This completes the series of articles designed to acquaint Angus breeders with genetic defects, problems which occur in every breed of every species.

PROGENY TESTING

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Valuable genetic material shouldn't be thrown away. Breeding powerful seed stock that can improve and perpetuate a breed takes years of hard work and careful selection, because most of the traits we select for—fertility, growth, milk production, etc.—are controlled by many genes. They're much harder and more costly to change than genetic defects caused by a single pair of genes. We can't afford to discard a lot of good genes because of one bad gene.

On the other side, confirmed carriers of genetic defects should not be used in registered herds—no matter how strong they are in other traits. A carrier's offspring stand a 50% chance of carrying the defective gene, so they shouldn't be used unless they're proven clean. And grandsons or granddaughters should not be used extensively unless they're proven clean, because they run a 25% chance of being carriers.

That's where progeny testing comes in. A carrier's superior traits can be passed on to benefit future generations, without spreading the defect, by using outstanding sons or daughters that have been progeny tested and declared clean. Rather than throwing out whole lines of cattle because of one undesirable gene, superior animals in that line can be tested. Cattle that successfully complete a test should be accepted by the industry as clean, and not be discriminated against, even if they're closely related to an affected or carrier animal.

Offers 90% Accuracy

Progeny testing involves breeding the bull or female in question to animals which carry, or stand a higher than average chance of carrying, a defective gene. This increases the chance of abnormal genes pairing up in the offspring. If even one defective calf is born, we can be 100% sure that the tested animal is a carrier.

If no abnormal calves are born in tests following American Angus Assn. guidelines, we can be 99% sure that the animal is not a carrier. Breeders should note, however, that an animal never can be labeled clean with 100% assurance. There's always a chance of nondetection no matter how many normal calves are born. But with 99% accuracy under association policies, that chance is very slim.

Association guidelines offer four ways to check a bull for the marble bone, mulefoot, double muscling or dwarfism genes: (1) Mate to abnormal females (marble bone cases usually don't live to sexual maturity, however), (2) mate to known carriers, (3) mate to daughters of a carrier bull and (4) mate to the bull's own daughters. The first three methods test only for a single defect; mating to a bull's own daughters checks for all recessives.

A female can be tested by breeding to an affected bull or to a known carrier and using embryo transfer.

Numbers Vary

When mating to abnormal animals, seven live calves (with no defects) are required to prove, with 99% accuracy, that the animal is not a carrier. Mating to known carriers requires 16 live offspring. Mating to daughters of a carrier or daughters of the bull on test requires 35 different daughters. Trials do not have to be completed in one year so long as the required number of matings are made.

Since mulefoot can be detected early in gestation, both bulls and females can be tested using super ovulation, embryo transfer and fetal removal after a minimum 60 days pregnancy, which saves time, money and number of cattle needed. The same number of fetuses are required as the number of live calves listed in the preceding paragraph.

When is a progeny test justified? They're expensive and time-consuming, but using tests to spot carriers is cheaper than trying to control a defect after it's spread through a herd or a breed. Such a test generally is warranted for superior animals that are related to a carrier.

Several factors should be considered when deciding whether to test. Will the breeder's program be built around one bull so that his genes will form the foundation of the herd? Will a large quantity of semen be sold, distributing a bull's genes throughout the industry? Will buyers pay premiums for the offspring because the bull has been tested? Will a female be used extensively enough to warrant the test?

Justification for Test

A yes answer to any of these questions may indicate sound justification to run a progeny test.

A sire-daughter test is a more powerful tool than a test for one specific defect. These matings check for all undesirable recessives, and bulls that successfully complete a test on 35 different daughters are declared genetic defect free. Sire-daughter tests can fill a specific and important industry need by providing a bank of solid clean genetic material, but they should be kept in proper perspective. Genetic defect-free bulls should be used as another tool in over-all breeding and merchandising plans.

However, this is the most expensive progeny test. It also produces a lot of inbred calves. And it takes three to four years to complete, delaying the use of good young sires. If a bull is tested as a yearling, he'd be at least four years old when his first daughters' calves are born. Breeders shouldn't hold back from using a young bull unless there's reason to suspect that he may be a carrier.

Because of time and money involved, sire-daughter tests probably will be limited

to a few bulls expected to be used widely in the industry through sons, also to bulls with outstanding genetic merit that are suspect because of an affected or carrier relative.

