Bulletin No. 262 - Economic Importance of Sarcosporidia, with Especial Reference to Sarcocystis tenella

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Economic Importance of Sarcosporidia, with Especial Reference to *Sarcocystis tenella*

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# TABLE OF CONTENTS

1. Introduction ..................................... 5  
   (a) Statement of problem .......................... 5  
   (b) Relations to human welfare:  
       Human infections ..................... 6  
       Economic relations. Direct. Indirect..... 6  
2. Kinds of animals infested .......................... 7  
   (a) Mammals. Domesticated. Wild ............... 7  
   (b) Birds. Domesticated. Wild ................ 7  
   (c) Reptiles .................................. 8  
3. Geographical distribution and incidence ........... 8  
   (a) Geographical distribution ................. 8  
   (b) Incidence and number of animals infected 8  
   (c) Location of parasites and severity of infections 11  
   (d) Seasonal infection ........................ 14  
4. Pathological effects ................................ 15  
   (a) General ................................ 15  
   (b) Effect on muscle fibers .................. 19  
   (c) Effect on other tissues .................. 24  
   (d) Sarcocystin. Nature and effects .......... 26  
5. Experimental results ................................ 30  
   (a) On mice and rats .......................... 30  
   (b) On lambs ................................ 31  
   (c) On birds ................................ 31  
   (d) On other mammals ........................ 32  
   (e) Immunity ................................ 32  
6. Losses .......................................... 33  
   (a) Direct .................................... 33  
   (b) Indirect .................................. 36  
7. Nature of Sarcosporidiosis ......................... 39  
   (a) Diagnosis ................................ 39  
   (b) Symptoms ................................ 40  
   (c) Treatment ................................ 41  
8. Prevention and Control ................................ 42  
9. Summary and Conclusions .......................... 44  
10. References ...................................... 47  

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Parasitologist and Zoologist
of the
Wyoming Agricultural Experiment Station
1913-1941.

Doctor Scott is still pursuing his studies on zoological problems of Wyoming. Having reached the age of 70, he has been on limited service since September, 1941. The science of parasitology has been greatly enriched by his work.

J. A. HILL, Director.
Economic Importance of Sarcosporidia, with Especial Reference to *Sarcocystis tenella*

By John W. Scott

**INTRODUCTION**

A previous Bulletin, No. 259, dealt with the life history of this important group of parasites, and our work added an appreciable contribution to an understanding of the life history of the sarcosporidia. The economic importance and effects produced by invasion of the parasites provided motives for setting up the original project of *Sarcocystis tenella*. It is therefore appropriate at this time to summarize our knowledge of the economic importance of the Sarcosporidia, including their relation to human welfare, the kinds of animals infested, the location of the parasites, the incidence and severity of infections, the pathological effects produced, the losses sustained, the symptoms, diagnosis and treatment, and to offer suggestions for prevention and control.

The very small size of the sarcosporidia makes it extremely difficult to follow their passage through the body, and only when the spores multiply in enormous numbers and are enclosed in cysts, is the aggregate visible to the naked eye. Not only is the life history difficult to follow, but the effects produced by the parasites are frequently either apparently harmless, indefinite, or obscure. However, evidence has gradually accumulated that their effects are insidious and, if present in large numbers, are very injurious. When large numbers of sarcocysts are present, the flesh is rendered unfit for food, and occasionally the pathological effects result in losses of animals, both direct and indirect. Like other one-celled, animal parasites, the effect of one, or even a considerable number is negligible. The wide distribution of the sarcocysts in the muscles of the body tends to produce a generalized rather than a specific effect. When present in enormous numbers, the muscle tissues become heavily involved, and lameness or stiffness results. If such a concentration of parasites occurs in a vital organ such as the heart, failure of that organ may occur and death result. It is the purpose of this bulletin to assemble, in the light of my own observations, all the facts that
we know of the economic importance of the Sarcosporidia, and since all treatments of sarcosporidiosis have proved ineffective, offer some suggestions for its prevention and control.

Gilmore, Kean and Posey (1942), reported the twelfth case of human sarcosporidiosis and the fourth with cardiac involvement. The twelve cases included two from Nancy, France, two from Panama, one from British West Indies, two from India, and one from the East Indies. Human infection is undoubtedly accidental, and apparently without any relation to the life history. Baraban and St. Remy (1894) noticed that sarcocysts found in the vocal cords of a beggar at Nancy produced no apparent clinical effect. Vuillemin (1902) made a comparative study of two human cases and found the sarcocysts corresponded to S. tenella. Darling (1909) discovered the parasites in a living host, but these disappeared within four months; the parasites were apparently abortive. Darling (1919) found sarcocysts in the tongue of an East Indian; the sporoblasts were similar to those found in 1909, and much smaller than those in sarcocysts of sheep, horse, hog or rat. He found no evidence of inflammation or degeneration in the neighborhood of the sarcocysts. Manifold (1924) found sarcocysts in human heart muscle, similar to those reported by Darling. Lambert (1927) found sarcosporidia infecting the myocardium of man, and compared the size of cysts and spores with the results of Darling and Manifold; the spores were smaller than those previously described, and no pathological effects were reported. Naidu (1928) states that an abscess on the left chest wall of a Hindu, which started as a small pustule, was excised and reported as a sarcosporidiosis. Many of the spores were almost straight, in some respects resembling those of Globidium. Gilmore, Kean and Posey (1942) reported an invasion of sarcosporidia in the myocardium of an eleven-year-old Panamanian child, and Hertig (1934) reported sarcocyst infection in the same location in a 26-day-old premature infant, a circumstance indicating transmission through the placenta.

In the century which has elapsed since the discovery of the Sarcosporidia there have been rather definite trends or viewpoints with regard to the economic and pathological importance of this
group. In the first period, lasting for approximately forty years, there was a tendency to exaggerate the importance of large sarcocysts in producing disease and death of domestic animals, particularly in sheep and goats. After the brilliant discoveries of Pasteur and others, there was a popular tendency to ascribe bacteria as causal agents of all disease, and minimize the effects of other organisms. However, following the discovery of the insect transmission of yellow fever in 1898 and the transmission of sarcosporidia by Theobald Smith in 1901 by feeding infected muscle, there was an aroused interest in the parasites infesting the muscle tissue. Since that time many critical, experimental and morphological studies have been made that give us a more complete picture of the economic importance and pathological significance of this obscure group of animals.

KINDS OF ANIMALS INFESTED

Sarcosporidia are found in the striated muscles of a large number of mammals, a considerable number of birds, and in a few reptiles. Sarcocysts have been reported from seven orders of Mammalia. However, they are most common in the Artiodactyla (even-toed), and in the Perissodactyla (one-toed), hoofed mammals, and in the Rodentia or gnawing mammals. They are also found in the Marsupialia (opossum), Carnivora (seal, dog, cat), Edentata (sloth), and Primates (apes, man). The even-toed hoofed mammals are especially heavily infested with sarcosporidia. Among our domestic animals these include sheep, goats, oxen, swine, camels, llamas, and the carabao. Wild species infested include deer, elk, reindeer, carabou, African and American antelopes, Grant’s gazelle, and the roebuck. Sarcosporidia are also common in the horse, as well as in such rodents as the rabbit, rats, mice, marmot, vole and hamsters. Darling (1915) reported sarcosporidia from the opossum and from the sloth (1915).

Erickson (1940) states that sarcocysts had been reported from eight orders, thirteen families, nineteen genera, and twenty species of birds. Besides being present in the domestic fowl and domestic duck, hosts of Sarcocystis include the wild mallard, gadwell, American pintail, shoveller, blue-winged teal, black duck,
turkey vulture, Wilson’s snipe, American redstart, rose-breasted grosbeak, olive-backed warbler, English sparrow, and seven species of birds from other continents. There is no doubt that if a careful search were made, sarcocysts would be found in many other species of mammals and birds, and probably in other reptiles.

Among the Reptilia, sarcocysts have been reported in the gecko (Tarentola) by Weber (1909) and by Chatton and Avel (1923); in the gecko (Platydactylus) by Bertram (1892); in the python by Tiegs (1931).

Besides the casual occurrence in man as noted above, sarcocysts have been reported from two other Primates; from the ape, Inuus, by Ratzel (1868), and from Macacus rhesus by Korte (1905) and by Castellani and Chalmers (1919). The reader is referred to the reference list at the end of this bulletin, and to the list found in Bulletin 259.

GEOGRAPHICAL DISTRIBUTION AND INCIDENCE

Sarcosporidia have been reported in both hemispheres and from every continent. These reports have come from widely scattered scientific centers, wherever the veterinarian, physician, or zoologist has been led to investigate the finer structure of muscles. If a comprehensive search were made for muscle parasites in other, more remote regions, probably few groups of parasites would have a more wide-spread distribution. No doubt sarcosporidia infest many species of mammals and birds from which no muscle parasites have been recorded.

The incidence of sarcosporidia is widely variable. In some species, as in man, sarcocysts are seldom found and infection is apparently casual, accidental, or a mere matter of chance. As pointed out in a previous bulletin (1943) such aberrant parasites are usually poorly developed, rather scarce, and give other evidence of being in an ill-adapted host. Such parasites play no important role in the life history, and are of academic rather than economic interest.
The question of what is a species in Sarcosporidia is still an unsolved problem. Species of *Sarcocystis* have been designated usually on the basis of size differences of spores and sarcocysts, the structure of the enclosing cyst wall, and chiefly on their presence in different hosts. Little is known of the natural classification of these parasites. Whatever the relationship of the named species, it is believed that sarcosporidia can complete the essential parts of their life-history in numerous species of mammals and birds. In these, such as *Sarcocystis tenella* of the sheep the incidence varies tremendously with the external environment, and probably to a considerable extent with the tolerance or natural resistance of the host. We have pointed out (1943) that infection with *S. tenella* varies with the season, with the climate, with moisture conditions, with concentration of flocks, and with various pasturing conditions. For example, in 1917 and 1918 after some years of more than average rainfall, sarcocysts were easy to find in the range sheep on the Laramie Plains, and approximately one hundred per cent were infested. From 1932 to 1937, during a series of drouth years, the range sheep in the same area showed much lighter infections, the incidence of infection dropped very low in many flocks, frequently below 50 per cent, and in some flocks, it was hard to find any sarcocysts. While the factors that cause variation in the incidence of sarcocysts in ducks must vary appreciably from those that obtain in the land-grazing sheep, the essential factors are probably closely parallel. The habits of the host species that harbor sarcosporidia vary, and it is to be expected that infestations will vary from host to host, and from individual to individual, dependent on factors previously reported.

