STUDIES ON BOVINE CONGENITAL DEFECTS

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A THESIS

submitted in partial fulfillment of the
requirements for the degree
MASTER OF SCIENCE

Department of Pathology

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1979

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DEDICATION

This work is dedicated to my whole family who has persevered with me through my years of schooling.

ACKNOWLEDGEMENTS

I wish to express my sincere thanks to Professor H. W. Leipold for his guidance, encouragement and assistance throughout the various facets of my research. I am also grateful to Professors S. M. Dennis and R. R. Schalles for their critical reading of the thesis and their suggested amendments. I thank the National Animal Production Research Institute and Ahmadu Bello University for the opportunity for graduate training at Kansas State University.

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INTRODUCTION

Bovine congenital defects pose a real problem to animal breeders. They result in loss in reproduction, production, and product quality. An example of this is dark cutting beef carcasses associated with double muscling in cattle. Most genetic defects are controlled by autosomal recessive genes; others are incompletely recessive, dominant or polygenic.

Muscular hypertrophy (double muscling) has existed in many cattle populations throughout the world for some time. In Italy and France it has been selected for by farmers as people in these countries prefer meat with less marbling. As a result the double muscled gene increased in frequency among the total population in France and Italy. Kidwell et al. (1952) were the first to use the words "muscular hypertrophy" for double muscling. The term double muscling is a misnomer as the muscles are not doubled but simply enlarged. Double muscling has not been reported in any Zebu breed.

Little is known of facial defects in cattle in contrast to sheep (Dennis and Leipold, 1972).

The dairy industry relies heavily on udder quality. A cow with defective udder does not produce as much milk as that with a normal udder. Strong selection pressure is

applied for udder conformation. The shape, size, and location of the udder determines, to a large extent, the economic value of a dairy cow. Polythelia is one congenital inherited teat abnormality in cattle. The appearance of several teats spoils the symmetry of the udder and also the glands associated with the extra teats frequently produce small amounts of milk and cause irregularities and difficulties in milking. Genetic analysis of inheritance of teat abnormalities would be valuable to dairy cattle breeders as an aid to selecting their breeding stock. This should help to decrease the frequency of deleterious genes from the dairy population and possibly eliminate them. There is also a genetic difference in the susceptibility of the bovine udder to mastitis. Apart from polythelia conditions like synthelia, teat aplasia, defective teat openings, and hypoplastic teats also occur.

Umbilical hernia of cattle is little understood. It has been considered to be genetic while some authors have incriminated environmental causes. Ranchers and veterinarians have regarded the condition as a simple problem that can be dealt with surgically. Bulls with umbilical hernia have impaired ability to serve and thus become infertile. Artificial insemination can spread the condition. Most hernia cases are not reported; persistent cases are either surgically treated or disposed of. The fact that umbilical hernia has not appeared in large numbers of herds indicates that it may be a dominant character.

Prevention by selecting against the trait will help in reducing its frequency or eliminating it altogether.

The four congenital defects studied in this thesis undoubtedly pose a real danger to the reproductive capability of cattle and efforts should be made for selecting against them.

The congenital defects studied in this research were:

1. Double muscling
2. Facial defects in polled Herefords
3. Teat abnormalities in Purebred Herefords
4. Umbilical hernia in Holsteins.

I. DOUBLE MUSCLING IN CATTLE

INTRODUCTION

Muscular hypertrophy or double muscling is an inherited syndrome in cattle that causes a reduction in fitness of affected individuals in each of several physiological functions. In severely affected cattle all muscles of the body are enlarged. The term "double muscling" is a misnomer as the muscles are enlarged and not doubled. There are a greater number of muscle fibers than normal in affected muscles. The mode of transmission is not clear. Research on double muscling has been limited with most observations having been made on private farms. Kidwell et al. (1952) were the first to use the words muscular hypertrophy to describe this abnormality.

Muscular hypertrophy has existed in many cattle populations throughout the world for some time. In Italy and France, it has been selected for by farmers as people in these countries have a preference for meat with less marbling. As a result the double muscled gene increased in frequency among the total cattle population in France and Italy. Cully (1807) connected the incidence of dark cutting carcasses with muscular hypertrophy in England. These carcasses came from the Shorthorn Dutch kind cattle. These cattle were imported from Holland and developed in England.

Muscular hypertrophy has been reported mostly in European breeds of cattle, with no reports in the Zebu cattle of the tropical regions of Africa, Asia, and South America. In places like the United States, double muscled meat does not find acceptance by packers because the scanty covering of fat results in meat drying out, thus reducing its keeping qualities. A farmer in Nebraska kept a good number of cattle with this condition. It is said to have appeared in the United States in the early 1930's (Weber and Ibsen, 1934).

Double muscling is being studied in cattle because of increased reports of decreased reproductivity. The continuing selection against the homozygous phenotype has not eliminated the gene from the cattle population but instead has led to a balanced polymorphism possibly due to the selective heterozygous advantage. Selection of breeding cattle particularly bulls, based on conformation correlated with higher yielding leaner carcasses, would be expected to favor an increase in the muscular hypertrophy gene in the cattle population. Although the pattern of inheritance is debated, most believe that it is due to a single pair of genes. Heterozygous cattle have variable expressivity ranging from apparently normal animals to those resembling homozygous double muscled animals. This has been the source of debate in the mode of transmission. Some authors consider that it is due to a recessive gene, others an incomplete dominant gene, and still others an incomplete

recessive gene. Muscular hypertrophy has both deleterious and useful effects.

REVIEW OF LITERATURE

Double muscling was referred to in most European countries in the late 19th and early 20th Centuries indicating that it existed in the cattle populations at that time. Kaiser (1888) observed it in Germany and named it "doppenllender" (double loin), a name that is still being used today. In France, Thierry (1898) called it "culdl poulain" (backside of a colt). In Italy, Brusaferro (1905) described it as "a groppa de cavallo" (rump of a horse). Later in France, Dechambre (1910, 1911) described the condition as "croupe de poulain" (rump of a colt) and "a"cuisse de cheval" (thigh of a horse). In France today, the term "culard" is used to describe double muscling (Lauvergne et al., 1963). The name most commonly used in Italy at present according to Ramondi (1956) is "a groppa doppia" (double rumped).

The double muscled condition has been observed in France, Belgium, Holland, Denmark, Norway, Switzerland, and Italy (Wriedt, 1929). It has been reported to be most prevalent among the Friesians, Ayrshires, Shorthorns, Charolais, and Piedmonts. In Britain, cattle with muscular hypertrophy are called Teeswater and Yorkshires (Cully, 1807). Reports of double muscling have come from England

where Mason (1962) observed a double muscled bull in an A.I. stud belonging to the Milk Marketing board. In Australia, Butterfield (1966) reported the incidence of the defect among crossbreds of Brahman and British cattle.

The condition is one of generalized and not localized hypertrophy or duplication of muscles as the name suggests (Pomeroy and Williams, 1962; Black, 1936; Schaper, 1937; Shrode and Lush, 1947; Kidwell et al., 1952).

Weber and Ibsen (1934) found no evidence of the condition being reported in the USA prior to 1934. They attributed the name "double muscling" to a purebred Hereford farmer in Eastern Nebraska. The defect appeared in his herd in the late 1920's, thus affirming the assertion that a recessive gene was responsible. Kidwell et al. (1952) reported cases of muscular hypertrophy in a farm in Louisiana. Kleberg (1967) reported it in Santa Gertrudi cattle in Texas.

Meat with less marbling from double muscled animals is preferred in France and Italy (Lauvergne et al., 1963). This has resulted in selection for double muscling. English Shorthorn are said to be responsible for the condition appearing in the Charolais breed in France. Ramondi (1965) estimated that 80 percent of bulls of the Piedmont breed in Italy carried muscular hypertrophy genes, as a result of selection for the character. The double muscle gene may have been introduced into Italy, through importation of a Charolais bull from France in 1886 (Luciano, 1903).

EXTERNAL APPEARANCE OF DOUBLE MUSCLED ANIMALS

All reports concerning inheritance of double muscling agree only to the extent that a single pair of genes is incriminated. There is disagreement as to whether the gene is dominant or recessive. Most agree, however, that either incomplete penetrance, incomplete dominance, or gene modifiers of some type are involved, because the phenotypic expression of double muscling is quite variable. Because of this variability, heterozygotes may appear normal to almost phenotypically homozygous. This makes it difficult for farmers to recognize heterozygous cattle and may inadvertently select them for breeding. Few, if any cattle, exhibit all the characteristics associated with double muscling. The defect is a syndrome.

The hindquarters are the most noticeable part of affected cattle. "The outline of the rump and round from the hip to the point where the quarter joins the leg is a convex line corresponding rather well to an arc of a circle whose center would be the articulation of the femur and tibia," (Dechambre, 1910). This gives the rump a sloppy appearance. The tail is attached more anteriorly and the tailhead is prominent and well-defined. The enlargement of rump muscles bilaterally on the swell of the sacral vertebrae gives rise to creases or depressions above the vertebrae that are responsible for the Italian description of "groppa doppia" or double rump. In the majority of cases,

massive musculature is accompanied by other well-defined signs. The muscles over the point of the hips are so enlarged that their size and movement become prominent as the animal walks. Double muscled cattle appear to be long and cylindrical in the middle region (Mason, 1962). Charlet and Poly (1965) reported similar findings. The longissimus dorsi muscles are also enlarged causing a depression above the backbone that is the basis for German term of "doppellender" or double loin. The medial gluteal muscles and to a lesser extent, the crease between the semimembranous and semitendinous muscles are obvious in newborn calves (Oliver and Cartwright, 1969).

