STUDIES OF BOVINE CONGENITAL DEFECTS

BY

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"Seek truth in even the most obscure matters so that once found, it will bring no fame to you but glory to The Creator."

R. Edwarde Rosell, 1644

INTRODUCTION

Congenital anomalies are defined as abnormalities of structure and/or function which are present at birth. Many or perhaps all structures or functions of the body may be affected by developmental aberrations and may vary from slight to severe. One defect may be obvious grossly while others may be discernible only after a careful clinico-pathologic and detailed necropsy examination.

Few studies have been undertaken of the nature and cause of congenital defects in cattle, and even less have dealt in detail with gross and microscopical aspects of these defects. The cause of congenital defects may be attributed to one of three factors: first, genetically - induced traits; secondly, abnormalities caused by environmental factors; and thirdly, the interaction of genetic and environmental factors.

The consequences of congenital defects are manifold and include economic losses to the livestock industry and their study may also provide biological models for allied disease processes in man.

The specific objectives of this study were: to review the literature of albinism and of congenital defects of the skeletal system; to determine the clinico-pathologic findings of these defects; to evaluate the gross and microscopic changes particularly of parts of the body affected as well as the endocrine and central nervous system; and to provide additional pathological information as well as pathogenetic and genetic data.

PAPER 1

OCULAR ALBINISM IN HERD OF NIGERIAN HOLSTEIN-FRIESIAN CATTLE

Various types of albinism, such as complete, incomplete, and partial, have been described in cattle.[1-10] This report describes the occurrence of ocular albinism in a herd of Nigerian Holstein-Friesian cattle.

MATERIALS AND METHODS

Four albino calves were encountered in the herd of Shika Research Farm of Ahmadu, Bello University, Zaria, Nigeria. One of the calves was available for slaughter and the eyes were retrieved at the slaughter plant. The eyes of a normal calf were also retrieved. The eyes were carefully observed and described grossly then fixed in 10% buffered, neutral formalin 20 times the volume of the eyes and later embedded in paraffin and sectioned at 8u thickness and stained with Haematoxylin and Eosin. Pedigree analysis was also used to establish a possible genetic pattern.

RESULTS

Herd data: Four Holstein-Friesian calves were encountered at Shika farm, three males and one female (Fig. 1). All four calves were sire-daughter matings by a purebred Holstein-Friesian bull (sire I-1 in Fig. 1). The first two dams were purebred Holstein-Friesians, and were identical twins of dam I-2 (Fig. 1) and their sire was also I-1 (Fig. 1). Dams II-3 and II-4 were halfbred Fulani X Holsteins and their sire was also bull I-1. Bull I-1 had sired 47 more calves in this herd, a sample of 12 examined clinically had normal eyes. The dams of the affected calves had normal eyes. The bull I-1 was not available for examination but reportedly had normal eyes.

The calves had coat color patterns like Holstein-Friesian

cattle, however, the coat color was a dilute grey instead of black (Fig. 2). All four calves had grey irises (Fig. 3).

One calf's eyes were retrieved at slaughter. The irises were dilute grey. The fundi had a faintly green tapetum lucidum and a grey tapetum nigrum. The ciliary body was grey. The optic disc appeared normal.

The histologic changes were as follows: The anterior layer of the iris and stroma lacked pigment (Fig. 4). The posterior layer of the iris had a reduced amount of pigment. Reduced amounts of pigment were also present in the ciliary body and tapetum nigrum. The albinotic eye had considerably less pigment compared to a normal eye (Fig. 5).

DISCUSSION

Various types of generalized and ocular albinism have been described in cattle.[1,3,4] Amongst the generalized forms of albinism is partial albinism which is characterized by a grey iris centrally and brown peripherally.[1-4] Another form of partial albinism has been illustrated in the recessively inherited Chediak-Higashi syndrome, which besides its albinotic features includes abnormally large membrane-bound organelles in various cells and increased susceptibility to infection.[7]

Incomplete albinism, inherited as an autosomal dominant character is characterized by pure white hair coat and skin, a few animals may have small pigmented areas on the body. The iris may vary from blue to grey to white and may contain brown sectors. Incomplete albinos have colobomas of the non-tapetal fundus and tapetal fibrosum hypoplasia.[1,3,4]

Complete albinism, inherited as a simple autosomal recessive trait, is characterized by pure white hair coat and white pink irides but a normal tapetum lucidum. Complete albinism has been reported in Holstein-Friesian[6,8] in a Guernsey calf,[5] in a Murbodner calf,[9] and in European Brown Swiss.[10]

SUMMARY

Ocular albinism combined with dilution of body color occurred in four Nigerian Holstein-Friesian calves. The calves exhibited grey irises and the general body color was dilute grey. Ocular examination revealed a normal tapetum lucidum and normal optic nerve. The condition is most likely caused by homozygosity of a simple autosomal recessive gene.

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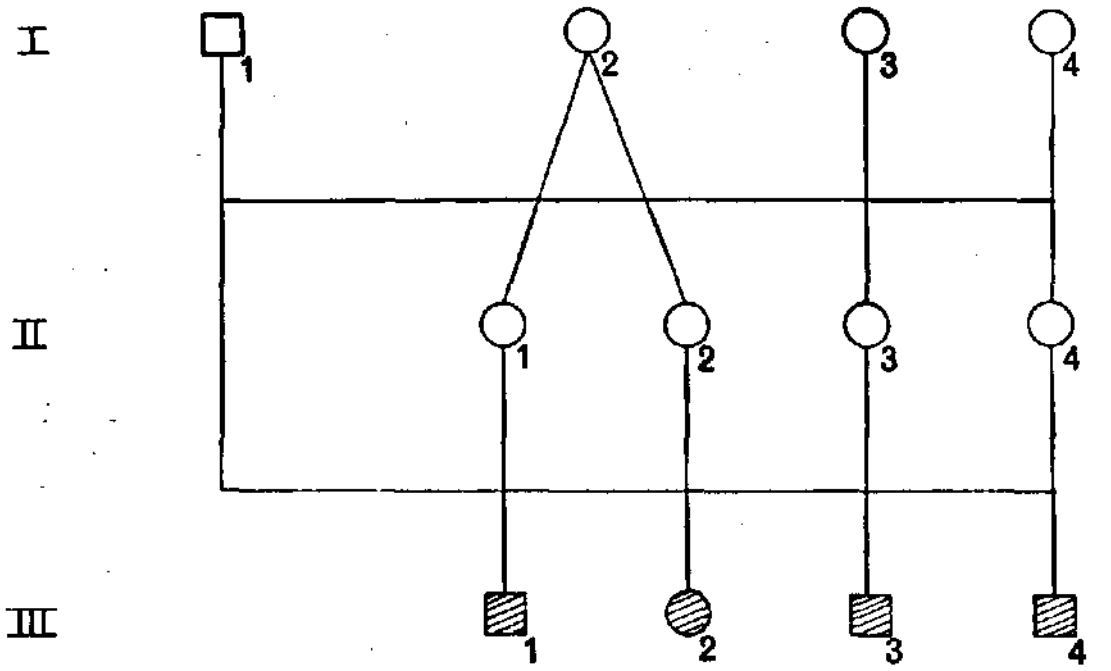


Fig. 1

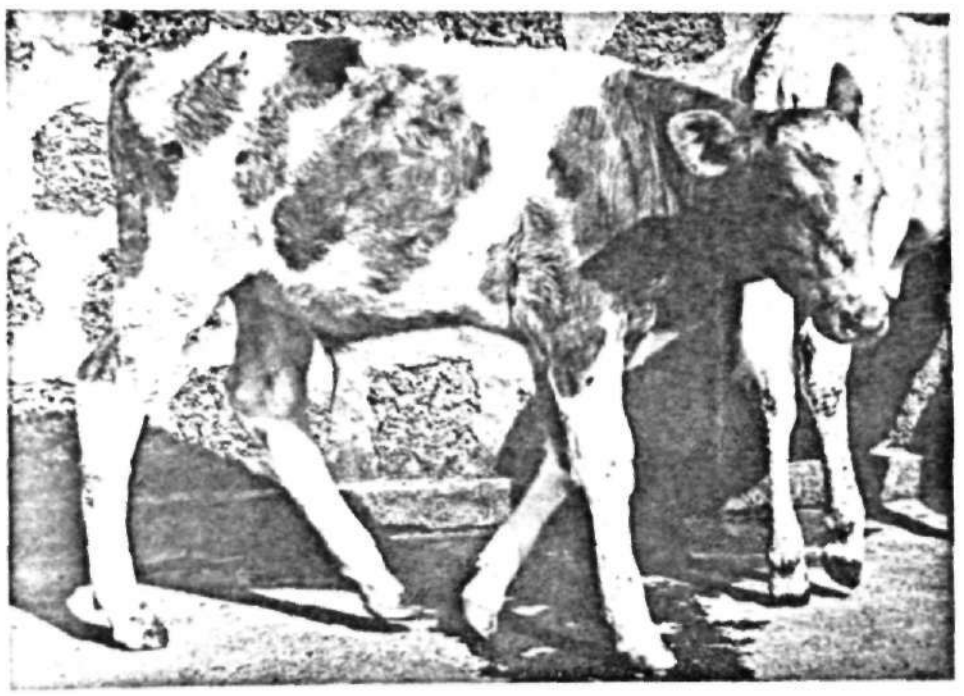


Fig. 2

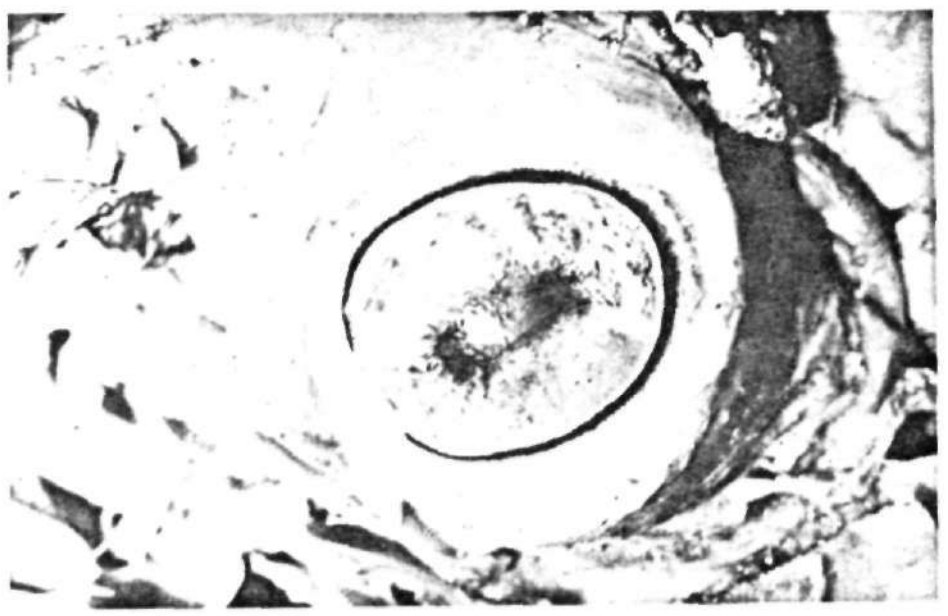


Fig. 3

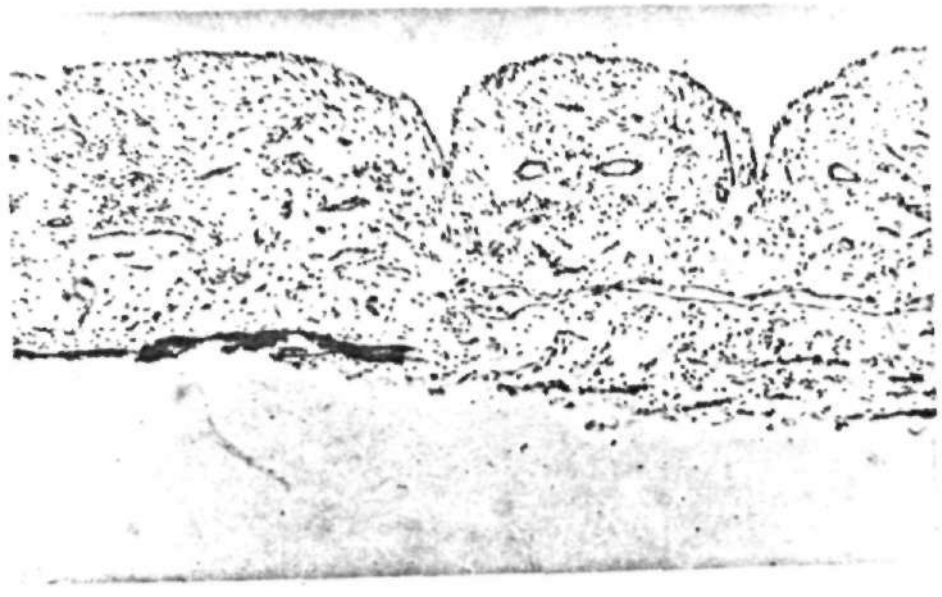


Fig. 4



Fig. 5

PAPER 2

OCULAR ANOMALIES OF INCOMPLETE ALBINO CATTLE: PATHOLOGICAL CHANGES

INTRODUCTION

The term Coloboma means absence of a part, was first applied to the eye by Walther (1821), indicates a condition where a portion of a structure of the eye is lacking. Later authors have variously described it as a notch, gap, hole or fissure in any of the ocular structures. A coloboma is considered typical if the defect is situated in or near the line of the foetal ocular cleft (vertical and through the optic disc), or atypical if it is situated elsewhere in the eye.

MATERIALS AND METHODS

Fifteen albino cattle and 3 normal cows controls were evaluated. Each animal was euthanized by intravenous administration of Sodium pentobarbital and underwent immediate necropsy examination. The eyes were removed and promptly fixed in 10% buffered neutral formalin twenty times their volume. The tissues were sectioned 8u thick, then routinely processed and stained by hematoxylin and eosin.

RESULTS

The typical control eye had the following histological morphology. The capsule of the eye can be divided for descriptive purposes into:

a) An outer fibrous tunic, composed of cornea and sclera.

b) A middle vascular layer, the uvea, composed of the choroid, The ciliary body and Iris.

c) An inner tunic, the retina.

The cornea was covered by stratified squamous epithelium anteriorly and adjacent to it is a structureless homogeneous membrane, Bowman's

membrane. The posterior surface of cornea was covered by simple low cuboidal epithelium and this epithelium was located adjacent likewise to a structureless homogeneous membrane (Descemet's membrane). The body or stroma of cornea, substantia proporia, consist of parallel bundles of collagenous fibrils termed lamellae and between these bundles are rows of flattened, branching fibroblasts termed Corneal cells.

The sclera was white, opaque, and composed of densely woven collagenic fibers. The junction of Cornea and Sclera is termed Corneal limbus. The point where optic nerve emerges from ocular capsule is the site of continuation of Sclera and duramater.

Ciliary body

Composed to ciliary smooth muscle and processes which is folded and highly vascularized. To this processes are attached the suspensory ligaments.

Choroid

Adjacent to Sclera. It is composed of chromatophores and pigmented posterior epithelium and rich in blood vessels.

Iris

The colored portion of the eye had the anterior and posterior layers highly pigmented and the stroma was lesser pigmented.

Retina

Composed of ten layers among which is the layer of light receptive cells, and rods and cones. The macula lutea area was devoid of rods and blood vessels and possessed an abundance of cones. The optic papilla lacked both rods and cones.

Albino Eyes

A total of 30 eyes from 15 incomplete albino cattle each had coloboma of the optic disc. Gross and histological findings of incomplete albino cattle are presented in Tables 1 and 2 and Figures 1 to 10. The cornea and anterior eye chambers appeared normal. The irises were grossly grey, blue or blue and white. The histological changes of the iris were characterized by reduced pigmentation. The anterior layer and the stroma of the iris were free of pigment. The posterior layer revealed reduction of pigment and abnormal clumping. The ciliary body was lightly pigmented. The typical coloboma was located at the ventral aspect of the optic disc (Fig. 1).

Other Lesions

One albino cow had numerous cysts of the liver (Fig. 2).

DISCUSSION

As early as 1920 an albino herd of cattle was described.[4] These albinos lacked pigment in the skin, eyes, horns or hoofs and the animals were extremely photophobic. The parents of the albinos presented a normal Holstein coat pattern. This suggested a recessive gene, but further data indicated dominance as the albino bull sired only albino calves when mated to unrelated grade Holstein cows.[4] Furthermore, the matings of albino females to a Holstein bull produced only albinos. The breeders records of this herd were lost and the data presented were based on memory. However, the author did not question the accuracy of the report given by the owner of the herd.[3]

A single case of a female calf with complete lack of pigment in skin, hair and iris was reported.[9] The pupils were red. The breeding

test indicated a recessive gene since the mating of an albino female resulted in a normal offspring.

Another herd of albino showed the following characteristics: complete lack of pigmentation in skin, iris and hair at birth, however at sexual maturity some pigment could be observed and this phenomenon was designated as "ghost pattern".[3]

Histological examination of two albino cattle's eyes from the herd revealed no pigment on the retina whereas the iris and ciliary body showed some pigmentation causing the pupils to be pink but the irises, grey. Breeding test were performed and data obtained indicated a recessive gene for extreme reduction of pigment.[3]

The occurrence of 22 albino animals in Brown Swiss Cattle was investigated in Germany.[2] Fourteen animals were closely examined and one female and male were purchased and mated and produced a male albino calf. The three animals were later slaughtered and specimens were taken from the eyes, skin, horns, claws, and hairs. The tissues were all unpigmented. The iris was white with a pinkish shine, the pupils were red and photophobia was extreme. The authors concluded that albinism is a recessive trait since test mating of albino X albino resulted in albino and albino X normal resulted in normal coat color. Furthermore, pedigree studies were done and in herd in which albinos occurred, common ancestors could be traced.

Three albino animals were reported in a Holstein herd.[8] The skin was described as pinkish and the muzzle, hoofs, and horns, unpigmented. The hair was creamy-white and no ghost pattern was observed. In daylight, the iris was greyish-blue and the pupil black, whereas in twilight, the pupils had a pinkish shine. The animals were

photophobic. The inheritance was thought to be recessive since both parents of all 3 animals were related.

The results of 43 matings demonstrated to Peterson that albinism was inherited as a simple recessive.[20] Further, it was shown that ghost pattern was due to a structural defect of the hairs rather than different degrees of pigmentation. The appearance of ghost pattern was associated with the factors for black (B) and was obvious at sexual maturity.

From Japan came a report of three albino Holstein calves[17] from a common sire and whose ancestors had been imported from the United States. In Wisconsin and Minnesota, the occurrence of albinos had been reported in these families.

In the Murbodner breed in Austria, a single albino calf was observed.[2] This calf, the result of an accidental mating of a son to his dam, showed no pigmentation at all and the iris was pink. Comparative body measurements and skin thickness did not reveal any differences from normal.

Three cases of albinos were reported in Brown Swiss Cattle.[23] In each case, the parents exhibited normal coat color. Breeding experiments were not carried out but a single autosomal recessive factor was presumed. These cases were related to earlier cases, so sporadic reappearance of same mutation was considered. The cattle were described as having pink eyes, pink skin, lack of pigment, and photophobic.

Albinism has also been reported in Hereford Cattle.[18] Three albinos were observed in a small herd of his full sisters and four of his half-sisters. Several animals were acquired from this herd for

breeding tests and from the brother-sister matings, three albinos resulted. The affected offspring had light pigmented areas on the inside of the hind legs but no evidence of ghost pattern. Clinically, the calves showed photophobia but were otherwise normal. Blood types of the sires and dams of the albinos were determined and were similar. Albinism and dwarfism were observed together in the same animals, but it was felt that the two traits were independent.[6] A blood abnormality was found in these partial albinos which was considered to be identical to that observed in the Chediak-Higashi Syndrome of man.[19] Increased susceptibility to disease was also observed. Breeding experiments performed with these cattle revealed a recessive gene.[18]

Chemical investigations of the pigment contents of bovine hair showed some melanin was present, not only in white hair from colored Herefords, Holsteins, and Guernseys but also in albino hair.[22]

In conclusion, general specific types of albinism have been reported in cattle.[10,11,12,13] They ranged from ocular albinism[10,11] over incomplete and partial albinism.[10,11,13,14]

In partial albinism, the iris is blue and white centrally, and brown peripherally, and the coat is usually characteristic of the breed or a dilute color.[5,10,11,12] A form of partial albinism is illustrated by the recessively inherited Chediak-Higashi syndrome, which besides its albinotic features, includes abnormally large membrane-bound organelles in various cells and an increased susceptibility to infection.[19]

Incomplete albinism, inherited as an autosomal dominant character is characterised by pure white coat, a few animals may have small pigmented areas of the body. The iris may vary from blue to grey to white and may contain brown sectors. Incomplete albinos have

colobomas of the nontapetal fundus and tapetal fibrosum hypoplasia.[5,10,11,12]

Complete albinism, inherited as a simple autosomal recessive trait, is characterized by pure white hair coat color and white to pink irides but a normal tapetum lucidum. Complete albinism has been reported in Holstein-Friesian,[17,18] in a Guernsey calf,[13] in a Murbodner calf,[21] and in European Brown Swiss.[24]

Coloboma of the eye in cattle have been described in Charolais cattle[1] as an incidental findings in a Slaughterhouse Survey, and associated with other eye anomalies in related albino Hereford Cattle.[5,11] In 1971 two separate cases of an ocular defect in imported Charolais heifers were investigated and in both cases the lesions found were diagnosed as ocular coloboma.[1] This condition is known to be hereditary in man and many species of animals.[1]

Relatively few ocular defects have been described in cattle.[16] Ocular defects reported in cattle include micropthalmia, anophthalmia, ankcyloblepheron, entropion, opacity of the cornea, cataracts, micropkia (hypoplastic lens), ectopia lentis and blindness. The majority of these defects were reported to be due to a single autosomal recessive trait.[15,16]

Ocular defects of incomplete albinism of Hereford cattle have included heterchromia irides, tapetum fibrosum hypoplasia and coloboma of nontapetal fundus.[5]

Descriptions of ocular defects in cattle have been included with abnormalities in other body organs, especially the central nervous system. Internal hydrocephalus has been associated with cataracts, corneal opacity, microphthalmia, retinal detachment, persistent

pupillary membrane, and retinal dysplasia.[5] Congenital defects of the caudal segments of the vertebral column and high Ventricular Septal defects have occurred with anophthalmia and microphthalmia.

The term Coloboma (mutilation), introduced by Walther (1821), indicates a condition where in a portion of a structure of the eye is lacking. The great majority of congenital defects of this native is found in the region of the embryonic cleft and is due to a disturbance of the mechanism of its closure; these are termed TYPICAL COLOBOMATA. Congenital defects of a similar appearance occurring elsewhere on the eye which have a different etiology are termed ATYPICAL COLOBOMATA.

Typical colobomata occur between time of invagination of the optic vesicle and closure of the cleft. They may be complete, implicating all the structures associated with the embryonic cleft, or partial when the defect is less extensive. The term thus includes a whole series of anomalies varying from a complete defect involving the optic disc, the retina and choroid, the ciliary body, the iris, and even the lens, associated in the more extreme cases with the formation of cysts arising from the lips of the fissure, to a localized and partial failure inner layer may form a cyst, the cavity of which is continuous with the primary optic vesicle. Initially the everted retina differentiates to some extent and an attempt at layer formation may be evident. The rods and cones are turned towards the cavity of the optic vesicle and bundle of nerve fibers run along the outside of the eye. These aberrant fibers are only temporary and never reach the optic nerve before they degenerate.

Depending on the extent of retinal eversion and the degree of nonclosure, a number of defects may arise.

Today it is generally accepted that typical Colobomata are due to some interference with the normal closure of the embryonic cleft, the aberration lying formally in the epiblast of the optic cup.

We have already seen that the development of the retina lags behind in the region of the embryonic cleft, and that initially the inner layer (the foresumptive retina) normally grows more quickly than the outer (pigmentary) layer of the optic cup. The result is that while the cleft is still open there is a slight eversion of the inner layer through the cleft. Normally the lips of the cleft meet in its central part and, since the cells are still in the pluripotential stage, the two layers fuse rapidly and completely. Peripherally the closure of the cleft leaves a slight notch on the rim of the optic cup for a little time so that proximally the cleft never closes where the hyaloid artery enters, and in this region the eversion of the inner layer persists for some time so that the fusion of the two outer layers is delayed and a short nonpigmented strip remains at the proximal end of the fissure. It is an accentuation of this process that results in the formation of typical Colobomata.

