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

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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 derived 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 interfere with reproduction that the veterinarian and livestock owner may be more fully informed of an important aspect of livestock disease.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achondroplasia</td>
<td>Three types: brachycephalic, dolichocephalic &amp; 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.</td>
</tr>
<tr>
<td>Missing phalanges</td>
<td>“Creeper calves” Absence of first two phalanges.</td>
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<tr>
<td>Acroteriasis congenita</td>
<td>“Amputated limbs” Hemimelia or Amelia. Forelegs terminate at elbow and hind legs at hock joint; Holsteins and Swedish cattle; Lethal.</td>
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<tr>
<td>Epitheliogenesis imperfecta neonatorum</td>
<td>“Skinless” Simple autosomal recessive; Raw areas devoid of skin mostly on limbs; Most dairy breeds; Usually lethal.</td>
</tr>
<tr>
<td>Hypotrichosis congenita</td>
<td>“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.</td>
</tr>
<tr>
<td>Impacted molars</td>
<td>“Parrot mouth” Milking Shorthorns. Lethal.</td>
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<tr>
<td>Achondroplastic micromelia</td>
<td>Russian cattle; Imperfections of lower limbs and shortened legs; Lethal.</td>
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<tr>
<td>Atresia ani</td>
<td>Imperforate anus; Lethal; Holsteins, Angus, Guernseys, Shorthorns, Ayrshires.</td>
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<tr>
<td>Atresia ilei</td>
<td>Norwegian cattle; Lethal.</td>
</tr>
<tr>
<td>Brachygnathia superior</td>
<td>Short maxilla; Simple autosomal recessive; Jerseys; May be lethal.</td>
</tr>
<tr>
<td>Brachygnathia inferior</td>
<td>Short mandible; Simple autosomal recessive; Shorthorns; Jerseys, Holstein, Ayrshire.</td>
</tr>
<tr>
<td>Agnathia</td>
<td>Absence of jaw; Lethal.</td>
</tr>
<tr>
<td>Sex linked lethal</td>
<td>Disproportionate sex ratios; Male or female lethal; Holsteins.</td>
</tr>
</tbody>
</table>
Abnormal skull: (61, 67)
Types:
1. Failure of fusion of frontal and parietal bones. Cerebral hernia in Holsteins.

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.
Most beef and dairy breeds. Usually lethal.

Mummification: (3, 23, 61, 62, 67)
Haematie 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 exophthalmos—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:
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 Müllarian 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.


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.

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<tr>
<td>Abnormal Wolffian or Gartner's Ducts:</td>
<td>Remnants of ducts become cystic.</td>
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<tr>
<td>Failure of fusion of Müllerian ducts:</td>
<td>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.</td>
</tr>
<tr>
<td>Hermaphrodites or Intersex:</td>
<td>Permanent joint contracture and fluid replacement of missing cerebral cortical tissue.</td>
</tr>
<tr>
<td>Hydranencephaly and Arthrogryposis:</td>
<td>Congenital absence of the epididymis, ductus deferens or seminal vesicles; Red Danish, Guernseys, Holsteins.</td>
</tr>
<tr>
<td>Aplasia segmentalis ductus Wölffii:</td>
<td>Sterile heifer born twin to a bull; 90% of bovine females born twin to a bull are sterile; Result of fusion of the placentae.</td>
</tr>
<tr>
<td>Freemartin:</td>
<td>Ectopia cordis, subaortic septal defect, ventricular septal defect.</td>
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<td>Cardiac anomaly:</td>
<td>Shorthorns, Herefords, Angus.</td>
</tr>
<tr>
<td>Persistent penile frenulum:</td>
<td>Types: 1. Duo-cecum large colon terminates in a rounded blind end. 2. Rumenal defects.</td>
</tr>
<tr>
<td>Digestive tract anomalies:</td>
<td>Inability to protrude penis or short penis; Guernsey, Holstein, Jersey.</td>
</tr>
<tr>
<td>Impotentia coeundi:</td>
<td>Angus, Hereford.</td>
</tr>
<tr>
<td>Curvature or deviation of penis:</td>
<td>Angus, Polled Hereford.</td>
</tr>
<tr>
<td>Preputial prolapse:</td>
<td>Urethral fistula.</td>
</tr>
<tr>
<td>Hypospadia:</td>
<td>“Crampy” “Spastic Syndrome” Holsteins, Guernseys, Ayrshires; Intermittent spastic contractions of back and leg muscles in mature animals.</td>
</tr>
</tbody>
</table>
Umbilical, scrotal, inguinal hernia:
(16, 61)
Holstein, Hereford.

