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ENZOÖTIC CEREBRITIS, OR "STAGGERS," OF HORSES.

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During the autumn and winter of 1890-'91, reports were published in various live-stock and agricultural papers, of severe losses of horses, not only in Kansas, but in adjoining States, from a new and comparatively strange disease, which was called "blind staggers," "mad staggers," or simply "staggers," according to the symptoms presented in different cases and the imagination of the reporter.

As soon as possible after my assuming the duties of Veterinarian to the Experiment Station, in November, investigation of this peculiar disease was begun. Owing to the rapidity of the disease, it was difficult to get a case to watch the progress of the disease; but one case was seen in the early stages, and this one had to be studied from a safe distance, as the least approach threw the animal into a frenzy of excitement. Other cases visited were dead, or dying, and afforded little opportunity to watch the progress of the disease.

It has been very difficult to get an estimate of the loss from this disease. A careful estimate gives 50 fatal cases within a radius of 20 miles from Manhattan, and the disease was generally prevalent in the State wherever corn was raised and fed.

Various theories as to the cause of the disease were advanced by veterinarians and stockmen. Some thought it the result of impaction of the stomach or intestines with coarse food, and immediately connected it with the so-called "corn-stalk disease" of cattle; but the fact that the "corn-stalk disease" was not as prevalent as usual among cattle, while "staggers" in horses was not known in other years when many cattle died in the stalk fields, taken with the different symptoms and post-mortem appearances, is convincing proof that the two are separate and distinct diseases.

Another theory was, that the disease resulted from irritation of the bowels, caused by various intestinal worms. The disease was found to occur in many subjects where no intestinal worms were found, so the worm theory was exploded.

Various theories assigned by authors of text-books upon diseases of animals as to the cause of cerebritis or cerebro-spinal meningitis, such as impure drinking-water, malarial or atmospheric influences, bad ventilation and sanitary surroundings, could not be given as the cause of the disease in Kansas. The fall and early winter weather was very pleasant and no malarial diseases were reported, and surely the ventilation of a Kansas corn-field could not be improved. This disease was not confined to any locality. It occurred upon the hills and in the river valleys, in stables with excellent sanitary surroundings, and was especially prevalent among horses pastured in stalk fields. The drinking-water in most cases was excellent.

Owing to the prevalence and rapidity with which the disease ran its course, it was supposed by some to be a contagious disease; but the many isolated cases, and many where one animal in a barn or herd would contract the disease and the others remain apparently healthy, and the fact that it did not sweep over a certain area, like a contagious or epizootic disease, would settle the question in the negative.

#### SYMPTOMS.

The peculiar nature of the disease made it difficult for an ordinary observer to notice any premonitory symptoms. In cases where horses were at work, the first thing noticed was a refusal of the feed and a desire for water, while sometimes drinking was performed with difficulty. Following this would be dullness and a drooping of the head and ears, partial or complete blindness, loss of consciousness, delirium and death, or, in a few cases, recovery. In some cases the brain did not seem as badly affected as the spinal cord, and the animal would not have good control of its hind parts. Some animals would press their heads against a post or wall with considerable force; others would be thrown into a frenzy by the least excitement,

and be dangerous to approach. There was but little fever, the temperature varying from 101° F. to 103° F., mucous membranes slightly injected, and in some instances of a yellowish tinge. The pulse varied with the condition of the animal from 40 to 100 beats per minute. Urine scanty and very high colored, often containing blood and pus, and in many cases passed involuntary; bowels constipated.

The ordinary symptoms are well described by Mr. John Dorman, of Wabaunsee, upon whose farm four horses died within a few days. He says:

"The first one was a six-year-old horse, which seemed all right when they came to the pump to drink Saturday night. Sunday morning he was missing and I went to hunt him up; found him in the pasture, in a slight depression; lay as he fell, and had died apparently without much struggle.

"No. 2 was a three-year-old; noticed her stumbling around in the pasture; she seemed blind; got her into the stable and she had a spasm and was crazy for a time and tore around a good deal; finally got quiet and stood with head and ears drooping, and didn't look right in the eyes. She had spasms frequently and died in about 48 hours.

"No. 3 was 10 years old; came to drink, and when she started away staggered and fell over a wood-pile; got up and ran into a hedge and then into a wire fence. Died in about 15 hours.