It would seem that the pluripotential stage has been passed and hence fusion cannot take place. While the outer pigmented layer remains stationary, this process of eversion proceeds and, since the choriocapillaris does not develop apart from the pigment epithelium, the wall of the eye within the sclera condensation is formed merely of two retina layers, the outer of which is inverted. Such a process is usually most accentuated about the middle of the fissure, and if the eversion continues the redundant of closure of which the evidence may be a small notch at the pupillary margin, a slight hypoplasia of the mesoderm of

the iris, a linear area of abnormal pigmentation in the fundus, or an exaggerated excavation of the optic disc.

Typical Coloboma of all types, whether partial, complete or cystic, despite their great individual variations are members of one continuous series and although a topographical division is useful from a clinical point of view, their nature, the mechanism of their formation and their hereditary characteristics are most economically considered together.

SUMMARY

The Pathological Changes of Ocular anomalies of incomplete Albino cattle showed iridal heterochromia grossly. Histopathological findings of irises showed only posterior layer were fairly pigmented and usually no pigment in the stroma nor the anterior layer. The ciliary body showed reduced amount of pigmentation and absence of Copra nigra on two cases. Choroid lacked pigmentation. Retina showed disorganization in those eyes with cystic Coloboma and rosette formation. Fundus anomalies induced Colobomata of varying sizes at the ventral aspect of the optic disc and the tapetum fibrosum was hypoplastic.

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Table 1: Gross Pathological Changes

No.	Iris Color	Ciliary Body	Fundus Tapetal	Fundus Non-Tapetal	Optic
1	Grey	Reduced pigment	Light green	Reduced pigment	Normal
2	Grey black section ventral	"	"	"	Coloboma
3	Blue periphery and white center	"	"	"	Coloboma
4	Coloboma iris white periphery and black center	"	"	"	Large Coloboma
5	Blue	"	"	"	Normal
6	Blue with grey and black spots	"	"	"	Normal
7	Irregular black and grey	"	"	"	Normal
8	Blue	"	"	"	Normal
9	Blue	"	"	"	Normal
10	Grey	"	"	"	Normal
11	Blue	"	"	"	Coloboma
12	Blue periphery white center	"	"	"	Coloboma
13	Blue	"	"	"	Coloboma

Table 1 (continued)

No.	Iris Color	Ciliary Body	Fundus Tapetal	Fundus Non-Tapetal	Optic
14*	Blue periphery white center	Reduced pigment	Light green	Reduced pigment	Large Coloboma
15	Light black	"	"	"	Coloboma
16	Black	Normal	Normal	Normal	Normal
17	Black	"	"	"	"
18	Black	"	"	"	"

*Both eyes had microphthalmia

Table 2: Histopatholical Findings in Incomplete Albino Cattle Eyes

No.	Cornea	Sclera	Iris	Cilary Body	Choroid	Retina	Fundus
1	Normal	Normal	Only posterior layer was pigmented	Reduced pigmentation	Lacked pigmentation	Normal	Hypoplastic tapetum coloboma at ventral aspect (4d.d.)
2	Normal	Normal	Only posterior layer was pigmented	Reduced pigmentation Absence of copra nigra	Lacked pigmentation	Disorganized	Coloboma at ventral aspect presence of rosette (5d.d.)
3	Normal	Normal	Dense pigmentation of posterior iris with other layers lacking pigment	Reduced pigmentation	Lacked pigmentation	Disorganized retina	Hypoplastic tapetum and large cystic coloboma (5d.d.) at ventral aspect.
4	Normal	Normal	Only posterior layer pigmented	Reduced pigmentation	Lacked pigmentation	Normal	Hypoplastic tapetum with coloboma at ventral aspect (2d.d)
5	Normal	Normal	Only posterior layer pigmented	Reduced pigmentation	Lacked pigmentation	Normal	Normal
6	Normal	Normal	Only posterior layer pigmented	Normal	Lacked pigmentation	Normal	Normal except hypoplastic tapetum

Table 2 (continued)

No.	Cornea	Sclera	Iris	Cilary Body	Choroid	Retina	Fundus
7	Normal	Normal	General pigment reduction on all layers	Reduced pigmentation	Reduced pigmentation	Normal	Hypoplastic tapetum and coloboma at the ventral aspect (2d.d)
8	Normal	Normal	Only posterior layer pigmented	Faintly pigmented	Lacked pigmentation	Normal	Coloboma dipping into choroid ld.d.)
9	Normal	Normal	Posterior layer only pigmented	Faintly pigmented	Lacked pigmentation	Normal	Remmant of hyaloid artery
10	Normal	Normal	Posterior layer pigments with scattered in the stroma and Anterior layer	Faintly pigmented	Lacked pigmentation	Normal	Coloboma at ventral aspect (3d.d.)
11	Normal	Normal	Only posterior layer is pigmented	Faintly pigmented Absence of copra nigra	Lacked pigmentation	Normal	Large cystic coloboma at ventral aspect (5d.d.)
12	Normal	Normal	Only posterior layer is pigmented	Faintly pigmented	Lacked pigmentation	Normal	Large cystic coloboma (5d.d.) at ventral aspect

Table 2 (continued)

No.	Cornea	Sclera	Iris	Ciliary Body	Choroid	Retina	Fundus
13	Normal	Normal	Only posterior layer is pigmented	Faintly pigmented	Lacked pigmentation	Normal	Coloboma (3d.d.) and hypoplastic tapetum
14	Normal	Normal	Posterior layer heavily pigmented	Faintly pigmented	Lacked pigment	Disorganized	Coloboma (4d.d.)
15	Normal	Normal	Posterior layer only pigmented	Faintly pigmented	Lacked pigment	Normal	Coloboma (3d.d.) and hypoplastic and tapetum
16	Normal	Normal	Normal	Normal	Normal	Normal	Normal
17	Normal	Normal	Normal	Normal	Normal	Normal	Normal
18	Normal	Normal	Normal	Normal	Normal	Normal	Normal

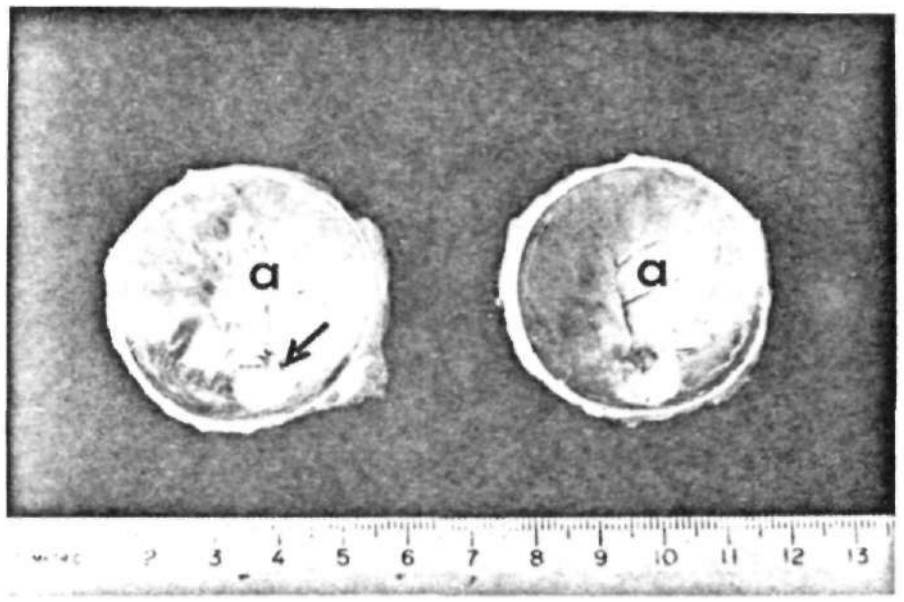


Fig. 1

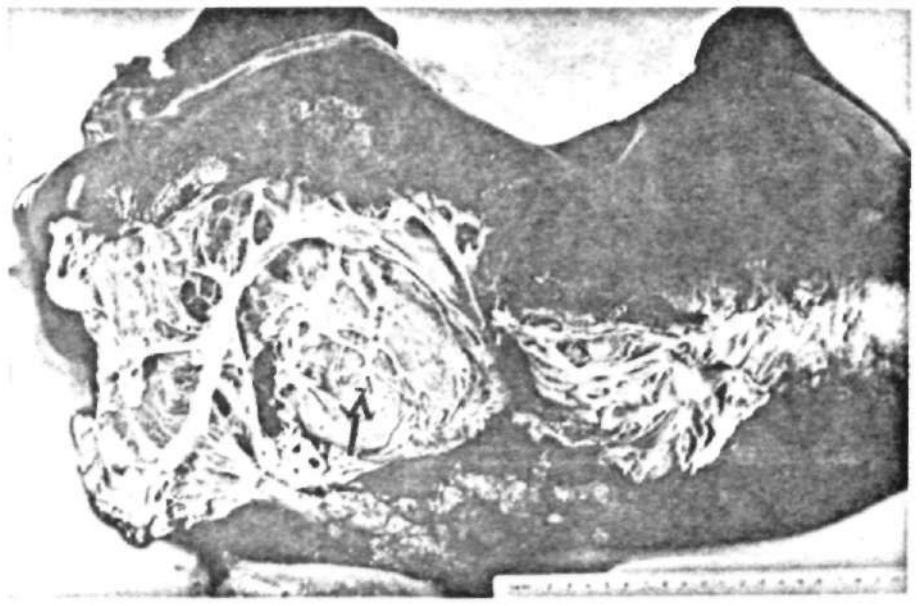


Fig. 2

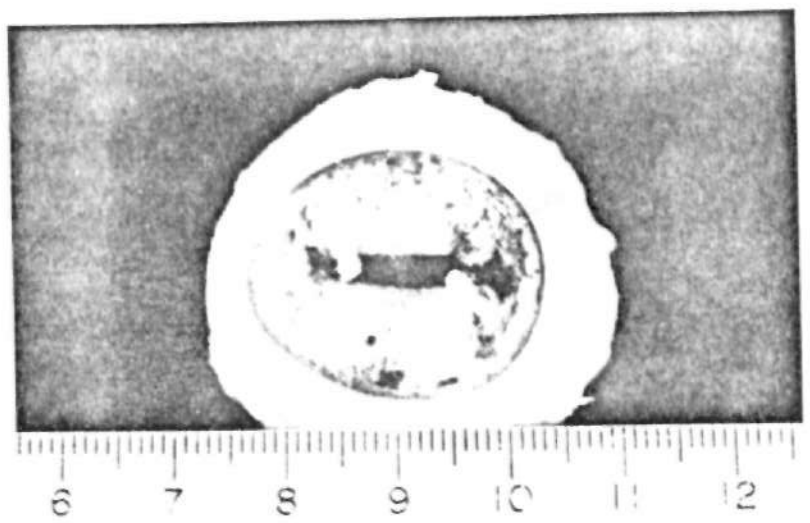


Fig. 3

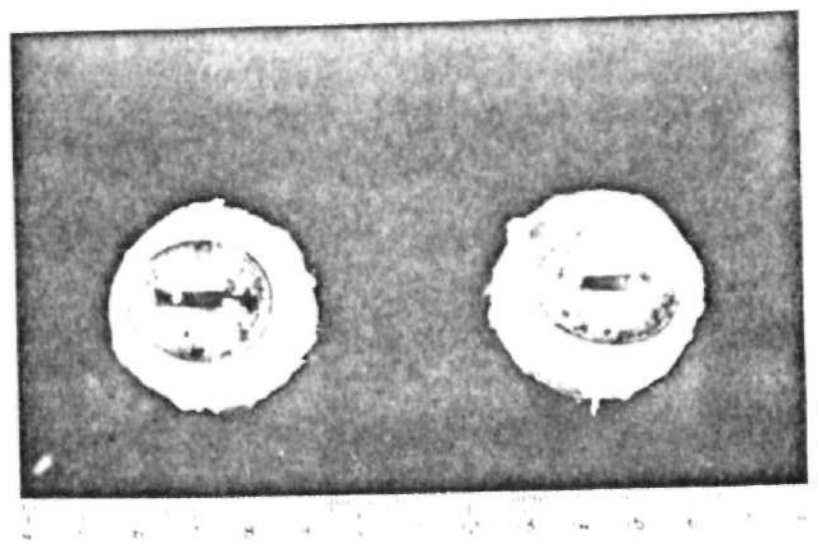


Fig. 4

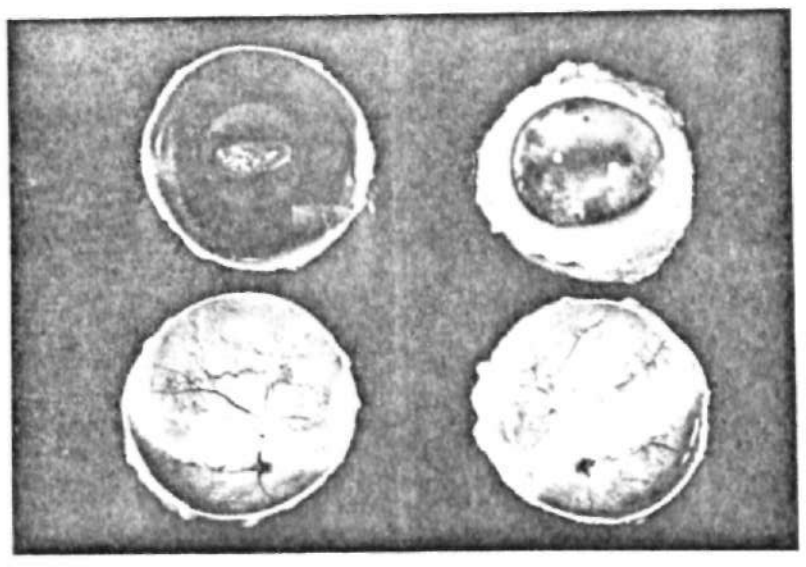


Fig. 5

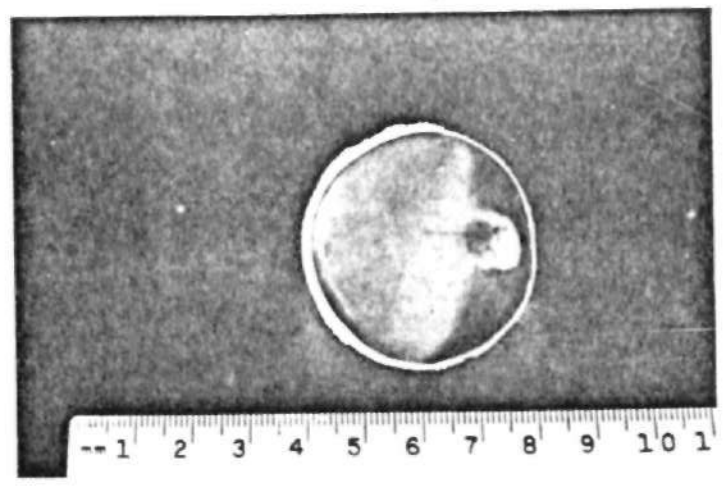


Fig. 6



Fig. 8



Fig. 10



Fig. 7

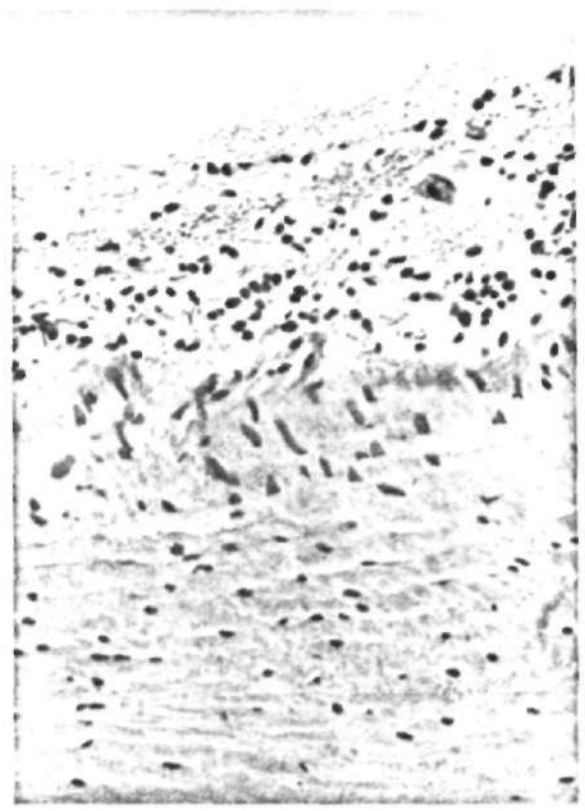


Fig. 9

PAPER 3

CONGENITAL DEFECTS OF THE SKELETAL SYSTEM IN CATTLE

INTRODUCTION

The skeletal system is involved in many congenital defects, either as isolated defect or the entire skeleton maybe affected. Although many different skeletal dysplasias have been described in man, relatively few dealt with cattle.[1-4, 6-9]

MATERIALS AND METHODS

Calves in this study were received as part of a long term study of nature, cause, and frequency of congenital defects in cattle. The calves were submitted to Kansas State University during a 16 month period from fall 1973 to fall 1974. Radiological and clinical signs were recorded. The calves underwent careful necropsy examination. Tissue samples of brain, bone and representative organs were selected and fixed in 10% phosphate buffered neutral formalin, sectioned at 6u and stained with hematoxylin and eosin. Bone sections were first decalcified with formic acid prior to histological sectioning.

The calves also underwent karyotyping according to the method outlined by Morrehead. Whenever possible extensive herd and breeding histories were obtained.

RESULTS

Two distinct generalized skeletal defects were encountered in cattle dwarfism and osteopetrosis in Hereford calves. Five localized skeletal defects were encountered, they included polydactyly in a Holstein-Friesian calf, camptodactyly in a Simmental calf, tibial hemimelia in Galloway calves, facial-digital syndrome in Angus calves, and syndactyly. Table 1 summarizes the features of these defects.

DISCUSSION

Out of a total of 1275 congenital defects studied in Kansas, most common were skeletal defects followed by those of the central nervous system and the muscular system.[1]

The 476 skeletal defects are recorded by affected body region. Generalized skeletal defects were chondrodystrophy in Angus and Hereford cattle.[3]

Regional skeletal defects included campylognathia, Roman nose, schistoprosopus and palatoschisis. Tetramelic arthrogryposis and palatoschisis in Hereford and Charolais cattle were frequently seen. Brachygnathia inferior was commoner than agnathia. Some rare syndromes like short-spine lethal and atlanto-occipital fusion were seen. Most frequent defects of the axial skeleton were kyphoscoliosis and taillessness.[1,3] Several types of appendicular skeletal defects were encountered, including polydactyly, monobrachia, adactyly, ectrodactyly, and syndactyly.[1,3,5,8]

There were generalized and regional skeletal defects which had a genetic cause.[1,2,3,5,6,8] Dwarfism (chondrodysplasia, bulldog calves, achondroplasia) was economically important in North American Aberdeen Angus and Hereford herds. A chondrodystrophy gene also was present in Charolais and in Holstein cattle in the midwestern sector of the United States.

Osteopetrosis is caused by homozygosity of a simple autosomal recessive gene, (lack of bone resportion) occurred in both sexes of red and black Angus cattle. Affected calves were stillborn, small, and had short, immobile mandibles. Long bones, although completely solid and lacking bone marrow cavities, fractured easily under lateral

pressure. Gross examination of longitudinal bisections of bones revealed absence of bone marrow cavities and a solid bone-within-bone appearance. Radiographs disclosed homogenous bone densities without distinction between cortical bone and bone marrow cavity. The cranial cavities reduced in size gave rise to cerebellar coning. The optic nerves were hypoplastic.[2,6]

Histologic and electromicroscopical examination of bone tissue disclosed persistence of primitive chondro-osseous tissue within the medullary cavity. Bone resorption was deficient. Osteoclasts were present but appeared to be inactive. Other histological changes included mineralized vascular walls and neurons in various areas of the brain including the hypothalamus which was most severely affected.[2,6]

Syndactyly in Holstein-Friesian cattle (fusion or nondivision of functional digits) resulted from a simple recessive gene with incomplete penetrance.[4,8,9]

The external features of syndactyly in Holstein-Friesian cattle include four groups: (1) normal overlaps, despite sy/sy genotypes; (2) partial syndactyly, two clearly defined hooves partially fused at the axial-coronary border, (3) intermediate syndactyly, a syndactylous hoof's dual origin indicated by dorso-axial groove, (4) complete syndactyly, a hoof with no indication of its dual origin.

The external fusion pattern includes right-left and front-rear gradients, which were paralleled by the osteological pattern.[8]

The most proximal synostosis involved the carpal and tarsal bones: *synosteosis of intermediate and carpal bones, synosteosis of* the first tarsal bone with the fused second and third tarsal bone.

The right metacarpal bone was brachydactylous in most

syndactylous animals. The distal trochleas were reduced to a single trochlea with one or two sagittal ridges. Some animals revealed tendency towards syndactyly by enlargement of the small metacarpalia with numbers of proximal sesamoid bones varying. The phalanges revealed horizontal synosteosis with second phalanges most frequently fused followed by fused third and first phalanges.

In the fetlock region, the three digital extensor muscles fused to a wide aponeurotic plate which inserted a phalanx II and III. The superficial and deep flexor tendons usually remained undivided. The vascular and nerve supply were also adapted to the syndactylous condition.

Syndactyly, an anatomical defect, is associated with hyperthermia.[8] During the 15 year period of study of syndactylous cattle, nine syndactylous cattle had serious heat adaptation problems and nine, slight discomfort during hot summer weather. Five syndactylous and five control cattle were subjected to standardized, moderate, temperature stress in a climate chamber. All syndactylous cattle developed clinical signs of hyperthermia, including elevated rectal temperatures (41.5° to 45° C), tachycardia, and tachypnea. Two syndactylous cattle developed oliguria and haematuria; one developed polyuria, and three became recumbent and paralyzed after exposure. One cow died without premonitory signs after 96 hours exposure. Blood chemical changes were slight, except for preterminal hyperglycaemia in two cows. Change in total leukocyte count was small. However, relative and absolute esinopenia, lymphopenia, and neutrophilia occurred in the syndactylous cattle. The only changes observed in the controls were attributable to normal adaptation. Post mortem examination revealed a wide-spread

parenchymatous degeneration.[8]

Reportedly, 0.2 and 3.6 percent of all calves born are affected with congenital defects.[1-4, 6-9] Congenital defects in cattle result in economic losses from decreased reproductive capacity, increased perinatal losses and reduced value of related calves when defects are genetic. Many congenital defects follow a simple pattern of Mendelian inheritance. Others are caused by environmental factors, and still others have no clearly established cause. Various parts of the body may be affected with a congenital defect. Some may be single isolated defects; however, it becomes increasingly obvious, that many defects occur together as part of a syndrome. Frequencies of defective anatomical structures vary with geographical region, breeds, and other factors. However, in most studies in cattle the most frequently encountered congenital defects are those of the central nervous, muscular, and skeletal systems.[7]

Studies of the literature of congenital defects in cattle revealed that about one-third of the defects involved the skeletal system, either the entire skeleton or smaller localized areas. Most congenital defects occurred as syndrome rather than as single entities. Skeletal dysplasias encountered were chondrodystrophy, persistence of secondary spongiosa, and osteopetrosis. The commonest genetic disease of localized skeletal regions was syndactyly in Holstein-Friesian cattle.[1,6,8]

SUMMARY

Recent research of bovine congenital defects of the skeletal system were reviewed. Generalized defect osteopetrosis seems to be

common in Angus calves. Syndactyly, the fusion or non-division of functional digits, is common in Holstein-Friesian cattle.

This study included the description of dwarfism in a Main-Anjou calf as a generalized skeletal defect. Osteopetrosis is a newly recognized skeletal defect in Hereford cattle.

Regional skeletal defects seen were: polydactyly, camptodactyly, tibial hemimelia, facial-digital syndrome, and syndactyly.

