THREE HEREDITARY ANOMALIES IN PIGS

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PARALYSED HIND LEGS.

A recessive sublethal factor found in a herd of Norwegian Landrace in Norway was reported by WRIEDT (1929) and MOHR (1929). Affected pigs had apparently paralysed hind legs. They could not rise and walk, but were able to crawl forward by means of the fore legs, which were apparently more normal. The pigs died a few days after birth. Litters showing the character had 71 normal pigs and 25 paralysed ones, and WRIEDT and MOHR assumed a recessive factor to be present.

At the foundation of the herd at the Pig Breeding Station at the Agricultural College of Norway, some animals descending from a bearer of the factor were purchased. Of these animals and their offspring a boar, Grom, his litter mate sister and his daughter with another litter mate sister carried the factor. In matings to Grom these two sows had in 3 litters 21 normals (12 ♀♀ + 9 ♂♂) and 7 paralysed (2 ♀♀ + 5 ♂♂). These findings are in accordance with the hypothesis of WRIEDT and MOHR. None of the carriers of the gene produced paralysed pigs in matings to unrelated sows and boars.

The paralysed pigs showed the same traits as described by WRIEDT and MOHR. Further, it was observed that the fore legs, too, were affected, but not in the same degree as the hind legs. The pigs were inclined to lie always on the same side. If laid on the other side, they managed to crawl a little but came to rest on the usual side. The side was not always recorded but in some cases the right side was noted.

Probably there were also some concomitant internal anomalies, as the affected pigs died a few days after birth in spite of all possible care. The birth weight was slightly under normal. The normal pigs averaged 1,13 kg. and the paralysed ones from the same litters 1,02 kg.

KOROVECKAJA (1938) has reported from Russia a hereditary anomaly, which in many respects resembles that described above. I have not read the Russian paper, having seen only a review in Animal Breeding Abstracts (1939). The character was called »recumbent». Affected pigs were unable to stand and were always lying on the right

side; if turned over on the left side, they attempted to turn back. They were able to flex and extend the limbs. The hind legs were powerless, but the fore legs could be used for crawling. The degree of the anomaly was variable. Even if tended very carefully the affected piglings usually died at an age of 2—4 weeks. Histological examinations of the nervous system revealed a degenerative atrophy of the motor cells. In litters containing affected piglings the ratio of normals to affected was 455 : 110, i. e. 19,47 %, which indicates a monogenic recessive. There was no sex-linkage. The expected number in general is 141,25 affected pigs (25 %).

The expected number of affected piglings in litters, containing affected piglings, is in fact a little higher than 25 %, because some litters, especially small ones, do not show any affected piglings, and thus we have a reduction in the number of normals. If x stands for the number of pigs in each litter, n for the number of litters of each litter size and p stands for the expected proportion of affected pigs, we can calculate the expected proportion by the following formula:

$$\frac{p \cdot \Sigma xn}{\Sigma xn[1 - (1 - p)^x]} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (1)$$

If the number of pigs in each litter had been 8, the expected proportion in litters containing affected piglings in matings of monogenic heterozygotes would be:

$$\frac{0{,}25}{1 - (3/4)^8} = 0{,}2778.$$

When the number in each litter is small, the deviation from the expected proportion may be considerable, but when the number is above 10, the difference is slight.

If the value of p is small, we have the interesting fact that:

$$1 - (1 - p)^x = px.$$

The limit value of formula (1) by decreasing values of p is accordingly:

$$\frac{\Sigma nxp}{\Sigma nx^2 p} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (2)$$

The last formula is approximately $1/x$ irrespective of the real value of p.

The grouping of litters is thus very often misleading as to the segregation ratio.

These calculations indicate a deficiency in the number of affected pigs in proportion to the expected number of »recumbent».

From Sweden and Germany hereditary anomalies are reported, which in some respects resemble the two mentioned, but in many respects they are different and are probably caused by different genes.

