# GENETIC ABNORMALITIES IN BEEF CATTLE

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## INTRODUCTION

Breeders of domestic cattle do so for profit. Because they are concerned primarily with increasing the production of desirable meat animals, they should be able to minipulate to best advantage the genetic variations affecting meat producing characteristics.

All animal breeders are concerned with inherited defects. These defects range all the way from congenital weaknesses to lethal abnormalities. It should be pointed out that all defects are not inherited. Some are due to improper nutrition, desease, and stress due to climatic conditions, age and abusive handling.

Breeders in the past have often felt that genetic defects should be "covered up" and that in time the problems disappear. Both of these beliefs are myth and this paper is an attempt to dispel auch beliefs.

The purpose of this paper is to summarize research data on bovine genetic abnormalities with hope that beef producers will benefit from its contents.

Genetic diseases originate in the gene carrying chromosomes of the cell nucleus. Each species has a characteristic number of chromosomes. Cattle have 30 pairs, and each chromosome is estimated to contain at least 500 genes.

Genes occasionally change into other kinds of genes. This is known as mutation. Frequency of gene mutation is not known for many loci in livestock.

Mutant genes are known to cause hydrocephalus and snorter dwarfism in cattle. Once an animal is born with a mutant gene, there is a chance that the animal will reproduce and perpetuate the new gene. New mutants can survive for many generations.

It should be noted that extreme mutants, such as those causing hydrocephalus, tend to be self eliminating because once occurring in pairs as they must eventually do, the animal possessing them dies before reproducing.

## REVIEW OF LITERATURE

### Modes of Inheritance of Abnormalities

Huston (1967) stated that there are four major hereditary patterns by which mutant genes become evident over two or more generations. These four patterns involve certain definite ratios of normal and affected progeny. Such ratios and their modifications form the basis of all accurate diagnoses.

1. The Dominant Pattern:

The dominant mutation pattern can be identified if mutation embodies the following characteristics:

The initial mating between two normal parents results in a mutant gene from one or the other parent being passed on to the offspring, using a bull for purposes of illustration. To classify under the dominant pattern, this resulting bull possessing the mutant gene must be abnormal. For example, such a mutant gene is the epilepsy gene occurring in Brown Swiss cattle. Cattle possessing one such mutant gene have an ataxia, which is most pronounced when the animal runs. These cattle are commonly described as weavers.

The second step in the dominant pattern is to mate the adult abnormal bull to normal females. An important means of diagnosing the dominant disease pattern is that half the calves resulting from these matings should be normal and the other half weavers.

A third generation of animals is then produced by mating daughters of the weaver bull to another weaver bull, resulting in five abnormal progeny for every three normal produced. Or if the third generation is produced by mating daughters of the weaver bull to a normal bull, the result is only one abnormal for every three normal calves.

Another characteristic of the dominant pattern is that in large series of mating, abnormals are produced generation after generation, without skipping a generation.

Disorders resulting from a dominant mutation are easy to control because elimination of abnormal animals eliminates all abnormal genes.

Of the heredity patterns the dominant pattern gives the highest frequency of abnormal animals, 50 per cent of the animals in the first generation after mutation. Most hereditary defects occur first at frequencies of less than 25 per cent and in most cases less than $12\frac{1}{2}$ per cent of the animals.

2. The Incompletely Dominant Pattern:

The pattern of the incompletely dominant mutation is similar to the dominant mutation with one characteristic difference.

In the case of dominant mutation, breeding of two normal parents produces an abnormal son. When that bull is mated to normal cows, normal and abnormal animals result in a 1:1 ratio. If those progeny are then mated to a normal bull, a diagnostic ratio of three normals to one

abnormal results; if they are mated back to their sire or to another abnormal bull (the inbreeding test), they produce five abnormal for every three normal.

The incompletely dominant pattern differs from the dominant. Among the abnormals are the ones that are much more abnormal than the others, so the actual diagnostic ratio for this pattern is four slightly abnormal and one highly abnormal for every three normal produced.

The occurence of two degrees of abnormalities is the means of distinguishing the dominant pattern from the incompletely dominant. As with the dominant mutation, disorders resulting from an incompletely dominant mutation is relatively easy to control because eliminating all abnormal animals eliminates all the mutant genes.

3. The Recessive Pattern:

Most of the genetic defects in cattle are caused by a recessive mutation. This is one of the most difficult hereditary patterns to control.

When two normal parents are mated, one passes a mutant gene to a son. However, in the recessive pattern, the son possessing the mutant gene appears completely normal. When mated to normal cows, he produces only normal progeny.

If his daughters are mated to normal "non-carrier" bull, only normal offspring result. But if his progeny are mated back to their sire or to another bull which appears normal but carries the mutant gene, the progeny occur in the ratio of seven normals to one abnormal.

The characteristics of this recessive pattern are that the abnormals typically come from normal parents. They usually occur in relatively small numbers. The trait skips generations even when families are large.

The hydrocephalus gene currently occurring in range cattle is characteristic of the recessive pattern. Recessives like hydrocephalus are difficult to control because the mutant gene is carried by normal appearing animals. It is only when an animal receives two mutant genes, one from each parent, that the abnormal is produced. Although eliminating such animals eliminates two abnormal genes, it is difficult to identify the normal appearing recessive gene carriers. They perptuate the mutation in animal populations.

4. The Overdominant Pattern:

The overdominant heredity pattern is another difficult to control mutation. It is typified by the snorter dwarf condition in beef cattle.