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Table 1: Generalized and Localized Congenital Skeletal Defects in Cattle

Types	Nature of Defect	Associated Defects	Etiology
A. Systemic Defects 1. Dwarfism in a Maine-Anjou calf	Stocky, chunky animal with abdomen close to the ground and short head. The long bones had knobby epiphyses and short shafts. The cranium had a short base.	Internal hydrocephalus vertebrae were compressed.	Genetic recessive trait pending breeding trial
2. Chondrodystrophy in a Holstein-Friesian calf	Calf died shortly after birth. Had domed shaped skull, reduced long bones with cartilagenous epiphyses.	Both fore and hind legs had missing phalanges, moderate palatoschisis and internal hydrocephalus.	Genetic - implicating a number of chromosomal foci.
3. Osteopetrosis in Hereford calves	Stillbirth, long bones were fragile and frectured easily under lateral pressure. Radiographical bones were homogeneous without bone marrow cavities. Reduced cranial cavity with multiple cysts in the frontal bone, brain lesions characterized by mineralized vessel walls and neurons.	Undersized with brachgnathia inferior.	Homozygosity of a simple autosomal recessive gene (resulting from in-breeding).

Table 1 (continued)

Types	Nature of Defect	Associated Defects	Etiology
B. Localized Defects			
1. Polydactyly in Holstein-Friesian calves	3 separate digits in the forelegs also 3 digits with fusion of lateral and middle digits in the hindlegs.	Polysyndactyly, missing proximal sesamoid and navicular bones.	No causative factor was evident in the calf described. (Normal bovine female karyotype)
2. Camptodactyly in a Simmental calf.	Medial digits of both hindfeet deviated from longitudinal axis and had contracture.	Absence of proximal and distal sesamoids in the forelegs. Both kidneys hypoplastic and contain numerous oxalate crystals.	No causative factor was evident in the calf described.
3. Tibial Hemimelia in Galloway calves	Agenesis of Tibia of only one leg. (hindled)	Agenesis of Patella, non closure of pelvic symphysis cranioschisis meningocele, internal hydrocephalus, cryptorchism, or nonfusion of Mullerian ducts.	Most likely inherited as simple autosomal recessive.
4. Facial Digital syndrome in Angus calves	Syndactyly of the legs, shortface, one nasal opening.	Kyphoscoliosis, internal hydrocephalus and spinal dysraphism.	Most likely inherited as a simple autosomal recessive.
5. Syndactyly in cattle	Partial or complete fusion of functional digits on one or more feet.	The muscles (a poneurosis of extensor muscle) and tendons adapted to the syndactylous defect.	Hereditary autosomal recessive.

PAPER 4

DWARFISM IN CATTLE

INTRODUCTION

The peculiar shape of the chondrodystrophic dwarf aroused interest of laymen, from the artist to the priest, long before it was studied by the medical profession.[1-14, 16-22]

Many different types of systemic skeletal congenital diseases have been studied in man[6,15] but few have been described and well studied in cattle.[14] In cattle chondrodystrophy, osteopetrosis, crooked calf disease, and acroteriasis congenita have been described.[14] Various types of dwarfism such as shortheaded, long-headed and Telemark were described and were considered to result from homozygosity of simple autosomal recessive genes.[1,2,6-14] In addition, the Dexter, comprest and compact mutants are generally considered to be dominants, and part of a complex of conditions from more than one locus. They appear to be related to the simple autosomal recessive types.[3-8,14]

This paper describes clinical, radiological, gross and microscopic features encountered in a Maine-Anjou dwarf and a chondrodystrophic dwarf in a Holstein-Friesian calf.

MATERIALS AND METHODS

A six-months-old female Maine-Anjou heifer was submitted for necropsy in connection with a long-term investigation of congenital defects in cattle. Prior to euthanasia by intravenous administration of sodium pentobarbital, blood samples were collected for chromosomal studies according to the method described by Moorehead, et. al.[15] The calf underwent necropsy and section of long bones were fixed in 10% phosphate buffered neutral formalin. Bone sections were decalcified with formic acid prior to routine histological sectioning. Representa-

tive samples of internal organs and brain were also fixed in 10% buffered neutral formalin, sectioned at 6u, and stained with hematoxylin and eosin.

A neonatal male Holstein-Friesian calf was submitted for necropsy and radiological examination. Sections of the epiphyseal regions of long bones were fixed in 10% buffered neutral formalin. Bone sections were decalcified with formic acid prior to routine histological sectioning. Tissue samples of internal organs and brain were also fixed in 10% buffered neutral formalin, sectioned at 6u and stained with hematoxylin and eosin.

RESULTS

The Maine-Anjou dwarf was 6-months old. It was a stocky, compact animal with abdomen close to the ground. The animal had wide-set eyes and a short head. The heart, stomach and lungs were considered oversized for this animal. The heart weighed two kilograms. The long bones had large knobby epiphyses and the shafts were short. The vertebrae were compressed and the cranial base was markedly shorter than normal. The brain revealed internal hydrocephalus (Fig. 1). The bones revealed narrowed and irregular epiphyseal plates. Chromosomal analysis disclosed normal 60, xx karyotype typical of bovine female (Fig. 2). The neonatal Holstein-Friesian calf born from normal parents weighed 8 kilograms and died shortly after birth. The skull was domed. The long bones were reduced in length with cartilaginous epiphyses showing distortion. Both the fore and hind limbs had missing phalanges. The face was abnormally shaped and palatoschisis was evident (Fig. 3). The brain revealed internal hydrocephalus. The long bones revealed epiphysis

consisting entirely of cartilage (Fig. 4). The endocrine organs were all within normal.

Radiographic Findings: The Holstein-Friesian calf showed no ossification centers in any of the epiphyses of the long bones while the metapheses and diaphyses were radiographically normal (Fig. 5). The facial bones were hypoplastic.

DISCUSSION

Congenital defects have always been recognized but for many years inadequately studied.[14] It is important for animal breeders that records be made of new malformations not recorded previously. In this instance dwarfism reported in the imported Main-Anjou breed is of special significance to the pedigree breeder. Dwarfism is a threat to the efficient production of beef as well as to owners since even living dwarfs are worthless on the market. Several diagnostic methods have been used in the past but they are not practical enough, the most practical test would be a chemical or morphological one of the blood if it could be found.

The internal hydrocephalus observed was due to compression of the midbrain and was probably induced by the short cranial base.

The chromosomal studies revealed grossly normal karyotype number for bovine female, so if this is a hereditary condition can only be confirmed by a breeding trial.

The literature of dwarfism is summarized in Table 1.

Achondroplasia was used in 1876 to describe some fetuses with dwarf-like features.[18,19] Chondrodystrophy was proposed to replace achondroplasia in order to emphasize that the condition was due to wrong

development of the cartilage and not to its non-existence.[13]

Chondrodystrophy in calves had been reported by several authors most of them in Europe. The term "bulldog" has been applied to calves that show chondrodystrophy. Twenty-five percent of calves in Dexter herds were born with large round heads, depressed nostrils and projecting lower mandibles with extreme shortness of the limbs. The vertebral column was short as was the trunk. These calves are often aborted at six, seven, or eight months.[19,20] Evidence pointed out that the "bulldog" condition in Dexter calves is inherited.[4] The upper lip is split and swollen tongue is thrust out with compressed nose. The tail is situated high on the back, and umbilical hernia is often present. The skin hangs loosely with many folds with much subcutaneous fat. The limbs are extremely short and the digits are separated wider than normal.

In general the abnormalities are constant and similar to those of achondroplasia in man. Malfunctioning of the pitutitary gland between the second and third months of intra-uterine life was thought to cause the condition.[4] A later review did not fully support this. The Dexter gene in homozygous state resulted in a "bulldog" calf.[8]

Twelve "bulldog" calves were reported in a herd of Nganda cattle in Uganda.[3] Nganda cattle like the Dexter breed originated from crossing two breeds of cattle.[3]

A similar but less extreme condition was described in Telemark Cattle of Norway. These calves were carried full term but, with the exception of one which survived for three months, they were unable to stand and died within a few days after birth from asphyxation due to mechanical obstruction of the respiratory tract. The pituitary was abnormal. The Telemark type was proven to be recessive in its

inheritance.[22]

Achondroplasia has also been reported in Holstein cattle in Germany.[6] The occurrence of a "Dexter Monster" in offsprings of a Jersey Holstein bull was reported.[5] There was no hard palate present, the hypophysis was not found, there was only one parathyroid and the thyroid was small while thymus gland was grossly large but microscopically normal active thymus.[5] The other reports added the occurrence of "bulldog" calves in Guernseys,[19] and Ayshires.[2] Chondrodystrophy occurred in Hereford, Angus, Charolais, Shorthorn, mixed breed, Holstein, Guernsey, and Jersey calves amongst the 1,275 congenital defects documented between 1964 and 1972 in Kansas.[14]

The other reports added the occurrence of "bulldog" calves in Guernseys,[20] and Ayshires.[2]

Chondrodystrophy occurred in Hereford, Angus, Charolais, Shorthorn, mixed breed, Holstein, Guernsey, and Jersey calves amongst the 1,275 congenital defects documented between 1964 and 1972 in Kansas.[7]

Each body system is susceptible to congenital defects, some more frequently than others. Skeletal system is involved in many congenital defects of cattle.

The term "chondrodystrophy" implies a defect due to poor nutrition of cartilage. Perhaps the term "chondrodysplasia" implying defective formation of cartilage or failure of a normal physiologic event in enchondral ossification would be preferable; however, these disturbances of skeletal growth found at birth or developing through calfhood and adult have been vaguely classified throughout the literature as the chondrodystrophies.

Chondrodystrophies are a heterogeneous group differing one from another in various ways.[1-4,7,9,16-20] Many may be inappropriately named. In some, physiologic alterations that produce the defects are known. In others, little is yet known about the altered metabolism. Also a few cases of the more unusual types have been reported but it is not certain whether they represent true entities or variations and subtypes.

Chondrodystrophy is a universal problem in all breeds of cattle and is an economic problem to the American Beef Cattle industry hence there is need for strict classification of this syndrome.

SUMMARY

A 6-month-old female Maine-Anjou heifer was affected with dwarfism. It was a stocky, compact animal with abdomen close to the ground. The animal had wide-set eyes and short head. The long bone revealed short shaft with knobby epiphyses, while the vertebrae were compressed. Chromosomal cultures of peripheral lymphocytes revealed no gross abnormalities.

A neonate, Holstein-Friesian male calf was affected with chondrodystrophy. The calf which died shortly after birth had a domed-shaped skull, reduced long bones with cartilaginous epiphyses, both fore and hind legs had missing phalanges and the face was abnormally shaped while a moderate palatoschisis was evident. The brain revealed internal hydrocephalus. Radiograph and microscopic studies showed absence of epiphyseal ossification centers.

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Table 1: Dwarfism in Cattle

Types of Dwarfism	Cause	Mode of Inheritance	Gross Findings	Microscopic Findings
1) Dexter Type	Heredity	Simple dominant autosomal heterozygous character	Small size, short legs short broad heads	
2) Bull Dog Type	Heredity	Homozygosity of a dominant autosomal character	Nonviable, usually aborted, there is severe anasarca umbilical hernia	
3) Telemark Type	Heredity	Recessive	Die shortly after birth due to suffocation. Cranium is domed, hydrocephalus, brachygnathia face, cleft palate, short neck, limbs very short	
4) Short-headed Type	Heredity	Recessive		
5) Lichocephalie (long headed)	Heredity	Recessive		
6) Compact or compressed	Heredity	Dominant		
Endemic Cretinism	Thyroid deficiency			

Table 1 (continued)

Types of Dwarfism	Cause	Mode of Inheritance	Gross Findings	Microscopic Findings
Pituitary Dwarfism	Hypopituitarism resulting from compression of the gland by tumors, atrophy, or embolism			
Normal dwarf (true dwarf)	Heredity	Recessive		
Renal dwarf	Renal osteodystrophy			
Rachitic dwarf	Rickets - deficiency			

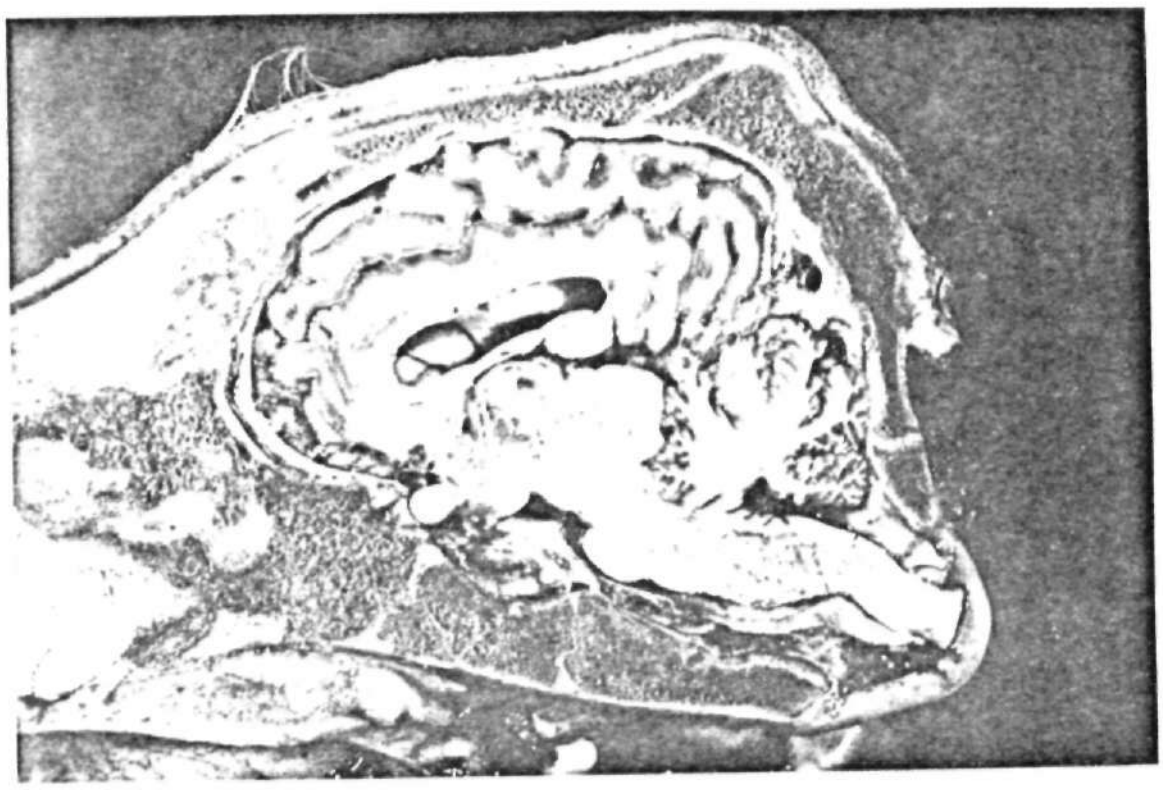


Fig. 1

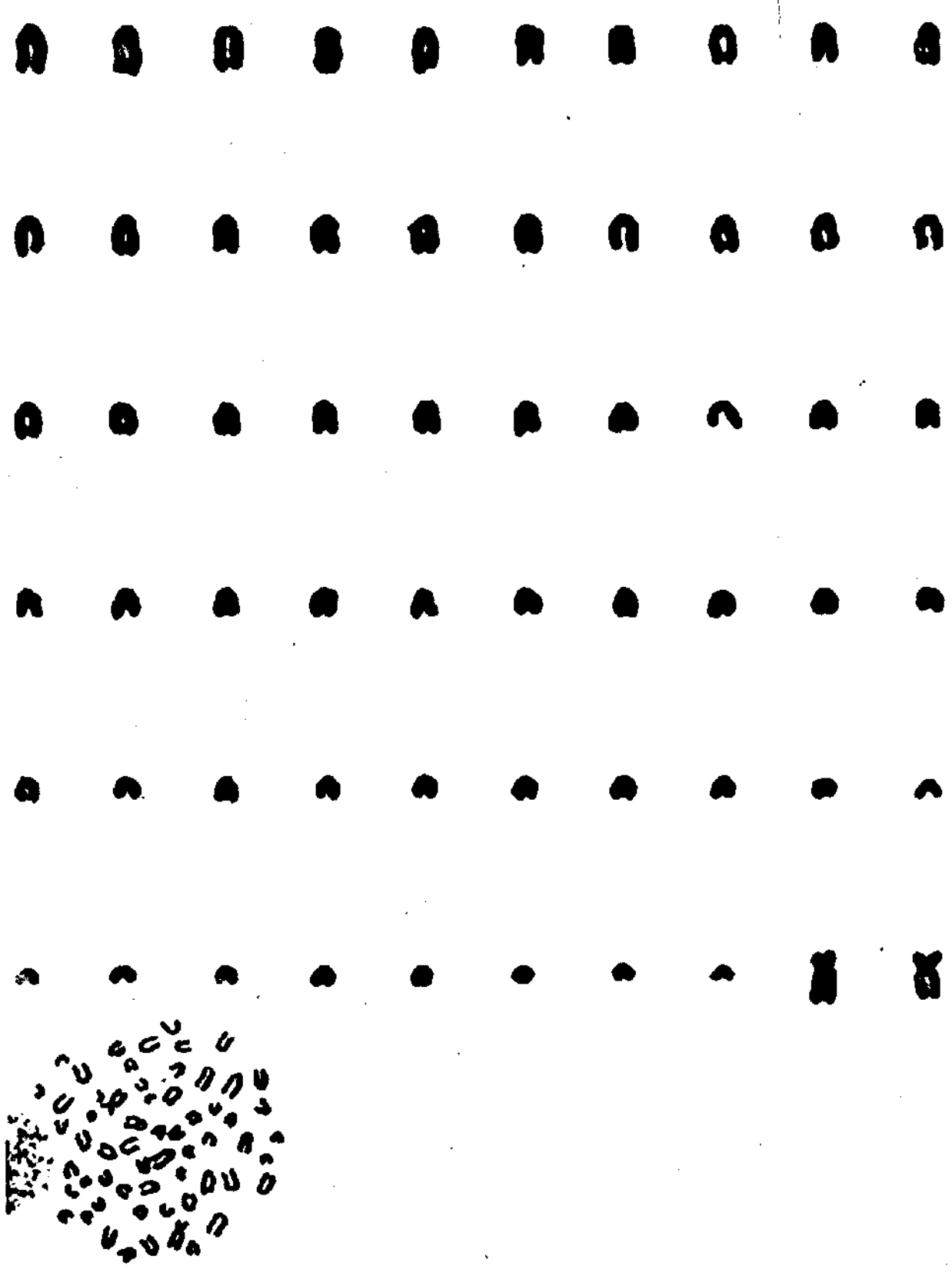


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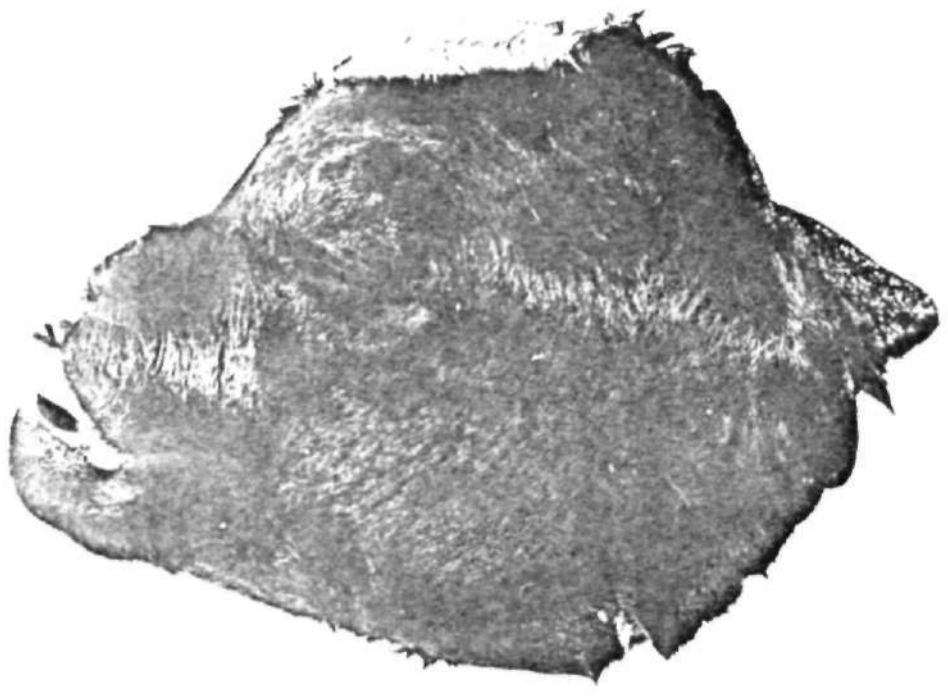


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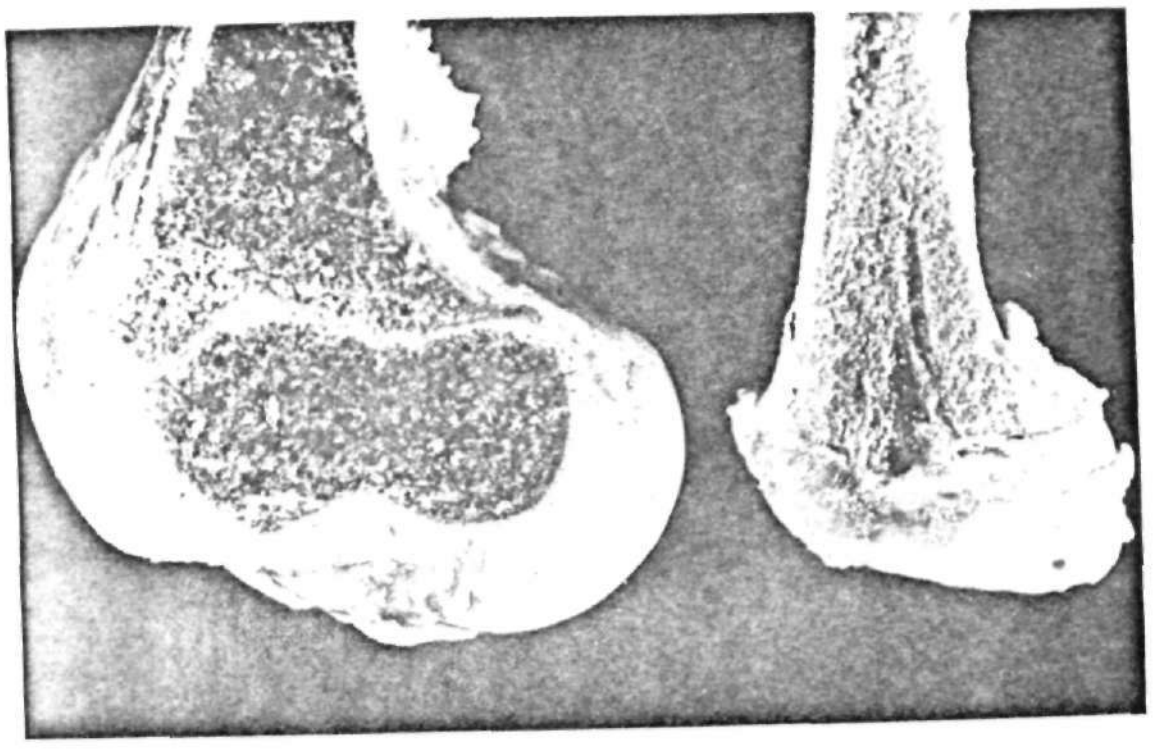


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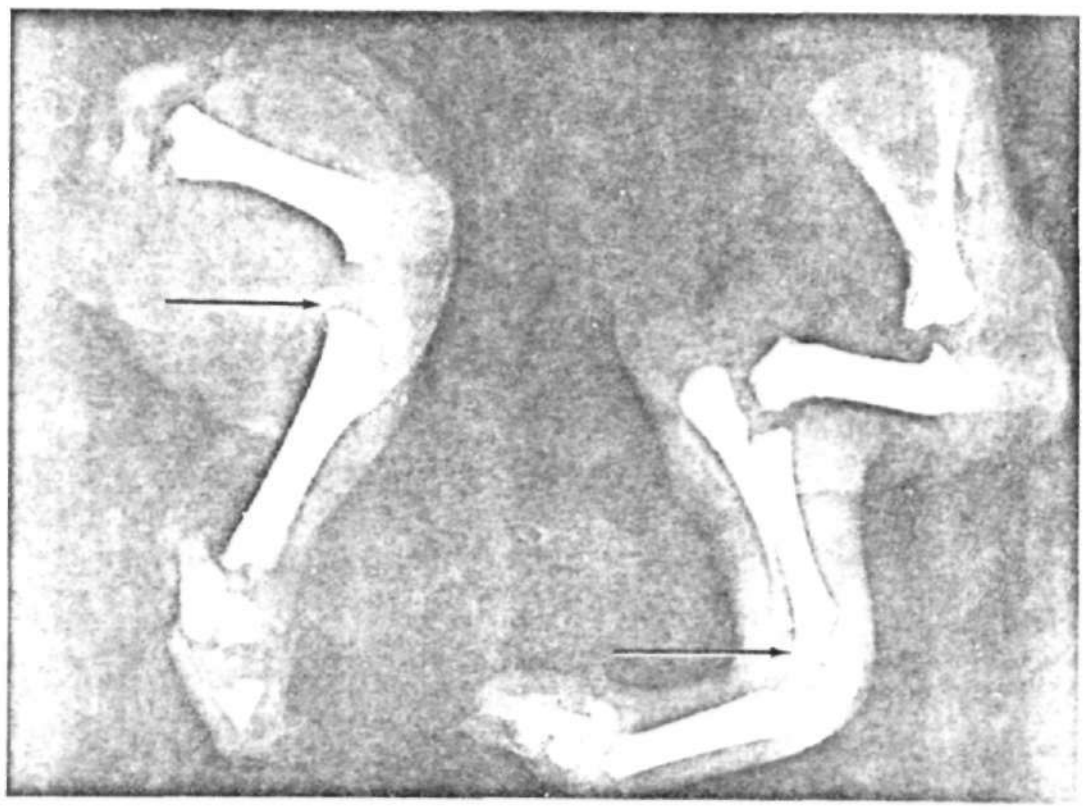


Fig. 5

PAPER 5

OSTEOPETROSIS IN HEREFORD CALVES

INTRODUCTION

Osteopetrosis, a generalized skeletal disease of man and animals is characterized by absence of bone marrow cavities due to defective bone remodeling. Congenital osteopetrosis was recently described in red and black Angus calves.[2-7] This report describes gross, microscopic, and radiographic changes of congenital osteopetrosis in Hereford calves.

