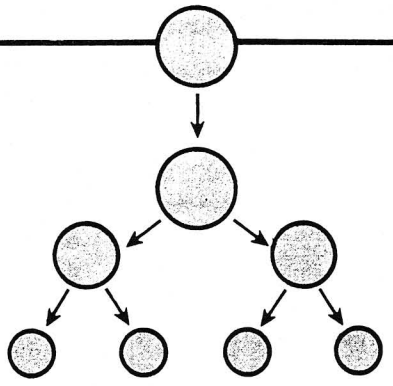
Progress in the purebred cattle industry demands that breeders understand their cattle thoroughly—the weaknesses as well as the strengths.

This article, an update of a series published in the Angus Journal several years ago, is designed to acquaint Angus breeders with genetic defects, problems which occur in every breed of every species. It is not meant to imply that the Angus breed has serious genetic defect problems or more defects than other breeds, because it certainly does not. Instead, the following discussion serves to complement and stress the American Angus Assn.'s open policy of informing breeders.

The policies of the Association were designed so that breeders can work together to keep Angus cattle as genetically clean and trouble-free as they are today. The better we understand genetic defects, the better we can control them. By being open, honest and educated, we can keep defects at an insignificant level and continue to produce the quality seed stock for which Angus breeders are noted.

(The following information was compiled by Marilyn Barr Pieper with the help of Dr. Horst Leipold.)



GENETIC DEFECTS

Part 1 of this article includes an overview of genetic defects and Association policy, then takes a closer look at syndactyly, osteopetrosis, hydrocephalus and heterochromia irides. Double muscling and progeny testing will be detailed in a future issue of the Journal.

Genetic defects occur in all breeds of all species. Inherited defects are, in fact, a natural part of cattle breeding. Genetic change is the tool of purebred breeders striving to improve their cattle. Good genetic changes benefit the entire industry—and outweigh the undesirable changes, such as genetic defects, by far. But any business that makes such rapid progress and change as the Angus business does should expect to face some adversities along the way.

Although occurrence of genetic defects in the Angus breed is minimal, it's impossible to avoid undesirable genes. They are in all living things. Studies indicate that every bull may carry a certain number of bad genes, maybe five or six lethal ones. Studies also indicate that man may carry a mutational load of eight or more hidden recessives than cattle.

Genetic defects affect only a minute number of Angus cattle and an even smaller number of breeders. But they should not be taken lightly. They can cause severe economic losses and setbacks in breeding programs. They can spread insidiously through a breed if not monitored and controlled. And they can hit anyone.

Comparative losses

Calf losses due to infectious disease and environmental problems are much more common than losses due to inherited defects. But modern science and technology enable breeders to control environmentally caused losses more effectively every day. This means genetic defects could increase in proportion and importance in the future if not carefully controlled now.

We cannot eliminate genetic defects, but we can control and breed around those found in Angus cattle. The responsibility lies with the seed stock producer, the purebred Angus breeder.

Genetic defects are results of mutations. Mutations are changes in genetic material—more specifically, changes in the code transmitted by a gene that gives instructions to build a specific protein. Mutations have played an important role in shaping all living creatures. In fact, the genetic part of the variation among individuals of a species in type, size, color, behavior, etc., is due to accumulation of mutations. An example of a desirable mutation is the change from horned cattle to polled. These "changed" genes then are transmitted from generation to generation.

Monitors defects

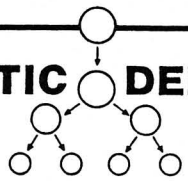
The Association monitors four Class I genetic defects, one Class II defect and one genetic factor that have been reported in Angus cattle. Class I defects are lethal, seriously disabling, or seriously affect reproductive performance.

Table 1. Different Phenotypes* and Genotypes** with Black (B) and Red (b) Gene Inheritance Resulting from Mating the Given Bulls and Cows (An example of simple recessive gene transmission where black is dominant to red.)