Starting with two normal parents, a mutant gene is passed to the offspring. In this pattern, the resulting son is not only normal, but is superior in appearance.

If the superior bull is mated to normal cows, all progeny are normal, but some are superior to others. When these progeny of the super bull are mated to another super bull, abnormal calves occur in the ratio of seven normals to one abnormal. Identification of overdominance can be made on the basis of superiority of some of the normal animals.

Defects caused by a overdominant mutation usually are evidenced rather quickly since superior animals are selected in preference to normal animals. The super animals are often mated together. An overdominant mutation is difficult to control because selection favors survival of the superior animals, those carrying mutant gene.

GENETIC DISORDERS:

Gruneberg et. al. (1968) observed that syndactyly was the partial or complete fusion of functional digits on one or more feet. Huston (1967) observed the same results. The best known type of syndactyly is a recessive trait in cattle. This type has been described in American Holstein-Friesian cattle (Huston 1961).

Rare variant types may be confused with the hereditary type. Leipold et. al. (1968) reported that such a variant has occurred in purebred Aberdeen Angus and caused needless alarm.

Charolais cattle are sometimes affected with arthrogryposis. Leipold et. al. (1969) noted that arthrogryposis is caused by a dominant gene with incomplete penetrance. The degree of involvement of the forelimbs varies from extreme flexion of carpal and fetlock joints to mild extension. The rear limbs vary from mild overextension to severe overextension of the hock joint. Most calves affected were born alive and at full term, some live only a few hours up to eleven days.

For many years various kinds of dwarfism have been observed in the Angus, Hereford and Shorthorn breeds of beef cattle. Dwarfism is extremely difficult to eradicate from these herds as the general problem is complex and much more research is required to understand the phases. Another problem retarding control of dwarfism is the reluctance of herdsmen to record its occurrence.

Dwarfs are inefficient individuals as they fail to grow and many die prior to maturity. Dwarfism lowers reproductive efficiency even when the the dwarfs are destroyed. It results in the same economic loss as reproductive failures which prevent the production of normal calves.

"Snorter Dwarfism" In Conventional Herefords:

Smith et. al. (1954) stressed that dwarfs, produced from normal appearing parents, are generally born alive. Many survive during the suckling period and variable periods of time following weaning. It is suspected that still-births and early calfhood death losses are more frequent in dwarfs than normal calves.

Most dwarf calves are recognizable at birth through their physical appearance. Dwarf calves possess a blocky appearance, shorter that normal cannon bones, bulging foreheads, prominent eyes, and protruding lower jaws. Some are born with contracted flexor tendons of the forelegs and are unable to stand naturally. Most dwarfs show evidence of incoordination and are characterized by a somewhat staggering gait. Dwarfism is most evident at three to four months of age as such calves do not grow normally and most develop distended paunches as a result of chronic bloat.

Dwarfs allowed to live usually die prior to two years of age. This generally due to bloat. For this reason this particular form of dwarfism is classified as a sub-lethal character (Gregory et. al. 1951).

Inheritance of Dwarfism in Conventional Herfords:

Johnson, et al. (1950) concluded that dwarfism in conventional Herefords is transmitted as a simple, monofactorial, autosomal recessive.

Cattle are of three kinds in regard to this type of dwarfism, normal non-carriers, normal carriers, and dwarfs. Inheritance is explained by using the symbols d to represent the dwarf gene and D to represent the gene for normal growth. An animal receives a unit of inheritance from each parent and then possesses two genes. Noncarriers are DD, carriers are Dd, and dwarfs are dd. Dd individuals cannot be distinguished from DD animals from their

appearance. The D gene is dominant or overshadows d when both are present in the same animal (Smith et al. 1954).

Smith et al. (1954) compiled a summarization of the six possible mating types for a pair of genes such as D and d.

| Mating types of parents | Offspring | |
|---|---|---|
| | Genetic Composition* | Appearance* |
| 1. DD x DD | All DD | All Normal |
| 2. DD x Dd | $\frac{1}{2}$ DD and $\frac{1}{2}$ Dd | All Normal |
| 3. Dd x Dd | $\frac{1}{4}$ DD; $\frac{1}{2}$ Dd; $\frac{1}{4}$ dd | $\frac{1}{4}$ Dwarfs |
| 4. DD x dd | All Dd | All Normal |
| 5. Dd x dd | $\frac{1}{2}$ Dd and $\frac{1}{2}$ dd | $\frac{1}{2}$ Dwarfs |
| 6. dd x dd | All dd | All Dwarfs |

* All ratios represent average expected values. Only the first three mating types occur under natural breeding conditions because the last three involve dwarfs and are not likely to occur in nonexperimental herds.

Carrier Identification:

The control of dwarfism which occurs in conventional cattle is dependent upon the identification and disposal of carrier animals in breeding herds. The three general methods which have been studied for the accomplishment of this are as follows: (1) breeding tests; (2) pedigree penalties; and (3) phenotypic differentiation of carriers and non-carriers, providing there exists a measurable expression of the dwarf gene in the carrier (Lush et al. 1952).

Breeding Tests:

Smith et al. (1954) concluded that breeding tests have proven reliable in identification of carrier animals. These tests are expensive because test herds must be maintained and delays the breeding program.

Carriers are identified when a cow produces or a bull sires a dwarf calf. Each cow used in the test must have produced at least one dwarf calf to be considered a known carrier.