How incidence of sarcosporidia varies may be illustrated by the following examples. Moulé (1886) found sarcocysts in 120 out of 150 mutton sheep. He also examined 100 muttons carefully, most of which were in poor flesh, and found sarcocysts in all except one. In 51 fat muttons, the sarcocysts were fewer, smaller, and less well formed than in the muscles of cachetic sheep. He found sarcocysts in three out of twelve goats. Moulé (1888) again reported *S. tenella* in 196 out of 200 lean muttons, and in 44 out of 100 fat sheep. He gives no clue as to the en-
vironment under which these sheep were raised. Bergmann (1913) in Sweden, reported results from the examination of sheep, swine, cattle, and horses, extending over a period of years. Of 342 lambs, about 3 months old, he found sarcosporidia in 20 per cent. In 100 lambs, one and one-half to two and one-half months old, he found S. tenella in eight, but no Balbiania; in 139 sheep, all over one year of age, he found S. tenella in 107 and Balbiania in 39. He examined 27,751 pigs, many of them about 7 months old and found sarcosporidiosis in 8,498 (30.62%); sarcocysts were found in 96 per cent of swine, one year of age or over. He examined 1,200 cattle over two and one-half years of age, in the course of one year, at the rate of 100 each month and found 88 per cent infected. During the same period, 1,011 calves from four to ten weeks of age were examined and 8 per cent had sarcosporidia. Also, 67 per cent of 480 horses and all of 15 reindeer were found infected. The youngest animals found infected were: a lamb, 6 weeks; a calf, 6 weeks; a pig, 10 weeks; and a colt, 10 months.

Numerous other records are available. Sabrazes and Muratet (1911) found that 90 per cent of the horses coming to the abattoir at Bordeaux were affected. Stroh (1921) reported that 92 per cent of 133 cattle two years of age or older, slaughtered in spring and summer at Augsburg, were infected with small cysts of sarcosporidia. Hasselman (1923) found sarcocysts in 100 per cent of pigs of Brazil, the age not given. In 1926, he reported finding Miescheria crusi, a parasite specific for cardiac muscle, in all of 33 ox hearts examined from four different cities of Brazil. Nakanishi (1929) examined 1,922 head of Korean cattle, and reported they were slightly infested, but calves were seriously infested. Oxen five to nine years old, enclosed in stalls from one to five years, were always heavily infested. Thevenoz (1932) selected at random 100 healthy cattle in a slaughter house and found that all were infected with sarcosporidia; 22 were from 16-24 months, 53 from 25-60 months, and 25 from 5-12 years of age. Wilson and McDonald (1938) examined 35 cows, 29 calves from 6-8 weeks old, 27 sheep, and one horse; they found sarcocysts in 30 cows, no calves, 8 sheep, and one horse.
The incidence of sarcocysts in birds has not been so fully reported. However, Leidy (1875), Barrows (1883), Stiles (1893), Splendore (1907), Crawley (1911), Bascom and Osterhud (1928), Vogelsang (1929) and Beaudette (1941), have reported the presence of sarcosporidia in various species of birds, and Erickson (1940) added four new records. Riley (1931) gave a historical review of Sarcocystis in ducks, and published three excellent figures. The presence in reptiles has been previously mentioned.

LOCATION OF PARASITES AND SEVERITY OF INFECTION

In general, sarcocysts may be found in all striated or voluntary muscles, including the heart and diaphragm, or in connective tissue intimately related to these muscles, but never in smooth muscle fibers. Leiserung (1865) noted numerous large cysts (Balbiania) on the oesophagus of a sheep. Manz (1867) found sarcocysts in almost all striated muscles of the ox, roebuck, mouse, rat, and most plentiful in swine. Führstenburg (1869) found large cysts in a French ram, partly in the connective tissue around the oesophagus, partly in the muscle fibers and some between the muscle and the mucous membrane. Some cysts were also found in muscles of the larynx, pharynx, palate, neck, head, arm, breast, intercostal, diaphragm, and belly. Zürn (1872) saw two large cysts on the covering of the brain. Moulé (1886) reported that sarcocysts were fewer in number and smaller in size in fat sheep than in the muscles of cachetic sheep. He also found sarcocysts in all the muscles of a mutton carcass. In 1888, he found cysts on the oesophagus of 272 sheep out of a total of 900 examined. Bertram (1892) stated that the youngest stage observed had a length of 40 micra and a width of 6 micra, while some cysts in the wall of the oesophagus were the size of hazel nuts. He observed small cysts only in tongue, masseter, larynx, pharynx, oesophagus, neck, intercostal, diaphragm, heart, belly, and hip muscles. Large cysts were found only in larynx, pharynx, oesophagus, tongue, and palatal muscles. He concluded that Balbiania and sarcocysts belong to the same species, and believed that pres-
Sarcozystis tenella was present in 182 out of 185 sheep. Both large and small cysts were found in old sheep, but only small cysts in eight months old lambs, and no cysts in embryos. Linstow (1903) reported Balbiania siamensis from the base of the tongue of Bos bubalis. Except in the locations mentioned sarcocysts in general show no important differences in the various muscles. Mason (1910) found no appreciable difference in the distribution of sarcocysts in the various muscles (oesophagus, larynx, head, tongue, neck, thorax, abdomen, sub-lumbar dorsal, gluteal, arms, forearms, thigh, leg, heart, diaphragm, tail), except that there were more on the oesophagus and in the gluteal regions and fewer in the diaphragm. I have noted (1930, et al) that the shape of the muscle fiber limits to a certain extent the shape of the enclosed sarcocyst, as for example in diaphragm and heart muscle. Alvarez (1926) noted that oesophageal sarcosporidiosis was very frequent in Zarazoza, the infestation possibly attaining 100 per cent. Numerous authors, Stiles, Riley, Crawley, Erickson and others, have observed sarcocysts in various skeletal muscles of birds and Bascom and Osterud (1928) found them in the skeletal muscles of the turkey buzzard. Thevenoz (1932) reported that the cardiac end of the oesophagus was the site of predilection in cattle, and that the number of cysts increased after birth to about 24 months of age, and then progressively decreased owing to the death of the parasites and the progressive immigration of the cattle which prevented re-infection. It may be worth noting that Arase (1938) determined the percentage of muscle infection with living sarcosporidia in pigs, steers, goats, horses, and rats near Dairen, and the organs of predilection were studied.

So far as I have been able to learn, the only studies relating to the number of sarcocysts present in a given volume of muscle have been made at this experiment station. To determine the relative incidence and severity of infection, the following method was used: A piece of muscle from a chosen location was fixed and sectioned at a given thickness, approximately in the plane of the muscle fibers. The number of sarcocysts appearing in each of a series of sections was counted and recorded. To prevent
duplication in counting sarcocysts that appeared in more than one section, the location of each sarcocyst in the section was platted, and by comparison of successive sections, any duplication was easily detected. The area of the sections was carefully measured and the average area of the sections was multiplied by the total thickness of the series of sections to give a known volume, containing a known number of sarcocysts. Adopting this procedure as a standard method enabled us to estimate the number of sarcocysts present in a cubic centimeter or cubic inch of flesh of any given animal. By this means we were able to obtain valuable information in regard to incidence of the parasite and severity of infection, following various kinds of experimental treatment. See Scott, 1918b, 1920b, 1930, and Scott and O'Roke, 1920.

The incidence of infection varies tremendously and is related to conditions favorable or unfavorable to the life history, as pointed out by the author, (1943). When lambs are raised with their ewes in a dry barn lot, with pure water in a trough, and feed available only from the racks, the infection is always relatively light and may fail altogether, with seldom more than a few hundred sarcocysts per cubic centimeter of flesh. If one of these lambs is subjected to additional treatment favorable to the life history of the parasite, some astonishing results may be obtained. For example in 1917, lamb 293, which was raised in such a dry lot, on 21 occasions was fed grass from a small pen two feet wide and six feet long forming part of the border of a pond in lot 3; here the grass grew luxuriantly and was repeatedly contaminated with sheep feces. After slaughter it was found that this lamb had 5,387 sarcocysts per ccm. of heart muscle. The same year, another lamb raised in the same dry lot, No. 296, was allowed to graze 21 times in a small enclosure ten by twelve feet in size, in lot 3. It thus had the opportunity to re-inflect itself many times through the medium of its own feces, and when killed the heart muscle contained 7,539 sarcocysts per ccm. of flesh.

Extensive data, which we have assembled, could be introduced here showing rather wide variation in the incidence of infection in lambs, raised under apparently identical conditions. At present we have no better explanation than to say that some individuals
are more tolerant of the parasite, or perhaps some lambs are more fastidious about eating grass contaminated with feces. After watching the feeding behaviour of lambs, I am inclined to think that the latter explanation may be of considerable importance.

As pointed out in a previous bulletin, restricted range, confinement in close quarters, or any unsanitary condition favoring the contamination of food by means of feces of infected animals, tends to increase infection, and since the infection is generalized, the pathological changes become more pronounced. In an old, lean ewe, kept on a ranch with other sheep under very unsanitary conditions, it was estimated that the flesh contained 88,000 sarcocysts per cubic centimeter, or over 1,400,000 sarcocysts per cubic inch of flesh. It is needless to say that this flesh was clearly unfit for food, though none of the parasites could be seen with the naked eye.

Previously (1918b) it was shown that the incidence of infection on the Laramie Plains is closely related to seasonal infection and that the age of sheep, of four years of age or less, could be determined approximately by a careful study of the size and number of sarcocysts present. Evidence from other sources, bearing on seasonal infection, has been reviewed in a recent bulletin on the life history of *S. tenella* (1943). Seasonal infection probably does not occur in the tropics and has been reported only in colder climates, as in Sweden and South Africa, where distinct seasonal changes are prevalent. Some of the work of Bergmann (1913) indicates that under certain circumstances infection in pigs may take place in winter in Sweden. It is generally assumed that the spores of sarcosporidia, particularly *S. tenella*, are unable to long withstand the rigors of a winter climate, and that this is sufficient to explain seasonal infection in the warmer months. The notorious unsanitary habits of swine may explain winter infection of pigs, kept in sties in Sweden.
Because of the generalized nature of the infection and the minute size of the muscle parasites, the effects produced are obscure and frequently pass unnoticed. Unless a large number of sarcocysts are present, no important noticeable effect has been observed. Even when a comparatively large number of sarcocysts are found in the muscles, the casual observer may see nothing abnormal. On account of the generalized nature of the infection, affecting the heart and diaphragm, as well as all the striated muscles of the body, the effects are usually insidious, and not readily diagnosed. However, the observations of a large number of workers and the evidence from numerous experiments with the toxin found in the sarcocysts, gives abundant proof that the sarcosporidia have a far greater pathological and therefore economic significance than has been generally supposed. The diagnosis, symptoms, and particularly the toxological effects produced have attracted the attention of parasitologists and veterinarians more than any other phase of the study of sarcosporidia. For this reason a somewhat detailed review of the results of these workers is both interesting and merited.