Bull		×	Cow(s)		=	Calves	
Genotype	Phenotype		Genotype	Phenotype		Genotypes	Phenotypes
BB	black	×	BB	black	=	all BB	black
BB	black	×	Bb	black	=	½ BB ½ Bb	black black
BB	black	×	bb	red	=	all Bb	black
Bb	black	×	Bb	black	=	¼ BB ½ Bb ¼ bb	black black red
Bb	black	×	bb	red	=	½ Bb ½ bb	black red
bb	red	×	bb	red	=	all bb	red

**Phenotype—the observable or measurable composition of an animal.*
***Genotype—the actual genetic composition of an animal.*

GENETIC DEFECTS



Dwarfism, osteopetrosis (marble bone disease), double muscling and syndactyly (mule foot) fit into this category. Heterochromia irides (white eye) is the single Class II defect. The single genetic factor is red color. Although it is not lethal and does not affect reproduction, growth, structure or function, red color is economically important because red calves are not eligible for registration under rules of the Association.

These six defects are thought to be transmitted by simple recessive genes. This means they are caused by a single pair of genes and only show up when both genes of the pair are recessives. (See Table 1.)

Simple recessive traits are the easiest to select against or control. Other traits such as small pelvic areas, leg problems, lack of mothering ability, prolapses and many others of economic importance are much harder to breed out or handle with minimal losses because they are controlled by many pairs of genes.

The mechanics

A calf inherits one gene in a pair from its sire and one from its dam. If both parents carry a recessive and pass it on to the offspring, the calf will be affected with the defect.

A calf also could inherit one recessive gene and one normal gene. This type of animal is called a carrier and, although it appears normal, can pass the defective gene on to its offspring. Usually there is no way to detect a carrier unless it is mated to another carrier or to an affected animal and a defective calf is produced.

Genetic defects are passed along from generation to generation. When two normal animals (with no recessives for the defect) are mated, all of the offspring will be normal.

If a normal bull is mated to carrier cows, statistically one-half of the offspring would be carriers. (Remember, carriers appear normal.)

If a carrier bull is mated to carrier cows, the problem usually becomes obvious. One-fourth of the calves would be affected, one-fourth would be normal and one-half would be carriers. In order for a calf to be affected, both parents must carry the recessive and pass it to the offspring.

The least likely event would be to use affected cattle in a breeding program. Marble bone and mule foot cattle usually die before they reach puberty, but it could happen with red color, double muscling or congenital reproductive abnormalities.

An affected bull mated to clean cows would produce all carriers. An affected bull mated to carrier cows would produce one-half carriers and one-half affected calves. And an affected bull mated to affected cows would produce all affected calves.

Open policy

The Association has the most comprehensive and open genetic defect control program in the industry. Some breed associations deal with genetic problems secretly, others have no program at all, and others simply refuse to admit that genetic defects do exist in their breed. But policies that cover up defective cattle certainly do more harm than good. Those breeds can only go backward in the long run.

Angus seed stock producers are charged with the responsibility of keeping our breed as free from undesirable genes as possible. This requires breeders not only to understand genetic defects but to report defective calves, cull carriers and be honest in dealing with customers.

As one Angus breeder who discovered a genetic problem in his herd says, "If we have to have a problem, it is to our long-term advantage to learn of it now so we can correct it. If we are to be truly successful in improving our cattle, we need to know all of their hidden problems along with all of the obvious beneficial economic advantages. This will make us even more aware of the tremendous responsibility, burdens and problems a purebred breeder faces."

Requires report

The Association requires members to report any abnormalities found in Angus cattle. Failure to comply could result in suspension or expulsion from the Association.

The Association strives to keep its members informed of bloodlines that carry defective genes so they can avoid introducing them into their herds. A complete list of animals reported by their owners as having sired or produced one or more of the six defects or factors monitored is published regularly in Angus Journal, and additions to this list are updated each month. The list currently includes 39 bulls and 31 cows as carriers of specific Class I defects, and 11 bulls as carriers of heterochromia irides (the single Class II defect). This list, however, is not a list of all Angus that are carriers of genetic defects. It includes only cases reported to the Association by the owner or owners of the animal or verified by a veterinarian.

