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Congenital and Hereditable Defects Which Interfere With The Reproductive Efficiency Of Domestic Cattle (Bos tarus)

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Congenital and Hereditable Defects Which Interfere With The Reproductive Efficiency Of Domestic Cattle *(Bos tarus)*

by

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INTRODUCTION

The terminology concerning defects or abnormalities in cattle can render confusion if there is not a distinction between the adjectives congenital and hereditable. The word congenital is derived from the Latin words *con* and *genitalis*. *Con* means with or together and *genitalis* means to beget or reproduction. Thus the word congenital describes those conditions which are present at birth as a result of the developmental process. Heredity is dervied from the Latin word *hereditas* or heirship. Thus hereditable indicates those conditions in the young which are present as a result of parental genotypes. Most, but not all inherited developmental defects are apparent at birth and therefore can be said to be congenital. *However, not all congenital defects can be accurately termed hereditable.*

The following discussion of congenital and hereditable defects in cattle is confined to those which interfere with the propagation of the species *Bos taurus*. There are three listings; hereditable defects, congenital defects, and defects of questionable hereditable nature. Those defects which have some evidence, though not definite, of being hereditable are included in the listing, questionable hereditable characteristics. The mode of expression in many hereditable conditions is not definitely known. Incomplete penetrance and environmental-genetic interaction preclude the definite categorization of many defects as being solely hereditable.

It is hoped that by bringing attention to the developmental conditions which interefere with reproduction that the veterinarian and livestock owner may be more fully informed of an important aspect of livestock disease.

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HEREDITABLE CONDITIONS

Achrondroplasia: (17, 20, 25, 36, 39, 52, 61, 67)	"Bulldog" "Dwarf" Three types: brachycephalic, dolichocephalic & compressed; Simple autosomal recessive; Short legs, short, broad concave face, chondrodystrophy; All beef breeds, most dairy breeds; May be combined with albinism in Herefords; Usually lethal; May be hydrocephalic.
Missing phalanges: (16, 61)	"Creeper calves" Absence of first two phalanges.
Acroteriasis congenita: (13, 32, 61)	"Amputated limbs" Hemimelia or Amelia. Forelegs terminate at elbow and hind legs at hock joint; Holsteins and Swedish cattle; Lethal.
Epitheliogenesis imperfecta neonatorum: (3, 61, 67)	"Skinless' Simple autosomal recessive; Raw areas devoid of skin mostly on limbs; Most dairy breeds; Usually lethal.
Hypotrichosis congenita: (7, 45, 61, 67)	"Hairless" Simple autosomal recessive. Types: 1. lethal hairless. 2. semi-hairlessness. 3. hypotrichosis with anadontia. 4. viable hypotrichosis. 5. hypotrichosis with missing incisors. 6. streaked hairlessness, may be dominant. Most dairy breeds.
Impacted molars: (61, 67)	"Parrot mouth" Milking Shorthorns. Lethal.
Achondroplastic micromelia: (67)	Russian cattle; Imperfections of lower limbs and shortened legs; Lethal.
Atresia ani: (61)	Imperforate anus; Lethal; Holsteins, Angus, Guernseys, Shorthorns, Ayrshires.
Atresia ilei: (57)	Norwegian cattle; Lethal.
Brachygnathia superior: (16, 35, 67)	Short maxilla; Simple autosomal recessive; Jerseys; May be lethal.
Brachygnathia inferior: (16, 67)	Short mandible; Simple autosomal recessive; Shorthorns; Jerseys, Holstein, Ayrshire.
Agnathia: (67)	Absence of jaw; Lethal.
Sex linked lethal: (67)	Disproportionate sex ratios; Male or female lethal; Holsteins.

