Genetic Defect FAQs

How long has RAAA recognized genetic defects?

Genetic Defect concerns have been part of Red Angus’ Rules and Regulations for more than a decade.

Most noticeable in requirements for registry “Animals known to have or exhibit any of the following traits [listed genetic defects] will not be eligible for registration…”

Also, in suggested Terms and Conditions of Sale, “all animals are guaranteed by the seller not to produce calves with the following defects [listed genetic defects] ….effective for a period of two years from date of sale…”

Have there been some recent changes to RAAA’s genetic defect policy?

Yes, updates and additions to Red Angus’ genetic defect policy were implemented following the January 2007 Board of Director’s meeting.

What prompted RAAA to make changes to their genetic defect policy?

Red Angus proposed additions to the Genetic Defect Policy due to increased incidences of genetic defects occurring in other US beef breeds – primarily, TH (Tibial Hemimelia) and PHA (Pulmonary Hypoplasia with Anasarca).

What do these additions consist of?

The most significant changes were:
1) The requirement to report affected calves.
2) The process through which carriers of a specific genetic defect would be made public.

Do other beef breed associations have a genetic defect policy?

Yes, due to the fact that all breeds are subject to genetic defects, the majority of beef breed associations have a genetic defect policy, with some breeds’ having implemented such policy 2-3 decades ago. The updated version of Red Angus’ Genetic Defect Policy is very similar to the policy used by other US beef breed associations.

I have heard there was a Red Angus calf diagnosed with a genetic defect at Kansas State University in 2006. Why didn’t RAAA inform the membership of the parentage of that calf?

Subsequent to the implementation of RAAA’s Genetic Defect Policy, Red Angus staff was made aware of a calf diagnosed with Osteopetrosis by Kansas State University (KSU). KSU referenced the calf as being Red Angus in a publication to veterinarians in Kansas with the intent to educate veterinarians with regards to the different characteristics of Osteopetrosis and the environmental mimic, BVD. Red Angus staff contacted KSU to determine if tissue samples of the Osteopetrosis calf were retained. KSU staff had discarded the samples; therefore, eliminating both the possibility of parent verification, and confirmation of Osteopetrosis by RAAA’s Genetic Defect consultant – both are critical steps in RAAA’s Genetic Defect Policy.
**Could you describe the typical sequence of RAAA’s Genetic Defect Policy?**

A Producer contacts RAAA to report abnormal calf.

B Typically, entire calf is transported to respective State or University Vet Diagnostic Lab, (VDL).

C If VDL diagnosis indicates genetic defect the following steps occur:
   1) Samples taken to rule out environmental mimics (BVD in case of Osteopetrosis)
   2) Tissue sent to Dr. David Steffan at UNL (RAAA’s Genetic Defect Consultant) for final confirmation of VDL’s genetic defect diagnosis.
   3) DNA samples sent to approved genomics lab to confirm that parentage of affected calf is the same as reported parentage.

D Positive results from steps 1-3 on two progeny result in RAAA notifying breeder/owner(s) of registered parent(s) of the intent to list parent(s) as a confirmed carrier of the identified genetic defect.

E Breeder/Owner(s) has 30 days to appeal the decision, investigate test procedures/results.

F Breeder/Owner(s) may waive their right to appeal, which ends the appeal period.

G At the end of the appeal process, the parent(s) will be listed as a genetic defect carrier.

**How long does it take to complete all of these steps?**

Typically this process takes approximately 60 days. However, several things can increase this timeframe: schedules of scientists involved in the process, tissue collection on reported sire and dam of abnormal calf, appeal to the executive committee, etc.

**Why doesn’t RAAA notify membership as soon as an abnormal calf is reported, and disclose the reported parentage?**

All abnormal calves are not genetic defects. Environmental mimics exist for genetic defects. In the case of Osteopetrosis, BVD affected calves can be indistinguishable from the genetic defect by the untrained eye. Furthermore, reported parentage is not always correct, i.e. pulling the wrong straw of semen out of the tank during artificial insemination, calf being sired by the clean-up bull as opposed to the AI bull, etc. Examples of both of these situations have occurred; therefore, listing animals before parentage and the genetic defect are confirmed would be premature. While all efforts will be made to deliver information to the membership in the most expedient manner, the accuracy of information provided must remain the top priority.

