XXV. ON THE EFFECT OF DEFICIENCY OF IRON IN THE DIET OF PIGS.

(PRELIMINARY COMMUNICATION.)

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(*Received February 16th, 1923.*)

In a previous paper [Elliot, Crichton and Orr, 1922], the importance of the inorganic constituents of food in nutritional disorders, with especial reference to rickets in pigs, was pointed out. In this paper, attention will be drawn to the important place that another inorganic constituent, namely iron, may take in the growth and development of the same animals.

In a large breeding establishment for pigs, it was the custom to keep the sows out in the open, on pasture, till within a fortnight of their farrowing date. They were then brought into the house and put in pens with cement floors, walls etc. Their food, when in the house, consisted of fishmeal, bruised maize, and "draff" or brewers' offal in certain proportions, together with an abundant supply of water.

This diet contained very little iron. The fishmeal was of the white variety, prepared from the bones and adherent flesh of white fish, without the blood, intestines or other organs and contained only a trace of iron. (Albu-Neuberg [1906] found only a trace of iron in anchovies.) Maize contains 0·008 % and barley, from which "draff" is obtained, 0·024 % iron [Forbes, 1909, p. 152].

The sows farrowed in due course and the pigs appeared to do well till they were from three to four weeks old. Then the following train of symptoms began to appear. The pigs became dull and listless, their skins became intensely white, they developed a hairy appearance, and they took on a "stocky" or thick-set look (due to oedema of the skin). In addition to these, the breathing became pumping in character and "thumps" or spasmodic jerking of the diaphragm developed. Sudden death became a common event, until whole litters perished one after the other. Cases would be seen standing up alive and, in a few minutes, they would be seen in respiratory convulsions on the floor.

On *post mortem* examination of cases at this stage, the following appearances would be found. The animals were in an apparently very fat condition, subcutaneous fat being specially abundant. The heart would be found so dilated as actually to fill almost the entire chest cavity, the lungs being crushed back and collapsed until they were confined in the angle between the bodies of the vertebrae and the ribs. There was great effusion into the pericardial cavity, as also into the pleural cavity, and the lungs were oedematous. There was great effusion of fluid also into the peritoneal cavity, and

this fluid contained large coagulated flakes. The liver was pale in colour and showed, all over its surface and substance, minute paler areas of the size of a millet seed. These areas were in the centre of the lobules. The pancreas was extremely white in colour, resembling a piece of chalk. The kidneys were also pale, and the spleen in most cases was slightly enlarged.

The blood and tissues were sterile. The blood was extremely watery and pale, the haemoglobin in many cases being about 15 %, and the red blood corpuscles about 3,000,000 per mm^3.

Microscopic examination of the tissues showed extreme fatty change in the centre of the lobules of the liver. There was also advanced fatty degeneration of the heart muscle and of the epithelium of the secreting tubules of the kidneys. (The urine contained a very slight trace of albumin.)

In the cases which continued to live, and these were few if the disease was at all advanced and if treatment was not adopted, the following appearances developed. The pig became emaciated and ceased to grow; it became very hairy and the skin assumed a cinnamon tint and became very dry and dirty. The animal became very apathetic and no longer desired food. Many of them died subsequently, and on *post mortem* examination of such cases the following appearances were found. Owing to the resorption of the effused fluid in various parts of the body, there were adhesions between the heart and the pericardium, the pericardium and the lungs and the chest wall. The lungs were collapsed and bound down with adhesions to the angle between the bodies of the vertebrae and the ribs. Often there was a pneumonic condition in their dependent margins. The lungs were also adherent to the upper surface of the diaphragm. In the abdomen the diaphragm was fixed by adhesions to the upper surface of the liver, the various lobes of the liver being also adherent to one another and to the coils of the intestine. These latter also were attached to the spleen and to one another. Especially was this seen in the large intestine, where the coils were gummed together leaving practically no furrow between them.

The condition of matters, just described, added to the mortality caused by an epidemic of distemper in the herd on account of the lung complications developed. It, alone or in conjunction with the distemper, gave rise to an outbreak of abscesses in the face, due to caries of the temporary teeth.

The following would appear to be the explanation of the train of events just described. While the sows were out at grass, they obtained abundant iron from the grass and the soil. When, however, they were taken into the pens a fortnight before farrowing, owing to the cement floors and the nature of the food, the supply of iron ceased. In spite of this, presumably owing to a certain amount of iron stored up, they were able to carry on for a period, but, after a time, this supply was insufficient for themselves and the requirements of the growing litters of pigs. The result was that the symptoms and effects of iron deficiency as described appeared in the pigs, and the sows themselves became very emaciated. In this latter connection, it may be

mentioned that the pigs of those sows which contrived in some way to keep up their condition suffered relatively less from the disease.

