

Sarcocystosis

*Sarcosporidiosis,
Equine protozoal
myeloencephalitis*

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Etiology

Sarcocystosis is caused by species of *Sarcocystis*, an intracellular protozoan parasite in the phylum Apicomplexa. These parasites have an indirect life cycle, cycling between a definitive and an intermediate host. Intestinal infections occur in the definitive host, and tissue invasion is seen in the intermediate host. More than a hundred species of *Sarcocystis* are parasites of domestic and wild animals. Many of these infections are asymptomatic, particularly in the definitive host.

Equine protozoal myeloencephalitis is usually caused by *Sarcocystis neurona*. *Neospora caninum* and/or *Neospora hughesi*, not discussed in this outline, have also been implicated in some cases. Symptomatic infections caused by *Sarcocystis* species are reported occasionally in other domestic animals.

Humans are a definitive host for *S. suis* found in pork, and *S. hominis*, found in beef. These parasites infect the intestines. Humans can be intermediate hosts for a variety of other *Sarcocystis* species. The parasites are found in the muscles. The species of *Sarcocystis* involved is, in many cases, unknown.

Geographic Distribution

Sarcocystis spp. occur worldwide, but individual species may be found in specific geographic regions. *S. neurona* is endemic only in North, Central and South America. Human intestinal sarcocystosis has been reported worldwide. Symptomatic cases of human muscle sarcocystosis have been seen mainly in Southeast Asia, probably due to the distribution of a definitive host. *Sarcocystis* spp. have also been found in human muscle tissues in the U.S., often as an incidental finding at autopsy.

Transmission and Life Cycle

Sarcocystis spp. have an indirect life cycle, and must develop in both an intermediate and a definitive host. In many cases, a *Sarcocystis* species cycles through a specific predator or scavenger, and its prey. Most species have a single intermediate host; however, *S. neurona* is less host-specific.

The definitive host becomes infected when it ingests encysted parasites (sarcocysts) in muscle tissues. Sarcocysts are oval, whitish cysts that vary in size from microscopic to visible. They are filled with hundreds to thousands of bradyzoites. The bradyzoites are released in the intestine of the definitive host, where they enter the lamina propria and immediately undergo gametogony to form oocysts. There is no asexual replication in the definitive host. The oocysts mature in the host's cells, then are shed in the feces. These oocysts already contain two sporocysts, each with four sporozoites. The oocysts may disintegrate, and sporocysts may be found in the feces.

Intermediate hosts become infected when they ingest oocysts or sporocysts. The sporozoites are released in the intestines and cross into the bloodstream. In many cases, they multiply asexually in the walls of small blood vessels before invading the skeletal or cardiac muscles or neural tissues, where they form the sarcocyst wall and multiply as merozoites for several generations. The merozoites eventually develop into bradyzoites within the sarcocysts. Only the bradyzoite stage is infectious.

Birds may act as transport hosts for *S. neurona*. Birds fed infective feces from opossums, the definitive host, shed infective sarcocysts in the feces. Insects such as flies and cockroaches may also be vectors for this organism.

Disinfection

Sarcocysts in pork can be destroyed by cooking at 70°C for 15 minutes, freezing at -4°C for 2 days, or freezing at -20°C for 1 day. Heating to more than 60°C for 1 minute is reported to destroy *S. neurona*. A recent report suggests that commonly used disinfectants in veterinary hospitals may not be able to kill *S. neurona*.



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Infections in Humans

Incubation Period

In human volunteers, the clinical signs occurred as soon as 3 to 6 hours in the intestinal form, then recurred 14 to 18 days later. The incubation period for the muscle form is unknown.

Clinical Signs

Myositis

If humans act as the intermediate host, myositis is the primary syndrome. The spectrum of illness ranges from acute self-limited infections to chronic, moderately severe disease. Painful muscle swelling has been reported, accompanied by erythema, muscle tenderness, generalized muscle weakness and fever. Bronchospasm has also been seen. Other reported symptoms include cough, arthralgia, transient pruritic rashes, headache, malaise, lymphadenopathy and muscle wasting. Chronic cases can have persistent or recurrent symptoms for up to seven years. Many infections may be asymptomatic.

