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Sarcocystosis of Animals and Humans

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Sarcocystosis of Animals and Humans

J. P. Dubey • R. Calero-Bernal • B. M. Rosenthal

Animal Parasitic Diseases Laboratory
Beltsville Agricultural Research Center
Agricultural Research Service
U.S. Department of Agriculture
Beltsville, Maryland

C. A. Speer

Healthwide Solutions
Vail, Colorado

R. Fayer

Environmental Microbial and Food Safety Laboratory
Beltsville Agricultural Research Center
Agricultural Research Service
U.S. Department of Agriculture
Beltsville, Maryland



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Preface

Since the publication of the first edition of the *Sarcocystosis of Animals and Man* in 1989, considerable progress has been made in understanding the biology of the genus *Sarcocystis*. Sarcocystosis is one of the most frequent infections of warm-blooded and poikilothermic animals worldwide. Completion of the life cycle requires two host species: an intermediate (or prey) host and a definitive (or predator) host. Hosts can harbor more than one species of *Sarcocystis*. In intermediate hosts, whether normally involved in the life cycle or aberrantly involved, some species of *Sarcocystis* cause reduced weight gain, poor feed efficiency, anorexia, fever, anemia, muscle pain, and weakness, reduced milk yield, abortion, neurologic impairment, and death. Such infections are of economic importance in intermediate hosts such as cattle, sheep, goats, and pigs. Disease has also been observed in avian species. Humans occasionally have been involved as aberrant intermediate hosts. In definitive hosts, some species of *Sarcocystis* can cause digestive disturbances, including nausea, vomiting, and diarrhea. Humans serve as a host for at least two such species. The underlying mechanisms giving rise to disease in both muscular and intestinal sarcocystosis are reviewed.

Outbreaks of a mysterious disease with severe symptoms have been reported in vacationers returning from the Malaysian islands. Symptoms included fever, myalgia, headache, and cough, associated with elevated levels of enzymes indicative of muscle degeneration and inflammation. Muscle biopsies in a few patients revealed intramuscular sarcocysts. In the absence of other etiologies, these infections have been identified as muscular sarcocystosis, presumably acquired from ingestion of water and food contaminated with *Sarcocystis* sporocysts of reptile origin. The evidence linking the infectious agent to *Sarcocystis nesbitti* of nonhuman primates is herein reviewed. Methods of diagnosis and detection are discussed with regard to future needs. The status of chemoprophylaxis, chemotherapy, immunity, and vaccination is reviewed and future needs discussed.

After the publication of the first edition, the causative agent of a fatal disease of horses, equine protozoal myeloencephalitis (EPM), was found to be a new species of *Sarcocystis*—*S. neurona* was described in 1991, and its life cycle was completed in 2000. Unlike most other species of the genus, *S. neurona* was found to have a wide host range of intermediate hosts but only opossum as a definitive host. Additionally, an EPM-like fatal disease was recognized in many other hosts, especially marine mammals. Furthermore, *S. neurona* has been used as a model by biologists to study organelle synthesis, because it is easily cultivated in cell culture and because the nuclear division is prolonged. We have summarized available literature on *S. neurona* biology, clinical diagnosis, and prevention in a separate chapter in this second edition.

There has been considerable progress in molecular aspects of *Sarcocystis*, including characterization of recombinant antigens for diagnosis and the development of methodologies and markers that make possible studies on molecular epidemiology. We have added a separate chapter on the molecular biology of *Sarcocystis*.

We have attempted to preserve the format used in the first edition to add recent developments, and exclude information that has become redundant. Our objective has been to include all literature, and provide a comprehensive review of biology, clinical disease, economic losses, public health concerns, diagnosis, treatment, and prevention. We have tabulated information on all *Sarcocystis* species by host and listed species that should be considered species enquirende/invalid.

It is hoped that this book will be useful to biologists, veterinarians, physicians, and researchers.

We would like to acknowledge those who made this book possible; we feel we cannot possibly list all of them. Ana Beatriz Cassinelli, Camila K. C. Cézar, Oliver Kwok, Yuging Ying, and Francisco J. López Acevedo helped with the bibliography, Petras Prakas and Liuda Kutkiené

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J. P. Dubey
Rafael Calero-Bernal
Benjamin M. Rosenthal
C. A. Speer
Ronald Fayer

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Authors

J. P. Dubey, MVSc, PhD, was born in India. He earned his veterinary degree in 1960 and master's in veterinary parasitology in 1963, from India. He earned a PhD in medical microbiology in 1966 from the University of Sheffield, England. Dr. Dubey received postdoctoral training from 1968 to 1973 with Dr. J. K. Frenkel, Department of Pathology and Oncology, University of Kansas Medical Center, Kansas City. From 1973 to 1978, he was associate professor of veterinary parasitology, Department of Pathobiology, Ohio State University, Columbus, and professor of veterinary parasitology, Department of Veterinary Science, Montana State University, Bozeman, from 1978 to 1982. He is presently a senior scientist, Animal Parasitic Diseases Laboratory, Beltsville Agricultural Research Institute, Agricultural Research Service, U.S. Department of Agriculture, Beltsville, Maryland.

Dr. Dubey has spent over 50 years researching protozoa, including *Toxoplasma*, *Neospora*, *Sarcocystis*, and related cyst-forming coccidian parasites of humans and animals. He has published over 1400 research papers in international journals, more than 250 of which are on sarcocystosis. In 1985, he was chosen to be the first recipient of the "Distinguished Veterinary Parasitologist Award" by the American Association of Veterinary Parasitologists. Dr. Dubey is a recipient of the 1995 WAAVP Pfizer Award for outstanding contributions to research in veterinary parasitology. He also received the 2005 Eminent Parasitologists Award from the American Society of Parasitologists. The Thomas/Institute for Scientific Information identified him as one of the world's most cited authors in plant and animal sciences over the last decade. In 2003, he was selected for the newly created Senior Scientific Research Service (SSRS) and is one of the few scientists and executives within the USDA's Agricultural Research Service; selection for this position is by invitation only, on approval by the Secretary of Agriculture. In 2010, Dr. Dubey was elected to the U.S. National Academy of Sciences, Washington, DC and inducted in the USDA-ARS Hall of Fame.

Rafael Calero-Bernal, DVM, MSc, PhD, was born in Badajoz (Spain). He earned his degree in veterinary medicine at the University of Extremadura, Spain in 2006. One year later, he attended the Official Master's program in meat science and technology at the same institution. In 2011, Dr. Calero-Bernal earned a PhD in European framework in veterinary medicine. Since 2008, he has been a professor in the Animal Health Department at the University of Extremadura, developing teaching curricula at the Faculty of Veterinary Medicine of the University of Lisbon (Portugal). He has been a researcher in the Spanish National Microbiology Centre and the Tropical Medicine National Centre, both part of the Instituto de Salud Carlos III (Spain). Dr. Calero-Bernal developed several research protocols at Istituto Superiore di Sanità (Italy), Instituto Nacional de Saúde (Portugal), Fundação Oswaldo Cruz (Brazil), and Centro de Referencia para el Control de Endemias (Equatorial Guinea). Currently he is a postdoctoral researcher in the Agricultural Research Service, United States Department of Agriculture. He has authored more than 50 articles and 4 books related to veterinary sciences. His research interests are wildlife parasites and zoonoses, especially meat-borne pathogens and tissue cysts forming coccidia.

Benjamin M. Rosenthal is a parasitologist with primary interests in the population genetics, phylogenetics, genomics, and epidemiology of zoonotic parasites. He is a graduate of Oberlin College and the Harvard School of Public Health. Since 1999, he has led a research program in veterinary and zoonotic parasitic diseases for the USDA's Agricultural Research Service, where his studies have encompassed toxoplasmosis, sarcocystosis, trichinellosis, and related diseases. His work uses genetic variation within and among parasitic microorganisms to (1) develop improved methods for their detection and diagnosis, (2) clarify their routes of transmission and define what risk they may pose to public health, and (3) establish an accurate understanding of their evolutionary relationships and origins. He mentors secondary, undergraduate, graduate, and postdoctoral students. His course work at the University of

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Maryland, College Park focuses on the ecology and evolutionary biology of infectious disease. Dr. Rosenthal developed microstatellite markers to characterize strains of *Sarcocystis neurona*, employed molecular diagnostics to differentiate among various suspected species of *Sarcocystis*, and provided the inference that *Sarcocystis nesbitti*, a newly recognized zoonotic species implicated in human tissue infections, might have its definitive host in a snake or related reptile.

C. A. Speer, PhD, earned a BS from Colorado State University in 1967 and MS and PhD degrees from Utah State University in 1969 and 1972, respectively. During 1972 and 1973, Dr. Speer was assistant professor of histology at the University of Texas Medical Center in Houston. From 1973 to 1975, he was a postdoctoral fellow of the U.S.-AID (Agency for International Development) Malaria Immunity and Vaccination Project at the University of New Mexico, Albuquerque. From 1975 to 1983, Dr. Speer was assistant and then associate professor of microbiology at the University of Montana, Missoula. From 1983 to 2000, Dr. Speer held several positions at Montana State University, Bozeman—as associate professor of veterinary science and director of the Electron Microscope Facility in the Department of Veterinary Science from 1983 to 2000 and as professor and head of Veterinary Molecular Biology from 1986 to 1994. In 1994, he founded and served as director of the Center for Bison and Wildlife Health until 2000. From 2000 to 2008, he served as Dean of the College of Agricultural Sciences and Natural Resources, director of the Agricultural Experiment Station, founder and director of the Center for Wildlife Health and the B. Ray Thompson Distinguished Professor of Cell and Molecular Immunology at the University of Tennessee, Knoxville. Currently, Dr. Speer is professor emeritus at Montana State University and the University of Tennessee and vice president of Strategic Planning for Healthwide Solutions located in Vail, Colorado.

Dr. Speer's scholarly activities have contributed to the education of numerous undergraduate and graduate students and postdoctoral fellows. He has published more than 220 research papers in refereed journals on malaria, Chagas' disease and Crohn's disease of humans and various infectious diseases of wildlife and livestock, including malaria, coccidiosis, trichomoniasis, equine protozoal myeloencephalitis (EPM), cryptosporidiosis, toxoplasmosis, whirling disease, and Johne's disease (paratuberculosis). He has also published on epitope analysis in leukocyte–endothelial cell interactions, genetic disruption of cell junctions, and expression of L-selectin by bovine T cells. He conducted research on human malaria and other protozoan diseases in the Brazilian Amazon, in India, and in Africa. He holds two international and two U.S. patents on the biochemical extraction and use of surface antigens to diagnose infectious diseases. In 1986, he received the Mershon Award for Excellence in Research from the Montana Academy of Sciences, the Honorary Alumnus Award in 1987 from the College of Natural Sciences, Colorado State University, Fort Collins and in 2002, the Gubernatorial Award for Biotechnology in Tennessee.

Ronald Fayer earned his master's and PhD degrees in 1964 and in 1968, respectively, from Utah State University. He joined the Agricultural Research Service of the U.S. Department of Agriculture in 1968 where he has worked on the life cycles, *in vitro* cultivation, pathological effects, epidemiology, immunology, and molecular biology of protist parasites of veterinary and public health importance, including *Sarcocystis*, *Cryptosporidium*, *Giardia*, *Microsporidia*, and *Blastocystis*. He has published over 400 peer-reviewed scientific articles, review chapters, and books, and shares four patents. He has served as president of the American Society of Parasitologists (ASP) and of the American Association of Veterinary Parasitologists (AAVP). For his scientific contributions he has received numerous awards, including the H.B. Ward medal from the ASP, the Distinguished Veterinary Parasitologist Award from the AAVP, USDA Superior Service Awards from two Secretaries of Agriculture, the rank of Distinguished Senior Professional conferred by the President of the United States, and other awards. He received a Fulbright Senior Scholar Award for research in Spain. He is currently working on molecular epidemiology of zoonotic protists at the Environmental Microbial and Food Safety Laboratory.

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Abbreviations

ALT	alanine aminotransferase
AAT	aspartate aminotransferase
BUN	blood urea nitrogen
CPK	creatinine phosphase kinase
CNS	central nervous system
h	hours
DPI	day postinoculation
H and E	hematoxylin and eosin
ELISA	enzyme-linked immunoabsorbent assay
IgG	immunoglobulin G
IgM	immunoglobulin M
IHA	indirect hemagglutination
IHC	immunohistochemical staining
IFA	indirect fluorescent antibody
ITS	internal transcriber space
LDH	lactic dehydrogenase
KO mice	interferon gamma gene knockout mice
LM	light microscopy
min	minutes
PAS	periodic acid Schiff reaction
PCR	polymerase chain reaction
PCV	packed cell volume
PI	postinoculation
PV	parasitophorous vacuole
PVM	parasitophorous vacuolar membrane
RAPD	random amplified polymorphic DNA
RBC	red blood cells
RFLP	restriction fragment length polymorphism
SAT	*Sarcocystis neurona* direct agglutination test
SBDH	sorbitol dehydrogenase
SCID	severe combined immune deficiency syndrome
SEM	scanning electron microscopy
TEM	transmission electron microscopy
WB	Western blot

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CHAPTER 1

General Biology

1.1 INTRODUCTION AND HISTORY

Sarcocystis was first reported by Miescher in 1843 as "milky white threads" in the skeletal muscle of a deer mouse caught in his house in Switzerland.[1191] Miescher did not specify the zoological name of the mouse. Subsequently, the parasite found in the house mouse (*Mus musculus*) was named *Sarcocystis muris* (Figure 1.1) and is believed to be the same as that discovered by Miescher. The life cycle of *Sarcocystis* remained unknown until 1972 (Table 1.1).

Historical accounts[803,1317,2020,2110,2111,2169,2170,2144,2167,2289] of *Sarcocystis* are given in the first edition of this book[393] and are not repeated here.

Sarcocystis species are coccidian parasites and belong to

Phylum—Apicomplexa; Levine, 1979
Class—Sporozoasida; Leuckart, 1879
Subclass—Coccidiasina; Leuckart, 1879
Order—Eucoccidiorida; Leger and Duboseq, 1910
Suborder—Eimeriorina; Leger, 1911
Family—Sarcocystidae; Poche, 1913
Subfamily—Sarcocystinae; Poche, 1913
Genus—*Sarcocystis*; Lankester, 1882
Type species—*Sarcocystis miescheriana* (Kühn, 1865) Labbe, 1899

1.1.1 Generic Diagnosis

Sarcocysts are found in muscles and in the central nervous system of homeothermic and poikilothermic animals. The life cycle includes: obligatorily 2 hosts, asexual multiplication in the intermediate host with "sarcocysts" in muscles and CNS, gamonts, fertilization, and endogenous sporulation of oocysts in the intestine of definitive hosts. Oocysts are not infective for the definitive host.

There is considerable debate concerning the number of species in the genus *Sarcocystis*. In the last review of the taxonomy of the genus, Odening[1317] listed 189 species; many of these are probably not valid. In the last chapter of this book (Chapter 24), we have summarized the current status. It appears that there are 196 valid *Sarcocystis* species; however, full life cycles are known only for 26 of them.

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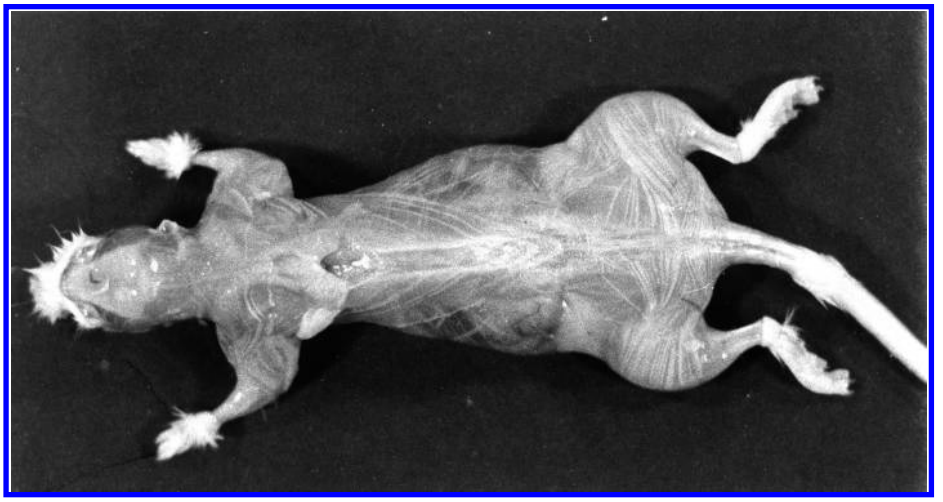


Figure 1.1 Macroscopic, criss-crossing, thread-like *S. muris* sarcocysts in skeletal muscles of a mouse (*Mus musculus*). (From Springer Science+Business Media: *Cardiovascular and Musculoskeletal Systems,* Sarcocystosis of the skeletal and cardiac muscle, mouse, 1991, pp. 165–169, Dubey, J.P.)

Table 1.1 Historical Landmarks Concerning Genus *Sarcocystis*

Year	Findings	References
1843	Sarcocysts found in the muscles of a house mouse	**1191**
1882	Genus *Sarcocystis* named	**1022**
1972	Sexual phase cultured *in vitro*	**571**
1972	Two-host life cycle found	**1478, 1479**
1973	Vascular phase recognized and pathogenicity demonstrated	**572**
1975	Multiple *Sarcocystis* species within a given host recognized	**803**
1975	Chemotherapy demonstrated	**576**
1976	Abortion due to sarcocystosis recognized	**577**
1981	Protective immunity demonstrated	**358**
1986	Vascular phase cultured *in vitro*	**1655**
1989	*Sarcocystis* species classified based on morphology of cyst wall	**393**
1991	Multiple host *Sarcocystis* species, *S. neurona* discovered as the etiological agent of Equine Protozoan Myeloencephalitis (EPM)	**404**
2015	First *Sarcocystis* genome annotated	**109**

1.2 STRUCTURE AND LIFE CYCLE

1.2.1 Structure

Sarcocysts (in Greek, Sarkos = flesh, kystis = bladder) are the terminal asexual stage found encysted, primarily in striated muscles of mammals, birds, and poikilothermic animals (intermediate hosts). The number and distribution of sarcocysts throughout the body vary greatly from host to host. Factors affecting the number and distribution of sarcocysts include the number of organisms (sporocysts) ingested, the species of *Sarcocystis*, the species of host, and the immunological state of the host. Although most sarcocysts develop in striated muscles of the heart, tongue, esophagus, diaphragm, and skeletal muscles, some sarcocysts have been found in the smooth muscles of the

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intestine. Sarcocysts of *S. mucosa* have been found in the gut of marsupials, apparently in smooth muscles.[108,1293] Infrequently, immature sarcocysts of some species (*S. cruzi*, *S. tenella*) also have been found in the muscularis layer of the gut.[360,367] Sarcocysts also have been found in the CNS and in the Purkinje fibers of the heart and muscle bundles, but always in relatively low numbers. They are found in different types of myofibers.[1412]

Sarcocysts vary in size and shape, depending on the species of the parasite (Figures 1.1 through 1.5). Some always remain microscopic (e.g., *S. cruzi*), whereas others become macroscopic (e.g., *S. gigantea*, *S. muris*). Microscopic sarcocysts vary from very long and narrow to short and wide. Macroscopic sarcocysts, which are nearly always in skeletal muscles or esophageal muscles, appear filamentous (e.g., *S. muris*), like rice grains (e.g., *S. rileyi*), fusiform (e.g., *S. fusiformis*) or globular (e.g., *S. gigantea*).

Sarcocysts are always located within a PV in the host-cell cytoplasm, often not visible under the LM (Figure 1.6). More than 1 sarcocyst may be found in 1 host cell. The sarcocyst consists of a cyst wall that surrounds the asexual metrocyte or zoite stages (Figures 1.7 and 1.8). The structure and thickness of the cyst wall varies among species of *Sarcocystis*, and within each species, as the sarcocyst matures. A connective tissue wall (secondary sarcocyst wall) surrounds the *S. gigantea*, *S. mucosa*, *S. hardangeri*, and *S. rangiferi* sarcocysts[691,692,893,1141,1164] (Figure 1.9). Histologically, the sarcocyst wall may be smooth, striated, or hirsute, or may possess complex branched protrusions (Figure 1.10). Internally, groups of zoites may be divided into compartments by septa that originate from the sarcocyst wall or may not be compartmentalized. When present, septa are usually less than 2 µm thick, but sometimes they are indistinct or not found (Figure 1.11).[392,935] The structure of the parasites within the sarcocysts varies with the maturation of the sarcocyst. Immature sarcocysts contain globular parasites called metrocytes (mother cells). Each metrocyte produces 2 progeny by an internal form of multiplication called endodyogeny (described in Section 1.3.1.1.1). After what appears to be several such generations, some of the metrocytes, through the process of

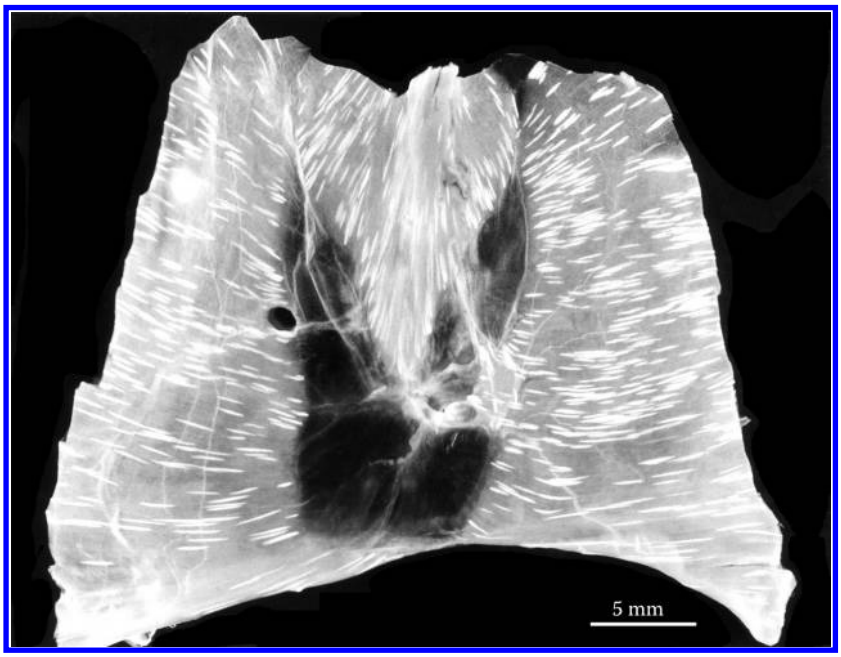


Figure 1.2 Macroscopic *S. campestris* sarcocysts in the diaphragm of a Richardson's ground squirrel (*Spermophilus richardsonii*). (From Cawthorn, R.J., Wobeser, G.A., Gajadhar, A.A., 1983. *Can. J. Zool.* 61, 370–377. With permission.)

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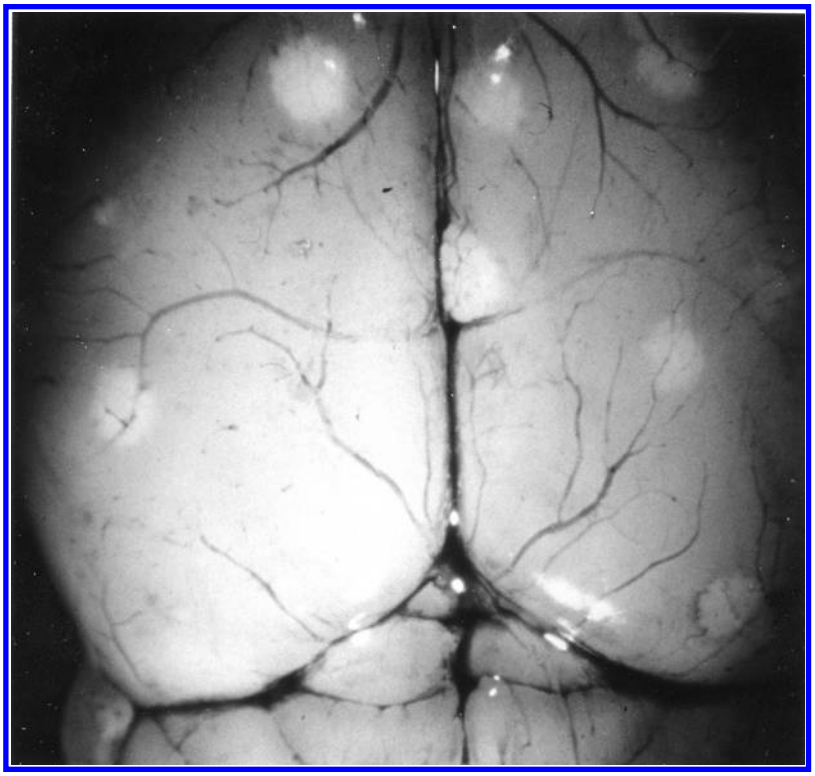


Figure 1.3 Globular *S. microti* sarcocysts in the brain of a vole (*Microtus agrestis*). (Courtesy of M. Rommel.)

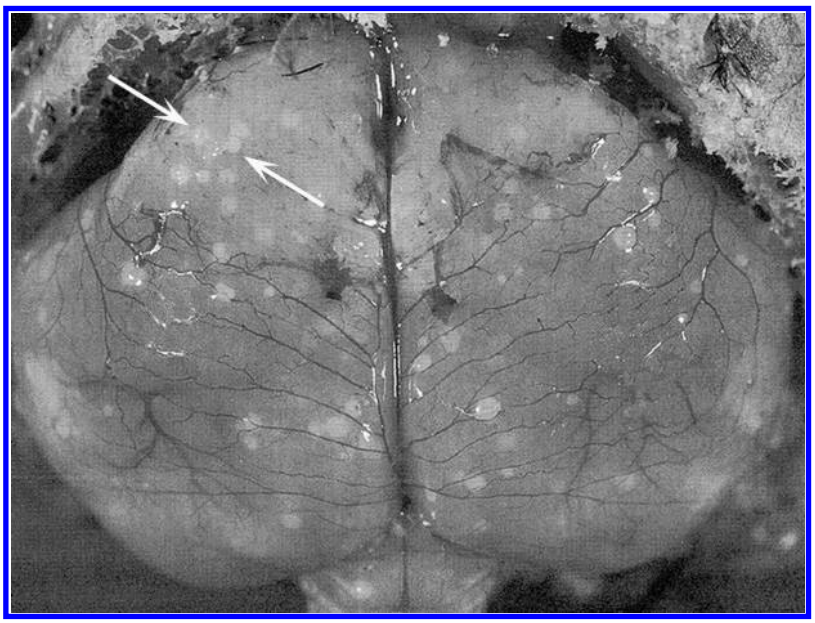


Figure 1.4 Globular *S. ochotonae* sarcocysts in the brain of a pika (*Ochotona* sp.). (From Odening, K. et al., 1998. *Zool. Garten* 68, 80–94. With permission.)

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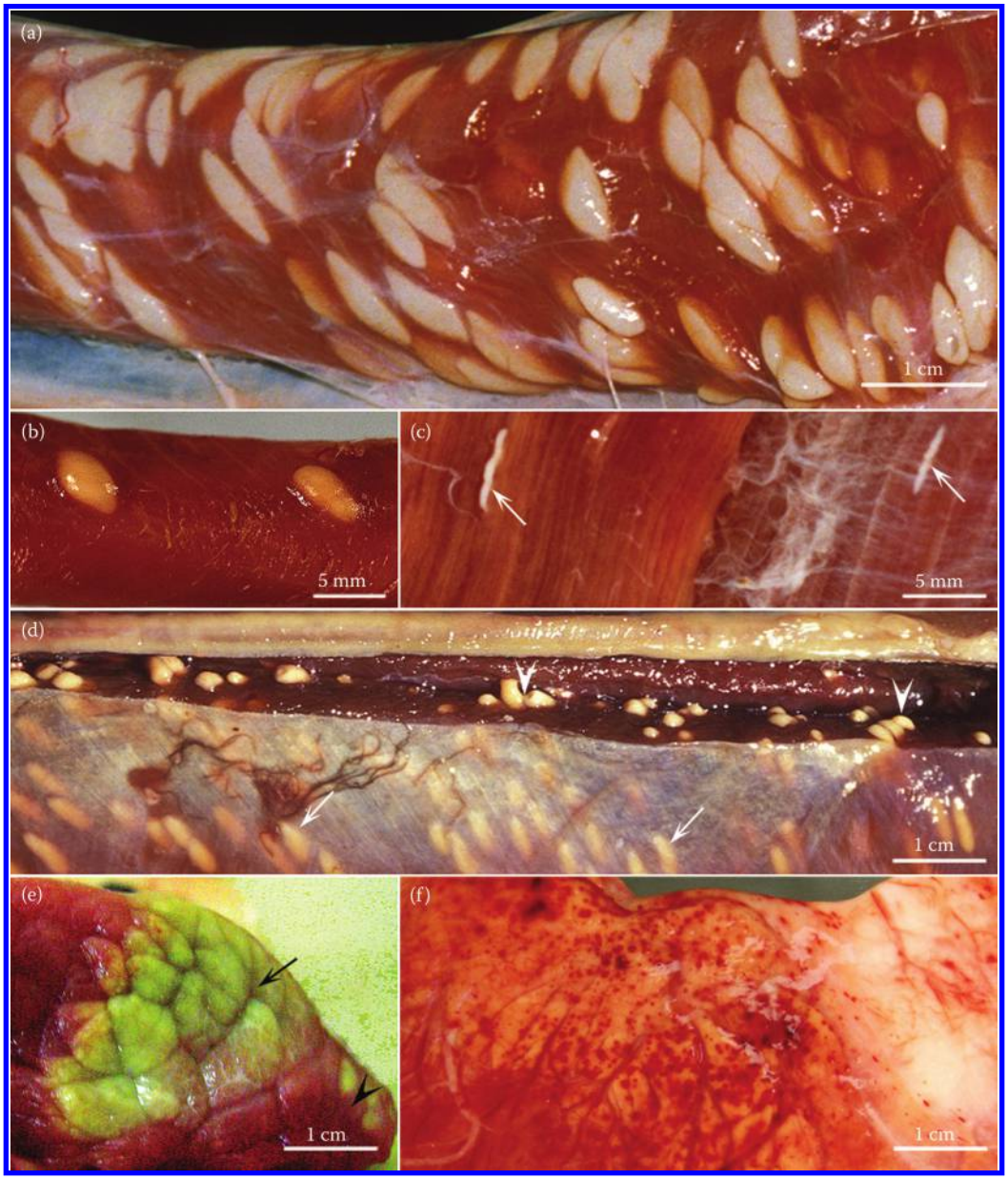


Figure 1.5 *Sarcocystis*-associated gross lesions in animals. Unstained. (a) Sarcocysts of *S. fusiformis* on the surface of the esophagus of a naturally infected water buffalo in Vietnam. (Courtesy of A. Uggla and L.T.T. Huong.) (b) Sarcocysts of *S. gigantea* on the surface of the esophagus of a naturally infected sheep in Sweden. (Courtesy of A. Uggla.) (c) Sarcocysts (arrows) of *S. hirsuta* in the flank muscle of a naturally infected cow in the United States. (Adapted from Dubey, J.P. et al. 1990. *J. Am. Vet. Med. Assoc*. 196, 1095–1096.) (d) Sarcocysts of *S. rileyi* in the pectoral muscle of a naturally infected mallard (*Anas platyrhynchos*) in Sweden. The sternum is on the top. Arrowheads point to sarcocysts in cut muscle. Arrows point to sarcocysts visible through the connective tissue. (Courtesy of A. Uggla.) (e) Eosinophilic myositis in the skeletal muscle of a naturally infected cattle in Belgium. Arrow points to a large greenish area and arrowhead points to smaller lesions. (Courtesy of the Laboratory of Veterinary Pathology, Ghent University, Belgium.) (f) Hemorrhages in the epicardium of the heart of a calf experimentally infected with *S. cruzi*. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res*. 43, 2147–2164. With permission.)

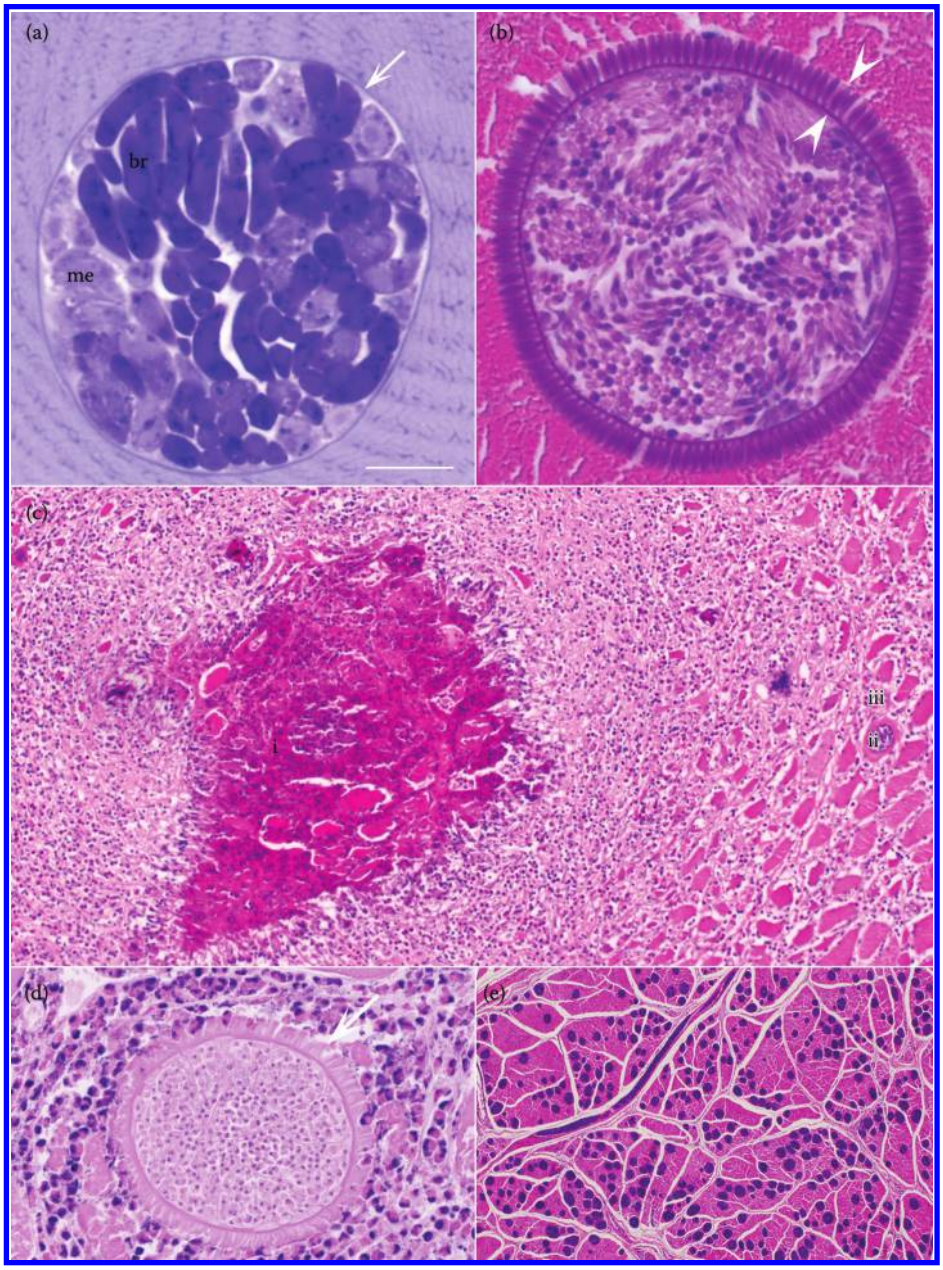


Figure 1.6 Sarcocysts and associated lesions. (a), toluidine blue stain. (b–e), H and E stain. Bar applies to all parts; bar in (a) and (b) = 10 μm, in (c) = 200 μm, in (d) = 25 μm, and in (e) = 350 μm. (a) *S. muris* in an experimentally infected *M. musculus* muscle. Note the thin sarcocyst wall (arrow), faintly stained metrocytes (me), and intensely stained bradyzoites (br). (b) *Sarcocystis* sp. in the skeletal muscle of a naturally infected turkey. (Adapted from Teglas, M.B. et al., 1998. *J. Parasitol*. 84, 661–663). (c) Eosinophilic myositis in a naturally infected cattle. Note an area of central necrosis (i), a peripheral rim of inflammatory cells (ii), and an intact sarcocyst (iii). (d) A degenerating *S. hominis*-like sarcocyst in an area of eosinophilic myositis. The bradyzoites have degenerated but the sarcocyst wall is mostly intact. (Adapted from Wouda, W., Snoep, J.J., Dubey, J.P., 2006. *J. Comp. Pathol*. 135, 249–253.) (e) Numerous *S. cruzi* sarcocysts in the tongue of an experimentally infected asymptomatic calf. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res*. 43, 2147–2164. With permission.)

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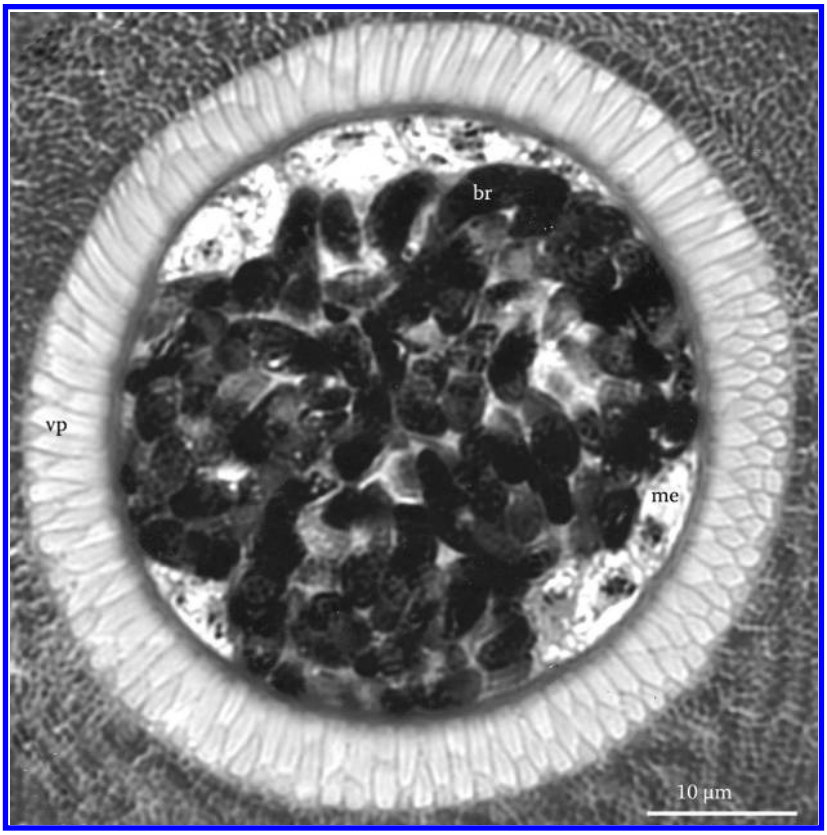


Figure 1.7 Cross section of a sarcocyst of *S. hominis* in the skeletal muscle of a cow. Villar protrusions (vp) on the sarcocyst wall enclose intensely stained bradyzoites (br) and pale staining metrocytes (me) arranged peripherally. 1 µm section, toluidine blue stain. (Adapted from Wouda, W., Snoep, J.J., Dubey, J.P., 2006. *J. Comp. Pathol.* 135, 249–253.)

endodyogeny, produce banana-shaped zoites called bradyzoites (also called cystozoites). Within the sarcocysts, metrocytes are generally located in the cortex, whereas bradyzoites are located in the medulla. In old, large sarcocysts, bradyzoites near the center of the sarcocyst are sometimes degenerate and are replaced by granules or globules. Live sarcocysts are probably fluid filled with metrocytes and bradyzoites, moving within the fluid. In histologic sections stained with H and E, metrocytes are paler than bradyzoites. The bradyzoites contain prominent amylopectin granules that stain bright red with PAS. In smears stained with Giemsa, metrocytes are oval to round in shape, and pink/magenta in color (see Figure 1.18).

1.2.2 Life Cycle

Sarcocystis has an obligatory prey–predator 2-host life cycle (Figure 1.12). Asexual stages develop only in the intermediate host, which, in nature, is often a prey animal. Sexual stages develop only in the definitive host, which is carnivorous. Intermediate and definitive hosts vary for each species of *Sarcocystis*. For example, there are 4 named species of *Sarcocystis* in cattle: *S. cruzi, S. hirsuta, S. hominis,* and *S. rommeli.* The definitive hosts for these species are canids, felids, primates, and unknown, respectively. Some species of *Sarcocystis* can complete the life cycle in the same host species, but not in the same animal[66] (see Chapter 22).

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Figure 1.8 TEM cross section of a *S. hemioni* sarcocyst in the skeletal muscles of a mule deer. The host-cell cytoplasm is separated from the sarcocyst by a thin parasitophorous vacuole (arrow). Note villar protrusions (pv), metrocytes (me), and bradyzoites (br).

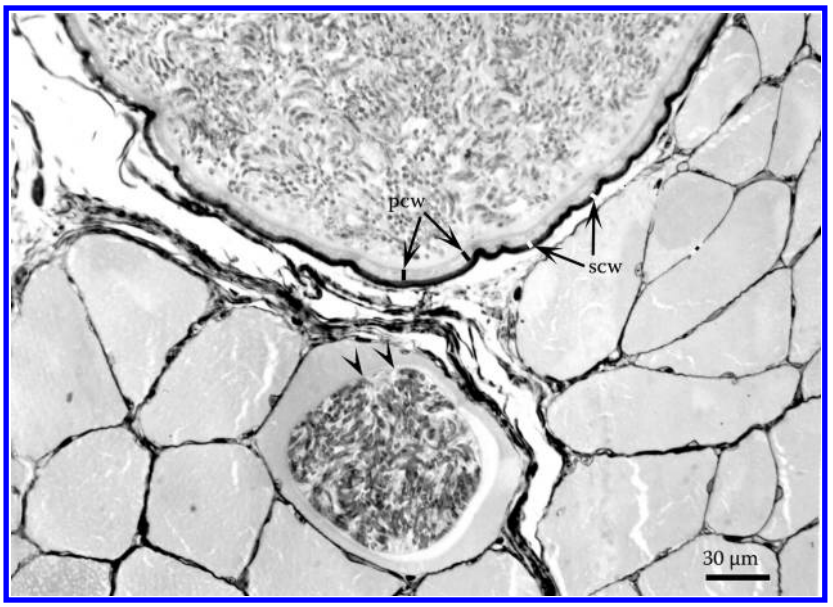


Figure 1.9 Sarcocysts of *S. gigantea* (top) and *S. arieticanis* (bottom) in the skeletal muscle of a sheep. The primary cyst wall (pcw) in *S. gigantea* is thick and surrounded by a connective tissue capsule (secondary sarcocyst wall, scw), while *S. arieticanis* (arrowheads) has no secondary sarcocyst wall. Gomori's methanine silver reaction, a connective tissue stain.

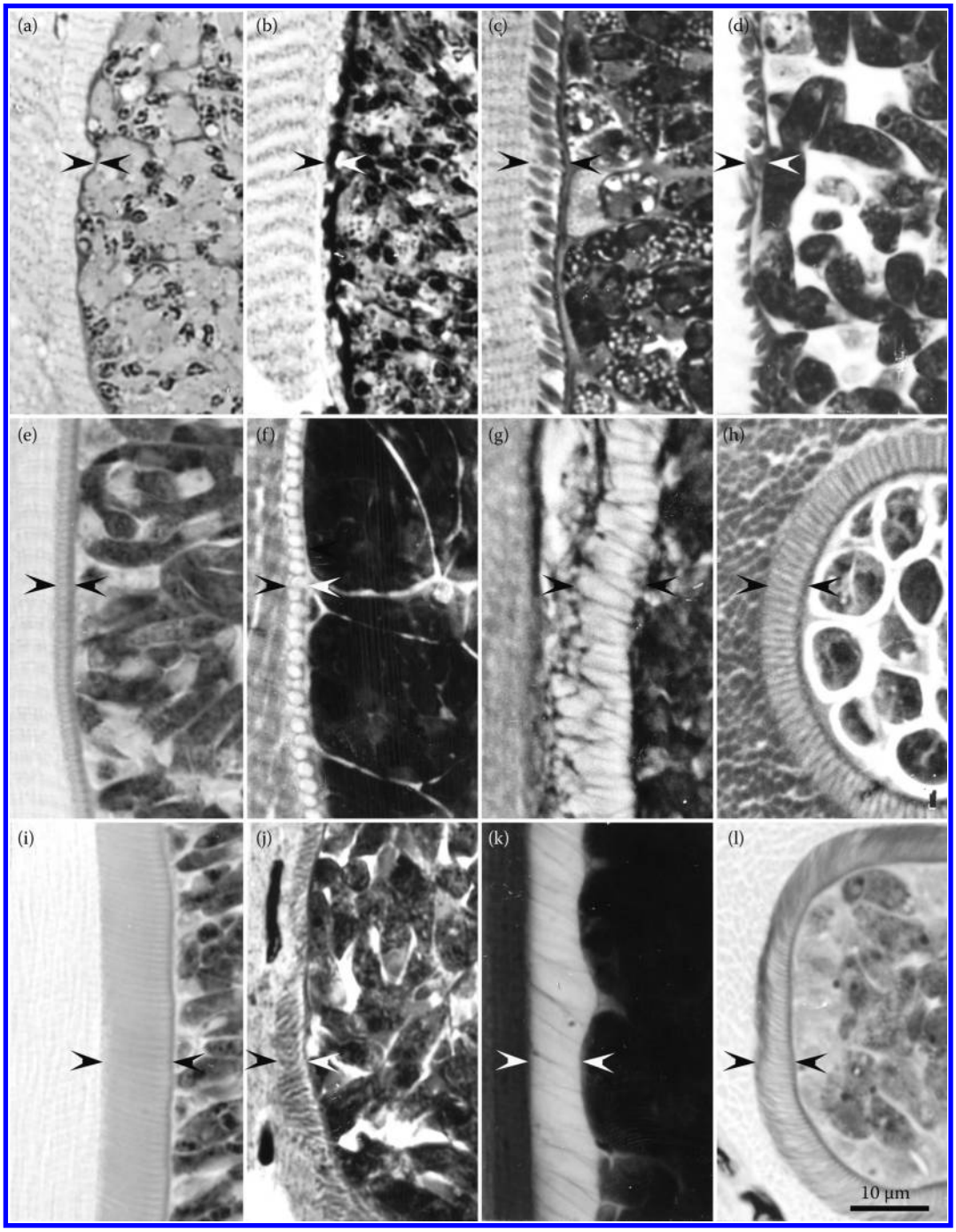


Figure 1.10 Light microscopic appearance of *Sarcocystis* sarcocyst walls. Bar applies to all parts. Arrowheads indicate the villar protrusions (vp) or lack of it on cyst walls. (a and b) Thin-walled *S. idahoensis*. The wall is smooth in (a) and irregular in (b). (c) *S. campestris* with vp. (d) *S. microti* with small vp of uneven length. (e) *S. odocoileocanis* with small stubby vp. (f) *S. hemionilatrantis* with inverted T-like vp. (g) *S. youngi* with vp of uneven width. (h and i) *S. hemioni* with thick wall with long vp. (j) *S. peromysci* with fine vp. (k) *S. arieticanis* with long, hirsute vp. (l) *S. sybillensis* with thick tufts of fine vp.

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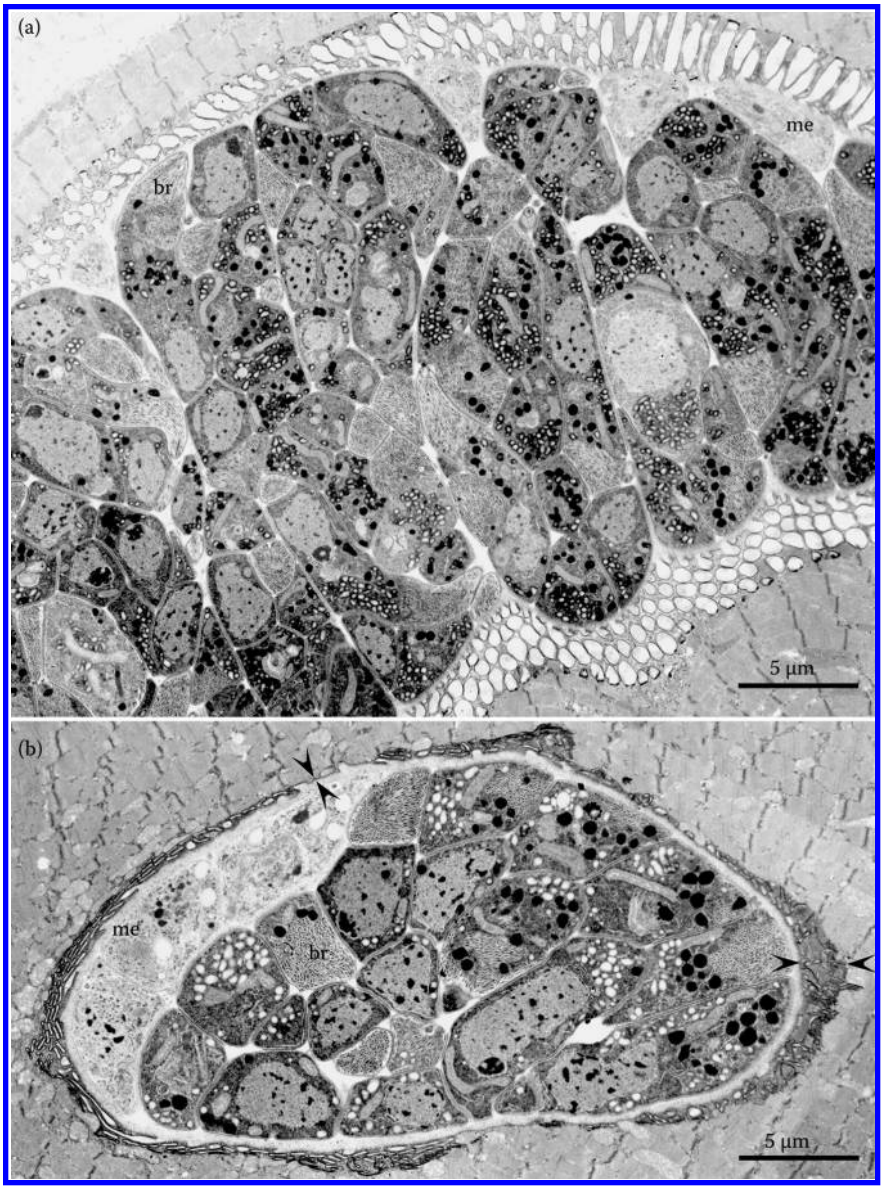


Figure 1.11 TEM of 2 mature sarcocysts in sheep at 103 DPI. Both sarcocysts contain numerous bradyzoites (br) and a few metrocytes (me). (a) *S. tenella*. The sarcocyst wall is uniformly thick. The bradyzoites are arranged in packets and separated by septa. (b) *S. arieticanis*. Septa were not found. The sarcocyst wall is thin at some places and thick at others (arrowheads).

For the following description of the life cycle and structure of *Sarcocystis*, *S. cruzi* will serve as the example. Examples from other species are discussed when information on *S. cruzi* is deficient or at variance.

Dogs, coyotes, red foxes, and possibly, wolves, jackals, and raccoons, are the definitive hosts, whereas bison (*Bison bison*) and cattle (*Bos taurus*) are the intermediate hosts for *S. cruzi*.[361,586] The definitive host becomes infected by ingesting muscular or neural tissue containing mature sarcocysts. Bradyzoites are liberated from the sarcocyst by digestion in the stomach and intestine.

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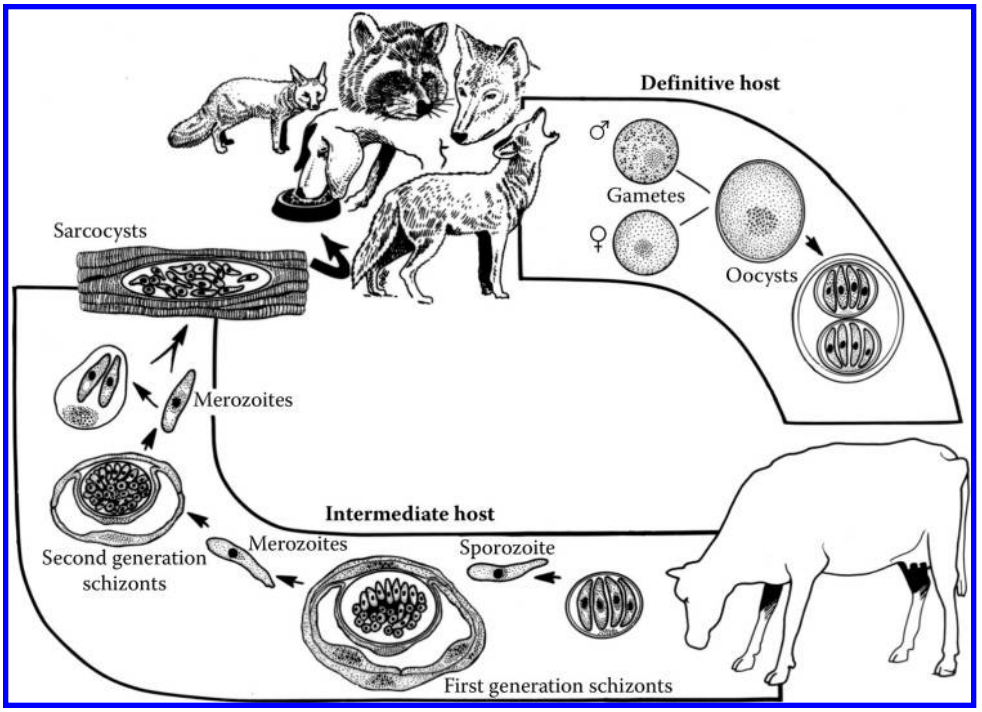


Figure 1.12 Life cycle of *S. cruzi*.

Bradyzoites move actively, penetrate the mucosa of the small intestine, and transform into male (micro) and female (macro) gamonts. Within 6 h of ingesting infected tissue, gamonts were found within a PV in goblet cells near the tips of villi or the lamina propria (Figure 1.13a). In certain species of *Sarcocystis* (e.g., *S. idahoensis*), gametogony is delayed for several days after ingestion of sarcocysts.[111,112] The ratio of macrogamonts to microgamonts is approximately 95:5. Macrogamonts are ovoid to round, 10–20 µm in diameter, and contain a single nucleus with compact chromatin. Microgamonts are ovoid to elongated, and contain 1 to several nuclei. The microgamont vesicular nucleus divides into several nuclei (usually up to 15), and, as the microgamont matures, the nuclei move towards the periphery of the gamont (Figure 1.13b–e). In *S. cruzi*, mature microgamonts, which are 7×5 µm, contain 3–11 slender gametes (Figure 1.13e). The microgametes, which are 4×0.5 µm in size, consist of a compact nucleus and 2 flagella. Microgametes liberated (Figure 1.13f) from the microgamont actively move to the periphery of the macrogamont, where their membranes fuse and only the nucleus of the microgamont passes into the macrogamont. After fertilization,[1598] a wall develops around the zygote and the oocyst is formed. The entire process of gametogony and fertilization can be completed within 24 h, but it is asynchronous. Thus, gamonts and oocysts can be found at the same time. The location of gametogony, and the type of cell parasitized, varies with species of *Sarcocystis* and stage of gametogenesis. For example, gamonts of *S. muris*, *S. hirsuta*, and *S. cruzi* initially develop in goblet cells at or near the surface of the intestine, whereas those of *S. idahoensis* develop in enterocytes next to the basement membrane adjoining the lamina propria. Occasionally, oocysts have been found in the mesenteric lymph nodes.[125,1604]

It is assumed that the infected enterocyte or goblet cell moves to the lamina propria, where sporulation occurs (Figures 1.14 and 1.15). Initially, the sporont was granular, eosinophilic, and filled the young oocysts. During the initial stages of sporont condensation, a clear cap-like structure was found in *S. cruzi*[360] and *S. idahoensis*.[111] When the sporont condensed further, a similar clear

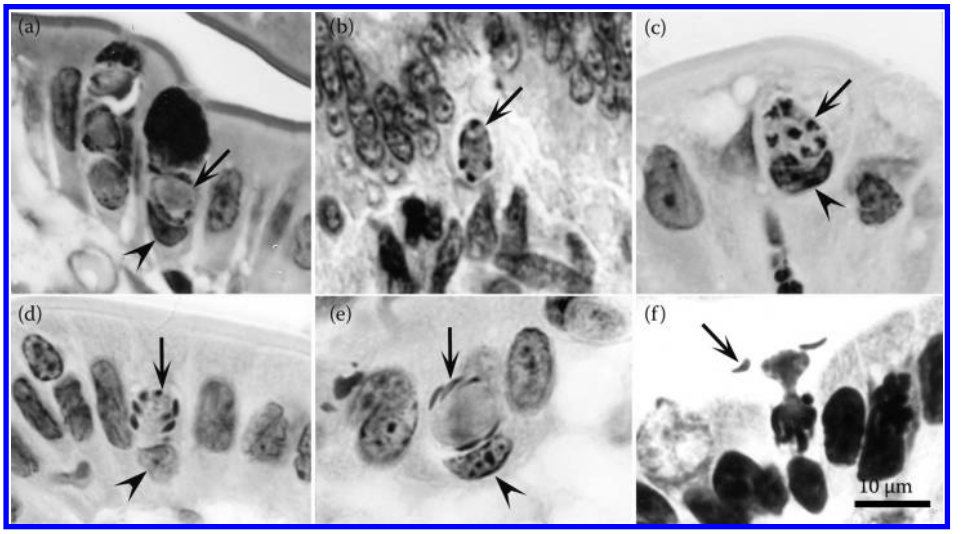


Figure 1.13 Gamonts of *S. hirsuta* (a, c, d, and f) and *S. cruzi* (b and e) in the small intestines of cats (*S. hirsuta*) and coyotes (*S. cruzi*), respectively. Each arrowhead points to a host-cell nucleus, and arrows point to parasites. (a) Four oval early gamonts in 2 adjacent goblet cells. (b) Microgamont (arrow) with 5 nuclei. (c) Microgamont with 8 nuclei. (d) Microgamont (arrow) with 7 peripheral nuclei. (e) Mature microgamont with gametes. (f) Ruptured microgamont liberating microgametes (arrow). (From Dubey, J.P., 1983. *Parasitology* 86, 7–9; Dubey, J.P., 1982. *J. Protozool.* 29, 591–601. With permission.)

area appeared at the opposite end. The oocyst contained one large nucleus with 1 or 2 prominent nucleoli, and several PAS-positive granules. As sporulation progressed, the nucleus elongated and became parallel to the longitudinal axis of the sporont.[111,174,1648] The elongated nucleus divided into 2 nuclei, 1 at each pole of the sporont. These nuclei underwent a second transverse division, and the sporont cytoplasm divided transversely into 2 sporoblasts (Figure 1.14). Each sporoblast contained 2 nuclei. The nuclei moved towards the opposite poles of each sporoblast, and the sporoblasts became surrounded by an eosinophilic wall; sporoblasts became sporocysts. Four sporozoites formed in each sporocyst, apparently by a third nuclear division. Because sporulation is asynchronous, unsporulated and sporulated oocysts are found simultaneously (Figure 1.14).

Sporulated oocysts are generally colorless, thin-walled (<1 µm), and contain 2 elongated sporocysts. An oocyst residuum and a micropyle are absent. Each sporocyst contains 4 slender sporozoites and a granular sporocyst residuum, which may be compact or dispersed, but a Stieda body is absent. Because sporozoites are flexed in the sporocyst, all 4 are often not seen in a single plane of focus (Figures 1.14 and 1.15). Each sporozoite has a central to terminal nucleus, and several cytoplasmic granules, but there is no refractile body.

The oocyst wall is thin and often ruptures (Figure 1.16). Free sporocysts, released into the intestinal lumen, are passed in feces (Figure 1.16). Occasionally, unsporulated and partially sporulated oocysts are excreted in feces. The prepatent and patent periods vary, but for most *Sarcocystis* species, oocysts are first excreted in the feces between 7 and 14 days after ingesting sarcocysts.

The intermediate host becomes infected by ingesting sporocysts in food or water. Sporozoites excyst from sporocysts in the small intestine. The fate of the sporozoite, from the time of ingestion of the sporocyst until initial development in mesenteric lymph node arteries, is not known. Sporozoites of *S. cruzi* were first found in the lumen and in the endothelium of arteries 4–7 DPI. At such time, free zoites have been seen in arteries in mesenteric lymph nodes. First-generation schizogony begins in endothelial cells as early as 7 DPI and may be completed as early as 15 DPI (Figure 1.17). Second-generation schizonts have been seen in endothelium from 19 to 46 DPI, predominantly in capillaries,

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Figure 1.14 Sporulating *S. cruzi* oocysts in the lamina propria of the small intestine of an experimentally infected coyote (a) unsporulated oocyst, (b) oocyst with 2 sporoblasts, (c) oocysts with 2 sporocysts, (d) sporulated oocyst with 2 sporocysts and a thin oocyst wall (arrowhead). Unstained.

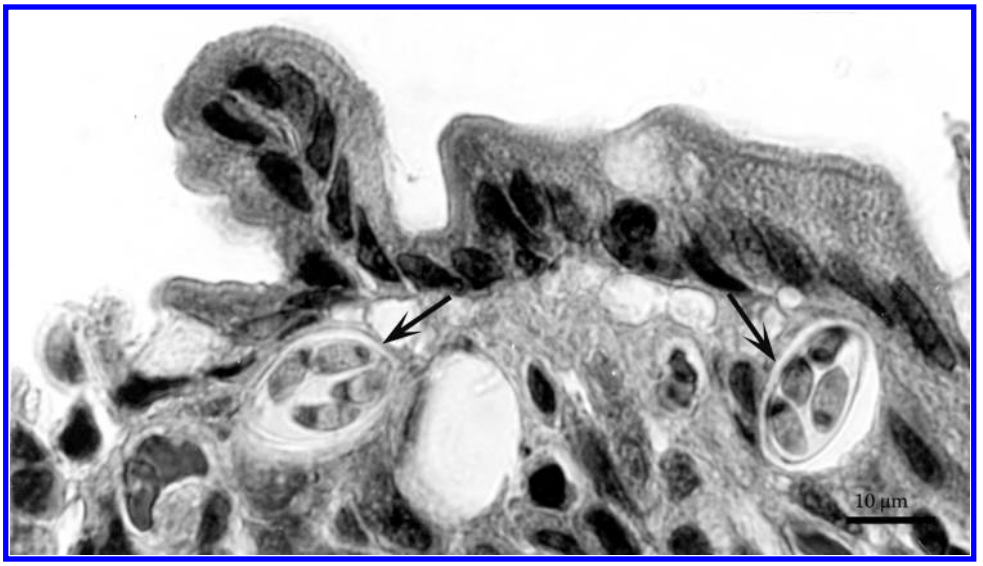


Figure 1.15 Two *S. cruzi* sporocysts (arrows), each with 4 sporozoites in the lamina propria of the small intestine of a dog. H and E stain. (From Dubey, J.P., 1976. *J. Am. Vet. Med. Assoc*. 169, 1061–1078. With permission.)

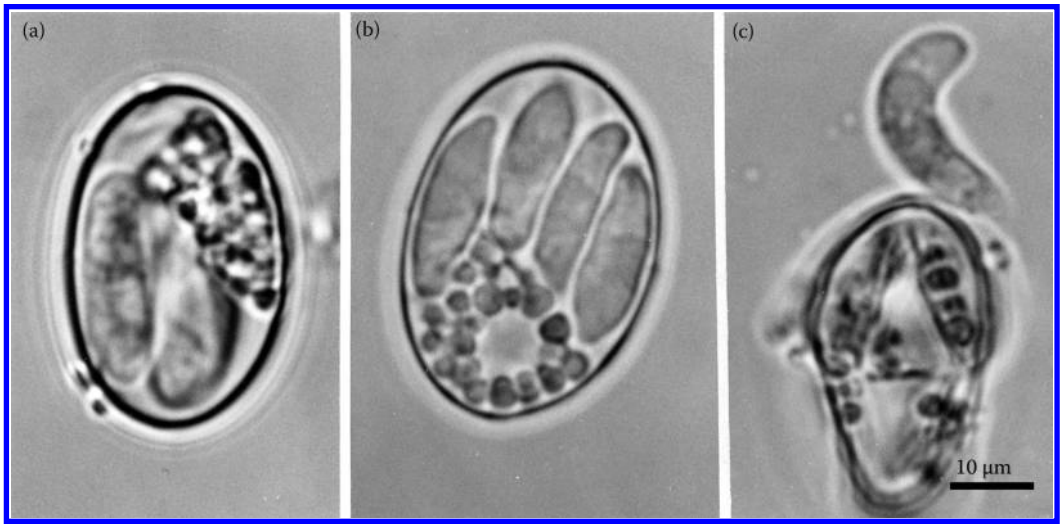


Figure 1.16 *In vitro* excystation of sporozoites of *S. cruzi* in trypsin-bile excystation fluid. (a) Untreated sporocyst. (b) Treated sporocyst with 4 sporozoites and sporocyst residuum. (c) One excysted sporozoite and collapsed sporocyst wall. (Adapted from Fayer, R., Leek, R.G., 1973. *Proc. Helminthol. Soc. Wash*. 40, 294–296.)

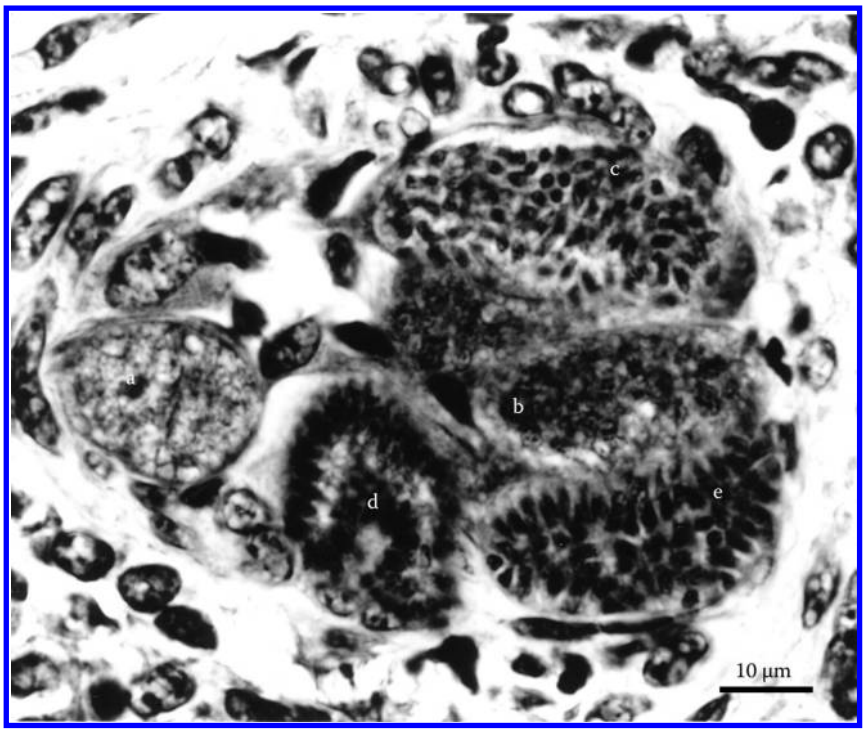


Figure 1.17 Development of first-generation schizonts of *S. cruzi* in a mesenteric lymph-node artery of an experimentally infected calf. Progressive developing stages are (a) lobulated nucleus, (b) undifferentiated nuclei, (c) differentiated nuclei, (d) developing merozoites, and (e) mature with merozoites. Iron hematoxylin plus Giemsa stain. (From Dubey, J.P., 1982. *J. Protozool.* 29, 591–601. With permission.)

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but also in small arteries, virtually throughout the body. These schizonts were most numerous in the glomeruli of the kidney (Figure 1.18). Immature schizonts stained with H and E are basophilic and stain lighter than the host tissue. The nucleoplasm of the schizont is diffuse and difficult to recognize in 5-µm thick-histological sections, but it appears clear in 1- to 3-µm sections (Figure 1.19a). The nucleus becomes lobulated (Figure 1.19b) and divides into several lobes. Merozoites form at the

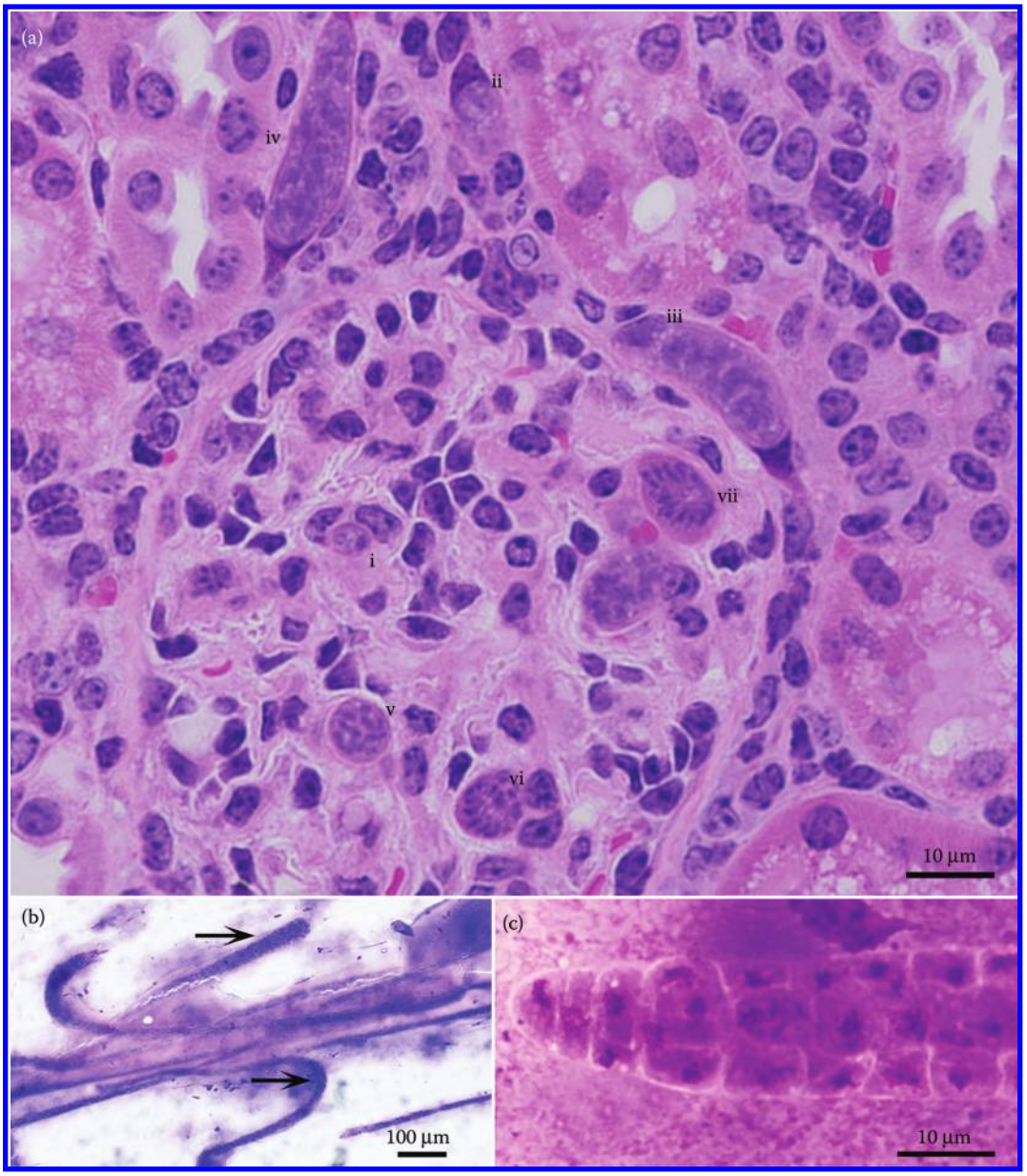


Figure 1.18 *Sarcocystis* schizonts and sarcocysts. (a) Second-generation schizonts within and outside the renal glomerulus of a calf experimentally infected with *S. cruzi*, in presumed order of development from uninuclate schizont (i) to mature (vii). Two schizonts (ii, iv) are located in capillaries in renal parenchyma. H and E stain. (b) Immature thread-like *S. speeri* sarcocysts (arrows) in a skeletal muscle smear of an experimentally infected KO mouse. 22 DPI. Giemsa stain. (c) Higher magnification of a terminal end of a sarcocyst in Figure 1.18b. Note rows of magenta-colored metrocytes.

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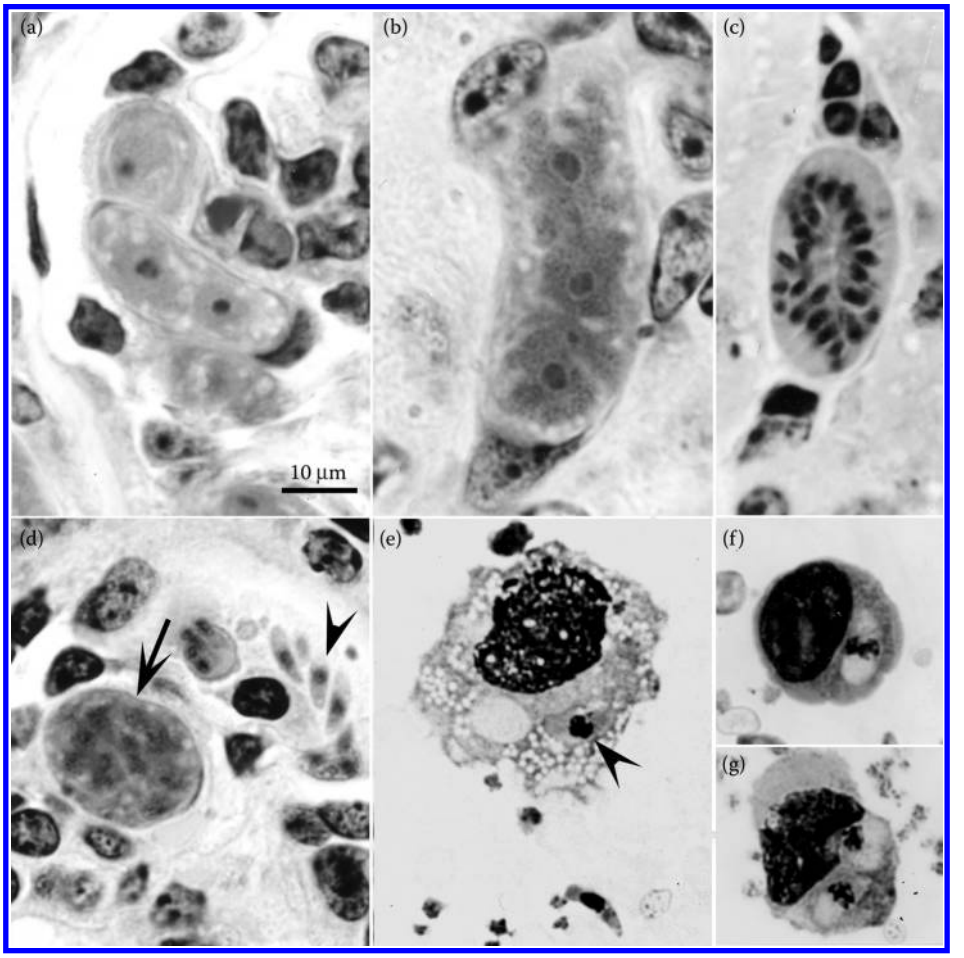


Figure 1.19 Second-generation schizonts (a–d) and intravascular zoites (e–g) of *S. cruzi* in experimentally infected calves. (a) Immature schizont with 1 or 2 nuclei. (b) Lobed nucleus with 3 prominent nucleoli. (c) Peripherally arranged merozoites. (d) Individual merozoites (arrowhead) and a multinucleated schizont (arrow). (e–g) Merozoites in peripheral blood. (e) Intracellular merozoite (arrowhead) in a monocyte and an extracellular merozoite. (f) Binucleated merozoite in a mononuclear cell. (g) Mononuclear cell with 2 merozoites. (From Dubey, J.P., 1982. *J. Protozool.* 29, 591–601. With permission.)

periphery (Figure 1.19c). The shape and size of schizonts vary considerably. Schizonts observed in skeletal muscle are longer than those seen in other tissues. Both first- and second-generation schizonts are located within the host cytoplasm, and are not surrounded by a PV.

Merozoites have been found in peripheral blood smears 24–46 DPI coincident with the maturation of second-generation schizonts (Figure 1.19). Merozoites in blood are extracellular or located within unidentified mononuclear cells (Figure 1.19e). Intracellular merozoites contain 1 or 2 nuclei, and some divide to form 2 merozoites, apparently by endodyogeny[582] (Figure 1.19f and g). Extracellular merozoites often appear degenerate. Division in blood cells has not been seen in some species of *Sarcocystis* (e.g., *S. hirsuta*). Individual merozoites have been seen in macrophage-like cells in tissues of animals infected with some species in cattle, deer, pig, and sheep.[75,362,379,1296]

The number of generations of schizogony and the type of host cell may vary with each species of *Sarcocystis*, but trends are apparent. For example, all species of *Sarcocystis* of large domestic animals (sheep, goat, cattle, pigs) form first- and second-generation schizonts in vascular endothelium,

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whereas only a single precystic generation of schizogony, has been found in the *Sarcocystis* species of small mammals. The species found in mice generally develop in hepatocytes, whereas *Sarcocystis* species in rats develop both in the endothelial cells and in the connective tissue cells of the spleen, kidneys and lungs.[1363] Schizonts of *S. neurona* in the horse are found only in the neural cells (see Chapter 3). *S. hemionilatrantis* represents a variation, with the first 2 generations of schizogony in vascular endothelium and an additional third or fourth generation in macrophages in muscles (Figure 1.20). Still other variations are seen in cattle, in which the second-generation schizont of *S. cruzi* develops in virtually every organ, whereas those of *S. hirsuta* are restricted to the muscles.

Sarcocystis schizonts divide by a special form of endopolygeny (Section 1.3.1.1.1) (Figures 1.21 through 1.23). Usually, schizonts are time-limited stages that disappear before sarcocysts form. However, schizonts of *S. falcatula* develop in blood vessels of birds (budgerigar) from day 2 to 5.5 months PI,[1626] with 2 distinct peaks of schizogony at 7–8 DPI and 28 DPI. All schizonts are structurally similar. The site of schizogony shifts progressively from capillaries to venules to veins.[1626] Merozoites liberated from the terminal generation of schizogony initiate the sarcocyst formation.

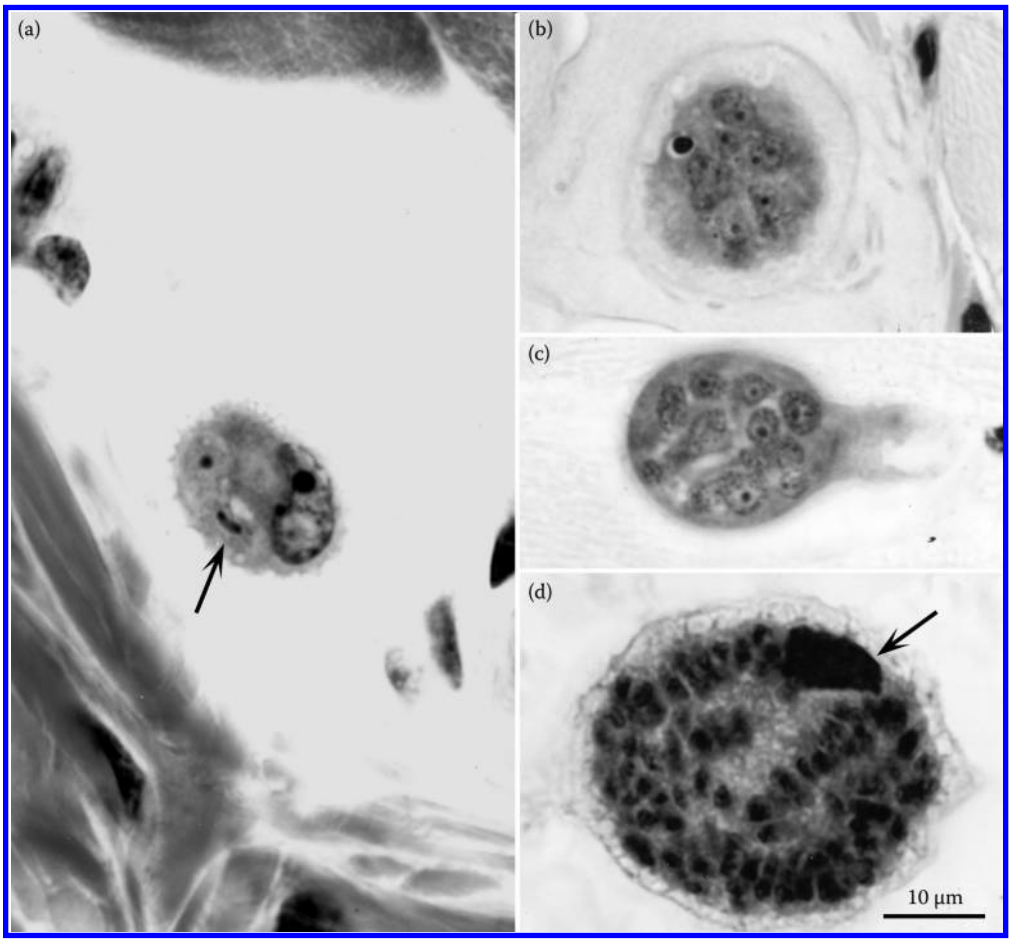


Figure 1.20 Third-generation *S. hemionilatrantis* schizonts in macrophages in histological sections of the skeletal muscles of a mule deer. H and E stain. (a) Binucleate schizont with 1 dividing nucleus (arrow). Myocytes are on the top right and connective tissue cells are at the bottom left, and the infected macrophage is in the edematous perimysial space. (b and c) Multinucleated schizonts. (d) Nearly mature schizont. The host-cell nucleus (arrow) is indented. (From Dubey, J.P., Kistner, T.P., Callis, G., 1983. *Can. J. Zool.* 61, 2904–2912. With permission.)

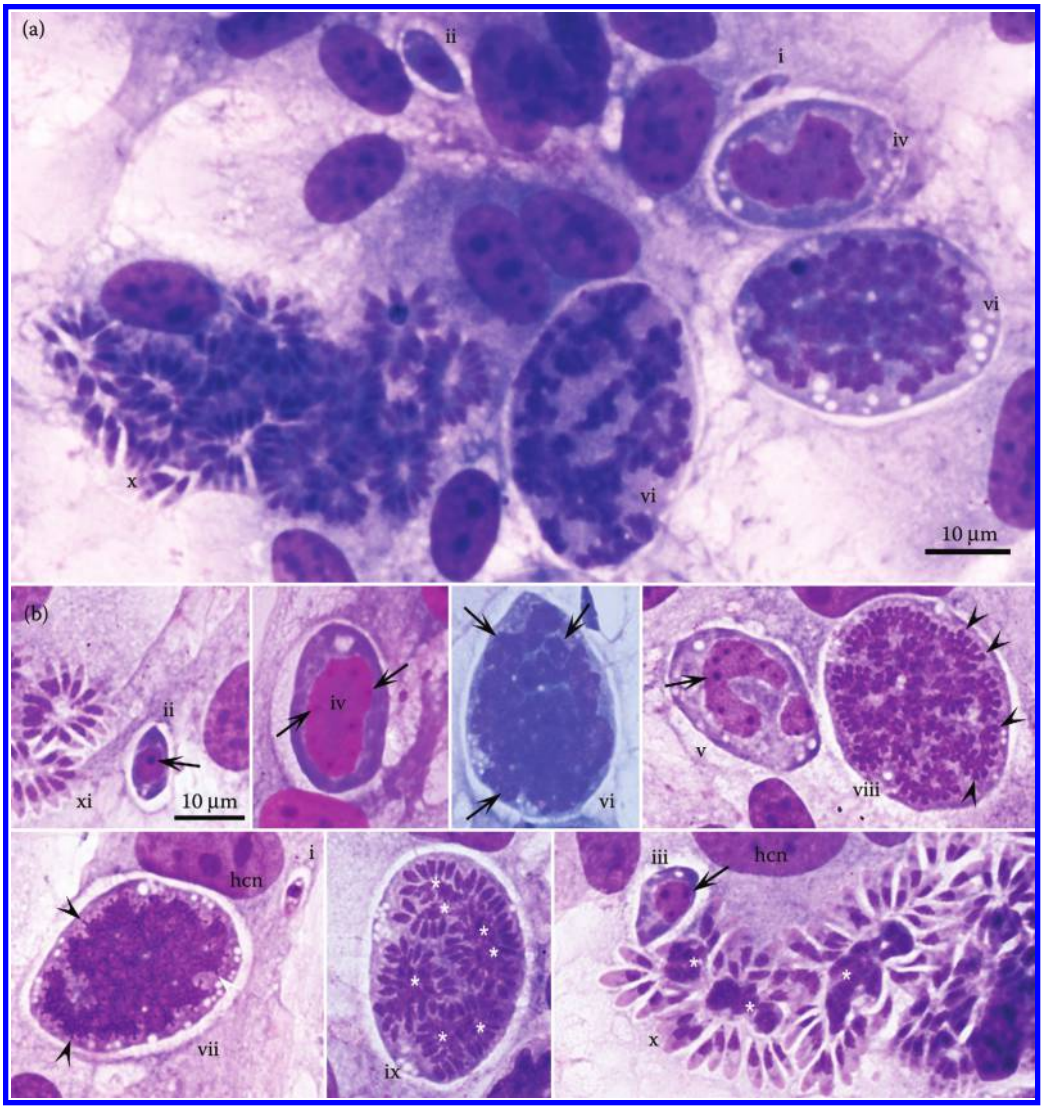


Figure 1.21 Development of *S. falcatula* schizonts in bovine turbinates cells. Illustrations are from 1 slide, 5 days after seeding with merozoites. Giemsa stain (Slide courtesy of D. S. Lindsay). hcn = host-cell nucleus. There is considerable variability in staining of different stages of the parasite (a) Different developmental stages in one field. (b) Stages selected from different fields to show details. Stages (i–xi), in presumed order of development. Intracellular merozoite (i) with a single nucleus, earliest schizont (ii) with a prominent nucleus and 1 nucleolus (arrow), an immature schizont with 3 nucleoli (iii), and a schizont with 7 nucleoli (iv). Thereafter, the nucleus has become lobulated (v, vi). In (vii, viii) the ends of merozoites are forming. In (ix) the merozoites are arranged in different groups. In (x) the merozoites have formed around residual bodies (*). Note different shapes and sizes of merozoites, some are more slender than others. Mature schizonts without residual bodies are shown in (xi).

The intracellular merozoite surrounded by a PV becomes round to ovoid (metrocyte) (Figure 1.24). After repeated divisions, the sarcocyst is filled with bradyzoites, the presence of which is the infective stage for the predator. Sarcocysts generally become infectious around 75 DPI, but there is considerable variation among species of *Sarcocystis*. Immature sarcocysts and schizonts are not infectious for the definitive host.

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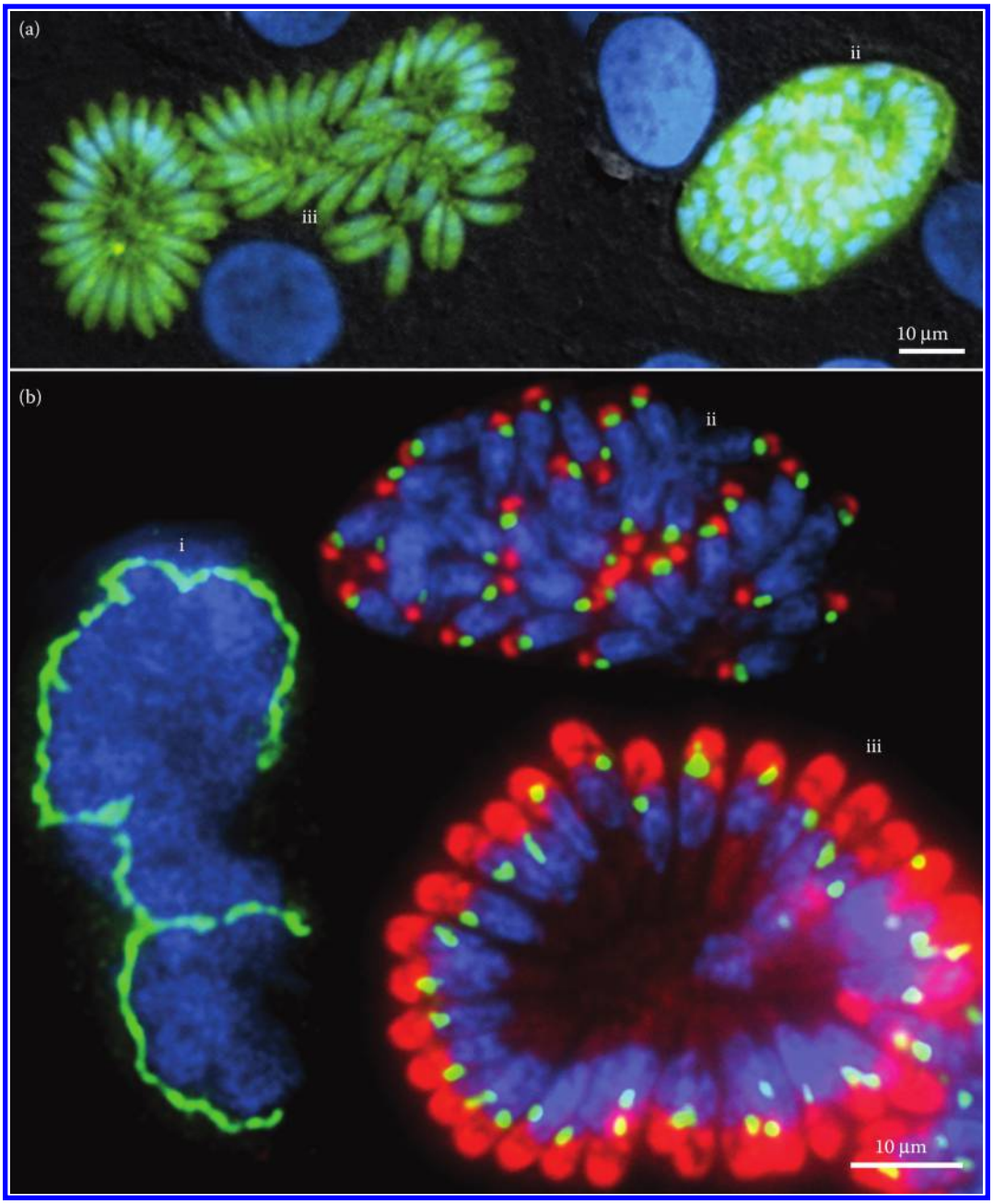


Figure 1.22 Immunofluorescence of *S. neurona* schizonts in BT cells. Note lobulated nucleus in (i), 24 h, merozoite formation in (ii), 48 h, and a mature schizont with merozoites in (iii), 72 h post infection. (a) Transgenic clone of *S. neurona* expressing yellow fluorescent protein (YFP). Differential interference contrast image with epifluorescence image overlay, showing a multilobed schizont and a mature schizont of a clone of *S. neurona* that stably expresses YFP. The host cell and parasite nuclei were stained with 4′,6-diamino-2-phenylindole (DAPI) (blue). (b) Genesis of apicoplast during schizogony of *S. neurona*. The apicoplast is stained green, micronemes are red, and nucleus/nuclei are blue. The apicoplast follows the nucleus and divides/fragments during the nuclear division. Micronemes (stained red) are absent in the stage expressed in (i). The nucleus is stained blue. (From Vaishnava, S. et al., 2005. *J. Cell Sci*. 118, 3397–3407. With permission.)