A list of animals that have been progeny tested is also published. Ten bulls have been tested free of all defects and 28 bulls and five cows have tested free of specific defects.

In addition, a code is printed on registration papers issued after Jan. 30, 1979, which designates reported carrier animals. (Congenital reproductive abnormalities are not included.) The following codes are used to indicate carriers of the respective defects: Syndactyly "s," double muscling (muscular hypertrophy) "h," dwarfism "d," osteopetrosis "m," and red "r." The code for bulls possessing multiple genetic defects is "x," and breeders can check the Association's list to determine which defects the animal carries.

Tested clean

Animals tested free of a specific genetic defect in accordance with Association rules are indicated by adding an "f" to the code letter for that defect. For example, "sf" means that the animal has tested free of syndactyly. Animals that are carriers of one defect but have tested free of another also are indicated. For example, "r-sf" means the animal transmits the red gene but tested free of syndactyly.

Bulls that have tested free of all genetic defects by mating to 35 or more daughters under Association guidelines are designated "gdf," genetic defect free. Females that have tested free of defects under Association guidelines also can be labeled "gdf." (Progeny testing is discussed in more detail later.)

These codes, however, do not mean an animal does not carry a genetic

defect that has not been discovered or has not been reported to the Association.

A.I. policy

Another important part of the Association's policy involves A.I. service certificates. A registered Angus bull confirmed as a carrier of any of the six defects is indicated as a carrier on all A.I. certificates issued for that bull.

Good genetic material should not be thrown away unnecessarily, but caution must be used to avoid undesirable genes.

Calves sired artificially by non-owned bulls that transmit dwarfism, marble bone, mule foot or double muscling are not eligible for registration if conceived 60 days or more after first notification in the Angus Journal list. The breeder who sold semen from the carrier bull is responsible for informing semen buyers.

The Association also supports studies by contributing financially to breeders who follow set guidelines to determine whether an abnormal calf is afflicted with a genetic disease. (Contact the office for details.)

Need effective reporting system

The Association's control program can be only as effective as its reporting system—the purebred Angus breeders. Angus seed stock producers are charged with the responsibility of keeping our breed as free from undesirable genes as possible. This requires breeders not only to understand genetic defects but to report defective calves, cull carriers and be honest in dealing with customers.

"A breeder should have the insight, education and foresight to handle it (reporting genetic defects) on his own," Dr. Leipold says. "It's really his responsibility. It's his material. It's his baby."

Purebred breeders are the only ones who can effectively monitor genetic defects. Animal identity is often lost in commercial herds and, when a defective calf is discovered, the sire often cannot be traced. As a result, introducing defective animals into commercial herds can lead to serious economic problems.

Few losses

Genetic defects should cause few, if any, calf losses in a registered herd. If no genetic defect problems are present in a herd, the breeder has only to avoid using carrier bulls and females sired by known carriers. This can be done by checking pedigrees to determine which animals could be carriers and which should be clean. (There is a rare possibility that a mutation will create a new genetic defect in the herd no matter how cautious a breeder is.)

On the other hand, what should be done if a defective calf is discovered? First of all, only a small fraction of dead or aborted calves are afflicted with genetic problems. Most calf losses are caused by nutritional problems or infectious diseases.

The suspected calf should be thoroughly inspected by a veterinarian. Breeders should aid in the postmortem by providing as much information about the calf, its ancestors and its environment as possible. Speed in calling the vet is important if the suspected calf

is dead, because tissue degenerates quickly, especially in hot weather.

If the diagnosis confirms a genetic problem, the occurrence must be reported to the Association.

Recommendations

Most geneticists recommend eliminating the offending bull from the herd. Breeders also may want to eliminate the dam or breed her only to clean bulls because she is a confirmed carrier. The breeder must then decide what to do with offspring of the sire because, statistically, one-half of his calves will be carriers.

