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# Tularemia in Sheep And in Sheep Industry Workers

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**Tularemia in Sheep  
And in Sheep Industry Workers  
In Western United States**

William L. Jellison, Ph.D.

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PUBLIC HEALTH MONOGRAPH No. 28

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## Introduction

Few if any diseases have as many distinct epidemic and epizootic patterns as tularemia, which is a specific infectious disease of man and animals caused by the micro-organism *Pasteurella tularensis* of McCoy and Chapin (1). Early in the history of the disease several of the epidemic types were referred to by such distinctive names as "deerfly fever," "rabbit fever," "plaguelike disease of rodents," and "glandular type of tick fever." After a common etiological agent was established for some of these the name "tularemia" was proposed by Francis (2).

The main epidemic types<sup>1</sup> that can be recognized in North America at the present time are:

1. Tularemia from wild rabbits—rabbit fever.
2. Tularemia from terrestrial rodents.
3. Tularemia from the biting fly (*Chrysops discalis* Williston)—deerfly fever, or Pahvant Valley plague.
4. Tularemia from tick bites—glandular type of tick fever.
5. Tularemia from the semiaquatic mammals, such as muskrats and beavers, and from contaminated water.
6. Tularemia from game birds.

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<sup>1</sup> Khatenever (3) outlines five main epidemic types of tularemia observed in the USSR, where rodents appear to be most important as sources of human infection, in contrast to North America, where rabbits are the chief source of infection. Khatenever's epidemic types are: (a) Trade outbreaks resulting from hunting, skinning, and preparing hides and carcasses of rabbits, water rats, and other wild game or furbearing animals; (b) agricultural or mouse outbreaks from more or less direct contact with mice by farmers during epizootics; (c) contact outbreaks from food contaminated by infected mice; (d) water outbreaks from direct contact with or ingestion of contaminated water, but not including cases from contact with water animals; and (e) tularemia transmitted by arthropods (ticks and insects).

Although these various types of tularemia are often interrelated, there are features of seasonal distribution, clinical manifestation, or occupational incidence that serve to distinguish them. It is becoming evident that virulence of the strains and, therefore, prognosis of the individual case may be related to the epidemic type involved.

Range sheep in western United States are grazed on lands that are also favorable for cottontails and jackrabbits. Most of the area is infested with the Rocky Mountain wood tick, *Dermacentor andersoni* Stiles, a well-known vector of tularemia. Epizootics of tularemia among sheep occur in the spring of the year, when sheep often become heavily infested with ticks. Four such outbreaks have been studied by the Rocky Mountain Laboratory. Lambing often takes place at this season, and it is the usual season for shearing. Masses of infected ticks and infectious tick feces are encountered in shearing, and loose ticks readily transfer from sheep to man. Herders are continually exposed to ticks on the range. If sheep sicken and die, they are usually skinned, and the pelts are saved. A herder or a member of a shearing crew may occasionally take a wild rabbit for food. Thus, in the sheep industry, there is gross exposure of man to ticks and to infected animals, and many human tularemia cases result from this exposure. Most physicians in the area are aware of this association and anticipate a few sheep-contact cases each spring. We now have records of 189 such human cases of tularemia. These observations and records appear to warrant the consideration of tularemia in sheep and in sheep industry workers as a separate epizootic and epidemic entity, and it is the purpose of this paper to bring together our information on the subject.

# Tularemia in Sheep

The earliest publication on tularemia in sheep was entitled "Tularemia in Sheep in Nature," and was prepared by Dr. R. R. Parker of the Rocky Mountain Laboratory and Dr. J. S. Dade of the Idaho State Sheep Commission (4). It stated:

In the spring of 1923 there were reported to the field station of the Public Health Service at Hamilton, Mont., several instances of heavy losses among sheep that were believed to be due to the wood tick, *Dermacentor andersoni* Stiles. These reports were from eastern Montana and southern Idaho and were the first complaints of probable tick-caused trouble in sheep that had been received. Each report was investigated by Parker either by personal visit to the locality concerned or by correspondence, and in one instance ticks from affected eastern Montana sheep were tested at the Hamilton laboratory.

All of the guinea pigs injected with ticks from sick sheep in eastern Montana died, but the nature of the infection was not determined. Later experience suggests a diagnosis of tularemia in these affected animals, but at that time it was not known that the wood tick was a carrier of *P. tularensis*, and the workers concerned were not yet familiar with tularemia in laboratory animals.

Within the year, Parker, Spencer, and Francis (5) demonstrated that the wood tick could acquire and transmit tularemia and that the infection was present in adult ticks collected in nature. In 1925, *P. tularensis* was recovered from ticks collected from sick sheep, and agglutinins were demonstrated in serums from two recovered sheep by Parker and Butler (6).

During the next few years, several serious outbreaks of disease associated with heavy tick infestations were experienced in Idaho. However, for various reasons no diagnosis was established.

## Blackwell Band

When an episode of sickness in sheep associated with heavy tick infestations occurred in 1928, tularemia was immediately suspected and conclusively proved by Parker and Dade (4). Although their first publication inadvertently omits mention of the locality, a later report (7) gives considerably more

detail. This outbreak occurred in the Blackwell band of sheep between Mountain Home and Boise, Idaho.

The significant history of this band was related as follows:

. . . This band consisted of 900 old ewes, 300 yearlings and 700 lambs. They had wintered at Grandview, south of Mountain Home and, in the early spring, were purchased by H. Blackwell. Delivery was made at Mountain Home about the middle of March. Thence they were trailed by way of Orchard and Round Butte to Black Creek, 15 miles south of Boise, where spring range had been leased. While en route they traversed a section near Round Butte and Orchard in which the losses has been experienced by the Thompson Brothers bands . . . in 1927, and where bands of the same owners were again affected in 1928.

The loss of lambs was noted for several days prior to reaching Black Creek and immediately on arrival they called on Dade for assistance and the band was visited on April 1, by Dade and Dr. Huffman. Dade recommended the removal of the band from the sage brush to a tick-free area, but local range of this character all being occupied, the owner was forced to hold his sheep on Black Creek. It was therefore necessary to treat each affected animal individually, either the ticks being hand-picked or Kreso dip applied to points of attachment. Affected animals with temperatures of from 106.0° to 107.8° F. were numerous. Many were scouring. The band was again visited by Dade and Parker, April 12, and though much improved, losses were still being experienced, and the band showed marked loss of condition. Three ewes were "down," several recently dead lambs were seen, and there were about twenty sheep which showed by high carriage of the head, by stiff-legged walk or the presence of scours that they were affected. Up to this date, 50 percent of the lambs, 25 percent of the yearlings, and a smaller percentage of the ewes had been affected. Sixty lambs and 15 ewes had died.

Dead jackrabbits were reported as numerous along the trail followed to Black Creek and on the Black Creek range.

