Black Disease

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Illustration on cover—

Figure 1. The type of pasture where Black Disease is found.
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SUMMARY

1. The cause of a heretofore mysterious disease of Oregon sheep has been found.

2. The cause of this disease is an anaerobic bacterium Cl. novyi B (B. oedemations).

3. This organism is identical with one found by Montana workers and is identical with the organism found in other countries.

4. The disease is definitely associated with fluke infestation.

5. Some of the symptoms and lesions are similar to, and are sometimes confused with, so-called hemorrhagic septicemia.

6. Treatment with 1 cc. doses of carbon tetrachloride or flukoid does not stop losses.

7. The use of copper sulphate on fluke-infested pastures will help prevent losses from this disease.

8. Satisfactory methods of treatment for this disease have not been found.

9. Methods of preventing this disease are being studied.
Black Disease
(Infectious Necrotic Hepatitis of Oregon Sheep)

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INTRODUCTION

As the common name of the disease with which this bulletin deals is not very well known and the scientific name is little understood, the title "Black Disease" is not very satisfactory. No real common name receives any considerable use. Sheep men simply describe losses as occurring suddenly without the appearance of a warning sickness. A good many of the sheep men and some veterinarians have confused this disease with so-called hemorrhagic septicemia, and have persisted in using hemorrhagic-septicemia vaccine in efforts to protect their animals. The disease in no way resembles so-called hemorrhagic septicemia, unless it might be that in both diseases small hemorrhages appear on the surface of the heart and under the endocardium or the lining of the heart cavities.

In the British possessions, the disease is commonly known as Black Disease, so-called because the insides of the pelts, when removed, take on a very dark or black appearance. Perhaps this common name will become popular in this country if sheep men consistently pelt animals dead from the disease.

The scientific name, infectious necrotic hepatitis, indicates that the disease is caused by a bacterium that lives on dead liver tissue, the sheep dying as a result of a toxin, or poison, produced by the bacteria. Scientifically, the disease is a real toxaemia caused by an organism growing on the dead liver tissue in the absence of oxygen.

The disease is quite widespread in Oregon (1), appearing on so-called fluke-infested pastures. More cases have been reported from Douglas, Lane, Benton, Linn, and Marion counties than from other fluke-infested counties, but no doubt such losses occur where flukes are common. So far as known, the disease does not appear on pastures where flukes are not found. Evidence indicates it is definitely associated with fluke infestation.

SYMPTOMS AND PATHOLOGY

Opportunities to study the symptoms of this disease have been rare. In those cases observed, rapid breathing seemed the most noticeable, while only a small rise in temperature was present. Farmers have reported that affected sheep lag behind and that they could be detected by the use of a dog, but these symptoms might easily be those of acute fluke infestation. A further study of the symptoms will be necessary before it can be said that there are any that are characteristic. Sheep men report finding sheep dead on the bedding ground, lying in a natural position, as if they had died in their sleep. No evidence of struggling can be found. In some cases, a foamy
discharge from the nose is present, and at times this is somewhat colored with blood. Animals dead for some time are considerably bloated. Dead sheep are usually found lying in shaded places along small creeks and in groves bordering swampy areas. This is because the disease appears during the dry months of the year and, where possible, sheep feed in such areas. The losses occur more or less irregularly.

Upon autopsy, these animals show what might be considered characteristic lesions. If the carcass is fairly fresh, a peculiar sweetish odor is present, not one characteristic of extensive decomposition, but one always found in these cases. Opening first the thoracic cavity, it is noted that the lungs are usually normal in appearance, although some congestion might be present. Often some clear-colored fluid is present in this cavity. The heart is enclosed in a pericardium that is distended with fluid, and when the pericardial sac is opened, it is found that some of the fluid has coagulated and formed what is known as a chicken-fat clot. This is said by the British workers to be always present. Examination of the heart usually discloses a few small hemorrhages under the epicardium and always many such hemorrhages under the endocardium, especially under that lining of the left ventricle.

Usually some small amount of fluid is present in the peritoneal sac. This may be tinged with blood, but in most cases is clear.

Figure 2. Shells containing Lymnaea (Galba) bulimoide Lea. Enlarged 3 times. Snails used by fluke in its reproduction.
Figure 3. Encysted form of liver fluke on leaves—actual size.
The liver is congested and contains from one to many necrotic areas varying in size, some being as large as a fifty-cent piece. These necrotic areas sometimes are not apparent until the liver is sliced. A few small holes may be present in the capsule of the liver as a result of the migrations of the fluke, but such holes are not always present. The surface of an infected liver often shows under the capsule several hemorrhages that, when opened up, reveal the presence of immature fluke. Fluke burrows also may be found on the surface, but are significant only in that they indicate the presence of immature parasites.