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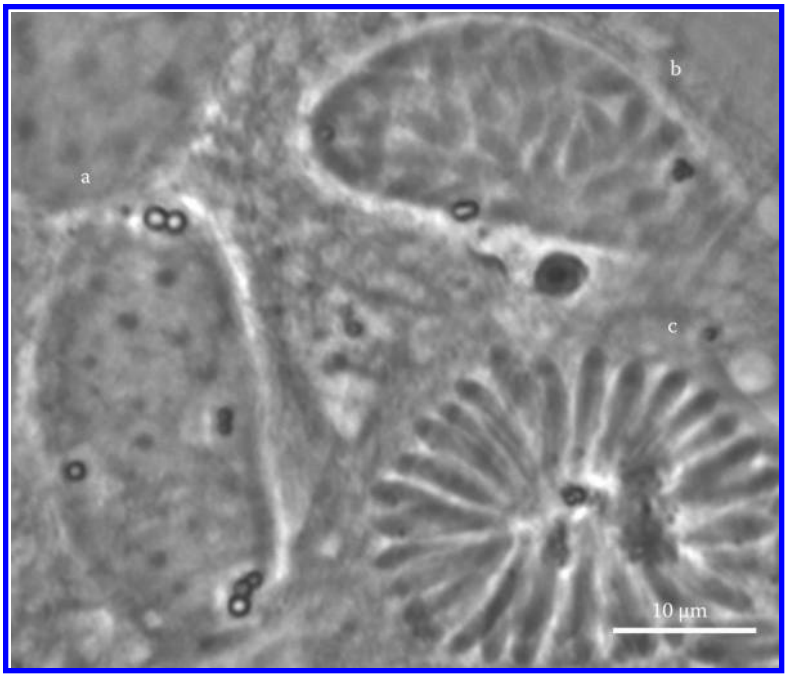


Figure 1.23 Same image as in Figure 1.22b. Phase contrast. (From Vaishnava, et al. 2005. *J. Cell Sci.* 118, 3397–3407. With permission.)

Sarcocysts may persist for the life of the host, but many begin to disappear after 3 months (Figure 1.25).

Rupture of sarcocysts may initiate host response, but the released bradyzoites do not initiate new development. In other words, there is no reactivation of chronic infection, irrespective of the immune status of the host. Experimentally, merozoites (from cell cultures) of certain species (e.g., *S. neurona*) are infectious to immunodeficient mice, but bradyzoites do not infect mice.[470]

1.3 ULTRASTRUCTURE

Sarcocystis spp. are single-cell, eukaryotic organisms that contain a nucleus, nucleolus, endoplasmic reticulum, ribosomes, a plastid, a Golgi complex, and a mitochondrion. They also have organelles that are characteristic of the phylum Apicomplexa, such as apical rings (also called conoidal or preconoidal rings), polar rings, a conoid (which may be absent in some apicomplexans, i.e., *Plasmodium*), a pellicle, subpellicular microtubules, micropores, rhoptries, and micronemes.

There are numerous reports on the ultrastructure of *Sarcocystis* spp. sarcocysts (see References **5, 6, 8–10, 23, 39, 82, 91, 96, 97, 99, 126, 127, 137, 172, 173, 190, 201, 220, 222, 224–226, 240, 244, 245, 253, 273, 289, 326, 327, 360, 367–370, 378, 380, 383, 385, 393, 394, 396, 446, 452, 453, 457, 461, 466, 467, 474, 478, 479, 525, 530, 533, 535, 536, 538, 540, 542, 543, 636, 673, 677, 691–696, 698, 707, 708, 787, 797, 802, 819, 824, 833, 862, 863, 865, 895, 926, 936, 938, 961, 963, 964, 1112, 1132, 1135, 1137, 1142, 1164, 1167–1176, 1178, 1227, 1273, 1274, 1290–1293, 1296, 1299, 1303, 1309, 1310, 1313, 1316, 1318, 1326, 1348, 1367, 1369, 1404, 1406–1408, 1504, 1518, 1519, 1552, 1561, 1564, 1565, 1567–1569, 1576, 1583–1585, 1597, 1609, 1610, 1633, 1651, 1654, 1681, 1684, 1708, 1752, 1793, 1794, 1796, 1803, 1825, 1845, 1867, 1868, 1871, 1875, 2227**), schizonts (see References **39, 75, 184, 356, 359, 366, 436, 534, 708, 808, 1142, 1178, 1306, 1329, 1347, 1363,**

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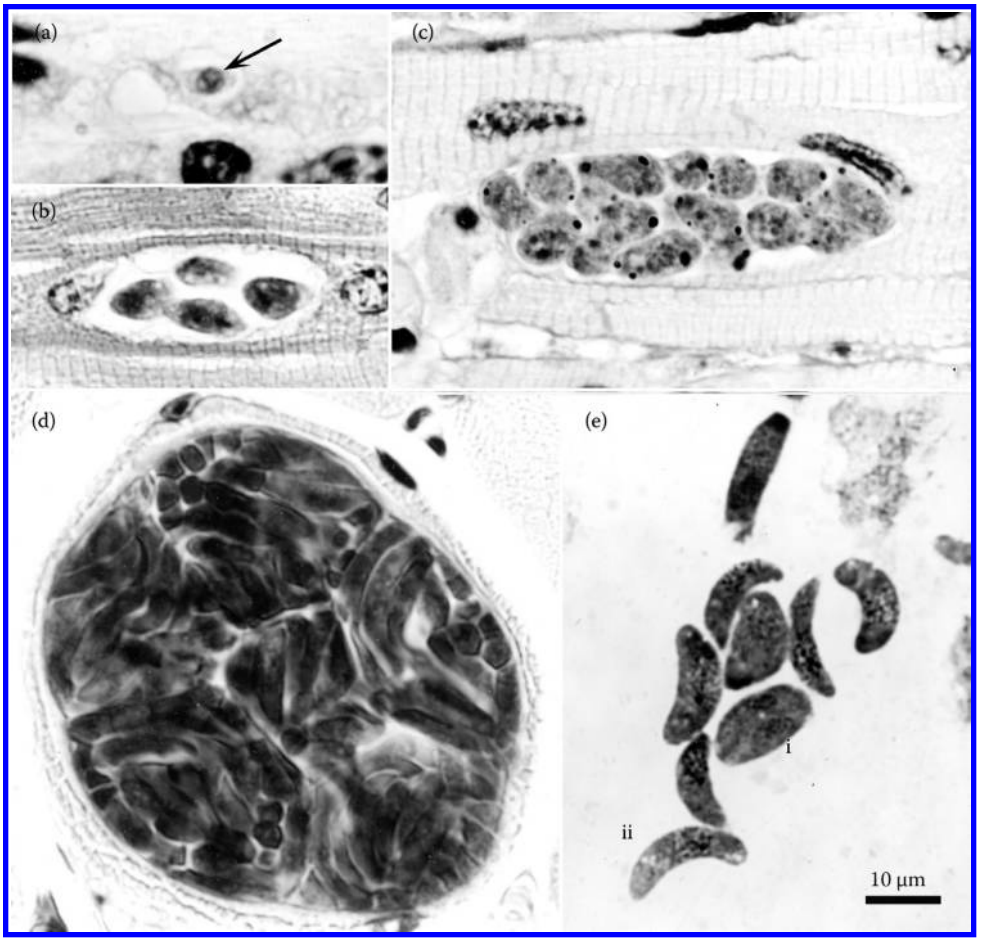


Figure 1.24 *S. cruzi* sarcocysts in myocytes of experimentally infected calves. (a–d), histological sections. (a and d), H and E stain, (c), PAS reaction, G, smear. Giemsa stain. (a) Merozoite in a parasitophorous vacuole (arrow), probably a unizoite sarcocyst. (b) Sarcocyst with 4 metrocytes. (c) Sarcocyst with numerous metrocytes. (d) Mature sarcocyst with banana-shaped bradyzoites. (e) Bradyzoites (i) and metrocyte (ii) with 2 organisms. Giemsa stain. (From Dubey, J.P., 1982. *J. Protozool*. 29, 591–601. With permission.)

1368, 1561, 1585, 1620, 1626, 1627, 1649, 1650, 1652, 1660, 1752, 1798), and gamonts, oocysts, and sporozoites (see References **86, 531, 537, 539–541, 543, 887, 888, 1018, 1142, 1165, 1177, 1178, 1364–1366, 1566, 1598, 1648, 1790, 1868**). We have summarized available information.

1.3.1 Sarcocysts

The ultrastructure section begins with sarcocysts because of the importance of this stage in transmission, and as a taxonomic aid. Each *Sarcocystis* species produces a sarcocyst wall that often has unique ultrastructural characteristics that can be used to distinguish it from other species within the same intermediate host.

Sarcocyst development begins when a merozoite enters a myocyte (Figure 1.26) or neural cell. The merozoite resides in a PV and is surrounded by a PVM which appears to develop immediately into a primary sarcocyst wall (PSW). The PSW consists of a PVM plus an underlying electron-dense layer. During transformation of a merozoite into a metrocyte, many of the organelles of the apical

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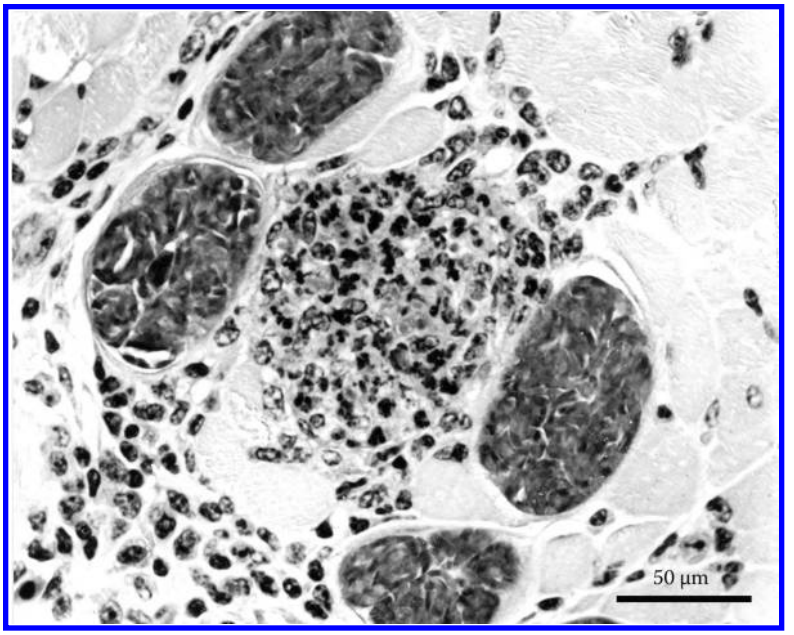


Figure 1.25 A degenerate sarcocyst in the center, surrounded by mononuclear cells and infiltrated by granulocytes, and 4 intact sarcocysts. H and E stain. (From reference Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res*. 43, 2147–2164. With permission.)

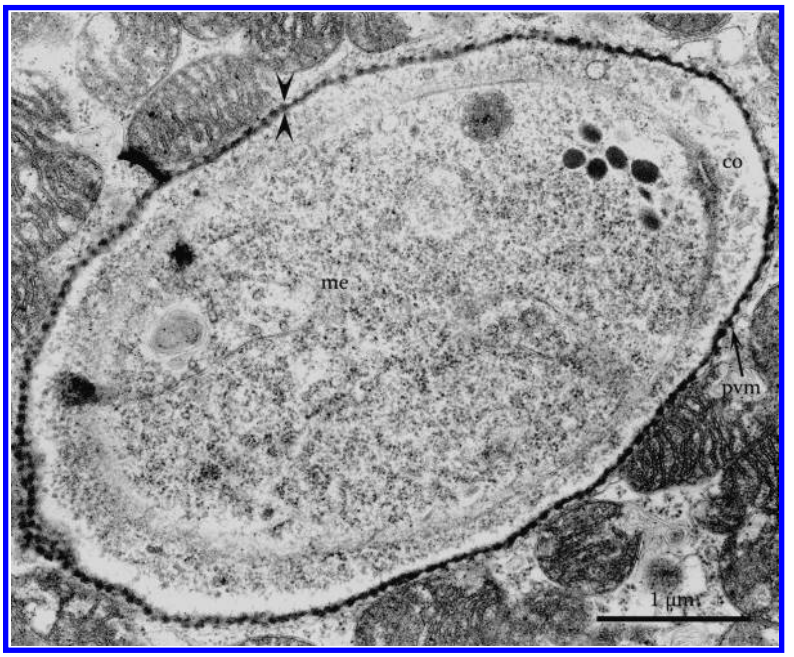


Figure 1.26 TEM of unizoite sarcocyst of *S. raushorum* at 9 DPI. The merozoite is in a late stage of transformation to a metrocyte (me). The infected myocyte has already formed a primary sarcocyst wall (opposing arrowheads), and remnants of the conoidal (co) complex are visible. (Courtesy of R. J. Cawthorn.)

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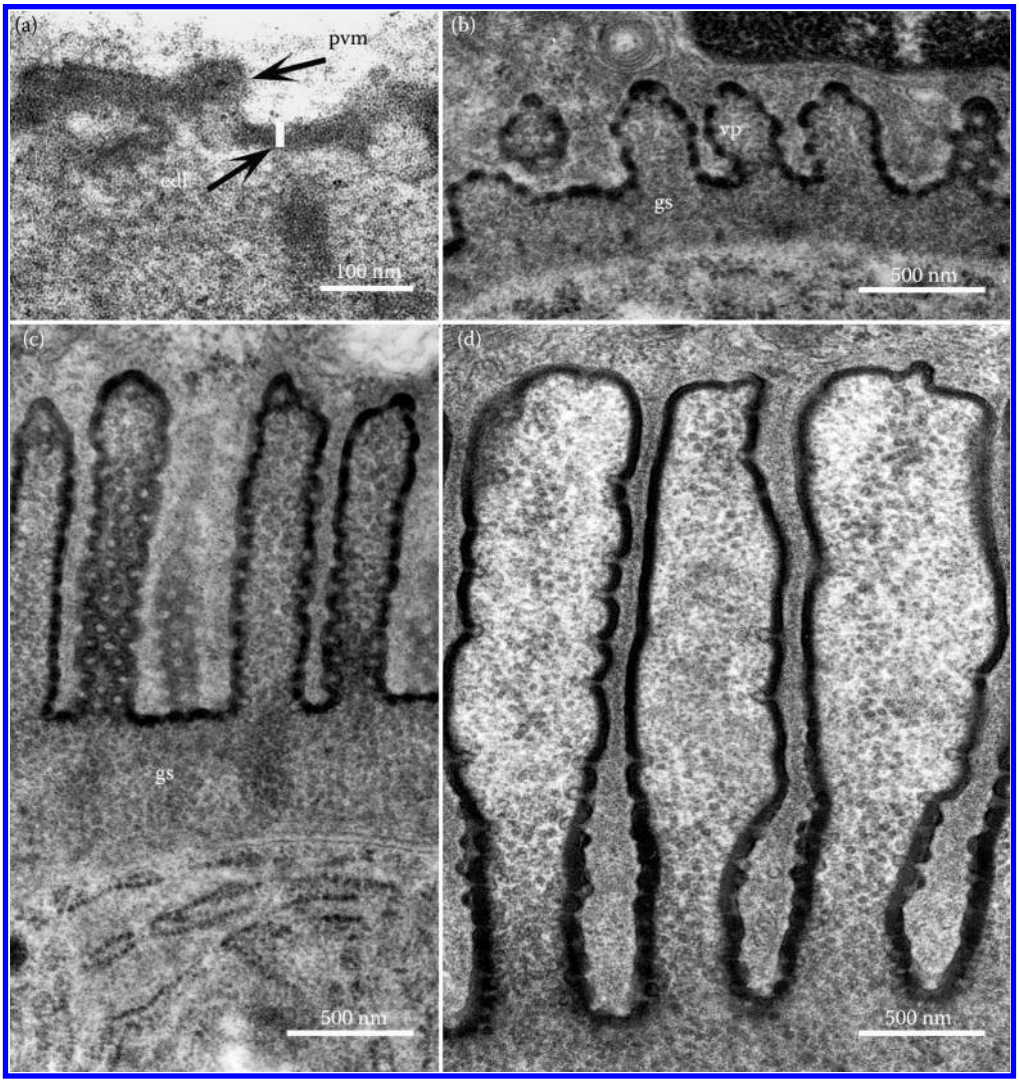


Figure 1.27 TEM of *S. singaporensis* sarcocyst wall formation. (a) Initial stage during at 25 DPI. The pvm of the sarcocyst wall has formed a bleb and is lined with an electron-dense layer (edl). (b–d), 30 DPI. (b) The primary sarcocyst wall has folded to form short villar protrusions (vp), (c). The vp are elongate with parallel sides and the granular layer is about twice as thick as in (b). (d) The vp are longer and club-shaped.

complex, such as micronemes, conoid, polar and apical rings, disappear (Figure 1.26), while ribosomes, endoplasmic reticulum, and mitochondrion become more abundant, and the nucleus becomes larger. A granular layer located immediately beneath the PSW develops early with the acquisition of minute granules and vesicles, which pinch off from invaginations in the PSW (Figure 1.27).

Mature sarcocysts of all *Sarcocystis* spp. have minute undulations in the PSW. In some species, the PSW may remain relatively simple (e.g., *S. rauschorum*), whereas other species may have highly complex (e.g., *S. fusiformis*) walls, in which the primary sarcocyst wall is folded or branched to form protrusions that project outwardly from the sarcocyst. The protrusions may contain minute granules, electron-dense bodies, microfilaments, microtubules, and small vesicles. *S. singaporensis* has a complex wall, which requires nearly 200 days to become fully formed. Figures 1.27 through

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1.30 show a series of TEMs in the development of the primary sarcocyst wall of *S. singaporensis*. At 25–30 DPI, the PSW initially consists of minute undulations only (Figure 1.27a), which soon becomes folded to form short villar protrusions (vp) (Figure 1.27b), elongate protrusions with parallel sides (Figure 1.27c), and then much larger club-shaped protrusions that are approximately 2.3 µm long (Figure 1.27d). At 176 DPI, the vp are sausage-shaped with a narrow stalk (Figure 1.28) and are 10 µm long. At 184 DPI, most sarcocysts have protrusions that are shorter and usually irregular, measuring approximately 3 µm long (Figure 1.30).

As the sarcocyst develops, the contents of the vp also change, which possibly reflects a change in the metabolic activity within the sarcocysts. The vp of early and intermediate sarcocysts are filled with small vesicles and membranous whorls, which pinch off from the PSW at indentations in the PSW (Figures 1.27b–d and 1.28a and b). In mature sarcocysts, the vp contain granules and clusters of vesicles (Figures 1.28b through 1.30).

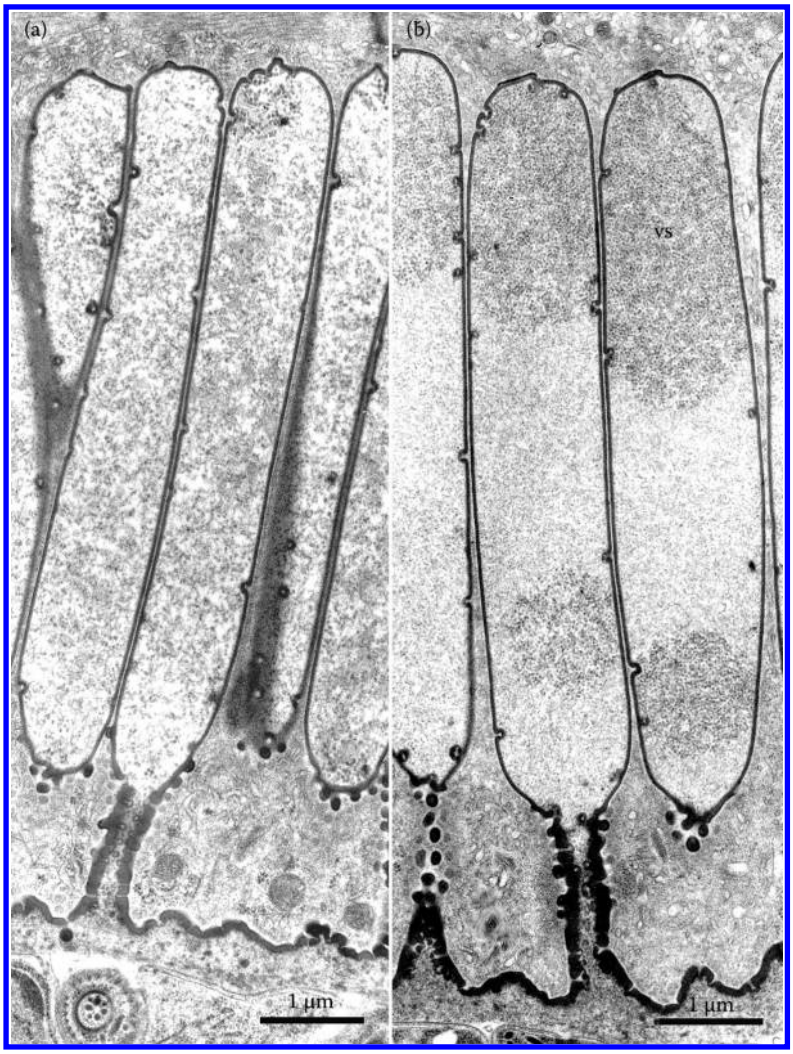


Figure 1.28 TEM of *S. singaporensis* sarcocyst at 176 DPI. (a, b) The vp have become sausage-shaped with a narrow stalk, and contain small vesicles (vs). The vp in (b) are slightly longer and wider, and contain more granules and aggregates of vesicles than in (a).

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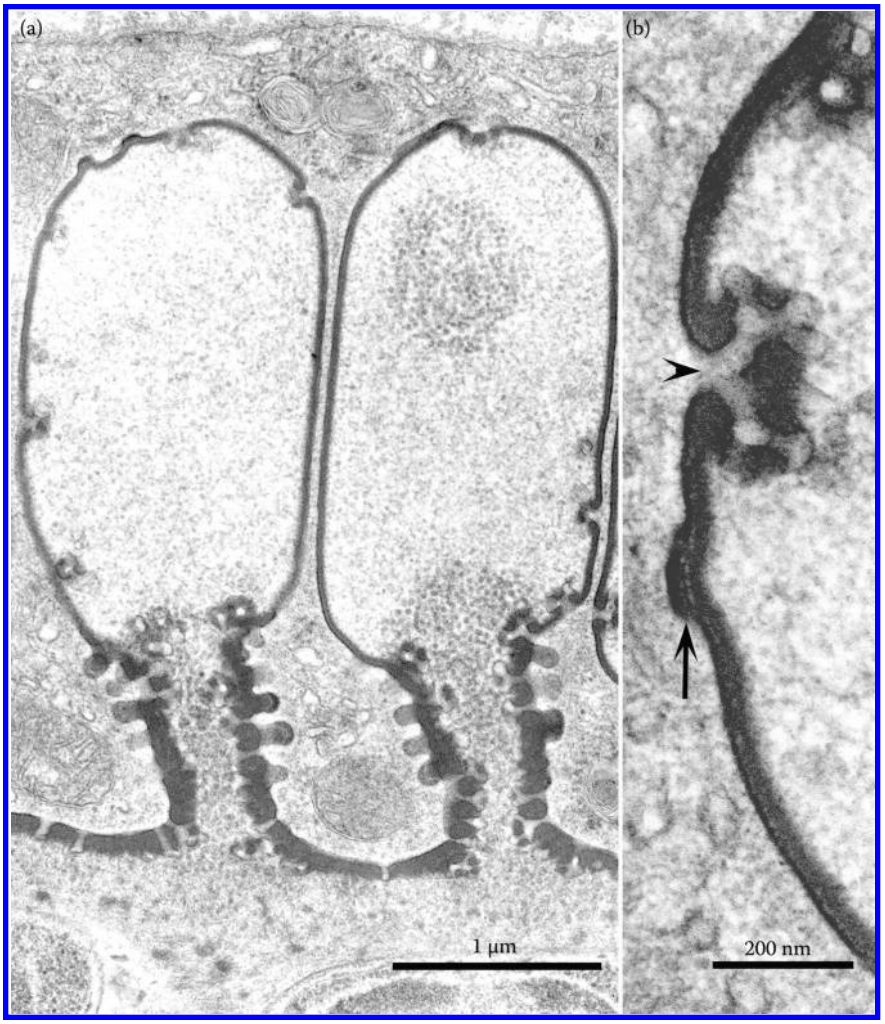


Figure 1.29 TEM of *S. singaporensis* sarcocyst at 184 DPI. (a) The vp have decreased in size to about one-fourth the length of those in Figure 1.28. (b) High magnification of the apex of a vp showing disk-shaped plaques (arrow) and indentations in the primary sarcocyst wall (arrowhead) that gives rise to small vesicles.

1.3.1.1 Metrocytes (Figures 1.31 through 1.35)

Metrocytes are ovoid, rapidly multiplying forms, which have an electron–lucent cytoplasmic matrix, numerous ribosomes, mitochondrion, 1 or more micropores, a pellicle consisting of 2 or 3 membranes, subpellicular microtubules, endoplasmic reticulum, fissures, 1 or 2 Golgi complexes, a few amylopectin granules, centrioles, electron-dense bodies, an occasional lipid body, and a diffuse nucleus (Figures 1.26, 1.31, 1.32, and 1.35). Metrocytes may also contain anlagen of 2 progeny formed by a process called endodyogeny. Metrocytes may arise from metrocytes or from dedifferentiated merozoites. Metrocytes in early stages of endodyogeny may also contain remnants of the conoid, micronemes, and rhoptries. Early sarcocysts contain only metrocytes (Figure 1.32), whereas relatively few metrocytes are contained in intermediate and mature sarcocysts (Figure 1.33).

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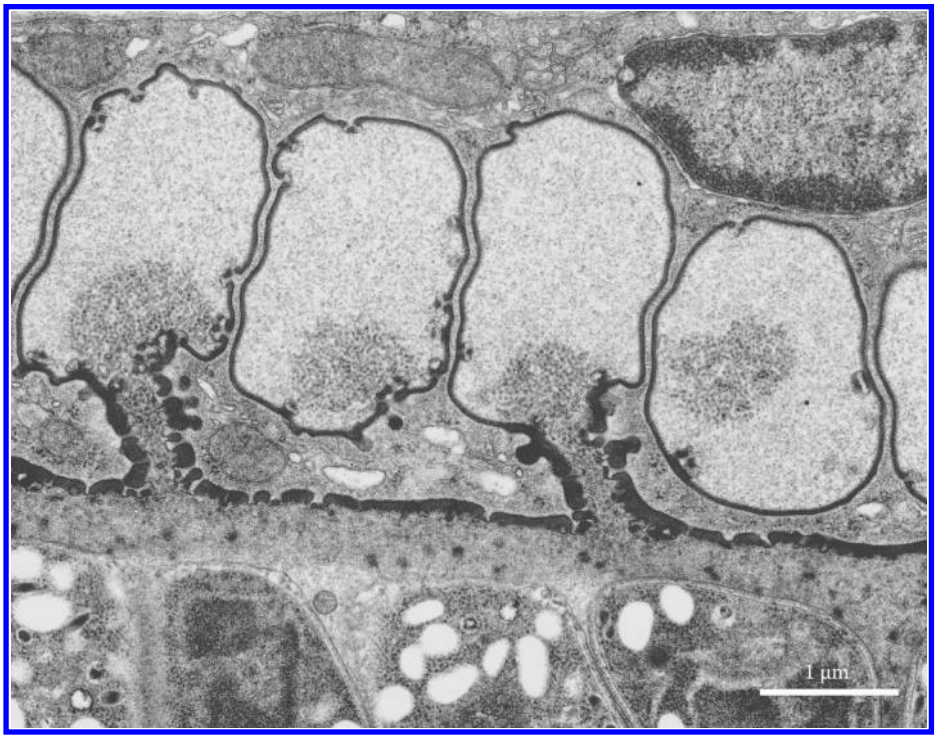


Figure 1.30 TEM of *S. singaporensis* sarcocyst at 184 DPI. Aged sarcocyst wall. The vp have become even shorter, with the distal portion irregular in shape.

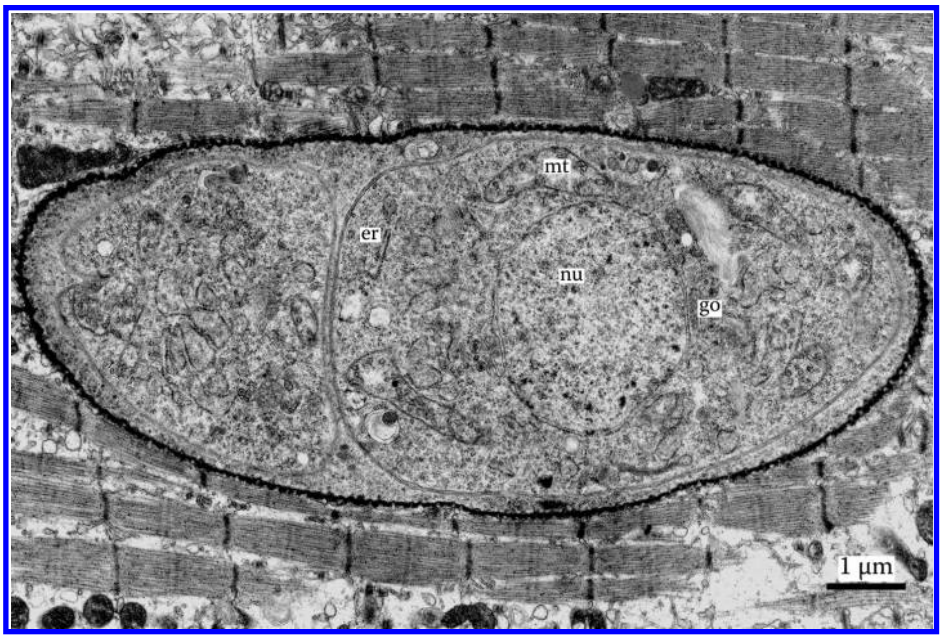


Figure 1.31 Young sarcocyst of *S. raushorum* containing 2 metrocytes with a large nucleus (nu), mitochondrion (mt), Golgi complex (go), ribosomes and endoplasmic reticulum (er). (Courtesy of R. J. Cawthorn.)

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Figure 1.32 Young sarcocyst of *S. raushorum* containing 8 metrocytes; a developing apical complex (arrow) is visible in 1 metrocyte. 15 DPI. (Courtesy of R. J. Cawthorn.)

1.3.1.1.1 Endodyogeny

Endodyogeny is a form of asexual multiplication in which 2 progeny develop within a preceding stage that is ultimately consumed in the process. A series of TEMs shows endodyogeny in metrocytes of *S. singaporensis* (Figures 1.33 and 1.34). The process usually begins with the transformation of the merozoite, metrocyte, or bradyzoite, from a fusiform to an ovoid shape. The parasite cytoplasm becomes electron lucent, and micronemes, rhoptries, amylopectin, and electron-dense bodies become localized or scattered, and some disappear. The Golgi complex divides into 2 parts, and 2 centrioles appear at the anterior margin of the large vesicular nucleus. The nucleus becomes bean-shaped, with each of the 2 lobes containing a spindle apparatus consisting of nuclear spindle microtubules and a centrocone. The centrocone is a sharply conical structure from which the spindle microtubules originate. The spindle microtubules pass from the centrocone into the nucleus via perforations (not nuclear pores) in the nuclear envelope, which remains intact during nuclear division. Each of 2 centrioles is situated immediately above and slightly lateral to each centrocone. The inner membrane complex and subpellicular microtubules of each progeny form a conical-shaped structure above each spindle apparatus in the anterior part of the metrocyte. The apical rings and polar rings appear early, along with the inner membrane complex and subpellicular microtubules.



Figure 1.33 TEM of *S. singaporensis* at 60 DPI. (a) A metrocyte in an early stage of endodyogeny; note the pair of centrioles (ce) at the anterior margin of the nucleus and micronemes scattered around the margin of the metrocyte. 176 DPI. (b) A metrocyte containing 2 developing zoites. The apical complex → of each zoite has begun to form immediately above each lobe of the U-shaped nucleus. Note spindle apparatus (arrowheads).

During this early stage of endodyogeny, a conoid is gradually formed at the tip of the developing complex. A centriole is often seen in close proximity to the developing conoid and may actually be responsible for its formation in much the same manner that centrioles give rise to a subsequent generation of centrioles.

As division progresses, the inner membrane complexes and subpellicular microtubules grow posteriorly, incorporating increasingly more of the dividing nucleus. The electron-dense areas, present at the edges of the inner membrane and at the ends of the subpellicular microtubules, probably represent areas in which components of these structures are assembled. As the inner membrane complex nears the posterior end of the metrocyte, the nucleus pinches into 2 nuclei, with each portion incorporated into a progeny. At this point, each progeny usually has a completely formed conoid, some micronemes and rhoptries, and various inclusion bodies. The progenies increase in volume,

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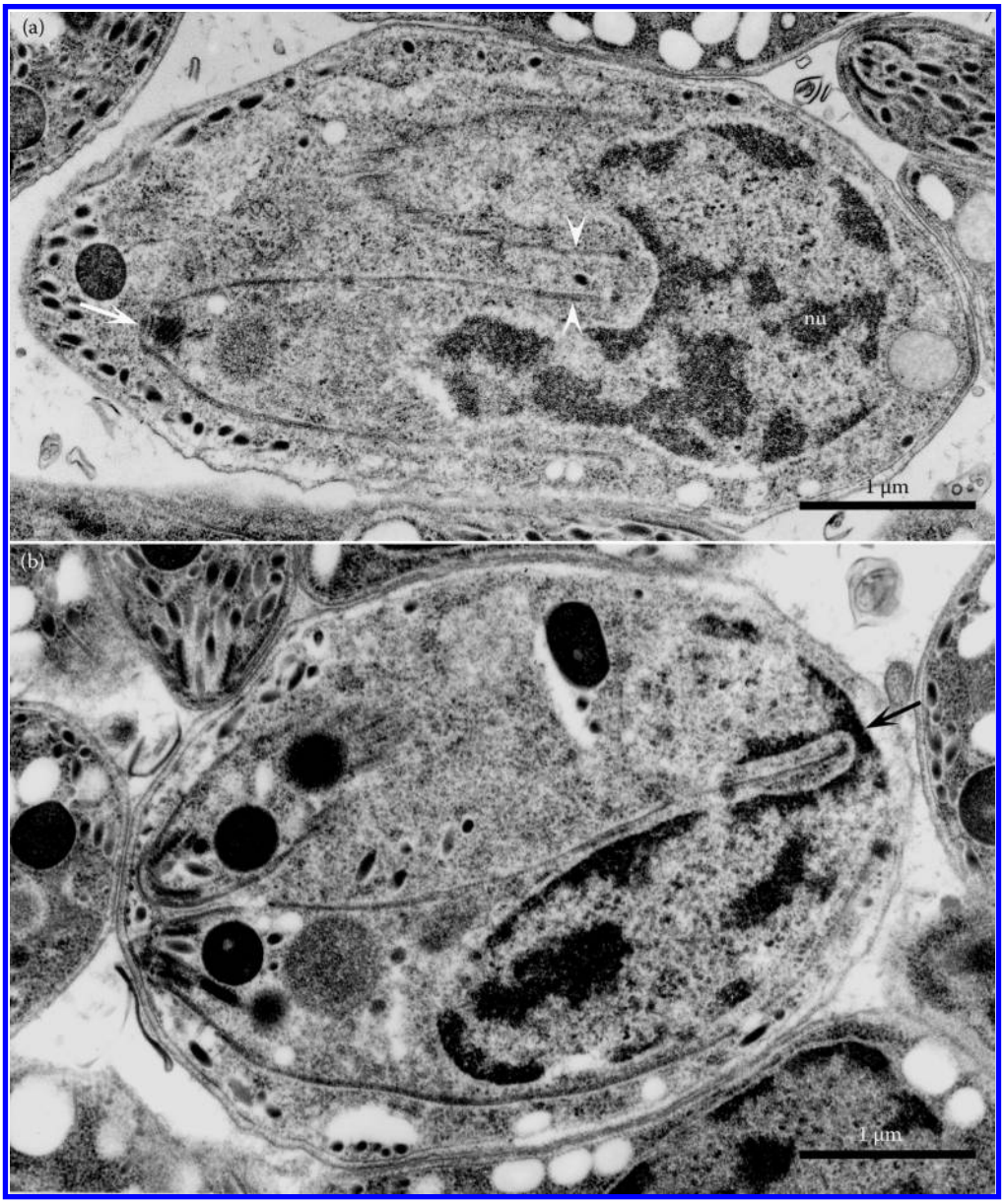


Figure 1.34 TEM of *S. singaporensis*. (a) Intermediate stage of endodyogeny showing a completely formed conoid (arrow), the inner membrane complex and subpellicular microtubules of each daughter zoite have extended further posteriorly (arrowheads), incorporating more of the parasite nucleus, 176 DPI. (b) TEM of advanced stage in endodyogeny; note that the 2 lobes of the nucleus are connected by a narrow isthmus (arrow) at 60 DPI.

acquiring more of the metrocyte cytoplasm and organelles, until they occupy most of the metrocyte. As the progenies approach the surface, the inner membrane complex of the metrocyte disappears, and the outer membrane becomes the outer membrane of each progeny. At this point, they separate into 2 progenies. During endodyogeny, various spheroidal structures appear in the anterior end, which probably represent anlagen of rhoptries and micronemes of the developing progeny.

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1.3.1.2 Bradyzoites (Figures 1.35 through 1.40)

1.3.1.2.1 Pellicle

The pellicle consists of 3 membranes, an outer plasmalemma and an inner double membrane complex (Figures 1.35, 1.39, and 1.40). The plasmalemma is a continuous unit that completely encloses the whole parasite, whereas the inner pellicular membranes are interrupted at the anterior and posterior ends and at micropores. The plasmalemma is separated from the inner membranes by an electron–lucent space, 15–20 nm thick. In *S. tenella*, the intramembranous particles are randomly arranged in the plasmalemma, but have an orderly arrangement in the inner pellicular membranes.[1406] The inner pellicular membrane complex is similar to tight intercellular junctions

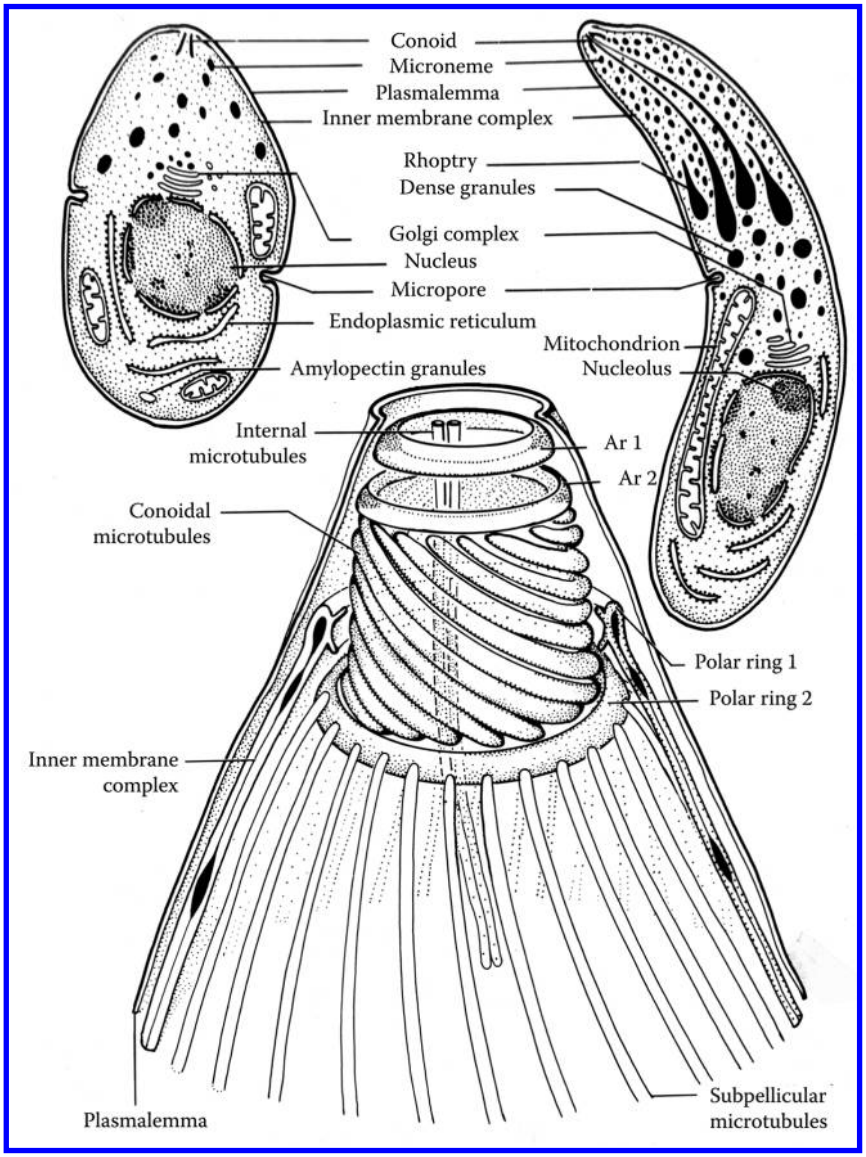


Figure 1.35 Schematic representation of typical metrocytes, bradyzoites, and apical complex of *Sarcocystis* spp.

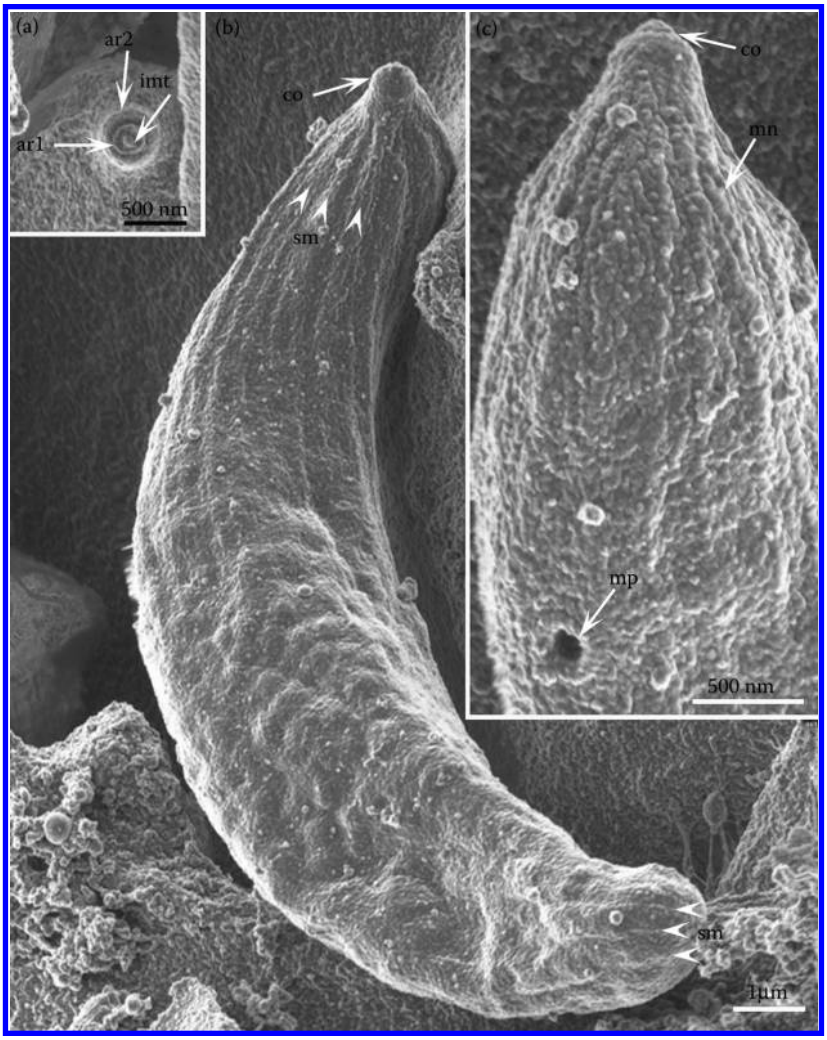


Figure 1.36 SEM of *S. fusiformis* bradyzoites. (a) Enface view of conoid (co). Note 2 inner microtubules (imt), and 2 apical rings (ar1, ar2). (b) Surface view of bradyzoite (br). Note subpellicular tubules (sm) arising from the polar ring of the co and extending the entire length of the br. The posterior half of the bradyzoite has irregular bumps. (c) Conoidal part of a bradyzoite with a conoid and micropore (mp); it is granular because of the numerous micronemes that are partly visible through the pellicle. (Adapted from Dubey, J.P. et al., 2015. *Parasitology.* 142, 385–394.)

of higher organisms and is composed of 11 rows of rectangular contiguous plaques (0.5×2 μm in size) that are arranged in a loosely coiled helical pattern. The plaques converge at the posterior end to form the posterior pore (posterior ring) which represents an interruption in the inner membrane complex. At 1.5–2 μm from the anterior tip, the rectangular plaques abut tightly to a conical inner membrane complex called the anterior cape. The anterior cape has an interruption at its anterior tip, which appears electron-dense in TEM, and forms the polar ring. At the anterior tip, the plasmalemma contains a rosette of intramembranous particles (1 central and 8 peripheral particles). The rosette of intramembranous particles may be involved in rhoptry secretion during host-cell penetration or serve as a receptor–processor system, which enables the parasite to recognize and penetrate host cells.

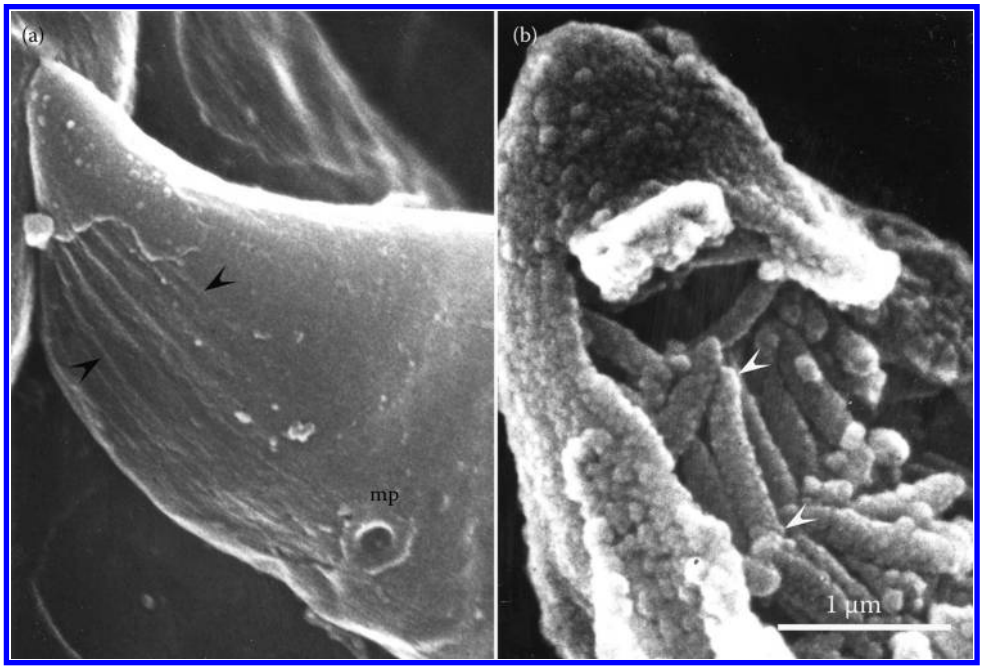


Figure 1.37 SEMs of the apical ends of *S. cruzi* bradyzoites. (a) Note opening of micropore (mp) and ridges (arrowheads) on surface created by underlying subpellicular microtubules. (b) Portion of pellicle has been removed exposing the micronemes (arrowheads). (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res*. 43, 2147–2164. With permission.)

1.3.1.2.2 Apical Rings

Two apical rings (also called preconoidal or conoid rings) are situated at the anterior tip, immediately beneath the plasmalemma and above the papillary conoid (Figures 1.36, 1.39, and 1.40). Bradyzoites of *S. tenella* may have 3 apical rings.[1406] The function of these apical rings is not known.

1.3.1.2.3 Polar Rings (Figures 1.35 through 1.40)

The polar rings of *Sarcocystis* species have been studied less extensively than those of other coccidia. A single polar ring appears to be present in *S. tenella* bradyzoites,[1408] whereas other species have 2 polar rings. Polar ring 1 appears as an electron-dense thickening at the anterior termination of the anterior cape of the inner membrane complex, and serves to anchor the subpellicular microtubules in those bradyzoites with a single polar ring.[1408] In those species with 2 polar rings, polar ring 2 is located immediately interior to and slightly posterior to polar ring 1, and serves an an anchoring point for the subpellicular microtubules (Figure 1.40). Two electron-dense projections are attached to the inner surface of polar ring 1 (Figures 1.35 and 1.40).

1.3.1.2.4 Microtubules (Figures 1.35 through 1.39)

Microtubules are found in virtually every stage of *Sarcocystis*. A few are cytoplasmic or nuclear microtubules, whereas most other microtubules are components of specialized structures, such as subpellicular microtubules, conoid, centrioles, flagellar axoneme, and the mitotic spindle apparatus. Bradyzoites of *Sarcocystis* species have 22 subpellicular microtubules that originate at evenly

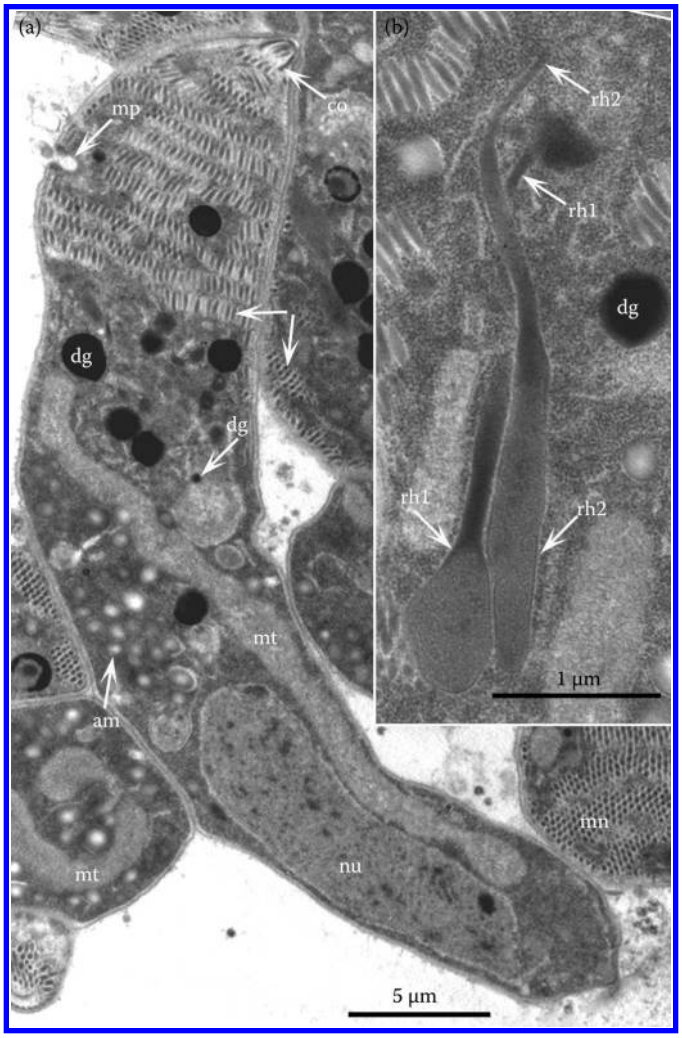


Figure 1.38 TEM of *S. fusiformis* bradyzoites. (a) Longitudinally cut bradyzoite (br) and parts of other br. Note conoid (co), numerous micronemes (mn), a micropore (mp), at least 8 dense granules (dg) of different sizes, numerous amylopectin granules (am), a convoluted mitochondrion (mt), and a large nulcleus (nu). (b) Part of a bradyzoite showing 2 rhoptries (rh1, rh2) that are twisted at their necks. Note variability in density of the rhoptry contents. (Adapted from Dubey, J.P. et al., 2015. *Parasitology.* 142, 385–394.)

spaced intervals around the polar ring, and extend posteriorly immediately beneath the inner membrane complex for about 1/2 to 2/3 the length of the zoite (Figures 1.35 and 1.39).

The subpellicular microtubules are similar to those found in a wide variety of plant and animal cells. They are cylinders approximately 25 nm in diameter. They usually contain 12 or 13 protofilament strands running longitudinally. They appear striated because the protofilaments appear to be composed of repeating subunits, probably α- and β-tubulin, arranged in transverse or helical rows. Subpellicular microtubules probably provide structural integrity to the overall shape of zoites, and may be involved in motility (gliding and flexing), transport of cytoplasmic components near the margin of the zoite, or serve to anchor protein molecules in the inner membrane complex. Treatment with the antimicrotubular agents—colcemid, colchicine, or vinblastine—stops motility of bradyzoites of *Sarcocystis*.[273]

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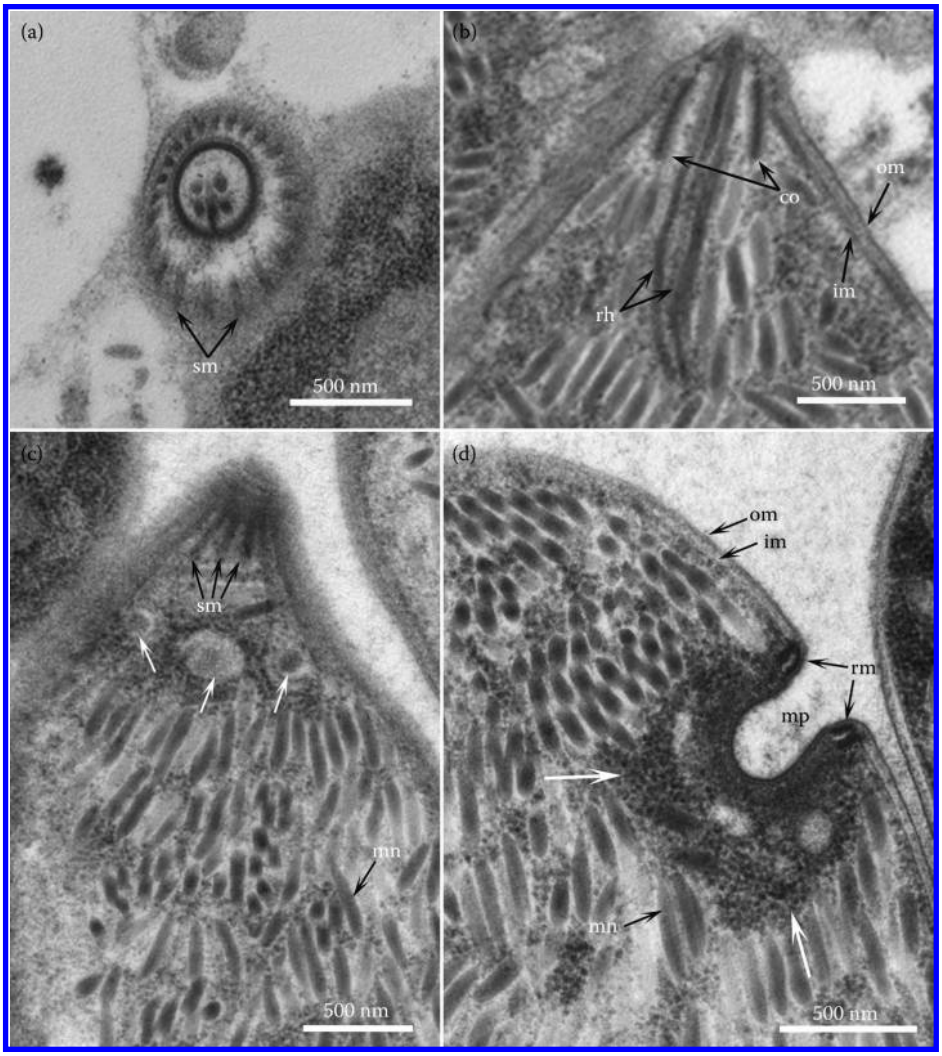


Figure 1.39 TEM of conoidal part of 4 bradyzoites. (a and b), *S. fusiformis*, (c and d), *S. cafferi*. (a) Cross/oblique section through the conoid. Note 22 subpellicular tubules (sm) originating from the polar ring. (b) Conoid with 2 rhoptries (rh) opening in the conoid. (c) Details of conoidal end with annular and polar rings, subpellicular tubules (sm), 3 ring-like structures at the conoidal end (white arrows). Note haphazard arrangement of micronemes (mn). (d) Details of pellicle with outer (om) and inner (im) membrane at the micropore opening and a rim-like (rm) structure is present at the opening. Electron-dense secretory material (white arrows) surround the micropore. Note numerous micronemes (mn). ((a and b) Adapted from Dubey, J.P. et al., 2015. *Parasitology.* 142, 385–394; (c and d) Adapted from Dubey, J.P. et al., 2014. *J. Parasitol.* 100, 817–827.)

1.3.1.2.5 Micropore

Micropores are present in all stages of *Sarcocystis* species, except microgametes. They consist of an invagination of the parasite plasmalemma into the parasite cytoplasm (Figures 1.36 through 1.39). The invaginated membrane is encircled on the cytoplasmic side by an electron-dense membranous collar, which the inner membrane interrupts, and is juxtaposed at a 90°-angle to it. Although most micropores appear to be inactive, some have appeared to be portals for ingestion of particulate

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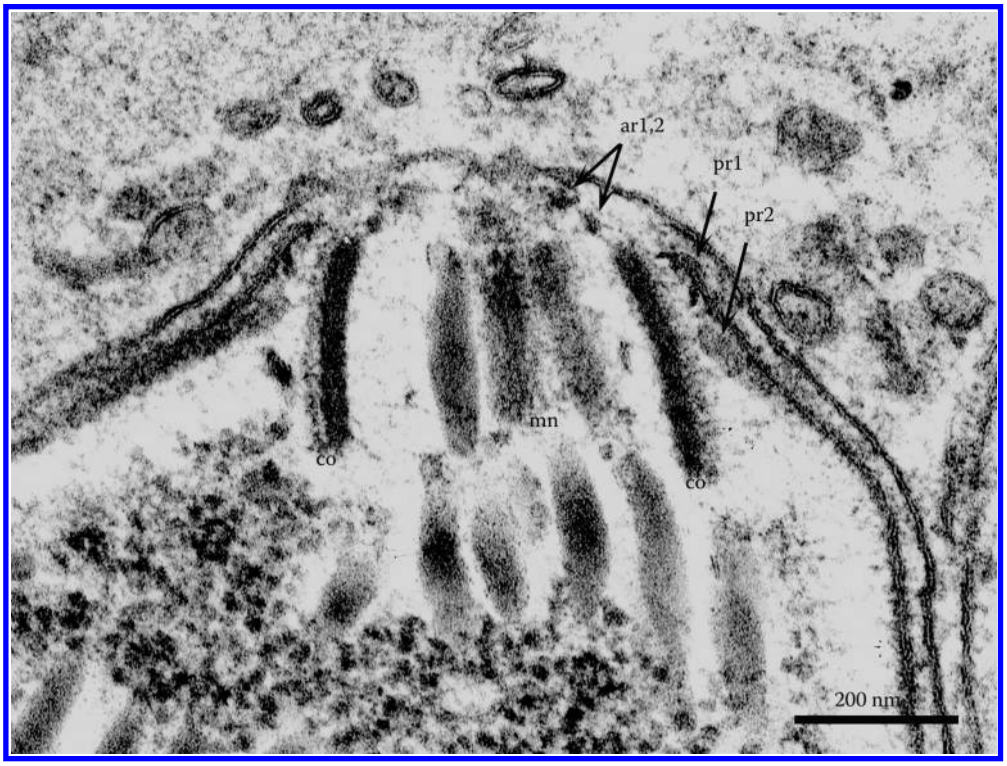


Figure 1.40 TEM of apical complex of *S. campestris* bradyzoite at 62 DPI. Note: apical (ar1,2) and polar (pr1,2) rings, conoid (co), and micronemes (mn).

matter in developing or multiplying stages such as metrocytes. Occasionally, electron-dense material surrounds the cytoplasmic side of the micropore (Figure 1.39d).

1.3.1.2.6 Rhoptries and Micronemes

Rhoptries and micronemes are electron-dense, membrane-bound, elongate structures occupying the anterior of the bradyzoite (Figures 1.36 and 1.38). These organelles are believed to constitute a single functional unit involved in secreting substances to aid penetration of cells and may secrete proteins that become antigenic or are inserted into the bradyzoite plasmalemma just anterior to the conoid.

Rhoptries consist of a narrow, duct-like anterior region found within the core of the conoid and a posterior club- or sack-like region. Most bradyzoites have only 2 rhoptries; in early studies, dense granules may have been mistaken as rhoptries.[474] To avoid misinterpretation, it is necessary to identify the elongated neck region, or a very clear bulbous end, because the rhoptry contents can appear differently within the same rhoptry (Figure 1.38). The contents of rhoptries are often electron-dense. Bradyzoites of all species of *Sarcocystis* contain numerous micronemes. Micronemes are elongate, rod-like structures that usually appear ovoid or round in cross section. In some Apicomplexa, narrow interconnections have been found between micronemes and rhoptries, but interconnections between the rhoptries and micronemes in bradyzoites of *S. tenella* are controversial. Micronemes appear to form within single membrane-bound vesicles in the apical region of bradyzoites, and apparently give rise to rhoptries. The arrangement of micronemes can vary within different regions of the bradyzoite. Their location in the bradyzoite is fluid, and they are considered to move towards the conoid.

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1.3.1.2.7 Dense Granules

Dense granules are electron-dense and are scattered throughout the bradyzoite; but most are located in the middle of the parasite (Figure 1.38). They are secretory vesicles, and their contents play a major role in the modification of the PV and regulation of immune mediation.

1.3.1.2.8 Conoid

The conoid is a hollow, papillary organelle located at the anterior tip of sporozoites, bradyzoites, and merozoites. The conoid of bradyzoites of *S. tenella* is made up of 20 microtubules arranged in a helical pattern. Some investigators believe the conoid may be involved in penetration of host cells, because, when it is protruded, the anterior tip becomes shaped like a long, narrow stylet that could facilitate active penetration into the cell. Within the core of the conoid are the ducts of rhoptries and 1 or 2 eccentrically located microtubules that extend for a short distance into the zoite cytoplasm (Figure 1.35). These microtubules may be attached to the conoid, and serve to protrude or retract the conoid.

1.3.1.2.9 Golgi Complex

The Golgi complex is well-developed in the stages that are most metabolically active, such as metrocytes and schizonts, but are reduced in size and complexity in bradyzoites of most *Sarcocystis* spp. The functions of the Golgi complex are probably similar to those described for other cells.

1.3.1.2.10 Endoplasmic Reticulum

Both smooth and rough endoplasmic reticula are present but small in bradyzoites, especially in the area just posterior to the nucleus.

1.3.1.2.11 Ribosomes

Bradyzoites have an abundant amount of free ribosomes, found from the conoid to the posterior tip, filling the bradyzoite cytoplasm not occupied by organelles or inclusion bodies.

1.3.1.2.12 Mitochondrion

A mitochondrion is found in bradyzoites, as well as in all other stages of *Sarcocystis* spp. Bradyzoites appear to have 1 long twisted mitochondrion (Figure 1.38). This organelle is double-membrane-bound, with tubular cristae, which are characteristic of most protozoans.

1.3.1.2.13 Apicoplast

The apicoplast is a membrane-bound, alga-derived, obligatory endosymbiotic structure. Considerable biologic curiosity has been focused on this nonphotosynthetic organelle. It is essential for the survival of the parasite, and has its own genome. In the past, it has been called a multivesicular body, multimembrane-bound vesicle, or Golgi adjunct. It is surrounded by 4 membranes.[1754] Its synthesis was studied in detail in *S. neurona* schizonts (Figure 1.22)[1770] (see Chapter 3).

1.3.1.2.14 Inclusion Bodies

Bodies that appear free in the cytoplasm, such as amylopectin, lipid bodies, and electron-dense bodies, are generally referred to as inclusion bodies (Figure 1.35). Although electron-dense bodies

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are seen in nearly all stages, their function is still not known. Some inclusion bodies probably serve as a site for storage of energy reserves, such as amylopectin, and others may store proteins, lipids, and, perhaps, enzymes, whose functions are unknown.

1.3.1.2.15 Size

Bradyzoites vary in size. Bradyzoites of species of *Sarcocystis* in livestock are approximately 3 times larger than those species parasitizing reptiles. They are often banana-shaped; in few species they are lanceolate (e.g., *S. wenzeli* of chicken).

1.3.1.3 Types of Sarcocyst Walls (Figures 1.21 through 1.30, 1.41 through 1.61)

Numerous ultrastructural reports have shown that the sarcocyst walls of *Sarcocystis* species vary—from being relatively simple to highly complex. After examining published reports, there appear to be at least 82 distinct types of sarcocyst walls (Table 1.2), which herein are referred to as Types 1–42. It is likely that additional types will be discovered. To avoid



Figure 1.41 TEM of *S. rauschorum* sarcocyst wall at 60 DPI. The primary sarcocyst wall consists of an undulating pvm and an electron-dense layer immediately beneath the pvm. At certain points, the pvm invaginates (arrow) into the granular layer; the latter contains fine granules and small vesicles (arrowheads) that arise from the pvm.

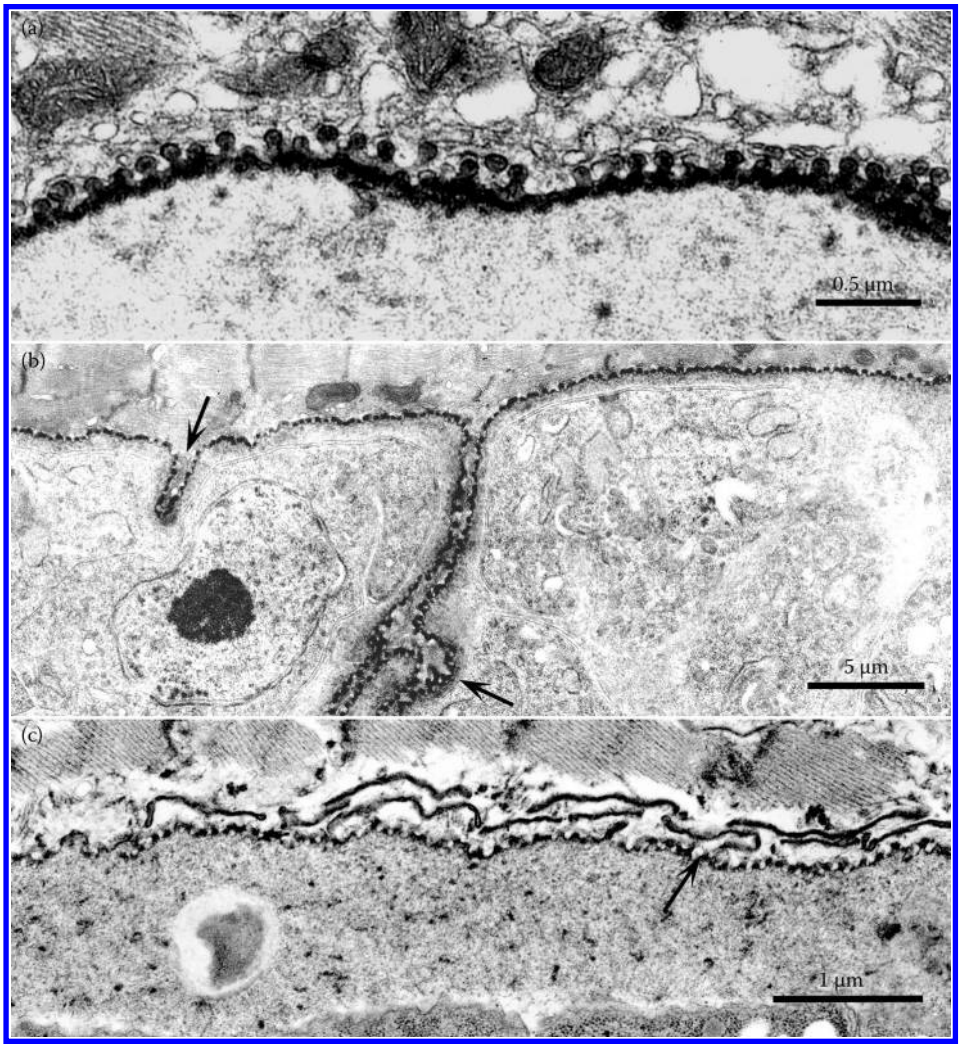


Figure 1.42 Types 1 and 2 sarcocyst walls. (a) *S. muris*, type 1a with the simplest structures with blebs on pvm. (b) *S. rauschorum*, type 1h. The sarcocyst wall invaginates (arrows) deeper into the sarcocyst at 60 DPI. (Courtesy of R. J. Cawthorn.) (c) Type 2 *S. wapiti* showing thin hirsute structures arising from the apex of the undulating primary sarcocyst wall (arrow). (From Speer, C.A., Dubey, J.P., 1982. *Can. J. Zool*. 60, 881–888. With permission.)

misinterpretations, the types reported earlier[393,432] were retained, and new types were added as subtypes (Figures 1.58 through 1.61). The wall types presented herein do not correspond in number with those reported by Beaver et al.[84] which were based on LM observations, and, therefore, were limited in the detail that could be observed.

The primary sarcocyst wall consists of a PVM and an electron-dense layer (edl) immediately beneath the PVM. A granular layer is immediately beneath the primary sarcocyst wall. Septa, which arise from the granular layer, traverse the sarcocyst, separating it into compartments (Figure 1.11a), which contain bradyzoites and metrocytes.

There are only a few reports that used SEM to study the ultrastructure of sarcocysts.[126,127,281–284,360,382,473,474,698,700,702,1176,1309,1316,1684] Compared to a TEM, an SEM provides little information because it is limited to examining surfaces (Figures 1.53 through 1.57).

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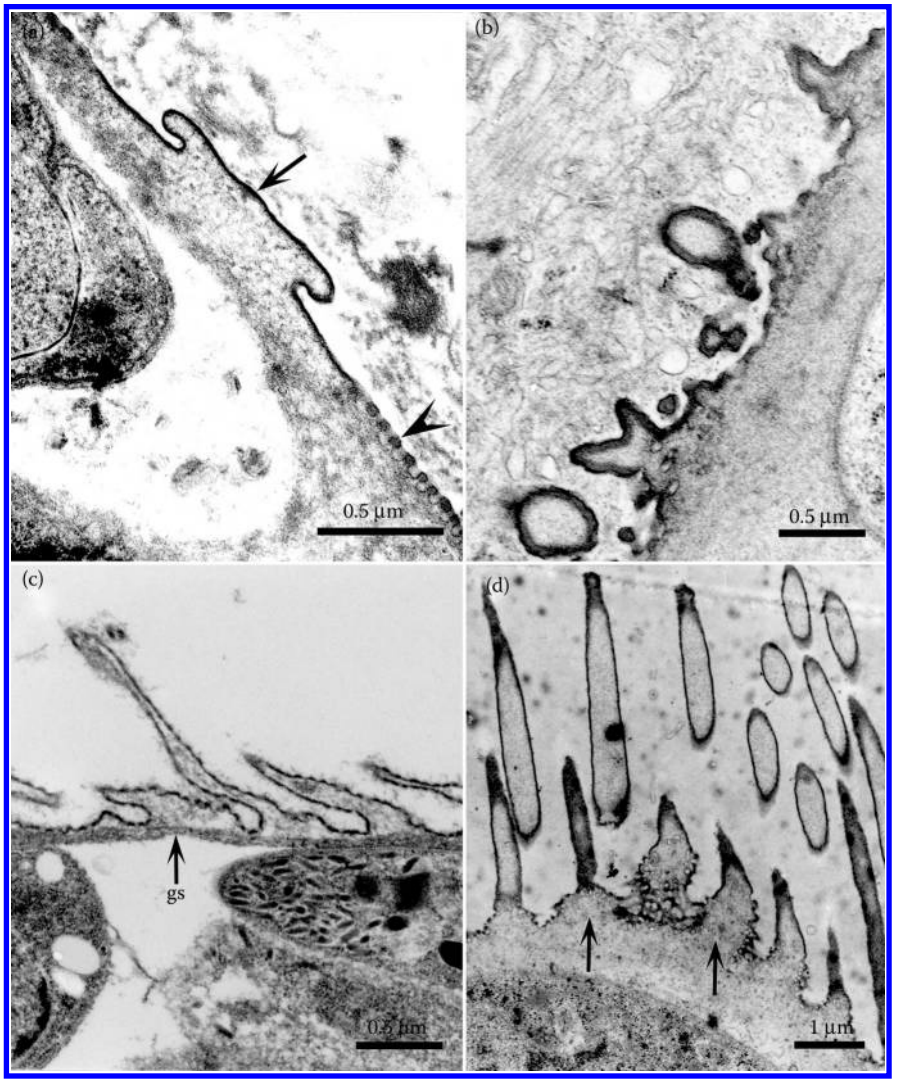


Figure 1.43 Types 3–6 sarcocyst walls. (a) Type 3 *S. ferovis* sarcocyst wall, showing undulating pvm with blebs (arrowhead) and a flattened mushroom-like vp (arrow). (From Dubey, J.P., 1983. *Proc. Helminthol. Soc. Wash*. 50, 153–158. With permission.) (b) Type 4 *S. sigmodontis* wall, showing short, irregularly shaped vp. (c) Type 5 *S. sulawesiensis* wall with hirsute vp and a relatively thin granular layer (gs). (From O'Donoghue, P.J., Watts, C.H.S., Dixon, B.R., 1987. *J. Wildl. Dis*. 23, 225–232. With permission.) (d) Type 6 *S. capreolicanis* wall, showing finger-like vp arising from a dome-shaped base (arrows). The pvm has minute undulations in the dome-shaped base, but is relatively smooth in the finger-like vp that are oriented perpendicularly to the sarcocyst wall. (Courtesy of R. Entzeroth.)

The sarcocyst surface of type 14 sarcocysts of *S. capracanis* has a honeycomb-like appearance, and the protrusions appear villus-like (Figure 1.54a and b).[382]

1.3.1.4 Gametogenesis

After ingestion by the appropriate definitive host, bradyzoites escape from sarcocysts, and penetrate enterocytes, usually goblet cells, in the small intestine (Figure 1.62a). Development

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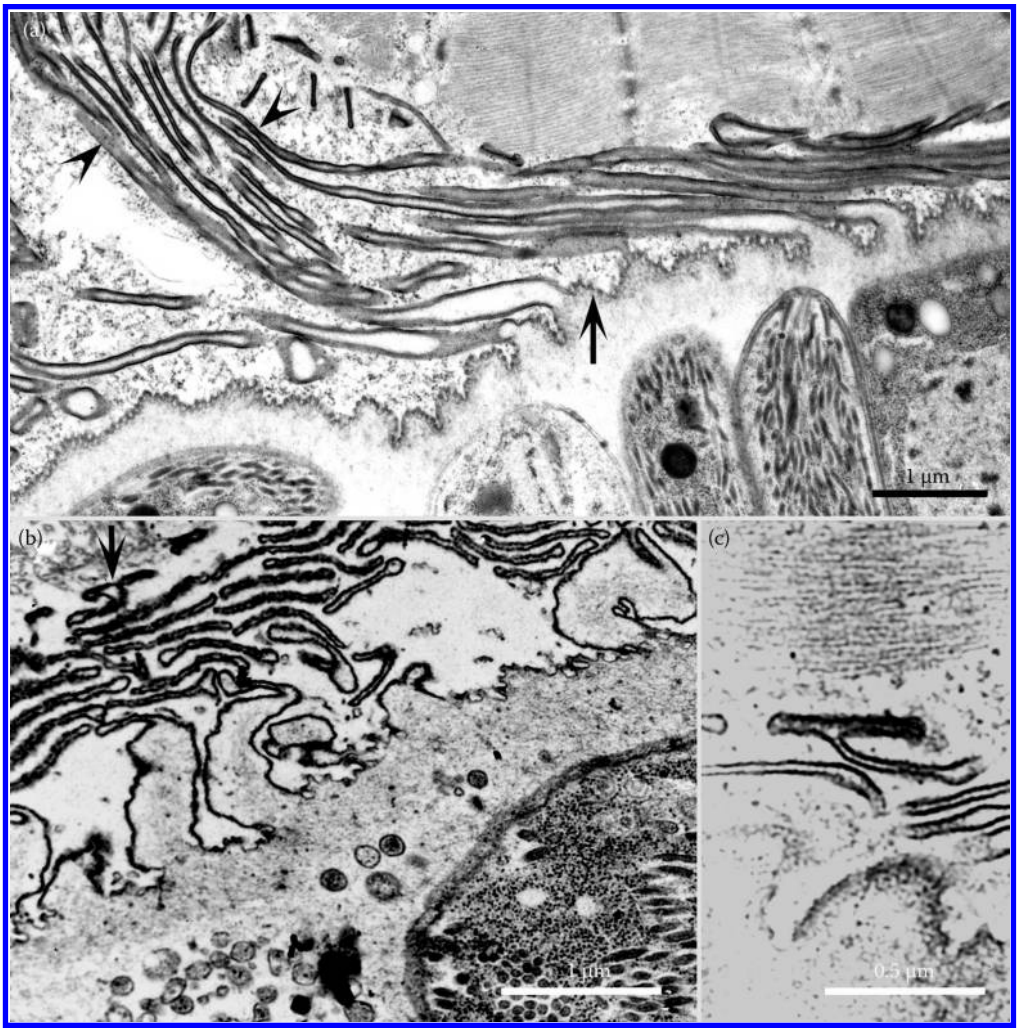


Figure 1.44 Types 7 and 8 walls. (a) Type 7 *S. arieticanis* sarcocyst consisting of a dome-shaped base (arrow) with an undulating pvm, an intermediate finger-like segment, and a thin, thread-like distal segment (arrowhead). The intermediate and distal segments are often bent 90° and run parallel to the sarcocyst surface, whereas in other areas, the protrusions are arranged collectively to form conical tufts. (b) Type 8a *S. gruneri*. This sarcocyst is similar to Type 7, except that the distal segment of the protrusion is branched (arrow). (From Entzeroth, et al., 1985. *Z. Parasitenkd*. 71, 33–39. With permission.) (c) Higher magnification of type 8a showing branched distal segment.

from bradyzoite to microgamonts or macrogamonts is rapid, being completed in 6–18 h or less. Bradyzoites are situated within a PV and begin immediately to develop into either a micro- or macrogamont. How the sex is determined or regulated is not known.[255] Bradyzoites quickly transform from a fusiform to an ovoid or spheroid form, and most of the organelles of the apical complex undergo dissolution. Micronemes become scattered in the gamont cytoplasm, and soon disappear. Simultaneously, the early gamont produces much endoplasmic reticulum, numerous ribosomes, mitochondrion, and a vesicular nucleus, with scattered heterochromatin and a nucleolus. Macrogamonts have 2 ultrastructural features that appear to be lacking in microgamonts. These are an exocytosis pore and electron-dense granules which are transported to a location near the surface

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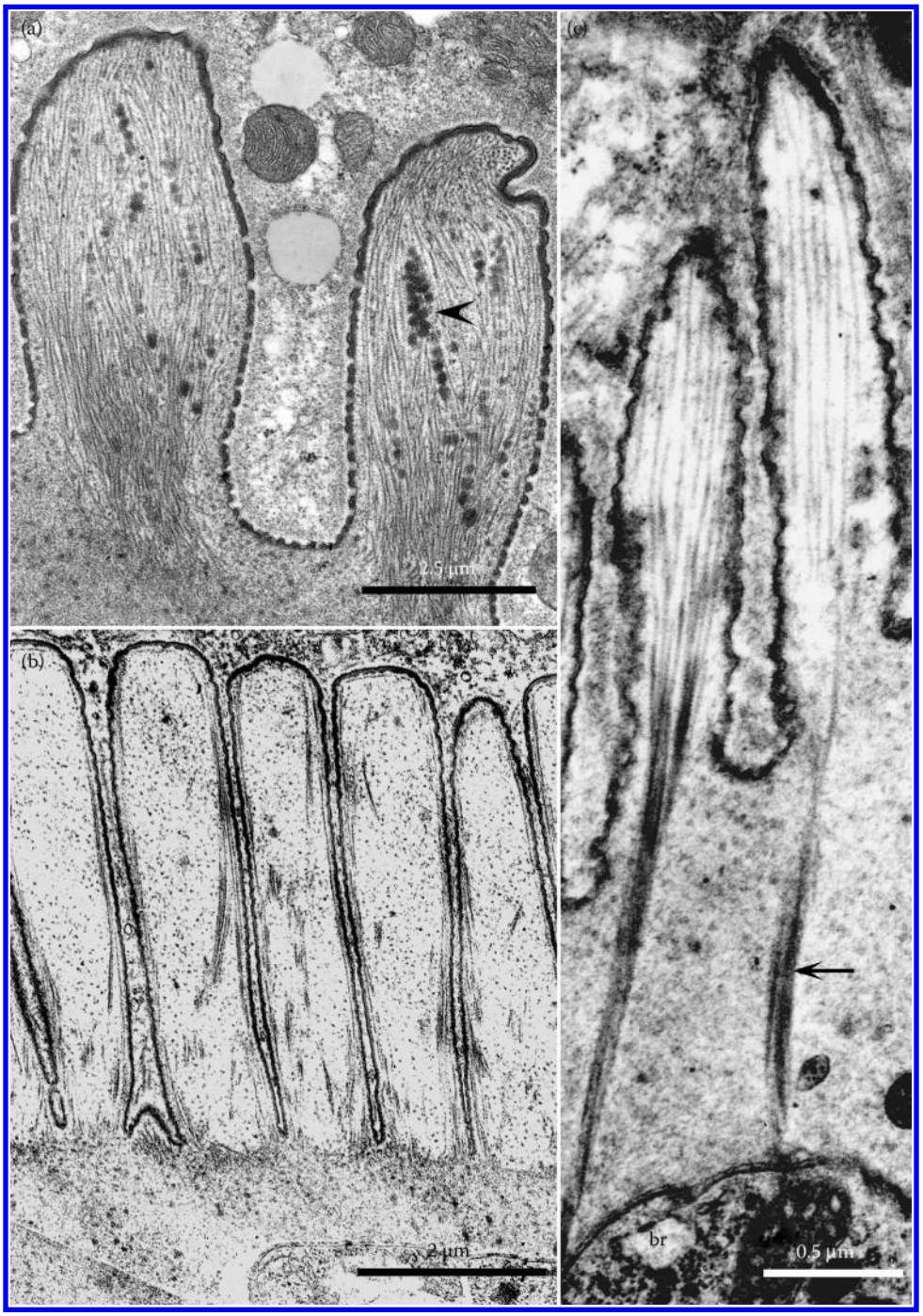


Figure 1.45 Types 9–11. (a) Type 9a *S. campestris* sarcocyst showing widely spaced vp. The pvm has minute undulations over the entire sarcocyst surface and the vp contain prominent electron-dense granules (arrowhead) and microtubules that extend from the villar tips into the granular layer. (b) Type 10a *S. odoi* sarcocyst wall, similar to Type 9a but with tightly packed vp. (c) Type 11a *S. fayeri* sarcocyst wall.The microtubules extend from the villar tips to the plasmalemma of bradyzoites (arrow). (From Tinling, S. et al., 1980. *J. Parasitol*. 66, 458–465. With permission.)



Figure 1.46 Type 11b *S. falcatula* wall, showing vp and microtubules (arrowhead) in cross section. The pvm is wavy and lined underneath with an electron-dense layer. Also see Figure 17.1, showing that microtubules do not extend to the pellicle of bradyzoites. (From Box, E.D., Meier, J.L., Smith, J.H., 1984. *J. Protozool*. 31, 521–524. With permission.)

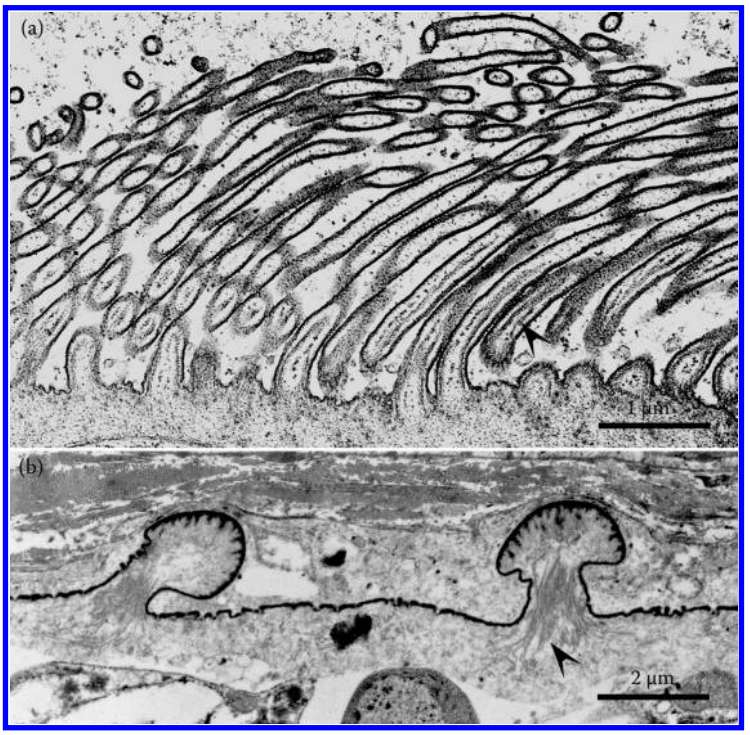


Figure 1.47 Types 12 and 13. (a) Type 12 *S. sybillensis* sarcocyst wall consisting of tightly packed, thin, finger-like vp with microtubules in the basal one-third of the protrusion. (b) Type 13 *S. mucosa* sarcocyst with widely spaced, mushroom-like vp with microtubules that extend from the core of the protrusion into ground substance (gs) (arrowhead). (From O'Donoghue, P.J. et al., 1987. *Parasitol. Res*. 73, 113–120. With permission.)

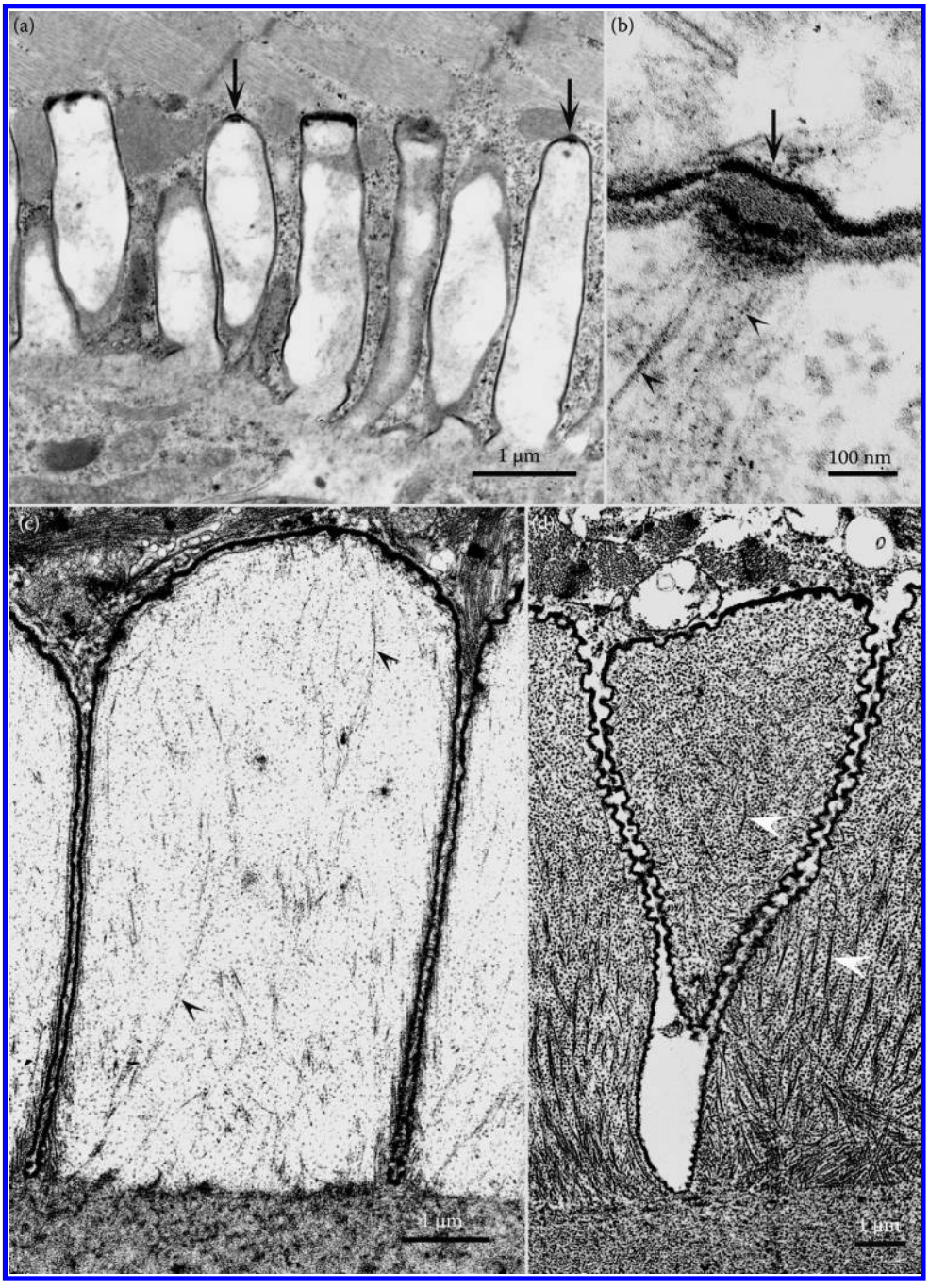


Figure 1.48 Types 14–16. (a) Type 14 *S. tenella* sarcocyst with tightly packed vp, the tips of which contain disc-shaped plaques (arrows). Microtubules are absent. (b) High magnification of a disc-shaped plaque (arrow) in *S. hemionilatrantis*. (From Speer, C.A., Dubey, J.P., 1986. *J. Protozool*. 33, 130–132. With permission.) (c) Type 15 *S. rangiferi* with tightly packed upright tombstone-like vp that contain widely scattered microtubules (arrowheads). In some species with type 15, the villar protrusions have interdigitating lateral margins. (d) Type 16 *S. youngi* sarcocyst wall with alternating conical and club-shaped vp with criss-crossing microtubules. (From Dubey, J.P., Speer, C.A., 1985. *J. Wildl. Dis*. 21, 219–228. With permission.)

