



Dr. Colin Palmer

#### Biography:

Dr. Colin Palmer is an Associate Professor of Theriogenology (Animal Reproduction) at the Western College of Veterinary Medicine. Originally from Nova Scotia, Dr. Palmer worked in mixed practices in Ontario and British Columbia and has owned/operated a practice in Saskatchewan. Dr. Palmer along with his wife Kim and children Lauren, Emily and Carter run a herd of purebred Red Angus cattle under the KC Cattle Co. name.

There has been much discussion of genetic defects within Angus circles lately. Most breeders seemed to show little concern until they discovered that a well-known AI sire had been listed as a carrier; or they had a few registrations suspended this spring because the calves were identified as potential carriers of a genetic defect. For the uninitiated, as of January 1, 2010, calves being registered with the Canadian Angus Association having a known carrier of any one of four genetic defects in the first two generations of their pedigree, must be declared free of the causative gene either by submitting a DNA sample from the calf, or from a parent in the intervening line if the grandparent is the known carrier. Breeders who decide to check the parent first, and let's say they find that it is a carrier, must also check the calf; or they may choose not to register the calf. If the parent is free of the causative gene, then the calf will be free and registration should proceed provided the appropriate fee is paid. Once that parent has been declared free, all future calves will be free of that gene as long as the other half of the mating equation is also free. A good source of information regarding genetic defects is the Canadian Angus Association Website. I borrowed heavily from it for this article.

All breeds of cattle have known genetic defects and several defects such as hairlessness (hypotrichosis), syndactyly (mule foot), tibial hemimelia (absence of a shin bone), and dwarfism appear in more than one breed of cattle. In fact, the Canadian Angus Association monitors 13 distinct genetic defects and encourages the reporting of all abnormal calves, in case new, previously unreported, genetic defects appear. The troublesome four; those for which we must test: Neuropathic hydrocephalus (NH – fluid in the head) and arthrogryposis

## Genetic Defects

multiplex (AM – Curly Calf Syndrome) affecting predominantly Black Angus lines of cattle; and osteopetrosis (OS – Marble Bone Disease) and alpha mannosidosis (MA – muscular weakness, lack of coordination and weakness) affecting predominantly Red Angus cattle, have been linked to cattle listed as “Active” within the Canadian Registry. Other conditions such as syndactyly, have not been heard of for some time and should remain that way, unless you are planning to use some Marshal Pride 256 semen you have kickin’ around in a line breeding scheme.

Neuropathic hydrocephalus is a brand new disease confirmed to be caused by a single gene mutation a little more than a year ago. Arthrogryposis, osteopetrosis, and alpha mannosidosis have been around for some time. Calves afflicted with arthrogryposis, hydrocephalus or osteopetrosis are obviously abnormal at birth. If they are born alive, they usually do not survive very long. Alpha mannosidosis has been recognized in the Galloway, Murray Grey and Angus breeds, but unlike the other conditions, affected calves may not be obvious at birth. Some die close to birth or are aborted; whereas, many only show signs after sexual maturity.

Very few affected calves have been born in Canada and most, if not all, have been in commercial herds. Probably the biggest reason for this is that intensive line breeding is not practiced by most Canadian purebred Angus breeders. We have been blessed with a huge gene pool and we have done a good job of sampling it. Commercial producers have a tendency to be less concerned about a small amount of inbreeding, particularly in large herds using multiple bull breeding systems. All of the genetic defects are caused by recessive genes, so in

order to have an affected calf, two, otherwise normal, carriers must be mated. Mating two carriers results in 1 in 4 calves being affected with the condition; 2 out of 4 being normal, but are carriers of the gene; and 1 out of 4 being absolutely normal, non-carriers. If there are a lot of carriers in the population, then chances are that a number of affected calves will be born in a single calving season. A likely scenario would be if a number of daughters in a herd sired by carrier bull are then mated with another carrier bull. Carriers of gene mutations can usually be traced back to a common ancestor. Affected calves are usually not noticed right away and it is only after a few to several generations of progeny have been produced and mated within the line, that the problem is discovered.

Calves born with genetic defects are nothing short of bad news. The financial losses associated with dead and nonviable calves are obvious; however, it is the seed stock producer(s) who suffers the most when his program is identified as the source. A lot of great cattle may be carriers and they will have produced many non-carriers, so it is unreasonable to condemn an entire breeding program based on the occurrence of a genetic defect; especially, now that we can identify carriers.

When setting a course of action to eliminate the causative genes from our breed there are two things that we must be thankful for. The first is the ability to DNA test. I believe that this technology is easily the best thing to hit science and medicine since the discovery of penicillin. Look at all the previously unsolved crimes that have been cracked; and all the other wrongs that have been made right in the last 15 years with this technology. The second is that all of these defects are caused by single recessive genes, not multiple genes as is the case with marbling and other carcass traits; and these defects are not linked to sex, or influenced by diet or environment. Simple inheritance, just like the dominant black and recessive red coat colour, of course, before the discovery of the wild gene. Now that the bovine genome has been mapped it is very

easy to develop a test for a bad gene. Breed associations around the world are jumping on the band wagon in effort to identify carriers of undesirable genes so as to finally eliminate them from their breed. Before DNA technology we had to do a number of test breedings of closely related animals to identify carriers. This was very expensive and often not done. Several years ago I put down a hairless (hypotrichosis) Polled Hereford calf. The breeder planned to cull both the sire and dam which had to be carriers, but I could certainly understand his greater concerns – how widespread was this gene in his herd, and if word got out that his cattle had this gene it could ruin him financially. With DNA technology he can now test his entire herd and only cull the carriers if he wishes, but at least he will know who they are. Genetic testing enables us to identify all of the clean animals to preserve the genetics and we can cull the rest. That sure beats culling all of the progeny. When a herd has identified known carriers and makes that information available to the buyer, then we can be confident that the rest of the cattle are clean.