Early workers, before the microscope was well developed, frequently made erroneous observations or attached undue significance to the Balbiania, which were evident to the naked eye. For example, Rainey (1857) made the mistake of describing sarcocysts in the muscle fibers as young stages of a tapeworm. Leiserung (1865) found numerous, yellowish cysts from the size of a pea to the size of a hazelnut, on the oesophagus of a sheep that had died suddenly, but concluded that too little was known of the “Rainey’s Corpuscles” to ascribe to them the death of the sheep, as had been done by Winkler who sent the material. Beale (1866) found sarcocysts in all or nearly all animals dying of cattle plague, and sarcocysts in smaller numbers in healthy animals. He found sarcocysts in immense numbers in a six months old calf that had died of cattle plague, but stated that the sarcocysts may not have had anything to do with the plague.

Cobbald (1866, 1879) ate the heart of a healthy sheep, calculated to have 1,000 parasites per ounce and the heart of a healthy
bullock in which the number of parasites was rather in excess of
those in the sheep; it was estimated he swallowed not less than
18,000 cysts, with no after effects, and he concluded that they were
entirely harmless to humans. Gerlach (1866) concluded that these
parasites do not usually influence health, but in large masses may
destroy movement by pressure on contractile muscle substance and
cause the muscle substance to diminish. Virchow (1866) de-
scribed an illness of swine characterized by lameness of the hind
quarters and a skin eruption, and doubtfully attributed this to
sarcocysts. Ratzel (1868) found sarcocysts in an ape that had
been lame and bedridden several weeks before its death, and sug-
gested that the sarcocysts were probably an important cause of
illness. In an imported French ram which continuously lost flesh
and was killed, and which showed emaciation and insignificant
swelling of the mesentery and lymph glands, Führstenberg (1869)
found on the oesophagus, large and small, yellow-white colored
cysts from the size of a small pea to the size of a small bean,
and from one-eighth to one-fourth inch in cross-section; cysts
were also found in various other muscles of the body, and no
other cause was observed to account for the condition of the ram.
Zürn (1872) saw sheep take epileptic fits and die, and relates that
cysts were found in various muscles of these sheep. Later dis-
ccoveries indicate that the sarcocysts probably had nothing to do
with the epileptic fits. Von Niederhausen (1873) observed the
death of an old goat whose muscles of the larynx were filled with
cysts, and consequently had considerable difficulty in breathing.
The appetite was good with no fever and no clinical symptoms
in the internal organs. The large cysts were from 6 to 14 mm.
in length and 3 to 7 mm. in width. He concluded the illness
was due to the sarcocysts. Leidy (1875) observed myriads of
sarcocysts measuring 0.15 inch in length in the flesh of a Mallard
duck, evidently unfit for food. Barrows (1883) observed a
similar condition in the muscles of a Parula in Uruguay. Laulanie
(1884), working on swine, reported interstitial myositis diffuse
in nature, found generally; the flesh was altered, not entirely
useless, but had lost a great deal of its food qualities; the
muscle elements were notably atrophied, and granulations promi-
ment: the muscle substance was reduced and in a measure author-
izes seizure of the carcass. There were other cases with no visible lesions. Railliet (1866a) felt there was no need of con-
demning the flesh, unless the parasites caused grave muscular lesions as described by Laulanie. Moulé (1886) states that when
many sarcocysts are present in the flesh of sheep, the flesh is watery, and when few sarcocysts are present, the flesh is normal in ap-
pearance. He and his colleague (1888) ate several bits of raw
beef containing a quantity of sarcocysts, but experienced nothing to indicate development. Our study of the life history of *S. tenella* (1943) indicates that this should be expected. Putz (1887) be-
lieved that Miescher's cysts ordinarily play no role as exciters of disease; however, he states that large cysts of herbivores, if
numerous in the region of the larynx, may produce severe illness and death, but in other locations no serious interference with health.

Minchin (1903) noted that sarcosporidia sometimes cause fatal epizootics among domestic animals, and mentions paralysis of the hind quarters in hogs. He believed the dangerous effects are caused by an active poison secreted by the parasites. Mettam (1905a) found *S. tenella* in large numbers in sheep dying from an affection that at first was believed to be ovine piroplasmosis. Lineaux (1907) described the symptoms of a sick horse which by extrication of muscle was proved to have sarcosporidia. He suspected the parasites might be the cause of grave ailments or disturbance. Ratz (1909) noted that the muscle tissue of the common fowl infested with sarcocysts showed fatty degeneration. Mason (1910) found no inflammatory reaction in camels when the cysts were intact, and believed that re-infection occurred by bursting of the cysts and the spores were carried in the blood stream. Alexander (1913) described the disease in sheep called "scrapie" and attributed it to *S. tenella*. Cadeac (1913) reviewed the literature on the occurrence, symptoms, lesions, diagnosis, and treatment of sarcosporidiosis in the horse and other domestic ani-
mals. McGowan and Rettie (1913) found sarcocysts in all sheep with "scrapie" and suggested that this disease may be a syndrome that applies when a sheep is dying of a mass infection of *S. tenella*. Later McGowan (1914) on the basis of association concluded that
sarcosporidia have a causal connection with “scrapie.” However, McFadyean (1918b) and Gaiger (1924) showed the fallacy of McGowan’s theory that *S. tenella* is the cause of “scrapie.” Darling (1915) after examining 1,000 animals and finding sarcocysts in the opossum, sloth and hawk, thought that sarcosporidia were of little pathogenic or economic importance. Hedinger (1915) after an eight months study of gal-lamziekte advanced the theory that it was due to the presence in the muscular tissue of enormous numbers of sarcosporidia. Walker (1918) found no degenerative changes in skeletal muscles and no pathological changes in peripheral nerves, except that in two sheep a fatty degeneration of skeletal muscles was observed. An oedema of inter-muscular connective tissue was found in some sheep in poor condition.

Croveri (1920) came to the conclusion that ordinarily the sarcosporidia cause no serious harm, but, if there is deficiency of nutrition or other disease present, complications of serious nature may arise and death result. Stroh (1921) examined twelve degenerated hearts of cattle with severe foot and mouth disease and found all heavily infected with sarcosporidia. He states that the question whether the degeneration of heart muscle arising in severe forms of foot and mouth disease has proceeded from a significant localized injury of the myocardium due to the invasion of the sarcosporidia, cannot be answered in the affirmative, but he thinks this may be possible. Guiart (1922) believes that, except in the case of *S. muris* in the mouse, the pathogenicity of sarcosporidia is proportional to the numbers of parasites involved. He mentions that of three human cases, two were attributed to *S. tenella*, and as a prophylaxis suggests avoiding eating raw meat. Rademaker (1923) stated that only in very infrequent cases do sarcosporidia lead to pathological alterations. Wroblewski (1923) concluded that the adult sarcocysts, as long as they are in the muscles, do not do as much harm as the young stages in the blood. He was of the opinion that the latter can produce a severe epizootic with picture of a severe anaemia, and a large per cent fatal. Leese (1928) mentions that sarcosporidia affect all emaciated camels slaughtered in Cairo; the cysts average 3 mm. but may reach 12 mm. in length. Chiwy and Colback (1926) working
in the Belgian Congo, state that the animal does not appear in-commoded, except when infested with a severe localization, as referred to later.

Osterud and Bascom (1928) reported sarcocysts in muscle fibers of the pectoralis major of the Turkey buzzard, but the adjacent fibers appeared normal and there was no apparent connective tissue reaction. Vogelsang (1929), while investigating sarcosporidia in the birds of Uruguay, saw no noteworthy inflammation processes in the muscles. However, he found that severely infested birds fly low and slowly. Babudieri (1932) states that cysts are found in practically all body muscles, and that gravity of infection depends upon the food, season, environment, sex, constitution, and complicating diseases. Erickson (1940) reported on a pintail duck in a weakened condition that died five days later; it was found heavily infected with S. rileyi in all skeletal muscles and in the heart. He thought that this may have been a factor in contributing to its death, and mentions that a large proportion of the ducks that come in for examination are sick, diseased or crippled. On the other hand Beaudette (1941) reported on sarcosporidiosis in a black duck and states that this bird was in good condition though its skeletal muscles were thoroughly infiltrated with sarcocysts. This resumé of the generalized effects of sarcosporidia as found in the literature, gives an idea of the obscure nature of the disease. We shall next take up the more specific effects on the muscle tissue.

EFFECTS ON MUSCLE TISSUE

As early as 1872, Siedamgrotzky, in a horse parasitized with sarcosporidia, found an increase in the muscle nuclei in neighboring muscle fibers, and a hyperplasia of the interstitial connective tissue which leads to a simple atrophy of the muscle fibers. Laulanie (1884) found the muscle elements were notably atrophied, granulations prominent, and the muscle so reduced that in a measure this condition justified the seizure of the flesh as unfit for food. Reick (1888) studied the pathological effects as seen in sections of tissue in two cases of sarcosporidiosis in the horse. He concluded that as a rule, sarcocysts produce no serious patho-
logical changes, but under some circumstances they may produce an acute, later chronic, interstitial myositis, with a secondary degeneration of muscle fibers. Pfeiffer (1891) believed that young animals were probably sick at the time of the invasion of the muscles. Pluymers (1896) concluded that sarcocysts are generally harmless, but exceptionally they become the cause of inflammatory lesions, more or less extended, with the destruction of muscular tissue, and may even cause death, if the organ attacked (heart, diaphragm) is a vital one. The inflammatory lesions appear only after complete development of the parasite and after rupture of the organ containing them, and the inflammation is always terminated by organization of tissue.

