Bacterial Investigation.

— A Study of a Disease of Swine —

by

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Thesis

for

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in

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1897.
During the fall and early winter of 1896 there was very prevalent an infectious disease among hogs in Champaign County and in adjacent regions of Illinois. This offered a favorable opportunity to become acquainted with pathological methods of investigation. It was soon found that there was a special interest in the occurrence and development of the outbreak in the vicinity. For these reasons the studies herein reported were undertaken.

Because of the great confusion in the minds of many and indeed existing in literature concerning swine diseases, it seems advisable to give a brief history of the studies and conclusions of eminent investigators concerning the disease or diseases described under the names of (1) Hog-Cholera, (2) Swine Plague, (3) Schweine-aechte, (4) Pneumococcic Infection, (5) Swine Pox, and (6) Ranget.

Detmers, in 1878-1879, published accounts of investigations.
made by him upon a swine disease to which he gives the name of "swine plague." [Report of Department of Agriculture, 1874; p. 361; ibid. 1874, pp. 367]

He attributed the special agent of the disease to a Bacillus called by him "Bacillus said". No adequate description is given of the Bacillus, but from his descriptions of the lesions, the intestines being primarily the seat of the disease, and from his references to the motility of the organism, he doubtless had the "Hog cholera" germ of Salmon.

Some years after this Schütz described a disease of dogs in Germany, which he called "Schweinereuche. [Abb. a d. Kaiser. Crem. 1886, pp. 376;]"

The question immediately arose whether this German infection was identical with Dentmers "swine plague", a point not agreed upon by Billings and Klein [Fortsche. d. Med. 1888, pp. 937].

The germ causing the disease was given the name of "Loeffler's Bacillus", Loeffler having described previously what seemed to be
the same organism.

Pictet and Joberg, however, distinguished this Loeffler-Schütz germ from that obtained by them from an epidemic at Huntsville, of a disease also called Schweinsmäulchen, because of its slow development on artificial media and the character of growth upon potato. [Comp. Rend. - 1888 t-CV1-2015]

In 1885 Salmon differentiated the "swine plague" of that time from "rougeth". [Report of Dep. of Agr. 1885 p.516]
the two having been confused by Pasteur. [Comp. Rend. 1882 pp.1120]

He showed that Pasteur's vaccination against rougeth did not confer immunity against swine plague and that there was danger of spreading the former disease.

Greater confusion was made when, in a publication in 1887 Salmon and Smith adopted the term "hog-cholera" for the disease known to Billings and Detmer as "swine plague" and using the term "swine plague" for a disease akin to their hog-cholera but claimed by them to be caused by a
distinct germ. [Report of Department of Agri. 1887 pp. 1] These two diseases they found very frequently associated, very nearly one third of the cases they had examined containing the two organisms.

At the same time Comte and Canteloube studied a swine disease occurring near Mentilly, France, which they called Pneumocentrosis infection (Pneumocentrosis). [Comp. Rind 1887 pp. 1281] [Sci. 1888 pp. 612] The lesions and the specific germ were very similar to those of Salmon's swine plague. Again, nearly at the same time, in 1888, Selander published in the "Centralblatt of Bacteriol" a description of an outbreak which occurred in Sweden and Denmark, under the name of "Swine Pied" a disease which he later came to consider as identical with Pneumocentrosis infection and Salmon's Swine cholera. [Annalee de L'institut Pasteur 1890 p. 345] Deleze's criticism was now made of Salmon by Billing...
for holding swine plague and hog cholera as separate diseases and because of Salmon's opposition to a scheme of Billings for inoculation against hog cholera, Salmon opposed the Billings' remedy on the ground that it might lead not to the extermination but to the spread of the disease. On account of the extreme hostility of Billings, then the head of the experiment station of Nebraska, toward the government 'Board of Animal Industry,' a Commission was appointed to find out, if possible, what germ Billings was dealing with and to test the virtue of his inoculations. Also to inquire into the claims of Detmold Concerning priority of description of germ etc.

