But is this disease, unknown in France, known to American veterinarians?

I have made some researches in regard to this, and I believe that it is the same as that which the breeders in the Western States designate by the name of "corn-stalk disease."

Billings speaks of it in his "Report on the Work of the Laboratory of Nebraska for the Year 1888." Although the description that he gives of it is rather summary—he had at his disposition only a small number of specimens sent from a distance—it corresponds pretty closely with what I have been able to observe on my side.

However that may be, it follows from the facts that I have just stated that American cattle are exposed to contract a micro-parasitic broncho-pneumonia hitherto unknown in France. That being well established, it may at once be asked, What sanitary measures ought to be taken in the circumstances? We have already a great deal to do in struggling with our indigenous maladies; is it necessary to run the risk of seeing the number augmented by the acclimatisation of the "Corn-stalk Disease"?

The Department of Agriculture asked my opinion on this point. An attentive observation of the facts and the experimental study of the condition of contagion impose the conviction that the disease has only very feeble contagious properties. In the three very important lots in which the disease showed itself, notwithstanding the considerable number of animals and the long duration and intimacy of the contact, the disease did not spread, and the cases remained isolated. If one considers, besides, that the American cattle imported into France are destined exclusively for the butcher, it seems scarcely probable that the disease in question could propagate itself in France.

At any rate the danger is not such that one could not ward it off by urgency should the event falsify these anticipations.

SWINE ERYSIPELAS.

By J. M'FADYEAN, M.B., B.Sc., F.R.S.E., Royal Veterinary College, Edinburgh.

THE term swine fever, as employed to designate a specific porcine disease, in a sense implies that there is only one febrile affection in the pig, but it is now well known to continental veterinary surgeons that there are at least three totally distinct contagious diseases of that species. With one of these—swine fever, we, in Great Britain, are only too familiar; it is the only one of the three affections of which the Contagious Diseases (Animals) Act takes cognisance, but the facts and observations that I am about to set forth prove that we have among pigs in this country a second affection for which there is no recognised name in the English language. That is the disease which is known in France as *Mal Rouge*, and in Germany as *Schweine-rothlauf*. It appears to me that the literal translation of the German name furnishes a convenient designation for the affection, and I have accordingly headed this article—Swine Erysipelas.

Swine erysipelas is distinguished from the disease which we term

swine fever by many well-marked differences, both clinical and pathological, but it is doubtful whether the two affections would yet have been differentiated had not bacteriological investigation placed the matter beyond dispute.

Swine erysipelas is caused by a minute bacillus, which during the height of the disease is present in great numbers in the blood. As in the case of several other diseases, priority in the matter of discovering this organism has been claimed by more than one investigator. Undoubtedly, the first full and accurate description of the microbe was given by Loeffler.¹ His first investigations were made in July 1882 on a piece of skin from the abdomen of a pig that had been killed while suffering from typical swine erysipelas. On staining sections from this piece of skin with alkaline methyl-blue solution, he found the vessels of the cutis filled with fine rods, which were surprisingly similar to those of Koch's mouse septicæmia, but somewhat shorter and a trifle thicker than the latter. At some places the bacilli formed long threads in the vessels, and they were especially closely heaped together in the vessels of the papillæ. As a result of this interesting discovery, Loeffler was stimulated to examine pieces of skin and organs from five other pigs that had died from the same disease some years previously, these parts having been taken from the body immediately after death and preserved in alcohol. In all of these pieces he found the same fine bacilli.

Some months later Loeffler had the opportunity to make a *post-mortem* examination of two pigs affected with swine erysipelas, and to cultivate the minute bacilli in a state of purity. He found that the growth in nutrient gelatine was very similar to, but still distinguishable from, that of the bacillus of mouse septicæmia. In both cases the culture spread out in the form of fine radial filaments from the needle track, but this radial growth extended farther outwards in the case of the mouse septicæmia organism.

Cultures of the bacillus of swine erysipelas were found to be pathogenic to mice and rabbits. Animals of the former species when inoculated subcutaneously died after 4 or 5 days, with reddening of the skin, swelling of the spleen, hæmorrhages into the lungs, and parenchymatous degeneration of the liver and kidneys; in all cases the fine bacilli were numerous present in cover-glass preparations of the blood and organs. When rabbits were inoculated at the ear an erysipelatous reddening and swelling of the skin started from the point of inoculation and spread over almost the whole body, and death ensued after 5-9 days. Attempts to infect swine by inoculation or feeding with cultures or portions of organs from diseased pigs had a negative result, but this was ascribed to the experimental animals having belonged to breeds that have a natural immunity or insusceptibility towards swine erysipelas. This lacuna in the evidence was subsequently filled up by Schütz; he succeeded in infecting young half-bred English pigs, which experience had shown to be very susceptible to swine erysipelas, by subcutaneous inoculation with a culture of the fine bacilli, and the experimental pigs died with the symptoms and lesions of the disease naturally contracted.