Abnormal skull: (61, 67)	Types: 1. Failure of fusion of frontal and parietal bones. Cerebral hernia in Holsteins. 2. Nasal openings fused. Lethal.
Hydrocephalus: (4, 9, 14, 20, 55, 61, 67, 68)	Simple autosomal recessive. Types: 1. Malformation of cranium—dome shaped skull. 2. Blockage of ventricular drainage. 3. Internal hydrocephalus, normal cranium. 4. Heredoencephalo-opthalmomyopathy. Most beef and dairy breeds. Usually lethal.
Mummification: (3, 23, 61, 62, 67)	Haematic or papyraceous mummification; Separation of maternal and fetal placenta with subsequent death and partial resorption of the fetus; Red Danish, Guernsey, Jersey, Holstein.
Paralyzed hindquarters: (67)	Red Danish cattle.
Short spine: (67)	Fusion of ribs and vertebra; Lethal; Norwegian cattle.
Ljutikow's lethal: (67)	Stillborn calves with no recorded abnormalities.
Fetal anasarca: (3, 16, 61, 67)	Subcutaneous edema and anasarca; Ayrshires.
Epilepsy: (16)	Brown Swiss; Attacks in calves disappear when mature.
Cryptorchidism: (49, 71)	Usually only left testicle.
Multiple eye defects: (16, 61, 63, 67)	Opaque lens, narrow iris, displaced lens in Jerseys; Cataracts and opacity of lens—Holsteins—Brown Swiss; Micro-opthalmia and blindness—Shorthorns (esp. white); Strabismus and exopthalmos—Jerseys and Shorthorns.
Prolonged gestation: (16, 54, 61, 67)	Types: 1. 310–350 days Holsteins and Ayrshire, autosomal recessive; Usually lethal. 2. Adenohypophysial aplasia. In Guernsey and Jerseys gross deformity of the head; Lethal.
Cerebellar defects: (16, 46, 61, 67)	Types: 1. Cerebellar hypoplasia. Herefords, Guernseys, Holsteins. 2. Cerebellar ataxia less severe than type 1. Jerseys, Shorthorns and Holsteins.

Spastic lethal: (34, 44)	Ataxia, incoordination and convulsions; No anatomical abnormality; Jerseys and Herefords.
Spastic paresis (Elso heel): (16, 69)	Spastic lameness of the hindlimbs. Holsteins and Angus.
Multiple ankylosis: Muscle contracture: Tendon contracture: (16, 29, 61, 67)	May be observed as separate syndromes or together; Limbs folded and head drawn back; Usually lethal; Holsteins, Shorthorns, Jerseys.
"Baldy" calves: (16)	Alopecia, loss of body condition, horns fail to grow, skin lesions; Holsteins.
Congenital porphyrinuria: (50, 61)	"Pink tooth" "Osteohaemochromatosis" Holsteins and Shorthorns, Herefords; Simple autosomal recessive.
Smooth tongue: (61)	Unable to nurse—sparse epithelium on tongue.
Sex limited genetic infertility: (37)	Jerseys and Holsteins; Jersey females have normal estral cycles and normal tracts yet are sterile; Holstein males have abnormal, infertile spermatozoa.
Segmental aplasia of the Müllerian Duct System: (27, 31, 32, 33a, 61)	"White Heifer Disease" Autosomal recessive sex-linked; Associated with white coat color in Shorthorns; Shorthorns, Angus, Holsteins, Jerseys, Guernseys, and Ayrshires. Types: 1. Hymenal constriction; absence of either the cranial part of the vagina, cervix, or the uterine body. 2. *Uterus unicornis.* 3. Imperforate hymen; infantile, congenital, small vulva.
Ichthyosis congenita: (47, 61)	Excessive keratinization of skin and loss of hair. Holsteins, Brown Swiss and Red Poll.
Thyroid dysfunction: (67)	Telemark cattle, shortened head and jaw; Lethal.
Ovarian hypoplasia: (49)	Recessive autosomal; Swedish Highlanders, Shorthorns.
Gonadless: (49, 61)	No ovaries, juvenile tracts; Autosomal dominant.
Testicular hypoplasia: (49)	Recessive autosomal with incomplete penetrance; Swedish Highland cattle and most beef breeds; Usually unilateral and involves left side.
Seminal defect: (41)	Only 5% intact spermatozoa; Jersey and Holsteins.
Genetic load or genetic incompatibility: (12, 22, 42, 51)	Expressed in early embryonic death; Inbreeding of zygote and dam; Maternal—fetal incompatibility.