"No. 4, two years old, came to water; noticed she was blind; ran into hedge. She only lived a short time. Took out the brain and sent to you.

"No. 5 was taken with what seemed to be the same disease, but didn't go blind. He seemed sleepy and didn't have good control of his hind parts; would reel around a good deal. He was sick for several days, but finally recovered.

"These horses were pastured in a large stalk field. Corn was moldy and wormy and was not husked very clean. Stalks were good, and field was considered good pasture."

Another work horse belonging to Mr. Stingley, of Manhattan, that was fed upon corn and prairie hay, contracted the disease. He was easily excited, and would attack a person, kicking and striking furiously, so as to render treatment impossible. He recovered after about a week's serious illness.

A horse about 20 years old, belonging to Wm. Mitchell, fed upon corn-chop and straw, was taken sick and seemed dull. He would stand with his head against some object, pushing, and finally was taken with spasms and, unable to rise, struggled violently, was covered with perspiration, and seemed in great agony; was killed by "pithing."

#### POST-MORTEM EXAMINATION.

Post-mortem examination of a number of cases gave the following results : Circulatory system normal; respiratory system in a normal condition, with the exception of a slight yellowish tinge to the mucous membrane in some cases ; digestive system with more or less irritation of the stomach and small intestines, but not sufficient to produce serious symptoms. The stomach and small intestines contained but a small amount of food, well digested;

the large intestines usually contained a large amount of fecal matter. In all cases the liver was congested, and in many cases inflamed and adherent to the diaphragm. These cases where the liver was badly affected showed a yellowness of the mucous membranes. In every case the liver was more or less congested. Kidneys, in some cases, showed considerable irritation; in others they appeared normal. Other internal organs apparently normal.

#### NERVOUS SYSTEM.

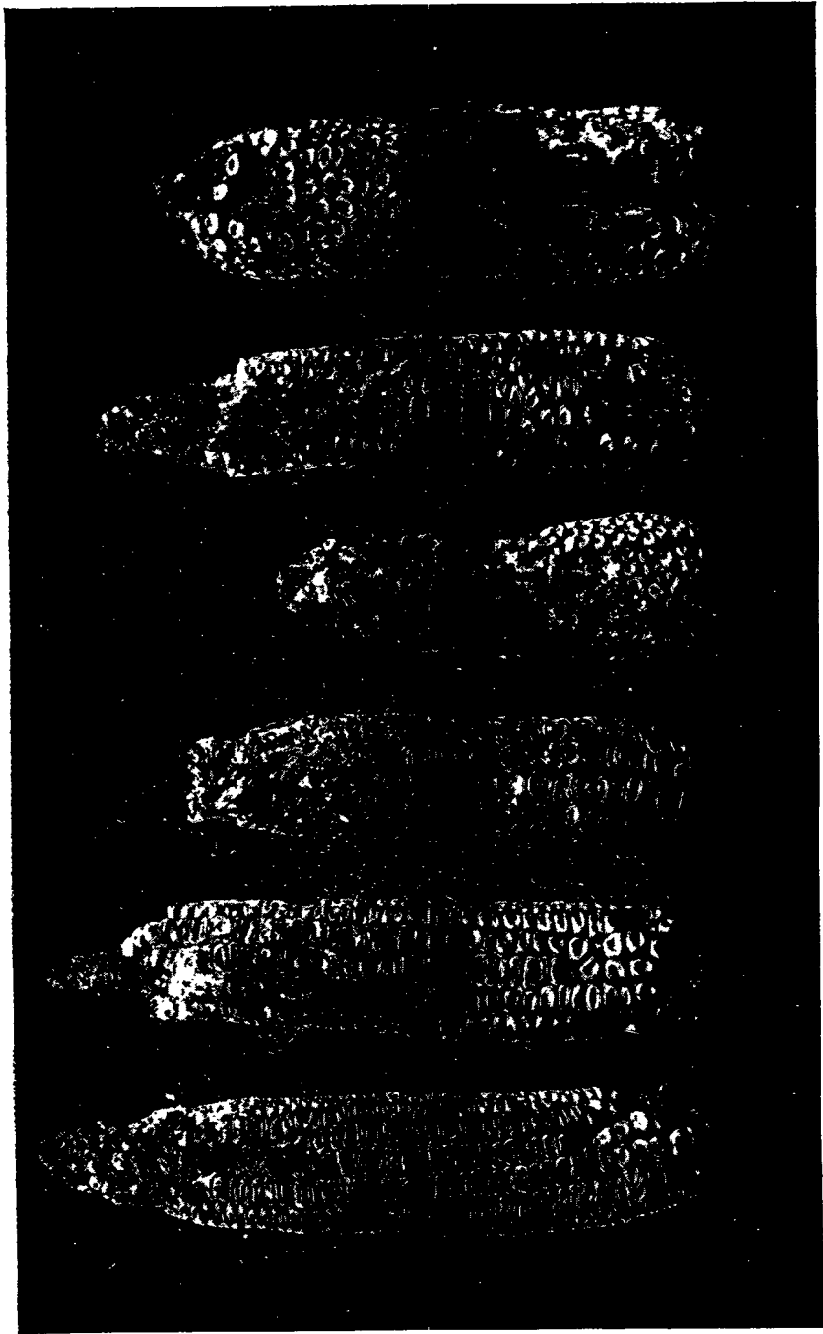
On exposing the brain, more or less inflammation of the meninges appeared, and effusion in the arachnoid space. The blood-vessels of the brain were congested. There was no bulging of any portion of the brain that I could discover, but a careful manipulation would reveal a soft spot toward the anterior portion of the right or left cerebral hemisphere; and in cutting into the white central substance, a serous abscess would be found, in which would be floating flocculi of broken-down brain substance, which presented the appearance, as one stockman said, "of a mixture of vinegar and curdled milk." In some cases, the abscess was only in process of formation. The location could be determined as being softer than the surrounding tissue, and upon cutting into the white central substance, a soft yellow spot would be found, which precedes the formation of the abscess. An examination of the spinal cord revealed no inflammation, abscess, or softening of the cord, and none of the cases upon which autopsies were held had exhibited symptoms of a spinal affection, such as loss of control of the hind parts.

