

Managing Genetic Defects in Beef Cattle Herds

Congenital defects are abnormalities present at birth. They are abnormalities of structure or function that can result in calf losses before or after birth. These defects can be caused by genetics, the surrounding environment, or a combination of these two factors. In some cases, the cause of defects is unknown.

Genetic defects are the result of an abnormal or mutated gene. They may impair animal health or cause a condition of abnormal function or structure. Hereditary defects occur in all breeds of cattle, but some defects are strongly associated with certain breeds. More than 200 different genetic defects have been identified in cattle. Most of them occur rarely and are of minor concern, but some increase in frequency to the point that they become a significant economic concern and need to be selected against. Genetic abnormalities may contribute to poor animal performance, structural unsoundness, semi-lethal disease, or lethal disease.

Specific Genetic Abnormalities

Genetic abnormalities in beef cattle differ in trait expression, mode of inheritance, and incidence rate. Examples of genetic defects include the following:

- ▶ achondroplasia (bulldog dwarfism)
- ▶ alopecia
- ▶ ankylosis
- ▶ arthrogryposis (palate-pastern syndrome, rigid joints)
- ▶ arthrogryposis multiplex (AM, curly calf syndrome)

- ▶ brachynathia inferior (parrot mouth)
- ▶ cryptorchidism
- ▶ dermoid (feather eyes)
- ▶ double muscling
- ▶ fawn calf syndrome
- ▶ hypotrichosis (hairlessness)
- ▶ hypotrichosis (“rat-tail”)
- ▶ idiopathic epilepsy (IE)
- ▶ mannosidosis
- ▶ neuraxial edema (maple syrup urine disease)
- ▶ neuropathic hydrocephalus (NH, “water-head”)
- ▶ oculocutaneous hypopigmentation (white eyes)
- ▶ osteopetrosis (marble bone disease)
- ▶ polydactyly (extra toes)
- ▶ progressive bovine myeloencephaly (weaver calf)
- ▶ prolonged gestation
- ▶ protoporphyria (photosensitivity)
- ▶ pulmonary hypoplasia with anasarca (PHA)
- ▶ syndactyly (mule foot)
- ▶ tibial hemimelia (TH)
- ▶ translocations

Table 2 details defect names, descriptions, modes of inheritance, and breeds affected for several known genetic defects in cattle.

Identifying Causes of Defects

When an abnormal calf is born, first you should try to determine if the defect is hereditary. Congenital defects that are environmentally caused rather than inherited often occur during a short period in a group of cattle that are managed the same way. Malnutrition, toxic factors, infectious disease, or extreme weather may be to blame for these congenital defects.

When a pregnant cow consumes the lupine plant between days 40 to 60 of gestation, her calf may be born with crippled-calf (crooked-calf) disease, a crooked-leg condition. Two-headed calves and calves with extra legs may be the result of fetal development mistakes. Bovine virus diarrhea (BVD) infection during pregnancy can lead to some hydrocephalus. Flexed pasterns can be caused by a large fetus developing in a small uterus. Each of these conditions may at first appear to be genetic defects but are actually caused by environmental conditions. In the cases of crippled-calf disease and flexed pasterns, the causes could be either environmental or genetic. Once it is determined that a specific environmental cause is responsible for a defect, management changes can be put in place to address these problems and reduce the risk of future incidence.

Some congenital defects are inherited. Most genetic abnormalities are determined by a single pair of recessive genes. See the example in **Figure 1**. In this instance, the parents are both heterozygous where there is one desired allele and one undesired allele per parent. This makes them “carriers” for the defect.

