A STUDY OF LETHAL AND SEMI-LETHAL ASPECTS IN A FRIESIAN HERD AT THE GEMMEZA EXPERIMENTAL STATION.

I.—Stunted or Vestigeal Tail

By

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SUMMARY

Between the period 1956-1959 cases of abnormalities appeared in a Friesian herd at the Gemeza Experimental Farm. The conditions reported here appeared in 17 purebred Friesian calves as well as 72 crosses with the native cattle expect for the halfbreds. No associations between the number of abnormal animals and either the type of breed or sex was found. When these abnormalities were categorized, no association was demonstrated between the frequency of type of abnormality and either the type of breed or sex.

The condition of stunted or vestigeal tail constituted 17.2% of the total abnormals and 4.2% of the total calves born. It has been shown that this condition is due to a single autosomal recessive gene. Facts in favour of this view may be breifly listed as follows: (*a*) Some bulls produced affected calves from certain dams, whereas others in the same herd and mated to the same dams did not. (*b*) Sires and dams of affected calves are frequently related. (*c*) Association of the defect with inbreeding in many cases. (*d*) Limitation of affected calves to particular sire progenies.

INTRODUCTION

Lethals or semi-lethals are always a source of economic loss to individual breeders. There effect occur at any time, during embryonic fetal, or post-natal time. Cases of hereditary abnormalities in cattle were investigated by several authors. Dropsical calves were observed by Donald *et al* (1952) in an Ayrshire herd and attributed this abnormality to a single autosomal recessive gene consistent with Mendelian expectations. Taillessness in cattle was reported by Gilmore and Fechleimer (1957) and Huston and Wearden (1958). The former authors noted that same tailless animals had malformed or missing sacral vertebrae, weak lions, and high pinbones and genitalia. The latter authors discribed the occurance of taillessness in the females of the dairy and beef breeds as accompanied by anterior and dorsal misplacement of the vulva and anus, concave rump and stiffness of the rear legs, whereas in males of the same breeds, the defect may be accompanied by misplaced or atretic anus, concave rump, stiffness of the rear legs, but the malformation of the vertebral column appeared to be variable.

Congenital spasms in Holstein cattle were reported by Johnson *et al* (1958) and in Herford cattle by Gregory *et al* (1962). Both authors have presented the evidence that this condition is inherited as a single autosomal recessive, the occurance of which in the progeny ceased when bulls known to be carriers were removed from the herd. Another abnormality, the double cervix condition was studied by Sittman *et al* (1961) who postulated that the inheritance of this sex limited defect is conditioned by a single recessive gene with low penetrance and variable expressivity. Albright (1960) described the occurance of multiple lipomatosis in two herds of the Holstein breed. The condition was first observed at the age of 3½ years and progressed extensively as the animals became older. In some instances the fatty like tumors caused serious malfunctioning of the mammary system resulting in lowered milk production.

It should be noted, however, that not all abnormalities are genetic in origin. Some are caused by environmental factors such as nutrition, vitamin defficiency and diseases.

SOURCE OF DATA

The conditions reported in this paper occured in a Friesian herd and its crosses with the native cattle at the Gemmeza Farm in the Central Delta. The foundation stock of the present herd consisted of few importations from Palestine in 1931 of non-pedegreed animals whose previous performance was unknown. This small herd was supplemental by two other importations, the first from South Africa in 1946, and the second from Holland in 1948. Contemporary to the Friesian Stock, a native herd of Damietta cows was kept for the purpose of grading and also acting as a control group. As a closed herd, inbreeding was carried out to a certain degree. Since 1956 and up to 1959 cases of congenital malformation appeared in the pure and all the grades from the second to the seventh generation (3/4 F up to 127/128 F). No abnormalities occured in the halfbreds, Some calves had more than one abnormality so that more abnormalities were expected than the total number of calves. From the pedegree sheets, these affected calves were traced back to sires ; Hollandaise, imported from Holland; Merston and Winverse from South Africa; and Ibn Nena whose dam Nena was imported from Palestine.

CLINICAL ASPECTS

The conditions reported here were described by the experts of the station and the authors. Post mortum examination of some malformed calves was performed by Dr. Hans Jorgen Hansen, the visiting professor at the faculty of Veterinary Medicine, Giza. These conditions were divided into the following five classes :

(1) Cases where calves showed a stunted or vestigeal tails : The post mortum examination as described by Dr. Hansen for eight calves is as follows, "the stunting tail reminds in its appearance that of the goat. In addition, three of the cases showed abscence of the anus with communication between rectum and vagina. In two cases the picture also included the underdevelopment of the lumber spine. Others suffered from congenital dilation of the brain verticles, double vagina and cervix and hypoplasia of the ovaries".