The other organs of the abdominal cavity present no abnormalities of any importance. The abomasum is often congested in the pyloric portion, and the blood vessels of the mesentery are usually filled. Occasionally, the first portion of the small bowel is congested for several feet.

In some cases that have gone several hours before examination, the kidneys are greatly softened. Usually, though, only a mild congestion is present.

THE CAUSE OF THE DISEASE

The cause of this disease has been proved to be due to the invasion and growth of *Cl. novyi B* (2) (*B. oedematiens*) (3) in fluke-damaged livers, resulting in the liberation of a lethal toxin. This organism was first isolated by Dodd (4) in New South Wales. More recently, it has been found in widely separated regions (2). It was first recognized in the United States by Marsh and his co-workers (5). Turner and Davesne (6) first identified this organism as *Cl. novyi B* (*B. oedematiens*).

CULTURAL METHODS AND BIOLOGICAL CHARACTERISTICS OF THE CAUSATIVE ORGANISM

Due to the insidious course of the infection and the relative absence of symptoms, it has been difficult to secure fresh specimens for bacteriological examinations.

During the late summer of 1938, an outbreak of sudden losses occurred in a band of Hampshires pastured on fluke-infested land in Linn County. One of these sheep was presented to the Experiment Station laboratory in a comatose condition. Upon necropsy, the lesions were found to be typical of Black Disease.

**Isolation.** Smears from the liver lesions showed the presence of an apparently pure culture of large Gram-positive rods with rounded ends, a few showing subterminal spores. A portion of the necrotic material was aseptically removed and sowed into several tubes of peptic digest broth (7), to which egg-meat medium (Difco) had been added. Some of the tubes were incubated anaerobically by the sodium hydroxide-pyrogallic acid method of Buchner (8). After 24-hours' incubation at 37° C., examination revealed that the growth consisted principally of Gram-positive rods, morphologically identical with those seen in direct smears of the affected liver, and a few coccus forms. Examination of the aerobic cultures revealed a predominance of Gram-positive cocci. The following steps were taken to obtain the anaerobe in pure culture. The coccus forms were eliminated by placing a
small quantity of the culture in sterile 10 cc. saline blanks, and heating in a water bath to 70° C. for thirty minutes (9). Five-tenths cubic centimeter of the contents were then successively inoculated into a series of 1 per cent peptic digest deep agar tubes and incubated at 37° C. for 48 hours (10) (11). Well-isolated colonies extending to within 8 mm. of the surface were apparent at this time. The cultures were apparently pure, as judged by the similarity of the colonies. The tubes were gently heated and the contents expelled into sterile Petri dishes by inserting a capillary pipette to the bottom of the tube and blowing air through the pipette. The agar was flamed and individual colonies were picked with a sterile capillary pipette and transferred to tubes of peptic digest broth to which egg-meal medium had been added. Prior to inoculation, the tubes of media were boiled to drive off absorbed oxygen. It was apparent that the organism grew more luxuriantly in media that had been treated as above.

Morphology. In smears made from 24-hour cultures, the organism somewhat resembled Clostridium welchii. However, the rods were longer and the ends more rounded. The bacilli usually occur singly. As a rule, there is a marked tendency to morphological variation in older cultures. Some appear as short rods, others beaded and partly disintegrated, while some are long filamentous forms. In young cultures the bacilli stained Gram-positive, but Gram-negative forms predominated in cultures four to five days old. Examinations for motility were made by placing cultures in flattened sealed capillary pipettes (12). The organisms were sluggishly motile.

The spores were oval, slightly bulging the cell wall. They were either central or subterminal in the vegetative cell, depending on the age of the culture. Sporulation did not occur freely in the cultures examined.

Cultural characteristics. Cultural characteristics of the causative organism have been determined as follows:

One per cent peptic digest agar shakes: In 24-hour agar cultures, the colonies were opaque and discreet, becoming diffuse, woolly, and flocculent with prolonged incubation.

Peptic digest broth: The growth was diffuse and showed auto-agglutination in about 48 hours, resulting in the settling out of flocculent mass and leaving a clear supernatant liquid.