Heterozygous x Heterozygous	$\frac{1}{2}$ D	$\frac{1}{2}$ d					
Carrier Sire x Carrier Dam							
Dd x Dd	$\frac{1}{2}$ D		<table style="border-collapse: collapse; width: 100%;"> <tr> <td style="border-right: 1px solid black; padding: 5px;">$\frac{1}{4}$ DD</td> <td style="padding: 5px;">$\frac{1}{4}$ Dd</td> </tr> <tr> <td style="border-right: 1px solid black; padding: 5px;">$\frac{1}{4}$ Dd</td> <td style="padding: 5px;">$\frac{1}{4}$ dd</td> </tr> </table>	$\frac{1}{4}$ DD	$\frac{1}{4}$ Dd	$\frac{1}{4}$ Dd	$\frac{1}{4}$ dd
$\frac{1}{4}$ DD	$\frac{1}{4}$ Dd						
$\frac{1}{4}$ Dd	$\frac{1}{4}$ dd						
D = normal allele	$\frac{1}{2}$ d						
d = affected allele							

Figure 1. Simple inheritance example for mating two carrier animals (Dd).

Table 1. Resulting offspring from Figure 1.

Percent	Genotype	Phenotype (of calves)
25%	DD = homozygous, dominant, non-carrier	Unaffected, normal
50%	Dd = heterozygous carrier	Unaffected, normal
25%	dd = homozygous recessive	Affected

Genetic tests for DNA markers can usually help identify inherited defects. Genetic tests for simple traits that are controlled by one gene are able to accurately assess whether an animal is a carrier (heterozygous, two different alleles) or homozygous (two same alleles) for marker of a certain phenotype (physical manifestation of a trait). Breed associations and genetic testing companies can provide testing protocols for genetic defects associated with a certain breed. Tests for a specific genetic defect generally cost around \$30 per animal. Combined tests that provide information on multiple genetic disorders are also available for slightly higher fees. If a defect is dominant, no test is needed because the animal would display the defect even if only one dominant allele was present. A small number of congenital defects are caused by genes with incomplete dominance, but some are caused by two or more sets of genes. If the defect is inherited as incomplete dominance, an animal that has only one undesired allele can usually be identified and testing is not needed.

Genetically caused defects tend to run in families. The sire and dam of a calf will likely have at least one common ancestor. The occurrence of multiple affected calves in a herd often results from the same sire and closely related dams.

Breeders must have good records to determine the cause of defects. These records must include calf parentage, description of abnormalities, photos or videos of the abnormalities when possible, and causes of death. If parentage is not known from records, it can be determined with DNA testing. When cause of death is not obvious, request veterinary assistance to obtain necropsies on dead cattle immediately. Necropsies are valuable for investigating possible causes of death and ties to congenital defects. Management groups and movement among paddocks or pastures should be recorded as well. Feed and forage analysis reports, notes of toxic plant presence, and herd health records help in determining the cause of any congenital abnormalities.

Managing Genetic Defects

Once carrier and affected animals are identified, producers can make selective breeding and culling decisions to manage a genetic defect within a herd. When a carrier animal is extensively used for breeding purposes, as in the case of sires for artificial insemination purposes, several thousand calves may be sired by the carrier bull before the abnormality expresses itself. Even more matings using the carrier sire may take place before affected calves are associated with the sire and genetic testing confirms the sire as a carrier.

In some cases, a genetic test may not be available during the initial occurrences of the condition. As a result, a diagnostic test may need to be developed based on data submitted from producers who observe the defect in their herds. Therefore, it is important to test sires whose semen will be marketed. It is also important to test donor dams in embryo transfer programs. Furthermore, producers must remain vigilant about observing calf crops for congenital defects by collecting appropriate records and animal samples for diagnostics. They should report problems with occurrences of defects to breeding animal suppliers and breed associations.

Consider the value of a carrier's genetic worth to the breeding program. An animal with one undesirable recessive gene may also have thousands of very desirable genes. Carriers that have superior genetics should be strategically paired with non-carriers in a terminal crossbreeding program where all calves are marketed for beef production and not for breeding purposes. Alternately, a superior son could be used as a herd sire that does not carry the defect.

In most cases, defect carriers should not be used to produce breeding animals; therefore, do not keep replacement heifers that are defect carriers. When you can utilize other cattle with similar or superior genetic merit that do not carry the defect, you should systematically work carrier females out of the herd and replace them with cattle that do not carry the defect. Be sure to send carriers directly to harvest to avoid transferring the defect to another breeding herd.

