XXXVII. IRON DEFICIENCY IN PIGS.

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In a previous paper [McGowan and Crichton, 1923] a preliminary account was given of this subject. It is now proposed to give the result of more extended observations.

Having regard to the previous findings, two pregnant sows, during the last few weeks of pregnancy and during lactation, were fed on an iron-poor diet. Their food consisted of distiller's draff, bruised maize and fish meal in the following amounts per day: maize, 2 lbs.; whitefish meal, 1 lb.; distiller's draff as much as they would eat. One sow received in addition 40 g. of ferric oxide per day. This was intimately mixed up with her food. The food was supplied three times a day. They had an abundant water supply. The sows and their pigs were confined during the whole time in concrete pens.

In the case of the sow receiving the iron, the pigs were excluded while the sow was feeding. Thereafter the food was removed. This procedure was carried out to prevent as far as possible the young pigs obtaining iron otherwise than through the milk of the mother. As, however, some of the food was scattered by the sow on the floor of the pen and as, more important, her faeces contained a considerable quantity of iron, the possibility of the pigs obtaining iron in this way could not be entirely excluded.

The young pigs were not allowed outside the pen, were not exercised, and were never separated from their mother except for the short time during which she was feeding.

An attempt was made to estimate the amount of iron in the milks of the two sows. This failed at the outset, owing to inability to obtain enough milk from the sows for this estimation and because of the absence of any reliable method of estimating the very minute quantities of iron present, if small samples of milk were used.

Two sows, Sow 62 and Sow 131, were brought into the pens a fortnight before their farrowing date on April 2 and were made the subject of the experiment already mentioned. The ferric oxide was administered to Sow 131.

They farrowed on April 2, Sow 62 having a litter of eight pigs, numbered consecutively from 1–8, and Sow 131 a litter of ten pigs, numbered from 9–18. As soon as they were born three pigs from Sow 62—pigs 6, 7 and 8—were placed with Sow 131, and four pigs from Sow 131—pigs 15, 16, 17 and 18—were transferred in their places to Sow 62.

Various points in connection with the two groups were observed during the course of the experiment and these are summarised in Tables I and II.

Table I. *"Non-iron" Sow.*

	March			April						May								June				Remarks
	14	20	28	2	6	15	19	24	29	4	7	10	14	17	22	25	31	4	7	12	22	
Sow 62.				Farrowed																		
Haemoglobin %	70	80	80		—	—	—	75	75	—	70	—	—	80	—	—	—	—	—	—	—	Alive and well
Red blood cells in millions per c.mm.	6·5	7·5	6·9		—	—	—	5·3	5·4	—	5·0	—	—	—	—	—	—	—	—	—	—	
Pig 1 (Sow).																						
Weight in kilos.	—	—	—	1·263	Dead	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Dead. Overlaid by sow
Haemoglobin %	—	—	—	70		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Red blood cells in millions per c.mm.	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Pig 2 (Boar).																						
Weight in kilos.	—	—	—	1·440	—	—	—	—	—	—	—	—	—	—	—	11·5	—	13·8	15·9	18·1	—	Alive. Typical iron deficiency
Haemoglobin %	—	—	—	60	60	55	50	40	40	35	—	40	40	40	35	40	60	60	70	—	—	
Red blood cells in Millions per c.mm.	—	—	—	—	—	—	3·3	2·8	2·7	3·0	—	—	—	—	—	—	—	—	—	—	—	
Pig 3 (Boar).																						
Weight in kilos.	—	—	—	1·390	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Dead in 59 days.
Haemoglobin %	—	—	—	80	60	40	40	30	40	30	—	40	25	25	40	35	—	—	—	—	—	Typical iron deficiency
Red blood cells in millions per c.mm.	—	—	—	—	—	—	3·0	3·0	2·5	2·5	—	—	—	—	—	—	—	—	—	—	—	
Pig 4 (Sow).																						
Weight in kilos.	—	—	—	1·320	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive. Typical iron deficiency
Haemoglobin %	—	—	—	70	70	40	40	35	—	45	—	40	35	40	50	60	60	70	—	70	—	
Red blood cells in millions per c.mm.	—	—	—	—	—	—	3·0	3·6	—	4·2	—	—	—	—	—	—	—	—	—	—	—	
Pig 5 (Boar).																						
Weight in kilos.	—	—	—	1·430	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Dead. Acute obstruction of intestine. Iron deficiency beginning
Haemoglobin %	—	—	—	60	70	40	Dead	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Red blood cells in millions per c.mm.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Pig 15 (Boar).																						
Weight in kilos.	—	—	—	1·240	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Iron deficiency at first, then about 29th April became a "runt" and so killed
Haemoglobin %	—	—	—	80	70	50	40	40	60	—	60	60	65	Killed	—	—	—	—	—	—	—	
Red blood cells in millions per c.mm.	—	—	—	—	—	—	4·5	3·6	6·8	—	6·5	—	—	—	—	—	—	—	—	—	—	
Pig 16 (Boar).																						
Weight in kilos.	—	—	—	1·330	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Dead. Overlaid by sow
Haemoglobin %	—	—	—	60	Dead	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Red blood cells in millions per c.mm.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
Pig 17 (Sow).										Fluid in abdomen, prolapse of anus												
Weight in kilos.	—	—	—	1·310	—	—	—	—					—	—	—	—	—	—	—	—	—	Dead in 38 days.
Haemoglobin %	—	—	—	70	55	45	40	40	40	30	25	30	Dead	—	—	—	—	—	—	—	—	Typical iron deficiency
Red blood cells in millions per c.mm.	—	—	—	—	—	—	4·1	2·7	3·0	2·8	3·0	—	—	—	—	—	—	—	—	—	—	
Pig 18 (Sow).																						
Weight in kilos.	—	—	—	1·290	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Dead in 48 days.
Haemoglobin %	—	—	—	70	60	40	30	30	25	25	20	20	20	20	Dead	—	—	—	—	—	—	Typical iron deficiency
Red blood cells in millions per c.mm.	—	—	—	—	—	—	1·0	2·0	2·0	2·0	1·8	—	—	—	—	—	—	—	—	—	—	