Intestinal sarcocystosis

When humans serve as the definitive host (*S. suis hominis* or *S. hominis*), the clinical signs may include fever, chills, sweating, diffuse abdominal tenderness, diarrhea, nausea and vomiting. Dehydration may occur as a result of the diarrhea and vomiting. Eosinophilic enteritis and rare cases of acute intestinal obstruction have been reported. Intestinal sarcocystosis is transient and usually self-limited; chronic enteritis has not been described. Many or most cases are thought to be asymptomatic.

Communicability

Humans infected with *S. hominis* or *S. suis hominis* can transmit the infection to cattle or pigs, respectively, by sarcocysts shed in the feces. Shedding begins after 10 to 13 days and can continue for up to six months. Humans serving as the intermediate host cannot spread the infection to others.

Diagnostic Tests

Intestinal infections can be diagnosed by detecting sporulated sporocysts in the feces, using zinc sulfate flotation. Sarcocysts may be found in the muscles by microscopic examination of a muscle biopsy. A CT scan or MRI can sometimes visualize sarcocysts in the muscles. A complete blood count, to reveal eosinophilia, may also be helpful.

Serologic tests include an indirect immunofluorescent antibody test and immunoblotting. Immunoblotting may be useful for muscle infections, but is not considered to be valuable for intestinal infections. Serologic tests may not be widely available.

Treatment

Intestinal sarcocystosis may not be treated, as the typical infection is self-limiting. Symptomatic myositis may be treated with antiparasitic agents such as metronidazole, cotrimoxazole and albendazole. Corticosteroids may be used to reduce inflammation. Asymptomatic cases may not require treatment.

Prevention

To prevent intestinal sarcocystosis, people should avoid eating raw or undercooked beef. Freezing the meat to -20°C can also help prevent transmission.

To prevent muscle sarcocystosis, food contaminated by feces or dirt should be avoided. Good personal hygiene, such as hand washing, may also help to prevent transmission. The definitive host is not known in many cases, particularly those that occur in Southeast Asia.

Morbidity and Mortality

Worldwide, the incidence of intestinal sarcocystosis is estimated to be 6-10%. This disease is more prevalent in cultures where raw meat is commonly eaten. Intestinal sarcocystosis is usually self-limited and transient, and complications are uncommon. Many cases may be asymptomatic. Chronic enteritis has not been described.

Myositis has rarely been described in humans, and asymptomatic muscle infections may go undetected. Approximately 20% of people in Malaysia have antibodies to *Sarcocystis* and, throughout Southeast Asia, 21% of autopsy specimens contain the parasite. More than 60 cases have also been reported in the U.S., often as an incidental finding. Many cases appear to be self-limiting and the prognosis is usually excellent. Chronic or recurring infections have, however, been reported; in one case, the symptoms persisted for seven years.

Infections in Animals

Species Affected

Definitive hosts

The definitive hosts are carnivores, including dogs, cats, mammalian wildlife, birds, reptiles and humans. The specific host varies with the species of *Sarcocystis*.

Dogs are the definitive hosts for *S. cruzi* (*S. bovicanis*) *S. miescheriana* (*S. suicanis*), *S. tenella* (*S. ovicanis*), *S. arieticanis*, *S. capracanis* and *S. hircicanis*. Wolves, coyotes, raccoons, foxes and hyenas are also definitive hosts for *S. cruzi*. Wolves, raccoons and jackals are also definitive hosts for *S. miescheriana*.

Cats are the definitive hosts for *S. hirsuta* (*S. bovis felis*), *S. porcifelis*, *S. moulei*, *S. gigantea* (*S. ovifelis*), *S. medusi-*

formis, *S. muris*, *S. leporuum*, *S. fusiformis*, *S. cymruensis* and *S. cuniculi*.

Non-human primates and humans are the definitive hosts for *S. hominis* and *S. sui hominis*. Opossums are the definitive hosts for *S. neurona*.