Harelip and cleft palate:
(70)
Shorthorns.

Multiple lipomatosis:
(1)
Large fat deposits in perineal region.
Holsteins.

Hypomyelinoses congenita:
(16, 61, 73)
Ataxia, incoordination; Absence of myelin in cerebel-
lum 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)
Australian 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 ab-
ormal 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 dur-
ing pregnancy.

“Acorn” calves:
(16)
Unknown maternal deficiency; Abnormal osseous de-
velopment 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 con-
sulted. Roberts (61) has classified congeni-
tal defects according to their develop-
mental origin. Embryonal defects may be
manifested in any of the categories.

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

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2. defects due to failure of fusion
   i.e. spina bifida
   schistosomus reflexus
   palatoschisis
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 disappear­
   ance of contiguous structures
   that normally follow a certain pattern
   i.e. persistent urachus
7. defects due to fusion of sexual character­
   istics
   i.e. hermaphrodites
8. defects due to fusion of twin parts

Some of the more common congenital ab­
normalities 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 re­
view of the clinical incidence of the de­
fects. Herschler, et al. (44) has found
6.26% abnormal calves in a study of ap­
proximately 5,000 individuals. They found
that of 312 abnormal dairy calves the most common abnormality was the still­
born 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 ab­
normalities with sire, twinning, and pro­
longed gestation. No significant associ­
ation of breed with number of abnormali­
ties was found; however, there was a
highly significant association of breed with the frequency of certain abnor­
malities.

The high incidence of abnormal calves is an interesting observation. If an infec­
tious process were to claim 6% of the
nation's calf crop it would immediately be subjected to research and control at­
tempted. However, it seems that most con­
genital 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 exami­
nations the following was found:

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

Carrol et al. (18) has reported on exami­
nation 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 hypo­
plasia 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 hydrocephalus 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 hereditary 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 intermittent 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 ethiological 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 undesirable 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 hereditary 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.”

**BIBLIOGRAPHY**

11. Binne, W. et al. A congenital deformity experimentally produced in calves by feeding lupine...
12. Bishop, M. S. H. Paternal contribution to embryo·

13. Bishop, M. S. H. Paternal contribution to embryo·


15. Blood, D. C. Arthrogryposis and hydranenceph·

16. Blood, D. C. Arthrogryposis and hydranenceph·


18. Carroll, E. J., Ball, L., and Scott, J. A. Breeding

19. Carroll, E. J.

20. Clark, R. T.


22. Conneally, P. M.

23. Davidson, J. G. and Roberts, S. J. Fetal mummi·

24. Dawson, F. L. M. Uterine pathology in bovine

25. Dollahon, J. C. and Koger, M. Inheritance of the

26. Geerts, S. H.

27. Fincher, M. G. and Williams, W. L. Arrested de.

28. Fitzgerald, T. C. A study of the deviated penis


32. Geerts, S. H. The pathology of the anterior u.

33. Ginther, O. J. Similar loin muscle lesions in a


44. High, J. W., Kincaid, C. M., Smith, H. S. Dodder

45. High, J. W., Kincaid, C. M., Smith, H. S. Dodder

46. High, J. W., Kincaid, C. M., Smith, H. S. Dodder

47. Julian, J. C. and Koger, M. Inheritance of the


52. Marlowe, T. J. Evidence of selection for the


54. Marlowe, T. J. Evidence of selection for the

55. Mullenax, C. H. and Mullenax, P. B. Congenital

56. Mullenax, C. H. and Mullenax, P. B. Congenital

57. Muller, N. Hereditary factors in infertility in

58. Norel, R. Hereditary factors in infertility in

59. Parish, N. R.

60. Parish, N. R.

61. Parish, N. R.

62. Roberts, S. J. The enigma of fetal mummi·


