FOETAL MALDEVELOPMENT IN A LITTER OF LARGE WHITE PIGS.

By J. W. RYLEY, B.V.Sc. (Senior Husbandry Officer, Animal Research Institute, Yeerongpilly),
E. L. MELVILLE, Q.D.D. (Senior Adviser, Pig Branch), and J. S. F. BARKER, B.Agr.Sc. (Assistant Husbandry Officer, Animal Research Institute, Yeerongpilly).

SUMMARY.

Congenital deformities in a litter of Large White piglets, characterized by "kinky tails," cleft palates, hare lips, split ears, deformed limbs, supernumerary digits, and urogenital defects, are described and illustrated.

Breeding work to determine the cause of the deformities was not conducted, but it is suggested that they have a complex genetic basis that may involve a threshold during development.

I. INTRODUCTION.

Congenital abnormalities of man and animals are well known. In the pig, Buchanan Smith, Robinson and Bryant (1936) recorded a total of 21 abnormalities believed to be genetic defects, while Lerner (1944) recorded nine genetic lethals. Foetal maldevelopment in pigs believed to be of nutritional origin has been described by Hale (1935), and Ross, Phillips, Bohstedt and Cunha (1944).

During an experiment on the effect of *Leptospira pomona* infection on the breeding performance of sows (Ryley and Simmons 1954), one of the uninfected control sows farrowed a litter showing "kinky tail," cleft palate and a number of other anatomical defects. The details are reported in this paper.

II. HISTORY.

Ten purebred sows and a boar AHS of the Large White breed were obtained from a stud piggery in southern Queensland. Eight were maiden sows, but the other two had each produced one litter showing no deformities. Three of the sows had slightly deformed tails, but although the possibility of "kinky tail" was considered, it was decided that injuries were probably responsible.

III. OBSERVATIONS.

All 10 sows were mated to the boar AHS. One of them (Sow 11), which herself had a normal tail, produced a litter of 11 piglets all of which had "kinky tails." In eight of these piglets, there were also other anatomical defects cleft palates, hair lips, split ears, deformed limbs and urogenital abnormalities, as shown in Table 1. Three of the piglets, including a mummified foetus, were born dead or died within a few minutes of birth. The birthweights of all the piglets are shown in Table 1. The mean weight of the eight living piglets was FOETAL MALDEVELOPMENT IN A LITTER OF PIGS.

1 lb. 15 oz., which is satisfactory for a first litter. Three (Numbers 1, 2, and 3) were destroyed five hours after birth because their hind limb deformities and cleft palates prevented their moving and suckling. Two (Numbers 7 and 8) lost weight rapidly and died within three days because of their inability to suckle. The other three (Numbers 9, 10 and 11), which had no visible abnormality except "kinky tail," survived and were weaned at eight weeks.

Та	ble	1.

SEX, BIRTH WEIGHTS, SURVIVAL AND DISTRIBUTION OF ANATOMICAL ABNORMALITIES IN A LITTER OF ELEVEN LARGE WHITE PIGLETS.

Piglet No.	Sex.	Birth Weight.	Survival.	Kinky Tail.	Cleft Palate.	Hare Lip.	Split Ears.	Deformed Limbs.	Super- numerary. Digits.	Urogenital Abno r- mality.
		Lb. oz.								
1	F	24	Destroyed	+	+	+	+	+	+	
2	F	$1 \ 15$	Destroyed	+	+	+	+	+	+	+
3	F	18	Destroyed	+-	+	+	+	+	+	
4	M	$1 \ 12\frac{1}{2}$	Born dead	+	+	+	—	+	+	+
5	F	$0 \ 14\frac{1}{2}$	Born dead	+	+	+	+	-	+ .	_
6		04	Mummified	+	+	+	+	+	+	
7	\mathbf{F}	19	Died	+	+	+	-	_	_	
8	M	$2 \ 2$	Died	+	+	+	_	-		-
9	M	$2 \ 2$	Weaned	+	-	—	-			1
10	M	2 3	Weaned	+				-		_
11	М	1 14	Weaned	+	-		-	—	-	-
		То	tals	11	8	8	5	5	6	2

Key: .. = Mummified foetus, sex not determined.

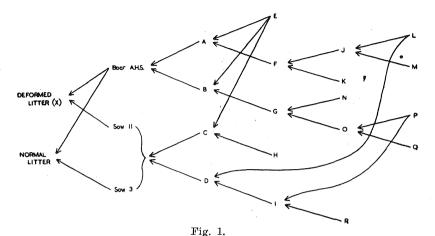
+ = Abnormality present.

- = Abnormality absent.

The other nine sows, including three with deformed tails, produced litters with no visible anatomical defects. These sows included a litter mate of Sow 11 (Sow 3) which also had a normal tail. The boar AHS did not show any defects. Four of these sows, including the litter mate of Sow 11, were remated to the same boar and again all produced normal litters. Sow 11 was remated to a Tamworth boar and farrowed a litter that did not show anatomical defects.