Intermediate hosts

Intermediate hosts include cattle, sheep, goats, buffalo, pigs, horses, poultry, birds, dogs, cats, rodents, rabbits and wildlife. *S. hominis*, *S. cruzi* and *S. hirsuta* are found in cattle. *S. sui hominis*, *S. miescheriana* and *S. porcifelis* are found in pigs. *S. tenella* (*S. ovicanis*), *S. arieticanis*, *S. gigantea* (*S. ovifelis*) and *S. medusiformis* are found in sheep. *S. capracanis*, *S. hircicanis* and *S. moulei* are found in goats. *S. fusiformis* is found in water buffalo. *S. canis* is found in dogs. *S. leporuum* and *S. cuniculi* are found in rabbits. *S. muris* and *S. cymruensis* are found in rodents. *S. neurona*, *S. fayeri* and *S. bertrami* are found in horses. Horses are intermediate hosts for *S. fayeri* and *S. bertrami*. They are aberrant intermediate hosts for *S. neurona*; sarcocysts do not develop in their muscles.

S. neurona can be found in an unusually large number of intermediate hosts including domestic cats, armadillos, skunks, raccoons, sea otters and probably other species. *S. neurona* causes disease mainly in horses, which are aberrant intermediate hosts for this organism. Symptomatic infections have also been reported in a pony, cats, a lynx, mink, raccoons, skunks, sea otters, harbor seals and a monkey. Birds may act as vectors/ transport hosts for *S. neurona*.

Incubation Period

Clinical signs are not usually seen in the definitive host. The prepatent period is usually 7 to 14 days. The incubation period for equine protozoal myeloencephalitis is not known with certainty, but could be as short as 10 to 12 days and as long as five years. Little is known about the incubation period for most *Sarcocystis* spp. in their intermediate host.

Clinical Signs

Most animals infected with *Sarcocystis* spp. are asymptomatic, and the parasites are seen mainly as an incidental finding at necropsy. However, clinical cases are occasionally reported, particularly in the intermediate host. Of the following diseases, only equine protozoal myeloencephalitis is diagnosed frequently; the remaining syndromes are rare.

Equine protozoal myeloencephalitis

Equine protozoal myeloencephalitis is a disease of horses usually caused by *Sarcocystis neurona*. This organism also infects many horses without symptoms. Clinical disease may begin insidiously or be acute and severe, and can be associated with a wide variety of neurologic signs including depression, head tilt, facial paralysis, seizures, visual defects, behavioral abnormalities, asymmetric or symmetric weakness, and ataxia of one or more limbs. In some cases, there may be discrete areas of spontaneous sweating or loss of reflexes and skin sen-

sation. Muscle wasting can also occur. Untreated animals may become recumbent, over a period of hours to years, and die. *S. neurona* has also caused rare cases of neurologic disease in a pony, domestic cats, a lynx, raccoons, skunks, mink, sea otters, harbor seals and a monkey.

Multifocal myelitis in horses

Multifocal myelitis caused by *S. fayeri* is a rare infection reported in horses. The symptoms in naturally infected horses include myositis, muscle atrophy, weight loss and decreased appetite. Fever, anorexia, mild depression, mild anemia and wasting have been reported in experimentally infected ponies and horses. Myositis caused by *S. bertrami* has been reported in horses in Europe but not in the U.S.

Sarcocystosis in ruminants

In cattle, *Sarcocystis* spp. can cause acute disease in calves, eosinophilic myositis, abortions and neurologic disease. Clinical signs reported in heavily infected cattle include fever, anorexia, wasting, decreased milk production, diarrhea, muscle spasms, weakness, hyperexcitability, pneumonia, hemorrhages, anemia, icterus, prostration and death. Pregnant cows may abort or give birth to still born calves. One group of feedlot cattle failed to thrive after recovery from acute sarcocystosis and eventually died of cachexia. In these calves, sarcocystosis was associated with the loss of the tailswitch hair. Rare cases of necrotic encephalitis have also been reported in cattle. *S. hominis* infections appear to be asymptomatic.

S. tenella is thought to be the most pathogenic of the *Sarcocystis* species in sheep. It can cause anorexia, fever, decreased weight gain, anemia and death in experimentally infected lambs, and has been associated with abortions in ewes. Neurologic signs including encephalomyelitis, muscle weakness, hindlimb paresis and ataxia have been seen in naturally infected sheep. After recovery from the acute illness, some sheep may lose their wool. Acute deaths can also occur without other symptoms. *S. medusiformis* and *S. gigantea* are thought to be nonpathogenic or cause only mild disease.