In France, as previously mentioned, this disease is known as *Mal Rouge*, and as early as December 1882, Pasteur, in the name of

¹ Arbeiten a. d. Kaiserl. Gesundheitsamte.

himself and his late assistant, Thuillier, communicated to the Academy of Sciences¹ a note regarding the discovery of its specific microbe. He described it as being "easily cultivated outside the body. It is so minute that it might escape even a very attentive observation. It is to the microbe of fowl-cholera that it most nearly approaches. Its form is again that of a figure of 8, but finer, less visible than that of cholera. Without action on fowls, it kills rabbits and sheep. Inoculated in a state of purity into the pig in inappreciable doses, it promptly produces the malady and death, with the characters habitual to spontaneous cases."

In this same communication Pasteur went on to refer to certain investigations by Dr Klein into the etiology of swine fever (pneumo-enteritis), and he rather contemptuously asserted that Dr Klein was entirely mistaken in supposing that the organism described by him had anything whatever to do with the causation of the disease. Klein in like manner criticised Pasteur's communications on the erroneous assumption that *Mal Rouge* and swine fever were identical diseases.

Regarding the bacteriological distinction between swine erysipelas and swine fever it is unnecessary for present purposes to say more than that the before-described fine bacilli described by Loeffler are not present in cases of the latter disease. It is, of course, not impossible that these bacilli might be found in an animal suffering from genuine swine fever, but in that case their presence would merely prove the evidence of a double infection. The etiological connection of any organism with swine fever is by no means so well established as is that of Loeffler's bacillus with swine erysipelas, but I can as the result of my own observations assert that the latter organism is certainly not the cause of swine fever. Loeffler's bacillus is an easily demonstrable organism by culture and by staining, and I feel justified in saying that it has never been present in any one of the numerous cases of swine fever that I have examined.

Although it is probable that the duality of swine fever and swine erysipelas would not yet have been generally admitted had not the causal organism of at least one of them been discovered, the two diseases are notably different both as regards their clinical history and their morbid anatomy. To this point I shall return later, but it may be well to state here that *in the majority of cases* swine erysipelas is an acute septicæmic disease, with redness of the skin as a prominent symptom during life, terminating in death within 48 hours, and without any constant visceral lesions—notably without pneumonia, or ulceration of the large bowel.

During the past summer, on the occasion of a visit to the Copenhagen Veterinary College, Dr Bang was good enough to show me preparations proving that while swine erysipelas in general runs a rapid course, terminating in death or recovery, there occur cases in which the disease lasts for weeks, possibly months, and is associated with the formation of verrucose growths on the mitral valve.

Since then an article by Dr Bang setting forth the interesting facts observed by him has been published in the *Deutsche Zeitschrift für Thiermedizin*, and I cannot do better than quote here the introductory portion of his paper. He says "For a number of years past I have

¹ Comptes Rendus, 1882, p. 1120.

observed cases of obstructive verrucose valvular endocarditis in pigs. It always appeared to me to be probable that this disease was of a bacterial nature, but it was not till the year 1888 that I could find the time necessary to make a close investigation of the matter. On examining cover-glass preparations from the soft superficial thrombus-like layer of such vegetations, I now immediately found a great number of fine bacilli and fine curved filaments, which stained easily with alkaline methyl blue, gentian-violet, and especially by the method of Gram. Vertical sections through the thickened cusps showed when stained by Gram's method a very pretty picture. Even with the naked eye one could see close to the surface of the cusp a broad deep blue seam, which showed sharply against the red carmine-stained ground work. With slight magnification one could see that the blue seam was lost inwardly in scattered specks, which, however, could seldom be followed deep into the vegetations. Under the oil-immersion lens the blue resolved itself into a pure culture of the above-mentioned fine bacilli and filaments. It could now also be seen that the blue seam did not reach quite to the surface of the vegetation, but that a very small seam of almost homogeneous thrombus matter, poor in bacilli, covered the bacillary growth and protected it against the blood stream."