**Will RAAA pull the registrations of confirmed genetic defect carriers and/or their progeny?**

No. Confirmed carriers will be listed on the RAAA website. In addition, their registration certificate (printed and web versions) will identify them as a genetic defect carrier with the [*D] notation after their registration number. Furthermore, the specific genetic defect will be listed under that animal’s name and above it’s pedigree.

In the case of progeny of a confirmed carrier: Any confirmed genetic defect carriers will be identified at their respective position within a pedigree by the [*D] notation after their registration number. In the web version, such carriers’ registration numbers are live links, which when clicked will identify the specific genetic defect.
Where can I find a list of confirmed carriers of a genetic defect?

The genetic defect carrier list is found at RedAngus.org / genetics / genetic defects / carrier list.

What is Osteopetrosis?

Osteopetrosis, also known as Marble Bone, is a genetic defect that causes abnormalities to the development of the bone marrow cavity and brain, ultimately causing death.

Is Red Angus the only breed with reported cases of Osteopetrosis?

No, Osteopetrosis has been reported in Hereford, Simmental, Red & Black Angus, and Holstein.

Is Osteopetrosis a new genetic defect?

Absolutely not, Osteopetrosis has been documented in cattle since the 1960s.

What are the characteristics of Osteopetrosis?

Calves are born premature (one month to one week early), if the calf is born alive it will die within 24 hours. The most noticeable phenotypic abnormality is a short lower jaw. Additionally, bones broken with ease due to an abnormal bone marrow cavity.

What is the inheritance pattern of Osteopetrosis?

The inheritance pattern of Osteopetrosis is typical of most bovine genetic defects, in that it is homozygous recessive – meaning both parents must transmit one Osteopetrosis affected gene to the offspring in order for Osteopetrosis to be expressed. The inheritance pattern and probability of phenotypic expression of Osteopetrosis is identical to red/black hide color. (see: RedAngus.org / about RAAA / history of Red Angus)

Is Osteopetrosis the only genetic defect RAAA monitors?

No. The complete list of genetic defects monitored by RAAA can be seen in the Rules & Regulations. However, Osteopetrosis is the only genetic defect RAAA has identified in the breed. However, through increased member education, it is possible that other genetic defects could be identified in the Red Angus breed.

How many animals has RAAA identified as carriers of Osteopetrosis?

Since the January 2007 update of RAAA’s Genetic Defect Policy, two bulls have been confirmed as carriers of Osteopetrosis.

The two animals identified as confirmed carriers of Osteopetrosis have a common ancestor in their pedigree. Is the common ancestor the source of the Osteopetrosis gene?
Instinctively, knowing the typical inheritance pattern of genetic defects one jumps to the conclusion that the first common ancestor is the source of the defective gene. However, if you look a little deeper into the two bulls' pedigrees you will notice that there are additional common ancestors. It is possible that any of these common ancestors is the source of the defective gene. Also, it is possible, though not as likely that the gene did not come from any of the common ancestors, but from animals that are not closely related. The fact is, at this point in time we can not determine where the defective gene came from; therefore, we should not unfairly point blame at any specific animal.

Bottom line…the only thing we can assume is progeny of the animals on the genetic defect carrier list have a 50% likelihood to be carriers of Osteopetrosis. Any assumption beyond that point is based on speculation…not sound science.

Isn’t the core reason to have a Genetic Defect Policy to determine where the defective gene came from?

The purpose of RAAA’s Genetic Defect Policy is to identify and report to members, confirmed carriers of genetic defects - not to identify the origin of the genetic defect.

Is there a DNA test for Osteopetrosis?

Not at the current time. However, RAAA staff is working with the beef industry’s leading molecular geneticists in the development of a DNA test for Osteopetrosis. This process requires significant sampling of affected calves, parents, and all known and available animals in the pedigree of affected calves. The cooperation of Red Angus breeders has been a vital step in the development of this important tool.

When will the DNA test be available?

This process is very similar to finding a needle in a haystack. The needle could be located on the outside – less time, or it could be located in a more difficult area – more time. We are fairly confident of our ability to find the gene that causes Osteopetrosis; however, the timeframe of doing so is not as certain.