A careful examination of the surroundings precluded the theories that the disease was the result of malarial or atmospheric influences, or bad water, and my attention was at once directed to the food. In every case that I have examined or heard of, the animals had been fed upon corn, or corn-chop, or were running in the stalk fields; and an examination of the corn revealed considerable quantities of mouldy corn. The season of 1890 was a very dry one in Kansas, and the corn crop nearly a failure. The corn was also injured by ravages of the green corn-worm (*Heliorthis armigera*), and wherever the ear was injured by this worm it was attacked by a mould. Commonly the tip of the ear was injured and mouldy; sometimes one or two rows of kernels upon one side would be eaten and mouldy; and in any place upon the ear where the kernels had been injured, this fungus would grow. In a very few cases this injury seems to have resulted from the presence of bacteria, the Burrill bacterial corn disease.\*

The mouldy ears would easily be recognized by an ordinary observer by a dense "felt-like" mould, of a white or grayish-white color, with which was mingled more or less "worm dust." The injured ears were smaller than the average, and the silk and husks would cling to the ear very closely about the injured spot, making it difficult to husk, and much moldy corn

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\*Bulletin No. 6, 1889, Agr. Exp. Station, Champaign, Ill.



CORN ATTACKED BY MOULD (*Aspergillus glaucus*).

Photographed by Kellerman and Mayo.

was left in the fields. This might account for the greater loss among horses that were pastured in the stalk fields. Owing to the short corn crop, much mouldy corn was fed to horses, that would otherwise have been fed to hogs.

Specimens of mouldy corn were handed to Prof. W. A. Kellerman, of this Experiment Station, and the mould was identified by him as *Aspergillus glaucus*, one of our common moulds.

As all evidence pointed to the mouldy corn as the possible cause of the disease, an attempt was made to get an extract which would contain the poisonous properties of the injured corn, but various concentrated watery and alcoholic extracts failed to possess any poisonous properties when given by the mouth or injected hypodermically. Failing in this, I next turned my attention to the search for a "germ" of some kind, as the probable cause of the disease. A careful microscopical examination was made of the blood, brain tissues, liver, spleen, and kidneys, together with plate and beef-broth cultures, but all failed to give any pathogenic bacteria, though in two cases where the inoculation was made from the liver, I obtained a growth of mould. This was thought to be accidental at the time, and as I was looking for a germ no attention was paid to it; but further experience leads me to believe that the mould was *Aspergillus glaucus*, and that the spores were obtained from the liver.

