Dwarfism in Cattle

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Dwarfism is one of a number of heritable defects in all animals. Spontaneous mutations account for the appearance and continued presence of defects. Examination of DNA profiles is currently the most useful tool to identify specific genes known or suspected to be responsible for a mutation.

A QUICK GUIDE TO GENETICS

All mammals have genetic factors that express their characteristics. Genes are combinations of chromosomes; each pair of chromosomes determines the physical makeup of an animal. The group of chromosomes—the genome—includes genes that determine species in general (the genotype) as well as individual traits like coat color (the phenotype). All these genes together comprise the DNA profile, unique to each animal. Genes are inherited; spontaneous mutations can occur, and the mutation becomes part of the heritable traits. Mutation was responsible for polled cattle: a polled calf was born, then another; breeding the two together concentrated the mutation, and so on until only polled calves were born.

Genes are either dominant or recessive. Dominant genes are the overriding traits by which we identify a type or breeds. All cattle have four legs; all zebu cattle have thoracic (shoulder) humps. Recessive genes appear under special circumstances: usually when two carriers of the gene breed. Two gray cows can produce a red calf only if there is a red cow somewhere in their histories.

Variations of gene dominance also exist: co-dominant, simple recessive, and others. A simple recessive gene “expresses” in this manner:

Simple Recessive Gene

carrier + carrier parents:

Dd-(carrier)—25%	Dd-(carrier)—25%
dd-(noncarrier)—25%	DD-(dwarf)—25%

carrier + noncarrier parents:

Dd-(carrier)—50%
dd-(noncarrier)—50%

Genes carry both good and bad traits. At least 200 gene defects have been found in cattle. In his 2013 article “The Truth: Every Living Thing is a Genetic Defect Carrier,” Jared Decker, University of Missouri assistant professor and beef genetics specialist, states:

> A new perspective about genetic defects is needed, because all animals are carriers of something.

Some negative traits are minor, like a tail crook; moderate, like a jaw misalignment; serious, like blindness, and some traits are incompatible with life. Some defects can be corrected with surgical intervention. The worst defects (“lethal”) cause calf death.

A number of genetic defects can be identified by DNA testing. Some are breed specific; others are seen in all cattle. As of mid-2014, about a dozen lethal defects can be identified by lab testing.

CAUSES OF DWARFISM

Noninherited

Caused by environmental conditions, these often occur with multiple calves, in one herd, at one time. Cows may be exposed to toxins during pregnancy. Infection, poor body condition, or very extreme weather conditions can be responsible for other birth defects in addition to dwarfism.

Genetic

"Snorter" dwarfs—these are typically what we perceive as dwarfs: short, stubby, compacted bodies with large, disproportional heads. They're called snorters because they usually have deformed sinus cavities that cause them to have difficulty breathing.

"Long-headed" dwarfs—these calves have normal size heads but display the same body style as the snorter calves.

"Bulldog" Dwarfism--this defect causes calves to either die in utero or very shortly after birth.
Their appearance is grotesque: they have heads shaped like bulldogs, fat bodies and short legs.

As of 2014, researchers have identified three genetic factors responsible for dwarfism; other types of dwarfism exist but those genes are yet to be identified. Dexter cattle can carry the "ACAN" gene; almost all carriers can be identified visually. Testing is available for Dexter and Dexter influence cattle.

Angus cattle can carry the "PRKG2 (D2)" gene. Carriers of this gene are normal in appearance; only affected calves can be identified visually, and the degree of dwarfism can vary. Testing for this gene is available for Angus and Angus influence cattle.

(SEE APPENDIX II FOR A FACT SHEET ON PRKG2 (D2) DWARFISM)

Recently researchers found the gene responsible for dwarfism in Japanese Brown cattle, one of the Wagyu breeds. Dwarfism found in Herefords as well as other breeds have more complicated genetic profiles, yet to be identified.

HISTORY OF ERADICATION EFFORTS

Irish Dexter Cattle

In the 1800s Irish breeders developed a type of cattle known for its hardy nature and small size. Starting with the small Kerry black cattle, the breed became known as Irish Dexter. The preferred body type was very stocky and short-legged. Unknown at the time, these animals were actually genetic dwarfs. Over time, it was noticed that some long-legged calves in the population. Calves were also born with a fatal defect that became known as "Bulldog" due to their compacted appearance. The percentage of births was 50% shortleg, 25% longleg, and 25% bulldog.