Because of the chance factor a bull may be a carrier even though he is stated to be a non-carrier as the result of a breeding test. The following table shows the percentage of carrier bulls which would be expected by chance to not sire dwarf calves when mated to carrier cows for various numbers of progeny in test mating (Lush et al. 1952).

| Number of calves from known carrier cows | Percentage of carrier bulls which would sire no dwarfs |
|---|---|
| 1 | 75 |
| 2 | 56 |
| 4 | 32 |
| 8 | 10 |
| 10 | 6 |
| 12 | 3 |
| 14 | 2 |
| 16 | 1 |

The reliability of a breeding test increases with the number of calves produced. Prospective herd sires must sire 15 or more calves all of which are normal, from carrier cows before they may be classified as non-carrier bulls. There is about one chance in 100 of mis-classifying a carrier bull.

Pedigree Penalties:

Claims have been made that some blood lines and herds are free from the dwarf gene. The reliability of the selection of sires on this basis is questionable. The dwarf gene may be present in a bloodline or herd but at such a low frequency that dwarf calves have not been produced. Pedigree errors may cause the dwarf gene to be introduced.

The probability of a normal animal being a carrier on the basis of its relationship to a dwarf is as follows:

| Relationship to a dwarf | Probability of being a carrier |
|---|---|
| 1. Parent | 100% |
| 2. Full-sib | 67% |
| 3. Half-sib | 50%+ |
| 4. Son or daughter of half-sib | 25%+ |

Compiled by Smith et al. (1954).

Pedigree analysis are likely to be inaccurate, however they should be used on a probability basis to screen breeding herd replacements. The analysis is extremely helpful in screening prospective sires before placing them on breeding tests.

Phenotypic Identification of Carrier and Non-carrier Conventioal Herefords: Blood test identification of carriers has been researched by Dr. L. C. Ferguson of the Ohio Experiment Station. No correlation has been determined at the present.

Gregory et al. (1953) concluded that dwarfism in conventional Herefords is a cretin type accompanied by the primary characteristics of the cretin syndrome. This involved a marked bulging of the frontal bones, and have postulated that there is likewise a less pronounced but measurable similar expression of the dwarf gene in the carrier animal.

An experimental herd for the study of dwarfism in conventional herefords was established at the Iowa Experiment Station in 1952. X-rays were taken on several different parts of the body of all newly born calves produced. The lumbar vertebrae of the dwarf calves showed a distinct abnormal condition, appearing as if strongly compressed endwise. A lesser degree of this same condition was observed in some of the normal calves, while others showed no visible abnormal bone structure. The normal calves possessing the moderately compressed abnormal vertebrae are suspected as carriers whereas those with normal lumbar vertebrae are thought to be non-carriers. The classifications of calves as carriers and non-carriers appear to fit expected genetic ratios.

Dwarfism in Comprest Herefords:

The comprest character is transmitted as a single, incompletely dominant gene. The carriers of this gene are the comprest individuals in

the Hereford breed. Carroll et al. (1951) reported that comprest Herefords can be recognized by their shorter stature and have been commonly referred to as small type Herefords.

The inheritance of the comprest character and its assosiated kind of dwarfism may be illustrated with the following symbols and genetic compositions (Stonaker et al. 1958).

C = gene for the comprest character

c = gene for conventional type

cc = conventional type individual

Cc = comprest individual

CC = comprest dwarf

This kind of dwarfism may be effectively controlled by the avoidance of double comprest matings. Conventional types should not possess or transmit the comprest gene. Control should not present a problem as comprest animals can be differentiated from conventionals quite accurately.

Duck-Legged Herefords:

Lush (1930) reported a short-legged type of Hereford observed in Texas. Mature duck-legged type of cattle seemed to differ from normals only in that their bodies were about four to six inches nearer to the ground. No external symptoms of hypothyroidism were observed to be associated with the duck-legged character. Endocrine gland studies of one duck-legged calf revealed no gross abnormalities although its pituitary was very small.

Smith et al. (1954) concluded that the duck-legged character appears to be transmitted as a single, autosomal, dominant gene.

It should be pointed out that it is doubtful that the duck-legged character has ever occurred in purebred Herefords.

Dwarfism in Angus Cattle:

Scientific reports on dwarfism in Angus cattle are limited. The most troublesome kind is generally believed to be the same as that which occurs in conventioal Herefords. Baker et al. (1951) reported that dwarf Angus calves possess a blocky appearance, shorter than normal cannon bones, bulging foreheads, prominent eyes and protruding jaws. Reports indicate that these dwarfs are considerably more active than Hereford dwarfs which are quite sluggish.

Three Angus dwarfs of the bloater type have been x-rayed at the Kansas Agricultural Experiment Station. They appeared to possess the same abnormality of the lumbar vertebrae as dwarfs produced by conventional Herefords.

Breeding tests for the identification of carriers is the only reliable method by which this dwarfism may be controlled. This control technique has been discussed in detail previously.

Baker et al. (1951) reported dwarfism in Angus cattle which is different from the one previously discussed. This different kind is not always recognized at the time of birth. However, they do exhibit exceptionally compact, low-set, thick bodies and possess short, wide heads at an early age. Retarded growth is evident at a few months of age. At this time the dwarfs are usually in poorer condition than normal calves. As the calves grow older their heads appear longer and narrower. Bloat is not reported to be a common characteristic.