Brooks (1903) observed the parasites of wild animals in captivity, and he and Miller in 1902 reported the case of an elk dead, due to the presence of sarcocysts in the heart muscle. Brooks states that this disease eventually causes the death of most of the elk; that he has reason to believe that sarcosporidiosis is extremely rare in wild animals while it is very frequent in members of the deer family in captivity; that in elk, caribou, and native deer, the parasites invade the heart causing in most cases a sort of parenchymatous degeneration of the heart muscle and infiltration. Ratz (1909) pointed out that infested muscle tissue in the common fowl showed fatty degeneration. Weber (1909) working on a lizard, found that degeneration of the muscle fibers seems uniquely related to the phenomenon of compression, that this degeneration stops at the end of the sac enclosing the parasite, and thinks that the toxin or enzymes are manifest only when the cuticle of the parasite is ruptured.

Mason (1910) states that in the absence of any wasting diseases, such as tuberculosis, typanosomiasis, strongylosis, etc., and when the parasites are numerous, camels appeared extremely emaciated with inflammation of the heart and its coverings and linings, with numerous small hemorrhages, gelatinous degeneration, and disintegration of muscle fibers. Cadeac (1913) claimed that S. tenella in the sheep is ordinarily inoffensive, but exceptionally it becomes the cause of inflammatory lesions with destruction of muscle tissues, and may even cause death if the heart or dia-
phragm is involved; that the inflammatory lesions do not appear until after the growth of the cyst is complete and after rupture of the muscle fiber, and are terminated by organization of infiltrated tissue. Fantham (1913) examined an African mouse bird and found it heavily parasitized with a generalized infestation of the muscles; the bird showed no obvious external symptoms and the effect on the chest wall was doubtful.

Viljoen (1918) studied the pathological lesions in muscle fibers in cattle in South Africa, and concluded that sarcosporidia are generally harmless parasites in cattle and that they overrun the animal only when it is debilitated through other causes. Beller (1924) made a careful study of the effects produced by sarcosporidia on the skeletal muscles, not infrequently found in slaughtered domestic animals. He arrived at the following conclusions: (1) There is in some slaughtered domestic animals a disease of the skeletal musculature produced by sarcosporidia. (2) The pathological anatomy is characterized by a perimysitis with an important share of eosinophile lencocytes. The muscle fibers react thereupon in the sense of a simple atrophy, in connection with an obliterating endartitis of the regional blood vessels as part compensation for the contractile substance destroyed, and the elastic tissue in the perimysium becomes hypertrophied. (3) The inflammation occurs at the stage of the diffuse infiltration of the parasites, while its healing in the muscle fibers runs a reactionless course. Hassleman (1926) found sarcocysts in 100 per cent of 300 ox hearts examined. The lesions found included colonies (cysts) of the parasites within the muscle fibers; rupture of the fibers; loss of chromophyllic staining reaction; loss of power of nuclear reproduction; localized or diffuse inflammation with infiltration of lymphocytes and mononuclears, or fibrous hyperplasia. Functional tests failed to reveal alterations of cardiac rhythm or extra systoles in two heavily parasitized animals, examined ante mortem.

Koegel (1926) in reporting on sarcosporidia in sheep and swine, states that the musculature can contain countless sarcocysts so that the flesh has a diseased appearance. Strongly infected muscle can be colored quite white, pale grey, red flecked, or striated greenish yellow. Many times the muscle becomes thinner and
the connective tissue thicker, or many times thickened and hardened. Calcifications have been observed up to two centimeters long and one millimeter broad. Such strong invasions of muscle can destroy important functions and lead to weakness and lameness, and as parasites of the heart and diaphragm may produce death of the host. Especially severe inflammatory changes occur in the neighborhood of the cyst. The high toxicity of the cyst, he thinks, is probably to blame for great emaciation of animals when many cysts are present. In 1928, Leese stated that when a muscle such as the heart becomes crammed with cysts, degeneration of the fibres results. Areas of degeneration in the heart appeared as white, oval bodies, resembling small abscesses. He thinks that the disease, when extensive, may help to cause weakness and emaciation in old camels. Tiegs (1931) in describing a sarcocyst from the python says that the parasite completely destroys that part of the cell it occupies but has no effect on adjacent fibers. Urizar (1913), in cattle, found that the muscle fibers in the vicinity of sarcocysts are oedematous and hazy, and the nuclei of these fibers may increase in number and volume to such an extent as to create the impression of a leucocytic infiltration, either diffuse or localized; but in preparations he has encountered in the vicinity of cysts groups of leucocytes, polynuclear neutrophiles and fat mononuclears in groups.

Hanel (1932) described in detail a peculiar case of sarcosporidiosis in a horse which resulted in the formation of tumors of the muscle, rather than in the more common diffuse form of infection. He notes that the muscles ordinarily are injured by the extension of cysts, by the formation of new cysts, and by the formation of a toxic substance liberated during the growth of the parasite. This injury, he states is most serious when cardiac and respiratory muscles are involved.

The preceding account represents some of the more important accumulated evidence that the sarcosporidia may and do cause serious diseased conditions, which may lead to death, with or without combination with other disease-causing factors. On several occasions I have observed the pathological effects produced by heavy infestation with sarcosporidia. The carcass of the old
ewe previously mentioned as harboring an extraordinarily large number of sarcocysts, probably weighed less than 30 pounds. Great emaciation was evident. The presence of over 88,000 sarcocysts per cubic centimeter of muscle gave the flesh a pale greyish color and a pronounced anaemia was clearly indicated. Before being killed this ewe was weak and unsteady on its legs and had very little strength. Undoubtedly it would have died in a short time. No evidence of any other disease was found. The previous history of this case is unknown, except that on the small ranch from which this ewe was obtained, a small number of sheep were kept under very unsanitary conditions. While this represents an extreme case, it indicates the end result of a long-persisting heavy infestation with sarcosporidia.

In the absence of other diseases, I have never seen an old, very heavily infected ewe that was in good flesh. I have seen a lamb less than one year of age with a pretty heavy load of sarcocysts (5,387 sarcocysts per c.cm.) that was in very good condition. Perhaps this lamb would have been fatter if relieved of this load of parasites. However, a mass infection with sarcocysts, if continued year after year, undoubtedly causes great destruction of muscle fibers, and consequent emaciation, anaemia, and other concomitant results as noted above. Some of these destructive disturbances are undoubtedly caused by growth and mechanical pressure of the sarcocysts, but some of the effects are believed to be due to the toxin produced.

Other evidence of the pathology of *S. tenella* has been observed. Two ewes of uncertain age were killed, one on July 16, and the other July 21, 1932. Balbiania could not be found, but both ewes showed interesting, pathological lesions in the lining of the heart. The endocardium of the left ventricle in particular, appeared splotched with hemmorhagic, inflammatory patches extending into the muscle, which upon examination proved to be heavily infested with sarcocysts. Nearly the entire surface was affected and this condition was most severe on the *musculi papillares* and the ventricular septum. A study of sections from one ewe revealed congestion and hemorrhages in the muscle, at some places extending from the endothelium into the muscle for a distance of
1.5 mm. Hemorrhages were most severe in sections through the *musculi papillares* and the ventricular septum. In several places there was necrosis of muscle fibers, but in no place was this extensive. In one small area an infiltration of leucocytes among the muscle fibers had occurred. A considerable number of leucocytes were found over the *musculi papillares*, underneath the endocardium. Sarcocysts were found throughout the muscle tissue, but not sufficient in number to indicate an especially heavy infection. However, the more heavily affected areas were near where pronounced lesions were present. An apparently young sarcocyst consisting of two cells was found, and several mature sarcocysts were found in Purkinje fibers located between the endocardium and the muscle tissue. An examination of three blood smears from this ewe failed to show any spores of *S. tenella*. The blood smears gave evidence of a moderate eosinophilia, which may be interpreted as a reaction to a parasitic toxin. Since these organs were normal, since no local infections or other parasites were found in this ewe, and since it is known that sarcosporidia do produce a toxin, it appears reasonable to believe that *Sarcocystis tenella* was responsible for the conditions found. The microscopic lesions of both ewes were identical, and a detailed study of sections from the second ewe was not made. Such conditions as were found undoubtedly affect the function of the heart muscle, and these observations furnish additional evidence of the pathological effects produced by *S. tenella*. As our knowledge of the sarcosporidia increases, we are more certain of the obscure and insidious, but nevertheless serious effects produced.

**EFFECTS ON OTHER TISSUES**

Besides the direct and indirect destructive effects on the muscle fibers which harbor the parasites, other tissues may experience harmful effects. Kitt (1900) states that sarcosporidiosis sometimes produces a hyaline and fatty degeneration and interstitial myositis and knot-like formations, with consequent weakness and lameness. In the first stage of myositis, there is an overwhelming cell infiltration, and in the second stage a connective tissue increase, a destruction of muscle fibers, calcification, and
changed appearance and function. Motas (1906) stated that the sarcocysts give origin to lesions of connective tissue analogous to those in muscular tissue which, although numerous, do not manifest themselves during life. Besnoit and Robin (1914) expressed the opinion that sarcosporidiosis of the skin corresponds to the habitual form of elephantiasis of older authors, but we have found no confirmation of this view. Schlegel (1918) found the neck ligaments of an ox, two and one-half years old, were grass-green throughout, and attributed this to Miescher's cysts. Adjoining muscles were pale reddish and strongly moisturized with serous fluid. Between the stretched connective tissue fibers were small spindle shaped bodies, which formed inflamed centers, and most masses consisted of leucocytes, fibroblasts and epithelial cells. Some centers showed an erythrocyte infiltration, other centers were more or less entirely calcified; still others showed clearly the structure of Miescher's cysts with a cheesy consistency. Sections from neighboring muscles showed that it was fairly well colonized with *S. Miescheriana*. Wroblewski (1923) concluded that the adult cysts of *S. tenella*, as long as they are in the muscles, do not do as much harm as the young stages in the blood; that the latter can produce a severe epizootic, with picture of a severe anaemia, and a large per cent of fatal cases.