Peptic digest egg-meat medium: Growth took place rapidly in this medium, producing a slight amount of gas, becoming turbid at first, but on continued incubation auto-agglutination occurred with subsequent settling of the bacilli on the meat. The meat was bleached but not digested. The odor was characteristic but not putrefactive in nature.

Gelatin: After seven days' incubation at 37°C., followed by twenty minutes in refrigerator at 4°C., the gelatin was softened but not liquefied.

Litmus milk: Cultures in this medium showed slight acidification but no coagulation after seven days' incubation.

Fermentation reactions. The basal medium used for the fermentation tests was Phenol Red Broth base Ph 7.4 (Difco) to which 0.25 per cent agar had been added. After sterilization, appropriate amounts of sterile concentrate solutions of various sugars were aseptically added to give a final 1 per cent sugar concentration. The tubes were boiled to expel the air, cooled, and just prior to inoculation 0.5 cc. of sterile horse serum was
added as an enriching substance. The tubes were incubated at 37°C. and examined periodically for evidence of growth, color change, and gas production. This strain was found to ferment dextrose and maltose with the production of acid and gas in both. Galactose, levalose, lactose, inulin, and salacin were weakly fermented, while saccharose, mannite, and glycerin were not. These results indicate that the fermentative powers of the organism are relatively weak.

Experiments in pathogenicity. Intramuscular injections of 36-hour peptic digest egg-meat medium cultures proved fatal to guinea pigs in doses of 0.02 cc., while 0.1 cc. proved fatal to sheep weighing about 70 pounds. In sheep there were no symptoms except a swelling at the point of inoculation, causing a severe lameness, when the injection was made into one of the quarters. The animal became apathetic and died within 18 to 60 hours, depending on the amount of inoculum.

On necropsy, sheep infected in this manner differed from naturally infected cases in that there was a marked subcutaneous hemorrhagic congestion and edema originating at the side of the inoculation and involving the entire quarter when the injection was made in the leg muscles. There was no evidence of gas formation. The lesions were otherwise similar to those found in naturally occurring cases, with the exception of the absence of liver lesions.

Other workers (3) (13) have proved most of the domestic and laboratory animals susceptible to this infection.
Toxin. The virulence, or the ability to produce toxin in peptic digest egg-meat medium, did not diminish appreciably with this organism after several months.

Cultures were grown in this medium for 48 hours at 37°C., filtered through gauze, and then through a Mandler filter. The filtrate was checked for sterility.

When injected intravenously into sheep, the average lethal dose was 2 cc. for a 70-pound animal. The period between injection and death was relatively long, averaging 26 hours, and was accompanied by no apparent symptoms. The lesions produced by the injection of toxin were similar to those produced by the injection of cultures. Subendocardial and subepicardial hemorrhages, and congestion of the pyloric portion of the abomasum, similar to the lesions occurring in naturally infected cases, were present.

The reaction of the filtrate varied between Ph 6 and Ph 6.2. It was strongly hemolytic for washed sheep corpuscles. Heating at 70° C. for 30 minutes apparently inactivated the toxin. Incubation at 37° C. in the presence of 0.3 per cent formalin also rendered it atoxic in from three to five days.

Neutralization by specific antisera. Turner demonstrated in his neutralization experiments that the antisera of Clostridium novyi was homologous regardless of the origin of the strain (3). In view of this fact, and to assure ourselves that the organism isolated by us was a strain of Cl. novyi B, antiserum was obtained from the Cutter Biological Laboratories.

One cubic centimeter of a 48-hour culture (equivalent to 10 average lethal doses for a 70-pound sheep) was mixed with 1 cubic centimeter of antiserum. The mixture was allowed to stand at room temperature for one hour before use. Three sheep were used in the following experiment.

Sheep A received an intramuscular injection of 1 cc. of culture plus 1 cc. of antiserum.

Sheep B was injected with 1 cc. of culture plus 1 cc. of normal horse serum.

Sheep C was injected with 1 cc. of culture plus 1 cc. of uninoculated media.

Sheep A did not show any reaction except a slight lameness, which disappeared within 48 hours. Sheep B and C died within 36 hours.

As the pathogenicity of the culture was neutralized by the specific antiserum, it was concluded that this organism, which has the morphological and cultural characteristics of Cl. novyi, is a variety of, or identical with, Cl. novyi B (B. oedematiens) of infectious necrotic hepatitis.