The report of the Commission acknowledged the existence of the two diseases, swine plague and hog cholera, but found that Billings had at that time but the hog cholera. Their report upon his inoculations remedy was not decisive, but rather disapproved of it for years of spreading the
In 1869, a monograph was published by Salmon, then the Chief of the Bureau of Animal Industry, upon Hog Cholera, Its History, Nature and Treatment. Much of the information contained in this volume had been published previously in reports of the Department of Agriculture, but this was a systematic and complete statement of the facts as set by Salmon. In the preface he asserts the existence of two very different and distinct epizootic diseases of swine which are widely prevalent and which had previously been spoken of under the one name of hog cholera or swine plague. These two names had therefore been used synonymously previous to 1886 when the differences between the two diseases were pointed out in the reports of this Bureau. It was then deemed best to apply the term hog cholera to that disease in which the intestines were found most affected and in which the symptoms came
meant to justify the appellation. The other malady appeared identical, not only in symptoms and lesions but in the microbe which causes it, with the disease recently described in Germany by both Loeffler and Schütz under the name of Schweineseuche or swine plague. For this reason it was considered best to call this affection swine plague in the reports and thus preserve uniformity and prevent confusion of ideas in reference to the disease of swine in different countries. The volume treats exclusively of hog cholera, mainly with symptoms and lesions found in autopsies.

In 1891, Smith, under the direction of Salmon, published a monograph on swine plague analogous to Salmon's on hog cholera. New again morphological characters and location of lesions served to differentiate the two diseases.

By these two publications of Salmon and Smith
and by the complete descriptions given by Schütt for Schweineschwarte, Selander for Swine pock, Cornil and Chavasse for Pneumonic Infection and from the work of Salmon upon Roupel, the points of distinction and similarity are plainly brought out. Let us now consider more precisely the conclusions these authors reach as to the identity or distinction of these diseases.

The exact position of Salmon in relation to the germ of hog cholera and swine plague can be presented in no better shape than by reproducing a comparative table found on page 105 in Smith's volume "Swine Plague."

<table>
<thead>
<tr>
<th>Swine-plague Bacteria</th>
<th>Hog-cholera Bacilli</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morphological</strong></td>
<td><strong>Morphological</strong></td>
</tr>
<tr>
<td>1. Beads 1.0 µ long and 0.5 to 0.6 µ wide (in saline mounts)</td>
<td>1. About 1.2 to 1.5 µ long and 0.6 µ wide</td>
</tr>
<tr>
<td>2. They show the so-called pico-stain</td>
<td>2. No characteristic polar stain</td>
</tr>
</tbody>
</table>
in certain conditions

3. Non-motile

Biological


5. Growth on gelatin gelée or absent.

6. Growth on gelatin gelée.

7. Tends to produce acid reaction of culture liquid.

8. Produces no fermentation of glucose.

9. Produces phenol and indol (?).

10. Rapidly destroyed in water in soil, by drying, etc.

Pathogenic

or on serous membranes.

12. Produces leptococcid: fibrinous and cellular inflammation of serous membranes and pneumonitis in small animals.

13. Mortalities of varieties are fatal to mice, rabbits, guinea pigs, and pigeons in very minute doses. Death in 16-20 hrs.


13. Most virulent varieties are fatal to mice, rabbits, and guinea pigs in minute doses. Death in 5-8 days.

The difference found in certain should be noted. Swine plague is primarily an infection of the lungs with the intestines secondary, while in hog cholera the reverse is true. As previously stated, three authors consider swine plague and swine plague as identical forms a morphological point of view but Smith...
...to hesitate in calling them identical, because of a difference in virulence.