MATERIALS AND METHODS

Two affected calves were received as part of a study of bovine congenital defects. Heads and legs were radiographed, then the calves underwent necropsy examination. Representative specimens of the skeleton were macerated in an autoclave, bleached with 3% hydrogen peroxide, and examined grossly. Tissue samples for histologic examination were taken from brain, liver, kidney, spleen, thyroid, and parathyroid glands, fixed in neutral buffered 10% formalin, embedded in paraffin, sectioned at 6u, then stained with H & E Stains. Longitudinal and cross sections were taken at various levels from long bones, then decalcified and stained with H & E.

RESULTS

Herd Histories -- Congenital osteopetrosis occurred in two Hereford herds in western Kansas. Four calves affected with small body size and markedly domed foreheads were born in herd 1 consisting of 35 grade Hereford cows and one purebred Hereford bull was used in the herd. The last calf was submitted for necropsy examination. The second calf submitted for necropsy examination came from a purebred Hereford herd of 120 cows. It was the only defective calf born in that

herd in 1974. The dam was a first calf heifer and she was a half sister to the bull. The purebred Hereford bull in herd 1 and heifer and sire in herd 2 were distantly related.

Pathologic Findings -- Both calves were stillborn, the male weighed 32 lbs. and the female weighed 46 lbs. Both presented immobile lower jaws which had brachygnathia inferior and markedly domed foreheads (Fig. 1). The molar teeth were not erupted and were impacted. The fontanelle was patent and had a diameter of 2.5 cm. Sagittal sections of the skull revealed small cranial cavities, with thickened cranial base and extremely thickened frontal bones which had a thickness up to 5 cm (Fig. 2). The frontal bone also had numerous cysts ranging in size from 2 millimeters to 3 cm (Fig. 2). Maceration disclosed agenesis or hypoplasia of foramina of the skull such as supraorbital, intraorbital anterior palatine, hypoglossal, ethmoidal, and external and internal acoustic meati were also hypoplastic. The longitudinal sections of ethmoid bone disclosed a thickened and dense bone. The mandibles were extremely thickened, did not articulate normally with the petrous temporal bones and lacked foramina such as mentale and the vertebrae were dense and revealed thick areas of cortical bone which enclosed two endochrondral bone cones with their bases at cranial and caudal epiphyses and joined apexes in the center of the vertebra. Longitudinal bisection of the long bones revealed lack of bone marrow cavities. Instead, the place of bone marrow cavities was occupied by cones of chondro-osseous tissue which bases were located at proximal and distal epiphyses and their apexes meeting at midshaft (Fig. 3). Some of the long bones were bent and had old fractures and calluses. The

metaphyseal areas of most long bones had fluid-filled cysts ranging up to 1 cm. in diameter (Fig. 3).

With the exception of brain, other tissues of the body were grossly normal. The gross abnormality of the brain consisted of compression of the cerebrum and coning of the cerebellum.

Histological sections taken longitudinally and transversely at various levels of the long bones disclosed the following changes. The periosteum was normal and thick lamellar concentric layers of bone made up the center cortex of the long bones. The inner aspects of the cortical bone had little or no bone resorption, however, pockets filled with loose mesenchymal tissue were numerous (Figs. 4 and 5). The inner cortex merged into the chondro-osseous tissue. The epiphyseal plates were normal. The metaphyseal areas had chondro-osseous tissue consisting of mineralized cartilagenous cores and osteoid (Fig. 6). Osteoclasts were present but appeared inactive. Blood vessels were sparse in the inner aspects of long bones. Numerous large cysts were present in metaphyseal areas of long bones. These cysts were lined by loose mesenchymal tissue and osteoclasts and contained eosinophilic material (Fig. 4). Flat bones from face and cranium were characterized by dense bone tissue lacking remodeling. However, numerous cysts with similar features to those in the long bones were present.

Thalamus, choroid plexus, periventricular areas and cerebellum had densely mineralized vessel walls and neurons (Fig. 7). Liver and spleen had numerous extra medullary foci of hematopoiesis. The thyroid and parathyroid appeared normal.

Radiological Findings -- Radiographs revealed dense medullary cavities in long bones without differentiation between cortex and

characterized by mineralized vessel walls and neurons. The solid medullary areas comprised of chondro-osseous tissue resembled early fetal steps of ossification.

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Fig. 1

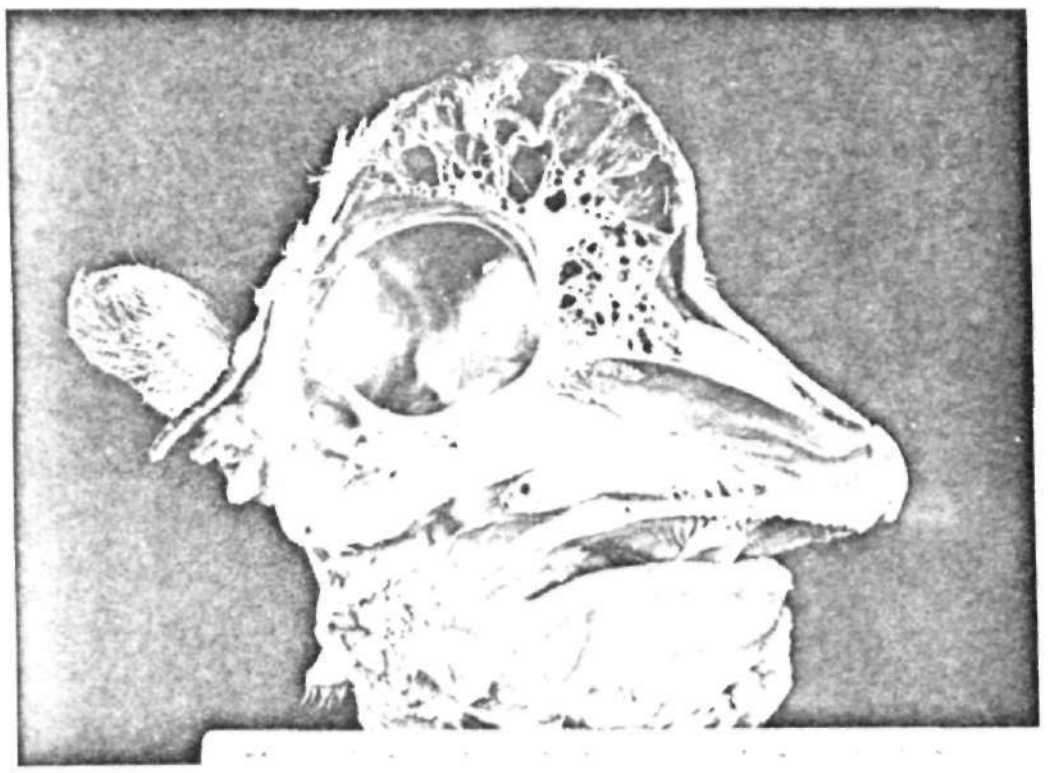


Fig. 2



Fig. 3



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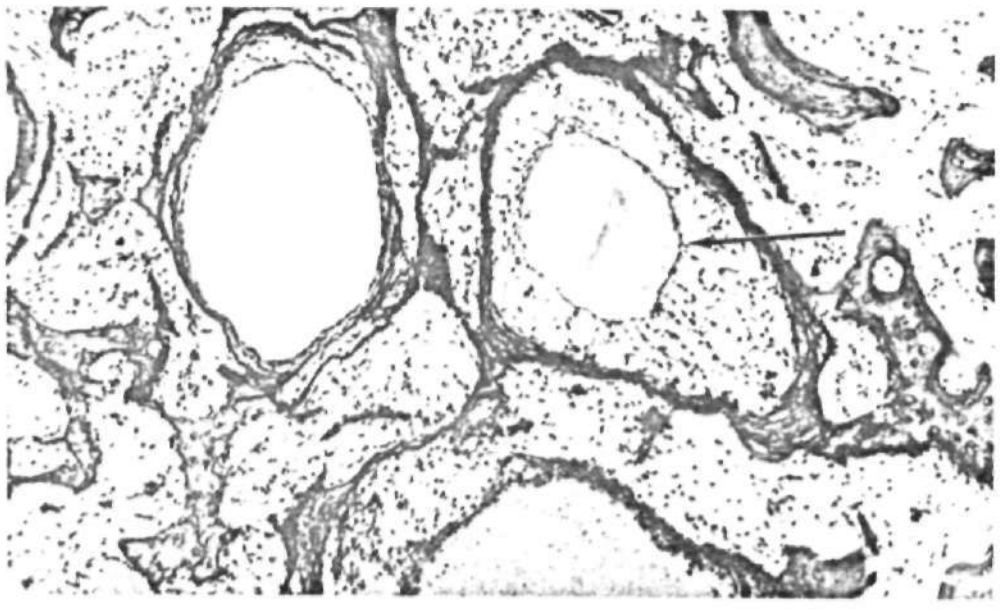


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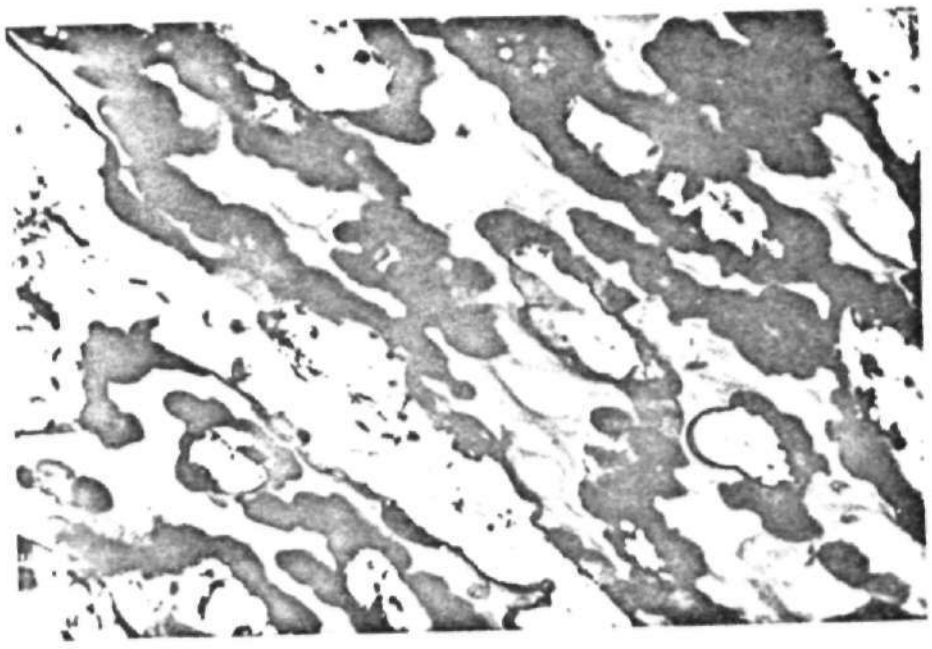


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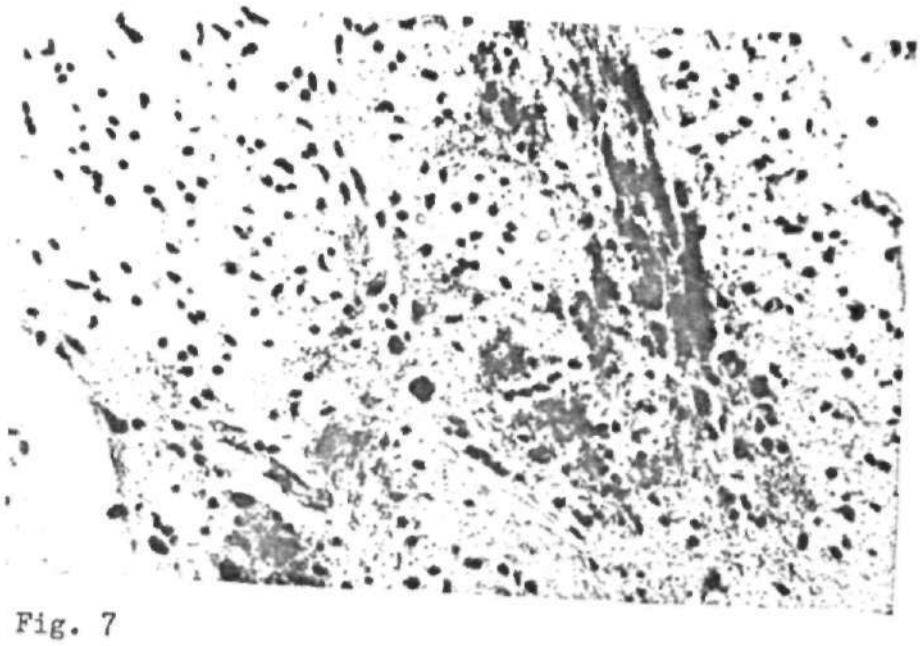


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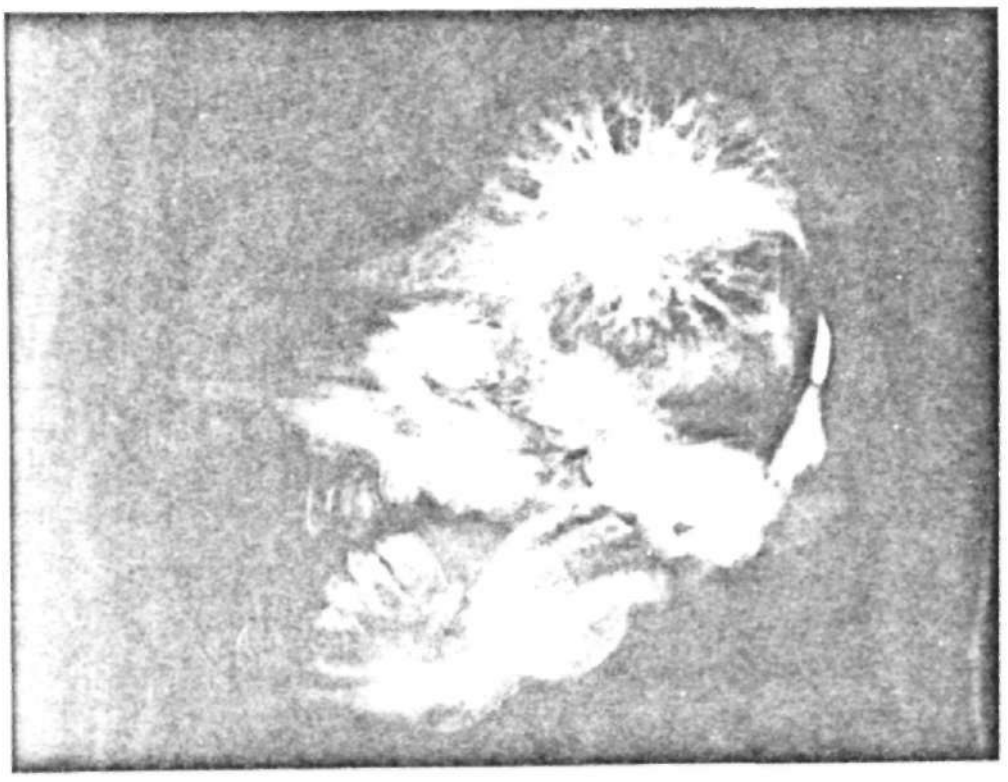


Fig. 8

PAPER 6

POLYDACTYLY IN TWO HOLSTEIN-FRIESIAN CALVES

Congenital defects of the appendicular skeleton in man are considered significant and are carefully recorded, but those in cattle, with exceptions, are sparsely documented.[1,2] Polydactyly, partial or complete duplication of digits (or digital parts) in cattle, has been reported several times, but anatomical descriptions are scarce.[3-8,10]

MATERIALS AND METHODS

A 3-month-old female Holstein-Friesian calf with polydactyly of all four legs was submitted for necropsy in connection with a long-term investigation of congenital defects in cattle. Before she was euthanized by intravenous administration of sodium pentobarbital, blood samples were collected for chromosomal studies according to the method outlined by Moorhead.[9] The calf underwent necropsy and the legs were radiographed, fixed in 10% buffered neutral formalin, and carefully dissected. Tissue samples of internal organs and brain were fixed in 10% buffered neutral formalin, sectioned at 6u, and stained with hematoxylin and eosin. The second calf, a Holstein-Friesian female calf was sent to slaughter and the 4 legs were sent to us in liquid nitrogen.

RESULTS

Calf 1

Herd History -- The calf was from clinically normal parents and the owner reported no previous history of polydactyly.

External Features -- The calf had polydactyly of all 4 legs and had difficulty in walking as indicated by skin abrasions on the dorsal region of the carpal joints of the forefeet and tarsal joints of the

hindfeet. The forelegs had 3 separate digits, but the most medial digit had a large angle to the other two and its 3rd phalanx seemed to be convexly shaped. In both hind legs there were three digits with fusion of lateral and middle digits but a dorsal median furrow indicated the separate digits (Figs. 1 & 2). There were 2 normal size dewclaws on the caudal aspect of each foot.

Radiographic Changes -- The lateral half of the distal epiphysis of the radius was much narrower than the medial half, which caused a lateral deviation of the leg distal to the carpus (Fig. 3).

Carpal bones were normal. Polydactyly involved metacarpals and phalanges. The first metacarpal was rudimentary. Second and third metacarpals were fused, and the fourth seemed to be completely separate from the second and third. One nutrient foramen supplied all the metacarpals. There were 3 complete sets of phalanges. The third and fourth digits each had 2 proximal sesamoids and 1 navicular bone. The second had only 1 proximal sesamoid and no navicular bone. No digits involved the first metacarpal (Fig. 3).

Radiographic findings from the left foreleg were the same except the left's second digit had 2 proximal sesamoids and no navicular bone (Fig. 4).

The right hindleg had its 2nd and 3rd metatarsals fused and the 4th metatarsal appeared to be partially fused at about midshaft (Fig. 5). The distal end of the second and third metatarsals were completely fused with an abnormal distal epiphyseal plate. The distal articular surface had a degenerative lesion involving the medial abaxial surface that appeared to cause osteoarthritis involving both it and the proximal end of the first phalanx of the second digit. The second

digit lacked both proximal and distal sesamoids. The fourth metatarsal did not fuse with the third. Except for the first phalanx (described for both the second and third digits, Fig. 5), the phalanges were normal in all digits.

The 3 metatarsal bones fused in the central portion of the left rear leg. On the distal end there was a separate fourth metatarsal and a fused second and third. The distal end of the second and third metatarsals were similar to those in the right leg (Fig. 6).

Dissection -- Arrangements of the extensor and flexor muscles in both forelegs and hindlegs were similar. The muscles were normal but the 3 extensor tendons and the superficial and deep flexor tendons were not fused in the region of the toe (as in syndactyly) but inserted at normal places except that the long digital extensor tendon branched and inserted normally on the extra digit. The medial tendon supplied only the extra digit while the lateral tendon supplied the fused digits. The flexor tendons each branched to supply the extra digit.

Chromosomal Studies -- Metaphases of lymphocytes revealed a normal bovine female karyotype of 60, XX.

Dissection of Calf 2

External Features -- The forelegs had 3 digits each with fusion of lateral and middle digits but a dorsal median furrow indicated the separate digits. The most medial digits had a large angle to the other two and its 3rd phalanx seemed to be convexly shaped. The lateral dewclaw was missing in each of the affected forelegs. Both hindlegs appeared normal.

Radiographic Findings -- Polydactyly of the forelegs involved

proximally the metacarpals and distally the phalanges. The second and third metacarpals seemed fused at their distal extremities but the fourth was observed to be completely separated from the second and third. There were 3 complete sets of phalanges. The third and fourth digits each had two proximal Sesamoids and 1 navicular bone. The second had no visible proximal Sesamoid nor navicular bone.

Dissection -- The lateral digital extensor tendon supply solely the extra digit while the long digital extensor tendon branched to supply the "middle" and medial digits. The medial digital extensor tendon inserted normally on the medial digit. Both the superficial and deep digital flexor tendons each branched to supply the extra digit.

DISCUSSION

Congenital defects of the appendicular skeleton are sparsely documented in cattle except for a few well-known syndromes.[3] Polydactyly, the duplication of digital parts, has been described in various breeds[4,5,6,7] and is attributed to an autosomal dominant gene with incomplete penetrance in French Black Pied cattle.[7] Differentiation within the mosaic of the post gastrula embryo is rapid, which is a useful morphogenetic concept to explain such limb malformations as polydactyly.[2]

The cases reported here add anatomical knowledge to bovine polydactyly.

Congenital defects may be caused by environmental or genetic factors or by interactions between them. No causative factor was evident in the calf described.

Identifying congenital defects is important to veterinarians and

herdsmen so they may establish causes and institute preventive measures. Many congenital defects are known, but others undoubtedly are still unrecognized.

SUMMARY

Two female Holstein-Friesian calves were affected with polydactyly of both forefeet, while the hind feed of the 1st calf exhibited polysyndactyly. Both calves had normal parents. Karyotypes prepared from peripheral blood revealed no gross abnormalities.