Hasselman (1924) observed centers of cellular reaction in cattle, in the interstitial tissue of the myocardium, even to a distance of three fields of view of the microscope (low power). Urizar (1931) stated that he had seen no leucocytic infiltration, either diffuse or localized, in the vicinity of the cysts, but in preparations has encountered near the cysts groups of leucocytic poly- nuclears, neutrophiles and fat mononuclears. Babulieri (1932) sums up his conclusions by saying that infections usually pass unnoticed but may lead to degenerative changes, granulomas, muscular paralysis, abnormal secretions in intestinal infection, etc. He is here evidently considering *Sarcosporidia* in the broader sense including *Globidium*. Salomon (1935), in a study of *S. tenella* in the sheep, found that haemotoxylin-eosin staining revealed strong infiltrations of round cells including eosinophile leucocytes. He regarded the presence of numerous sarcocysts in the muscle
as the cause of this condition. Gilmore, Kean and Posey (1942) reported the twelfth human case of sarcosporidiosis, the fourth with cardiac involvement, and the second case for the Isthmus of Panama. Koegel (1926), in a report to farmers in regard to important diseases of useful animals, produced by Protozoa, stated that especially severe inflammatory changes are produced in the neighborhood of the cyst, if the contents flow out in these places. The contents of the cysts possesses a high toxicity, and he thinks that this toxicity is probably to blame for the great emaciation of animals with many cysts. Our own observations indicate that great emaciation is at least partly due to the destruction of muscle fibers by invasion of the parasites in enormous numbers.

From this brief account, it is clear that extensive invasion of the muscle fibers involves not only destructive effects on muscle fibers, but also involves serious pathological changes in other tissues.

SARCOCYSTIN, THE TOXIN OF SARCOSPORIDIA

The study of the toxic effects produced by sarcosporidia has received a great deal of attention. Pfeiffer (1890) showed that injection of an emulsion of *S. tenella* in sheep serum produced symptoms of illness in mice, rabbits, and sheep. Laveran and Mesnil (1899b) following the lead of Pfeiffer, showed that the sarcosporidia of sheep contain a powerful poison which they named "sarcocystin." They used both fresh and dried sarcocysts; they put the water extract through a porcelain filter and the glycerine extract through a paper filter. Rieval and Behrens (1903a) studied the sarcosporidia in the llama, and confirmed Laveran and Mesnil in regard to the properties of the poison which they named "sarcosporodin." They concluded that the poison did not have the chemical and physiological properties of a toxin, but stood nearer the enzymes; that it was a neuro-toxin, as it had a specific effect on the central nervous system, producing lameness. Weber (1909) agreed with Rieval and Behrens that the sarcocyst does not secrete toxin; that at most it encloses enzymes whose action is only manifest when the cuticle of the parasite is ruptured.
Teichmann and Braun (1911b) undertook to test whether the toxin of *S. tenella* is a true poison. They dried and pulverized the sarcocysts, dissolved this material in a salt solution, and injected the fluid either subcutaneously or intravenously. They concluded that *S. tenella* contains a true toxin, "sarcosporidotoxin." Knebel (1912) saw cysts up to the size of a pea, which were completely free from bacteria. Using the method of Teichmann and Braun, he proved by experimental inoculation that such cysts contained a toxin which he regarded as more than the primary protozoan toxin. Cominotti (1913) concluded after a series of experiments that *S. tenella* contains a poisonous substance, "sarcosporodin," which in rabbits and sparrows produces a significant toxic action. Mesnil, Chatton and Perard (1913) showed for the first time that *S. miescheriana* of pork is toxic for rabbits, but not for the guinea pig, rat, or mouse.

Various methods have been used in preparing the toxin. This explains in part the different results obtained. Rieval and Behrens emulsified the sarcocysts in physiological salt solution. The method of preparation used by Teichmann and Braun has been given. Sabrazes and Muratet (1911), working with *S. bertrami* from the horse, dissected out cysts from the oesophagus, and boiled the cysts until the walls broke and the contents escaped. By proceeding thus, aseptically in a closed vessel, there was obtained a pulp deprived of all microbes. Remaining in glycerine did not modify the toxicity. A small dose from 10 cysts produced morbid conditions; a large dose of 100 cysts produced a cholera-like diarrhoea and death. The grade of toxicity was measured by the number of sarcocysts used. Cominotti (1913) following the general procedure of Teichmann and Braun, dried the contents of cysts by vacuum desiccation for 24 hours; the dried material was pulverized in sterilized mortars, and the toxicity was determined or measured by weight. For injection, he dissolved the dried substance in normal salt solution.

Sato (1926, 1927) working on *S. blanchardi* of cattle, dried the mature cysts in a vacuum, pulverized the dried material, and prepared water, normal saline, and glycerine extracts; the solutions were shaken for two or three hours and then centrifuged;
the clear fluid contained the sarcosporidiotoxin. The fluid extract contained besides the toxic principle, an antagonistic acting substance that gave an albumin reaction. The toxicity was greatly reduced by filtration. Arrue (1927) recommends a method for the study of sarcosporidia proposed by Cesario. This consists of finely mincing the flesh and mixing with it a quantity of salt, which by its hygroscopic action attracts the sporozoites. After setting aside for an hour, the material in centrifuged; the spores are said to unite in a clot, which is separated from the excess of salt by washing with physiological solution, followed by a new centrifuging. He states that this method is convenient for the preparation of sarcocystin from S. tenella. He made only two tests of material prepared in this way, and the value of this method is yet undetermined.

The nature and properties of the toxin that is now generally referred to as "sarcocystin," has received the attention of a number of investigators in this field. It will serve our purpose to refer to some of the most important work that has been done. Rieval and Behrens (1903a) emulsified sarcocysts from the llama in physiological salt solution, and found that a subcutaneous injection of 2 cc. of this solution into a rabbit caused death in seven hours; also, that injection of 1 cc. of the solution, and feeding an additional amount caused death in eight hours. Sub-injections from these rabbits into other rabbits showed that death was not due to bacteria. The poisonous effect on the rabbit was not due to the flesh of the llama, as was shown by other inoculations. These authors confirmed Laveran and Mesnil in regard to the properties of the toxin, and concluded that it was a neuro-toxin, as it appeared to have a specific effect on the central nervous system. Teichmann (1911b) states that the sheep sarcocyst contains a powerful toxin that has the following properties: Thermolabile, filterable, soluble in physiological salt solution, and toxic only for the rabbit, whose natural immunity to the antitoxin does not rest upon contents of its serum. Other observations indicating nature and properties of sarcocystin will appear in later references, as indicated by effects produced and experimental results.
Erdmann (1910a) mentioned the severe toxic effect of sarcocystin, causing the destruction of epithelium, following the feeding of _S. muris_ to mice. Teichmann (1910) and Teichmann and Braun (1911) investigated the poisonous action of the sheep sarcocyst. They found that _S. tenella_ contains a poison for rabbits, with a lethal dose of 0.0002 gm. of dried material. The poison was found localized in the central nervous system, bound up with the lipoids, and could be retrieved from the brain by means of ether, water, or alkaline alcohol. The extract from the sheep sarcocyst produced an agglomeration of the blood cells of sheep, guinea pig, man, horse and pigeon, but not of the rabbit, but the stuff that produces this agglomeration is not identical with the toxin. Cominotti (1913), who found that _S. tenella_ contains a substance highly poisonous to rabbits and sparrows, describes in some detail the effects produced. The animal begins to show the effects by appearing stupid; at first localized and then general paralytic symptoms appear; a rise in temperature occurs accompanied with diarrhoea; paralytic symptoms cease suddenly and animals later die in from one to three hours. A sparrow was killed by 0.00005 gram; a guinea pig was not harmed by 0.01 gram; in sheep 0.001 gram produced a temperature of 40.9 degrees Centigrade, but only indifferent loss of appetite. A second injection produced a rise in temperature.

Sato (1927), working with _S. blanchardi_ from the ox, used water, normal saline, and glycerine extracts of dried cysts and cyst contents. He found that the toxin is inert when swallowed or injected into the bronchi; that the effect is weak when injected into the brain, and weaker still when injected into the peritoneum, but very active in susceptible animals when injected into the blood. The rabbit is particularly susceptible, the m.l.d. _intra venis_ dose being 0.00005 grams per kilo body weight. Toxic symptoms were excitement, paralytic collapse, severe diarrhoea in about half an hour, and death in about ten hours. This author enumerates the pathological changes in detail and infers that the toxic principle acts upon the adrenal glands, liver, intestinal walls and heart.

The toxic effects of sarcocystin can be destroyed in a number of ways. Laveran and Mesnil (1899b) stated that water extract
of the toxin heated to 100 degrees Centigrade for five minutes lost its effect; that heating to 85 degrees C. for 20 minutes had a similar effect on the toxin. Activity of the poison diminished when heated from 55-57 degrees C. for two hours. The glycerine extract was more resistant to heating. Teichman (1910) found that the toxin loses its effect when heated to 100 degrees C.; and that the toxicity is reduced by union with lecithin. Cominotti (1913) reported that the poison is destroyed at 58 degrees C. in 10 minutes, at 70 degrees in five minutes, and is killed very quickly when heated to 100 degrees C.; that it is not destroyed by drying for five months. Sato (1927) claimed that the toxic principle was destroyed by heating to 50 degrees C. for 30 minutes, by carbon dioxide, and by acids, but is resistant to alkalis.

EXPERIMENTAL RESULTS

Some of the properties and characteristics of arcocystin are further elucidated by experimental results on a considerable number of animals and birds. Pfeiffer (1890) found that an injection of an emulsion of S. tenella in sheep serum produced symptoms of illness in mice, rabbits and sheep, which in some cases led to death. Laveran and Mesnil (1899b) reported that rats, mice, sheep, frogs, and turtles did not react, but that dogs and pigeons lost weight after a strong dose. Cominotti (1913) made use of goats, sheep, rabbits, guinea pigs, mice and sparrows in his experiments. He found by repeated injections of extracts of S. tenella it was possible to produce anaphylactic shock in guinea pigs which are especially resistant to the toxic action of the sarcosporidia. A significant toxic action was produced in rabbits and sparrows, but on the other animals mentioned, the effect was only slight or even unnoticeable. Sato (1926) stated that young guinea pigs, mice and canary birds showed certain susceptibility.