Comparing the symptoms of Salmon's log-cholera and Selander's sick-fever, there seems no difference, and growth upon potato, the log-cholera germ growing abundantly while Selander's germ resembles typhoid fever. However, from his study of Selander's germ, brought to life by the Professor of Veterinary Science in the University of Stockholm, upon small animals, Salmon doubts its identity with log-cholera. Pneumococcic infection described by Comit and Cittadeneze, they consider as closely allied to log-cholera while the germ of Pneumococcic infection as described by Reitel & Jocher is related closely to small plague. This variation is especially peculiar because of the fact that all four of the scientists studying the Pneumococcic infection studied it from the same disease and at the same time.

Salmon, then, embraces them thus:
In reply to a letter from Smith asking his present view concerning these diseases, he also 'rabbits septicæmia and jaw cholera,' he replies: 'Hog cholera is a disease wholly distinct from either swine plague or hog cholera; hog cholera = swine pest = Schweinepest = (probably) Schweinepest juven. (English); swine plague = Schweinseuche; rabbit septicæmia and jaw cholera are probably races of the swine plague species (septicæmia).
"The French pneunmonenteritis has not been satisfactorily described."

On account of the opposition to such a classification, taken by Detmers, Bellings, Jeffries, Slaander &c., there has of late been extensive comparative studies carried on chiefly by investigators in Europe.

Slaander considered the germ brought by Lundgren and studied by Salmon not to be the typical swine pest—a thing admitted as possible by both Salmon and Lundgren—and in an article published in 1898 sustained the opinion that Salmon's hog cholera, Cornil & Fantonnel's pneunmonenteritic infection, and his swine pest are the same [Annales de l'Institute Pasteur 1890 pp. 524-47].

If this be true, then, a demonstration of the identity of swine plague and hog cholera would prove all the diseases (except rough) identical, if we accept the Salmon.
Smith classification. Such a demonstration has been attempted very recently by Silberschmidt [Annals of L'Institut Pasteur 1895. p. 65]. He received cultures of the two germs from Smith through Metchnikoff and found their differences, both morphological and biological, exactly as described by Smith and Salmond. The dog cholera, compared with swine plague, was found to be (1) larger, (2) grows more rapidly upon gelatine, (3) more abundant upon agar, (4) grows upon potato, (5) produces gas and (6) has greater resistance to physical and chemical agents. In testing their pathological effects it was found that originally dog cholera was less virulent, but, following Lelander's method of successive inoculations in rabbits, its power was increased to equal that of swine plague. Thus it is shown that difference in virulence is not sufficient to identify a species. A comparative study of the toxins showed nothing but a qualitative difference. A study of morbid symptoms and lesions in rabbits, guinea pigs, mice and fowls
showed great similarity in pathological action. He was able to immunize rabbits from both diseases by inoculating repeatedly, small but increasing doses of toxin of each disease obtained (1) from a filtered culture of the germ, (2) from a culture having been heated at least one half hour at or above 60° C., (3) from heated toxic blood of rabbits dead of the disease.

It was then found that an animal immunized against the more virulent disease withstood large injections of the live and active culture of the less virulent and although immunity from the less virulent did not assure against an injection of the more virulent live culture, yet it greatly retarded its action, death ensuing only after a much longer time than in a check animal not immunized.

If, therefore, vaccination against hog cholera also means vaccination against swine plague then swine plague and hog cholera are identical. This line of reasoning is the same as employed in smallpox vaccination. Inoculation with cow
pox confers immunity against small pox and cow pox is
generally considered but a modified form of small pox from this
fact. If Cookbank's supposition is true that Cow pox and
Small pox are two distinct diseases then our supposed proof
of identity in this case is at fault. It seems, however,
very strange that two really distinct diseases should produce
the same poison. And for practical purposes it is sufficient
to call diseases identical if the fatal poison against which
medical skill should be directed is the same. It remedy
must be obtained for the most virulence attack, be it hog
choler or swine plague; then hog cholera, swine plague,
schwarzewelt, pneumoenteric infection and swine pest will
all be subdued.