HALLQVIST (1933) reported from the Swedish Large White Breed in Sweden a lethal anomaly, which is named »bent legs». The fore legs were stiff and bent towards the longitudinal axis of the body. Sometimes the hind legs, too, were affected, but in a slightly different manner. When affected, the hind legs were stiff and placed at an approximately right angle to the body, but not bent. HALLQVIST maintains that »bent legs» is a complete parallel to the case found by ROBERTS (1926) in sheep.

The affected pigs were usually stillborn or died soon after birth. Data for 32 litters containing affected piglings showed 220 normals to 46 defective (17,29 %), i. e. a ratio which deviates from the expected 25 % in a monogenic recessive. This suggests that some disturbing influence is in operation. As mentioned above, we should rather expect a little above 25 % in litters containing affected piglings.

From herds of improved German Landrace in Germany a sublethal anomaly called »thick legs» has been reported. The fore legs of new-born piglings were thick and bent inwards and could not be stretched. The degree of manifestation was very variable. Histological examinations demonstrated that the thick-legged condition was caused by an abnormal growth of subcutaneous connective tissue. Primarily it was caused by a developmental disturbance of the circulatory system and resembled elephantiasis. Few affected piglings survived for more than a few hours. The affection has been reported by WALTHER, PRÜFER and CARSTENS (1932), WENZLER (1936) and CARSTENS, WENZLER and DÜRR (1937).

The data suggest a monogenic recessive. WENZLER reports in 14 litters 83 normals to 28 »thick-legged» (25,2 %). Apparently this is the result of matings of evidently heterozygous animals. It is not quite clear how the distinction is made between heterozygous and homozygous sows. The anomaly was revealed by matings of a certain boar

to his daughters and all the experimental matings were by the same boar to his daughters. The boar was also the carrier of a factor causing atresia ani and possibly a third factor causing blindness. A linkage between »thick legs» and atresia ani was probable.

SCROTAL HERNIA.

A comprehensive report on scrotal hernia in swine and its inheritance in a herd of Duroc Jersey and Poland China is given by WARWICK (1926 and 1931). WARWICK suggests a digenic recessive. In some respects the data tally with the hypothesis, but in other respects they argue against it. Strongly opposed to it are the results of the selection for hernia. After selection in 7 generations the percentage of herniated boars was about the same as in the second generation of selection. The average for the 5th—7th generation is 52,1 % herniated males, and in the second generation 42,8 %. This suggests rather a type of incomplete dominance. Such a character as scrotal hernia is influenced by many conditions, hereditary and non-hereditary, and it is very probable that the manifestation cannot be complete. Pure breeding is likely to fail as to the 100 per cent manifestation of the character, notwithstanding the fact that the genetic constitution of the animals is 100 per cent pure. In addition we may most probably have cases of sporadic and non-hereditary origin.

In the above-mentioned paper by KOROVECKAJA (1938) a report is given of the inheritance of scrotal hernia in swine. No conclusion is drawn as regards the genetic basis of the anomaly, though no doubt it is hereditary. Litters containing affected piglings had 745 normal males to 186 affected males, or a ratio of 4 : 1 (20 %). Actually a classification of litters in a group containing affected piglings cannot give an unbiased estimation of the underlying segregation ratio. If the ratio in each litter be 15 : 1, litters containing affected piglings would give 23 % affected males in a litter of 5 males. With the same litter size the same classification of litters would give 23 % affected males, when the distribution of scrotal hernia was quite at random and not hereditary at all, and a frequency of scrotal hernia of 15 : 1. If the frequency of scrotal hernia had been very slight, we should according to formulae (1) and (2) expect 20 % affected animals in a litter of 5 males.

Litters containing affected males had a considerable excess of males. The sex proportion was 57,1 % ♂♂. The Russian author

suggested that the homozygous females died as embryos, but this explanation is probably not correct. The deviation is probably caused by the grouping of litters.

