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## FIELD AND LABORATORY NOTES ON A FATAL DISEASE OF CATTLE OCCURRING ON THE COASTAL PLAINS OF TEXAS

(LOIN DISEASE)



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\*In cooperation with School of Veterinary Medicine, A. and M. College of Texas.

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A disease of cattle manifesting itself by a sudden and complete breakdown of the organs of locomotion and locally called loin disease or down-in-the-back is herein described.

This disease is prevalent from May to September in the low flat area bordering on the Gulf of Mexico and affects cattle over 18 months of age in case such cattle have been subsisting entirely on the native vegetation for a period of at least one year. It is not observed in dairy cattle fed a dairy ration.

The outbreaks of the disease simulate an infectious disease, but in repeated experiments pathogenic organisms could not be demonstrated.

The cause of the disease is tentatively ascribed to toxins produced by bacterial action in carcass material on the prairie and the consumption of such putrid material by cattle.

It is tentatively recommended to feed sweet bone meal to cattle in order to stop them from eating dangerous carcass material; also to thoroughly clean all pastures of animal carcasses.

Investigation of this disease is not yet complete and experiments are under way to definitely prove whether putrid carcass material is the sole source of the toxin and to establish the best methods of preventing the disease.

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## FIELD AND LABORATORY NOTES ON A FATAL DISEASE OF CATTLE OCCURRING ON THE COASTAL PLAINS OF TEXAS

(Loin Disease)

### A PRELIMINARY REPORT

H. Schmidt

#### HISTORY AND DISTRIBUTION OF THE DISEASE

A rapidly fatal disease of cattle, known locally as "loin disease" or "down-in-the-back," occurring on the prairie region along the Gulf of Mexico in the State of Texas has been known to cattlemen in that territory for a long time. Occasionally reports of this hitherto undescribed<sup>1</sup> malady of cattle reached the Division of Veterinary Science of the Texas Agricultural Experiment Station as early as 1910. These reports gradually became more numerous and calls for help more insistent, but it was not until 1918 that a small fund was provided to investigate this malady. The funds available were entirely inadequate to do much effective work and for that reason the activities of the Division of Veterinary Science were limited to making occasional calls whenever a report came in with the view of collecting material for microscopical and bacteriological study.

This method of procedure proved entirely unsatisfactory for two reasons. In the first place, many times the animal could not be reached in time to satisfactorily collect material. In the second place, range cattle were so cheap that cattlemen would seldom take the trouble to call on the Division for help. For these reasons it was impossible to make any satisfactory progress and it, therefore, became necessary to change the method of attack in spite of the handicap of limited funds available. It was, therefore, decided to place a man in the field during the seasonal prevalence of the disease in order to collect data on its occurrence and specimens for laboratory study. This plan was inaugurated in May, 1921, and as many cases studied as possible with our limited force.

As the investigation progressed it was learned that the disease centered in Harris County, but reports of its occurrence, apparently to a lesser degree, also reached the Division from Galveston, Colorado, Brazoria, Austin, Waller, Fort

<sup>1</sup>Dr. A. T. Kinsley, who was in the field with the writer during the 1921 outbreak published his observations on this disease in *Veterinary Medicine*, Chicago, Vol. XVI, No. 7, p. 24.

Bend, Matagorda, Wharton, Lavaca, and Hardin Counties. It is not yet definitely known whether the occurrence of the disease is limited to these counties; however, it appears that the disease is limited to the low, flat area bordering on the Gulf of Mexico and extending inland to the north to a distance of about 100 miles and ranging in elevation from sea level to about 250 feet above. This area is traversed by numerous creeks, bayous, and drainage ditches, but on the whole the drainage is very poor and many small, shallow surface ponds are found. The land, however, does not become boggy, but few bogs occurring excepting along the creeks and bayous. In the low places, especially along the bayous, in and around the surface ponds and in the drainage ditches along the roads, the coffee bean, (*Daubentonia longifolia*), grows very abundantly. Over the entire area grasses and many different kinds of weeds grow very luxuriantly. The soil is usually non-calcareous.

Cattlemen who have been in the cattle business in the affected area for a long time say that this disease has been prevalent in Harris County for the last twenty years, or even longer. One man stated that he observed the disease during the Spanish-American War, at which time he was buying steers for a Galveston firm and which were being shipped to the American forces in Cuba. At that time he frequently observed that steers would get down and die without any apparent cause after he had bought them and before he could deliver them to the Galveston firm. He is convinced that the disease occurred in Harris County three or four years previous to that time. Others say that the disease was first observed in the pastures of Calvin Ehrhardt at Bammel, Texas, in the northeastern part of Harris County, and that it was introduced by him with cattle which he is reported to have acquired in Louisiana. From Mr. Ehrhardt's pastures the disease is reported to have spread westward. Answers to inquiries indicate, however, that this disease is not and never has been prevalent in Louisiana. How this spread may have taken place cannot yet be stated, as the cause of the disease has not yet been determined. Mr. Ehrhardt cannot positively say that he was the first man to have observed the disease and Mr. J. J. Reibenstein, Catspring, in Austin County, observed the disease in 1902. In this year, Mr. Reibenstein lost 18 head of cattle from this disease and in the following year he lost 16 head from it. It will be noticed that this was only four years after the Spanish-American War, and if the above statements are correct, the disease would have spread from the northeastern part of Harris County westward across Waller County to Austin County, a distance of about 80 miles. The signif-

icance of this fact cannot be estimated until the cause of the disease and the manner as well as the rapidity with which it is spread are known. However, in searching for the cause, one must ever bear in mind that the manner and especially the rapidity with which it is spread bear some relation to the cause of the disease. Knowing the rapidity of spread and the distribution of a disease, one is justified in drawing some conclusions as to its probable cause, especially in the case of insect-borne diseases.

### EPIZOOTOLOGY

Although the disease has been prevalent in this territory for a long time it was not until about 1917 that it began to cause such serious losses as to endanger the cattle industry of that section. Some pastures soon became so "infected" that they were entirely abandoned, while adjoining pastures remained entirely free from the disease. Although not all pastures in this area were "infected," the disease did not remain localized, for some pastures are known to have become "infected" in recent years. The following case may serve as an example. In 1919 the Martin-Settegast Cattle Company observed the first case of this disease on their ranches. In 1920 the cases were more numerous, while in 1921 the losses were very heavy. Cattlemen agree, and the writer has found it to be the case, that animals are found down only in certain parts of a pasture, especially around and in close proximity to ponds. On the other hand one must not forget that these ponds are very numerous and one need not go far in order to find such a pond. This observation probably bears no direct relation to the disease, but one must keep an open mind and consider the water supply of the animals when looking into the nature of a disease as obscure as loin disease. Nevertheless, there are hundreds of acres in some pastures where not a single animal has ever been found down with the disease. The disease seems to be localized within certain pastures.

Here again one must consider the frequency with which animals graze over such apparently unaffected areas. One must not forget that the cause of the disease may enter the animal today, but the disease itself need not become manifest until several days or even a fortnight later. In the meantime the animal may have traveled many miles away from the place at which the cause of the disease was picked up. Since no accurate data have been kept by cattlemen in the affected area showing the exact distribution of the disease, contradictory reports are often obtained, thus making it indeed difficult to point out suspicious or dangerous pastures with any degree of certainty.