It was suspected that the outbreak originated
from inoculations, introduced by Detwiler, for the prevention
of swine plague as all the diseases in question are called
by him. The same plant, in general as that of Billings,
was adopted i.e. inoculating with attenuated bacilli which
gave a very light form of the disease and yet conferred immunity
from all other forms. I could find no ground for the
suspicion. The epidemic was wide spread through our
Illinois and Indiana and the fairs in the fall when traveling
show-herds are being exhibited, are means enough for scattering
the disease. Mr. Hinton living one mile east of Turkey
claims to be the first to have the disease in this section and
he states that he obtained it by allowing his herd to go for
shelter under a corncrib where, two years previous a large
number of hogs had died of cholera. The ground under the
creek was always dry and deep with dirt. The animals
showed symptoms seven days after the exposure. That the
germ could have lived two years even in such a favorable place
is, however, not likely.

Thus seemed to be the usual two forms of the disease,
the acute and the chronic. In the latter the animals was
fish seized with a spasmodic cough especially noticeable when the animal was disturbed. The head and ears drooped and the back was humped up. Generally there was a slight greenish exudation from the eyes and now and then a persistent diarrhea set in with the discharge colored according to the diet, though constipation was as frequent. Toward the end the use of the hind limbs seemed to be impaired and often the hind quarters rendered useless. If not so extreme, the gait became unsteady, the animal staggering back and forth in its gait. Breathing became difficult and accelerated and the hog became greatly emaciated. Death usually occurred in 2-4 weeks if no recovery.
They represent the two types found— one intestinal in its nature, the other a lung trouble. Both cases were rather acute in form.

"Case II. Symptoms: Spasmodic cough, brought on by the slightest movement; respiration accelerated; inspiration long, expiration short, the air being expelled with a jerk which shook the animal's body. The pulse fast and weak. The mucous membranes of the eyes injected and an exudation from them which adhered to the edges of the lids and in some cases glued them together so that the animal could not see. There was a slight discharge of mucus from the nostrils after coughing; appetite lost; would drink a little water. The bowels in the early stages were loose but later they became torpid; urine scanty, and of high color; as the disease advanced the pig showed weakness of its hind legs and it was with some difficulty it could walk. This pig was sick about a week, was found dead in the morning. A post mortem was made about ten hours after death. This
and no spots or discolorations of any kind on the outside of
the skin; in cutting through the skin and flesh it had
a natural appearance; the peritoneum was of a dark bluish
color in patches with effusion of blood in the tissues; an
opening the colon and caecum, patches of ulcers were found,
and the coat of the bowel somewhat thickened, with
extravasation of blood around the diseased parts; the small
intestine and stomach were healthy; the pelvic portions
of the kidneys were congested; the spleen was normal; the
liver was somewhat enlarged, soft in parts and easily torn.
We were somewhat surprised on opening the chest to find
the lungs in a healthy condition as the symptoms would
indicate lung trouble. The heart was also healthy. On
opening the larynx the vocal chords and the mucous membrane
were found to be very much thickened, which might account
for the difficulty the animal had in breathing. From the
post mortem appearances I would consider the cause of
death to be peritonitis with colitis and ulceration of the colon and cæcum.

"Case II presented the same symptoms as case I and as it was nearing its end it was killed and a post-mortem made at once. The external skin was healthy, and free from spots of discoloration. Incutting into the abdominal cavity a quantity of clear yellowish fluid escaped; the peritoneum was not discolored, and on opening the large intestine it was found to be healthy also; two small ulcers were found near the ileoceleal valve; no other sign of disease was found in the intestines; the kidneys were very much inflamed and enlarged; on opening the chest the lungs were found to be very much inflamed, with patches of red hepatization; the posterior portion of the right lung was in this condition; there was considerable enlargement of the pulmonary vascular system, with dark colored fluid blood; in no part of the cavity did we find effusion save a little serous material.
the characteristic plastic exudation of inflammatory process being as yet undeveloped. It seemed almost impossible that an animal could have lived with its lungs in such a condition. The pericardium showed signs of slight inflammation, the spleen and liver were not altered in appearance and seemed perfectly healthy."