Dr. E. A. Tunnicliff (14), of the Montana Veterinary Research Laboratory, has compared this strain with strains isolated at that laboratory and has reported that they seem to be identical.

Cultures sent to R. F. Montgomerie (15) of The Wellcome Physiological Research Laboratories of England proved to be Cl. novyi B (B. oedematiens).
RELATIONSHIP TO LIVER FLUKE INFESTATION

In 1930, attempts were made at this station to reproduce this disease in sheep. Organisms similar to the one recently isolated were obtained and fed to sheep together with the encysted forms of the liver fluke. This same organism was also fed to sheep at a time when it was known that the immature fluke were burrowing into the liver. These experiments gave negative results and, as sudden losses were only one part of the fluke problem, research efforts were directed toward other phases of the work. This summer, losses were reported from registered ram lambs on fluke-infested pastures, and while only two autopsies were held, an organism was isolated and immature parasites were found.

British (3) workers have quite definitely proved the association of the disease caused by the bacteria with the lesions produced by the immature worms. Experiment station workers here have long believed in this association, but were working more with the parasitological than with the bacteriological side of the problem, and therefore had no proof of this association until this spring.

Since there will be much confusion as to the causes of losses due to acute liver rot and this Black Disease, it is deemed advisable to include in this publication information on the life history of the liver fluke and the symptoms and pathology produced by it.

LIFE HISTORY OF LIVER FLUKE

(Fasciola Hepatica)

Snails as hosts. The life history of the liver fluke was first worked out in England by Thomas (16) in 1881. He proved that the parasite utilized a small fresh-water snail as one step in its reproduction process. In this country several species of such snails have been found to harbor the parasite. The two snail hosts in Oregon are Galba bulimoides Lea and Galba ferruginea Haldermann. These fresh-water snails are quite common and found in all parts of the state where wet, swampy pastures are found. These snails are small and spoken of as right-handed—right-handed in that when the snail is held with the spire, or pointed end up, the opening is on the right. This helps distinguish them from many other snails that are found in the same pastures. They are dark brown in color and vary in size depending on their age. Full-grown snails average 10 mm. in length. The species bulimoides is more obese than ferruginea, and seems to be the preferred host of the parasite in this state.

Snails are attached by miracidia, embryos from hatched fluke eggs, at a very early age. Half-grown snails often have been found that would discharge cercariae, the form of the parasite that is taken in with the food of the sheep.

Snails do not always stay in the water, but may be found wandering around over the mud and in the grass. As many as twenty have been found in a single cow track. Snails are said to bury themselves in the ground during dry weather, but also have power of migrating from one field to another.

According to Baker (17), the average snail lives about five years, and in this climate reproduces at all times of the year. Very small snails have been found during all months of the year.

Rediae and cercariae. Rediae and cercariae are the forms that develop in the snail. The cercariae are of especial interest to livestock men because
these are the forms that enter the digestive tracts of their animals. The cercariae, as they swarm from the snail, are milk-white in color and about the size of an ordinary pin head. They swim about by lashing a tail that is about twice as long as the body is wide, and are quite easily seen with the naked eye if one knows what is being looked for. Cercariae encyst on almost any object they come in contact with. They are capable of encysting on the water, and it is thus possible for them to be transferred for considerable distances by currents that are present in the small creeks and channels found in swampy pastures. The wall formed upon encysting serves as a very definite protection against the elements.

Cercariae are very long lived under favorable conditions and have been known to remain alive on hay as long as eight months (18). How long they would live under pasture conditions in Oregon has not been definitely determined.

After cercariae are taken in with the feed, they burrow through the walls of the intestine and can be found for the next five days wandering about in the peritoneal sac and burrowing through the capsule of the liver. During their wanderings in the peritoneal sac, very little pathology is produced, but once they enter the liver considerable damage is done. Liver cells are destroyed, blood vessels are interfered with, and as a result the appearance of the liver changes from a natural color to a grayish color. The surface is roughened and adhesions are present. Often the surface of the diaphragm is involved, and that next to the liver is covered with tags. The surface of the liver has a mottled appearance, showing hemorrhages under the capsule and burrow tracks of flukes.
If the animal lives through the acute stages of fluke infestations, the liver gradually changes back to a somewhat more normal appearance. After the flukes enter the bile ducts the liver has a pitted appearance, the pits being the result of the healed fluke burrows. The bile ducts are greatly enlarged and thickened, and when opened many flukes are usually revealed.