Dr Bang found that these bacilli in form, size, and staining reactions corresponded with the bacilli of swine erysipelas, and that they were identical with the latter he was able to prove to his own satisfaction by inoculation and culture experiments. Mice inoculated subcutaneously with small pieces of the soft thrombus matter from the vegetations always died in a few days (generally four), and in the blood and spleen and other organs the same bacilli were always present in large numbers. Finally, in gelatine cultures made from such mice these bacilli grew with the highly characteristic "tube-brush appearance" of the bacilli of swine erysipelas.

On several occasions during the past two or three years I had sent to me examples of vegetative endocarditis in the pig, but I had never examined any of these microscopically, concluding that they were, as is frequently the case in the human subject, caused by the presence of pyogenic organisms (*staphylococcus pyogenes aureus*, *streptococcus pyogenes*). I was therefore much interested in Dr Bang's discovery, and determined to take steps to ascertain whether the cases of endocarditis occurring in this country, or any proportion of them, were also caused by the bacillus of swine erysipelas. Thanks to the trouble taken by the gentlemen hereafter mentioned, I have during the past quarter had the opportunity to examine three such hearts; two of these were sent unpreserved, and from them I was able not only to demonstrate the presence of the fine bacilli in the vegetations, but also to obtain pure cultures of these same organisms. The third heart was sent in an excellent state of preservation, and while it was thus impossible to obtain cultures from it, there was no difficulty in demonstrating the presence of the minute bacilli unmixed with any other organism.

The first heart, which was sent by Mr Harry Olver of Tamworth, was from a pig that had been found dead. The animal was one of four bred by the owner, and it had never been out of his possession or premises. It had always been weakly, and at the date of its death

was much smaller than its three companions, but otherwise it had never shown any definite symptoms of disease.

Both cusps of the mitral valve were studded with huge warty vegetations which greatly reduced the size of the auriculo-ventricular orifice. The growths were greyish white in colour, soft, and friable. On shaving away a little of the soft superficial portion of the valve, and staining a preparation made by rubbing the freshly exposed surface on a cover-glass, a great number of minute bacilli, agreeing in size and shape with those of swine erysipelas, were discovered. Along with these there were numbers of other, morphologically different, organisms—probably putrefactive bacteria.

Though not very sanguine of success, I resolved to attempt to isolate the swine erysipelas bacilli directly by culture in artificial media; and, in case that should fail, also to inoculate two rabbits and two pigeons subcutaneously with fragments from the vegetations on the valves.

The culture experiments were made as follows. A small piece was dug out with sterilised scalpel and forceps from one of the valvular excrescences, the superficial layer being rejected as likely to be most seriously contaminated with adventitious organisms. The fragment was crushed and triturated on a sterilised glass plate with a few drops of sterilised water, and a slanting glycerine-agar tube was inoculated with a platinum needle from the thin pulp thus obtained. A second tube was inoculated from the first, and a third from the second, and the three tubes were then placed in an incubator at 30° C. At the end of 48 hours the surface of the agar in the first and second tubes had been entirely overspread by an abundant mixed growth, in which it was not possible to recognise anything suggestive of the appearance of a swine erysipelas culture. The third tube also showed a mixed growth, but at most places this was still composed of discrete colonies; and on close inspection there were recognisable between the large white and yellow colonies a number of very minute almost transparent specks of growth. When examined under a low power of the microscope these were found to be lobate colonies like those of the swine erysipelas organism, and a cover-glass preparation made from one of them by means of the platinum needle proved that they were composed of minute bacilli. Numerous stabbed cultures in tubes of nutrient gelatine were therefore made from the small transparent colonies on the surface of the agar, and at the end of a week the commencing radial growth from the needle track showed beyond any doubt that cultures of the swine erysipelas organism had been obtained.

The inoculation experiments with the rabbits and pigeons did not yield the result anticipated. The two rabbits did not subsequently show any notable disturbance of health, but they fell off in condition, and both died on the 16th day after inoculation. The autopsy showed identical lesions in the two cases. The skin had cicatrised at the point of inoculation, and covered a pea-sized nodule composed of caseo-purulent matter, which, when stained with methyl-blue solution, showed diplococci, but no bacilli of swine erysipelas. The peritoneal cavity contained a large quantity of clear fluid, in which no organisms could be found by staining. The spleen was enlarged to three times the normal, dark, and pulpy. Cover-glass preparations from the pulp showed diplococci. Liver congested. Kidneys normal. Chest

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Cultures of Swine Erysipelas Bacilli in nutrient gelatine (from a Photograph by Mr T. Greig Smith). All three tubes are 3rd cultures from Mr Olver's case, referred to in the text. Tube A represents the growth nine weeks after inoculation; tubes B and C are about a week older. The characteristic "tube-brush appearance" is best seen in tube A.