Not infrequently the organs, lungs, liver, spleen and heart were found adherent to the body walls and to each other, so that in some cases the organ was torn in separating it. In such a case the body cavity was often filled with a fluid secretion containing a variety of germs.

After an examination of some fifteen cases it was found that the interiors of a large per centage of them were scarred, in a few cases the walls being almost corroded through generally the ulcers were button-like and yellow. In almost as large a percentage the lungs were affected, often a large portion of the organ being completely hepatized. The spleen
and liver never showed much enlargement or abnormality except in chronic cases when they often adhered to the body walls. The attempt to make cultures in the field from the liver and spleen led to contamination to such a degree that this plan was abandoned and either the animal was brought to the laboratory and dissected or a small portion of the spleen and liver was cut out, immediately wrapped in a cloth moistened in 7% aqueous solution of corrosive sublimate and placed in a fruit jar which had been disinfected with a similar solution. Inoculations upon artificial media were then made in the laboratory from these organs.

The most frequent contamination was a large coecus which formed on an agar plate a thick round colony much like the cholera germ. It stained very quickly and very deeply with methyl blue or gentian violet. Another form often found was a very small oval little elongated and often in the form of figure-of-eight. They stained solid and easily with
methyl blue. Sometimes instead of the figure of eight form, four or more were united, in the direction of their long axis into a rod, the divisions being very inconspicuous.

 Aside from the ovals, round or heart-shaped spheroids showed the bacillus, though this process revealed it before we got it in culture. As seen in the smear, the germ is a small oval about 1.4 long and 0.8 wide which takes the methyl blue stain quite readily but leaving a central zone unstained. This polar stain is very marked in the germ direct from the tissue.

 Then the germ was obtained in culture it was put through the usual tests with the following results: The organism is a distinct oval with rounded ends, often showing a polar stain or at least has a central band that takes the stain less readily than the ends. There is great variation in the size and general appearance, the average size being 1.4 x 0.8 though frequently they are found twice that size. These large germs stain deeper and led to the idea that the culture was contaminated
Frequent plating did not, however, get rid of them. In the culture in active growth, there is greater diversity in size than in an old culture. The old cultures or cultures whose multiplication is slow, show the polder strain much more readily than in the actively growing cultures. Sometimes the spores are connected in short chains but generally they occur singly. A drop of liquid culture suspended on a cover glass and held from the slide by means of a rubber ring shows the organism to be non-motile. This thing, the readiest means of distinguishing the bacillus of hog cholera from that of swine plague, especially pains was taken in the observation. Cultures grown in liquid media at a temperature of 37°C and examined when first opalescent upon a slide warmed to the same temperature failed in every case to reveal motility except in genera known to be contaminant. No spore formation has been observed. The bacillus grows moderately in bouillon making it uniformly cloudy in twenty-four hours after inoculation. This is a slight, white, granular precipitate which increases in
amount for 4-5 days. When a culture has stood for 4-5 days without disturbance, a ring is formed upon the glass plate above the surface of the liquid. In gelatine the growth is very slight. In a stab culture there is a beaded line of growth following the needle's course. The spherical colonies never attain more than \( \frac{1}{10} \) mm. in diameter. The growth never extends out over the surface of the medium. A colony on the surface of a gelatine plate is thin and yellowish white. It is never more than one mm. in diameter. It does not liquefy. A colony on an agar slant or plate is a grayish white, very thin, with smooth margins and glistening surface. There is a faint concentric structure due perhaps to inequality in capacity of growth. The growth is abundant and rapid. In a stab the line of the gelatine wire can be traced in twenty-four hours by a light line of colonies. There is no beaded appearance as in the gelatine. But rather a feathery aspect. The surface of the medium is soon covered with the germ. Upon potato there is but
slight growth. Development is most rapid at 37°C. A culture
set outside a window for a night when the thermometer
registered -15°C was found dead in the morning. Again a hog
that had died and been frozen at a temperature of -26°C
gave no culture report artificial's medium. A culture subjected
to 58°C for 15 min. is killed. Again a culture placed in an incu-
borator registering 49°C was found dead in twenty-four hours. An
agar culture which had been perfectly dry for three weeks,
when scraped with a sterilized knife and the flakes put in broth,
return the germ.

