Curly Calf Syndrome Discussed

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On September 17, 2008 the American Angus Association (AAA) made an announcement that rattled the seedstock industry. One of the most influential bulls in the Angus breed, G A R Precision 1680, was fingered as a carrier of a lethal genetic abnormality dubbed Curly Calf Syndrome (CCS). Given that Precision 1680 had sired thousands of sons and daughters, that have in turn produced thousands upon thousands more offspring, it is clear that the number of animals carrying the genetic abnormality in this and other countries' beef cattle populations is substantial.

What are the symptoms of CCS? Dr. David Steffen from the University of Nebraska, the scientist who first documented and named the syndrome, provides this description "The spine is bent and twisted in affected calves. The calves are small and appear thin due to limited muscle development. Legs are often rigid and may be hyper extended (common in rear limb) or contracted. In some cases the rigid limbs result in calving difficulties. Additional unique feature are recognized during laboratory examination." It also should be noted that in all cases affected calves are born dead. Below are pictures of affected calves.

What causes CCS? Though the lead scientists studying the syndrome, Dr. Steffen and the University of Illinois' Dr. Jon Beever, have yet to provide a concrete conclusion, they have stated that it is most likely due to a single recessive gene. If that is the case, a 2 x 2 Punnett Square can be used to illustrate the outcome of a particular mating. In the two examples below we have mated a male (♂) carrier (e.g., Precision 1680) to a female (♀) carrier and a non carrier in scenarios A and B, respectively.

A)

♂ \ ♀	**C**	**c**
C	CC	Cc
c	cC	cc

B)

♂ \ ♀	**C**	**C**
C	CC	CC
c	cC	cC

In these examples, C is the normal gene while c is the lethal recessive. The cells with single letters contain one copy of each of the sire's (left column) and dam's (top row) genes. Since we have used a male that carries the lethal gene (c) in both examples each Punnett Square has a C and c on the sire side. As explained earlier, in example A we have mated the sire to a carrier female (Cc) while in B he is mated to a non carrier female (CC).

Through the use of Punnett Squares we can readily visualize what the resulting offspring will look like from our example matings. In scenario A we can see that the 4 potential genotypes from the mating are CC, Cc, cC and cc—each with an equal probability (1/4) of occurring. Since the presence of C has complete dominance over the expression of c (i.e., C completely covers up the symptoms of c) we know that only the calf receiving cc will show the symptoms of CCS, the other 3 will appear normal. Because they received the c gene (Cc and cC), however, 2 of the 3 normal calves in appearance will be carriers of CCS. In example B we can see that all of the resulting offspring will appear normal, while half of them (2 of 4) will be carriers. The above examples also work to illustrate other situations where a single recessive is involved, such as polled/horned or red/black.

What are the implications of CCS to ASA members? Obviously, with the heavy usage of Angus genetics by ASA members, we need to be vigilant about addressing this situation in our own population. Fortunately, technology has evolved to the point where the impact of genetic abnormalities like CCS can be largely mitigated. Currently the AAA is working with Drs. Steffen and Beever, two of the industry's leading experts on genetic abnormalities, to develop a test for carriers of CCS. Once the test is developed, carriers can be easily identified making it unnecessary to remove entire lines.

We will keep you abreast of the progress made toward developing a CCS test. We will also be crafting policy over the next few months designed to reduce the influence of CCS on our population. Your can be sure that ASA is committed to using all practical means available to keep our population as free as possible of genetic abnormalities. At the same time, we must recognize that a population the size of ours will never be completely free of genetic defects. Even if we could eliminate all existing abnormalities (and we can't) new ones would crop up via mutation. Nevertheless, given the rapidly evolving technology in this area and recent policy implementations (e.g., we now routinely test our fifty most heavily used bulls for genetic defects where tests are currently available) we are assured to reduce the frequency of genetic abnormalities in our population compared to that of the past.

Finally, if you have an animal that you feel shows signs of having CCS, or any genetic abnormality, please get in contact with Marilyn, Marty, Jerry or me. With the network of experts who advise us in this area, we will guide you through the protocol required in making a determination about the defect. By reporting anything suspicious you are looking out for the welfare of us all—and we appreciate it immensely. ◆