The shoulders are enlarged due to an increase in the external muscles and enlargement of the muscles medial to the shoulder blade. This tends to push the shoulder blade away from the body, creating an open shouldered condition. Double muscled cows are more masculine than their normal counterparts, the reverse is the case with bulls. Double muscled bulls are more feminine than normal bulls of the same age, size, and environmental conditions. There is reduced percent of bone in both sexes especially the cannon bones in both front and rear limbs (Butterfield, 1966).

The head of affected cattle is smaller and lighter (Charlet and Poly, 1965). The neck seems to be shorter, thicker, and arched higher above the withers. The rear legs tend to be straight from the hocks downward. Double muscled cattle have a characteristic stance, the front

legs are more forward and the hind limbs are pulled more to the rear giving a distinct stretched appearance (Brusaferro, 1905; Paci, 1935). This posture explains the "saddlebacked" appearance by Kidwell et al. (1952). Macroglossia may be present in newborn calves. The tongue fills the whole mouth and the calf experiences difficulty with feeding. In some cases the tongue may be adhered and any attempt to nurse the calf may tear the septum. Macroglossia, bowed and crooked legs are temporary. They disappear at about 90 days of age (Oliver and Cartwright, 1969). Paci (1935) and Ramondi (1956) also described the tongue as being thick, inert, and extending beyond the lips by about 5 to 10 cms. in extreme cases. Oliver and Cartwright (1969) observed two cases with severe macroglossia.

The vulva is positioned away from the normal near-vertical plane and more towards a horizontal plane, this causes difficulty with breeding. The testicles of affected bulls are lighter in weight than those of normal bulls of the same age (Charlet and Poly, 1965). The testicles seem to be carried closer to the abdominal wall.

It has been indicated by most authors that as an affected animal grows older, the expression of the trait becomes less apparent, especially after maturity. Lauvergne et al. (1962), however, reported that some cattle appear normal up to 12 months of age before exhibiting signs. The calves mentioned by Lauvergne and associates were not homozygous for the defect. They must have been talking

about heterozygotes that Charlet and Poly (1965) described as intermediate double muscled. Generally, double muscled cattle grow faster and mature early, and this gives them a smaller size than their normal contemporaries. Lauvergne et al. (1963), Charlet and Poly (1965), and Oliver and Cartwright (1969) indicated that the trait is more marked in males than females. This was especially true after puberty and first calving. Variability of expression of the double muscled gene has been mentioned. Smith (1949) working in an experimental breeding herd in Kansas stated that cattle he classified as heterozygotes had no signs of the double muscled gene. Charlet and Poly (1965) reported a system for classifying double muscling by French research workers:

a) Hypertrophy of the tongue
b) Muscular hypertrophy of the shoulders
c) Body width in relation to height
d) Reduction of the paunch
e) Inclination of the rump
f) Tail attachment
g) Muscular hypertrophy of the thighs
h) Impression of the superficial grooves between muscles
i) Position of the legs
j) Fineness of the bone

Affected cattle are given a score for each of the above characteristics; 0 if normal, 1 if intermediate, and 2 if double muscled. An animal with a total score of 0-6 is

normal, 7-12 intermediate, and 13-20 double muscled. Oliver and Cartwright (1969) used the following classification:

1) Animals with no evidence of the condition.
2) Animals with a slight evidence.
3) Animals with rather strong evidence.
4) Animals that are definitely double muscled.

SLAUGHTER AND CARCASS CHARACTERISTICS

Cattle with muscular hypertrophy have a higher dressing percentage than normal cattle and the carcass has less inter- and intramuscular fat, a higher ratio of muscle to bone, and more tender meat (Oliver and Cartwright, 1969). Heterozygotes yield proportionately more carcass (Ramondi, 1964; Theissen, 1973). The economic return for heterozygotes in Italy according to Ramondi is higher than that of normal cattle due to the increased quantity and quality of lean meat. West et al. (1973) reported that heterozygous steers excelled in carcass characteristics and had more tender meat than their normal counterparts. Meat from the longissimus and semimembranosus muscles of normal heifers was more tender than meat from heterozygous phenotypes, while the opposite was true for bullocks. The meat from normal phenotypic heifers was juicier than that of heterozygous heifers (Carrol et al., 1978). Butterfield (1966) found the amount of dissectable fat in a rapidly growing

muscular hypertrophic bull to be 5 - 6 percent of live weight, a value equal to that of an emaciated animal and only 60 percent that of a normal animal. Under conditions of nutritional stress, double muscled animals will have to metabolize muscle protein instead of fat. Relative difference in food intake exists between the muscular hypertrophic animal and its normal counterpart, due to a smaller digestive tract in the double muscled animals (Rollins et al., 1969). An alternative explanation is that the reduced intake of food may be due to reduced fat synthesis leading to maintenance of higher blood acetate that Baile (1968) found might depress appetite in ruminants (Holmes and Robinson, 1970). Holmes and Robinson also noted a low concentration of NEFA (non esterified fatty acids) in double muscled animals, suggesting a low ability to mobilize fatty acids in response to short term alterations in energy availability. They found that double muscled cattle have a marked visible reduction in muscling at 85 percent of maintenance in three weeks, while normal cattle had no change. Normal cattle consumed more dry matter per unit of body weight, and required less feed per unit of gain than double muscled cattle (Charlet and Poly, 1965). Dechambre (1911) reported an average dressing percentage of 64 for muscular hypertrophic cattle. Many calves of affected animals are lost through dystocia.

Double muscled animals produce more meat protein on a

body weight basis (Ramondi, 1956; Rollins et al., 1969). Little is known about the biochemistry or physiology of the trait. Comparing normal and affected muscles, Ashmore and Robinson (1969) found no degenerative changes in affected muscles. No significant differences were observed between the concentrations of DNA, RNA, protein, or in serum creatinine phosphokinase activity of the two muscular types. The hypertrophied muscle had decreased succinic dehydrogenase activity. Degeneration is not a part of the double muscled syndrome, rather it is associated with a disproportionate number of glycolytic type fibers (Holmes and Ashmore, 1970). Since these are, on the average, larger than oxidative type fibers, this may explain the gross hypertrophy of the skeletal musculature. Smith (1949) found the dressing percentages of affected and normal cattle to be 66.7 percent and 63.5 percent, respectively, at 18 months of age and fed the same quality feed. The cutability of double muscled cattle is higher than that of normal cattle. Cully (1807), speaking of the Tees River cattle of England, was quoted as saying: "One of these creatures will feed to a vast weight and though fed ever so long, yet will not have one pound of fat about it, neither within or without." Youatt (1834) also said: "Always fleshy, but never fat, and the flesh being of bad quality." Double muscled carcasses are regarded to be undesirable under American Marketing System (Weber and Ibsen, 1934). Many authors and researchers

including Kidwell et al. (1952) found reduced fat deposits in affected cattle. Smith (1949) reported that the hindquarter represented 48.7 percent and 46.6 percent of the side weight of double muscled and normal cattle, respectively. Ramondi (1956) supported Smith's work by reporting that double muscled cattle had 3 percent less fat, 8 percent more muscle, and 5 percent less bone than normal carcasses. Charlet and Poly (1965) found that affected cattle had 9 percent more meat, 6 percent less fat, and 2.3 percent less bone than their normal counterparts. Vissac (1962) reported 73.4 percent and 61.6 percent muscle in double muscled and normal cattle, respectively. Front and rear muscles are affected to the same degree (Rollins et al., 1969); they also reported the ratios of double muscled to normal weights for carcass fat and offal fat. Their results suggested that more than one gene is involved in the laying down of carcass and mesenteric fat. Butterfield (1966) observed that affected cattle contained only 50 percent as much total fat as the normal carcasses. He also found 20 percent more muscle and 20 percent less bone in affected individual animals. Pomeroy and Williams (1962) found no significant difference in bone weights of double muscled and normal cattle.

There is essentially no difference in palatibility of meat from hypertrophic muscular animals and normal ones. Butterfield (1966) doubted the assertion that subcutaneous

fat depots are reduced more than the intermuscular depots. Although moderate degree of muscular hypertrophy may be desirable by butchers the more extreme form causes dystocia. Results show that the hypertrophic effect is reflected by an increased nitrogen and potassium content (on a fat free basis) and by diminished connective tissue (hydroxoproline) and intramuscular fat. The effect is less pronounced in regions of early developing musculature (Lawrie et al., 1963). The findings confirm subjective estimations that muscles of hypertrophied cattle are more tender than normal, and that this tenderness is retained to a much later age. Hypertrophied muscles have a lower potassium/sodium ratio (Lawrie et al., 1964). They also reported that the iodine number of fat extracted from hypertrophied muscles was invariably higher than in corresponding normal animals.