The bacillus is pathogenic to mice, rabbits, and guinea-pigs
given in order of greatest susceptibility. A mouse inoculated
subcutaneously with 0.3 cc. usually died in about twenty-four hours.
A large white rabbit succumbed after a subcutaneous injection of
0.5 cc. of virulent culture, in four days. There was no swelling or
even inflammation at the point of injection. There was a slight
diarrhoea and, near the end, vomiting. The spleen was very much
gorged with blood and very tender. The liver was dotted with light spots. The lungs were normal with the exception of one congealed spot on the caudal tip of the left lobe. Guinea pigs succumb to larger doses. The younger animals are more susceptible than the mature. A half-grown guinea pig inoculated subcutaneously with 0.5 c.c. of virulent culture remained entirely well, with the exception of a large hard lump at the point of inoculation for ten days after the injection. At that time the pus from the lump, obtained by cutting the latter open, showed the germ in abundance together with masses of disintegrating phagocytes. Six hours after this operation, the animal was very sick and was found dead the next morning. There had been copious vomiting of a granular fluid. The spleen was found somewhat enlarged and the liver spotted. The lining of the stomach was inflamed. Smears from the liver and spleen did not show the organism but cultures from these organs together with those from the heart's blood returned the
Subsequent inoculations proved fatal in 7-10 days.

It was found that 0.5 cc. in half grown guinea pigs proved fatal in about the same time as 1 cc. in full grown animals.

In order to find the relation that this germ bears to that of swine plague and hog cholera, a culture was sent to Professor Smith of Harvard University with the request that he send me a culture of the true hog cholera bacillus. He most kindly did so and in reply said: "The culture sent me is new to me... It has not the character of swine plague but approximates hog cholera."

In comparing the two germs it was found that, in size and tendency toward polar staining, the germ found here entirely agrees with hog cholera, but is morphologically different from it, and identical with swine plague. It being non-motile. It and hog cholera grew identical on gelatine and in bouillon except that the ring is less apparent than from the real hog cholera. On potato the growth almost fails as does the growth of the Smith...
germ. I could find neither acid nor alkaline reaction in the organism. It is a gas producer in glucose solution but not such an abundant one as the Smith germ. It produces phools like swine plague. It is quite resistant to drying, high temperatures, like hog cholera but seems to multiply differently in the blood, producing nodules in the blood vessels. It produces puerperal fever, destruction and inflammation of the serous membranes of the intestine and stomach; also ulceration of the intestine like hog cholera and destruction of the lungs like swine plague. In virulence it resembles the hog cholera. It can differ from hog cholera in the two very essential characters of mobility and production of phools. Its pathogenic action also seems to favor swine plague.

There is a marked difference between it and hog cholera in the character of growth upon agar. The Illinois germ forms a thin, dry, and evenly spread colony with very smooth margin and polished, glistening surface with the thickest part near the margin, while the Smith germ forms a thick colony and p
greatest depth in the centre, with a rather irregular margin and a glistening, though irregular, surface.

In order to get a clearer idea of the relation between the various diseases before mentioned and the germ found here, the following table is referred to.