This type of dwarfism in Angus cattle is transmitted as a single autosomal, recessive gene.

Dwarfism in Shorthorn Cattle:

There are two kinds of dwarfism which occurs in Shorthorns of which neither appears to be the same as those which have been previously discussed in other breeds.

Breeders report that a specific kind of dwarfism is associated with the compact character in the Shorthorn breed. Stonaker and Tom (1944) described compact individuals as recognizable at birth as they appear shorter in head, neck, body and legs. Some have a tendency to be heavy in the shoulders and are crooked legged.

Double compact matings result in the production of conventional, compact, and dwarf progeny in an expected ratio of 1:2:1. Punnett (1936) concluded that the dwarf of this double compact mating produces the monstrous "bull-dog" calf. Surrarrer (1943) observed the same results. Bull-dog calves are generally born prematurely and are nonviable. The body is greatly distorted and there is a pronounced shortening of the long bones. This character is transmitted as a single, incompletely dominant gene. The carriers of the gene possess short legs which is characteristic of the Dexter type.

Compact Shorthorns appear to be similar to the Dexter cattle. On this assumption, the inheritance of the compact and bull-dog character may be illustrated with the following symbols and genetic compositions.

Co = gene for the compact character

co = gene for conventional type

coco = conventional type individual

Coco = compact individual

CoCo = Non-viable bull-dog calf (dwarf)

Conventional type Shorthorns should not possess or transmit the compact gene. It appears that this kind of dwarfism may be controlled by the avoidance of double compact matings in the Shorthorn breed.

Brandt (1941) concluded that recessive achondroplasia is another bulldog type dwarf although these dwarfs are less extreme than the double compact matings. These dwarfs are usually carried full term and are born alive. The carriers of the recessive achondroplasia gene appear normal and are not recognizable by physical appearance.

The "stumpy" character in Shorthorn cattle is a non-lethal kind of dwarfism. These dwarfs are characterized by a curly hair coat at the time of birth and the tail switches smaller than normal. The legs are shorter than normal and is more apparent in the forelegs than the hind legs. The hoof-heads are enlarged and the feet often turn outwards. The body and head of the stumpy dwarf appears to be of normal length. Growth was retarded in stumpy calves and they were in poorer condition than normal calves.

Stumpy calves appear to be produced by a single autosomal recessive gene (Gregory et al. 1952).

There are wide anatomical and physiological differences occurring within a given species. There is an eventual limit of deviation from the norm beyond which the organism cannot survive. Death of the organism may occur at any stage of development, immediately following fertilization, during embryonic differentiation, at parturition, or postnatally. We speak of any cause of death as a lethal effect. Among the many causes of death are gene changes which are incompatible with development or survival. These genes are known as lethal genes. Some genes have a detrimental effect on the organism but under favorable environmental conditions is not lethal. These genes are called semilethals.

Bogart et al. (1959) projected that the following four points tend to indicate a hereditary defect.

1. If it had previously been reported as hereditary in the same breed of livestock.
2. If it occurred more frequently among near relatives than among animals not so closely related.
3. If it occurred where there had been inbreeding.
4. If it occurred more than one season when different rations had been fed.

Abnormalities may be due to nutritional deficiencies and the following points indicatesuch (Bogart et al. 1959).

1. If it occurred when the ration of the mother was known to be deficient.
2. If it disappeared when an improved ration was fed.
3. If it had previously been reliably reported to be due to a nutritional deficiency.

Further, lethals may be divided into three groups (Bogart et al. 1959).

1. True lethals, those which are expressed before or shortly after birth.
2. Delayed lethals, those which are expressed later in life.
3. Partial lethals, those in which the animal inherits the character which becomes lethal only under certain conditions.

Lethal Traits in the Bovine:

Hutt (1964) described some Monogenic Lethal Characters of Domestic Cattle, All Simple Autosomal Recessives, Unless Marked D (dominant) or S (sex-linked).

(D) Achondroplasia, dominant

Achondroplasia, recessive

Epithelial defects

Hairless

Acroteriasis (amputations)

Paralysis of hind legs

Muscle contractures

Short spine

Congenital dropsy

Short lower jaw

Cerebellar hypoplasia

Missing phalanges

Hydrocephaly, internal

Congenital spasms

Prolonged gestation

Adenohypophyseal aplasia

(DS) Streaked Hairlessness

Ichthyosis congenita

Keratogeneses imperfecta

Hypotrichosis and anadontia

(D) Fused nostrils, malformed skull

General ankylosis

Atresia ilei

Porphyria (semi-lethal)

Achondroplasia, dominant, is due to an incompletely dominant gene that is lethal to the homozygote. Extreme bull-dog calves are aborted before the eighth month. The legs are very short. In some cases the hooves seem to project from the body. The cranium is vaulted, the head shortened and the tongue protrudes (Hutt 1964).

Achondroplasia, recessive resembles the dominant, but is less extreme. The calves are usually born alive at full term, but are unable to stand, and die within a few days (Hutt 1964).

Epithelial defects are the result of a recessive mutation (Rice et al. 1957). There are areas on the body competely devoid of skin. The homozygous recessive animals die at an early age.

Hairless calves are generally confined to the dairy breeds. Surrarrer (1943) reported that the hairless trait is lethal.

Acroteriasis is the genetic amputation of limbs in cattle. The limbs are terminated near the elbow and hock joints and the skin is neatly patched over the rounded stump. Associated defects include reduction of the jaw, cleft palate, and absence of most teeth (Hutt 1964).