Purebred breeders and breed associations share responsibility for controlling genetic defects in seedstock populations. In seedstock herds, use available DNA diagnostic tools to test suspect animals or those known to have ancestors that are carriers. When carriers are retained in the breeding herd, test all progeny to determine carrier status before marketing them as breeding animals.

Seedstock producers have an obligation to be honest and notify the customers they supply with breeding animals as well as their respective breed associations when carriers of genetic defects become known. Many breed associations mandate genetic defect reporting among their members. Breeders should photograph any calf born with a suspected defect and then contact the breed association to arrange for tissue collection and reporting. Breed associations may also have rules regarding registration eligibility or required denotations on registration papers for animals carrying or affected with genetic defects.

Make sure buyers understand the consequences of using offspring from known carriers. Serious ethical and legal problems can be involved in marketing known carrier cattle or progeny of known carriers. Marketing carriers without informing the buyer can not only harm breeder reputation but may also reflect negatively on the entire breed.

Good monitoring and control measures by seedstock operations will help control genetic defects in commercial cattle populations. Even with seedstock level management of genetic defects, commercial cattle producers may still need to cull carrier animals within their herds if the incidence rate of a genetic defect rises to a level where it becomes an economic problem. Yet in most cases, careful sire and/or breed selection can be an effective approach for managing genetic defects in a commercial herd without the need for extensive cow herd culling. For more information about beef cattle genetics or specific genetic defects, contact your local MSU Extension office.

Table 2. Genetic defects in cattle.

Condition	Description	Inheritance	Breeds Affected ¹
Achondroplasia (bulldog dwarfism)	Homozygous calf may be aborted at 6 to 8 months gestation; has compressed skull, nose divided by furrows, and shortened upper jaw for a bulldog facial appearance; heterozygous calf is small and heavy-muscled	Incomplete dominant	
Alopecia	Lethal abnormality very similar to hypotrichosis; takes laboratory analysis to distinguish; calves have kinky, curly hair that is soon lost in patches around the head, neck, and shoulders; skin changes and anemia occur in all cases; death before 7 months of age due to anemia	Simple recessive	Polled Hereford
Ankylosis	Abnormal union of any joints in calf; cleft palate frequently occurs	Recessive	
Arthrogryposis (palate-pastern syndrome; rigid joints); Arthrogryposis Multiplex (AM; curly calf syndrome)	Pastern tendons are contracted; joints of all four legs fixed symmetrically; upper part of mouth not properly fused together (cleft palate); calves usually die shortly after birth; AM in Angus includes twisted malformation of spine and fixed leg joints	Simple recessive; many environmentally caused forms	Angus (AM), Red Angus (AM), Charolais, many other breeds
Brachynathia inferior (parrot mouth)	Cattle have a short lower jaw; under- or over-shot jaw with varying degrees of expression when polygenic	Simple recessive; polygenic (more common)	Angus, Simmental
Cryptorchidism	Retention of one (unilateral) or both (bilateral) testicles in body cavity instead of descending into scrotum	Sex-limited trait; polygenic	Many breeds
Dermoid (feather eyes)	Skin-like tissue masses occur on eye or eyelid; cattle may become partially or completely blind	Polygenic	
Double muscling	Muscle enlargement with large grooves between muscle systems; particularly evident in hind leg; cattle usually grow slowly; fat deposition (internal and external) is reduced; typically fine-boned; considerable variation in expression	Simple recessive; dominant in Piedmontese	Belgian Blue, Piedmontese, Angus, Red Angus
Dwarfism	At least three types in cattle; snorter dwarfism: most common; short, blocky appearance; deformed bone growth in nasal passages causing difficult breathing; long head dwarfism: small size but normal nasal passages; compress dwarfism: extremely compressed body conformation with one compress allele and one normal gene; dwarf and death at or soon after birth with two compress alleles	Simple recessive (snorter dwarfism; long head dwarfism); incomplete dominance (compress dwarfism); environmental causes	Angus, Hereford, Brahman, Dexter
Fawn Calf Syndrome	Abnormal crouched posture at birth, resembling a deer fawn, with the feet placed more to the rear than normal, hocks pulled up and back and the spine slightly arched; flat down on pasterns during first days of life; tend to be taller and more slender with poor foot conformation; poor muscle development; difficulty in some with movement and suckling; can result in calf death but most can walk, suckle, and survive; poor growth performance; early onset of degenerative arthritis	Simple recessive	Angus
Hypotrichosis (hairlessness)	Partial to almost complete lack of hair; hair grows in and falls out, so appearance varies over time; non-lethal	Simple recessive; low frequency	Hereford
Hypotrichosis (rat-tail)	Form of congenital hypotrichosis; colored hair anywhere on body is short, curly, malformed, and sometimes sparse; abnormal tail switch; often confused with premature birth; slower post-weaning weight gains	Interaction between two loci where at least one gene is for black color and must be heterozygous at the other locus involved	Matings of Simmental with Angus; Hereford can carry