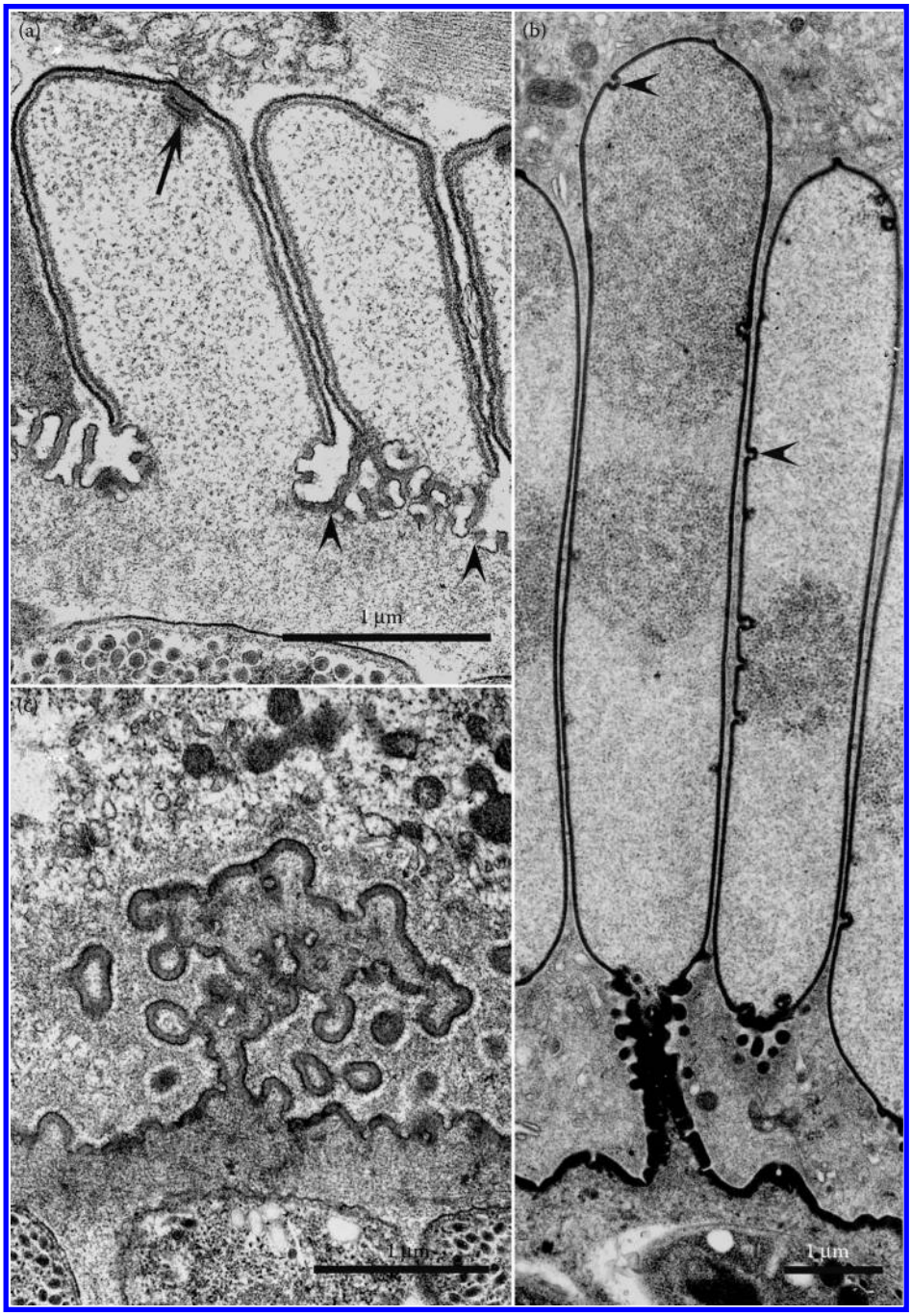


Figure 1.49 Types 17–19. (a) Type 17 *S. odocoileocanis* with disk-shaped plaques (arrow) at the apex and lateral margins of the vp and a highly branched primary sarcocyst wall at the base of the protrusions (arrowheads). (b) Type 19 *S. singaporensis* with club-shaped vp consisting of a cylindrical stalk and a sausage-shaped distal segment. In older sarcocysts, the distal segment may be shorter and irregular in shape (see Figure 1.30). Invaginations of the primary sarcocyst wall are numerous in the stalk (arrowheads) and widely scattered in the distal segment. (c) Type 18 *S. dispersa* with an irregularly shaped vp which may appear T-shaped. (From Sénaud, J., Cerná, Z., 1978. *Protistologica*. 14, 155–176. With permission.)

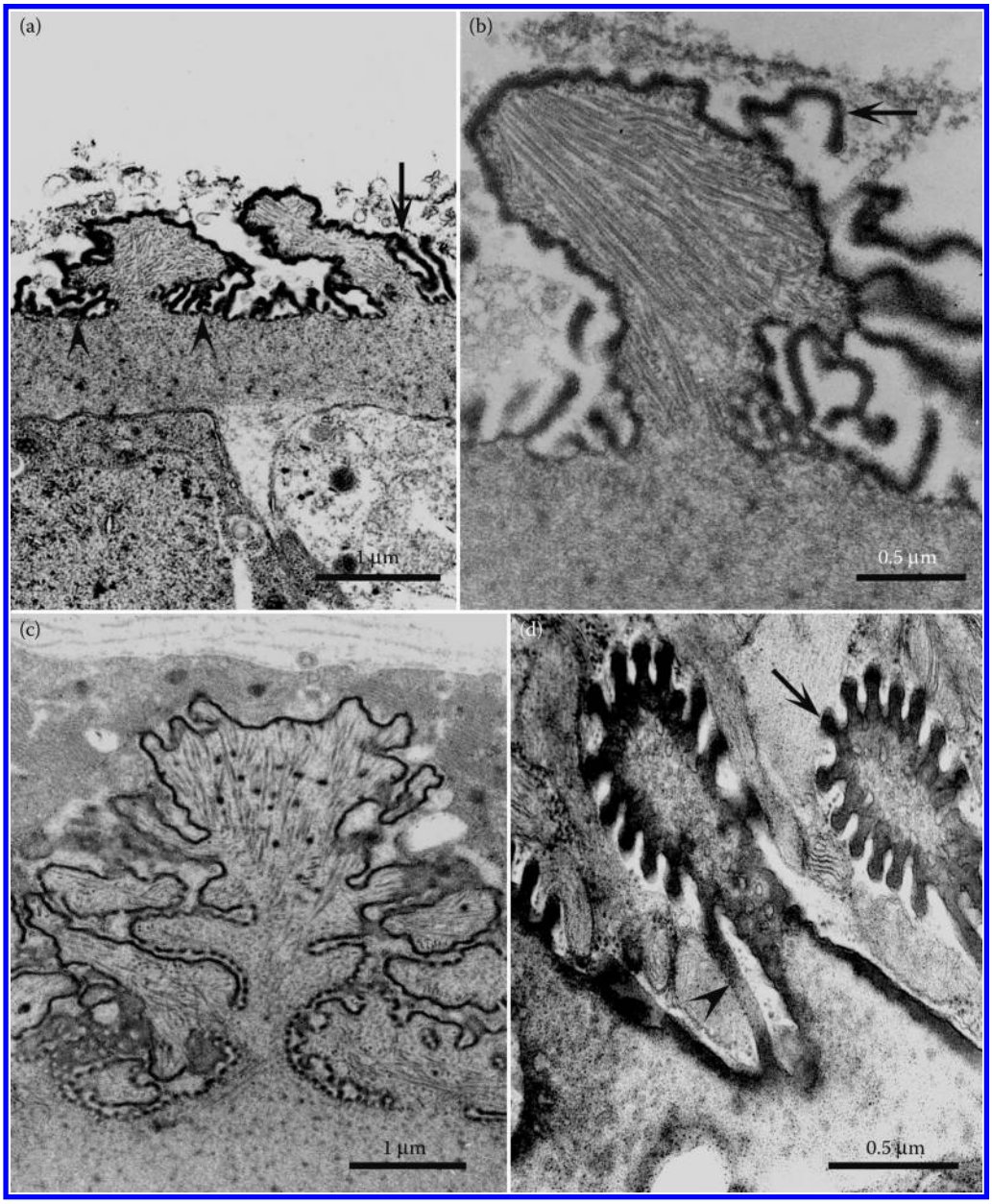


Figure 1.50 Types 20–22. Type 20 (a, b). *S. medusiformis* sarcocyst wall with angular vp that vary from pyramidal, trapezoidal, or mushroom-like. Serpentine projections arise from the surface of the vp (arrow) as well as from the base (arrowheads) and some connect the surface to protrusions. (a) Fine bristles (arrow) arising from the surface of the protrusions as well as from the surface of the serpentine projections. (b) The villar core contains tightly packed microtubules. (c) Type 21 *S. gigantea* wall that has cauliflower-like vp containing microtubules or microfilaments and fine or coarse granules. Also see Figures 1.53 and 1.57. (d) Type 22 *S. villivillosi* has protrusions with a proximal, sharply conical base and a distal segment that is cocklebur-like with short, radiating projections. The conical base is in oblique section. Cross-sections of the distal segment appear as cogwheels (arrow). Some of the projections from the conical base connect with the surface of the sarcocyst wall (arrowhead). Vesicles are located within the protrusions, especially in the distal half. (From Beaver, P.C., Maleckar, J.R., 1981. *J. Parasitol*. 67, 241–256. With permission.)

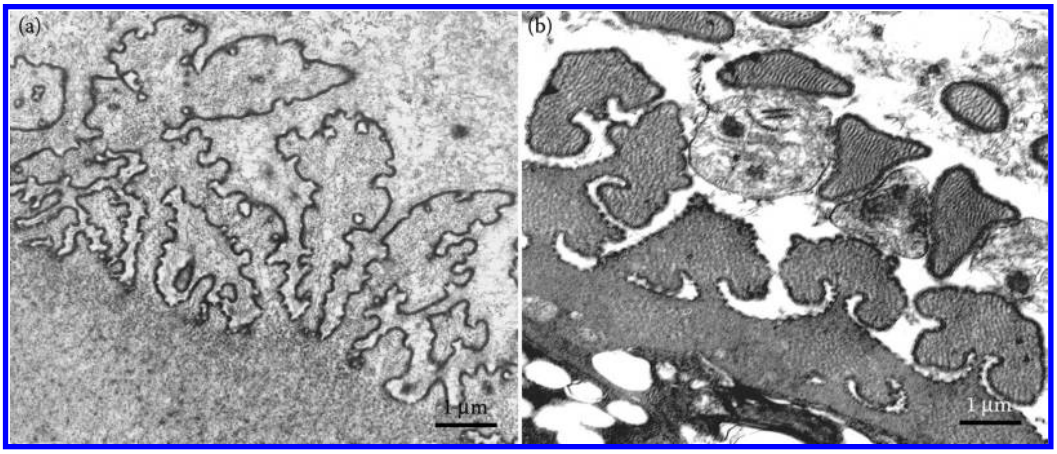


Figure 1.51 Types 23 and 24. (a) Type 23 *S. rileyi* consisting of anastomosing, cauliflower-like vp that contains fine granules and microfilaments. (Adapted from Dubey, J.P. et al., 2003. *J. Eukaryot. Microbiol.* 50, 476–482.) (b) Type 24 *S. cornagliai* sarcocyst type with mushroom-like projections arising from mushroom-like vp. (*Sarcocystis* sp. from mountain goat, courtesy of W. Foreyt, see Foreyt, W.J., 1989. *J. Wildl. Dis*. 25, 619–622.)

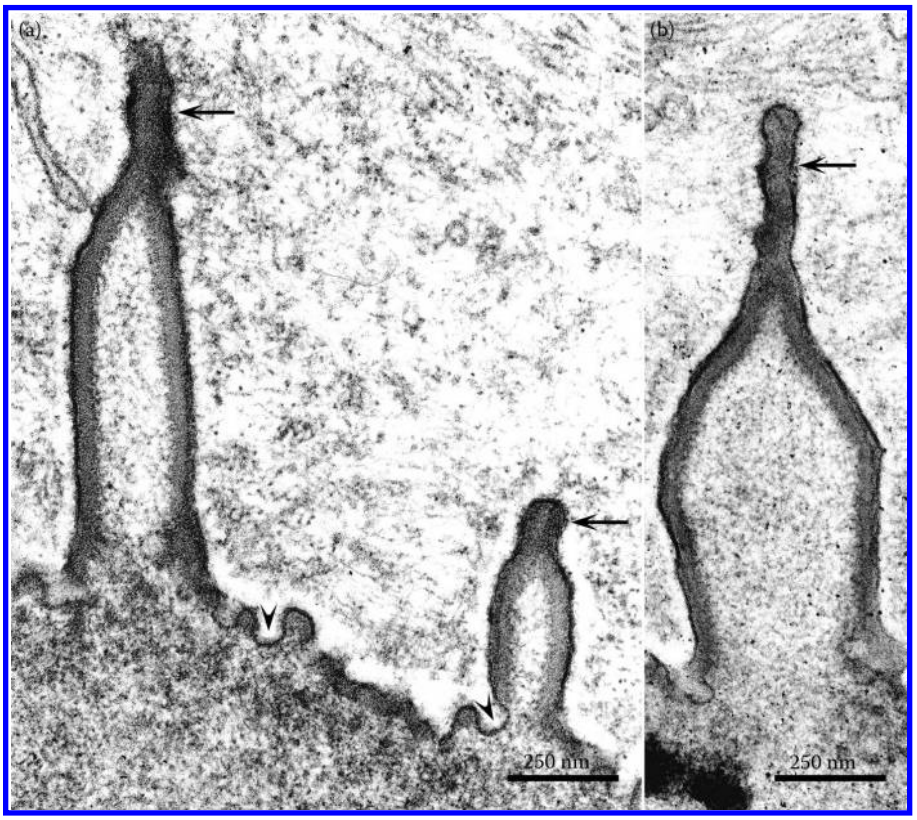


Figure 1.52 Two images (a, b) of type 38 *S. speeri* with steeple-like vp (arrow). The pvm is lined by an electron-dense layer, absent in invaginated areas (arrowheads). (Adapted from Dubey, J.P., Speer, C.A., Lindsay, D.S., 1998. *J. Parasitol.* 84, 1158–1164.)

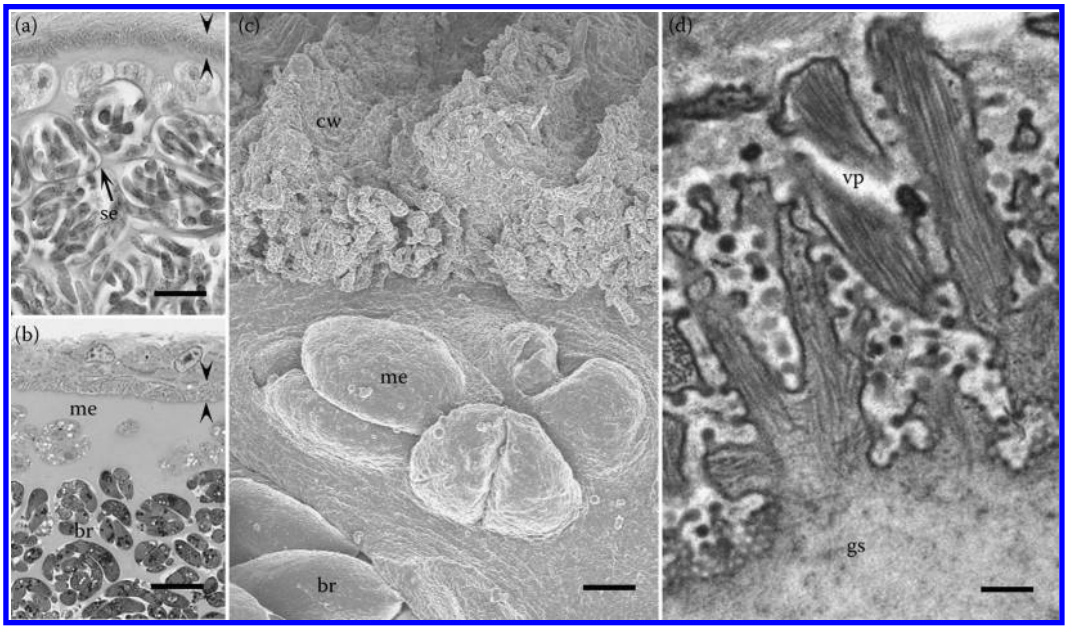


Figure 1.53 Structure of sarcocyst wall of *S. cafferi* observed with different techniques. (a and b), light microscopy; (c), SEM; and (d), TEM. Bar in (a) and (b) = 10 μm, in (c) = 2 μm, and in (d) = 300 nm. Note thin cyst wall (opposing arrowheads) in (a) and (b). (a) H and E stain, 5 μm section. (b) Toluidine blue stain, 1 μm section. (c) Surface and side views of the sarcocyst wall. (d) Branching of vp. Also note metrocytes (me), bradyzoites (br), and septa (se). (Adapted from Dubey, J.P. et al., 2014. *J. Parasitol.* 100, 817–827.)

of the gamont and discharged through the exocytosis pore (Figure 1.62b). In *S. cruzi*, the exocytosis pore is similar in size to micropores, but differs ultrastructurally (Figure 1.62b). The exocytosis pore protrudes slightly to produce a flattened dome on the surface of the gamont. The plasmalemma is invaginated at the apex of the dome, and the plasmalemma continues uninterrupted through the invagination. The single inner membrane is interrupted at the site of the micropore, allowing the invaginated plasmalemma to extend into the gamont cytoplasm. The margin of the interruption in the inner membrane is electron-dense, thick, and projects outward at approximately a 45° angle, instead of inwardly, as in micropores. The membranes surrounding the electron-dense bodies fuse with the invaginated membrane of the exocytosis pore, which causes the electron-dense bodies to be discharged into the PV, where it fuses to form a thin electron-dense layer (Figure 1.62b). This layer eventually becomes continuous to form the oocyst wall. Oocyst wall-forming bodies (WB), typical of *Eimeria* spp., and certain other coccidia, are lacking in macrogamonts of *Sarcocystis* spp. However, 2 types of electron-dense bodies in macrogamonts of *S. singaporensis* were interpreted as WB.[1364,1366] In *S. singaporensis*, the oocyst wall appears to be formed before fertilization and the WB disappear with formation of the oocyst wall.[1364]

Bradyzoites of *S. muris* have an exocytosis pore, through which dense granules are discharged into the PV.[529] The contents of the dense granules may stimulate the adhesion of the host-cell endoplasmic reticulum to the PV, to form a 3-membrane complex, or cause antigenic mimicry, preventing a host-cell immune response or inflammation.[529]

In most *Sarcocystis* species, macrogamonts begin development in the epithelium, but as development proceeds to an intermediate stage, the host cell degenerates and lyses (Figure 1.63), and the macrogamonts enter the lamina propria, where they undergo oogony and sporogony. In addition to forming the oocyst wall, the contents of the dense granules may also contain enzymes that cause

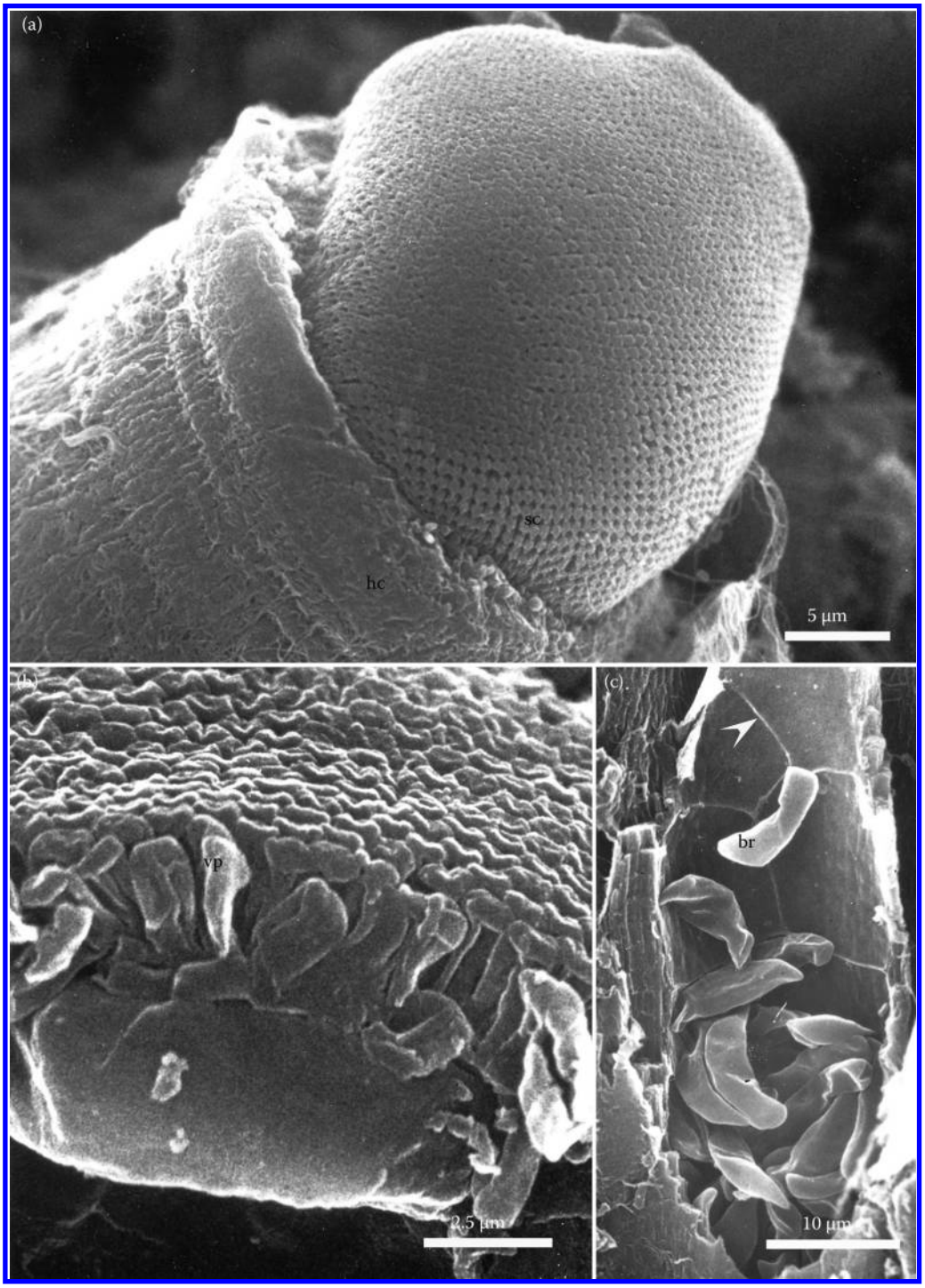


Figure 1.54 SEMs of sarcocysts. (a) *S. capracanis* sarcocyst (sc) protruding from the host cell (hc). The vp give a honeycomb appearance to the surface of the sarcocyst. (b) Cross-sectional view of the margin of a *S. capracanis* sarcocyst showing vp. (From Dubey, J.P. et al., 1984. *Int. Goat Sheep Res.* 2, 252–265. With permission.) (c) Ruptured *S. cruzi* sarcocyst showing bradyzoites (br) and septa (arrowhead). (From Dubey, J.P., 1982. *J. Protozool.* 29, 591–601. With permission.)

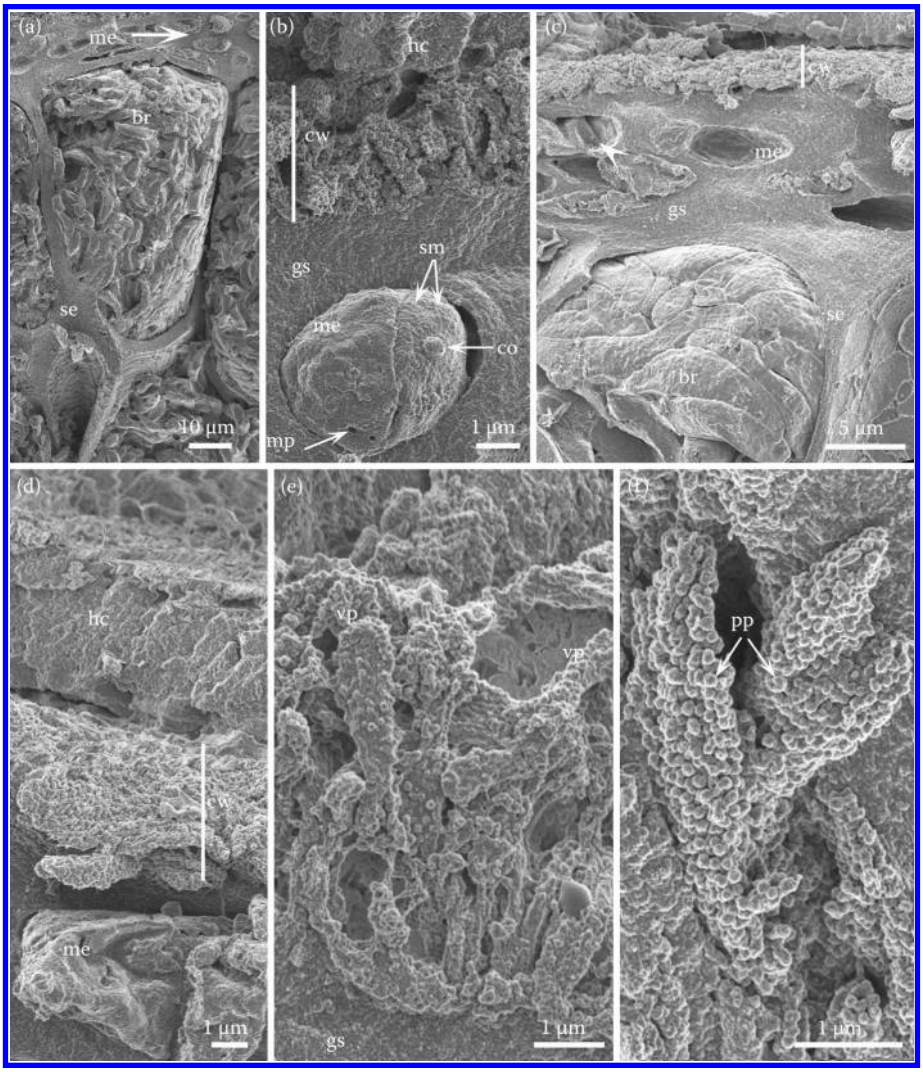


Figure 1.55 SEM of *S. fusiformis* sarcocysts. (a) Sectioned part of a sarcocyst. Note bradyzoites (br) are arranged in sacks enclosed by thick septa (se). Metrocytes are located in the gs. Empty spaces represent location of fallen metrocytes (me) during processing. (b) Sectioned sarcocyst. Note host cell (hc) enclosing the cyst wall (cw), thick, finely granular gs, and a single or group of metrocytes embedded in the ground substance. Part of outer covering of the metrocytes has ripped off showing a conoid (co), subpellicular tubules (sm), and micropore (mp). (c) Partly exposed sarcocyst. Note that vp are folded over the cyst wall (cw). The gs has several groups of metrocytes, 1 of which has 2 zoites (arrowhead). Also note a group of bradyzoites (br) enclosed in se. (d) Note combed appearance of the cw. (e) Higher magnification of the cw with irregularly shaped villar protrusions. (f) Higher magnification of the vp to show nearly identical papillomatous structures (pp) on the vp. (Adapted from Dubey, J.P. et al., 2015. *Parasitology.* 142, 385–394.)

lysis of the enterocyte, enabling the parasites to enter the lamina propria. Because the oocyst wall is being formed during this time, it seems likely that the electron-dense granules may contain cytolytic substances that cause lysis of the host cells. Neighboring cells are apparently not harmed.

In contrast to macrogamonts, microgamonts of some species of *Sarcocystis* can complete their development within the intestinal epithelium enterocyte or goblet cell (Figure 1.64). Several

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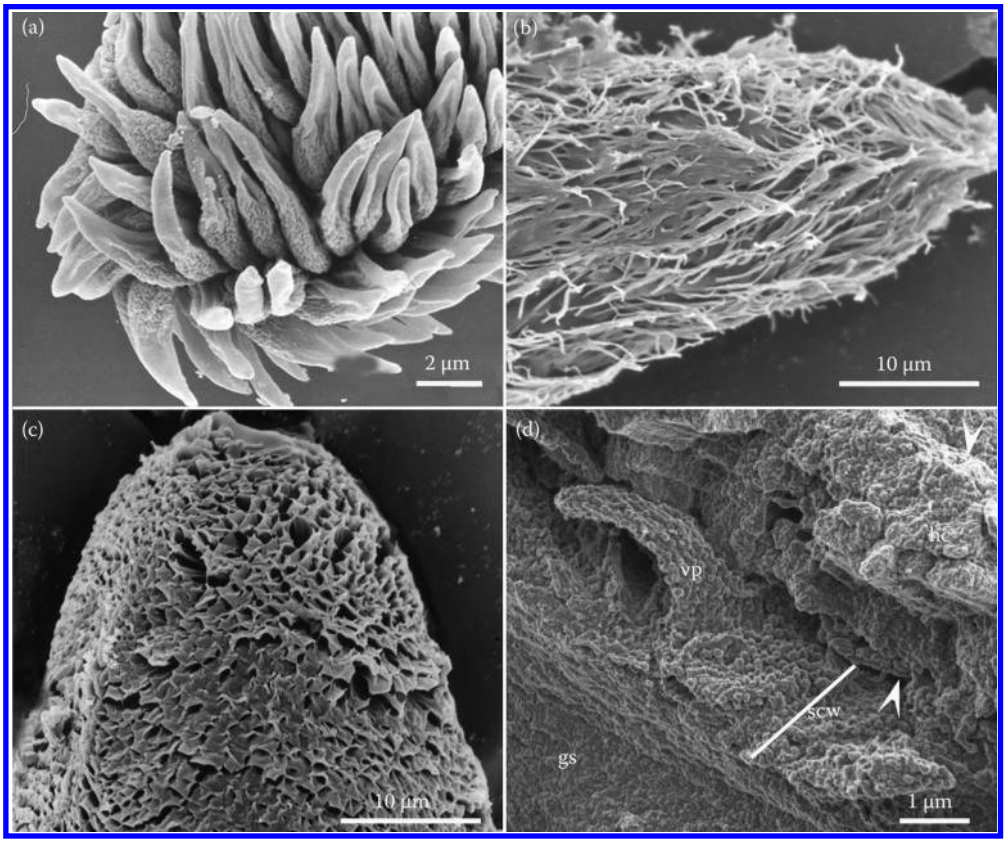


Figure 1.56 SEM of sarcocyst walls of 4 *Sarcocystis* species. (a) *S. mihoensis* with vp like a sea anemone; the vp are tapered towards the free end. (b) *S. arieticanis* with thread-like vp that are folded over the sarcocyst wall. (c) *S. tenella* with scale-like vp. (d) *S. fusiformis* shown at the surface view of the sarcocyst wall (scw). Note papillomatous growth on villar protrusions (vp), and host cell (hc). (Figure 1.56a–c courtesy of M. Saito, see Saito, M., Shibata, Y., Kubo, M., Itagaki, H., 1997. *Sarcocystis mihoensis* n. sp. from sheep in Japan. *J. Vet. Med. Sci.* 59, 103–106.)

ultrastructural features distinguish microgamonts from macrogamonts. Early microgamonts increase in size and become ovoid (Figure 1.65), have a well-developed endoplasmic reticulum, mitochondrion, numerous ribosomes, and a vesicular nucleus; but in contrast to macrogamonts, they lack electron-dense bodies and an exocytosis pore but do contain a mitotic spindle apparatus (Figure 1.64). The microgamont increases in size to about 10 µm in diameter, the inner membrane disappears, and the nucleus becomes lobulated, with each lobe situated near the gamont surface. A single microgamete develops immediately above each nuclear lobe. Two centrioles become basal bodies that form 2 flagella, each of which projects outward from the gamont. The chromatin becomes more compact and dense and, eventually, is situated in that portion of the nucleus nearest the gamont plasmalemma. The microgamete buds at the surface of the gamont (Figure 1.66), incorporating the dense portion of the nucleus and a mitochondrion and, eventually, the microgamete plasmalemma, separates from that of the gamont. Fully formed microgametes have a plasmalemma, 2 basal bodies associated with the base of each of 2 flagella, a dense nucleus, a mitochondrion, and several longitudinally oriented microtubules that extend from near the basal bodies to a point about midway along the nucleus (Figure 1.66). In *S. singaporensis*, the final stage of maturation on microgametes coincided with the degeneration of the parasitized enterocytes and loss of their brush border.[1365]

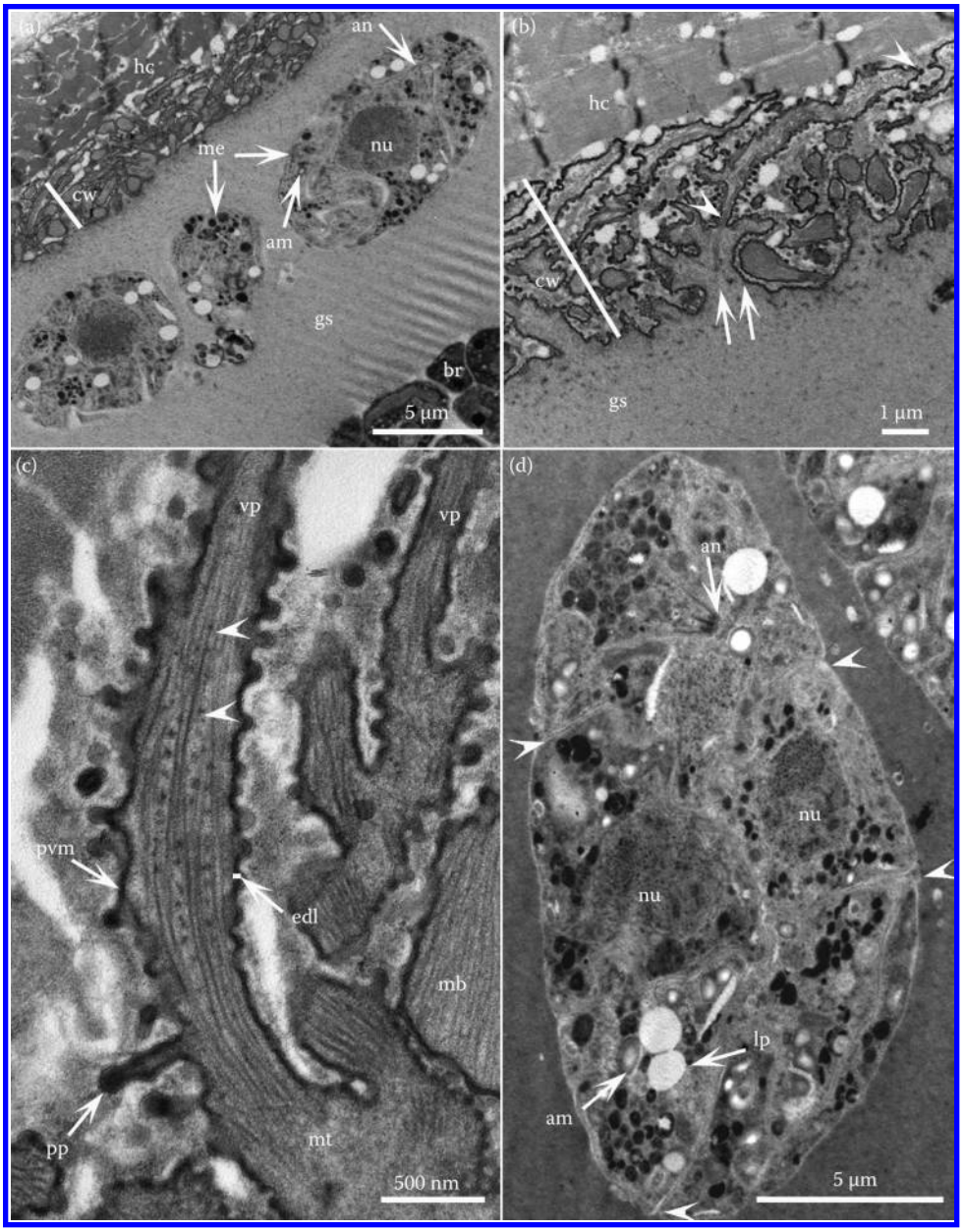


Figure 1.57 TEM of *S. fusiformis* sarcocyst wall. (a) The cyst wall (cw) is poorly demarcated from the host cell (hc). Three metrocytes (me) are located in gs. The me on the far right has a daughter anlagen (an), a few amylopectin (am) granules, a large nucleus (nu), and numerous electron-dense granular structures. (b) Highly branched vp. Double arrows point to branching of vp, 1 of the vp is very long (arrowheads) and juxtaposed against the host cell. The gs has a few granules at the junction of vp and the gs. (c) Longitudinal view of a vp with longitudinally oriented microtubules (mt). Arrowheads point to few indistinct structures, probably microtubules cut in cross section. Note dumbbell-like endings of pappilomatous structures (pp). (d) A metrocyte with 2 nuclei (nu), several am, lipid bodies (lp), fissure-like structures (arrowheads), and absence of micronemes and rhoptries. Note zoite anlagen (an) with its conoid. Numerous unidentified electron-dense structures of varying sizes are present. (Adapted from Dubey, J.P. et al., 2015. *Parasitology.* 142, 385–394.)

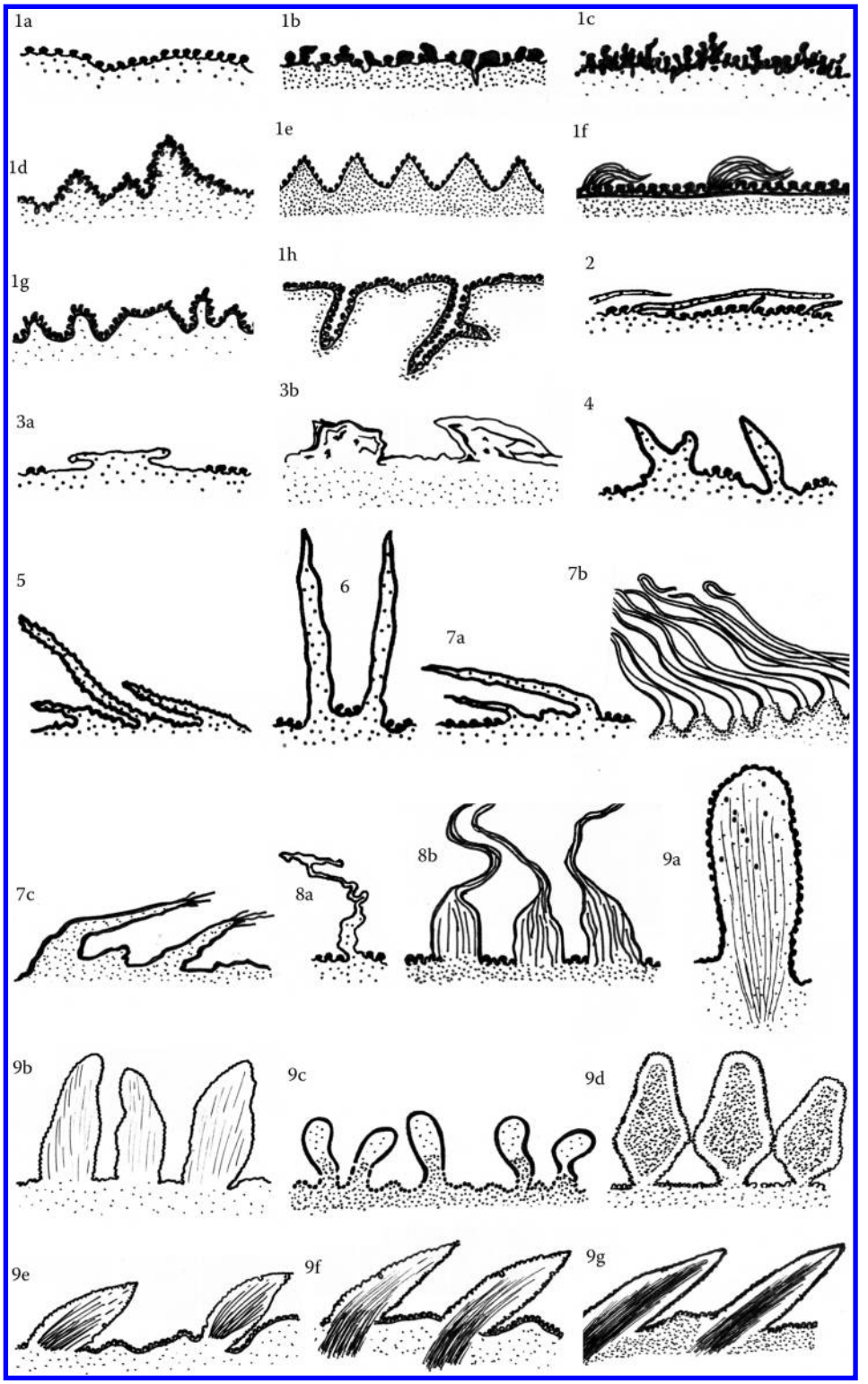


Figure 1.58 Line drawings of different sarcocyst wall types, 1a–9g (see Table 1.2 for description).

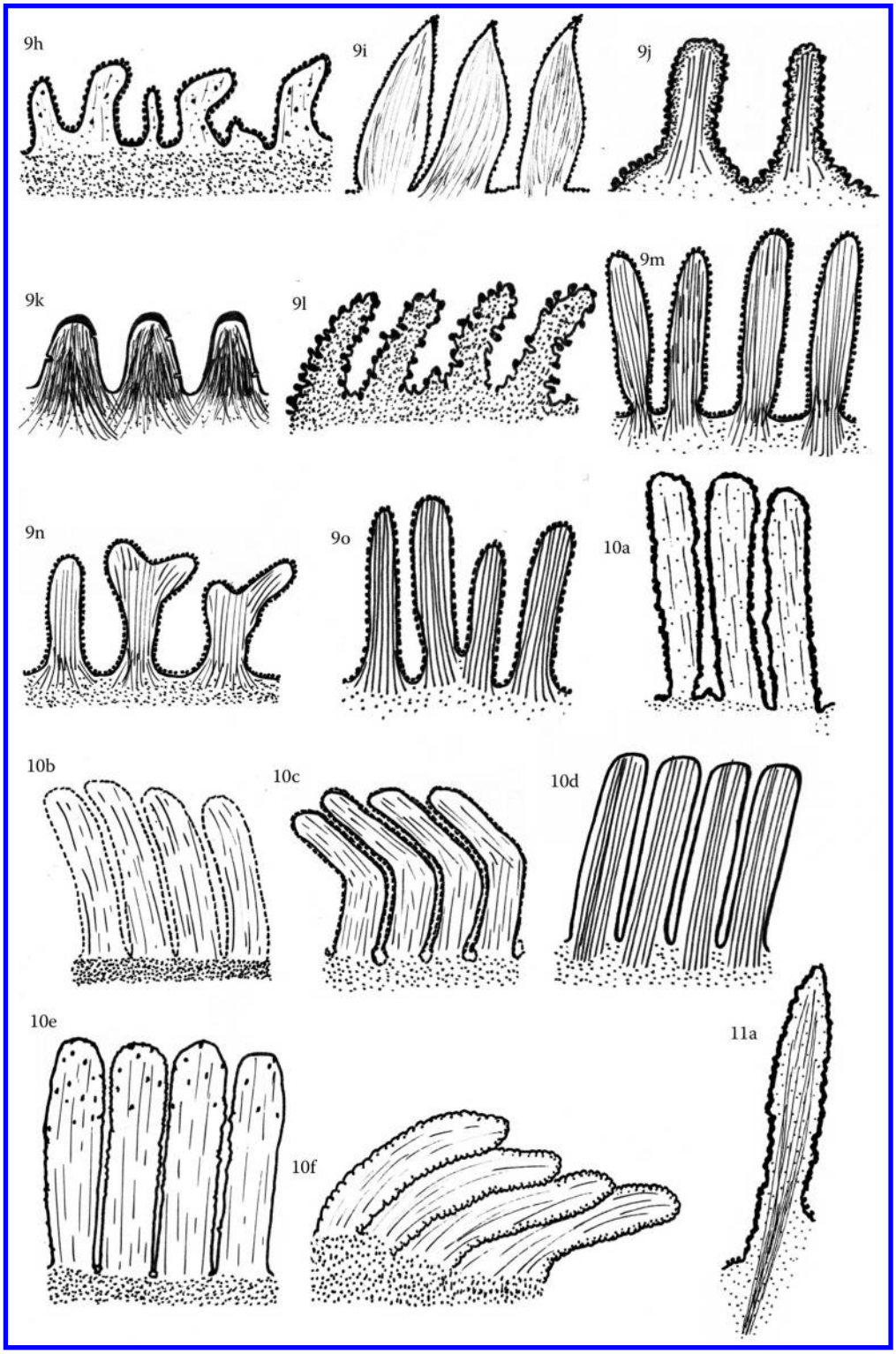


Figure 1.59 Line drawings of different sarcocyst wall types, 9h–11a (see Table 1.2 for description).

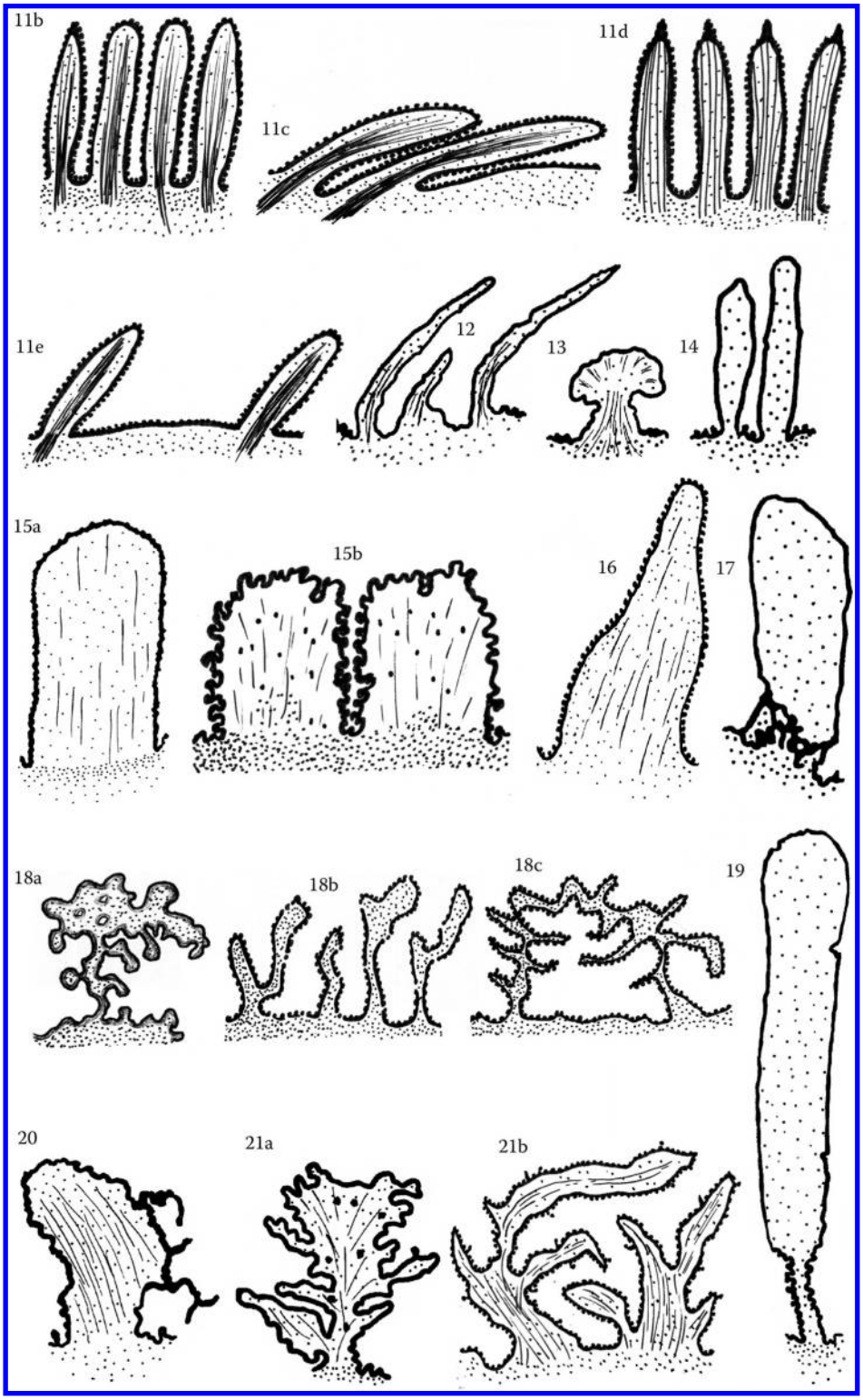


Figure 1.60 Line drawings of different sarcocyst wall types, 11b–21b (see Table 1.2 for description).

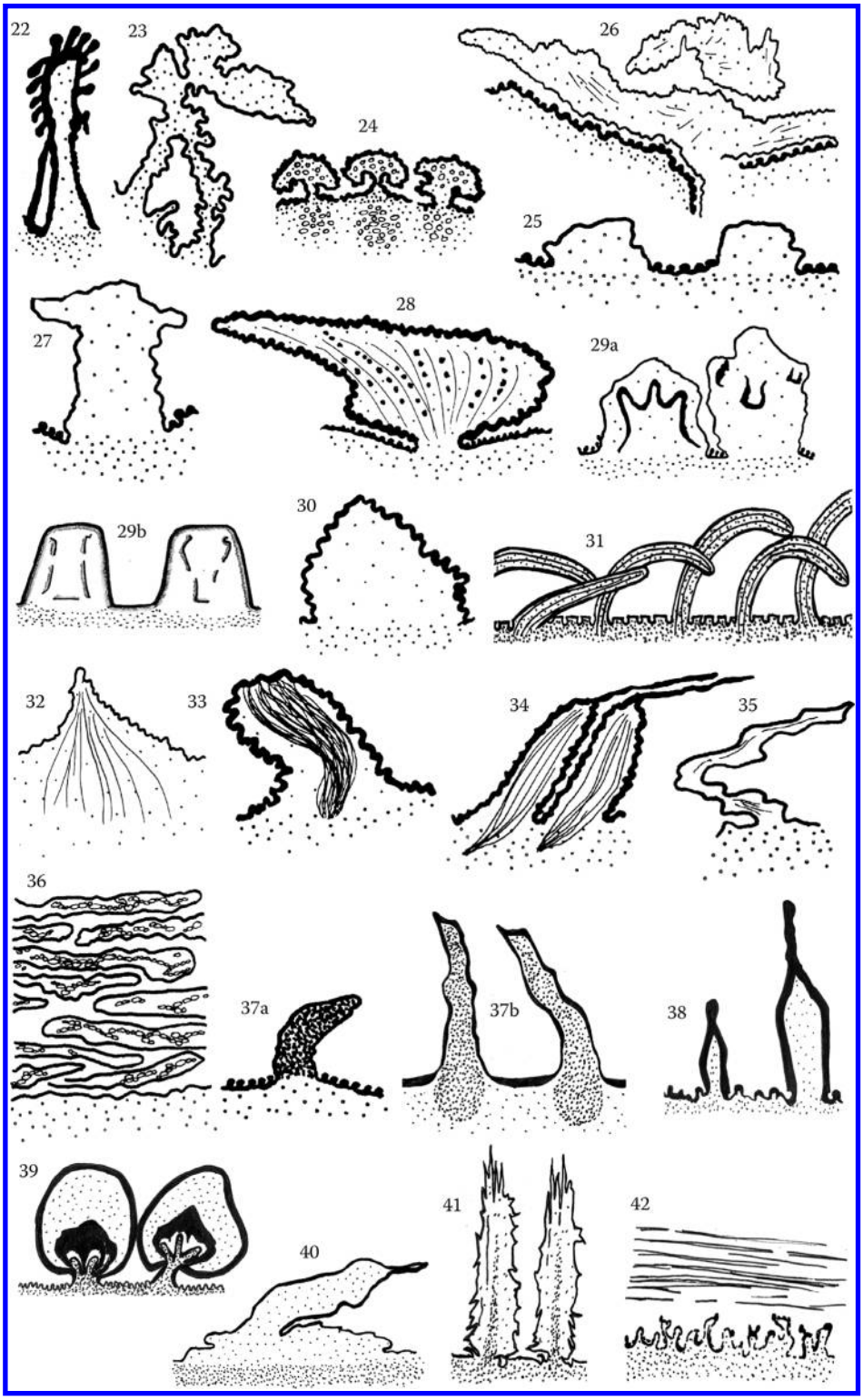


Figure 1.61 Line drawings of different sarcocyst wall types, 22–42 (see Table 1.2 for description).

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Table 1.2 Characteristics of *Sarcocystis* Sarcocyst Wall Types

Type	Subtype	Sarcocyst Wall	Representative Species	Location in the Book
1	a	Pvm with minute undulations or knob-like blebs with rounded ends and spherical vesicles with short stalk inpocketing into host cell, gs smooth	*S. muris*	Figure 1.42b
	b	Pvm with pleomorphic blebs, conical, irregular	*S. schneideri*	Chapter 22
	c	Similar to type 1a but pvm with hyphenated blebs	*S. cymruensis*	Table 21.2
	d	Similar to type 1a but blebs interspaced with conical protrusions	*S. wobeseri*	Table 17.3
	e	Similar to type 1d but pvm with conical vp at uneven distances and blebs with conical to round tops; the sarcocyst wall appears spiny by LM	*S. sehi*[a]	Table 21.5
	f	Similar to type 1a but blebs interspersed with filliform vp lined with smooth mt	*S. melis*[a]	Table 19.1
	g	Pvm with stump-like protrusions at irregular distances	*S. cornixi*	Table 17.3
	h	Similar to type 1a, but invaginations into the interior of the sarcocyst	*S. rauschorum*	Figure 1.42a
2		Similar to type 1a, except that thin, hirsute structures arise from some of the apices of the undulating pvm	*S. wapiti*[a]	Figure 1.42c
3	a	Pvm undulated, vp flattened mushroom-like	*S. ferovis* [a]	Figure 1.43a
	b	Like type 3a but irregular mushroom-like vp not flattened	*S.* sp. from black jackal [a]	Table 19.1
4		Irregularly shaped vp with a finely granular core, pvm undulating at the base of the vp and smooth or undulating over the surface of the vp	*S. sigmodontis*	Figure 1.43b
5		Pvm highly folded to form hirsute vp with granular cores, gs very thin	*S. sulawesiensis* [a]	Figure 1.43c
6		Vp finger-like, arising from a dome-shaped base, pvm with minute undulations in the dome-shaped base, but relatively smooth over the surface of the vp. The vp with fine granular core and oriented perpendicularly to the sarcocyst surface	*S. capreolicanis*	Figure 1.43d
7	a	Elongated vp arising from a dome-shaped base, without fibrilar elements, and folded over the cyst wall	*S. cruzi*	Figure 7.2a
	b	Vp with 3 distinct regions: a dome-shaped base, an intermediate finger-like segment, and a thin thread-like distal segment. The intermediate and distal segments are often bent 90° and run parallel to the sarcocyst surface. In certain areas, however, the vp are arranged collectively in conical tufts. The core contains coarse granules in the base segment and fine granules in the intermediate and distal segments	*S. arieticanis* [a]	Figure 1.44a
	c	Vp very long, slender, folded over the cyst wall. Villar tips with a sharp spike-like structures	*S. stehlinii* [a]	Table 22.1
8	a	Similar to type 7, except that the basal and intermediate segments appear as 1, vp narrow abruptly to form the branched distal segment	*S. gruneri* [a]	Figure 1.44b and c
	b	Similar to type 8a, but vp have mt	*S.* sp. from Sika deer [a]	Table 18.1
9	a	Pvm with minute undulations, lined by thick edl, vp conical or tongue-shaped, close together, mt extending from tip to the gs, mt with edg	*S. campestris*	Figure 1.45a
	b	Same as type 9a but vp with indistinct mt without edg	*S. greineri*	Table 20.2
	c	Same as type 9a but pv wide apart at uneven distances, vp of variable sizes and without mt and edg	*S. felis*	Figure 15.1

(*Continued*)

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Table 1.2 (*Continued*) Characteristics of *Sarcocystis* Sarcocyst Wall Types

Type	Subtype	Sarcocyst Wall	Representative Species	Location in the Book
	d	Same as type 9a but vp leaf-like with a narrow stalk, wide in the middle, and rounded tapered end. The interior of the vp filled with vesicular electron-dense material	*S. gongyli*	Table 22.1
	e	Same as type 9a, except vp at irregular distances, folded over the cyst wall, vp with invaginations of the pvm, mt in vp denser at the base and without edg	*S. ramphastosi*	Table 17.3
	f	Same as type 9e but mt of vp extending deeper in gs and the mt much denser in gs versus in vp	*S. sulfuratusi*	Table 17.3
	g	Same as type 9f but mt in vp very dense both in the gs and in vp	*S. ursusi*	Table 19.1
	h	Same as type 9a, but with straight to sloping vp, with indistinct mt and dark granules	*S. kitikmeotensis*	Table 19.1
	i	Vp elongated and spear-like, mt without edg, numerous indentations of the pvm on the entire villar surface	*S. markusi*	Chapter 5
	j	Vp upright finger-like with knob-like structures arising from the pvm	*S. cameli* [a]	Figure 12.2a
	k	Vp short, stubby with criss-crossing mt, extending to gs. Villar tips with thick edl	*S. wenzeli* [a]	Figure 16.1
	l	Vp vertical to sloping with fine microtubules not extending into the gs	*S. phoeniconaii*	Table 17.3
	m	Vp similar to type 9d, but with profuse mt without edg extending into the gs	*S. mephitisi*	Table 19.1
	n	Vp similar to type 9m, but some villar tips bifurcated	*S. inghami*	Table 19.1
	o	Vp sloping to straight, mt without edg and extending from villar tips into the gs	*S. jaypeedubeyi*	Table 21.3
10	a	Similar to type 9a, but vp tightly packed, upright, few mt, not extending into gs	*S. odoi*	Figure 1.45c
	b	Similar to type 10a, but vp with profuse fine mt throughout the villar core	*S. hominis*	Figure 7.2
	c	Similar to type 10a, but vp elongated and bent at an angle and vesicles on pvm at the base of villi	*S. dubeyi*	Figure 10.1f
	d	Similar to 10b, but mt extending into the gs	*S. hemioni*	Table 18.1
	e	Similar to 10a, but vp with patchy electron-dense material on pvm and the core at the villar tips	*S. tarandi*	Table 18.1
	f	Vp folded over the cyst wall	*S.* sp. from black jackal [a]	Table 19.1
11	a	Vp similar to types 9e, but mt extend from the villar tips to the plasmalemma of metrocytes/bradyzoites	*S. fayeri* [a]	Figure 1.45b
	b	Similar to type 11a, but vp microtubules not extending deeper in gs, never reaching bradyzoites/metrocytes	*S. falcatula*	Figures 1.46, 17.1
	c	Similar to type 11b, but vp folded over the cyst wall giving it a thin wall appearance	*S. bertrami*	Chapter 11
	d	Vp similar to 11b, but with stylet-shaped villar tips	*S. lindsayi*	Table 17.2
	e	Vp similar to type 11b, but wide apart	*S.* sp. from sea lion [a]	Table 17.3
12		Tightly packed, thin, finger-like vp with mt in the basal one-third of the vp	*S. sybillensis*	Figure 1.47a
13		Vp widely spaced, mushroom-like with mt extending from the core into the gs. The pvm has irregularly spaced indentations	*S. mucosa* [a]	Figure 1.47b
14		Vp tightly packed, cylindrical-shaped, the tips of which contain disk-shaped plaques	*S. tenella*	Figure 1.48a and b

(*Continued*)

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Table 1.2 (*Continued*) Characteristics of *Sarcocystis* Sarcocyst Wall Types

Type	Subtype	Sarcocyst Wall	Representative Species	Location in the Book
15	a	Upright, closely packed "tombstone-like" vp with round tops, and a core containing scattered granules, scattered thin mt	*S. rangiferi*	Figure 1.48c
	b	Like type 15a but vp with deeply indented margins	*S. africana*[a]	Table 18.2
16		Similar to type 15, except that the sarcocyst wall consists of alternating conical and club-shaped vp with criss-crossing mt	*S. youngi*	Figure 1.48d
17		Rectangular vp with disc-shaped plaque at the apex and lateral margins and the pvm highly branched at the base of vp	*S. odocoileocanis*	Figure 1.49a
18	a	Vp branched and irregularly shaped, appearing T-shaped in appropriately cut sections, with a granular core	*S. dispersa*	Figure 1.49b
	b	Like type 18a, but vp elongated and U-like branching, and indentations on pvm	*S. muriviperae*	Table 21.1
	c	Vp highly branched anastomosing	*S. zamani*[a]	Table 21.2
19		Vp club-shaped with a cylindrical stalk and a sausage-shaped distal segment. In older sarcocysts, the distal segment may be shorter and irregular in shape. Invaginations of the pvm are numerous in the stalk and widely scattered in the distal segment. In young and intermediate sarcocysts, the core is filled with numerous small vesicles and membranous whorls, whereas in mature sarcocysts the core contains fine granules with localized areas of small vesicles.	*S. singaporensis*	Figures 1.28–1.30, 1.49c
20		Vp angular that may be pyramidal, trapezoidal, or mushroom-like. Serpentine projections arise from the surface of the vp as well as from the surface of the sarcocyst. The entire sarcocyst surface is covered by tiny bristles that project outward at 90° from the pvm. Vp core contains tightly packed mt	*S. medusiformis*[a]	Figure 1.50a and b
21	a	Vp with cauliflower-like branches containing microtubules with edg	*S. gigantea*	Figure 1.50c
	b	Vp highly branched, appear mesh-like by SEM, and like a dead tree by TEM, uniform papillomatous projection on vp with door knob tops	*S. fusiformis*	Figures 1.53, 1.55, 1.57
22		Vp with a proximal, sharply conical base and a distal segment that is cocklebur-like with short, radiating projections. Cross sections of the distal segment appear as cogwheels. Some of the projections from the distal segment connect with the margin of the sarcocyst wall. The core contains numerous vesicles especially in the distal half	*S. villivillosi*[a]	Figure 1.50d
23		Vp with anastomosing branches that contain fine granules and fine mt	*S. rileyi*	Figure 1.51a
24		Vp mushroom-like with a core of tightly packed mt, and mushroom-like protrusions also arise from the surface of other protrusions	*S. cornagliai*	Figure 1.51b
25		Similar to type 1a but blebs interspersed with dome-shaped small elevations at irregular distances and containing few mt	*S.* sp. from badger[a]	Table 19.1
26		Vp flattened and very irregular linguiform containing small invaginations on pvm. Pvm at villar tips lined by thick electron-dense material	*S. hardangeri*	Table 18.1
27		Vp palisade-like and thick T-shaped smooth surface	*S. sibirica*	Table 18.2

(*Continued*)

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Table 1.2 (*Continued*) Characteristics of *Sarcocystis* Sarcocyst Wall Types

Type	Subtype	Sarcocyst Wall	Representative Species	Location in the Book
28		Vp undulating, palisade-like or rhombic-like with a stalk at the base, expanded laterally, mt with edg	*S. hirsuta*	Figures 7.4, 10.1d
29	a	Vp molar tooth-like by SEM. By TEM interior of vp with variable-shaped condensations of electron-dense structures	*S. danzani*[a]	Table 18.2
	b	Similar to type 29a but vp with smooth margins and hollow on one side	*S. phacochoeri*	Table 18.2
30		Parasite induced host-cell encapsulation, vp irregularly triangular with indented margins looking like shark teeth	*S. dubeyella*	Table 18.2
31		Vp up to 14 μm long, folded over the sarcocysts surface, vp with mt in the core without edg. Battlement-like invaginations of pvm	*S. suihominis*[a]	Chapter 6
32		Vp thorn-like with mt radiating into the gs	*S. ippeni*[a]	Figure 12.2b
33		Vp thumb-like with a compact central bundle of mt in the core and with small invaginations on the surface	*S. hippopotami*[a]	Table 18.2
34		Vp finger-like with mt penetrating the ground substance, small invaginations on the surface and hirsute at the tip	*S. giraffae*[a]	Table 18.2
35		Vp kinked finger-like with scattered mt in the interior	*S. klaseriensis*[a]	Table 18.2
36		Vp in rows parallel to the cyst wall surface and strap-like with chain-like osmiophilic structures in the interior	*S. camelopardalis*[a]	Table 18.2
37	a	Pvm with invaginations deep in to the gs. Vp spine-like, arising at irregular distances. Interior of vp edg expanding into the middle of gs with irregularly indented margins and condensed matrix	*S. simonyi*	Table 22.1
	b	Vp slender, up to 7 μm long and lined by electron-dense material from the tip to knob-like ending inside the gs	*S. gallotiae*	Table 22.1
38		Vp steeple-shaped surmounted by a spire	*S. speeri*[a]	Figure 1.52
39		By SEM, vp pallisading, like a sea anemone. By TEM, vp elongated with tapered end. Condensation of electron-dense material at the base of vp giving mushroom-like appearance	*S. mihoensis*[a]	Figure 1.56a
40		Vp with flat wart-like elevations. Short pit-like parts on pvm between vp. Vp without mt and tipped ends	*S. melampi*[a]	Table 18.2
41		Vp upright, finger-like corrugated pvm. Villar tips with deep invaginations giving serrated appearance. Villar cores with electron-dense material that appears thick in the middle	*S.* sp. from Hispanic ibex[a]	Table 18.2
42		Macroscopic with secondary cyst wall. Pvm lined with thick edl, numerous irregular vp	*S. hoarensis*[a]	Table 22.1

Pvm = parasitophorous vacuolar membrane, gs = ground substance, vp = villar protrusions, mt = microtubules, edl = electron dense layer, edg = electron dense granules.
[a] Unique types reported only for 1 *Sarcocystis* species.

1.3.1.5 Fertilization

Fertilization usually occurs within 18 h PI.[819,1364,1598] Microgametes penetrate the macrogamont by an unknown process (Figure 1.67). The plasmalemma of the microgamete fuses with that of the macrogamont to create a cytoplasmic bridge through which the microgamete nucleus enters the macrogamont (Figure 1.67). Evidently, soon thereafter, the cytoplasmic bridge closes and the gamete nuclei fuse to form a zygote. The remnant of the microgamete becomes vesicular and eventually disappears *in situ* (Figure 1.68). What proportion of microgametes succeeds in fertilizing the macrogamete is unknown.

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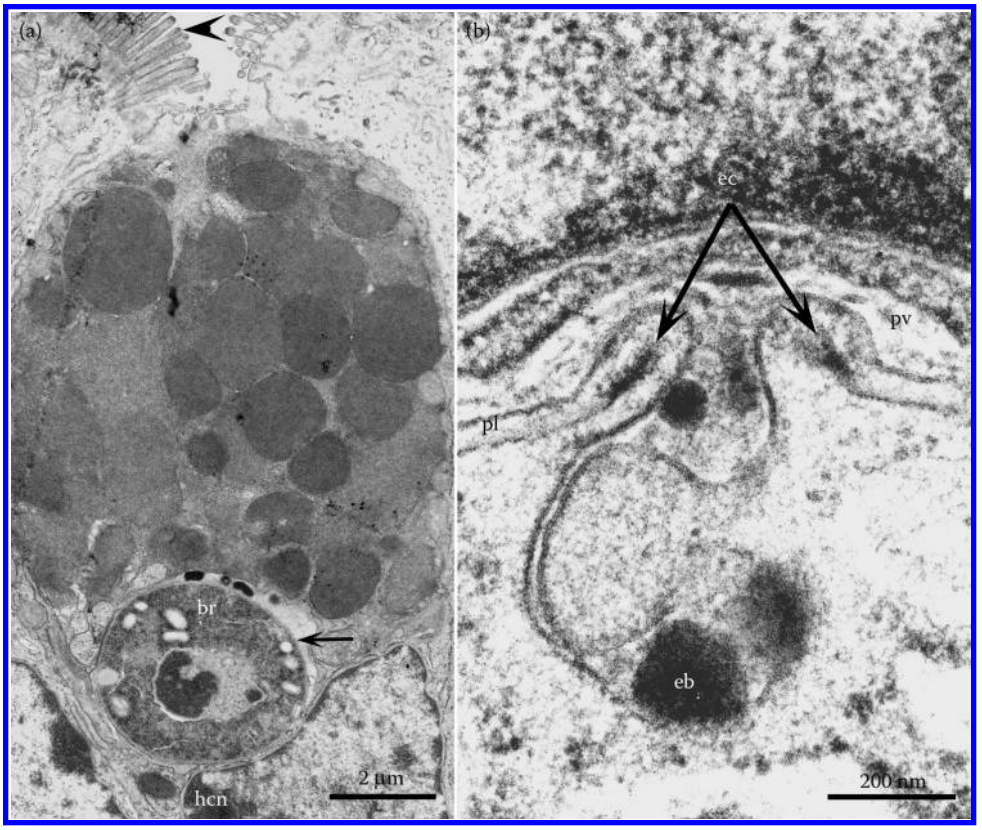


Figure 1.62 TEM of *S. cruzi* bradyzoites transforming to gamonts. (a) Bradyzoite (br) located between mucin droplets and nucleus of intestinal goblet cell of an experimentally infected coyote. Arrowhead points to microvillus border. (b) Exocytosis pore discharging electron-dense bodies into parasitophorous vacuole.

1.3.1.6 Oocyst

Unsporulated oocysts consist of a sporont surrounded by an oocyst wall. The oocyst wall has an electron-dense, finely granular outer layer, and an inner layer of 1–4 membranes (Figure 1.68).[803] Electron-dense projections arise from the outer surface of the oocyst wall (Figure 1.68). The sporont is limited by a plasmalemma and a single inner membrane and contains a nucleus, endoplasmic reticulum, mitochondrion, ribosomes, Golgi complex, amylopectin bodies, micropores, and an occasional electron-dense body (Figure 1.68).

1.3.1.7 Sporocyst

There are no reports on the ultrastructural changes during sporulation of oocysts of *Sarcocystis* spp. The sporocyst walls are composed of a thin continuous outer layer, and a thick inner layer consisting of 4 plates joined at sutures similar to those in related coccidia. The sporocyst wall of *S. cruzi* has a lamellar outer layer, approximately 50 nm thick, consisting of alternating electron-dense and electron-lucent layers and an electron-dense inner layer, approximately 130 nm thick, that is separated from the outer layer by a single membrane (Figure 1.69a). The inner layer has alternating bands (≈5 nm thick) of electron-dense and electron-lucent material perpendicular to the surface

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Figure 1.63 TEM of 2 *S. cruzi* macrogamonts in a degenerate goblet cell in the small intestine of an experimentally infected coyote.

of the sporocyst. A thickening (150–180 nm thick) is seen at the margin of each plate. A thin strip of electron-dense material (≈20–30 nm thick) is interposed between the thickenings of 2 apposing plates forming a suture (Figure 1.69b). In SEMs, the sutures appear as ridges on the surface of the sporocyst (Figure 1.69a). Treatment of sporocysts with NaOCl (commercial laundry bleach) usually results in the removal of the outer layer of the sporocyst wall (compare Figure 1.70a and b). Sporulated oocysts and sporocysts are located in the lamina propria. The mechanism of migration and release of sporocysts in the intestinal lumen is unknown.

During excystation, bile salts and/or trypsin act upon the sutures, causing the plates to separate from the interposed strip. The edges of the plates curl inward and openings appear where plates had been adhered to one another, enabling the release of sporozoites (Figures 1.69 and 1.70). However, *S. muris* sporocysts can excyst in mouse peritoneum, in the apparent absence of trypsin or bile.[683,1503] *S. neurona* sporocysts are equally infective to mice by the oral and the subcutaneous routes, indicating that trypsin is not necessary for excystation of some *Sarcocystis* species.[470]

1.3.1.8 Sporozoites

Sporozoites of *S. cruzi* are banana-shaped and have all the ultrastructural features described earlier for bradyzoites, such as conoid, apical rings 1 and 2, polar rings 1 and 2, micronemes,



Figure 1.64 TEM of an early *S. cruzi* microgamont in the small intestine of an experimentally infected coyote showing a portion of the spindle apparatus (arrow). Note profuse endoplasmic reticulum (er).

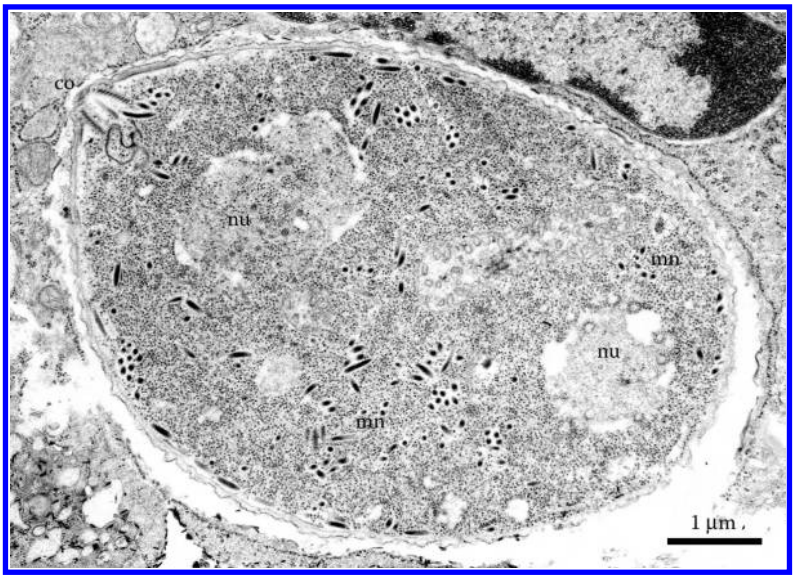


Figure 1.65 TEM of an early *S. cruzi* microgamont in the intestine of an experimentally infected coyote showing 2 lobes of a nucleus. Note the nuclear pores and scattered micronemes (mn) and conoid (co) of the original bradyzoite.

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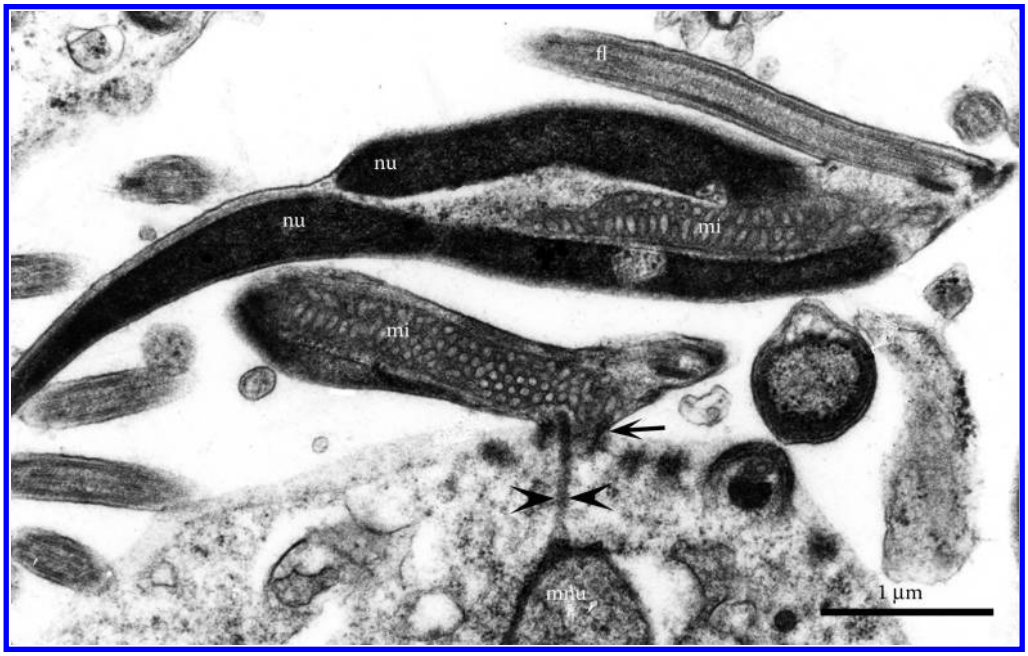


Figure 1.66 TEM of 2 *S. suihominis* microgametes in longitudinal section, 1 of which is still connected (arrow) to the surface of the microgamont in which a nuclear bridge (double arrowheads) extends from the residual nucleus into the microgamete. Note the flagellum (fl), nucleus (nu), and mitochondrion (mi) of the microgametes. (From Mehlhorn, H., Heydorn, A.O., 1979. *Z. Parasitenkd*. 58, 97–113. With permission.)

rhoptries, micropore, nucleus, mitochondrion, Golgi complex, endoplasmic reticulum, ribosomes, amylopectin granules, pellicle, and subpellicular microtubules. Sporozoites differ, however, from bradyzoites and merozoites by possessing a virus-like crystalloid body, and structures similar to rhoptries and micronemes in the posterior half of the body (Figure 1.71). The crystalloid body consists of electron-dense and electron-lucent granules (≈38 nm in diameter), and is usually located in the posterior half of the sporozoite. Some sporozoites have several crystalloid bodies randomly scattered throughout the sporozoite, with some bodies anterior to the sporozoite nucleus. Also, various other organelles and inclusion bodies, such as rhoptries, micronemes, and amylopectin granules, may be dispersed among the granules of the crystalloid body. This body is probably analogous to the homogenous refractile bodies of *Eimeria* spp., in which the body is believed to represent an energy or amino acid reserve.[1991] The crystalloid body was not found in *S. neurona* sporozoites.[1079]

1.3.1.9 Schizogony

Sarcocystis schizonts multiply by endopolygeny, in which the nucleous becomes multilobbed, and numerous merozoites bud simultaneously at the surface of the schizont (Figures 1.21 and 1.22).

Schizonts differ from other intracellular stages in the life cycle of *Sarcocystis* spp. in that they are located free within the cytoplasm of the host cell and are not surrounded by a PV (Figures 1.72 through 1.82). Schizonts also lack subpellicular microtubules. Early phases of sporozoite entry and development of *S. singaporensis* sporozoites into schizonts were reported.[887,891] *S. singaporensis* sporozoites entered several types of rat cells but developed only in rat endothelial and pneumocytes,

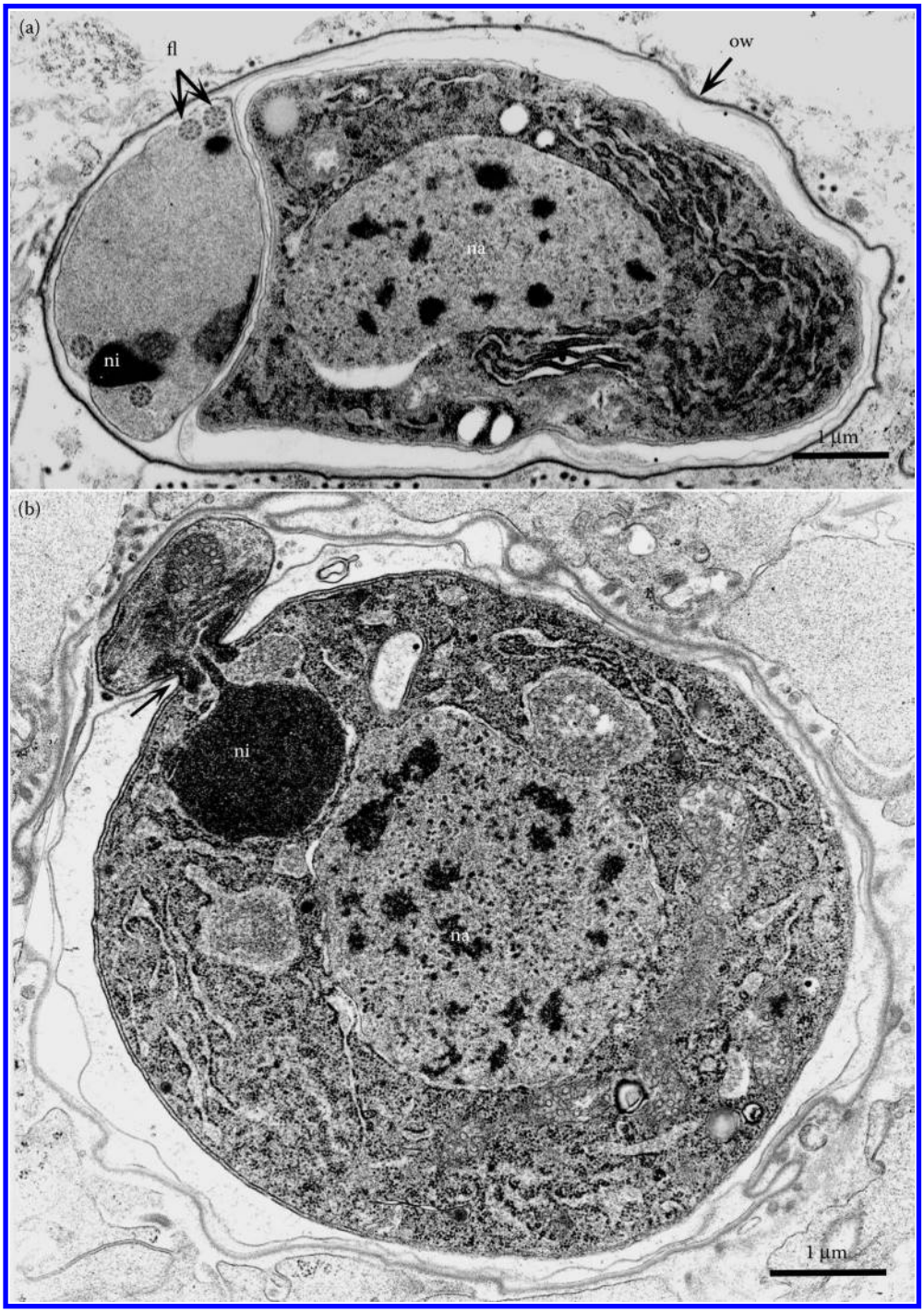


Figure 1.67 TEM of *S. cruzi* fertilization. (a) Early stage in fertilization of macrogamont by a microgamete. Note the oocyst wall (ow), a large nucleus (na) of the macrogamete, and small nucleus (ni), and flagella (fl) of microgamete. (b) Intermediate stage in fertilization in which the microgamete nucleus (ni) is entering the macrogamont through a cytoplasmic bridge (arrow) created by fusion of the plasmalemma of the microgamete and macrogamont. (Adapted from Sheffield, H.G., Fayer, R., 1980. *Proc. Helminthol. Soc. Wash.* 47, 118–121.)

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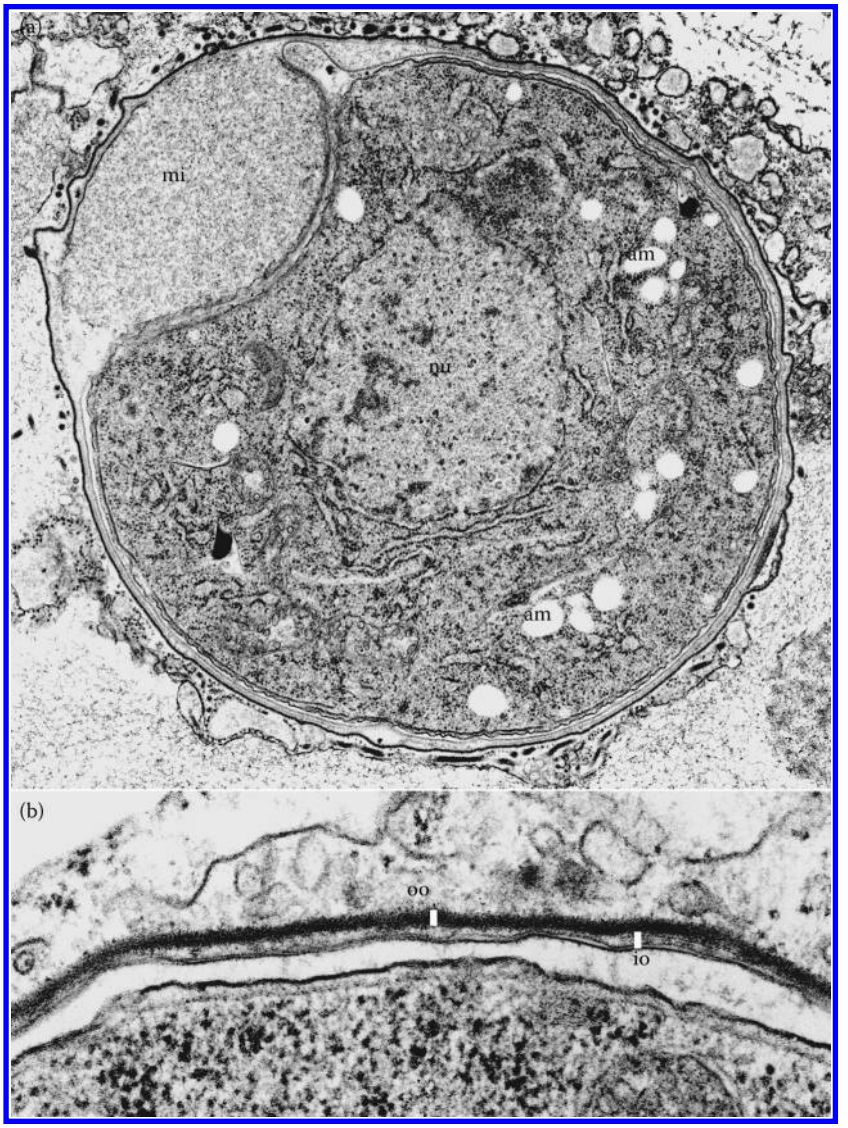


Figure 1.68 TEM of *S. cruzi* zygote. (a) A zygote, soon after fertilization, showing a remnant of the microgamete. Note the large nucleus (na) and amylopectin granules (am) of the macrogamete. (b) The oocyst wall and pellicle of the zygote. Note that the oocyst wall consists of an inner layer (io) of 2 membranes and an electron-dense outer layer (oo).

indicating that specific receptors or physiological conditions may be needed for development of schizonts. Most sporozoites entered cells within 2 h of inoculation but schizont development was delayed until the third day. At 1 h PI, some sporozoites were located within PVs, and the findings were confirmed ultrastructurally, while some were located free in the cytoplasm; these intravacuolar sporozoites had disappeared by 2 DPI. Even young schizonts, with an enlarged nucleus and condensed crystalloid body, were seen free in the host cytoplasm. The formation of a PV and subsequent escape may be an adaptation from the free-living state to the intracellular state.

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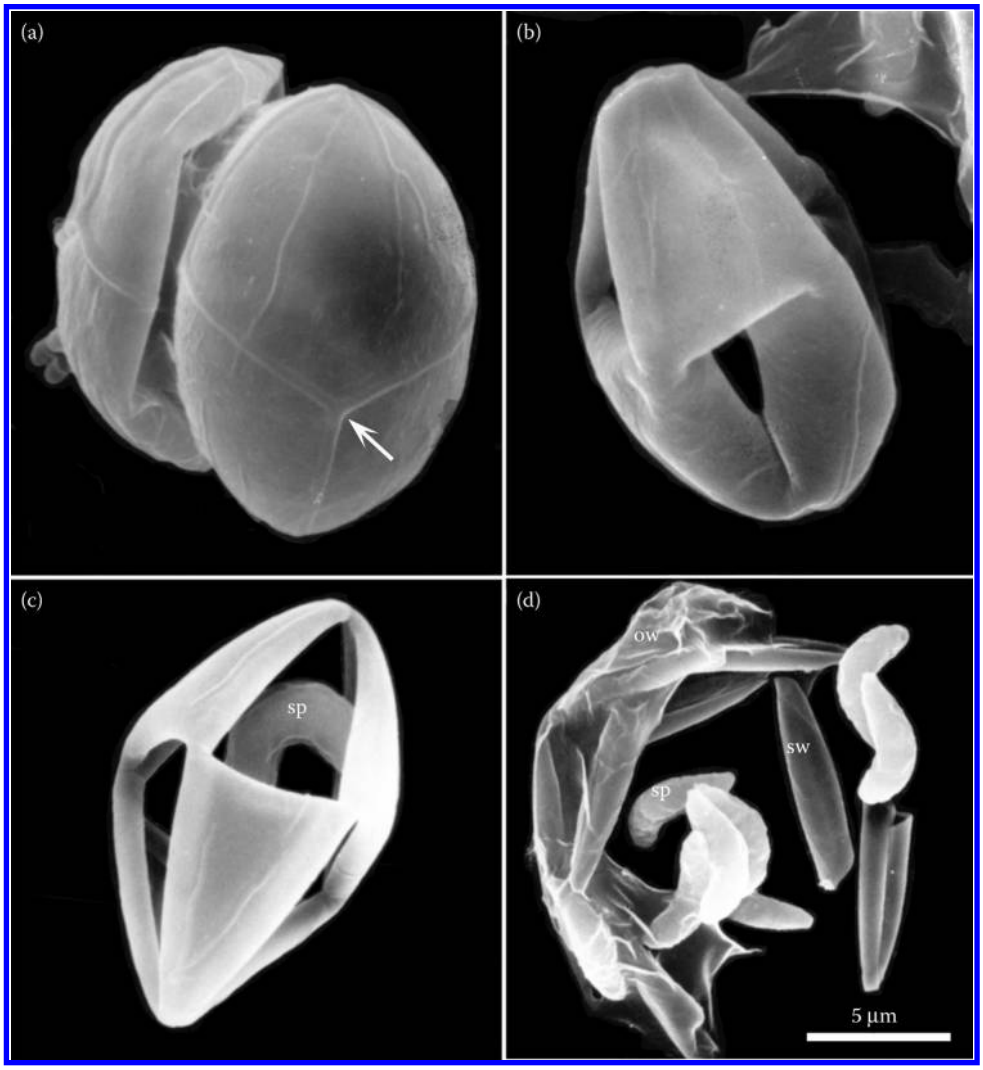


Figure 1.69 SEMs showing sporocysts and excystation of *S. miescheriana* sporozoites. (a) Two sporocysts held together by a thin oocyst wall. Junctions between apposing plates appear as ridges (arrow) in the sporocyst wall. (b) Intermediate stage in excystation in which the apposing plates in the sporocyst wall have partially separated from each other in response to excysting fluid. (c) A single sporozoite (sp) is visible within a sporocyst in which the walls have almost completely separated. (d) Final stage in excystation in which the plates that formed the sporocysts wall have separated and released sporozoites. (From Strohlein, D.A., Prestwood, A.K., 1986. *J. Parasitol.* 72, 711–715. With permission.)

Soon after entering the appropriate host cell, the sporozoite transforms into an ovoid schizont ($\approx 10 \times 8$ µm), which contains a large nucleus with a single nucleolus, a double pellicular membrane, mitochondrion, endoplasmic reticulum, ribosomes, electron-dense bodies, Golgi complex, micropores, autophagic vacuoles, scattered micronemes and rhoptries, and lipid, amylopectin, electron-dense, and multivesicular bodies (Figures 1.72 through 1.75). Autophagic vacuoles contain parasite cytoplasm, ribosomes, and micronemes, indicating that this may be 1 of the sites for recycling the micronemes and other cytoplasmic components. Subsequently, the crystalloid body and

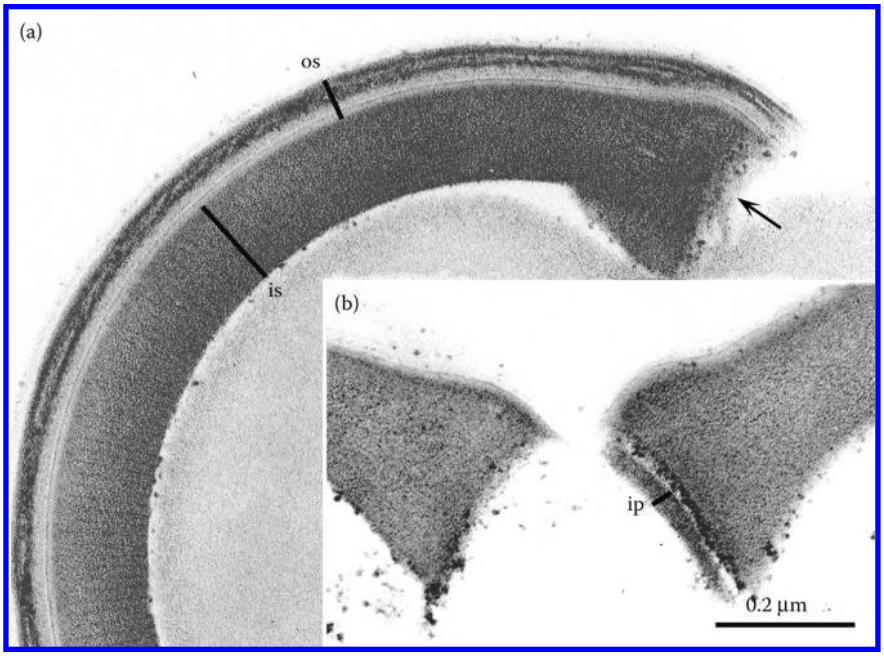


Figure 1.70 TEM of *S. cruzi* sporocyst wall. (a) The outer layer (os) is lamellated with alternating electron-lucent and electron-dense layers. The inner layer (is) is cross-striated and has a thick end at its margin (arrow). (b) Site of apposition between 2 plates of the sporocyst wall after exposure to excysting fluid. Note that the plates have separated and the interposed strip remains attached to the fish lip-like thickening of 1 plate; also note that exposure of the sporocyst to sodium hypochloride prior to incubation in excysting fluid has removed the outer layer of the sporocyst wall.

most organelles disappear. In more advanced stages of the parasite, the nucleus becomes irregularly shaped with several nucleoli, and the micronemes are completely absent (Figure 1.75).