Greenbaum and Young (1953) found that administration of growth hormones affected certain muscles in rats. Pectoralis major and suprasphinatus muscles are among the fastest reacting group of muscles. Pomeroy and Williams (1962) suggested that an endocrine-enzyme imbalance under genetic control may result in specific acceleration of muscle growth. This may explain the fast growth rate of double muscled calves. The location and function of a muscle may influence its growth (Lawrie et al., 1963).

Kidwell et al. (1952) stated that cooking and palatability tests revealed no difference between hypertrophied and normal cattle. People in the United States tend to

look upon hypertrophied cattle with disfavor because of the small amount of fat in the carcasses (Schrode and Lush, 1947). Kidwell et al. (1952) associated dark cutting carcasses with the "Tees water" or "Yorkshire" cattle that were later reported to have the double muscled gene. Unusual pigmentation of the brain coupled with less vascularization in the pia matter in the frontal lobes was noted during examination of brains of hypertrophied animals by Kidwell et al. (1952). This condition was unilateral.

There is a positive correlation between total hemoglobin content of tissues and the brilliance and color values of the meat (Guilbert, 1937). Guilbert also found that the total hemoglobin content of black cutting beef was within the normal range. Black cutting beef when placed in an atmosphere of oxygen under pressure changes color from dark purple to bright red (Guilbert, 1937). Several factors have been incriminated as causes of dark cutting carcasses including age, exercise, excitement, feed, delayed bleeding, peculiar pigmentation, and heredity. Guilbert examined these factors by subjecting cattle to be slaughtered to these conditions and to his surprise found no correlation with dark cutting carcasses, though delayed bleeding could cause some dark coloration. Since dark cutting beef could not be produced experimentally, hereditary factors became apparent as possible causes. These findings proved the statements of Kidwell et al. (1952) and Cully (1807) and others, that muscular hypertrophied cattle have dark cutting

carcasses, thus establishing a link between muscular hypertrophy and dark cutting carcasses. At all ages and in all muscles, the proportion of red fibers is lower than normal (Pomeroy and Williams, 1962). Most muscles were of the white type and were larger than normal. The term muscular hyperplasia may be a more accurate description of muscular hypertrophy. Dennis (1971) reported a case of congenital muscular hypertrophy in a lamb. The skin on sectioning was thin with little visible subcutaneous fat, the muscles were paler than normal, with the bundles clearly demarcated by intermuscular connective tissue.

Hypertrophied muscles contain more glycogen than normal muscles due to greater muscling. Metabolic differences between normal and hypertrophied cattle is probably not related to synthesis of body constituents by the liver (Beitz et al., 1969). Hypertrophied muscles contain less red fibers and more white fibers. There is decreased activity of oxidative enzymes and increased activity of glycolytic enzymes; this means that the double muscled cattle have a greater capacity to produce lactic acid faster than normal cattle. Ramondi (1956) reported a case of muscular degeneration associated with rigidity, trembling, and difficulty of movement in Italian Piedmont calves. This was substantiated by Holmes et al. (1972) in which a double muscled heifer after being forcely exercised could not stand and had to be destroyed, the quadricep muscles of the leg were hard. There was high levels of CPK (creatinine phosphokinase)

in the blood of the hypertrophied heifer that existed for quite a long time. Potassium concentration also rose. All these changes were postulated to be due to reduced microcirculation resulting from change in musculature from red to white fibers in hypertrophied cattle. This proves the susceptibility of double muscled cattle to exercise.

ADVANTAGES AND DISADVANTAGES OF DOUBLE MUSCLED CATTLE

One of the big disadvantages of this defect is reduced reproductive efficiency. Sterility may result from an infantile reproductive tract in the female. The uterine horns and vagina are shorter than normal but wider. Double muscled animals mature slowly. Lauvergne et al. (1963) brought out the possibility of muscular hypertrophy being responsible for absence of estrus in affected cows. The gestation period of hypertrophied cattle is about 10 days longer than average. Calves of hypertrophic cattle are heavier than normal, hence the dystocia rate is high (Paci, 1935). Ramondi (1965) reported that mating of cattle homozygous for double muscling produced 13.2 percent stillbirths, 11.3 percent of dystocia requiring traction, and 3.7 percent weak calves. Fifty nine percent of hypertrophied cows were reported to require delivery by caesarean sections (Charlet and Poly, 1965). Thirty eight percent of the calves died at birth and only 33 percent of calf crop was weaned. Charlet and Poly (1965) also reported reduced lactation in

affected cows with only 50 percent of normal production.

Semen produced by hypertrophied bulls is poor quality with a lot of dead spermatozoa. Defective bulls have lighter testicles tucked up against the abdominal wall. The penis is narrow and in a case reported by Charlet and Poly (1965), the glans penis was adhered to the sheath thus preventing copulation. Normal bulls produce a greater volume of spermatozoa than defective bulls, but the semen of the latter is more concentrated. Neuvy and Vissac (1962) reported semen quality in 14 double muscled bulls was similar to that of normal bulls; they obtained 70 percent conception rate using artificial insemination. Paci (1935) found that muscular hypertrophic animals gain 15 percent faster than their normal contemporaries. This was supported by Charlet and Poly (1965).

Calves of double muscled dams are more susceptible to disease (Paci, 1935). Ramondi (1956) reported without substantiating data numerous cases of death among double muscled calves which was attributed to heart weakness. Rickets was observed in double muscled calves (Paci, 1935; Hanset, 1961; Lauvergne, 1963). Ramondi (1956) observed that muscular degeneration occurred more frequently among hypertrophied cattle.

MODE OF INHERITANCE

The mode of inheritance of double muscling is debated

by many authors and geneticists. Some say it is incomplete dominance, others incomplete recessive. Heterozygotes vary phenotypically from almost normal to the almost double muscled form. This phenotypic variability has been interpreted by many as a variable expression of the muscular hypertrophic gene. Matings between homozygotes produce only double muscled calves and matings between homozygous double muscled and normal individuals yield all intermediates. These heterozygotes behave as explained above. The allelic nature of the gene among different breeds is evident since double muscled calves have been sired by Charolais bulls crossed to Red polled, Santa Gertrudis, and Charolais cows. This proves that the gene is same in all affected breeds (Oliver and Cartwright, 1969). The double muscled gene is neither dominant nor complete recessive, even though the genetic nature is characteristic of monofactorial or single gene inheritance. Recessive mendelian genes should only express themselves in the homozygous condition meaning that in the presence of the dominant gene its effects are masked. Simple mendelian dominant and recessive genes produce only normal and abnormal calves phenotypically. The fact that the double muscled gene expresses itself to some degree in presence of the normal gene for muscling suggests that the normal muscling gene is only partially dominant to the double muscled gene (Oliver and Cartwright, 1969). They considered the double muscled gene to be incompletely

recessive with variable expressivity in the heterozygote. Weber and Ibsen (1934) on the basis of their data suggested that the character might be conditioned by a recessive gene but concluded that the mode of inheritance was not satisfactorily established, particularly with reference to modifiers. Wriedt (1929) believed that the character was incompletely dominant and that homozygous dominant cattle were either sterile or very poor breeders. The heterozygotes he found to be fertile. This was in sharp contrast to Weber and Ibsen's findings. Kidwell et al. (1952) postulated that the double muscled gene was incompletely recessive with variable expressivity. Mason (1962) prefers to refer to the muscular hypertrophic gene as having intermediate type of inheritance rather than simple dominant or recessive. The double muscled condition is mediated by a single genetic factor, with only one pair of genes involved. The condition is incompletely recessive (Oliver and Cartwright, 1969). The double muscled gene is likely being given increased selective preference in many beef cattle populations today because of increased emphasis on meat type and carcass cutability. This indicates the need for a team research approach for elucidating the pathogenesis, pathology, and inheritance of double muscling in cattle.

MATERIALS AND METHODS

Six cattle were examined clinically for signs of double muscling; four Angus sired by one bull and two crossbred Charolais cattle. Of the four Angus cattle, a bull and a cow were brought to Kansas State University for clinicopathologic examination. The Charolais crossbreds came from a herd with previous history of double muscling.

The double muscled Angus bull was mated to the double muscled Angus cow and three normal Hereford cows.

RESULTS

Clinical Examination

The most prominent phenotypic characteristic was the stance of affected cattle. The forefeet were more cranial and the hindfeet more caudal, giving a "stretched" appearance. The shaft of the long bones appeared to be shorter than normal and were thinner. The muscles over the point of the hips were enlarged, their size and movement were prominent when the animal walked.

The hindquarters presented an impression of extreme thickness and muscling. The outline of the rump and round from the hip to the point where the quarter joins the leg is convex corresponding to an arc of a circle whose center would be the articulation of the femur and tibia. The tail was attached more cranially than normal with a prominent

head. Enlargement of the rump muscles on both sides of the sacral vertebrae caused a depression immediately above the vertebrae. The muscles outside of the rounds were enlarged resulting in definite intermuscular grooves. These grooves were prominent between the biceps femoris and semitendinosus muscles. A smaller groove or crease was evident between the biceps femoris and vastus lateralis muscles.

Affected cattle appeared to be longer from shoulder to hip and shallower from topline to underline giving the trunk a cylindrical appearance. Shoulder muscles were also enlarged but to a lesser degree. The neck seemed to be shorter, thicker, and arched higher above the withers, due to enlargement of the neck muscles. The head was smaller and lighter than normal. The vulva in affected cows was smaller than normal. Testicles of the bulls appeared to be smaller than those of normal bulls and located closer to abdominal wall. The double muscled defect was more prominent in the bull.