Longtime Dexter breeder Carol Davidson states:

> With the advent of the science of genetics, it was discovered that the very trait that made a Dexter a Dexter (a stout cow with short legs), was unfortunately due to a form of dominant lethal genetic mutation. As long as the mutation was the primary selection criterion, all three types would continue, and Dexters would never breed true. Rather than give up the appearance they preferred, owners chose to live with the problem.
>
> Once the mutation was better understood, many started to breed their shortleg Dexters with longleg Dexters as they found they still got the same proportion of shortlegs but avoided the bulldog calves. For those matings, the statistical proportions were ½ shortleg, ½ longleg. Until very recently, owners continued to breed both ways, with only the shortleg considered the real Dexter, and many (most) of the longlegs discarded. Since 1980 in North America, some owners have chosen to breed small longlegs, working to produce a size and look similar to the original selection criteria, but without using the lethal mutation.

Hereford Cattle

As with the Irish Dexters, selective breeding played a part in spreading the dwarfism gene. Roy T. Berg, University of Alberta, in his article “Beef Cattle Breeding: Past, Present, and Future” states that:

> “Dwarfism had reached a level of serious concern by 1955, particularly in the Hereford breed which was the dominant beef breed in Alberta at that time. There was pressure on me as a budding geneticist to help solve the Dwarfism problem. My approach was not popular. I felt that Dwarfism was caused by what breeders had been doing, not by any act of God or disease. The most likely cause was the emphasis, particularly in the Show Ring, on a ‘Compact’ type characterized by short legs, short and thick body accompanied by very slow growth rate and reduced body size.

The dwarfism defect was first reported to the American Hereford Association in the 1950s. It took seven years to interview breeders, review their herd records, examine pedigrees, and clear Hereford bloodlines. When they finished, the first carrier identified was a bull named St. Louis Lad, born in 1899. From that bull, 40,000 carriers were identified.

Aided by the open communication between the association and its breeders and partnering with university researchers, there are now no dwarfs in the registry of the American Hereford Association.

THE MODERN APPROACH

"Mutations that cause abnormalities always will be a reality of the livestock business. Fortunately, with advances in genomics technology and thoughtful management by Associations and breeders, we can mitigate the adverse effects substantially."

Kent Andersen, former executive vice president of the North American Limousin Foundation

The Role of Breed Associations

Breed associations can collect and disseminate information and maintain records of test results. Associations have taken different paths to manage known genetic defects. The American Hereford Association boasts no genetic carriers of dwarfism in their registry; their policy is that carriers cannot be registered. The American Angus Association allows registration, but denotes carriers. Several breed associations formed partnerships with testing labs to deliver results to the association, who then notifies owners.

The Role of Breeders

During the 2009 Beef Improvement Federation meeting in Sacramento, CA, Jonathon Beever, University of Illinois in Urbana-Champaign geneticist and one of the nation's top authorities on genetic defects in cattle, told producers of four options in dealing with a potential genetic defect carrier in a cowherd population. He said producers could:

- Ignore the issue and risk future problems.

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.

- Completely eliminate the genetic source, which would be contrary to overall breed improvement.

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.

- Retain superior carriers; only breed to noncarriers. Retain noncarrier progeny. Slaughter/Spay/Castrate carriers.

In most cases, defect carriers should not be used to produce breeding animals. With carriers with superior genetics, strategically mate these animals to noncarriers. When other cattle with similar or superior genetic merit that do not carry the defect can be utilized, systematically work carrier females out of the herd and replace them with cattle that do not carry the defect. A superior son could be produced for use as a herd sire that does not carry the defect.

- Accurately identify the carriers through genetic testing, and then manage the problem.

Test sires from which semen will be marketed as well as donor dams in embryo transfer programs. Carrier females are safe to use as embryo recipient cows. Keep appropriate records and animal samples for diagnostics, and report defect occurrence to breeding animal suppliers and breed associations. 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. Make sure buyers understand the consequences of using offspring from known carriers.

TESTING

In the US, at least two veterinary diagnostic laboratories perform dwarfism testing. The Veterinary Genetics Laboratory at the University of California, Davis offers the Dexter Bulldog Dwarf test (the ACAN gene) using tail hairs. Forms and instructions are available at: https://www.vgl.ucdavis.edu/services/cattle.php.