Table II. *"Iron" Sow.*

		March			April						May								June			
		14	20	28	2	6	15	19	24	29	4	7	10	14	17	22	25	31	7	12	22	Remarks
Sow 131.	Haemoglobin %	70	80	80	Farrowed	80	80	—	80	75	—	80	—	—	80	—	—	—	—	—	—	Alive and well
	Red blood cells in millions per c.mm.	5·9	7·5	6·0		—	—	—	6·4	5·7	—	6·0	—	—	—	—	—	—	—	—	—	
Pig 6 (Sow).	Weight in kilos.	—	—	—	1·360	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive and well.
	Haemoglobin %	—	—	—	70	60	60	60	60	70	70	—	65	75	70	80	70	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	4·0	4·4	3·5	5·0	—	—	—	—	—	—	—	—	—	—	—	ciency
Pig 7 (Boar).	Weight in kilos.	—	—	—	1·540	—	—	—	—	—	—	—	—	—	—	—	—	16·3	20·4	22·7	28·4	Alive and well.
	Haemoglobin %	—	—	—	60	70	70	60	70	70	—	—	70	75	75	80	80	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	3·7	4·9	5·8	—	—	—	—	—	—	—	—	—	—	—	—	ficiency
Pig 8 (Boar).	Weight in kilos.	—	—	—	1·440	—	—	—	—	—	—	—	—	—	—	—	—	15·9	19·3	21·3	23·4	Alive and well.
	Haemoglobin %	—	—	—	70	60	60	60	60	60	70	—	60	75	70	75	80	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	5·7	4·4	5·7	5·5	—	—	—	—	—	—	—	—	—	—	—	ciency
Pig 9 (Boar).	Weight in kilos.	—	—	—	1·240	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Killed on May
	Haemoglobin %	—	—	—	70	60	45	40	45	40	—	40	40	40	40	40	40	—	—	—	—	25th. Slight
	Red blood cells in millions per c.mm.	—	—	—	—	—	3·7	Diarrhoea, big belly				3·0	—	—	—	—	—	—	—	—	—	iron deficiency
Pig 10 (Sow).	Weight in kilos.	—	—	—	1·450	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive and well.
	Haemoglobin %	—	—	—	60	60	70	—	60	65	70	—	80	75	80	70	80	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	—	—	4·8	6·8	7·1	—	—	—	—	—	—	—	—	—	—	ciency
Pig 11 (Sow).	Weight in kilos.	—	—	—	1·250	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive and well.
	Haemoglobin %	—	—	—	60	70	70	60	80	70	—	—	80	75	80	75	75	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	—	5·6	7·0	6·4	—	—	—	—	—	—	—	—	—	—	—	ciency
Pig 12 (Sow).	Weight in kilos.	—	—	—	1·050	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive. Slight
	Haemoglobin %	—	—	—	70	70	60	50	60	60	—	—	40	40	60	80	70	—	—	—	—	iron deficiency
	Red blood cells in millions per c.mm.	—	—	—	—	—	—	4·5	4·1	3·6	—	—	—	—	—	—	—	—	—	—	—	
Pig 13 (Boar).	Weight in kilos.	—	—	—	1·250	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive and well.
	Haemoglobin %	—	—	—	70	70	60	65	—	—	60	—	60	70	70	70	80	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	—	4·1	—	—	5·0	—	—	—	—	—	—	—	—	—	—	ciency
Pig 14 (Boar).	Weight in kilos.	—	—	—	1·250	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	Alive and well.
	Haemoglobin %	—	—	—	70	70	65	—	70	—	65	—	60	65	80	80	75	—	—	—	—	No iron defi-
	Red blood cells in millions per c.mm.	—	—	—	—	—	—	—	5·0	—	6·0	—	—	—	—	—	—	—	—	—	—	ciency