Sarcocystosis in pigs

Clinical signs associated with sarcocystosis in pigs may include weight loss or reduced weight gain, dyspnea, muscle tremors and abortions. There may be purpura of the skin, most often on the legs and buttocks. Natural infections with *S. sui hominis* rarely result in significant disease; however, experimental infections with large doses of organisms in suckling piglets can result in severe disease and deaths.

Sarcocystosis in dogs and cats

Clinical signs are unlikely to be seen in the cats or dogs serving as the definitive host. Experimental enteric infections are asymptomatic or mild.

A *Sarcocystis* species, termed *S. canis*, has been linked to encephalitis, hepatitis and generalized coccidiosis in

young dogs. The neurological signs included depression, generalized weakness, recumbency, nystagmus and seizures. Sarcocystosis of the lungs has been reported in association with canine distemper in a dog.

Meningoencephalitis or encephalitis associated with *Sarcocystis* has been reported in two domestic cats and a lynx. Sarcocysts have also been found in the muscles of cats at necropsy.

Communicability

The definitive host usually begins shedding oocysts or sporocysts within 7 to 14 days, and continues to excrete them in the feces for several months. The oocytes or sporocysts can infect the intermediate host for that species of *Sarcocystis*. Intermediate hosts cannot transmit the infection unless they are eaten, and unless their tissues contain the bradyzoite stage. This stage usually appears 2 to 4 months after infection.

Diagnostic Tests

Sporocysts or oocysts found in the feces are suggestive of intestinal *Sarcocystis* infection. *Sarcocystis* spp. are indistinguishable morphologically and are difficult to detect during fecal flotation.

In intermediate hosts, the asexual stages may be found in affected tissues by microscopy. They can be distinguished from the tachyzoites of *Toxoplasma gondii* or *Neospora* spp. with anti-*Sarcocystis* antiserum. Immunoblotting and a direct agglutination test have been used to detect *Sarcocystis* antibodies.

Equine protozoal myeloencephalitis can be confirmed at necropsy by demonstrating protozoa in the CNS lesions. Serologic tests used in horses include direct agglutination, an immunofluorescent antibody (IFA) test and immunoblotting. Only immunoblotting is free of cross-reactivity with other species of *Sarcocystis*. Antibodies found in the CSF are highly suggestive of equine protozoal myeloencephalitis; serum antibodies are less valuable. A polymerase chain reaction (PCR) assay can detect *S. neurona* DNA.

Treatment

Equine protozoal myeloencephalitis can be treated with sulfonamides and pyrimethamine. Other drugs that have been used include diclazuril, toltrazuril and Ponazuril®. In addition, anti-inflammatory medications or vitamin E may be given, and supportive care may be necessary. The use of immunomodulators has been proposed, based on a suspicion that *Sarcocystis* may be more common in immunocompromised patients. Treatment for extended periods may be necessary.

Amprolium has been used prophylactically to decrease the clinical signs in experimental infections in cattle. Amprolium and salinomycin have been used in experimentally infected sheep. Therapeutic treatment of cattle and sheep has, to date, been ineffective.

Prevention

Horses are aberrant intermediate hosts in equine protozoal myeloencephalitis. They become infected by exposure to the feces of opossums, the definitive hosts. To avoid attracting opossums, and also to prevent fecal contamination, grains, fruit, garbage, pet food or other food attractants should be kept in closed metal containers. Opossums may also be attracted to bird feeders and fallen fruit. A partially buried fence and electric fences can be used to discourage opossums from entering paddocks. Opossums may be trapped and relocated. Birds may be able to serve as transport hosts for *S. neurona*. Excluding birds from horse barns may be helpful. Intermediate hosts for *S. neurona* could spread the infection to opossums in the area; dead cats, armadillos, skunks, and raccoons should be disposed of promptly. Heat treating feed and steam cleaning horses' environments may reduce the risk of infection. A vaccine has recently become available.

Livestock, which act as intermediate hosts for various species of *Sarcocystis*, become infected by exposure to the feces of the definitive host. Feed supplies should be kept covered, and dogs and cats should be kept out of livestock housing and feed storage buildings. Exposure to human feces should be prevented. Amprolium has been used prophylactically to decrease the clinical signs in experimental infections in cattle. Amprolium and salinomycin have been used in experimentally infected sheep.