Reports on cases in man and animals show that scrotal hernia in some cases no doubt has a hereditary origin. In man dominant cases are reported, though there is no reason to believe that the mode of inheritance is always the same in the same species, and that all cases of scrotal hernia are hereditary.

At the Pig Breeding Station we have had some cases of scrotal hernia, but not many, and most of them seem to be sporadic, although in a few cases a genetic basis may be assumed. The total number of all cases of hernia is given in Table 1.

TABLE 1. *Herniated pigs at the Pig Breeding Station, the Agricultural College of Norway 1932—39. The two breeds, Large White and Norwegian Landrace, had approximately the same frequency.*

Year	Number of litters	Living pigs at 21 days		Herniated				
				Inguinal	Scrotal ♂♂		Umbilical	
		♀♀	♂♂	♀♀	Number	% of males	♀♀	♂♂
1932...	17	62	87	0	5	5,75	0	3
1933...	38	165	161	2	5	3,03	2	0
1934...	58	202	229	1	0	0	0	0
1935...	76	310	334	0	10	2,99	2	3
1936...	60	239	283	3	13	4,24	4	2
1937...	50	204	230	0	0	0	1	1
1938...	54	234	244	0	3	1,23	1	3
1939...	61	222	287	0	1	0,35	0	0
Total	414	1638	1855	6	37	—	10	12
Litter average		3,95	4,48	0,01	0,09	—	0,02	0,03
Per cent.....		46,89	53,11	0,17	1,06	—	0,29	0,34
Per cent of each sex.....		100	100	0,37	1,99	—	0,61	0,65

The frequency of hernia is low. The 37 cases of scrotal hernia were sired by 17 different boars, only 2 boars had no offspring with scrotal hernia. The accumulation of cases in certain years is the result of some boars, obviously inheriting a tendency to scrotal hernia, but

no definite conclusion can be drawn as to the mode of inheritance. The segregation ratio in 31 litters containing scrotal hernia was 135 females, 143 normal and 37 affected males (20,6 %). In a segregation ratio of 15 : 1 and a litter average of 5,8 males, the expected ratio is above 20 % [see formula (1)], and when the scrotal hernia is distributed at random with a frequency of 6,25 % per male, the expected ratio is also 20 % at the same litter size.

As in the Russian report a high excess of males in litters containing affected animals is noteworthy. The sex proportion in these litters is 57,14 %, as compared with 53,11 % in all litters. An ordinary calculation of the error of difference is of no use, as the skew sex proportion is obviously the result of the grouping. Litters with a large number of males have, as a matter of fact, a greater probability of one or more cases of scrotal hernia. In order to eliminate the differential fertility in the sows, I have grouped the litters (110) of 24 sows, which had litters containing affected animals and litters with no scrotal hernia, and have examined the two groups separately. Litters without affected pigs had in 83 litters at 21 days 360 males and a sex ratio of 51,28 %. Litters with affected pigs had in 27 litters 157 males, of which 33 were affected and had a sex ratio of 57,93 %. If we group the litters into two large groups according to the number of males in the litters we have the following:

Number of males in litter	1 — 5 ♂♂	6 — 9 ♂♂
Observed number of males	232	285
» » » affected males ..	16	17
Calculated affected males	14,8	18,2

There is absolutely no tendency towards an excess of affected males in litters with a large number of males, rather the contrary.

There is a considerable excess in the litter size and in the sex ratio in litters containing affected pigs as compared with litters having no affected animals. But this is obviously due to the grouping into affected and non-affected litters. I have calculated the distribution of the affected litters and the number of males on the assumption that all males in the 110 litters had the same probability of scrotal hernia. The formulæ, which I have obtained by the aid of Dr. PER OTTESTAD, are very important in genetic researches when dealing with some type of selection or other.

Number of litters containing one or more herniated individuals $= \Sigma n\,[1 - (1 - p)^x]$ ..... (3)

Number of males in litters containing
one or more herniated individuals $= \Sigma nx\,[1 - (1 - p)^x]$ ..... (4)

where n stands for the number of litters, x for the number of males in a litter and p for the probability of scrotal hernia ($p = 33/517 = 6{,}38\ \%$). The results are given in Table 2.