The milk of swine contains 0·009 % of iron as compared with 0·002 % in the cow, thus showing a high demand in the pig [König, 1920]. There is a storing up of iron in the body of the unborn mammal in anticipation of the poverty of milk in this essential nutrient. During the whole of the suckling period, the young animal is drawing upon this reserve and lowering the percentage in its body [Bunge, 1889]. Hess, Unger, and Supplee [1921], show that the iron content of milk varies with the feed of the animal. Thus cow's milk contains twice as much iron when the animals are fed on pasture as when they are fed on a mixture of bean meal, linseed meal, hominy, gluten meal, and bran together with dried beet pulp, molasses and straw (an anti-scorbutic-free ration).

At the time when it became evident that deficiency of iron was the cause of the disease, there were in the piggery two groups of pigs, first, those over three weeks old, already in the grip of the disease with the organic changes in the liver, heart, kidneys and lungs, and second, those under three weeks old down to those newly born, in which the disease had not begun or where at least it was not so advanced. The mothers of both sets, as well as those pigs which were already feeding for themselves, were at once put on to large doses of ferric oxide, which was administered in their food. The effect of this was noticeable almost at once in several directions. The sudden deaths ceased at once. The pigs became more lively and had more appetite for their food, those of them, which had arrived at the stage of feeding for themselves, now squealing whenever they heard a bucket rattled. Examination of the blood of about a hundred of them, before and after the administration of the iron, showed that the haemoglobin had risen in about three weeks from 20–30 % to 70–80 %. This return of the haemoglobin to about normal did not, and could not be expected to, remove the disease symptoms entirely, owing to the organic changes in the liver, lungs and elsewhere which its lack had produced. No amelioration could for instance be expected in the case of the collapsed adherent lungs, the adherent pericardium and the adhesion of the various organs in the abdomen. The iron, however, had a marked effect in removing the fatty changes in the various organs. This was especially noticeable in the liver, which became normal in consistence and colour, except where, in some cases, the process of regeneration of the liver tissue could be seen in raised darker coloured ring-like areas—like "fairy rings"—on the surface and in the substance of the liver.

The effect of the administration of the iron to sows, before their pigs became visibly affected with the disease, may be judged of by the fact that, apart from the cessation of the sudden death condition, the pigs now were normal and healthy and the pigs of such litters at eight weeks old were about three times the size of the diseased pigs at fourteen weeks old (37 pounds as compared with 14 pounds).

The disease described here would appear to be the same as that discussed

by (amongst others) Wither and Carruth [1918] as cotton seed poisoning. The symptoms and *post mortem* appearances are alike and Wither and Carruth used iron salts to treat the disease with beneficial results. They did not however use iron salts with the idea that the condition was due to a deficiency of iron but apparently with the view that "the iron salts combine with or facilitate the oxidation of the harmful substances in the cotton-seed meal." The only other reference in the literature which we can find bearing on what may be this disease, occurs in Hutyra and Marek [1913] where a description is given of a disease called enzootic hepatitis of pigs. The disease apparently occurs when the pigs are from two to four months old and the pathological appearances, so far as they are mentioned, resemble closely those of the disease we have described. It caused great losses in Russia and during the year 1906 "the losses from this disease in Eastern Prussia were greater than those due to Swine Plague."

The disease discussed in this paper is a very important one in the pig industry. In the instance we have described it occurred not as an experimental production, but in the ordinary course of events in a pig rearing undertaking and we have found evidence of its occurrence in a considerable number of other like concerns. Its appearance depends on the nursing sows being fed on food which for various reasons does not contain enough iron to keep pace with the demands of the growing pigs. The subject matter of this investigation may have an important bearing on the pathology of chlorosis and on the pathology and aetiology of "Wet" Beri-beri in human beings. The fact that in the latter condition the symptoms and *post mortem* appearances are very similar to those described above in the pigs and that while rice bran contains 0·232 % of iron, polished rice contains only 0·003 % [Forbes, 1909, p. 152] is suggestive in this connection. The problem is being further inquired into from these directions, as also from the general stand-point and its relation to the cotton-seed poisoning so prevalent in the United States.

Orr [1922] has emphasised the importance of the mineral content of the food in connection with the etiology of rickets. He has not however specifically mentioned iron. Sollmann [1907] points out that human milk contains about three times as much iron as the artificial foods commonly fed to infants. It is probable that deficiency of iron may be a factor in conditions of malnutrition in infants as well as calcium and phosphorus.

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