On April 1, when Dr. Dade and Dr. Huffman visited the band, they autopsied a dead lamb that had been acutely ill and another that had been dead for 12 to 24 hours. Both tissues and ticks from each lamb produced tularemia in guinea pigs when tested by Dr. Parker at the Rocky Mountain Laboratory. Infected guinea pig tissues were sent to Dr. Edward Francis of the Hygienic Laboratory at Washington, D. C. Dr. Francis recovered cultures of *P. tularensis* and confirmed the diagnosis of natural infection with tularemia in sheep.



On their April 12 visit, Dade and Parker obtained specimens for testing from 6 dead, sick, or convalescent sheep. Tularemia was produced in test guinea pigs by tissues of 1 dead sheep and by ticks of 2 sick sheep. Blood samples from 5 of the sick and convalescent animals agglutinated *P. tularensis* at maximum dilutions of 1/160, 1/640, 1/640, 1/1,280, and 1/1,280, respectively. The 8 animals in which tularemia was demonstrated included 4 lambs, 1 yearling, and 3 old ewes.

Thus, a disease entity in range sheep associated with heavy tick infestations and which had been repeatedly observed causing serious loss of condition and numerous fatalities was identified as tularemia.

### Ringling Epizootic

A more complete study of an outbreak of tularemia in sheep near Ringling, Mont., in 1934 was made by this laboratory and the Montana Livestock Sanitary Board as reported by Philip, Jellison, and Wilkins (8). The significant facts, not quoted verbatim, regarding this epizootic were related as follows:

A band of yearlings numbering 1,320 had been lambed in the spring of 1933. In November of that year it was moved to winter range near Melville, about 35 miles southeast of Ringling. The band left Melville April 1 and was driven overland, arriving near Ringling on April 8.

Presence of wood ticks was first noticed by the herder on April 7 when the flock was about 10 miles from Ringling.

On April 13, a few "gaunt looking" sheep were noticed. On April 16, 2 were definitely sick and about 30 more were sick the next day. On April 18, so many looked ill that the owners became alarmed, thinking some poisonous plant might be responsible, and moved the band, with the exception of 38 which were too severely affected to travel. Two, possibly 4, sheep died that day and by April 21, 17 had succumbed. On April 25, 60 head of moribund sheep and some 400 others, obviously ill, were scattered singly or in varying-sized groups over an area a mile square. Though "deticking" operations were begun by the owners, additional prostrated sheep continued to appear until about April 30.

The last moribund yearling was necropsied May 8. Except for this animal and a few that were still convalescent, the epizootic had ended. A count of dead carcasses in the epizootic area totaled 200 or about 15 percent of the flock.

The band of ewes (mothers of the affected yearlings) that had not been taken to winter range but had been pastured continuously near Ringling did not become

ill, although they too were infested with quite a few ticks.

The affected band was visited April 23 and 24 by Dr. H. F. Wilkins of the Montana Livestock Sanitary Board and Dr. Fred Stimpert of the State Hygienic Laboratory. Three or four blood samples obtained from sick animals on April 24 agglutinated *P. tularensis* at low dilutions. In view of the heavy tick infestations, a tentative diagnosis of tick-borne tularemia was made by Dr. Wilkins, who also recommended dipping all the animals. On April 25 a field laboratory was set up near the affected band for the collection and processing of specimens.

Numerous autopsies were performed on sheep that were found moribund or dead. Tissues were preserved in glycerine or in buffered glycerine solution for testing at the Rocky Mountain Laboratory. Blood samples for agglutination tests were taken from apparently normal, sick, moribund, convalescent, and recovered sheep. Ticks were collected from sheep and from sagebrush areas for testing.

Tissues from 6 moribund sheep were tested for tularemia by injection of guinea pigs. Tissues from 5 of the 6 sheep produced typical tularemia infections, and cultures of *P. tularensis* were established. Tissues yielding infection were: external lymphatic glands, 4 of 6 tests; spleen, 1 of 4 tests; and liver, 1 of 4 tests.

*P. tularensis* was recovered from 6 of 9 groups of ticks taken from sick sheep. Ticks from 5 animals selected as normal and ticks from sagebrush produced no infection.

Serum samples from 36 of the yearlings were tested for agglutinins. Twenty-seven of these were positive, giving complete agglutination at maximum dilutions varying from 1:80 to 1:2,560, as shown in table 1.

Table 1. Results of agglutination tests on range yearling sheep, Ringling, Mont., 1934

Condition of animals	Positive (4+ at 1:80 or higher)	Negative
Apparently normal.....	1	3
Ill.....	7	1
Very ill.....	4	0
Moribund.....	4	1
Recovering.....	7	1
"Recovered".....	4	3

It is likely that part of the animals selected as "recovered" had not been ill but were stragglers that remained behind with the affected sheep when the still healthy animals were removed from the area. It is also quite possible that some sheep were sick from other causes.

The outbreak was associated with an unusual prevalence of wood ticks, and local residents were agreed that jackrabbits and ground squirrels occurred in greater abundance than had been observed for several years. The sheep had been driven through a sagebrush area where ticks were abundant. *P. tularensis* was also recovered from jackrabbits, *Lepus townsendii*, found dead in the vicinity.

Several employees of this ranch were engaged in picking ticks from the sheep, in skinning dead sheep, and in otherwise handling the sheep and contaminated material. About 10 other men were engaged in the dipping operations. These men worked barehanded with little precaution against infection. None of them became ill. Seven of them were bled on April 30 and all gave negative agglutination tests for tularemia. A case of tickborne tularemia in a 4-year-old child occurred at a nearby ranch.

### Epizootic in Canada

An outbreak of tularemia in sheep at Whitla, southeastern Alberta, Canada, was reported by Gwatkin, Painter, and Moynihan (9), who visited the flock on April 25, 1942. Five sick sheep were in evidence and a number of others had already died. The flock was visited again on May 7. The significant history of this group as related by the authors was as follows:

The trouble occurred in a group of 850 yearling ewes. They had recently been brought from another part of the ranch which was covered with low scrub and was ideal land for ticks. The land on which they were feeding at the time of our first visit also had a certain amount of low scrub land. Twenty-four animals died and 5 or 6 seriously affected ewes recovered. Some animals had been observed to have been less seriously affected but we were unable to determine the number from the owner. They did not go off their feet and eventually recovered. We did not see any of these mild cases on either of our visits. No new cases developed between the first and second visit to the ranch. The sheep had been well fed and cared for and, with the exception of the affected ones, were in

excellent condition. There had been no trouble in previous years on this ranch. The animals examined were all heavily infested with wood ticks (*D. andersoni* Stiles) and keds (*Melophagus ovinus* L.). The sandy ground near the waterhole was covered with fully engorged ticks and some of these, as well as ticks from affected sheep, were collected for examination. On our second visit all ticks on the ground had disappeared. No drag ticks were taken on the first occasion and, as none got on us while walking through the scrub, it was evident that they were not plentiful at that time. We were unable to collect any on the second occasion although the weather was bright and the scrub was assiduously dragged for them.

Infection was demonstrated in the ticks and tissues of one sheep, and a high agglutination titer was obtained in a serum sample from a recovering animal. Twenty-four sheep died in the course of this epizootic.