TABLE 2. *Number of pigs (21 days old) and sex ratio in litters of 24 sows, which had litters containing scrotal hernia and litters without.*

	Total	Litters without herniated	Litters containing herniated	
			found	calculated
Number of litters	110	83	27	28,2
» » females	456	342	114	119
» » males	517	360	157	152
» » » herniated ..	33	—	33	33
Litter averages, females	4,15	4,12	4,22	4,21
» » , males	4,70	4,34	5,81	5,39
Sex ratio %, males	53,13	51,28	57,93	56,14

There is a slight regression coefficient of + 0,058 female per male. The calculated number of females (4,21) is found by computing the probable number of females to 5,81 males. The calculated sex ratio is found accordingly. From the table will be seen that the difference between found and calculated number of males and that between found and calculated sex ratio are slight. Probably very few of the cases have a genetical basis. There is a slight excess of males in herniated litters. But this excess is not large enough to justify the above-mentioned Russian hypothesis that homozygous females die during the foetal period.

With a single exception all the boars used gave about 20 % herniated males in litters containing herniated animals. This percentage is the expected value when scrotal hernia is not hereditary and the frequency is low and the average number of males in litters is about 5 (see formula 2). The exceptional boar is probably a mutation and strongly indicates that scrotal hernia in some cases may be incompletely dominant. The boar, No. 39, belonging to Norwegian Landrace, was born in a litter without herniated pigs. The dam of the boar had no herniated pigs in 3 litters with a total of 22 males. The sire had in 16 litters by 14 sows 73 male offspring and only a single case of scrotal hernia. As to the pedigree we believed this boar to be

free from hernia, but from the purchaser we got information that the apparently normal boar had 15 herniated males out of a total of 36. We mated the boar to some of our sows. In three litters we got 4 herniated out of 13 males. The 4 herniated were all in the same litter, consisting of 5 males.

As far as I can see, the best explanation of the results is that scrotal hernia is caused by incompletely dominant factors, probably only one factor, and that the manifestation ratio (»Penetranz») in males is low, probably under 50 per cent. The factor has no influence on the sow.

The experiment of WARWICK may be explained in the same way. There is, however, no reason to believe that all cases of scrotal hernia are inherited in the same manner, and some cases may have no hereditary basis.

Incomplete dominance is a frequent mode of inheritance. WRIEDT (1927) reported a case of *umbilical* hernia in pigs of undoubtedly this type. An apparently normal Large White boar, Syver Kalnes 81, had in 1924 in a total of 8 litters with 73 piglings, 25 umbilically herniated individuals (4 ♀♀ + 18 ♂♂ + 3 sex not given). Herniated pigs were found in all litters. The sows never had umbilical hernia in matings to other boars. The uneven sex ratio in herniated piglings may be due to a special influence of the factor.

In eliminating scrotal hernia and umbilical hernia from a herd, attention should be paid to the fact that incomplete dominance may be the mode of inheritance. The best mode of procedure is to eliminate litters containing herniated pigs. Both the sire and dam of such a litter have to be tested further in order to ascertain if the hernia is transmitted from the sire or the dam or from both. If a sow has herniated offspring with different boars, it would be well to eliminate the animal. A single case or very few cases among the offspring of a boar, which is mated to many sows, need not signify that the boar is the transmitter. If there is an obvious surplus of hernia, the boar should also be eliminated.

ATRESIA ANI.

In a herd it sometimes happens that a pigling is born without an opening of the rectum. The anus is completely closed. Similar cases are reported from other animals and man. In the literature are often found descriptions of sporadic cases. The anomaly is usually called atresia ani. In swine the anomaly is variable in its manifestation and the malformation is different in sows and boars. Typical in the boars

is the closed rectum, and after a couple of days the abdomen is usually swollen owing to obstruction. If not treated surgically the affected pigs die. In the sows the anus is closed, but usually there is an opening in the ventral side of the rectum with a communication to the sheath, resulting in a type of cloaca. Urine and faeces are evacuated through the vulva. These sows can live and grow up without any special treatment and are fertile. In a few cases there is no opening between the rectum and the sheath, and in such cases, if not treated surgically, the sows die (KINZELBACH, 1932).