Paralysis of hind legs is due to an autosomal, recessive lethal gene. Calves are born at full term and are normal except for inability to stand because of paralysis of hind legs. Efforts to save calves is useless because all affected calves must be eventually destroyed.

Rice et al. (1957) reported that muscle contracture is a simple autosomal recessive in cattle. This defect may cause death to the dam as the fore limbs are often bent with rigid joints.

Short spine calves have normal limbs. Before this condition was shown to be hereditary, many breeders thought such calves were sired by elks (Rice et al. 1957).

Cerebellar hypoplasia is a simple autosomal recessive trait. Affected calves are born alive but cannot stand, and lie with their limbs rigidly extended. The cerebellum is greatly reduced in size. Stormont (1958) reported that none have survived.

Missing phalanges is an absence of the first two phalanges of the lower limb. The affected calves have hooves but cannot stand. This is an example of a lethal gene with rather localized effect (Rice et al. 1957; Hutt 1964).

Hydrocephaly internal, is another recessive, autosomal lethal. In hydrocephalic calves fluid accumulates in the ventricles of the brain. Affected calves are usually born alive at full term. They display an enlargement of the cranium, are unable to stand, and usually die within two days (Hutt 1964).

Stormont (1958) reported that congenital spasms result in the incoordination of the legs. Spasmodic movement of the head is characteristic and survival of those affected is nil. The disorder is a simple autosomal recessive.

Burris et al. (1952) indicated that prolonged gestation is known as a simple recessive, autosomal trait. This trait causes gigantism of the calf and may cause death to both the calf and dam. Lasley et al. (1961) reported the same observations.

Adenohypophyseal aplasia is a type of hereditary prolongation of gestation. In this type the fetuses display varied abnormalities (Lasley et al. 1961).

Streaked hairlessness is a sex-linked dominant lethal (Hutt 1964). The hair is lacking on vertical streaks over the hip joints and in some cases extending up the sides. This genetic defect is a sex-linked, dominant,

lethal in males. The deficiency of male offspring from streaked cows has led to the conclusion that streaked hairlessness is caused by a gene that is sex-linked, incompletely dominant, lethal to the hemizygote, and thus sex-limited.

Ichthyosis congenita is a condition of the skin causing calves to be hairless and to appear covered with horny plates resembling big scales. This trait results from an excessive production of keratin and is due to a simple autosomal recessive. Keratogenesis imperfecta is similar to Ichthyosis and is also a simple autosomal recessive (Stormont 1958).

Hypotrichosis implies hairless. Surrarrer (1943) reported that one type in cattle is due to a recessive gene. A second type of hairlessness is thought to result from a recessive, sex-linked gene. The second type is completely hairless at birth, even without eyelashes. Anadontia refers to an absence of teeth.

Anderson et al. (1957) reported that porphyria is an abnormality of metabolism which causes the red pigment, porphrin, to be excessively produced. The porphrin accumulates in the lbood, bones, and teeth. Urine turns reddish brown and teeth are pink. Affected animals are extremely sensitive to sunlight and exposure results in severe blistering. An animal may live a normal life if protected from the sun. This trait is transmitted in cattle as an autosomal recessive (Hutt 1964).

TABLE I

INHERITED LETHALS AND ABNORMALITIES IN CATTLE

This table was compiled by Bogart (1959).

| | ABNORMALITY | DESCRIPTION | MODE OF INHERITANCE |
|---|---|---|---|
| 1. | Achondroplasia | Bulldog. Extremes are aborted at 3 and 8 months gestation. Delivery may be very difficult. Lethal | Three kinds. One is incompletely dominant with the heterozygote smaller than normal and bulldog, a complete monstrosity as the lethal. One type is born alive. The other is simple recessive. |
| 2. | Acroteriosis | Also called amputated. Eyes protrude. Ears short, lower jaw short, forelegs terminated at elbow and rear legs at hock, with ends of all four blunt, covered with skin. Lethal. | Hereditary, but mode of inheritance unknown. |
| 3. | Agnathia | Short, lower jaw; lethal. | Simple recessive |
| 4. | Ankylosis | Legs rigidly bent due to bones of joints fusing. Some cases of jaw fusions noted. Lethal. | Probably recessive |
| 5. | Brachygnathia | Short lower jaw. | Probably recessive |
| 6. | Bulldog head | Short, broad skull, orbits large, upper jaw short, impaired vision. | Simple recessive |

| | | | |
|---|---|---|---|
| 7. | Bowed pasterns | Bowed outward | Probably recessive |
| 8. | Cancer eye | Occurs mostly in white-eyed hereford. Cancer develops in nictitating membrane. Usually occurs late in life. Results eventually in death. | Hereditary, but mode of inheritance not determined. Seldom occurs in animals with pigmented eyelids. |
| 9. | Cataract | Lens shows opaque body beneath cornea. | Simple recessive |
| 10. | Cerebellar hypoplasia | Poor coordination. Cerebellum redimentary, excessive fluid. Some walk like ballet dancers. | Probably simple recessive |
| 11. | Curly hair | Hair in tight curls, viable. | Simple dominant |
| 12. | Digital anomaly | One toe shorter, toes spread, animals lame. | Simple recessive |
| 13. | Double ears | Double ear is thin flap of cartilage parallel to main axis of ear and projecting out of back surface. | Simple dominant |
| 14. | Double muscle | Thighs deep, full, with deep groove. Homozygous is undesirable, but heterozygous has selective advantage. | Incomplete recessive |
| 15. | Dropsy | Degrees of dropsy expressed. Severe cases may cause loss of dam. | Simple recessive |