One of the herdsmen for this flock became ill on April 29, 8 or 9 days after he had skinned 3 dead sheep, and the case was reported as ulceroglandular tularemia by Bow and Brown (10). This is the only human case we have on record that was definitely associated with any confirmed outbreak of tularemia in sheep. *P. tularensis* was also demonstrated in ticks and tissues of 1 jackrabbit, *Lepus townsendii*, and ticks from 1 ground squirrel, *Citellus richardsoni*, found dead in the epizootic area.

### Idaho Epizootic, 1949

The Arco Desert <sup>2</sup> in central Idaho had long been recognized as one of the main centers of tickborne disease in sheep, presumably tularemia. In the spring of 1927, it was estimated that 3,000 to 5,000 sheep died in this general area. The first epizootic on this desert in which tularemia was definitely diagnosed occurred in 1949 (11, 12) on a ranch near Rupert, Idaho. The history of this episode, which is typical of those observed previously, follows (12):

Lambing in the flock, which then consisted of about 290 ewes, started at the home ranch near Rupert, on December 25, 1948, and continued through January 1949. The flock, which totaled about 500 ewes and lambs, was kept at the ranch until April 15 when it was turned out on the range, a semidesert area 6 to 12 miles north of the town of Paul. There were several large, abandoned dry-land farms on this range that had grown up to cheat grass (*Bromus* sp.), but most of the

<sup>2</sup> A large desert area south of Arco, north of Rupert, east of Blackfoot, and west of Shoshone, referred to on some maps as the Snake River Plain.

range had never been under cultivation and in these areas sagebrush predominated.

The first sick animals were noticed on April 28, 13 days after the sheep were turned out on the range. A ewe and a lamb were found dead the following day. At this time, the sheep were grazing in high sagebrush just south of the prominent butte some 12 miles north of Paul. More dead sheep were observed on May 1, and 12 sick lambs, some of which eventually recovered, were brought back to the ranch during the next few days.

Heavy infestations of wood ticks, *Dermacentor andersoni* Stiles, were observed on the sheep, and ticks were abundant in the grazing area. At the recommendation of veterinarians of the Idaho Department of Agriculture, the flock was sprayed on May 5 by a commercial concern using an insecticide containing benzene-hexachloride and DDT, in addition to other ingredients. The flock was then moved from sagebrush range to grass areas on the dry-land farms where the ticks were less abundant. The outbreak quickly subsided.

Preceding and during the epizootic, the flock watered at two temporary ponds on the range and also received some water hauled from the home ranch wells.

A total of 7 or 8 ewes and 35 lambs died during the outbreak, a mortality of 8 to 10 percent. No unusual illness was observed in a small band consisting of 2 bucks, 10 ewes, and 16 lambs kept on the home ranch throughout the spring and summer.

On May 5, a sick lamb was shipped to the State Veterinary Laboratory at Boise, Idaho, where it was slaughtered and autopsied. Tissues from this animal were injected into guinea pigs. The guinea pigs died, and spleen and liver tissues of one were sent to the Rocky Mountain Laboratory with the request that they be tested for the presence of *Pasteurella tularensis*.

These tissues produced infection and death in all test guinea pigs. All had lesions characteristic of tularemia, and one culture of *P. tularensis* was isolated.

During the epizootic, blood specimens from six of the affected sheep were sent to the Idaho Department of Public Health at Boise for agglutination tests. The individual titers as given by McArthur and Brown (11) were: 1:640, 1:1,280, 1:1,280, 1:2,560, 1:5,120, and 1:5,120, respectively.

By the time a diagnosis had been established, the epizootic had largely subsided and the sheep were back on the open range, where further studies were not feasible. However, when the lambs were brought in for shipment in September, 5 months after the outbreak, blood samples were taken, and in November, when the ewes were brought in, they also were bled and all serums were tested for agglutination of *P.*

*tularensis* antigen. The results of these tests are shown in table 2.

The highest dilution of serums at which complete agglutination occurred was 1:80 for 4 of the lambs. However, serums from 2 of the mature sheep gave complete agglutination at dilutions of 1:160.

Table 2. Results of agglutination tests on range sheep, Rupert, Idaho, 1949

Agglutination reaction	148 lambs		283 mature sheep	
	Number	Percent	Number	Percent
3+ or 4+ at 1:20 or higher.....	23	15.5	56	19.8
Lower titer or negative..	125	84.5	227	80.2

Although these titers are not especially high, they are diagnostic in well-controlled tests and show that a diagnosis of spring outbreaks can be established in the fall when flocks are brought in from the range and are accessible in shipping pens or feed lots. Agglutinins for *P. tularensis* are extremely persistent in man and remain at diagnostic levels for 8 to 10 years after infection. They seem to be less persistent in sheep.

Apparently wool damage as a result of tularemia infection in sheep had been overlooked because few or no followup studies had been made. The owner of this band identified certain of the animals that had been sick by their poor or broken fleeces. A few lambs had completely shed their fleeces during the convalescent period and others had great patches of wool missing. It is possible that some of the damage may have resulted from application of the insecticide used to control ticks, but later (1952) we observed similar damage to wool in lambs that had not been sprayed or dipped but which were recovering from tularemia.

Wool samples were saved from 8 of these lambs with broken fleeces, that is, some portion of the fleece had been completely shed. These samples were submitted to the Montana Wool Laboratory, at Bozeman, for examination. In taking the samples, a piece was clipped at the border of both the shed and un-

shed portions of the fleece. The report on these samples stated that a distinct break in the wool was evident in 7 of the 8 samples. Representative of these analyses are the following (12):

*Lamb No. 47:* Sample  $\frac{3}{8}$  blood. Small lock was 4 inches long. Remainder of sample  $1\frac{1}{4}$  inches long. Indicates the portion  $2\frac{1}{4}$  inches long was mostly shed. Each portion sound. First growth is cotted and weathered. Second growth shows distinct crimp and is soft handling.

*Lamb No. 122:* Sample  $\frac{1}{4}$  blood. Length  $3\frac{1}{4}$  inches. Break  $2\frac{1}{4}$  inches from the tip (first growth). Portion on each side of break is sound. Weathered portion is harsh handling and felted. Short portion appears normal.

*Lamb No. 140:* Sample  $\frac{3}{8}$  grade—about  $2\frac{1}{2}$  inches long. Break in middle of sample. Some samples indicate complete shedding or loss of fiber outside of break. The weathered exterior portion is sound but appears dead or oxidized. The later growth is sound, appears normal having distinct crimp and good body (or substance).

Not all of the lambs with broken fleeces gave positive agglutination tests. However, of 17 lambs with evident wool damage, 7, or 41 percent, were positive, whereas only 15 percent of the entire flock gave positive reactions.

Any serious illness in a sheep may cause damage to the wool, and it is emphasized that this is an additional economic loss to owners of tularemia-affected bands.

No case of human infection was associated with this flock of infected sheep, although the owner, the herder, and the dipping crew experienced gross exposure in handling sick and dead animals and in handpicking ticks from sick animals.