When the obstruction occurs it often gives the impression that anus is closed by a film. In fact the rectum is closed and does not reach the epidermis. It ends usually from 0,5 cm. to 4 cm. from the epidermis. As a rule a piece of connective tissue connects the closed end with the epidermis. When the rectum ends near the epidermis, the boars can be saved by surgical treatment. The rectum can be fastened to the epidermis. Usually the boar will grow up and be fertile.

A single case is reported (KINZELBACH, 1932) of a boar with atresia ani and a connection between rectum and urethra. Liquid faeces passed through the opening and the pig lived some days longer than is usual in such cases.

The older reports of atresia ani in pigs did not imply that the cases were hereditary. In later years some cases are reported, which undoubtedly have a genetic basis. From Hohenheim, Germany, KINZELBACH (1932) gives a report of atresia ani in herds of Schwäbisch-Hällisch Landrace. KINZELBACH gives a comprehensive report of earlier cases, on the anatomy of the anomaly and the breeding results. Later reports on the same affection are given by WALTHER and co-workers (1932) and CARSTENS and co-workers (1937). KINZELBACH considered the mode of inheritance to be irregular and the segregation obscure. He found great deviations from the segregations of a monogenic recessive.

By a surgical operation some atresia ani boars survived and were able to grow up and were bred to sows with cloaca, but the percentage of affected pigs was not substantially higher than in litters from apparently normal parents. This result indicates that normals and affected pigs may have the same genotype. If we assume a monogenic mode of inheritance, we have probably 3 different genotypes of normals, and of affected pigs we have one genotype if the character is recessive, and 2 genotypes if it is dominant. There is reason to believe it is an

incompletely dominant character. From KINZELBACH (1932) I have grouped some of the more important results, which strongly indicate a dominant mode of inheritance.

TABLE 3. *The inheritance of atresia ani. Some data from* KINZELBACH *(1932).*

Parents	Number of litters	Offspring normals	Offspring affected	Per cent affected
A. Normal ♀ × affected ♂ (full brother)	2	10	3	23,1
Normals from mating *A*	3	15	1	6,3
B. Affected ♀ × affected ♂	2	7	4	36,4
Normals from mating *B*	1	5	1	16,7
Normal ♀ × affected ♂ from mating *B*	3	18	5	21,7
Affected ♀ × affected ♂ from mating *B*	1	5	3	37,5

If the character is recessive all the offspring from an affected male mated to an affected female should have the same possibility of giving affected pigs among their offspring. But there is a striking difference in the percentage of affected, when the offspring of mating *B* are grouped in mating. Normals from mating *B* had 16,7 % and affected from mating *B* had 37,5 % affected in their offspring. Because of the inbreeding depression, the fertility was rather low and the number of offspring was accordingly small, but it is obviously enough to ascertain that the affected animals used in mating *B* were not homozygous for atresia ani, as the offspring showed a considerable genetic variability.

The mode of inheritance is probably an incomplete dominance of one or more factors. This mode of inheritance is frequent in animals and plants. Through the works of TIMOFÉEFF-RESSOWSKY it is known in particular in *Drosophila funebris.*

CARSTENS and co-workers (1937) have recorded further results of their investigation, and report a manifestation quota (»Penetranz») of 73 %. Linkage between atresia ani and »thick leg» was probable.

KOCH and NEUMÜLLER (1932) reported a hereditary anomaly of the snout in the offspring of a Berkshire boar. Some of the affected pigs showed taillessness and atresia ani. The segregation ratio was not clear, but the data indicate an incompletely dominant mode.