Failing to find a germ upon which to lay the blame, a feeding experiment was begun. On June 30, a large two-year-old colt was put up and fed upon mouldy corn and good prairie hay. The colt was very thin in condition. He was given a good feed of mouldy corn, picked out from the college crib. After feeding the corn three times daily for two weeks, no change was noticed, except a gain in flesh and general appearance; so a change was instituted. The corn was thoroughly moistened, and kept in a moderately warm place until the mould had begun to form spores. I commenced feeding the fresh, mouldy corn to the colt on July 16. His pulse and temperature were taken twice daily, morning and night. His temperature varied from 101° to 102½° F., standing at 101½° most of the time, with pulse at 48 beats per minute. On the evening of July 26, his temperature registered 102½° F., pulse, 53. He seemed slightly uneasy, but ate his feed and drank as usual. He died during the night, and with apparently little struggling. Early the following morning an autopsy was held, with the following result:

Body fairly well nourished; no apparent diminution in weight during latter part of experiment visible; mucous membranes normal in color.

The stomach was partly filled with partially-digested food, and was considerably irritated toward the pyloric end. This irritation extended through the small intestines, giving them a dark-red appearance. The large intestines were normal; spleen and pancreas normal; but the liver was much engorged, and easily broken down, being difficult to handle on this account. The capsule was considerably thickened, and adherent to the diaphragm.

Both kidneys were enlarged, the left more than the right, and on the sur-

face were dark spots, varying in size from one to three millimeters. These spots appeared to be of a hemorrhagic character, and extended into the medullary substance of the kidney. The pelvis of the left kidney contained a small quantity of pus, from the rupture of a small renal abscess.

The bladder contained a small amount of urine, in which was found some pus, mucus and blood, which gave it a dark, thick appearance. On examining the circulatory system a large *ante-mortem* clot about five inches long was found in the left auricle, extending into the ventricle, and had evidently caused the death of the animal. The respiratory system was normal. On exposing the brain the cerebral meninges were engorged, and in cutting into the white central substance of the left cerebral hemisphere a spot of yellow softening was found about the size of a pea. This spot was examined microscopically for foreign substances, but without success. Up to this stage of investigation I had been unable to find any literature regarding infection by moulds, as no experiments have been performed in this country, but after the close of the feeding experiment I found a record of experiments\* performed by M. Kaufmann, of the Lyons Veterinary School, of France. Kaufmann reviews the work done up to that time. In 1869 Grohe and Block had produced fatal infection in rabbits by injecting into their veins the spores of *Penicillium glaucum* and *Aspergillus glaucus*. Cohnheim and Grawitz doubted the results and failed to reproduce them, but later, in 1880, Grawitz succeeded in producing infection by spores adapted to all alkaline medium.

Under the direction of Chauveau, Kaufmann attempted to prove that the spores of *Aspergillus glaucus* were infective without any previous adaptation. The following is the result of one of his experiments, which was corroborated by many others. He says :

"On May 12, on damp bread I sowed the spores of *Aspergillus glaucus* procured from the surface of a solution of gum-arabic. This cultivation placed in a water bath, and kept at a temperature of 35° C., furnished numerous spores in 48 hours. In order to obtain an abundance of spores, I made a new cultivation on bread reduced to broth, with an acid reaction, using for this purpose spores obtained by the preceding cultivation. This second crop, like the first, furnished spores in abundance in 48 hours. I left these cultivations in the bath until May 19, and on the evening of that day I put a quantity of spores of the second generation in water enough to make it look slightly turbid. Into the jugular vein of rabbit (No. 1), I injected one centilitre of this fluid, and into another rabbit (No. 2), two centilitres. During the night of the 23d, 24th, rabbit No. 1 died, while rabbit No. 2 was very ill, turning its head towards the side and foaming at the mouth. At the autopsy, there were found in both rabbits the typical lesions of infection by moulds such as Grawitz had described. The kidneys were highly congested in places, and on their surfaces were a multitude of white nodular points. On section from the periphery towards the hilum, it was noted that each white point on the surface was prolonged towards the medullary portion by a white line. Examined microscopically, in all these nodules, the mycelium was found to be felted. In rabbit No. 1, the mycelium tubes were yet