Igenity (formerly known as Agrigenomics/GeneSeek), a division of Neogen, Inc, offers testing for the PRKG2 (D2) gene. This test also uses tail hairs. Forms and instructions are available at: http://www.neogen.com/Agrigenomics/pdf/Submission-Forms/AG088_GeneseekOrderForm-Beef.pdf

Both UC Davis and Igenity accept submissions from individuals as well as veterinary clinics. Veterinarians may have access to other diagnostics labs throughout the country. Tests cost approximately $25. Both labs, upon request, will send results to both you and the breed associations.

CLOSING COMMENTS

I expected to finish this article a few days after I began researching, but it's been very, very hard to understand the subject of genetics and its ties to dwarfism. I hope that I've been able to relay this information without making too many mistakes; please feel free to correct me. I am not a scientist or veterinary professional, but a teacher and artist who likes to learn.

The most important "take-away" fact is this: zebu cattle do not have an identifiable gene or genes related to dwarfism. DNA research hasn't been conducted yet. Therefore, either or both tests may NOT be reliable for zebu cattle. Positive results have been found using these tests, showing some relation. The ACAN and the PRKG2 (D2) tests are the best and only identifier available to zebu breeders at this time. I've chosen to submit samples to both labs.

I urge everyone to have your sires and dams tested. If either is identified as a carrier, have the progeny tested. Each calf will have a 50% possibility of being a carrier or noncarrier. Superior carrier calves can be carefully bred to noncarriers until a noncarrier replacement is born. This requires responsible culling of carrier calves, removing them from the gene pool. Breeders have other options than slaughter: castration, spaying (inexpensive and safe); cows can also be used as embryo transfer recipients.

Ethical, responsible breeders dedicated to our breed will test and inform breed associations. Only with clear, open, and honest cooperation between breeders, owners, and associations can we clear our breed from genetic dwarfism.

APPENDIX I: GENETIC DEFECTS IN CATTLE

Condition	Description	Inheritance	Breeds Affected
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 shoulder; skin changes and anmia occur in all cases; death before 7 months of age due to anemia	Simple recessive	Polled Hereford
Ankylosis	Abnormal union of any joint in calf; cleft palates frequently occur	Recessive	
Arthrogryposis (palate-pastern syndrome; rigid 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 or (parrot mouth)	Cattle have a short lower jaw; under- or overshot 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)	Skirt-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 alleles 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 day 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

Condition	Description	Inheritance	Breeds Affected
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
Idiopathic 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
Neuroxial 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	
Protoporphyria (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, Chianina, 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

Condition	Description	Inheritance	Breeds Affected
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 hove 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

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

American Angus Association®
PRKG2 Gene Mutation for Dwarfism (D2) Fact Sheet

The following fact sheet was developed to respond to questions commonly asked by American Angus Association members. Additional information may be found online at www.angus.org.

What is PRKG2 Gene Mutation for Dwarfism (D2)?
D2 was recognized as a specific strain of dwarfism on September 7, 2007. There are several types of dwarfism, but all dwarfs appear shorter and some smaller than normal. The legs are short and body is short, and the animal may appear to have a potbelly and a thick or blocky shape. The head may be normal (long-nosed or long-headed dwarf) or the face may appear shortened. Muscling is often normal and, thus calves may be a thick appearance.

What causes D2?
D2 is caused by a recessive mutation on a single cattle chromosome. Cattle that are homozygous for the mutated gene will exhibit D2.

What is a D2 carrier?
For the purpose of this response, a D2 carrier is an Angus or Angus-cross cow, heifer, bull or steer that carries the recessive D2 mutation in their DNA.

Why are carriers of D2 important?
Carriers of D2 used in breeding programs (registered or commercial) are responsible for propagating the recessive mutation within the cattle population.

What does a D2 carrier look like?
A D2 carrier looks perfectly normal; there is nothing in the way an animal looks (its phenotype) that indicates that the animal is a carrier of the D2 mutation.

If a cow has a D2 calf, what does that mean?
If a cow has a D2 calf, and if it is the cow's natural calf, it means that the cow is a carrier of the D2 mutation and the sire of the calf is also a D2 carrier.

If a recipient cow has a D2 calf, what does that mean?
If a recipient cow has a D2 calf, it means only that both the donor cow and the sire of the calf are carriers of the D2 mutation. It doesn't tell you anything about the D2 carrier status of the recipient cow.

If a bull sires a D2 calf, what does that mean?
If a bull sires a D2 calf, it means that the bull is a carrier of the D2 mutation and that the dam of the calf is also a D2 carrier.