Cattlemen have also observed that when cattle are dying in one pasture and are then moved to another pasture they will stop dying either immediately or within 7 to 10 days thereafter, although they may later again begin to die in the pasture to which they have been moved. An interesting case of this kind was reported from Austin County by Dallas Hillbolt. He and his father each had 1000 acres of land separated only by a railroad with a fence on either side of the right-of-way. In 1917 the son had 300 head of cattle in his pasture and the father about 150 head in his. About the latter part of April the son's cattle began to die from loin disease and by July 10, 75 head had been lost. On this date both father and son decided to move their cattle to Brazoria County in order to escape further losses. Up to this time the father had not lost any cattle. The cattle were rounded up on said date and shipped to Brazoria County, where all cattle were released in a 48,000-acre pasture. Here the son's cattle stopped dying immediately while the father lost 15 head within the next two weeks, presumably from loin disease.

It is said that losses cease entirely as soon as cattle are moved to timbered pastures and that no losses are known to have ever occurred in timbered areas. This is said to be the case in Harris County and is staunchly maintained by all cattlemen there. Also in Austin County cattlemen contend that this is the case. Information gathered in that county illustrates this point very clearly. The southern part of this county is low and flat and almost entirely devoid of timber. Losses are quite heavy from this disease in the southern part of the county, but cease quite suddenly as soon as the northern hilly and timbered part is reached. It is quite true, however, that in the northern part of Austin County we have a more elevated country combined with timber, while in Harris County we have a low flat country combined with timber in certain parts.

Cattlemen also contend that working the cattle, that is, rounding them up as is done in dipping or branding or shifting them from one pasture to another, has a tendency to check heavy losses. Losses are also said to be heaviest soon after a heavy rain and during showery weather, while a long dry spell has a tendency to check losses considerably. In this connection it is interesting to note that as the disease begins to make its appearance in late April or early May, the rainfall of the affected area begins to increase noticeably as is shown by the observed average monthly precipitation during the last 25 years. This increased precipitation will continue till about the end of August, when it begins to decrease perceptibly. Comparing this with the incidence of the disease one finds that the disease makes its appearance in the latter part of



April or the early part of May and continues unabatedly until the early part of August, after which time only sporadic cases occur during the next three or four months, and then cease entirely. It is apparent, therefore, that the prevalence of the disease coincides with the season of greatest rainfall and also with the season when the vegetation is at its best. However, one must also consider that the environment must be favorable in order that some diseases may develop and flourish, and that dry, hot weather lasting only for a fortnight or so may indeed noticeably check the occurrence of some diseases.

The age of the animal plays an important role. Of all the animals observed the writer has never seen one affected that was less than 18 months old, with one exception where the owner said the animal was a yearling. Likewise, the disease has never been observed in dairy cattle. These two facts, coupled with the fact that cattle, moved into the affected area from the outside, do not contract the disease till they have been in the affected area for a year or longer, seem to have a very important bearing upon the cause of the disease. It will be a most important task to clear up the possible relation that these observations may bear to the cause of the disease. The most plausible explanation would be that there is something lacking in the vegetation that the animal needs, but the fact that affected animals are usually in prime condition seems to lend weight to the contrary. An animal in continued good health probably secures in the feed it consumes and may ever have stored in the body tissues all elements necessary to maintain this condition. Now, should this normal supply of necessary elements gradually or even suddenly cease, one would expect the animal to manifest this condition by a gradual change in its health and physical condition. In loin disease, however, this is not the case. On the contrary, the animal is apparently in good health and condition and then goes down suddenly, almost without warning. The animal may appear to be in perfect health at night and the next morning may be found down and unable to get up.

The sex of the animal, on the other hand, is immaterial. One will find bulls, steers, cows with calf at foot, pregnant cows, and heifers suffering from the disease. The majority of the animals are mature and range in age from 4 to 6 years. The territory in question is essentially a cow country and for that reason one will find that the greater number of animals dying from loin disease are cows. The affected animals are, as a rule, in the pink of condition, robust and fat. Only seldom will a thin animal be found suffering from the disease.

Some cattlemen report losses as high as 15 per cent dur-

ing some years while others estimate their losses at a much lower figure. The losses may vary considerably from year to year and from pasture to pasture.

### SYMPTOMS

When an animal is observed in the early stages of the disease and before it goes down, one will notice that the animal only reluctantly follows the herd and frequently lies down to rest. The walk of such an animal is difficult to describe, varying but little from normal. A person not familiar with the habits and disposition of such an animal would not notice anything wrong at all. Again the observer may fancy that he can see a change in the animal's habits and disposition, but upon taking a second look he is no longer so confident and may even conclude he was wrong. But soon the condition becomes more pronounced; the animal does not walk quite as actively as when normal; it appears a little stiff; and the back may be slightly arched. An incoordination of movements seems to be developing. The cowman refers to this condition as "not trailing well behind." The animal experiences ever-increasing difficulty in getting up after having lain down to rest. Within a very short time, as early as 12 hours and seldom later than 24 hours after these symptoms have developed, the animal will be found down and unable to get up. When assisted up at first, that is, at a time when the animal is just at the stage of being unable to rise unassisted, it may still be

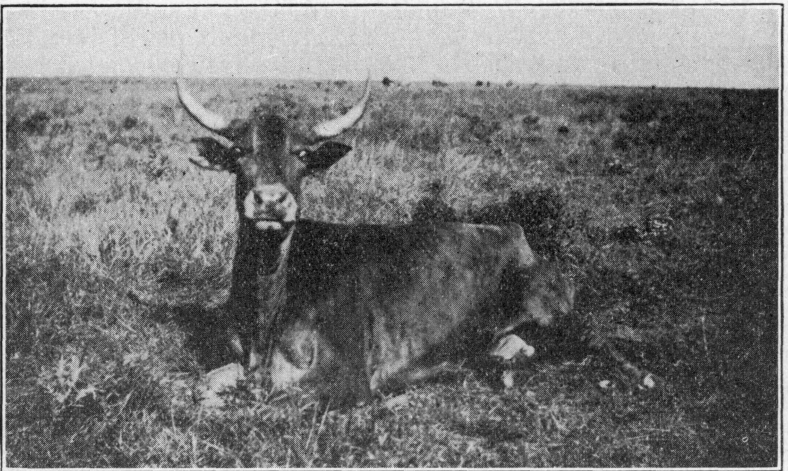


Figure 1. Animal suffering from loin disease just found on the prairie.

able to walk a mile or so, but eventually the animal will go down again. One will notice that when the animal is down it will not switch its tail although flies may be tormenting it at the time. The tail has become paretic. When an animal in this stage of the disease is approached on the range, it will make strenuous efforts to get up, but once down all efforts to rise again are useless.

The animal otherwise makes the impression of being perfectly healthy. The eyes are bright, the pulse, respiration and body temperature are normal, and the animal rests on its sternum. The feces are normal in color and consistency, but they are frequently covered with more or less clear mucus or may even be bloodstained. When the animal is examined as to the condition of the muscular functions, one will observe that the tail is paretic. When the tail is pricked with a pin the animal will not move it away or switch it, but one can observe the animal making strenuous efforts to do so; however, it can only raise the end of the tail an inch or so off the ground. Cattlemen riding the prairie and seeing an animal lying down never approach it when they see it switch its tail, for they know that such an animal is not suffering from loin disease. When pricked along the rump, back, or sides the animal will instantly react with contractions of the muscles of this region and make an effort to rise. The animal at this stage will eat and drink. One may even observe such animals crawling round in a circle on their knees to graze, dragging their hind legs after them.