Intermediate schizonts contain anlagen of developing merozoites and a highly lobulated nucleus (Figure 1.76). A spindle apparatus appears in association with each lobe of the nucleus (Figure 1.77). The spindle consists of several microtubules that extend completely across the nucleus from 1 centrocone to the other, and of shorter diverging microtubules that cover an arc of 35°. Each of the 2 centrioles is located immediately above and slightly lateral to the apex of each centrocone. In some species of *Sarcocystis*, 1 or 2 multivesicular bodies may be situated laterally along each centrocone, just outside the nuclear envelope. Coincidental with the appearance of centrocones, the conoid, apical rings, a single-unit membrane, and subpellicular microtubules of a merozoite anlagen form immediately above the centrioles (Figure 1.77). As each merozoite anlagen elongates, the single-unit membrane, and its associated subpellicular microtubules, extend posteriorly, incorporating a part of the nucleus, which eventually pinches off from the rest of the nucleus. Cytoplasm, ribosomes, endoplasmic reticulum, and mitochondrion, are also incorporated into each merozoite anlagen. Micronemes also appear at this stage in association with the Golgi complex or a multivesicular body, between the apical end and the nucleus (Figures 1.78 through 1.80), and continue to be formed until the merozoite is complete. The double-membrane pellicle folds in around the developing merozoites until they appear to bud at the surface of the schizont (Figures 1.78 through 1.80). The double-membrane pellicle becomes closely associated with the single-unit membrane of each merozoite anlage to form the merozoite pellicle. Usually, 1 of the 3 membranes disappears so that the fully formed merozoite has a double membrane pellicle.

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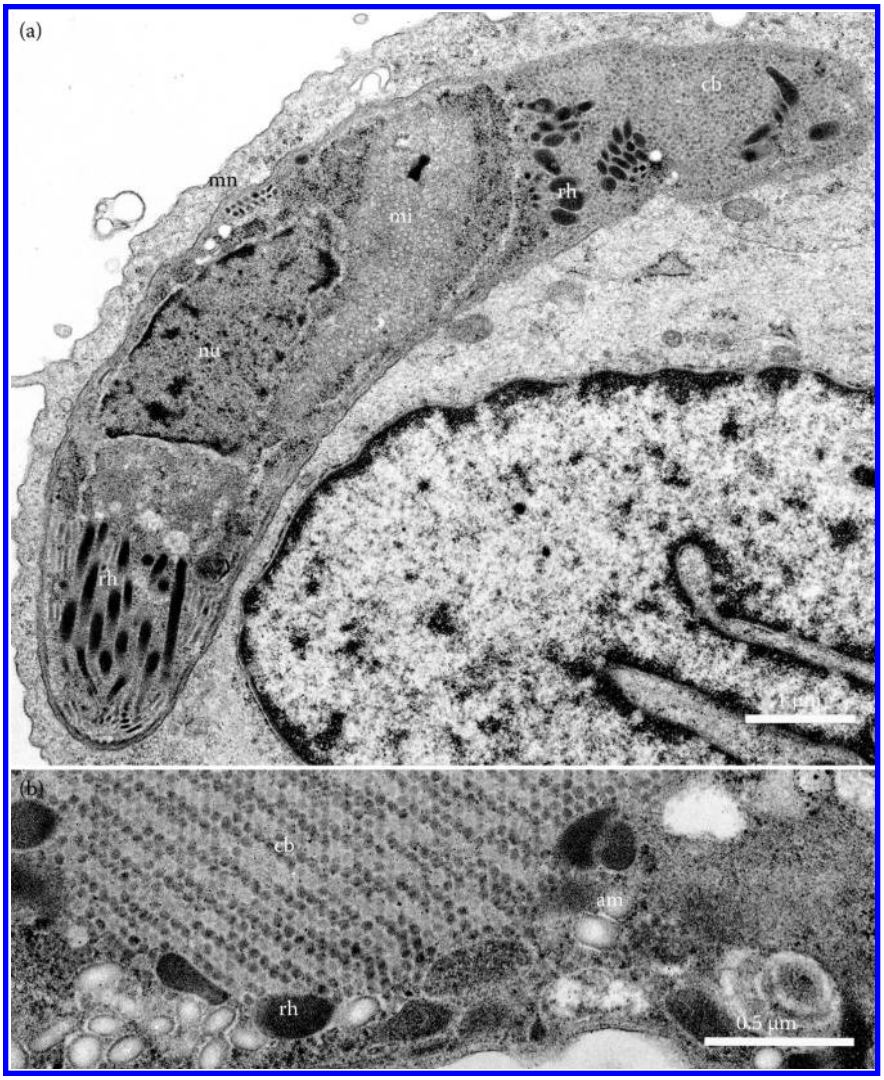


Figure 1.71 TEM of *S. cruzi* sporozoites. (a) Intracellular sporozoite free in the cytoplasm of a cultured cardio pulmonary arterial endotelial (CAPE) cell. Note the absence of a parasitophorous vacuole, and the presence of a large mitochondrion (mi), crystalloid body (cb), and numerous rhoptries (rh). (b) High magnification of a portion of a sporozoite showing the crystalloid body, amylopectin granules (am) and rhoptries.

Merozoites of all *Sarcocystis* species contain virtually all the organelles observed in bradyzoites, except rhoptries (Figure 1.82). The merozoite is twisted slightly to form a helical pattern on its surface (Figure 1.83a). This helical pattern is probably due to a series of rectangular, contiguous strips (plaques) of the inner membrane, aligned in longitudinal rows, as has been described for other coccidia. At the anterior tip, concentric ridges are visible by SEM, which are formed by the apical rings and polar ring 1, which are located just beneath the merozoite plasmalemma (Figure 1.83b and c).

In those species of *Sarcocystis* that undergo schizogony in blood vessels, the host cell appears to be an endothelial cell. In renal glomeruli, the host endothelial cell is completely surrounded by



Figure 1.72 TEM of a *S. falcatula* merozoite transforming into an early schizont in a pulmonary capillary cell of an experimentally infected budgerigar. Note a central, spheroidal nucleus with a large nucleolus, conoid (co) pushed against the host-cell nucleus, a few micronemes (mn), a mitochondrion (mt), and apicoplast (arrow). (Adapted from Speer, C.A. Dubey, J.P., 1999. *J. Parasitol.* 85, 630–637.)

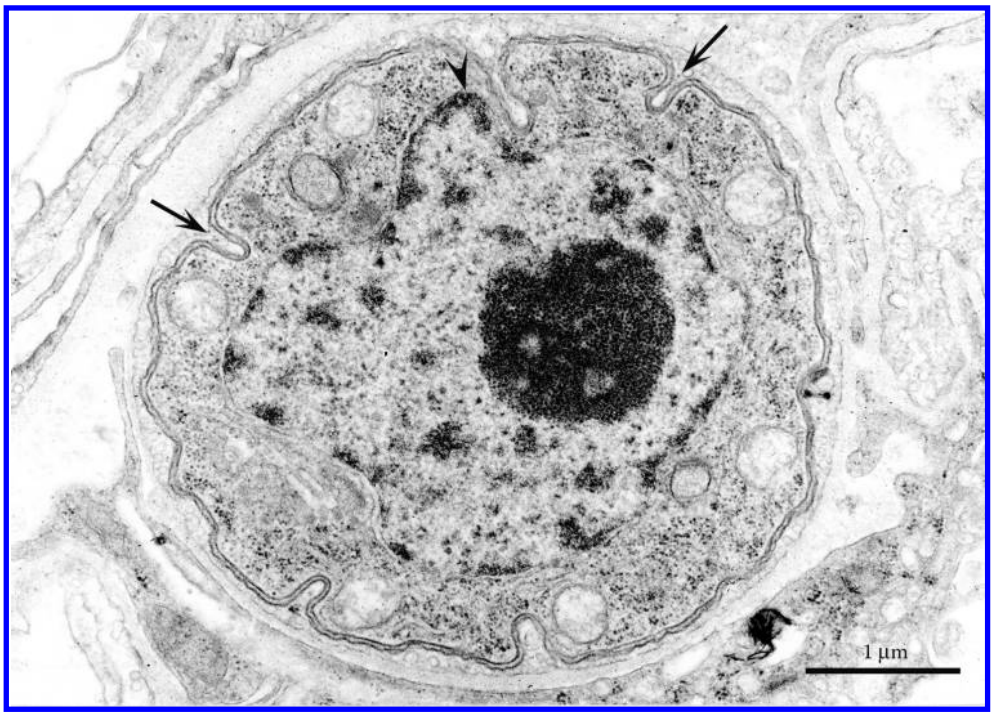


Figure 1.73 Cross section of early schizont of *S. falcatula* in a pulmonary capillary cell of the experimentally infected budgerigar in Figure 1.72. Note the infolded pellicle (arrows) of the schizont. The nucleus has begun to lobulate (arrowhead). (Adapted from Speer, C.A., Dubey, J.P., 1999. *J. Parasitol.* 85, 630–637.)

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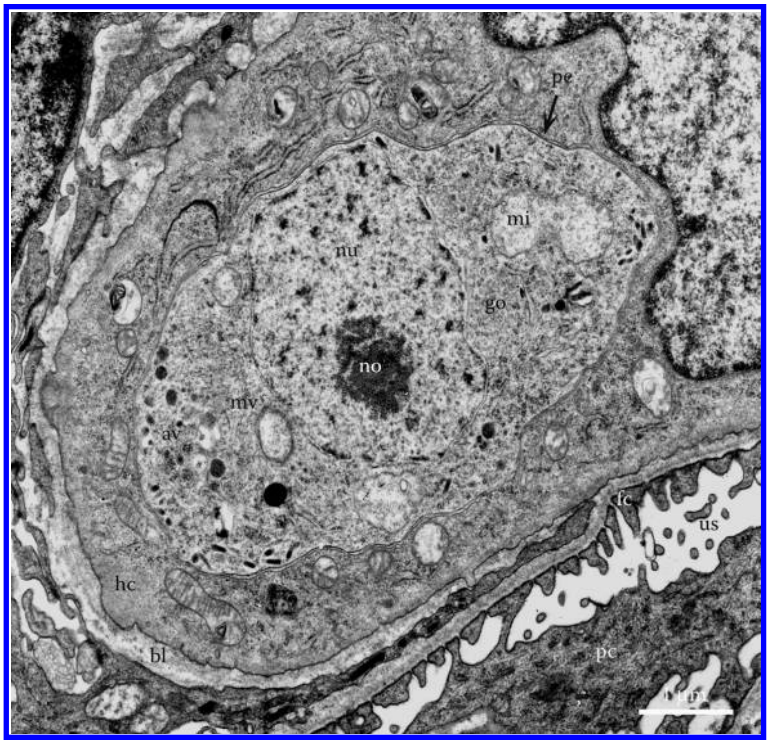


Figure 1.74 TEM of a uninucleate *S. tenella* schizont lying free in the cytoplasm of a glomerular capillary endothelial cell in the kidney of an experimentally infected sheep. (From Speer, C.A., Dubey, J.P., 1981. *J. Protozool.* 28, 424–431. With permission.)

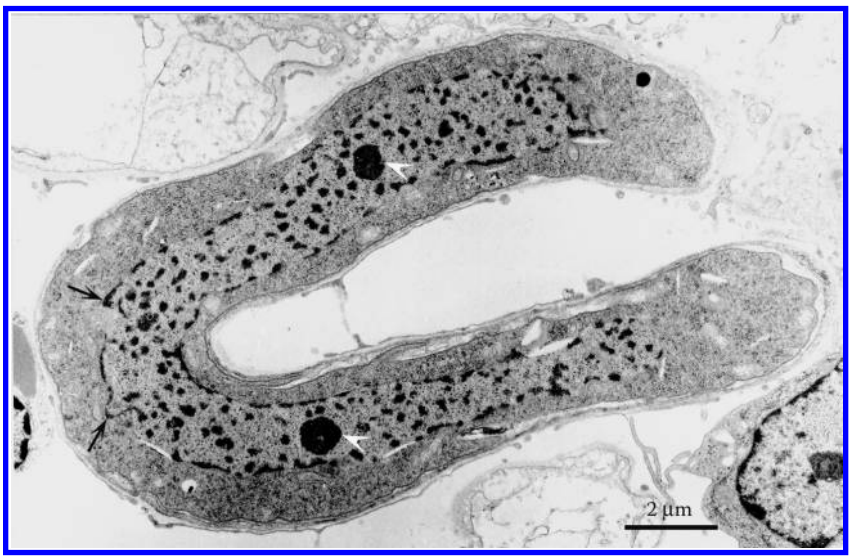


Figure 1.75 TEM of an elongate *S. falcatula* schizont conforming to the shape of the pulmonary capillary in a pulmonary capillary cell of the experimentally infected budgerigar in Figure 1.73. Note the elongated nucleus with 2 nucleoli (arrowheads) and nuclear spindles (arrows). (Adapted from Speer, C.A., Dubey, J.P., 1999. *J. Parasitol.* 85, 630–637.)

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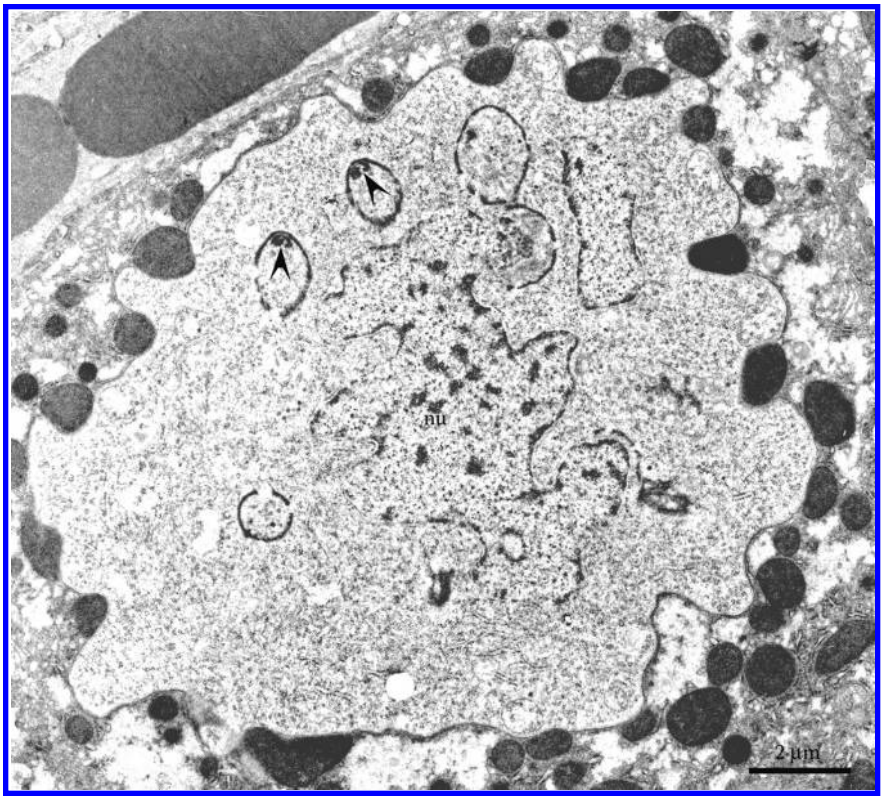


Figure 1.76 TEM of *S. rauschorum* schizont with irregular nucleus and developing apical complexes (arrowheads) in merozoite anlagen.

a basal lamina, on one side of which is a typical capillary endothelial cell, and on the other side are foot processes of podocytes and the urinary space (Figure 1.81). Merozoites may invade cells, normally not parasitized by schizonts. For example, *S. singaporensis* merozoites were shown inside red blood cells of rats.[1368]

Merozoites population differs structurally and biologically. Jäkel et al.[888] reported 2 types of merozoites in schizonts that developed 11–13 DPI of rats with *S. singaporensis* sporocysts. Type 1 merozoites were PAS-negative and these produced schizonts in capillary endothelial cell cultures. Type 2 merozoites were PAS-positive and these induced unizoite sarcocyst-like structures when inoculated in myoblast cultures.[888]

1.4 TAXONOMIC CRITERIA

Before the discovery of the life cycle of *Sarcocystis* in 1972, the 2 major criteria for naming a new species were the structure of sarcocysts and the species of the host. Because the age of sarcocysts and the method of fixation greatly influenced the sarcocyst structure, some species are not clearly described. Life cycle studies have indicated that some structurally similar sarcocysts (e.g., *S. tenella* of sheep and *S. capracanis* of goats) are actually different species, based on host specificity for the intermediate host. Furthermore, some species of *Sarcocystis* (e.g., *S. falcatula* and *S. neurona*) can infect numerous species of hosts belonging to different orders (discussed later). Because it is often very difficult to complete the life cycle of the *Sarcocystis* species of large animals

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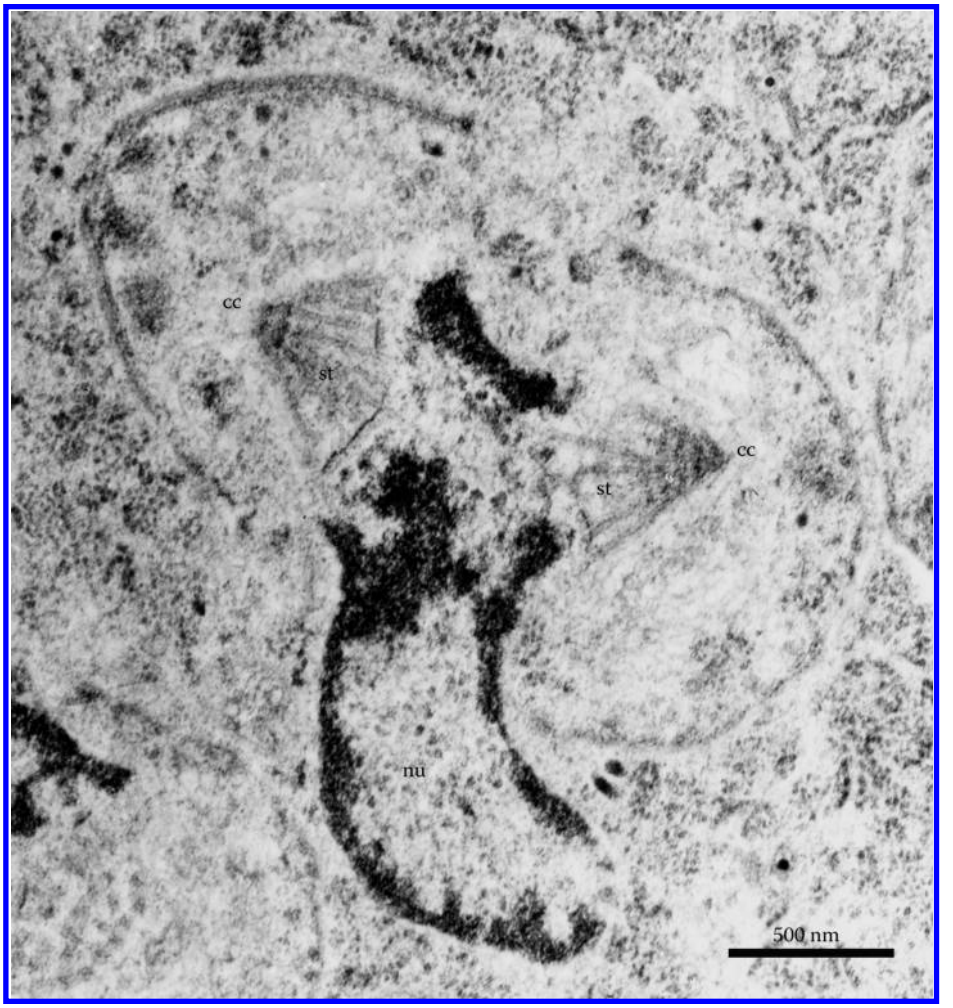


Figure 1.77 TEM of spindle apparatus and developing apical complexes (cc), and spindle microtubules (st) of 2 *S. tenella* merozoites. (From Speer, C.A., Dubey, J.P., 1981. *J. Protozool.* 28, 424–431. With permission.)

under laboratory conditions, the relative value of different criteria that might be used to validate the *Sarcocystis* species will be discussed. Consequently, multiple factors contribute to the naming of species, relying on what information is actually available, including known host species from natural and/or experimental infections, sarcocyst morphology, and, most recently, on DNA data. As new data become available, the taxonomic status of some species has changed and the status of others is expected to change.

1.4.1 Sarcocysts

The shape and size of the sarcocyst vary with the age of the sarcocyst, the type of host cell parasitized, and the techniques used for study. For example, sarcocysts of the same species in cardiac muscles and in the CNS are always smaller than those in skeletal muscles.[360,367,382] The size and shape of sarcocysts will also vary, depending on fixation (they are smaller in fixed specimens than

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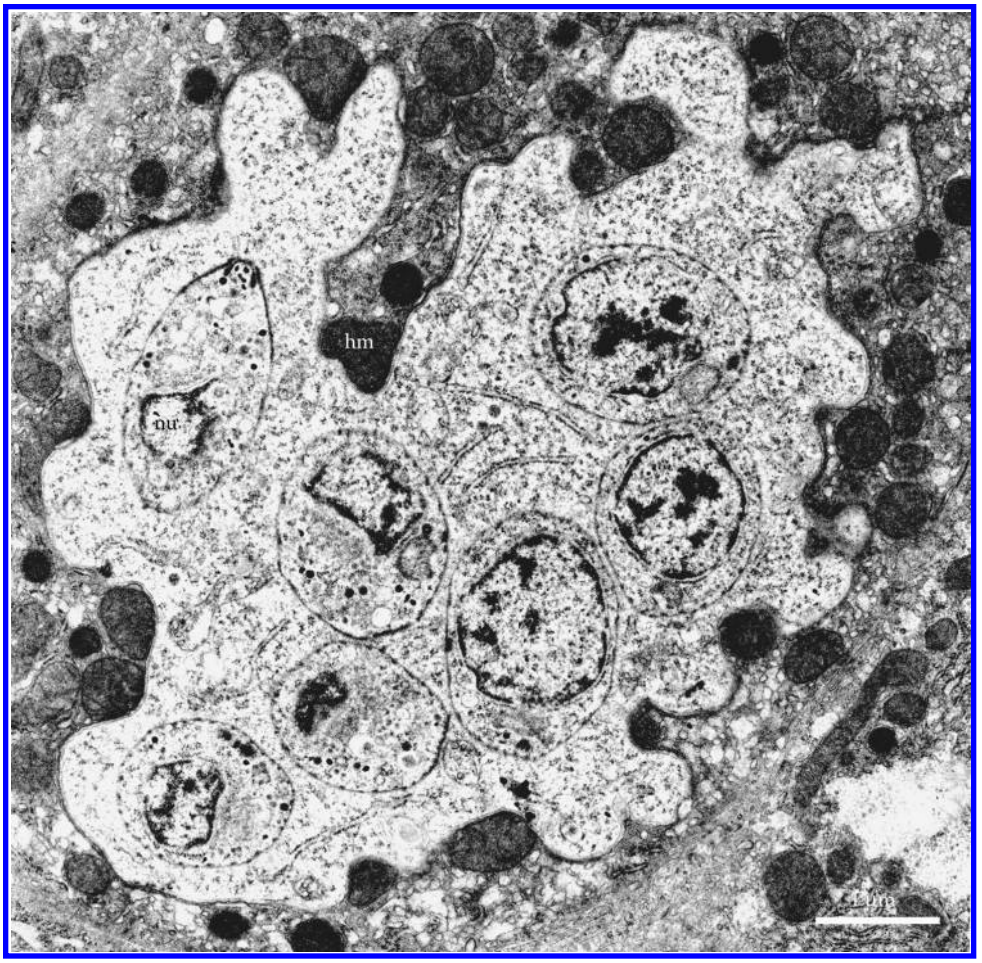


Figure 1.78 TEM of advanced *S. rauschorum* intermediate stage schizont in which each developing merozoite contains a nucleus but the merozoites are still located within the original parasite membrane. Note numerous host-cell mitochondria (hm) surrounding the schizont.

in live specimens), and possibly might vary with the type of fixative. Because they are often located in contractile muscles, the sarcocyst size will vary, depending on whether the host cell was relaxed or contracted at the time of fixation.

Some *Sarcocystis* species continue to grow in size for several years after they have reached infectivity. For example, *S. bertrami* of the horse and *S. medusiformis* of sheep do not attain their maximum length (15 mm) in their hosts until they are approximately 4 years old, whereas *S. cruzi* attains a maximum size of 0.5 mm at approximately 4 months PI. The shape of sarcocysts also varies with relation to the location. For example, *S. gigantea* sarcocysts in the esophagus are globular to pear-shaped, whereas sarcocysts of the same species in the diaphragm are elongate and slender (see Chapter 8). Sarcocysts may continue to grow in size, but the wall structure stabilizes once bradyzoites have formed.[1597]

Metrocyte morphology is not a useful criterion for speciation because metrocytes are often irregularly shaped and their size is highly variable, depending on the stage of division. The structure of bradyzoites also varies. Bradyzoites of some *Sarcocystis* species are densely packed within

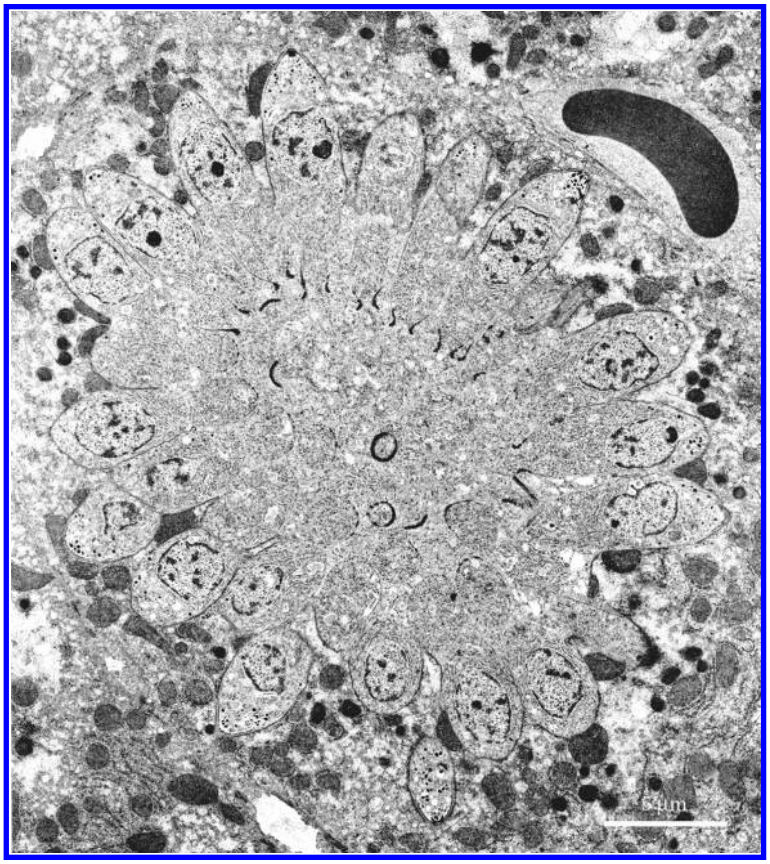


Figure 1.79 TEM of nearly mature *S. rauschorum* schizont in which the merozoites are still attached but are protruding at the margin of the meront.

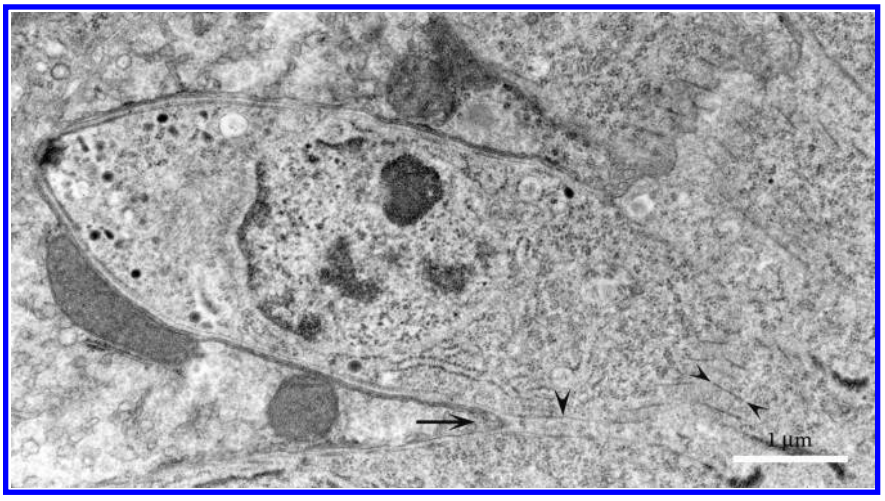


Figure 1.80 Higher magnification of a portion of Figure 1.79 showing a budding *S. rauschorum* merozoite; note that the merozoite plasmalemma (arrow) has invaginated just posterior to the merozoite nucleus. Single arrowhead points to the inner membrane complex of the merozoite and opposing arrowheads point to subpellicular microtubules.



Figure 1.81 TEM of a mature *S. tenella* schizont in a glomerular capillary endothelial cell. Note the endotelial cell (en), basal lamina (bl), urinary space (us), podocyte nucleus (pn), mesenchymal area in glomerulus (ms), and lumen (lu) of the blood vessel. (From Speer, C.A., Dubey, J.P., 1981. *J. Protozool.* 28, 424–431. With permission.)

sarcocysts, whereas those of other species are sparsely found. Such conditions may affect size or shape. Because bradyzoites in most *Sarcocystis* species are banana-shaped, with great variation in curvature, it is difficult to measure them accurately. Measurements obtained from live preparations are unreliable, because the size varies considerably depending on the pH and osmolarity of the medium used, the weight of the cover glass, the volume of fluid under the coverslip, movement during measurement, measured when fixed or live, and the effect of birefringence. The banana-shaped bradyzoites of some *S. cruzi* become globular in acid–pepsin solution used to digest away surrounding host tissue. Therefore, measurements of zoites fixed *in situ* within sarcocysts are more reliable than measurements of zoites liberated by digestion. The width of the bradyzoites at the level of the nucleus may be more reliable than the length of bradyzoites, because in histologic sections it is difficult to measure bradyzoites cut longitudinally along their entire length. Overall, unless the

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Figure 1.82 TEM of *S. tenella* merozoite. Note the absence of rhoptries. (From Speer, C.A., Dubey, J.P., 1981. *J. Protozool.* 28, 424–431. With permission.)

size differences of bradyzoites are obvious, this criterion should not be used for identification. The structure of organelles, such as mitochondrion, rhoptries, and micronemes, is not a good taxonomic criterion.

The structure of the sarcocyst wall can sometimes be a useful criterion for speciation. For example, all 4 species of ovine *Sarcocystis* (*S. gigantea*, *S. tenella*, *S. medusiformis*, and *S. arieticanis*) have sarcocyst walls characteristically different from one another, even though the sarcocyst wall of *S. tenella* is similar to that of *S. capracanis* of the domestic goat. The sarcocyst walls of various species of *Sarcocystis* are illustrated in Figures 1.42 through 1.51.

1.4.2 Schizonts

The structure of the schizont is of limited taxonomic value because it varies greatly, depending on the developmental cycle (e.g., first versus second generation) and the host-cell parasitized. For example, second-generation schizonts of *S. cruzi* and *S. tenella* are slender and elongated in skeletal muscles but are globular in renal glomeruli.[360,382]

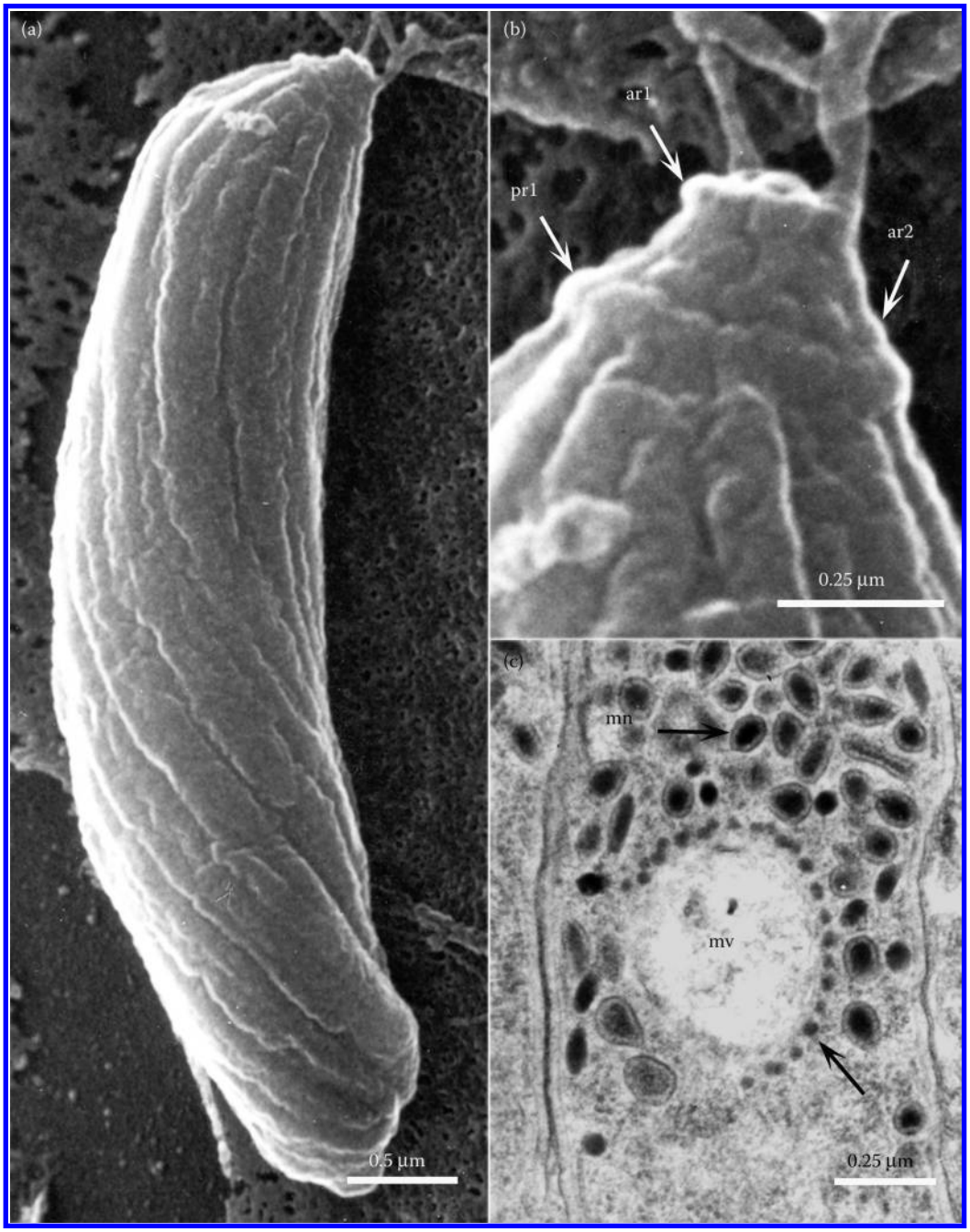


Figure 1.83 SEMs and TEM of merozoites. (a) A helical pattern is seen on the surface of a *S. cruzi* merozoite obtained at 57 days after sporozoites were inoculated into cultured CPAE cells. (b) High magnification of the anterior tip of the merozoite is seen in Figure 1.83a. Note the ridges created by underlying apical rings 1 and 2, and polar ring 1. (c) Anterior region of *S. tenella* merozoite shows multivesicular body (mv) which appears to give rise to micronemes (mn) at its margin (arrow). (From Speer, C.A., Dubey, J.P., 1981. *J. Protozool.* 28, 424–431. With permission.)

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1.4.3 Oocysts and Sporocysts

The structure of the oocyst and sporocysts, traditionally used to determine species of other coccidia, is of little or no taxonomic value in *Sarcocystis* because

a. Except for minor variations in size, all *Sarcocystis* sporocysts and oocysts are structurally similar.
b. Sporocysts of numerous species of *Sarcocystis* in a given host overlap in dimensions. For example, dogs are definitive hosts for 22 species and cats are host for 12 species, and sporocysts of most canine species are about 15 × 10 µm and those of feline species are about 12 × 10 µm (see Chapters 14 and 15).

The prepatent and patent periods vary tremendously and are of no systematic value in *Sarcocystis*. For example, the prepatent period for *S. cruzi* is 7–33 days, and sporocysts can be excreted for many months.

1.4.4 Host Specificity

The species of *Sarcocystis* are generally more host-specific for their intermediate hosts than for their definitive hosts (Table 1.3 through Table 1.5). For example, for *S. cruzi*, the ox and bison are the only intermediate hosts, whereas the dog, wolf, coyote, raccoon, jackal, and fox, can act as definitive hosts. Coyotes and foxes also serve as efficient definitive hosts for other dog-transmitted species (e.g., *S. tenella* and *S. capracanis*). None of the species transmissible via dogs are proven to be transmitted by cats and vice versa (Table 1.4). However, *S. muris* transmitted via domestic cats can be transmitted experimentally by ferrets,[1484] and a species of *Sarcocystis* in chickens is reported to be transmitted by dogs and cats, although details require confirmation (see Chapters 14 through 16).

Table 1.3 Attempted Transmission of *Sarcocystis* Species of Large Animals to Other Animals

Sarcocystis Species	Intermediate Host	Attempted Transmission to	Results[a]	References
S. arieticanis	Sheep	Goat	Negative	**816**
S. tenella	Sheep	Goat	Negative	**354**
S. tenella	Sheep	Ox	Negative	**1468**
S. capracanis	Goat	Sheep	Negative	**1341,1392**
S. hircicanis	Goat	Sheep	Negative	**1768**
S. cruzi	Ox	Sheep, monkey, rabbit, pig, rat	Negative	**578**
S. cruzi	Ox	Bison	Positive	**361**
S. cruzi	Bison	Ox	Positive	**586**
S. cruzi	Ox	Buffalo	Negative	**882**
S. bertrami	Donkey	Horse	Positive	**1131**
S. ferovis	Bighorn sheep	Domestic sheep	Negative	**368**
S. wapiti	Wapiti	Ox	Negative	**359**
S. sybillensis	Wapiti	Ox	Negative	**359**
S. gracilis	Roe deer	Sheep, mice	Negative	**538**
S. odocoileocanis	White-tailed deer	Goat	Negative	**1064**
S. odocoileocanis	White-tailed deer	Sheep, ox	Positive	**266**

[a] Sarcocysts in the muscles of recipient animals.

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Table 1.4 Excretion of Sporocysts by Carnivores Fed *Sarcocystis*-Infected Meat

Intermediate Host	*Sarcocystis* Species	Sporocysts Excreted[a]	Sporocysts Not Excreted[a]	Prepatent Period (days)	Year	Reference
Cattle (*Bos taurus*)	*S. cruzi*	11 dogs, 2 foxes, 2 raccoons	16 cats, 3 Macaca mulatta, 4 skunks, 2 ferrets (*Mustela putorius*), 6 rats, 6 guinea pigs, 6 rabbits	8–15	1976	**578**
	S. hominis	4 Rhesus monkeys, 4 baboons, 1 human	Cats, dogs	12	1976	**805**
	S. hirsuta	Cats	Rhesus monkey, baboon, human, dog	9		
	S. cruzi	Dogs	Cats	11–12	1985	**1223**
	S. hirsuta	3 cats	1 human	12	1990	**397**
	S. cruzi	8 dogs	8 cats	10–12	1992	**654**
	S. cruzi	4 dogs	2 cats	12–16	1994	**1372**
	S. hominis	2 *Macaca fascicularis*	2 dogs, 2 cats	10	1998	**1521**
Horse (*Equus caballus*)	*S. equicanis*	3 dogs	6 cats	8	1975	**1481**
	S. fayeri	10 dogs	10 cats	10–15	1977	**351**
	S. bertrami	1 dog	1 cat, 1 raccoon	9–10	1983	**1131**
	S. bertrami	1 dog	2 humans, 3 cats	13	1982	**833**
Donkey (*Equus africanus asinus*)	*S. bertrami*	1 dog	1 cat, 1 raccoon	9–10	1983	**1131**
Water buffalo (*Bubalus bubalis*)	*S. fusiformis*	4 cats	1 dog, 2 monkeys	8–14	1978	**327**
	S. fusiformis	4 cats	4 dogs	14–16	1995	**861**
	S. buffalonis	7 cats	4 dogs	10	1997	**863**
	S. levinei	2 dogs	2 cats	16–18	1997	**862**
Camel (*Camelus dromedarius*)	*S. cameli*	3 dogs	3 cats	10–14	1980	**822**
	S. cameli	3 dogs	5 cats	9–10	1995	**826**
	S. sp.	2 dogs	4 cats	9–10	1996	**568**
	S. camelicanis	13 dogs	13 cats	11	2009	**11**
Llama (*Llama* spp.)	*S. aucheniae*	1 dog	1 cat	11	1984	**1561**
	S. sp.	4 dogs	4 cats	9–16	1984	**722**
Chickens (*Gallus domesticus*)	*S. horvathi*	NS	Dog, cat, polecat, martin, goshawk	6–9	1982	**1822**
	S. wenzeli	Dog, cat	NS	15		
	S. sp.	3 dogs, 3 cats	NS	10–11	1974	**714**
	S. wenzeli	4 dogs, 4 cats	NS	8–13	1994	**1109**
Pig (*Sus scrofa*)	*S. suihominis*	2 humans	Dogs, cats	9–10	1976	**806**
	S. miescheriana	6 dogs, 2 raccoons	4 cats, 2 opossums	12–14	1980	**1424**
	S. sp.	4 dogs	2 cats	12	1984	**762**
	S. miescheriana (wild pig)	2 dogs, 2 red foxes, 1 wolf, 2 arctic foxes	2 tigers (*Panthera tigris*), 2 wild cats (*Felis chaus*), 2 bears (*Ursus arctos*)	10–12	1976	**546**
	S. miescheriana (wild pig)	Dogs	Cats	13–18	1981	**73**

(*Continued*)

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Table 1.4 (*Continued*) Excretion of Sporocysts by Carnivores Fed *Sarcocystis*-Infected Meat

Intermediate Host	*Sarcocystis* Species	Sporocysts Excreted[a]	Sporocysts Not Excreted[a]	Prepatent Period (days)	Year	Reference
	S. miescheriana (European wild boar)	Dogs	Cats and rats	9–10	1989	**33**
	S. suihominis	NS	2 dogs, 2 cats	–	1998	**1520**
Sheep (*Ovis aries*)	*S.* sp.	4 dogs	4 cats	12–13	1976	**350**
	S. medusiformis	5 cats	12 dogs	15–30	1979	**240**
	S. tenella	2 dogs	4 cats	12–13	1982	**553**
	S. arieticanis	12 dogs	1 cat	12	1985	**817**
	S. gigantea, *S. medusiformis*	4 cats	4 dogs, 4 ferrets, 2 humans	11–14	1986	**613**
	S. tenella	4 dogs, 4 foxes	1 cat	9–11	1987	**615**
	S. microps	4 dogs	4 cats	13–14	1988	**1807**
	S. arieticanis	2 dogs	2 cats	12	1996	**1517**
	S. mihoensis	6 dogs	2 cats	11	1997	**1519**
	S. tenella	2 dogs	2 cats	12	2008	**1753**
Rabbit (*Oryctolagus cuniculus*)	*S. leporum*	13 cats	4 dogs	11–20	1977	**581**
	S. cuniculi	2 cats	2 dogs, 1 fox, 1 weasel (*Mustela nivalis*), 1 krestel (*Falco tinnunculus*)	10	1977	**1706**
	S. sp.	Cats	Dogs	NS	1979	**238**
	S. cuniculi	3 cats	NS dogs	12–13	1980	**1253**
Goat (*Capra hircus*)	*S.* sp.	Fox, wolf, dog	Cat, lion, ferret, krestel (*Falco tinnunculus*)	NS	1975	**103**
	S. sp.	Dogs	Cats	NS	1979	**238**
	S. capracanis	5 dogs	4 cats	10–11	1983	**68**
	S. capracanis	30 dogs	30 cats	12–15	2011	**1226**
Richardson's ground squirrel (*Spermophilus richardsoni*)	*S. campestris*	2 badgers	1 cat, 1 skunk	9	1983	**172**
Rat (*Rattus norvegicus*)	*S. cymruensis*	Cats	Dogs, ferrets	4	1978	**41**
	S. sp.	Cats	Dogs	NS	1979	**238**
Moose (*Alces alces*)	*S. alceslatrans*	3 dogs	2 cats, 1 coyote	10–14	1983	**245**
Roe deer (*Capreolus capreolus*)	*S.* sp. type 1 and 2	10 dogs, 2 foxes	16 domestic cats, 2 wild cats, several birds	10–14	1978	**548**
Alpine ibex (*Capra ibex*)	*S.* sp.	1 dog, 1 fox, 1 wolf	1 cat, 1 ferret	12–21	1975	**103**
Mouflon (*Ovis musimon*)	*S.* sp.	4 dogs	2 cats	12–13	1991	**1277**
Fallow deer (*Dama dama*)	*S.* sp.	5 dogs	2 cats	10–11	1988	**1404**
Mule deer (*Odocoileus hemionus*)	*S.* sp.	1 dog	1 cat	9	1983	**379**
Red deer (*Cervus elaphus*)	*S.* sp.	2 dogs, 1 fox	2 cats	18	1985	**61**

(*Continued*)

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Table 1.4 (*Continued*) Excretion of Sporocysts by Carnivores Fed *Sarcocystis*-Infected Meat

Intermediate Host	*Sarcocystis* Species	Sporocysts Excreted[a]	Sporocysts Not Excreted[a]	Prepatent Period (days)	Year	Reference
	S. cervicanis	3 dogs	2 cats, 1 man	11–12	1981	**796**
Wapiti (*Cervus elaphus*)	*S. wapiti*	1 coyote, 1 dog	1 cat	10	1982	**1651**
Yak (*Poephagus grunniens*)	*S. poephagi*	Cat	Dog	NS	1990	**1815**
	S. poephagicanis	Dog	Cat	NS		

NS, not stated.
Additional information: There are other studies (see References **266, 267**) in which authors detected at least 2 *Sarcocystis* species in venison (white-tailed deer, *Odocoileus virginianus*) and which resulted in yield of sporocyst by several dogs, cats and 1 red fox. Subsequently, after isolation in dog and not in cat, the description of a new species, *S. odocoileocanis,* was revealed.
[a] In several cases the number of animals fed was not reported or not clearly specified.

Some species of *Sarcocystis* transmitted by opossums have a wide range of intermediate hosts, but opossums are the only known definitive hosts (see Chapters 3 and 20).

The species of *Sarcocystis* vary considerably in their biology; therefore, caution is needed before making generalized statements concerning the genus.

1.4.5 Molecular

Molecular characterization can aid speciation. However, the specificity/accuracy depends on the genes analyzed (see Chapter 23). The 18S rRNA gene used in earlier investigations is now considered of limited taxonomic help. For example, structurally and biologically different *S. neurona* (Chapter 3) and *S. falcatula* (Chapter 17) were found to be almost identical in their 18S rRNA sequences.

1.5 PATHOGENICITY

1.5.1 Intermediate Hosts

Not all species of *Sarcocystis* are pathogenic for intermediate hosts. Generally, species transmissible via canids are more pathogenic than those transmissible via other definitive hosts. For example, sheep have 4 universally recognized species of *Sarcocystis*: *S. tenella*, *S. arieticanis*, *S. medusiformis*, and *S. gigantea*. Of these, *S. tenella* and *S. arieticanis*, transmitted via canids, are pathogenic, whereas *S. gigantea* and *S. medusiformis*, transmitted via cats, are nonpathogenic.

The severity of clinical sarcocystosis is dependent on dose, as shown in Table 1.6. The size or weight of the host does not appear to be relevant to resistance or susceptibility of clinical disease. Mature cattle are as susceptible to a specific dose of sporocysts as calves. However, stress can play an important role in the severity of illness and the susceptibility to infection. Pregnancy, lactation, poor nutrition, weather, or other stresses, can influence the severity of clinical sarcocystosis.

1.5.1.1 Clinicopathological Findings

Cattle usually do not develop acute sarcocystosis under experimental conditions unless 200,000 or more *S. cruzi* sporocysts have been ingested at a given time. Except for fever (≥40°C) between 15

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Table 1.5 Attempted Transmission of *Sarcocystis* Species of Small Mammals to Other Small Mammals

Sarcocystis Species	Intermediate Host	Definitive Host	Transmission Attempted to	Results[a]	References
S. atheridis	Laboratory mouse (*Mus musculus*), Barbary striped grass mouse (*Lemniscomys barbarus*)	Nitsche's bush viper (*Atheris nitschei*)	Norway rat (*Rattus norvegicus*)	Negative	**1620**
S. cernae	Common vole (*Microtus arvalis*)	Krestel (*Falco tinnunculus*)	House mouse (*Mus musculus*)	Negative	**185**
S. clethrionomy-elaphis	Voles (*Microtus* spp., *Clethrionomys glareolus*)	Aesculapian snake (*Zamenis longissimus*)	Tundra vole (*Microtus oeconomus*)	Positive	**1135**
			Günther's vole (*Microtus guentheri*)	Positive	
			Bank vole (*Clethrionomys glareolus*)	Positive	
			Common vole (*Microtus arvalis*)	Positive	
			Wood mouse (*Apodemus sylvaticus*)	Negative	
			Sand lizard (*Lacerta agilis*)	Negative	
			Common wall lizard *(Podarcis muralis)*	Negative	
S. cymruensis	Norway rat (*Rattus norvegicus*), deer mouse (*Peromyscus maniculatus*), greater bandicoot rat (*Bandicota indica*)	Cat (*Felis catus*)	Dog, ferret, house mouse (*Mus musculus*), white-footed mouse (*Peromyscus leucopus*)	Negative	**41**
			Bank vole (*Clethrionomys glareolus*)	Positive	**740**
S. dispersa	House mouse (*Mus musculus*)	Barn owl (*Tyto alba*)	Voles (*Microtus arvalis*)	Negative	**183**
S. gerbilliechis	Subfamily Gerbillinae (*Gerbillus* spp., *Psammonys* spp., *Pachyuromys* spp.)	Arabian saw-scaled viper (*Echis coloratus*)	Cairo spiny mouse (*Acomys cachirinus*)	Negative	**885**
			House mouse (*Mus musculus*)	Negative	
			Norway rat (*Rattus norvegicus*)	Negative	
			Multimammate mouse (*Mastomys natalensis*)	Negative	
S. idahoensis	Deer mouse (*Peromyscus maniculatus*)	Gopher snake (*Pituophis melanoleucus*)	House mouse (*Mus musculus*)	Negative	**113**
			White-footed mouse (*Peromyscus leucopus*)	Negative	
S. muris	House mouse (*Mus musculus*)	Cat (*Felis catus*), ferret (*Mustela putorius*)	Norway rat (*Rattus norvegicus*)	Negative	**1155,1503, 1839**
			Golden hamster (*Mesocricetus auratus*)	Negative	
			Guinea pig (*Cavia porcellus*)	Negative	
			Meadow vole (*Microtus pennsylvanicus*)	Negative	
			Mongolian jird (*Meriones unguiculatus*)	Negative	
S. muriviperae	House mouse (*Mus musculus*)	Palestinian viper (*Vipera palaestinae*)	House mouse (*Mus musculus*)	Positive	**1142**
			Günthers vole (*Microtus guentheri*)	Negative	
			Jirds (*Meriones unguiculatus*,*M. tristrami*)	Negative	
			Multimammate mouse (*Mastomys natalensis*)	Negative	
			European rabbit (*Oryctolagus cuniculus*)	Negative	

(*Continued*)

Table 1.5 (*Continued*) Attempted Transmission of *Sarcocystis* Species of Small Mammals to Other Small Mammals

Sarcocystis Species	Intermediate Host	Definitive Host	Transmission Attempted to	Results[a]	References
S. rauschorum	Varying lemming (*Dicrostonyx richardsoni*)	Snowy owl (*Nyctea scandiaca*)	Norway rat (*Rattus norvegicus*)	Negative	**173**
			House mouse (*Mus musculus*)	Negative	
			Red-neck vole (*Clethrionomys gapperi*)	Negative	
			White-footed mouse (*Peromyscus leucopus*)	Negative	
			Brown lemming (*Lemmus sibiricus*)	Negative	
S. singaporensis	Norway rat (*Rattus norvegicus*)	Python (*Morelia reticulatus*)	Golden hamster (*Mesocricetus auratus*)	Negative	**140,1869**
			Multimammate mouse (*Mastomys natalensis*)	Negative	**140**
			Vole (*Microtus arvalis*)	Negative	
			Mice, amphibians, rodents[b]	Negative	**886**
			Rats[c]	Positive	
			Other snakes[d]	Negative	
			Rhesus monkey (*Maccaca mulatta*)	Negative	**85**
			House mouse (*Mus musculus*)	Negative	
			8 *Rattus* spp. and 3 *Bandicota* spp.[e]	Positive	**767**
			15 species of *Muridae*[f]	Negative	
S. villivillosi	Norway rat (*Rattus norvegicus*)	Python (*Morelia reticulatus*)	Rhesus monkey (*Macaca mulatta*)	Negative	**85**
			8 *Rattus* spp. and 3 *Bandicota* spp.[e]	Positive	**767**
			15 species of *Muridae*[f]	Negative	
S. zamani	Norway rat (*Rattus norvegicus*)	Python (*Morelia reticulatus*)	House mouse (*Mus musculus*)	Negative	**85**
			Rhesus monkey (*Macaca mulatta*)	Negative	
S. zuoi	Norway rat (*Rattus norvegicus*)	King rat snake (*Elaphe carinata*)	Mouse (*Mus musculus*)	Negative	**855**

[a] Sarcocysts in the muscles of recipient animals.
[b] *Bufo regularis, Rana mascareniensis, Chalcides ocellatus, Mabuya quinquetaeniata, Acanthodactylus boskianus asper, Ptychodactylus hasselquisti, Gerbillus gerbillus, Meriones shawi isis, Jaculus jaculus, Acomys cahirinus cahirinus, Mus musculus praetextus.*
[c] *Rattus rattus frugivorous, Rattus norvegicus, Arvicanthis niloticus, Nesokia indica.*
[d] Snakes of Families Elapidae, Viperidae, Colubridae, Boide.
[e] *Sigmodon hispidus, Cricetulus griseus, Mesocricetus auratus, Clethryonomys glareolus, Arvicola terrestris, Microtus arvalis, Gerbillus perpallidus, Meriones unguiculatus, Saccostomus campestris, Apodemus agrarius, Apodemus flavicollis, Mastomys natalensis, Acomys dimidiatus, Mus musculus, Rattus surifer.*
[f] *Rattus novegicus, Rattus rattus, Rattus argentiventer, Rattus tiomanicus, Rattus exulans, Rattus losea, Rattus villosissimus, Rattus colletti, Bandicota indica, Bandicota bengalensis, Bandicota savilei.*

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Table 1.6 Abbreviations for Figures 1.26 through 1.81

am—amylopectin granule	mp—micropore
ap—apical complex	mt—microtubule
ar1,2—apical rings 1 and 2	na—nucleus of macrogamont
br—bradyzoite	ni—nucleus of microgamete
cc—centrocone	no—nucleolus
ce—centriole	nu—nucleus of parasite
co—conoid	oo—outer layer of oocyst wall
eb—electron-dense body	os—outer layer of sporocyst wall
edg—electron-dense granule	ow—oocyst wall
edl—electron-dense layer of primary sarcocyst wall	pe—pellicle
gs—ground substance	pi—plasmalemma of parasite
go—Golgi complex	pvm—parasitophorous vacuolar membrane
hc—host-cell cytoplasm	pr—polar rings
hm—host-cell mitochondrion	pv—parasitophorus vacuole
hn—host-cell nucleus	pcw—primary sarcocyst wall
im—inner membrane complex	rh—rhoptry
io—inner layer of oocyst wall	se—septa
ip—interposed strip in sporocyst wall	sm—subpellicular microtubule
is—inner layer of sporocyst wall	sp—sporozoite
me—metrocyte	sw—sporocyst wall
mi—mitochondrion	vp—villar protrusion
mn—microneme	

and 19 DPI, clinical signs have not been observed until 24 DPI.[366,572,591] Beginning the fourth week after inoculation, cattle can develop anorexia, diarrhea, weight loss, weakness, muscle twitching, prostration, and, sometimes, death. Pregnant animals can undergo a premature parturition, abortion, or produce a stillborn fetus.[577] Some or all of these clinical signs can last from a few days to several weeks.

Clinical laboratory findings indicate anemia, tissue damage, and clotting dysfunction. Anemia with packed cell volumes (PCV) below 20% has been found in animals with moderate-to-severe infections. The mechanism of anemia is not fully understood.[234–236,584,629,1097,1098,1422] Elevation of clotting factors during acute sarcocystosis can affect anemia.[299]

Serum bilirubin, LDH, AAT, SDH, and CPK are generally elevated for brief periods during the acute phase.[366] BUN becomes elevated approaching terminal sarcocystosis. Prothrombin time is longer, whereas platelet counts, clotting time, activated partial thromboplastin time, and thrombin time, are generally not altered.[629,1097,1422] Acquired Factor VII deficiency and platelet dysfunction are found in some acutely infected cattle.[1422]

As infections become chronic, other signs become apparent. Growth is adversely affected. Animals can become hyperexcitable; they might hypersalivate and they might lose hair, especially on the neck, rump, and tail switch[591] (Figure 1.84). Some become emaciated. Some eventually develop CNS signs, including recumbency, opisthotonous, nystagmus, cycling gait while laterally recumbent, and, occasionally, death. Clinical disease in relation to phases of the life cycle of the parasite is summarized in Figure 1.85.

With few exceptions, clinical signs similar to those seen in *S. cruzi*-infected cattle have been seen in goats, sheep, and pigs infected with large doses of *S. capracanis*, *S. tenella*, and *S. miescheriana*, respectively (see Chapters 6 through 9). In sheep and goats, neural signs were more prominent than in cattle, but hypersalivation was not seen in sheep or goats.

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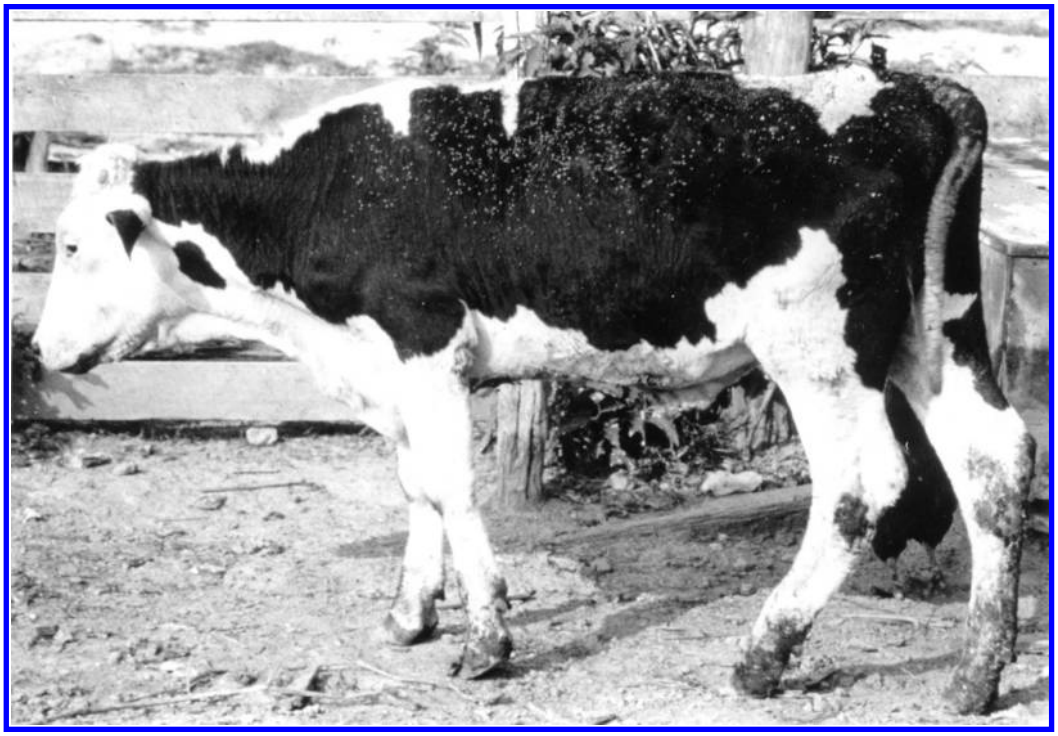


Figure 1.84 An emaciated steer naturally infected with *S. cruzi*. Note the loss of hair at the tip of the tail. (Adapted from Giles, R.C. et al., 1980. *J. Am. Vet. Med. Assoc.* 176, 543–548.)

1.5.1.2 Gross Lesions

Edema and focal necrosis in gut-associated lymph nodes are seen about 15 DPI.[366] The next gross lesions are seen at about 26 DPI. The most striking lesion seen at this time is hemorrhage[366,911] (Figure 1.5f). Hemorrhages are most evident on the serosal surface of viscera, in cardiac and skeletal muscles, and in the sclera of the eyes. On necropsy, characteristics of acute sarcocystosis include skeletal muscles that appear mottled or striped with pale areas interspersed with areas of dark hemorrhage. Hemorrhages vary from petechiae to ecchymoses several centimeters in diameter. Following acute infection, body fat becomes scanty, yellow-hued, and gelatinous. Body cavities contain straw-colored fluid, and organs become icteric. In chronically affected animals, the most notable lesion is serous atrophy of fat, especially pericardial and perirenal fat, with white flecks of mineralization.

1.5.1.3 Microscopic Lesions

The earliest lesion observed is hypertrophy of endothelial cells, associated with the development of first-generation schizonts in arteries and arterioles[366,371] (Figure 1.86). This is followed by perivascular and interstitial infiltration of many organs with mononuclear cells (Figure 1.87). The mononuclear cells are seen as early as 11 DPI and can be present for several months. The predominant inflammatory cells are lymphocytes and macrophages, with a few plasma cells and eosinophils. Hemorrhage can be generalized and often not associated with inflammation. Necrosis may be found in many organs, especially in skeletal muscles, heart, and kidneys, probably associated with

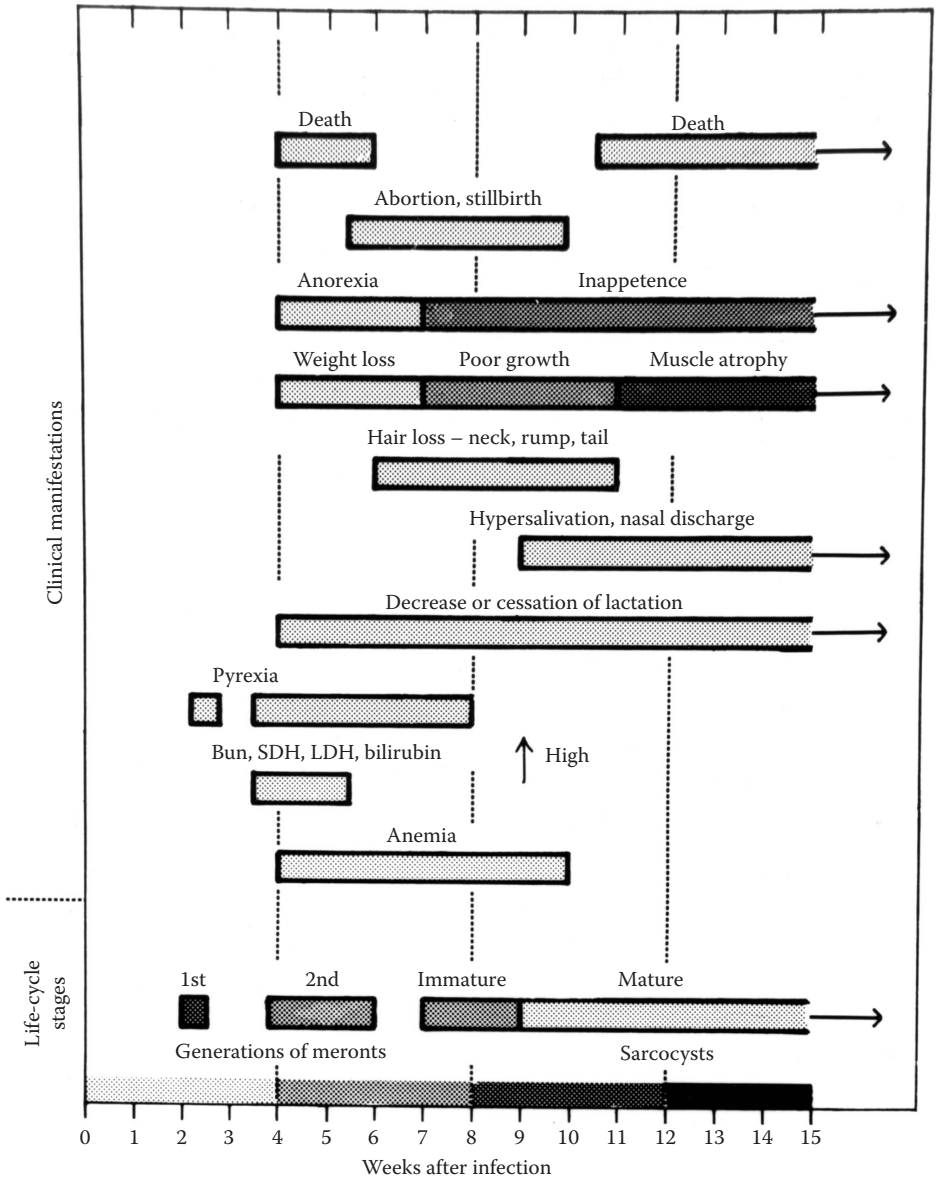


Figure 1.85 The chart compares the life-cycle stages of *S. cruzi* with the weeks after infection and the clinical signs of sarcocystosis in cattle. (Adapted from Fayer, R., Dubey, J.P., 1988. *Transplacental Effects on Fetal Health*. Alan R. Liss, Inc., New York, NY, pp. 153–164.)

vasculitis (Figures 1.87 through 1.92). The overall predominant lesion in sarcocystosis is inflammatory rather than degenerative.

Degenerating sarcocysts might be surrounded by mononuclear cells, neutrophils (Figure 1.93), eosinophils, giant cells, or a combination of these cells. In most livestock species, the cellular response is mainly mononuclear. How sarcocysts are removed from host tissue is not known. It is likely that some sarcocysts rupture spontaneously and inflammation follows. However, macrophages and plasma cells can be seen around seemingly intact live sarcocysts. However, many sarcocysts can be seen with no adjacent inflammatory reaction. There is no reactivation of chronic infection.

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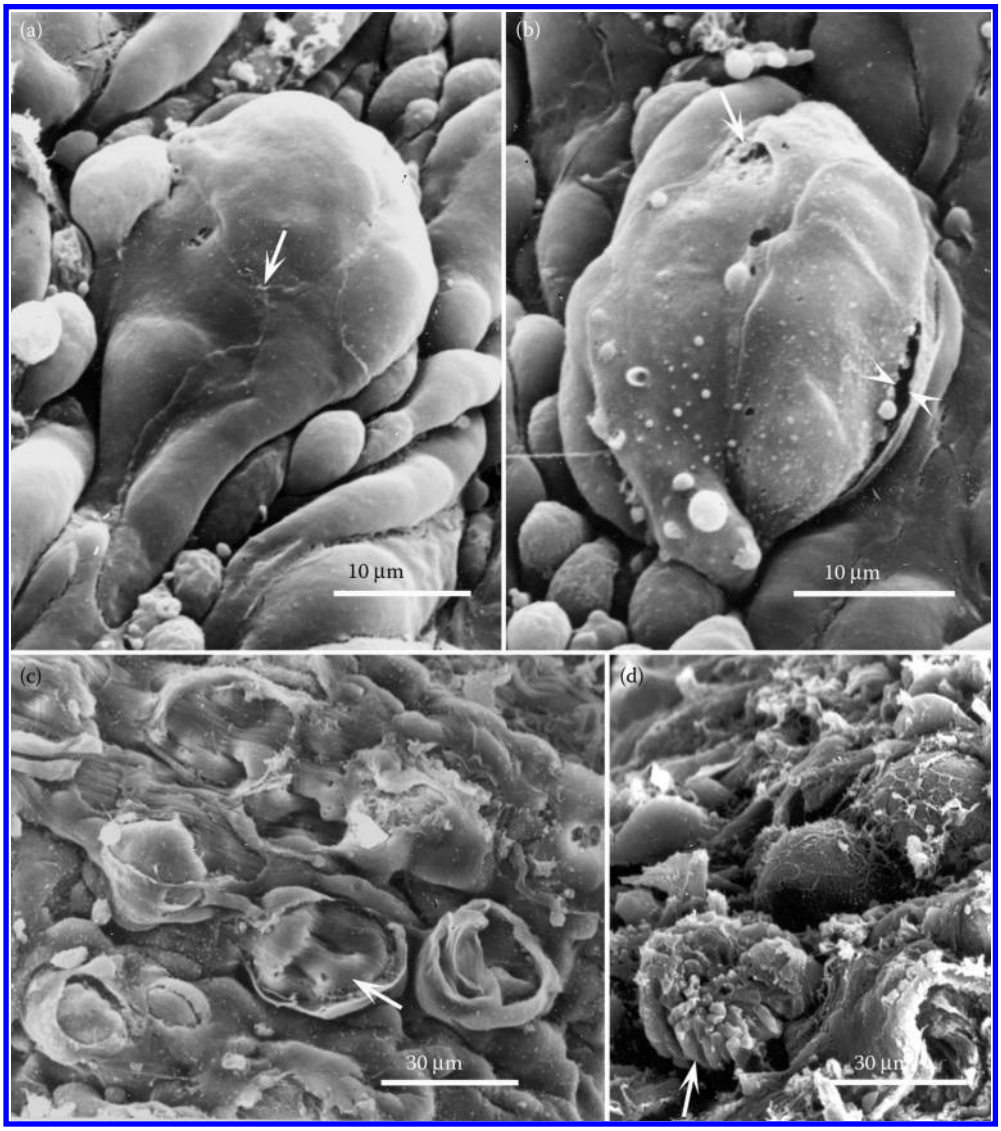


Figure 1.86 SEMs of ovine mesenteric arteries infected with first-generation schizonts of *S. tenella*. (a) The protuberance in the center includes 4 endothelial cells demarcated by intercellular junctions (arrows) covering a host cell containing a schizont (not visible). (b) Perforation (arrow) and separation of 2 endothelial cells (arrowheads) covering a schizont. (c) Sloughing of endothelial cells exposing the internal elastic membrane and host cells harboring schizonts (arrow). (d) Most of the endothelium has sloughed, exposing a schizont and merozoites (arrowheads). (From Speer, C.A., Dubey, J.P., 1982. *Can. J. Zool.* 60, 203–209. With permission.)

1.5.1.4 Definitive Hosts

Sarcocystis generally does not cause illness in definitive hosts. Dogs, cats, coyotes, foxes, and raccoons, fed tissues infected with numerous species of *Sarcocystis* excreted sporocysts, but were otherwise normal. A few dogs and coyotes vomited or were anorexic for 1–2 days following ingestion of meat, but such signs might have resulted from the change in diet from laboratory chow to raw meat. However, human volunteers who ingested beef and pork infected with *S. hominis*

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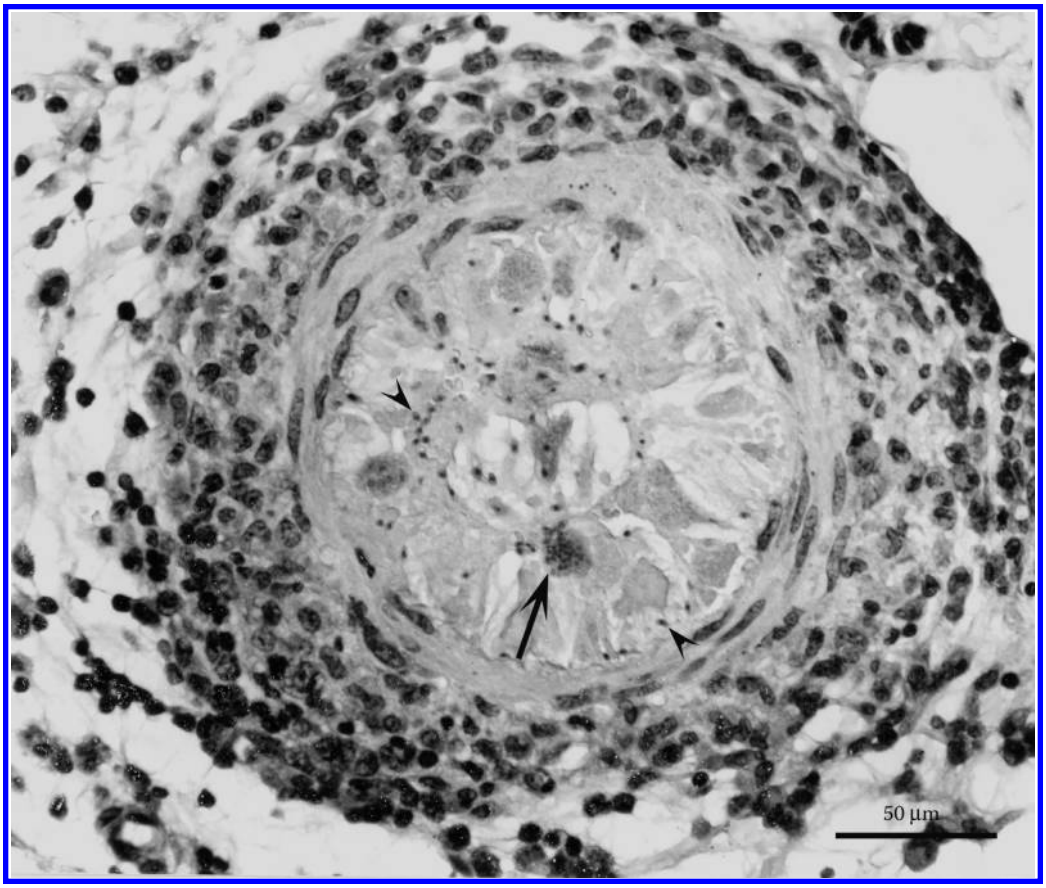


Figure 1.87 Necrosis and vasculitis in a mesenteric lymph node artery of a sheep infected with *S. tenella*. The endothelial cells have been destroyed by rupture of second-generation schizonts (arrow), releasing merozoites (arrowheads) into the lumen. Mononuclear cells are in and around arterial walls. H and E stain. (From Dubey, J.P., 1988. *Vet. Parasitol.* 26, 237–252. With permission.)

or *S. suihominis*, respectively, developed symptoms, including vomiting, diarrhea, and respiratory distress. These symptoms were more pronounced in volunteers who ate infected pork than in those who ate infected beef (see Chapters 4, 6, and 7).

1.6 PATHOGENESIS

1.6.1 Tissue Necrosis

Schizonts cause necrosis of cells and tissues, depending on the species of *Sarcocystis*, location, and the multiplication potential.[366] For example, *S. falcatula* of birds multiplies extensively in endothelial cells and produces several generations of schizogony.[1626] The physical damage alone as a result of vasculitis might result in the death of birds heavily infected with *S. falcatula*. The same hypothesis might apply to *S. idahoensis* infection in deer mice, in which hepatocytes are destroyed as a result of schizont multiplication.[112] However, localized tissue necrosis does not appear extensive enough to cause the severe illness or death seen in large animals (cattle, sheep, goats, and pigs).

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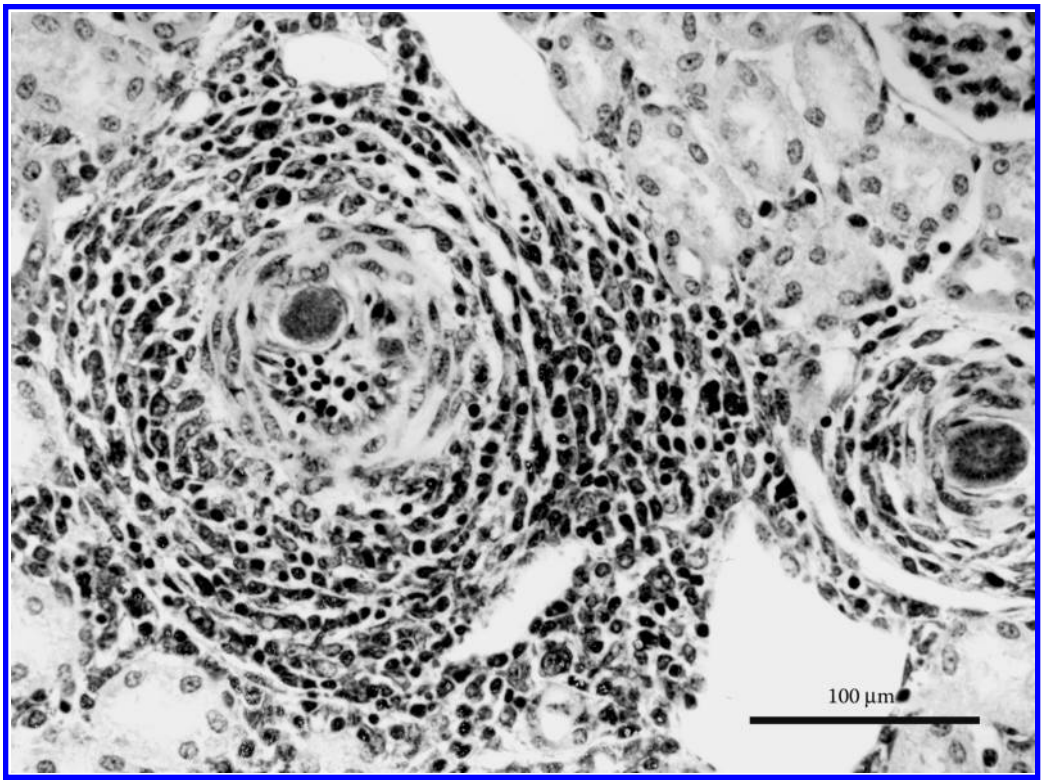


Figure 1.88 Severe mononuclear cell infiltrations in tunica adventia and tunica media in arteries in the renal cortex of a calf infected with *S. cruzi*. The vascular lumen is almost occluded by hypertrophied endothelial cells and first generation schizonts. Giemsa stain. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res.* 43, 2147–2164. With permission.)

1.6.2 Inflammation

The perivascular mononuclear cell infiltration seen around *S. cruzi*-infected arteries at 7–11 DPI[366] indicates a host reaction to antigens liberated from sporozoites or immature schizonts, or the expression of parasite antigens by host cells, because merozoites are not liberated from these first-generation schizonts until 14 DPI or later. An intense inflammatory reaction is usually observed about the time when the second-generation schizonts mature and rupture, during the fourth to sixth week PI. The myositis, during the penetration of myocytes by merozoites, might be related to products liberated from merozoites or myocytes.[366] The intense mononuclear cell infiltrations in the kidneys, liver, lungs, and other organs, are probably stimulated by similar parasite antigens.

1.6.3 Immune Regulation of Pathogenesis

Vascular endothelial cells have MHC II antigens[1634] and can process antigen for presentation to lymphocytes.[1151] Adherence of thymus-derived (T) lymphocytes to endothelial cells via the lymphocyte function-associated molecule, LFA-1, or other mechanisms[785], is the first step toward emigration of these cells from the blood into the tissues. Lymphocytes also recognize specific MHC II antigen on vascular endothelial cells.[1402] In a rabbit model, a strong correlation was found

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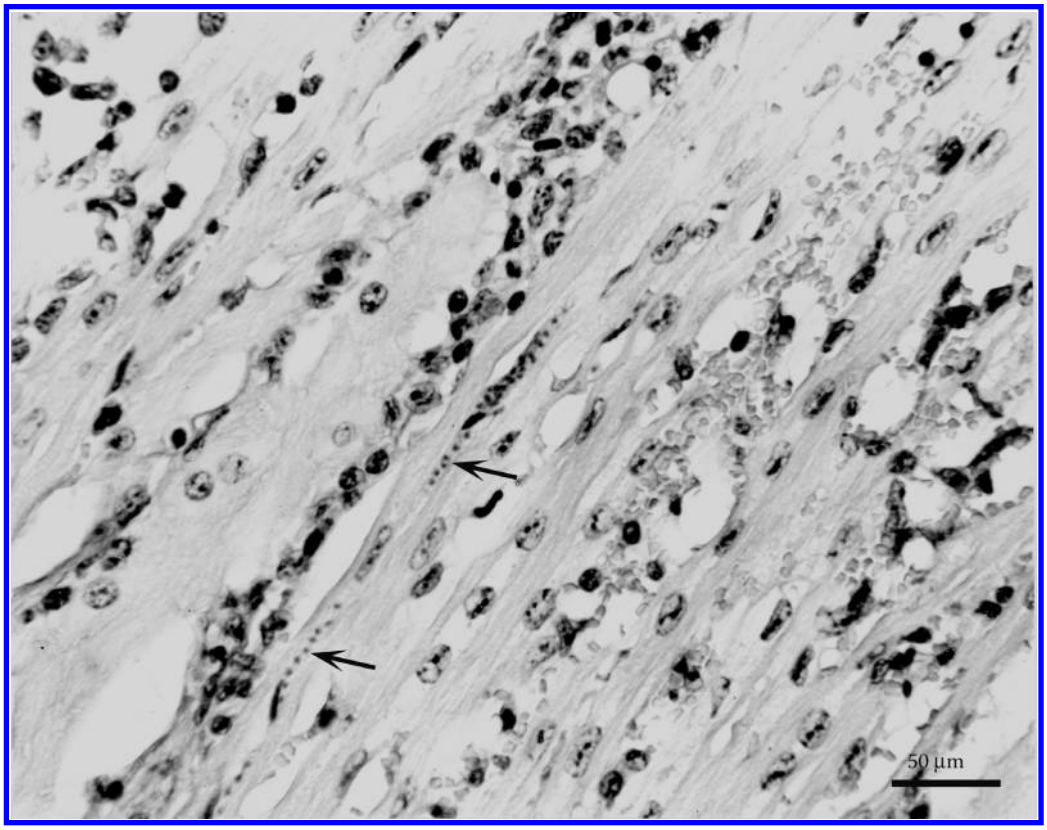


Figure 1.89 Hemorrhage and mild mononuclear cell infiltration in the myocardium of a calf experimentally infected with *S. cruzi.* Arrow points to merozoites and schizonts. H and E stain. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res.* 43, 2147–2164. With permission.)

between the magnitude of the *in vivo* immune response and the vascular endothelial expression of MHC II antigens.[1635]

Thus, during acute *S. cruzi* infections, endothelial cells might participate in eliciting an immune response, leading to pathogenesis by processing and presenting antigens immunogenically, which results in sensitization of T lymphocytes.[1659] These antigen-sensitive T lymphocytes might then recognize antigen plus MHC II[1402] on vascular endothelium and give rise to destructive immune responses, such as local delayed hypersensitivity reactions involving mononuclear cell infiltration of vascular tissue.[366,1634,1635] Cytokines induced by *S. cruzi* infection can adversely affect growth regulatory hormones and interfere with weight gain.[594]

1.6.4 Edema

Ascites and edema in tissues are probably related to hypoproteinemia and vasculitis.[366,1626]

1.6.5 Fever

The peaks of fever coincide with maturation of schizonts and release of merozoites into the bloodstream.[591] Fever is probably related to the release of pyrogen from mature rupturing schizonts directly on the hypothalamus, or indirectly by stimulating the release of prostaglandins.

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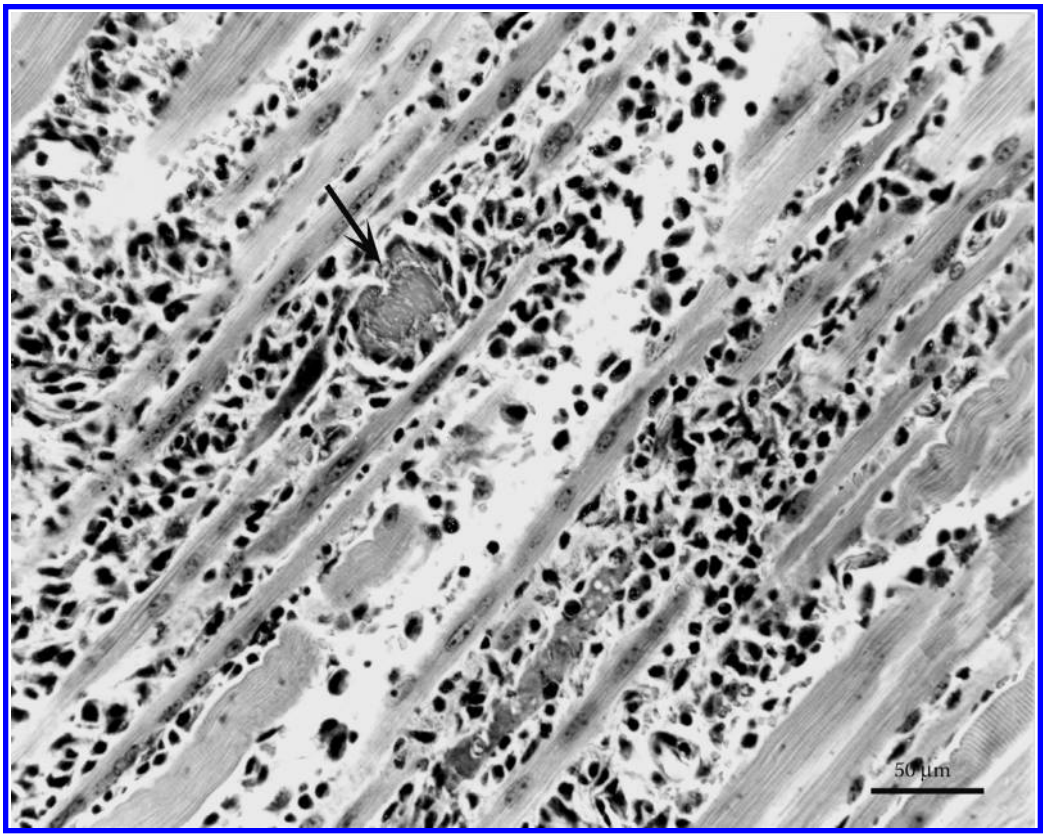


Figure 1.90 Severe necrosis, infiltration of mononuclear cells, and mineralization (arrow) in semitendinosus muscle of a calf infected with *S. cruzi*. H and E stain. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res*. 43, 2147–2164. With permission.)

1.6.6 Anemia

Anemia is the most evident clinical finding of acute sarcocystosis in cattle, sheep, goats, and pigs, but the underlying mechanism is unknown.[359,366,584,1036,1097,1098,1422] Although the anemia is regenerative, few or no reticulocytes are found. The anemia is normocytic, normochromic, and primarily hemolytic. Hemorrhage can account for part of the loss of blood cells. In addition, many red-blood cells are removed from circulation and sequestered in the spleen, probably via immunologic mechanisms. It is possible that some unknown toxic factors, or metabolite released from schizonts, or infected host cells, contribute further to the anemia. The increased prothrombin time and increased fibrin degradation product concentration in infected calves indicate intravascular coagulopathy, which can result in capillary extravasation.[304]

1.6.7 Abortion

Abortion and fetal death can result when animals become infected with pathogenic species of *Sarcocystis* during pregnancy. Most cattle, sheep, swine, goats, and roe deer that developed clinical sarcocystosis from experimental infections induced in mid to late gestation aborted,[70,357,548,549,577,1037,1672] whereas most infected animals without signs of infection carried their fetus to term.[357,1254] Lesions were not found in fetal tissues from any of these experimentally

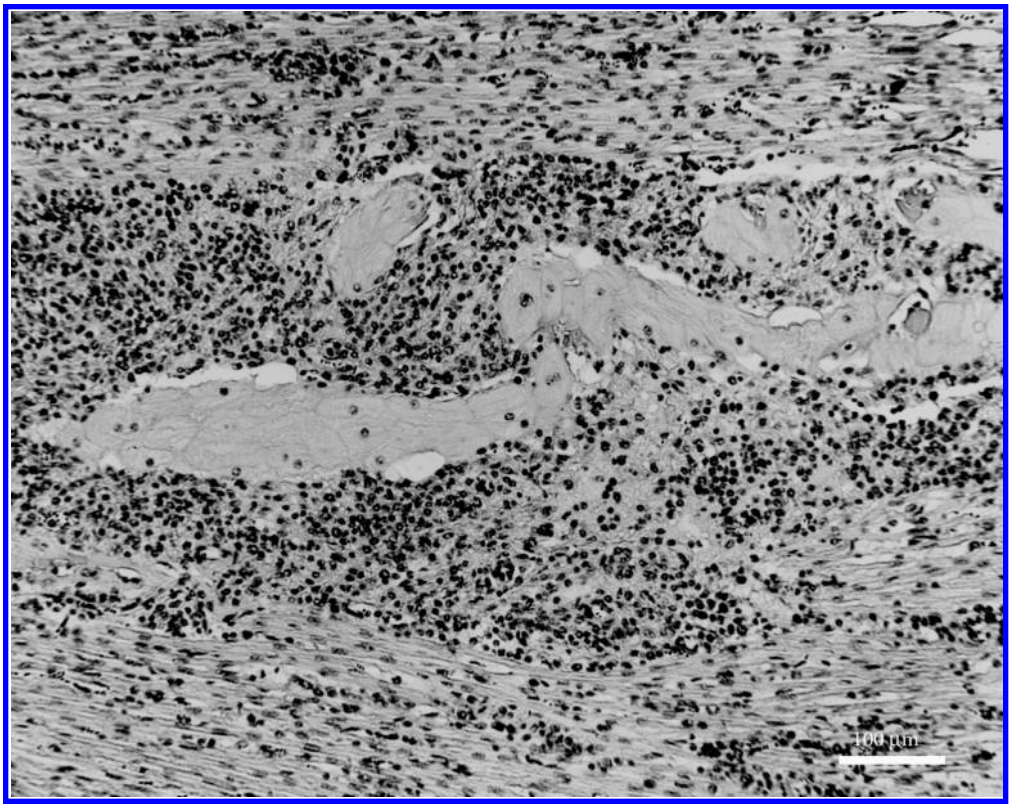


Figure 1.91 Severe myocarditis involving Purkinje fibers and myocardium of a calf experimentally infected with *S. cruzi*. H and E stain. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res.* 43, 2147–2164. With permission.)

infected host species, with the exception of 1 lamb. Parasites were found in only 2 of 18 bovine fetuses and only 4 schizonts were found in fetal membranes of 3 sheep. In contrast, parasites and lesions were found in maternal placentomes of cattle, sheep, and goats. Thus, results from these experimentally infected animals indicate that although *Sarcocystis* is present in the maternal placenta, it rarely infects the fetus or fetal membranes. Unlike experimentally infected cattle, those with natural infections have had parasites, lesions, or both, in the fetuses (Figure 1.94).[30,844,908,913,988,1152,1246,1558,1741] Unlike experimental infections, parasites, as well as lesions, were found in the placentas in several natural infections.[256,365,1246] Furthermore, some naturally infected cows were asymptomatic, although organisms were detected in the fetus or placenta.[365] It is not possible to confirm that the natural infections and the experimental infections were due to the same species of *Sarcocystis*, although the parasites were morphologically similar, parasitized the same type of host cells, and, in some cases, responded serologically to the same antigen. Thus, the question remains as to whether differences observed between experimentally infected animals and naturally infected animals were due to different species or strains of *Sarcocystis*.