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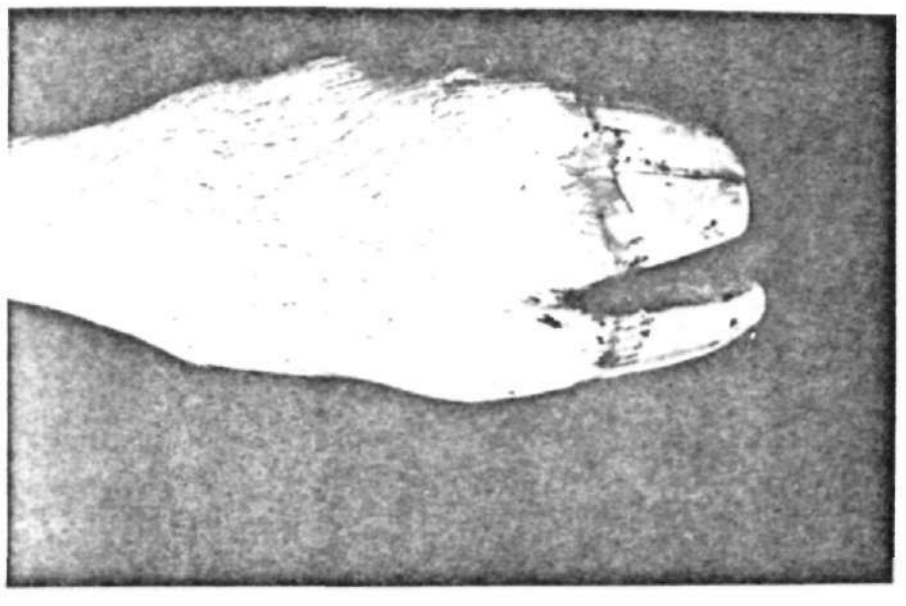


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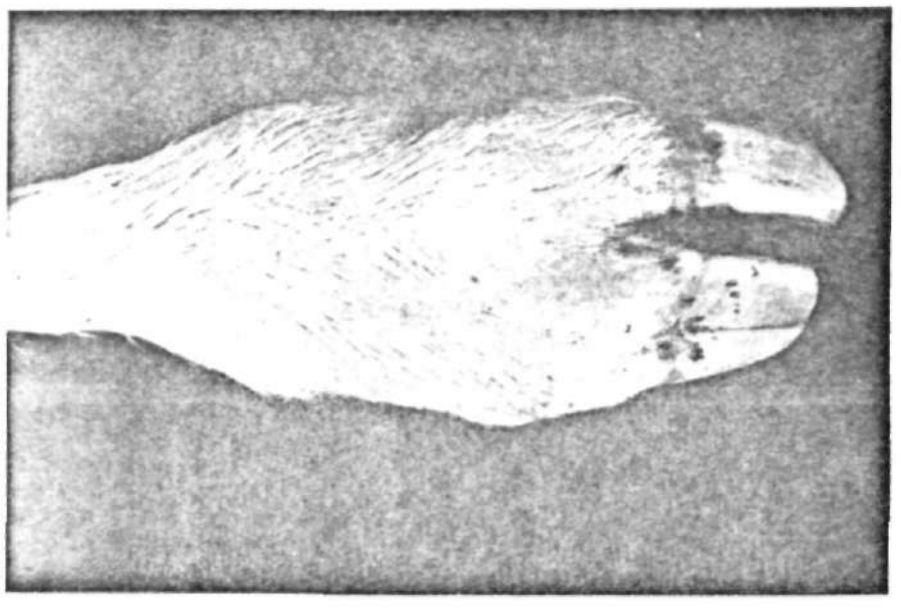


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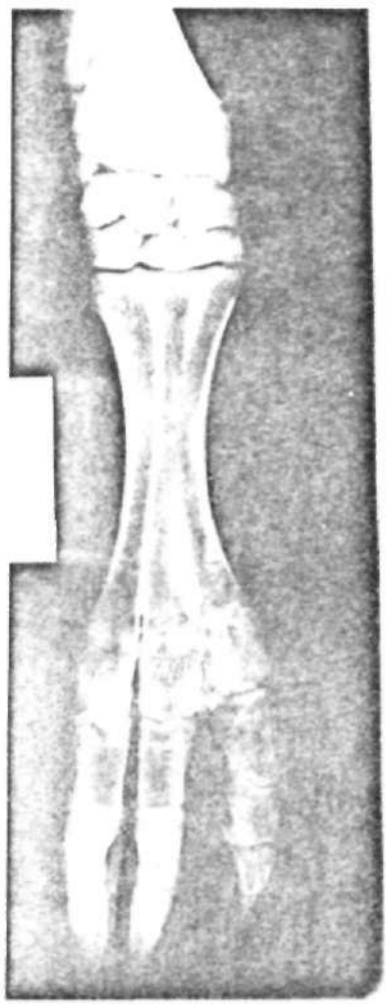


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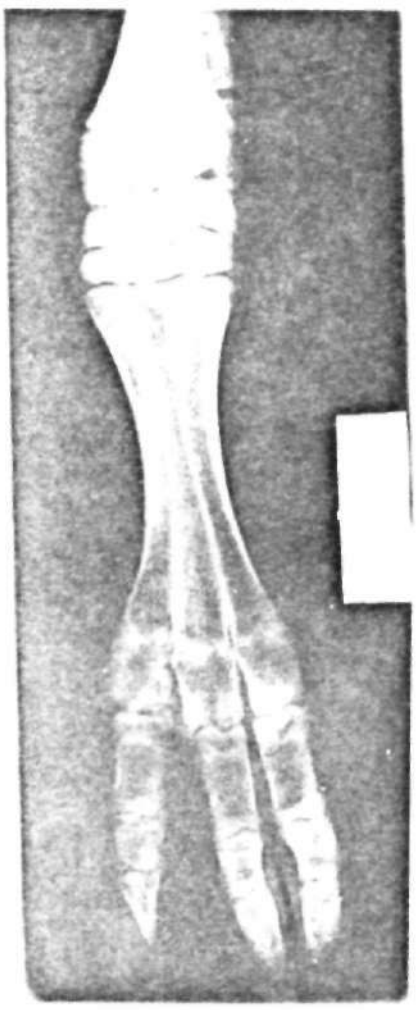


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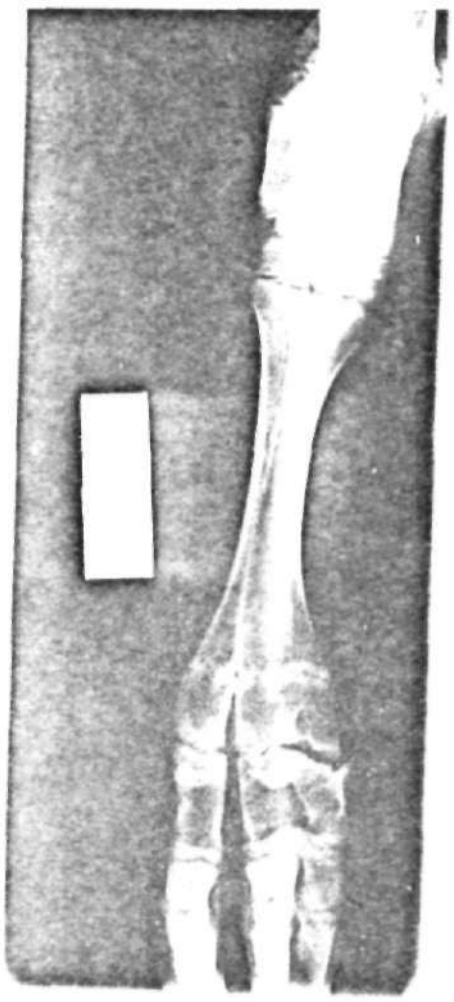


Fig. 5

PAPER 7

CAMPTODACTYLY IN A SIMMENTAL CALF

INTRODUCTION

Camptodactyly, permanent and irreducible flexion of one or more digits, is well documented in man.[1] A literature search revealed no record of camptodactyly in cattle.[2] This report describes camptodactyly associated with bilateral hypoplastic kidneys in a neonatal Simmental female calf.

MATERIALS AND METHODS

The deformed neonatal Simmental calf was submitted for necropsy as part of a continuing study of bovine congenital defects. All four legs were radiographed. Tissues were collected at necropsy from central nervous system, kidney and leg muscles. The muscles and tendons of the two hind legs were carefully dissected. After dissection, the legs were macerated.

RESULTS

Gross Anatomical Findings: -- Medial digits of both hindfeet deviated from longitudinal axis and had contracture (Figs. 1 & 2).

Radiographic Changes: -- The outstanding radiologic features in the calf includes the following: Both front legs had metacarpals lacking fusion at the midshaft (Fig. 3). There are no proximal or distal sesamoids in either forelegs (Fig. 3).

The tarsal bones were quite abnormal with unusually shaped fibular tarsal and tibial tarsal bones with an extra center of ossification located at the dorsal limits of the tibial tarsal. The metatarsals were not fused at any point. The phalanges of the 3rd digit had abnormal alignment possibly due to contracture of the tendons of this digit. No proximal sesamoids for either the medial or lateral

digits were observed (Fig. 4).

Histopathology Findings: -- The central nervous system and muscle tissues were within normal. The kidneys were hypoplastic and contained numerous oxalate crystals.

Dissection: -- The superficial and deep digital flexor tendons were shorter than normal.

DISCUSSION

Rare congenital defects, like the common ones, may have hereditary origins or may be caused by environment or may reflect a combination of both. However, because of their rarity and because genealogic records are needed to prove hereditary involvement usually are not available or often are inadequate, their etiologic factors likely will be revealed gradually after many years of painstaking collection of details about each isolated occurrence. Fortunately, rarity of these congenital defects causes them to be of modest economic importance, except when they resemble a more common defect about which more is known and for which there is usually more concern, especially if it is hereditary.

SUMMARY

Camptodactyly, permanent and irreducible flexion of one or more digits, was diagnosed on both hind feet of a neonatal Simmental calf. Abnormal formation of the tarsal bones, nonfusion of the 3rd and 4th metatarsals, absence of proximal and distal sesamoids and contracted tendons involving the 3rd phalanges were associated features in the defect. In addition, there was bilateral hypoplasia of the kidneys. The cause of the defect was not determined.

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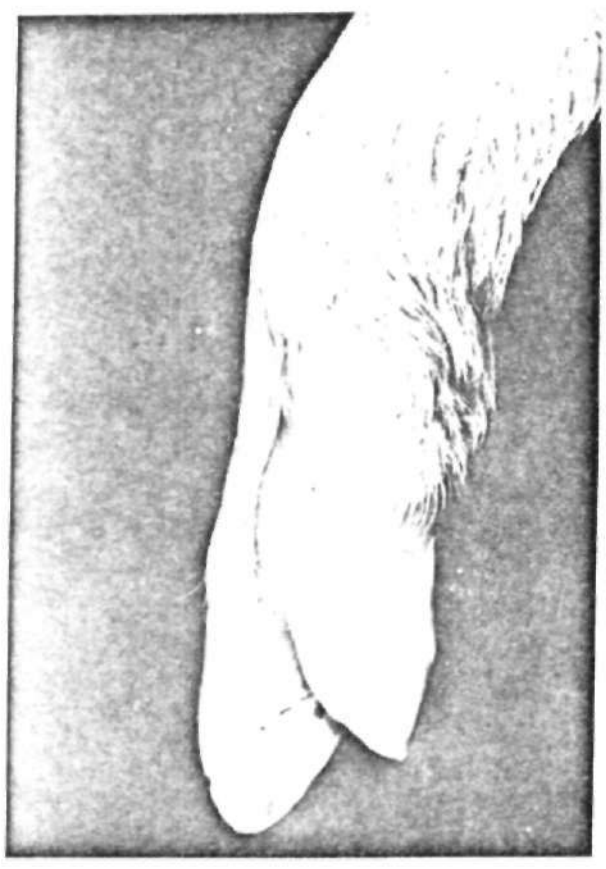


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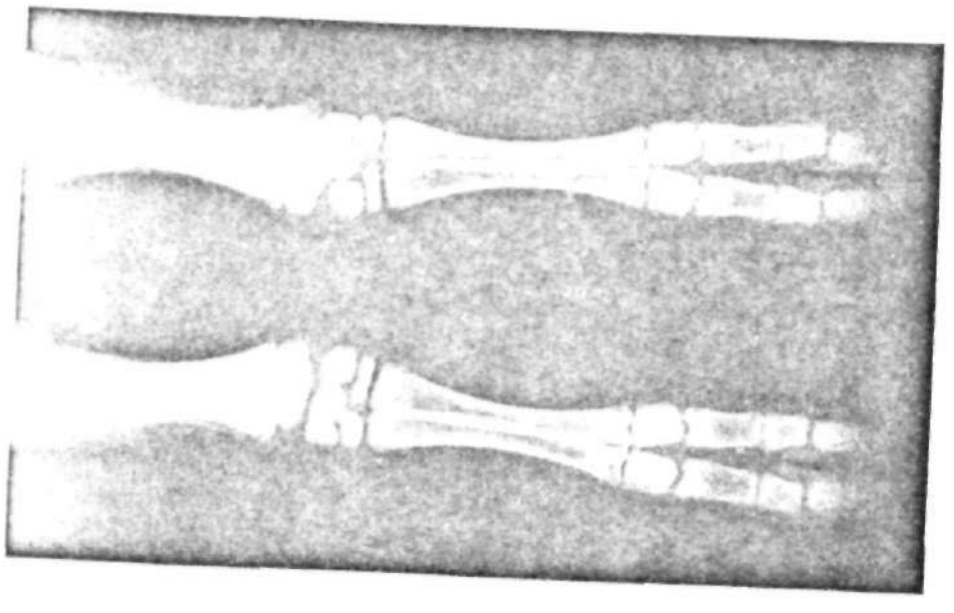


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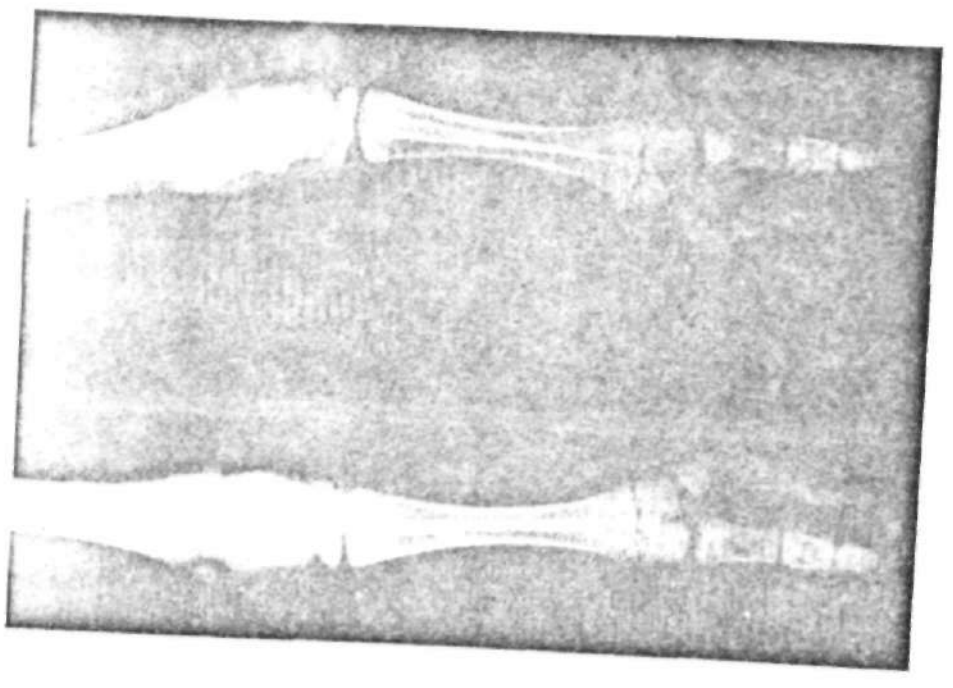


Fig. 4

PAPER 8

TIBIAL HEMIMELIA IN GALLOWAY CALVES

Congenital skeletal defects may affect the entire skeleton or localized skeletal regions and may be single, multiple, or associated with congenital defects of other organs to constitute a syndrome. Such a syndrome consisting of bilateral tibial hemimelia (literally, half a hindlimb), cryptorchidism, and ventral abdominal hernia was described in a calf in Scotland more than two decades ago.[8] This report adds the gross and microscopic changes, and genetic data in seven affected Galloway calves and genetic data for two other similarly affected Galloway calves.

MATERIALS AND METHODS

Seven deformed neonatal Galloway calves were submitted for necropsy as part of a continuing study of bovine congenital defects. Tissue specimens were collected at necropsy from central nervous system, skeletal, muscle, and internal organs, including those of the endocrine system. Radiographs were taken of the hind limbs and pelvis. The muscles of the hind limbs were carefully dissected.

Herd and breeding histories were obtained by personal visits and by mail.

RESULTS

Herd Histories -- Tibial hemimelia occurred in 3 herds, 1 in Kansas, 1 in Wyoming, and 1 in Texas. Herd 1 had 6 affected calves in fall of 1973 and spring of 1974, 5 of which were submitted for necropsy (calves 1-5, Table 1), the first record of abortion or congenital defects in this herd. Herd history concerning vaccination, nutritional deficiency, disease, or exposure to toxic plants was inconclusive. Four affected female calves (IX_1 to IX_4, Fig. 1) were

from dams that were half sibs ($VIII_1$ to $VIII_4$, Fig. 1). Bull A sired 4 calves, Bull B sired 2 affected calves (XI_{27} and XI_{28}, Fig. 1). Bulls A and B and dams of affected calves were related to Bull C (Fig. 1). Herd 2 in Wyoming, one affected calf was submitted (calf 6, Table 1) but no further history was available. Herd 3 from Texas had 2 affected calves, one of which was necropsied (calf 7, Table 1), and its sire and dam were related to Bull C in Herd 1.

The defect affected both sexes, there were 4 females and 3 males. Five calves were born alive and full term, but died within 24 hours of birth. Two were born dead and full term.

Radiologic Changes -- The outstanding radiologic feature in 5 calves was bilateral agenesis of the tibia and the symphysis of the pelvis was not closed (Fig. 2). In addition, there was bilateral agenesis of the patella. There were variable remnants of fibula present. Two calves had closed pelvic symphysis and had no patellas and short tibias.

Pathologic Changes -- The gross pathological changes are listed in Table 1. In addition, the following observations were made. All calves had ventral abdominal hernia (Fig. 3). None of the calves had a patella. The femurs of all 5 calves affected with agenesis of the tibias had one trochlea which was round and without subdivisions for accommodation of patella or tibia. The fibula in these calves was 3 to 4 cm. long and between 0.5 to 1 cm. wide. The fibula was attached to the distal end of the femur and to the talus by short and taut ligaments (Fig. 4). The talus had no trochlea. The remaining bones of the hind leg were normal.

Muscles that normally insert on the tibia inserted instead on

the femur. Muscles that originate at the tibia originated at the femur instead. Meningoceles were present in six calves (Fig. 5). Internal hydrocephalus was present in six calves (Fig. 6). The hydrocephalus in calves 1, 2, 3, 4, 5, and 7 was caused by a prolapsed vermis. The mesencephalic aqueduct was abnormally dilated in 6 calves. There were no additional microscopic lesions.

DISCUSSION

Congenital skeletal defects may involve the entire skeleton as in dwarfism and osteopetrosis, or a single bone structure, as in syndactyly. The latter defects may occur as single isolated defects but they frequently are associated with congenital defects of other body regions.[2,3,5,6]

The congenital disease described here is a syndrome consisting of brain, skeletal and reproductive system defects (Table 1). All these factors are easily recognized in the field.

Herd histories and occurrence of the defect in relatives confirm the genetic basis for tibial hemimelia.

A search of the literature revealed a single calf of unspecified breed but a photograph suggested a Galloway.[8]

The fact that this anomaly has only been seen in one breed of cattle suggests a hereditary basis and the occurrence in Herd 1 (Fig. 1) suggests autosomal recessive inheritance. We are conducting a breeding trial to verify that hypothesis.

The genealogy (Fig. 1) is a method used to find the common source of a simple autosomal recessive mutant gene. All parents of affected calves are heterozygotes, receiving the mutant gene from a

common ancestor, in this case from parents in Europe unless an identical mutation occurred in the United States, a highly unlikely event. A 9-generation pedigree analysis of 6 affected calves from Herd 1 was performed. Only one animal, Bull C (Fig. 1), was found to be common to all 12 parents of affected calves. Bull C was calved in 1947 in Scotland and appears in the American Galloway Herd Book with both American and Scottish registration numbers.

The 6 affected calves in one herd in one breeding season represents a serious economic loss. The previous report[7] helped us to classify our findings and make recommendations to the owners, which confirms the importance of documenting isolated cases. It remains to be seen if tibial hemimelia is found in Galloway cattle abroad.

A fascinating aspect of this defect is its localized effect. Congenital absence of distal extremities in humans associated with bony scalp defects.[1,4] The striking feature and point of difference in the calves of our report is the congenital absence of the tibia and patella with bone both proximal and distal to it within normal limits. A possible explanation of this defect is offered by an embryonic neuropathy due to a neural crest injury.[7]

SUMMARY

Tibial hemimelia is part of a congenital syndrome that may include agenesis of patella, tibia, nonclosure of the pelvic symphysis, cranioschisis, meningocele, internal hydrocephalus, cryptorchidism, or nonfusion of Mullerian ducts was found in 7 Galloway calves. This congenital defect is most likely inherited as a simple, autosomal recessive.

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Table 1. Defects Associated with Tibial Hemimelia in 7 Galloway Calves

Calf No.	Identification in Fig. 1	Sex	Reproductive System	Central Nervous System			Skeletal System		
				Cranioschisis	Meningocele	Internal Hydrocephalus	Tibia Right	Tibia Left	Pelvic Symphysis
1	IX_1	Female	Nonfusion of Mullerian ducts	0.5 cm frontal bone	2.0 cm	Moderate	Agenesis	Agenesis	Open
2	IX_2	Female	Nonfusion of Mullerian ducts	0.5 cm frontal bone	2.0 cm	Moderate	Shortened	Shortened	Closed
3	IX_3	Female	Nonfusion of Mullerian ducts	0.5 cm frontal bone	3.0 cm	Moderate	Agenesis	Agenesis	Open
4	IX_4	Female	Nonfusion of Mullerian ducts	0.5 cm frontal bone	2.0 cm	Moderate	Agenesis	Agenesis	Open
5	IX_{28}	Male	Bilateral Cryptorchidism	2, each 0.3 cm frontal bone	2, each 1.0 cm	Marked	Agenesis	Agenesis	Open
6	Not Shown	Male	Bilateral Cryptorchidism	None	None	None	Agenesis	Agenesis	Open
7	Not Shown	Male	Bilateral Cryptorchidism	0.25 cm frontal bone	0.25 cm	Marked	Agenesis	Agenesis	Open