## Idaho Epizootics, 1952

Several large bands of sheep, designated here as flocks A, B, C, D, and E, were affected with tularemia on the Arco Desert in central Idaho during the spring of 1952. We were first informed of this outbreak by Dr. F. X. McArthur of the Idaho Livestock Disease Control Laboratory at Boise. Dr. McArthur forwarded two refrigerated jars of guinea pig tissues to the Rocky Mountain Laboratory. One sample was from an animal that had been injected with tissues of a lamb from flock A at Oakley, Idaho. This lamb had died and portions of the liver had been forwarded to the Boise laboratory by Dr. R. M. Thornburg of Burley, Idaho. Inocu-

lum from this sample produced typical lesions of tularemia in guinea pigs, and a culture of *P. tularensis* was established. The strain proved to be highly virulent for domestic rabbits as has been found characteristic of strains isolated from sheep. The other sample sent by Dr. McArthur contained tissues from a guinea pig that had been injected with ticks, *D. Andersoni*, removed from sheep of flock D, Ketchum, Idaho. We were unable to demonstrate infection in this sample.

Although the outbreaks had subsided by the time our tests were completed, two trips were made to the area to obtain additional samples and to obtain firsthand information on the extent of the epizootic.

Histories and laboratory findings on the several flocks follow.

### Flock A

Headquarters ranch for the flock A sheep is near Oakley, Idaho. Four separate bands of these sheep were grazed on the Arco Desert during the spring of 1952 and one band was severely affected by the epizootic. The immediate history of this band as related to us by the owner was as follows:

One-half of the band was turned out to graze on the desert north of Milner, Idaho, on April 5. The remainder was turned out on April 7. The total band contained about 2,200 ewes and lambs.

The first sign of illness was observed about April 20 when a few lambs were noticeably stiff. By April 27, or 28, about 400 to 500 lambs were conspicuously ill and a few had died.

On April 28 or 29, two sick lambs heavily infested with ticks were brought into the Thornburg Veterinary Clinic at Oakley, Idaho, where autopsies were performed and tissues sent to Dr. Scott B. Brown, Inspector-in-Charge, State of Idaho Sheep Commission, Boise, Idaho. A tentative diagnosis of tularemia was made by Dr. R. M. Thornburg. (Dr. Brown tested these tissues in experimental animals. Test animals died and showed lesions suggestive of tularemia. Tissues from test animals were forwarded to the Rocky Mountain Laboratory, where cultures of *P. tularensis* were isolated after animal passage.) Following the recommendations of Dr. Thornburg, this band and two others were sprayed on May 1 with a formulation containing benzenehexachloride to kill the wood ticks which were extremely abundant on the animals. One band was sprayed with lindane, which also contains benzenehexachloride. The general condition of the sheep improved markedly after spraying and losses moderated. About 28 lambs died in this band and a total of about 20 lambs died in the other 3 bands.

Not more than 8 or 10 died after all the sheep were sprayed. Deaths among the ewes were few, but the herder thought that 2 or 3 had died during the outbreak.

On June 13, this band was examined in the hills south of Oakley, Idaho, where it had been grazing for some time. Ticks were scarce in this area, and only a few ticks were found on the animals, although they showed evidence of previous heavy tick infestations by encrusted areas along the chin, throat, chest, and belly. Forty-one ticks collected at this time were tested for infection, with negative results. Many of the lambs had badly broken fleeces and a few had shed almost completely. Retarded growth and underweight were evident in some animals. Stiffness was noticed in a few of the lambs but otherwise all of the sheep appeared to be healthy.

Agglutination tests were made on blood samples from 71 lambs selected at random. Complete agglutination was observed at the following titers: 1:20, 33 samples; 1:40, 32; 1:80, 30; 1:160, 20; and at 1:320, 7 samples.

Complete agglutination of *P. tularensis* antigen at a serum dilution of 1:20 is accepted as diagnostic when many of the animals show much higher titers. Presence of the disease had been confirmed by isolation of *P. tularensis* from one sick animal. On the basis of these samples, about 46 percent of the lambs experienced an infection with tularemia during the epizootic.

When the sheep in flock A were bled in June 1952, there were two sheep dogs in camp which the herder said had been ill about the same time as the sheep. Both dogs were bled and both were positive for *P. tularensis* by agglutination tests at titers of 1:20 and 1:80, respectively.

When Dr. R. M. Thornburg of Burley, Idaho, made a diagnosis of tularemia in this band of sheep he supplied the owner with streptomycin for treatment of some of the sick animals. The owner reported the following results:

In one instance, 9 sick lambs were brought in from the range in a truck. These were not treated and only one or two survived. A few days later 30 sick lambs were treated by injection with a single dose of streptomycin, about  $\frac{1}{2}$  gram each. Most of these animals showed marked improvement the following day and only two died.

Although this is not a well-controlled experiment, the results are encouraging in view of the well-known effectiveness of streptomycin in therapy of human tularemia. We have no information that any other antibiotics have been tried on sheep affected with tularemia.

### Flock B

The flock B sheep were divided into two bands for summer grazing. The immediate history of these prior to the outbreak as related to us by the owner, was as follows:

The bands were turned out on the Arco Desert about April 9. The first sick animals were noticed 2 weeks later (April 23), when the sheep were west of Paul and north of the prominent hill on the desert known locally as the butte. Wood ticks were abundant in the sagebrush where the sheep were grazing and on the sheep. [This is the same location where sheep became sick in 1949 as reported by Jellison and Kohls (12).] About 60 sick lambs from a band of 1,250 ewes and lambs and 30 sick yearlings from a band of 1,800 yearlings and dry ewes were trucked to the home ranch for care and observation. An estimated 50 lambs and 15 yearlings died on the range or among these brought to the home ranch.

On June 14, the small band of sheep at the home ranch was examined. This band included the animals brought in from the desert when ill and a number of orphan lambs which had never been on the range. All of the animals appeared well and active. Some showed evidence of having had heavy tick infestations earlier in the season but no ticks remained on them. Many of the lambs had badly broken fleeces.

Agglutination tests for *P. tularensis* on serum samples from this group gave the following results: Complete agglutination at a titer of 1:40 was observed in 25 of 61 samples from lambs and in 3 of 4 samples from ewes, or a total of 28 out of 65 samples. Thus, serums from 43 percent of these animals gave a positive agglutination test for tularemia at diagnostic titers. These results from a selected group are comparable to the random sample from flock A in which 46 percent gave positive tests for tularemia.

It was not practical to bleed any of the sheep which were out on the range at this time (June 13) but in late July the sheep were driven into stockyards at Ketchum, Idaho, for separation and shipment of lambs. On July 20, 13 lambs and 13 ewes were selected from the flock and bled. These animals had broken fleeces or were conspicuously thin or undersized. Six lambs and 6 ewes gave positive

agglutination tests, indicating that tularemia was prevalent in the band during the spring.

#### *Flock C*

The flock C sheep were on the Arco Desert during the spring season but they had been sprayed just before they were turned out on the range. Losses were light during the tick season; only 9 animals died, according to the owner's estimate. On July 20, this flock was examined on summer range near the home ranch in Stanley Basin. Ten lambs and 4 ewes showing broken fleeces or other evidence of previous illness were bled. Serum samples from 2 of the lambs gave positive agglutination tests, both at a titer of 1:80. All of the ewes were negative.