<table>
<thead>
<tr>
<th></th>
<th>Hog Cholera</th>
<th>Swine Plague</th>
<th>Schinewazech</th>
<th>Pneumococcus</th>
<th>Leishmaniasis</th>
<th>Illinois Germ</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>White</strong></td>
<td>Salmon's monograph &quot;Hog Cholera&quot; 1889</td>
<td>Salmon's monograph &quot;Swine Plague&quot; 1891</td>
<td>Utzschneider &amp; Batt. 1888</td>
<td>Comp. Rev. Lab. 1887</td>
<td>Comp. Rev. Lab. 1888</td>
<td>Salmon in monograph &quot;Cholera&quot; pub. 1885; grown by Dr. Green</td>
</tr>
<tr>
<td><strong>Growth on Agar</strong></td>
<td>Colonial circular, gray-white, very flat convex.</td>
<td>Colonial, glistening, convex with a stringy, glistening, very flat convex.</td>
<td>Colonial, white, opaque with red dots and black reflections.</td>
<td>Colonial, white, opaque with red dots and black reflections.</td>
<td>Colonial, white, opaque with red dots and black reflections.</td>
<td>Colonial, gray-white, the milk magnesium; no growth.</td>
</tr>
</tbody>
</table>
| **Growth in Bouillon** | Moderate growth, with pronounced "H"-shaped growth. | Rapid growth, with a film and deposit. | Becomes very turbid in 12 hrs, with heavy precipitate. | Liquid remains opalescent, ring not as marked as in 


<table>
<thead>
<tr>
<th></th>
<th>Cholera</th>
<th>Plague</th>
<th>Schweinepest</th>
<th>Pneumococci</th>
<th>Typhoid</th>
<th>Cattle</th>
<th>Sheep</th>
<th>Edema</th>
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<td><strong>Growth on</strong></td>
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<td><strong>Points</strong></td>
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<td>Growth at</td>
<td>No growth.</td>
<td>In abundant growth.</td>
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<td></td>
<td>Room temperature</td>
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<tr>
<td><strong>Serotype</strong></td>
<td>1cm. by 0.5 cm.</td>
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<td>1cm. by 0.3 cm.</td>
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<td></td>
<td>Long by 0.6 cm.</td>
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<td>Long by 0.3 cm.</td>
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<tr>
<td><strong>Motility</strong></td>
<td>Mobile</td>
<td>Non-mobile</td>
<td>Mobile</td>
<td></td>
<td>Slowly mobile</td>
<td>Non-mobile</td>
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<tr>
<td><strong>Action toward starch</strong></td>
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<td>Slight tendency toward Potassium</td>
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<td>Colorless</td>
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<td>Colorless</td>
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<tr>
<td><strong>Gas Production</strong></td>
<td>Produce gas</td>
<td></td>
<td>Produce no gas</td>
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<tr>
<td><strong>Oxidase on milk</strong></td>
<td>No change in milk</td>
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<td>Do not coagulate</td>
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<td><strong>Spore formation</strong></td>
<td>No.</td>
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<td>No.</td>
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<td>No.</td>
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<td>No.</td>
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<tr>
<td><strong>Thermal death point</strong></td>
<td>70°C for 0.5 min.</td>
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<td>55°C for 15 min.</td>
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<td>67°C for 15 min.</td>
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<td><strong>Effect of</strong></td>
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<td><strong>Desiccation</strong></td>
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<td></td>
<td>In desiccation, in three days</td>
<td></td>
<td>Continuous drying</td>
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<td></td>
<td>Usually destroys</td>
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<td>at 25°C does not destroy but released.</td>
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<td><strong>Bacterial serum</strong></td>
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<td></td>
<td>Not the same as serum of plague or Schweinepest.</td>
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<td>Direct from Logcholera, but identical with Schweinepest.</td>
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<td><strong>Clinical signs</strong></td>
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<tr>
<td><strong>of relationship or identity</strong></td>
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<td></td>
<td>Identical with the swine pest germ, also with Schweinepest and Schweinepest.</td>
<td></td>
<td>Perhaps a variety of Logcholera.</td>
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as will be seen that the description of the Illinois organism
varies less from Salmon's hog cholera than does Salmon's description
of S. typhosa - a disease that he would class as a variety of hog
cholera. (Second communication from Smith, says it, 1889.)

"In general I should state that it should not be announced
as a new disease but rather as a variant of the one now
known as hog cholera."