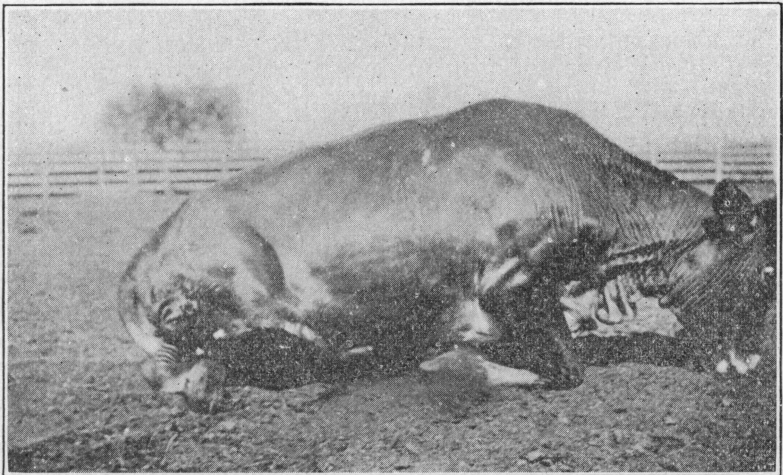


Figure 2. Animal making an effort to rise after it had been down with loin disease for ten hours.

The rapidity with which further symptoms develop depends somewhat upon weather conditions and the care given the animal. If the animal be turned over frequently it may remain bright and rest normally on its sternum for some days following. Soon, however, within 24 to 48 hours, one will observe that the muscles of the neck also become affected; the head will now be found resting against the side of the animal presenting a typical "milk fever position." If the head be raised



Figure 3. The "milk fever position" in three-year-old bull suffering from loin disease. Animal down for about 42 hours.

by an assistant into its normal straight position and then released, it will immediately drop back against the side of the animal again. If the head be turned too far by the assistant before being released, and become overbalanced, the animal will not be able, despite all efforts, to balance it again, but it will now fall flat on its side with outstretched head and legs to remain there. Frequently one will observe, when the animal is resting in a milk fever position, if the horns of the animal be grasped and its head raised into its normal resting position, that the neck of such an animal shows a sharp crook about at the point of the third and fourth cervical vertebrae. This can sometimes also be observed when an animal is found which is still barely able to handle its head. One may notice in such a case that the animal, if it raises its head at all, will do so slowly and in the normal manner until it reaches a point where it apparently can go no farther and the neck shows this sharp crook. The animal will now be seen to slowly raise the point of the nose until it is almost on a level with the poll, and now, with a sudden snap, the crook disappears and the

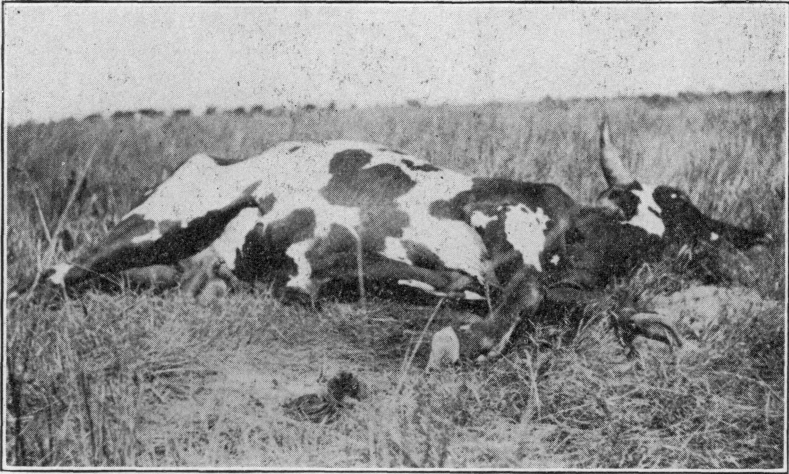


Figure 4. The same animal as above photographed a few minutes later after the animal had been pushed over on its side.

neck becomes straightened out. This movement is apparently always accomplished with difficulty. As time goes on, however, the animal will no longer be able to raise its head and will, therefore, when it begins to struggle, come to lie on its side to remain there. Although the animal goes flat there is no paralysis of the legs. In a few cases hyperesthesia of the skin was observed.

Once the animal is flat, the progress of the disease will depend to some extent upon the attention given the animal. Food, as a rule, will no longer be taken voluntarily for at least a number of days, but water will be taken when the animal is raised on to its sternum,—if not voluntarily, it will at least be swallowed when poured into the animal's mouth as in drenching. Rumination ceases when the muscles of the neck become affected and the animal assumes the milk fever position, or has gone flat. Some animals may retain their normal resting position upon the sternum for as long a period as two weeks, taking food and water during that time, but eventually they will all go flat. If the animal be well bedded, frequently turned over and watered regularly it may begin to eat again in a week or ten days, and once such an animal begins to eat again its chances for recovery are good indeed. Should such an animal recover, which may happen after the animal has been down for several weeks, one will find that it suddenly gets up and walks away. Such cases of recovery are rare in-

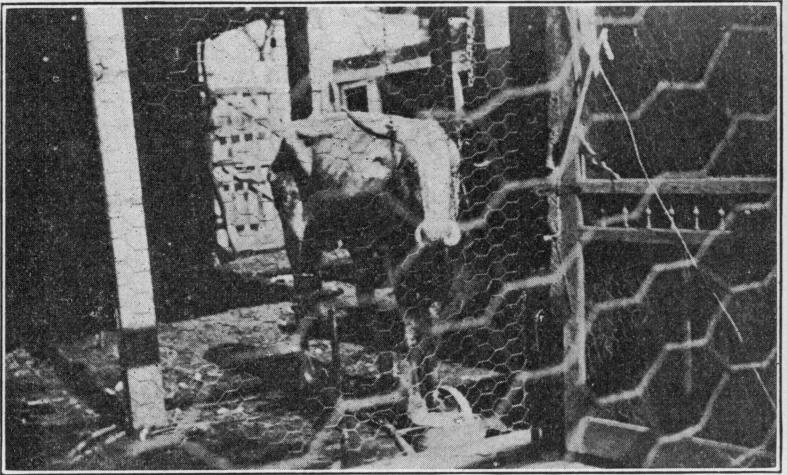


Figure 5. This cow had been down for three weeks with loin disease. She suddenly got up one morning and immediately charged the writer, who happened to be standing near. This photograph was taken a few minutes after the cow got up.

deed. Animals recovering from this disease are not permanently immune, for they may have another attack as early as the following year and then usually die from it.

On the other hand, if the animal be found down on the prairie and left there exposed to the hot sun without food or water, the progress of the disease will be very rapid. In this case the animal may be flat after being down for only 12 hours and be dead at the end of 36 hours after going down. No cases are on record where the animal dies suddenly without first going down. It will thus be seen from the above that the disease may run a slow course, which may be described as chronic, or the disease may terminate fatally more rapidly and one may then speak of it as taking an acute course.

### PATHOLOGICAL ANATOMY

If one should sacrifice an animal suffering from loin disease after it has become moribund and all hope for recovery has disappeared and examine the body carefully for lesions produced by the toxin, one would find a variable picture presenting itself. The autopsy lesions here reported have all been taken from such animals. It may be well to mention here that no pathognomonic lesion will be found, that is, a lesion to which one can point and confidently say that if that lesion is present one is dealing with loin disease.