Both of the positive lambs had badly broken fleeces. In comparison with the other bands, this one was very little affected with tularemia. The difference may have been due to choice of range area or possibly due to the residual action of the insecticide in preventing tick infestation.

#### *Flock D*

According to Dr. McArthur, about 100 sheep had died in another band of 2,500 during the tick season. Some ticks from these sheep had been sent to the Boise Laboratory and inoculated into test animals, with fatal results. Tissues from these test animals were forwarded to the Rocky Mountain Laboratory but no infection was demonstrated in them.

#### *Flock E and Others*

Information was received from Tom Fica, deputy sheep inspector for the State of Idaho, Ketchum, Idaho, that a fifth band of sheep was seriously affected during tick season and that many animals had died. Of the E band, 3 ewes and 1 sick lamb were bled on June 15. The lamb and 1 ewe were positive for tularemia at diagnostic titers.

Dr. J. S. Allen, Sr., Idaho Falls, reported in a personal communication that he assisted in the treatment of still another flock during an outbreak of disease on the Arco Desert during the 1952 tick season. No blood samples were obtained from this band for diagnostic studies.

Owners of flocks involved in the 1952 outbreaks agreed that the weather was favorable during the outbreaks but that snow or pro-

longed cold rains would have greatly increased their losses when the sheep were weakened and sick.

In contrast to the agglutination tests on affected flocks showing a high percentage of positive reactions are the results on serums from 135 sheep taken for Q fever studies at Gooding, Idaho, March 28 and 29, 1951. Tularemia infection was not suspected in this flock, and none of the 135 samples gave any detectable agglutination at dilutions of 1:20 or higher.

#### **Human Infections During Epizootics**

During the 1952 Arco Desert studies, no human cases of tularemia attributable in any way to the occurrence of the disease in sheep came to our attention. Individuals exposed to risk of infection included owners, herders, and tick control crews engaged in spraying and dipping sheep, and in some instances, even hand picking ticks from sick animals.

#### **Convalescent Pathology**

In 1952, the first sick sheep in several affected bands were observed about April 20 to 24. The epizootic was at its height about April 27 to May 1 and had largely subsided by May 5. When the flocks were first examined by us on June 13 and 14 none of the sheep appeared acutely ill, although many showed broken fleeces, retarded growth, or emaciation as a result of recent illness.

We had not previously tested convalescent sheep, infected in nature with tularemia, for the persistence of infection in their tissues.<sup>3</sup> Two lambs from flock B were selected and brought to the laboratory alive for autopsy and testing.

One lamb, whose serum sample gave complete agglutination of *P. tularensis* antigen at a dilution of 1:160, was autopsied on June 23, approximately 30 days after the epizootic had subsided. The lamb had a badly broken fleece and was thin and retarded in growth. Its weight was estimated at 40 pounds, compared with 80 to 100 pounds for healthy lambs in this flock.

<sup>3</sup> The pathology of acute tularemia infection has been described for three naturally infected and one experimentally infected sheep by Lillie, Francis, and Parker (13).

At autopsy, the only conspicuous gross lesions noted were in the prescapular lymph nodes. The right node contained a dry necrotic center about 2 cm. in diameter; the left prescapular node contained a mass of thick pus. (The greatest tick concentration on infested sheep is usually along the lower neck, brisket, and navel areas. In a tick-transmitted infection the prescapular nodes are the ones most likely to show involvement.)

The following tissues were used for inoculums: bone marrow, brain, kidney, liver, prescapular lymphatic node, muscle, and spleen. Four mice were used for testing each tissue. All test mice remained well and, except for those injected with brain emulsion, were discarded after 30 days. Three of the four test mice injected with brain tissue died the day after inoculation. The fourth died on the seventh day with lesions suggestive of tularemia. A culture of *P. tularensis* was established from subsequent transfers.

The other lamb was killed and autopsied on July 8, approximately 1 month after the height of the epizootic. At this time it weighed 60 pounds and had completely shed its fleece. The following lesions were noted at autopsy:

The right prescapular node was enlarged and contained a thin-walled pocket at one extremity that was filled with dry greenish exudate. The left prescapular node was slightly enlarged and contained a small tract of dry necrotic material. The peritoneal cavity contained about 100 cc. of amber-colored fluid with strands of fibrous residue. The surface of the liver and the peritoneal surface of the diaphragm showed multiple small fibrous tags. There were multiple contiguous fibrous adhesions on the spleen capsule. The left lobe of the liver had a small strip and small focal areas of necrosis. Two lymph nodes in the omentum along the lesser curvature of the abomasum showed dry, caseous nodules, the largest of which was about 3 x 2 cm. and almost entirely replaced the node. The intermediate lobe of the right lung showed an area of atelectasis. These lesions suggest an earlier bacterial infection with resulting peritonitis.

The following tissues were tested for infection by maceration and injection into mice (4 mice were used for each tissue): brain (3 samples), right and left prescapular node, precrucal

node, omental node, liver, peritoneal fluid, spleen, kidney, heart muscle, lung, bone marrow, and muscle tissue. No infection resulted in the test mice.

On July 22, two convalescent lambs were selected from flock D at Bellevue, Idaho, for transport to the laboratory and autopsy. Serum samples from both were positive for tularemia at diagnostic titers. Both lambs had badly broken fleeces.

One lamb was killed and autopsied August 1, approximately 3 months after the epizootic had affected the band. It was an especially small animal and weighed only 48 pounds. The following conditions were noted:

The peritoneal surface of the rumen and abomasum was slightly roughened. The visceral surface of the rumen showed a caseous nodule about 6 mm. in diameter. There were several small, white subcapsular foci on the liver, and a few yellow necrotic streaks. One omental node was enlarged and contained several discrete yellowish necrotic areas and a caseous mass about 8 mm. in diameter. In this lamb the prescapular nodes were apparently normal, in contrast to the other three, which had enlarged nodes containing dry necrotic or caseous masses.

The following tissues were tested for infection by maceration and injection into mice: prescapular nodes, liver, spleen, kidney, lung, omental node, muscle, brain, and bone marrow. Test animals injected with kidney emulsion died of tularemia, and a culture of *P. tularensis* was established. A retest of this kidney tissue after 7 days' storage in a refrigerator again produced tularemia in test mice. All other tissues tested gave negative results.

The other lamb was autopsied on August 12. It weighed 71 pounds. The following conditions were noted:

The fleece had shed on the lower sides and belly. A 1-cm. subcutaneous abscess containing thick greenish exudate was present at the point of the right shoulder. The right prescapular node was enlarged about 2 times and contained a small pocket of necrotic exudate. The left prescapular node contained a thickly walled-off cavity 1 cm. in diameter filled with green caseous exudate. There was a fibrous band about 3 cm. long between the diaphragm and the liver at a fissure between liver lobes.

There were a few subscapular white foci on the liver about 1 mm. in diameter.