Swine Erysipelas Bacilli (Reichert's $\frac{1}{4}$ oil-imm., Oc. 3). From a 2nd nutrient gelatine culture obtained from the case of Mr Frank Carless.

organs also normal. Cover-glass preparations from the heart blood showed no organisms. Nutrient gelatine and glycerine-agar tubes inoculated from the blood, peritoneal fluid, and spleen pulp remained sterile.

The two pigeons inoculated at the same time as the rabbits never showed any symptom of disturbance, and both are still alive, three months after inoculation.

As stated above, another result had been anticipated for these experiments. The fragments of valvular growth used for inoculation were known to contain a mixture of organisms, and the death of the rabbits was probably due to the diplococcus demonstrable in the spleen and at the seat of inoculation. That this organism did not grow in the tubes inoculated from the rabbits may be explained on the supposition that it was anaerobic. However that may be, the fact remains that neither rabbits nor pigeons contracted swine erysipelas although inoculated with material rich in the bacilli of that disease.

The second heart was sent by Mr Frank Carless of Stafford. It was from a seven-months-old pig which died after an illness of two days. There was no history of swine fever. The mitral valve was covered with soft warty excrescences identical with those present in the preceding case, and a cover-glass preparation from one of the vegetations showed numerous fine bacilli. In this case no animals were inoculated, but slanting glycerine-agar tubes were inoculated in the manner already described, and plate cultures in gelatine were made at the same time.

By the third day discrete colonies of swine erysipelas bacilli were recognisable in the second and third agar tubes, and from these stabbed cultures in gelatine were made without difficulty. In due time these gelatine tubes showed the characteristic "tube-brush appearance." The original plate cultures furnished numerous colonies of swine erysipelas bacilli, with a few colonies of other organisms in the first plate. The former were easily recognised under a low power of the microscope by the slender branching out-runners radiating from the central spot of growth.

The third heart was sent by Mr Snaith of Bishop Auckland, with the following history. "On the last day of September I examined a pig's heart with large growths on the mitral valve; and being unable to examine it minutely at the time, I immersed it in a very strong solution of carbolic acid. On examining it the other day I found that it was well preserved. The pig was one of three, and was suspected to have died from swine fever. They had all been on the premises for six months, and were quite isolated. The other two appeared quite healthy. In the one from which the heart was taken there were no bowel lesions, but the lower part of each lung was consolidated. On section the hepatised part had a speckled appearance, and there was some effusion into the interlobular connective tissue. The pig was in fair general condition, but it had had a cough for some time."

The mitral valve in this heart was affected precisely like the two already described. It was, of course, impossible to make cultures from the vegetations, but a microscopic examination of sections from them when stained by the method of Gram shows an appearance

for which the passage quoted from Dr Bang's article might stand as a description.

The structure of the new growths is well seen in sections stained by picro-carmin. Towards its free surface each excrescence is composed of an almost homogeneous yellow-stained material, which is evidently altered fibrin and blood corpuscles. A thin, granular, opaque pellicle, in which the outlines of red blood corpuscles are still recognisable, covers this broad homogeneous layer at most places, and the deep aspect of this same homogeneous stratum passes along an irregularly sinuous line into a brownish granular layer in which the degenerating fibrin is being invaded and destroyed by the agency of round-cells. This again at its deeper part passes gradually into delicate connective tissue with stellate and spindle-shaped cells and well-formed blood-vessels.

In sections stained by Gram's method with gentian violet the bacilli are seen to be mainly confined to the stratum of homogeneous degenerated blood clot; they are most abundant in the superficial part of this stratum, and under a low power they appear to be absent from the parts already invaded by leucocytes. Under a high magnification occasional bacilli are found to be still present in the latter situation, but they disappear before the deeper spindle-celled part of the growth is reached. The organisms are present both as short bacillary forms and as filaments of considerable length, the latter being arranged in distinct bundles.