(2) Calves born weak with twisted legs, unable to stand (Lameness in hind limbs) or nurse and die within few days after birth. Some of these calves were born blind.

(3) Calves were born blind.

(4) Still born calves with no other observable defects.

(5) Cases where calves showed amputated limbs, cleft palate, under development of one eye (microphtalmia) and of the ears (Microtia) and the underdevelopment of the alimentary canal, This condition is termed (Acroteriosis Congenita) and all the calves were born dead (Fig.1).

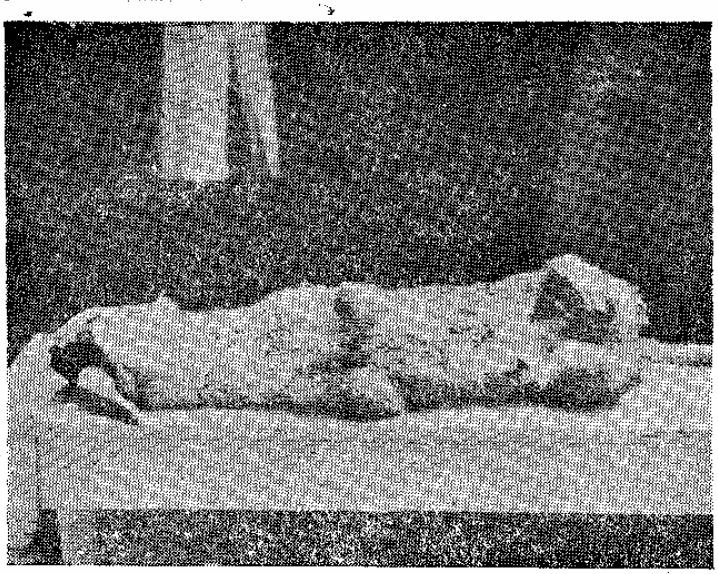

Fig. 1.—See Text.

ANALYSIS OF THE DATA

The chi square test of independence was performed using Yates correction (Snedecor, 1950). The hypothesis of independence was rejected if the differences between the observed and calculated values were significant and an association between the factors studied was assumed. The factors tested were of genetic in origin, breed, sex and their association with the type of abnormality. Since the affected crossbred calves appeared in each generation was small, all crossbred animals were grouped under one category to be matched with the pure Friesian calves. If possible the coefficient of inbreeding was calculated for each calf.

RESULTS AND DISCUSSION

The proportion of abnormal animals by breed (pure Friesians and crosses) are presented in table (1). Of a total number of 383 calves 89 or 23.2% were abnormal which is almost the same percentage obtained for each of the two breeds. The chi square test failed to demonstrate any association between the number of abnormal animals and type of breed.

TABLE 1.—Number and Percentage of Abnormal Animals by Breed

	Friesian		Crosses		Total	
	No.	%	No.	%	No.	%
Abnormal	17	22.4	72	23.5	89	23.2
Normal	59	77.6	235	76.5	294	76.8

X^2=.0401; df=1: P=.8—.7

Inspection of table 2 shows that there is no association of either the Friesians and the crosses with any specific type of abnormality, *i. e.*, there is no definite type of abnormality associated with a specific breed. Nevertheless, only 29.4% of the total abnormalities in the Friesians were weak, twisted legs, whereas the figure is 46.7% for the crosses. On the other hand the total number of still born calves in both breeds was 23 of which 5 (21.7%) were Friesians and 18 (78.3%) were crosses.

TABLE 2.—Proportion of abnormalities in each class by breed

Category of Abnormality	Friesian		Crosses		Total	
	No.	%	No.	%	No.	%
1. Stunted or vestigeal tail	2	11.8	14	18.4	16	17.2
2. Weak, twisted legs	5	29.4	35	46.1	40	43.0
3. Blind	3	17.6	8	10.5	11	11.8
4. Still born	5	29.4	18	23.7	23	24.7
5. Acroteriosis Congenital	2	11.8	1	1.3	3	3.2
Total	17	100.0	76	100.0	93	100.0

X^2=3.0400; df =4; P=.6—.5

Sex was the second genetic factor tested. From tables 3 and 4 the statistical analysis failed to demonstrate any association between sex and either the number of abnormal animals or frequency of type of abnormality which means that the two sexes had approximately the same proportion of affected calves.