Upon removing the skin from the carcass one will probably find bruised places on different parts of the body caused by the animal's lying and struggling on the ground. Frequently one will find no lesions at all; all organs and the meat in such cases have the appearance of a perfectly healthy and well nourished animal like one regularly slaughtered for the consumption of its meat. At other times lesions will be found in some of the organs, but these lesions vary greatly in character and extent. Let them be described in some systematic manner:

The lungs and trachea are nearly always normal; only seldom will petechial hemorrhages be found. In the heart, lesions are encountered more frequently. In about 25 per cent of all cases hemorrhages will be found on the endocardium of the left ventricle. Very rarely are such hemorrhages present on the endocardium of the auricles or the right ventricle. These hemorrhages vary in size from a pin point to very extensive patches, thus sometimes covering three-fourths of the surface of the left ventricle. In color they vary from a pale red to a deep red. When such hemorrhages are small and few in number, they may be present on the papillary muscles only. The myocardium, epicardium, and pericardium have been found normal in all cases examined.

The liver is usually normal in color and consistency. Only occasionally will this organ show a yellowish tinge or be even pronouncedly yellow. In only four per cent of all cases examined so far were hemorrhages about the size of a pin head found under Glisson's capsule. Now and then one will find liver flukes in the bile ducts. The gallbladder, as a rule, is well filled with bile and is frequently slightly, or even much enlarged. The bile itself is clear and of a greenish color. The mucous membrane of the gallbladder is sometimes found congested and in about 15 per cent of the cases hemorrhages will appear thereon. These hemorrhages vary in size from a pin point to that of a pea, are irregular in outline and usually very numerous.

The spleen was always found normal in size, color, and consistency with the veins of the capsule well filled and standing out prominently.

The kidneys show lesions more frequently than any other organ, but even here the lesions are not consistently found and vary in character and extent. In about 30 per cent of the cases a congestion of the kidneys is present which is especially well marked in the medullary portion. Upon close scrutiny one will find in about 40 per cent of the cases, small pin point

red dots in the cortex. These are the congested glomeruli of the kidneys. This congestion of the glomeruli does not always occur simultaneously with the congestion of the medullary portion. Only rarely will one find hemorrhages.

The urinary bladder may be empty or contain some urine. Lesions on the mucous membrane of the urinary bladder have never been observed by the writer.

The writer has never found lesions on the first three compartments of the stomach. In about 40 per cent of the cases a parasite, *Paramphistomum cervi*, was found. The fourth stomach has sometimes been found to contain small pieces of bone. The mucosa of the abomason may be diffusely reddened and congested. Occasionally one will find ulcer-like areas or old scars on the mucosa. In about 10 per cent of the cases one will find distributed over the mucous membrane a few circumscribed areas closely studded with sharply defined deep red petechia varying in size from a pin point to that of a pin head.

Bile will be found in the small intestines in about 50 per cent of the cases. The amount of bile varies; more frequently one will find it in considerable quantities mixed with excessive amounts of mucus and some food material. The mucous membrane is often congested. The congested areas may be small and circumscribed or diffuse and extensive. The color of these areas varies from a deep red to a pale red.

On the mucous membrane of the large intestines one may find more or less circumscribed or diffuse reddened areas and streaks, either on the ileo-caecal valve, the caecum, or the colon. The ingesta are sometimes mixed with mucus. No lesions are found on the mucous membrane of the rectum. The rectal contents are more or less covered with clear mucus.

The lymph glands do not show any constant and characteristic lesions. The precrucial, prescapular, portal and some groups of the mesenteric lymph glands may be found slightly congested around their periphery, but these four groups need not be simultaneously affected. Occasionally one or the other lymph gland of the body may be found more or less congested or even hemorrhagic, especially a small lymph gland located in the small curvature of the abomason.

In a few cases one can find relatively large hemorrhagic spots on the omentum or the peritoneum, varying in color from a pale red to a bright red.

The meninges of the brain may be found congested, but other lesions of the brain have not been observed. The spinal cord has always been found normal macroscopically. In a few cases the spinal fluid was found either milky or tinged with blood.



## EFFORTS TO ESTABLISH THE ETIOLOGY OF LOIN DISEASE

The first symptoms of loin disease appear with but very little warning; it strikes suddenly. In this respect it resembles an intoxication. Such an intoxication would naturally have to come about by the toxic material entering the body from the outside and in such quantities as to produce an impairment of the health of the animal. And since the toxic material would have to be introduced from the outside, one will have to search for it either in the food, in the water, or in both.

With the first possibility in mind the range has been searched for possible poisonous plants, but with the exception of the coffee bean, which is not eaten by cattle, none were found. Nor did the epizootology of the disease suggest such a plant. If it were a poisonous plant, why do cattle under 18 months of age not die from the disease? They have at this time passed at least one entire grazing season on the affected area without having contracted the disease. Surely there was opportunity to take up such a poisonous plant during this time. There is no record, so far as the writer knows, of a plant which is poisonous to animals of a certain age only. Why do dairy cattle not contract it while running on the affected range? And why do cattle, brought into the affected area from the outside, not develop the disease until they have passed at least a year therein? Such facts would not suggest a poisonous plant.

However, one must not limit his investigation to known poisonous plants, plants readily detectable by the naked eye, but one must go beyond that and look into the nature of the microscopic plant life encountered on the range. The writer has in mind here especially that microscopic plant life generally classed as bacteria. Here several possibilities present themselves. In the first place one must consider the probability of its being an infectious disease. Here again one must consider two possibilities. On the one hand a general infection may take place, in which case the microorganisms must be found in the tissues of the animal in considerable numbers. This the writer was unable to do. On the other hand the microorganisms could gain access to the animal body through small wounds or abrasions, remain localized at the point of entrance, but disseminate their toxin throughout the entire animal body much as we see in tetanus. In such a case it would be extremely difficult to locate the point of infection and therewith the microorganisms unless there were some lesions produced at the point of entrance. Such lesions, however, could not be found.

TABLE 1

Lesions Found in Cattle Suffering from Loin Disease and Slaughtered in a Moribund Condition.

No.	Lymph Glands	Heart	Lung	Liver	Gall Bladder	Spleen	Kidney	4th Stomach	Small Intestines	Large Intestines	Remarks
R. H.					Large		Congested		Bile Present		
7	Precrural Prescapular Mesenteric congested				Large				Small congested areas. Bile present		
14	Mesenteric congested	Hemorrhages between valve folds			Very large		Pinpoint red spots in cortex		Bile present congested		
20	Precrural Prescapular Mesenteric congested	Hemorrhages in l. ventricle and auricle	Petechial Hemorrhages; also in trachea at bifurcation		Large		Hemorrhages. Congested; Two infarcts	Congested	Small congested areas	Small congested areas	
21	Precrural congested	Hemorrhages at origin of aorta			Enlarged; Many petechia on mucosa						Liver flukes present; Hemorrhages on omentum
24					Large						
25	Precrural Mesenteric congested			Pinhead hemorrhages under capsule Portal lygl. congested	Large; hemorrhages on mucosa			Not opened	Not opened	Not opened	
26							Pin point red spots in cortex		Congested areas	Congested areas	Paramphistomum found; Gulf Coast Tick found
27					Petechia on mucosa; much bile present		Pinpoint red spots in cor- tex; congested		Much bile present		Many paramphistoma found
28					Very large		Pinpoint red spots in cor- tex; congested				One paramphistomum cervi found
31											One tapeworm found. Hyperestheis of skin
32								Ulcer-like areas	Small congested areas in jejunum	Small congested areas in caecum	Paramphistomum cervi found
33					Large; mm. congested slightly			Old ulcers	Not opened	Not opened	