Before discussing them, the symptoms and pathology of the condition of iron deficiency may be briefly stated: this will facilitate reference in the tables. The disease, although actually commenced some time previously, as will be seen later, usually declares itself when the pigs are about three weeks old. The pigs affected are very pale in colour, very fat and stocky in build and become more and more listless. Often there is blueness of the ears and along the back of the body. Spasmodic breathing develops—"thumps"—and a great many of the animals die quite suddenly at this stage. Diarrhoea is a very prominent feature and great thirst is very often present. The temperature is normal or subnormal. The haemoglobin may be as low as 20 %. On examining such cases, post mortem, the heart is found to be greatly dilated, there is great excess of pericardial fluid, the lungs are oedematous with sometimes effusion into the pleural cavities, and there is great effusion into the peritoneal cavity. The liver is studded all over the surface and in sections with minute white dots, indicative of fatty change in the centre of the lobules. In the tables, the term "typical iron deficiency" means that in such cases all the most important symptoms and signs just enumerated were present.

The occurrence of iron deficiency in the pigs of the two groups may now be presented in the form of a table—Table III.

Table III. *Sow* 62, "*Non-iron*" *Sow*.

Pig	Lived	Died	Cause of death	Iron deficiency present
1	5 days	Yes	Overlaid	...
2	3 months +	No	...	+
3	54 days	Yes	Iron deficiency	+
4	3 months +	No	...	+
5	16 days	Yes	Acute intestinal obstruction	? + beginning
15	46 ,,	,,	Killed as "runt"	+ then recovered
16	5 ,,	,,	Overlaid	...
17	39 ,,	,,	Iron deficiency	+
18	49 ,,	,,	,,	+
		Sow 131, "*Iron*" *Sow*.		
6	3 months +	No	No	No
7	,, ,,	,,	,,	,,
8	,, ,,	,,	,,	Trace
9	,, ,,	,,	,,	No
10	,, ,,	,,	,,	,,
11	,, ,,	,,	,,	,,
12	,, ,,	,,	,,	Trace
13	,, ,,	,,	,,	No
14	,, ,,	,,	,,	,,

The great preponderance of cases, and these fatal, of iron deficiency in the "non-iron" group will be observable. Other points in connection with these tables, especially the occurrence of the iron deficiency in the "iron" group will now be discussed.

During the whole time of the experiment the haemoglobin titre and the red blood cell count of the two sows remained within slight limits practically steady. This is especially noteworthy in connection with Sow 62. (See Tables I and II.)

The haemoglobin of the young pigs from both sows estimated[1] at birth with blood obtained from the umbilical vessels, ranged from 60–80 %, and was the same for the pigs of both sows.

With the exception of pigs 9 and 12, which will be specially referred to, all the pigs of Sow 131—the "iron" sow—showed throughout the experiment a constant level of haemoglobin titre from 60–80 % and a red blood cell count ranging from 4–6 millions per c.mm. This was quite distinct from what occurred in the case of the pigs of the "non-iron" sow—Sow 62. Here the haemoglobin titre fell within the first fortnight below 50 and remained there, reaching in some cases as low as 20 %. Apparent exceptions where, after the primary fall, there was a subsequent rise, as took place in the cases of pigs 15, 2 and 4, will be discussed later. Again in this group the red blood cell count was lower than in the "iron" group, being in most cases 3 million per c.mm. or below. (See Tables I and II.)