The following tissues were tested for infection by maceration and injection into mice: right and left prescapular node, spleen, liver, kidney, brain, bone marrow, muscle, lung, heart, and mandibular lymph node. No infection resulted in any of the test animals.

Discovery that tularemia infection persisted in lambs several months after apparent recovery suggested the possibility of infected lambs serving as sources of infection to workers in packing plants where animals are sent for slaughter or in stockyards along the way. We already had available serum samples from 51 employees in the stockyards and 71 employees in packinghouses at Ogden and Salt Lake City, where many of the sheep from Idaho are processed. These samples were taken in connection with Q fever surveys by Dr. H. G. Stoenner in 1951. Only one sample, from an employee at the livestock yards in Salt Lake City, gave a positive test for tularemia. The single positive test does not suggest any unusual incidence of infection in the workers.

There is also a possibility of infection surviving in the wool of sheep. In the summer of 1951, as part of the epidemic studies on Q fever, the employees of several wool processing plants in Portland, Oreg., were bled. These factories were receiving fresh wool from central Idaho. Twenty-six serums were tested for tularemia; 25 were entirely negative at 1:20 and higher dilutions, and 1 gave a partial agglutination at 1:20 and 1:40 only, but it could not be considered diagnostic, again suggesting no unusual incidence of infection.

### Symptomatology

There is probably little in the symptomatology of tularemia in sheep, especially in an individual animal, to differentiate it from numerous other diseases of sheep. Parker and Dade (4) noted the following symptoms in animals they examined in which infection was demonstrated:

- Acutely ill.
- Down and unable to rise.
- Lame for several days.
- Loss of weight and high carriage of head.
- Coughing and showed loss of weight.
- Scouring had been severe.

Philip, Jellison, and Wilkins (8) observed the following in their study of an affected band:

. . . slight dejection at first, stiffness in gait, desultory feeding, tendency to trail behind when band was moving, head held low, respiration rate noticeably increased, some rise in temperature, and diarrhea often in evidence. As the disease progressed, labored breathing was markedly evident and, in the later stages, often was accompanied by grunting, groaning, grinding of teeth; the walk became increasingly stiff, with a hunched spraddle-legged appearance of the hind quarters. The temperature in some animals was as high as 107° F. In the very severe cases diarrhea was usually present at this stage, accompanied frequently by a resultant fly-blown condition with quantities of maggots in evidence in the feces-soiled perianal region. Extreme restlessness and complete prostration finally ensued in the more severe cases, death following in a few hours. In a number of instances, the removal of infesting ticks before prolonged prostration had occurred resulted in complete recovery in a few days.

The prostrate stage of severe cases could easily be confused with tick paralysis. Paralyzed animals, however, when placed on their feet, are incapable of standing unaided and also show slight or no febrile reaction. By contrast, the Ringling sheep . . . could be roused to move for short distances or raised by hand to stand alone before lying down again; also fever was a marked symptom. Only one sheep of the Ringling band (46) showed symptoms of paralysis. Whether or not its condition was due to tick paralysis is uncertain. When seen on May 8, it was alert and apparently recovering although there was still a lack of coordination of the right legs. Its general condition and appearance were not indicative of an acute tularemia, but its serum, secured that date, agglutinated *Bact. tularense* in a dilution of 1:640. Recovery of the other affected sheep was uneventful and without observed sequelae.

A few sick sheep showed considerable mucous discharge from the nose but this is a very common symptom in ill sheep.

Rather similar symptoms were recorded by Gwatkin, Painter, and Moynihan (9) in Canada who state:

The affected animals were thin and weak but there was no evidence of paralysis. Temperatures ranged from 104° to 106.4° F. They were heavily infested with ticks and keds. Nasal discharge was noted. Breathing was very rapid, and in some cases labored, and the pulse was rapid and weak. All had diarrhea and the discharge was dark and fetid. They were stiff in movement but were fairly active until the last stages, although easily exhausted. Grinding of the teeth was noticed in the worst case after it went off its feet.

Flock symptoms seem more distinctive and reliable for a clinical diagnosis and are characterized by lagging behind of affected animals



with progressive illness, prostration, death. The affected animals will be heavily infested with wood ticks around the head, chest, and belly. Because of the association with ticks, epizootics are strictly seasonal, occurring in the spring months. Illness and fever rather than paralysis should distinguish tularemia from tick paralysis, which also may affect a flock in sud-

den outbreak proportions. The heavy tick infestations associate tularemia outbreaks with special habitats, that is, with sagebrush areas in range land. It is not yet recognized as a disease of feed lot animals. The presence of unusual numbers of dead, or sick, tick-infested jackrabbits in the epizootic area should also suggest tularemia.

## Human Tularemia and the Sheep Industry

Among the first human cases of tularemia reported in the sheep industry were two which originated in the general vicinity of Miles City, Mont. (14). Both were of the ulceroglandular type, with onsets in August and May, respectively. One patient gave a history of being bitten by ticks; the other reported no definite source of infection except that he worked with sheep and had not handled rabbits.

Four Montana cases of tularemia from sheep contact were reported by Winter, Farrand, and Herman (15). All patients had marked pulmonary symptoms but all recovered. The possibility of a primary pneumonic tularemia has been denied by some workers, who insist that lung involvement is a secondary process. We can only state that pulmonary complications are very common in sheep-contact cases, and the handling of sheep, sheep pelts, or wool loaded with highly infectious tick feces would afford abundant opportunity for infection by inhalation.

From the time the late Dr. R. R. Parker recognized tularemia as a tickborne disease, he kept records of all cases in western United States that came to his attention and he sent many letters of inquiry to physicians and State health officers requesting data on such cases. Information on cases associated with the sheep industry has been kept separately because of the interest of the Rocky Mountain Laboratory in tularemia as a disease of sheep. Additional information was obtained in 1944 by means of a questionnaire sent to physicians throughout the sheep-raising area of the western States. This questionnaire especially requested information on sheep-contact cases

of tularemia. A separate circular letter was addressed to members of the sheepshearers union, requesting information on illnesses associated with their work.