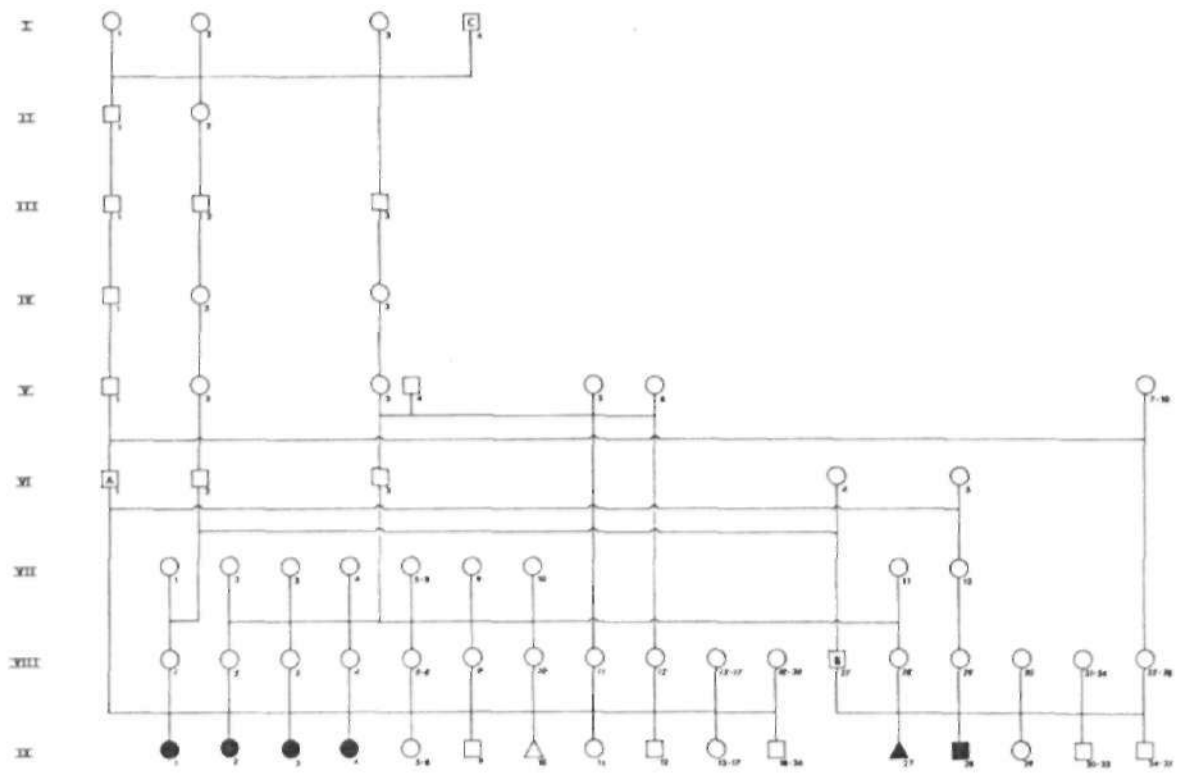


Fig. 1

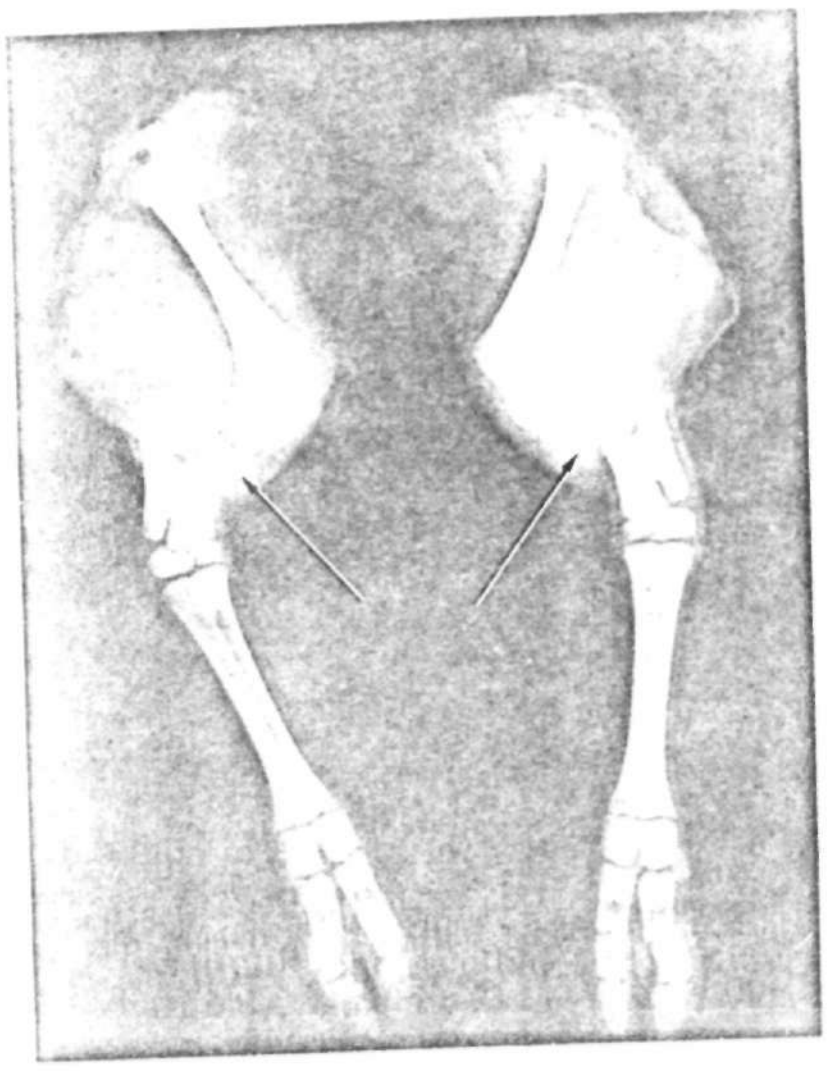


Fig. 2

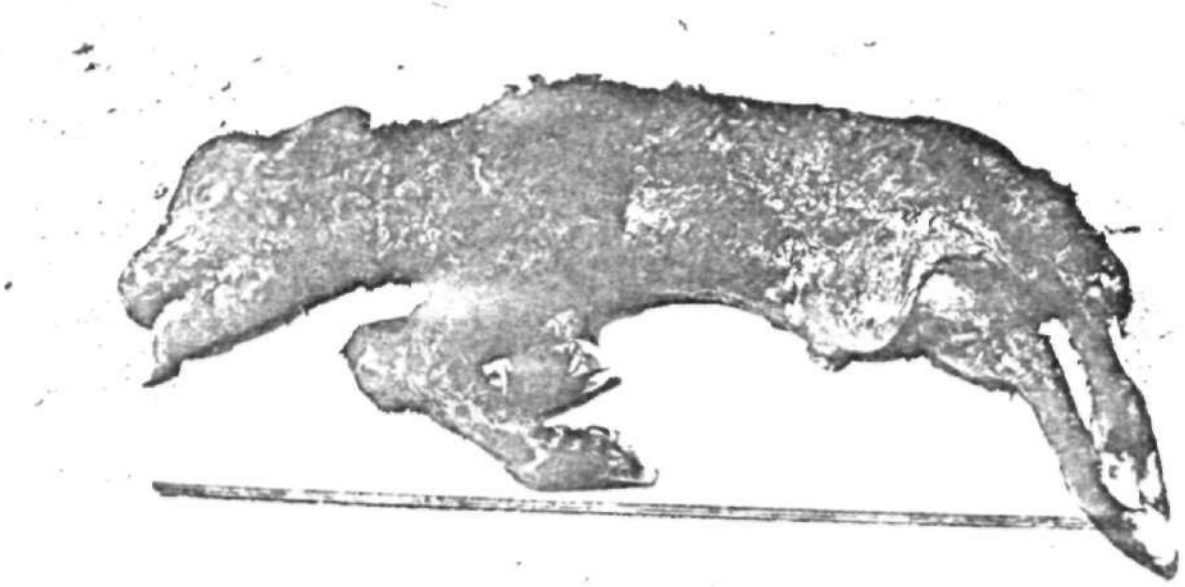


Fig. 3

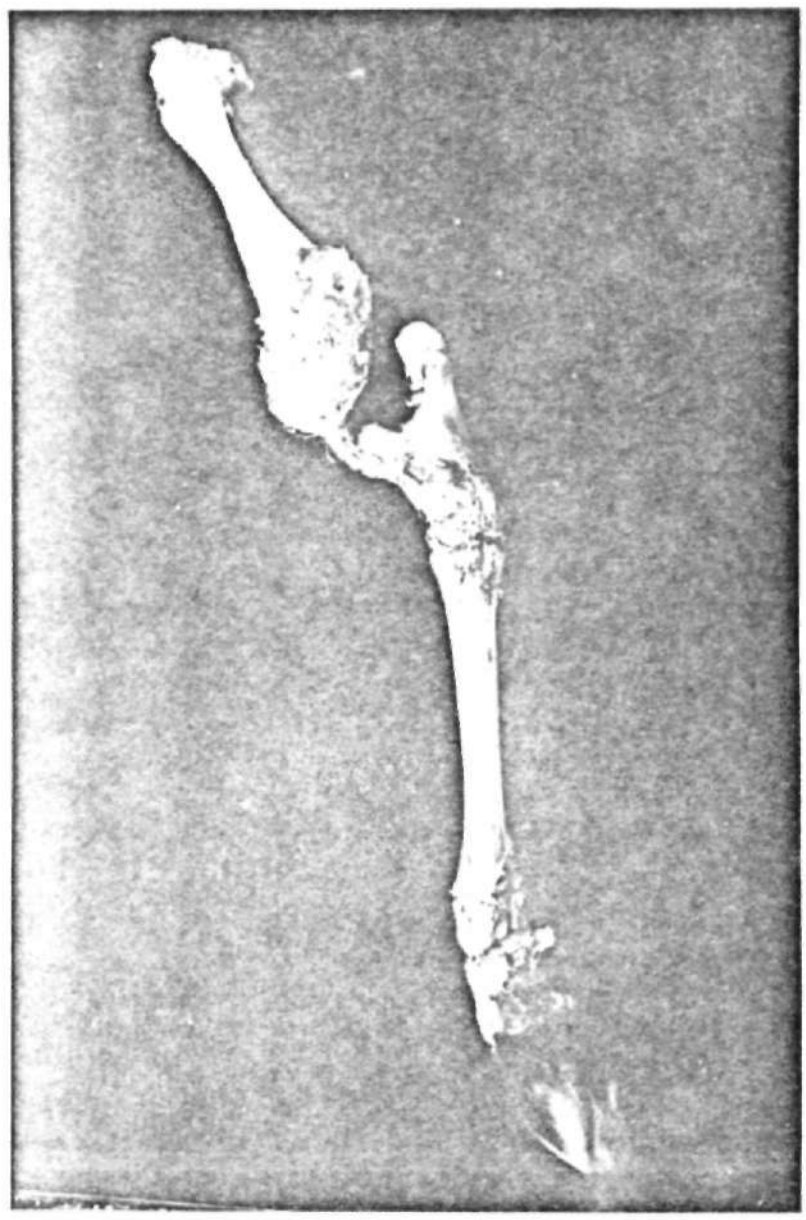


Fig. 4



Fig. 5

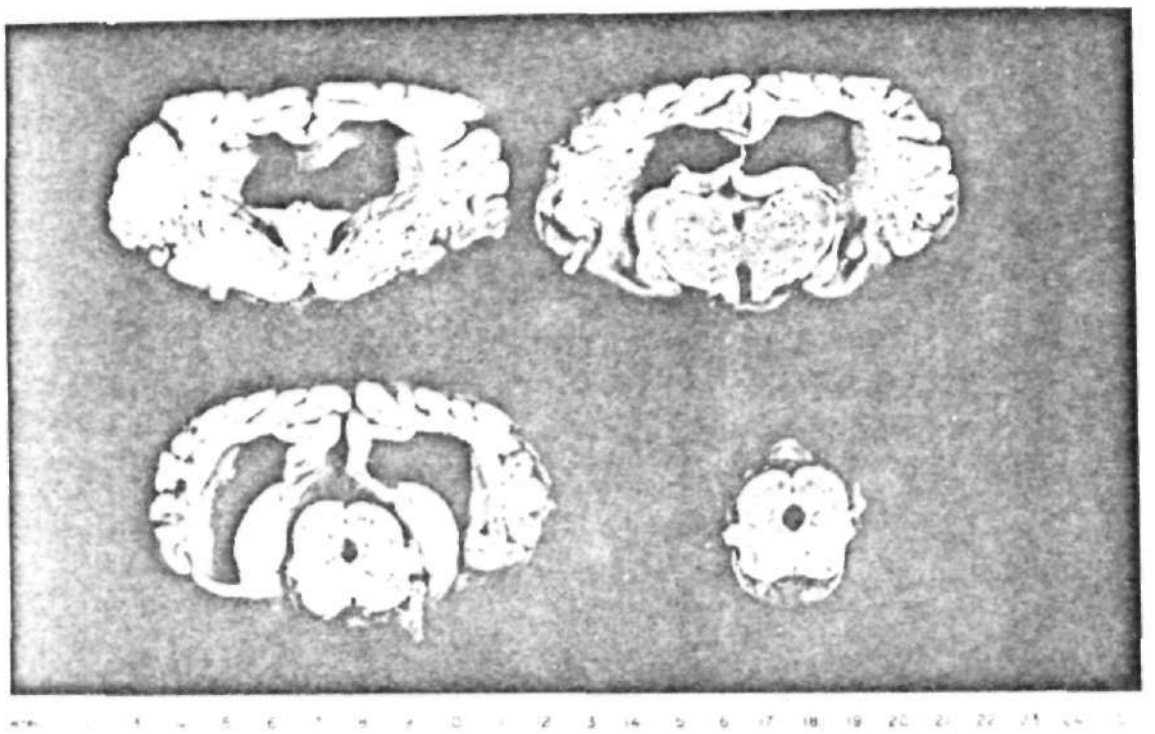


Fig. 6

PAPER 9

FACIAL DIGITAL SYNDROME IN ANGUS CALVES

INTRODUCTION

Congenital skeletal defects may affect the entire skeleton or localized skeletal regions and may be single, multiple, or associated with congenital defects of other organs to constitute a syndrome.[2] This report describes such syndrome in Angus calves comprised of skeletal and central nervous system defects.

MATERIALS AND METHODS

Two deformed neonatal Angus calves, from a herd in southwestern Kansas, were submitted to the Department of Pathology, Kansas State University, for necropsy as part of a continuing study of bovine congenital defects. Heads and legs were radiographed. At necropsy, tissue samples for histopathological examination were collected from central nervous, skeletal and muscualr system. The muscles of the legs were dissected, and the head and appendicular skeleton were macerated for osteological studies. Herd and breeding histories were obtained by personal visits.

RESULTS

Herd Histories -- This syndrome occurred in the fall of 1973 in a purebred Angus herd in southwest Kansas. Two affected calves, a male and a female, were born, died within 3 hours of birth and were submitted for necropsy (Table 1, VII-1, and VII-2, Fig. 1). Two other heifers in the same groups of cattle aborted at the fifth and sixth months of pregnancy, but their fetuses were not submitted for diagnostic examination. Herd history on vaccination, nutrition, disease, or exposure to toxic plants was inconclusive. The two affected calves (male and a female) were from half sisters bred (VI-2 and VI-3) to bull

VI-1 (Fig. 1). This bull was bred in this herd to 57 cows and heifers (including 7 half sisters shown in Fig. 1). Additionally, 6 more half sisters of V-3 had been inseminated by another bull. Except for the two affected calves with this syndrome, all calves in the herd in fall of 1973 were normal.

Radiologic Changes -- The outstanding radiologic feature in the 2 calves was the fusion of the metacarpal bones with single distal epiphysis. The front legs had only 2 horizontally fused phalanges for each syndactylous digit, with the 2nd phalanges missing. The metatarsal bones of the hind legs were fused with a single distal epiphysis. The digits had single 1st, 2nd, and 3rd phalanx in both hind legs quite typical of complete syndactly (Fig. 2). The skull had marked shortening of the maxillary and nasal bones so that the anterior limit of the nasal bones was dorsal to the 1st premolar within the mandible. The mandible and cranial vault were normal.

Pathologic Changes -- Both calves were affected with facial hypoplasia and tetrameric syndactyly (Figs. 3 to 5). The gross pathological changes are listed in Table 1. In addition, the following observations were made. The calves had no distinct mouth. The site was filled with the tongue which can be seen for a good length uncovered. The eyes seem slightly smaller than normal. The brains had no olfactory tracts and revealed also marked internal hydrocephalus. The histopathological observations included the absence of central canal, tilted ventral median fissure and abnormally shaped dorsal in the spinal cord and ventral spinal horns, all of which constitute spinal dysraphism.

The three extensor tendons in both fore and hind legs were fused to a single aponeurotic-like sheath in the region of the toe.

The superficial and deep flexor tendons fused at the area of their insertion. The muscles were within normal histologically.

Underdevelopment of interdigital vessels and nerves were also observed.

DISCUSSION

Many congenital defects can be explained as being caused by a simple pattern of Mendelian inheritance. Others are caused by environmental factors and still others have no clearly established cause. Various parts of the body may be affected with a congenital defect. Some may be single isolated defects, however, it becomes increasingly obvious, that many defects occur together as a part of a syndrome. The frequencies that various parts of the body may be affected may vary according to geographical region, breeds and other factors.[2] The congenital disease described here is a syndrome consisting of central nervous and skeletal defects (Table 1).

Herd history and occurrence of the defect in related calves point to a genetic basis for the syndrome. The fact that this anomaly has only been seen in one breed of cattle suggests a hereditary basis and the occurrence in the herd (Fig. 1) suggest autosomal recessive lethal inheritance. A breeding experiment is needed to verify this hypothesis.

The genealogy (Fig. 1) is a method used to find the common source of a simple autosomal recessive mutant gene. The parents of affected calves are heterozygotes, receiving the mutant gene from a common ancestor.

A fascinating aspect of this defect is its peculiar type of

Syndactyly which is different from the one extensively reported in Holstein-Friesian and also that reported in another purebred Angus.[3] The features described in this report closely parallel Apert's syndrome (a type of acrocephalo-syndactyly) in man.[1]

SUMMARY

Facial-digital syndrome that may include syndactyly of the legs, short face, one nasal opening, kyphoscoliosis, and internal hydrocephalus and spinal dysraphism was found in 2 Angus calves. This congenital defect is most likely inherited as a simple, autosomal lethal recessive.

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Table 1. Facial-Digital Syndrome in Two Angus Calves

Born in a Southwestern Kansas Herd, Fall 1973

Case	Breed of Calf	Sex of Calf	Breed of Dam	Breed of Sire	Central Nervous System	Skeletal System Face	Feet Affected RF	LF	RR	LR	Remarks
1	Angus	Female	Angus	Angus	Spinal dysraphism hydrocephalus	Hypoplastic face, one blind ending nasal opening	sy	sy	sy	sy	Kyphoscoliosis and contracture legs
2	Angus	Male	Angus	Angus	Spinal dysraphism hydrocephalus	Hypoplastic face, one blind ending nasal opening	sy	sy	sy	sy	Kyphoscoliosis 10 cm. inguinal hernia covered with peritoneum

KEY

sy = syndactylous

N = normal

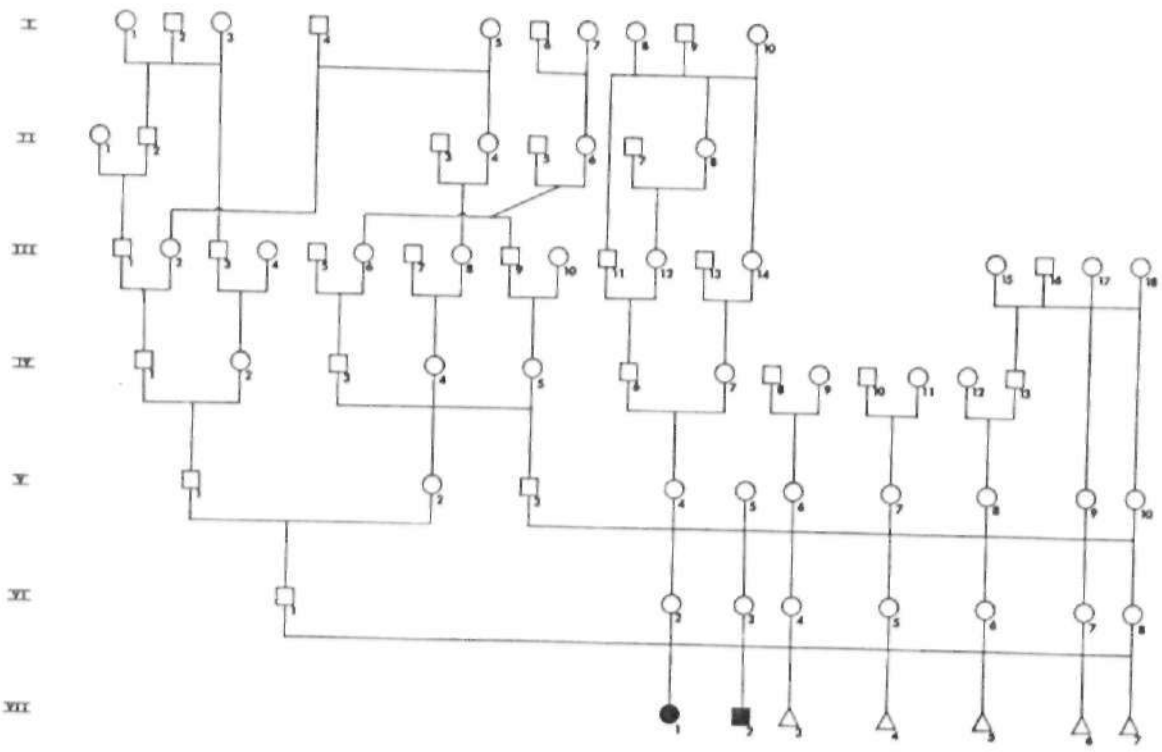


Fig. 1

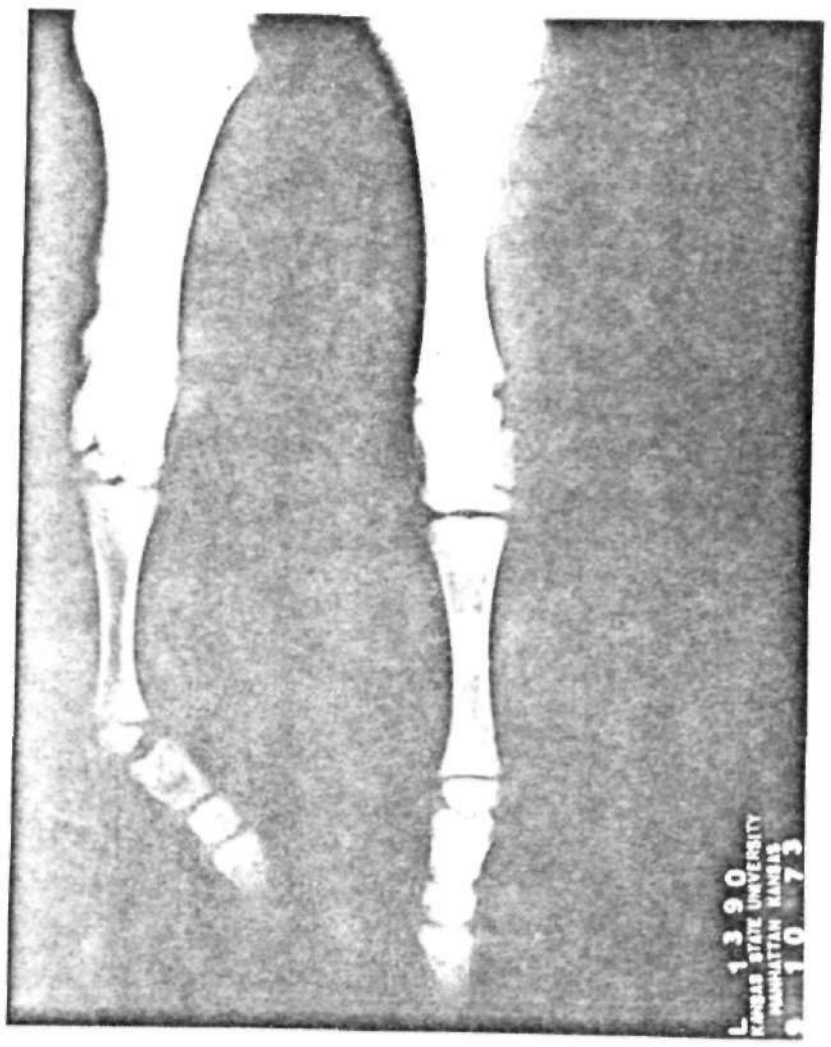


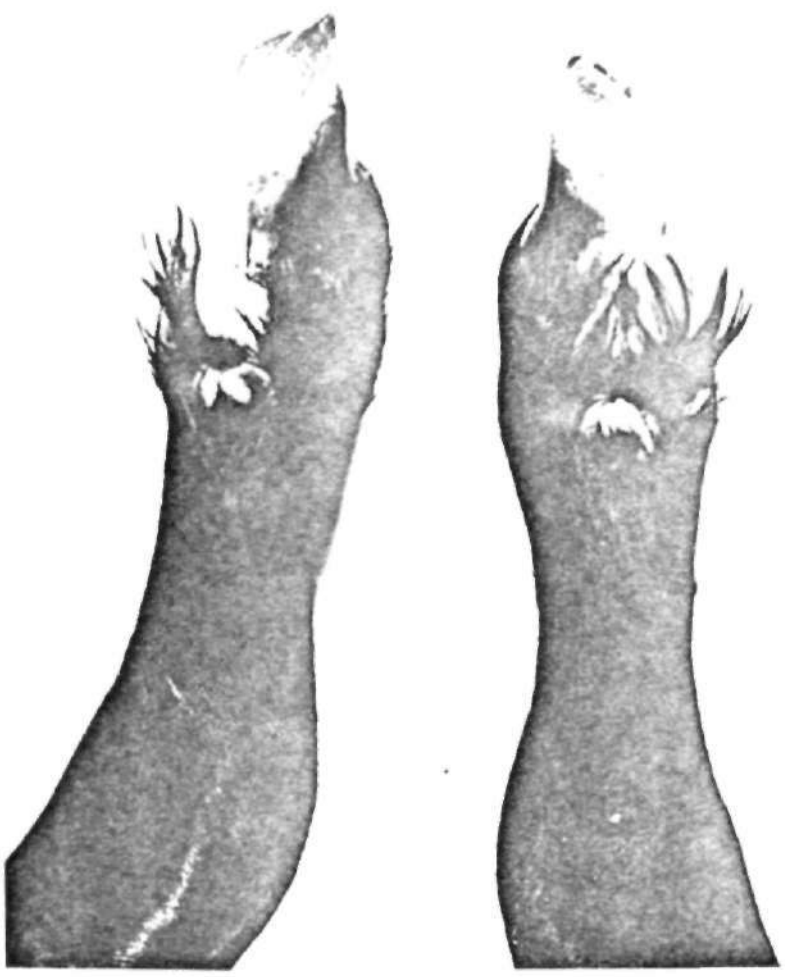
Fig. 2



Fig. 3



Fig. 4



PAPER 10

SYNDACTYLY IN CATTLE

INTRODUCTION

Syndactyly, the partial or complete fusion of functional digits involving one or more feet, has been described in several breeds of cattle, including Simmental, Indian Hariana cattle, German Red Pied, a Japanese native breed, Aberdeen Angus, and American Holstein-Friesians.[1-5,8] The best-known and most common type is a recessive hereditary syndactyly described in American Holstein-Friesian cattle.[5]

Syndactyly in cattle has been reported from many countries; Austria, Czechoslovakia, France, Germany, Japan, India, Italy, New Zealand, and the United States as reviewed recently.[5]

This report describes syndactyly in purebred Hereford, purebred Holstein, and crossbred Chianina calves.