There are many unanswered questions and observations that appear contradictory within the subject of how sarcocystosis affects fetal health. Some fetuses are infected and have lesions, others are unaffected. Some fetal placentas are infected and have lesions, others are unaffected. Some maternal placentomes are infected and have lesions, others are unaffected. Most pregnant animals have overt clinical illness at the time the fetus is affected, others do not. Several possible mechanisms have been hypothesized by which sarcocystosis can directly or indirectly affect

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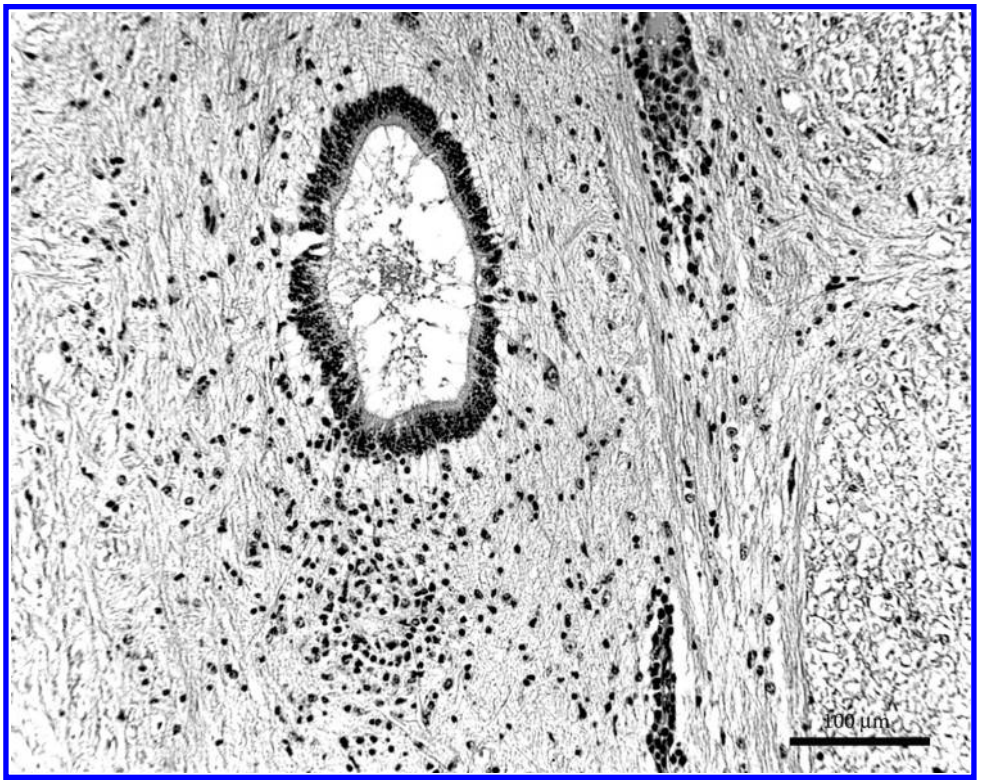


Figure 1.92 A glial nodule and perivascular infiltration of mononuclear cells in the spinal cord of a calf infected with *S. cruzi*. H and E stain. (From Dubey, J.P., Speer, C.A., Epling, G.P., 1982. *Am. J. Vet. Res.* 43, 2147–2164. With permission.)

fetal health.[592] In naturally infected bovine fetuses, parasites were found in virtually all organs, most often in the brain. Immature and mature schizonts and free merozoites were found, usually within endothelial cells of capillaries, but occasionally free in the lumen of a vessel or in neural tissue.[30,71,365,844,908,913,988] Neural lesions include nonsuppurative encephalitis or meningitis, with small foci of glial cells surrounding a central necrotic area throughout the gray and white matter of the cerebrum, cerebellum, or brainstem, with some perivascular mononuclear cell infiltration and occasional microthrombi in vessels in reaction foci. Other affected organs are similar to those of infected postnatal animals, including nonsuppurative myocarditis, pneumonitis, hepatitis, and renal glomerulitis accompanied by focal necrosis and hemorrhage.

One premature lamb born to an experimentally infected ewe had cerebral congestion and edema, as well as areas of leukoencephalomalacia in the cerebrum and midbrain.[1254] Another lamb, from a naturally infected ewe, was found with encephalitis characterized by mononuclear cell infiltration and gliosis.[386] Abortion and stillbirths were reported in sheep from Brazil[1390] and Denmark[17] (see Chapter 8), and in yak from China.[1816]

1.6.7.1 Placental Infection and Lesions

Free merozoites and schizonts, usually associated with lesions, have been observed in maternal or fetal placentas of some cattle, sheep, and goats. Immunofluorescent staining of placentomes from 6 experimentally infected cows revealed numerous merozoites and schizonts in the maternal, but

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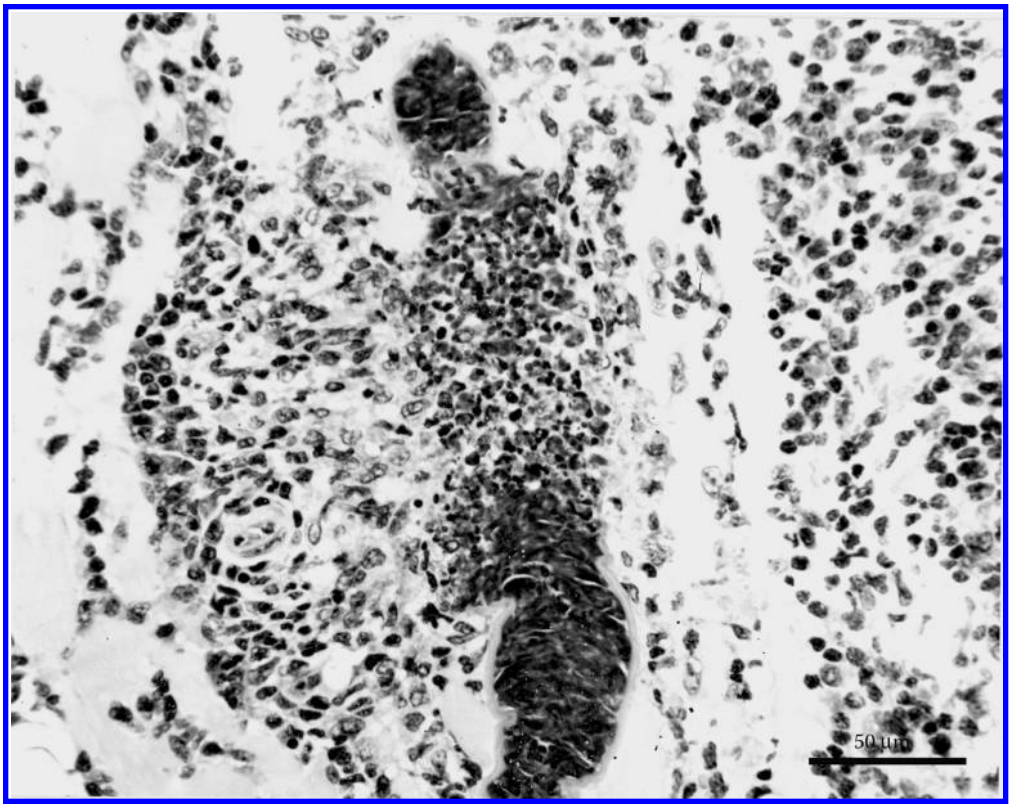


Figure 1.93 Part of a degenerating *S. capracanis* sarcocyst being replaced by neutrophils in the diaphragm of a vaccinated-challenged goat. Mononuclear cells surround the sarcocyst. H and E stain. (From Dubey, J.P., 1983. *Vet. Parasitol.* 13, 23–34. With permission.)

not the fetal, placentome.[70] Placentomes of all 6 cows were atrophied. Maternal placentomes from naturally infected aborting cows were also found to contain numerous parasites, but were hemorrhagic and necrotic with calcification of placental villar epithelial cells and of the fibrous connective tissue.[256] In other natural infections resulting in abortion, fetal placental lesions were mild to severe, with accompanying mononuclear cell infiltration.[365,1246] Of 11 experimentally infected sheep, only 4 schizonts were found in fetal membranes from 3 ewes.[1037] In experimentally infected goats, schizonts were found in the maternal placentome and endometrium, but not in fetal membranes.[357] Lesions were restricted to the maternal placentome and endometrium with heavy mononuclear cell infiltration and some focal necrosis.

1.6.7.2 Possible Mechanisms Causing Abortion or Fetal Death

Based on the forgoing descriptions of parasite locations and lesions, there appear to be several possible ways in which sarcocystosis might initiate abortion or fetal death (Figure 1.95).

One hypothetical sequence of events leading to labor might begin in the hypothalamus of the fetal brain, with transmission via the pituitary to the fetal adrenal, where cortisol provides the fetal trigger to maternal endocrine changes.[195] In turn, there might follow a release of steroids and prostaglandins from the placenta, the placentome, and the fetal membranes, which stimulate the myometrium to contract and the cervix to dilate. Under normal conditions, it is thought that there is a rise in concentration of plasma cortisol secreted from the fetal adrenal as it grows and

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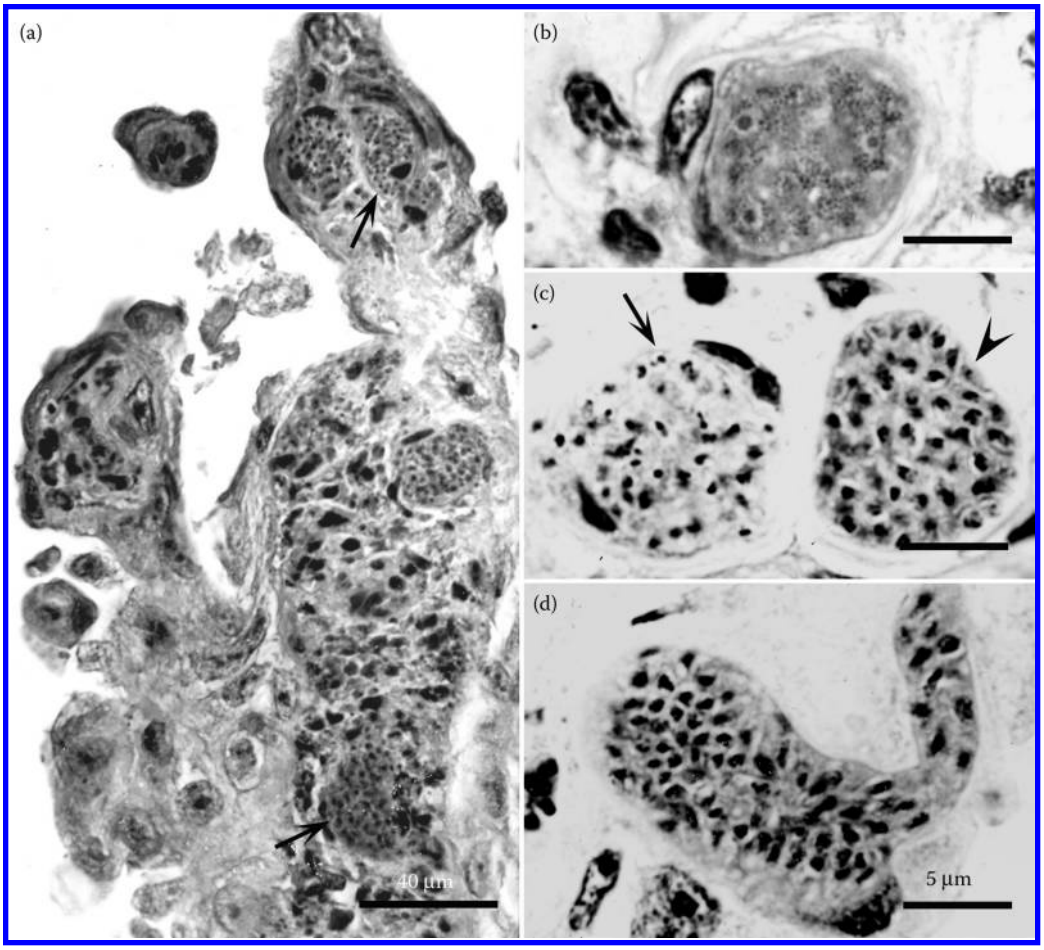


Figure 1.94 Necrosis and schizonts of *Sarcocystis* in a fetal placental cotyledon of a naturally infected cow. (a) Necrosis, schizonts (arrows), and numerous merozoites. (b) Early schizont with lobulated nucleus. (c) A degenerating (arrow) and normal (arrowhead) schizont. (d) An irregular-shaped schizont. H and E stain. (From Dubey, J.P., Bergeron, J.A., 1982. *Vet. Pathol.* 19, 315–318. With permission.)

develops, with the highest concentrations during the last few days of fetal life.[195] Mechanisms that result in high adrenocorticotrophic hormone (ACTH) or cortisol concentrations in the fetus leading to premature parturition or abortion are not known. One sequence of events could begin with anemia in the dam resulting in hypoxia in the fetus. Hypoxia could also result from insufficient blood-flow through the maternal placentome due to occlusion of capillaries by schizonts, localized hemorrhage, or intravascular coagulation. Evidence of hypoxia is the finding of leukoencephalomalacia in some fetuses. Vasopressin, known to stimulate ACTH release, rises severalfold with the approach of spontaneous parturition and in response to hypoxia.[195] Hypoxia itself has been shown to raise the concentration of immunoreactive ACTH in fetal plasma at all times during gestation, when it was studied. Isolated fetal adrenal cells appear to be responsive and secrete large amounts of cortisol in response to ACTH, a form of GTP, or dibutyrylcyclic AMP at various times during gestation.

Vascular damage associated with sarcocystosis in the dam might lead to changes in the concentration of hormones associated with the maintenance of gestation. The plasma concentration

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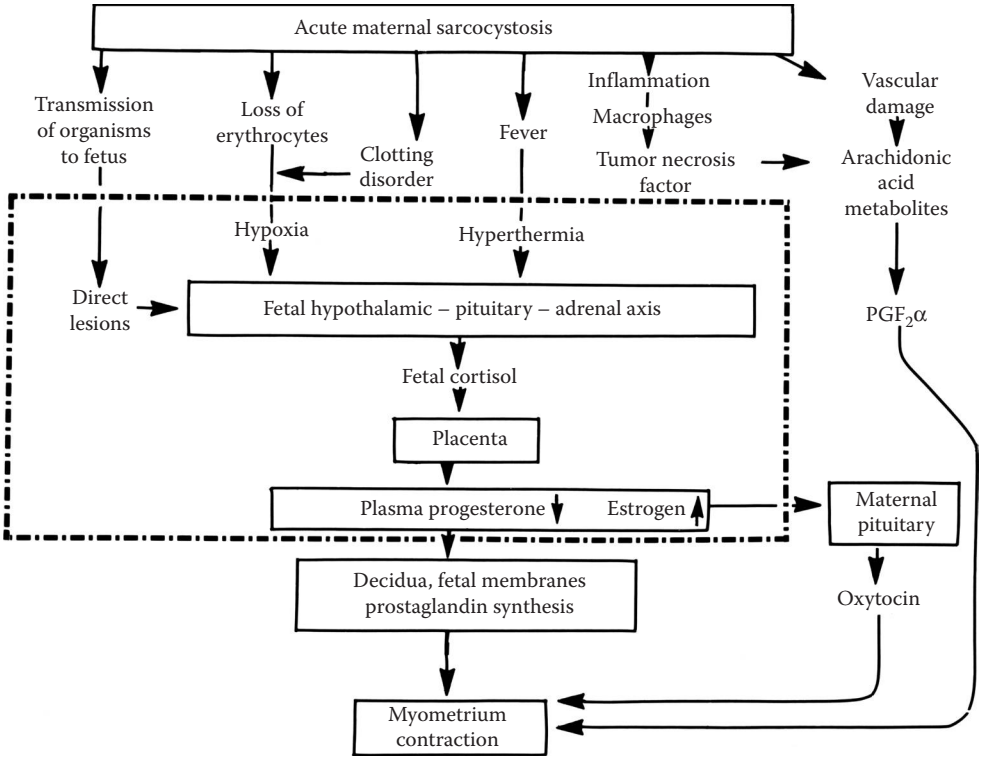


Figure 1.95 A hypothetical diagrammatic presentation of events leading to *Sarcocystis*-induced abortion. Fetal hormonal events are shown within the area marked by dotted lines. (Adapted from *Transplacental Effects on Fetal Health*. Alan R. Liss, Inc., New York, NY, pp. 153–164.)

of progesterone, E1, E2, PGFM, and alpha-fetoprotein, was monitored in pregnant cows experimentally infected with *S. cruzi*.[55,56]

Progesterone production after day 50 of gestation in sheep is primarily in the placenta. It acts by maintaining the myometrium refractory to stimulation by oxytocin or PFG2α. Decrease in placental progesterone at term follows and is caused by a rise in fetal cortisol levels. Progesterone withdrawal initiates increased PGF2α secretion and parturition.

In late pregnancy, estrogens usually promote uterine contractility, increase responsiveness of the uterus to agonists such as oxytocin and PGF2α, inhibit progesterone production, and stimulate prostaglandin synthesis and release. Maternal estrogen increases in response to fetal adrenal activity.

Unfortunately, changes in progesterone, E1 and E2 concentration in the experimentally infected cows could not be interpreted as cause or effect. Although the changes preceded or were concurrent with abortion, similar changes were documented at the time of normal parturition. Prostaglandins initiate labor by stimulating myometrial contractions and by softening the cervix. Two specific lines of observation suggest that events in acute sarcocystosis might result in increased concentration of prostaglandins in the peripheral circulation of the dam or in the maternal placentome and fetal placenta. First, release of second-generation merozoites is associated with damage of capillary endothelium, an event known to trigger the arachidonic acid cascade leading to PGF2α release. Parasitic disruption of cell membranes and the action of inflammatory cells could also stimulate platelet activity, including the release of PGF2α by platelets, and by damaged endothelium. Second, when second-generation merozoites mature, numerous mononuclear inflammatory cells are found perivascularly throughout the body and in the placenta. Many of these cells are macrophages, and

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in vitro studies indicate that macrophages can be stimulated by *Sarcocystis* lysates to release the tumor necrosis factor (TNF).[592,2009] One activity of TNF is the triggering of an arachidonic acid cascade leading to PGF2α release. Because the findings in identifying those events leading to high maternal plasma PGF2α concentration preceding bovine abortion were inconclusive, studies need to be conducted to clarify the relationship between sarcocystosis and prostaglandins.

Fever has been associated with premature parturition and sometimes regarded as the cause. However, it is not clear if fever itself is a cause or an effect. Pyrogens from infectious agents such as protozoa, or from degenerating body tissues, can cause fever. Pyrogens can act directly on the hypothalamus to raise the thermostatic control or can act indirectly by stimulating leukocytes to produce endogenous pyrogen. Endogenous pyrogen is thought to cause fever by inducing prostaglandin E1 formation, which elicits the fever reaction. Thus, it is not clear if fever directly affects the fetus, or if the initiators of fever—in this case, *Sarcocystis* pyrogens or prostaglandins—induce lesions. Prostaglandins were elevated during acute sarcocystosis also in pigs.[300]

In addition to the forgoing hypotheses leading indirectly to death or premature parturition of the fetus, invasion of fetal tissues by the parasite might result in specific lesions with similar end results. Distribution of parasites throughout the brain with associated lesions might stimulate the fetal hypothalamic–pituitary–adrenal axis or destroy significant brain tissue leading to death. Evidence of fetal myocarditis, pneumonitis, and hepatitis, indicate other possible avenues leading directly to fetal death.

1.6.8 Eosinophilic Myositis and Sarcocystosis

Eosinophilic myositis (EM) is a specific inflammatory condition of striated muscles, principally due to accumulations of eosinophils. It has been found mainly in cattle,[12,151,309,526,654,724,725,871,907,1046,1460,1472,1511,1778,1943] occasionally in sheep,[907] and rarely in pigs and horses. The affected animals might appear clinically normal. Most of findings are at the abbatoir level; thus, EM is generally detected at the time the surface of the carcass is inspected or when it is cut into prime cuts or into quarters.[907] Sex and breed have no influence on the prevalence. The condition was reported with equal frequency in steers, cows, and heifers. Virtually all striated muscles, including skeletal muscles, the muscles of the eye, larynx, and heart, can be affected.

In the United States, federal meat inspection regulations require that parts, or, in severe cases, the entire carcass, affected with EM be condemned. Generally, EM has been found in 1 of 100,000 slaughtered cattle, but in some feedlots up to 5% of the cattle were condemned.[907]

Two types of lesions are found associated with EM.[907] The most common lesion found on the surface of muscles is multifocal, spindle-shaped to round, and 5 to 15 × 1 to 3 mm in size. The color of the lesion can vary. Green lesions result from accumulations of eosinophils, principally between myocytes (Figure 1.1e). With the progression of EM, eosinophils and myocytes degenerate, resulting in granulomas with a central area of necrosis. Later, the central necrotic tissue becomes surrounded by zones of giant cells, epithelial cells, eosinophils, lymphocytes, and fibrocytes.[907]

A second type of lesion is less prevalent but more conspicuous. It can be up to 15 cm long, bright green to pale yellow, and firm in consistency.

The etiology of EM remains uncertain. Because sarcocysts are found in the same location as EM, *Sarcocystis* has been traditionally considered to be the cause of EM. The *Sarcocystis*-induced etiology of EM was supported by the finding of *Sarcocystis*-specific IgE antibody in lesions.[526,724,725] Because of the small number of cases of EM compared with the high prevalence of intramuscular cysts, the confirmation of *Sarcocystis* as the cause, and EM as the effect, requires more substantial evidence. It is necessary to note that EM has never been seen in any experimentally infected ox, sheep, or other animals. In experimentally infected animals, degenerating sarcocysts are often surrounded by mononuclear cells and neutrophils. Because most of

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these observations were made in animals infected less than a year of age and with only 1 dose of sporocysts, repeated natural infections can result in a different reaction, perhaps involving other cell types such as eosinophils.

Recently, EM-like lesions were induced in 2 calves by repeated injections of *Sarcocystis* antigen with adjuvants.[1777] EM has been found associated with all 3 species of *Sarcocystis* in cattle, *S. cruzi*, *S. hirsuta*, and *S. hominis*.[654,1778] In the study from Belgium, of 97 beef samples condemned because of EM, sarcocysts were found in 27; most (82%) of the sarcocysts were *S. hominis*.[1778] In the study from Colorado, United States, sarcocysts were found in 32 of 363 EM lesions and all of them were *S. cruzi*.[654] *S. hirsuta* sarcocysts were found in all 18 cattle that were condemned because of grossly visible sarcocysts.[397]

1.6.9 Chronic Sarcocystosis and Toxins

Little is known of the pathogenesis of chronic sarcocystosis. Anatomic lesions are insufficient, especially in cattle, sheep, and pigeons,[1099] to explain the etiology of CNS signs. Although sarcocysts in muscles or in the CNS are well adapted, usually without any host reaction, some sarcocysts probably rupture from time to time and might release toxic products. It has been known for about a century that an aqueous extract of bradyzoites is toxic (sarcotoxin) when inoculated into rabbits. Of mice, guinea pigs, chickens, and rabbits injected with *S. cruzi*, extract toxic reaction was observed only in rabbits.[1061,1513,1516,1527,2271] The crude *S. cruzi* extract was water soluble, acid–alkali stable, and thermolabile. Harada et al.[781] commented on 27 outbreaks of food poisoning in humans after eating uncooked horse meat in Japan. They proposed that the cause was a toxin associated with a 15 kDa protein from *S. fayeri*; freezing the meat for 2 days at −20°C abolished the toxic property of the infected meat. It is not clear how, or if, sarcotoxin might be released from intact sarcocysts and, if released, what role it might play in chronic sarcocystosis. Although lectins are also associated with bradyzoites, their role, if any, in causing disease is unknown.

It is possible that substances released from *Sarcocystis* stimulate production of TNF.[584] TNF is known to be associated with wasting disease. Poor weight gain and low feed efficiency in sarcocystosis are regulated through growth-regulating hormones.[505,506] Any relationship between TNF and the growth-regulating hormones has not been established.

Attempts have been made to characterize antigenic/biochemical properties of extracts prepared from sarcocysts, particularly *S. gigantea* of sheep, because of the availability of macroscopic sarcocysts from sheep esophagus and their easy separation from the host tissue.[1140,1527,1734,1746–1748,1945,2133,2176] Lectins and species-specific polypeptides were demonstrated. However, the role of these substances in the pathogenesis of sarcocystosis is not clear.

1.7 IMMUNITY

Cellular and humoral immune responses in infected animals indicate that *Sarcocystis* species are immunogenic in intermediate hosts. Unfortunately, most information is available only from responses directed against antigens derived from bradyzoites. Because bradyzoites are obtained from the terminal stage (sarcocysts), only cross-reactive antigens are actually tested.

1.7.1 Antigenic Structure

Relatively large numbers of highly purified parasites are needed to study the proteins and antigens of *Sarcocystis* spp. Parasites can be obtained from experimentally infected animals or from *in vitro* cultivation. Parasites such as bradyzoites should be obtained from experimentally infected animals, and not from naturally infected ones, to insure accurate species identification. Whether

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parasites are obtained from *in vivo* or *in vitro* sources, biochemical analyses should also include controls of solubilized noninfected host cells or tissues.

There is little information concerning the proteins and antigens of *Sarcocystis* spp., and most information is on *S. cruzi*, *S. neurona*, and *S. muris*. Proteins and antigens of *S. cruzi* sporozoites, merozoites, and bradyzoites were studied with sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS–PAGE), and Western blotting with immune serum or monoclonal antibodies, in the hope of developing a commercial diagnostic test for bovine sarcocystosis that did not materialize because of lack of interest by industry. Bradyzoites and merozoites had similar proteins with Mr 15.7 kDa, several between Mr 18–21.4,29, and 110 kDa; proteins with Mr 15.7,29,49.9 and 54K kDa were common to sporozoites and merozoites. Only 2 proteins (Mr 15.7 and 16.5 kDa) were present in all 3 parasite stages.

Based on SDS–PAGE analysis of *S. cruzi* merozoite proteins, some appeared, disappeared, and then reappeared during *in vitro* development of merozoites. Merozoites harvested at 36 and 48 DPI (called 36D and 48D merozoites) from bovine pulmonary artery endothelial cells each had a unique protein of Mr 50 and 70 kDa, respectively. Merozoites harvested at 36 and 60 DPI had many proteins with similar molecular weights as well as several unique proteins.[393] For example, 36D merozoites had a 43-kDa protein that was not present in 60D merozoites, and 60D merozoites had a prominent 63.1-kDa protein not present in 36D merozoites. A 63.1-kDa protein first appeared in 37 or 38D merozoites and was also present in 38, 40, 48, 52, and 60D merozoites but was absent in 41 and 58D merozoites. Two other proteins also showed temporal variation in appearance. A 43-kDa protein was present in 31, 36, and 52D merozoites, whereas a 42.3-kDa protein was present in 29, 40, and 48D merozoites, and both proteins were present in 33, 38, 41, 58, and 60D merozoites.

In Western blot analyses, antibodies in bovine serum reacted with increasing numbers of antigen bands as the animals progressed through the course of infection. Proteins with Mr 21,40, and 50 kDa might be potent antigens because serum reacted strongly with them beginning early in infection.

Monoclonal antibodies (MAbs) have been generated against *S. cruzi*[154,1659] by immunizing female BALB/cBY mice 8–10 weeks of age with 10^6 merozoites or sporozoites per mouse. Merozoites were mixed 1:1 (v/v) in complete Freund's adjuvant and given by intraperitoneal injection. Mice were intravenously injected 1 month later with 8×10^5 merozoites or 5×10^5 sporozoites, respectively; 3 days later the spleens were removed and fused to the myeloma cell line P3/X63-Ag8.6.5.3 and selected as previously described. Hybridoma culture supernatants were screened on acetone-fixed merozoites or sporozoites by an IFA, cloned by limiting dilution, re-tested, and expanded in culture. MAbs were tested for their ability to react with whole parasites in a live IFA or with parasite proteins in Western blots.[393]

A variety of reactivities occurred when either antisporozoite or antimerozoite MAbs were used to detect antigens of *S. cruzi* on Western blots of whole parasite (sporozoite, merozoite, or bradyzoite) antigens separated by SDS–PAGE. Several MAbs reacted in IFA with acetone-fixed sporozoites and merozoites, and 3 MAbs reacted with the surface of live sporozoites or merozoites. In Western blots, the surface-reactive MAbs reacted with polypeptides ranging from Mr 20 to 74 kDa. Interestingly, only 1 of the MAbs analyzed reacted with blots of antigens prepared from homologous (eliciting) stages, although several did react with the surface of homologous stages in the live IFA. Thus, certain epitopes on the surface of both merozoites and sporozoites might have been irreversibly denatured in the blotting procedure, labile to certain proteases or nonprotein in composition (e.g., lipid, glycolipid). In addition, several epitopes on the surface of live merozoites appeared to be constituents of molecules within sporozoites. Such molecules can be precursors of surface components of merozoites or might simply share these epitopes on unrelated molecules. Antimerozoite MAbs also identified shared epitopes in sporozoites and bradyzoites that appeared to be located on molecules of both similar molecular weight (i.e., 40 kDa) and distinct molecular weight (i.e., 40 vs.

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20 and 60 kDa). An MAb reactive with the surface of *S. cruzi* merozoites also identified epitopes on sporozoite and bradyzoite antigens, indicating that some of the 20- to 60-kDa sporozoite molecules may be precursors to internal or surface antigens of bradyzoites. Further investigations will be needed to elucidate the molecular relationships and functions of these molecules in the development of the major stages in the life cycle of *S. cruzi*.

Several MAbs generated against *S. cruzi* also revealed several patterns of fluorescence on infected bovine cardial pulmonary artery endothelial (CPA) cells.[393] For example, all 3 surface-reactive MAbs reacted with single, intracellular or extracellular merozoites and with merozoites in mature schizonts but did not react with schizonts in intermediate stages of development. Thus, the antigens identified by these 3 MAbs were expressed only by mature merozoites and not by intermediate schizonts. Results from the IFA indicated that intracellular merozoites shed the antigens into the host-cell cytoplasm. These antigens were then expressed on the surfaces of the host cell, as well as on adjacent noninfected CPA cells. Such observations indicate that active processing of antigen by neighboring CPA cells might occur after indirect acquisition of parasite antigens from adjacent infected CPA cells. Similar antigen processing and presentation might also occur in endothelial cells in infected animals.

For characterization of antigens/antibodies of *S. neurona*, see Chapter 3.

Three surface antigens of Mr 27, 43 and 90 kDa have been identified by ^{125}I labeling and immunoprecipitation of *S. muris* bradyzoites with specified rabbit or mouse antiserum.[1] Surface antigens of *S. muris* and *S. miesheriana* bradyzoites and sporozoites have been reported.[1642,1643,2336] Two major proteins were found in micronemes of *S. muris*.[1403]

Monoclonal antibodies have also been generated against some other *Sarcocystis* species for species differentiation and diagnostics.[1285] All 6 monoclonal antibodies against *S. gigantea* and *S. tenella* of sheep were species-specific.[2335,2337] One of these monoclonal antibodies was directed against micronemes of *S. tenella*, whereas 2 antibodies against *S. gigantea* reacted with micronemes and amylopectin granules.[1285] Species-specific and cross-reactive epitopes have been characterized in *S. muris*.[545,1183,1733,1998] Jäkel et al.[891] characterized monoclonal antibodies against *S. singaporensis* and their role in invasion and escape of the parasites *in vitro*. Tenter et al.[1731] used isoelectric separation of species-specific proteins to differentiate *S. gigantea*, *S. tenella*, and *S. arieticanis* of sheep.

1.7.2 Humoral Responses

Cattle,[577,580,589,626,681,1087] pigs,[1286,1287,1289,1461,1813,1826,1827] sheep,[666,1250,1294] mice,[1,186,193,666,839,1044,1729,1730] and rats[890] inoculated with *Sarcocystis* sporocysts developed IgG antibodies starting 3–5 weeks PI with respective species of *Sarcocystis*. These antibodies were detectable by IHA,[193,1087] ELISA or dot-ELISA,[666,1287,1729,1730] IFA,[186,1730] or complement fixation[1250] tests. The IgM antibodies appeared earlier than IgG antibodies, but were short-lived, usually declining to low levels by the time sarcocysts matured. The IgG antibody concentration in serum peaked during the early period of sarcocyst formation and persisted at a relatively high concentration during the chronic infection. There was no anamnestic antibody response in calves orally challenged with sporocysts 3–5 months after primary infection.[666] IgA and IgG2 antibodies were not found.[666]

The onset and persistence of *Sarcocystis* antibodies varied with the species of the host, species of parasite, source of antigen, and serological test. Although *Sarcocystis* species share antigens, the antibody titers were higher using antigen from homologous species of *Sarcocystis* than from heterologous species.[666] For example, antibody titers in pigs inoculated with *S. miescheriana* were higher when the antigen was derived from *S. miescheriana* than from *S. muris* of the mouse or *S. gigantea* of sheep.[1286]

Both bradyzoites and merozoites can be used in ELISA, but titers were higher by using the merozoite antigens.[1545,1549]

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1.7.3 Cellular Responses and Immunosuppression

As might be expected of an intracellular parasite, immune cells are mobilized during the *Sarcocystis* infection. The predominant cells infiltrating visceral and muscular tissue during *Sarcocystis* infection are lymphocytes and macrophages.[366,911] This mononuclear cell infiltration begins during the third week of infection and can last for several months, long after the parasite is no longer demonstrable in visceral tissues. Not only visceral tissues are affected, but lymphocytes from peripheral circulation also show a blastogenic response when stimulated with antigen-specific *Sarcocystis*. In calves and sheep inoculated with *Sarcocystis*, this blastogenic response was evident 2 weeks after infection, coincident with the release of first-generation merozoites in cattle and sheep.[666]

Whether these cellular events participate in the recovery of the host from sarcocystosis has not been established, and passive transfer of resistance via cells or antibodies has not been reported. In certain animals, sarcocystosis can depress immunity.[628] Goats with subclinical infection with 1000 sporocysts were more susceptible to intestinal coccidial infections than controls.[389]

The intense cellular response seen in immune animals that survive lethal challenge indicates cell-mediated immunity against *Sarcocystis* (Figure 1.93). Cytotoxic antibodies or metabolites are known to destroy second-generation extracellular merozoites[1650] (Figure 1.96).

Immune responses might differ with the strain of *Sarcocystis* and the strain of the host, as demonstrated with *S. singaporensis* infection in rats.[890] The strain S5, originally obtained from feces of a wild python in Thailand, was highly pathogenic for laboratory rats and less pathogenic for wild rats. After 10 serial passages between pythons and Fischer's rats, the *S. singaporensis* strain (designated S1) became completely nonpathogenic for wild rats, irrespective of the dose.[890] Different classes of immunoglobulins were characterized in brown rats during acute and chronic infection with the modified S1 strain of *S. singaporensis*. Some strains became more virulent after serial passage in the laboratory.[892] Immune responses were higher for hypervirulent strains versus avirulent strains.[892] These findings should be considered when evaluating results of *Sarcocystis* infection in different hosts. Little is known of the comparative virulence of strains of *Sarcocystis* circulating in nature.

1.7.4 Protective Immunity and Vaccination

Cattle, sheep, goats, and pigs inoculated orally with a dose of sporocysts that resulted in subclinical infection were protected against a challenge dose that normally would have been lethal. This protective immunity persisted for 80 days, but not 120 days, in pigs,[1827] at least 252 days in cattle,[590] 274 days in goats,[372] and at least 90 days in sheep.[612] The size of the immunizing dose is important. Goats and pigs dosed with 100 or more sporocysts of *S. capracanis* and *S. miescheriana*, respectively, were protected; those dosed with 10 sporocysts were not protected. The degree of protection was better with 1000 sporocysts than with 100 sporocysts.[358,1880] Immunized animals had only mild illness after a putative lethal challenge, even though parasites from the challenge inocula underwent some development in the host.[358,1880] The protective immunity was induced only by homologous *Sarcocystis* species because cattle experimentally infected with *S. hirsuta* were not protected against challenge infection with *S. cruzi*.[590] The interruption of the second-generation schizogony by chemoprophylaxis did not affect the outcome of protective immunity. Cattle inoculated with lethal doses of sporocysts and treated with amprolium from days 21 to 35, survived acute sarcocystosis and developed protection against lethal challenge.[590]

The stage of the parasite responsible for protective immunity is not known but is likely to be either the sporozoite or first-generation schizonts. Pigs were vaccinated with live, killed, or fractions of *S. miescheriana*-bradyzoites-induced antibodies but provided no protection.[1289] Similar results were obtained in mice immunized with killed *S. dispersa* bradyzoites. Inoculation with *S. muris* sporocysts irradiated with 5 or 10 krad induced protective immunity in mice.[1044] Irradiated vaccines have not been tried in large animals.

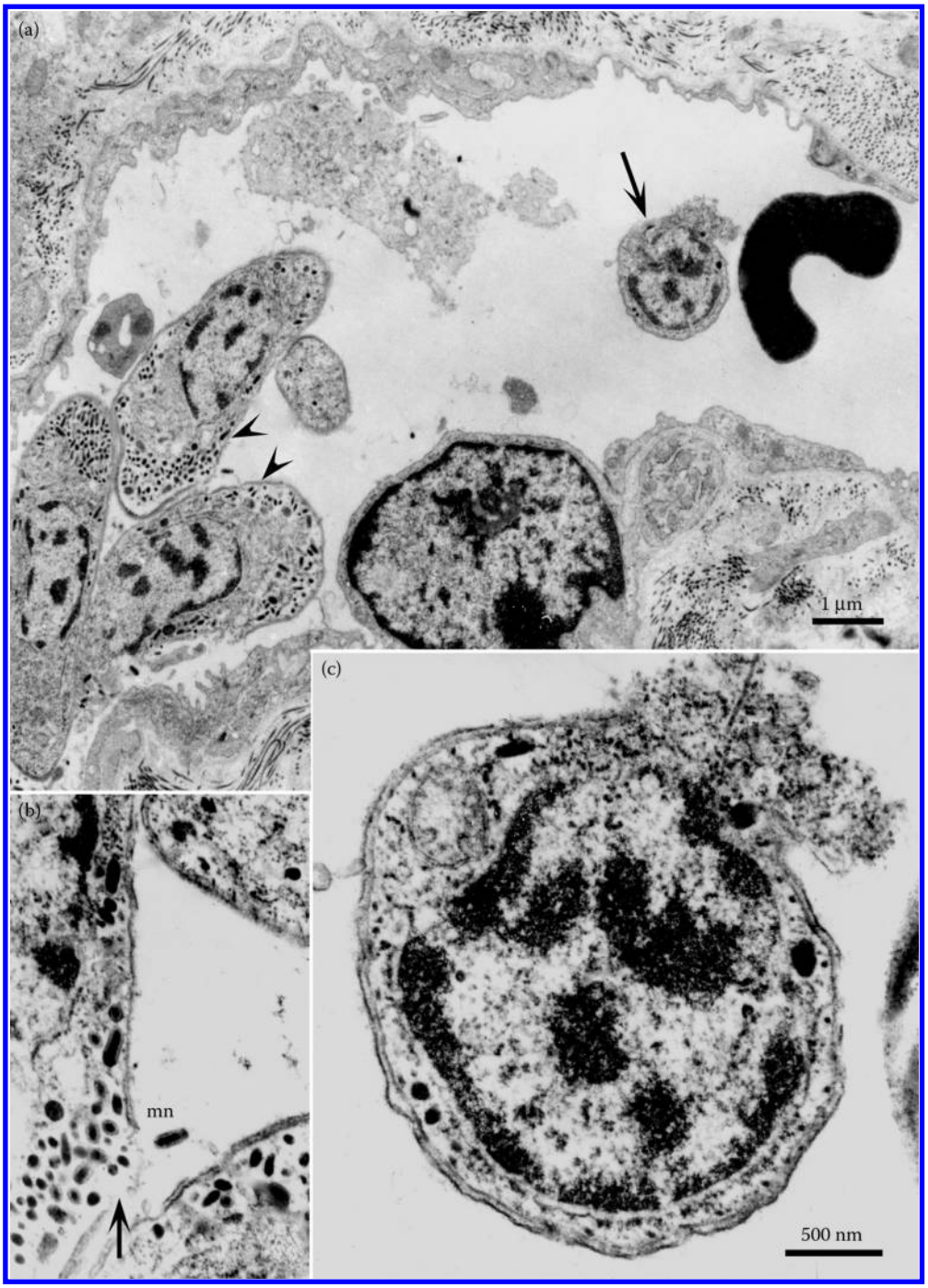


Figure 1.96 TEM of *in vivo* lysis of *S. cruzi* merozoites in a venule in the kidney of an experimentally infected calf. (a) Five merozoites are present in the vascular lumen. Cytoplasm is extruding from 1 merozoite (arrow) adjacent to a red-blood cell (dark, U-shaped). (b) Higher magnification of the merozoites (2 arrowheads) showing partial lysis of double pellicular membranes (arrow). Micronemes (mn) are being released from a pellicular lesion. (c) Higher magnification of a merozoite in (a), showing cytoplasm escaping (top right side) from a lysed merozoite. (From Speer, C.A., Dubey, J.P., 1981. *J. Parasitol*. 67, 961–963. With permission.)

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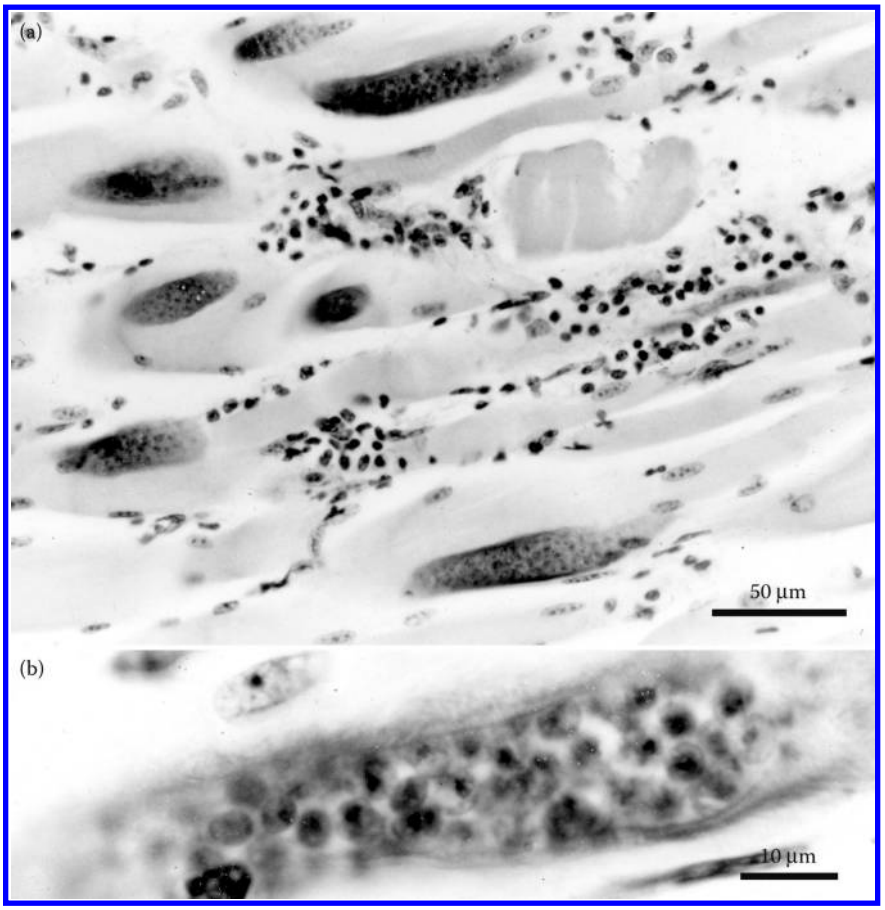


Figure 1.97 Myocarditis in a steer naturally infected with *S. cruzi*. H and E stain. (a) Myocardial cell degeneration (arrow), infiltration by mononuclear cells, and 7 immature sarcocysts. (b) Higher magnification of a sarcocyst to show spheroid metrocytes. (Adapted from Giles, R.C. et al., 1980. *J. Am. Vet. Med. Assoc.* 176, 543–548.)

The administration of colostrum has no protective response in cattle and sheep, as these animals remain susceptible to clinical sarcocystosis from birth and throughout their lives despite repeated natural infection.[577,1250]

The persistence of live sarcocysts is probably not essential for the maintenance of protective immunity. Goats were protected from challenge infection even when sarcocysts were no longer demonstrable or were markedly reduced in number.[372]

1.8 DIAGNOSIS

Diagnosis of acute sarcocystosis is difficult. The disease is generalized in nature, with no specific signs; finding parasites in tissues of acutely infected cattle is unlikely. There is no commercially available or standardized serologic test, and virtually all cattle, regardless of clinical involvement, have been found with some sarcocysts in muscles. A diagnosis of bovine sarcocystosis is based on the elimination of other causative agents, a good epidemiologic evaluation of the herd and its relationship to other animals (especially dogs), and clinical findings.

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A presumptive diagnosis of acute sarcocystosis can be made if anemia, anorexia, fever, excessive salivation, abortion, loss of body hair (especially at the tip of the tail), increased levels of LDH, SBDH, CPK, BUN, and bilirubin, or lowered PCV are present. Finding *Sarcocystis* antibody or antigen in serum can aid diagnosis of acute sarcocystosis. There is a parasitemia during acute sarcocystosis. Although merozoites were found in buffy coat smears of cattle with acute sarcocystosis from 16 to 46 DPI[363]; this method is tedious, time consuming, and not practical for routine diagnosis. An ELISA to detect circulating antigen in mice and pigs has been reported.[1286] This method might eventually prove useful in diagnosis of acute sarcocytosis in naturally infected animals. The detection of humoral antibodies can aid diagnosis, even though titers sometimes do not correlate with the clinical state. Comparison of antibody titers in acutely ill animals—with those in animals not showing clinical signs—can be useful.[626,681] The more animals tested within an affected herd, the greater the ability to interpret trends in serologic data.

Sarcocystis antibodies have been detected by IHA, ELISA, dot-ELISA, and IFA tests.[393,488,1213,1215,1216,1700] At present, there is a lack of standardization of these tests. Because antigens are obtained from sarcocysts in the muscles of experimentally infected animals and consist of lysate of the bradyzoites,[1087] variations in preparative methods yield antigens varying greatly from one batch to another. Although antigens obtained from merozoites might be more suitable for serologic diagnosis of acute sarcocystosis than antigens from bradyzoites, merozoite antigens have not been utilized for diagnostic purposes. Serologic cross reactivity among *Sarcocystis* species is inconsistent. Although *S. muris* bradyzoite antigens from mice are more easily obtained than *S. cruzi* bradyzoite antigens from cattle, *S. muris* antigens are not suitable for the diagnosis of acute sarcocystosis in cattle. Antigens from *S. cruzi*, however, have been found suitable to detect antibodies against *S. hirsuta* in cattle and *S. tenella* in sheep.[666]

Finding schizonts in biopsies of muscles and lymph nodes, or in additional tissues post mortem, can aid diagnosis. However, schizonts are often too few to be found in histologic sections, because they might disappear by the time clinical disease is obvious. Finding large numbers of immature or mature sarcocysts at the same stage of development can also aid diagnosis (Figure 1.97). Most clinical outbreaks are likely to occur between 5 and 11 weeks following the initial ingestion of sporocysts. Coupled with the history of feeding raw beef to local dogs (or the consumption of a carcass by dogs or wild carnivores), this time period might be a useful guide. Factored into this guide is the consideration that it might take as long as 2–3 weeks for a carnivore to excrete sporocysts after ingesting beef.

In histologic sections, *Sarcocystis* must be differentiated from *Toxoplasma gondii* and other closely related coccidians. *Sarcocystis* schizonts develop in endothelium of blood vessels. The immature schizonts are basophilic structures with or without differentiated nuclei (Figure 1.98). Ultrastructurally, *Sarcocystis* schizonts are free in the host-cell cytoplasm, whereas all stages of *T. gondii* are separated from the host-cell cytoplasm by a PV and can develop in virtually any cell in the body. Because each *T. gondii* organism divides into 2, there is no immature stage in the intermediate host.

The diagnosis of *Sarcocystis*-induced abortion presents additional problems because the parasite is not consistently found in fetal tissues. Sarcocystosis produces gliosis and placental necrosis; therefore, brain and placenta should be examined (Figure 1.98). A detailed description of a case of congenital infection was described in a stillborn lamb from Denmark.[17] Within the placental cotyledons, schizonts were in both the lamina propria and submucosa. The chances of diagnosis are improved if numerous fetal tissues are examined.

IHC with specific antibodies is very useful in diagnosis of parasites in histological tissue sections. However, the specificity depends on the purity of reagents, antigenic variability among the strains of *Sarcocystis* used, and hosts used to produce antibodies.[436] IHC is only an aid, and other factors should be considered for final diagnosis. The IHC procedure is detailed in Chapter 2, and has been commonly employed for the diagnosis of *S. neurona* (Chapter 3). Cross reactivity among

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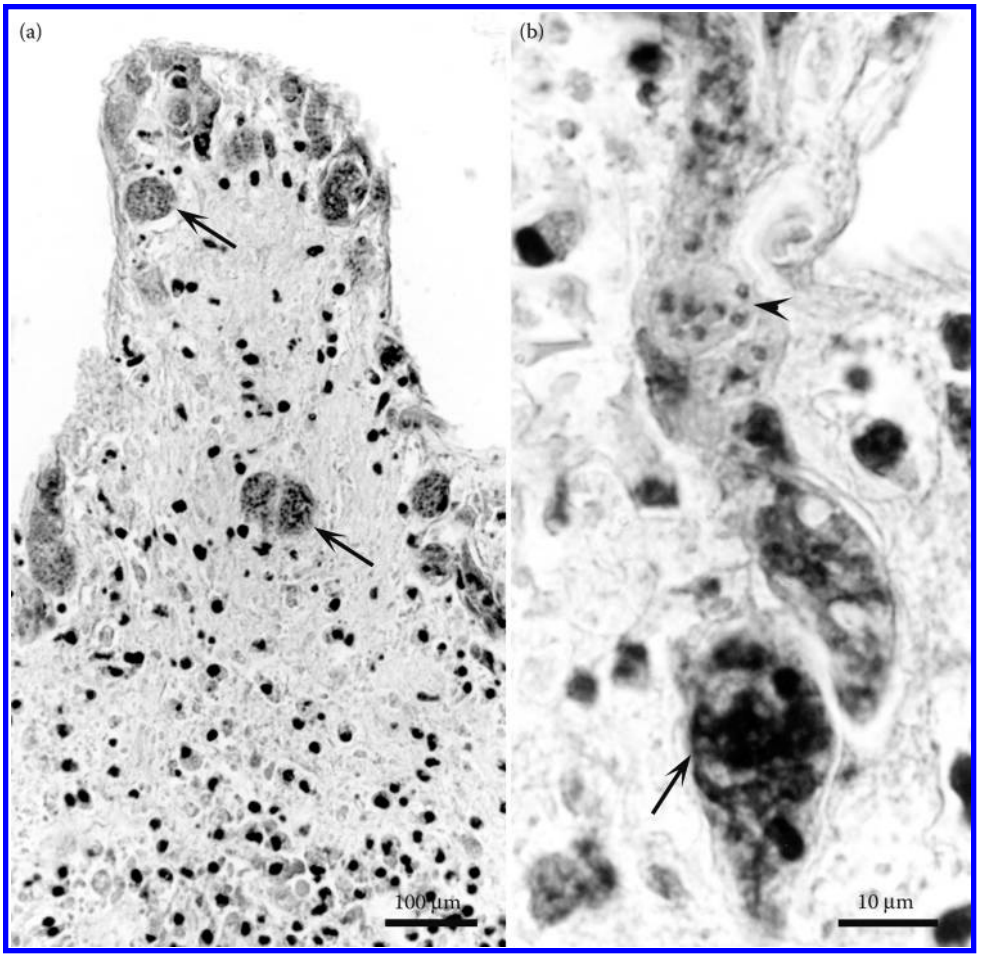


Figure 1.98 Placenta of an aborted cow naturally infected with *S. cruzi*. (a) Several schizonts (arrows) are located in the lamina propria of a villus exhibiting necrosis and infiltration of macrophages. (b) Higher magnification showing deeply stained immature schizont (arrow) and mature schizonts with merozoites (arrowheads). H and E stain.

Sarcocystis species using polyclonal antibodies can be problematic. For example, anti- *S. cruzi* bradyzoite polyclonal rabbit antibodies stains not only *Sarcocystis* species, including *S. neurona* and *S. canis* (see Figure 3.11), but also *T. gondii*.[724,726] Additionally, bradyzoites or bradyzoite extracts are often lethal for rabbits. However, species-specific *S. neurona* antibodies were prepared in rabbits using cell cultured merozoites.[420] Although specific monoclonal antibodies have been characterized against some *Sarcocystis* species, these are not available commercially for routine diagnostic use.

1.9 ECONOMIC LOSSES

Millions of dollars have been lost each year from condemnation or downgrading of meat containing grossly visible sarcocysts[241,591] but economic losses from clinical and subclinical infections are difficult to calculate because: (1) nearly 100% of cattle and sheep are infected, thus it is difficult to differentiate affected and nonaffected animals that are infected with microscopic sarcocysts; (2)

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no dollar values are available for the losses due to poor feed efficiency, failure to grow, reduced milk and wool production, reproductive problems, and obvious clinical disease; and (3) clinical disease is difficult to diagnose and has been recognized in relatively few outbreaks involving cattle in natural conditions. The wide prevalence of pathogenic species of *Sarcocystis* in domestic animals leaves no doubt that clinical disease could be recognized when better diagnostic tests are available. Economic losses due to *S. neurona* are discussed in Chapter 3.

1.10 TRANSMISSION

Ingestion of sporocysts and oocysts in food or water is the only major mode of transmission to the intermediate host. Although transplacental infection has been documented in cattle and sheep in nature by finding schizonts in some aborted fetuses, it is rare. It is possible that all transplacentally infected cattle and sheep die because *Sarcocystis* sarcocysts have never been found in newborn calves or in lambs. Sarcocysts has been found only once in a 3-day-old foal.[269] The possibility of lactogenic transmission via milk or colostrum was tested experimentally, but evidence of transmission was not found.[587]

The ingestion of mature sarcocysts (i.e., those containing bradyzoites) is the only known means by which the definitive host acquires *Sarcocystis* infection.

1.11 EPIDEMIOLOGY

Sarcocystis infection is common in many species of animals worldwide. Virtually 100% of adult cattle in the United States are infected with this parasite. Several conditions exist that permit such an unusually high prevalence of *Sarcocystis* in animals:

1. A host may harbor any of several species of *Sarcocystis*. For example, sheep may become infected with as many as 5 species, and cattle may have as many as 4 species of *Sarcocystis*.
2. Many definitive hosts are involved in transmission. For example, cattle sarcocystosis is transmitted via felids, canids, and primates. *S. cruzi*, the most common species of *Sarcocystis* of cattle worldwide, is transmissible via dogs, coyotes, foxes, wolves, and raccoons. Wild carnivores, such as coyotes, may travel many miles for food each day and spread the parasite over long distances.
3. *Sarcocystis* oocysts and sporocysts develop in the lamina propria and are excreted over a period of many months.
4. Sporocysts or oocysts remain viable for many months in the environment.[1160,1547] They can be further spread or protected by invertebrates.[1111,1625]
5. Large numbers of sporocysts can be excreted. For example, dogs ingesting a relatively small amount of beef (250 g) excreted 100–6000 sporocysts per gram of feces.[579] With an average fecal output of 250–350 g/day, a dog may excrete from 250,000 to over 2 million sporocysts per day. Coyotes and foxes are even more efficient producers of *S. cruzi* sporocysts. As many as 200 million sporocysts were recovered from the intestinal scrapings of a coyote that was fed 1 kg of naturally infected beef.[354] *S. cruzi* is not the only species that produces large numbers of sporocysts. As many as 90 million *S. tenella* sporocysts were recovered from the feces of a dog fed naturally infected mutton.[611]
6. There is little or no immunity to reexcreting of sporocysts.[579,801,1479] Therefore, each meal of infected meat can initiate a new round of production of sporocysts.
7. Oocysts or sporocysts are resistant to freezing and, thus, can withstand winter on the pasture.[95,173,352,575,811,1042,1918] They are also resistant to disinfectants.[79,1159] Apparently sporocysts can be killed by drying and by 10 min exposure at 56°C. Sporocysts can survive for months even at low humidity.[1547]
8. Unlike many other species of coccidia, *Sarcocystis* is passed in feces in the infective form and is not dependent on weather conditions for maturation and infectivity.

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Not all species of *Sarcocystis* are highly prevalent. Most species of *Sarcocystis* transmissible via cats have been found less frequently than those transmissible via canids. There may be several reasons for this. One reason may be that cats are very poor producers of *Sarcocystis* sporocysts. Cats fed several grams of macroscopic sarcocysts of *S. muris* and *S. gigantea* containing millions of bradyzoites produced relatively few sporocysts. Another reason may be that sarcocysts of feline-transmitted species, such as *S. gigantea* and *S. medusiformis*, require several months or years to become infective. Such species are not able to complete the cycle in young animals. Still another reason may be that some host species are inherently more susceptible to infection with some agents than are others. For example, the pronghorn (*Antilocapra americana*) is rarely found infected with *Sarcocystis*. Whether breeds of livestock species vary in susceptibility to *Sarcocystis* is not known, but certain strains of mice appear more susceptible to infection than others.[1486]

1.12 CONTROL

There is no vaccine to protect livestock against clinical sarcocystosis. However, experimental studies indicate that cattle, sheep, goats, and pigs can be immunized by small numbers of live sporocysts. Thus, there is hope of developing a vaccine for sarcocystosis in the future. For the present, prevention is the only practical method of control.

Excretion of *Sarcocystis* in the feces of definitive hosts is the key factor in the spread of *Sarcocystis* infection. Therefore, to interrupt this cycle:

1. Carnivores should be excluded from animal houses and from feed, water, and bedding for livestock.
2. Uncooked meat or offal should never be fed to domesticated carnivores. Freezing can drastically reduce or eliminate infectious sarcocysts in meat. Overnight freezing in a household freezer is effective in killing sarcocysts, based on the thickness of the meat.[672,722,1280,1486,1666] Exposure to heat at 55°C or higher temperatures for 20 min kills Sarcocystis.[242,574,972]
3. Dead livestock should be buried or incinerated. This is particularly important where dead animals are left in the field where carnivores can have access to the carcasses.
4. The prophylactic use of anticoccidials may be another practical method of controlling sarcocystosis in livestock.
5. Rodent bait containing lethal doses of *S. singaporensis* sporocysts have been suggested as a method to control rat populations[886,889] but this has not been approved for use in some countries; *S. singaporensis* is thought to be host-specific for rats.

1.13 CHEMOPROPHYLAXIS AND CHEMOTHERAPY

Several drugs routinely used as anticoccidials in poultry are effective against sarcocystosis in cattle, sheep, and goats, when administered continuously for a month, starting at or before inoculation with *Sarcocystis*.[576,813,1040,1041] Amprolium (100 mg/kg body weight) administered from 0 to 30 DPI, prevented acute disease or death in cattle infected with *S. cruzi* and in sheep infected with *S. tenella*; 50 mg/kg body weight of amprolium was less effective in sheep than the larger dose.

Calves treated with 100 mg/kg of body weight of amprolium from 21 to 35 DPI with 100,000 sporocysts had only mild signs of acute sarcocystosis, indicating that second-generation schizonts and any subsequent asexual stages preceding sarcocyst development were affected.[590]

Salinomycin (1 or 2 mg/kg body weight), administered from 1 to 30 DPI also prevented deaths in sheep with *S. tenella*. Halofuginone (0.22 mg/kg body weight) similarly reduced or prevented acute sarcocystosis in goats infected with *S. capracanis*, and in sheep infected with *S. tenella*; the drug was administered in feed from day 5 before inoculation and continued up to 36 DPI.[813,1797]

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Lasalocid, decoquinate, and monensin, each incorporated in feed at 33 mg/kg of feed, had minimal or no effect on the course of acute sarcocystosis in cattle inoculated with 250,000 *S. cruzi* sporocysts; the medicated food was given from day 7 before inoculation through 80 DPI.[618] For chemotherapy, only halofuginone has been found effective.[813,1797] Halofuginone (0.67 mg/kg body weight) given once or 4 times, prevented death in goats and sheep acutely ill with sarcocystosis. Once the clinical signs of acute sarcocystosis appeared, the administration of salinomycin, sulfadoxin and trimethoprim, lasalocid, robenidin, or spiramycin, did not prevent death in sheep and goats.[813,1041,1797]

Although oxytetracycline (30 mg/kg body weight), given intravenously, prevented death in 2 sheep with acute sarcocystosis, this drug may be too expensive.[813]

Most anticoccidials affect *Sarcocystis* schizonts. Results with *S. muris* infections in mice provide further insight. Of the 4 phases of infections examined, sporozoite migration (2–10 DPI), schizogony (11–17 DPI), merozoite migration (18–27 DPI), and sarcocyst formation (28–50 DPI), anticoccidials were most effective against schizogony.[1486] Of the 12 anticoccidials tested in the *S. muris* model, including all 4 phases of *S. muris* infection, amprolium, monensin, arprinocid, and sulfaquinoxaline plus diaveridine had no antisarcocysticidal activity, and lasalocid, halofuginone, sulfadoxine plus trimethoprim, and sulfadimethoxine had only marginal anti-*S. muris* activity, whereas Zoalene®, primaquine diphosphate, sulfaquinoxalin plus pyrimethamine, and Bay® G 7183 had excellent anti-*S. muris* activity. Of particular interest is the activity of sulfaquinoxalin plus pyrimethamine against immature and mature sarcocysts, because no other drugs are known to be effective against sarcocysts.[1486,1487] Results with the murine *S. muris* model also indicate that results with 1 species of *Sarcocystis* may not be directly applicable to other species of *Sarcocystis*. For example, amprolium was effective against *S. cruzi*, but not against *S. muris*.

Chemotherapy of the pathogenic species *S. neurona* is discussed in Chapter 3.

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CHAPTER 2

Techniques

2.1 EXPERIMENTAL INFECTION OF INTERMEDIATE AND DEFINITIVE HOSTS

Intermediate hosts are infected by feeding sporocysts that can be mixed in grain or suspended in a small volume at the back of the tongue to avoid spillage. The age of the animal does not appear to affect infectivity; 1-day-old calves, goats, or lambs, as well as older animals are easily infected with *Sarcocystis*. Inoculated animals should be kept in isolation for 10 days, and all the waste and bedding should be incinerated to kill sporocysts that might pass through the gut unexcysted and be excreted in the feces[136,381,1257] where they can be infectious to other animals.

Definitive hosts are infected by feeding intermediate host muscle tissue with mature sarcocysts containing bradyzoites; immature sarcocysts containing only metrocytes are not infectious. Time to maturity varies with each species of *Sarcocystis*. For example, in sheep, *S. tenella* and *S. arieticanis* become infectious within 3 months, whereas *S. gigantea* sarcocysts do not mature for more than 1 year or longer. The numbers of sarcocysts of some species (e.g., *S. miescheriana*, *S. capracanis*, *S. muris*) are reduced dramatically after 3 months.

2.2 ISOLATION, PURIFICATION, AND PRESERVATION

2.2.1 Bradyzoites

Bradyzoites can be released from sarcocysts by incubation in digestive fluid (trypsin or acid pepsin). The sarcocyst wall is dissolved by digestive fluid, but the released bradyzoites survive in it.[878] The following method[567,1087] is useful for obtaining *S. cruzi* bradyzoites:

a. Select heavily infected tissue by microscopic examination. The number of sarcocysts in different tissues may vary among animals; heart and tongue are the most heavily infected organs.
b. Remove as much connective tissue and fat as possible, and grind muscle in a meat grinder or mince into small pieces.
c. Suspend about 50 g of ground meat in 100 mL of warm (37°C) digestion fluid (pepsin, 2.5 g; concentrated HCl, 10 mL; water to make 1000 mL; dissolve pepsin in water, centrifuge to remove undissolved pepsin), and incubate at 37°C on a magnetic stirrer for approximately 10 min or longer. The objective is to release bradyzoites from sarcocysts that were broken during grinding, and this method is best suited for heavily infected tissues. Lightly infected tissues may have to be digested longer in acid–pepsin solution, and, in that case, the digestion fluid should be isotonic.[567]
d. Pour the homogenate through cheesecloth to remove large particles and centrifuge at 400*g* for 5–10 min using a floating-head centrifuge and 50-mL tubes.
e. Discard the supernatant fluid and suspend the sediment in Hanks' balanced salt solution (HBSS) at pH 7.4, preferably at 5–10°C.

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f. Recentrifuge at 400*g* for 5–10 min and discard the supernatant. The pellet has 2 layers, the whitish bradyzoite layer at the bottom and the brownish host tissue layer on the top. Remove host tissue by gently aspirating it from the whitish layer; this separation is not always possible.
g. To further clean zoites of host tissue, mix concentrated zoite suspension with isotonic Percoll® solution, one part of zoite suspension with two parts of Percoll. To prepare isotonic Percoll, mix Percoll with 9% NaCl solution (9 parts Percoll + 1 part NaCl).
h. Centrifuge Percoll-zoite suspension at 400*g* or more for 10 min. The bradyzoites settle at the bottom and host tissue floats in Percoll.
i. Pour off Percoll and wash bradyzoite pellet three times in HBSS by centrifugation.
j. Bradyzoites recovered by the above procedure remain viable.

2.2.2 Sporocysts

Sarcocystis sporocysts, after sporulating in the intestinal lamina propria, are normally released into the intestinal lumen over several months. To optimize the recovery of sporocysts:

a. Euthanize the definitive host 3–7 days after oocysts or sporocysts are first passed in the feces.
b. Remove the small intestine, cut it lengthwise, and spread over paper towels with the luminal side up.
c. Lightly scrape the intestinal epithelium with a glass slide, removing only the tips of the villi, where most sporocysts are concentrated.
d. Suspend scrapings in water and then homogenize in a blender for about 2 min at top speed.
e. Centrifuge the homogenate at 400*g* for 10 min.
f. Discard supernatant and suspend the sediment in water. Repeat the above process until most sporocysts are released from the host tissue.
g. Concentrate sporocysts by centrifugation, filter through a 25-µm stainless-steel sieve, suspend in HBSS, and centrifuge.
h. Suspend the pellets in HBSS–antibiotic mixture (given below).
i. Although most sporocysts are concentrated toward the tips of the villus, some are located deep in the villus. To obtain these sporocysts, deeply scrape the intestine and homogenize the intestinal scrapings in water.
j. Emulsify homogenate in 5.25% sodium hypochlorite solution (1:1 ratio) in a cold bath for 30 min.
k. Centrifuge at 400*g* for 10 min and discard supernatant.
l. Suspend the sediment in water and wash by centrifugation until the smell of chlorine is gone.
m. Suspend sporocysts in HBSS–antibiotic mixture (penicillin, 10,000 U; streptomycin, 10 mg; Fungizone®, 0.05 mg; and Mycostatin®, 500 U/mL [mixture = PSFM] of HBSS–PSFM mixture).
n. Sporocysts stored in PSFM solution at 4°C remain viable for 12 months or more (1 L of antibiotic solution can be prepared and stored at –20°C in 100-mL aliquots. Each aliquot is thawed as needed).
o. Sporocysts can also be obtained from feces of definitive hosts by flotation methods used for other coccidia.

2.3 DIAGNOSTIC TECHNIQUES

2.3.1 Examination of Feces for Sporocysts

The number of sporocysts in feces is usually low; therefore, concentration methods are often necessary to detect *Sarcocystis* in feces. Either sugar or salt (NaCl or $ZnSO_4$) solutions of specific gravity 1.15 or more can be used to float sporocysts free of fecal debris. Sugar solution is less deleterious to sporocysts than salt solutions, and has been used with the following method:

a. Mix 5–10 g of feces thoroughly in 50–100 mL of Sheather's sugar solution (sugar, 500 g; water, 320 mL; add liquid phenol, 6.5 mL if the solution needs to be stored for more than few days, to prevent fungal growth). To thoroughly dissolve sugar, heat water to 70°C and stir solution continuously,

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then cool before use. To obtain a homogenous suspension, the feces should be mixed well in about 5–10 mL of water before adding sugar solution.

b. Filter through cheesecloth.
c. Centrifuge fecal suspension in 50-mL centrifuge tubes with a cap at about 400*g* for 10 min.
d. With a pipette, remove several small drops from the very top, put drops on a glass slide and cover the drop with a coverslip.
e. Let the slide coverslip lie flat for about 5–10 min so that the fecal particles can settle and the sporocysts can rise to the top before the slide is examined.
f. Examine at magnification of ×100 or more. *Sarcocystis* sporocysts are small (usually 15 × 10 µm), thin-walled, and of lower density than most other parasite eggs or oocysts; therefore, they lie just beneath the coverslip and are often missed (Figure 2.1).
g. To collect sporocysts from feces, aspirate the top 5 mL of the solution from the 50-mL centrifuge tube. Mix the aspirate with 45 mL of water and centrifuge at 400*g* for 10 min. Discard the supernatant fluid and resuspend the sediment in water, centrifuge, and repeat the process. Suspend the final sediment in PSFM solution.



Figure 2.1 *Sarcocystis* sporocysts in sugar solution float of dog feces. (a) Sporocyst (arrow) and a roundworm egg, *Toxascaris leonina* (arrowhead). (b) Sporocyst on the right and an oocyst on the left. (From Streitel, R.H., Dubey, J.P., 1976. *J. Am. Vet. Med. Assoc*. 168, 423–424. With permission.)

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2.3.2 Examination of Muscles for Sarcocysts

Sarcocysts can be detected in muscles by gross inspection, examination of unstained squash preparations, histologic examination, or by a digestion method. Each of these methods has its advantages and disadvantages. Only a few species of *Sarcocystis* form macroscopic sarcocysts. Examination of unstained squashes is useful to study sarcocyst structure (Figure 2.2); by this method, one can examine relatively more tissue than by histologic examination. However, this method is unsuitable for fixed tissue. Histologic examination is necessary to study sarcocyst morphology, but this method is the least sensitive because only a small amount of tissue can be sampled.

Digestion of host tissue is the most sensitive method to detect light *Sarcocystis* infection. In the digestion procedure, host tissue (50 g) is incubated in 10 volumes of either 1% trypsin solution at pH 7.4 or HCl–pepsin solution for 1–4 h.[132,243,392,878,1775] The digest is examined for bradyzoites. By this method, one may detect even a few sarcocysts in 50 g of tissue because several hundreds or thousands of bradyzoites are released from sarcocysts as the host tissue and sarcocysts are digested. The bradyzoites can be used for antigen, and the species can be identified by molecular methods. Disadvantages of this method are: (1) it is not possible to distinguish species of *Sarcocystis* because the sarcocyst wall is dissolved; (2) metrocytes are not resistant to digestion, therefore, immature sarcocysts may not be detected; and (3) it may not be possible to detect sarcocysts with relatively small bradyzoites (5–6 μm). To obtain intact sarcocysts, we suggest the digestion of small amount of tissue for 8–10 min.[547,1896,2072]

The following method is used to isolate individual sarcocysts from infected pigs (Heydorn, A. O., personal communication):

a. Remove fat and connective tissue from 100 g of heavily infected swine muscles (102–110 DPI after experimental infection).
b. Grind it in a meat grinder and suspend ground meat in 1 L of digestion fluid (0.3% trypsin [Merck], 0.05% ethylenediaminetetraacetic acid [EDTA] in phosphate buffer saline [PBS], pH 7.4–7.6).

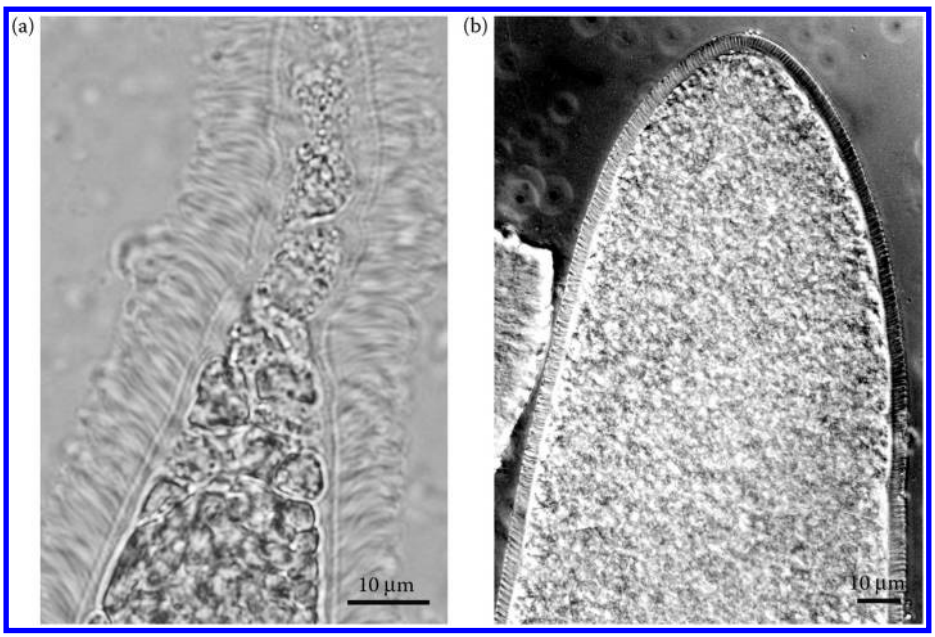


Figure 2.2 Sarcocyst walls in unstained wet preparations. (a) *S. cruzi.* Although the sarcocysts appear thin in H and E-stained sections, numerous elongated, hirsute protrusions are visible in this preparation. (b) *S. tenella* with striated wall.

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c. Stir gently at room temperature with magnetic stirrer for 45–60 min.
d. Sieve through a coarse metallic sieve (or tea strainer) and save the filtrate.
e. To stop digestion, add 1 L of PBS with 15 mL of fetal calf serum.
f. Sediment sample in a tall glass cylinder for 15–20 min. Decant and save 100 mL of the sediment. Refill with PBS, repeating sedimentation and decanting 3–4 times, using the PBS as the diluent.
g. Examine the sediment for intact and broken sarcocysts.

2.3.3 Serological Techniques (for *S. neurona* Diagnosis, See Chapter 3)

2.3.3.1 Preparation of Soluble Antigen from Bradyzoites

In IHA and ELISA tests, soluble antigen is prepared by the following method.[666]

a. Obtain bradyzoites as described and then freeze and thaw the suspension 3 times or more.
b. Suspend the bradyzoite suspension in Dulbecco's phosphate-buffered saline (DPBS).
c. Homogenize the bradyzoite suspension in a homogenizer (Polytron®, Brinkmann Instruments, Westbury, NY) at low speed until the zoites are disrupted. Let the bradyzoites stand at 4°C for 1–2 h to leach out soluble material.
d. Centrifuge at 16,000–20,000*g* for 30 min. Collect the supernatant fluid.
e. Dialyze the supernatant fluid overnight at 4°C against DPBS without added calcium and magnesium, using a dialysis membrane with a 6000- to 8000-M_r cut-off.
f. Filter and sterilize the dialysate through a 0.2-µm filter unit.
g. Estimate the protein concentration using a Bio-Rad® Coomassie blue dye binding assay or an alternative method.
h. Store the antigen in aliquots at –70°C.

2.3.3.2 Method for ELISA Test

Method initially used for *S. cruzi* with crude bradyzoite lysate antigen was reported earlier.[393] Newer methods of ELISA, using cell culture-derived merozoites antigens (whole or recombinant), are discussed in Chapter 3.

2.3.3.3 Immunohistochemical Staining

Formalin-fixed paraffin-embedded tissues can be used in this technique. Although parasite antigens can be detected even 1 year after fixation in 10% formalin, fixation for short periods (24 h) is recommended. Sections are cut 3–5 µm thick. It is necessary to use grease-free clean slides, otherwise the sections detach from the slide; also, an addition of chrome-gelatin in the water bath helps adherence of sections to the slide. To avoid mortality due to toxicity of bradyzoites, rabbits can be infected with merozoites or oocysts; the latter are preferred because oocysts can be cleaned of fecal matter and treated with 5.25% sodium hypochlorite solution (Clorox®) to remove host material.

We carry out the following procedure:

a. De-paraffinize slides in old xylene I for 30 min. Discard xylene I. Pour xylene II into xylene I container. Soak 5 min.
b. Pour fresh xylene into xylene II. Soak slides in fresh xylene for 5 min.
c. Soak in 100% ethanol I for 5 min. Discard ethanol I. Pour ethanol II into ethanol I container. Soak 5 min. Pour fresh 100% ethanol into ethanol II container. Soak another 5 min.
d. Soak in 95% ethanol for 5 min.
e. Quench endogenous peroxidase in 3% H_2O_2 in methanol for 15 min. (20 mL 30% hydrogen peroxide and 180 mL methanol).

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f. Soak in 80% ethanol for 5 min.
g. Soak in 70% ethanol for 5 min.
h. Soak in 50% ethanol for 5 min.
i. Soak in saline for 5 min.
j. Soak in warm saline (37°C) for 5 min.
k. Pepsin digestion: incubate slides in 0.4% pepsin in 0.01 N HCl for 15 min at 37°C. (0.8 g pepsin, 0.333 mL 6 N HCl, 200 mL saline, prewarmed to 37°C for 30 min).
l. Soak in 0.75% Brij-PBS for 5 min twice (2.5 mL 30% Brij-35 per liter PBS).
m. Block nonspecific binding with 0.5% Na caseinate in Brij-PBS for 10 min.
n. Drain and, without rinsing, apply the properly diluted primary antibody to the tissues for 30 min at 37°C.
o. Soak slides in Brij-PBS for 5 min.
p. Treat tissues with Dako EnVision Rabbit peroxidase solution® at 37°C for 30 min.
q. Soak slides in Brij-PBS for 5 min twice.
r. Apply Dako AEC substrate chromogen solution® to slides at 37°C for 15 min.
s. Collect chromogen in hazard waste bottle, and soak slides in Brij-PBS for 5 min.
t. Rinse slides in running tapwater for 2 min. Rinse in deionized water for 2 min. Twice.
u. Counterstain with Mayer's hematoxylin for 1 min.
v. Wash slides in running tapwater for 1 min.
w. Soak slides in Scott's tapwater substitute ($MgSO_4 7H_2O$ 20 g, $NaHCO_3$ 2 g, water 1 L) for 1 min.
x. Soak in running tapwater for 1 min. Soak in deionized water for 2 min. Twice.
y. Apply Crystal Mount® and incubate slides overnight at room temperature.
z. Apply Permount® and coverslip, if desired.

2.4 *IN VITRO* CULTIVATION

The first stages to be grown *in vitro* were sexual stages[86,569,571,1177] (Table 2.1). Only a few species of *Sarcocystis* have been grown *in vitro.* After that, an *in vitro* system was developed to grow large numbers of merozoites of several species of *Sarcocystis* that infect livestock.[1655,1656] The development of merozoites to sarcocysts in cultured cells has not yet been reported.

Two factors determine the success of *in vitro* growth experiments. First, a large supply of sterile stage-specific parasites (i.e., sporozoites, merozoites, or bradyzoites) must be readily available. Second, the selection of the cell line to be tested for its ability to support growth of the parasite should be based on the cell or tissue in which the parasite develops *in vivo.* For example, if schizonts develop *in vivo* in mouse hepatocytes, then a mouse epithelioid liver cell line should be tested. Because other coccidia have been shown to readily develop in some specific cell types, such as kidney cells, even though they do not normally develop in such cells *in vivo*,[1655] the literature on *in vitro* cultivation of coccidia should serve as a guide for possible cell types to test for *in vitro* growth of *Sarcocystis* spp.

Table 2.1 *In Vitro* Culture of *Sarcocystis* spp. Gamonts

Species	Host	Type of Cells Successful	Cells Unsuccessful	Reference
S. muris	Mouse	MDCK, FE, MF	Not stated	**539, 544**
S. muris	Mouse	CL, DK	HF, SK	**86**
S. suihominis	Pig	HB	CL, DK, SK	**86**
S. tenella	Sheep	DK	HF, SK, CL	**86**
S. capracanis	Goat	DK	HF, SK, CL	**86**
S. sp.	Grackle	MDCK, EBK, EBTr, ETK, ECM	ECK	**569, 571**
S. suihominis	Pig	HEI, HSF	–	**1177**

Abbreviations: MDCK = Madin–Darby canine kidney, FE = feline embryo, CL = cat lung, DK = dog kidney, SK = swine kidney, MF = mouse fibroblasts, HF = human fibroblasts, EBK = embryonic bovine kidney, EBTr = embryonic bovine trachea, ECK = embryonic chicken kidney, ECM = embryonic chicken muscle, ETK = embryonic turkey kidney, HEI = human embryonic intestine, HSF = human skin fibroblasts.

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2.4.1 Excystation and Cultivation of Schizonts (for *S. neurona* Cultivation, See Chapter 3)

One of the most important preliminary steps in achieving *in vitro* development of sporozoites to merozoites is to obtain a relatively high rate of sporozoite excystation from sporocysts. After the sporocysts have been isolated from the definitive host, purified, and concentrated, they can be stored until used at 4°C in HBSS with antibiotics.[1039] Several factors are important in storing and successful excystation of sporocysts, and these conditions may differ with different species of *Sarcocystis*. For example, *S. cruzi* sporocysts should not be stored in aqueous $K_2Cr_2O_7$, which is routinely used to store coccidian oocysts, because it has a deleterious effect on excystation.[176,1039] McKenna and Charleston[1159] reported no excystation of *S. gigantea* sporocysts after 5 day exposure in 2% sulfuric acid, compared with 85% excystation by storage in tapwater and 15% excystation after storage in 2.5% $K_2Cr_2O_7$.

Pretreatment of sporocysts with NaOCl helps remove contaminating microorganisms from sporocysts and in subsequent excystation. Traditionally, 5.25% NaOCl (available commercially as undiluted laundry bleach) has been used for this purpose, and the treatment was done in an ice bath to avoid the deleterious effect of heat generated during the procedure. Horn et al.[846] reported higher efficiency of excystation of *S. gigantea*, *S. tenella*, *S. arieticanis*, and *S. hircicanis* by using 6%–8% NaOCl at room temperature for 20 min instead of an ice bath. The NaOCl treatment removes the outer lipid layer of the *Sarcocystis* sporocysts. Incubation in 1%–3% formic acid for 24–72 h at 4°C was reported to enhance excystation.[2282] The mechanism of excystation of *Sarcocystis* species is not fully understood. Trypsin, required for excystation of the *Eimeria* species, is not necessary for *Sarcocystis*, whereas bile is important.[573] Also, different concentrations (5%–15%) of bovine bile have been used for excystation of sporocysts.

The following method has proven useful for excysting sporozoites from sporocysts of *S. cruzi*, *S. capracanis*, and *S. tenella*:

a. Treat the sporocysts with 2.6% (v/v) NaOCl at 4°C for 30 min (i.e., Clorox, Purex®) to kill other microorganisms that would be certain to contaminate the *in vitro* cultures. A significantly greater excystation rate can be obtained by treating the sporocysts of *S. capracanis*, *S. cruzi*, and *S. tenella* with 2.6% NaOCl than with 1.3% or 5.3% NaOCl.[1039] Treatment of the sporocysts with cysteine HCl for 18 h may also improve the rate of excystation.[1039] Cysteine HCl and NaOCl evidently have similar beneficial effects on excystation, but NaOCl acts more rapidly (30 min vs. 18 h), and it must be used anyway to disinfect the sporocysts.
b. Wash the sporocysts several times (usually at least four times) by centrifugation and resuspension in cold saline or culture medium (i.e., RPMI 1640 without serum) until the odor of NaOCl can no longer be detected or until the pH color indicator in the culture medium remains unchanged in the presence of sporocysts.
c. Suspend the sporocysts in excysting fluid and incubate at 37°C for 1.5–4 h. The excysting fluid should consist of a balanced salt solution (i.e., HBSS, pH 7.2–7.4), containing 2% (w/v) trypsin or chymotrypsin (activity of 1:250 or 1:300) plus 5%–10% bile or 2.5%–5% (w/v) bile salt (i.e., sodium taurocholate obtained from the appropriate intermediate host such as bovine, ovine, etc.).
d. Remove the excysting fluid by washing the excysted sporozoites at least twice in the culture medium without serum, and resuspend in 5–10 mL culture medium.
e. To remove debris and sporocyst walls, the sporozoites may be passed through a nylon-wool column.[1026] Too much debris and sporocyst walls in the inoculum may have a deleterious effect on parasite development.[1655]
f. Count the number of sporozoites with a hemacytometer and resuspend them at the appropriate concentration in the culture medium containing 5% (v/v) fetal bovine serum, 2 mM *l*-glutamine, 50 µg/mL dihydrostreptomycin, and 50 U/mL penicillin G.
g. Inoculate 2 to 5×10^4 sporozoites per square centimeter of the monolayer of the cell line to be tested and incubate at 37–39°C in 5% CO_2. Sporozoites of *S. cruzi*, *S. capracanis*, and *S. tenella* will develop in bovine pulmonary artery endothelial cells (CPA; American Type Culture Collection,

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Rockville, MD) or bovine monocytes (BM; see Reference **1653** for details concerning the BM). The CPA cell line will produce approximately seven times more merozoites of *S. cruzi* than the BM line,[1657] but the BM line is especially useful for investigating immunological parameters such as opsonizing antibodies and lymphokines.[1653]

h. Remove the culture medium on day 1, and then at 2- to 7-day intervals after sporozoite inoculation, replace it with a culture medium containing 1% or 2% fetal bovine serum (FBS).
i. The infected cell cultures might require maintenance for long periods to allow sufficient time for the parasites to undergo one or more generations of schizogony. Long cultivation periods can be achieved by varying the FBS concentration in the culture medium. As the cell cultures become sparsely populated due to parasite development (usually first observed at 24–28 DPI), then treat them with cell culture medium (CM) containing 10%–20% FBS, which can then be reduced to 1%–2% FBS once the monolayer has reestablished itself.
j. Parasite development can be monitored and photographed at various intervals after sporozoite inoculation by direct microscopic observation with an inverted phase-contrast microscope or by examining double-coverslip preparations[1373] with a compound phase-contrast microscope.

Schizonts of only a few species of *Sarcocystis* have been grown in cell culture, and the selection of cell type for culture is an important consideration (Table 2.2). Schizonts of *S. capracanis*, *S. cruzi*, and *S. tenella* develop similarly *in vitro*, except that *S. cruzi* produces considerably more merozoites per culture vessel than do *S. capracanis* or *S. tenella*.[1656,1657] *S. cruzi* and *S. tenella* develop to schizonts in BM and CPA cells, whereas *S. capracanis* develops in BM only. Soon after inoculation, sporozoites of all three species penetrate cells (Figure 2.3a) and gradually transform in approximately 2–3 weeks into ovoid or spheroidal schizonts with irregularly shaped nuclei, with small nucleoli, and small cytoplasmic granules (Figure 2.3b and c). More advanced stages appear to have numerous nuclei and nucleoli (ultrastructural studies

Table 2.2 Species of *Sarcocystis* Schizonts Cultivated *In Vitro*

Host	Species	Type of Cells Successful	Cells Unsuccessful	Schizonts Days	Reference
Ox	*S. cruzi*	BM, CPA	Mouse macrophages	18–1,320	**31, 1657, 1659**
Ox	*S. hirsuta*	BPE	BM	14–62	**177**
Rat	*S. singaporensis*	Rat brain endothelial cells, rat pneumocytes	Hepatoma, fibroblats, myoblasts	3–18	**887, 891**
Sheep	*S. tenella*	BM, CPA	MDBK, OM	14–50	**1656**
Goat	*S. capracanis*	BM	MDBK, OM, CPA	60–100	**1656**
Budgerigar-*Didelphis virginiana*	*S. falcatula*	BT	–	3–4	**1070**
Budgerigar-*Didelphis-albiventris*	*S. falcatula*	BM	–	7	**418**
Budgerigar-*Didelphis-marsupialis*	*S. falcatula*	CV-1, BT	EK	4	**441**
Budgerigar-*Didelphis-albiventris*	*S. falcatula*	BT, Vero	ED, Hep-2	4–6	**510**
Budgerigar-*Didelphis-albiventris*	*S. lindsayi*	EK, BT, CV-1	–	2–3	**425**
Mouse-*Didelphis virginiana*	*S. speeri*	BM, EK	CV-1	3	**423**
Mouse-*Didelphis marsupialis*	*S. speeri*	BT	–	15	**427**

Abbreviations: BM = bovine monocytes, BPE = bovine pulmonary endothelial, BT = bovine turbinates, CPA = cardiopulmonary endothelial cells, CV-1 = African green monkey kidney, ED = equine dermal, EK = equine kidney, Hep-2 = human hepatocytes, MDBK = Madin–Darby bovine kidney.

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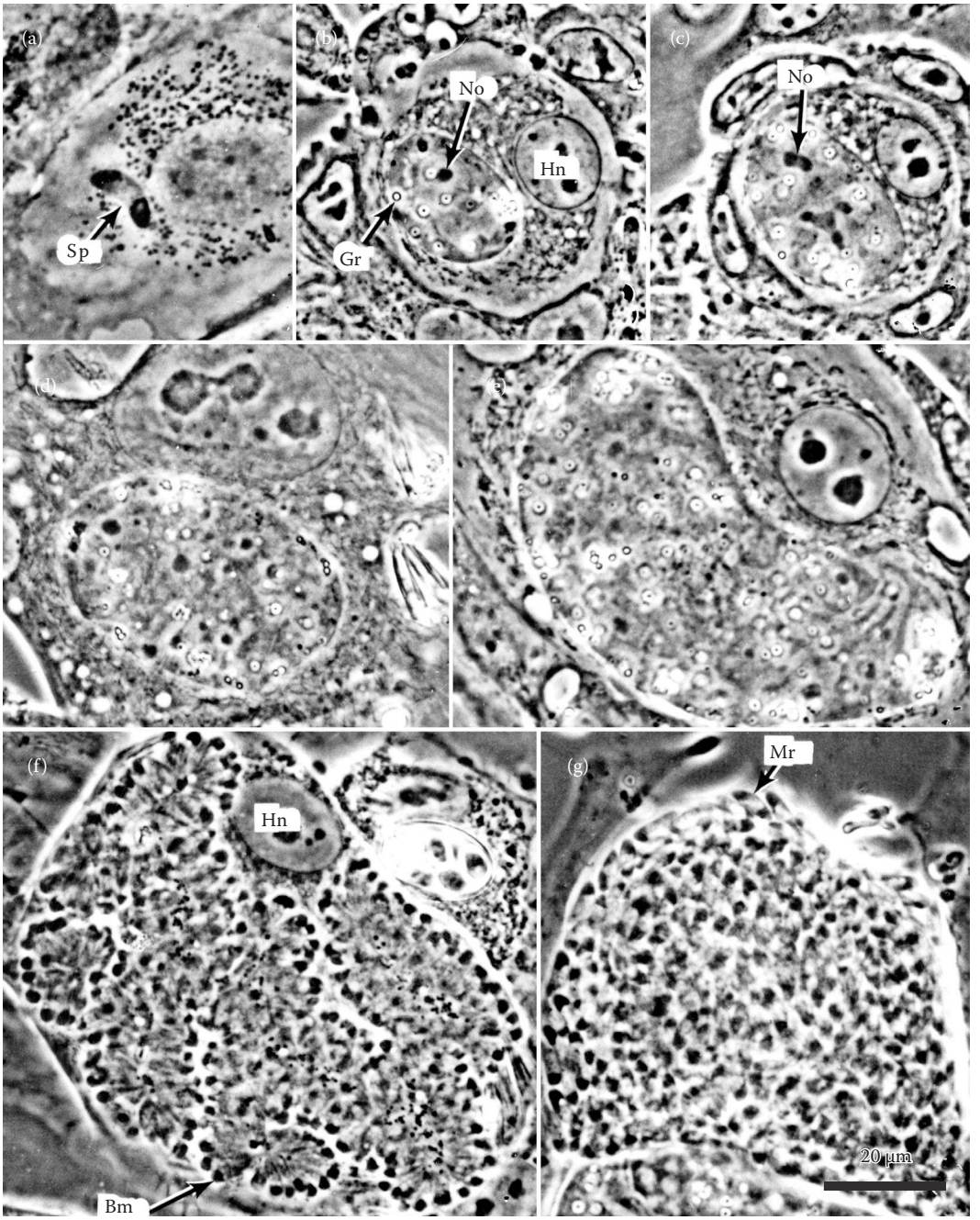


Figure 2.3 Phase-contrast photomicrographs of the development of sporozoites to large schizonts of *S. cruzi* in cultured bovine monocytes (a, f, g) and CPA cells (b–e). Day after sporozoite inoculation is given in parentheses. (a) Intracellular sporozoite (24). (b) Young schizont (18). (c) Young schizont with irregular nucleus and nucleoli (18). (d) Small intermediate schizont (23). (e) Large intermediate schizont (42). (f) Large schizont with budding merozoites (35). (g) Mature large schizont (35). (From Speer, C.A. et al., 1986. *J. Parasitol.* 72, 677–683. With permission.)