| | | | |
|---|---|---|---|
| 16. | Duck-legged | Cattle very short-legged. | Dominant |
| 17. | Dwarfism | Described in most major breed of beef cattle. Several distinct kinds. Some appear normal except for being small. Some are cretins, short heads, wide through shoulders, breathing hard, etc. Cannot always be identified at birth. Some are long-headed. | Recessive |
| 18. | Edema | Watery enlargement of legs, muzzle, and belly. | Hereditary, but mode not determined |
| 19. | Epileptic fits | Lowering of head, tongue chewing, foaming at the mouth, collapse into coma. | Simple dominant |
| 20. | Epithelial defects | Defective formation of skin below knees, one or more claws underdeveloped, deformed integument of muzzle and the mucous membranes of nostrils, tongue, palate, and cheek. Lethal. | Simple recessive |
| 21. | Female sterility | Sex-limited with only females showing it. Probably zygote abortion in late cleavage or early blastocyst. | Simple, sex-limited recessive |

| | | | |
|---|---|---|---|
| 22. | Flexed pasterns | Usually with front legs. Toes may turn under in severe cases. Metatarsal inclination and bowed pasterns. | Simple recessive; hereditary, but mode not determined |
| 23. | Fused teats | Faulty placement of fore and rear teats, fused in severe cases. | Simple recessive |
| 24. | Hairlessness | Born hairless or semihairless. Skin thick and shows cracks. Appears to be two kinds: one lethal, while the other lives but grows slowly and never has normal hair. | Simple recessive |
| 25. | Hernia | Umbilical rupture. Appears in males 8 to 20 days of age. Disappears at 10 weeks to 7 months. | One or more pairs of recessive genes; sex-limited |
| 26. | Hydrocephalus | Water on the brain. Malgrowth of diencephalon with constriction of third ventricle to narrow channel. Hydrocephalus restricted to the two cavities of the olfactory bulbs. Lethal | Not known; probably recessive |
| 27. | Hypoplasia | Epithelial layers of testes, tubules, and follicles underdeveloped. Sterile if bilateral. | Simple recessive |

| | | | |
|---|---|---|---|
| 28. | Impacted molars | Molars misplaced and deranged. Terrific stress on jaw which might cause break in bone. Lethal | Simple recessive |
| 29. | Imperforati anus | No exterior opening of the anus. | Recessive |
| 30. | Lameness | Lameness in calves | Not determined |
| 31. | Missing teat | Only one teat on left side. | Recessive |
| 32. | Mummified fetus | Calves die at eighth month gestation but carried to term. | Apparently simple recessive |
| 33. | Muscle contracture | Calf born at full term with contracture of neck and limbs. May lead to death of cow at delivery. Lethal | Simple recessive |
| 34. | Night blindness | Animals do not see well at night but have good vision during day. | Not determined |
| 35. | Paralysis | Posterior paralysis. May have muscular tremors and blindness. Dies shortly after birth. Lethal | Simple recessive |

| | | | |
|---|---|---|---|
| 36. | Polydactylism | Extra toes. Defect of front feet. Affected animals go lame. | Probably recessive |
| 37. | Prolonged pregnancy | Pregnancies up to 403 days. Calves born dead. | Simple recessive |
| 38. | Screw tail | Distinct kinks in lower part of tail. | Simple recessive |
| 39. | Short spine | Entire vertebral column is short but legs are normal length. | Recessive |
| 40. | Skull defect | Frontal opening in skull. Brain tissue protrudes. | Not determined |
| 41. | Spasms | Animals appear normal at birth but in a short time go into spasms and die. Lethal. | Simple recessive |
| 42. | Sterility | (a) Malformation and underdevelopment of sex organs.<br>(b) Endocrine imbalance resulting in cystic overies, and aspermia.<br>(c) Adolescent infertility<br>(d) Ovarian hypoplasia<br>(e) Absence of gonads.<br>(f) Incompletely developed.<br>(g) Testes hypoplasis.<br>(h) Sigmoid flexure will not straighten. | Not determined; most of these are recessive |

| | | | |
|---|---|---|---|
| 43. | Strabismus | Cross eyes. Not evident at birth but identified by 12 months of age. | Simple recessive |
| 44. | Streaked-hairless | Streaks of hairlessness in perpendicular rows in cows. These cows produced a ratio of 2 heifers: 1 bull calves. | Incomplete dominant; sex-linked |
| 45. | Syndactylism | One toe on each front foot instead of two. | Simple recessive |
| 46. | Tendon contracture | Tendons pulled rigidly. Calves either born dead or die shortly after birth. | Simple recessive |
| 47. | White heifer disease | Presistent hymen or incomplete cervix. Horns of uterus become distended with fluid. Associated with white Shorthorn. | Probably recessive |
| 48. | Wry tail | Tail twisted to one side at tail head. Direction not genetic. | Simple recessive |

## SUMMARY

There are four major hereditary patterns by which mutant genes become evident over two or more generations. These four patterns involve certain definite ratios and their modifications form the basis of all accurate diagnoses.

The dominant pattern is identified by the mating of two normal parents resulting in a mutant gene from one or the other parent being passed on to the offspring. The resulting offspring must be abnormal to classify under the dominant pattern of mutation.