\*Archives Vétérinaires, 1882, p. 861, Fleming.

perfectly recognizable; they were felt and partitioned in every respect similar to those described by Grawitz. In rabbit No. 2, which lived a day longer, the mycelia had almost completely disappeared. Some fragments were noticeable which were easily broken up. In the liver were also numerous white spots, which contained mycelia in process of destruction. The lungs showed a small number of white nodules, but no mycelium tubes could be found, only granules, which were doubtless the product of disintegration of the mycelia under the influence of the inflammation its presence produced in the lung tissue. Similar white points were also found beneath the pericardium, and in the walls of the stomach.

"In these two rabbits the spores of *Aspergillus glaucus*, cultivated on bread which had an acid reaction, produced a mortal infection exactly similar to that which Grohe and Block obtained, and also similar to that induced by Grawitz', with their malignant varieties previously adapted to the character of the blood by gradual cultivations.

"The spores which I injected into the rabbits did not undergo any process of adaptation to enable them to live in the blood, nevertheless they germinated and vegetated in the organism. Previous adaptation is therefore needless in order to render the spores of *Aspergillus glaucus* infective."

Kaufmann mentions the experiments by Koch and his assistants, Loeffler and Gaffky, which are analagous to those obtained by himself. These German investigators believed they had discovered the cause of Grawitz's failure. Kaufmann concludes as follows:

"1. The *Aspergillus glaucus* grown on bread may produce fatal infection in the rabbit, even in the extremely small dose of 1.10 milligrammes. Subsequently it was found that .05 milligramme of spores was sufficient to kill a large rabbit.

"2. That its previous adaptation to a liquid and alkaline medium, and to a temperature of 39 deg. C., is not requisite to confer infectious properties.

"3. That if this adaptation exercises any influence, it is accessory and very slight.

"4. The spores exposed to the temperature of the air for nearly six months preserve all their infective properties."

An experiment on the same plan as those of Kaufmann was then undertaken. In the place of bread broth I used "bran mash," having sterilized the bran by heating it in the sterilizer to 300° F. for an hour. It was mixed with distilled water and boiled for an hour, then allowed to stand for 24 hours, boiled again for an hour, and when cool was inoculated by sowing spores from a previous culture obtained directly from the mouldy corn. In a wet chamber at the ordinary temperature of the room the mold grew rapidly, and in 48 hours had produced an abundance of spores. These spores were gathered and added to distilled water until it was slightly turbid, and .5 cc. of this fluid was injected into the left internal saphena vein of a full-grown Guinea-pig on the morning of September 8. The temperature of the pig just before the inoculation was 102 1/3 °F. On the morning of the 9th the pig appeared quite sick, ate but little, remained crouched in the corner of his cage, his hair bristling, and breathed rapidly, with temperature 103° F. No other change was observed, except a rise in temperature to 103½° F. on the morning of September 10, when it was de-



cided to hold an autopsy, with the following results: On the surface of the lungs were a few hemorrhagic spots, but I could find no mycelia of *Aspergillus glaucus* in them. In the kidneys were a number of white spots, and still more in the liver. The liver was much enlarged and of a dark purplish color. On examining these spots under the microscope, after cutting and washing away the tissue, one or two mycelia of *Aspergillus glaucus* would be found, ranging from two to four mm. in length, and constructed and partitioned in two or three places. Surrounding these mycelia were large numbers of leucocytes. Other organs were normal.

#### OTHER OUTBREAKS OF THIS DISEASE.

In the Report of the United States Department of Agriculture (Veterinary Division), for 1881-'82, I find reported two outbreaks of a disease which resembles the one under consideration. Both outbreaks were investigated by Dr. H. J. DeTmers, and pronounced cerebro-spinal meningitis. The first of these two outbreaks, where five out of six animals died, was in a pasture in which were the "rotten remnants of two oat stacks, fit only for manure." These oat stacks had furnished the principal food for the horses during the summer. There were also some Jamestown weeds in the pasture, and the water was not of the best. All these, taken together, Dr. DeTmers concludes, were the cause of the disease. No other horses in the locality were affected, and probably many others drank water fully as bad. Might not the mouldy oat stacks be the cause?