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have shown that the nucleus is highly lobulated, but by light microscopy the lobes appear as separate nuclei) (Figure 2.3d and e). Eventually, these schizonts form large structures in which merozoites bud radially from 8 to 12 residual bodies (Figure 2.3f). Large mature schizonts contain 150–350 merozoites (Figure 2.3g) and may be present at 2.5–10 weeks or more after sporozoite inoculation. Merozoites frequently glide rapidly through the host-cell cytoplasm, escape through the host-cell plasmalemma, and some of these immediately penetrate and glide through the cytoplasm of cells in the vicinity of the original host cell. Within a few minutes after being penetrated by 1 or more merozoites, these previously uninfected cells retract their cellular processes, become spheroidal, and some detach to float freely in the culture medium. Spheroidal host cells that remain attached transform within a few hours to flattened, epithelioid cells. Some of the merozoites that enter other cells or remain within the cytoplasm of the original host cell develop into small schizonts (Figure 2.4a–d).

Small schizonts contain 36–100 merozoites and may be present at 16–138 days or more after sporozoite inoculation, but are most numerous at 30–52 days. Several generations of small schizonts

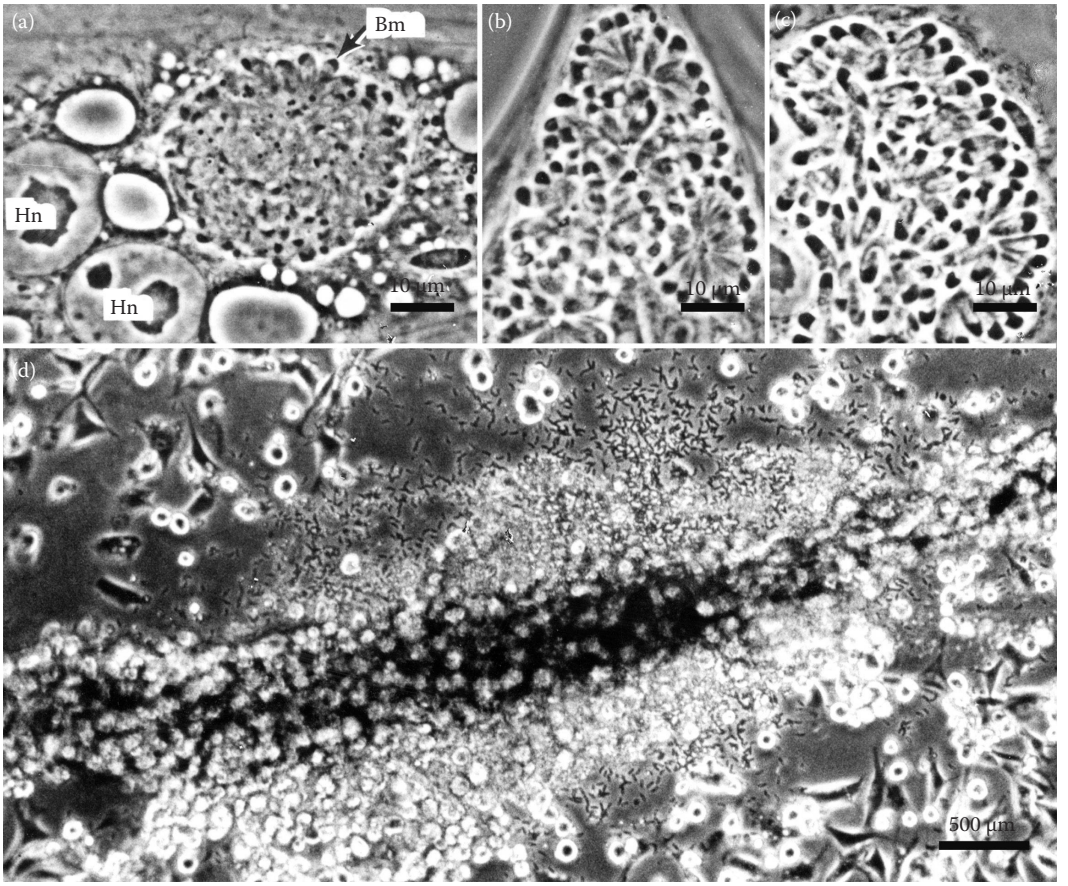


Figure 2.4 Phase-contrast photomicrographs of schizonts and merozoites of *S. cruzi* in cultured CPA cells. Day after inoculation of sporozoites given in parentheses. (a) Merozoites in early stage of budding at surface of schizont (42). (b) Schizont with merozoites budding from four residual bodies (48). (c) Mature schizont (42). (d) Large area of CPA culture showing numerous infected cells and extracellular merozoites (76). ([a–c] From Speer, C.A. et al., 1986. *J. Parasitol.* 72, 677–683; [d] Speer, C.A., Burgess, D.E., 1987. *Parasitol. Today* 3, 2–3. With permission.)

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occur during this time, but the precise number of generations cannot be determined because schizogony is asynchronous. Merozoites bud radially from 4 to 8 residual bodies (Figure 2.3a and b), are highly motile, easily exit the host cell, exhibit pivoting and gliding movements, and readily penetrate other cultured cells in the immediate vicinity. Noninfected CPA cells adhere to CPA cells that are infected with small schizonts to form aggregates of cells, and some of these aggregates reorganize into branching and, occasionally, anastomosing capillary-like structures, which become centers of intense schizont activity and merozoite release. Cells that adhere to infected cells serve as host cells for additional schizogonous generations. Eventually, relatively large, elongated aggregates of cells (Figure 2.4d) are formed, within which nearly all cells are infected with schizonts and merozoites (Figure 2.5). Production of *S. cruzi* merozoites cycles during the cultivation period; peak merozoite numbers are present at 36 and 44 DPI and at 44 and 50 DPI for CPA and BM, respectively.[1657]

The host-cell type *in vivo* for large and small schizonts of *S. cruzi*, *S. capracanis*, and *S. tenella* has been considered, but not proven, to be vascular endothelial cells. The recent success in culturing merozoites *in vitro* indicates that the preferred host cell *in vivo* may indeed be an endothelial cell, because approximately 7–12 times more merozoites of *S. cruzi* and *S. tenella* were harvested from CPA than from BM.[1656,1657] In contrast, *S. capracanis* underwent schizogony in BM, but not CPA cells.[1656]

Ultrastructural studies of infected BM and CPA cells revealed that sporozoites, schizonts, and merozoites of *S. cruzi* were located free in the host cell cytoplasm (i.e., parasites were not surrounded by a parasitophorous vacuole)[1655,1658] (Figures 2.5 and 2.6), which substantiates this finding in earlier studies of *in vivo* infections with *S. cruzi* as well as other species of *Sarcocystis*.[356,1649] Certain endothelial cells are known to be part of the reticuloendothelial system and are capable of phagocytosing and digesting ingested matter and microorganisms. If such reticuloendothelial cells serve as host cells for schizonts of certain *Sarcocystis* species, being located free in the host cell cytoplasm may represent a means of escaping a potentially hostile host-cell environment.

This system for culturing merozoites of *Sarcocystis* species[356,1649] can be particularly useful in elucidating the biochemistry, as well as the mechanisms, of immunity and pathogenesis, especially because the parasite develops *in vitro*, as well as *in vivo*, in cell types (i.e., endothelial and mononuclear cells) that have been shown to participate in other immunologic and pathologic mechanisms.[1402,1634,1635] With minor modifications (i.e., selecting the appropriate cell line), this system may prove useful for culturing other species of *Sarcocystis* that are known to develop slowly within endothelial or endothelial-like cells in the intermediate host.

2.4.2 Cultivation of Gamonts

Fayer[569,571,2005] was the first to describe the *in vitro* development of bradyzoites of *Sarcocystis* to microgamonts, macrogamonts, and oocysts (Figure 2.7c and d), which was also the first description of such stages in the life cycle of *Sarcocystis.* Becker et al.[86] were able to achieve even greater *in vitro* development of this phase of the *Sarcocystis* life cycle, in which the oocysts of four species of *Sarcocystis* underwent sporulation in various mammalian cell lines. Gamonts of several other species have been grown in cell culture.[537,539,541,898,1177]

The ultrastructure of gametogony, oogony, and sporogony has been described for several species of *Sarcocystis* developing in cell cultures.[86,1177,1790] The ultrastructural features of gamonts and oocysts of five different species of *Sarcocystis* formed in cultured cells were essentially identical to those formed in the intestines of the natural hosts. These studies showed that bradyzoites developed directly into microgamonts and macrogamonts, usually within 18 h after inoculation. Each microgamont had a large, irregularly shaped nucleus, which gave rise simultaneously to 20–30 microgametes. At 22 h after inoculation, oocysts began to undergo sporulation to form sporocysts.

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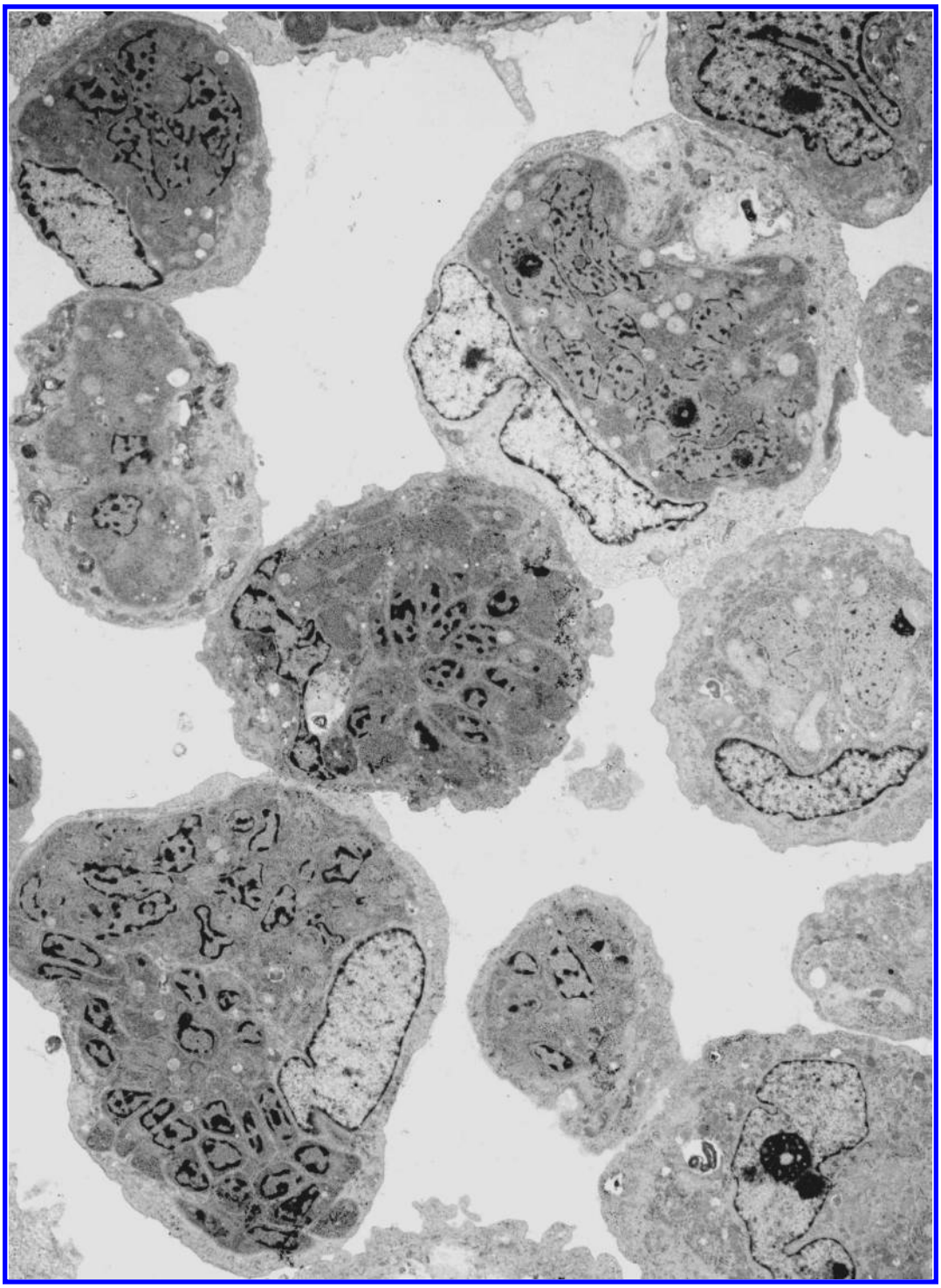


Figure 2.5 TEM of several CPA cells infected with schizonts of *S. cruzi* in various stages of development. 78 DPI.

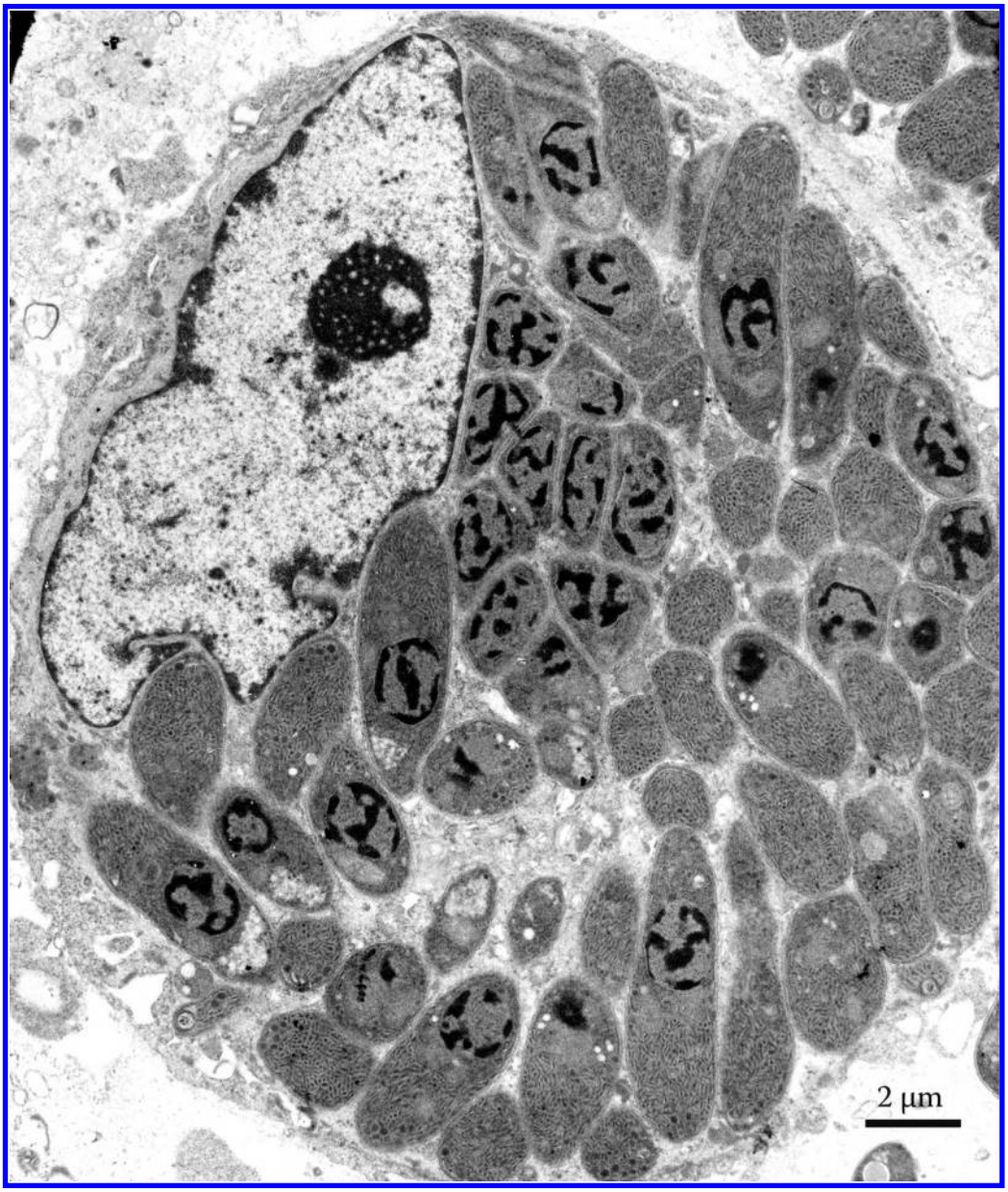


Figure 2.6 TEM of a mature schizont with merozoites of *S. cruzi* in a bovine CPA cell 35 days after sporozoite inoculation. (From Speer, C.A., Burgess, D.E., 1987. *Parasitol. Today* 3, 2–3. With permission.)

2.5 MOLECULAR METHODS

2.5.1 PCR from Tissues and Purified Tissue Cysts

Parasite DNA can often be extracted from infected host tissues, in quantities sufficient for successful PCR, without first isolating tissue cysts. This direct method will be most successful when infections are heavy, and when the tissue is fresh, or frozen before extraction.

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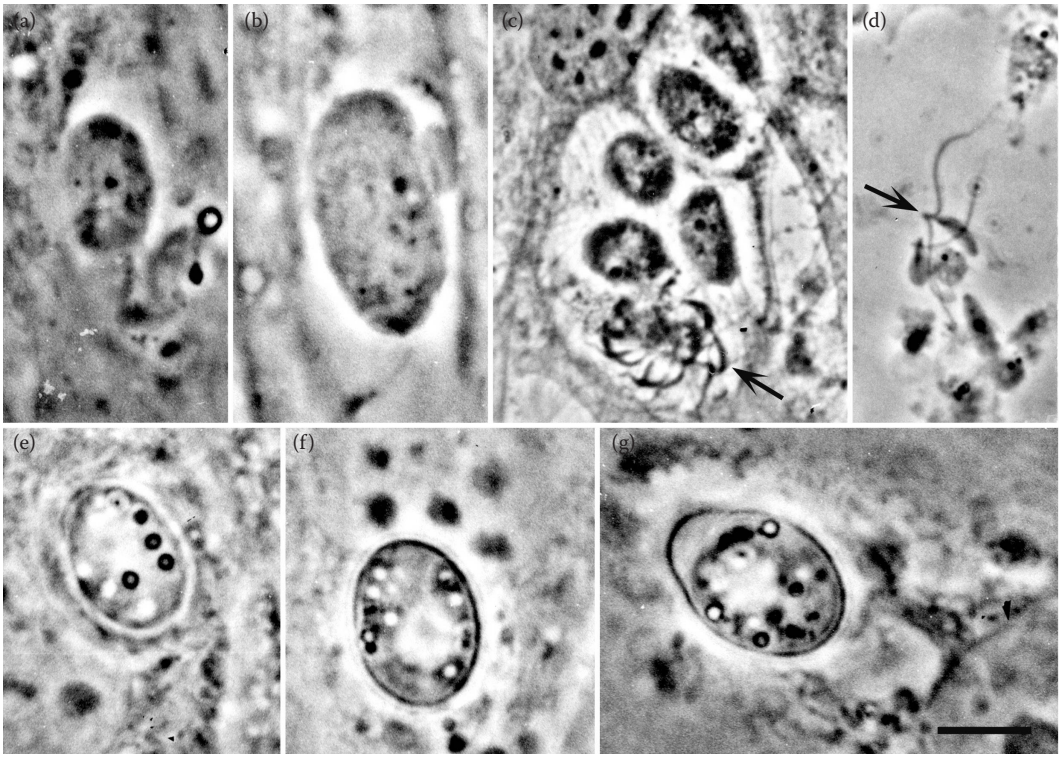


Figure 2.7 Phase-contrast photomicrographs of gamonts and oocysts in cultured cells. (a, b, e, f, g) Gamonts and oocysts of *S. muris* in mouse kidney cells. (c and d) Gamonts of *Sarcocystis* sp. from grackles in embryonic bovine trachea cells. Arrowheads point to host-cell nuclei. (a) Early microgamont. (b) Advanced microgamont. (c) Mature microgamont (arrow) and five macrogamonts. (d) Microgamete (arrow) with two flagella. (e) Macrogamont with four refractile granules (arrow). (f) Unsporulated oocyst with oocyst wall (arrow). (g) Unsporulated oocyst with slightly condensed sporont. (From Fayer, R., 1972. *Science* 175, 65–67. With permission.)

Purer preparations, which may be necessary in the case of light infections or when downstream applications may be impaired by contamination with host DNA, can be obtained by first isolating sarcocysts from the surrounding host tissue. Proteins and nucleic acids can be effectively extracted from mechanically isolated sarcocysts or by digestion in solutions of either trypsin or pepsin, as indicated earlier.

2.5.2 Preservation of Tissues That Cannot be Immediately Processed

When DNA cannot be extracted immediately, freezing at −20°C can safeguard the tissue. Preservation in 70%–95% ethanol can be used, but this diminishes subsequent success, and even residual ethanol has a low flashpoint, rendering such tissues hazardous for transport. Immersion in RNALater or Trizol reagent can help maintain nucleic acids until they can be extracted in the laboratory.

2.5.3 Options for Transporting DNA Prior to Analysis

Aqueous extracts of DNA should be kept cold before being analyzed, including during shipping. Drying a DNA pellet or applying an aqueous DNA solution to FTA (Whatman®) cards

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(impregnated with DNAse inhibitors) enable DNA to be transported at ambient temperatures (at significant cost savings). In any event, templates should not be subjected to x-ray or direct sunlight.

2.5.4 Extraction of DNA from Sporocysts

DNA can be successfully extracted from sporocysts.[1844] Clean sporocysts can be stored on filter paper in Eppendorf tubes submerged in 500 μL distilled water at –20°C before DNA extraction.

The quality of DNA obtained depends upon the purity of sporocysts and the method used to extract. It is often difficult to obtain DNA from intact sporocysts. Excystation of sporozoites facilitates DNA extraction because sporozoites, as opposed to intact sporocysts, are easily solubilized. Several methods have been used to excyst *Sarcocystis* sporozoites as explained earlier in Section 2.4.1.

Sporocysts can be crushed between the coverslip and glass slide to release sporozoites from sporocysts. DNA can then be extracted via a variety of methods; phenol–chloroform extraction methods, and the Invitrogen Easy-DNA kit® (using the tissue and blood protocol, as specified by the manufacturer) routinely yield success.

2.5.5 Freeze–Thaw Methods to Free Sporocysts from the Oocyst

Liquid nitrogen may be employed to accomplish cycles of freezing and thawing, and some success has been reported by using a "bead beater" to break the oocyst wall (although damage to DNA can also be expected when doing so). The methods used to extract DNA from other coccidian oocysts/sporocysts are applicable to *Sarcocystis*. The method for *Toxoplasma gondii* oocyst DNA extraction was published previously.[464]

Two rounds of PCR were needed to obtain sufficient product to visualize restriction fragment-length polymorphisms (RFLP) in ethidium bromide-stained agarose gels.[1844,1858] Less product might suffice for other downstream purposes, such as sequencing or visualizing by more sensitive means. In each round of PCR, primer 2L was employed: 5′-GGA TAA ACC GTG GTA ATT CTA TG 3′. The reverse primer in the first round (2H) is 5′-ACC TGT TAT TGC CTC AAA CTT C, and the reverse primer in the second round (3H) is 5′ GGC AAA TGC TTT CGC AGT AG.

Genes used to characterize *Sarcocystis* species are given in Tables 23.1 and 23.2. Additionally, genes/methods used to distinguish *Sarcocystis* species for each host or group of hosts are indicated in Chapters 3 through 22.

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CHAPTER 3

Unique Multiple-Host *Sarcocystis* Species

3.1 INTRODUCTION

Most *Sarcocystis* species have a 2-host life cycle as explained in Chapter 1. Therefore, *Sarcocystis* infections are discussed by hosts or group of hosts. However, 2 species, *S. neurona* and *S. canis* have a wide host range and their biology is slightly different than other *Sarcocystis* species. *S. neurona* was discovered in 1991 after the publication of the first edition of this book. Since then, more than 300 papers have been published on this subject. The following information is drawn largely from 2 reviews.[438,475]

3.2 *SARCOCYSTIS NEURONA* INFECTIONS IN ANIMALS

Sarcocystis neurona Dubey, Davis, Speer, Bowman, de Lahunta, Granstrom, Topper, Hamir, Cummings, and Suter 1991.

3.2.1 Introduction and History

An equine clinical syndrome was referred by 3 different names in the 1970s—"segmental myelitis"[1491,2123] "Equine Protozoan Encephalomyelitis,"[87] and "Equine Protozoal Myeloencephalitis (EPM)"[1146]; the term EPM has been used by most authors. The etiology of EPM was not ascertained until 1991, when the protozoan was cultivated and named *Sarcocystis neurona*[404]; historical developments are summarized in Table 3.1. An EPM-like disease has also been recognized in several other hosts.

3.2.2 Biology

3.2.2.1 Hosts

S. neurona has a wide host range relative to other species in the genus *Sarcocystis* (Figure 3.1, Table 3.2). The North American opossum (*Didelphis virginiana*) and the South American opossum (*D. albiventris*) are its known definitive hosts; whether *S. neurona* can infect other species of South American opossums continues to be unknown. Several other animal species are its intermediate or aberrant hosts (Figures 3.2 and 3.3, Table 3.2). In some hosts, only schizonts have been identified with certainty, and these are considered aberrant hosts. Intermediate hosts are those in which sarcocysts mature. Laboratory-raised opossums excreted sporocysts after feeding naturally infected skunk, armadillo, raccoon, and sea otter muscles,[207,440,445,1723,2390] confirming that these are proven intermediate hosts. One study[1108] reported brown-headed cowbirds as

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Table 3.1 History of *S. neurona* and Equine Protozoal Myeloencephalitis (EPM)

Contribution	Reference
1. Clinical syndrome first recognized without a defined etiology	**1491**
2. Protozoa first identified in lesions	**270, 88, 348**
3. EPM defined as a disease	**87, 1146**
4. Antiprotozoal chemotherapy introduced	**87, 1146**
5. Protozoa causing EPM considered to be a *Sarcocystis* species	**349, 1611**
6. Protozoa first isolated in cell cultures and named *Sarcocystis neurona*	**404**
7. *In vitro* development described	**307, 308**
8. *Sarcocystis neurona* Western blot developed for diagnosis	**728**
9. PCR primers introduced for diagnosis and molecular characterizations	**600, 729**
10. Opossum (*Didelphis virginiana*) proposed as the definitive host for *S. neurona*	**601**
11. Clinical syndrome simulating EPM induced in horses by feeding sporocysts from opossums	**603**
12. *Sarcocystis neurona* separated from *S. falcatula* based on susceptibility of immunodeficient mice to *S. neurona*	**412, 1114, 1115**
13. Opossum proven to be definitive host by inducing EPM-like disease in immunodeficient mice fed sporocysts	**412**
14. *In vitro* and *in vivo* testing of chemotherapeutic agents begun	**439, 1068**
15. *Sarcocystis neurona* isolated from CNS of a non-equine host	**1072**
16. South American opossum (*Didelphis albiventris*) from Brazil found to be another definitive host for *S. neurona*	**442**
17. Genome of *S. neurona* annotated	**109**

Source: Modified from Dubey, J.P. et al., 2001. *Vet. Parasitol*. 95, 89–131.

intermediate hosts for *S. neurona*, but this finding needs confirmation. *S. neurona*-like sarcocysts were reported in muscles of 1 horse,[1239] 2 dogs,[472,1780] 1 mink,[1445] and 1 bobcat.[1788]

3.2.3 Structure and Life Cycle

3.2.3.1 Structure

Schizonts and sarcocysts are tissue stages in intermediate hosts (Figures 3.4 through 3.10).

Schizonts divide by endopolygeny where the nucleus becomes multilobed before merozoites are formed (Figure 1.22). The schizogonic cycle may be asynchronous; schizonts of different maturity can be found in a single cell (Figure 3.6). Only 1 morphologic type of schizonts is known.

3.2.3.2 Mouse Modeling Schizogony

Only schizonts are produced in the KO mouse. After oral inoculation of *S. neurona* sporocysts, sporozoites excyst in the small intestine, and parasitemia has been detected 1–8 DPI.[430] By 1 DPI, sporozoites can be detected histologically in mesenteric lymph nodes. Mature schizonts with merozoites were first detected histologically starting 8 DPI.[431] Beginning 13 DPI, schizonts were seen consistently in the brain. The other organs parasitized were the lung, heart, liver, intestine, retina, and kidney. Although all regions of brain and spinal cords were parasitized, over 90% of organisms (Figure 3.3) were seen in the cerebellum of the KO mice.[637] The infectivity of sporocysts in the KO

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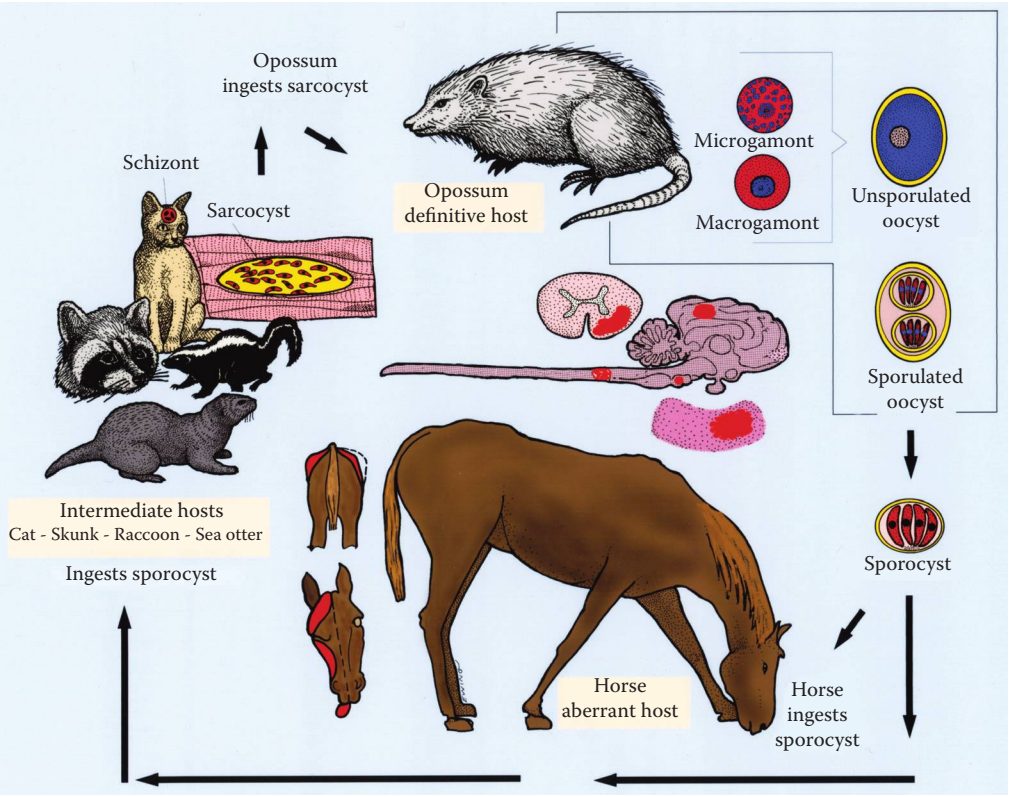


Figure 3.1 Life cycle of *S. neurona.* Opossums are the definitive host, and other animals are aberrant/intermediate hosts. *S. neurona* parasitizes and causes lesions (in red) in the brain and spinal cord of horses. Affected horses can have neurological signs, including abnormal gait, dysphagia, and muscle atrophy.

Table 3.2 Intermediate/Aberrant Hosts of *S. neurona*

Proven Intermediate Hosts[a] (See Table 3.5)	*S. neurona* Isolated from Host Tissue by Bioassay in Mice or Cell Culture (See Table 3.5)	*S. neurona*-Like Sarcocysts in Sections	Clinical Disease Reported (See Tables 3.9 and 3.10)	Only Antibodies Found (See Table 3.7)	Only *S. neurona* DNA Detected in Sections
Cat	Horse	Dog	Horse	Ring tailed lemur	Fur seal
Skunk	Sea otter	Horse	Pony	Blue eyed black lemur	Elephant seal
Raccoon	Pacific harbor seal	Mink	Zebra	Black and white ruffed lemur	Dolphin
Armadillo	Harbor porpoise	Bobcat	Raccoon	Beaver	Sperm whale
Sea otter	Raccoon	Fisher	Cat		Killer whale
			Ferret		
			Dog		
			Skunk		
			Lynx		
			Sea otter		
			Pacific harbor seal		
			Sea lion		
			Red panda		
			Fisher		

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.

[a] Laboratory-raised opossums shed sporocysts after consuming infected muscle; sporocysts were infective to KO mice.

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Figure 3.2 An 18-year-old quarter horse gelding with ataxia and atrophy of hindquarter muscles. This horse improved after antiprotozoal therapy for EPM, and then relapsed 1 year later to the present state. (Adapted from Dubey, J.P. et al., 2001. *Vet. Parasitol.* 95, 89–131.)

mice by the oral route paralleled the subcutaneous route, irrespective of the strain of *S. neurona*.[434] All infected KO mice died, and the dose did not affect clinical signs.[203,207] The strain of the KO mouse (C57BL/6-Black or BALB/c White-derived) did not affect the outcome of the disease, and all affected mice died by 70 DPI; clinical signs were more severe in C57Bl/6-genetic background as compared to the BALB/c line.[470] SCID mice were not susceptible to *S. neurona* infection.[1114,1580] Thus, the genetic background and specific cellular immune components of mice play a role in their susceptibility, and the parasite's ability to replicate and cause disease. Cell-cultured merozoites were infective to KO mice, but bradyzoites from muscles of raccoon were not.[470]

3.2.3.3 Raccoon Modeling of Sarcocyst and Bradyzoite Development

Development of *S. neurona* was studied in 10 raccoons euthanized 1–77 DPI with SN37R strain *S. neurona* sporocysts derived from experimentally infected opossums[1673]; the SN37R strain had been passaged 10 cycles through laboratory-raised raccoons and opossums, ensuring the absence of extraneous species. Parasitemia was detected 3 and 5 DPI. Individual zoites, interpreted as sporozoites, were seen in histological sections of intestines at 1 and 3 DPI. Schizonts with merozoites were consistently detected in 5 raccoons euthanized 7–22 DPI. Schizonts were not detected in 2 raccoons euthanized 37 and 77 DPI. At 22 and 37 DPI, the sarcocysts were immature; they were up to 125 µm long at 22 DPI and up to 270 µm long at 37 DPI. Sarcocysts at 77 DPI were mature. The sarcocyst wall was up to 2.5 µm thick and the slender villar protrusions were 2.5 µm long.[1673] None of these raccoons had clinical signs. In another study, 2 raccoons were each fed 5 million sporocysts; 1 raccoon became lame and was euthanized 14 DPI.[445] The second raccoon developed neurological signs and was euthanized 22 DPI. Histologically, both raccoons had encephalomyelitis associated with schizonts. Additionally, sarcocysts were seen in the muscles of the raccoon euthanized 22 DPI.[445]

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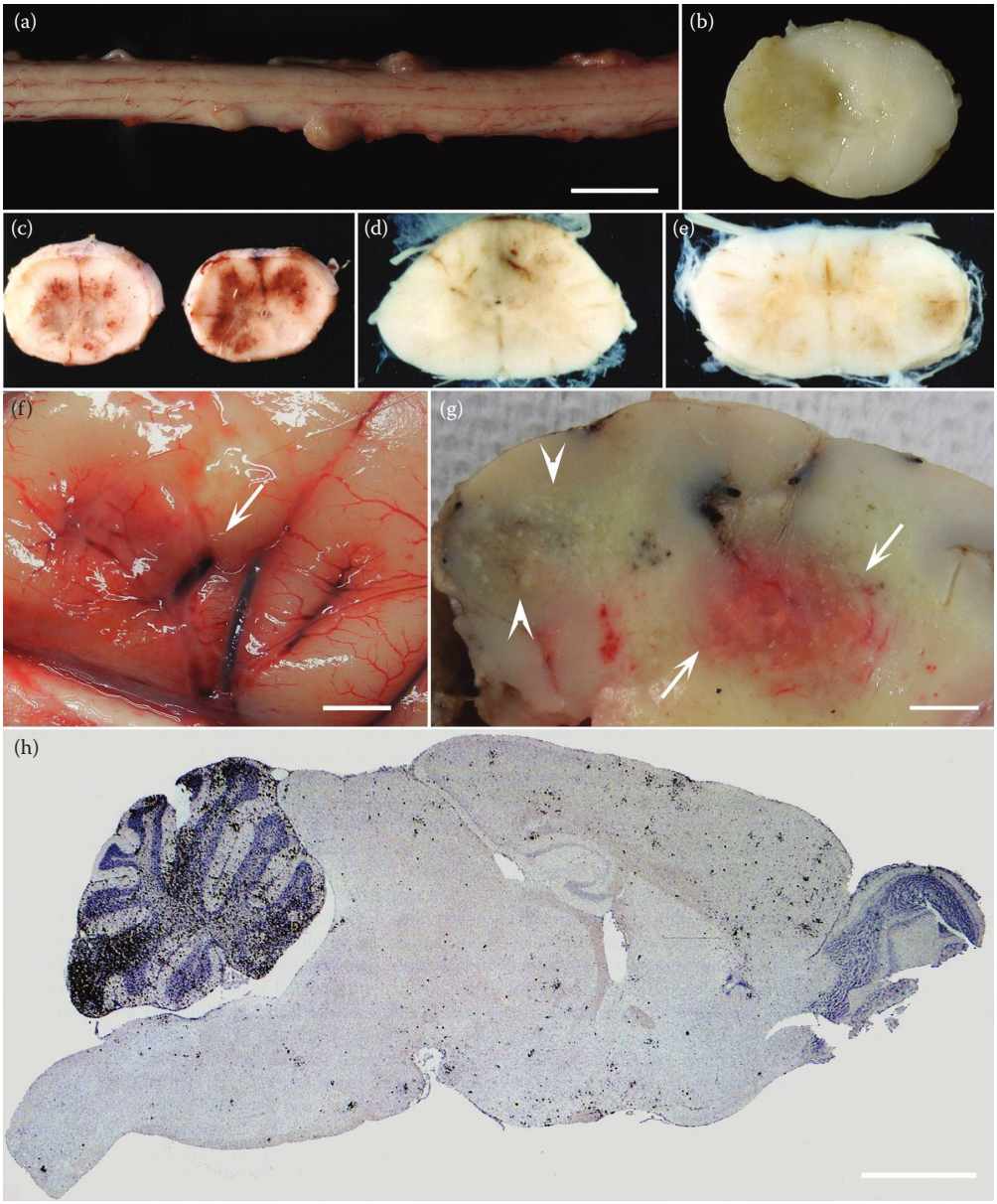


Figure 3.3 *S. neurona*-associated lesions. (a) Nodular growths between spinal nerve roots of the spinal cord of a dog in Reference **670**. (Specimens courtesy of Shelley J. Newman and Amanda Crews.) Unstained. Bar = 1 cm. (b) Cut section of the spinal cord in Figure 3.3a. The protruding lesion is extended into the central canal. Unstained. (c–e) Cross-sections of spinal cords of horses with histologically confirmed cases of EPM. Note the varying degree of hemorrhage and discolored areas, indicative of malacia. Unstained. (Figure (c) from Reference **438**, (e) from Reference **307**.) (f and g) Surface and cut view of cerebrum of a 20-year-old Paint horse with EPM confirmed histologically and by PCR. The horse had a 6-day history of muscle fasciculations, bruxism, difficulty in eating and drinking, and circling to the left with head pressing. Note hemorrhagic and yellow discolored areas, indicative of necrosis. Bar = 5 mm. (Courtesy of Dr. Uneeda Bryant.) (h) Longitudinal section of brain of an interferon gamma gene KO mouse experimentally infected with *S. neurona*. All red dots are merozoites and schizonts. Although the entire brain, from olfactory bulbs to the medulla oblongata, is parasitized, most parasites are concentrated in the cerebellum. Immunolabeling with *S. neurona* antibody. (Adapted from Fritz, D.L., Dubey, J.P., 2002. *Vet. Pathol.* 39, 137–140.)

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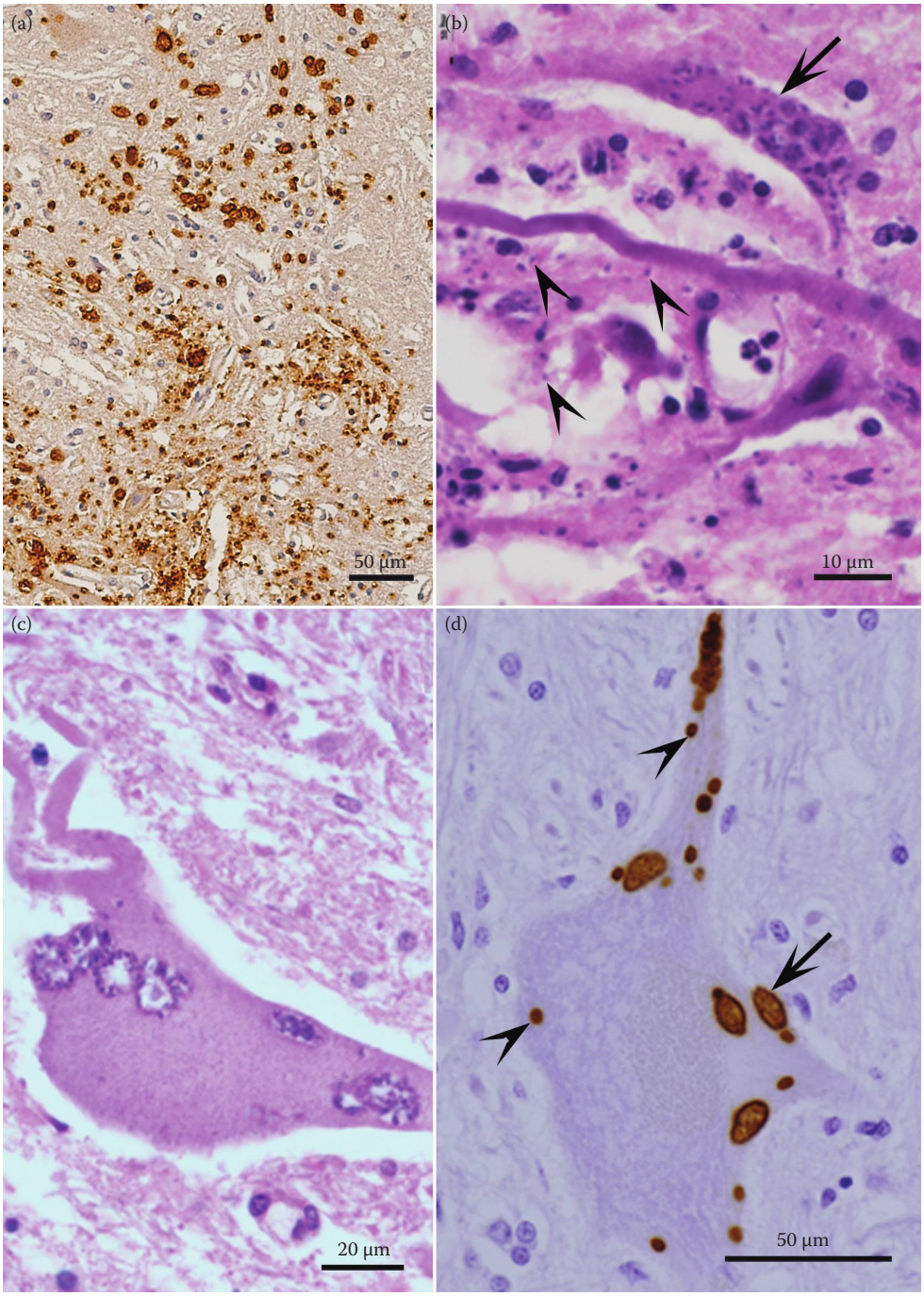


Figure 3.4 Histological sections of spinal cord of a horse with EPM. *S. neurona* was first named from the parasite observed in this horse with massive infection of the spinal cord. (a,c), immunostaining with *S. neurona* antibody, (b,c), H and E stain. (a) Severe myelitis and numerous parasites (all red stained structures). (b) Myelin degeneration, and numerous parasites in an axon (arrow). Merozoites (arrowheads) are 2–3 µm long (arrowheads) and difficult to see. The empty spaces are due to myelin degeneration. (c) Neuron with several schizonts. (d) Intraneuronal merozoites (arrowheads) and schizonts (arrow) in an intact neuron.

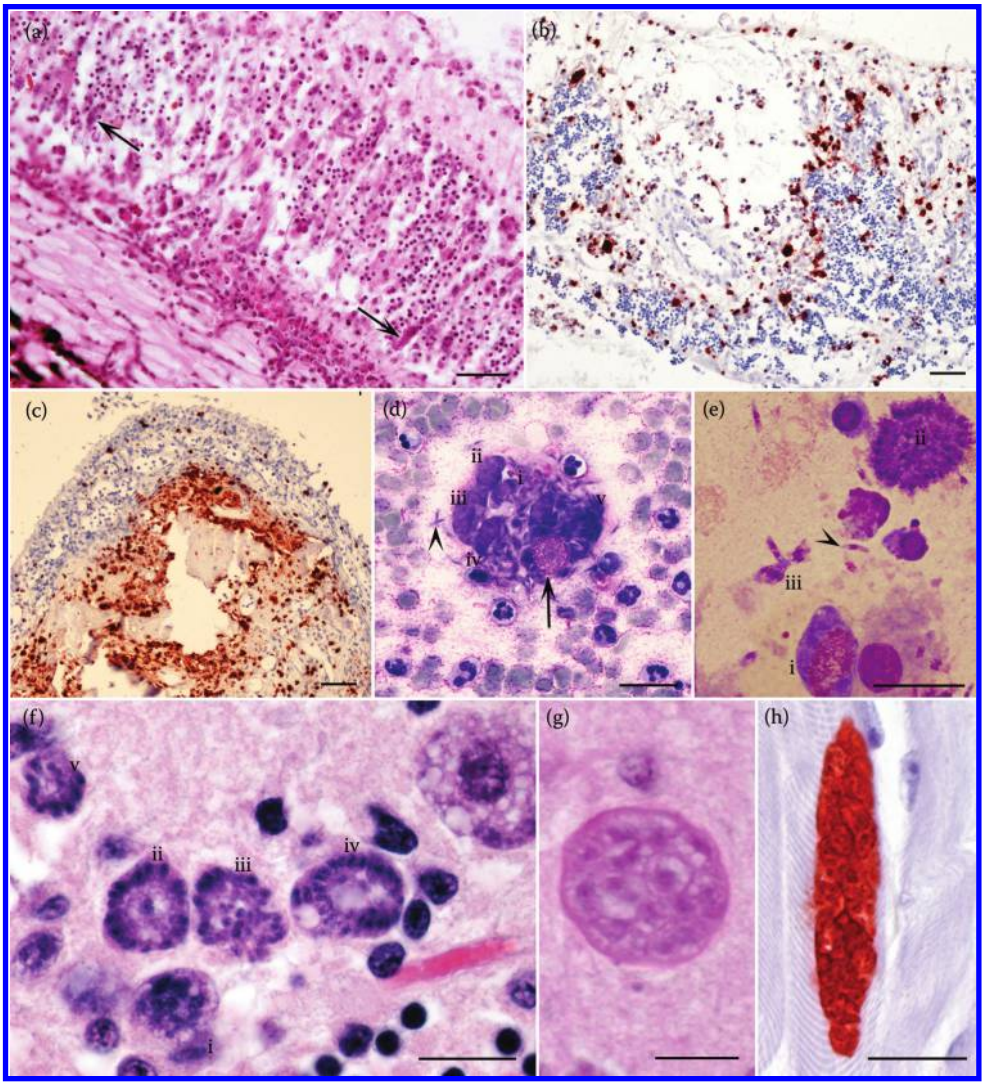


Figure 3.5 *S. neurona*-associated lesions in different hosts. (a, b, c, f, g, h), histological sections, (d and e) touch smears. Bar in (a,b and c) = 50 μm, (d, e, f, and h) = 20 μm, (g) = 10 μm. (a) Eye of a sea otter with 2 foci of retinitis (arrows). Numerous *S. neurona* schizonts and merozoites are present but not visible at this magnification. The retinal architecture is preserved. H and E stain. (From Reference **468**.) (b) Eye of a dog with retinitis and necrosis. The entire retina was riddled with numerous schizonts and merozoites (all red stained structures). Immunostaining with *S. neurona* antibody. (From dog reported in Reference **472**.) (c) Nasal septum from a ferret reported in Reference **145**. The lamina propria is severely parasitized by schizonts and merozoites. The epithelium is intact and mildly parasitized. Immunostaining with *S. neurona* antibody. (d) Smear from pus oozing from a dermal ulcer in a dog reported in Reference **401**. Note the 4 immature schizonts (i–iv), a mature schizont (v), several free merozoites (arrowhead). Arrow points to host cell nucleus. Compare size of merozoites with host red blood cells and neutrophils. Giemsa stain. (e) Touch smear from a grossly visible encephalitic lesion in a red panda. Note immature schizont (i), schizont with budding merozoites (ii), mature schizont with merozoites radiating from a residual body (iii), and free merozoites (arrowhead). Diff–Quick stain. (Courtesy of Timothy Walsch.) (f) Immature and mature schizonts (i–v) in the cerebrum of a sea otter. The section was stained deeply with H and E to reveal the structures. (g) Immature sarcocyst in the brain of a sea otter. Note the eosinophilic globular metrocytes and sarcocyst wall for differentiation from schizonts in Figure 3.5f. (h) Immature sarcocyst in the skeletal muscle of a sea otter. Immunostaining with *S. neurona* antibody. (Figure f–h, adapted from Thomas, N.J. et al., 2007. *J. Comp. Pathol.* 137, 102–121.)

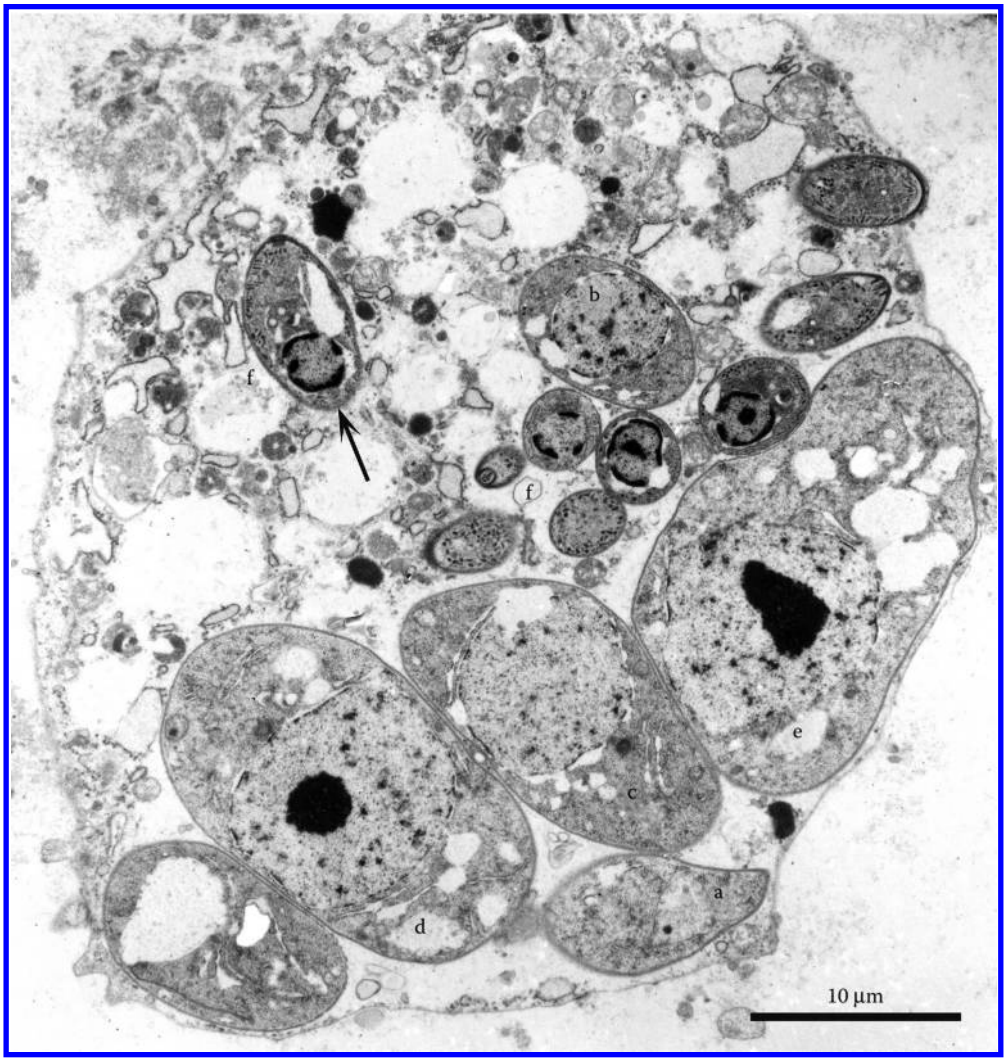


Figure 3.6 TEM of an infected neural cell in the brain of a raccoon naturally infected with *S. neurona*. Note asynchronous schizogony with 6 developing schizonts, in presumed order of development (a–e), and 9 merozoites (f). Arrow points to a longitudinally merozoite with a conoid at 1 end and a subterminal nucleus. The host cell is deteriorated but parasite structures are fairly well preserved. (Adapted from Dubey, J.P. et al., 1991. *J. Helminthol. Soc. Wash*. 58, 250–255.)

3.2.3.4 Feline Modeling of Sarcocysts and Bradyzoite Development

Sarcocysts also developed in cats after oral inoculation with *S. neurona* sporocysts.[429] At 36, 45, and 57 DPI, sarcocysts were immature. At 144 DPI, sarcocysts were up to 700 µm long, and opossums that were fed these sarcocysts excreted *S. neurona* sporocysts. Sarcocysts were also found in the muscles of 1 cat 43 days after the cat was inoculated parenterally with 10 million *S. neurona* merozoites; an opossum fed the sarcocysts from cat muscle did not excrete sporocysts.[157] Sarcocysts found in another cat euthanized 50 DPI were infective to an opossum, as evidenced by sporocyst excretion. Thus, bona fide *S. neurona* sarcocysts have been produced in cats following inoculation with culture-derived merozoites.

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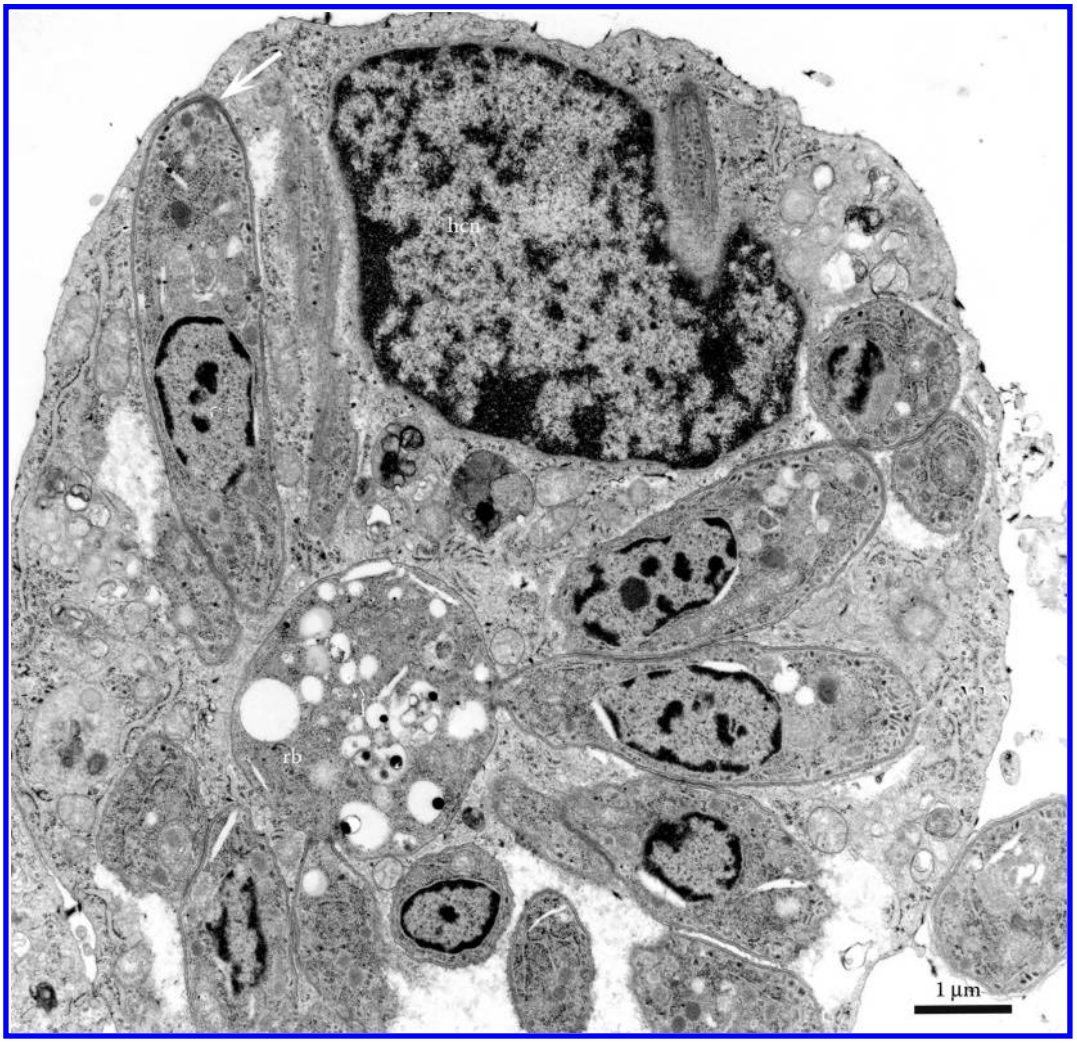


Figure 3.7 TEM of a BT cell culture infected with *S. neurona*. Mature schizont with non conoidal end of merozoites still attached to a residual body (rb). Note the 1 longitudinally cut merozoite (arrow) that has separated from the schizont, and the host cell nucleus (hcn). (Adapted from Dubey, J.P., 2004. *Infectious Diseases of Livestock with Special Reference to Southern Africa*. Oxford University Press, Ni City, South Africa, pp. 394–403.)

3.2.3.5 Equine Modeling of Early Parasite Infection

Attempts were made to study migration and development of *S. neurona* in 6 ponies after oral inoculation with 250,000,000 sporocysts[493]; the ponies were serologically negative to *S. neurona* and were euthanized 1, 3, 5, 7, and 9 DPI. Viable *S. neurona* were isolated by bioassay in KO mice, and in cell culture inoculated with tissue homogenates of pony mesenteric lymph nodes at 1, 2, and 7 DPI, liver at 2, 5, and 7 DPI, and from lungs at 5, 7, and 9 DPI. The parasite was not detected histologically in tissues of any pony. However, encephalitic lesions were detected in sections of brain and spinal cords of 2 ponies euthanized 7 and 9 DPI. One pony euthanized 9 DPI had IgM antibodies to *S. neurona*.[493]

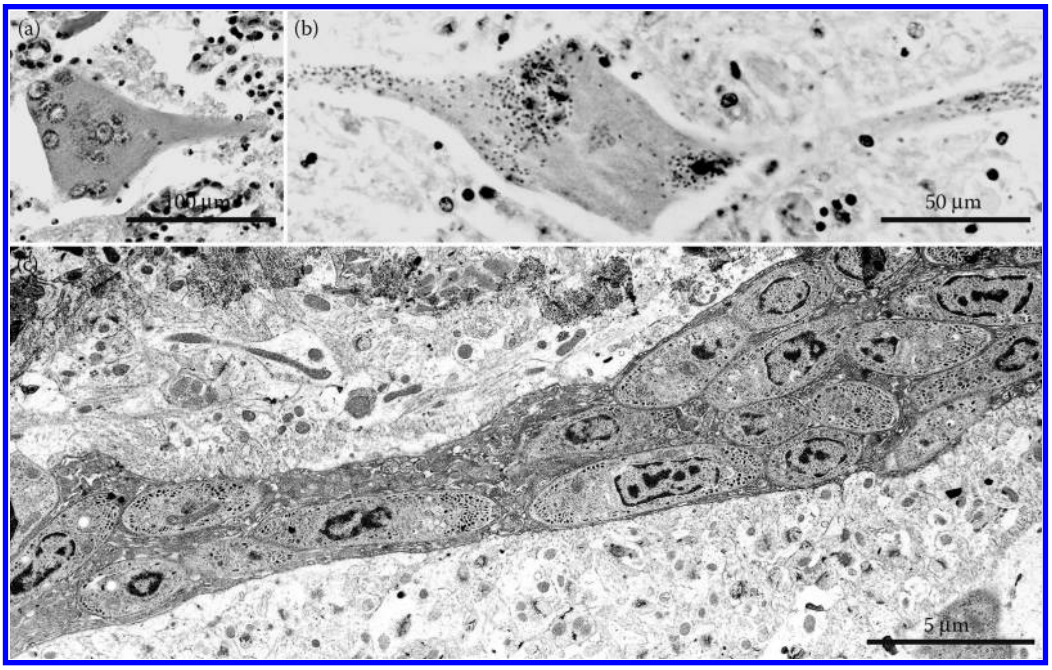


Figure 3.8 (a) A heavily infected neuron in the spinal cord of a horse with numerous schizonts. (b) Neuron with numerous merozoites. (From Dubey, J.P. et al., 1974. *J. Am. Vet. Med. Assoc.* 165, 249–255. With permission.) (c) *S. neurona* merozoites free in the cytoplasm of an unmyelinated axon in the cerebellum of an experimentally infected KO mouse. (Adapted from Fritz, D.L., Dubey, J.P., 2002. *Vet. Pathol.* 39, 137–140.)

3.2.4 Naturally Occurring Stages and Their Structure

In naturally infected horses with neurological signs, *S. neurona* schizonts have been found only in the CNS. Both neural and inflammatory cells in the CNS can be parasitized (Figure 3.8). As many as 13 schizonts and several hundred merozoites were present in 1 neuron.[348] As with other *Sarcocystis* species, the schizogonic cycle can be asynchronous; schizonts of different maturity can be found in a single cell (Figure 3.6). Mature schizonts in the CNS are up to 30 µm long and they can be oval, round, elongated, or irregular in shape. Ultrastructurally, merozoites were either stumpy (7.7 × 3.1 µm) or slender (7.3 × 1.7 µm), and they contained the same organelles as described in the merozoites of other *Sarcocystis* species, including the absence of rhoptries.[1661]

Sarcocysts have been demonstrated in skeletal and cardiac muscles and in the brain (Figure 3.5). Sarcocysts in the brain are often round and smaller in size than in skeletal muscles.[1196,1739] Additionally, sarcocysts in myocardium are smaller than those in skeletal muscles. Ultrastructurally, the mature *S. neurona* sarcocyst wall has vp that are up to 2.8 µm long and 0.4 µm wide.[435] The vp have microtubules that extend from tip to the base but have no granules (Figure 3.10). Few of these microtubules extend deeper in the ground substance, but they are electron-lucent and not prominent compared with the other *Sarcocystis* species of the horse in North America, *S. fayeri* (Figure 3.10). Bradyzoites in sections are 4.8–6.5 × 1.0–1.3 µm in size, and contain only 2 rhoptries.

S. neurona sporocysts are 11.3 × 8.2 µm in size. Sporozoites are slender, and have 2–4 rhoptries but no crystalloid body.[1079] The absence of a crystalloid body in sporozoites is notable, because the crystalloid body was present in *S. cruzi* and other species of *Sarcocystis* sporozoites examined ultrastructurally.[393]

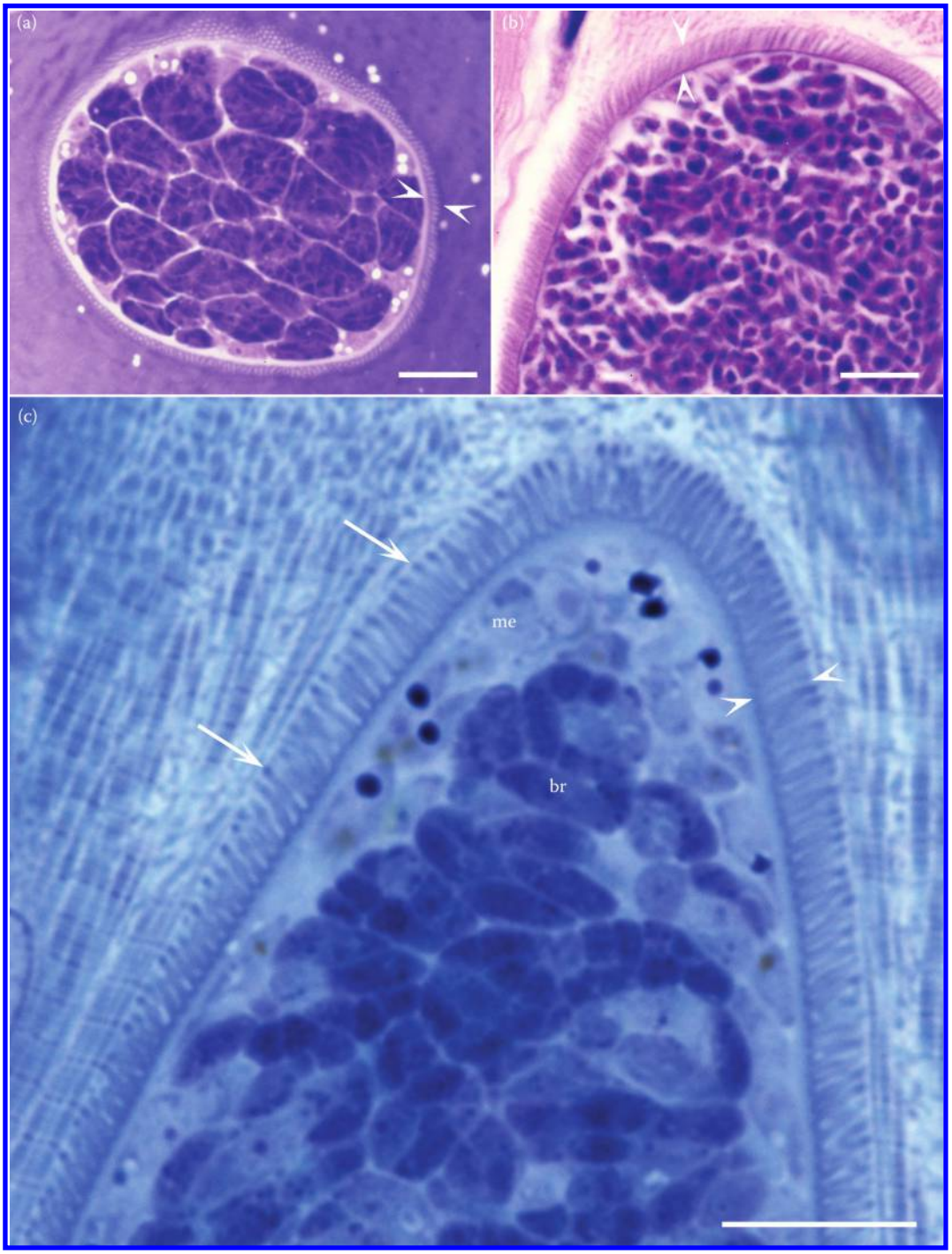


Figure 3.9 *S. neurona* sarcocysts in histological sections of skeletal muscle. Arrowheads point to striated cyst wall. Arrows point to thickening of the villar tips. (a) Cat, 144 DPI. (Adapted from Dubey, J.P. et al., 2001. *J. Parasitol.* 87, 1323–1327.) Toluidine blue stain. (b) Raccoon, 77 DPI. H and E stain. (c) Toluidine blue stain. (b and c, adapted from Stanek, J.F. et al., 2002. *Procyon lotor. J. Parasitol.* 88, 1151–1158.) Bars = 10 μm.

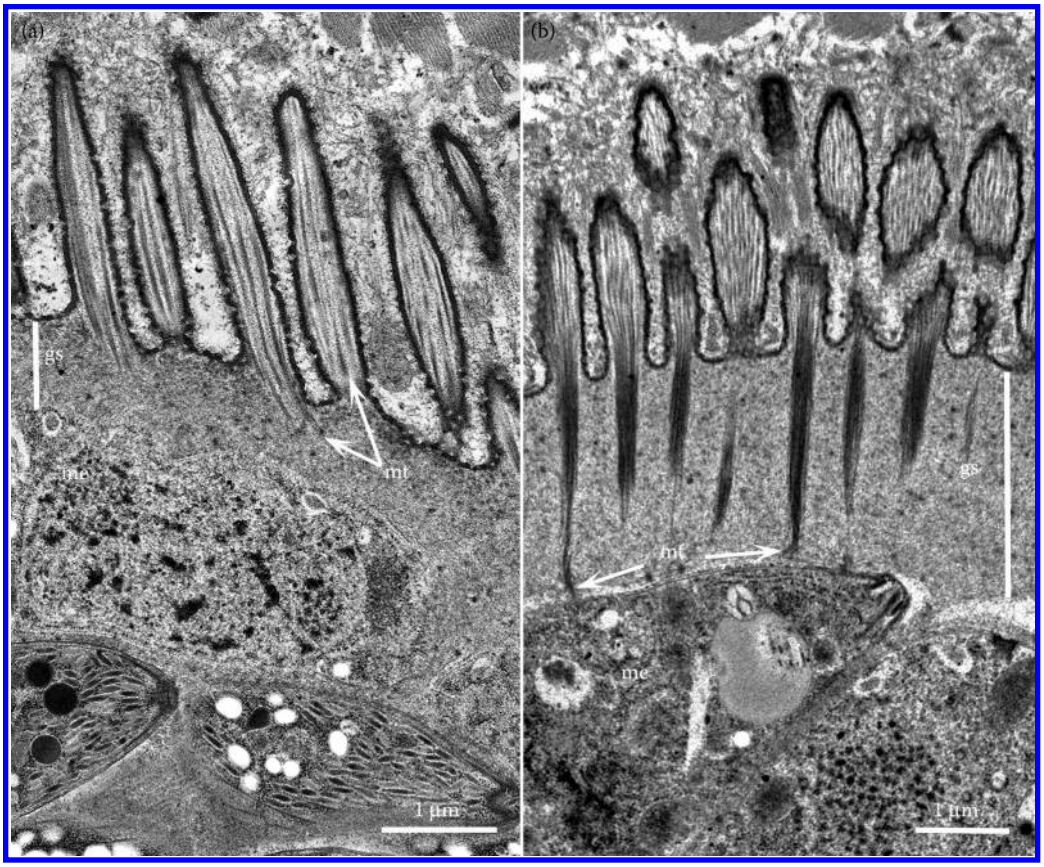


Figure 3.10 Comparison of the cyst walls of *S. neurona* (a) and *S. fayeri* (b) sarcocysts by TEM. The cyst walls, including the ground substance layer (gs) of *S. fayeri*, are thick, the microtubules (mt) are more electron-dense and extend up to the pellicle of the zoites, whereas the cyst walls of *S. neurona* are comparatively thin, the microtubules are few, and never extend deep in the gs. (Adapted from Stanek, J.F. et al., 2002. *J. Parasitol.* 88, 1151–1158; Saville, W.J.A. et al., 2004. *J. Parasitol.* 90, 1487–1491.)

3.2.5 Excretion of Sporocysts by Definitive Hosts

Large numbers of sporocysts can be excreted in feces of opossums (Table 3.3). Opossums are the definitive hosts for *S. neurona* and 3 other named species, *S. speeri*, *S. falcatula*, and *S. lindsayi*.[137,416,433,1722] Cheadle et al.[202] provided morphologic measurements of possibly 5 species of *Sarcocystis*-like sporocysts in the feces of 17 naturally infected opossums. Sporocysts of *S. neurona* were 10.7 × 7.0 µm, *S. speeri* were 12.2 × 8.8 µm, strain-1085 of *Sarcocystis* sp. were 10.9 × 6.8 µm, and *S. falcatula* were 11.0 × 7.1 µm; thus, the differences were within 2-µm range. Additionally, the identity of sporocysts of each species was from naturally infected opossums, and the species identification was not definitive.

Sporocysts of another organism were reported in feces of 10 opossums; sporocysts were 19.4 × 10.5 µm in size and had Stieda body-like structures at both poles.[202] Based on the large size, these are unlikely to be *Sarcocystis* sporocysts. Bioassay and molecular methods have been used to distinguish sporocysts of different species in opossum feces. The KO mice and budgerigars have been used to differentiate viable *S. neurona* and *S. falcatula* in opossum feces; *S. neurona* and *S. speeri* are not infective to budgerigars, and *S. falcatula* is not infective to KO mice.[412] *S. speeri*

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Table 3.3 Prevalence of *Sarcocystis* Sporocysts in Feces/Intestines of Opossum (*Didelphis virginiana*) in the United States

Location	Year	No. Tested	With Sporocysts (%)	*S. neurona*	Other Species	*S. neurona* Isolates	Reference
LA, MD, VA, GA, FL, PA	1998–99	44	24 (54.4)	14 (31.8%)-bioassay in KOM	19 (43.1%) *S. falcatula*-bioassay in budgerigars, 8 (18.8%) *S. speeri*-bioassay in KO mice	SN8-15OP	**421**
MS	1999–00	72	24 (33.3)	19 (26.3%)	Not investigated, 1 opossum infected with *S. speeri*	SN16 to 34OP	**443**
MI	1996–02	206	37 (17.9)	23 (11.1%) PCR–RFLP	4 (1.94%) *S. falcatula* based on PCR–RFLP	Not stated	**513**
CA	2005–08	288	53 (18.4)	17–PCR–ITS-1	Not stated	OP-1-13	**1463**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
CA = California, FL = Florida, GA = Georgia, LA = Louisiana, MD = Maryland, MI = Michigan, MS = Mississippi, PA = Pennsylvania, VA = Virginia.

and *S. neurona* in KO mice can be distinguished immunohistochemically.[421] Molecular methods have been developed to distinguish *S. falcatula* and *S. neurona*.[1722] Few *S. neurona* sporocysts are lethal for KO mice,[470] and 1000 *S. speeri* sporocysts are pathogenic for KO mice.[416]

S. neurona sporocysts have been found in 6%–31% of opossums tested in the United States (Table 3.3). Three of the surveys listed in Table 3.3 examined factors associated with *S. neurona* positivity (Table 3.4). Season, body condition, and presence of young in pouch were associated with the presence of sporocysts. How these factors affect the presence of sporocysts in opossums is uncertain, because *Sarcocystis* does not multiply in definitive hosts, and little is known of immunity to reinfection. The availability of infected intermediate hosts during spring might account for the results shown in Table 3.4.

The South American opossum, *D. albiventris*, is also a host for *S. neurona*.[442]

Table 3.4 Factors Associated with *Sarcocystis* Infections in Opossums in the United States

		Percentage of Positive (n)		
Factor		**Mississippi[1467]**	**Michigan[513,514]**	**California[1464]**
Age	Adult	28 (14/50)	14.5 (30/206)	6.7 (14/206)
	Juvenile	22.7 (5/22)	3.3 (7/206)	3.7 (3/81)
Gender	Female	29.4 (10/34)	9.7 (20/206)	4.4 (6/136)
	Male	23.6 (9/38)	8.25 (17/206)	7.5 (11/146)
Female with pouch	Present	33.3 (6/18)	No data	8.3 (2/24)
	Absent	25 (4/16)	No data	5.2 (4/76)
Season	Spring	34.2 (13/38)	3.3 (7/206)	9.1 (14/154)
	Summer	No data	9.2 (19/206)	
	Autumn	No data	3.3 (7/206)	2.2 (3/133)
	Winter	17.6 (6/34)	1.9 (4/206)	
Total number tested		72	206	288
Total positive (%)		26.3 (19)	17.9 (37)	5.9 (17)

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.

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3.2.6 Transplacental Transmission

Currently, there is no documented case of congenital *S. neurona* infection in land mammals. *S. neurona* DNA was recently found in tissues of 8 of 13 marine mammal neonates, supporting congenital infection.[210] These eight animals included three fetuses removed from the uteri of a naturally infected (with demonstrable DNA in tissues of mother) pygmy sperm whale (*Kogia breviceps*), a harbor seal (*Phoca vitulina*), and a Steller sea lion (*Eumatopias jubatus*). Additionally, five fetuses (four harbor seals and one harbor porpoise) were beach-cast carcasses, too young to have nursed.[210]

3.2.7 *In Vitro* Cultivation, Cell and Molecular Biology

3.2.7.1 In Vitro *Cultivation*

Viable *S. neurona* has been isolated from many hosts (Table 3.5). Numerous cell lines can support the growth of *S. neurona* (Table 3.5). In most EPM horses, the number of *S. neurona* in CNS tissue is low. Therefore, culture flasks seeded with CNS homogenates should be incubated for at least 2 months, because some strains are slow to adapt in cell cultures. Once established, *S. neurona* can complete schizogonic development in 3 days.[393,1770,2321] *S. neurona* has also been isolated from sporocysts purified from the intestines of opossums, either directly in cell cultures[511,1262] or by feeding sporocysts to KO mice and then recovering *S. neurona* in cell cultures from mouse brains. The isolation of *S. neurona* from sporocysts, via KO mice, is advantageous, because it removes *S. falcatula*, a common parasite in opossum feces; *S. falcatula* is not infective to KO mice.[412,1115] Cell cultures infected with *S. neurona* are useful for *in vitro* screening of anti-*S. neurona* compounds and other aspects of biology.[130,661,1118,1119,1993,1994]

3.2.7.2 Cell Biology

Because the parasite is easy to cultivate in many cell lines and can be genetically manipulated, *S. neurona* has been used as a model to study several aspects of cell biology. Biologically, *S. neurona* has many characteristics in common with *Toxoplasma gondii*, and multiple reagents and techniques developed for *T. gondii* have been applied in studying the biology of *S. neurona*. In turn, the *S. neurona* culture system has contributed to the understanding of organelle development, which is difficult to resolve with the *T. gondii* cultures. One example is the information on plastid replication. Compared with *T. gondii* (<6 h), the nuclear division in *S. neurona* merozoites is prolonged (3 days), and, thus, events during parasite development can be easily followed. Nuclear and plastid replication were followed[1770] in BT cells infected with the SN3 isolate of *S. neurona* (Figure 1.22). In this culture system, 64 merozoites were formed in 3 days. During schizogonic development, the nucleus continued to grow and became lobulated; 5 cycles of chromosome replication occurred without nuclear division.[1770] After the sixth and the final division, the nuclear lobes bifurcated into 2, giving rise to 64 merozoites. The apicoplast in *S. neurona* was a 4-layered, tubular structure without microcristae, and was closely associated with the nucleus at all stages of division. During nuclear division, the apicoplast stayed associated with the nuclear lobes, like a flexible hose. When the nucleus divided, the apicoplast fragmented, and 1 lobe was incorporated into the budding merozoite.

3.2.7.3 Gene Discovery and Characterization

There is limited information concerning the molecular composition of *S. neurona*. The initial efforts to generate nucleic acid sequence data for *S. neurona* were conducted in the context of phylogenetic analyses and included the use of RAPD assays[517,518,522–524,729,1722] and sequence analysis of

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Table 3.5 *In Vitro* Cultivation of *S. neurona*

Host	Year	Tissue[a,b]	Cell Type Initial Culture[c,d]	Strain Designation	Reference
Horse (*Equus caballus*)	1990	Spinal cord	M617	SN1	**404**
	1990	Spinal cord	M617	SN2	**307**
	1991	Spinal cord	M617	SN3	**727**
	1991	Spinal cord	M617	SN4	**307**
	1992	Spinal cord	M617	SN5	**729**
	1998	Spinal cord	M617, Esp	SN6	**420**
	1997	Spinal cord	M617, EK, Esp	SN7	**444**
	1999	Spinal cord	ED (not BT, DT)	SN-MU1,2	**1119**
	1994–95	Spinal cord	M617	UCD1,2,3	**1113, 1114**
	2009	Brain	Not stated	H1756, H1801	**1464**
	1997–98	Spinal cord or brain	ED	MIH 1-8	**1107**
Sea otter (*Enhydra lutris*)	NS	Brain	CV-1, BT	SN-OT1	**1072**
	1999	Brain	MA104	SO SN_1	**1195**
	2009	Brain	MA104	SO1-3	**1196**
	2004	Brain	MA104	8 isolates	**1197**
	2005–08	Brain	MA104	23 isolates[e]	**1464**
Pacific harbor seal (*Phoca vitulina*)	1998	Brain, CSF	MA104	HS1	**1194**
	2011	Brain	MA104	PV16-19; PV22	**210**
Harbor porpoise (*Phocoena phocoena*)	2006	Brain	Not stated	HP060325	**1464**
	2011	Brain	MA104	PP11	**210**
Raccoon (*Procyon lotor*)	2001	Muscle[b]	Not applicable	SN37R	**445, 1636**
Cat (*Felis catus*)	2000	Muscle[b]	Not applicable	Sn-Mucat-2	**157, 1765**
Armadillo (*Dasypus novemcinctus*)	2000	Muscle[b]	Not applicable	Not stated	**205**
Sea otter (*Enhydra lutris*)	2001	Muscle[b]	Not applicable	SN-OT-2[f]	**440**
Brown-headed cowbird (*Molothrus ater*)	1999	Muscle[b]	Not applicable	MICB1	**83**
South American opossum (*Didelphis albiventris*)	1999	Intestine[a]	EK,BT	SN35-OP, SN36-OP	**442**
North American opossum (*Didelphis virginiana*)	1999	Intestine[a]	ED, M617	SN8 to 15OP	**421**
	1999	Intestine[a]	ED, BT, EK, M617	SN16 to 34OP	**442**
	2005–08	Intestine[a]	MA104	OP 26, 48, 68, 134, 166, 187, 201, 212, 226, L49, GA3, GA7, OPBF2	**1464**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
NS = not stated.

[a] By feeding sporocysts to KO mice, and *in vitro* cultivation from infected tissues of KO mice.
[b] By feeding naturally infected muscle to laboratory-raised opossums and the bioassay of sporocysts shed into KO mice.
[c] *In vitro* cultivation directly from sporozoites released from sporocysts.
[d] BT = bovine turbinate, CV1 = African green monkey, DT = deer testes, ED = equine dermal, EK = equine kidney, ESp = equine spleen, M617 = bovine monocytes, MA104 = monkey kidney.
[e] SO 4387, 4413, 4530, 4653, 4697, 4711, 4725, 4755, 4786, 4834, 4928, 4970, 4972, 5002, 5073, 5110, 5226, 5259, 5263, 5274, 5278, 5283, 5296.
[f] Designated here.

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the 18S rRNA locus[290,600,1116] to identify species-specific genetic markers. These investigations led to the development of PCR-based tests to detect *S. neurona* and/or distinguish it from other closely related species.[601,1722]

Early studies to identify and characterize protein-encoding genes of *S. neurona* utilized traditional molecular biology approaches. Sequence analysis of random clones from a cDNA library produced from the UCD-1 strain, and standard immunoscreening of a cDNA library produced from the SN3 strain, independently identified the *S. neurona* major surface antigen SnSAG1,[499,849] which is homologous to the gene family of SAG/SRS surface proteins that have been investigated extensively in *T. gondii*.[917,1045,1812] Polyclonal antibodies against *T. gondii* enolase 2 (ENO2), followed by mass spectrometry, were used successfully to identify the *S. neurona* ENO2 homolog.[123,1829] Biochemical characterization of *S. neurona* merozoites demonstrated the presence of serine protease activity at defined molecular weights, but the encoding gene(s) was (or were) not identified.[72]

To better facilitate gene discovery in *S. neurona*, an expressed sequence tag (EST) sequencing project was performed using cDNA libraries constructed from the merozoite stage of the EPM isolates SN3 and SN4.[848,1054,1055] This project generated[848] partial gene sequences from *S. neurona* with another 6332 ESTs produced from the closely related *S. falcatula* for comparative purposes. The resulting database of sequences represented only a portion of the genes present in *S. neurona*, but it permitted more efficient approaches to identify and select parasite sequences for further investigation. While a majority of *S. neurona* genes were chosen for study based on their homology to *T. gondii* molecules, predicted structural features of the encoded protein and cDNA abundance (i.e., number of ESTs) were also useful criteria for selection of interesting genes.

Along with the major surface antigen, SnSAG1 that was identified by traditional approaches, 3 additional SnSAG paralogues—designated SnSAG2, SnSAG3, and SnSAG4—were revealed in the ESTs generated from the SN3 strain of *S. neurona*.[849] The SnSAGs are located on the surface of the extracellular merozoite stage of *S. neurona* and are present throughout intracellular development of the schizont as well. Additionally, analyses of the bradyzoite and sporozoite life cycle stages demonstrated that expression of the SnSAGs is differentially regulated during the life cycle,[668] similar to the SAG/SRS surface antigens in *T. gondii* and *N. caninum*. The specific function(s) of SnSAGs is unknown. However, evidence from *T. gondii* has implicated several of the TgSAGs as cell adhesins and modulators of host immunity,[483,958,1437,1812] so it seems reasonable to hypothesize that SnSAGs perform a similar role. Because the SnSAGs elicit strong humoral responses in infected animals,[498,499,849,1062] they have been used as target molecules to develop serologic tests for detection of antibodies against *S. neurona*.[500,837,1862] Interestingly, it is apparent that all strains of *S. neurona* do not possess the same repertoire of SnSAG genes. This antigenic diversity was initially revealed in an *S. neurona* isolate from a horse in Missouri.[1119] Further examination of this strain, designated Sn-MU1, revealed that it lacked the SnSAG1 gene that is transcribed abundantly in the UCD-1 and SN3 strains.[870] Subsequent immunologic analysis of a collection of 14 *S. neurona* strains, using antiserum against SnSAG1, demonstrated that this surface antigen is not expressed by multiple strains, and the lack of expression was shown to be due to the absence of the SnSAG1 gene.[850] Those parasite strains that lack SnSAG1 were found to express 1 of 2 alternative major surface antigens that were called SnSAG5 and SnSAG6.[263,1821] While it is conceivable that additional alternative major SnSAG paralogues exist, analyses of much more extensive collections of parasite strains suggest that SnSAG1, SnSAG5, or SnSAG6 will be predominant in the *S. neurona* strains circulating in nature.[1464,1820] The genes for these 3 SnSAG paralogues seem to be mutually exclusive to one another, because all strains of *S. neurona*, that have been analyzed, possess sequence for only SnSAG1 or SnSAG5 or SnSAG6; no strain has been found that possesses more than 1 of these genes. Analyses of synonymous versus nonsynonymous mutations in the SnSAG1 and SnSAG5 gene sequences suggest that there may be evolutionary advantages to altering some regions of these surface proteins.[512]

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The *S. neurona* surface protein 1 (SnSPR1) gene is a seemingly novel sequence that is not paralogous to the SnSAG surface antigens but does encode a merozoite surface protein.[1877] Instead of relying on homology to select the sequence for investigation, this gene was chosen for study based on its abundance in the *S. neurona* EST collection, and the prediction that the encoded protein had an amino-terminal signal peptide and a carboxyl-terminal glycolipid anchor addition consistent with a cell-surface protein. In addition to localizing to the merozoite surface, SnSPR1 was shown to be a low molecular weight protein (approximately 14 kDa) that is present throughout the intracellular development of the *S. neurona* schizont. Despite being an abundant protein based on the number of ESTs that match the SnSPR1 sequence, this protein does not appear to elicit the robust immune response seen for the SnSAG surface antigens.

Genes encoding secretory proteins of *S. neurona* have been identified based on homology. A putative SnMIC10 sequence exhibited approximately 30% identity to the TgMIC10 and NcMIC10 orthologues of *T. gondii* and *N. caninum*, respectively, and examination of the native protein in merozoites using antiserum, raised against recombinant SnMIC10, revealed characteristics consistent with it being a microneme protein of *S. neurona*.[835] A dithiol-dependent nucleoside triphosphate hydrolase (SnNTP1) was similarly identified, based on sequence similarity to the 2 TgNTPase isoforms of *T. gondii*.[1876] Although localization to the apical end of the merozoite was unexpected for SnNTP1, it was found to be part of the secreted fraction of *S. neurona* consistent with it being a dense granule protein. Interestingly, both SnMIC10 and SnNTP1 are not expressed for much of *S. neurona* intracellular development, with these 2 proteins seen only in late schizonts containing newly forming daughter merozoites.[835,1876] While this was expected for SnMIC10, since microneme proteins participate in host-cell invasion and should not be needed during endopolygeny, it was unanticipated for SnNTP1 because the TgNTPases are important for intracellular growth of *T. gondii*.

3.2.7.4 Molecular Genetic Tools

To enhance the study of *S. neurona*, basic methods have been established for DNA transfection, transient expression of transgenes, and selection of stably transformed clones of this parasite. Luciferase, β-galactosidase (β-gal), yellow fluorescent protein (YFP), and red fluorescent protein (RFP) have been used successfully as reporter molecules in *S. neurona*.[656,657,1770] Mutant dihydrofolate reductase–thymidylate synthase (DHFR–TS) that confers resistance to pyrimethamine can be used to achieve stable transformation of *S. neurona*. This selection system was used to produce transgenic *S. neurona* clones that stably express either β-gal or YFP, which are useful for monitoring parasite growth or invasion rates *in vitro*.[656] Additionally, fluorescence-activated cell sorting (FACS) of YFP expression can be used to select stable clones of *S. neurona* that do not contain a drug resistance gene. Recently, the hypoxanthine–xanthine–guanine phosphoribosyltransferase gene of *S. neurona* (*SnHXGPRT*) was successfully disrupted by double crossover homologous recombination using a gene-targeting plasmid.[293] The SnHXGPRT-deficient mutant clones (SnΔHXG) were selected by their resistance 6-thioxanthine (6-TX), a toxic analog of xanthine, and could be complemented with the *T. gondii HXGPRT* gene, rendering the SnΔHXG parasites resistant to mycophenolic acid (MPA). Thus, the SnΔHXG clone provides an efficient system for both positive and negative selection of stable transgenic *S. neurona*. Collectively, the molecular genetic capabilities that have been developed for *S. neurona* make a variety of new experimental approaches possible, including gene knockouts, complementation studies, and gene regulation assays. Indeed, the value of these molecular tools has been demonstrated in studies examining the development and segregation of the *S. neurona* apicoplast during endopolygeny[1770] and identification of sequence elements involved in promoter function.[657]

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3.2.7.5 Population Genetics

Compared to other prominent parasites within the Apicomplexa, there have been only limited analyses performed to examine the population genetic structure of *S. neurona* strains circulating throughout the Americas. As stated previously, initial efforts focused on developing markers capable of distinguishing between the different *Sarcocystis* species that infect the opossum definitive host, including *S. neurona*, *S. falcatula*, *S. lindsayi*, and *S. speeri*. The first molecular characterization was performed using 18S rRNA marker on *Sarcocystis* strains isolated from horses and established that *S. neurona* resolved phylogenetically within the family Sarcocystidae, suggesting a close relationship to *S. muris*.[600] Follow-up work at the 18S locus indicated that *S. neurona* was synonymous with *S. falcatula*, the parasite that cycles between opossums and birds.[290] Phylogenetic resolution between *S. neurona* and *S. falcatula* was finally achieved upon DNA sequencing the ITS-1 locus within the 18S rRNA gene array, which identified 12 nucleotide differences between the 2 parasites.[1116] In the absence of gene-specific molecular markers, panels of random amplified polymorphic DNA (RAPD) markers were next developed to differentiate *S. neurona* isolates from other related coccidia[729] and within the *S. neurona* species.[523,1722] These investigations led to the development of PCR-based tests used to detect *S. neurona*, and/or distinguish it from other closely-related coccidia.[601,1722]

During the last 15 years, a large collection of *S. neurona* strains have been isolated from a variety of geographic regions and host species (Table 3.5). To ascertain the true genetic diversity among circulating strains, and to address whether specific *S. neurona* strains are associated with increased disease risk, a serious effort has been pursued to develop a panel of genetic markers capable of resolving the parasite's population genetic structure, its evolutionary biology, and the extent to which it expands in nature.