TABLE 3.—Number and Percentage of Normal and Abnormal Calves by sex

	Male		Female		Total	
	No.	%	No.	%	No.	%
Abonrmal	52	26.3	41	21.7	93	24.0
Normal	146	73.7	148	78.3	294	76.0
Total	198	100	189	100	387	100

X^2=1.097 ; df =1; P=.3—.2

TABLE 4.—**Frequency of Abnormalities in Each Category by sex**

Category of Abnormality	Male		Female		Total	
	No.	%	No.	%	No.	%
Stunted or Vestigeal tail	9	17.3	7	17.1	16	17.2
Weak, twisted legs	19	36.5	21	51.2	40	43.0
Blind	4	7.7	7	17.1	11	11.8
Stil born	18	34.6	5	12.2	23	24.7
Acroteriosis congenita	2	3.9	1	2.4	3	3.2
Total	52	100.0	41	100.0	93	100.0

$X^2 = 7.7998$; df = 4 ; $P < .05$

It will be seen also from table 3 that out of 387 calves, 93 or 25% were abnormal. The sex ratio is 55.9% males and 44.1% females.

Genetical aspects of the condition " stunted or vestigeal tail"

The stunted or vestigeal tailed calves constitute 17.2% of the total abnormals (Table 4) and 4.2% of the total calves born. Table 5 shows the incidence of this abnormality among calves whose pedegrees are illustrated in Fig. (2). Thirteen affected calves were sired by bulls A and B. These calves trace back to two or three generations to three male ancestors : Hollandaise (I), Ibn Nena (III) and Winverse (IV). Bull I imported from Holland, produced three daughters (3, 4 & 5) and two sires (A and II) which were known to produce abnormal calves. When sire A was mated to two of his half sibs they proudced abnormal calves. On the other hand, when this bull mated unrelated cows to him (1, 6 & 7) abnormal and normal calves were produced. It could be concluded, therfore, that the occurrance of abnormality in most cases is associated with inbreeding which creates the presumption that the condition is inherited. In all cases, non of the parents of abnormal animals showed any of the stunted or vestigeal tails. In other words, the prents were carriers but phenotypically normal. There fore, the condition would have to be due to a resessive gene. As both sexes showed the abnormality, then it could not be sex linked.