Breeding Trials

The union between the double muscled Angus bull and the double muscled cow produced a healthy calf which was delivered by caesarean section. Of the three calves obtained by crossing the double muscled Angus bull and normal Hereford cows, only one was delivered by caesarean section. Only one of them survived.

The Angus calf had double muscled characteristics. The stance, muscles of the rear quarters and round were characteristic.

The heterozygote calf produced from mating the double muscled Angus bull to the normal Hereford cow, had some double muscling characteristics; stance, round and rump muscular hypertrophy and small head.

DISCUSSION

The mode of inheritance of double muscling has not clearly been accepted. Weber and Ibsen (1934) thought double muscling was due to a recessive gene. They based their argument on the fact that the trait appeared suddenly in a herd in Nebraska. Oliver and Cartwright (1969) believed that the gene for normal muscling was partially dominant to that for double muscling, and that the double muscled gene was an incomplete recessive with variable expressivity.

From the present study, if the double muscling gene were truly recessive, then the heterozygote crossbred calf would have appeared phenotypically normal, but that was not so. Double muscling characteristics such as stance and muscular hypertrophy were evident. The calf was midway between normal and double muscled. The expression of some double muscled characteristics by the heterozygous crossbred calf indicated that the gene for normal muscling

is only partially dominant to the double muscled gene. The homozygous Angus calf had the same double muscled characteristics of its parents. The defect was better expressed morphologically in the male than in females; this supported earlier findings by Oliver and Cartwright (1969).

In conclusion, the double muscled gene can be said to be incompletely recessive with variable expressivity; this makes it easier for selection against the trait. Some heterozygotes, however, appear to be similar to their normal parents. The variable expressivity of the gene is evident in the heterozygous condition varies from near normal to almost the expression of the homozygous double muscled. In cases where heterozygotes appear to be phenotypically almost normally muscled identification becomes difficult and such cattle could be passed for breeding purposes if their pedigrees are not carefully checked or not known. Cattle suspected of carrying the trait should not be used for breeding.

SUMMARY

Six cattle were examined, four Angus and two crossbred Charolais cattle. The four Angus cattle came from the same farm and were sired by the same bull. Two double muscled Angus were mated. The double muscled Angus bull was mated to three normal Hereford cows; only one calf was obtained alive by caesarean section. The homozygous Angus calf was

also delivered by caesarean section. The defect was more severe in bulls than in cows. The heterozygote crossbred calf had some double muscled characteristics.

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Figure 1: Progeny of mating of homozygous double muscled Angus cattle.

Figure 1

II. FACIAL DEFECT IN POLLED HEREFORD CATTLE

INTRODUCTION

Little has been reported about congenital facial bone defects in cattle. Considerable work, however, has been done on non-osteogenic defects of the head and face such as hydrocephalus, paralysis of the trigeminal nerve leading to a twisted face, cerebellar hypoplasia.

Congenital defects may be lethal, semi-lethal or non-lethal. They may severely reduce the economic value or usefulness of calves. What worries animal breeders and veterinarians is the cause of congenital defects. If known, a possible way of eliminating the condition may be tentatively established. If these defects are caused by environmental agents, change in environment, and management may alleviate the problem. The critical period of susceptibility to congenital defects such as cleft palate and arthrogryposis ranges from 40-60 days gestation in cattle. Some bone defects have been found to be caused by alkaloids ("crooked calf syndrome" is an example).

Congenital abnormalities, defects of structure or function present at birth, are caused by hereditary or environmental factors or by their interaction. A hereditary defect is a pathological or pathophysiological result, determined by a mutant gene or chromosomal aberration. Such defects may be lethal, semi-lethal, or subvital.

Most hereditary defects in cattle result from single pairs of recessive genes. Cases of dominant as well as multiple gene action are known (Leipold, 1974). Genetic-environmental-interaction defects are produced by specific environmental factors only on a specific genotype.

In this section six heifers with a specific facial bone defect are reported.

REVIEW OF LITERATURE

Reportedly 0.2 to 3.6 percent of all calves born have congenital defects (Dennis, 1974; Priester et al., 1970). In a survey by Kansas State University involving 588 herds from 1964 to 1972, 1275 congenital defects were reported: 476 (37.3%) skeletal, 238 (18.6%) organs of special sense, 190 (14.9%) muscular, and 155 (12.1%) central nervous origin. Of the skeletal defects, axial skeletal defects (48.3%) were most common (Greene et al., 1973).

Ingestion of certain weeds such as Veratrum californicum results in teratogenic effects in cattle and sheep. Leipold et al. (1977) surveyed congenital defects on Kodiak Island in Alaska and found a variety of defects. Although some were suspected of being hereditary such as slow gait with a synchronous hopping motion of the hindlimbs, torticollis, etc., they could not be classified as hereditary due to lack of breeding records and pedigrees.

Occipito-atlanto axial malformations are rare in domestic animals. Mayhew et al. (1978) described congenital occipito-atlanto axial malformation in Arab foals. They found that the occiput, atlas, and axis were symmetrically malformed and there was occipito-atlantal fusion. The sutures and synchondroses in the cervical vertebrae were not yet fused. The hinged synovial joints at the occipito-atlantal junction were replaced by immovable synarthroses. Fusion was present ventrally but not dorsally across the notch that normally separates the anterior articular process of the atlas. The opposing surfaces of the atlas and occiput were modified. At the atlanto-axial articulation the spinal cord was compressed and distorted laterally and degenerative changes were found in the white matter. The dura had evidence of hemorrhage and fibrosis.

Leipold (1974) described malformations of the occiput, atlas, and axis in cattle. Hereditary basis for occipito-atlanto axial malformation is assumed but not yet determined.

Ojo et al. (1975) reported two cases of facial digital syndrome in Angus cattle. The metacarpal bones were fused with single distal epiphysis, both calves were syndactylous. Facial hypoplasia and kyphoscoliosis were observed. The legs of the male calf were contracted. Neither calf had a distinct mouth, the site being filled with the tongue, that was opened for a good part of its length. The eyes were smaller than normal. There was no central canal in the

spinal cord, tilted ventral median fissure, and abnormally shaped dorsal and ventral horns. Herd history and occurrence of the defect in related calves indicated a hereditary basis. The parents of the affected calves were heterozygotes who received the mutant gene from a common ancestor. Skeletal related defects of the brain were anencephalia, cranioschisis, and encephalocele (Leipold, 1974). The spinal cord was involved with spina bifida and spinal dyraphism. Dennis and Leipold (1972) reported 74 (18.3%) cases of agnathia in lambs out of 401 lambs examined.

MATERIALS AND METHODS

The six calves about one year old were all Polled Herefords. Four of these calves came from the same farm and were sired by the same bull. The calves were tagged (78-2465, 2466, 2467, 2513, 2514, and 2515), euthanatized and subjected to a careful standardized necropsy.

The heads were photographed and radiographed. After the skin was removed they were rephotographed to indicate any deviation of the facial bones in situ. Each head was examined grossly before sectioned (seven sections from each head): 1st section - 2 cms before the molars; 2nd section - 1 cm before the molars; 3rd section - at the 1st molar; 4th section - at the 2nd molar; 5th section - at the 3rd molar; and the 6th section - at the last molar. Two normal calves (78-2465, 78-2515) served as controls.

RESULTS

Controls

Routine necropsy revealed an umbilical abscess 6 x 12 cm in size in one control calf. It was hard and fibrous. All other organs appeared to be normal. Hair balls were found in the abomasum measuring about 7 cm in diameter. Ecchymotic hemorrhages spotted the abomasal wall at the abomasal-rumenal junction. The other control had no lesions.

No. 78-2466

Examination of the head indicated depression of the frontal bone on the right side, the right occipital arch was elevated about 0.5 cm, and bilateral cataracts. The kidneys were smaller than usual.

No. 78-2467

Both frontal bones were depressed adjacent to the orbits. Both eyes had cataracts.

No. 78-2513

Both frontal bones were depressed. The mesenteric lymph nodes were enlarged and the Peyer's patches were hypoplastic. The processes of the sphenoid bone bilaterally protruded into the cranial cavity depressing the cerebral

hemispheres (Figures 1 and 2). The cerebellum was compressed, internal hydrocephalus was present, and both eyes had cataracts. Hair balls were present in the rumen.

No. 78-2514

The facial deviation was prominent close to the occular region with unilateral protrusion of the sphenoid bone into the cerebral hemispheres (Figure 1). The frontal sinus was compressed caudally. The brain had mild internal hydrocephalus. The heifer also had bilateral cataracts. There was enlargement of the mesenteric lymph nodes and hypoplasia of the Peyer's patches.

DISCUSSION

The defect appeared suddenly in a herd. The farmer observed a heifer calf with a misshaped head and neck. Calf No. 2466 acted retarded and was small in size, No. 78-2467 and 78-2514 were not noticed to be affected until examined carefully.

Nothing was found in the literature about this unusual facial defect. Before incriminating a hereditary cause more studies are needed. Environmental factors such as teratogenic viruses and plants should first be eliminated. Maternal effects should also be considered. The occurrence of this defect in a herd suggests it may be inherited.