TABLE 1. (Continued)

No.	Lymph Glands	Heart	Lung	Liver	Gall Bladder	Spleen	Kidney	4th Stomach	Small Intestines	Large Intestines	Remarks
34	Precrural hemorrhages	Extensive hemorrhages in left ventricle		Yellowish tinge			Pinpoint red spots in cortex; congested	Many petechia	Small congested areas; some bile	Many heavily congested areas and streaks	Large subperitoneal hemorrhage on pancreas; paramphistoma
35								Old ulcers	Much bile present	Extensive heavily congested areas	Many paramphistoma. Hyperesthesia of skin
36					Large				Not opened	Not opened	
37	Superior gastric congested				Much enlarged		Congested			Heavily congested areas and streaks	Paramphistoma found
38					Many small and large hemorrhages; large		Pinpoint red spots in cortex	Many pinpoint hemorrhages	Ilio-caecal valve congested; some bile		117 Paramphistoma
39									Congested	Congested	11 Paramphistoma
43		Large hemorrhagic areas in left ventricle					Congested	Diffusely red	Ilio-caecal valve diffusely reddened		20 Paramphistoma 2 Gulf Coast Ticks
50				Pronouncedly yellow			Congested		Yellow stained mucus		Many paramphistoma Bright red hemorrhages on omentum
51	Superior gastric hemorrhagic	Hemorrhages in left ventricle					Pinpoint red spots in cortex		Some bile present		Paramphistoma found
55		Hemorrhages in left ventricle			Hemorrhages on mucosa		Pinpoint red spots in cortex		Not opened	Not opened	Spinal fluid milky in lumbar region
56					Enlarged		Pinpoint red spots in cortex	Petechiated	Congested areas; Much bile		
57							Pinpoint red spots in cortex				No exhaustive post mortem made. Spinal fluid bloody in lumbar region
58	Mesenteric marked congestion				Mucous membrane congested		Pinpoint red spots in cortex congested		Much bile present		

But there is still another possibility to be considered. It is possible that the microorganisms do not enter the animal body at all, but multiply outside of the animal body, produce and liberate their toxin there, and this in turn gains access to the animal body. We can here cite a parallel case, that of botulism, occurring widely distributed over the world. The microorganism must then have to be looked for in the food consumed by the animal or in the water taken up. Failing in every other direction, the writer turned his attention especially to the latter possibility.

### LOIN DISEASE PROBABLY NOT DUE TO BACTERIAL INFECTION

In the early part of the investigation, when material was hard to obtain, the possibility that the disease was of an infectious nature was especially considered, it being constantly kept in mind that whatever organism might be encountered, it must be possible to reproduce the disease with it in all its details. A diligent search was, therefore, made for microorganisms in the blood, heart, liver, spleen, lymph glands, spinal cord and brain, both in smears made from these organs and in cultures. Laboratory animals and cattle were also inoculated, especially with blood and serous fluid from the pericardial sac. The results were always negative. In May, 1921, the writer had an opportunity to autopsy an animal in the field that had been down for ten days. Blood was drawn from the jugular vein of this animal and the animal then destroyed. Smears were also made from the heart, liver, spleen, and kidneys. Fifty cubic centimeters of the blood drawn from the jugular vein of this animal were injected subcutaneously on the following day into a cow with negative results. Unfortunately no specimens of the organs for culture purposes were secured. When the smears were examined numerous clusters of bacteria were found in all of them. Since the animal from which these smears were made had been down for 10 days, it was thought worth while to consider the possibility that the organisms responsible for the disease did not develop in the animal body in such numbers as to be readily detectable until the animal had been down for some time. Such animals, however, could not be secured.

During the severe outbreak of 1921 it was possible to make a thorough study of the disease. A large number of animals were available for autopsy and a thorough survey of the epizootology could be made. The latter point has already been discussed in the previous pages of this report. Unfor-

fortunately, only two cows were available for inoculation during this outbreak. These were kept under observation for two months in a small enclosure, during which time they were, at different times, inoculated with blood, spinal fluid, saline extracts from the spleen, kidney, liver, and spinal cord and also drenched with the contents of the small intestines of animals suffering from loin disease. The animals were finally returned to their respective owners, but nothing untoward happened to them during the next several months that they were kept under observation. At the same time numerous smears were made from the organs of sick animals and diligently searched for microorganisms, but none could be demonstrated that could be held responsible for the disease. Numerous cultures, both aerobic and anaerobic, were made from the organs of animals autopsied within a day or two after they were found down, using such media as plain nutrient bouillon, plain nutrient agar, brain media (von Hibler), liver media, and a combination of the latter two. Plates and tubes were also incubated in anaerobic jars and still the results were negative with one possible exception. In this case, No. 32, a small organism was obtained on plates that was very pathogenic for mice, guinea pigs, and rabbits. Its pathogenicity was then tested for cattle. For this purpose a twenty-four-hour bouillon culture was prepared and with 50cc of this culture a four-year-old cow was injected intravenously at 1:30 p. m., on November 9, 1921. Let us call this cow CS1A. The following facts were recorded at the time:

At 8:00 a. m., 11-10-21, cow very weak and down. The cow made four attempts to get up before she succeeded in regaining her feet. Breathing labored and accompanied by a light roaring noise. Number of respirations per minute 18; body temperature 97.2 F.; watery diarrhea.

At 1:00 p. m., same day, cow lying down and regained her feet upon the second attempt to rise. At 5:00 p. m., cow lying down again; this time in a different place.

November 11, 1921, 8:00 a. m.: The animal evidently had been up during the night, for on this morning she was down in a different part of the lot from where she was last seen the previous night. Cow unable to rise. Number of respirations per minute 18; body temperature 98.6 F. The head was held in its natural position, but a slight crook appeared in the neck. Animal bright and alert; switches its tail. Some well-formed feces of a normal color had been passed during the night. At 11:00 a. m., same day, animal still down resting its head against the side of the body as in milk fever. When the head is lifted into its normal position, the animal will hold it there

for a short time and then allow it to drop back against the side of the body. The alertness of the early morning has disappeared and the previously naturally nervous animal no longer takes notice of its surroundings. At 1:00 p. m., on the same day, animal flat, but still able to sit up when assisted upon its sternum. At 5:00 p. m. the animal flat, and can still sit up when assisted. When the head is lifted into its normal resting position, the animal resting on the sternum, a decided crook appears in the neck and when the head is released it mechanically drops back against the side of the body.

November 12, 1921, 8:00 a. m.: Animal flat, when assisted on to the sternum it drops its head against the side of the body to remain there a few minutes, after which time the animal goes flat again. Body temperature 97.6 F. The animal died at 10:00 a. m. on the same day.

Autopsy findings: The animal was autopsied at 1:00 p. m., 11-12-21. Bruises under the skin at the sternum and on the sides of the body. Prescapular and precrucial lymph glands normal. Peritoneum, no lesions.

Spleen, not enlarged; veins of capsule, injected; some circumscribed and also some quite extensive, pale, washy hemorrhages on capsule. On section, pulp firm; trabeculae and Malpighian bodies plainly visible; color normal.

Liver, firm; normal in color; no hemorrhages or other lesions. Gallbladder contained 640 cc bile of a greenish color. Mucous membrane of gallbladder greenish in color; no hemorrhages.