**Table 3. Occupational incidence of tularemia in the sheep industry, by State, 1934-52**

Occupation	Colorado	Idaho	Montana	Nevada	Oregon	Utah	Wyoming	Total
Shearer.....	14	9	4	1	1	15	20	64
Owner.....	1	4	34				15	54
Herder.....	3	4	7	2	2	5	19	42
Housewife.....			3				1	4
Other or unknown.....	2	2	12				9	25
Total.....	20	19	60	3	3	20	64	189

**Table 4. Incidence of tularemia in the sheep industry, according to month, by State, 1934-52**

Month	Colorado	Idaho	Montana	Nevada	Oregon	Utah	Wyoming	Total
January.....								
February.....							1	1
March.....	1		1			1		3
April.....	2	2	10			4	8	26
May.....	11	8	31	1	2	2	29	84
June.....		5	7	1		3	10	26
July.....			5		1	2	4	12
August.....		1	3					4
September.....	1						2	3
October.....							1	1
November.....							1	1
December.....								
Unknown.....	5	3	3	1		8	8	28
Total.....	20	19	60	3	3	20	64	189

**Table 5. Incidence of tularemia in the sheep industry, according to year, by State, 1934-52**

Year	Colorado	Idaho	Montana	Nevada	Oregon	Utah	Wyoming	Total
1934 and previous	1	4	6	1	1	1	4	18
1935							4	4
1936							1	1
1937	2	3			1			6
1938	1						1	2
1939		1				1		3
1940	2	2					4	8
1941		1	7			1	15	24
1942	4	4	24	1		1	8	42
1943	3	1	15				6	25
1944	4	1	4			2	5	16
1945	1			1		4	2	8
1946	1		1			4	3	9
1947					1		1	2
1948			2				2	4
1949		2	1			1	2	6
1950						1	2	3
1951						1	1	2
1952						1		1
Unknown	1					2	2	5
Total	20	19	60	3	3	20	64	189

**Table 6. Sources of tularemia infection in the sheep industry, by State, 1934-52**

Source of infection	Colorado	Idaho	Montana	Nevada	Oregon	Utah	Wyoming	Total
Sheep	5	7	40		1	7	27	87
Tick bite	4	4	13	1	1	7	16	46
Rabbit	1			1		1	3	6
Deerfly		1			1		2	4
Other or unknown	10	7	7	1		5	16	46
Total	20	19	60	3	3	20	64	189

**Table 7. Clinical types of tularemia infection in the sheep industry, by State, 1934-52**

Type of infection	Colorado	Idaho	Montana	Nevada	Oregon	Utah	Wyoming	Total
Ulceroglandular		8	28	2	2	5	28	73
Typhoidal	6	1	12			2	14	35
Glandular	1	7	8			1	8	25
Oculoglandular	1	1	1				1	4
Other or unknown	12	2	11	1	1	12	13	52
Total	20	19	60	3	3	20	64	189

Although the data from the above sources are admittedly incomplete, we have on file information on 189 human cases of tularemia associated with the sheep industry. When these data are analyzed and tabulated, some idea is obtained of the geographic distribution (map, p. 13), occupational incidence (table 3), seasonal and yearly incidence (tables 4 and 5), sources of infection (table 6), and clinical types (table 7) of infection experienced.

Shown on the map is the geographic distribution of the recorded cases for which the county where infection was supposedly acquired is known. In addition, there were many cases in which the county was unknown but the State was given. The numbers of cases for the several States, with a figure in parentheses showing the number of cases for which county data is lacking, are as follows: Colorado 20 (0); Idaho 9 (10); Montana 47 (13); Nevada 2 (1); Oregon 3 (0); Utah 13 (7); Wyoming 43 (21); or a total of 137 (52).

The occupational incidence of tularemia within the sheep industry based on these case data is rather evenly shared among shearers, 64 cases; owners, 54 cases; and herders, 42 cases (table 3). As a general rule, the shearers have contact with the animals only during a 4- or 5-month season, March to July, but this period includes the entire active season of the adult ticks. Four housewives on sheep ranches had tularemia. Women frequently help in the care of sheep and lambs and are exposed to ticks or contaminated materials brought into the house on work clothes.

In 46 cases, the source of infection (table 6) was not stated, but it was attributed to sheep in 87 cases, to tick bite in 46, to rabbits in 6, and to deerflies in 4 cases. Some of the sheep-contact cases were attributed to skinning dead sheep, and many of the others occurred in shearers who became grossly contaminated with crushed ticks and tick feces in the wool of sheep. Shearers' hands are often cut and abraded, thus allowing ample exposure to infection. Twelve of the sheep-contact cases were more specifically attributed to lambing.

In their daily routine, herders are highly exposed to ticks. Loose ticks readily transfer from sheep to man, so the high incidence of infection from tick bite is to be expected.



Both cottontails and jackrabbits are often abundant on sheep ranges and around sheep corrals, and contact with these animals accounted for 6 cases. Deerfly bites are a common source of infection in midsummer in certain western States, and 4 cases from this source were recorded.

The ulceroglandular type of infection predominated, with 73 cases. There were also 25 glandular cases; 4 oculoglandular; and 35 typhoidal (table 7). With many of the infections contracted from tick bite or through wounds on the hands and arms, the ulceroglandular type of infection is most likely. Although 35 cases were of the typhoidal type, suggesting infection from ingestion or oral contamination, in no instance was eating of mutton even suspected as a source of infection. Three persons on one ranch in Montana were thought to have acquired their infection from a contaminated water supply, but field investigations gave no convincing evidence as to the source of infection (16). However, in another

instance, we were able to associate a number of rural cases with a contaminated domestic water supply (17).

The information on seasonal incidence, shows a marked concentration of cases in the spring months (table 4). This is coincidental with the active period of adult wood ticks and with the shearing season. Of the 161 cases for which date of onset was recorded, 148 occurred in April, May, June, and July; 84, or approximately 52 percent, occurred in May. Some of the summer and fall cases were attributed to deerfly bites and to handling old pelts.

During the period 1934-52, from 1 to 42 cases of tularemia have been reported each year, an average rate of over 9 cases per year (table 5). There are records of 18 cases previous to that time, the first having been reported in 1926. For the years 1941-45, there were reported 24, 42, 25, 16, and 8 cases, respectively, but this probably represents a period of greater case-finding activity and interest rather than any great increase in the actual number of cases.

## Serologic Evidence of Tularemia in Sheep Industry Workers

Sheepshearing is an itinerant and largely seasonal occupation. Crews of 10 to 15 men, under the direction of a foreman and with complete equipment, start work in the southwestern States in February or March. They move north as the season advances, stopping a few days or weeks at each of the ranches along their scheduled route. Camp is often set up on the range in places distant from cities or medical care. The season ends in June or July in northern Montana and Washington.

The foreman follows much the same route each year, but the membership of his crew is fluid; men may work with a different crew each season. Sheepshearers have a well-organized union, membership in 1949 was over 1,000. Wages are comparatively high and the work is hard. Work is not interrupted for minor illnesses. For a more serious illness the man stays around camp, moves along with the crew when it changes location, and resumes work at the earliest possible time. Only se-

riously ill individuals consult a doctor or are hospitalized. Illness of several shearers at one time seriously disrupts the teamwork and schedule of the crew, and a long illness during the short working season is a major economic loss to the shearer.

With a background of numerous case histories of tularemia in shearers, a survey of crews was made by the Rocky Mountain Laboratory in the State of Montana in 1950 near the end of the shearing season in cooperation with LaVor Taylor, president of the shearers' union.

Shearing crews were contacted in the field at their work. Blood samples and brief histories of illness, if any, were taken. Blood samples and histories were also taken from wool tiers, "wool trompers," camp cooks, herders, wranglers, and owners who were present at the scene of operations.

Blood samples suitable for testing were obtained from 140 shearers and from 41 persons otherwise employed in the sheep industry in

the spring of 1950. During the spring of 1951 Dr. Herbert Stoenner and his associates, of this laboratory, made an extensive survey for Q fever in central and southern Idaho among persons engaged in various livestock industries. Sixty-six shearers and 70 other persons with part- or full-time employment in the sheep industry were bled and the blood samples were tested for *P. tularensis* agglutinins.