MATERIALS AND METHODS

Nine syndactylous calves were submitted for necropsy as part of a continuing study of bovine congenital defects.

Herd and breeding histories were obtained by personal visits and by mail. Peripheral blood lymphocyte cultures were used (a modification of Moorhead's method)[7] to determine the karyotype. The legs of all calves were radiographed. Legs of 4 calves were removed after euthanasia then fixed in 10% buffered neutral formalin and later dissected; muscle tissues were taken for histopathology. The dissected legs later were macerated for osteological studies.

RESULTS

History -- Between spring 1973 and fall 1974, nine neonatal calves affected with syndactyly of one or more feet were observed (Table 1). Herd histories were as follows: Calves 1 and 2 were

observed in two Holstein herds in New Mexico. Both were bred artificially by 2 different Holstein sires. The Hereford calf originated in a purebred Hereford herd in Nebraska, no other information was available. Calves 4 to 9 are crossbred Chianina calves (Table 1; Fig. 1) sired by 2 related Chianina bulls. The 2 Chianina bulls (II-6 and II-7 in Fig. 1) have a common paternal grandsire. The first Chianina bull (II-6 in Fig. 1) sired 2 calves in 2 different herds in Iowa. The first one, calf number 4 in Table 1 (IV - 1 in Fig. 1) was from a grade Holstein dam. The second one, a bull calf was from a grade Angus cow (IV-2 in Fig. 1).

The second Chianina bull sired 4 syndactylous offspring (Calves 6 to 9 in Table 1, IV-3, IV-4, IV-18, and IV-19 in Fig. 1). Two syndactylous calves were encountered in 1 herd in Nebraska (IV-3 and IV-4 in Fig. 1). The remaining 65 cows in this beef herd, inseminated with semen of 3 Charolais bulls and an unrelated Chianina bull, all had normal calves. Calf number 8 (IV-18, Fig. 1) was encountered in Missouri in a commercial herd of 34 grade mixed beef cattle and 1 grade Holstein cow. Only the Holstein cow had been inseminated with semen from bull III-4. The last calf (IV-19 in Fig. 1) occurred in a herd in North Dakota. The dam was a crossbred Charolais Angus cow.

Clinical and Radiographic Findings -- Calves 4 to 9 underwent examination of their karyotypes. All 6 calves had normal karyotypes. All 9 calves were examined clinically and radiographically. The external features of the syndactylous hooves were as follows: All the affected hooves had the appearance of a truncated cone with the base at the coronary seam (Figs. 2-6). Three of affected calves had a front foot incompletely syndactylous (Fig. 2). The lateral dewclaws

were enlarged in all syndactylous legs of 8 calves (Fig. 6). Calf seven had a subdivided lateral dewclaw on both front feet (Fig. 7). All calves walked slowly with a stepping gait. Those with 4 syndactylous feet walked with considerable difficulty.

The fusion or nondivision of the phalanges and other osteological deviations determined from radiographs of the front feet are listed in Table 2. Typical radiographs are shown in Figures 8 to 13. In addition, the 2 calves with syndactyly of all 4 legs had the right metacarpal about 1.25 cm. shorter than the left. The hind feet of calves 6 and 8 had completely fused phalanges.

Dissection of Syndactylous Legs -- The legs of 4 calves underwent dissection (calves 1 to 3, and 5 in Table 1). The muscles and tendons adapted to the syndactylous defect as follows: In the fetlock region the three digital extensor tendons formed a wide aponeurotic place inserted on phalanx II and III and from each side received a branch from the deep suspensory ligament; the superficial and deep flexor tendons usually remained undivided and were normally inserted. The superficial, digital, flexor tendon often sent a branch to the lateral dewclaw. The vascular supply of the syndactylous legs also adapted to the malformation by the dorsal metacarpal artery terminating at the fetlock. The distal perforating metacarpal artery was absent from the severely fused syndactylous legs. The single digit was supplied by a lateral and a medial volar digital artery. The common volar digital artery was usually rudimentary, and veins were similar. The two main digital nerves of the fused foretoes were formed by the median nerve and the main nerve supply to the hind toes arose from the lateral and medial plantar nerve.

DISCUSSION

Two types of syndactyly were noted in cattle. An uncommon type, in Angus cattle, involved vertical synosteosis of phalanges.[3] A common type, occurring in U. S. Holstein-Friesian cattle, is characterized by horizontal synosteosis of phalanges.[4,5] In the cases reported here the right forefeet always were syndactylous. Two of 9 calves had all 4 feet syndactylous, which is rare. Syndactyly in purebred Herefords had been reported only once before.[5] Cases 4 to 9 reported here involved crossbreds (Holstein/Chianina and Angus/Holstein). Both had synosteosis patterns as well as muscle and tendon arrangements similar to that seen in syndactylous Holstein-Friesian cattle, strongly suggesting an allelic gene in these breeds with the cause a hereditary autosomal recessive. We are seeking more information in breeding trials.

In addition, syndactylous Holstein-Friesian cattle have a shorter than normal life expectancy. Frequently, these cattle died from heat stress.[6] It remains to be demonstrated if these exotic breeds affected with syndactyly do have similar heat susceptibility.

The economic importance of the condition is stressed by the fact that new breeds frequently are started from few importations.[2] In addition, many of these bulls may be related accentuating the possibility of transmitting autosomal recessive traits. Congenital defects such as syndactyly may reach large numbers unless heterozygous bulls are located early and removed from stud service and are labeled as heterozygous as had been done in this case.

SUMMARY

Syndactyly, the partial or complete fusion of functional digits

on one or more feet, as described in a purebred Hereford and in crossbred Holstein/Chianina and Angus/Chianina were similar by external muscle, and tendon arrangements, while osteologic features were like much commoner hereditary form occurring in U. S. Holstein-Friesian cattle.

The syndactyly in these crossbreds is most likely inherited as a simple, autosomal recessive.

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Table 1. Syndactyly in Nine Calves

Calf	Breed	Sex	Breed of Dam	Breed of Sire	Feet Affected* RF	LF	RH	LH
1	Holstein	Female	Holstein	Holstein	sy**	sy	Normal	Normal
2	Holstein	Male	Holstein	Holstein	sy	sy	Normal	Normal
3	Hereford	Male	Hereford	Hereford	sy	sy	Normal	Normal
4	Crossbred	Female	Holstein	Chianina***	sy	Normal	Normal	Normal
5	Crossbred	Male	Angus	Chianina	sy	sy	Normal	sy
6	Crossbred	Female	Angus x Holstein	Chianina	sy	sy	sy	sy
7	Crossbred	Male	Angus x Holstein	Chianina	sy	sy	Normal	Normal
8	Crossbred	Male	Holstein	Chianina	sy	sy	sy	sy
9	Crossbred	Female	Angus x Charolais	Chianina	sy	sy	Normal	Normal

RF* = Right front
LF = Left front
RH = Right hind
LH = Left hind
sy** = syndactylous
*** Calves 4 and 5 were sired by bull II-6, Figure 1
Calves 6 to 9 were sired by bull II-7, Figure 1

Table 2. Radiological Findings in Forefeet of 9 Syndactylous Calves

Calf	Carpal Joints RF	Carpal Joints LF	Metacarpus RF	Metacarpus LF	Small Metacarpals RF	Small Metacarpals LF	Proximal Sesamoids RF	Proximal Sesamoids LF	Phalanges I RF	Phalanges I LF	Phalanges II RF	Phalanges II LF	Phalanges III RF	Phalanges III LF
1	N	N	1T	2T	N	N	2	3	F	S	F	F	F	F
2	N	N	1T	2T	E	N	3	4	F	S	F	S	F	S
3	N	N	1T	2T	N	N	3	4	F	S	F	S	S	S
4	N	N	1T	2T	N	N	3	4	F	N	F	N	F	N
5	N	N	1T	1T	E	E	2	2	F	F	F	F	F	F
6	N	N	1T	1T	E	E	2	2	F	F	F	F	F	F
7	N	N	2T	2T	N	N	2	2	F	F	F	F	F	F
8	N	N	1T	1T	E	E	2	2	F	F	F	F	F	F
9	N	N	1T	1T	N	N	3	3	F	F	F	F	F	F

N = normal
T = trochlea
F = fusion
S = separate
E = enlarged

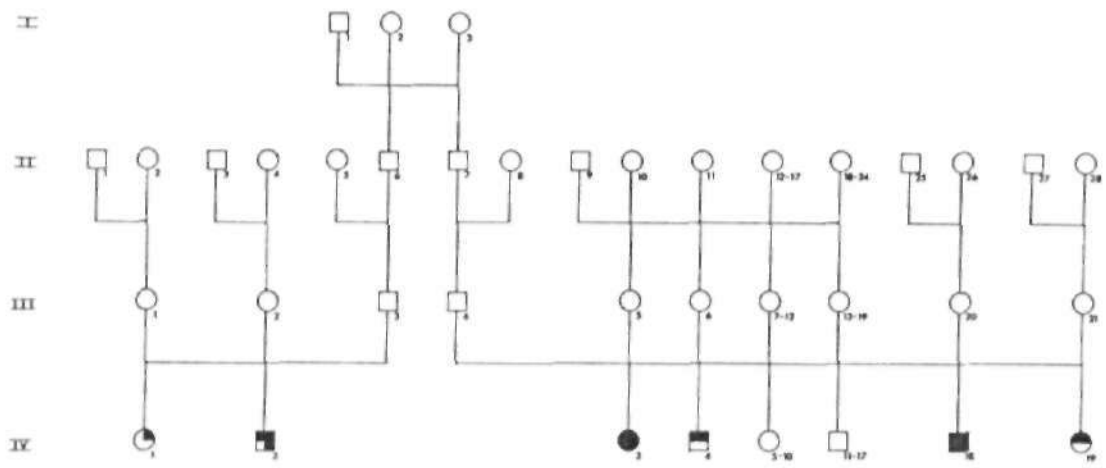


Fig. 1

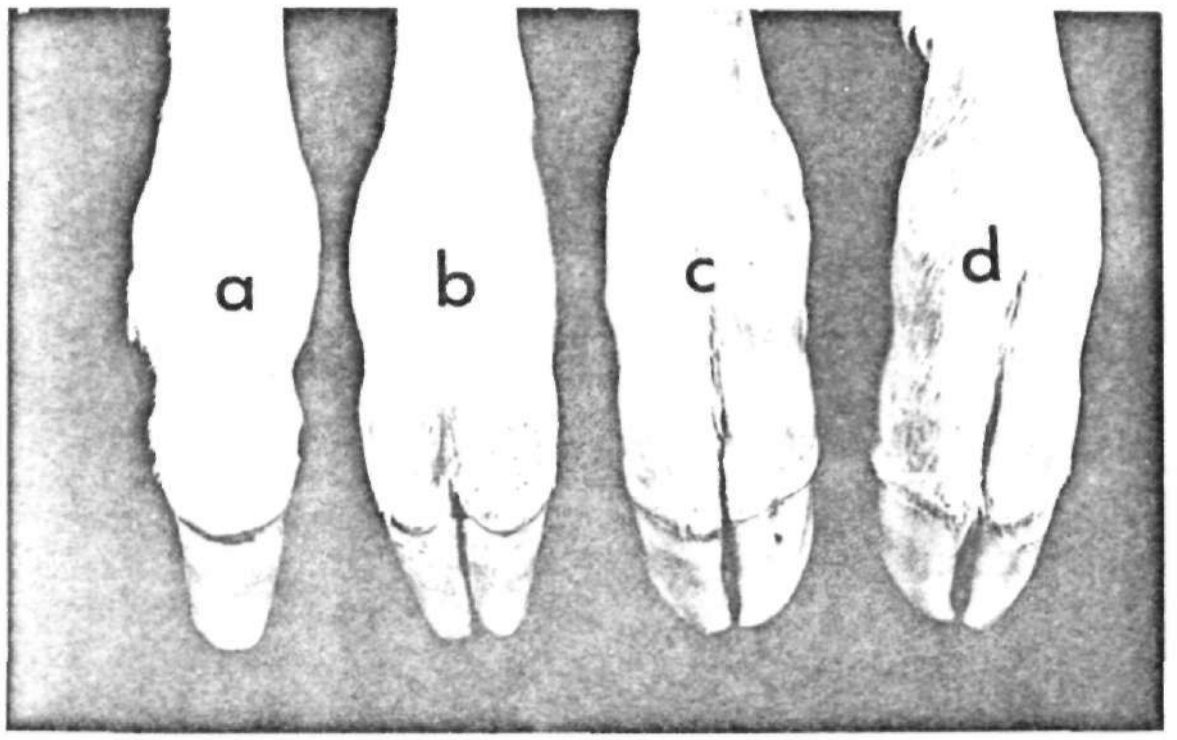


Fig. 2



Fig. 4



Fig. 3

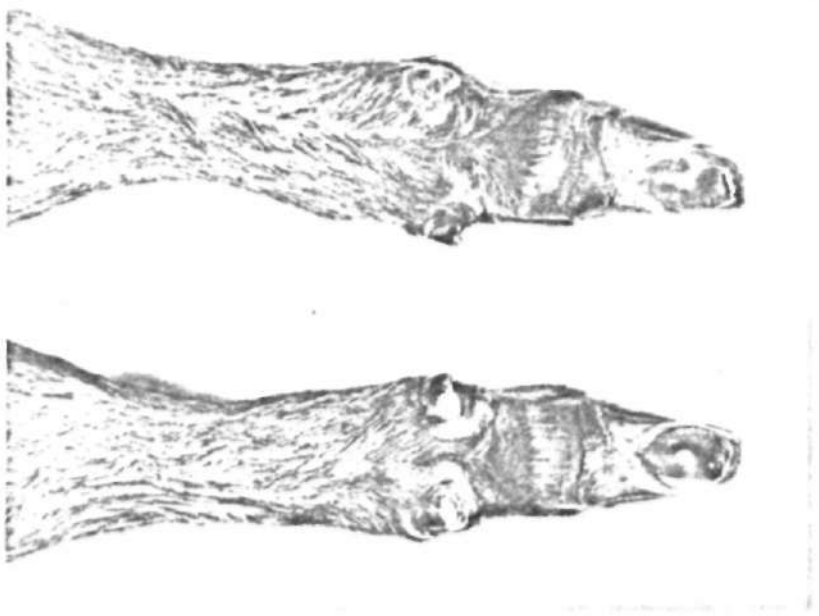


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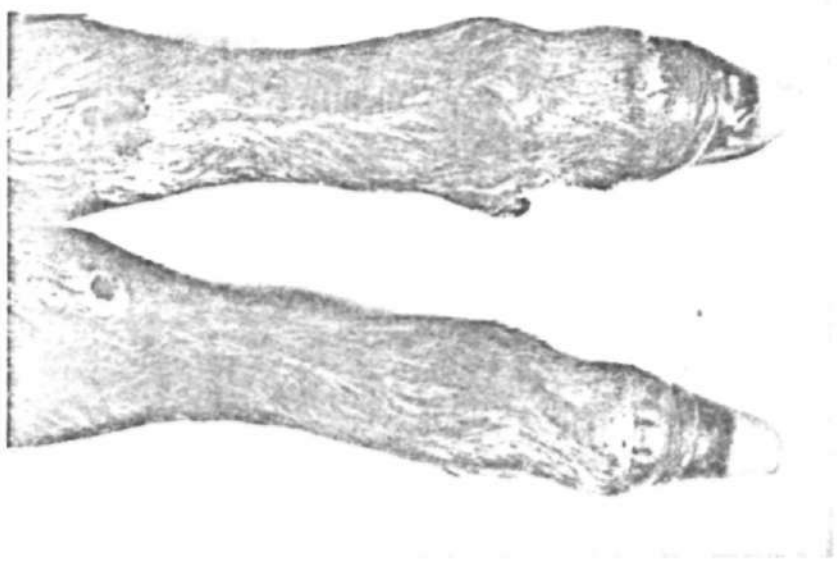