Condition	Description	Inheritance	Breeds Affected ¹
Ideopathic Epilepsy (IE)	Neurological disorder causing seizures	Simple recessive	Hereford
Mannosidosis	Lethal nervous disease; failure to thrive; progressive incoordination; aggressive disposition when disturbed; death typical before sexual maturity reached	Simple recessive	Angus, Murray, Grey, Galloway, Salers
Neuraxial edema (maple syrup urine disease)	Calf is normal size at birth but may not be able to stand or lift head; sudden touch or loud noise may cause vigorous extension of the legs and neck; muscle spasms of neck and legs may last for 1 to 2 minutes; death typical within 5 days	Simple recessive	Hereford, Shorthorn
Neuropathic Hydrocephalus (NH; water head)	Fluid accumulation in brain ventricles (internal: water head) or cranium (external); bulging forehead; calves usually die shortly after birth	Simple recessive; environmental causes	Angus, Red Angus (external), Hereford (internal), many other breeds
Oculocutaneous Hypopigmentation; Heterochromia Irides (white eye)	Hair coat is bleached color; iris is pale blue around pupil with tan periphery	Simple recessive	Angus
Osteopetrosis (marble bone disease)	Bone tissue fills marrow cavity of the long bones; brittle bones that break easily; calves have short lower jaws, protruding tongues, and impacted molar teeth; calves usually born dead 2 to 4 weeks preterm	Simple recessive	Angus, Red Angus, Simmental, Holstein
Polydactyly (extra toes)	One or both front feet or sometimes all four feet have outer dew claw develop into extra toe	Polygenic	Simmental, Holstein
Progressive bovine myeloencephaly (weaver calf)	Calves develop a weaving gait at 6–8 months of age and get progressively worse until death at 12–20 months of age	Simple recessive	
Prolonged gestation	Fetus fails to trigger parturition; calving must be induced or the calf removed; calf is often extremely large and often dies	Simple recessive	
Protoporphyrin (photosensitivity)	Sensitivity to sunlight; development of scabs and open sores upon sunlight exposure; liver function affected; seizures possible	Simple recessive	Limousin
Pulmonary Hypoplasia with Anasarca (PHA)	Fluid collection in skin and body cavities at birth; lethal to calves; usually causes dystocia because of added weight; lungs underdeveloped	Simple recessive	Shorthorn, Maine-Anjou, Chianino, Simmental
Syndactyly (mule foot)	One or more hooves are solid in structure rather than cloven; front feet most often affected; can occur in all four feet; cattle cannot tolerate hot temperatures; high mortality rate in calves	Simple recessive	Chianina, Angus, Simmental, Holstein
Tibial Hemimelia (TH)	Calves born with twisted legs with fused joints, large abdominal hernias, and skull deformity	Simple recessive	Shorthorn, Maine-Anjou, Chianina, Simmental, Galloway
Translocations	Part of a chromosome breaks off and attaches to another chromosome; carriers have reduced conception rates and increased abortion rates	Simple recessive	1/29 in Simmental, Charolais, and Blonde D'Aquitaine; 14/20 or 14/21 in most Continental breeds

¹Specific defects have been noted in the breeds listed. However, these defects could occur in other breeds.

References

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