Kidneys, firm; greyish in color (cloudy swelling); hyperemia; no hemorrhages. Urinary bladder, empty. Its mucosa showed hemorrhages the size of a pin point to that of a pin head.

The mucous membrane of the abomason showed sharply defined hemorrhages from the size of a pin head to four times that large.

The contents of the small intestines, mixed with bile. Mucous membrane, more or less hyperemic about one-half the length of the small intestines. The aggregated follicles, not enlarged.

Large intestines: Ileo-caecal valve, hyperemic and swollen. In the caecum, hyperemic streaks and folds near ileo-caecal valve and apex. Lymph glands in abdominal cavity, normal.

Lungs: Extensive pneumonia present. Mucosa of trachea, slightly hyperemic.

The pericardial sac contained a large amount of reddish fluid. Epicardium, covered with fibrin and some hemorrhages. Myocardium, firm and pale (cloudy swelling). Endocardium, a few hemorrhages about the size of a dime.

Microscopical examinations of smears made from the different organs showed numerous microorganisms similar to the one injected into the animal. The same organism was recovered in pure culture from the animal.

To further test this organism, a twenty-four-hour bouillon culture was prepared and 35 cc of this culture injected into the jugular vein of an eighteen-months-old heifer, CS1B. This heifer showed some depression during the next several days but eventually recovered without ever showing symptoms resembling those observed in loin disease.

On 11-25-21 at 5:00 p. m., an aged cow, CS2, was inoculated intravenously with 75 cc of a thirty-hour bouillon culture of the organism recovered from the heart of CS1A. At 8:00 a. m., 11-26-21, this cow was down and could not be induced to get up. Body temperature 97.2 F.; number of respirations, 72 per minute. Feces, normal in consistency and streaked with blood. At 11:00 a. m., the cow was up and walking around in the lot. At 5:30 p. m., on the same day, the cow was lying down again and could not be induced to get up. On the following day, at 8:00 a. m., the cow was found dead.

Autopsy findings: Bloody discharge from the nostrils. Numerous large hemorrhages under the skin in the subcutaneous tissue. Prescapular and precrural lymph glands, normal. Numerous hemorrhages on peritoneum, on both the parietal and visceral layer, especially on all four stomachs. Lymph glands in peritoneal cavity, normal.

On mucous membrane of fourth stomach, numerous hemorrhages, the majority about the size of a bean and consisting of a circular ring-shaped hemorrhagic line about 2 mm wide with a non-hemorrhagic center.

Mucous membrane of small intestines, hyperemic; no hemorrhages present. No bile in small intestines.

Mucous membrane of the large intestines, especially that of the colon, hyperemic.

Liver, greyish in color, friable (cloudy swelling). Gallbladder, normal in size; no hemorrhages on mucosa.

Spleen, apparently normal.

Kidneys, greyish in color; friable (cloudy swelling). No hemorrhages present. A few small hemorrhages on mucosa of urinary bladder. Urine, straw colored.

Parietal pleura, covered with numerous large hemorrhages. Extensive pneumonia in lungs.

Many hemorrhages on epicardium; myocardium, greyish in color, friable (cloudy swelling). Endocardium of left ventricle almost entirely covered with one large hemorrhagic area. No hemorrhages on endocardium of other heart cavities.

No lesions were found in the muscles of the legs and along the back, but the loose connective tissue accompanying the sciatic nerves showed profuse hemorrhages.

The organism injected into the animal was recovered from the heart, liver, spleen and lungs in pure culture. No cultures were made from the kidneys.

Although the clinical symptoms and lesions observed in cow CS1A closely resembled those found in cattle suffering from loin disease, the symptoms and lesions observed in cow CS2 were so different as to preclude the possibility that the organism injected into these animals was the causative organism of loin disease, and for this reason no further experiments were undertaken with it.

Thinking that the negative results thus far obtained might be due to the media—plain agar, glucose agar, liver agar, blood agar—used for purifying the cultures made in the field or inoculated in the laboratory from specimens brought in from the field, not being suitable for the growth of the causative organism, a mass culture was made by inoculating liver bouillon from original brain media cultures made from the spleen and either the heart or kidney of six different animals. This mass culture was incubated for three days, the fluid portion then strained through a cloth and a two-and-one-half year old cow and a two-year-old goat drenched with it. The cow received 1000 cc and the goat 300 cc of this fluid. No symptoms developed in either of these animals. Rabbits and guinea pigs were also inoculated subcutaneously with culture fluid obtained by straining the fluid from brain media cultures inoculated with tissue from the organs of animals suffering with loin disease. No attempt was made to first purify these cultures for fear that the organism might get lost in the attempt. The rabbits and guinea pigs thus inoculated never became sick during the next two weeks following the inoculation.



Experiments were also undertaken in which the intestinal and rumen content of animals suffering with loin disease were used in an attempt to find the cause. The content of the rumen of case No. 34 were carried to the laboratory, the fluid portion strained off and with this a healthy cow was drenched. No symptoms developed in this cow. No animal was available to which the remaining solid content could be fed.

In another experiment the small and large intestines were removed from case No. 28 and the content, with the exception of that of the rectum, placed in a bucket. The intestines were then flushed with water and the flushings combined with the intestinal content. This material was then used to drench a healthy cow. No symptoms developed.

#### LOIN DISEASE NOT IDENTICAL WITH BOTULISM

The possibility that loin disease was related to botulism was also considered and, therefore, some animals down with the disease were injected with varying amounts of polyvalent botulinus anti-toxin. Thus one animal that was found down in the pasture was immediately given 80 cc polyvalent botulinus anti-toxin, intravenously. No improvement was observed and 36 hours later the animal was given another injection of 100 cc botulinus anti-toxin. The animal died some six hours after the last injection.

Another animal was given 80 cc polyvalent botulinus anti-toxin in the early stages of the disease and before it was down. The animal was cast for this operation and when released could not get up again. On the following day this animal was given another 80 cc polyvalent botulinus anti-toxin, intravenously; on the third day another 80 cc, and finally, on the fourth day, another intravenous injection of 100 cc polyvalent botulinus anti-toxin. The animal died on the fifth day.

A third animal was found down in the pen in the morning where it had been placed the previous night, apparently in good health. At 4:30 p. m., on the following day, the animal was given an intravenous injection of 200 cc (500 units) polyvalent botulinus anti-toxin. No beneficial effect could be observed from the injection. The animal gradually grew worse and was finally destroyed for autopsy four days later.

#### LOIN DISEASE NOT DUE TO TRYPANOSOMES

Although the symptoms of loin disease do not resemble those usually observed as due to trypanosomes, it was

considered worth while to test this theory. Trypanosomes could never be demonstrated in smears from the organs of animals suffering with loin disease, and cattle given injections of large quantities of blood from such animals always failed to show any reactions following the injections. Neither did any of the numerous rabbits given injections of blood and extracts of organs from animals suffering with loin disease show any suspicious symptoms, or trypanosomes in their blood. Guinea pigs were given intraabdominally, injections of 10 cc of blood freshly drawn from animals suffering with loin disease. The blood of these guinea pigs was later examined for trypanosomes. These parasites could never be demonstrated and all guinea pigs lived and thrived.