If a breeder decides to keep the bull's daughters, they should be bred only to clean bulls. This way, the frequency of carrier offspring would be halved each generation. (Fifty percent of the daughters are carriers. When mated to clean bulls, 25 percent of the bull's granddaughters would be carriers. The next generation, only 12.5 percent would be carriers, etc.) Elimination of the problem this way is a slow process, however, and some breeders prefer to eliminate all daughters of the carrier bull and replace them with clean females.

If the carrier bull has produced an outstanding son, he should be progeny tested before extensive use as a sire. Good genetic material should not be thrown away unnecessarily, but caution must be used to avoid undesirable genes.

Keep positive attitude

Ethics in dealing with carrier cattle is an area which cannot be stressed strongly enough. Breeders who sell carriers or descendants of carriers owe their customers complete honesty. A breeder who informs his buyers that an animal has, for example, a 25 percent chance of transmitting a certain defective gene can only inspire the customer's trust and respect. And that is what the purebred business is all about.

Breeder perspective and attitude is perhaps the most important part of dealing with genetic defect problems. One Angus breeder hit the nail on the head when he stressed, "Think positive! We all know the vast majority of our cattle are clean. The problem can only turn into a 'monster' by breeder neglect and attitude."

SYNDACTYLY-Mule Foot

Syndactyly is commonly known as mule foot because most affected animals have fused or uncloven hooves that resemble mules' feet (see Figure 1). The genetic form of mule foot originated as the result of a mutation, and it is passed along by a simple recessive gene.

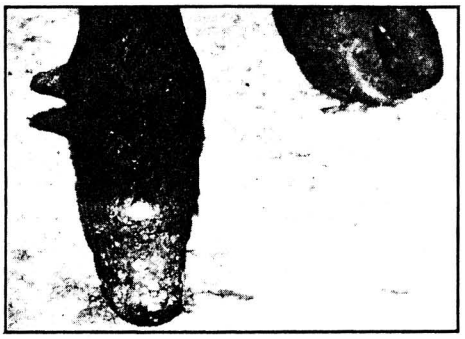


Figure 1. A typical case of mule foot. Photo courtesy Kansas State University Pathology Dept.

Mule foot is one of the most common genetic defects of U.S. cattle. It is most frequently seen in Holsteins, but the recessive gene is present in Angus and several other breeds. The defect also has been found in many cattle breeds abroad in addition to lambs, pigs, dogs and cats. And a similar form of syndactyly occurs in humans.

Mule foot was added to the Association's list of Class I defects in 1976. Since then, Angus breeders conscientiously have monitored and progeny tested for the defect, and it is well under control in today's Angus population.

Although it is a simple autosomal recessive trait, mule foot has some irregularities.

First, the expression of mule foot varies. One, two, three or all four feet may be affected, and this follows a distinct pattern. The right front foot is always first and most severely affected. If two feet are affected, it will be the two front feet. The right hind foot is next, and the left hind foot is last and least likely to be fused.

Other irregularities

But hooves are not the only thing different about these cattle. The genetic form always involves horizontal fusion of the bones of the feet (see Figure 2). This is one way to differentiate between an inherited and noninherited problem.

Bone fusion results in stiff and insecure joints. It follows the same pattern as hoof fusion—that is, if X-rays of the two front feet are compared, the bones of the right one usually are more fused than those of the left, etc.

The second irregularity is incomplete penetrance—some affected animals appear normal. The hooves show no external signs of fusion; however, X-rays reveal bone fusion. This is rare, and these animals are called escapers.

Four groups of hoof fusion severity have been distinguished (see Figure 3): (a) Hooves show no external signs of fusion. (b) Hooves are partially fused at their axialcoronary border. (c) Hooves are fused but have a groove from the toe bed to the tip of the hooves and show signs of dual embryonic origin. (d) Hooves are completely fused and show no signs of originating as normal hooves.

Not all calves from one bull are likely to have the same number of feet affected or to the same degree of severity.