Test for Single Defect

Tests for a single defect are quicker and more economical than sire-daughter matings. The association's test policies were especially designed to allow a breeder to prove an animal free of a single undesirable gene when that problem has cropped up in related animals.

To date, six Angus bulls have completed sire-daughter matings and have been declared genetic defect free. Two bulls and one female have tested free of the mulefoot gene, 21 bulls have tested free of dwarfism, and 18 bulls have been found free of the red gene.

This is the last article of the genetic defect series. These articles were not published to imply that the Angus breed has serious defect problems or more defects than other breeds. It certainly doesn't.

The series was written to complement the American Angus Assn.'s open policy to get information out to breeders where it will do the most good. The association's policies are designed so that we can work together to keep our cattle as clean and trouble-free as they are today, to maintain the Angus breed's No. 1 spot in the industry. Only by being open, honest and educated can we do that.

Darrell L. Wilkes, a partner in Wilkes' 6 Bar D Angus Ranch, Hawk Springs, Wyo., who is working toward his PhD in animal breeding at Ohio State University, submitted the following letter. It deals with spread of genetic defects and seems an appropriate way to bring the JOURNAL'S genetic defect series to a close. —The Editor

As an Angus breeder and enthusiast, I appreciate the efforts of the ANGUS JOURNAL staff to inform breeders of genetic defects found in Angus cattle. The series of articles on this topic have been accurate, complete and easy to understand. While it is true that all breeds of cattle have such defects, not all breed associations are committed to solving their problems with such vigor as our association—reason to be proud, without a doubt. As an Angus breeder, I feel it my duty to point out a slight misconception which seems to be fairly widespread with regard to these genetic defects.

A typical misconception is that these defects are becoming more common. This is an unfounded fear in most cases. According to the Hardy-Weinberg law of genetic equilibrium, the frequency of any particular gene will remain constant indefinitely unless some sort of selection is acting on the animals which carry that gene.

In order for a gene to increase in frequency in a population, individuals which carry that gene must have increased fitness compared to non-carriers. In other words, in order for a defect such as syndactyly, osteopetrosis, dwarfism or mannosidosis to increase in frequency, carriers must leave more offspring than their non-carrier counterparts. This is not likely to be the case with these genetic diseases.

No Abnormal Spread

I am trying to point out that these genes do not *spread* any more than normal genes *spread*. In fact, since the homozygous recessive individuals (those with two abnormal genes for that trait) are usually genetically dead (that is, they leave no offspring), the frequency of these defective genes is actually on the decline. Think about it this way; every time an affected individual dies, the population looses two of these defective genes.

Some of the readers may be thinking that the use of A.I. will help spread the disease (raise the gene frequency), but this is not true. So long as the presence of the gene in an individual does not bestow him with some other superior quality, and there is no evidence that it does, carrier bulls will not appear in A.I. books with any higher frequency than they occur in the general population. For example, if 5% of the bulls in the general population carry some mutant gene, then we would not expect any more than 5% of the bulls in A.I. books to carry that particular mutant gene. It may be hard to believe that some high-fallutin' genetic theory is based on common sense, but it really is.

Final Point

One final point should be discussed before the argument is complete. That is, spontaneous mutation is *not* a vital force in changing gene frequency. (Spontaneous mutation is a change in the genetic blueprint of an animal which results in a "clean" animal producing offspring with a defective or mutant gene.) When a football player drops a pass in the end zone, the first thing he does is accuse his defender of pass interference. Similarly, when a highly promoted bull sires a defective calf, the first response is to claim that a mutation occurred and that his ancestors are "clean." This is rarely true. None of us will live long enough to see gene frequencies changed by spontaneous mutation. Hence, this is not a valid argument in 99.99% of the cases.

In summary, we as cattle breeders should continue our battle against genetic defects. Inasmuch, we should try to understand as much as possible about the mechanisms through which genetic defects arise. Programs to identify carriers will be of great merit in helping to eliminate some deleterious genes from the population. Where practical, these programs should be considered (as in the case of mannosidosis where carriers can be identified by a blood test—see February 1981 ANGUS JOURNAL, Page 23). As responsible cattle breeders of a truly meritorious breed of cattle, it is our duty to become more aware of the problems which exist. So long as our association continually strives to inform us of these problems, we will be in a stronger position to truly insure our future.