The relationship of bilateral cataracts and hypoplastic Peyer's patches suggests a viral fetal infection in utero. Sections of affected heads indicated deviation of the nasal septum and gross assymetry (Figures 3 and 4).

SUMMARY

Six Polled Hereford calves from the same herd with a facial defect were examined; four were affected and two were normal. The head deviated from the midline due to depressed facial bones. Processes of the sphenoid bone projected into the cerebral hemispheres. All affected calves had cataracts and two had hypoplasia of the Peyer's patches. The cause was unknown; probably viral.

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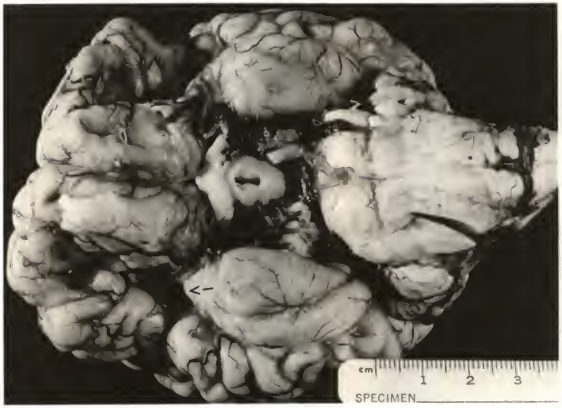
Figure 1: Brain from one of the affected heifers showing indentations made by overgrown processes of the sphenoid bone.

Figure 2: Cross section of figure 1.

Figure 1



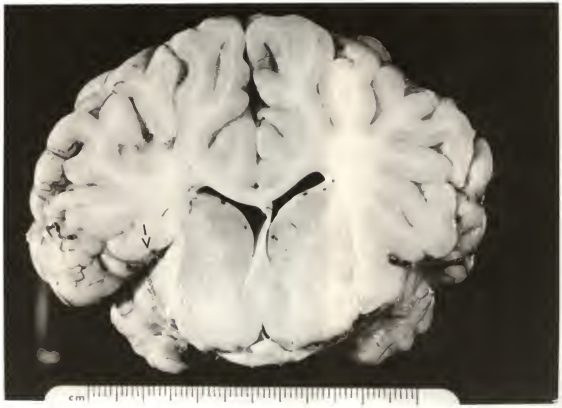
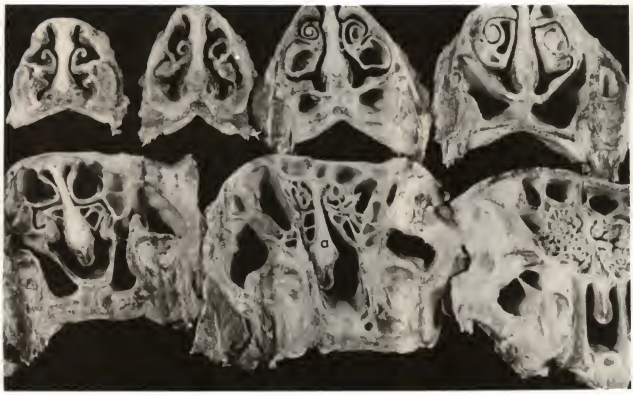


Figure 2

Figure 3: Sections showing deviation of nasal septum.

Figure 4: Normal sections.



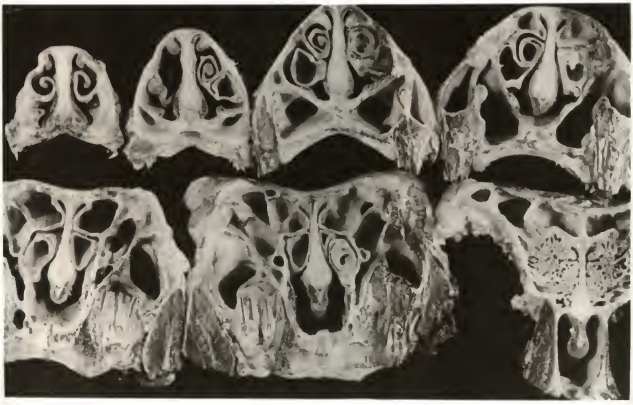


Figure 4

III. TEAT ABNORMALITIES IN CATTLE

INTRODUCTION

The dairy cattle industry relies heavily on udder quality and a strong selection pressure is applied for udder conformation by both breeders and dairy breed associations. Shape, size, and location of the udder determines to a large extent the economic value of a dairy cow. Cows with udder and teat abnormalities are hard to milk and their production is about 30 percent lower than comparable cattle with normal udders under the same environmental conditions (Heizer, 1932).

Polythelia is one congenital and inherited defect of the udder in cattle, goats, sheep, and swine. The majority of dairy cattle breeders in the United States look upon polythelia with disfavor, since the appearance of several teats spoils the symmetry of the udder and also because the glands associated with the extra teats frequently produce small amounts of milk and cause irregularities and difficulties in milking. However, a few breeders believe supernumerary teats are associated with abundant milk and butterfat production.

Genetic analysis of inheritance of teat abnormalities would be valuable to dairy cattle breeders as an aid to selecting their breeding stocks. This should help to decrease the frequency of deleterious genes from the population and possibly their elimination. Initiating such a

study requires knowledge of gene frequencies of the various deleterious traits. Cows with large pendulous udders are more prone to mastitis due to mechanical injury to the quarters and teats. There is also a genetic difference in the susceptibility of the bovine udder to mastitis.

Udder and teat abnormalities occur in goats and pigs and have an adverse effect on reproductive efficiency and ability to care for the newborn. The dairy goat has increased in popularity and in its ability to produce milk, and along with this increase in milk production has come a similar increase in mastitis and udder abnormalities. The dairy goat is an important milk producing animal in parts of the Middle East, Africa, Europe (especially France), and even the United States where goats capable of giving 8 liters per day are becoming common. In France some of the best nannies give more than 2,000 liters in a 10 month lactation period. In swine the big problem is inverted nipples and unevenly spaced teats. Inverted nipples are unproductive while uneven spacing leads to a concentration of teats posteriorly and little milk. High piglet mortality prevails within litters of affected sows. Similar mammary gland defects exist in man and other primates.

Mammary gland defects are detrimental to the dairy industry and the only control is by intensifying selection against them by breeders and breed associations. Farmers undoubtedly are invaluable in helping to control udder

defects by checking the gene frequencies and eliminating these genes from the breeding population. Apart from polythelia other defects of the udder and teats are: teat aplasia, teat fusion, defective teat openings, hypoplastic udders and teats.

REVIEW OF LITERATURE

1. Cattle

At the period in the evolutionary development of dairy cattle, females normally have four milk secreting glands or mammae. Associated with each of these glands is the papilla mamma commonly called a teat or nipple that serves as an outlet for the secretion produced in the mammary gland. It is not uncommon to observe individual cows having a variable number of additional teats, in addition to the four normal teats, some that are orifices of small glands, others opening into one of the normal glands. This condition of polythelia is often referred to as supernumerary teat(s).

Polythelia was observed by cattle breeders long ago but their occurrence in the literature is limited to only twenty-five years. The condition was first reported by May in 1862. In his report he mentioned that one or two small pseudoteats may occur on the rear glands, this he considered favorable. Sutton (1889) also described polythelial conditions in cattle and other animals. Bateson

(1894) stated that normally the cows four teats are of about equal size. Not infrequently there are six teats of which four are large and may be said to be "normal" and the other two are located posteriorly to the others, their size and position vary greatly, sometimes near the other teats. According to Bateson: "I have seen them very high up, almost in the fold between the udder and the thighs. Very frequently, however, there is only one extra teat making five in all, such an extra teat being so far as I know always on one side of the udder." Buchard (1897) compared the number of supernumerary teats occurring in growing cattle with those found on the mammary glands of embryos; 38 percent of all animals examined had abortive teats. Hannerberg (1904) made an extensive study of the frequency of supernumerary teats in various breeds of cattle of European origin. He examined 2,373 females and found that approximately 39 percent had supernumerary teats. Mackenzie and Marshall (1925) examined 276 cows at Cambridge University Farm and at seven other dairy farms for supernumerary teats and observed a 50 percent prevalence. Juler (1927) reported 1,472 observations in dairy cattle in Germany and found that about 23 percent had so-called "after" teats. Emmerson (1928) examined the udders of 370 beef and dairy cattle and found that 44 percent had supernumerary teats. Ivanova (1928) studied about 4,000 cattle in the Soviet Union and found that 800 cows had supernumerary teats. Leroy (1928)

observed among 90 experimental cows representing different breeds in France that only 28 did not possess supernumerary teats. Turner (1930-31) examined 40 female and 46 male bovine fetuses and reported that fifty percent of the females had supernumerary teats while only 17 percent of the males had more than four rudimentary teats. Gifford (1934) conducted a survey in which data was collected on the various breeds of cattle at the Missouri Agricultural Experiment Station. These investigations had begun earlier in 1925 and had been continued at regular intervals. He broadened the scope of his investigation to include 230 herds of cattle from different locations in Missouri.