CONDITIONS WITH QUESTIONABLE HEREDITABLE NATURE

Abnormal Wölffian or Gärtner's Ducts: (61)	Remnants of ducts become cystic.
Failure of fusion of Müllerian ducts: (61, 64, 65)	Swedish Highlanders, Shorthorns Guernseys, Herefords, Holsteins, Brown Swiss. Types: 1. Persistent septum in the external os. 2. True double external os of the cervix. 3. Complete or true double cervix. 4. Incomplete or false double cervix. 5. Vaginal septum. 6. *Uterus didelyphys.*
Hermaphrodites or Intersex: (61)	
Hydranencephaly and Arthrogryposis: (15)	Permanent joint contracture and fluid replacement of missing cerebral cortical tissue.
Aplasia segmentalis ductus Wölffii: (61)	Congenital absence of the epididymis, ductus deferens or seminal vesicles; Red Danish, Guernseys, Holsteins.
Freemartin: (32, 61)	Sterile heifer born twin to a bull; 90% of bovine females born twin to a bull are sterile; Result of fusion of the placentae.
Cardiac anomaly: (8, 20, 44a, 61)	*Ectopia cordis,* subaortic septal defect, ventricular septal defect.
Persistent penile frenulum: (18, 19)	Shorthorns, Herefords, Angus.
Digestive tract anomalies: (48, 56, 58, 66)	Types: 1. Duo-cecum large colon terminates in a rounded blind end. 2. Rumenal defects.
Impotentia coeundi: (3, 49)	Inability to protrude penis or short penis; Guernsey, Holstein, Jersey.
Curvature or deviation of penis: (18, 28)	Angus, Hereford.
Preputial prolapse: (3, 18)	Angus, Polled Hereford.
Hypospadia: (18)	Urethral fistula.
Progressive posterior paralysis: (6, 61)	"Crampy" "Spastic Syndrome" Holsteins, Guernseys, Ayrshires; Intermittent spastic contractions of back and leg muscles in mature animals.

Umbilical, scrotal, inguinal hernia: (16, 61)	Holstein, Hereford.
Harelip and cleft palate: (70)	Shorthorns.
Multiple lipomatosis: (1)	Large fat deposits in perineal region. Holsteins.
Hypomyelinogenes congenita: (16, 61, 73)	Ataxia, incoordination; Absence of myelin in cerebellum and brain stem. Jerseys, Shorthorns, Herefords, Angus-Shorthorns.
Spondylarthrosis: (5)	Sacral-lumbar vertebra. Young bulls.
Bleeding disease: (10)	Multiple coagulation defect. Holsteins.
Sacrococcygeal agenesis: (53)	Hereford-Holstein.
Adolescent infertility: (32)	Infertile period from first estrus until conception; Period varies with breed.
Neuronopathy and pseudolipidosis: (72)	Austrailian Aberdeen-Angus; Appears in growing calves; Incoordination and ataxia.

TERATOGENIC AGENTS

Pre-natal radiation: (26, 59)	Irradiation of 31–32 day fetus in utero causes abnormal limb development; Irradiation in 5th and 8th month results in reduced spermatogenesis in males.
Hypovitaminosis A: (55)	Hydrocephalus .
Lupine and lead: (11)	"Crooked calf syndrome" Front legs flexed, articular surfaces malaligned; *Lupinus sericeus* + lead fed during pregnancy.
"Acorn" calves: (16)	Unknown maternal deficiency; Abnormal osseous development of skull and skeleton. Usually die.

NON-INHERITED ABNORMALITIES

Congenital defects may be the result of teratogenic agents, embryonal accidents or genetic mutation. These abnormalities are not genetically transmitted from one generation to another although it may be argued that the individual was genetically susceptible to the accident. The large number of developmental abnormalities does not make it feasible to discuss each recorded congenital abnormality. For a more comprehensive discussion of these abnormalities and others the books by Roberts (61) and Arthur (3) should be consulted. Roberts (61) has classified congenital defects according to their developmental origin. Embryonal defects may be manifested in any of the categories.

1. defects due to excessive division
   i.e. polydactylia, polydontia

2. defects due to failure of fusion
   i.e. spina bifida
   schistosomus reflexus
   palatoshisis
3. defects due to arrest in division
   i.e. cyclopia
4. defects due to complete local failure of tissue growth
   i.e. ectromelia
   vertebral or costral abnormalities
   anopthalmia
5. defects due to arrest in assumption of final form or posture
   i.e. ectopia cordis
6. defects in the persistence and disappearance of contiguous structures that normally follow a certain pattern
   i.e. persistent urachus
7. defects due to fusion of sexual characteristics
   i.e. hermaphrodites
8. defects due to fusion of twin parts

Some of the more common congenital abnormalities are listed below.