Another characteristic of the dominant pattern is that in large series of mating, abnormals are produced generation after generation, without skipping a generation. Abnormals occur in a 1:1 ratio.

Disorders resulting from a dominant mutation are easy to control because elimination of abnormal animals eliminates all abnormal genes.

The incompletely dominant pattern differs from the dominant pattern. Among the abnormals are the ones that are much more abnormal than the others, so the actual diagnostic ratio for this pattern is four slightly abnormal and one highly abnormal for every three normal produced.

Disorders resulting from an incompletely dominant mutation is relatively easy to control because eliminating all abnormal animals eliminates all mutant genes.

Most of the genetic defects in cattle are caused by a recessive mutation. This is one of the most difficult hereditary patterns to control.

The characteristics of this recessive pattern are that the abnormals typically come from normal parents. They usually occur in relatively small numbers. The trait skips generations even when families are large.

Recessives like hydrocephalus are difficult to control because the mutant gene is carried by normal appearing animals. It is only when an animal receives two mutant genes, one from each parent, that the abnormal is produced. Although eliminating such animals eliminates two abnormal genes, it is difficult to identify the normal appearing recessive gene carriers.

The overdominant hereditary pattern is another difficult to control mutation. It is typified by the snorter dwarf condition in beef cattle.

Starting with two normal parents, a mutant gene is passed to the offspring. In this pattern, the resulting offspring is not only normal, but is superior in appearance.

As selection favors the superior animals, they are perpetuated. When a super bull is mated to progeny of another super bull, abnormal calves occur in the ratio of seven normals to one abnormal.

Identification of overdominance can be made on the basis of superiority of some of the normal animals. An overdominant mutation is difficult to control because selection favors survival of the superior animals, those carrying a mutant gene.

There are three general methods which have been studied for carrier identification.

Breeding tests have proven reliable in identification of carrier animals. These tests are expensive because test herds must be maintained and delays the breeding program.

Prospective herd sires must sire 15 or more calves all of which are normal, from carrier cows before they may be classified an non-carrier bulls. There is about one chance in 100 of mis-classifying a carrier bull.

The reliability of the selection of sires on the basis of pedigree penalties is questionable. The mutant gene may be present in a herd at such a low frequency that the undesirable trait has not yet expressed itself. Pedigree errors must also be considered.

The pedigree analysis is extremely helpful in screening prospective sires before placing them on breeding tests.

Phenotypic identification of carrier animals is relatively easy in some cases, but in general carrier animals cannot be identified by simply observing the animal. X-rays of the lumbar vertebrae of dwarf calves showed a distinct compressed condition. A lesser degree of this same condition was observed in some normal calves, while others showed no visible abnormal bone structure. The normal calves possessing the moderately compressed abnormal vertebrae are suspected as carriers. The classifications of calves as carriers and non-carriers appear to fit expected genetic ratios.

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## LITERATURE CITED

Anderson, D.E., J.L. Lush and D. Chambers. 1957. Studies on bovine ocular squamous carcinoma (cancer eye).
J. of Animal Science 16:739.

Baker, M.L., C.T. Blunn, and M. Plum. 1951. "Dwarfism" in Aberdeen-Angus Cattle.
J. of Herdity. 42:141 - 143.

Bogart, R. 1959. Inheritance of lethals and Abnormalities in Farm Animals. Improvement of Livestock. pp. 77 - 113.

Brandt, G.W. 1941. Achondroplasia in Calves.
J. of Heredity. 32:183 - 186.

Burris, M.J. and C.T. Blunn. 1952. Some factors affecting gestation length and birth weight of beef cattle.
J. of Animal Science. 11:34 - 41.

Carroll, F.D., P.W. Gregory, and W.C. Rollins. 1951. Thyrotropic-hormone deficiency in homozygous dwarf beef cattle.
J. of Animal Science. 51:435 - 440.

Gregory, P.W., W.C. Rollins, and F.D. Carroll. 1952. Heterozygous expression of the dwarf gene in beef cattle.
Southwestern Vet. 5:345 - 349.

Gregory, P. W., C.B. Roubicek, F.D. Carroll, P.O. Stratton, and N.W. Hilston. 1953. Inheritance of Bovine Dwarfism and the Detection of Heterozygotes.
Hilgardia. 22:407 - 450.

Gregory, P.W., W.C. Rollins, P.S. Patengale and F.D. Carroll. 1951. Phenotypic Expression of Homozygous Dwarfism in Beef Cattle.
J. of Animal Science. 10:922 -933.

Gruneberg, H. and K. Huston. 1968. The Development of Bovine Syndactylism.
J. of Embryol and Exptl. Morphol. 19:251 - 259.

Huston, Keith. 1967. Problems in Diagnosis of Genetic Diseases and Defects.
North Central Regional Dairy Cattle Breeding Project. 1967:Winter.

Huston, K., F.E. Eldridge, and J.W. Mudge. 1961. Genetics of Syndactylism in Cattle.
J. of Dairy Science. 44:1197.

Hutt, F.B. 1964. Dwarf Calves.
Animal Genetics. pp 50 - 53 and 167 - 195.

Johnson, L.E., G.S. Harshfield, and W. McCone. 1950. Dwarfism, An Hereditary Defect in Beef Cattle.
J. of Heredity. 41:177 - 181.