Dogs and cats, which usually act as definitive hosts, become exposed by eating infected raw or undercooked meat. Dogs and cats should not be allowed to eat raw meat, offal or dead animals.

Morbidity and Mortality

Sarcocystis spp. are estimated to infect 10-100% of all ruminants. In some countries, these parasites are also found in many pigs. In the U.S., porcine sarcocystosis is less common and seems to be decreasing as a result of confinement rearing. Natural infections in food animals are usually asymptomatic but clinical cases are occasionally reported. One outbreak of neurologic disease affected 10% of a flock of sheep. Experimental infections with large doses of organisms in young pigs have resulted in mortality rates of up to 50%. In naturally infected animals, deaths are rare.

Equine protozoal myeloencephalitis is a common neurologic disease in the U.S. Most cases are seen in young horses but animals of any age can be affected. Cases usually occur sporadically but small outbreaks, with morbidity rates greater than 25%, have been reported. Stress seems to increase the risk of clinical disease. In the U.S., approximately 30-60% of all horses have been exposed to *S. neurona*. All of the horses may be exposed on individual farms. Clinical disease is much less common: the average incidence is 14 cases per 10,000 horses per year. Approximately 60-75% of horses improve with treatment. A smaller percentage recov-

ers fully; up to 30% of horses return to their original level of performance. Approximately 10-28% of horses relapse, days to months after treatment. The reasons for the high relapse rate are unknown.

Sarcocystis spp. rarely cause myositis in horses in the U.S., but intramuscular sarcocysts were found in 0.5-21% of the horses examined in a slaughter plant. In Morocco, 46% of horses had intramuscular sarcocysts at necropsy.

Intestinal sarcocystosis is usually inapparent in dogs and cats; however, clinical disease can be seen when they serve as intermediate hosts. Rare serious or fatal cases of neurologic disease, hepatitis or generalized disease have been reported in young dogs. Rare cases of neurologic disease caused by *Sarcocystis* spp. (including *S. neurona*) have been documented in cats and a lynx. Recent surveys in Florida, Michigan and Ohio reported that 5-10% of cats have antibodies to *S. neurona*. On some farms with cases of equine protozoal myeloencephalitis, 40% of the farm cats were seropositive.

Post-Mortem Lesions

Sarcocysts can be found in skeletal and cardiac muscles, as well as the central nervous system (CNS) in all species. In most cases, the parasite is an incidental finding at necropsy.

In pigs and cattle, sarcocysts are most common in cardiac muscle, the diaphragm and esophagus. In sheep, sarcocysts may also be found in the tongue. The cylindrical, whitish cysts vary in size from a few micrometers to a few centimeters, and may or may not be visible to the naked eye. They are found along the length of the muscle fiber and may resemble a grain of rice. Post-mortem lesions in symptomatic cattle may also include cachexia, hepatitis and myocarditis. Hemorrhages of the serosa of the viscera, myocardium and skeletal muscles have been reported in sheep infected with *S. tenella*.

The lesions of equine protozoal myeloencephalitis may include focal discoloration, hemorrhages or malacia in the CNS.

Multifocal meningoencephalitis has been reported in the CNS of symptomatic dogs and cats, with sarcocysts visible on microscopic examination. Gross lesions may not be seen. Sarcocysts have also been found in the muscles, lungs and other tissues.

Internet Resources

Animal Health Australia. The National Animal Health Information System (NAHIS)

<http://www.aahc.com.au/nahis/disease/dislist.asp>

Food and Agriculture Organization of the United Nations [FAO] - Manual on Meat Inspection for Developing Countries

<http://www.fao.org/docrep/003/t0756e/T0756E05.htm>

Fort Dodge Equine Myeloencephalitis Vaccine Information

<http://www.questgel.com/epm/01fdvaccine.htm>

Medical Microbiology

<http://www.gsbs.utmb.edu/microbook>

The Merck Manual

<http://www.merck.com/pubs/mmanual/>

The Merck Veterinary Manual

<http://www.merckvetmanual.com/mvm/index.jsp>

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