I have never had a D2 calf. Does that mean my cows are non-carriers?
Not necessarily.

What is the risk of having a D2 calf if I breed a D2 carrier cow to a D2 carrier bull?
Every time you breed a carrier to a carrier, there is:
- A 25% risk of having an affected D2 calf;
- A 50% risk of having an otherwise normal-appearing calf that carries the D2 mutation;
- A 25% chance that you will have a normal-appearing, non-carrier calf.

If I breed a D2 carrier cow to an D2 carrier bull and have three live calves, will the fourth calf have D2?
The risk is the same every time you breed a carrier to a carrier. There is always a 25% risk of having an affected D2 calf, a 50% risk of having a carrier calf, and a 25% chance of having a non-carrier calf.

If I breed a D2 carrier cow to a non-carrier bull, what is the chance of having a D2 calf?
Zero. You will never have a D2 calf if you breed a carrier cow to a non-carrier bull. (excluding the possibility of a spontaneous mutation)

If I breed a D2 carrier cow to a non-carrier bull, what is the risk of having a carrier calf?
Every time you breed a carrier cow to a non-carrier bull there is:
- A 50% risk of having a normal-appearing calf that carries the D2 mutation; and
- A 50% chance you will have a non-carrier calf.

Is there a test to identify D2 carriers?
Yes. A DNA test is available to determine if an animal carries the D2 mutation in their DNA. The type of DNA sample required to perform the test varies from lab to lab but includes; hair root samples, blood-spot or FTA cards, whole blood in "purple–top" tubes, tissue samples from ears and semen samples.

A video on ***www.angus.org*** explaining how to collect the sample can be found **here**.

What do I do with the confirmed non-carrier females in my herd?
If the females are tested non-carriers and they are bred to non-carrier bulls, they will never produce affected D2 calves or carriers. These non-carrier females can be used throughout your breeding program with no risk of propagating the D2 mutation.

What do I do with confirmed female carriers in my herd?

You have several options:

- If you have a cow that carries the D2 mutation and you want to produce calves from her; you must make a commitment to test all offspring retained for breeding; (check policy regarding registration requirements)
- If you have both a registered and a commercial herd, retain your carrier cows in the commercial herd, breed to a non-carrier bull, and test any calves retained for breeding purposes;
- If you always breed your carrier cows to a non-carrier bull, you will never have a D2 calf. Then, treat the resulting calves as market animals, not as breeding stock.
- Use your D2 carrier cows as ET recipients. As a recipient female, she has no genetic effect on the embryo calf she raises.

D2 potential carrier report & potential carrier management tool

AAA Login users can access interactive tools to generate a report of owned animals and their PRKG2 Gene Mutation for Dwarfism (D2) status based on the D2 test results received to date. From the AAA Login menu, go to the "interactive" section and click on "Potential Carrier Report AM/NH/CA/DD/M1/D2" or "Potential Carrier Management Tool (PCMT)." The PCMT can identify those animals in your herd that have the most descendants in your herd and would be the most logical animal to start a testing scheme should you decide to test for a particular genetic condition. If you are not a current AAA Login user, you can sign up to create an online profile at *www.angusonline.org.*

What is the AAA registration policy regarding D2?

		One or both parents test D2C (confirmed carriers)
Heifers		All calves must be tested and can be registered regardless of the test outcome.
Bulls		All bull calves registered after 8/29/2011, must be tested and only those that test D2F can be registered.
E.T. Calves		Registration is based on the date of birth, sex of calf and if they are sired by a bull that is an A.I. sire as described below.
Steers		No test required.
A.I. Sires that are confirmed carriers		Calves cannot be registered that are conceived more than 60 days after the date a non-owned bull (a bull that would require an A.I. Service Certificate) is listed as a carrier animal (D2C).
Definitions		D2C - D2 Carrier, has been tested and carries the D2 mutation. D2F - D2 Free, has been tested and does not carry the D2 mutation. D2P - D2 Potential Carrier, animal that traces to one or more confirmed tested carrier animals in its pedigree that have no intervening ancestors that have been tested free of D2.

Testing Procedures

Submit Samples through American Angus Association/AGI

Use **AAA Login** to order test. Samples are submitted to the American Angus Association and archived for future testing requests. Login at ***www.angusonline.org*** and use menu option: Order--Testing for AM/NH/CA/DD/D2/M1.

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