Lasley, J.F., B.N. Day and J.E. Comfort. 1961. Some genetic aspects of gestation length and birth and weaning weights in Hereford Cattle.
J. of Animal Science. 20:737 - 741.

Leipold, H.W., W.F. Cates, O.M. Radostits and W.E. Howell. 1969. Spinal Dysraphism. Arthrogryposis and Cleft Palate in Newborn Charolais Calves.
Can. Vet. Jour., Vol. lo, no. 10, October, 1969. 268 - 274.

Leipold, H.W., K. Huston, M.M. Guffy, S.M. Dennis. 1968. Syndactyly in an Aberdeen Angus Calf.
North Central Regional Dairy Cattle Breeding Project NC-2. (1968):1685 - 1687.

Lush, J.L. 1930. "Duck-Legged" Cattle on Texas Ranches.
J. of Heredity. 21:83 - 90.

Lush, J.L. and L.N. Hazel. 1952. Inheritance of Dwarfism.
The American Hereford Journal. March 1, 1952:32 - 34.

Punnett, R.C. 1936. The Experiments of T.H. Riches Concerning the Production of Monsters in Cattle.
J. of Genetics. Vol. XXXII:65 - 72.

Rice, V.A., F.N. Andrews, E.J. Warwick, J.E. Legates. 1957. Lethal Effects of Genes.
Breeding and Improvement of Farm Animals. pp 285 - 296.

Smith, W.H. and L.A. Holland. 1954. Dwarfism in Beef Cattle.
Dwarfism in Beef Cattle. pp 1 - 24 (unpublished).

Stonaker, H.H. and R.C. Tom. 1944. "Compact" Shorthorns.
J. of Heredity. 35:247 - 250.

Stonaker, H.H. 1958. Breeding for beef. A summary of results of experimental beef cattle breeding at the Colorado Agr. Exp. Sta.
State Univ. Ext. Bull. 501 - S.

Stormont, C. 1958. Genetics and disease.
Advances in Veterinary Science, Vo. 4. Academic Press, New York. pp 137 - 162.

Surrarrer, T.C. 1943. Bulldog and Hairless Calves.
J. of Heredity. 34:175 - 178.

# GENETIC ABNORMALITIES IN BEEF CATTLE

by

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AN ABSTRACT OF A MASTER'S REPORT

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The purpose of this study was to assemble as much research data as possible with the hope that beef producers would benefit from its contents. Breeders in the past have felt that genetic defects should be "covered up" and that in time the problem would disappear.

Genetic disorders originate in the gene carrying chromosomes of the cell nucleus. Each species has a characteristic number of chromosomes, the bovine having 30 pairs and each chromosome is assumed to carry at least 500 genes.

Genes may rarely change to other kinds of genes. This is known as mutation. Frequency of gene mutation is not known for livestock.

When an animal is born with a mutant gene, there is some chance that the animal will reproduce and perpetuate the new gene. New mutants may survive for many generations. New mutants are added to those surviving from previous generations and a gradual accumulation may take place in populations.

There are four major hereditary patterns by which mutant genes become evident.

1. The dominant mutation pattern gives the highest frequency of abnormal animals, 50 per cent of the animals in the first generation after mutation. Most traits due to mutation occur first at frequencies of less than 25 per cent and in most cases less than $12\frac{1}{2}$ per cent of the animals. Abnormal traits that occur more often than in one of every eight animals usually should not be diagnosed as hereditary "disease."

2. The pattern of the incompletely dominant mutation is like the dominant mutation with one characteristic difference. The occurrence of two degrees of abnormalities is the means of distinguishing the dominant

pattern from the incompletely dominant. Incompletely dominant abnormalities occur at a diagnostic ratio of four slightly abnormal and one highly abnormal for every three normal individuals produced. As in the case of the dominant mutation, the disease resulting from an incompletely dominant mutation is relatively easily controlled because eliminating the abnormal animals eliminates all the mutant genes.

3. Most of the genetic defects and diseases in cattle are caused by a recessive mutation. The recessive pattern is one of the most difficult hereditary patterns to control. The characteristics of the recessive pattern are that the abnormals typically come from normal parents and they usually occur in relatively small numbers. The trait skips generations even when families are large. The recessive pattern fits the hydrocephalus gene that occurs in range cattle. Recessive hereditary diseases such as hydrocephalus are difficult to control because the mutant gene is carried by normal appearing animals. Only when an animal receives two mutant genes, one from each parent, is the abnormal produced. Although eliminating such animals eliminates two abnormal genes, it is difficult to identify the normal-appearing recessive gene carriers. They perpetuate the mutation.

4. The overdominant heredity pattern is another important but difficult to control mutation. It is typified by the "bull-dog" dwarf condition in beef cattle. Defects caused by an overdominant mutation usually are evidenced rather quickly since superior animals are selected in preference to normal animals. The "super" animals are often mated together. As the result of super matings, abnormal calves occur in the ratio of three normals to one abnormal. Identification of overdominance

can be made on the basis of superiority of some of the normal animals. An overdominant mutation may be difficult to control because selection favors survival of the superior animals, those carrying a mutant gene.

It is important that cattle breeders diagnose hereditary defects at which time they occur in breeding herds. Also, cattle breeders should possess correct knowledge of the type of inheritance involved for the abnormality in question to be able to select against undesirable gene. Breeders will not eliminate all or perhaps any undesirable gene, but they can prevent nearly all traits from causing major loss to the industry.