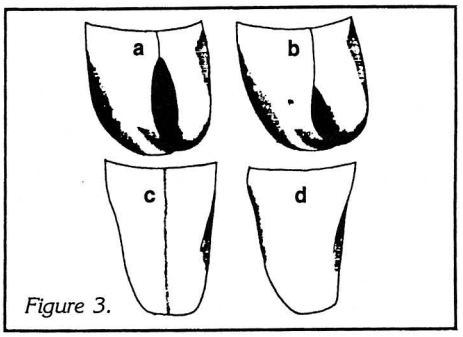


Figure 3.

More serious problems

Fusion or non-division of the hooves occurs during early gestation and, in most cases, the physical signs of syndactyly are easily recognized at birth. But while uncloven hooves are the most obvious physical signs of syndactyly, they are not the most serious problem.

"Fused hooves are just a trivial expression of a dramatic biochemical abnormality," says Dr. Horst Leipold, pathologist at Kansas State University, Manhattan. Further research is needed to determine just what biochemical changes the recessive gene causes, however.

Mule foot renders animals economically useless. They cannot cope with stress, especially heat. They cannot stand temperatures of 80° or higher

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and often die of heat stroke. In addition, they frequently develop signs of hyperthermia, including elevated temperature, fast heart rate and rapid breathing. They are unthrifty and frequently die soon after birth. And whether an animal has one or four feet affected does not seem to affect its ability to adapt to stress.

Mule foot cattle that are given special care and live to sexual maturity are capable of reproduction, and affected cattle have been mated in the Kansas State University research herd. In the general population, though, mule foot animals seldom live long enough to reproduce, which helps control spread of the defect. The recessive gene is not likely to be passed along by affected animals but is handed from generation to generation by carrier animals.

May not be syndactyly

Breeders should not be too hasty in dubbing a calf syndactylous or in calling a sire a carrier. A fused or single hoof on the end of a leg does not necessarily mean mule foot—conditions exist that look like syndactyly but are not. For example, a condition has been observed where fused hooves are correlated with facial defects; the face is not formed right and the legs are not formed right. And there are forms of syndactyly that may not be inherited.

As with all calves suspected of having a genetic defect, mule foot suspects should be examined by a veterinarian or diagnostician, and the exam should include X-rays of the feet.

If veterinary inspection confirms inherited syndactyly, parentage verification should be obtained by blood typing. Any sire or dam that has produced an affected animal should be listed as a carrier.

Researchers at Kansas State University have studied mule foot for some 20 years, and a small herd of affected and carrier animals is maintained there. One area of current research involves the pattern of syndactyly in beef cattle vs. dairy cattle. Although it is more frequent in some dairy breeds, studies indicate that it is more severe in beef cattle. Dairy animals usually have one foot affected, sometimes two or three, but rarely four. On the other hand, beef animals more often have all four feet affected.

Modern progeny tests

Another area of successful research involves methods to speed up and cut

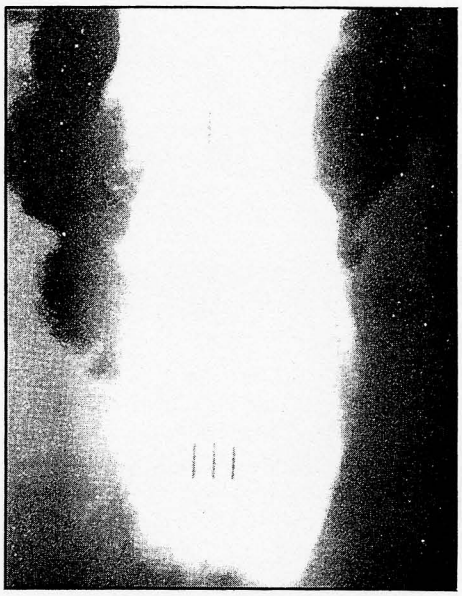

Figure 2. Radiograph of a mule foot. Note abnormal development of toe bones and abnormal fusion. Photo courtesy Kansas State University Pathology Dept.

costs of progeny testing for syndactyly. Since it is a hereditary defect present at birth, test matings can be used to identify carrier animals.

But rather than mating a bull to 35 different daughters of confirmed carrier sires and waiting until calves are born to determine the outcome, new procedures include embryo transfer and early fetal removal. This cuts number of cattle needed, time and cost, and makes it more feasible to test bulls before they are widely used or females before they are used for embryo transfer.