The writer (1926) found that feeding muscle heavily infested with S. tenella had a severe toxic effect on the lining of the intestine of young white rats. Large sarcocysts from the oesophagus of sheep (Balbiania) were fed to a number of young rats for the purpose of demonstrating intestinal stages of these parasites. The spores failed to develop and the results were negative, so far as
the life history was concerned. However, the effect on the young rats was significant. After being fed from two to seven Balbiania, a healthy young rat that previously has fasted 15 to 24 hours, soon loses interest in food or in its surroundings, and appears ill; it hunches up and is disinclined to move about. Killing such rats at intervals from 1\(\frac{1}{4}\) to 28 hours after feeding showed the fate and progress of the spores along the alimentary canal. For example, in rat 56, which was killed two hours after it was fed five Balbiania, the empty spore cases were found from 19 to 23 inches below the stomach. During the progress of the spores, digestion apparently took place, and the sarcocystin was set free. Extensive cytolytic effects were found, causing destruction of the epithelium and villi. In regions of the toxic effects, the contents of the intestine consisted of a bloody serous exudate, which distended the intestine and included cell debris, spores, bloody serum, etc. The jejunum was empty and practically free from any cytolytic lesions. From these experiments it was clear that mass infection with *S. tenella* causes extensive destruction of the epithelium and villi in certain regions of the intestine, and that this powerful toxic effect is caused by sarcocystin. Similar results were found after mass feeding of Balbiania to mice. Laveran and Mesnil, Chatton and Perard, have each experimented with rats, but reported no reaction; probably the doses administered did not approach the mass feeding with Balbiania used in our experiments.

In 1926, two lambs were fed Balbiania, with the idea of obtaining intestinal stages of *S. tenella*. The spores set free by digestion did not develop. No destructive or cytolytic effects were noted on the epithelium. Cominotti (1913), Stackman (1926) and several other workers have tried the effects of sarcocystin on sheep and goats without serious result.

Among birds, sparrows (Cominotti) and canaries (Sato) have been proved to be susceptible to the poison, and the hen, pigeon and dove (Teichmann) immune. Among other animals, Reick (1888) showed that dogs were not harmed when injected subcutaneously with sarcocysts from the horse or sheep. Laveran and Mesnil (1899b) showed that 0.01 milligram of dried extract of the sarcosporidia of sheep was fatal for rabbits; that whether
a water or glycerine extract was used, 0.01 gm. of the pulverized and 0.05 gm. of the fresh material were equally potent. Cominotti (1913) reported the minimum lethal dose of *S. tenella* extracts for rabbits is 0.0001 gram, Chiwy and Colback (1926) found that 0.1 milligram of dried extract of the sarcosporidia of cattle was enough to kill a rabbit. Darling (1910b) made an intra-muscular inoculation of fresh sporozoites from the opossum into a guinea pig; the development of the spores was not typical and produced sporozoa not unlike those found in a guinea pig after feeding with sporozoites of *S. muris* from the rat. Cominotti found that by repeated injections it was possible to produce anaphylactic shock in guinea pigs, which are especially resistant to the toxic action of sarcosporidia. Sato likewise found that young guinea pigs showed certain susceptibility. Stockman (1926) found that inoculation with sarcocystin from sheep, which produced death in rabbits, had no effect on sheep. Arrue (1927) made sub-cutaneous injections of large quantities of *S. tenella* into rabbits with the result that they revealed only a light and passing restlessness. Nakanishi (1929) found that a one per cent glucose or a normal saline extract, was toxic for the rabbit but not for a calf when injected into the jugular vein.

An immunity to the toxin can be developed in susceptible animals. Rieval and Behrens (1903) stated that it was possible to immunize the rabbit against this poison. Teichmann (1910) also reported that rabbits could be immunized against the poison and Teichmann and Braun (1911b) added the further information that the toxin produces an anti-toxin in the rabbit organism, which may be immunized and this immunity may be passively transmitted; the immune serum contains a complement-binding antibody against the sarcosporidian extract. They further reported that animals immune to the toxin include guinea pigs, rats, mice, sheep, dog, hen, dove, frog, turtle, paramoecium, canary, cat and ape. Cominotti (1913) makes the statement that it is possible to attain active immunization in rabbits through injection of progressive doses of sarcosporidial dry substance. By treatment with a series of intra-venous injections of solutions of sarcosporidial dried substance, there is produced finally an immunization. Sato
(1926) found that rabbits can be highly immunized by repeated injections of the toxin, and in 1927 reported that rabbits can be rendered highly immune in the usual manner, but the immune serum does not always neutralize the toxin in vivo though it does in vitro.

Considerable differences will be noticed in the experimental results recorded above. These differences are not necessarily contradictory, and are probably due to the methods of preparing the toxin, the size of the dose administered, the purity or impurity of the extract, the method of administering the toxin, and perhaps other factors.

LOSSES FROM SARCOSPORIDIOSIS

Losses due to infestation with sarcosporidia are both direct and indirect. Since the parasite lives within a muscle fiber and may destroy the fiber by growth and pressure, it is evident that a heavy infection will sooner or later destroy a large per cent of the muscle fibers, and so interfere with the normal functioning of the muscle. This sort of destructive effect will be in proportion to the number of muscle fibers invaded. An extreme case is afforded by the ewe already referred to, in which there were more than 88,000 sarcocysts per cubic centimeter of flesh. This animal was so emaciated, and the voluntary muscles so reduced that it might be designated as consisting of "nothing but skin and bones." In the absence of any other recognized disease, this animal probably represented a direct loss due to sarcosporidiosis. It is also true that as the degree of infestation increases the amount of toxin produced likewise increases, and both effects are probably cumulative. Besides our own work, the literature is replete with records of losses attributed to sarcosporidial infection.

Leiserung and Winkler (1866a) noted that in a flock of sheep that died suddenly, large cysts from the size of peas to hazelnuts were found on the oesophagus. In the light of our present knowledge of sarcosporidia, these parasites may have been instrumental in bringing about an unhealthy condition, but death could hardly be attributed to this cause. Dammann (1867) described the illness and death of a nine-year-old ewe. He found large cysts in the
muscles of the palate, pharynx and glottis, which apparently interfered with breathing and produced some inflammation. Death was explained as due to oedema of the glottis, which developed in consequence of inflammation of the pharynx and in consequence of cysts in the roof of the palate. He found no other cause of death, and came to the conclusion that sarcosporidia were responsible. Likewise von Niederhausen (1873a) observed the death of an old goat heavily infested with sarcocysts which he believed to be the cause of death. Brooks (1903) reported the case of an elk that had died from Miescher's cysts in the heart muscle. He found that a large part of the elk herd at the New York Zoological Park had been infected. “Eventually it caused death in these animals though it is generally supposed to be entirely innocent. I have every reason to believe that the disease is extremely rare in wild animals in the open while it is frequent in members of the deer family in captivity. * * * * In our animals—elk, caribou and native deer—contrary to the general rule as stated by most veterinary and medical authorities, the infection invades the heart muscles in most cases, and it is through the invasion of this viscus that it causes the death of the animal.” The close confinement of these wild animals, within a restricted area, thus permitting opportunity for almost continuous and cumulative infection, and re-infection, no doubt accounts for the fact that these animals were overrun with the parasites, while the same species under wild conditions are relatively free from sarcosporidia.

Moussu and Coquet (1909) described the case of a young four-year-old horse, in good condition and health until February, 1907, when a small swelling appeared in the neighborhood of the wethers; soon other swellings appeared on the neck, chest, belly, etc., and movement became difficult. In September the horse became worse and fed with difficulty. The tongue became hard, stiff, increased in size, rigid and immovable, resembling actinomycosis of the ox. The animal appeared as if suffering with rheumatism, with stiff limbs, flexions incomplete, steps short, temperature normal, appetite good, respiration and pulse regular, contraction of heart soft, but it did not appear to suffer pain. Prehension, mastication and swallowing were difficult, though
there was a continual flow of saliva. The condition was diagnosed as due to sarcosporidia, since the muscles were ridged with calcareous grains which were in reality parasites which had undergone calcereous infiltration. At autopsy, it was found that in general the superficial muscles were attacked. Some muscles had undergone fibrous degeneration; others had calcified nodules in the midst of tissue in a state of slight myositis. Muscles more extensively attacked had a paler color. Erdmann (1914) stated that meat containing *S. tenella* does not seem injurious to man but the spores may cause a regular fever; that the sarcosporidia of domestic animals appear relatively harmless, but *S. muris* is rapidly fatal to mice.

Schneider (1917) reported the case of a two-year-old ox that first became lame in the hind quarters, and then after four days, lameness developed in the whole body. Breathing was short, sharp, and appetite was lacking. After death nothing abnormal was found in the internal organs. The musculature for the most part was flecked with a yellow-greenish color, soft and watery in consistency, and stippled with clear grey, long, pointed parasites. Microscopically, numerous sarcocysts were visible to whose presence the symptoms were attributed. Creech (1922) studied sarcosporidiosis of swine, and found that it was associated with advanced, degenerative changes in the musculature. He described cases of "soft" and "mushy" hams, and discussed the cause of degeneration and the nature of the toxin. He concluded that degenerative changes in the muscle are always associated with an abundance of sarcosporidia; that the lesions may be due to excessive numbers or to toxic products eliminated from the parasites. Hadwen (1922) found that in diseased or aged reindeer, the meat was so altered that it could not have been passed for food. The distribution of the parasites was uneven. The sarcocysts, similar to *S. tenella* were found mostly deep in the muscles over the periosteum of the flat bones, such as the scapula. Pits in both bone and tendon were produced. Owing to the granular nature of the lesions, reindeer owners call it "cornmeal disease." Hadwen named this parasite *Fibrocystis tarandi*, from the location and host in which it is found.
Piettre (1922) in a book on food inspection, stated that sarcosporidiosis entails total withdrawal from consumption in caseous or calcareous degeneration, with the same right to condemn as all other generalized suppurations in all muscular tissue. When the giant form of the parasite (Balbiania) is concerned, one takes the invaded portion (oesophagus), or the whole carcass if many groups of muscle are involved. It appears in fact, he states, that the contents may be toxic, and that the presence of sarcocysts in the flesh is displeasing. Nevertheless sterilization permits use of the meat, provided it is lightly infested. Bergeon (1924) reported that among the parasites that cause rejection of the flesh of the buffalo at Tonkin, sarcosporidia are the most common. The regions of the body where Sarcocystis bubali is found most frequently, are the tongue, neck, shoulders and thigh.

In the United States, Edelmann, Mohler, and Eichorn (1933) consider sarcosporidia as harmless parasites, and if not visible the meat may be used with impunity. However, if visible, or if present in large numbers in all the muscles, or if the meat shows greenish or yellowish spots, or if it is oedematus, the entire carcass is to be condemned. If confined to certain muscles, these are to be condemned. All muscles inhabited by Balbiania are to be condemned. Houdemer (1927) reported that sarcosporidia are extremely frequent in butchered animals in Indo-China, and provide the motive for seizing and condemning part or the entire carcass of buffaloes.