As cleft palates, hare lips, polydactyly, kinky tail and defects of the ears in swine have been recorded either separately or in various combinations (Buchanan Smith *et al.* 1936), the pedigrees of Sow 11 and the boar AHS were examined. These pedigrees are presented in diagrammatic form in Fig. 1. Boar "E" is the grandsire of the boar AHS through both maternal and paternal lines, and grandsire of the sow through the paternal line. The coefficient of relationship between Sow 11 and the boar is 14%, while the inbreeding coefficient of the deformed litter is 7.4%.

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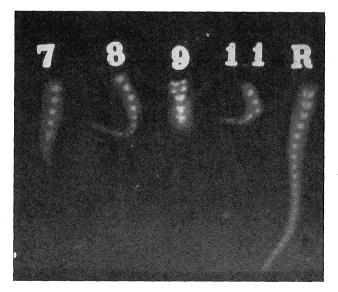


Diagrammatic Pedigree of the Litter of Deformed Large White Pigs.

IV. DESCRIPTION OF DEFORMITIES.

The deformities in each piglet are listed in Table 1 and four of the piglets are illustrated in Fig. 3.

"Kinky tails."—All 11 piglets had abnormalities of the tail varying from short thickened tails bent dorsally and anteriorly over the back to tails of normal length and thickness but bent at angles. X-ray photographs showed that these abnormalities were associated with either a reduction in the number of coccygeal vertebrae or a fusion of some of these vertebrae (Fig. 2).





X-ray Photograph of the Tails of Piglets 7, 8, 9 and 11, Showing Reduction in Number and Fusion of Coccygeal Vertebrae Compared With Normal Tail "R" From a Newborn Piglet in a Normal Litter.

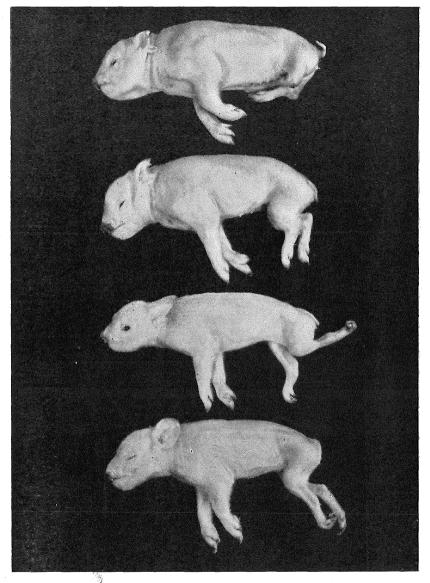


Fig. 3.

Piglets 1, 2, 3 and 4 (from top to bottom) Showing "Kinky Tail," Split Ears, Deformed Limbs and Supernumerary Digits.

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Cleft palates.—Eight of the piglets had cleft palates. These clefts were irregular, usually on both sides of the raphe and varying from 0.5 in. to 3.0 in. long. This abnormality is shown in Fig. 4. In one piglet (Number 8) the cleft was unilateral.

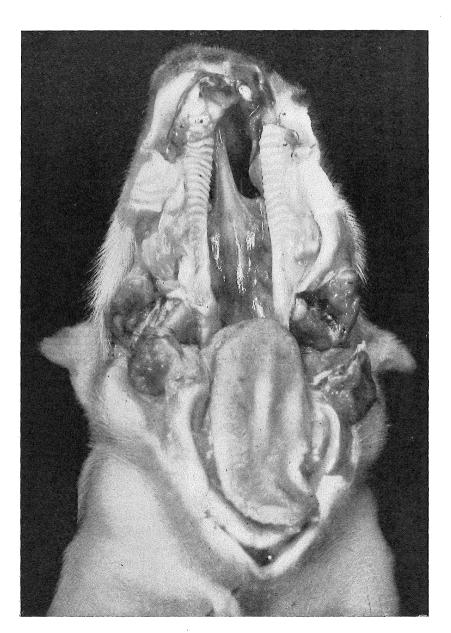


Fig. 4. Dissected Buccal Cavity of Piglet 1, Showing Bilateral Cleft Palata

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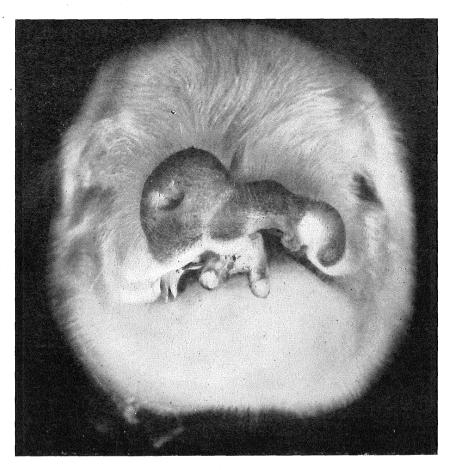


Fig. 5. Unilateral Hare Lip and Distorted Incisor Teeth in Piglet 1.

Hare lips.—All eight piglets with cleft palates also had hare lips. The cleft in the lips extended to the nostrils on both sides except in Piglet 1 (Fig. 5), where it was unilateral. The incisor teeth were large and distorted.

