poisonings, or imbalances causing deformities are known. It's also well-documented that feeding on certain plants -- particularly of the lupine family -- can cause the fetus to develop abnormally.

These are usually rare and quite localized. Information and guidance should be sought from extension livestock specialists, veterinarians, or university plant toxicologists for the history of the area in this regard.

Drought is often a culprit triggering these outbreaks. And cattle can develop perverse appetites, particularly if under stress. Cattle licking the tops of discarded car batteries or craving known poisonous plants reveal a fundamental deficiency. Steps should be taken to correct the problem.)

**2.** If possible, blood should be drawn from the defective calf along with blood samples from the sire and dam if they have not been blood typed, in order to definitely determine parentage. Blood typing is conducted by the Cattle Blood Typing Laboratory at Ohio State University, Columbus.

**3.** The breeder should contact Don Painter, at the Association office, who will in turn contact Dr. Horst Leipold of the College of Veterinarian Pathology at Kansas State University, Manhattan. Should Dr. Leipold agree there is a strong possibility of a genetic problem, the calf must then be packed in ice, crated, and sent by Air Freight to the College of Veterinary Medicine, Dept. of Pathology, Manhattan, Kansas.



*Calf afflicted with Mule Foot (Syndactyly). Three feet are affected: right fromt and both rear hooves. the left front hoof is cloven.*

If the distance is not too great, it's recommended the live calf be delivered to the college.

**4.** Do not destroy the animal nor dispose of it or any semen ampules or straws from its sire. Keeping the animal alive is desirable, though recognize there are those genetic defectives exhibiting a short lifespan and high mortality.

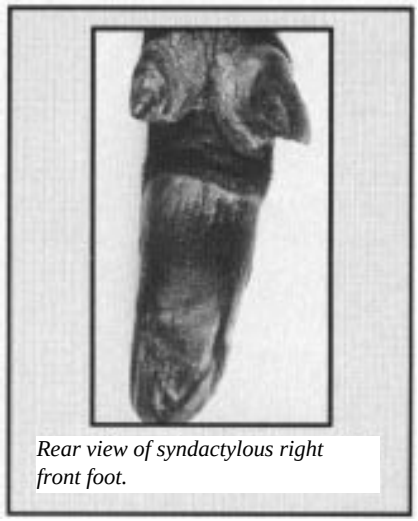
**5.** The breeder must file a complete report of the situation with the American Angus Assn. If the Association feels its program for reporting defects has been followed, within reason, the breeder will at that time be reimbursed in an amount not to exceed $50.

The nationally acknowledged authority on genetic defects is Dr. H. W. Leipold of the Department of Pathology, College of Veterinary Medicine, Kansas State University

Excerpts from a paper written for the *Angus Journal* by Dr. Leipold, Dr. Neal Woollen, and Dr. Dave Steffen of the KSU department of animal pathology are cited in this discussion. Thefocus is on genetic defects that might have some impact on Angus breeders.

Quoting these authors, it's important to realize many congenital defects are of an unknown cause. "Most congenital defects are so rare and of unknown cause that they should not worry a herd owner.

"(Most defects) are caused by homozygosity of a simple autosomal recessive gene. *(Autosomal: a chromosome other than a sex chromosome.)* This means the



*Rear view of syndactylous right front foot.*

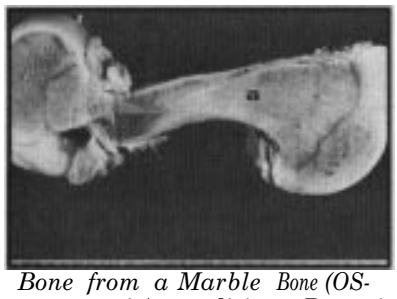
individual possesses two recessive genes for a particular trait. This also means that both parents should be considered carriers for that condition."

The authors point out a catalog of toxic plants that create abnormal fetuses or calves. Most are of the lupine family, Again, local knowledge is best applied in this area of husbandry.

Bovine Virus Diarrhea (BVD) is known to trigger congenital defects. Defective brain development, intra-uterine growth retardation, and impaired immunity are documented from BVD infections.

The Bluetongue virus has been experimentally transmitted to pregnant heifers by insects, report Drs. Leipold,Woollen, and Steffen. Such infections have produced joint contractures, jaw defects, water-on-the-brain, and "dummy calf' syndrome where inactivity, dullness, and behavorial disturbances are exhibited.