Fig. 5



Fig. 7

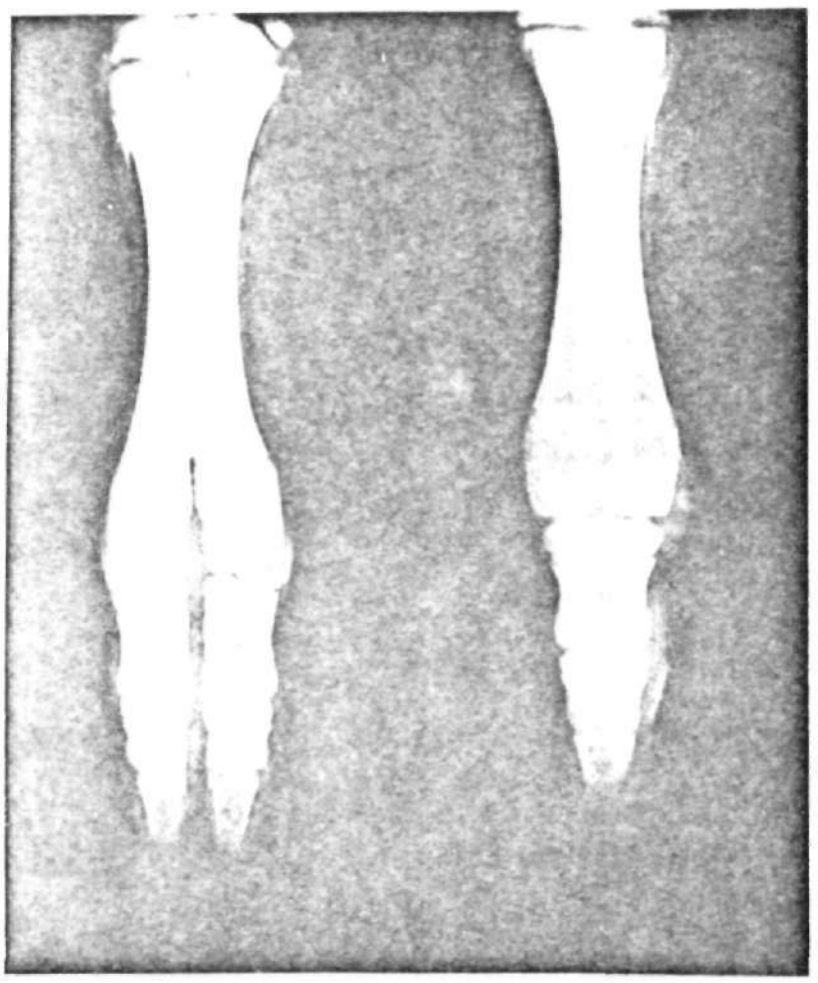


Fig. 8

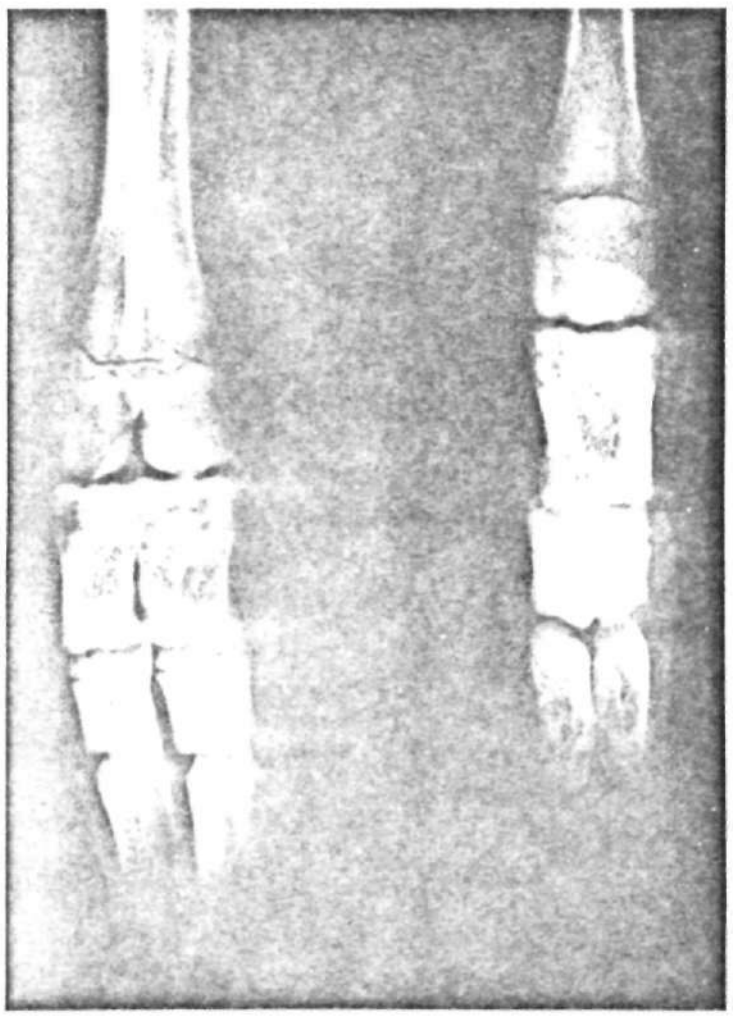


Fig. 9

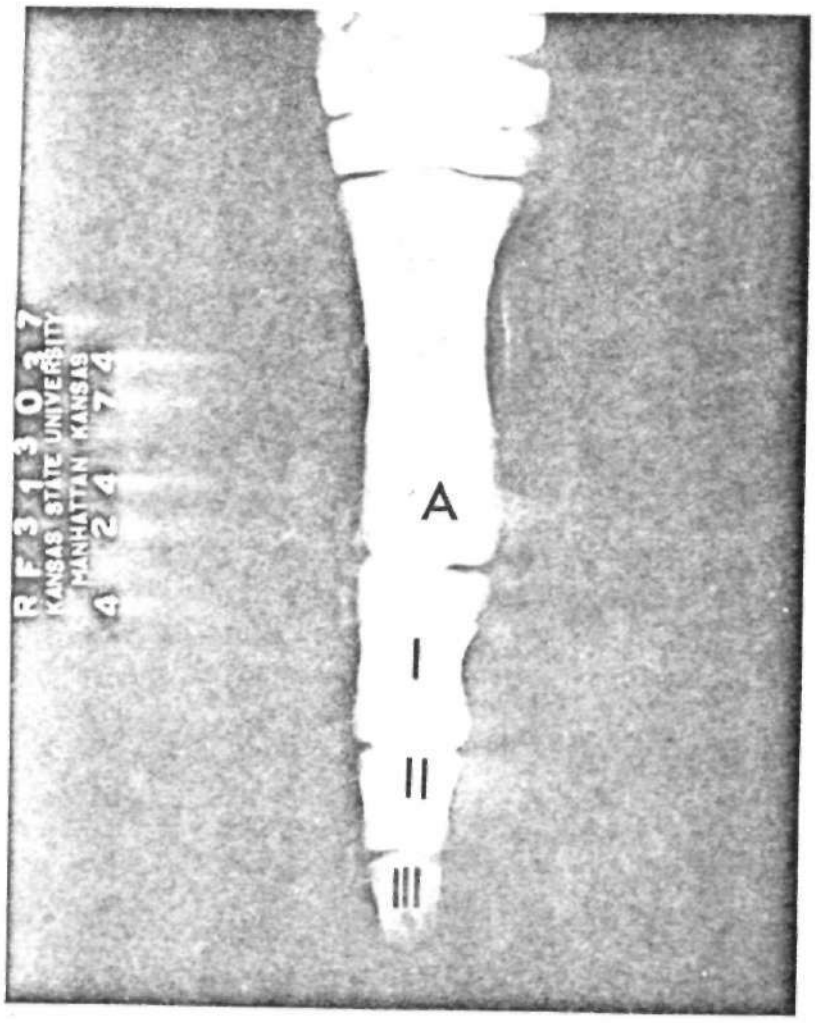

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Fig. 10

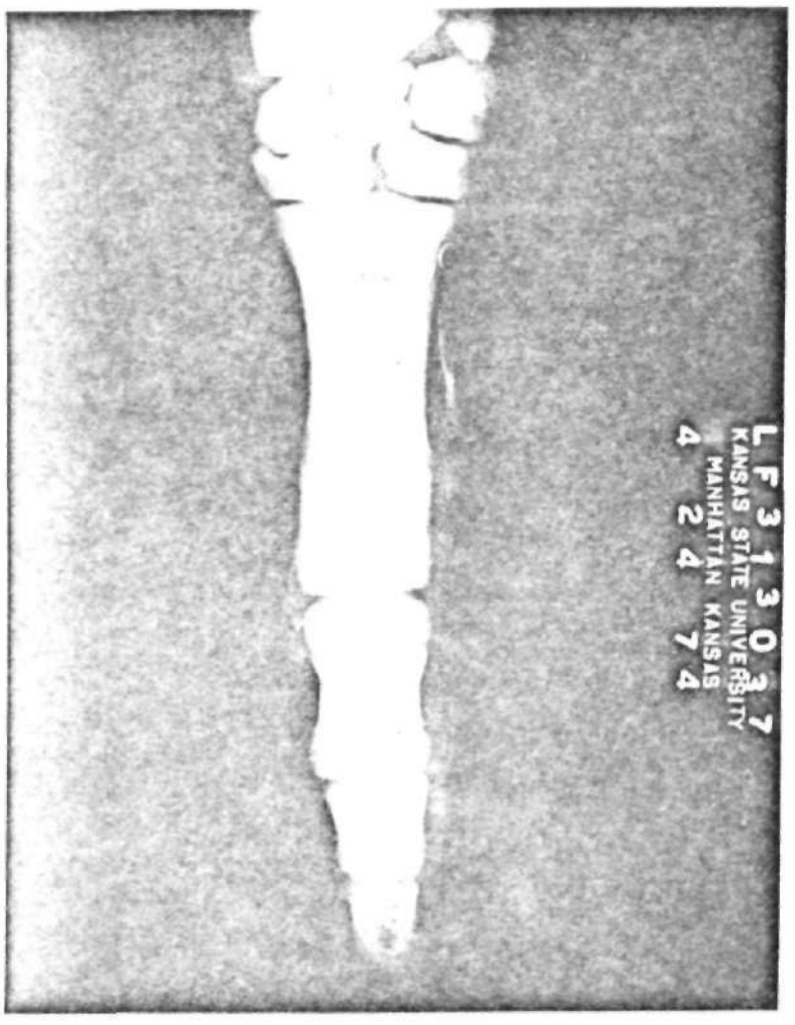


Fig. 11

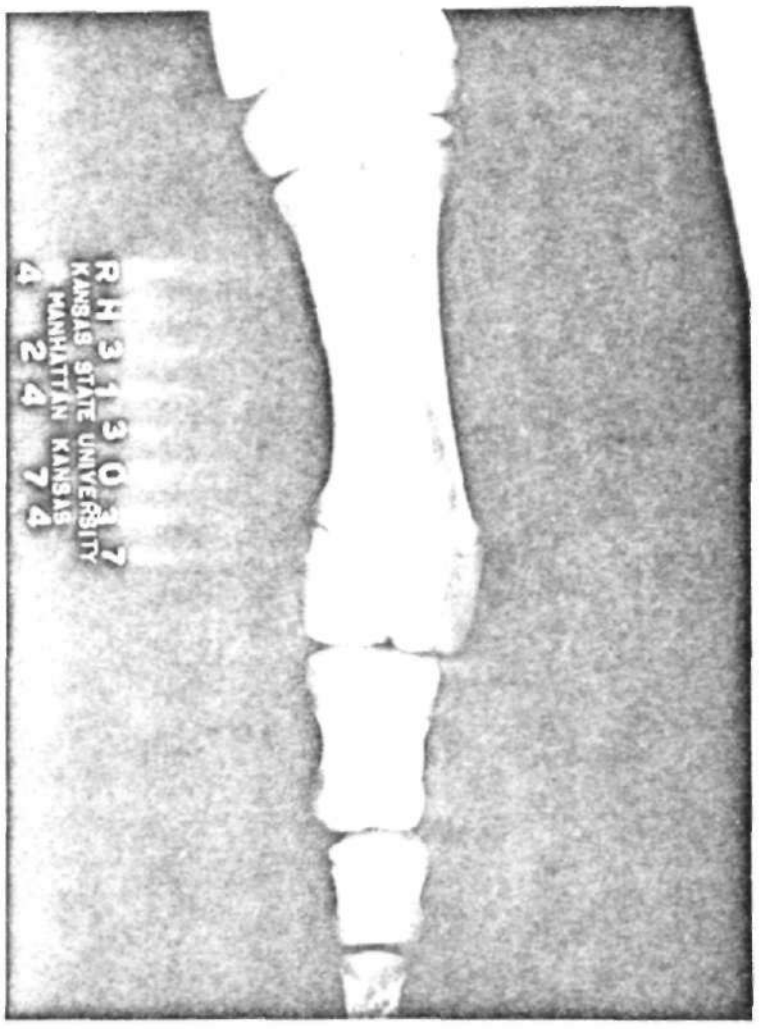


Fig. 12

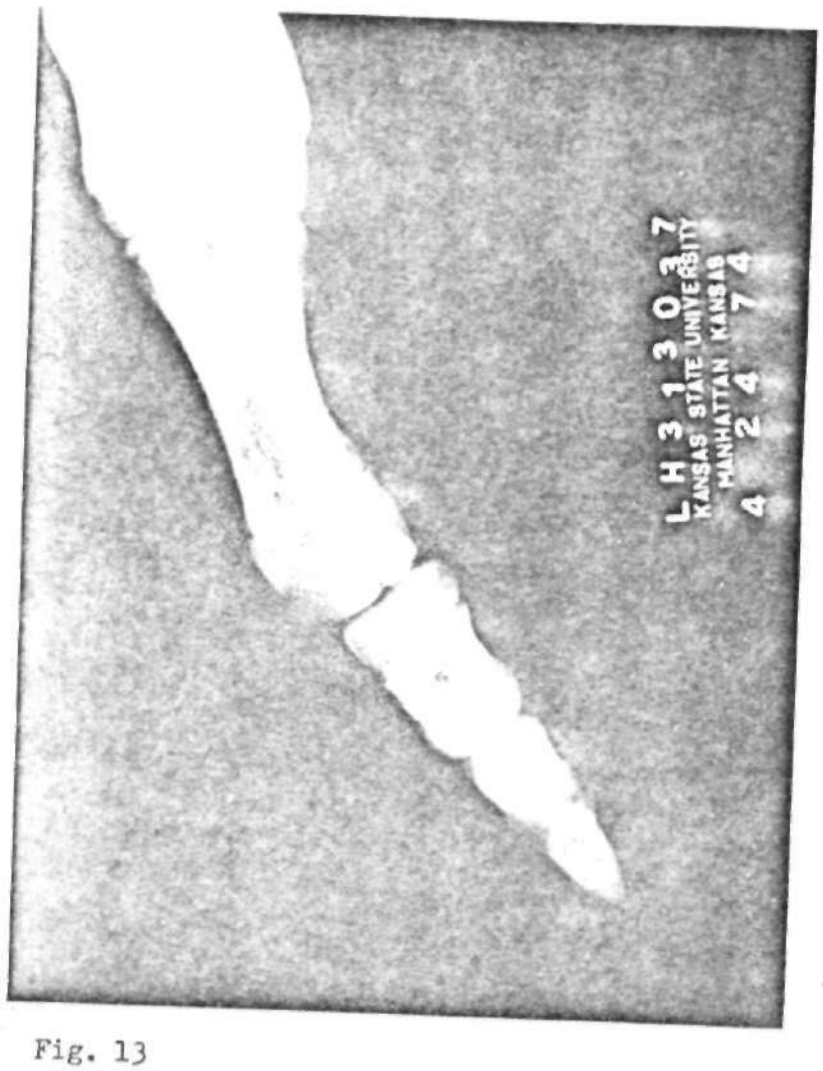


Fig. 13

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APPENDIX

REVIEW OF LITERATURE:

ALBINISM IN CATTLE

As early as 1920 an albino herd was described.[3] These albinos showed no pigment in the skin, eye, horns or claws and the eyes were extremely photophobic. The parents of the albinos showed normal Holstein coat color. This suggested a recessive gene, but further data indicated dominance as in F, albino bull sired only albino calves when mated to unrelated grade Holstein cows.[3] Furthermore, the matings of albino females to a Holstein bull produced only albinos. It should be mentioned that the breeders records of this herd had been lost and the data presented were based on memory. However, the author did not question the accuracy of the report given by the owner of the herd.[3]

A single case of a female calf with complete lack of pigment in skin, hair and iris was reported.[8] The pupils had a red color. The breeding test indicated a recessive gene since the mating of an albino female resulted in a normal offspring.

Another herd of albino showed the following characteristics: complete lack of pigmentation in skin, iris and hair at birth, at sexual maturity: some pigment could be observed and this phenomenon was designated as "ghost pattern".[2]

Histological examination of two albino cattle's eyes from the herd revealed no pigment on the retina whereas the iris and ciliary body showed some pigmentation causing the pupils to be pink but the irises, grey. Breeding test were performed and data obtained indicated a recessive gene for extreme reduction of pigment.[2]

The occurrence of 22 albino animals in Brown Swiss Cattle was investigated in Germany.[1] Fourteen animals were closely investigated and one female and one male were purchased and mated and produced a male albino calf. The three animals were later slaughtered and samples were

taken from the eyes, skin, horns, hooves, and hairs. The tissues were all unpigmented. The iris was white with a pinkish shine, the pupils were red and photophobia was extreme. The authors concluded that albinism is a recessive trait since test mating of albino X albino resulted in albino and albino X normal resulted in normal coat color. Furthermore, pedigree studies were done and in herd in which albinos occurred, common ancestors could be traced.

Three albino animals were reported in a Holstein herd.[7] Their skin were described as pinkish and the muzzle, hooves, and horns, unpigmented. Their hairs were creamy-white and no ghost pattern was observed. In daylight, the irises were greyish-blue and the pupil black, whereas in twilight, the pupils had a pinkish shine. The animals were photophobic. The inheritance was thought to be recessive since both parents of all 3 animals were related.

The results of 43 matings demonstrated to Peterson that albinism was inherited as a simple recessive.[19] Further, it was shown that ghost pattern was due to structural anomalus of the hairs rather than different degrees of pigmentation. The appearance of ghost pattern was associated with the fact for black (B) and was obvious at sexual maturity.

From Japan came a report of three albino Holstein calves[16] from a common sire and whose ancestors had been imported from the United States. In Wisconsin and Minnesota, the occurrence of albinos had been reported in these families.

In the Murbodner breed in Austria, a single albino calf was observed.[20] This calf, the result of an accidental mating of a son to his dam, showed no pigmentation at all and the iris was pink. Campara-

tive body measurements and skin thickness did not reveal any differences from normal.

Three cases of albinos were reported in Brown Swiss Cattle.[22] In each case, the parents exhibited normal coat color. Breeding experiments were not carried out but a single autosomal recessive factor was presumed. These cases were related to earlier cases, so sporadic reappearance of same mutation was considered. The cattle were described as having pink eyes, pink skin, lack of pigment, and photophobic.

Albinism has also been reported in Hereford Cattle.[17] Three albinos were observed in a small herd of registered Hereford cattle where a young bull was mated to two of his full sisters and four of his half-sisters. Several animals were acquired from this herd for breeding tests and from the brother-sister matings, three albinos resulted. The affected offspring had light pigmented areas on the inside of the hind legs but no evidence of ghost pattern. Clinically, the calves showed photophobia but were otherwise normal. Blood types of the sires and dams of the albinos were determined and were similar. Albinism and dwarfism were observed together in the same animals, but it was felt that the two traits were independent.[5] A blood abnormality was found in these partial albinos which was considered to be identical to that observed in the Chediak-Higashi Syndrome of man.[18] Increased susceptibility to disease was also observed. Breeding experiments performed with these cattle revealed a recessive gene.[17]

Chemical investigations of the pigment contents of bovine hair showed melanin was present, not only in white hair from colored Herefords, Holsteins, and Guernseys but also in albino hair.[21]

In conclusion, general specific types of albinism have been

reported in cattle.[10,11,13] They ranged from ocular albinism[10] over incomplete and partial albinism.[10,11,13]

In partial albinism, the color of the iris is blue and white centrally, and brown peripherally, and the coat color is usually characteristic of the breed or a dilute color.[4,10,11] A form of partial albinism is illustrated by the recessively inherited chediak-higashi syndrome, which besides its albinotic features, includes abnormally large membrane-bound organelles in various cells and an increased susceptibility to infection.[18]

Incomplete albinism, inherited as an autosomal dominant character is characterised by pure white coat color, a few animals may have small pigmented areas of the body. The iris may vary from blue to grey to white and may contain brown sectors. Incomplete albinos have colobomas of the nontapetal fundus and tapetal fibrosum hypoplasia.[4,10,11]

Complete albinism, inherited as a simple autosomal recessive trait, is characterised by pure white hair coat color and white to pink irides but a normal tapetum lucidum. Complete albinism has been reported in Holstein-Friesian,[16,19] in a Guernsey calf,[13] in a Murbodner calf,[20] and in European Brown Swiss.[23]

The findings of the various authors investigating bovine albinism are summarized in Table 1.

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Table 1 (continued)

Reference	Year	Breed	Mode of Transmission	No. of Albino Observed	Iris Color	Pupil Color	Hair	Sk
	1968	Hereford, Shorthorn, Guernsey, Holstein	Dominant incomplete	6				
	1968	Guernsey	Recessive	1	White	Pink	White	White

REVIEW OF LITERATURE:

CONGENITAL SKELETAL

DEFECTS IN CATTLE

Congenital defects have always been recognized but for many years inadequately studied. Defects generally are classified by the body system primarily involved. The entire skeleton or a localized region may be involved and the defect may be single or multiple or may be associated with congenital defects of other organs or systems.

Many different types of systemic, skeletal congenital diseases have been studied in man (49), but in cattle few have been described and still fewer have been well studied (39). In cattle chondrodystrophy, osteopetrosis, crooked calf disease and acroteriasis congenita have been described (39). Various types of dwarfism such as short headed, long-headed and telemark were described and were considered to result from homozygosity of simple autosomal recessive genes (16). In addition, the Dexter, comprest and compact mutants, are generally considered to be dominants, and part of a complex of conditions from more than one locus. They appear to be related to the simple autosomal recessive types (16-18).

Osteopetrosis in black and red Aberdeen Angus calves is characterised by small size and birth weight, brachygnathia inferior with impaction of the lower molar teeth, misshapen coronoid and condyloid processes, open fontanelle, thickened cranial bones, agenesis or hypoplasia of major foramina of the skull, and lack of bone marrow cavities due to persistence of the primary spongiosa (30,33). Crooked calf disease was shown to be caused by ingestion of lupines (Lupinus Sericeus and Lupinus Candatus) (51). A similar skeletal deformity of arthrogryposis, kyphosis, torticollis, scolliosis, and cleft palate, was due to homozygosity of recessive genes (52,53). Acroteriasis congenita, a simple recessive defect, has been reported in Europe and consisted of low birth weight, amputation of all four legs, defects of the facial

skeleton, cleft palate, brachygnathia inferior, microtia, and hydrocephalus (6,48).

Single defects of the skeletal system were conveniently classified according to body region affected (15). A number of isolated case reports have been described (1,7,9,43,45,57,59) but studies of overall incidence and etiology are missing (10,19,44,50,58). Palatoschisis occurred as an isolated defect or may be associated with other defects such as arthrogryposis (32). Cheilognathoschisis reported in Shorthorn cattle was inherited as a simple autosomal recessive trait (56).

Several congenital defects of the jaws have been described such as camplognathia, the lateral deviation of the face (42). Brachygnathia inferior (reduction of the length of the mandible) (14), has also been reported to be caused by chromosomal aberration (20). Inferior brachgnathia was also a feature of systemic congenital osteopetrosis (33). Agnathia or aplasia of the mandible is rarely observed in cattle (5,8,46). Craniofacial dysplasia in the French Limousine breed involved deficient ossification of the frontal sutures, convet profile of the nose, inferior brachygnathia, bilateral exophthalmus, scoliosis of the upper jaw, macroglossia, and associated defects of omasum and heart (3).

"Short spine lethal" is a rare defect of the axial skeleton, characterized by reduction and fusion of ribs (36). Likewise, perosomus elumbis, the agenesis of the spinal column caudal to the thoracic area, is a rare defect (39). Other reported defects of the spinal column include kyphosis which is dorsal deviation, lordosis (ventral deviation), scoliosis (lateral deviation), and their combinations. These skeletal defects are commonly associated with arthrogrypotic deformities (32,39). Spina bifida associated with posterior bimelic arthrogryposis of the

hind legs has been reported in German breeds of cattle and once in an Angus calf in the United States (39,40). Agenesis (anury) and partial agenesis (brachyury) of the caudal part of the spinal column are common defects of unknown cause in cattle and often associated with congenital defects of eye and heart (21,39,47). Wrytail has been described in cattle as a simple autosomal recessive trait (2).

Congenital defects of the leg include polymelia (duplication of whole limbs). This defect has been reported rarely (26,37,38). Adequate studies of osteologic embryonic duplications are also rare (34,35). Amelia which is agenesis of extremities, abrachia and apodia refer to absence of front and rear legs respectively (12,13). Calves with abrachia had dysplasia of the spinal cord segment corresponding to leg defect (13). Hypoplasia of the appendicular skeleton is referred to as micromelia, whereas peromelia is the failure of distal appendicular parts to develop; phocomelia refers to absence of the proximal appendicular parts with distal parts developed (11).

Other defects described were tibial hemimelia, a recessive lethal involving localized areas of the extremities (60), reduced phalanges, also a lethal recessive (23), extrodactyly, partial or complete absence of phalanges (27), and adactyly, a recessive lethal in Shorthorn calves (31). Development of additional digits, polydactyly, has been described occasionally (25,38). Fusion or nondivision of the functional digits is termed syndactyly. Syndactyly is one of the commonest skeletal defects of Holstein-Friesian cattle in the United States and has been shown to be due to a recessive trait with incomplete penetrance. The fusion follows a particular pattern; the right foot is affected, followed, in order of frequency by the left front, right rear and left rear foot.

The anatomical defects in hereditary bovine syndactyly are accompanied by a functional defect which needs special environmental condition for expression. They have little tolerance to heat (28). Syndactyly has also been described in an Angus calf in United States and exotic breeds in other countries (24,29,39).

Congenital disorders of joints may be generalized or restricted to one joint. Although joint diseases are of great importance, there is a scarcity of genetic reports. Hipdysplasia has been reported in Hereford and Charolais breeds and it appears to be hereditary (4,15). Ankylosis, abnormal union of the ends of bones forming the joint arthrogryposis and congenital contractures of muscles have been reported many times as ankylosis (41). An hereditary lethal type of ankylosis, restricted to the mandibular joint, has been encountered in calves (14). Osteoarthritis of the stifle joint in Holstein-Friesian and Jersey breeds has been described as an autosomal recessive trait (54). This inherited degenerative osteoarthritis usually involves gradual onset of lameness in both hind legs in older animals. Crepitation of the stifle joints can usually be heard and felt.

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STUDIES OF BOVINE CONGENITAL DEFECTS

by

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Congenital defects are defined as abnormalities of structure and function present at birth. Many or perhaps all structures or function of the body may be affected by developmental aberrations that may vary from slight to severe. Furthermore, other structures and or functions of the body may be affected. One defect may be obvious grossly but others may be discernible only after careful clinico-pathologic and necropsy examination. Reportedly, 0.2 to 3.6 percent of all calves born are affected with congenital defects. Skeletal congenital defects in cattle result in economic losses from decreased reproductive capacity, increased prenatal losses and reduced value of related calves when defects are genetic. Many congenital defects follow a simple pattern of Mendelian inheritance. Others are caused by environmental factors, and still others have no clearly established cause. Various parts of the body may be affected with a congenital defect. Some may be single isolated defects; however, it becomes increasingly obvious, that many occur together as part of a syndrome. Frequencies of defective anatomical structures vary with geographical region, breeds and other factors. This study considered two albino traits and seven skeletal defects in cattle.

Ocular albinism combined with dilution of body color occurred in four Nigerian Holstein-Friesian calves. The calves had general body color that was dilute grey and exhibited grey irises, a normal tapetum lucidum and normal optic nerve. Ocular albinism in Nigerian Holstein-Friesians is most likely caused by homozygosity of a simple autosomal recessive gene.

Thirty-six eyes of fifteen incomplete albino cattle and 3 normal cattle were examined grossly and microscopically. The main

lesions of incomplete albinism in cattle are reduction of pigment and coloboma of the optic disc.

In this study skeletal defects are recorded by body region affected. Generalized skeletal defects encountered were two cases of chondrodystrophy, in a Maine Anjou and a Holstein-Friesian, and osteopetrosis in two Hereford calves. Calves affected with osteopetrosis were stillborn, undersized, and had brachygnathia inferior and impacted molar teeth. Long bones were fragile and fractured easily under lateral pressure. The bones were homogeneous without bone marrow cavities. Longitudinally bisected bones had "bone cones" that nearly filled the medullary areas. Cranial cavities were reduced; the frontal bone had multiple cysts. The solid medullary areas comprised of chondro-osseous tissue resembled early fetal steps of ossification.

Various parts of the body may be affected with a congenital defect. Although some may be single isolated defects but in this study it became increasingly obvious that many defects occur together as part of a syndrome. Regional skeletal defects studied were: polydactyly, camptodactyly, tibial hemimelia and syndactyly.

A 3-month-old, female Holstein-Friesian calf was affected with polydactyly of both forefeet and the left hind foot, while the right hind foot exhibited polysyndactyly. Karyotypes prepared from peripheral blood revealed no gross abnormalities. Another Holstein-Friesian calf had polydactyly of both front legs.

Camptodactyly, defined as permanent and irreducible flexion of one or more digits, was diagnosed on both hind feet of a neonatal Simmental calf. Abnormal formation of the tarsal bones, nonfusion of the 3rd and 4th metatarsals, absence of proximal and distal sesamoids

and contracted tendons involving the 3rd phalanges were associated features. In addition, there was bilateral hypoplasia of the kidneys and fetal oxalosis. The cause was not determined.

Tibial hemimelia was encountered in seven Galloway calves and is part of a congenital syndrome that may include agenesis of patella, tibia, nonclosure of the pelvic symphysis, cranioschisis, meningocele, internal hydrocephalus, cryptorchidism, or nonfusion of Mullerian ducts. The defect was most likely inherited as a simple, autosomal recessive.

Facial-digital syndrome in two Angus calves included syndactyly of the legs, short face, one nasal opening, kyphoscoliosis, and internal hydrocephalus and spinal dysraphism. This defect is most likely inherited as a simple, autosomal recessive.

Syndactyly, the partial or complete fusion of functional digits on one or more feet, was studied in a purebred Hereford, in purebred Holstein, and crossbred Holstein/Chianina and Angus/Chianina calves. Syndactyly in these crossbreds is most likely inherited as a simple, autosomal recessive.