1. *Schistosomus reflexus*
2. *Microcephalus*
3. *Cyclopia*
4. *Crania bifida*
5. *Spina bifida*
6. *Campylorrhachis scoliosa*
7. *Persosomus elumbus*
8. *Persosomus horridus*
9. *Amorphus globosus*
10. Conjoined twins
11. Duplication of parts
12. Crooked or wry calves

DISCUSSION AND CONCLUSIONS

A study of congenital and hereditable conditions should necessarily include a review of the clinical incidence of the defects. Herschler, *et al.* (44) has found 6.26% abnormal calves in a study of approximately 5,000 individuals. They found that of 312 abnormal dairy calves the most common abnormality was the stillborn calf (39.6%); general anomalies which included weak and small calves made up 28.2%; 25.9% were muscle, bone, joint and cartilage abnormalities; 2.3% were nerve or eye abnormalities and 4% were epithelial defects. There was no significant association of abnormalities with sex, number of services required per conception, or level of herd production. There was a significant association of abnormalities with sire, twinning, and prolonged gestation. No significant association of breed with number of abnormalities was found; however, there was a highly significant association of breed with the frequency of certain abnormalities.

The high incidence of abnormal calves is an interesting observation. If an infectious process were to claim 6% of the nations calf crop it would immediately be subjected to research and control attempted. However, it seems that most congenital or hereditable disorders are usually disregarded or are observed only with curiosity or passing interest.

Zemjanis *et al.* (74) has reported on the clinical incidence of genital abnormalities in the cow. In a total of 20,913 examinations the following was found:

Ovarian hypoplasia	1.9%
Freemartinism	.1%
White heifer disease	.04%
Duplex cervix (2 cases)	
Uterus unicornis (2 cases)	

Carroll *et al.* (18) has reported on examination of bulls for soundness. Defects found in 10,940 examinations were:

Penile deviation	190
Persistent penile frenulum	57
Hypospadia	19
Hypoplastic testes	146
Scrotal hernia	17
Small testes	814
Cryptorchid	14
Segmental aplasia or hypoplasia of the Wolffian ducts	20

Prenatal mortality is seldom diagnosed since it often occurs in early gestation and the interval between estrus cycles may be only slightly lengthened. However, early embryonic death is probably the most important single cause of reproductive failure. A number of investigators have estimated prenatal mortality as being from 39–59.4% in cows with a history of infertility and from 14.9% to 21.0% in

normal females. (42) Most of these early embryonal deaths occur between 16 and 34 days after breeding. Fosgate and Smith (as cited by Hanly) have reported a mean pregnancy loss of 6.38% in cows found pregnant at 34 to 50 days. The variation of the loss at each month after the first was not significant.

Our understanding of embryonal accidents and genetic failure lags behind our knowledge of many infectious agents. The influence of environment and other factors on the incidence of congenital abnormalities in the bovine is not well defined. In other species the use of immunizing agents on pregnant females such as hog cholera virus in swine and blue tongue virus in sheep have been shown to result in teratogenic effects. Certain plants may elicit teratogenic effects such as the congenital cyclopian deformity seen in lambs as a result of pregnant ewes grazing on *Veratrum californicum*. Experimental hypovitaminosis A in pregnant cattle has produced ocular defects and hydrocephlus in calves. (55) There are few other recorded instances of teratogenic agents in the bovine. It may be possible that some of the congenital deformities in the bovine are the result of yet unknown teratogenic agents.

Recently the theory of paternal influence on early embryonic mortality has been advanced. (12) It seems logical that lethal effects could be manifested in early embryonic death. Each union of an ovum and a sperm creates a genotype different in some way from any other. This process allows for biological selection of the fittest and aids in the process of evolution. It seems feasible that some of the genotypes are not viable and that the resultant lethal effect is a justifiable expense in the total evolutionary process.

The infrequent appearance of many congenital and hereditable defects makes it difficult for complete study of etiological factors. Gilmore (32a) has indicated the following general guidelines for diagnosis and evaluation of such conditions. Hereditable characteristics are usually seen in the intermittant appearance of affected offspring by the same sire out of several different but usually related dams. The sudden appearance of a defect in a high percentage of the calf crop would strongly suggest nutritional or other environmental agents as eithiological factors. Isolated cases of multiple anomalies in a single individual involving tissues and organ systems derived from more than one germ layer are usually embryonal accidents.

It is a speculative question as to which is the more important—hereditable or congenital defects. Certainly the opportunity for continued expression of undesirable defects is greater in hereditable traits. With the advent of frozen semen and artificial insemination the inadvertent dissemination of undersirable genes is an an important consideration. The test mating of a young bull on about 20 of his first daughters is likely to uncover any hidden recessive characteristics. This would not be an impractical practice in most artificial breeding programs.

The following statement by Fincher and Williams (27) may well summate consideration of congenital and hereditable defects in the bovine. "One principle is clear; it is as much the duty of the veterinarian to the community and to the state, to use whatever influence and power he possesses to prevent the spread of infectious disease. Each leads eventually to the same port."

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