One may interpret the distribution of the organisms in the vegetations of the valve by supposing that the bacilli find, in the fresh surface layers of blood clot a most congenial pabulum, and a place in which they can grow and multiply unmolested by the leucocytes or other phagocyte cells. But as the excrescences grow by the addition of successive deposits of fibrin and blood corpuscles at their free surface, destruction and removal of the fibrinous material is being effected in their deeper parts by wandering cells, and this again is closely followed by the formation of cicatricial tissue through the agency of fibroblast cells. The leucocytes in invading the degenerated fibrinous deposits on the surface of the vegetations appear to compass the destruction of the bacilli.

Very brief investigation has thus sufficed to show that we have in Great Britain in addition to swine fever a disease which in other countries is a formidable scourge of the porcine species; and one is naturally desirous of knowing to what extent it prevails with us, and whether some outbreaks of the disease are not overlooked through confounding them with swine fever. Further inquiry is needed to settle these points, but the three cases cited do not lend any support to the view that the two diseases are confounded, by the veterinary profession at any rate. Moreover, all three cases are remarkable in that there was no evidence that the disease possessed either the formidably contagious character or the virulence universally ascribed to it on the Continent. It will be strange indeed if further inquiry should show that this is due to insusceptibility on the part of the breeds found in this country; for continental observers are perfectly united in recognising that, while the commoner breeds possess a certain degree of immunity, the finer breeds which have been crossed with English blood are remarkably susceptible to the disease.

Veterinary surgeons called upon to investigate outbreaks of contagious disease in pigs must keep their minds open to the fact that swine fever is not the only disease of that class known, and it may not be out of place to conclude this article by indicating briefly the chief distinctions between the three recognised epizootic diseases known to affect these animals.

(1) *Swine Fever*.—Attacks pigs of any breed or age, but is most fatal in young animals. Has an incubative period of 8-10 days. Discoloration of the skin is a common but not a constant symptom. Generally attended with gastro-intestinal disturbance (constipation or diarrhœa), hurried respiration, and cough. Seldom fatal within less than a week, and often lasts for 2 or 3 weeks or more. Save in very acute cases, *bowel lesions are constant*, and pneumonia is almost equally constant. The bowel affection may take the form of a diffuse diphtheritic inflammation of the mucous membrane; sometimes of the small, generally of the large, intestine. In more chronic cases the mucosa of the large intestine is beset with well defined, "ringed," patches of necrosis—these so-called "ulcers," or with nodular elevations ("buttons") covered with necrotic mucous membrane. The lung lesion is a catarrhal form of pneumonia, frequently attended with larger or smaller hæmorrhages into the lung tissue. Spleen as a rule not notably enlarged.

(2) *Swine Erysipelas*.—Coarser breeds (Yorkshire included) are least susceptible; generally attacks animals from 3-12 months old; older pigs are sometimes attacked, but sucking pigs seldom or never. Incubative period 3-4 days. Discoloration of the skin a very common symptom; sometimes confined to the under surface of the body and inner surface of the limbs, sometimes general, occasionally absent; at first bright red, later bluish or brownish red. Illness sets in suddenly with high fever, great depression, weakness, paralysis of the hind quarters, and sometime severe nervous disturbance. Death generally occurs on the second or third day of illness, sometimes within 24 hours, occasionally after a week, or even months (from chronic endocarditis). Is readily spread by mediate and immediate contagion. Fatality 50-75 per cent. or even more. Lesions revealed at *post-mortem* are:—*enlargement of the spleen*; acute inflammation of the gastro-intestinal mucous membrane, with minute hæmorrhages into the same; parenchymatous inflammation of the liver, heart, and muscles; hæmorrhagic nephritis. *Lungs in general free from pneumonic lesions*, but congested; and *no diphtheritic inflammation or formation of "ringed" ulcers in the large intestine*.

(3) *Swine Plague*.—This disease was first differentiated as a specific affection in Germany by Lœffler and Schütz (*Schweine-seuche*). Accurate information concerning relative susceptibility of different breeds and ages, and period of incubation is not available. Symptoms during life are, moderate fever, redness and swelling of the skin, cough, and disturbed respiration. The primary and essential lesion, according to Schütz, is *inflammation of the lung*, followed by *necrosis of the hepatised portions*, and, if the animal survives, by *caseous degeneration of the necrotic areas*. Spleen normal or only slightly enlarged; *no gastro-intestinal lesions*. Sometimes fatal within 24-48 hours, sometimes has a duration of 8 days, or even weeks.