It will thus be seen from these haemoglobin estimations that the disease was present in the animals for some considerable time prior to the onset of the sudden deaths, which directed attention to its presence. This point is of some importance in connection with cotton seed poisoning, in that in this condition the occurrence of the sudden deaths, in what are alleged to have been the healthiest and best animals, is adduced by some as evidence that cotton seed injury is in reality caused by a poisonous substance acting presumably at the moment of death. In the condition of iron deficiency, in a similar way, the uninitiated consider that the pale, fat, stocky pigs, the subjects of iron deficiency and practically moribund, are in the best of health and much better than the leaner and more active and ruddier pigs which have an abundant iron supply. The sudden death on which importance is laid in both instances is, of course, due to the great dilatation of the heart, and the water-logged condition of the body which are culminating features in a disease which has existed for some time unrecognised.

Reference may now be made to the exceptional cases in both groups. In pig 15, the haemoglobin titre, after a preliminary fall rose again. This rise was associated with a corresponding rise in the red blood cell count, but also and more important, with a stoppage of growth of the animal. It became a runt and on being killed showed none of the acute signs of iron deficiency. In particular, the liver showed no evidence of disease.

This association of the disease with growth of the animal is more particularly brought out in the cases of pigs 2 and 4. Here, during the period 2nd April to the 22nd May, it was particularly noticed that these pigs were growing much faster than their litter mates 7 and 8 on Sow 131. Unfortunately, their weights were not taken. It was then noticed that while pigs 7 and 8 were progressing uniformly, pigs 2 and 4 were at a standstill or even getting less in weight. This stoppage of growth was associated with a rise in the

[1] Examination was made with an instrument standardised for human blood. Normal sow's blood gave with this a reading of from 70 to 80 %.

haemoglobin titre and the assumption of a "runtish" look. The weights of the two lots were now taken (see Tables I and II) and it was found that pigs 7 and 8 were heavier than pigs 2 and 4 and that they were putting on weight at a greater rate. It may be mentioned that pigs 7 and 8 showed no variation in their haemoglobin titre throughout the whole experiment.

With the fact of the association of the disease with the rate of growth before us, we now come to a discussion of the exceptional cases in the "iron" group. These are pigs 10 and 12. At the outset, it may be noted that these are milder cases than those which occurred in the "non-iron" group. Neither of the pigs died, although one indeed was killed when it was about two months old. This was done because the animal had served its purpose in the experiment, and because, being a waster, it was not profitable to keep it. The occurrence of the disease at all in this group has however to be explained. As we know from practical experience of a year's duration in a pig farm attached to the Rowett Institute with about 40 breeding sows, the use of ferric oxide effectively prevents the appearance of the disease. It cannot therefore be, unless the experiment was performed on an exceptional sow, that the administration of the iron to the sow caused a rise in the iron content of the milk. Otherwise, these cases should not have arisen. We have already alluded to the reason why the iron content in the milk of the two sows was not estimated directly. On the other hand, attention, as far as this was practicable, was paid to preventing the pigs from obtaining iron otherwise than through the milk. It has been already mentioned, however, that iron absorption by this means could not be altogether excluded and it is probable that the young pigs of this litter obtained some iron by nibbling the faeces of the mother and the scattered food on the floor of the pen. This would to some extent prevent the disease appearing, but another factor has to be taken into consideration in this case. From experience in the large piggery, it is well recognised that the pigs most liable to the disease are those which are growing fastest, and conversely slow-growing pigs and runts in litters where the rest are dying from the disease escape it. In the present case, Sow 131 suckled throughout the whole experiment 9 pigs, while Sow 62 suckled only 6, which number was soon reduced by deaths from the disease. Again it was very noticeable that the pigs on Sow 62 were growing much faster than those on Sow 131. This was particularly observed in reference to the litter mates 2 and 4 and 7 and 8, the first two on Sow 62 and the last two on Sow 131.

It would appear therefore that the reason for the occurrence of the disease in the pigs of Sow 131 was that the feeding of the iron to the sow did not increase the amount of iron in the milk. On the other hand the reason why the disease did not appear in all her pigs as in the case of Sow 62, and appeared in such a mild degree was that the young pigs on Sow 131 obtained iron by eating the faeces of their mother and were less liable to the disease in that they were growing slower.

These conclusions are confirmed by the precautions taken to prevent the disease in the large piggery already referred to. A year ago, as many as

70 pigs died in a month from the disease. At the present time, while the number of breeding sows has increased and while farrowing is proceeding at a greater rate, it is difficult, owing to the precautions taken, to obtain a case of the disease for demonstration purposes.