To test a bull, the new methods require only seven fetuses to be recovered from seven affected (homozygous recessive) cows or one superovulated affected cow. To test a female, she is bred to an affected bull, and seven fetuses must be recovered. In both cases, the fetuses can be transferred into four recipients, removed by Caesarean section after 60 days of gestation and readily identified as normal or affected. This proves the animal to be clean or to be a carrier with 99.6 percent accuracy.

Syndactylous embryos have been recovered and identified as early as 31 days of gestation, but 60 days leaves less margin for error or doubt in identification of normal and affected embryos. The Association requires a minimum of 60 days gestation.

To date, five Angus bulls have been progeny tested free of the syndactyly gene, and ten bulls have tested free of all genetic defects monitored. Five females, tested by the new methods, have been proven free. ■

OSTEOPETROSIS-Marble Bone Disease

Osteopetrosis is a lethal genetic disease that affects the bones of growing fetuses. Abnormally solid bones and lack of bone marrow cavity associated with the disease have led to the more common name "marble bone disease."

Marble bone has been reported in Angus and Hereford cattle. Many features of the cattle disease are similar to inherited osteopetrosis in human babies.

Marble bone is caused by a simple recessive gene that is present at a low level in U.S. Angus cattle today. Although genetic defects cannot be eliminated, Angus breeders already have proven that marble bone can be easily controlled.

In the early 1970s, as many as 150 cases of marble bone per year were seen in Angus cattle. But by 1976, only two cases were reported. This shows that it can be easily controlled and kept at an insignificant level when cases are properly diagnosed and reported, breeders are informed and carrier bulls are eliminated from the industry.

Most dynamic tissue

Bone is the most dynamic tissue in the body. Shape, size and maintenance of normal bone involve almost daily turnover of tissue—it grows and changes constantly. A finely tuned balance of continuous formation and removal of

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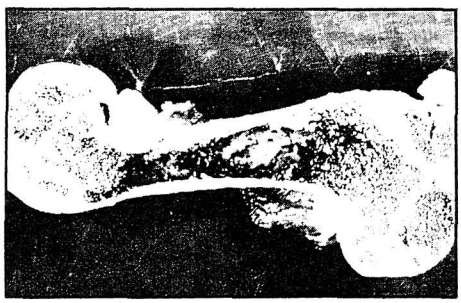
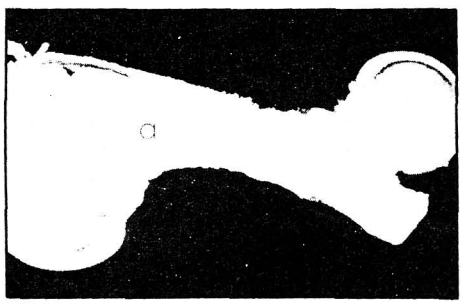
bone tissue is maintained in normal animals. If this balance is disturbed, disease results.

During normal fetal development, a cartilage precursor is laid down, then it is changed to bone. This bone is resorbed and remodeled, and the bone marrow cavity is formed in the center of the bone. (Bone marrow is a soft fatty substance involved in renewal of the blood supply.)

In marble bone cases, the first bone formed in the fetus is retained. New bone tissue is continually formed, but old tissue is not removed. It is not remodeled or strengthened, and cavities for bone marrow are not carved out. Instead, the bone is solid. Contrary to expectations, these solid bones are weak and brittle, and they will break easily



TOP: Section of normal bone. BOTTOM: Bone affected by marble bone disease. Note the lack of bone marrow cavity (a) in the diseased portion. Photo courtesy of Kansas State University Pathology Dept.



because they are filled with an immature mixture of cartilage and primitive bone. The disease may affect the entire skeletal system.

Subtle killer

Marble bone disease is a subtle killer that often goes undetected and can pass unknown from generation to generation. In fact, it has probably been around for a long time without being recognized as a genetic disorder.