There are three kinds of supernumerary teats: caudal - at the rear of the udder; intercalary - between normal teats; ramal - ramifications or branches of normal teats. Supernumeraries anterior to normal teats have not been observed. In general each of the three supernumerary types have a rather definite maximum number of possible locations on the udder for attachment. The caudal type may have four; one pair just posterior to normal teats and a second pair posterior to the first pair of supernumeraries and placed high on the rear quarters of the udder but may be near the median line. The intercalary type are attached to the floor of the udder and may occur at various points between normal teats on each half of the udder. The ramal type appears at the base of normal teats or on the side of a teat and may be attached medially, laterally, caudally or cranially to a

normal teat. One or more of the three types of supernumerary teats may occur on a single udder. In the investigation by Gifford (1934) 4,831 cattle were observed including cows and heifers of Holstein-Friesians, Jerseys, and Guernseys, and a number of mixed breeds; 1,249 had supernumerary teats and were classified as caudal, intercalary or ramal. The caudal teats were more numerous than the intercalary or ramal types. Of the 1,249 cows, 1,186 or 94.96 percent possessed one or more caudal teats; this was 24.55 percent of all cows and heifers observed. There were 737 or 15.25 percent of the cows with only one caudal, 431 or 8.92 percent with two caudal, 15 or 0.31 percent with three and only 3 or 0.06 percent with four caudal supernumeraries. The intercalary only appeared on 56 or 1.16 percent of the cows, of these, 49 cows had only one while the remaining 7 had two. The ramal type was found to be relatively rare with only 33 cows or 0.68 percent being affected; 24 had one, 8 had two, and one possessed three. A cow with only three normal teats was observed. The teat on the left half of the udder served as an outlet for milk secreted from the entire half of the mammary gland. Erizan (1931) reported three cows with only two teats. The first had two normally developed teats and the arrangement indicated they formed the anterior pair, both teats functioned normally and there was no indication of supernumerary teats. Emmerson (1928) reported an unusual type of udder with only two teats to drain the four glands.

A number of dairy sires also have supernumerary teats. The patterns in relation to the normal rudimentary teats are similar to the female patterns except that the extra teats are usually located on the scrotum. Of 135 bulls examined by Gifford (1934) 19 or 14.07 percent possessed one or more supernumerary teats. Turner (1931) observed a male fetus from a Hereford dam with only three rudimentary teats.

Gifford stated that the frequency of the polythelial gene was higher in European breeds than in American breeds. Skjervold (1960) noted that some supernumerary teats may be unassociated with glands and that others were orifices of small glands and some may have opened into one of the normal mammary glands. Some of the earlier investigations dealt with possible correlation between milk production and supernumerary teats. Ivanova (1928) concluded from data of 1,385 cattle that supernumerary teats result from a dominant autosomal gene, and that this gene is identical with one that raises milk production 15 percent. Gifford (1934) found no concrete association between extra teats and milk yield. The exact mode of inheritance of supernumerary teats is still unknown. It has been suggested that recessive genes are responsible for the intercalary teats but has not been proven. In Norway 6,268 Norwegian red and white cows were surveyed for polythelia and 40.06% had some type of supernumerary teats (Skjervold, 1960). The frequency was highest for one small nipple (13.53%). Only 0.43 percent

of the cows had more than 2 caudal teats, the frequency of the right as well as the left side dropped steadily from code 0 to code 7. Lush (1950) postulated that the degree of accordance between the right and left side may be used to test the repeatability of the trait. Since neither the genes nor the dominant or epistatic effect change during the individual's lifetime, repeatability should be at least as large as the heritability. In this case the repeatability will be larger because it includes the permanent effects of the environment. Skjervold (1959) calculated a repeatability of 0.52 for the supernumerary trait in 6,268 cattle sampled. The degree of asymmetry and distribution of size and number of caudal teats suggest that this is a case of quantitative inheritance. Skjervold found no correlation between size or number of caudal teats and the frequency of intercalary teats observed. Intercalary teats were found in 1.93 percent of all cows; 1.8 percent of cows with non-caudal teats had intercalary teats. Thirty-four cows or 0.55 percent of all cows had one ramal teat and more had two or more ramal teats. No correlation between frequency of ramal teats and number or size of caudal teats was observed. The sample (6,268 cows) was divided into 25 progeny groups with at least 25 progeny in each. A significant difference in size and number of caudal teats was found between progeny groups by analysis of variance. This difference was also significant when calculated separately for the left and right side

of the udder. Heritability of number and size of caudal teats was separate for left side of the udder which was 0.154, and for the right side, 0.170; average of right and left side was 0.176. Coefficient of heritability was lower than the coefficient of repeatability. No significant difference between intercalary and ramal groups was established.

Supernumerary teats (SNT) have probably deserved special attention due to the unfounded recommendations for surgical removal (Gold, 1943; Moore, 1941; Trimberger, 1958; Yapp, 1959; Kodagali, 1966) being considered as a harmful anomaly that causes generalized impression of unfavorable characteristics. Incidence varies from 6.8% in Hariana Bulls (Singh and Prabhu, 1966) up to 68.88 percent in Normand Cows (Leroy, 1928). Ivanova (1928) found that 251 cows with SNT produced 14.6 percent more milk than 444 cows without the characteristic. Singh and Prabhu working with the Zebu found that milk production was sometimes higher in cows with SNT, sometimes in cows without, and at times no difference in milk production. They also indicated that there was higher frequency of intercalary supernumerary teats. According to Gifford (1934), David (1935), Skjervold (1960), and Weiner (1962) the higher frequency of SNT was in the caudal position. Figueiredo et al. (1975) studied 3,730 females and 94 males from several cattle breeds and various cross breeds. SNT was found in 10.9 percent crossbred males and 19.5 percent in crossbred females, and 35.3 percent in Holstein cows. There was no statistical difference between the occurrence

of SNT on the left or right side of the udder. Only three SNT or 1.4 percent had evidence of milk production in cross-bred mongrel cows, while no functional SNT was found in Holsteins. There was no statistical association between mastitis and occurrence of SNT; however, the average milk production of Holstein cows with SNT was higher statistically than those without. In Britain, SNT are not ablated, and not milked from, they are often regarded as being functionless. Out of 276 cattle examined by Mackenzie and Marshall (1925), 154 had extra teats. They agreed that along with extra teats were extra glands (polymastia) and that these extra glands had no connection with the normal mammary glands. The milk produced by the extra glands, if not drained off, is absorbed into the circulation and lactose recovered from the urine as is seen during the drying period when the young are being removed. In one cow examined by Mackenzie and Marshall (1925), the extra gland in which milk had not been drawn was larger than the normal gland and had evidence of degeneration. They confirmed the assertion that 50 percent of cows in Britain had extra teats. Heizer (1932) discovered an abnormality of the udder in which the udder was poorly shaped with two quarters and teats on the right side and only one teat on the left and the left was almost the same size as the right half. Heizer examined the pedigree back to an earlier ancestor with the defect. The parents of this ancestor were apparently normal phenotypically. He recorded the abnormality only in Guernseys, and evidence

suggests simple mendelian recessive as the mode of inheritance as the condition appeared in progeny of phenotypically normal individuals. By the time Heizer wrote his paper it was being planned to prove homogozygosity by crossing an affected male to an affected female. Johnson (1945) reported fused teats in a herd of Hereford cows in South Dakota. Four of the fused teats were on the right side and one on the left in five cows. The cow with the defect on the left had a full sister with defective right teats. No rudimentary teats were observed in males. The pedigree chart of the herd was studied and it was established that when related sires were used, the defect increased dramatically; removal of related sires from the herd resulted in a decrease of affected animals. This established a simple monofactorial recessive mode of inheritance. Heterozygotes were found not to have normal spacing between teats. Lush (1950) presented evidence that inheritance plays a large part on whether an affected cow developed mastitis or not. The ability of a cow to combat mastitis depends, in part, to the anatomical structure of the mammary gland since anatomical structure is highly hereditary. Culling bulls whose progeny are known to have had a high incidence of mastitis reduces mastitis in a herd. Heritability estimates based on mastitis may measure genetic factors controlling seriousness of the disease in infected animal rather than factors controlling animal resistance against infection (Schmidt and Van Vleck, 1964). According to

Young et al. (1960) there is a high genetic correlation between leucocyte count and clinical mastitis that suggests that many genes influencing leucocyte count influence clinical mastitis. Cows with pendulous udders are more prone to mastitis.

2. Goats

Udder problems of dairy goats are similar to those of the dairy cow. Anomalies of the goats udder are common and may be hereditary but the hereditary studies are inconclusive. Smith and Roguinsky (1977) reported that teat ablation takes place shortly after birth. Frequently abnormal teats are fused or have two orifices at the tip. The American dairy goat association advised dairy goat owners to remove affected nannies and bucks, thus applying a strict selection pressure against these teat defects.

3. Pigs

Arrangement of nipples in swine is controlled by heritable factors. There are many variations in nipple shape, some of which do not affect functional efficiency; some such as "cone-shaped" and "stunted" nipples affect function. The stunted are too short to be of practical use. The "mushroom", "mulberry", and "fissured" types are of ample size. The "inverted" and "hollow" types are described as being crater-like because of the cavity they form on the

mammary surface. Total suppression of nipples in swine is common (Heizer and Turner, 1932, 1930). Creighton (1877) reported complete absence of mammae (amastia) in humans as one of the rarest congenital deformities. Congenital retraction of nipples is common in humans. Defects may also occur in the form of openings along the side or base of nipples. Openings at base of nipple in humans has been observed by Williams (1891). Inverted nipples in swine are of considerable importance because they are non-functional and may occur in large numbers in the same sow. They cannot be extended by manipulation. The defect is inherited and according to Nordby (1934) the mode of inheritance indicates recessiveness. The hereditary influence functions probably as an inhibitor to normal nipples. If this proves to be the case farmers should select against the condition by avoiding using affected boars and sows for breeding purposes.