To answer these important questions, a series of genetic markers of varying resolution have been developed. The first set of widely applied sequence-specific markers for population genetic analyses were derived from 2 RAPD markers and were described.[1722] An RFLP at the 33/54 locus was used to distinguish *S. neurona* from *S. falcatula*, and nucleotide sequence polymorphisms at the 25/396 locus identified 2 major alleles that resolved North American from South American *S. neurona* strains.[1492] To estimate whether the *S. neurona* population genetic structure was genetically diverse or clonal, microsatellites markers were developed[44,45] and applied 12 highly polymorphic microsatellite markers against 34 predominantly North American *Sarcocystis* samples collected from both definitive and intermediate hosts isolated from diverse geographical origins.[1693] This important study[1693] established that substantial allelic and genotypic diversity exists among circulating *S. neurona* strains and showed that 1 genotype is more prevalent than expected for a strictly outbred population, indicating that some degree of clonal expansion has occurred within the species.[45]

The identification of widespread *S. neurona* infections in marine pinnipeds and the discovery of a large epizootic that resulted in the death of approximately 2% of the federally listed threatened southern sea otter population, during a 3-week period in 2004, fostered the necessity to develop a more comprehensive genotyping scheme to determine the transmission dynamics of the parasite and whether specific genotypes of *S. neurona* circulating in the marine ecosystem were more pathogenic.[440,1072,1077,1194,1195,1197,1494] In 2010, a panel of gene-specific molecular markers of varying phylogenetic resolution were identified to increase the discriminatory power of the molecular markers used for *Sarcocystis* population genetic analyses. This work established a comprehensive multilocus sequence typing (MLST) approach capable of resolving strains at the genus, species, and intraspecies levels.[1464,1821] The markers consisted of a plastid-encoded RNA polymerase b gene (*RPOb*) and a cytochrome c oxidase 1 (*cox1*) gene encoded, respectively, within the apicoplast and mitochondrial organellar genomes, both of which are maternally inherited and exist as useful

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markers to detect genetic exchange (or hybridization) between strains based on incongruity between nuclear and organellar genome phylogenies. Hence, when 2 nearly identical MLST genotypes possess different organellar genomes, outcrossing is supported. In addition, the discovery of a family of polymorphic SnSAG surface antigen genes, orthologous to the highly informative SRS genotyping markers encoded by *T. gondii*,[849,850] identified a series of intraspecific genetic markers (annotated SnSAG1, 3, 4, 5, and 6) that possessed sufficient allelic diversity to chart the parasite's population genetic structure and produce the first genetic history model for *S. neurona*.[1820] The MLST sequence-level analysis identified 12 genetic types, 2 of which were predominant (56/87; 64%) and accounted for the majority of infections in the United States, based on the inheritance of different allele combinations encoded by the SnSAG genes among the 87 *S. neurona*-infected samples collected from varying hosts and geographic locations.[1820] This data supported a population structure that is both clonal and punctuated by a series of genetic types that evolved by sexual recombination through the definitive opossum host. Such an intermediate population structure is similar to that described by *T. gondii*.[737] To explain the existence of dominant clonotypes within the population structure for *S. neurona*, it was shown that *S. neurona*, like *T. gondii*, is capable of same-sex mating, and established selfing (or sexual amplification of a single clone) as a genetic mechanism for the clonal propagation of the species. Unlike *T. gondii*, the tissue stages of *S. neurona* are not infectious between intermediate hosts, and the sexual life cycle is obligatory. The extent to which uniparental mating versus outcrossing is impacting the population genetic structure of *S. neurona*, or its capacity to generate and expand specific strains capable of causing disease epidemics, is not currently known.[1820] This lack of knowledge certainly underscores the necessity to expand sample collection among intermediate and definitive hosts, to increase the number of informative polymorphic phylogenetic markers to improve the resolution of the current MLST typing scheme, and to pursue whole genome comparative studies in order to produce an accurate genetic history model for the species and assess the extent to which genetic recombination is impacting the parasite's population genetics in this genome-sequencing era.

Recently, a novel Type XIII *S. neurona* genotype was discovered; it was associated with severe disease in marine mammals in the north eastern Pacific Ocean.[210]

3.2.7.6 S. neurona *Genome*

As a genus, *Sarcocystis* arguably exists as the most successful protozoan parasite in nature, largely because all vertebrates, including birds, reptiles, fish and mammals can be infected by at least 1 *Sarcocystis* species. Despite its widespread prevalence and the relative ease of constructing drug-resistant, transgenic and gene-specific mutants in *S. neurona* that can be propagated *in vitro* in a variety of vertebrate cell lines, development of this parasite as a model system for genetic analyses has been hampered by the lack of a physical or genetic map, because *S. neurona* chromosomes do not condense and cannot be resolved by pulse–field gel electrophoresis. The recent whole genome shotgun sequencing of the *S. neurona* SO SN_1 genome, has rectified this knowledge gap and produced the first molecular karyotype for the genus, which should greatly facilitate future genetic and comparative genomic studies on this important pathogen.[109]

Combining Roche 454 and Illumina Hi-Seq reads at ~375X coverage, the *S. neurona* genome assembled into a molecular karyotype of 116 genomic scaffolds with a combined size of 127 Mbp, which was over twice the size of the *N. caninum*, *T. gondii*, and *H. hammondi* genomes.[1456,1806] The existence of a high proportion of repetitive Type II transposons, DNA- and LINE-element sequences, totaling 31 Mbp, accounted for nearly half of the increased genome size, with the remaining due to increased average intron length and intergenic region sizes, that were >3X that of *T. gondii*. The largest scaffold was 9.2 Mbp in length, and an additional 3.1 Mbp of sequence was encoded within 2950 unscaffolded contigs (that were each >500 bp in size). When RNA–Seq

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data was combined with the genomic data into an annotation pipeline, a complement of 7093 genes were identified, 5853 of which were expressed during merozoite growth. The predicted gene complement was similar to that found in *N. caninum*, *T. gondii*, and *E. tenella*; however, only limited chromosome-wide synteny was observed for homologous genes between *S. neurona* and *T. gondii*, with the largest block comprised of only 43 genes. This established that significant genome rearrangement between *Sarcocystis* spp. and that of *T. gondii* and *N. caninum* has occurred, which are largely syntenic across all chromosomes. In addition to its nuclear genome, the *S. neurona* apicoplast genome was largely conserved across the coccidia, with a few key differences. *S. neurona* has lost its *rpl36* gene, it had only one copy of the *tRNA-Met* ORF, and has two distinct RNA polymerase C2 genes. In common with *T. gondii*, it was missing *ORF A*. Furthermore, genome comparisons showed that *S. neurona* shared more orthologs with *T. gondii* (3169 genes) than with *Eimeria* spp. (1759 genes), indicating that *Sarcocystis* spp. is more closely related to *T. gondii*, than to *Eimeria* spp. Additionally, *Sarcocystis* spp. encoded 1285 (18%) genes that showed no detectable homology with any other species, underscoring the opportunity for investigators to now identify new gene families within the *Sarcocystis* genus, that promote their success in nature.

The tissue-encysting coccidia have evolved many families of dense granule (GRA), rhoptry kinase (ROPK), microneme (MIC), and surface protein adhesins, known collectively as the SRS, to promote their ability to disseminate infection among their intermediate hosts in order to establish long-term, chronic infections bearing infectious, transmissible sarcocysts. Comparative genomic, transcriptomic and metabolic data analyses between *S. neurona* and other coccidian genomes, has established that the *S. neurona* invasion machinery is largely conserved, but that it only has a limited set of ROPK and GRA proteins. Fifteen ROPK orthologs have been identified, which is significantly smaller than *E. tenella* (n = 27), *N. caninum* (n = 44), and *T. gondii* (n = 55). Moreover, none of the ROPK proteins previously identified as important murine virulence genes (ROP5, ROP18), or those that have the ability to alter host immune effector function by altering STAT3/6 (ROP16) or MAP kinase signaling (ROP38), are encoded within the *S. neurona* genome, indicating that *S. neurona* pathogenesis and infectivity is not dependent on the inactivation of these pathways, and may also explain why this parasite is not infectious to rodents in nature. In addition, *S. neurona* did not encode orthologs of the majority of GRA proteins expressed by *T. gondii* and *N. caninum*, including GRA6, GRA15, GRA24, and GRA25, which are known to regulate NFAT4, NF-kB, p38a MAP kinase and CXCL1/CCL2 levels, respectively, during acute infection. In fact, only 2 GRA protein orthologs have been identified in *S. neurona*, GRA10, and GRA12. This data underscores the differences between *S. neurona* and its related coccidia (*T. gondii* and *N. caninum*), and may indicate that *Sarcocystis* does not require an expanded repertoire of GRA and ROPK genes to promote its ability to recrudesce infection simply because, once encysted, it appears to undergo a terminal commitment to its gamont stage, thus requiring access to its definitive host to complete its life cycle.

3.2.8 Serologic Prevalence

3.2.8.1 Equids

Prevalence of antibodies is dependent on the distribution of opossums in the area, type, and age of horses sampled, geographical location, and the serological test used (Table 3.6). Sera in several studies were tested in 1 laboratory, by using immunoblots against low molecular proteins (marked WB[b] in Table 3.6). In these studies, prevalence was around 50% of horses tested. Seroprevalence increased with age.[93,114,419,1496,1535,1750,1863] In these surveys, the horses tested were mostly older than 1 year of age. It is likely that some of the foals tested had maternal-acquired antibodies.

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Table 3.6 Seroprevalence of *S. neurona* Antibodies in Equids

Location	Year Sampled	Type, Source	No. Tested	Test, Cut-Off	Positive		Reference
					No	%	
Argentina	1996	Chaco Province	76	WB[a]	27	35.5	**419**
Argentina	2006–10	9 provinces	640	WB[a]	167	26.1	**1220**
Brazil	1998	Thoroughbreds	101	WB[a]	36	36.0	**417**
	NS	10 states	961	ELISA SAG4[b]	669	69.6	**838**
	NS	Rio Grande do Sul	181	ELISA-crude extract	61	33.7	**1397**
Canada	2001	Western Canada	239	WB[a]	0	0	**454**
Costa Rica	NS	7 provinces	315	SnSAG2 ELISA[b]	133	42.2	**292**
France	NS	2 farms in Normandy	50	SAT	18	36.0	**1396**
India	2003	3 states	123	WB[a]	1	0.8	**146**
Korea	NS	Thoroughbred Jeju island	191	WB	0	0	**752**
Mexico	NS	Durango state	495	rSnSAG2/4/3 ELISA	240	48.5	**1863**
Spain	NS	Galicia	384	SnSAG2 ELISA[c]	9	2.3	**32**
USA PA	1993–94	Thoroughbred farms[d]	117	WB[a]	53	45.3	**92**
OR	NS	Horses from 4 regions, >3 months old	334	WB[a]	149	45.0	**114**
OH	1993–94	37 breeds, statewide[e]	1056	WB[a]	560	53	**1535**
CO	1995–96	593 horses and 15 ponies	608	WB[a]	204	33.6	**1750**
OK	2001	Banked sera	798	WB[a]	712	89.2	**93**
WY	NS	Wild horses	276	WB[a]	18	6.5	**454**
				SAT, 1:50	39	14.1	
CA, FL, MO, MT	NS	Sera, diagnostic labs	208	IFA, 1:100	49	26.0	**1779**
MI	1997	1056 horses, 63 ponies, 3 donkeys, 1 mule from 98 herds	1121	WB[f]	627	56.0	**1496, 1499**
40 states	2010–11	40 states, sera submitted to diagnostics laboratory	3123	IFAT, 1:80	865	27.6	**1433**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
CA = California, CO = Colorado, FL = Florida, MI = Michigan, MO = Missouri, MS = Mississippi, MT = Montana, OH = Ohio, OK = Oklahoma OR = Oregon, PA = Pennsylvania, WY = Wyoming.
NS = not stated.

[a] Western blot, Granstrom et al.[728]
[b] Hoane et al.[836]
[c] Reactive to 30–35 kDA protein. Of these, 26.1% reactive to 16–17 kDA protein, 5 (3.62%) reactive to rSnSAG2 ELISA, 4 (3%) reactive in SnSAG4/3 ELISA.
[d] 20% of horses from each farm with a population of 580 horses; 4-week-old to 26-year-old, one 4-week-old might have colostrally acquired antibodies.
[e] Every 36th sample submitted to diagnostic laboratory for equine infectious anemia.
[f] WB, blot treated with bovine antibodies against *S. cruzi* antigen.[1495]

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In 1 study, foals born to 33 seropositive mares were bled before suckling, 1 day after colostrum ingestion, and again at monthly intervals.[247] All foals were seronegative before suckling, all became seropositive 1 day after suckling, and 31 of 33 became seronegative by 9 months of age. The decay of antibody was probably related to the concentration of IgG in the colostrums. In another study, the median time of decay of maternal-acquired *S. neurona* antibodies in foals was 96 days, and these antibodies disappeared by 230 day of birth.[344] These results are in contrast to another study[1397] that reported antibodies in 61 (37.3%) of 181 mares at parturition, and in presuckle blood samples of 6.6% foals born to these mares. These observations need confirmation, because current evidence indicates that transplacental or lactogenic transmission of *S. neurona* in horses is very uncommon or absent. In an epidemiologic investigation of *S. neurona* seropositivity of horses in California tested by IFAT, there was no evidence for the transplacental transfer of *S. neurona* antibodies or parasite; all 366 presuckling foal sera were seronegative.[345] Another study of 174 foals born to mares on a farm with very high seroprevalence to *S. neurona* (90%) found only a single foal with a presuckle antibody titer.[1432]

Opossums are the only known definitive hosts for *S. neurona*, and they are found only in the Americas. Theoretically, therefore, horses in other countries should have no exposure to *S. neurona*. Consistent with this, *S. neurona* antibodies were not detected by immunoblotting in any of 191 horses from South Korea,[752] and in only 1 of 123 horses born in India.[146] However, seropositivity in horses from France[1395,1396] and Spain[32] is puzzling. In 28 French horses with EPM-like clinical signs, *S. neurona* antibodies were found by WB.[1395] In a followup study, they found seropositivity in 18 (36%) of 50 healthy horses tested by the SAT (*S. neurona* direct agglutination test). Even more intriguing are results from horses tested from Spain. Of 138 horses tested by immunoblotting, 26.1%–82.6% of horses were seropositive, based on the antibodies directed against immunodominant molecules at low (16–17 kDa) or high (30–35 kDa) molecular weights (MW); notably, Granstrom et al.[728] used antibodies directed against proteins lower than 16 kDA for interpretation of the WB. Retesting of these 138 samples with the rSnSAG2 ELISA and rSnSAG4/3 ELISAs, revealed that only 5 sera (3.6%) were reactive in rSnSAG2 ELISA, and only 4 (3%) were reactive in rSnSAG4/3 ELISAs; only 1 serum was strongly reactive in both types of ELISA tests.[32] Further, sera from 246 horses were tested by rSnSAG2 ELISA; 9 (2.34%) of 384 were seropositive. None of these 384 horses travelled out of Spain. Consequently, it was suggested that the immune-reactivity observed in the immunoblots was due to cross reactivity with another species of *Sarcocystis* that infects horses in Spain.

It has been suggested that there might be another definitive host for *S. neurona* in Europe, or there might be cross reactivity among *Sarcocystis* species in horses to account for the seropositivity in European horses.[1396] In this respect, another species of *Sarcocystis*, *S. bertrami* (with thin-walled sarcocysts—see Chapter 11) is found in horses in Europe.[393] Whether there is cross reactivity between *S. bertrami* and *S. neurona*, and if the type of antibody testing, or antigen preparations, account for this reactivity, has not been completely investigated. In the United States, only 1 non-*S. neurona* species of *Sarcocystis*, *S. fayeri*, has been found in horses. To resolve the question of cross reactivity between *S. fayeri* and *S. neurona*, 3 seronegative ponies were orally inoculated with 10^5, 10^6, or 10^7 *S. fayeri* sporocysts collected from dogs fed infected muscle obtained from horses in Texas.[1539] The fourth pony was an uninoculated control. Sera from ponies were tested by employing 3 tests (WB, IFA, and SAT) before dosing sporocysts, and then weekly, thereafter. With the WB using the strict interpretation criteria that were previously established,[728] antibodies specific to *S. neurona* were not found in any pony at 0, 2, 37, and 79 DPI. This negative reactivity was further confirmed using a recombinant SnSAG1 protein.[752] By SAT, only 1 sample collected 37 DPI from the pony dosed with 10^7 *S. fayeri* sporocysts was positive at 1:50 dilution. By contrast, all 3 inoculated ponies were seropositive by IFA, upto 1:400 dilution, whereas the control pony was seronegative.[1539] This experiment provides conclusive evidence that *S. neurona* infection can be distinguished from *S. fayeri* infection. It is important that the WB results are interpreted correctly,

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because it was apparent that these *S. fayeri*-infected ponies had antibodies that recognized numerous *S. neurona* antigens.[836]

3.2.8.2 Cats and Other Animals

Antibodies were found in 1%–40% of cats in the United States (Table 3.7). As a contrast, *S. neurona* antibodies were not found by SAT in any of the 502 domestic cats from Brazil.[448]

Antibodies (IFAT, 1:25) were found in 2 of 63 capybaras (*Hydrochoerus hydrochaeris)* and 8 of 11 free-living jaguars (*Panthera onca*) from Brazil.[1771,2397]

Antibodies to *S. neurona* were not found in experimentally[208] or naturally exposed[847] opossums.

Antibodies were detected in skunks following oral inoculation of sporocysts.[204]

Table 3.7 Serological Prevalence of *S. neurona* in Nonequid, Nonmarine Mammals in the United States

Host	Location	Year	Type	No.	Test, Cutoff	No. Positive (%)	Reference
Cat (*Felis catus*)	VA			232	IFAT, 40	22 (9.0)	**852**
	PA			209		10 (5.0)	
	MS	NS	Stray	9	WB	1 (11.1)	**1765**
	MI	1999–01	Pets	196	IFAT, 20	10 (27)	**1497**
	OH	2001	Horse farms	35	SAT, 25	14 (40.0)	**1674**
		2001	Spay/neuter	275	SAT, 25	27 (10.0)	
	FL	NS	Humane shelter, feral	100	WB	5 (5.0)	**684**
Skunk (*Mephitis mephitis*)	CT	NS		24	SAT, 50	11 (46)	**1201**
Raccoon (*Procyon lotor*)	CT			12		100	**1201**
	PA, MA, FL, NJ			99	SAT, 50	58 (59.6)	**1078**
	CT			12		12 (100)	**1201**
	VA	2001–02	Feral	469	SAT, 50	433 (92.3)	**779**
Opossum (*Didelphis virginiana*)	CT			7		0	**1201**
	FL	NS	Feral	20	SAT, filter paper	0	**208**
Beaver (*Castor canadensis*)	MA			62	SAT, 25	4 (6.0)	**915**
Ring-tailed lemur (*Lemur catta*)				52	SAT, 50	2 (1.9)	**1850**
Black-and-white ruffed lemur (*Varecia variegata*)				4		1 (25.0)	
Black-eyed lemur (*Eulemur macaco flavifrons*)				6		0	
Armadillo (*Dasypus novemcinctus*)	FL	NS		2	SAT, 1:50	2 (100)	**208**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
CT = Connecticut, FL = Florida, NJ = New Jersey, MA = Massachusetts, MS = Mississippi, OH = Ohio, PA = Pennsylvania, VA = Virginia.
SAT = *S. neurona* agglutination test, IFAT = indirect fluorescent antibody test, WB = Western blot.

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3.2.9 Clinical Infections

3.2.9.1 Horses, Ponies, and Zebras

EPM is often a progressively debilitating disease affecting the CNS of horses.[87,88,138,141,217,270,328,333,438,602,777,1082,1090,1091,1096,1128,1147,1148,1210,1225,1489,1554,1555,1578,1755,1761,1992,2085,2115,2121,2122,2153–2155,2177,2178,2246,2262,2285,2297,2377–2388] The clinical signs can vary from acute to insidious onset of focal or multifocal signs of neurologic disease involving the brain, brainstem, spinal cord, or any combination of the areas of the CNS. Some horses affected with EPM have abnormal upper airway function, unusual or atypical lameness, or even seizures. In severe cases, the horse can have difficulty with standing, walking, or swallowing, and the disease can progress very rapidly. In some horses, the disease appears to stabilize or remain static for a time period.

The early clinical signs, of stumbling and frequent limb interference, are often easily confused with a lameness of either the thoracic and/or the pelvic limbs. In many horses, the disease tends to have a gradual progression of clinical signs, including ataxia, but, in some horses, mild clinical signs followed by a rapidly progressive course have been observed. Blood monitoring of these horses, using either Western blot, IFAT or ELISA testing, often results in dramatic changes in antibody concentration. On physical examination, the vital signs are usually normal, although variations in body condition, from very thin to obese, along with clinical depression, might also be observed. Neurological examination often reveals an asymmetric weakness, ataxia and spasticity, involving all 4 limbs. Frequently, areas of hypoalgesia or complete sensory loss may be noted. The most frequent brain or cranial nerve deficits observed in horses appear to be head-tilt, depression, facial nerve paralysis, difficulty in swallowing, upper airway dysfunction, such as dorsal displacement of the soft palate and laryngeal hemiplegia, although signs are not limited to these areas (Figure 3.2). Gait abnormalities are often a result of damage to the brainstem or spinal cord, and can be quite variable, depending on the location and severity of the lesion.

Most horses affected with EPM are bright and alert at presentation; however, any horse with signs of neurologic disease is a candidate to have EPM. At the time of initial examination, most horses have normal blood values. One of the most helpful clinical signs is that horses with EPM often have asymmetric gait deficits, with focal muscle atrophy. This can be a useful differentiating feature, and may help direct one towards a clinical diagnosis of EPM, rather than another neurological disease.

The pathogenesis of EPM is not clear. Clinical signs of EPM are dependent on the area of the CNS parasitized. For example, involvement of the cerebrum can cause depression, behavioral changes, or seizures. Lesions in the brainstem and spinal cord often cause gait abnormalities, incoordination caused by involvement of ascending and descending tracts, and any of a variety of signs attributable to damaged cranial nerves. Severe damage in the gray matter that innervates muscles of the limbs can produce weakness, followed by atrophy of innervated muscles. The quadriceps and gluteal, as well as the temporalis muscles, are often atrophied; *S. neurona* has not been found in affected muscles.

Factors governing severity of EPM are unknown.[438,475] Clinical EPM is often reported in well-cared race horses 3–6 years of age. Clinical EPM does not seem to be associated with poor nutrition or known concurrent infections. There are no confirmed reports of clinical EPM in horses younger than 6 months of age. EPM-like disease in a 2-month Appaloosa colt that had facial paralysis beginning 2 days after birth was reported.[731] *S. neurona* antibodies were found in its CSF by immunoblot, and it responded favorably to pyrimethamine and trimethoprim–sulfadiazine medication.

EPM has been confirmed histologically only in horses born and raised in the Americas, coincident with the geographical range of opossums. However, EPM has been diagnosed in horses exported from the Americas. In Japan, *S. neurona* was confirmed by IHC in a thoroughbred 15 months after importation from Kentucky.[945] From the published reports, it appears that EPM is rare

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in equids other than horses. One case was diagnosed in a 10-year-old pony from Maryland.[387,422] EPM was also reported in an 8-year-old Grant's zebra (*Equus burchelli bohmi*) in California.[1117]

Prevalence of EPM in horses was estimated at 0.5%–1% of the horse population.[438] Clinical disease is sporadic, and more than 1 case is seldom seen at a particular farm. However, clusters of cases have occurred in a few instances, which would suggest that all of the risk factors necessary for disease were at those facilities. EPM has been reported in siblings.[1761]

The pathogenesis of *S. neurona* in horses is unclear, because it has been difficult to reliably induce disease experimentally in horses. Attempts at inducing EPM in horses are summarized in Table 3.8. Although horses developed clinical disease, attempts to demonstrate *S. neurona* in histological sections were essentially unsuccessful. Several factors, including the stage of the parasite inoculated, dose, age of the horse, immunological status, endogenous and exogenous stress during experiment, were examined by different investigators. Clinical outcome was not affected when sporocysts were derived from naturally infected versus experimentally infected opossums. The dose did not significantly affect outcome, because feeding as few as 100 sporocysts induced clinical disease.[1636] Administration of corticosteroids to horses also did not affect the outcome of the experiments.[272,1538] A transport stress model proved valuable in helping to induce clinical EPM. In a series of experiments, young horses selected from northwest Canada, where opossums are not present, were transported by road for 55 h and then dosed with *S. neurona* sporocysts as the horses were unloaded from trucks. In the transport-stressed horses, seroconversion to *S. neurona* was sooner and clinical signs were more severe than in horses not stressed. A second transport-related stress after a 2-week rest did not affect the severity of EPM.[1538,1540] Arabian foals with severe SCID, lacking T and B cells, were successfully infected with *S. neurona*, with demonstrable live parasites in blood and tissues; however, these foals did not develop clinical signs of EPM.[1083,1579] Parasitemia was demonstrable in an immunocompetent horse dosed daily with *S. neurona* for 112 days.[1500] All 5 horses inoculated with cultured *S. neurona* merozoites directly in the subarachnoid space seroconverted, but only 1 developed clinical signs.[1074] *S. neurona* can circulate in equine tissues within lymphocytes.[1080] To facilitate dissemination of *S. neurona* merozoites, and their entry into the CNS, equine peripheral blood mononuclear cells (PBMCs) were first infected with merozoites *in vitro*, and, after 5 h incubation, were inoculated intravenously back into the horses.[501,502] Five of the 6 inoculated horses developed signs of clinical EPM starting the first week PI. This experiment was repeated with 8 additional horses, with similar results. [1833]

Immunity is considered to play an important role in the clinical outcome of EPM, but data from naturally and experimentally infected horses is not conclusive.[642,644,1121,1279,1430,1574,1662,1663,1756,1833,1855,2267] Interferon gamma is essential for controlling the development of EPM, as evidenced by studies in mice. Immunocompetent outbred and inbred BALB/c or C57Bl/6 mice are not susceptible to *S. neurona* infection, whereas the parasite is fatal to either BALB/c or C57Bl/6-derived KO mice.[412,470,1502,1830,1831,2303] Clinical disease develops in KO mice. Both KO and nude mice are susceptible to infection and disease development, whereas SCID mice do not show signs of parasite survival or disease development.[1114,1580] The humoral response is less critical in protection, as suggested by the fact that B cell-deficient mice are not susceptible to infection, and mice that die of severe disease have antibodies to *S. neurona*.[1832]

3.2.9.2 Marine Mammals (Sea Otter, Seal, Porpoise, Sea Lion)

3.2.9.2.1 Sea Otter (Enhydra lutris)

Reports of *S. neurona* infection in sea otters are summarized in Table 3.9. Among marine mammals, causes of mortality are best known for sea otters because, during long periods of time, all dead sea otters recovered by wildlife services were examined at necropsy. Sea otters are resident on

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Table 3.8 Attempts to Induce Clinical Disease in Horses after Inoculation with *S. neurona*

No.	Type	*S. neurona*	Clinical Disease	Duration (days)	Diagnosis		Reference
					Bioassay	Histology	
5	3–6-month-old foals	Naturally infected opossums (NIO)	Clinical signs in all, 28–42 DPI, neurological, lesions in spinal cord of 4	40–110	Negative	Negative	**603**
8[a]	3–4 years	NIO	Spinal cord lesions in 7 of 8 horses	90	Negative	Negative	**272**
9[a]	Foals	NIO	Clinical signs more severe in stressed[b] horses	44–63	Negative	Negative	**1538**
20[a]	4–5 months	Experimentally infected opossums (EIO)[c]	Dose response 10^2, 10^3, 10^4, 10^5, 10^6, 4 horses per group Clinical signs in all, histologic lesions inconsistent	30	Negative	Negative	**1636**
24[a]	4–5 months	EIO[c]	Stressed horses[b], second time[d]	40	Negative	Negative	**1540**
6		EIO[c]	100–1000 sporocysts daily for 112 days		Positive, blood of 1 horse	No data	**1500**
1	Arabian 5-month-old SCID foal	NIO	Knuckling 39 DPI, euthanized 53 DPI. *S. neurona* isolated from brain and blood	53	Positive		**1083**
2	Arabian	NIO	Neurological signs in 1	90	Negative	Negative	**1579**
4	Arabian	500 million merozoites from culture, inoculated intravenously	3 horses neurological	41, 73, 80, 471	Positive, brain of 1 horse	Negative	**1579**
1	Arabian SCID foals	500 million merozoites from culture, inoculated intravenously into 1 foal	No clinical signs	21,32	Li, Sp, Sk of 1 foal, H and Sk of second foal	Negative	**1579**
2	Arabian SCID foals	NIO	No clinical signs	96	Li, Lu, Sp, H, Sk	Negative	**1579**
5	2–17 years, mixed breed	5×10^5 to 5×10^6 merozoites inoculated intrathecally	No clinical signs	132	Not done	Negative	**1074**
3	1–10 years	100, 1,000 and 10,000 cultured merozoites daily for 15 days	All horses developed clinical signs	90	Not done	Positive	**502**

(*Continued*)

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Table 3.8 (*Continued*) Attempts to Induce Clinical Disease in Horses after Inoculation with *S. neurona*

No.	Type	*S. neurona*	Clinical Disease	Duration (days)	Diagnosis		Reference
					Bioassay	Histology	
8	1–19 years, several breeds	6000 merozoites from culture inoculated intravenously, daily for 14 days	All horses developed clinical signs	55	Not done	No data	**1833**
15[a]	2–15 years, standardbred and mixed breeds	612,500 sporocysts NIO	Mild ataxia in 3	84		Negative	**1092**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
SCID = severe combined immunodeficiency disease.
H = heart, Li = liver, Lu = lung, Sk = skeletal muscle, Sp = spleen.
[a] Horses obtained from Northwest Canada outside the range of opossums.
[b] Transport stress model. Horses transported for 55 h and dosed soon after arrival.
[c] Isolate SN-37R-obtained by serial passage between laboratory raised opossums and raccoons.
[d] Horses transported after rest period.

the coasts of California, Washington, and Alaska in the United States, British Columbia in Canada, and in Russia, but *S. neurona* has been definitively identified only in sea otters from California and Washington, which is consistent with the distribution of opossums. Data in Table 3.9 are based on dead or sick animals. *S. neurona* high titer antibodies were found in 2 of 74 sea otters from Kodiak, United States, but not in 89 otters from Bering Island, Russia.[713]

Most diagnosed cases were from southern sea otters (*Enhydra lutris neresis*) from California, compared with northern sea otters (*Enhydra lutris kenyoni*) from Washington (Table 3.9). Since 1992, more than 1000 sea otters were examined at necropsy, and protozoal encephalitis was identified as an important cause of their mortality.[978,1197,1739,2068,2375] Among numerous causes of mortality in 105 sea otters, *S. neurona* encephalitis was diagnosed in 7 individuals, which was an underestimate because diagnosis was made only when protozoa were identified in lesions based on conventional histological examination; immunohistochemistry was not performed.[978] In a detailed study of encephalitic lesions, *S. neurona* was identified alone in 22 of 39 cases of encephalitis, and dual infection, with *T. gondii*, in another 12 otters among 344 sea otters. Most of these deaths were sporadic.[1739]

An epizootic of protozoal encephalitis in sea otters has been recorded.[1196,1197] Within 1 month, 63 sick and dead otters were recovered near Morro Bay, California. This outbreak provided the opportunity for epidemiologic and etiological investigations. Seizures, muscle tremors, and paresis were noted in live stranded otters. A few animals had tachycardia and respiratory distress.[1197] Of these otters, 15 were studied in detail. Antibodies (IFAT >2560–81,920) to *S. neurona* were present in all 14 adult otters; 1 pup was seronegative (<1:40). Viable *S. neurona* were isolated from the brains of 8 adults. Predominant gross findings were enlarged heart with mottling, lymphadenopathy, and adipose petechiation. Microscopically, meningoencephalitis associated with schizonts and myositis associated with sarcocysts predominated. Immature and mature sarcocysts were seen in muscles, heart, and brain.[1196,1197]

In an epidemiological investigation, land-based runoff was associated with *S. neurona* mortality in sea otters.[1590] *S. neurona* was identified in 33 of 205 sea otters; however, it is not certain if these were new cases or previously included in the reports listed in Table 3.7. Increased runoff due to rain in the prior 1–2 months was 12 times more likely to be associated with *S. neurona* mortality.[1590]

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Table 3.9 Reports of Clinical *S. neurona* Infections in Sea Otters in the United States

Location	Year	No. of Sea Otters Examined	No. Positive	Observations, Tissues Parasitized	Bioassay	Reference
Zoo, OR	1997	1	1	Antemortem diagnosis, biopsy muscle, CSF–WB positive, medicated with pyrimethamine, euthanized. Schizonts in brain, sarcocysts in muscle	Not done	**1494**
Monterey Bay, CA	2000	1	1	Meningoencephalitis, schizonts in brain, sarcocysts in muscle and heart	Positive	**1072**
Grays Harbor County, WA	2000	1	1	Convulsions, died. Meningoencephalitis, schizonts in brain. Coinfected with *T. gondii*	Positive	**1077**
Monterey Bay, CA	1999	1	1	Pup estimated 16-weeks old. Meningoencephalitis, schizonts in brain	Positive	**1195**
CA	1998–01	105	7	Only conventional histology. Encephalitis, schizonts in brain. Causes of mortality detailed. *T. gondii* in 17 otters	Not done	**978**
CA, WA	1985–04	344	39	Encephalitis in 17 otters from CA and in 5 from WA. *S. neurona* alone in 22 otters, dual *S. neurona* and *T. gondii* in 12, *T. gondii* in 7. Sarcocysts in muscles of all of 18 otters examined	Not done	**1739**
CA	2004	3	3	Lymphadenopathy, enlarged heart. Encephalitis, schizonts in brain. Sarcocysts in brain and muscle, proven by TEM	Positive	**1196**
Morro Bay, CA	2004	63 otters dead in an epidemic	8	16 necropsied and tested for *S. neurona*. *S. neurona* antibodies in 15, 1 pup seronegative. *S. neurona* isolated from 8, *T. gondii* isolated from 6. Encephalitis and myositis in 15	Positive	**1197, 1820, 1821**
Copalis Beach, WA	2010	1	1	Retinitis and encephalitis with schizonts	Not done	**468**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
CA = California, OR = Oregon, WA = Washington.

Encephalitis is the predominant lesion in sea otters, and both *T. gondii* and *S. neurona* can be found together, sometimes in the same histological section. In general, the *S. neurona*-associated lesions are more severe than those associated with *T. gondii*.[1739]

3.2.9.2.2 Pacific Harbor Seals (Phoca vitulina richardsi)

There are few reports of *S. neurona* mortality in seals. Encephalitis was found in 7 seals; 6 of them were stranded on the California coast, and the seventh was captive.[1023] Among these seals was a 1-week-old captured juvenile with tremors that lasted 17 days. Schizont-associated encephalitis was found in all 7 animals, and the diagnosis was confirmed immunohistochemically. *S. neurona* antibodies were found by immunoblot in 4 of 5 seals tested. Four seals had myocarditis, and sarcocysts were found in the heart of 1 seal.

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Subsequently, viable *S. neurona* was isolated from an adult seal that was stranded near Monterey, California.[1194] The seal had encephalitis in association with *S. neurona* and *T. gondii* infection. Barbosa et al.[210] likewise isolated viable *S. neurona* from the brains of 4 harbor seals in 2011 and one in 2012 that stranded with severe encephalitis along the Washington state coastline.

Antemortem diagnosis and treatment were reported in a 20-year-old captive seal that developed tremors and dysphagia.[1267] Findings of *S. neurona* antibodies in serum and CSF and negative tests for other infections suggested the diagnosis of *S. neurona*. The animal improved clinically after treatment with ponazuril at 10 mg/kg/oral for 3 months.[1267]

3.2.9.2.3 Pacific Harbor Porpoise (Phocoena phocoena)

Barbosa et al.[210] identified *S. neurona* infection in 32 of 46 (70%) harbor porpoises that stranded along the Washington state coastline between 2006–2012; the majority of these infected animals had mild to severe forms of encephalitis upon histological examination. Notably, however, several animals that were PCR positive had no inflammatory lesions. One isolate recovered in 2011 was genotyped at multiple loci and identified to be a common Type VI strain that is widely distributed throughout the United States.

3.2.9.2.4 California Sea Lion (Zalophus californianus)

S. neurona infection was diagnosed antemortem in a California sea lion stranded ashore in Marin County, California.[165] The animal was lethargic. Acute myositis was diagnosed based on histological examination of biopsy, serum biochemical tests, and finding of elevated antibodies to *S. neurona* (IFAT >1:5120–1:20,480). The ALT and CK values were elevated. Treatment with ponazuril (10 mg/kg/oral for 5 days) resulted in clinical improvement. The animal was returned to its habitat 3 months after it was found stranded.

The finding of *S. neurona* in 9 of 10 asymptomatic sea lions is most interesting. Of the 10 adult healthy California sea lions euthanized to protect fish stocks, 9 were infected with *S. neurona*, and 1 had concurrent *T. gondii* infection.[680] These results were based on the detection of the parasite DNA in samples of heart, brain, muscle, and lymph nodes.

3.2.10 Other Marine Mammals

S. neurona infection has also been documented by molecular PCR and histology in a wide range of other marine mammals, including 11/25 (44%) Guadalupe fur seals (*Arctocephalus townsendi*), 3/8 (38%) Steller sea lions (*Eumatopias jubatus*), 1/5 (20%) Northern fur seals (*Callorhinus ursinus*), 3/3 (100%) Northern elephant seals (*Mirounga angustirostris*), a Pacific white-sided dolphin (*Langenorhynchus obliquidens*), a killer whale (*Orcinus orca*), and a pygmy sperm whale (*Kogia breviceps*).[210,680] These results indicate that *S. neurona* infects a vast array of marine mammal orders, including pinnipeds, cetaceans, and mustelids.

3.2.11 Miscellaneous Animals

Results are summarized in Table 3.10. Unusual findings are commented upon.

3.2.11.1 Raccoons (Procyon lotor)

There are several reports of *S. neurona* encephalitis in raccoons (Table 3.10). These raccoons were examined at necropsy because of suspicion of rabies. A concurrent morbillivirus infection (MVI) is considered to aggravate severity of disease. However, in 2 raccoons, evidence for MVI

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Table 3.10 Reports of Clinical *S. neurona* Infections in Nonequid and Nonmarine Animals in the United States and Canada

Host	Location	No. Age	Clinical Signs	Diagnostic Methods	Organs Parasitized	Reference
Cat (*Felis catus*)	California	1, 13-week-old	Lame after fall, euthanized	IHC	B, Sc	**411, 422**
	Illinois	1, 4-month-old	Neurologic, post surgery	IHC	B, Sc	**450**
	Indiana	1, 5-month	Neurologic, antemortem dignosis	PCR	CSF	**104**
Canada lynx (*Felis canadensis*)	New York Zoo	1, 13-year	Encephalitis	IHC	B	**617**
Dog (*Canis familiaris*)	Illinois	1, 6-year-old	Myositis	IHC, PCR	Sk	**1780, 1987**
	Maryland, Illinois, Arkansas, Oklahoma, Louisiana	7, retrospective	Neurologic, dermal, pneumonia	IHC, PCR	B, K, Lu, skin,Sc	**460, 1988**
	Mississippi	1, 1.5-year-old	Ataxia, neurological	IHC, PCR	B	**249**
	Mississippi	1, 2-month-old	Neurologic	IHC	B, E, L, H, I, Nt, T,	**472**
	Tennessee	1, 2-year-old	Acute onset of parapresis	IHC, PCR	Sc	**670**
Raccoon (*Procyon lotor*)	Ohio	1	Neurological, euthanized	IHC	B	**398, 402, 422**
	Oregon	2	Encephalitis and myocarditis, no evidence of MVI	IHC	B, H	**778**
	New York	1	Neurologic, MVI	IHC	B	**1677, 422**
	Illinois	1	Neurologic, MVI	Hist.	B	**1742**
Mink (*Mustela vison*)	Oregon	2	Neurologic	IHC	B	**408**
Ferret (*Mustela putorius furo*)	British Columbia, Canada	1	Neurologic, respiratory, MVI	IHC, PCR	A, B, H, K, Li, Lu, Nt, Ln, Mo, Sk, Sp,	**145**
Fisher (*Martes pennanti*)	Maryland	1	Neurologic	IHC, PCR	B	**671**
Striped skunk (*Mephitis mephitis*)	Indiana	1	Neurologic	IHC, PCR	B, Li, Lu, Nt	**153**
Bald eagle (*Haliaeetus leucocephalus*)	Missouri	1	Neurologic signs	IHC	B	**1333**

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
A = adrenal, B = brain, H = heart, I = intestine, Li = liver, Lu = lung, Mo = blood monocytes, Nt = nasal turbinates, Sk = skeletal muscle, Sc = spinal cord, Sp = spleen, T = tongue.
MVI = concurrent Morbillivirus Infection.

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was not found; in these animals, the encephalitis was granulomatous rather than pyonecrotic or pyogranulomatous, as seen in animals with concurrent MVI.[778] The most severe disease diagnosed was in a juvenile raccoon.[398] The raccoon had neurological signs, was unsteady, and could not maintain its balance. Within a week of onset of clinical signs, the raccoon's condition deteriorated, it was euthanized, and a complete necropsy was performed. Protozoa-associated lesions with numerous *S. neurona* schizonts and free merozoites were confined to cerebrum.

3.2.11.2 Mink (Mustela vison)

On a farm in Oregon, United States, 50 of 15,500 minks died within 2 weeks.[408] The affected minks were weak, ataxic, and anorectic. Complete necropsy was performed on two 1-month-old female minks. Grossly, a discolored area was found in the cerebrum of 1 of the 2 minks. Microscopically, both minks had severe meningoencephalitis associated with numerous schizonts of *S. neurona*. Minks with progressive neurologic disease can also have a variable number of sarcocysts present.[1445]

3.2.11.3 Domestic Cat (Felis catus)

Three reports of *S. neurona*-like infection in cats need explanation because of unusual presentations. The first cat diagnosed was a 3-month-old that became ill 2 weeks after a fall, and developed hemiparesis.[411] The second cat developed neurological signs 3 days after routine surgery for castration.[450] In both cats, lesions and *S. neurona* schizonts were confined to CNS. Additionally, *S. neurona*-like sarcocysts were seen in the brain of the second cat. In the third cat, the diagnosis was made antemortem, and, thus, a detailed clinicopathologic could not be documented.[104]

Efforts to induce clinical disease in cats were unsuccessful. Four inoculated with 10 million culture-derived *S. neurona* merozoites[157] and 5 cats orally inoculated with >1000 *S.neurona* sporocysts[429] became infected but remained subclinical; 3 of these cats had been administered rather high doses of methylprednisone acetate.[429]

3.2.11.4 Dog (Canis familiaris)

Of the 10 dogs diagnosed with *S. neurona*-like infections (Table 3.10), 3 reports need an explanation because of unusual presentations. The first case was a dog from Illinois.[401] Protozoa were seen in the skin of this dog. The dog was misdiagnosed as having an *S. canis* infection.[400] Subsequently, it was diagnosed as *S. neurona*, based on immunohistochemistry.[460] This dog had pustular dermatitis with many merozoites and schizonts found in a smear made from the oozing fluid (Figure 3.5d).

A 2-month-old dog was thought to be congenitally infected with *S. neurona*-like organisms; it had focal severe encephalitis, retinitis (Figure 3.5a and b), and myositis associated with schizonts and merozoites.[472] Mature sarcocysts were found in its skeletal muscle. Schizonts were also present in sections of nasal turbinates, but without inflammation.

Also, a 2-year-old dog had acute onset of paraparesis.[670] At necropsy, large nodular masses were found between nerve roots of the thoracic spinal cord (Figure 3.3). Microscopically, the nodular growth had mixed cellular infiltrates and necrosis associated with protozoa, thought to be *T. gondii*. However, these structures were *S. neurona* schizonts (not *T. gondii*) in different stages of maturation; they reacted strongly to *S. neurona* antibody.

Attempts to induce clinical disease in dogs were unsuccessful; 2 dogs orally inoculated with 1,000,000 sporocysts remained subclinical, despite administration of corticosteroids (J.P.D, unpublished).

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3.2.11.5 Ferret (Mustela putorius furo)

The case of an infected ferret is unusual for 2 reasons. First, the animal had massive infection of *S. neurona* affecting the nasal turbinates.[145] Second, the protozoal infection might have been triggered by vaccination with live modified MVI vaccination. The ferret had been administered 1 dose of live canine distemper virus vaccine (CDV) 1 week before onset of rhinitis. In dogs, vaccination with CDV can trigger *T. gondii* infection.[464]

3.2.11.6 Canada Lynx (Felis lynx canadensis)

A 13-year-old lynx from a zoo had granulomatous encephalitis, both in grey and white matter throughout the cerebral cortex.[617] Schizonts were seen in neuropil as well as in vascular endothelium.

3.2.11.7 Fisher (Martes pennanti)

A wild-caught, ataxic fisher was found to have no fear of humans and was attacked by a dog.[671] After euthanasia, a complete necropsy was performed to exclude rabies. Numerous *S. neurona* schizonts were identified in encephalitic lesions, and the diagnosis was confirmed by PCR. Additionally, mature *S. neurona*-like sarcocysts were identified in the skeletal muscle.

3.2.11.8 Striped Skunk (Mephitis mephitis)

Disseminated *S. neurona* infection was diagnosed in a juvenile skunk with concurrent MVI.[153] Schizonts were seen in sections of brain, lung, liver, and the nasal epithelium.

3.2.11.9 Bald Eagle (Haliaeetus leucocephalus)

Sarcocystis-associated encephalitis was diagnosed in a captive bald eagle.[1333] Confirmation of *S. neurona* infection was needed because this was a unique report of this parasite in an avian species, and because another *Sarcocystis* species, *S. calchasi*, was diagnosed to cause encephalitis in birds[1329,1843] (see Chapter 17).

3.2.12 Diagnosis

3.2.12.1 Horses

3.2.12.1.1 General Observations

Although many neurologic disorders affect the horse, EPM remains the most commonly diagnosed infectious equine neurologic disease in the Americas.[438,1224,1225] A complete neurologic examination and implementation of a thorough diagnostic plan to rule out differential diagnoses are essential prerequisites to laboratory testing and appropriate interpretation of clinical signs and death. Many ancillary diagnostic procedures may be required to differentiate primary musculoskeletal disorders and other neurologic diseases from EPM.

3.2.12.1.2 Blood Chemistry

EPM does not produce consistent detectable changes in complete and differential cell counts or serum chemistry values. General analysis of CSF is also not informative. *S. neurona* merozoites have not been confirmed in CSF.

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3.2.12.1.3 PCR

PCR testing of equine CSF may provide information regarding the presence of *S. neurona* DNA in the CNS. However, the sensitivity of the PCR test appears to be much lower than initially estimated, possibly because intact merozoites rarely enter CSF. Nonetheless, the PCR test may be a useful adjunct for the postmortem diagnosis of EPM in selected cases, when it is used on tissues collected at necropsy.

3.2.12.1.4 Serological Tests

Several serological tests have been used to detect antibodies to *S. neurona* in animals. The WB test, also called the immunoblot test, was the first assay developed for detection of antibodies against *S. neurona*, and diagnosis of EPM.[728] This assay has been available for over 2 decades and continues to be offered by several diagnostic testing laboratories.[230,1091,1198] Methods and data used to validate WB were reviewed.[438] Subsequent to its development, the *S. neurona* WB test has been modified to try to improve the diagnostic accuracy and/or to make the assay semiquantitative. Use of bovine serum against *S. cruzi* to "block" cross-reacting antigens was reported to yield sensitivity and specificity approaching 100%.[1495] However, evaluation of this modified WB protocol by other investigators found a lower sensitivity/specificity of 89% and 69%, respectively, when applied to a more extensive sample set.[279,343] A WB method used by a commercial testing laboratory (Neogen, Inc.) involved measuring the intensity of the antibody/antigen response to yield a unitless number referred to as the "relative quotient" (RQ). This test is no longer offered by the company.

Development of the WB test provided a tremendous boost to EPM diagnosis, but this technique is primarily a research tool that is fairly laborious and requires significant expertise to interpret accurately. Consequently, multiple "second generation" serologic assays, that provide greater throughput and are more informative, have been developed. Two assays developed, using whole *S. neurona* merozoites as antigen, were the SAT[1076] and the IFAT.[343,346] The usefulness of the SAT was further evaluated in experimentally infected cats, raccoons, and ponies.[447,1539] Both the SAT and the IFAT can be used to obtain an end-point antibody titer, which is a large improvement over the nonquantitative result provided by the WB test. The SAT is advantageous because it can be used for detection of antibodies in multiple different animal species, and the assay has been employed in a variety of research studies (Table 3.5). However, the SAT has not been routinely employed for EPM diagnosis and is not presently offered as a commercial test. The IFAT was optimized and validated at the University of California, Davis,[343,346] and testing is currently available from the University of California Veterinary Diagnostic Laboratory. Although specialized instrumentation is needed (i.e., a fluorescence microscope), interpretation of parasite fluorescence requires expertise, and concerns have been raised about cross reactivity with the nonpathogenic species *S. fayeri*,[1540] serologic results from the IFAT will be generally accurate when the assay is performed by individuals with proper training.

ELISAs are easy to perform, allow for relatively high-throughput testing, and provide a more objective interpretation of results relative to the other existing assays, particularly the WB. Several ELISAs have been developed using *S. neurona* antigens expressed as recombinant proteins in *E. coli*[500,836,1862] or in Baculovirus.[753] Thus far, these ELISAs are all based on the family of related *S. neurona* merozoite surface antigens (SnSAGs),[499,849] which are good serologic targets, because they are abundant and immunogenic. Importantly, antigenic diversity has been found among different strains of *S. neurona*,[263,850,870,1119,1821] so all of the SnSAGs are not equally appropriate for use in serologic assays.

ELISAs based on the SnSAG2, SnSAG3, and SnSAG4 surface antigens, have been shown to be accurate for detecting and quantifying antibodies against *S. neurona* in equine serum and CSF samples.[836,1862] To help overcome the negative impact of antigenic diversity in the *S. neurona* population

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and the varied immune responses that occur in different horses, fusion of SnSAG3 and SnSAG4 into a single chimeric protein (rSnSAG4/3) and concurrent analysis with 2 ELISAs (rSnSAG2 ELISA and rSnSAG4/3 ELISA) have been employed for commercial testing of equine samples (Equine Diagnostic Solutions, LLC). Although homologues of the SnSAG antigens are present in the related parasite *S. falcatula*,[1821] this does not hinder the specificity of the SnSAG2 and SnSAG4/3 ELISA results, since *S. falcatula* is incapable of infecting and causing seroconversion in horses.[271] Furthermore, extensive validation studies have shown that the SnSAG ELISAs are specific and do not cross-react with serum from horses infected with other species of *Sarcocystis*.[32,836] Hence, these surface proteins are valuable immunologic markers that accurately detect infection with *S. neurona*. Recently, a trivalent ELISA rSnSAG2/4/3 with highly comparable results to independent rSnSAG2 and rSnSAG4/3 tests have been developed.[2403]

An ELISA based on the SnSAG1 surface antigen was described[500] and has been offered for EPM diagnosis by Antech, Inc. This test provides an end-point titer and values greater than 1:100 in serum are reported to indicate active infection. This test and cut-off point have not been rigorously evaluated; however, the cut-off values used for a positive diagnosis appear to be arbitrary. More critically, the SnSAG1 protein has been shown to be absent from multiple *S. neurona* isolates,[850,870,1119] which explains the relatively low accuracy observed with this antigen in independent studies.[836,912] An assay that combines SnSAG1 with the two alternative major SnSAGs, SnSAG5[263] and SnSAG6,[1821] is presently offered as a stall-side test (Pathogenes, Fairfield, FL, USA), but no published reports have validated this assay so it is unclear whether the test accurately detects antibodies to *S. neurona*.

Because EPM occurs only in a small proportion of horses infected with *S. neurona*, the simple detection of serum antibodies against this parasite has minimal diagnostic value. Detection of antibodies in CSF is more informative, but this analysis is potentially confounded by blood contamination of the sample during collection as well as the passive transfer of antibody from the plasma across an intact and healthy blood–CNS barrier.[646,648] The blood–CNS barrier (BCB) can also be compromised in neonatal foals. Of 13 foals born to seropositive mares, antibodies were detected in CSF of 12 of 13 tested in *S. neurona* WB test 2–7 days after birth.[248] The CSF antibodies declined over time, but were detectable in 3 foals between 62 and 90 days; the foals were asymptomatic.[248] These obstacles to accurate immunodiagnosis of EPM have been reduced in recent years through the development of semiquantitative assays (e.g., IFAT and ELISA) and use of diagnostic methods that reveal intrathecal antibody production, which indicates active *S. neurona* infection in the CNS. The blood contamination of CSF had no effect on the diagnosis of EPM by using the IFAT.[606]

The Goldman–Witmer coefficient (*C*-value) and the antigen-specific antibody index (AI) are algorithms that use antibody endpoint titers in paired serum and CSF samples to assess whether the amount of pathogen-specific antibody in the CSF is greater than would be present from normal passive transfer across the BCB. Difficulties in the interpretation and value of these tests to diagnose EPM were reviewed previously.[438] Use of the SnSAG2 ELISA, and the *C*-value and AI algorithms, to examine a sample set of 29 clinical cases, demonstrated that these methods provide accurate diagnosis of EPM.[648] This study also confirmed prior findings that modest blood contamination of the CSF, up to 10,000 red blood cells/μL, will have minimal effect on test results.[606]

Many horses with EPM have CSF antibody titers that are profoundly higher than what should be present due to normal passive transfer across the BCB. Studies using the SnSAG2 and SnSAG4/3 ELISAs to examine a large collection of horses with neurologic disease showed that a simple serum:CSF ratio, calculated from the endpoint titers, can serve as a proxy for the more laborious and expensive *C*-value or AI.[912,1453] These studies found that the optimal serum:CSF titer ratio was 100:1, which approximates the normal partitioning of proteins (e.g., albumin or total IgG) between the blood and the CSF.[648,1498] Serum:CSF ratios equal to or lower than 100:1 were indicative of intrathecal antibody production against *S. neurona*, and yielded EPM diagnostic accuracy of

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approximately 93% sensitivity and 83% specificity for a collection of 128 cases examined.[1453] Of 44 EPM cases, 33 had serum:CSF ratios of 25:1 or less, with 9 horses exhibiting CSF antibody titers that approached or exceeded their serum titers (i.e., ratio of 1.6:1 or lower). Although IgM antibodies can be detected in CSF or the blood of horses experimentally infected with *S. neurona*,[1263] this test has not been used in the diagnosis of EPM in horses.

Based on statistical modeling of IFAT results from a sample set of naturally and experimentally infected horses, it has been proposed that EPM diagnosis can be achieved from serum antibodies alone, with minimal additional information provided by CSF testing.[344,347] This idea was not supported by the previously described studies using ELISAs[912,1453] which showed that serum antibody titers are not good indicators of EPM. This discrepancy is not likely due to differences in the serologic assays, but rather can be attributed to differences in the authenticity of the sample sets used for the studies. Specifically, the sample set used by Duarte et al.[344,347] had only 12 seropositive horses in the non-EPM population (N = 97), which does not represent the seroprevalence of *S. neurona* that has been documented in many parts of the United States and Central and South America (Table 3.6). In most regions where *S. neurona* is endemic, there will be numerous seropositive horses that exhibit signs of neurologic disease not due to EPM, and some of these horses will have serum antibody titers that are quite high. When sample sets that better represent the normal equine population are tested, it becomes apparent that serum antibody titers against *S. neurona* do not provide accurate EPM diagnosis.[912,1453]

3.2.12.2 Necropsy Examination

3.2.12.2.1 Gross Lesions

When present, the gross lesions of EPM in the horse are confined to the CNS. Acute lesions consist of multifocal, randomly distributed foci of hemorrhages, whereas subacute and chronic lesions show areas of discoloration ranging from pale to dark tan areas, and foci of malacia, respectively.[438] Although the brainstem is more often involved than other areas of the brain (Figure 3.3), the lesions are more frequently seen in the spinal cord. In rare cases, lesions can be present in both the brain and the spinal cord of a horse.

3.2.12.2.2 Microscopic Lesions

Microscopically, the predominant lesions range from multifocal to coalescing areas of hemorrhage, nonsuppurative inflammation, and small foci of necrosis. Perivascular cuffing by mononuclear cells is evident in some of the affected areas, particularly in the meninges. The numbers of *S. neurona* stages present are often few and difficult to locate in routine histological sections stained with H and E. Developmental stages of *S. neurona* are more easily seen if organisms are present in neurons rather than in inflammatory cells. The types of host cells infected are not definitively known, except that schizonts and merozoites are found in neurons and in macrophages.

3.2.12.2.3 Immunohistochemical Staining

S. neurona schizonts and merozoites are stained specifically with polyclonal rabbit antibodies raised against culture-derived *S. neurona* merozoites.[420,422,2044] The reactivity is less intense using polyclonal rabbit antibodies against bradyzoites and sporozoites (Dubey, J.P.-own observations). The reactivity of *S. neurona* sarcocysts to *S. neurona* antibodies is highly variable; it was found that immature sarcocysts reacted brilliantly, whereas mature sarcocysts were stained irregularly or not at all within the same histological section.[472,1673,1739] Monoclonal antibodies against *S. neurona* can also be used for immunostaining.[1120] In most EPM cases organisms are sparse or undetectable even when diagnosis is confirmed by bioassay in cell culture or *S. neurona* DNA is detected. Not

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all *S. neurona* organisms in EPM cases stain by immunohistochemistry, possibly due to antibody recognition of specific parasite antigens or lack of antigens in the tissue sections. For example, the Sn-Mu1 isolate (SnSAG1 minus) did not stain using polyclonal antibodies prepared against the Sn-UCD1 isolate (SnSAG1 positive).[1119] However, multiple isolates and histopathology sections from a variety of hosts containing *S. neurona* merozoites or schizonts are recognized using Mab 2G5-2, which targets a conserved *S. neurona*.[1196]

3.2.13 Treatment

Treatment of horses suspected to have EPM should be done as quickly as possible after clinical signs of the disease are recognized. Treatment appears to result in successful recovery in 70%–75% of the affected horses, although these estimates are somewhat suspect without postmortem confirmation.

Antiprotozoal drugs, such as sulfonamides and pyrimethamine, have been used to treat EPM since 1974, when the protozoal etiology of the disease was recognized. After the first cultivation of *S. neurona* in 1991, it became possible to test antiprotozoal drugs *in vitro*. Subsequently, when the KO mouse model was established in 2000, it became possible to test drugs against *S. neurona in vivo*.[438] Since then, various compounds have been developed and licensed for treatment of EPM in the United States. In addition to ponazuril (Marquis, Bayer), which was the first FDA-approved EPM medication, diclazuril (Protazil, Merck) and a sulfadiazine/pyrimethamine combination drug (ReBalance, PRN Pharmacal) are currently approved for treatment of EPM.

3.2.13.1 Ponazuril

Ponazuril (Marquis®, Bayer Animal Health) is widely used to treat EPM. The dosage commonly used is 5 mg/kg per day for a minimum of 28 days. Ponazuril is a benzeneacetonitrile compound that is related to the herbicide atrazine and may act by inhibiting apicoplast and/or mitochondrial function in the parasite.[1202] Ponazuril is technically considered to be a coccidiostat.[1092,1431] The significance of this mode of action *in vivo*, however, is totally unknown and is probably unimportant because efficacy studies between coccidiocidal and coccidiostatic drugs provide similar outcomes.

Ponazuril is well absorbed orally and achieves a steady state concentration of 0.16 ± 0.06 mg/L in the CSF of horses treated with 5 mg/kg body-weight.[643] *In vitro* studies have documented anti-*S. neurona* activity of ponazuril.[1073,1202] *In vivo* studies in KO mice inoculated with *S. neurona* sporocysts indicated reduction in the severity of clinical signs if the drug was administered 4 DPI.[624] Prophylactic administration of Ponazuril at 5 mg/kg also reduced severity of clinical signs in experimentally infected horses.[647] The time to reach steady-state concentrations of Ponazuril in the CSF of horses is approximately 1 week. For this reason, an initial loading dose is recommended in an effort to achieve therapeutic concentrations more quickly. While the clinical value of this approach has not been demonstrated, maximum CSF concentrations of Ponazuril were achieved within 28 h when horses were given a loading dose of 15 mg/kg body weight (3 times the normal dosage). Additionally, feeding 60 mL of corn oil immediately before administering Ponazuril results in blood concentrations that are 25% higher than if no corn oil is given. A field efficacy study of 101 horses demonstrated approximately 60% efficacy, with 8% of cases relapsing within 90 days if treatment is stopped after 28 days.[645] It is expected, although not proven, that the relapse rate (already low) will be even less if treatment is continued for longer than 28 days. Animals typically responded within 10 days of beginning treatment, although some horses may take up to 3 weeks, following the initiation of treatment, before tangible evidence of response is noted. Therefore, a refractory patient often continued to improve even after treatment stopped at 28 days. The baseline neurologic score did not influence outcome in that study. However, success was defined as improvement by 1 clinical grade,

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which may be considered unacceptable in severe cases.[645] In clinical cases, the animal should be reevaluated at the end of the treatment period, and then a determination made whether further treatment is needed. In general, if there has been a clinical response, yet the horse remains abnormal, a second month of Ponazuril is recommended. If finances are limited, treatment can be stopped after 28 days, but the horse should be reexamined after 1 month to ensure that there is no deterioration.

Toxicity studies have found Ponazuril to be very safe, with no systemic toxicity, even at high doses (30 mg/kg body-weight) for up to 56 days.[950] Uterine edema was noted in mares given 30 mg/kg body-weight each day for 30 days[950]; however, treatment of breeding stallions with 10 mg/kg body-weight did not affect androgenic hormone production nor spermatogenesis.[1819] Ponazuril has been used without obvious problems in pregnant mares, but the use of Ponazuril in pregnant animals is off-label, and owners should be made aware of this fact. As stated earlier, Ponazuril was used successfully to treat a sea lion with *S. neurona*-associated clinical disease.[1267]

3.2.13.2 Diclazuril

Diclazuril (Protazil®, Merck Animal Health) is also a benzeneacitonitrile compound that is chemically similar to Ponazuril. Diclazuril is administered at 1 mg/kg body-weight as alfalfa-based pellets that are top dressed in the daily grain ration. In a study of 49 horses, Diclazuril had a success rate of 58%, when success was defined as improvement by a minimum of 1 clinical grade.[1092] Recent investigation of Diclazuril pharmacokinetics has shown that a dose of 0.5 mg/kg body-weight (50% of the recommended dose) is sufficient to attain concentrations in plasma and CSF that will inhibit *S. neurona* growth in culture.[860] Further study is needed, but these findings suggest that effective treatment of EPM might be possible with lower doses of Diclazuril.

Diclazuril is absorbed quickly, especially the sodium salt, and has been found in serum 1 h after feeding to horses[324] Diclazuril has anti-*S. neurona* activity *in vitro*[1071] and *in vivo*.[439] Diclazuril in pelleted feed (50 parts per million), starting 5 days before or 7 days after feeding *S. neurona* sporocysts to mice, and continuing therapy for a month, resulted in the absence of *S. neurona* stages in the mice. Therapy was less effective when Diclazuril was given 12 days or more after feeding sporocysts. These results indicate that Diclazuril can inhibit the early stages of *S. neurona* and might be useful as a prophylactic against *S. neurona* infections in horses.[439]

3.2.13.3 Nitazoxanide

The broad-spectrum antimicrobial nitazoxanide (NTZ) and its other derivatives have anti-*S. neurona* activity *in vitro*.[662] NTZ was approved for the treatment of EPM and marketed as Navigator® (Idexx Pharmaceuticals, Inc.). Although treatment with NTZ was effective against EPM, there were prominent concerns about adverse side-effects (e.g., colic). Subsequently, NTZ was removed from the market and is no longer available.

3.2.13.4 Decoquinate

Decoquinate has been used to treat coccidiosis in poultry, cattle, sheep, and goats. *In vitro* testing has determined that it is effective against *S. neurona* at low concentrations.[1081] Decoquinate, in combination with the immunomodulator levamisole, has been investigated for treatment of EPM in 1 published study, with a very high rate of clinical improvement reported after only 10 days of treatment.[503] There are substantial concerns with regard to these findings, however, including case selection and assessment, and the diagnostic standard applied. Substantial additional research using confirmed cases needs to be performed before the use of this drug combination can be considered.

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3.2.13.5 Sulfonamide and Pyrimethamine

The sulfonamide and pyrimethamine (S/P) combination has been used widely for the last 4 decades, but a premixed version of this combination has recently achieved FDA approval and can be purchased commercially (ReBalance®, PRN Pharmacal). The sulfonamide component of this compound competes with para-aminobenzoic acid to inhibit dihydropteroate synthetase activity, while pyrimethamine targets dihydrofolate reductase, which collectively inhibit folate metabolism. The synergistic effects of these compounds block synthesis of nucleic acids and amino acids, ultimately leading to parasite death. A field efficacy study reported a success rate of 57% after several months of treatment.[1092] The weaknesses of S/P suspension in the treatment of EPM include the prolonged duration of treatment required to affect a positive response, and the toxicity of the compound. Toxicity includes anemia, leukopenia, fetal loss, and fetal abnormalities. A benefit of S/P combinations is the lower cost, yet this may be offset by the extended dosing interval required.

Pyrimethamine has historically been given in combination with sulfa drugs in the treatment of EPM. There is, however, some evidence to suggest a synergistic effect of pyrimethamine when used with the benzeneacitonitrile-group antiprotozoals (Ponazuril, Diclazuril). While not empirically evaluated in horses with EPM, so that its effectiveness is not known at this time, it might be beneficial to add this drug to the treatment regimen in refractory cases.

3.2.13.6 Pyrantel Tartrate

Based on *in vitro* activity of pyrantel tartrate against *S. neurona* merozoites,[980] administration of pyrantel tartrate was considered for prophylactic use against EPM. However, this compound had no anti-*S. neurona* activity in experimentally infected KO mice.[1075] Similarly, daily administration of pyrantel tartrate for 134 days had no anti-*S. neurona* activity in experimentally infected horses.[1501]

3.2.13.7 Supportive/Ancillary Therapy

A variety of ancillary and supportive therapies may be indicated for the treatment of horses with EPM.[1581] Ancillary treatments may include various anti-inflammatory drugs such as phenylbutazone, flunixen meglumine, dimethylsulfoxide (DMSO) or steroids. Corticosteroids can be used to help stabilize horses with serious neurologic abnormalities during the early period of treatment. Long-term steroid treatment should be avoided due to their unknown effects upon immune clearance of *S. neurona*. However, it has been observed that some horses initially worsen slightly and transiently with treatment; hence, prophylactic use of nonsteroidal anti-inflammatory drugs to ameliorate this "treatment crisis" is sometimes advised. In seriously affected horses, prophylactic anti-inflammatory treatment is probably advisable for the first week. Additional ancillary treatments, such as Vitamin E, homeopathic medications, etc., have not been demonstrated to have any value, and use of these compounds or approaches is not recommended.

Immunostimulants have been recommended by some on the presumption that immunosuppression is a component of the pathophysiology of EPM. Levamisole (1 mg/kg orally daily), EqStim (5 mL IM on day 1, 3 and 7, then monthly), or Equimune IV, (1.5 mL intravenously weekly for 3 weeks) have all been advocated; however, there is no specific information to suggest that these have any positive effect. Another potential beneficial immune modulator is Parapox Ovis Virus Immunomodulator (Zylexis, Pfizer Animal Health). While licensed as an aid in the treatment of horses with EHV-1 and EHV-4, Parapox Ovis has been demonstrated to increase interferon gamma secretion in treated horses. As interferon gamma is considered a key cytokine in protection against *S. neurona* infection, this compound might be beneficial in selected animals.

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3.2.13.8 Duration of Treatment

The duration of treatment for EPM is difficult to determine, and when to terminate treatment in a particular horse remains problematic. Duration of treatment appears to be more important than peak concentrations (as long as they exceed minimum inhibitory concentrations). Therefore, horses should be evaluated after 1 month of treatment with Ponazuril or Diclazuril. If improvement has been noted, but clinical signs remain, then a further month of treatment is recommended. If finances are limiting, or the horse appears clinically normal, then treatment can be discontinued. However, the horse should be examined one month after treatment is completed to ensure that there has been no relapse. Alternatively, a 1–2-month course of S/P can be given to help minimize the chance of relapse.

Treatment until the CSF–WB becomes negative has been advocated by some clinicians in the past. Experience has dictated that this is an unachievable goal for most cases. Antibodies can have a lengthy half-life, so horses will exhibit positive titers for long periods. The effects of treatment upon CSF or serum antibody concentrations is unclear, and, at the present time, repeating these tests appears to have little value in determining treatment success or duration.

3.2.14 Relapse of Clinical EPM in Horses

Horses can relapse with clinical EPM, even after prolonged therapy, remarkably with clinical signs similar to first episode. The cause of relapse is unknown because the encysted stage, sarcocyst, has not been confirmed. Whether schizonts can remain dormant in equine tissues is unknown.

3.2.15 Epidemiology of EPM in Horses

Few epidemiologic studies have assessed the risk factors associated with clinical EPM in horses in the United States.[1224,1536,1537,2283,2284] In 1998, a National Animal Health Monitoring System (NAHMS) study examined data collected from 1178 U.S. equine industry operators representing 83.9% of horses from 28 U.S. states and 51.6% of operations with 3 or more horses. A total of 164 operators reported information concerning the most recent EPM Case and a Control Horse, and 974 operators completed survey questions regarding a Control Horse from NonCase Premises. An extensive statically valid risk assessment was performed, and results were reported.[1224] The following conclusions were drawn:

a. EPM risk on operations is closely tied to environmental and management factors that impact exposures to opossums, their feces, and their environment. This finding suggests opossums, or contamination from these animals, may be more commonly found on premises where EPM has occurred. Similarly, climate and terrain factors impact suitability of opossum habitat as well as the likelihood that *S. neurona* sporocysts will survive in the environment.
b. Housing of horses, choice of bedding material, methods of storing concentrate feeds, and equine stocking density, all could impact the likelihood of horses being exposed to *S. neurona*, either through direct exposure in the environment or indirectly through exposure to contaminated feed and bedding.
c. Horses that were used primarily for racing had a greater risk compared with other horses, as did thoroughbred and warmblood horses.
d. When comparing horses diagnosed with EPM to non-neurologic controls, young horses (1–5 years old) and older horses (>13 years) had a higher risk of developing EPM than horses <1 year and 6–13 years of age; the youngest horses had nearly 5 times greater odds of developing EPM, and horses 18 months to 5 years old had twice the odds of developing EPM, when compared with horses >5 years old. Horses that were transported <1 month before disease onset or the date of interview did not have a detectable difference in disease risk compared with horses that had been transported at >6 months before these dates, but there was about 2.6 times greater odds of disease if owners reported transporting horses 1–6 months previously. There was also a 4-times greater odds of disease if

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owners could not remember when horses were transported, in comparison with horses transported >6 months previously.

Finding age-related differences in the likelihood of disease is suggestive of repeated exposure to *S. neurona* over time, and the subsequent development of specific immunity. Several signalment variables were associated with EPM occurrence in individual horses (e.g., age, sex, breed, and primary use), but only 1 of the variables marking hypothetical stressful events (e.g., participating in competition) was considered most important.

e. The variables most strongly associated with EPM occurrence were maximum stocking density of turnout areas, number of resident horses, primary use of resident horses, finding evidence of raccoons or skunks on premises, and being located near a marsh. It was expected that EPM cases were more likely to be identified on premises with more horses, because the likelihood of identifying a case increased with the horse population size. Accordingly, operations with >20 horses were 2–3 times more likely to identify an EPM case, when compared with smaller operations. Operations that had 2:1.4 horses/acre in turnout areas, were 5–10 times more likely to report an EPM case than were operations where turnout areas were less densely aggregated, or operations that did not have turnout available. Operations where the primary use of resident horses was for show, modest mean temperatures had the lowest odds of disease, and those with moderate temperatures (>9.4°C and <12.0°C) had the highest odds of disease, followed by those with the lowest mean annual temperatures. Operations that housed horses in stalls or paddocks during the day had small increases in the odds of EPM occurrence, when compared with operations that housed horses in pastures. Operations that stored concentrate feeds in rodent-proof containers had about 3 times lower odds of detecting EPM than those that did not. Operations that used wood products (e.g., shavings, chips, or sawdust) as the predominant bedding material had about 1.4 times lower odds of detecting EPM than did those that used other bedding materials (e.g., straw, corn stalks).

f. Horses used for breeding had about half the odds of disease compared with those used for pleasure, but there were no detectable differences in the odds of disease among horses used for pleasure, show or competition, farm or ranch work, or other uses. Thoroughbred and warmblood breeds had about 4 times greater odds of disease, compared with quarter horses; there was no detectable difference in disease risk when comparing other breeds. As expected, regional ecology was also associated with the likelihood of EPM occurrence in this final model; horses were twice as likely to have EPM if they resided in states from region A, than if they lived in the other 3 regions.

3.2.16 Prevention and Control

Preventing contamination of feed and water with opossum feces is essential to prevent EPM in animals. Opossums can excrete millions of sporocysts in their feces for months. Sporocysts are resistant to environmental influences, and most commonly used disinfectants do not kill *S. neurona* sporocysts (Table 3.11). Heating to 60°C for 1 min will kill sporocysts, but exposure to 55°C for 5 min will not. Although survival of sporocysts in different environmental conditions outdoors has not been tested, sporocysts remained viable at 4°C for months (Table 3.11).

Currently, there is no vaccine for EPM. A killed whole *S. neurona* merozoite vaccine was conditionally marketed by Fort Dodge in the United States, but the product is now withdrawn because of the difficulty of obtaining efficacy data.

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Table 3.11 Resistance of *S. neurona* Sporocysts

Reagent/Condition	Duration	Viability[a]	Reference
5.25% NAOCl (bleach)	1 h	Killed	**449**
12.56% phenol (Wex-cide)	6 h	Live	
2% chlorhexide (Nolvasan)	6 h	Live	
1% iodine (Betadine)	6 h	Live	
5% O-benzyl-p-chlorphenol (TB-plus)	6 h	Live	
Formalin 10%	6 h	Live	
6% benzyl ammonium chloride (NPD)	6 h	Live	
Ammonium hydroxide			
100%	1 h	Killed	
10%	6 h	Live	
Thermal exposure			
60°C	1 min	Killed	
55°C	5 min	Live	
55°C	15 min	Killed	
50°C	60 min	Killed	
4°C	34 months	Live	**515**
4°C	44 months	Killed	

Source: Adapted from Dubey, J.P. et al., 2015. *Vet. Parasitol*. 209, 1–42.
[a] Determined by bioassay in KO mice.

3.3 *SARCOCYSTIS CANIS* DUBEY AND SPEER 1991 INFECTIONS IN ANIMALS

3.3.1 Introduction

Historically, a sarcocystosis-like illness was diagnosed in a dog from Maryland, United States.[405] In October 1989, 5 of the 8 pups born to a Rottweiler bitch became ill. Pup #1 had surgery for entropion (inverted eyelid) at 7 weeks of age and developed diarrhea and became anorectic. It was anemic and had slightly elevated liver enzymes. The pup was euthanized. At necropsy, only the liver and small intestine were collected for histological examination. The liver was enlarged grossly and had multiple foci of discoloration. Histologically, there was severe hepatic necrosis, associated with a *Sarcocystis*-like organism. Pup #2 died of massive toxoplasmic lympadentitis. The cause of illness in pups #3–5 was undetermined.

Dubey and Speer[400] named the parasite associated with hepatitis *S. canis* to differentiate it from *S. neurona* found in the neural cells of horses.[404] Subsequent investigations indicated that *S. neurona* can cause illness in several hosts other than horses (see previous sections in 3.2), and the diagnosis could be confirmed by PCR and an IHC test using *S. neurona*-specific antibody. The *S. neurona* antibody does not react immunohistochemically with *S. canis* in the liver of dogs.[460]

Since the initial report of *S. canis*-associated hepatitis in the Maryland dog, similar cases were diagnosed in a chinchilla, black bear, polar bear, sea lion, horse, Steller sea lion, and a Hawaiian monk seal from the United States (Table 3.12). Additionally, a case was diagnosed in a striped dolphin from Spain and in a dog from Costa Rica (Table 3.12). In all of the reports listed in Table 3.12, hepatitis was the only condition diagnosed and the parasite was confined to hepatocytes. Because the parasite has not been cultivated in cell culture or in laboratory animals, there are no specific

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Table 3.12 Reports of *S. canis*-Like Infections in Animals

Host	Country	Signalment	Clinical	Diagnosis	Reference
Dog (*Canis familiaris*)	Maryland, USA	10-month-old Rottweiler	Diarrhea, listless, anorexic	Histology[a] (*S. neurona* antibody negative)	**400, 405, 460**
	Kansas, USA	3-month-old mixed breed, four dogs from 3 months old to 17 weeks old	Suspected toxoplasmosis	Histology[a] (*S. neurona* antibody negative but 3 more reacted positively	**460, 1759**
	Colorado, USA	12-week-old Golden Retriever	Fever, lethargy, elevated enzymes, eosinophilia	Histology[a]	**28**
	Heredia, Costa Rica	10-week-old Rottweiler	Diarrhea, excessive salivation, convulsions, concurrent canine distemper	Histology	**98**
Polar bear (*Ursus maritimus*)	Anchorage, Alaska, USA	Two adult bears	Lethargy, anorexia	Histology, TEM	**663**
Black bear (*Ursus americanus*)	South Dakota, USA	2-year-old	Found dead	Histology	**1874**
	British Columbia, Canada	13-year-old, captive	Acute onset of vomiting and hepatic signs	Histology, PCR	**305**
Sea lion (*Zalophus californianus*)	Florida, USA	10-year-old	Found dead	Histology, TEM, also sarcocyst	**1184**
Chinchilla (*Chinchilla* sp.)	Georgia, USA	7-month-old	Found dead	Histology	**1443**
Horse (*Equus caballus*)	California, USA	4-year-old Trakehner	Fever, abdominal discomfort, enzymes elevated	Histology[a], TEM	**306**
Striped dolphin (*Stenella coeruleoalba*)	Mediterranean coast, Spain	80 kg adult female	Found dead	Histology[a]	**1465**
Hawaiian monk seal (*Monachus schauinslandi*)	Hawaii, USA	21-month-old	Gastrointestinal	Histology[a], TEM	**1860**
Steller sea lion (*Eumetopias jubatus*)	Alaska, USA	1100–1500 kg	Found dead	Histology, PCR	**1818**

[a] Immunohistochemistry negative with *S. neurona* antibody.

diagnostic tests. Partial DNA characterization of the parasite from the liver of a polar bear that died of massive protozoal hepatitis revealed that the 18S and ITS-1 were distinct from *S. neurona* and other *Sarcocystis* species.[460] Based on IHC staining and DNA characterization, it was revealed that out of the 10 cases of *S. canis*-like infections in dogs, only the 2 dogs listed in Table 3.12 were *S. canis*.

T. gondii, *N. caninum*, and *S. neurona* infections are frequent in marine mammals: That is why a differential diagnosis with respect to *S. canis* is a major concern.[455]

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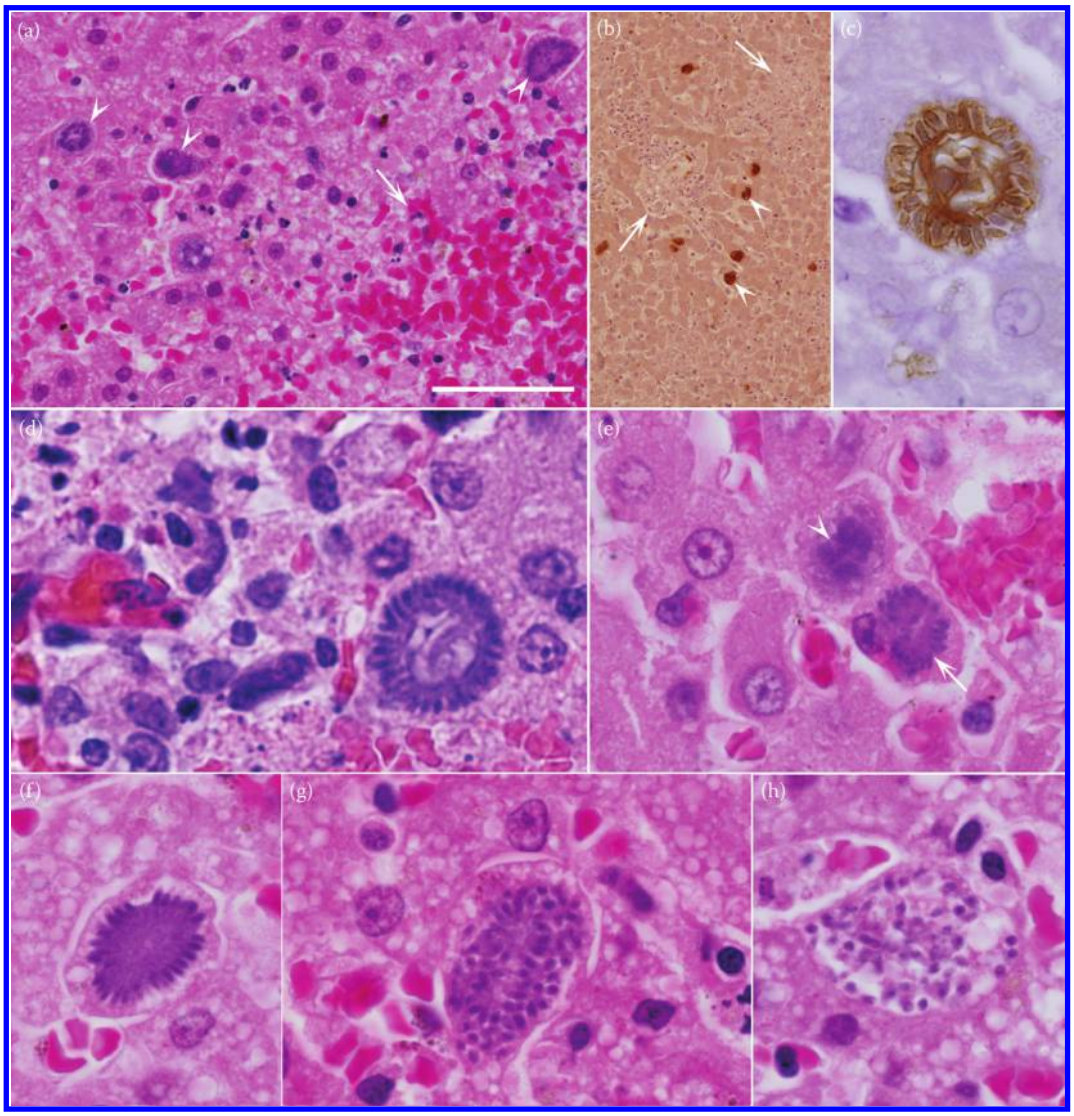


Figure 3.11 *Sarcocystis canis*-associated lesions in a sea lion (a,f,g,h described in Reference **1184**) and black bear (b,c,e described in Reference **1874**). (a,d–h). H and E stain. (b and c immunostaining with anti-*S. cruzi* rabbit polyclonal antibody. Bar in (a) = 100 µm, bar in (b) = 200 µm, and bar in (c–h) = 20 µm. (a) Hemorrhage (arrow) and several schizonts (arrowheads) at the periphery of the lesion. (b) Necrosis (arrows) and several schizonts (arrowheads). (c) Schizont with staining of both the residual body and merozoites reacting to *S. cruzi* antibody. (d) Inflammatory focus on left and a schizont on the right. (e) Two immature schizonts. Note lobulation of the nucleus (arrowhead) and budding of merozoites from the nucleus (arrow). (f) Merozoites budding from a large residual body. (g) Mature schizont without a residual body. (h) Ruptured schizont with cross sections of merozoites. Merozoites in (g) and (h) are indistinguishable from tachyzoites of *Toxoplasma gondii*.

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3.3.2 Structure and Life Cycle

Only the schizont stages are known and the parasite multiplies in hepatocytes (Figure 3.11). The schizogonic development is typical of other *Sarcocystis* species. After merozoite development, a large eosinophilic residual body is left. By TEM, merozoites lack rhoptries. As indicated earlier, *S. canis* does not react with antibodies to *S. neurona*, *T. gondii*, and *Neospora caninum* antibodies, but react to *S. cruzi* antibodies (Figure 3.11c).

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CHAPTER 4

Sarcocystosis in Humans (*Homo sapiens*)

4.1 INTRODUCTION

There are 2 known species of *Sarcocystis*, *S. hominis,* and *S. suihominis*, for which humans serve as the definitive hosts. Humans also serve as the accidental intermediate or aberrant host for several unidentified species of *Sarcocystis.*

4.2 INTESTINAL SARCOCYSTOSIS

4.2.1 *Sarcocystis hominis* (Railliet and Lucet 1891) Dubey 1976

This species is acquired by ingesting uncooked beef containing *S. hominis* sarcocysts. The structure and life cycle are described in Chapter 7.

S. hominis is only mildly pathogenic for humans. A volunteer who ate raw beef from an experimentally infected calf developed nausea, stomachache, and diarrhea 3–6 h after ingesting the beef; these symptoms lasted 24–36 h. The volunteer excreted *S. hominis* sporocysts between 14 and 18 days after ingestion. During the period of patency, he had diarrhea and stomachache.[807] Somewhat similar but milder symptoms were experienced by other volunteers who ate uncooked, naturally infected beef.[40,821,1478]

Similar symptoms were reported in people from Brazil[1382] who ingested raw beef or raw kibbe made from uncooked beef. Of 7 volunteers in Brazil, who ate raw kibbe (beef), 6 began to excrete *S. hominis* oocysts/sporocysts 10–14 days later, and continued to excrete them for 5–12 days.[1382]

4.2.2 *Sarcocystis suihominis* (Tadros and Laarman 1976) Heydorn 1977

This species, acquired by eating undercooked pork, is more pathogenic than *S. hominis.* Its structure and life cycle are described in Chapter 6.

Human volunteers developed hypersensitivity-like symptoms—nausea, vomiting, stomachache, diarrhea, and dyspnea—within 24 h of ingestion of uncooked pork from naturally or experimentally infected pigs. Sporocysts were excreted 11–13 days after ingesting pork.[40,821,1394,1478] Similar symptoms were reported in people from China[1053,1057,1884] who ate *S. suihominis*-infected raw pork.

In India, the prevalence of *S. suihominis* in pigs and humans is high in an economically deprived sect,[64,1638] probably linked to slaughter practices. Street sweepers raise pigs that clean the streets and eat everything, including human waste. When the pigs are slaughtered, usually in the backyards of the owners, who live in slum conditions, the owners' children often gather around the slaughter to help with chores. In turn, the children are invariably rewarded with the tail of the slaughtered pig, some of which were found to be infected with *S. suihominis* sarcocysts and which the children consumed on the spot of slaughter.

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4.2.3 Other Zoonotic *Sarcocystis* Species

In China, 2 volunteers who ate raw water buffalo meat excreted sporocysts; the buffalo had been fed sporocysts from a human who had ingested raw meat from a naturally infected bovine, but it is not clear if this buffalo was free from environmentally derived infection of a possible human *Sarcocystis* specific for the water buffalo. Two human volunteers who ingested raw *Sarcocystis*-infected buffalo meat became ill but did not excrete sporocysts[211]; the illness could be due to other foodborne microbes.

4.2.4 Natural Prevalence of Sporocysts in Human Feces

Reports on this topic are summarized in Table 4.1.

4.3 MUSCULAR SARCOCYSTOSIS

Until 1999, sarcocysts found in striated muscles of human beings were incidental findings, mostly reports of individual cases. Reports until 1978 were reviewed critically[84] and are included here for the sake of completeness (Table 4.2). Subsequent reports were summarized[595,596] (Table 4.3). Additionally, serologic findings were summarized[1410] for people from Malaysia,[1740] the former Yugoslavia,[1607] and Australia.[1264] All infections in humans until 2013 were reported as intramuscular sarcocysts of unknown species. The morphologies of 7 distinct types of sarcocysts were described from light microscope observations of histological sections from human tissues.[84]

A series of 3 outbreaks of acute sarcocystosis was first reported in 1999. In the first outbreak, 10 members of a United States Air Force team deployed for 1 week to a remote jungle village about 80 km northeast of Kuala Lumpur[36] became ill within 3 weeks after returning from deployment. They had fever, myositis, and bronchospasm, with elevated liver enzyme levels and eosinophilia. Seven members, 1 of whom was asymptomatic, from the jungle cohort were tested for eosinophilia, sedimentation rate, and serum enzyme levels, including ALT, CK, LDH, and AST, and virtually all had at least some levels elevated above the normal. Extensive laboratory investigation did not reveal a specific diagnosis. Myositis and immature sarcocysts were observed histologically in a muscle biopsy 3 months after the onset of the illness (Figure 4.1).

Another outbreak of acute muscular sarcocystosis involved college students and teachers who visited Pangkor Island off the west coast of peninsular Malaysia.[14,877] Of the 92 persons who attended a retreat on the island, 89 became symptomatic, with onset of fever (94%; 57% relapsing), myalgia (91%), headache (87%), and cough (40%), within 26 days of their return to the mainland. Eight persons, who had visible facial swelling for 4–6 weeks, showed changes, consistent with inflammatory edema, in their mastication muscles by "whole body" magnetic resonance imaging (MRI). Similar findings were observed in the back muscles of 4 persons and the calf muscles of 2 persons. A sarcocyst ~190 μm long was observed in cell culture inoculated with muscle homogenate from a patient, and a sarcocyst was observed in a histologic section of muscle from another patient. Neither had elevated serum CK levels, but both had eosinophilia.