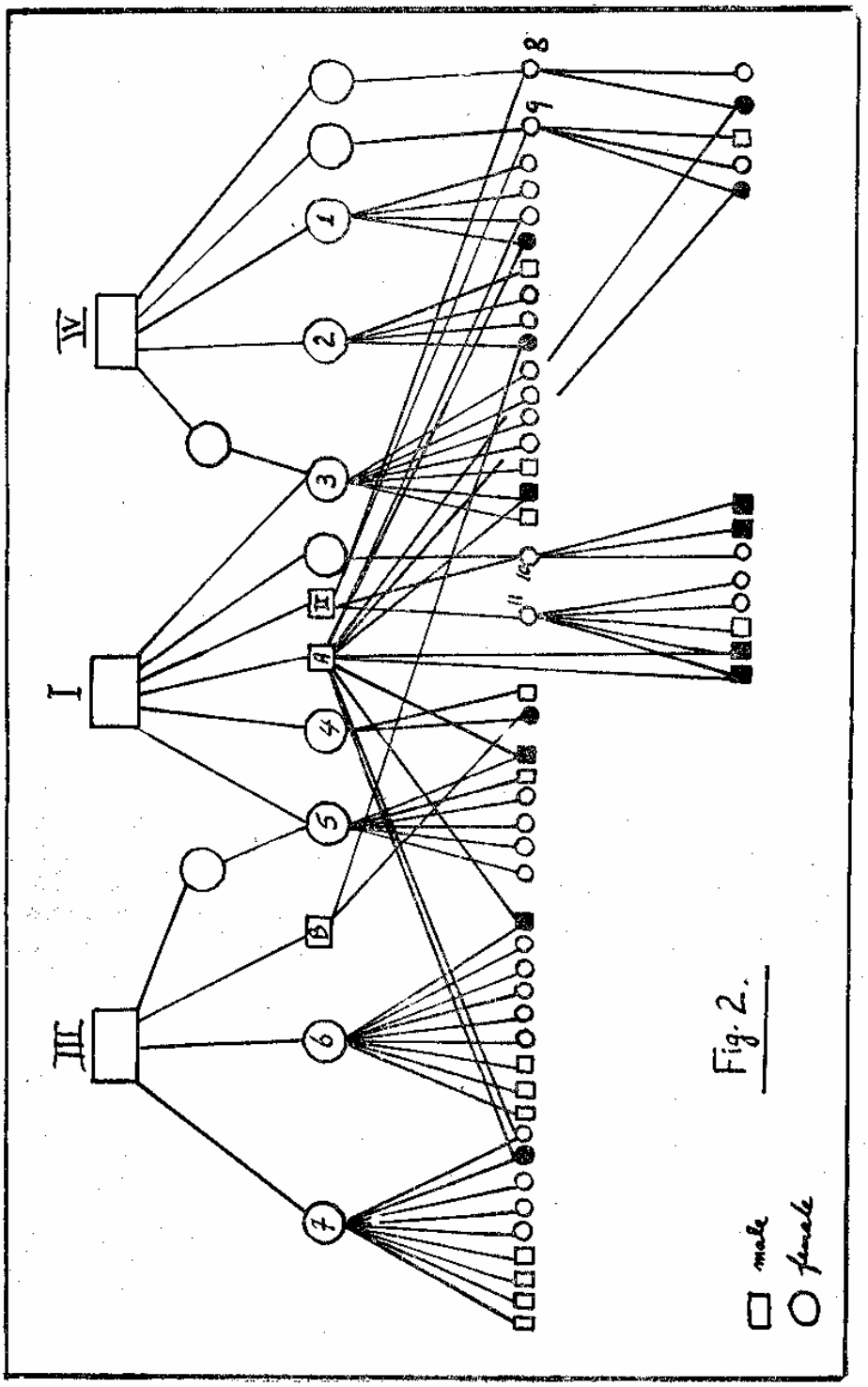

Fig. 2.—Pedegree showing relationship of Bulls and Cows Producing Stunted or Vestigeal Tailed Calves.

TABLE 5.—**Progeny of Daughters of Sires I, II, III and IV Known to Produce Stunted or Vestigeal Tailed Calves**

Sires of Cows	No. of daughters	Sires of calves	Stunted or Vestigeal		Normal		Total
			M	F	M	F	
I	3	A & B	2	1	—	—	3
(Hollandaise)	15	A & B	—	—	6	9	15
II	4	A, C & D	4	2	—	—	6
(Nardi)	18	A & B	—	—	10	8	18
III	2	A	1	1	—	1	3
(Ibn Nena)	5	A & B	—	—	2	4	6
IV	2	A & B	—	2	—	1	3
(Winverse)	8	A & B	—	—	5	3	8
Unknown	3	A & B	2	1	—	1	4
TOTAL	14 produce affected and normal calves 46 produce normal calves.		9	7	23	27	66

In Table 5 each set of half sib daughters produced abnormal and normal calves when mated to the same sires. Altogether, there are 66 calvings from fifty daughters of transmitting bulls mated to transmitting bulls and 16 abnormal and 50 normal calves were found. The expected ratio would be seven normal to one affected which disagrees with the obtained ratio. No allowance was made for cows culled which would have had the chance to produce calves, had they been in the herd.

Further information may be added to table 5. The 46 daughters that produced normal calves from sires A and B, have also produced 35 normal calves from other sires known to have not produced affected calves. This postulates that these 46 daughters are homozygous. Finally as no abnormalities occured in the halfbred calves, their native dams would be expected as being homozygous. The incidence of this condition had ceased when all bulls which sired effected calves were slaughtered.

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دراسة بعض العوامل المميتة وشبه المميتة فى قطيع من الفريزيان بمزرعة أبحاث الجميزة

أولا ـ الذيل الأثرى

صلاح برادة ، عباس الاتربى وصادق فهمى

الملخص

فيما بين سنة ١٩٥٦ و ١٩٥٩ درست حالات التشوه التى ظهرت فى قطيع من الفريزيان بالجميزة . وقد ظهرت الحالات فى ١٧ عجل فريزيان نقى و٧٢ خليط فريزيان وبلدى . ولم يوجد ترابط بين المشوه والنوع أو الجنس . وكذلك بين نوع المشوه والنوع والجنس وكانت حالة الذيل الأثرى تمثل ٢ر١٧٪ من جملة حالات التشوه و ٢ر٤٪ من جملة العجول المولودة . وأن هذه الحالة ترجع الى جين متنحى .