The precautions taken are briefly as follows. The sow's ration contains a large quantity of ferric oxide. This ensures a rich source of iron in the pen to which the young pigs have access. The young pigs are encouraged by every means possible to eat of this food. Thus they are separated from the sow for stated periods daily. This prevents them continuously sucking the sow and makes them hungry, in which case they are induced to eat the iron-containing food lying about. Their appetite is further increased by their being given exercise outside in a grass field which gives them an opportunity of obtaining iron by rooting in the soil and eating grass. The exercise further minimises the risk of their becoming too fat which, although a direct consequence of the disease, would appear also to predispose to it.

The disease dealt with here is one of the suckling stage, being either initiated then, or having its most fatal or dangerous period at that time. As the young pigs grow older and their diet becomes less and less milk and more and more extraneous food, the disease is not so liable to occur. Moreover, as this happens, pigs already affected begin to get better. This is illustrated in the cases of pigs 2 and 4 (see Table I) where after being affected acutely, and without alteration of the diet, recovery took place. That the diet, although poor in iron, contained sufficient for their needs, is shown by the fact that the sow herself remained on this diet for the duration of the experiment without any lowering in the titre of her haemoglobin. That milk although an ideal food in every other respect, is a very poor food as regards its iron content need not be stressed.

The ration by which the disease was experimentally produced above is admittedly one poor in iron: but the disease occurs very widely and can be produced on quite a variety of diets, provided other conditions are favourable. An instance may be cited where it occurred on the following diet: fish meal, $1\frac{1}{2}$ lbs.; white sharps, $2\frac{2}{7}$ lbs.; bran, $1\frac{1}{14}$ lbs.; bruised oats, $1\frac{1}{4}$ lbs.; potatoes, 8 lbs.; turnips 10 lbs.; treacle $\frac{1}{3}$ lb. per sow per day. This diet in virtue of the bran contained a fair amount of iron. The sows and pigs however were confined in cement pens and no attention was paid to exercise or encouragement of the young pigs to eat as soon as possible. In this case, too, the young pigs were receiving extra cow's milk; but this had no effect in prevention of the disease as might be expected.

The disease is not of an infectious nature. No bacterial or toxic condition, although both have been carefully sought for, can be assigned as having a causal relation. For the early diagnosis of the disease the estimation of the haemoglobin would appear to be the most reliable method. Early diagnosis is important, for the longer the disease is allowed to go on without appropriate treatment the more difficult it becomes to influence, and the larger will be the percentage of runts and wasters which survive.

In a previous paper [1923] it was mentioned that the disease might have an important bearing on the subject of "wet" beriberi in human beings. The resemblance is very striking, including the suggestive fact that while rice bran contains 0·232 % of iron, polished rice contains only 0·003 %. We know of no proof that "wet" and "dry" beriberi are aetiologically the same and it may quite well be that they are distinct in origin. In the present case, moreover, the disease was produced in pigs, the mothers of which were fed to a very large extent on draff[1], a material, which in consideration of its composition, must contain a fair amount of water-soluble vitamin. The question of the lack of the anti-scorbutic vitamin does not arise here. On the other hand, considering the food supplied and the conditions in which the sow and her pigs were kept, absence of fat-soluble vitamin would seem at first sight to be a likely possibility. The disease produced has, however, nothing in common with the type of disease associated with the lack of this vitamin. The specific curative and preventive effects, moreover, of iron in the shape of ferric oxide have to be considered. In our previous communication, to quote an example of this it was recorded that, on the administration of ferric oxide to animals already affected, the sudden deaths ceased and the symptoms abated, and together with this the haemoglobin titre as estimated on about one hundred animals, before and after the administration of the iron, rose in three weeks from 20–30 % to 70–80 %.

We have previously drawn attention to the resemblance of this disease to cotton seed poisoning [1923], in respect of the symptoms, post mortem appearances, and the beneficial effects resulting from the use of iron. We have made further observations on this subject, but we propose to deal with them in a separate paper.

Summary and Conclusions.

1. A condition in suckling pigs is described and experiments in connection therewith detailed. This condition is attributable to a lack of iron in the sow's milk.

2. It can be readily prevented and cured by the administration of ferric oxide in the food put into the sow's pen. This result in all probability is brought about by the provision in this way of an easily accessible and rich store of iron for the young pigs, which they should be induced by every means possible to partake of as soon and as liberally as possible. In all probability the effect is not produced by an increase of the iron in the milk by the sow being fed on such food.

3. The possible relation of the condition to "wet" beriberi and to avitaminosis in general is discussed.

[1] Draff is malted barley after extraction with water. The material is fed wet and thus contains a considerable amount of the extraction water.

REFERENCE.

McGowan and Crichton (1923). *Biochem. J.* **17**, 204.