The most frequent method attempted to immunize
animals against hog cholera is by the inoculation of attenuated
forms of the bacillus. The animal will undergo a mild attack
of the disease which will make him immune from all attacks
afterwards. Pasteur succeeded in attenuation the germ of
cholera, by growing it through many generations at its maxi-
mum temperature limits. (Cord and Cauterized) followed the same
plan with the germ of pneumatico, infections with the
same result. [Comp. Proc. 1888, page 812,7.]

Smith claims that
attenuated forms of the disease plague germ, are often found in
the upper air passages of a certain percentage of healthy
swine, cattle, dogs and cats and even in the saliva of the human
mouth.

An attempt was made with the firm found due
to attenuate it by subjecting it to such agents as it is nature
would much - (1) A variation in temperature, (2) subjecting it to high
temperature, (3) growing it in close contact with the air by forcing
a stream of air through the liquid medium, (4) by inoculation with
the firm cultivated in a somewhat refractory organism. Only the
third yielded anything indicative success. It was inoculated
with 0.3 cc. of a culture of a firm which had been subjected to a
current of air for but two generations, instead of drying in 24 hours
as did the check animal, lived for eight days. Upon dissection
it was found that death had resulted from a subcutaneous tumor
at the point of inoculation. Autopsy from the firm showed the genus in
abundance. Inoculations from the genus obtained from local
tumors upon somewhat refractory guinea pigs, proved even
more virulent and fatal than did the check culture not so taken
This is in accordance with Mitchnickoff’s experiments upon rabbits, vaccinated against dog cholera, namely, that the serum of vaccinated rabbits does not possess the property of attenuating the microbe of dog cholera. (Annales de l’Inst. Pasteur "Study of Immunity," 1877, p. 1.)

Because of the close resemblance of the fowl cholera germ and because it was found prevalent at the same time as the swine disease, it was thought that some connection might exist between the two—that inoculation with the fowl cholera germ might confer partial immunity against dog cholera. It was found, however, that the fowl cholera produced but slight local reaction in Canis and that animals inoculated with fowl cholera were no less sensitive toward dog cholera than was a check animal not so inoculated.

Inoculations with attenuated forms are however always open to the great objection shown by Salmon and referred to in the "Report of the United States Board of Inquiry."
Namely, that of spreading the disease. It may be as easy, perhaps easier to increase the virulence of an organism than to decrease it. An attenuated germ may then become fatal either from its action upon a very susceptible subject or through various accidents to which it may be subjected.

A mode of conferring immunity successfully carried out by Schauder [Annals de Clin. Pasteur 1893 page 5457, by Belcheim-Schmidt [Annals de Clin. Pasteur 1195-page 66] by Metchnikoff [ibid. 18-page 7] and by Salmon [N.Y. Cholera 1879-page 108], by inoculation with the toxins of the bacillus in ever increasing doses, if practical, relieves the danger of spreading the disease, in as much as the germ is not used outside of the laboratory. To conduct this experiment, a flask with 300 cc. of peptone bouillon was inoculated with the Illinois germ and placed so that a current of air passed through the liquid. Growth with such a current of air is very nearly twice as rapid as
in quire liquid. After seven days the culture was passed through a Beeksfeld filter which removed all the germ. The filter was so modified that it failed to grow any germ put in it. We would grow in it very slowly. This toxic liquid was injected under the skin of a rabbit in doses of 0.4 cc - 1.0 cc - 3.0 cc - 4.5 cc - 4.0 cc with the intervals of two days each. After a week's rest 0.5 cc of the virulent culture was inoculated. The rabbit lived eight days, while a rabbit not so toxicated and inoculated with the same amount of virulent culture died in four days. Some account should be taken of individual susceptibility yet it seems that the toxin injected did prolong life.

If we admit with Salmon that the degree of immunity conferred by preventative injections depends upon the quantity of antigenic product formed by the specific microbe, then perhaps it is explained why total immunity was not conferred in the above case for as previously shown the germ is not a very virulent one.