Koegel (1926) states that sarcocysts, as parasites of the heart and diaphragm, may produce death of the host. Chiwy and Colback (1928) are of the opinion that sarcosporidiosis of the heart could easily be the cause of death, which often occurs, if great marches are required of the animal in transport.

Riley (1931) is of the opinion that the edibility of ducks heavily infested with sarcocysts is not known; but their flesh would be rejected on aesthetic grounds as well as danger from toxic substances.

The fore-going account represents some of the more common ways in which direct losses from sarcosporidia may occur. There are numerous other ways in which indirect losses, which may be
combined with direct losses, are traceable to infestation with sarcosporidia. As early as 1884, Laulanie noted that the flesh is altered, not entirely useless, but loses a great part of its food properties. Putz (1887) described the case of a horse that was lame for six months in the fore limbs, especially on the right side, with movement very difficult; movement of the hind limbs was relatively free, and there was no evidence of pain. When killed, the muscles of the right side, especially of the front limb, were more heavily infected with sarcocysts and more degenerated. He attributed the symptoms and conditions found in the muscles were due to Miescher's cysts, now known as S. bertrami.

Smith (1901) noted that heavily infected mice were ill and frequently slow in movement. Ostertag (1905) described cases of sarcosporidiosis in cattle with symptoms of lameness. He noted post-mortem muscular changes and large numbers of Miescher's sacs. In one case, he observed paleness, loss of striations, and disintegration of muscle fibers. Watson (1909a), working with horses and cattle, stated that sarcocysts are an important factor in disease, invading the entire musculature of the host with serious and fatal consequences. Mason (1910) observed that sarcocysts were numerous in nearly all old, emaciated camels. In the absence of other wasting disease, when parasites were numerous, the camels appeared extremely emaciated, with inflammation of the heart and its coverings and its linings, with numerous small hemorrhages, with gelatinous degeneration of the heart, and with disintegration of voluntary muscle fibers. He concluded that in this way, sarcosporidia may set up emaciation, heart dilation, etc.

Sabrazes, Marchal, and Muratet (1910) described a case of fibrosarcoma and sarcosporidiosis in a horse. They were unable to discover sarcosporidia among the tumors, but only in their neighborhood. In conclusion, the authors refer to the chronic irritation theory of the genesis of neoplasms, and suggest that the chronic irritation induced by the sarcosporidia in this case may have been the cause of sarcomatosis, just as other irritants, traumatic, physio-chemical, biological, microbial, etc., are said to provoke neoplasms. Croveri (1920) believed that ordinarily the sarcosporidia cause no serious harm, but if there is a deficiency
of nutrition, or if either bovine pest or trypanosomiasis is present, complications arise of a serious nature and even death results. Hadwen (1922) found that reindeer, especially the older ones, are commonly infested with sarcosporidia. In diseased or aged deer, the meat is so altered that it could not be passed for food. These sarcocysts were similar to *S. tenella*.

Woolridge (1923) found that sarcocysts in the sheep may give rise to symptoms of anaemia, emaciation, general unthriftiness and sometimes difficult respiration, but in many cases no symptoms are exhibited. If localized in the oesophagus wall, tongue, pharynx, cheek, neck, abdomen, thigh and occasionally over the pleura and peritoneum, the infestation may cause difficult breathing and consequently set up emaciation. At times, he states, sarcosporidia cause dyspnoea from oedema of the glottis. Koegel (1926) thought that strong invasions of muscle can destroy important functions and so lead to weakness and lameness, and that as parasites of the heart and diaphragm, sarcocysts may produce death of their host. Chiwy and Colback (1926) raise the question whether animals with the heart infested are capable of long marches over hilly and mountainous ground or for long distances. For if very numerous in an animal, the muscle movements show harmful effects and lameness (paralysis), and when in the heart muscle, the sarcosporidia produce a kind of myocarditis or even endocarditis. In 1928, these authors state that sarcosporidiosis of the heart in cattle could easily be the cause of death, which often occurs, if great marches are required of the animals in transport. Mrowka (1925) found lambs, sheep, milk cows, calves, and young foals were affected with lameness in Peru. He mentioned very acute, acute, sub-acute, and chronic cases, and observed, “It is an interesting fact that wherever illness accompanied by lameness is found, sarcosporidia are present.” Drabble (1928) reported a case of sarcoma associated with sarcocysts of *S. tenella* in the sheep and concluded that anything that causes inflammation or chronic irritation is a potential factor in the ecology of cancer. Hence, parasitic infestation cannot logically be rejected as a potential factor in the causation of malignant growths, and therefore the association of parasites with neoplasms in the same organs.
and tissues must always be regarded with suspicion. In view of
the uncertainty about the cause of cancer, this opinion is specu-
lative.

Freund (1930) reported a case of sarcosporidiosis in a calf,
in which a dispute arose as to usability of such flesh and who
would be responsible for the purchase price. From the decision
made it was concluded in general, that in generalized sarcos-
poridiosis of the flesh, the whole body is prohibited from con-
sumption as human food, because the normal state has been lost;
also an unsavory food is furnished. The butcher, who has agreed
with the beef handler for the delivery of normal flesh, is not
obliged to accept and pay for this abnormal flesh since he cannot
again sell or work over the unfit food material, if he himself would
become liable. The seller, who has delivered a beef animal with
no marked defects, cannot collect pay since the selling transaction,
on its part, has not been fulfilled.

Human beings may also furnish evidence of serious losses.
Gilmore, Kean, and Posey (1942), after the death of an eleven-
year-old child infected with sarcosporidiosis, found cardiac involve-
ment in which a few endocardial hemmorhages were present. They
add that it is unlikely that this was the cause of death, which was
not definitely determined.

**DIAGNOSIS, SYMPTOMS, TREATMENT**

In any generalized infection like sarcosporidiosis, any symp-
toms produced, by their very nature, tend to be obscure and non-
specific in character. Diagnosis is therefore difficult and frequently
impossible in the live animal. Sarcosporidiosis, being a wasting
disease of the striated muscles, develops under conditions in which
it is frequently associated with other diseases. Hence diagnosis
becomes increasingly difficult. Mild, and usually rather heavy
infestations go entirely unperceived. Nevertheless, from the path-
ological lesions and effect produced, we know that the disease may
lead ultimately to anaemia, great emaciation and death, as well as
have other serious effects. The difficulty in recognizing the dis-
ease has been frequently mentioned in the preceding pages. A
few investigators have attempted a more definite diagnosis.
Hendrickx and Lineaux (1899) described a case of psorospermiosis in the upper lip and tongue of a four-year-old horse. Part of the tongue was extricated and disclosed the sarcocysts. They could find no other cause for the disease. A similar diagnosis of sarcosporidiosis in a young horse by Moussu and Coquot (1909) has already been described under the head of losses sustained. Cominotti (1913) states that the clinical picture, characterized by destruction of movement and lack of anatomical changes, determines the view of Prowazek, Rieval and Behrens, that sarcosporidiosis affects the central nervous system. Experiments with sarcocystin have confirmed this conclusion. Leese (1928) found that the disease when extensive may help to cause weakness and emaciation in old camels, but that diagnosis is difficult during life.

Symptoms of sarcosporidiosis frequently have been observed. Putz (1887) attributed the lameness of a horse, especially on the right side, to a heavy infection of sarcocysts in the muscles of that side. The muscles showed marked degeneration in the regions most affected. Cominotti (1913) who experimented extensively with the effects of sarcocystin, found that the symptoms of sarcosporidial poisoning appeared within four to eight hours after injection; that the clinical picture of sarcosporidial poisoning is characterized by destruction of movement, and in lack of anatomical changes, determines the view that sarcosporidia affect the central nervous system. Place (1918) reported that ewes and lambs became very weak in the hind quarters and finally died. He attributed this in part to sarcosporidia, in part to a bacterium, and in part to chemical changes in a drying food. His report is not very convincing. Chiwy and Colback (1926), working particularly on cattle, reported that the symptoms often pass unnoticed; that the animal does not appear incommmoded except when infested with a severe localization of the parasites resulting in tumors, paralysis, muscular pain, hardening of the tongue or lips, or when there is a generalized, heavy infection of the muscles.

Kinsley (1928) described in considerable detail, sarcosporidiosis in a twelve-year-old mare in foal, evidenced first in February by a swelling in the parotid region. Drooling and lack
of appetite developed and swelling gradually subsided. About two weeks later the lips and tongue were extensively swollen, interfering with prehension and mastication; in from two to three weeks, the swelling subsided leaving the tongue and lips dense and hard. The mare foaled May 1, and was well fed and had good care, but became emaciated and weak, so the colt was removed. The mare went down, unable to rise, and was destroyed. The autopsy revealed numerous white and grayish white areas in the muscles of the tongue, lips, and skeletal muscles, particularly of the front legs and thorax. The involved muscles were dense and hard. The white areas involved the muscle fibers and were identified as sarcosporidia. Kinsley states that other conditions may have been factors in this case, but the history, symptoms and autopsy findings did not reveal any other pathological entity.

Very little has been attempted in the way of cure or treatment. This can be readily understood from the difficulty in diagnosis and the obscure nature of the disease. A few references are given. Hendrickx and Lineaux (1899) administered potassium iodide to the case mentioned above and the horse recovered. Moussu and Coquot (1909) stated that treatment with iodine produced no curative effect. Mason (1910) found that treatment of camels with sodium arsenate had no effect on the cysts. Cadeac (1913), who discusses treatment of sarcosporidiosis in the cow, sheep, goat, swine, and horse reported that treatment with 15 grams of potassium iodide per day ameliorates the disease without injury. He states that other medicaments are probably inefficacious. Wooldridge (1923) suggested treatment with potassium iodide, and Babudieri (1932) states that treatment with iodine is the only therapeutic measure of any value. How valuable this treatment is has never been clearly demonstrated. In view of the amazing results claimed from medication with sulfa compounds, an experimental test should be made of their effect, if any, on sarcosporidiosis. However, due to the life history and location of these parasites, it is not likely that such treatment would produce any noticeable beneficial results.
LIMITATIONS OF THIS REPORT

The Sarcosporidia in the broad sense is treated as an independent sub-class of the Sporozoa. This group is composed of the order *Sarcosporidia*, in the restricted sense, and the order, *Globidia*. The order *Sarcosporidia* includes two families, the *Sarcocystidae*, which includes only one genus *Sarcocystis* composed of numerous species, and the *Fibrocystidae*, which includes two genera, *Fibrocystis* and *Besnoiti*, each composed of two, possibly more species. The order *Globidia* has one family, *Globididae*, which includes two genera, *Globidium* and *Ileocystis*. The *Sarcosporidia, sens. str.* includes the parasites of striated muscles, or of connective tissues related to these muscles, such as the tendons and the periosteum of the flat bones. The *Globidia* include parasites found in the mucosa or submucosa of the alimentary canal or in the skin of mammals. The spores are very similar to those found in sarcocysts, hence the supposed relationship of the two groups. Very little is known of the *Globidia* and this report deals primarily with *Sarcosporidia, sens. str.*, and more particularly with the genus *Sarcocystis*. We have made no attempt to include the *Globidia* in this account. *Globidium gilruthi* of sheep and goats was described by Chatton in 1910. Occasionally, we have found this parasite in the abomasum of Wyoming sheep. The cysts when mature rupture into the stomach and if numerous, may cause hemorrhages with serious symptoms. Trippitt (1925) found this parasite in as many as 92 per cent of British sheep.