The second outbreak occurred in Texas, in 1882, and was very disastrous, 500 head of horses dying in the vicinity of Corpus Christi. Dr. DeTmers concludes that the cause of this outbreak was impure drinking water. What this impurity was is not learned. Some of the stockmen of the locality thought the disease the result of pasturing while the dew was on the grass. The land to which this outbreak was confined was a narrow strip along the coast, which had been inundated, and was "covered by such plants as prefer to grow on ground inclined to be wet," a most admirable place for various fungi. In other years, when there was a much smaller rainfall, they were not troubled with the disease.

In 1886 a serious outbreak of a disease, with symptoms exactly, similar as far as one can judge from descriptions, occurred in Virginia and North Carolina, and was investigated by Dr. Harbaugh, of the Bureau of Animal Industry. The outbreak occurred during July, August and first part of September, but was entirely over when visited by Dr. Harbaugh, the first of November. Dr. Harbaugh says that every case of the disease he could trace directly to mouldy food. No mules contracted the disease, nor did any horses suffer that had been fed upon feed shipped in from the West. He also says as to the cause of "staggers": "The evidence of fungi is too plain to be overlooked by anyone who thoroughly investigates this matter, and all circumstances connected therewith." Other outbreaks of "staggers"

were reported in connection with the above by farmers and stockmen, which were supposed to have been caused by mouldy food.

Whether the disease called "staggers" in Kansas is identical with what is commonly known as "bottom disease," where animals are pastured upon river bottoms, I cannot say. The symptoms described of "bottom disease" are much like those of staggers, and the conditions for the disease so favorable that it would lead one to believe that, if not identical, they are closely related.

The symptoms of "staggers" are almost identical with those of cerebro-spinal meningitis, and by some veterinarians have been diagnosed as such; but the post-mortem results differ much. In "staggers" the formation of a cerebral abscess and inflammation of the liver and kidneys, and I should consider them separate diseases, having different though probably closely-related causes. For the disease commonly called "staggers," from the prominent cerebral symptoms, I should prefer the name of *Enzoötic cerebritis*. Though spinal symptoms occur, the cerebral predominate largely, and this would distinguish it from other cerebro-spinal affections.

#### CONCLUSIONS.

The disease variously known as "staggers," "mad staggers," etc., as occurring in Kansas during the past fall and winter, is caused by feeding corn which has been attacked by a mould--*Aspergillus glaucus*. The spores of this mould gain entrance to the circulation, and find lodgment in the kidneys and liver. The latter is more affected than the kidneys, (probably on account of the lower pressure of the circulation.) The spores germinate here, and cause inflammation of these organs. The cerebral symptoms are the result of the formation of an abscess in the cerebrum. This abscess is caused by an interference with the blood supply, probably from spores or mycelia of the mold in the circulation. The spores of *Aspergillus glaucus* seemed to retain their infectious properties for about six months, from October, 1890, to March, 1891. Mules, cattle and pigs do not contract the disease.

*Treatment.*--In this disease, an ounce of prevention is worth many pounds of cure. The method of prevention is obvious: Do not feed mouldy corn, or turn horses into fields where mouldy corn can be had. In feeding ear corn from the crib, care should be exercised to pick out the mouldy ears, or break off the mouldy tip. In case the corn has been shelled, it can be poured into water, and the mouldy kernels, floating, can be skimmed off.

After an animal has been taken sick, treatment is very unsatisfactory. The animal should be kept as quiet as possible, in a clean, dry, well-ventilated and strong box-stall. A purgative may be given, of about seven drachms of aloes. One drachm of the iodide of potash or three drachms of the bromide of potash can be given in sufficient water every three hours, and cold applications to the poll by means of wet cloths are helpful. In case the spinal cord is affected, a moderate blister can be applied along the

spine. Care should be taken to excite the animal as little as possible, and to avoid choking it in giving medicines, as it is often difficult for the animal to swallow.

NOTE.--The Veterinarian of the Kansas Experiment Station would like to have farmers and stockmen of the State report concerning various diseases affecting live stock; giving the kind of stock, their age, food, and surroundings, the number affected, the symptoms and nature of the disease as far as known. All questions concerning diseases of live stock will be answered, so far as possible.