V

aluable 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 se-
lect 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 regis-
tered 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 grand-
daughters 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 spread-
ing the defect, by using outstanding sons or
dughters 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. guide-
lines, we can be 99% sure that the animal is
not a carrier. Breeders should note,
however, that an animal never can be label-
ed 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
two 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 us-
ing embryo transfer.

Numbers Vary

When mating to abnormal animals, sev-

en 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
dughters 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 mat-
ings are made.

Since mulefoot can be detected early in
gestation, both bulls and females can be
tested using super ovulation, embryo trans-
er 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 pre-
ceding 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 re-
lated 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 test-
ed? 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 re-
cessives, and bulls that successfully com-
plete a test on 35 different daughters are
declared genetic defect free. Sire-daughter
tests can fill a specific and important in-
dustry need by providing a bank of solid
 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 prog-
eny 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 daugh-
ters’ 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

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

by Marilyn Barr
Assistant Director, Communications & Public Relations
American Angus Assn.
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 mulefoot gene.

This is the last article of the genetic defect series. The 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 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 loses 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-falutin’ 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 insures our future.