Eggs of the fluke appear in the droppings of infested animals, usually not earlier than eleven weeks after the cercariae are taken in with the feed. Eggs that pass with the feces, or droppings, of infested animals are capable of resisting very adverse circumstances for long periods of time. Under favorable conditions eggs hatched in fourteen days, while others, also taken from gall bladders of infested animals, hatched over a period of thirteen months and twenty days (1).

**METHODS OF CONTROL**

Methods of controlling Black Disease include (a) destruction of flukes by either treatment of pastures or treatment of infested animals, (b) development of immunity by the use of fluke extract or of vaccines, antitoxins, or toxoids.

**Treatment of pastures.** In pastures where only small areas are involved, treatment with copper sulphate is effective in killing the snails that act as intermediate hosts. The use of copper sulphate for this purpose is not always practical, though, for these reasons: It is too expensive to treat large areas. Some swampy areas are so covered with vegetation that the
treatment would not be effective. Again, the pasture treatment would not be effective because of the lack of cooperation on the part of neighbors. Snails are easily transported on leaves, sticks, etc., and treated pastures would become quickly reinfested from neighboring fields and streams.

Drainage would be very effective in destroying snails, but has this objection: Grass grows abundantly in the wet places during the summer months because of the presence of moisture; drainage then would not only make undesirable places for snails, but would also reduce the production of feed. Where practical, the use of copper sulphate is advised, as there can be no doubt that snail destruction does decrease the possibility of fluke infestation.

![Figure 7. Sheep dead in 24 hours from Black Disease. Insert shows causative organism (X 2000).](attachment:image)

**Treatment of infested animals.** The use of carbon tetrachloride in treating fluke-infested sheep and goats is very satisfactory except for two things. The one cubic-centimeter dose does not destroy immature parasites and occasionally does kill sheep. Just why sheep should be susceptible to one cubic-centimeter doses is not known. If sheep are not susceptible, fifty times the medical dose can be used without producing damage. Although cattle are always susceptible and occasionally sheep, no such instances have been reported in goats. Many efforts have been made to reproduce this peculiar condition in sheep, but to date only negative results have been obtained.

In an effort to avoid losses from carbon tetrachloride poisoning, it is always advised that the treatment be used on a few representative animals first. If no symptoms or losses are produced at the end of four days, other sheep of the flock may be treated. Not enough information is available to determine any satisfactory method of preventing the condition or over-
coming it, once it is known to exist. When it is possible to produce this condition artificially, the solution to the problem should be found.

If sheep are dying of acute liver rot or immature liver-fluke infestation, large doses of carbon tetrachloride will cause the destruction of the parasite. Montgomerie (19) has shown that ten cubic centimeters will kill flukes that have been in the liver five weeks, while five cubic centimeters will kill those that are eight weeks old and older. Experimentally infested sheep with as many as 650 cysts did not develop symptoms of acute liver rot. In our animals it was found that about twenty per cent of the cysts fed reached the liver.

Immunity by use of fluke extract. Others (20) have shown it possible to produce immunity to liver-fluke infestation in rabbits. In our work with sheep, while some immunity might have been produced, it was possible to infest and treat some sheep as many as five times. Reactive substances are produced in fluke infestations for this was clearly demonstrated when freshly ground fluke were injected into the jugular veins of normal and previously infested cattle and sheep. No symptoms were produced in normal animals, while typical symptoms of shock appeared in those having been infested with the parasites and those animals having parasites in the liver. Clearly the symptoms and lesions were those of shock and one six-months-old calf was down almost before the needle could be removed. Symptoms were relieved in a few minutes by the injection of adrenalin. Kellaway (21) has shown that fluke extracts contain two antigenic substances.

Sheep dead from shock and those injected with histamine show exactly the same lesions found in necrotic hepatitis with the exception of the necrotic areas in the liver.

Use of vaccines, antitoxins, and toxoids. British workers (3) report excellent results from the use of vaccines, antitoxins, and toxoids. These products will be given trials in Oregon as soon as opportunities are presented. Montana workers (22) also reported in a personal communication on the use of a toxoid.

DISCUSSION

Black Disease is not a new disease of Oregon sheep, but one for which the cause has been newly isolated. Since the name of the disease and the symptoms are not closely associated in the minds of the sheep men, considerable educational work is anticipated before the best results will be had as far as control is concerned. It is hoped that widespread distribution of this publication will help in this educational work.
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