It is noteworthy that in KINZELBACH's case atresia ani was apparently linked to »thick leg», and in KOCH and NEUMÜLLER's case a connection was found between atresia ani and an affected snout and taillessness.

DAVIDENKOV (1940) points out how an apparent linkage may be brought about by modifiers without any connection between the genes. In recent years workers in genetics have also made it clear that genes operating early in the development of the foetus may often have a pleiotropic effect and give the impression of a linkage between characters which in fact are brought about by the same gene. In cases of irregular and obscure mode of inheritance we thus have to be very cautious as to assumptions of linkage. A proportionally high percentage of coincidence between characters does not always imply an existing linkage.

The foundation stock of Large White of the Pig Breeding Station consisted of a boar and 3 sows. The sows were litter mates and unrelated to the boar. These were bred without any defective offspring, but when the offspring was inbred, some cases of atresia ani appeared. It was utterly out of the question that the foundation boar alone carried the gene. The anomaly was probably caused by complementary factors, one of which presumably had risen by mutation.

Table 4 gives a survey of the occurrence of atresia ani in the herd.

TABLE 4. *Offspring of the 3 boars giving atresia ani at the Pig Breeding Station, Aas.*

	Litters without atresia ani			Litters with atresia ani			
	Sows	Litters	Live born	Sows	Litters	Normals	Affected
Jarl ...	9	12	111	12	14	108	29
Frikk ..	19	24	236	5	8	68	11
Ergo ..	20	26	246	3	3	33	3

Only Jarl and Frikk are concerned in this investigation. The boar, Ergo, which had only 3 cases of atresia ani in his offspring, was of another breed (Norwegian Landrace) and was not related to the other cases. If Ergo inherited atresia ani, the mode of inheritance was probably some type of recessive inheritance or other.

Jarl and Frikk were apparently normal. They were sons of two full sisters. Their sires were unrelated. From the table it can be seen that Jarl inherits a higher percentage of atresia ani than Frikk. In Table 5 is given an extract from Table 4 of litters containing atresia ani.

TABLE 5. *Percentage affected in litters containing atresia ani by Jarl and Frikk.*

	Jarl	Frikk
Percentage of a. ani in females	19,64	15,00
» » » » » males	22,22	13,51
» » » » » all	21,16	14,29

The frequency of atresia ani in females and males is approximately equal. The difference in frequency between Jarl and Frikk is considerable. Jarl has 50 % more affected offspring than Frikk.

Two full sisters, daughters of Frikk, with cloaca, grew up and were bred to Jarl, Frikk and an unrelated boar. The fertility was low, but we succeeded in getting 4 litters from each. The result was interesting as they were full sisters and daughters of Frikk.

TABLE 6. *Offspring of the two sows with cloaca. The Pig Breeding Station, Aas.*

Sire	Sow No. 136			Sow No. 137			Percentage A. ani
	Litters	Normals	A. ani	Litters	Normals	A. ani	
Jarl	1	2	3	1	9	1	26,7
Frikk.....	2	6	1	2	15	3	16,0
Aros	(1)	(4)	0	(1)	(12)	0	0
Total of Jarl and Frikk...	3	8	4	3	24	4	20,0
Percentage ...	—	—	33,3	—	—	14,3	

Mated to Frikk, their father, the two sows had 16 % affected offspring, but mated to Jarl they had 26,7 %. The ratio between Jarl and Frikk is about the same as in the total.

A monogenic mode of inheritance is, in my opinion, out of the question. Jarl was mated to his full sister and had in two litters 21 offspring, all normals, later the same sow was mated to Frikk and had in two litters 2 cases of atresia ani out of 25 offspring. It seems as if 2 different factors were necessary in producing atresia ani in the offspring of Jarl and Frikk. A grouping of the sows, which had litters both with Jarl and Frikk and had affected pigs among their offspring, revealed a different percentage of atresia ani in sows which had affected offspring with one of the boars, and sows which had affected pigs with both of the boars.