"I think osteopetrosis is far more dangerous than mule foot, for instance, because it is more insidious," Dr. Leipold contends. That is because breeders usually see and recognize defects such as mule foot, while marble bone calves often are simply labeled abortions, and infectious disease or environmental problems (which are much more common) are thought to be the cause.

Affected calves are born between 251 and 272 days of gestation and are born dead or die shortly after birth. The only outward signs of marble bone are small size (usually 30-70 lb.), a short lower jaw, a protruding tongue and impacted molar teeth. Also, since the remodeling process (which plays a role in changing the shape of bones) is deficient, the bones which house the brain are misshapen and small, and the brain is flattened and compressed, with abnormal mineral deposits that slow vital body functions and probably cause death. Calves may have a soft spot on their foreheads where the skull bones fail to unite.

However, these signs are hard to detect in small calves. Plus many aborted calves are not carefully examined and, in some cases, are never even found. A good share of marble bone cases probably go undetected.

Inspect all aborted calves

All aborted calves should be closely inspected and, if any of these signs are present, the calf should be sent to a diagnostic lab. Ideally, the whole calf should be sent, but the head and one leg will suffice. If there will be a delay in sending the calf, it should be frozen.

Even examination by a veterinarian or diagnostician has not proven 100 percent effective in pinpointing marble bone cases. Infectious diseases and environmental problems are so much more common that the doctor usually is not looking for the genetic defect. A routine necropsy will not reveal marble bone affliction, because the outer part of affected bones appears normal. The genetic problem can be verified only by splitting a long bone (such as the leg bone), and that is not part of a standard necropsy.

To help assure correct diagnosis, breeders should alert the examining vet if marble bone is suspected. Sire and dam pedigree information also should be supplied to help determine whether the calf's ancestry involved any reported genetic defects.■

HYDROCEPHALUS

Hydrocephalus is the most common and best documented central nervous system defect of cattle. The disease affects virtually all major breeds of beef and dairy cattle as well as humans and other animals.

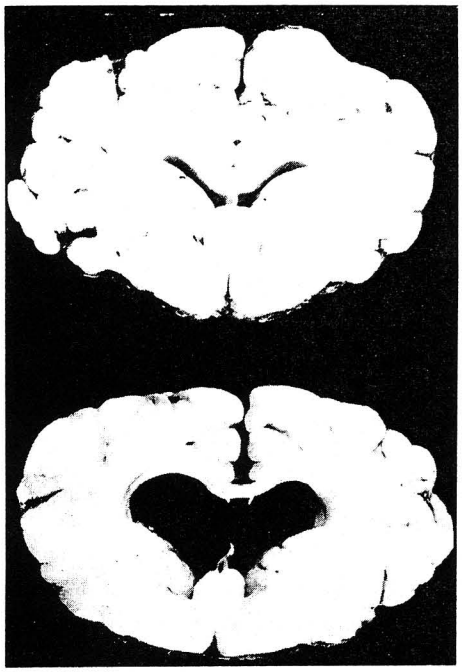
Hydrocephalus involves the build-up of excess fluid in the brain. This fluid causes the ventricles (cavities) of the brain to swell, reduces or thins the brain tissue and sometimes causes the bones of the head to separate. (See photo.)

Inherited hydrocephalus has not been documented in Angus cattle. It is a problem in some other major breeds, though, and has caused substantial losses in some Hereford herds. The defective gene is passed along as a simple autosomal recessive trait, causing the brain to develop wrong during gestation. This is called primary hydrocephalus.

Genetics is only part of the story, however. Hydrocephalus also occurs as a secondary result of environmental factors, other abnormalities or other inherited defects. Angus calves have had the disease as a result of cysts, tumors or other blockages in various parts of the central nervous system (CNS), associated with meningitis (inflammation of the membranes of the brain and spinal cord) and as a secondary problem to the inherited defect mannosidosis. (Mannosidosis is a lysosomal storage disease due to the presence of defective genes which cause the absence of an enzyme the body needs to function, and in turn causes the build-up of an unwanted substance. See the February 1981 Journal for details.)