### CONSUMPTION OF TOXIC CARCASS MATERIAL RESPONSIBLE FOR LOIN DISEASE

Failing entirely to get any tangible results from all theories considered thus far, the writer turned his attention to the carcasses left on the prairie. It was observed that many animals in the affected area had a habit of chewing bones. The mere fact that one occasionally saw an animal chewing a bone did not, at first, arouse suspicion that the trouble might be found there. Closer attention was, therefore, paid to the bone-craving habit of these animals and it was observed that the animals did not only consume sweet bones, as was at first thought they did, but that they would even consume putrid bones. Thus it was found that the bones of animals autopsied on the prairie would soon be consumed by other cattle running in the pasture. It was, therefore, decided to feed these bones to cattle experimentally and observe results. To obtain such putrid bones from the prairie, it was found necessary to place them in some kind of an enclosure in order to keep the cattle away from them.

Preliminary experiments were started in 1921. A small quantity of bones was at first obtained and ground up. One-half pound of such bone was fed to a cow, but no symptoms developed. In October, 1921, bones were obtained from an animal that had died of loin disease on August 4, 1921. When these bones were collected a considerable amount of dried and foul smelling meat was still adhering to them. The bones were ground and with several grams of them one liter of brain media was inoculated and incubated for three days. The liquid portion of the culture was then strained off and a two-and-one-half year old steer drenched with it. No symptoms developed in this animal.

## FEEDING EXPERIMENTS WITH PUTRID BONES

Feeding ground bone to cattle in larger quantities was then undertaken. For this purpose, more bones were collected from animals that had died from loin disease, the carcasses of which had been left on the prairie to rot. When the experiment was begun a supply of 70 pounds of bone was on hand. Two three-year-old steers were used in the experiment which was begun on April 24, 1923. Thirty-five pounds of bone was allotted to each animal. The ground bone was fed mixed with wheat bran and corn chops, as otherwise the animals would not eat it. Small quantities were given at first. By May 1, each animal was receiving and consuming regularly two pounds of ground bone daily. On May 13 the available supply of bone allotted to steer CS8 was exhausted. He ate it well on that day, but when on the following day he was offered two pounds of ground bone allotted to steer CS9 (these bones were from a different carcass) he refused to eat it. Previous to this, on the morning of May 12, the attendant noticed that steer CS8 was not walking as usual, but when on the same day the writer looked at the animal the peculiar walk could not be detected. When the attendant was again questioned about the matter he said, "The steer did not act like he used to." The steer was kept in the same lot for further observation and on May 16, at 1:00 p. m., the attendant observed from a distance of about 100 yards that the steer, lying down and resting apparently normally, suddenly belowed and struggled to get up. Once on his feet, he made a run of about 15 yards and then dropped to the ground again. Here he made frequent attempts to get up without succeeding. The writer saw the animal an hour later. It made the impression of a perfectly healthy animal resting normally on its sternum, but it could not get up. At 6:00 p. m., the animal's condition was as follows: body temperature, 102.0 F.; respiration and pulse, normal; the animal could not get up and was resting on its sternum.

May 17, 1923, 7:00 a. m.: Animal, flat. When assisted on to its sternum it remained there. Body temperature, 100.4 F.; respiration and pulse, normal. At 11:00 a. m., the animal was visited again and was again flat; at 1:00 p. m. the animal was still flat and was now assisted on to its sternum. Water was offered to the animal, but it did not drink. The animal was then offered some hay, of which it ate a little. The animal's bowels had acted and the feces were normal in color and consistency. At 5:00 p. m., the animal was again flat. When assisted on to its sternum a crook was noticed in the neck. The animal was offered some water and it drank

two gallons from a bucket. The animal could not switch its tail.

May 18, 1923, 8:00 a. m.: Animal, flat. Assisted on to its sternum the crook in the neck appeared more pronounced than on the previous night, but it could still hold its head up. The animal was observed to ruminate. Body temperature, 101.2 F. Feces had been passed during the night and these were covered with much mucus and a few streaks of blood. Up to this time the animal, when assisted on to its sternum, could, at all times, hold its head up in its natural position. On this morning, however, when the animal was to be turned

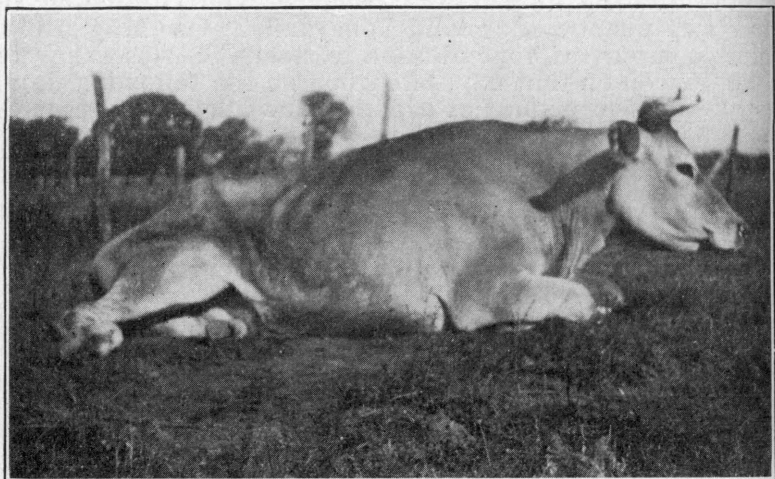


Figure 6. Steer CS8 fed ground bone from prairie. This photograph was made after the animal had been down twenty-four hours.

over on its other side and a rope placed around its horns for this purpose, it began to struggle and while doing so suddenly dropped its head against its side where it remained in a typical milk fever position. It could be plainly observed that the animal was making strong efforts to raise its head again, but could not do so. When the head was now raised by an assistant and released when in its normal position, it would either drop back against the side of the body again, or it would turn in the opposite direction and the animal would go flat. When the latter occurred the head would strike the ground with the nose first. The condition of the animal did not improve during the day.

May 19, 1923, 8:00 a. m.: Animal flat and in same condition as on previous night. Water and food had not been

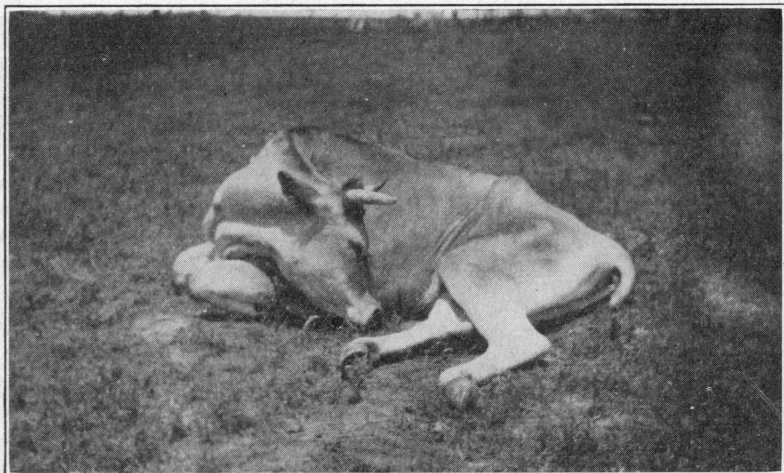


Figure 7. Steer CSS fed ground bone from the prairie. This photograph shows the milk fever position assumed by the animal after it had been down forty-eight hours.

taken for twenty-four hours. Body temperature, 101.8 F.; respiration, 18 per minute. Feces had been passed during the night. These were well-formed, like those of a horse, slightly bloodstained and covered with much clear mucus.