Deformed limbs.—Four piglets showed deformities of the hind limbs. In Piglet 1, the hind limbs were bent under the abdomen. It was able to move, but had little control due to inability to stand on the hind limbs. Piglet 2 had an antero-medial twist in the stifle joint, and in Piglet 3 there was overextension of the left stifle joint and marked flexion of both tarsal joints. Piglet 4 showed flexion of both the stifle and tarsal joints of the left limb and over-extension of the right tarsal joint.

Supernumerary digits.—Six piglets had supernumerary digits; in five the forelimbs were involved and in one (the mummified foetus) the hind limbs. These abnormalities in Piglets 1 and 2 were bilateral, Piglet 1 having a

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supernumerary digit on the medial aspects while in Piglet 2 the medial digit was partially bifid. The abnormalities in Piglets 3 and 4 were present in the right forelimb only, Piglet 3 having a partly bifid medial digit and Piglet 4 a supernumerary digit on the medial aspect. Piglet 5 had two feet on both forelimbs and the mummified foetus (Number 6) had two feet on both hind limbs.

Split ears.—Five piglets had bilateral split ears as shown in Piglets 1, 2, and 3 in Fig. 3.

Urogenital defects.—Two of the piglets showed urogenital defects. Piglet 2 had two small kidneys on the left side joined by a common ureter but with no connection between the parenchymatous tissue. The kidney on the right side showed no abnormality. Piglet 4 had one testis near the posterior pole of the right kidney and the other partly down the inguinal canal.

V. DISCUSSION.

Foetal maldevelopment such as cleft palates, hare lips, malformed hind limbs and blind and eyeless piglets has been associated with vitamin A deficiency of brood sows during the first 30 days of gestation (Hale 1935). However, all sows in this experiment (Ryley and Simmons 1954) were fed a ration containing 5,000 I.U. of vitamin A per lb. and 2-3 lb. per day of freshly cut lucerne for two weeks prior to mating and during the whole gestation period. In addition, none of the piglets showed any abnormalities of the eyes, whereas 42 piglets born during the experiments of Hale (1935) were all blind. Some had no eyes, some had one and others had one large and one small eye. It is therefore unlikely that vitamin A deficiency *per se* was responsible for the abnormalities we observed.

Other nutritional deficiencies have been associated with congenital abnormalities in laboratory animals. Warkany, Roth and Wilson (1948) described shortening of limb bones, syndactylism and cleft palate in rats fed insufficient riboflavin. Gross abnormalities in rat embryos associated with pantothenic acid deficiency have been described by Lefebreres-Boisselot (1951, quoted by Hogan 1953). Ross et al. (1944) described congenital malformations in pigs such as fused toes, club feet, syndactylism, paralysis agitans, vestigial limbs, and "kinked tail." As the addition of good quality lucerne meal to the basal ration prevented their occurrence, they concluded that the abnormalities were of nutritional origin. None of these abnormalities, except "kinked tail," was seen in the litter described in this paper, and in addition the sow's ration included freshly cut lucerne. As the other nine sows mated to the boar AHS produced litters showing no defects, it seems unlikely that a nutritional factor per se was responsible.

Annett (1938) described cleft palates, split ears and deformed hindquarters in two of a litter of 14 purebred Tamworth piglets. A photograph in that paper shows a piglet similar to one depicted in Fig. 3. He considered that the defects were genetic in origin. As three of our original 10 sows showed ''kinky tails'' there may be some hereditary factor for these deformities carried in the parent stud. At the time the deformed litter was observed, it was not

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possible to continue breeding in an attempt to determine the genetic basis, if any, of these deformities. The coefficient of inbreeding of the deformed litter is 7.4%. This is not a high coefficient, and even assuming simple inheritance of the defect, the chance that all 11 pigs of the litter would be affected is very remote.

In a series of Large White litters studied by Donald (1949), one litter was grossly deformed and contained piglets with abnormal tails, hind limbs, snouts, digits and urogenital organs. In addition, the "kinky tail" piglets in the other litters he studied showed a high incidence of abnormalities of the kidneys, ureters and genitalia. He concluded that the "kinky tail" character is genetic in nature, and the genes controlling it are of variable penetrance and expressivity. He thought that, in the Large White inbred strain studied, there were minor genes with a recessive effect as well as a major gene with incomplete dominance. Donald therefore suggested the hypothesis that pigs, in general, may be characterised by a slight tendency to the "kinky tail" defect, which is exhibited when the appropriate developmental accidents occur.

As all 11 piglets in the litter described in this paper were affected, it seems likely that the genetic basis is complex and that a "threshold" may be involved. During the pregnancy that produced the abnormal litter, some developmental accident could have occurred, allowing the developmental path to cross this threshold from the normal to the abnormal. As there are no breeding data other than the occurrence of this one defective litter, and as the developmental genetics of the pig have not been investigated, it is not possible to speculate further.

VI. ACKNOWLEDGEMENT.

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