Results of serologic tests of blood samples taken in the two surveys are given in table 8.

Of 140 serum samples from shearers in Montana 23, or 16.4 percent, gave complete agglutination at a titer of 1:20 or higher. Not a single positive at this titer was found in the 41 samples from other sheep industry workers although several gave low titer reactions, which are probably indicative of past infections.

The results from the Idaho survey are

**Table 9. Positive serologic tests in serums from sheepshearers—Surveys in Montana, 1950, and Idaho, 1951**

Specimen No.	Initials of sheep-shearer	Years shearing	Previous diagnosis suggestive of tularemia	Complete 4+ agglutination titer at dilution:
Montana, 1950				
2	L. T.	37	None	1:80
11	A. A. W.	4	None	1:20
14	C. C. B.	30	None	1:20
15	P. M.	6	None	1:80
16	L. B.	20	"Tularemia, 1949"	1:80
38	C. M.	10	"Typhoid, 1945"	1:160
50	F. B.	24	"Spotted fever, 1949"	1:160
59	C. K.	30	"Probably tularemia, 1920"	1:20
60	J. A.	22	"Tularemia, 1939"	1:20
70	P. J.	18	"Tularemia, 1949"	1:320
73	N. N. N.	23	"Shearers' pneumonia, 1948"	1:40
75	C. E. N.	28	"Ulceroglandular infection, 1930"	1:40
85	A. C. A.	19	"Ulceroglandular infection, 1938"	1:40
95	K. T.	10	"Tick fever, 1940"	1:20
114	D. S.	21	None	1:20
125	J. C. A.	30	None	1:40
127	E. D. A.	8	None	1:20
133	R. F.	5	None	1:20
138	L. B.	21	"Ulceroglandular infection, 1939"	1:40
140	C. T.	8	None	1:320
183	P. J. S.	25	None	1:20
185	W. F. D.	30	None	1:20
187	I. D.	24	"Tularemia, 1945"	1:40
Idaho, 1951				
402	W. A. A.			1:80
404	R. P. S.	22	None	1:160
439	L. R.	3	"Tularemia"	1:160
459	A. B.	24	"Pneumonia, 1950"	1:160
488	J. C.	15	"Suspected tularemia, 1938"	1:20
517	R. R.			1:40
520	G. V.	8	Current case(?)	1:320
524	C. C.	5	"Tularemia, 1948"	1:80

comparable to results of the Montana survey. Of 66 samples from shearers, 8, or 12 percent, were positive at a titer of 1:20 and a few others gave partial agglutination at 1:20, which is

**Table 8. Results of serologic tests of blood taken in two surveys of sheep industry employees, 1950 and 1951**

Survey	Number tested	Results		
		Negative	Positive <sup>1</sup>	
			Number	Percent
Montana, 1950:				
Shearers----	140	117	23	16.4
Other-----	41	41	0	0
Idaho, 1951:				
Shearers----	66	58	8	12.1
Other-----	70	70	0	0

<sup>1</sup> At titer of 1:20 or higher.

highly suggestive of past infection. Again, not a single definite positive, 4+ at 1:20 or higher, was encountered in any of the 70 samples from sheep industry workmen other than shearers. Only 6 of the 31 positive individuals gave a definite history of illness with a diagnosis of tularemia; one thought he "probably had tularemia in 1920" and another "suspected tularemia in 1938" (table 9). Three had experienced severe ulceroglandular infections, but if a specific diagnosis had been established they had not been informed of it. During the surveys we encountered several active cases of influenzalike illness that gave positive agglutinations at a moderate titer, but in no instance did we find a frank case of typical glandular or ulceroglandular tularemia infection among the shearers or other employees.

Perhaps the most striking result of the surveys is that not a single one of the 31 individuals with a positive agglutination titer suggestive of past infection with tularemia had come to our attention through the reports of physicians or State health departments, although 64 cases in shearers had been reported from such sources, and some of them had been treated by physicians who have reported other tularemia cases to us.

Very little information was obtained in this survey as to locality where the infection was acquired. Places mentioned by various individuals as the locality where they contracted some illness were Rawlins, Dixon, Lander, Evanston, and Kemmerer, Wyo.; Huntley, Mont.; Antonito and Craig, Colo.; and Shoshone, Idaho.

## Discussion and Summary

Tularemia in epizootic proportions occasionally occurs in range sheep in the western United States and in the adjacent areas of Canada. Every outbreak studied so far has been associated with heavy infestations of wood ticks, *Dermacentor andersoni*. Infection has been repeatedly demonstrated in ticks from affected sheep and we conclude that ticks are the source of infection. It is likely that a high population of ticks builds up in some area where there is an abundance of rodents or rabbits on which the immature ticks feed and from which they acquire their infection. Then, at a favorable time during the tick season, a band of sheep moves through the tick-infested area and picks up a few infected ticks and many more noninfected ones. The burden of ticks, along with the infection, produces the observed symptoms and often, death. The feeding habits of this tick preclude the possibility of sheep-to-sheep transfer of infection except perhaps in rare instances when partially engorged ticks leaving a sheep that had died of tularemia might reattach themselves to another sheep. The prompt recovery of most sheep after removal of the ticks suggests that

infection is not the sole cause of death. Losses are greater among lambs and yearlings than among mature sheep. Infection has been found to persist in lambs several months after apparent recovery.

Losses can be avoided by keeping sheep out of tick-infested sagebrush areas in the spring months. Dipping or spraying with a persistent repellent or acaricide before the tick season begins may be effective. Should the disease appear, prompt destruction of the ticks by spraying or dipping the sheep is recommended. In one recorded instance, treatment of sick animals with streptomycin appeared to be effective.

Since range sheep are grazed on areas that are favorable for various kinds of wild animals which serve as reservoirs of tularemia and that are also favorable for ticks and deerflies, it is obvious that persons caring for sheep have considerable exposure to infection. Risk of infection is increased at times of epizootics, when sick animals are treated and dead animals are skinned.

Sheepshearers experience gross exposure to

infection from ticks, tick feces, and debris in the wool of sheep and they must occasionally shear animals that are actually sick. This exposure has resulted in at least 189 cases of tularemia of which we have record. At least 7 cases were fatal. Human infection from contact with sheep or from tick bite is apt to be severe. The majority of those who had had tularemia attributed infection to "contact with sheep" or to tick bites. The ulceroglandular type of infection predominated and the greatest incidence was during the spring months. The cases were largely restricted to Colorado, Idaho, Montana, Utah, and Wyoming.

In North America, sheep are the only large mammals among which tularemia is known to occur in truly epizootic proportions and which serve as important sources of human infection.

The geographic distribution and seasonal incidence of tularemia in sheep industry employees and the epizootic nature of the disease in sheep characterize an epidemiological entity distinct but yet closely related to the tickborne tularemia. The economic loss and the number and severity of human cases warrant the consideration of this epidemiological type of disease as a veterinary medical and public health problem.

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