4. Sheep

Ewes having more than two nipples are observed frequently. Graham Bell (1904, 1912, 1923) worked with multinippled ewes and selected for the trait. He started by collecting a multinippled flock of ewes, buying his sheep from non-descript farm flocks in the vicinity of his home in Nova Scotia. He discovered that the incidence was readily affected by selecting the breeding sires and dams and by selecting for multiple nipples, the gene frequency increased.

This trait has little practical value in sheep husbandry either by association with high fertility or increased milk production (Phillips et al., 1946). Using rams with two rudimentary nipples reduced sharply the frequency of the gene.

Castle (1924) through analysis of Bell's work concluded that multiple nipples (supernumerary teats) in sheep are inherited. Bell (1904, 1912, 1923) discovered that his multiple nippled herd produced only progeny with the trait; this led him to conclude that a recessive mode of inheritance was responsible. The particular grade of multiple nipples borne by the parents is inherited by their progeny (Castle, 1924). The low nipple number has a tendency to dominate in crosses (Castle, 1924). By analyzing Bell's work Castle (1924) reported that twinning was not hereditary but was influenced by maternal age. Yearling ewes rarely have twins. A greater number of nipples often found in sheep depends upon a dominant gene (N) which increases the basic number to about four or more. This gene has variable expression ranging from appearance of only one additional nipple to two (Wassin, 1931).

The size of the additional nipples may depend on the action of other genes, for example meat breeds of sheep possess the most highly developed additional nipples. There is also a gene (I^N) which inhibits nipple number (N) (Wassin, 1931). Additional nipples in sheep are located cranial to the basic pair and they are rarely functional (Wassin, 1931).

He also reported that there was no linkage between the genes for wattles, piebaldness, and multiple nipples. Through his years of selection Bell (1904-1923) developed ewes with four functional teats. Miller (1934) produced ewes with four nipples from a flock of 12 ewes (all with four functional nipples) using a ram with four rudimentary nipples. These ewes had no special advantage for milk production and lamb weights over those with two nipples. Ritzman (1933) conducted breeding trials in which Rambouillet were crossed to the four-nippled ewes of Graham Bell. The idea was to retain the physical characteristics of the Rambouillet and at the same time gain the high fecundity of the four-nippled ewes, largely through twinning and development of four functional nipples.

MATERIALS AND METHODS

In a Polled Hereford herd a fused teat defect was observed in one line of cattle. All affected cows were sired by the same bull or his son.

RESULTS

The first cow had fused teats on the right half of the udder (Figure 1) and the second had fused teats on the left side. One cow had agenesis of the right hindquarter and another had a fused teat on the left side.

DISCUSSION

Heizer (1932) examined a herd of Guernseys with fused teats. Examination of the pedigrees of affected cows revealed that the great grandparents were phenotypically normal. This indicated that the trait was recessive; the gene being carried by both parents. In the herd studied here the defect suddenly appeared and was considered to have been introduced by both parents. The fact that heterozygotes did not show the trait in the herd indicated the recessiveness of the trait. In conclusion, fused teats or synthelia is probably a simple autosomal recessive trait.

SUMMARY

A specific line of a purebred Polled Hereford herd had synthelia. Four cows were found to have the defect at the time of examination. Pedigree examination indicated the defect was introduced by both parents. It was concluded the trait is probably inherited as a simple mendelian autosomal recessive.

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Figure 1. Fused teat on right udder half of a purebred polled Hereford (arrow).

IV. UMBILICAL HERNIA IN HOLSTEIN CATTLE

INTRODUCTION

Umbilical hernia in cattle is little understood. Some consider it to be genetic and other incriminate environmental causes. Umbilical hernia has been misjudged by ranchers and some veterinarians; they often regard it as a simple problem that can be dealt with surgically. The dairy industry has tended to regard umbilical hernia to be of little significance until its genetic inheritance was revealed. Bulls with severe umbilical hernia are infertile as they are unable to serve cows.

Umbilical hernia may be spread rapidly by artificial insemination with semen of affected bulls. Semen from a carrier bull can spread the defect to several of its progeny, and that's why umbilical hernia should be selected against just as any other undesirable trait. Most progeny testing programs in most artificial insemination stations do not, as a rule, apply a strict selection against umbilical hernia. In cases where the hernia is not large, dairymen and ranchers tend not to report the defect. Thus, most hernia cases are not reported; breeding bulls purchased from such herds could be carriers and thus spread the defect to unaffected herds. The fact that umbilical hernia has not appeared in large numbers of herds supports the fact that it is probably a polygenic character.

REVIEW OF LITERATURE

Little work has been done on umbilical hernia and as such is little understood. From 323,961 cases submitted to 12 United States and Canadian veterinary colleges involving five species (cattle, horses, swine, dogs, and cats) 1,315 had umbilical hernia and 57 scrotal hernia (Priester et al., 1970). This survey revealed that females were at excess risk for congenital umbilical hernia in cattle, horses, and dogs. In swine and horses, males were at excess risk for inguinal hernia. In general, umbilical hernia was about 4 to 12 times more common than inguinal hernia except in swine, where the frequency per 1,000 patients for both defects exceeded the rates in other species.

Cattle: Holstein-Friesian cattle have been found to have an unusually high rate for umbilical hernia. This was further demonstrated by the case study submitted by the 12 veterinary colleges in North America (Priester et al., 1970). The Angus and Hereford cattle are reported to have a significantly lower frequency of umbilical hernia. In cattle the defective umbilicus is frequently hereditary (Murray, 1923). The higher frequency in larger animals indicates that they have a more severe form of involvement generally than smaller animals. Minor hernias are usually under reported. The excess risk for umbilical hernia in Holstein-Friesian cattle agrees with earlier studies (Warren and Atkeson, 1931; Gilman and Stringham, 1953;

Angus and Young, 1972). The low incidence of umbilical hernia in Hereford and Angus breeds reported by the survey has not been supported by earlier work. This low incidence in the two breeds may be due to visual selection in beef versus record selection in dairy. Transmission has been postulated as being dominant, dominant with low penetrance or recessive. Umbilical hernia could also occur sporadically (Hayes, 1974).

The cause of congenital hernias has been attributed to normal anatomic variations, polygenic inheritance and infectious disease. Direct spread of hernias, the high incidence and absence of relationship between males and females suggested a dominant mode of inheritance (Angus and Young, 1972). The assumption by Angus and Young was based on an experimental research in which bulls descending from a certain phenotypically normal bull were mated to phenotypically normal cows. The distribution from small to large hernias suggested that the gene responsible for umbilical hernia is not penetrant (Angus and Young, 1972). Hernias may also be due to recessive or dominant genes with low penetrance (Gilman and Stringham). Cases of sporadic hernias are probably due to environmental factors. Some also result from one or several recessive genes with penetrance controlled by threshold environmental effects or modifying genes (Lerner, 1954). Hernias may thus be inherited in various ways. Varied inheritance of umbilical hernia is due to several genes controlling umbilical ring closure

mutating in differently; some dominant and some recessive. Environmental factors preventing ring closure could also produce phenocopy hernias indistinguishable from genetic hernias. In Iowa over a period of five years in an experimental station umbilical hernia occurred in 1 cow, 122 pigs, 2 dogs, 1 horse, 1 sheep, and 2 cats. This suggested that the defect in cattle was either infrequent, pathologically unimportant or that it disappeared naturally at an early age. Warren and Atkeson (1931) in Idaho supported the suggestion that umbilical hernia was inherited in a simple Mendelian ratio of 1:1 among males and that it was sex-limited. According to this hypothesis, it appeared that one of a pair of autosomes carried a sex-limited dominant factor for hernia.

Swine: In the United States, 0.6 percent of male hogs and 1.16 percent of females had umbilical hernia, and about a million herniated pigs, approximately half umbilical and half inguinal, are born each year (Warwick, 1926). In the dominant umbilical hernia, penetrance is high but incomplete; this facilitates identifying affected animals early. Inguinal hernia is more common than umbilical hernia but this can be disputed as umbilical hernia in adult hogs old enough for sale are not reported. In contrast inguinal hernia occurs in young piglets and are reported for surgical repair. Youatt (1847) believed hernia in pigs was inherited but was unable to comment further because of little attention being paid to the condition at that time. Warwick

(1926) concluded from the data he obtained that inguinal hernia in swine was inherited as a recessive and commented that males were more susceptible than females because of the anatomical differences.

Horses: Quarter horses have a high rate for umbilical hernia and females have a higher rate than males (Hayes, 1974). The hereditary nature of umbilical hernia in horses was reported by Dollar (1912).