TABLE 7. *Frequency of atresia ani in litters of sows which had litters both with Jarl and Frikk.*

	Jarl		Frikk	
	Normals	A. ani	Normals	A. ani
Sows giving A. ani with Jarl:				
Sow No. 69	5	1	7	0
» » 70	7	1	10	0
» » 40	9	1	12	0
» » 43	7	2	8	0
Total	28	5	37	0
Percentage	—	15,2	—	0
Sow giving A. ani with Frikk:				
Sow No. 25	21	0	23	2
Percentage	—	0	—	8,0
Sows giving A. ani with Jarl and Frikk:				
Sow No. 45	13	9	13	3
» » 136	2	3	6	1
» » 137	9	1	15	3
Total	24	13	34	7
Percentage	—	35,1	—	17,1

From the table a rule may be established. The frequency of atresia ani in the offspring of Jarl was twice that of Frikk, and sows giving affected pigs with only one of the boars had in general half the frequency of the sows which had affected offspring with both of the boars. Jarl had 15,2 % and 35,1 %, respectively, while Frikk had 8,0 % and 17,1 %. Jarl and Frikk were obviously of different genotypes, and sows giving affected pigs with both boars were obviously of another genotype than sows which had affected pigs only with one of the boars.

Owing to the fact that the manifestation quota is lower than *one* and is probably different in homozygotes and heterozygotes, it is impossible to set up a factor hypothesis, which excludes all other possible hypotheses. The number of offspring of domestic animals will always be limited and insufficient to obtain full certainty. I have suggested 2 pairs of factors with a somewhat low manifestation quota, but it is impossible to say if one or both or neither of them are chiefly dominant.

The factor carried by Frikk is either heterozygous or has a lower manifestation quota than the factor carried by Jarl.

One of the factors, or perhaps both, must have originated by mutation in the offspring of the foundation animals. The foundation animals were mated, without any single case of atresia ani in the litters. The rather intense inbreeding of the animals has apparently increased the rate of mutation.

Some observations are difficult to explain on the basis of dominance. Jarl was mated to his litter mate sister and had 21 offspring, all normal. A sow out of this mating was mated to her sire, Jarl, and had in two litters 14 normals and 5 affected with atresia ani, and the same sow mated to an unrelated boar had in 3 litters 34 offspring, all normals. This result is apparently due to a monogenic recessive mode of inheritance. But the percentage of affected in the offspring of the 2 »cloaca» sows argues against such a hypothesis. Certainly, if the factors were dominant the two »cloaca» sows should have had affected offspring in all matings, but if one factor is dominant and the other recessive, we should expect the result found.

It is necessary to point out that the mode of inheritance seems to be complicated. As mentioned under the description of the case from Hohenheim, it is possible that the factor or factors are active at an early stage of the development, and that the character observed is a secondary phenomenon. Inbreeding seems to play a rôle. It looks as if inbreeding in some or other way predisposes to the anomaly.

When the breeding results are so obscure, it may seem to be somewhat daring to suggest a factor hypothesis at all, but working with domestic animals, we have to devise plans for eliminating the deleterious characters, and then some theory or other of the mode of inheritance is indispensable. This theory may or may not be correct, but if it works effectively in eliminating the character it is sufficiently correct. When working with laboratory stocks such as mice and guinea pigs, we can simply eliminate the whole stock, if some deleterious hereditary character appears and the mode of inheritance is obscure. Replacing live stock is very expensive. We cannot eliminate a whole herd or groups of herds because of the appearance of a few cases of a deleterious hereditary character. Elimination has to be effected at the lowest cost. As already mentioned, Jarl was mated to his litter sister and had 21 offspring, all normal. A breeder bought a boar from this mating and later we obtained information that this boar was probably a carrier of atresia ani.