As it was feared that the animal would not live very much longer, it was destroyed for autopsy at 9:00 a. m., 5-19-23.

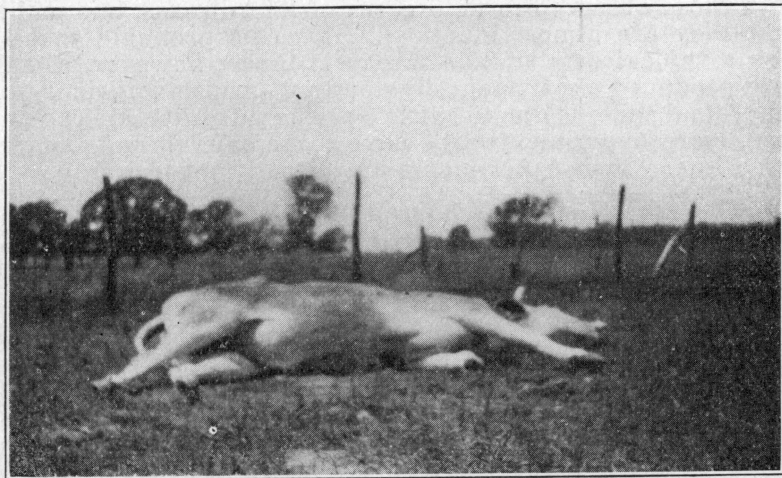


Figure 8. Steer CS3 fed ground bone from prairie. This photograph shows the animal flat forty-eight hours after it went down. Compare text.

Autopsy findings: No hemorrhages on peritoneum. Mucosa of pyloric end of abomason, slightly hyperemic.

Small intestines contained much bile and much sticky mucus on the mucous membrane; otherwise, they were empty. Mucosa slightly hyperemic in a few limited areas.

Large intestines, no lesions.

Few mesenteric lymph glands, slight peripheral hyperemia. Abdominal cavity contained about 50 to 75 cc clear yellowish fluid, which coagulated soon after exposure to air.

Liver: Left lobe atrophied to one-half normal size. On cut surface a distinct yellowish tinge. No other lesions.

Gallbladder contained 880 cc bile. Mucosa, normal.

Spleen, normal on cut surface. Capsule studded with hypertrophic areas. Blood vessels of capsule prominent.

Kidneys, hyperemic, especially in medullary portion. Cortex showed many pin point red dots; otherwise, kidneys apparently normal.

Heart: Endocardium of both auricles well marked and extensive hemorrhages; otherwise, normal in color and consistency.

Pleura, lungs and trachea, no lesions.

Comparing the symptoms and lesions found in steer CS8 with those observed in field cases of loin disease, one will see that they are almost identical. It seems probable that this was a typical case of loin disease. Since, however, a single case produced experimentally does not furnish sufficient proof that loin disease is due to cattle's eating putrid bone in the pasture, more experiments will have to be carried out along the same line. Such experiments are already projected.

The second steer, CS9, used in the above described experiment, consumed the 35 pounds of bone allotted to him, but at no time did he show symptoms of any kind, much less come down with loin disease.

From this experiment no definite conclusion can be drawn as to the period of incubation of loin disease. From the data obtained it can either be a minimum of 3 1-2 days or a maximum of 23 1-2 days.

While working on the bone theory an effort was made to determine at what time the toxin-producing bacteria enter the bone. Bones were, therefore, brought to the laboratory

from fresh field cases of loin disease, and cultures, both aerobic and anaerobic, were made from the bone marrow. In no case could microorganisms be demonstrated.

This would indicate that there is no organism present in the bones of animals suffering from loin disease at the time of their death. The bacteria producing the toxin responsible for loin disease, provided that the case produced in steer CS8 was a true case of loin disease, must, in that case, gain access to the bones after the animal has died. Probably the bacteria grow even more rapidly and abundantly in the flesh during decomposition than in the bones.

Another interesting problem is to determine whether the bacteria gain access to the bones and flesh from the soil after the animal has died, or whether the bacteria also inhabit the intestinal tract of the animal and invade the flesh and bones from that point after the animal dies, or whether both cases may happen. If the bacteria enter the flesh and bones from the soil only, then the possibility of spread of the disease would be very limited. On the other hand, if the bacteria may also inhabit the intestinal tract without producing their deadly toxin there during the life of the animal, but only after the animal has died from any cause whatever, then, in that case, the disease could be spread from place to place by the transportation of the animals harboring such bacteria in their intestinal tract and an outbreak of the disease may occur wherever such an animal dies and other cattle are wont to consume its putrid flesh and bones.

### RECOMMENDATION

If no other than the toxic cause of loin disease exists, the results obtained in the above experiment clearly indicate that the logical procedure to stop the losses from this disease would be to check the bone-craving habit of the animals on the range. Feeding the animals sweet bone meal in sufficient amounts to supply the deficiency necessary for the upkeep and normal development of the body should stop the disease in all cases. Scrupulously cleaning the range of all carcasses and carcass material, not only of cattle, but also of all other animals, should have a tendency to decidedly check, if not entirely prevent losses, depending upon the degree of thoroughness with which the cleaning is carried out.

Note: Upon a recent visit of Sir Arnold Theiler, Director of Veterinary Education and Research, Union of South Africa, to the Texas Agricultural Experiment Station, Sir Arnold saw some of the above data and expressed the belief that loin disease is identical with lamziekte, a disease very prevalent in South Africa. Not all data have been published (but see 3d and 4th Reports, Nov. 1915; 5th and 6th Reports, April 1918; and 7th and 8th Reports, April 1918, by the Director of Veterinary Research, Union of South Africa) on lamziekte, but Sir Arnold stated that the disease is contracted by the animals when eating putrid bone on the veld, and that he had been able to produce lamziekte by feeding the animals such bones, but in no other way. The bone-craving, in turn, is due to a lack of phosphorus in the soil and vegetation. Since lamziekte is contracted by eating bones, it only remained to stop the animals from doing this in order to prevent the disease. This was successfully accomplished by feeding the animals three ounces of sweet bone meal daily all the year round. In order to prove his contention, Sir Arnold administered this quantity of bone meal to each individual animal daily with the result that of a large number of animals thus fed, all stopped the habit of chewing bones in the pasture and none contracted the disease, while the control animals, which had not received the bone meal and were otherwise kept under like conditions, continued the habit of chewing bones and all developed the disease.

Not only did he stop the disease by this method, but he was also able to show that the animals receiving the bone meal for some time before they had reached maturity, made a much better growth and in the end developed into much better animals, weighing several hundred pounds more than the control animals which did not receive bone meal. Sir Arnold suggested that bone meal be mixed with stock salt at the rate of two pounds of bone meal to one pound of stock salt and placed in troughs in the pasture near watering places so that all animals may have easy access to them at all times. A sufficient number of troughs should be provided so as to prevent the stronger animals from hooking away the weaker ones.

While working on this trouble Sir Arnold also found that another disease of South Africa, known there as styfziekte, a disease probably identical with "creeps," could be prevented in the same way.

Considering, then, Sir Arnold's description of lamziekte, one must observe that the symptoms of this disease and loin disease are very similar, if not identical. It has, however, not yet been shown experimentally that the two diseases have a common cause. The organism responsible for the production of the loin disease toxin has not even been cultivated, up to the present.

The Texas Agricultural Experiment Station, Division of Veterinary Science, has projected a number of experiments which will likely throw some more light on the subject and also indicate the best method of feeding bone meal to cattle.