The same acknowledgments made in Wyoming Bulletin No. 259 are due in this report.

PREVENTION AND CONTROL

In looking over the literature of sarcosporidia, one is amazed at the absence of suggestions for the prevention and control of sarcosporidiosis. With the single exception of the few suggestions made by the writer (Scott and O'Roke, 1920), we have found no direct reference to the subject. However, this might have been expected, since there was so little agreement on what were the essential facts pertaining to the life history of the group. The ranchman and the farmer are primarily interested in how to pre-
vent or control any pest that causes losses to their flocks and herds. Medication is sometimes the answer, but here is a case, where any satisfactory measures for prevention and control could not be made until we knew the path of natural infection and the essential features of the life cycle. Bulletin No. 259 (1943) by the writer not only had significant scientific interest, but by clearing up important steps in the life history and by controlled experiments, laid the ground work for satisfactory prevention and control, based on scientific knowledge.

A long series of infection and grazing experiments has demonstrated that there are no practical methods by which we can altogether prevent infection with *Sarcocystis tenella*. There is good reason to believe that mild or moderate infections cause no great harm. On the other hand, our own observations and the references cited in preceding pages offer abundant proof that heavy infections are of significant importance and serious consequence. Now that the type of life history is fairly well known, and that much is known about the pathological effects produced by heavy infections, some practices are indicated by which heavy infections can be prevented and the sarcosporidia kept under control.

The following suggestions refer particularly to Wyoming conditions, but for the most part may be applied elsewhere:

(1) *First is sanitation.* Prevent as far as possible contamination of food with feces. Confinement to small areas or crowding together while grazing, increases the chances of ingesting contaminated hay or grass. The practice of preventing fecal contamination of food is also valuable in the control of other parasites and parasitic diseases.

(2) *Restricted grazing should be avoided.* Large flocks when first turned out of the corral or when first started out from their bedding ground in the morning, should be hurried along and quickly scattered out, as this will lessen the chances of infection. If crowded together for any length of time, more of the grass will be contaminated with feces, and the grass will be cropped closer to the ground.
(3) Feeding from the ground other than grazing should be avoided during the infective season. This applies particularly to the time of year during which infection occurs, that is, during the spring and summer months. In winter, on the Laramie Plains, infection seldom, if ever occurs. This may be due to the arid conditions, the presumed detrimental effect of sunlight on the spores, or possibly it may be related to temperature.

(4) Since moist or wet pastures tend to favor infection, it is suggested that, when a flock is in care of a herdsman, it be kept on dry pasture early in the morning; later, when feces are less plentiful, the flock may be taken to lower ground with less danger of infection.

(5) Whatever the type of animal under control, avoid any unsanitary conditions that permit continuous, frequent, or repeated contamination of food and drink by means of feces. If confined within stables or pens, construct the mangers or feed racks in such a way that fecal contamination is reduced to a minimum.

SUMMARY

1. The Sarcosporidia treated in this bulletin are those Sporozoa that are found in the striated muscle fibers, and in the connective tissue attached or otherwise related to these muscles. They belong chiefly to the genus Sarcocystis; a few species belong to the genera, Fibrocystis and Besnoiti. Besides occasionally infesting human hosts, they have many economic relations, both direct and indirect.

2. The Sarcosporidia are known to infest a large number of mammals, many birds, and a few reptiles, including many of our domestic species, a considerable number of which are used as food for human consumption.

3. As a group they have a world wide distribution, dependent upon the distribution of their hosts. This includes every continent and arctic as well as temperate and tropical zones.

4. Sarcocysts have been found in all striated or voluntary and heart muscles of the body, and in connective tissue related to
these muscles. They appear to have a predilection for the muscles of the oesophagus and the heart, but other muscles may be infested with equal abundance. They may be present in large numbers in the striated muscles of the oesophagus, but entirely absent in the smooth, involuntary muscles in the wall of the stomach less than two inches away.

5. Species of the genus Sarcocystis may infest at times as high as 100 per cent of sheep, oxen, horses and swine. The number of sarcocysts present may vary from a few to an enormous number per cubic centimeter of flesh. The severity of infection is largely dependent on the number and size of the sarcocysts present. The incidence of infection depends upon climate, season, restrictions in grazing, and other factors.

6. Seasonal infection, including late spring, summer and early fall months, has been reported from South Africa, Sweden and Wyoming. While infection has occasionally been noted at other seasons, the months indicated appear to afford the most favorable conditions for fecal contamination of food.

7. Light or moderate infections seldom produce any noticeable effects. Because of the generalized nature of sarcosporidiosis, the pathological effects in heavy infections tend to be obscure and are not often observed.

8. The sarcocyst in a muscle fiber destroys that part of the fiber which it occupies, and through growth pressure may interfere with the functioning of other adjacent muscle cells. In very heavy infections, atrophy of the muscle fibers occurs, calcifications may develop, and great emaciation may result in weakness and lameness, especially in old animals. Death may result from heavy infestations of the heart and diaphragm.

9. Large sarcocysts, such as Balbiania, after atrophy of adjacent muscle cells, become enclosed in connective tissue and frequently exert pressure on other tissues. Under certain conditions the parasites set free a toxin which may give rise to inflammation, hemorrhages, and other serious pathological effects.
10. The sarcosporidia of sheep and other animals contain a powerful toxin, which has been named “sarcocystin.” This poison is highly toxic for rabbits, mice and sparrows, and probably less toxic for rats, sheep, and some other animals. Sarcocystin is a neuro-toxin as indicated by its effect on the nervous system.

11. The administration of the toxin of sarcosporidia in various ways, to numerous kinds of experimental animals, has proved that sarcocystin has a highly toxic action, frequently producing death.

12. An immunity can be produced in susceptible animals, by repeated injections of the toxin. This immunity may be passively transmitted.

13. The losses due to sarcosporidia are both direct and indirect. These losses vary from the unnoticed atrophy of a few muscle fibers to great emaciation, or lameness, paralysis and death. Between these two extremes, the losses due to the pathological effects of the parasites on muscles and other tissues are undoubtedly far greater than usually suspected. It is the writer’s opinion that the Sarcosporidia are of far greater economic importance than is usually supposed.

14. The generalized character of sarcosporidiosis makes it difficult to diagnose in the living animal, and the symptoms are likewise usually vague. In the absence of other diseases, the symptoms include lameness, weakness, anaemia, great emaciation, paralysis and death. Biopsy or post-mortem examinations are required to complete the diagnosis.

15. All treatments so far tried are probably of little, if any value in curing or alleviating the disease.

16. Suggestions for prevention and control are included. These center around keeping the food and drink from being contaminated with feces of infected animals. A study of the life history has shown that the feces of infected animals contain an infective stage of the parasite.
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(See Wyoming Station Bulletin No. 259 for additional references not found in this list)


May, 1943  *Economic Importance of Sarcosporidia*  49


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May, 1943  Economic Importance of Sarcosporidia  53


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**ANNUAL REPORTS—**
20th to 52d, inclusive (1909-10 to 1941-42, inclusive, except 21st, 22d, 27th and 33d.)

**INDEX BULLETINS—**
G, H, and I.

No. **STATE FARMS BULLETINS—**

No. **CIRCULARS—**

No. **BULLETINS—**
112. The Poisonous Properties of the Two-Grooved Milk Vetch (*Astragalus bisulcatus*).
116. Winter Grains.
163. Results with Tree Planting at the Sheridan Field Station.
185. Barley Tests at the Sheridan Field Station.
205. Economic Studies of Irrigated Farms in Big Horn County.
209. Forty Years of Weather Records.
212. Steer Feeding in Southeastern Wyoming.
220. Study of Psyllid Yellows in Wyoming.
221. Occurrence of Selenium and Seleniferous Vegetation in Wyoming.
223. Corn Production on the Campbell County Experiment Farm.
227. Sugar Beet Tops, Cottonseed Cake and Mono-Calcium Phosphate in Rations for Steers.
228. Type of Farming and Ranching Areas in Wyoming.
229. Vegetative Composition, Density, Carrying Capacity and Grazing Land Values in the Red Desert Area.
231. Poisonous Plants and Livestock Poisoning.
232. Breastbones of Turkeys.
May, 1943  
Economic Importance of Sarcosporidia  

234. Cellar Wintering of Bees.
237. Roughage Feeding of Dairy Cattle.
238. Wintering Bees in Wyoming.
239. The Two-Queen Hive and Commercial Honey Production.
240. Salinity Conditions in the Big Horn River During the Years 1938 and 1939.
241. Livestock Poisoning by Oat Hay and Other Plants Containing Nitrate.
243. Practical Results from the State Experiment Farms.
244. Bacterial Ring-Rot of Potatoes.
245. Sulphur Dusting for the Control of Psyllid Yellows of Potatoes.
246. Hybrid Corn Adaptation Trials in Wyoming, 1940.
248. Influence of Cereal Grains Upon Quality of Meat in Turkeys.
249. Coccidia Infesting the Rocky Mountain Bighorn Sheep in Wyoming.
250. Vegetable Culture and Varieties for Wyoming.
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253. Range Forage Production in Relation to Time and Frequency of Harvesting.
254. Crossbreeding for Lamb and Wool Production.
255. Lungworms of Domestic Sheep and Bighorn Sheep in Wyoming.
256. The Use of Wheat in Livestock Feeding.
257. Utilizing Self-Feeding Methods for Fattening Lambs on Sugar Beet By-Products and Other Home-Grown Feeds.
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