When the mode of inheritance is so obscure, it is almost impossible to give security that a particular individual is a non-carrier, especially when young breeding animals are concerned. Older animals can be tested, but according to our experience, the number of offspring needed is greater than may be expected within a reasonable time. The purchasing of breeding animals would be impossible if the buyer required the animal to be free from genes of hereditary anomalies. When the mode of inheritance is complicated, it is very dfficult for a breeder to give any guarantee, even if only a single character is concerned.

Even if the character is a clear-cut monogenic recessive it is difficult to provide evidence of homozygosis of the normal allelomorph. The calculation of the probability was deduced by BERGE (1931 and 1934).

The procedure involving the least expense in eliminating atresia ani in the described case would be: not to take breeding stock from litters with affected pigs. The mother of the litter should be eliminated as soon as possible. As the males are more expensive and are difficult to replace, the boar should be tested further in order to ascertain if the case is hereditary or sporadic. The most efficient method is to test a boar with a sow which has previously had affected offspring. But a high grade certainty cannot be obtained when the manifestation quota is low. If two factors are necessary in producing atresia ani, the boar has to be tested to many sows, before we can obtain sufficiently high degree of probability that the boar is free from both factors, and we cannot expect to reach the usual standards of measuring the significance of a mean. Our usual demands on the error of the mean have to be reduced, because it is impossible to obtain the required number of offspring.

CONGENITAL BLINDNESS.

Occasionally reports are found on congenital blindness in swine, and a hereditary basis is sometimes suggested (CARSTENS and coworkers, 1937). In 1934 we had the opportunity to examine a case of congenital blindness reported by the owner of a herd of Norwegian Landrace. Most of his gilts farrowing in the autumn of 1933 had blind offspring. In some litters all the pigs turned blind within 8 days from birth. Usually the pigs were not born blind, but the eyes were under-normal in size and developed an inflammation and became blind. In addition, 10 out of 14 blind males examined either lacked testes or had

considerably undersized testes on one or both sides. Four of the blind males and the normal males had apparently normal testes.

An investigation revealed that the probable cause was xerophthalmia, due to a deficiency of vitamin A. All the sows had been mated to the same boar. The breeder suspected the case to be hereditary and the boar as a carrier. Two of the sows were mated again to the same boar and were brought to the Pig Breeding Station at Aas. They farrowed in due time, but none of the pigs showed any sign of blindness. An investigation of the feeding revealed that especially in the first part of the gestation period, which resulted in blind pigs, the sows had received food lacking in vitamin A. The food consisted mostly of boiled potatoes. When the sows got a balanced ration, there was never any sign of blindness among the offspring.

This case demonstrates that not all congenital anomalies are hereditary, even if it appears to be so. Purely physiological conditions in the gestation period may be the cause of affections which seem to be hereditary.

SUMMARY.

This paper presents some data on the inheritance of three deleterious characters in pigs.

As to the character, described as »paralysed hind legs», the data confirmed the findings of WRIEDT and MOHR that the mode of inheritance was a monogenic recessive. A similar character is described from Russia under the name of »recumbent».

The inheritance of scrotal hernia was apparently complicated and some cases were probably not hereditary. It is made clear that the grouping of litters into groups containing affected individuals is often misleading as to the true segregation ratio of a character. The hypothesis suggested by WARWICK of a digenic recessive is criticized. Data published by WARWICK and observations made by the author suggest rather a monogenic incomplete dominant, although there is no reason to believe that all cases of scrotal hernia in pigs are inherited in the same manner.

Researches on the inheritance of atresia ani are reported, and a review of some earlier investigations is given. A hypothesis of 2 pairs of factors is suggested, but it is impossible to say if one or both or neither of them are chiefly dominant. The manifestation quota is apparently low, as affected and non-affected sows had about the same segregation ratio in their offspring.

The eliminating of deleterious characters, when the mode of inheritance is partly unknown, is discussed.

The Pig Breeding Station was established in 1932 under the management of Professor PER TUFF. Some of the experiments described here were conducted under his supervision. Since 1935, when Professor TUFF moved to Oslo, the Pig Breeding Station has been under the management of the author.

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