Fluid build-up

The CNS includes the brain and spinal cord. A network of blood vessels in the brain secretes a clear fluid called CSF (central nervous system fluid) which surrounds and permeates the entire CNS, supporting, protecting and nourishing it. By secretion and absorption of CSF, these vessels regulate the pressure in the brain. In normal animals, the CSF flows from inside of the



This photo provided by Kansas State University's College of Veterinary Medicine illustrates the difference between a normal brain (top) and an abnormal brain (bottom) affected with hydrocephalus.

brain (ventricles) and is resorbed outside of the brain into the venous system.

Most hydrocephalus cases result from obstruction of the fluid flow somewhere along its pathway—the fluid cannot escape from the brain and accumulates there. Also, over-production of CSF or inadequate resorption can lead to hydrocephalus.

The excess fluid occupies space normally taken up by brain tissue, causing the brain to be under-developed. The cerebellum often is reduced to nearly half of its normal size.

Hydrocephalus varies considerably in how much and what parts of the brain are affected. It may be external, with the fluid accumulating in the space around the brain; internal, with the fluid contained in the ventricular system; or it may be present in both locations.

Enlarged heads

Hydrocephalic calves usually are aborted during later gestation, are stillborn or die shortly after birth. They are generally small, with enlarged and sometimes dome-shaped heads, and swollen protruding tongues.

Facial muscles are small and irregular in size, and facial features are refined and narrow. Eyes and optic nerves are small. Most major skeletal muscles are affected—especially the thigh muscles—and they are soft, pale and spongy. Other abnormalities associated with hydrocephalus include cleft palate, eye problems and heart defects.

Some calves may live a short time, but they are usually weak and retarded. They often elicit a characteristic bawl and have been labeled "bawlers."

If hydrocephalus is acquired later in life, an animal may exhibit depression, incoordination, abnormal responses, paralysis, prostration and sometimes convulsions. They frequently lose their vision.

But even with these characteristic signs, hydrocephalic calves are not always easily recognized. Calves that exhibit any of the symptoms listed should be taken to a vet, who should remove the brain and send it to a diagnostic lab for further study. It is important to get a careful necropsy to accurately diagnose and determine the cause of the condition.

Discovered long ago

Hydrocephalus is not a new disease. It was originally documented in man in 1514. It was first described as a lethal defect of cattle in 1959 when an outcross bull was introduced into a previously closed herd at the New Mexico Agricultural Experiment Station.

Causes of hydrocephalus other than genetics are not fully defined or understood. Viral agents may well play an important role in causing the disease, but the only virus confirmed as a culprit is BVD-MD (bovine viral diarrhea-mucosal disease). ■

HETEROCHROMIA IRIDES-White Eye

Heterochromia Irides, a partial albinism that affects various breeds of cattle, including Angus, has been the subject of several years of studies conducted by Dr. Leipold. According to him, the problem occurs in calves of both sexes and is identified by skin, coat, and iris color characteristics.

The hair of the entire body surface is chocolate brown rather than the normal rich black color typical of Angus cattle. The muzzle, hooves, dew claws and scrotum in males are also brown, with skin surface brownish to grey in color. This discoloration is particularly obvious at the glabrous skin, such as around eyelids, ear openings, the muzzle, and anal and reproductive openings.

The most distinguishing factor, however, involves iris color. While Angus cattle usually have a dark black iris, heterochromia irides calves have a light, usually two-colored iris. When viewed closely, the iris of an affected animal has a double-ringed appearance—an outer, faintly brown ring and an inner light blue ring circling the pupil. The pupils appear constricted in daylight, causing a "slit-like" appearance. From a distance the eyes appear white.

A few calves have been observed to have normal black Angus color, but have bilateral discoloration in the ventral parts of their irises. These discolorations are white and blue spots.

Based on findings in his studies, Dr. Leipold determined that heterochromia irides is a genetic defect due to a homozygosity of a simple, autosomal recessive gene.

Observed only on a very limited scale in Angus cattle, heterochromia irides is classified by the Association as the single Class II genetic defect. AJ