This recitation of what might happen should not panic breeders into thinking defects are lurking in every herd ready to spring to life. Their incidence from genetics is extremely low and should not inspire a witch hunt where pedigrees are traced back for decades nor animals with genetic



*Bone from a Marble Bone (OSteopetrosis) condition. Bone is sliced in half showing absence of bone marrow cavity.*

merit are needlessly destroyed. As Darrell L. Wilkes and Dr. Thomas B. Turner, The Ohio State University *Angus Journal,* April, 1982, p. 99) point out:

**"For some reason, most breeders believe that such defects tend to spread—to become more common. Because we cannot see the genes, we cannot detect a carrier without a progeny test or at least a blood test. We are left with the illusion that these carriers are secretly infiltrating our gene pool and that someday they will mount their offensive and wipe out the population. Very simply stated this is not true!**

**"If we take the case of a defect being controlled by only one pair of genes, as is commonly the case, there is no reason to believe that the, 'bad' gene will spread any more than tbe 'good' gene will spread. As long as the carriers have not been endowed with exceptional merit in other traits more so than 'clean' cattle, the frequency of the 'bad' gene will remain constant. In fact, the frequency will probably decrease a bit because we obviously cull all affected individuals and usually cull their heterozygous parents as well."**

The experts from KSU's animal pathology department have developed a checklist they review when a defect is brought to their lab.

"If a defective calf is submitted, histories should include breed, age of parents, parentage of affected and unaffected control calves, geographic region, season, type of pasture, soil type, exposure to or suspected exposure to poisonous plants such as lupine, feeding and management practices, breeding records, medical and vaccination records of the dam, disease status of the herd, periods of stress, drugs administered, congenital defects observed previously, and history of any similar congenital defects in neighboring herds.

Breedina records may be analyzed for evidence of genetic transmission. Autopsies are performed and defects classifed by the body system primarily involved. Serum samples are taken to check for Bovine Virus Diarrhea and other viral antibodies. Samples of brain and other tissues are taken for possible virus isolation..."

"If it is determined that a bull carries an undesirable recessive gene, most breed organizations proceed to label the bull as a heterozygote and remove him from service. If he is not removed, public notice should carry information listing the congenital defect."

**Number of offspring needed to test a sire**

| For a probability level of: | 95% | 99% | 99.9% |
|---|---|---|---|
| **Homozygous abnormalities** (testing for one trait only) | 5 offspring | 7 | 10 |
| **Heterozygotes** (testing for one trait only) | 10 | 16 | 24 |
| **Father-daughter matings** (testing for all undesirable recessive traits | 22 | 35 | 52 |

The American Angus Assn. monitors these defects: Dwarfism, Osteopetrosis (Marble Bone Disease), Double Muscling, Syndactyly (Mule Foot), and Heterochromia Irides (White Eye).

The first four are considered Class I Defects which are lethal or seriously disabling. Heterochromia Irides is a Class II Defect, meaning it is non-lethal and less severe than Class I. The red gene is a Genetic Factor, not a defect.

Here are descriptions of genetic defects monitored by the Association:

**Class I** (Dr. Leipold, *et al):*

1. Mule Foot... "Syndactyly... usually follows a specific fusion pattern. If one leg is affected, it is in the right front followed by the left front. Barely are four feet affected."

2. Dwarfism ... "(t)here is still an occasional dwarf diagnosed. The brachycephalic (short-headed) dwarf is frequently referred to as a 'snorter' dwarf because of its labored breathing."

3. Osteopetrosis ... "(a) recessive hereditary defect characterized by small body size and weight, shortened lower jaw with impacted molar teeth, misshapened jaw, thick cranial bones, lack of development of the skull, and lack of bone marrow cavities. Calved affected...are stillborn at 251 to 272 days of gestation."

4. Double Muscling... "Congenital defects of muscle are common in cattle and all are economically important. Muscular hypertrophy varies widely and few calves have all the characteristics, the most no table being the rounded outline of the hindquarters. The tail is attached more forward than normal. Muscles of the shoulder, back, rump, and hindquarter are separated by deep creases. Necks . . . are shorter and thicker; heads are smaller and lighter. Many double-muscled cattle stand in a stretched position. The long bones tend to be shorter. The tongue may be larger than normal. Additional features are infantile reproductive organs, impaired reproduction, delayed sexual maturity, lengthened gestation, and high birth weight combined with calving problems. Double-muscled calves are less vigorous and are particularly susceptible to bone and joint problems."

Class II

White eye... (Heterochromia Irides is also referred to as Oculocutaneous Hypopigmentation (in scientific literature). Its characteristics are:

1. Deviation of normal coat coloring from black to diluted dark yellow to a rich, dark brown.
2. Any change in the iris of the eye from normal dark black to blue-gray, giving the iris a double-ringed appearance when closely viewed. From a distance the eyes appear white.
3. Nose or muzzle is a pale gray to brown.
4. The underlying skin under the hair coat is gray, not black.
5. The obvious coat coloring abnormality of these cattle gets even lighter with age.

Dr. Leipold, *et al,* describe skin surfaces of the eyelids, ear openings, muzzle, anal, and reproductive orifices, as appearing grayish-brown with afflicted calves.

In conclusion, an approach toward genetic defects is not difficult to develop. Dr. Tom Turner and Jeanne Osborne, OSU graduate student (Letter, *Angus Journal* March 9, 1964) state: "(I)t is our opinion that the controversy over detrimental recessives has become too emotionally charged for objective reasoning in many cases. Granted, we cannot simply forget about them, but we must put them in the proper perspective; There are many traits that we must be concerned with as breeders; we should not get bogged down with concern over rare detrimental recessive genes."

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