Dogs: Five canine species have a high rate for umbilical hernia (Airedale, Terrier, Basenji, Pekingese, Pointer, and Weimaraner). Umbilical hernia in dogs has a sporadic pattern and an occasional concentration within families (Phillips and Felton, 1939). The Basenji breed is predisposed to both umbilical and inguinal hernias (Fox, 1963). The high risk of umbilical hernia in bitches and mares was uniform throughout their respective breed categories that suggested some common sex-linked or sex-influenced factor indigenous to each species.

Cats: Cats have similar breed rate for both umbilical and inguinal hernias as dogs.

Man: Stengel and Fox (1921) considered hernias in humans to be due to strain or muscular weakness as they occur more frequently in males than females. Davenport (1911) writing about hernia in man stated that such weakness or liability to hernia is inherited. There is insufficient data to determine how with certainty. Davenport (1911) reported that hernias in man are inherited as

dominant. Many writers such as Hobday (1914) have referred to hernias as being inherited. Accidents, digestive disturbances, excessive pressure, and muscular weaknesses have been incriminated as causes of hernia.

Development of Umbilical Hernia: Umbilical ring closure is controlled by a number of genes (Angus and Young, 1972), a defect in any could result in non-closure of the ring leading to herniation. During the early stages of development, the intestines rest partly in the umbilical cord because of their early rapid growth. Later the body walls enclose this area and the umbilical ring contracts forcing the intestines back into the abdominal cavity. If this does not occur, umbilical hernia results. A cross section of the umbilical cord in the amniotic portion revealed the amnion as the investing membrane.

Hernia Repair: A calf with the umbilicus positioned ventrally of a pendulous abdomen is more predisposed to umbilical hernia. Surgery has almost always been the choice of treatment. Other methods such as elastic bandage and wooden clamps have been used for repairing hernias. With both techniques, the umbilical hernia must be reducible. The bandage is usually applied at 6-8 weeks of age with the calf remaining in a standing position. The contents of the hernia are first returned to the abdominal cavity. The method has been successful (Anderson, 1976). In the second method, a wooden hernia clamp made from a 3.175 cm to 3.81 cm dowel stock and 25.4 cm to 30.56 cm

long, depending on the size of the hernia is split in two and held by elastic bands. The contents of the hernial sac are returned to the abdominal cavity, the two halves of the clamps are placed parallel to each other on both sides of the hernial sac and held tightly by elastic bands. The clamps are further held in position by strong Vetafil sutures, both to the front and back of the hernial ring. The sac eventually necroses and falls off and the hernial ring closes by scar tissue.

MATERIALS AND METHODS

Animals: Two related Holstein steers with umbilical hernia were examined and euthanatized and immediately necropsied.

Necropsy Procedures: The steers were subjected to a standardized necropsy with special attention being paid to the umbilical hernia and ventral abdominal wall. Sections of hernial ring and sac and adjacent abdominal muscles were fixed in 10% buffered neutral formalin, trimmed, embedded in paraffin, cut at 6 microns, stained with hematoxylin and eosin, and mounted on glass slides.

Pedigree Analysis: Analysis of the common sire of the two steers was drawn and evaluated.

RESULTS

Palpation of the hernia revealed a soft sac without

obvious contents (Fig. 2). The skin was carefully removed, the sac was exposed and opened to expose a small cavity containing peritoneal fluid but no intestines or omentum. The sac was removed to expose a hernial ring about 3 cm in diameter. The hernial ring was situated slightly caudal to the middle of the linea alba with the abdominal muscles forming the lateral and caudal hernial wall.

Histopathological examination of the hernial sacs and rings and adjacent abdominal muscles and connective tissue revealed no significant findings.

No gross or microscopic lesions were found in any of the other body systems.

The common sire (H-97) had 32 offspring including 11 with umbilical hernia; 9 females and 2 males (Fig. 2). The diameter of hernias in the females ranged from 5 to 10 cm in contrast to 3 cm in the steers. H-97 was not inbred and was mated to apparently unrelated normal females.

DISCUSSION

The umbilical ring normally closes before birth. Closure is reported to be influenced by several genes that may mutate dominantly or recessively (Angus and Young, 1972). Non-closure may also be influenced by environmental factors.

The fact that H-97 sired 9 herniated females and 2 males from unrelated cows suggests that the mode of inheritance of umbilical hernia in Holsteins is unknown. The

trait might be polygenic. The mode of inheritance in males may be dominant (Angus and Young, 1972) and questionable in females.

That umbilical hernia has not appeared in a large number of herds supports a dominant mode of inheritance. If it was a recessive it would become widespread in spite of selection and would present a problem of control. Dominant inheritance in males would enable cattlemen to control umbilical hernia by carefully selecting bulls for natural and artificial service. Breeders should avoid using herniated bulls and surgical treatment should be restricted to non-breeding cattle.

SUMMARY

Pedigree analysis of a Holstein bull siring 11 calves with umbilical hernia (9 females and 2 males) from a total of 32 calves from unrelated grossly normal cows. The diameter of umbilical hernia in females varied from 5 to 10 cm, in males 3 cm. The mode of inheritance is questionable in females but may be dominant in males. Necropsy of the two affected males revealed a simple, uncomplicated hernia.

Control of umbilical hernia in cattle appears to be possible by carefully selecting breeding bulls and by not using surgically corrected herniated bulls.

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Figure 1: Pedigrees of offspring affected with umbilical hernia. Affected calves were sired by bull IV-1.

Figure 1

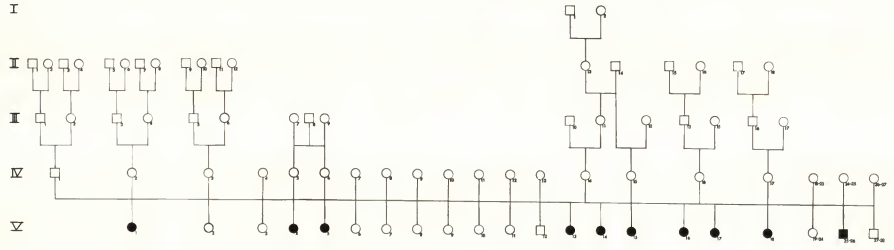

Figure 2: Herniated calf. Progeny of bull IV-1.

Figure 2

V. BOVINE CONGENITAL DEFECTS: GENERAL DISCUSSION

At least three of the four defects studied were genetic. Double muscling is due to an intermediate mode of inheritance. Oliver and Cartwright (1969) reported that double muscling was due to an incompletely recessive gene with variable expressivity in heterozygous cattle. Examination of muscles of affected cattle revealed lack of fat as seen on the live animal by the presence of deep grooves or creases. Palatability of double muscled meat varies little from that of normal meat (Carroll et al., 1978); the only difference being preference. Italians and French prefer double muscled meat with its less marbling, while Americans prefer marbled muscles. Cattle with this trait should be selected against because of the adverse effects on reproduction and production. Teat abnormalities being due to autosomal recessive genes are hard to select against as the heterozygotes go unnoticed.

The only selective method is progeny testing bulls or by sire daughter matings. To prove a bull 99.9% free of any defect seven normal progeny should be produced by crossing to defective cows. Sire daughter matings require 35 daughters; if any resulting progeny are defective, the bull is heterozygous.

It has not been determined whether the facial defect is hereditary or not, as there are no reports in the literature. More work is required before such conclusions can be drawn; for the moment we can only say it is a congenital defect.

Umbilical hernia may be dominant in males can be detected easily and this explains its low incidence among herds. The mode of inheritance in females is questionable. Of 32 calves, 9 heifers were affected in contrast to 2 bulls. Umbilical hernias may be controlled by many genes.

STUDIES ON BOVINE CONGENITAL DEFECTS

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ABSTRACT OF A THESIS

submitted in partial fulfillment of the
requirements for the degree
MASTER OF SCIENCE

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KANSAS STATE UNIVERSITY
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1979

Four congenital defects of cattle including three genetic, were studied.

Six animals, four Angus, and the remainder Charolais crossbreeds, were examined for double muscling. An Angus bull and cow homozygous for the trait were crossed and the resulting calf was double muscled with almost all the features of its homozygous parents. The Angus bull was also crossed to three normal Hereford cows. One calf was viable, one was born weak and died, and the third was stillborn. The heterozygous calf had some double muscling characteristics that indicated the variable expressivity of the gene.

A facial polled defect was reported from a herd in Iowa. The defect appeared de novo, in the herd. Since this was the first time that the defect was observed no etiologic conclusions could be drawn. Depression of the facial bones was prominent in the four affected cattle. In two, there was protrusion of the sphenoid bone processes into the cerebral hemispheres. Internal hydrocephalus was present in two and cataracts in all four cattle.

Four animals with fused teats were reported in a herd of purebred Polled Herefords. The condition was found only in a particular line. The pedigree examination indicated that the trait was introduced earlier by some old grandparent of the herd. Carrier cows appeared to be normal, indicating the recessiveness of the trait.

A bull (H-97) sired 32 calves from unrelated cows; 11 had umbilical hernia. Of the 11, 9 were heifers. The fact

that cows mated to bull H-97 were unrelated to themselves or the bull indicated that umbilical hernia was due to complete dominance in the male, though the dominance may be incomplete or the gene may have low penetrance. An earlier assumption that carrier bulls were heterozygous and cows homozygous suggested a high gene penetrance that is probably incomplete. The mode of inheritance in females remains questionable.