Outbreaks in 2011 and 2012 involved at least 99 persons who vacationed on Tioman Island off the east coast of peninsular Malaysia; all were Europeans except for 2 Canadians and a British resident of Singapore.[559,560] The strict case definition of myositis, eosinophilia, and negative trichinellosis serology most likely underestimated the number of positive patients at 68. Myositis required at least 1 of the following: a complaint of muscle pain with a serum CK level greater than 200 international units per liter, muscle tenderness documented on physical examination, or histologic evidence of myositis in a muscle, confirmed by biopsy; 62 patients were considered probable, and 6

Table 4.1 Intestinal Sarcocystosis in Humans

Country	Human Subjects	Species	Source of Sarcocysts	Clinical Signs	Year of Diagnosis	Reference
Netherlands	12 of 72 persons 5 of 17 autopsies	*I. hominis*[a]	NI	NI	1962	**1011**
Germany	2 volunteers 4 volunteers	*S. hominis* *S. suihominis*	Beef Pork	1 of 6 persons had severe influenza-like symptoms and mild diarrhea	1972	**1478**
Poland	29-year-old F 7 (assumed children) 92 unidentified	*Sarcocystis* sp. *Sarcocystis* sp. *Sarcocystis* sp.	NI	Symptoms very scanty	1973	**1398**
Poland	200 persons	*I. hominis*[a]	NI	NI	1973	**1399**
Germany	1 volunteer	*S. tenella*	Sheep	None. No infection resulted from ingestion of macroscopic cysts from sheep	1974	**1480**
Germany	22 of 300 persons	*I. hominis*[a]	NI	10 of 22 persons had gastric and intestinal symptoms; 12, no clinical signs	1975	**897**
Germany	8 of 506 persons	*Sarcocystis* sp.	NI	Intermittent enteritis with diarrhea, rheumatic phenomena, or asymptomatic	1975	**610**
Poland	13 of 125 children 7 to 18 years old	*Sarcocystis* sp.	NI	NI	1976	**1401**
Germany	1 volunteer 3 volunteers	*S. hominis* *S. suihominis*	Beef Pork	Diarrhea and stomachache, bloating, inappetence, nausea, vomiting, diarrhea	1977	**807**
Slovakia	A 12-year-old female	*I. hominis*[a]	NI	Asymptomatic	1978	**678**
United States	2	*S. cruzi*	Beef	Not infectious, asymptomatic	1978	**1038**
Netherlands	5	*S. hominis*	Beef	Natural infection	1979	**1714**
	1	*S. hominis*	Beef	Experimentally infected with 15,000 sarcocysts		
France via tropical areas	2% of 3500 samples	*S. hominis*		NI	1980	**314**
Thailand	30-year-old M 3-year-old F 60-year-old F 9-year-old M 19-year-old M 70-year-old M	*S. hominis* *S. hominis* *S. hominis* *S. hominis* *S. hominis* *S. hominis*	Beef Beef Beef Beef Beef Beef	All 6 had acute fever and acute abdominal pain, five had leukocytosis. Bacterial infection may have contributed to severity of illnesses	1982	**152**
China	48-year-old M	*Sarcocystis* sp.	Pork	Abdominal distension, pain, diarrhea, constipation, stomachache, dyspnea	1982	**1884**
	Adult M	*Sarcocystis* sp.	Unknown	No information		

(*Continued*)

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Table 4.1 (*Continued*) Intestinal Sarcocystosis in Humans

Country	Human Subjects	Species	Source of Sarcocysts	Clinical Signs	Year of Diagnosis	Reference
Germany	8 of 403 persons	*Sarcocystis* sp.	NI	Intestinal complains	1983	**667**
China, Yunnan Province	2 of 12 persons	*Sarcocystis* sp.	Pork	Suspected schistosomiasis, abdominal distension, fatigue, diarrhea	1983	**1885**
China, Yunnan Province	Adult M volunteer	*S. suihominis*	Pork	No symptoms but excreted sporocysts infectious for pigs	1986	**1057**
China	1 volunteer, 2 monkeys	*S. hominis*	Beef	NI	1990	**1059**
Slovakia via North Vietnam	14 of 1228 workers	*Sarcocystis* sp.	Unknown	None	1991	**1685**
Tibet	20.5%–22.9% of 926	*S. hominis*	Beef	Asymptomatic	1991	**1865**
	0.6%–7% of 926	*S. suihominis*	Pork	Asymptomatic		
Laos	>10% of 1008	*S. hominis*	NI	NI	1991	**679**
China	123 of 414	*S. suihominis*	Pork	Asymptomatic	1991	**1888**
Australia	2 of 385 persons	*Sarcocystis* sp.	NI	NI	1993	**1181**
India	14 of 20 children, 3–12 years old	*S. suihominis*	Pork	Asymptomatic	1994	**64**
Thailand	23.2% of 362 Thai laborers	*Sarcocystis* sp.	NI, usual consumption of raw beef and pork	Asymptomatic	1996	**1828**
China	3 volunteers 2 volunteers	*Sarcocystis* sp. *Sarcocystis* sp.	Cattle Water buffalo	All had clinical symptoms, including abdominal pain, distension, watery diarrhea, and eosinophilia	1999	**210**
Brazil	6 of 7 volunteers	*S. hominis*	Kibbe (beef)	Diarrhea	2001	**1382**
Spain	1	*S. hominis*	Raw beef	Abdominal discomfort, loose stools	2001	**218**
China, Guangxi	27 (22 M, 5 F) of 501 persons older than 2 years	*S. suihominis*	Pork	8 of 27 had diarrhea and abdominal pain	2004	**1051**
China, Guangxi	32 of 489	*S. suihominis*	Pork	Asymptomatic	2005	**1720**
China	1 volunteer	*S. suihominis*	Pork	Distension, diarrhea, fever, pain	2007	**1053**

(*Continued*)

Table 4.1 (*Continued*) Intestinal Sarcocystosis in Humans

Country	Human Subjects	Species	Source of Sarcocysts	Clinical Signs	Year of Diagnosis	Reference
Thailand:						
Ubon Ratchathani	4.6% of 479	*Sarcocystis* sp	NI	NI	2007	**1764**
Khon Kaen	8% of 1124	*Sarcocystis* sp.	NI	NI		
Argentina	31-year-old HIV patient	*Sarcocystis* sp.	NI	Diarrhea, systemic muscular sarcocystosis	2008	**1782**
Argentina	0 of 69 children	NI	NI	Asymptomatic	2008	**1645**
China	2 volunteers	*Sarcocystis* sp.	Water buffalo	Became ill but did not excrete sporocysts	2011	**211**
NE Thailand:						
Khon Kaen	1 of 253 persons	*Sarcocystis* sp.	NI	NI	2013	**124**
Jordan	19-year-old M	*S. hominis*	Shawarma (beef)	Abdominal discomfort, diarrhea for 3 weeks	2014	**1278**
Malaysia	1 of 269 from indigenous subtribes	*Sarcocystis* sp.	NI	Asymptomatic	2014	**1035**

Source: Modified from Fayer, R., Esposito, D.H., Dubey, J.P., 2015. *Clin. Microbiol. Rev.* 28, 295–311.
NI = Not indicated.
[a] *Isospora hominis* was an early name used to identify sporocysts in feces and stages in lamina propria before the *Sarcocystis* species were known.
M = male, F = female

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Table 4.2 Summarized Early Reports of Humans with Sarcocysts

Country	Patient Age, Gender	Sarcocyst Location	Biopsy or Autopsy	Sarcocyst Wall Type[a]	Original Author and Year[b]
Sudan	36-year-old, M	Abdomen	A	2	Kartulis (1893)
France	Adult, M	Larynx	A	1	Barbaran and St. Remy (1894)
France	Adult, unknown	Skeletal muscle	A	1	Vuillemin (1902)
Malaysia	30-year-old, M	Tongue	A	4	Darling (1919)
UK	Unknown	Heart	A	6	Manifold (1924)
India	55-year-old, M	Chest	B	2	Vasudevan (1927)
USA via West Indies	32-year-old, F	Heart	A	7	Lambert (1927)
Indonesia	20-year-old, M	Cheek	B	4	Bonne and Soewandi (1929)
China	Adult, M	Leg	B	1	Feng (1932)
UK	Adult, F	Heart	A	5	Hewett (1933)
Panama	11-year-old, F	Heart	A	7	Gilmore et al. (1942)
Panama	48-year-old, F	Heart	A	7	Kean and Grocott (1943)
Brazil	32-year-old, F	Heart	A	7	DeFreitas (1946)
USA via Germany	31-year-old, M	Heart	A	7	Arai (1949)
USA via Puerto Rico	34-year-old, M	Heart	A	7	
India	37-year-old, M	Leg	B	2	Dastur and Iyer (1955)
	20-year-old, M	Leg	B		
Sudan	45-year-old, M	Foot	B	3	McKinnon and Abbott (1955)
UK via India	60-year-old, M	Pectoral	B	2	McGill and Goodbody (1957)
Brazil	40-year-old, F	Heart	A	6	Koberle (1958)
Italy	17-year-old, F	Heart	A	5	D'Arrigo and Squillaci (1962)
Indonesia	51-year-old, M	Pectoral	B	4	Van Thiel and Van den Berg (1964)
UK via SE Asia	51-year-old, M	Leg	B	4	Mandour (1965)
Angola	30-year-old, F	Chest	B	2	Liu and Roberts (1965)
India	22-year-old, F	Leg, arm	B	2	Gupta et al. (1973)
SE Asia	21-year-old, M	Leg	B	3	Jeffrey (1974)
UK via Malaysia	34-year-old, M	Pectoral	B	4	
Malaysia	34-year-old, M	Larynx	B	4	Kutty and Dissanaike (1975)
Malaysia	12-year-old, F	Pharynx	A	4	Kutty et al. (1975)
India	56-year-old, M	Arm	B	3	Agarwal et al. (1976)
India	54-year-old, F	Leg	B	3	Thomas (1976)
Malaysia	20-year-old, M	Foot	B	4	Prathap and Dissanaike (1976)
Malaysia	23-year-old, M	Neck	A	4	Prathap and Dissanaike (1978)
Unknown	Unknown	Skeletal muscle	A	4	Frenkel (1976)
USA via Asia	40-year-old, M	Arm	B	4	McLeod et al. (1979)
Uganda	50-yr-old, M	Leg	B	1	Beaver (1979)
India	50-yr-old, M	Arm	B	3	
Singapore	Unknown	Skeletal muscle	B	4	
Singapore	Unknown	Skeletal muscle	B	4	
Costa Rica	9-yr-old, M	Heart	A	6	

Source: Complied from Beaver, P.C., Gadgil, R.K., Morera, P., 1979. *Am. J. Trop. Med. Hyg*. 28, 819–844.
A = Autopsy, B = Biopsy, F = Female, M = Male.

[a] Beaver et al.[84] reported 7 sarcocyst wall types that are not comparable with nomenclature stated by Dubey et al.[393] that classified the cysts wall by using TEM. Types reported by Beaver are: Type 1, thick, radially striated wall; large zoites often sparse in center; metrocytes. Type 2, sarcocysts generally large; thin, smooth wall; medium size zoites; septa. Type 3, sarcocysts small and medium size; thin wall, medium size zoites; septa. Type 4, sarcocysts long, medium size; thin wall; small zoites; septa evident in center. Type 5, sarcocysts small; thin wall; medium size zoites; in myocardium. Type 6, sarcocysts small to medium; thin wall; large zoites; in myocardium. Type 7, sarcocysts small; thin wall; small zoites; in myocardium.

[b] Original author and year can be found in Reference **84** and not all are included in the present Bibliography section.

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Table 4.3 Summary of Reports of Muscular Sarcocystosis in Humans not Included in the review by Beaver[84] and Presented in Chronological Order of Publication

Country	No. of Patients, Age, Gender	Sarcocyst Location	Biopsy or Autopsy	Year of Diagnosis	Reference
USA	0 of 297 diaphragm specimens	Diaphragm	A	1978	**1738**
Malaysia	58-year-old, M[a]	Tongue	B	1981	**1374**
India	2 persons	Leg Gluteus	B B	1983	**20**
Denmark	4 of 112 specimens[b]	Muscle	A (not clear)	1985	**736**
Malaysia	45-year-old, M[a] 53-year-old, M[a]	Nasopharynx Nasopharynx	B B	1987	**1375**
Malaysia	1 person	Skeletal muscle	B	1988	**1376**
Australia via Thailand	31-year-old M	Skeletal muscle	B	1990	**1361**
Egypt	1 person	Skeletal muscle	B	1990	**2**
Malaysia	45-year-old, M[a] 67-year-old, F[a] 32-year-old, F[a]	Tongue Tongue Pectoral muscle	B B B	1992	**1377**
Malaysia	21 16–57 years	Tongue	A	1992	**1838**
Australia	0 of 50 specimens	NI	A	1992	**1762**
Belgium via Brazil, Kenya and Tanzania	31-year-old, M	Thigh	B	1995	**1773**
India	40-year-old, M 14-year-old, F 52-year-old, F 23-year-old, F	Thigh Arm Thigh Arm	B B B B	1996	**1179**
Malaysia	44-year-old, F[a] 19-year-old, F	Thigh Calf	B B	1998	**1602**
Malaysia	7 Adults, M	1 muscle	B	1999	**36**
Argentina	31-year-old HIV patient	Liver, blood smear	B	2008	**1782**
Thailand	66-year-old, M[a]	Larynx	B	2011	**1024**
India	20-year-old, M 50-year-old, M	Arm Neck	B B	2012	**1100**
India	47-year-old, M	Leg	B	2013	**60**
Malaysia, Pangkor Island	89 people	Leg	B	2013, 2014, 2014	**14, 877, 1034**
Malaysia, Tioman Island	39 F 29 M 4–72 years	Skeletal muscle from 15 patients; 1 PCR positive	B	2014	**560, 1624**
Malaysia, Tioman Island	3 F 3 M	Febrile myositis syndrome	NTE	2014	**1726**

Source: Modified from Fayer, R., Esposito, D.H., Dubey, J.P., 2015. *Clin. Microbiol. Rev.* 28, 295–311.
NTE = No Tissue Examined, A = Autopsy, B = Biopsy, F = Female, M = Male.
[a] Cancer patients.
[b] Tissues examined by trichinoscopy.

were confirmed by histological observation of intramuscular cysts compatible with sarcocysts. The onset of symptoms that clustered during the second week and the sixth week after returning from the island included fever (82%), myalgia (100%), and headache (59%), similar to the symptoms of visitors to Pangkor Island, as well as fatigue (91%) and arthralgia (29%). Blood eosinophilia and elevated serum CK levels were first observed during the fifth week postdeparture.

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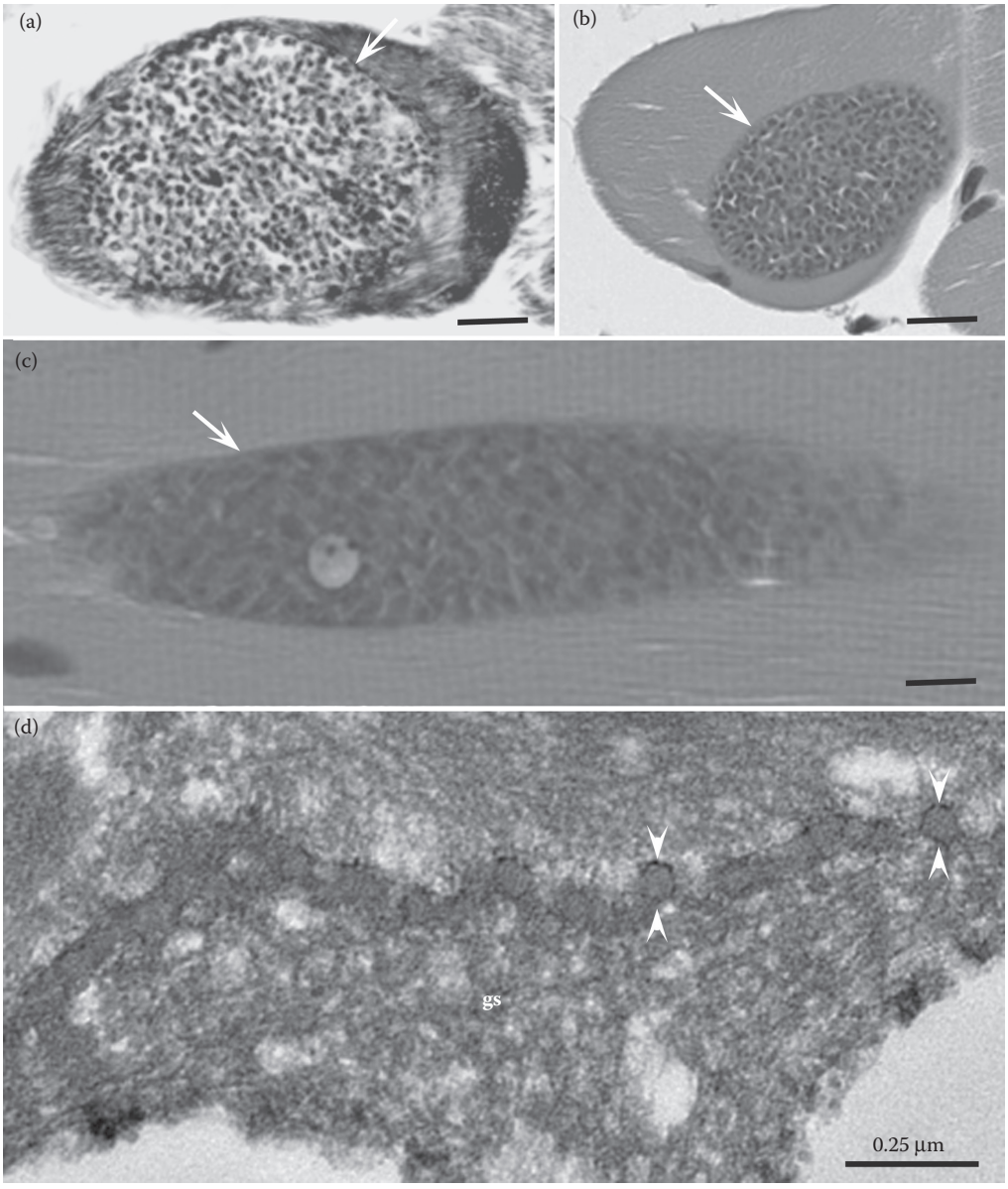


Figure 4.1 Sarcocysts in skeletal muscle biopsies from 3 humans with clinical sarcocystosis. (a, b, c) light microscopy, H and E stain. Bar = 10 µm. The sarcocysts are thin-walled (arrows). (d) TEM. The parasitophorous vacuolar membrane has tiny blebs (opposing arrowheads), and gs is relatively thin. ((a) Adapted from Arness, M.K. et al., 1999. *Am. J. Trop. Med. Hyg*. 61, 548–553. With permission; (b, c, d) from 2 patients reported in Esposito, D.H. et al., 2014. *Clin. Infect. Dis*. 59, 1401–1410; (b and c) Courtesy of Clifton P. Drew; (d) Courtesy of Cynthia S. Goldsmith.)

Additional details from 39 German patients with a history of travel to Tioman Island during 2011–2014, some of whom were included in the overall series during 2011–2012 (28 patients), were later reported.[1624,1891] These patients had a median duration of illness of 2.2 months (range 0–23 months), and 17% of patients had symptoms lasting >6 months; 2 patients had unresolved but diminished symptoms at 13 and 23 months. Severity of pain on a scale of 0–10 was a median

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of 6 (range 0–10). This represents the first data from multiple patients that indicate the duration of muscular sarcocystosis illness. Recently, an induction of Th2 cytokine polarization and biphase cytokine changes have been reported for muscular sarcocystiosis.[2402]

4.3.1 Evidence Linking Acute Sarcocystosis to *S. nesbitti*

S. nesbitti was originally described in muscles from a rhesus monkey from India, based on LM (see Chapter 5), but its taxonomic validity is questionable due to the lack of a good morphologic description. The first report of *Sarcocystis* in monkeys (*Macaca fascicularis*) in China was based on LM and TEM studies.[1859] Based on a perceived resemblance to *S. nesbitti*, the authors named the organisms they found in monkey muscles *S. nesbitti.* Morphological similarities suggested that a single species of *Sarcocystis* might infect *Macaca mulatta, M. fascicularis*, *Cercocebus atys*, and *Papio papionis*, as well as humans.[1743] When muscles containing sarcocysts identified as *S. nesbitti*[1859] were examined by molecular methods,[1743] DNA sequence data from 2 sarcocysts clustered with that of *S. atheridis* and *S. singaporensis* suggested that a snake might be a definitive host for *S. nesbitti*. The DNA sequence of *Sarcocystis* in feces from a cobra[1034] clustered with *Sarcocystis* from other snakes and with the *S. nesbitti* sequences obtained from monkey muscle.[1743] Sarcocysts were found in muscle biopsies from the swollen jaw of 1 patient and from the leg of another associated with the outbreak on Pangkor Island.[1034] The DNA sequences for *Sarcocystis* from these patients varied 1% from each other, and a BLAST result found they shared 99% homology with *S. nesbitti* from the muscle of *M. fascicularis.*

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Information pertinent to the subject matter of this chapter may be found in the following references:

Intestinal sarcocystosis: **40, 64, 124, 152, 210, 211, 213, 218, 314, 597, 610, 667, 678, 679, 807, 821, 866, 897, 919, 1011, 1012, 1035, 1038, 1051, 1053, 1057, 1059, 1181, 1278, 1382, 1394, 1398, 1399, 1401, 1478, 1480, 1638, 1639, 1645, 1685, 1707, 1714, 1720, 1764, 1782, 1828, 1865, 1884, 1885, 1888, 1954, 2095, 2222, 2314, 2332.**

Muscular sarcocystiosis: **2, 14, 20, 36, 60, 84, 559, 560, 595, 596, 736, 877, 1024, 1034, 1059, 1100, 1179, 1264, 1361, 1374–1377, 1410, 1602, 1607, 1624, 1725, 1726, 1738, 1740, 1743, 1762, 1773, 1781, 1838, 1859, 1891, 1916, 2104, 2161, 2264, 2324, 2348, 2355, 2389, 2402.**

Miscellaneous: **40, 1897, 1908, 1909, 1935, 1954, 2012, 2066, 2077, 2078, 2096, 2134, 2140, 2309, 2334.**

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CHAPTER 5

Sarcocystosis in Nonhuman Primates

5.1 INTRODUCTION

Although there are numerous reports of muscular *Sarcocystis* infections in New World monkeys[943,961,1428] and Old World monkeys,[477,795,939,943,1104,1112,1175,1737] only 3 species have been named (Table 5.1). Morphological studies of sarcocysts indicate that there are more. In a review of histologic sections from 744 monkeys, including 374 captive wild monkeys, 4 structurally distinct sarcocysts were found in 21% of the wild captive monkeys, but none was found in laboratory-raised monkeys.[943] Identification of the species of the sarcocysts in primates is difficult because the definitive hosts are not known. Mehlhorn et al.[1175] reported 3 species of *Sarcocystis*, with thin-walled cysts found in rhesus monkeys (*Macaca mulatta*), baboons (*Papio cynocephalus*), and tamarins (*Sanuinus oedipus*), were ultrastructurally distinct (Table 5.1).

5.2 *SARCOCYSTIS KORTEI* CASTELLANI AND CHALMERS 1909

Intermediate hosts include *Macaca mulatta*, *Erythrocebus patas*, *Cercopithecus mitis*, and probably others.

The sarcocysts in skeletal muscles are up to 800 μm. The sarcocyst wall, which appears hairy, is 5- to 6-μm thick. The bradyzoites are 9–14 × 3–4 μm in size.[1104]

5.3 *SARCOCYSTIS NESBITTI* MANDOUR 1969

This species was found in of 2 of 44 *Macaca mulatta* from India.[1104] Sarcocysts were found in skeletal muscles but not in the heart. They were up to 1100 μm long. The serrated cyst wall was 1.2- to 1.6-μm thick. A similar parasite was found in 12 (24%) of 51 *M. mulata* imported into New York, United States, but the origin of the monkeys was not stated.[987] Sarcocysts were found in all muscles examined, but in all instances no more than 17 sarcocysts were found. Sarcocysts were thin-walled by LM. By TEM, vp of the sarcocyst wall were up to 60 nm long. Bradyzoites were 4.3–7.8 μm long.

In China, sarcocysts were found in 1 of 69 *M. fascicularis* that were born on a farm.[1859] Sarcocysts were 2.3–14 mm long, and up to 0.1 mm wide. By LM the sarcocyst wall was thin and smooth. By TEM the sarcocyst wall had small vesicular invaginations, 19–380 nm in diameter and 65–120 nm in depth, type 1.[1859] Bradyzoites were 7.5–10 × 1.25 μm by LM, and 3.3–6.5 × 1.0–2.79 by TEM. Yang et al.[1859] reviewed the morphology of sarcocysts found in monkeys presented by other authors, and concluded that the species they found was *S. nesbitti*; they did not mention references.[987] Because there are no photographs of the parasite named by Mandour,[1104] it is impossible to know if the same parasite was present in the above reports.

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Table 5.1 Prevalence of *Sarcocystis* in Nonhuman Primates

Country	Host[a]	Origin	No. Tested	No. Positive (%)	Remarks	Reference[b]
Africa	*Macaca mulatta*, *Erythrocebus patas*, *Cercopithecus mitis*	NS	3	3	*S. kortei*	**1104**
China	*Macaca fascicularis*	Monkey farm	69	1 (1.4%)	*S. nesbitti*	**1743, 1859**
Germany	*Macaca mulatta*	Wild caught	20	2 (10.0%)	Cysts up to 1 mm, thin walled, TEM type 1, 6 μm long bradyzoites	**1175**
Germany	*Papio cynocephalus*	Wild caught	2	1 (50.0%)	Cysts up to 800 μm, TEM type 1a, folded vp, 6.4–9.0 μm long bradyzoites	**1175**
Germany	*Saguinus oedipus*	Wild caught	9	2 (22.2%)	Cysts up to 350 μm, TEM type 1a, papillary vp, 4–5 μm long bradyzoites	**1175**
India	*Macaca mulatta*	NS	44	2 (4.5%)	*S. nesbitti*	**1104**
Japan	*Saimiri sciureus*	Imported from animal dealer	37	7 (18.9%)	Histology in femoral muscle	**1695**
Japan	*Saimiri sciureus*	NS	10	8 (10.0%)	SEM, thin-wall sarcocysts	**961**
Malaysia	*Macaca fascicularis*, *Macaca irus*	NS, used in animal experimentation	50	7 (14.0%)	TEM, wavy and invaginated cyst wall	**939, 1423**
Mexico	*Macaca mulatta*	NS	1	1	Eosinophilic myocarditis, no description of cysts	**795**
South Africa	*Papio ursinus*	Wild caught	94	44 (46.8%)	Thick and palisade-like wall	**1153**
South Africa	*Cercopithecus pygerythrus*	NS	134	11 (8.2%)	*S. markusi*	**1112**
USA	*Macaca mulatta*	NS, used in animal experimentation	4	3 (75.0%)	Different stages of development	**477**
USA	*Macaca mulatta*	Imported from animal dealer	370	4 (1.1%)	*S. kortei*	**765**
USA	*Erythrocebus patas*	NS	1	1	Thick and striated wall	**1428**
USA	14 different species from Old and New World monkeys	Wild caught and laboratory born	774	0% in laboratory born, 21% in wild captive	4 structurally distinct sarcocysts	**943**
USA	*Macaca mulatta*	Imported from animal dealer	51	12 (24.0%)	TEM, description of the sarcocysts	**987**

NS = not stated.

[a] Host species: Rhesus macaque (*Macaca mulatta*), crab-eating macaque (*Macaca fascicularis*), cynomolgus monkey (*Macaca irus*), baboon (*Papio cynocephalus*), chacma baboon (*Papio ursinus*), Patas monkey (*Erythrocebus patas*), common squirrel monkey (*Saimiri sciureus*), blue monkey (*Cercopithecus mitis*), velvet monkey (*Cercopithecus pygerythrus*), tamarin (*Saguinus oedipus*).

[b] There are several other reports on the presence of *Sarcocystis* in monkeys.[943]

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DNA was extracted[1743] from 2 unfixed *S. nesbitti* sarcocysts from the monkey from China.[1859] Characterization of the 18S rRNA gene indicated a close relationship to *S. singaporensis*, which cycles between pythons and rodents, suggesting that snakes are likely a definitive host for *S. nesbitti*. The genetic sequences from *S. nesbitti* matched the sequences derived from a sarcocyst from 3 symptomatic cases of *Sarcocystis* in a human from Malaysia.[14,877] It is probable that snakes may act as definitive hosts for *S. nesbitti*, because recently, based on amplification of the 18S rRNA genes, sequences were found matching this species in sporocysts excreted by reticulated python (*Braghammerus reticulatus* syn. *Python reticulatus*) and monocled cobra (*Naja kaouthia*) from Malaysia.[1033] No bioassay was carried out. Evidence—or lack of it—to link *S. nesbitti* to human infections is discussed in Chapter 4.

5.4 *SARCOCYSTIS MARKUSI* (MARKUS, KAISER, AND DALY 1981) ODENING 1997

5.4.1 Intermediate Host

South African Vervet monkey (*Cercopithecus pygerythrus*).

5.4.2 Definitive Host

Unknown.

5.4.3 Structure and Prevalence

Sarcocysts were found in muscles of 11 of 134 *C. pygerythrus* in South Africa.[1112] The sarcocysts' size was not stated. Bradyzoites were 10–12 × 2–3 µm. Sarcocysts had unique vp, type 9i. Although the size of the vp was not stated by the authors, measurements from the illustrations indicate that vp were approximately 10 × 2.5 µm. The vp contain many smooth microtubules. The PVM was lined by an electron-dense layer that was invaginated.

5.5 CLINICAL SARCOCYSTOSIS IN NATURALLY INFECTED MONKEYS

There are 4 clinical reports of sarcocystosis in monkeys, all in *Macaca mulatta*, all from the United States, and all with different clinical presentations. The first case was a young male rhesus weighing 3.6 kg that was asymptomatic before being introduced into the colony.[1737] Three months later, the monkey developed myositis, with stiffness of all limbs and edema of the scrotum. A necropsy was performed because of the deteriorating condition. Severe myositis was associated with numerous sarcocysts. There were lesions similar to eosinophilic myositis. The sarcocysts were up to 650 µm long. The sarcocyst walls were 4–5 µm thick with perpendicular vp, similar to those described for *S. kortei*. Bradyzoites were 9–12 µm long. These authors also reviewed literature and concluded that clinical sarcocystosis is relatively rare in monkeys.

The second case was a 22-month-old rhesus that became anorexic and dull.[1020] The monkey was euthanized in moribund condition. Gross lesions included stippling and streaking on the myocardium and with facial ecchymosis and edema. The most striking findings were numerous intravascular schizonts in many organs. Some schizonts occluded capillaries in the brain (Figure 5.1). The presence of schizonts was confirmed by TEM. Immature sarcocysts with metrocytes were seen in skeletal muscles. The sarcocyst wall appeared to be thick (Figure 5.1). PCR amplification placed the organism in the genus *Sarcocystis* but final identity was not confirmed.

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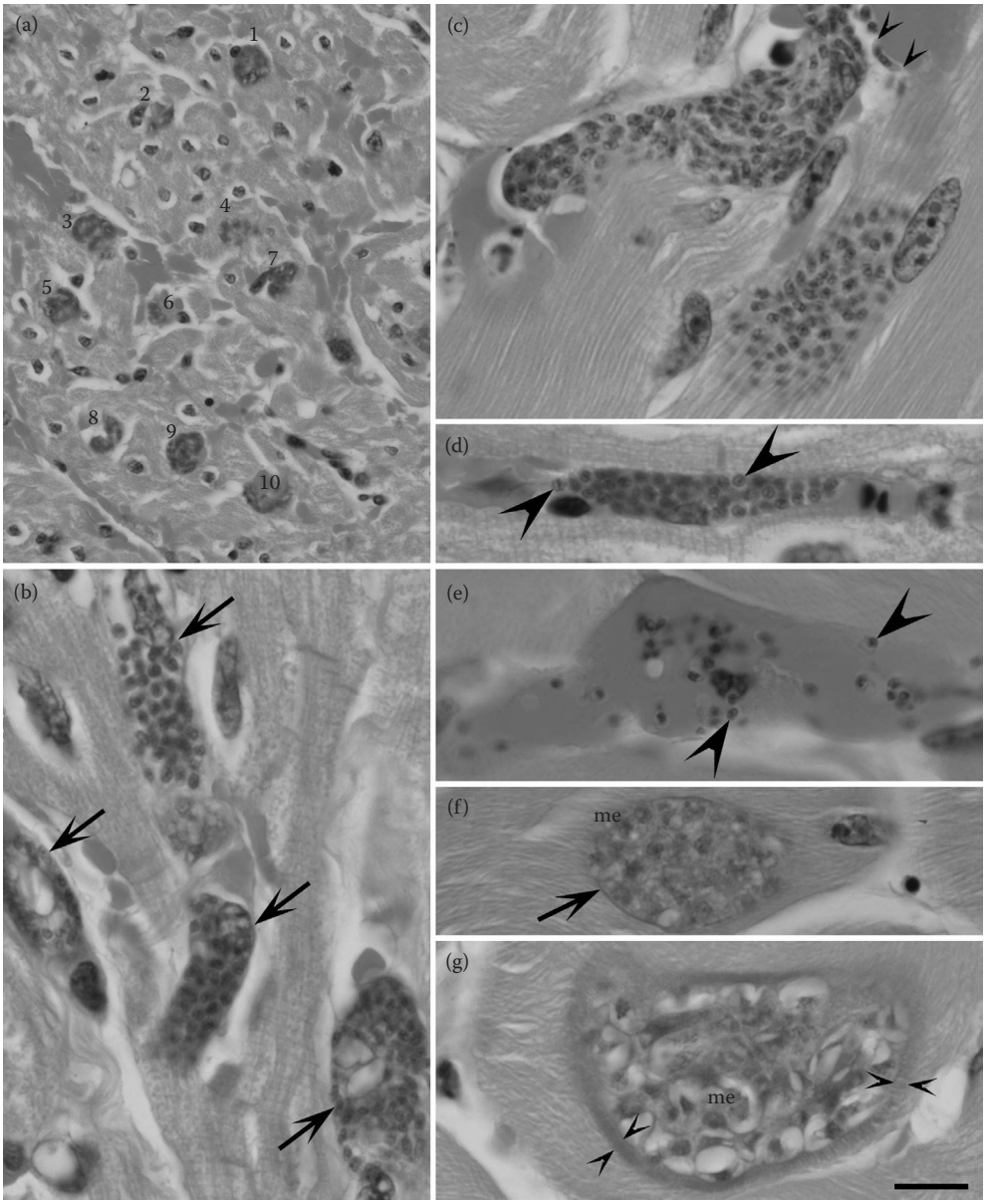


Figure 5.1 *Sarcocystis* sp. schizonts in heart (a–e) and sarcocysts in skeletal muscle (f, g) of a rhesus monkey (*Macaca mulata*), H and E stain. (a) Ten schizonts that appear like sarcocysts at this magnification. (b) Four intravascular schizonts (arrows). (c) Two schizonts (arrowheads point to a longitudinally cut merozoite). (d) Elongated schizont (arrowheads) in a capillary (all merozoites in cross-section). (e) Blood vessel with free intravascular merozoites (arrowheads). (f) Early immature sarcocyst with a very thin sarcocyst wall (arrow) enclosing metrocytes (me) that stain faintly with H and E stain. (g) Immature sarcocyst enclosing me with a thick sarcocyst wall (between arrowheads). Bar applies to all parts; bar in (a) = 50 μm, bars in (b–g) = 10 μm. (Courtesy of the authors in Lane, J.H. et al., 1998. *Vet. Pathol.* 35, 499–505.)

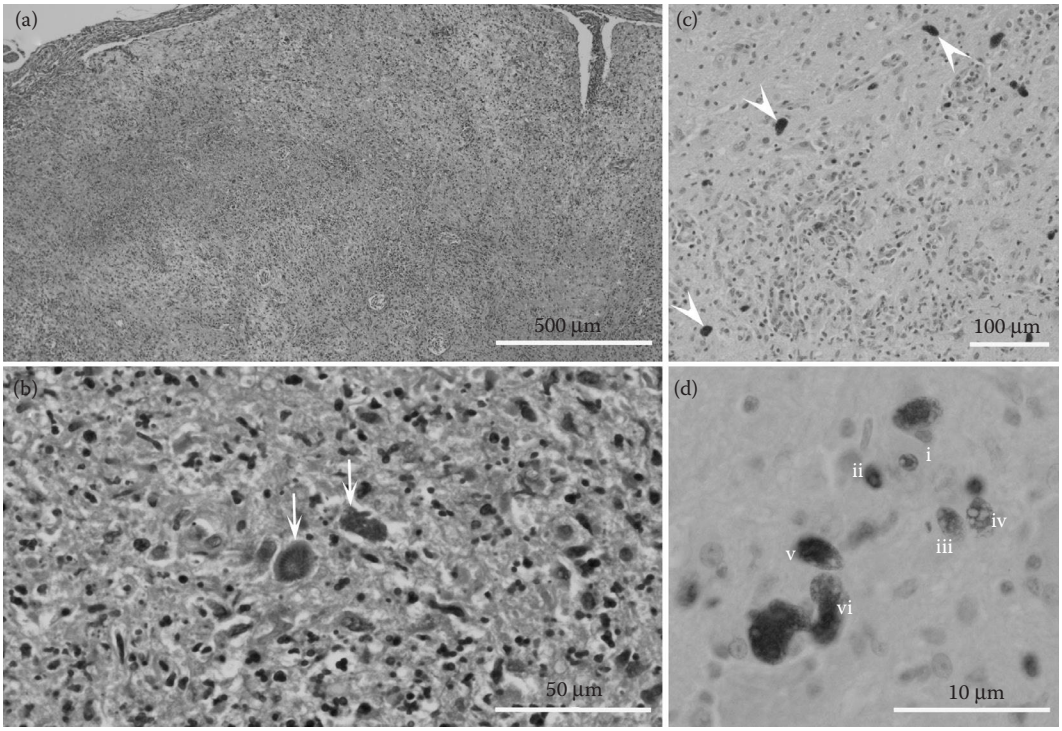


Figure 5.2 *Sarcocystis* myelitis in spinal cord of a *Macaca mulatta*. (a) Severe, extensive inflammation in grey and white matter and obliterating the spinal canal. H and E stain. (b) Higher magnification to indicate necrosis and infiltration with mixed leukocytes. Arrows point to schizonts. There are many free merozoites that are not visible at this magnification. H and E stain. (c) Vasculitis and glial cell proliferation. Arrowheads point to schizonts. Immunohistochemical staining with anti-*S. cruzi* rabbit antibodies. (d) Higher magnification of Figure 5.2c. Note the developing meronts (i–vi, in presumed order of development). The meront nucleus enlarges and becomes lobed (Adapted from Klumpp, S.A. et al., 1994. *Am. J. Trop. Med. Hyg*. 51, 332–338.)

The third case was a 2-year-old monkey euthanized as part of a clinical trial. Diffuse, pale streaking of several muscles was noted.[723] Enormous numbers of sarcocysts were seen in skeletal muscles, some associated with myositis (Figure 5.2). The sarcocyst wall was thick, with vp 2.7–6.3 µm long and 0.5–1.1 µm wide. Bradyzoites were 10–13 µm long. PCR amplification of the 18S rRNA gene showed a 91% similarity to *S. hominis*, but sequencing of the ITS-1 fragment showed little homology to any previously published sequences; therefore, the authors suggested a new/unique *Sarcocystis* species.

The fourth case was a rhesus born in captivity and used for research in simian immunodeficiency virus (SIDV). It was inoculated with SIDV at 18 months of age. Seven months PI with SIDV, the animal became lethargic and had a head tilt. After euthanasia, a necropsy was performed. Histologically, the most prominent lesion was in the lumbar spinal cord, with severe inflammation and obliteration of the central canal (Figure 5.3), marked by necrosis, mixed leukocyte cell infiltrations, perivasculitis, and proliferation of astrocytes (Figure 5.3). Both immature and mature schizonts were in the lesions. Ultrastructurally, schizonts divided by endopolygeny, and merozoites, lacked rhoptries.[968] The parasite reacted to polyclonal *S. cruzi* antibodies but not to *S. neurona*, *T. gondii*, and *N. caninum* antibodies.

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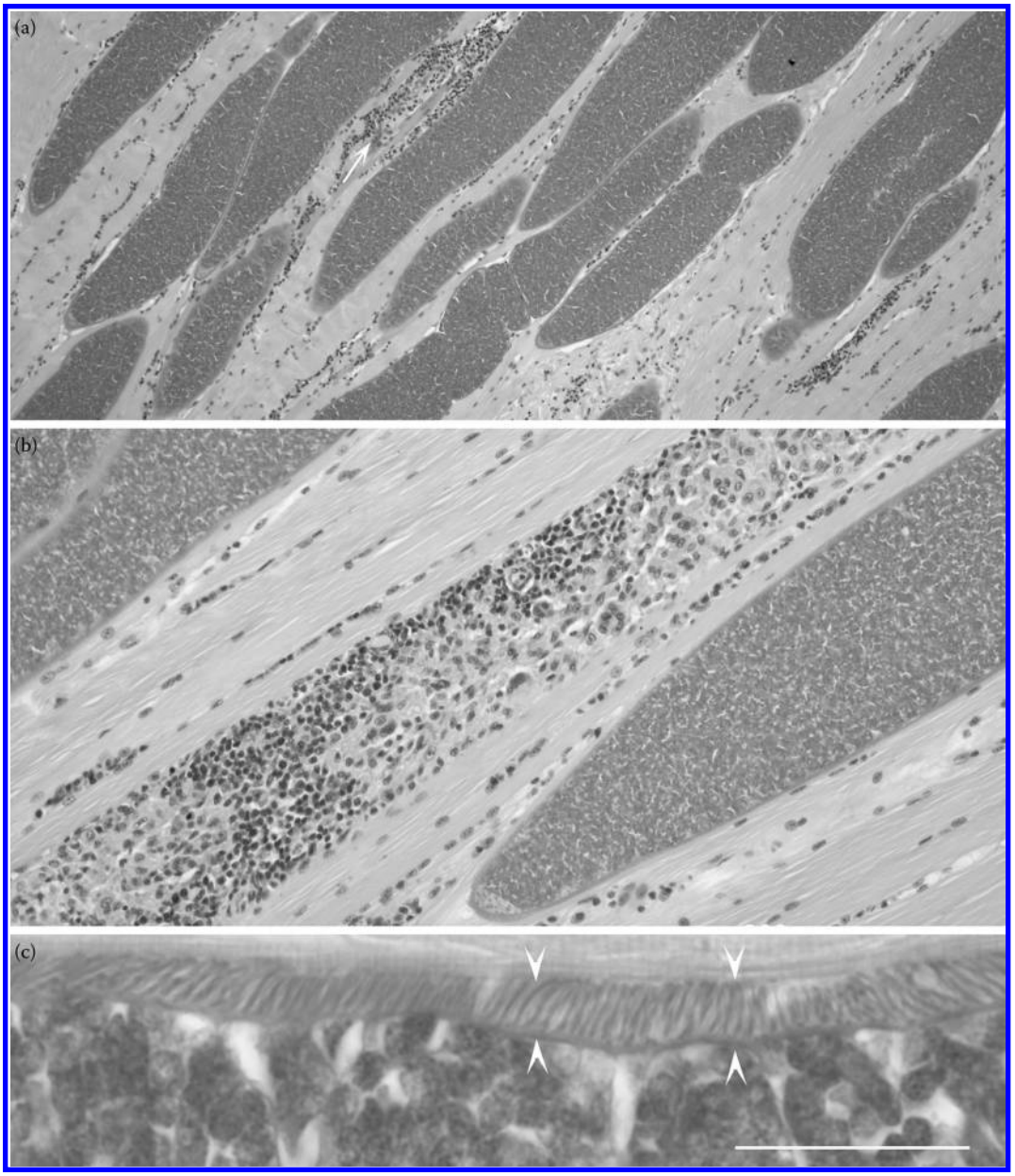


Figure 5.3 *Sarcocystis*-associated myositis in a rhesus monkey. (a) Numerous sarcocysts in skeletal muscle. Arrow points to an inflammatory focus. (b) Inflammatory focus with mononuclear cells and giant cells. (c) Sarcocyst wall with elongated villar protrusions on the sarcocyst wall (arrowheads). H and E stain. Bar applies to all parts; bar in (a) = 500 μm, in (b) = 200 μm, and (c) = 20 μm. (Courtesy of Gozalo, A.S. et al., 2007. *Vet. Pathol.* 44, 695–699.)

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Information pertinent to the subject matter of this chapter may be found in references: **14, 477, 723, 765, 795, 877, 939, 943, 961, 968, 987, 1020, 1033, 1104, 1112, 1153, 1175, 1423, 1428, 1695, 1737, 1743, 1859**.

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CHAPTER 6

Sarcocystosis in Pigs (*Sus scrofa*)

6.1 INTRODUCTION

There are 2 valid species in pigs: *S. miescheriana* and *S. suihominis* (Figure 6.1). *S. miescheriana* is type species of the genus.

6.2 *SARCOCYSTIS MIESCHERIANA* (KÜHN 1865) LABBÉ 1899

6.2.1 Definitive Hosts

Dog (*Canis familiaris*), raccoon (*Procyon lotor*), wolf (*Canis lupus*), red fox (*Vulpes vulpes*), and jackal (*Canis aureus*).[1188]

6.2.2 Structure and Life Cycle

Sarcocysts are up to 1500 µm long and 200 µm wide and are found in skeletal and cardiac muscles. The sarcocyst wall is 3–6 µm thick and appears radially striated. The vp on the sarcocyst wall are up to 5 µm long and 1.3 µm wide, type 10b (Figure 6.1a and c).[547,1176] Bradyzoites are up to 20 µm long.

Information on endogenous development is summarized in Table 6.1. There are 2 known generations of schizogony, which develop much more rapidly than those of cattle and sheep. First- and second-generation schizonts mature within 13 DPI; immature sarcocysts are found as early as 27 DPI.[75,814,832]

6.2.3 Pathogenicity

S. miescheriana can cause weight loss and purpura of the skin, especially of the ears and buttocks, dyspnea, muscle tremors, abortion, and death, depending on the number of sporocysts ingested.[74,75,547,549,550,1880] Ingestion of less than 1 million sporocysts generally results in subclinical infection. However, 1 pig fed 200,000 sporocysts died at 60 DPI, probably related to heart failure.[301] Stress of pregnancy may modify the severity of infection. Of 5 pregnant sows experimentally infected with 50,000 sporocysts each, all became ill: 2 aborted 12–14 DPI, 1 died, and 2 became moribund and were euthanatized.[549] A similar number of sporocysts fed to finishing pigs caused no clinical signs, but resulted in weight gains that were 11%–27% less than uninfected controls.[118] Administration of 25,000 or 15,000 sporocysts neither reduced weight gains nor produced clinical signs.

The effect of subclinical sarcocystosis on meat quality has been studied.[296–298] Pigs inoculated with 50,000 *S. miescheriana* sporocysts weighed 5–12 kg less than uninfected pigs, when examined 3 months PI; however, the quality of meat as determined by water-absorbing and water-binding capacity, pH, rigor values, color brightness, and back-fat ratios, was not reduced, compared with

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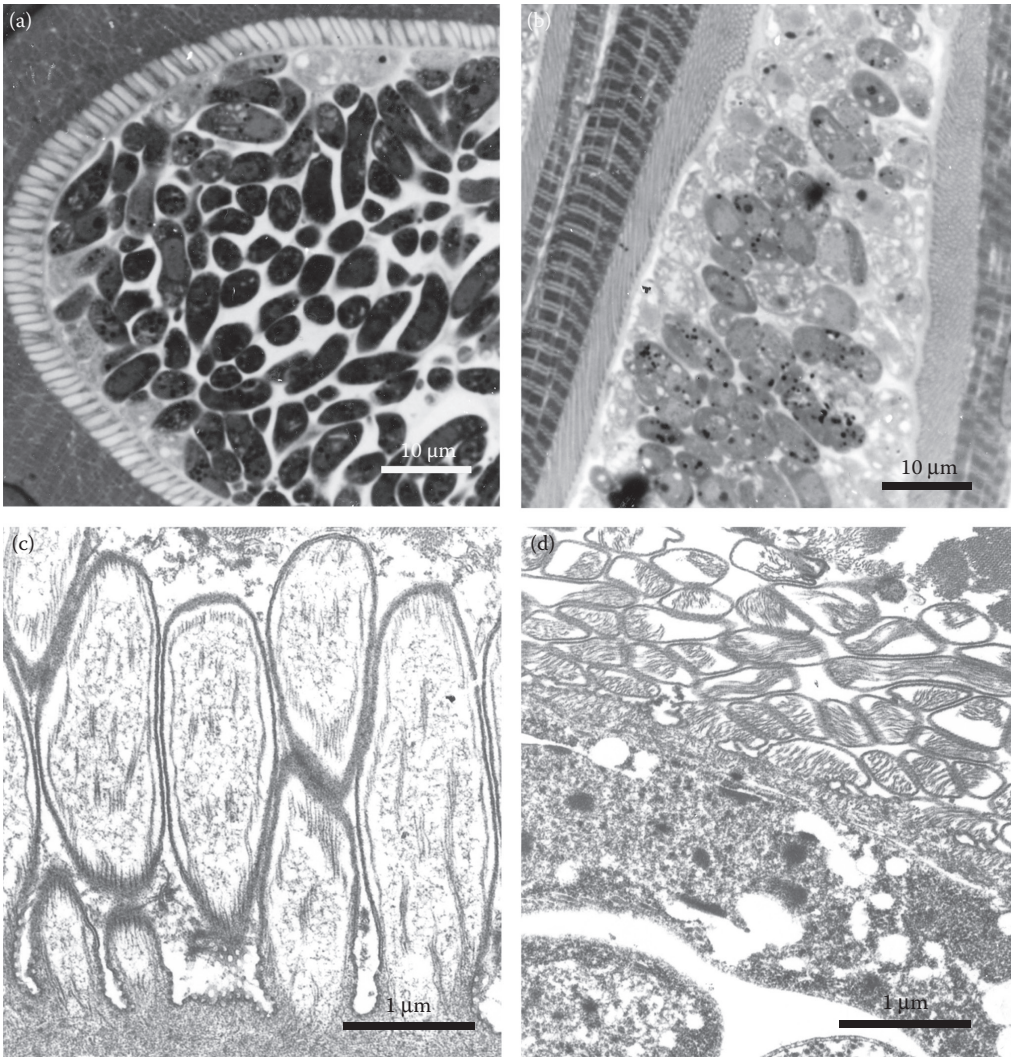


Figure 6.1 Sarcocysts of *S. miescheriana* (a and c) and *S. suihominis* (b and d) in skeletal muscles of pigs. The vp of *S. suihominis* are thinner and longer than those of *S. miescheriana*. a and c, Toluidine blue stain. c and d, TEM.

that of uninfected pigs.[296,298] Host genetics could affect susceptibility to *Sarcocystis* infection. The number of bradyzoites were 20 times higher in European Pietrain (n = 14) than in the Chinese Meishan (n = 11) pigs that were fed 50,000 *S. miescheriana* sporocysts and monitored for severity of disease.[1457] The Pietrain pigs developed more severe disease than the Meishan pigs and 2 Pietrain pigs died, versus no deaths in the Chinese pigs. In a subsequent study, the authors provided evidence for genes regulating resistance to *S. miescheriana*.[1459]

6.2.4 Protective Immunity

Pigs immunized once with 1000–50,000 *S. miescheriana* sporocysts orally become refractive to lethal challenge with large numbers of *S. miescheriana* sporocysts.[1488,1559,1560,1813,1880] This protection was found when the challenge infection was initiated at 80 DPI, but not at 120 DPI.[1880] Pigs fed

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Table 6.1 Comparison of Development at Stages of *Sarcocystis* Species in Pigs

	S. miescheriana	*S. suihominis*
	First-Generation Schizonts	
Location	Liver endothelium	Liver endothelium
Duration (DPI)	6–7	5–6
Size of meronts (μm)	Unknown	Unknown
No. of merozoites	Unknown	Unknown
Size of merozoites (μm)	Unknown	Unknown
	Second-Generation Schizonts	
Location	Many organs	Many organs
Duration (DPI)	7–13	7–20
Size of meronts (μm)	7–17 × 11–79	Unknown
No. of merozoites	20–94	50–90
Size of merozoites (μm)	Unknown	6.5 × 3.2
	Parasitemia	
Duration (DPI)	Unknown	Unknown
Blood-cell multiplication	Unknown	Unknown
	Sarcocysts	
Wall (μm)	Thick, striated (2.7–6)	Thick, striated (4–9)
First seen (DPI)	27	27
Maximum length (μm)	Up to 1500	Up to 1500
Infective (d)	58	56
Bradyzoites (μm)	17.1 × 3.8	15 × 4.5
Metrocytes	10.8 × 4.6	9.8 × 4.9
Main references	**51, 75, 707, 814**	**51, 806, 809, 1174**

multiple low doses of sporocysts (trickle infections) also developed protection.[1559] This protective effect resulting from feeding of *S. miescheriana* sporocysts was not protective against a challenge infection with *S. suihominis* sporocysts.[550]

6.3 *SARCOCYSTIS SUIHOMINIS* (TADROS AND LAARMAN 1976) HEYDORN 1977

6.3.1 Distribution

Europe, India, Japan.

6.3.2 Definitive Hosts

Humans (*Homo sapiens*) and nonhuman primates (*Macaca mulatta*, *Macaca irus*, *Pan troglodytes*, and *Papio cynocephalus*).

6.3.3 Structure and Life Cycle

Sarcocysts are up to 1500 μm long.[547] The sarcocyst wall is 4–9 μm thick[547] and appears hirsute, with vp up to 13 μm long, type 31 (Figure 6.1b and d).[1174] The bradyzoites are approximately 15 μm long.

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Life-cycle data is summarized in Table 6.1. The 2 generations of schizogony develop at about the same time as—and are structurally similar to—those of *S. miescheriana*.[806,808,809] Immature sarcocysts were first seen 27 DPI, and bradyzoites were seen 56 DPI.[1174]

6.3.4 Pathogenicity

S. suihominis is pathogenic for pigs. Pigs fed 50,000 or more sporocysts became ill, and half of those fed 1 million sporocysts died.[806] Clinical signs in pigs were similar to those in pigs infected with *S. miescheriana*.[806]

6.3.5 Genetic Diversity

There are few reports on the genetic diversity of *S. miescheriana*, which appears to be very low. Thus, minimal differences have been detected for the 18S rRNA gene in isolates from domestic pigs and wild boars in Iran, Lithuania, China, Switzerland, Germany, and the United States.[161,168,956,1854]

6.4 *SARCOCYSTIS PORCIFELIS* DUBEY 1976

The validity of this species is uncertain. This species was named[349] for the parasite described from the former USSR (Union of Soviet Socialist Republics).[716] *Sarcocystis*-infected esophagi from pigs were fed to 4 cats.[716] The cats excreted sporulated oocysts 5–10 days after ingesting the infected swine tissues. The sporocysts were 13.5 × 7.6 µm and each contained 4 sporozoites measuring 9.5 × 3.8 µm. The sporocysts from cat feces were fed to 8 littermate pigs. All pigs became ill and 1 died 89 DPI. Sarcocysts were found in skeletal muscles and the heart. Other pigs were killed 3.5 months after ingestion of sporocysts, and sarcocysts were found in them.[716] The structure of the sarcocysts was not described.

In Romania, sarcocysts were found in the muscles of a wild pig during examination for *Trichinella*.[1694] Sarcocysts were 800 µm long. Infected meat was fed to 2 cats. Both cats excreted sporocysts from day 9–13 after ingesting pork. The sporocysts were 14.5 × 8.9 µm.

A few sarcocysts of a third type, different from *S. miescheriana* and *S. suihominis*, were reported in wild boars from Lithuania.[33] But no data on morphological description was provided. Authors comment that, most likely, this finding could be an artifact (Kutkiené, L., pers. comm.).

In Japan, 1 case of *S. porcifelis* was reported in slaughtered pigs.[1323] The sarcocysts had thin and smooth walls, as can be seen in Figure 3 provided by authors; no additional details are given.

In India, a macroscopic sarcocyst was found in the diaphragm of a naturally infected domestic pig.[791] The sarcocyst wall was broken during separation from the host tissue. The sarcocyst was 4.1 mm long, and the wall was smooth and thin, with uniform radial striations that were 0.85 µm long. The bradyzoites were 7 × 3 µm in size. The authors called it *S. porcifelis*. Until now, this is the best morphological description of the sarcocyst.

Note: *Sarcocystis* sporocysts have never been reported in feces of cats that have been fed pork. In a national survey, in the United States, for *Toxoplasma gondii* infection, 2049 samples of pork were fed to cats and feces of cats were examined for oocysts[458]; *Sarcocystis* sporocysts were not found (unpublished). In other investigations, sporocysts were not found in feces of cats fed *Sarcocystis*-infected pork from domestic pigs[353] or from wild pigs.[73]

6.5 PREVALENCE OF *SARCOCYSTIS* AND ECONOMIC IMPACT

The prevalence of sarcocysts in domestic pigs (Table 6.2) is lower than in wild pigs (Table 6.3). The prevalence in finishing pigs or market-age pigs is lower than in adult culled pigs.

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Table 6.2 Surveys of *Sarcocystis* spp. in Domestic Pigs

Country	Year	No.	Method	No. Positive	% Positive	Species	Reference
Australia	1974	169	Histology	7	1.4 (<1 year) 16.7 (>1 year)	NS	**1244**
Austria	1978–79	712	Trypsin digestion	130	18.3 5.5 (fattening) 31.6 (sows)	*S. suihominis* (4.1%) "*S. suicanis*" (13.6%) Mixed (0.7%)	**832**
Chile	NS	100	Pepsin digestion	79 (esophagus)	85.0 73.2 (<1 year) 100.0 (>1 year)	NS	**721**
China, Guangxi	2004	42	NS	30	71.4%	NS	**1720**
China	2011	4	Histology, PCR	4 isolates	–	*S. miescheriana*	**1854**
Czech Republic	1979–80	2000	Trypsin digestion, trichinoscopy	0	0.0	NS	**764**
Czech Republic	NS	335	Trypsin digestion	8	4.0	NS	**1086**
Czech Republic	NS	1409	Inspection, pepsin digestion	Not clear	0.9 Not clear	NS	**1101**
Egypt	NS	105	Bioassay in dogs	NS	NS	*S. miescheriana*	**1873**
Germany	1977	1000	Trichinoscopy	419	41.9	NS	**810**
Germany	1975–77	1175	Trypsin digestion	219	18.6 9.7 (fattening) 35.5 (breeders)	*S. suihominis* (60.7%) "*S. suicanis*" (48.3%) Mixed (n = 11 cases)	**115**
Germany	NS	NS	NS	NS	3.0–30.0	*S. suihominis*	**156**
Germany	1997	2041 sows	ELISA	592	29.0	*Sarcocystis* spp.	**291**
Ghana	1997	60	Trichinoscopy	17	28.3	NS	**1387**
India, Haldwani, Rudrapur and Aligarh	NS	890	Pepsin digestion	605	67.9	*S. suihominis* (43.14%) *S. miescheriana* (47.11%)	**1526**
India, Haryana	1981–82	157	Pepsin digestion, trichinoscopy, bioassay in dog and cat	108	68.8	*S. miescheriana* after successful bioassay in dog	**761**
India	NS	37	Histology	2 (cysts in brain)	5.4	NS	**759**
India, Bihar	1968–80	170	Pepsin digestion	91	53.5	NS	**1508**
India, Bombay	1966	125	Histology	23	18.4	*S. miescheriana*	**315**

(*Continued*)

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Table 6.2 (*Continued*) Surveys of *Sarcocystis* spp. in Domestic Pigs

Country	Year	No.	Method	No. Positive	% Positive	Species	Reference
India, Assam	1991–92	372	Pepsin digestion	283	76.1 25.0 (<1 year) 79.6 (>1 year)	*S. suihominis (56.06%)* *S. miescheriana (34.85%)*	**319**
India, Punjab	NS	229	Pepsin digestion	168	73.3 33.3 (<1 year) 83.9 (>1 year)	*S. suihominis* (53.6%) *S. miescheriana* (31.8%) Mixed infections (14.5%)	**50**
India, Uttar Pradesh	NS	NS	Pepsin digestion	NS	NS	*S. miescheriana*-like	**18**
India, Madhya Pradesh	1987–88	200	Trypsin digestion	168	84.0	*S. suihominis* (49.5%) *S. miescheriana* (34.5%) Mixed (4.5%)	**1638**
India, Uttar Pradesh	NS	296	Trypsin digestion, microscopic examination	228 1	77.0	*S. suihominis* (48.9%) *S. miescheriana* (40.2%), *S. porcifelis*	**790, 791**
Italy, Reggio Emiglia	NS	272 fattening	Pepsin digestion	15	5.5	NS	**143**
Italy, Sardegna	NS	328	Pepsin digestion	271	82.6	NS	**37**
Japan, Saitama	1996–97	600 breeding	Trichinoscopy	5	0.83	*S. suihominis*	**1520**
Japan, Hokkaido	1992	144	Trichinoscopy	17	0.0 (fattening) 16.5 (sows)	"*S. suicanis*"	**1334**
Japan, Okinawa	1991–92	300	Pepsin digestion, pathology	107	35.7 13.0 (fattening) 47.0 (breeding)	*S. porcifelis* (1 case) *S. miescheriana* (106 cases)	**1323**
Japan, Saitama	Unknown	300	Trypsin digestion, pathology	17	0.0 (fattening) 8.5 (older)	*S. miescheriana*	**1510**
Philippines	1994	225	Histology	1	0.4	Unidentified, immature cysts	**219**
Philippines	NS	33 (2–5 years)	Histology	9	27.0	*S. miescheriana*	**224**
Poland	1967–77	NS	Trypsin digestion	NS	Incidence 0.328 in 1976 0.363 in 1977	NS	**317**
Thailand, Bangkok	NS	839	Trypsin digestion	99	11.8	NS	**1234**
Serbia	NS	788 (6 months)	Trypsin digestion	193	24.5	NS	**1388**

(*Continued*)

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Table 6.2 (*Continued*) Surveys of *Sarcocystis* spp. in Domestic Pigs

Country	Year	No.	Method	No. Positive	% Positive	Species	Reference
Slovak Republic	Unknown	1409	Pepsin digestion, trichinoscopy	13	0.9	NS	**1101**
Spain, Northwest	NS	100	Pepsin digestion, trichinoscopy	43	43.0	NS	**1384**
Spain, Santiago de Compostela	NS	100	Pepsin digestion, ELISA, HAI	5	5.0	NS	**1385**
Spain, Zaragoza	1978–80	484	Pepsin digestion, trichinoscopy	178–484	36.7–100	NS	**1530**
Switzerland	NS	1 two-year-old boar	Histology, PCR	1 isolate	–	*S. miescheriana*	**168**
Thailand	NS	295	Pepsin digestion	35	11.8	NS	**1234**
Uruguay		269 (90–140 kg)	Pepsin digestion, pathology	154	57.2	"*S. suicanis*"	**635**
USA, Georgia	NS	168 sows	Pepsin digestion, bioassay in dog, cat, raccoon and opossum	28	16.6	"*S. suicanis*"	**1424**
USA, Iowa	1990–92	893 sows	Pepsin digestion, pathology	163	18.2	*S. miescheriana*	**409**
USA, Ohio	1977	236 sows	Trypsin digestion, bioassay in dogs and cats	8	3.4	No species with dog and cats as definitive host	**353**
USA, Maryland	NS	39	Pepsin digestion	17	43.6	NS	**878**
USA, Michigan	1973	103	Pepsin digestion, pathology	7	6.8 0.0 (<1 year) 12.7 (>1 year)	NS	**1586**

Note: *S. suicanis* is now called *S. miescheriana*.

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Table 6.3 Surveys of *Sarcocystis* spp. in Wild Swine

Country	Year	No.	Method	No. Positive	% Positive	Species	Reference
Bulgaria	1972–75	NS	Trichinoscopy	NS	66.7	NS	**1187**
Germany	NS	200	Trichinoscopy	140	70.0	NS	**335**
Germany	NS	103	Trichinoscopy	46	45.0	NS	**546**
Iran	2007	–	Pathology, PCR	1 isolate	–	*S. miescheriana*	**956**
Lithuania	2005–11	51	Trichinoscopy	45	88.2	*S. miescheriana*	**1414**
Lithuania	1981–86	715	? (In Lithuanian)	353	49.4	"*S. suicanis*" *S. suihominis*	**33**
Lithuania	1998–2001	55	Histology	49	89.1	NS	**1102**
New Zealand	NS	50	Pepsin digestion, pathology	5	10.0	NS	**238**
Poland	NS	30	Trichinoscopy	20	66.0	NS	**546**
Poland	NS	166	Trichinoscopy		24.7	–	**1763**
Romania	1995	NS	Trichinoscopy, bioassay in cats	NS	NS	*S. porcifelis*	**1694**
Slovak Republic	2005–07	20	Trypsin digestion, Histology	17	85.0 (0.5–2 years)	NS	**869**
Slovak Republic	2006–09	30	Trypsin digestion	25	83.3	NS	**869**
USA, Southeast	NS	192	Pepsin digestion	62	32.0 40.0 (adults) 10.0 (immature)	*S. suicanis*	**73**
USA, Texas	1973–74	5 (6 mo–4 years)	Histology	3	60.0	NS	**250**
USA, 29 states	2012–14	1006	Histology, pepsin digestion, PCR	251	25.0%	*S. miescheriana*	**161**

Note: *S. suicanis* is now called *S. miescheriana*.

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Several food-safety institutions (e.g., European Food Safety Authority) encourage monitoring and characterization of zoonotic *Sarcocystis* species in animals and foodstuffs.[1727] Although carcass condemnation for *Sarcocystis* has been reported, there is no documentation of any species of *Sarcocystis* in swine, with a macroscopic sarcocyst, so it is unclear if sarcocysts are actually present or whether there has been misidentification. Greve[735] reported association between *Sarcocystis* infection and lymphadenopathy in the pelvic cavity during inspections in Danish abattoirs; it has been suggested that calcified sarcocysts are found only in domestic pigs and not in wild boars.[335] The basis for documentation is uncertain.

Only 1 report was found of clinical sarcocystosis in a naturally infected pig. Clinical fatal sarcocystosis attributed to *S. miescheriana* was reported in a pig from Switzerland.[168] The affected pig was a 2-year-old boar that had fever, anorexia, and died after a short period of illness. Histopathological examination revealed severe myocarditis associated with *S. miescheriana*-like schizonts; sarcocysts were not seen. Molecular characterization of DNA extracted from paraffin embedded myocardium indicated *S. miescheriana* infection.[168]

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Information pertinent to the subject matter of this chapter may be found in References: **75, 74, 79, 97, 119, 120, 296–298, 317, 349, 350, 353, 546, 547, 549, 550, 567, 583, 707, 716, 734–736, 806, 808–810, 812, 814, 819, 960, 1057, 1174, 1177, 1188, 1244, 1286, 1287, 1289, 1383, 1478, 1485, 1488, 1559, 1560, 1688, 1727, 1813, 1826, 1827, 1880, 1881, 1892, 1911, 1914, 1918, 1982, 2011, 2069, 2109, 2149, 2259, 2274, 2276–2278, 2290, 2320, 2323, 2363**.

Prevalence: **18, 33, 37, 40, 49, 50, 73, 115, 143, 156, 168, 219, 224, 238, 250, 291, 315, 317, 319, 335, 353, 409, 546, 635, 721, 735, 759, 761, 764, 790, 791, 810, 832, 869, 878, 956, 1086, 1101, 1102, 1187, 1234, 1244, 1323, 1334, 1384, 1385, 1387, 1388, 1414, 1424, 1508, 1510, 1520, 1526, 1530, 1586, 1638, 1694, 1720, 1763, 1873, 1916, 1930, 1938, 1953, 1967, 1982, 2037, 2038, 2054, 2075, 2091, 2124, 2231, 2242, 2297, 2311, 2325.**

Experimental: **74, 75, 120, 297, 298, 299–303, 322, 806, 954, 989, 1052, 1053, 1057, 1168, 1413, 1425, 1426, 1457, 1458, 1459, 1637, 1640, 1641, 1900, 1906, 1925, 1928, 2001, 2021, 2046, 2052, 2067, 2084, 2087, 2129, 2189, 2232, 2275, 2374.**

Morphology and pathology: **51, 52, 209, 707, 814, 809, 989, 1174, 2152, 2216.**

Molecular: **161, 719, 956, 1854**.

Miscellaneous: **209, 567, 1892, 1906, 1911**.

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CHAPTER 7

Sarcocystosis in Cattle (*Bos taurus*)

There are 4 species of *Sarcocystis* in cattle: *S. cruzi*, *S. hirsuta*, and *S. hominis* (Figure 7.1), with canids, felids, and primates, respectively, as definitive hosts; and a fourth species, *S. rommeli*, with unknown life cycle.[471]

7.1 *SARCOCYSTIS CRUZI* (HASSELMANN 1926) WENYON 1926

7.1.1 Intermediate Hosts

Cattle (*Bos taurus*), North American bison (*Bison bison*),[361,162] European bison (*Bison bonasus*),[608] and banteng (*Bos javaticus*).[1307]

7.1.2 Definitive Hosts

Dog (*Canis familiaris*), coyote (*Canis latrans*), red fox (*Vulpes vulpes*), crab-eating fox (*Cerdocyon thous*),[1475] raccoon dog (*Procyon lotor*, *Nyctereutes procyonoides*),[1512] and wolf (*Canis lupus*).

7.1.3 Structure and Life Cycle

Sarcocysts are microscopic, <1000 µm long, and are formed in virtually all striated muscles, Purkinje fibers of the heart, and in the CNS.[360] The sarcocyst wall is thin (<1 µm) and its surface is covered with long, narrow, ribbon-like vp (Figures 7.1a and 7.2a).[127,360,1171,1348] The vp are up to 3.5 µm long and up to 0.3 µm wide at the base.[638] They taper towards the tip, which is blunt. There are no supporting tubules or microtubules in the villar core; therefore, the vp are often folded over the surface (Figure 7.2a), type 7a. The sarcocyst wall surface has numerous small invaginations, which are hexagonal when viewed from the surface. The primary cyst wall shows small invaginations (contributing to a dentate appearance), and zipper-like structures.[638]

The life cycle that was used as an example in Chapter 1, is summarized in Table 7.1.

7.1.4 Pathogenicity

S. cruzi is the most pathogenic species found in cattle.[366,572,577,918,1269] It can cause fever, anorexia, anemia, weight loss, hair loss, weakness, muscle twitching, prostration, abortion, reduced milk yield, hypersalivation, neurologic signs, and death, depending on the isolate and the number of sporocysts ingested.[63,167,232,681] Ingestion of 1000 or fewer sporocysts under experimental conditions caused little or no clinical signs. Doses of 5 million or more are uniformly fatal. Cattle fed 200,000 sporocysts became clinically ill, some died of acute sarcocystosis, and some survivors did not grow to their full potential.[591] In experimental infections, cattle had no clinical signs until the fourth week, irrespective

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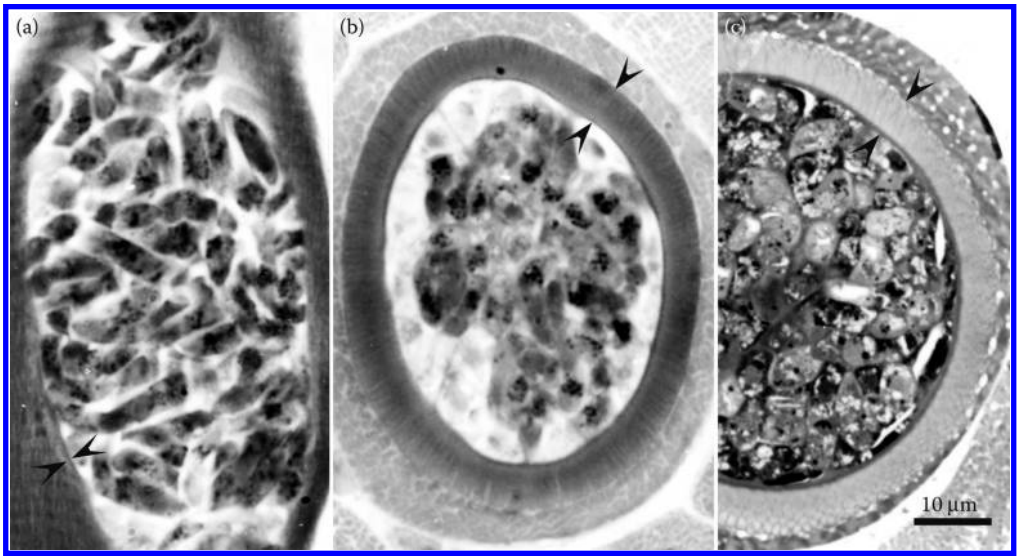


Figure 7.1 Sarcocysts of *S. cruzi* (a), *S. hirsuta* (b), and *S. hominis* (c) in skeletal muscles of cattle. Note outer and inner layers of sarcocyst walls (arrowheads). (a and c), Toluidene blue stain and (b), PAS reaction.



Figure 7.2 TEM of sarcocyst walls of *S. cruzi* (a) and *S. hominis* (b) in experimentally infected calves. (a) Filamentous villar protrusions, without microtubules, folded over the primary sarcocyst wall. (b) Finger-like vp with microtubules.

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Table 7.1 Comparison of Developmental Stages of *S. cruzi*, *S. hirsuta*, and *S. hominis* in Cattle

	S. cruzi	*S. hirsuta*	*S. hominis*
	First-Generation Schizonts		Unknown
Location	Arterioles of several organs	Mesenteric and intestinal arterioles	—
Duration (DPI)	7–26	7–23	—
Peak development (DPI)	15	15	—
Size of meronts (μm)	41.0 × 17.5	37.2 × 22.3	—
No. of merozoites	>100	>100	—
Size of merozoites (μm)	6.3 × 1.5	5.1 × 1.2	—
	Second-Generation Schizonts		Unknown
Location	Capillaries of several organs	Capillaries of striated muscles, heart	—
Duration (DPI)	19–46	15–23	—
Peak development (DPI)	24–28	16	—
Size of meronts (μm)	19.6 × 11.0	13.9 × 6.5	—
No. of merozoites	4–37	3–35	—
Size of merozoites (μm)	7.9 × 1.5	4 × 1.5	—
	Parasitemia		Unknown
Duration (DPI)	24–46	11	—
Intraleukocytic			—
Multiplication	Yes	No	—
	Sarcocysts		—
First seen (DPI)	45	30	—
Maturation time (DPI)	86	75	—
Size	183–417 × 20–98 μm	>8 mm × 1 mm	0.7–2.6 mm × 50–150 μm
Wall (μm)	Thin (<1.0), type 6/7	Thick (3–7), type 28	Thick (>6), type 10/15/16
Villar protrusions	Ribbon-like, wide at the base. Without microtubules, folded over the surface, >3.5 μm × 0.3 μm	Narrow stalk, expanded laterally and tapered distally, 8 μm long, bent at 45–90°	Finger-like, 0.7–3.0 × 6.8–8.1 μm
Metrocytes	Globular, 4.5 × 9.6 μm	Globular, 3 × 5 μm	Globular or oval, 4–5.5 × 8–9 μm
Bradyzoites	Crescentic	Banana shape	Banana shape, 2.5 × 7–9.0 μm
Location	Mostly in heart, high frequency in skeletal muscle, also found in CNS	Not in heart or CNS	Not in heart
Main references	**225,360**	**126,127,360,395**	**391,395,884,1840**

of dose. Beginning as early as 24 DPI they developed persistent fever, anorexia, weight loss, weakness, muscle twitching, prostration, and some died. Pregnant cows aborted,[844,1152] and milking cows had reduced yield and poor quality milk. Cattle that did not recover lapsed into chronic sarcocystosis, in which there was continued inappetance, weight loss, hypersalivation, hyperexcitability, and loss of hair, especially on the neck, rump, and tail switch (Figure 1.84). Some months after infection, a few animals developed signs of CNS involvement, including nystagmus, opisthotonus, and lateral recumbency with a running gait. All cattle that developed CNS disorders eventually died.

Gross and microscopic lesions were described in Chapter 1.

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7.1.5 Natural Outbreaks

In 1961, a febrile illness was noticed in a dairy herd.[256] Within 8 weeks, a total of 25 animals were affected and 17 died. Acutely ill cows had intermittent anorexia, drop in milk yield, diarrhea, transient fever, nasal discharge, hypersalivation, and hemorrhagic vaginitis. Of 17 pregnant cows, 10 aborted in the last trimester of pregnancy and only 1 of the 10 survived. Erosions were found on the tongue and buccal mucosa of the salivating cows. Chronic cases were marked by emaciation, pale or icteric mucous membranes, mandibular edema, exophthalmos, cessation of lactation, and sloughing of the tail switch. Cachectic animals became recumbent, with muscle tremors over the body resembling those of hypocalcemia. Gross and histologic lesions resembled those described for acute experimental sarcocystosis. Numerous schizonts were present in the endothelium of many organs in 11 of 16 animals. Because neither the stage nor clinical signs of sarcocystosis had been described, this outbreak was called Dalmeny disease, after the small Canadian town in which it occurred. In retrospect, however, it was an outbreak of acute sarcocystosis.[256]

Subsequently, cases of natural clinical sarcocystosis in cattle have been reported in Canada,[1161] England,[227,604,1016] Ireland,[232] Norway,[1019] Australia,[167] and the United States.[626,627,681,1238] Unlike Dalmeny disease, these were infections in yearlings or younger calves. A well-documented outbreak was reported in Kentucky, United States.[681] Approximately 30 of 41 heifers in a 1-hectare lot had diarrhea, were losing weight, and consuming less feed, and had rough coats. The condition worsened to include severe laminitis, hypersalivation, extreme nervousness, and marked hair loss from the ears, lower limbs, and the tail switch, giving a "rat tail" appearance (Figure 1.84). Within a month, more than half the animals were severely debilitated and eight died. In the next 5 weeks, two-thirds died or were euthanatized, only a third were salvaged for the market.

Macroscopic erosions were found in several tissues. The skin of the carpus and tarsus, just above the coronary band, was denuded and abraded; interdigital erosions were present, and the hoof wall was cracked, necrotic, and about to separate from the coronary band; the cornea had erosions and opacities; erosions and shallow ulcers were found in the muzzle, lips, tongue, hard palate, esophageal mucosa, and omasum; the lungs were congested. Histologic examination revealed intense mononuclear cell infiltrates in the muscles and muscular organs, with accompanying myodegeneration and numerous immature sarcocysts (Figure 1.96). The *Sarcocystis* IHA antibody titers in affected heifers were fourfold greater than in unaffected cows on the same farm.[681] Hay contaminated with feces from farm dogs was implicated as the source of infection.[626,681]

The association between sarcocystosis and ulcerations in the mouth and foot, seen in naturally infected cattle, has not been explained. Neither buccal nor pedal lesions were seen in over 100 experimentally inoculated cattle. Naturally infected cattle may be more susceptible to secondary infections.

Sporadic cases of bovine abortion and neonatal mortality have been reported in New Zealand,[1246,1741,1791] Australia,[908,1152,1242,1246] Canada,[256] the United States,[30,365,844,988,1371,1558] and Cuba,[251] in which fetal lesions and/or protozoa were found. Fetal encephalitis, myocarditis, and hepatitis were often associated with the outbreaks. Intravascular schizonts resembling those of *Sarcocystis* were found in some of the fetuses in all of the reported outbreaks. Apart from focal placentitis and hypersalivation in a few cows, no clinical abnormalities were identified.

An unusual necrotizing encephalitis was found in 2 cattle from South Africa.[1774] A 2-year-old heifer developed convulsions and died suddenly. Eleven of 15 young calves developed severe clinical signs over 5 days, and 1 calf died of neural signs. Both of these animals were necropsied. Multiple areas of necrosis and vasculitis were found in both animals. Schizonts, considered to be first generations, were identified in blood vessels in affected areas.

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7.1.6 Protective Immunity

Cattle are susceptible to clinical sarcocystosis from birth and throughout their life, despite repeated natural infections. However, it has been possible to establish a protective immunity under experimental conditions.[590] Calves infected with 50,000 or 100,000 *S. cruzi* sporocysts were protected from illness and death that would have resulted from challenge infections with large numbers of *S. cruzi* sporocysts given 70–252 days later. Calves treated prophylactically with amprolium from 21 to 35 days after infection to reduce second-generation schizonts were also protected.[590] Infection with the nonpathogenic *S. hirsuta* did not protect calves against *S. cruzi* infection.

7.1.7 Molecular Studies and Genetic Diversity

Several studies were focused on molecular diagnostic and characterization of *S. cruzi* infecting cattle. Initial genomic probes[957] were later applied[1272] for the diagnosis of acute sarcocystiosis in experimentally infected cattle. An RAPD assay was developed for specific diagnosis of *S. cruzi* infections.[1095]

Methodologies focused on the amplification of 18S rRNA, 28S rRNA, ITS-1, rRNA and *cox*1 genes allow differentiation of species.[608,1218,1477,1493,1856] Epidemiological surveys based on molecular identification of *Sarcocystis* species are accumulating; most are compiled in Table 7.2.

The most comprehensive study on genetic variability of *S. cruzi* found a very low/restricted genetic diversity in isolates from the United States and Uruguay, and also minimal differences with sequences available in international databases, belonging to isolates from remote locations like Australia, China, or Germany.[1493] These findings suggested that the dispersion of parasites of veterinary importance is closely related to human movements; in this sense, by domesticating dogs and cattle, humans may have profoundly influenced the ecology and evolution of *S. cruzi*.

7.2 *SARCOCYSTIS HIRSUTA* MOULÉ 1888

7.2.1 Definitive Host

Cat (*Felis catus*).

7.2.2 Structure and Life Cycle

Sarcocysts as large as 8 mm long and 1 mm wide (Figures 7.1b and 7.3) have been found in naturally infected cattle,[126] and others as large as 800 µm long and 80 µm wide have been found in experimentally infected cattle.[362] Sarcocyst walls as thick as 7 µm have been seen, some radially striated, others hirsute.[362,673] The vp are up to 7.0 µm long and 1.5 µm wide, and have been seen to contain numerous microtubules.[127,128] They can be closely packed (0.03 µm apart) or widely spaced (1 µm) (Figure 7.4), type 28. By SEM, wart-like structures were on the vp.[126]

Information on the life cycle and structure is summarized in Table 7.1. There are 2 known generations of schizogony. First-generation schizonts have been found only in small arteries of the intestine and mesentery, and second-generation schizonts have been found only in capillaries of the heart and skeletal muscles.

7.2.3 Pathogenicity

S. hirsuta is mildly pathogenic.[371] Calves fed 100,000 or more sporocysts became febrile, had diarrhea, and were mildly anemic; none died. At necropsy, hemorrhagic lesions were not seen.[371]

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Table 7.2 Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Argentina	2004	Abattoir	H, D, E	90	90 (100.0%) IFAT	100.0% dissection	—	93.3% histology	*S. cruzi*	—	**1213**
Argentina, Buenos Aires	2006	Farm	Serum	172 calves at birth and their mothers	IFAT: 3 calves (1.7%) before ingestion of colostrum; 172 cows (100.0%)	—	—	—	*S. cruzi*	Vertical transmission: 1.7%	**1215**
Argentina	2005	2 farms	Blood	112	IFAT: close to 100.0% in both farms	—	—	—	*S. cruzi* antigen used	Increment of titers in calves with time	**1216**
Argentina	NS	Abattoir	H (10 g), loin (10 g)	380	378 (99.5% in heart)	Muscle squash	135 (35.5%)	LM, TEM	99.5% of thin walled (*S. cruzi*), 32.1% of thick-walled (*S. hominis*, *S. hirsuta*, *S.* sp.)	IFAT antibodies in 99.7% (379/380)	**1217**
Australia	1989–90	Abattoir	E	714	371	52.0%	—	—	—	—	**1542**
Australia	1989	Abattoir	E, D, masseter	202	125 (62%)	Pepsin digestion	—	LM, histology	1 thick-walled, *S. cruzi* in all infected cattle	D: 53% E: 57% Masseter: 52%	**1541**
Belgium, Gent	1987	Abattoir	H, E, D	100	97 (97.0%)	Pepsin digestion: 97% E, H: 89, D: 75	—	LM, histology	Thick-walled: 56%, Thin-walled (*S. cruzi*) in all	—	**1787**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Belgium	2006	Retail stores	Mince loin	67	63 (94.0%)	—	63 positives	—	*S. cruzi*: 49%; thick wall: 91%, of those, 97.4% was *S. hominis*	—	**1776**
Belgium	1994–2007	Abattoir	Carcasses	97 carcasses affected with eosinophilic myositis	—	—	PCR and sequencing	LM	Extralesional: 80% of *S. hominis* and 20% unknown *S.* sp. Intralesional: 81.8% *S. hominis*, 4.5% *S. cruzi*, and 9% each *S. hirsuta* and unknown *S.* sp.	—	**1778**
Brazil	NS	NS	H (50 g)	50	50 (100.0%)	Homogenization in saline	—	LM	*S. cruzi* (but no morphologic study was done)	—	**275**
Cambodia	1994–95	Abattoir	Not clear (in Vietnamese)	Not clear	36.4%	Not clear	Not clear	Not clear	Not clear	Not clear	**857**
Czech Republic, Bohemia		NS	E	200	174 (87%)	Trypsin (87.0%)	—	Gross examination (29.0%), trichinoscopy (62.5%)	*S. cruzi*, *S. hominis*, *S. hirsuta*	40% single infections, 42% two species, and 18% three species	**764**
Czech Republic, Bohemia	NS	Abattoir	E	208	167 (80.3%)	Trichinoscopy	—	LM, H and E	*S. cruzi* (40.0%), *S. hominis* (5.7%), *S. hirsuta* (19.2%), mixed (4 cases)	—	**332**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Egypt	1989–90	Abattoir	H, D, T	10	4 (40.0%)	—	—	Histology	NS	—	**564**
Egypt, Assiut	NS	Abattoir	H, E, D, ocular muscle	100	94 (94.0%)	Muscle squash	—	LM	*S. cruzi*, by morphology and the success in bioassay in dog; cats did not shed sporocysts. Macroscopic cysts (4.0%) are suspected to be *S. hirsuta*	By ELISA using *S. fusiformis* antigen: 98.0%.	**1551**
Egypt, Sharkia	NS	Abattoir	E	81	24 (29.6%)	Gross, muscle squash	RAPD-PCR	LM, TEM	Only microcysts: thin (*S. cruzi*, 29.6%) and thick (*S. hominis*, 4.9%) walled	—	**54**
Estonia	NS	NS	NS	2892	1537 (53.1%)	NS	NS	NS	NS	NS	**1193**
Germany, South	1977–78	Abattoir	E, D, T	1020	1007 (99.7%)	Trypsin (99.7%)	—	Trichinoscopy (57.9%)	*S. cruzi* (65.6%), *S. hominis* (63.6%), *S. hirsuta* (34.5%)	—	**116**
Germany	NS	Retail stores	100 g beef	257	174 (67.7%)	—	67.7% by PCR, 69.6% multiplex	40% (52/130) by LM	28.5% thin walled (*S. cruzi*) and 24.6% thick walled (*S. hominis*, *S. hirsuta*, *S.* sp.)	Mixed infection: 28%	**1221**
Hungary	2014	Abattoir	E, H	151	100 (66.0%)	—	PCR	—	*S. cruzi* (64%), *S. hominis* (19%), *S.* sp., *S. rommeli* (17%)	—	**1143**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
India	NS	Abattoir	E, T, D, H, ocular muscle, M	238	191 (80.25%)	Trypsin digestion	—	LM	*S. cruzi*, *S. hominis*, *S. hirsuta*	—	**884**
India	NS	Animals submitted previously to experimental infection with *Clostridium chauvoei*	Brain	30	12 (40.0%)	—	—	Histology	NS	NS	**1616**
India	1994–95	Abattoir	H	200	98 (49.0%)	—	—	Gross, histology	*S. cruzi*, *S. hominis*	NS	**323**
India, Bangalore	NS	NS	E, H, D, T	100	84 (84.0%)	Pepsin digestion	—	LM, Histology	*S. cruzi* (78.5%), *S. hominis* (21.4%), *S. hirsuta* (34.5%)	E: 84.5%, H: 79.7%	**277**
Iran, Kerman	2005–06	Abattoir	H, D, T, M	480	480 (100%)	Pepsin digestion of 10 g	—	—	NS	No macroscopic cysts	**1283**
Iran, South West	2009	Abattoir	H, D, T, M	344	480 (100%)	Pepsin digestion of 50 g: 100%, Muscle squash: 94.7%	—	—	NS	The most frequently infected tissue was heart: 93.3%	**773**
Iran, Isfahan	2008	Abattoir	E, D	100	92 (92.0%)	Muscle squash	—	Gross inspection, LM, TEM	*S. cruzi* in 89%, *S. hominis* or *S. hirsuta* (identified as thick-walled cysts) in 21%	—	**1282**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Iran, Babol	NS	Retail stores	5 g muscle	1 isolate	—	—	PCR, sequencing	LM, histology	*S. cruzi*	—	**928**
Iran	NS	Abattoir	Intercostalis, D	1 cow	—	—	—	Gross inspection, LM, TEM	3–5 mm length cysts of *S. hirsuta*	First report in Iran	**1601**
Iran, North Khorasan	2013	Abattoir	H, D, meat, liver	32	32 (100.0%)	Pepsin digestion	–	–	NS	Bradyzoites were found in 100% of 32 livers	**1524**
Iran, Kurdistan	NS	Abattoir	E	130	56 (43.1%)	Muscle squash	—	LM	—	—	**1449**
Italy, Milan	NS	Abattoir	H, masseter	154	91 (59.1%)	—	—	LM, histology	*S. cruzi*, *S. hominis*, *S. hirsuta*	–	**1553**
Italy	2008–09	Abattoir	22 different muscles	50	48 (96%)	—	—	LM, histology	Thin-walled, *S. cruzi*	—	**150**
Italy	2008	Abattoir	H, E, D	384	300 (78.1%) by histology	—	PCR	LE, TEM	By PCR: *S. cruzi* (74.2%), *S. hirsuta* (1.8%), *S. hominis* (42.7%), *S. hominis*-like (18.5) present divergence 0.7%–1.4% with *S. hominis*	E: 11.7%, E + H + D: 62.0%, E + H: 10.0%, E + D: 9.0%, D: 3.7%, H: 2.3%, H + D: 1.3%	**331**
Japan	Not clear (in Japanese)	Not clear (in Japanese)	H, D	105	44 (41.9%)	—	—	Histology Heart: 97.7%, Diaphragm: 18.2%	*S. cruzi*	Bioassay in dog and cat; only sporocysts were shed by dogs	**1223**

(*Continued*)

Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Japan, Niigata	1989	Abattoir	H, T, masseter	25	25 (100.0%)	Trypsin digestion	—	LM, SEM	*S. cruzi*	H: 100.0%, T: 4.0%, masseter: 8.0%	**1852**
Japan, Aichi	1989	Abattoir	H, masseter	30	17 (56.7%)	Trypsin digestion, squeezing	—	LM, histology	*S. cruzi* (94.0%); *S. hirsuta* (23.6%)	H: 56.7%; Masseter: 53.3%	**872**
Japan, Hokkaido	1993	Abattoir	H	83 autochthonous, 185 imported	13 (15.7%) in autochthonous, and 92 (49.7%) in imported	Muscle squash	—	LM	*S. cruzi*	Bioassay in dogs	**1335**
Japan	1996–98	Abattoir, retail stores	Loin	482	72 (14.9%)	—	—	LM, histology	*S. cruzi*, only one case presented thick wall	Origin of cattle: Japan (6.3%), America (36.8%), Australia (29.5%)	**1337**
Japan, Saitama	NS	Abattoir	D	5	4 (80.0%)	—	—	LM, TM, SEM	*S. hominis* (other species not specified)	Bioassay in two cynomolgus monkeys: shed sporocysts 10 DPI	**1521**
Japan, Saitama	1999–00	Abattoir	D (100 g)	840 (3–10 yr old)	2 (0.23%)	—	—	LM, SEM, TEM	Thick-walled (7–10 μm) but not recognized species. Possibly, *S. hirsuta*	—	**1522**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Lithuania	Not clear (in Lithuanian)	Abattoir	H, D, chest muscle	NS	Not clear (100.0%)	NS	—	NS	*S. cruzi* (100% hearts, 34.6% diaphragm, 12.1% chest muscle), *S. hominis* (0% hearts, 65.4% diaphragm, 87.9% chest muscle), *S. hirsuta* (1 case)	—	**738**
Malaysia	2011–12	Abattoir	E, H, T, M, D	102	37 (36.2%)	Squeezing: 37 (36.2%)	7 cases sequenced	Histology	*S. cruzi*	Skeletal muscle and diaphragm (27%), tongue and esophagus (24.3%), and heart (8%)	**1029**
Mexico, Mexico DF	NS	Retail meat store	H	100	94 (94.0%)	—	—	LM	NS	—	**1185**
Mongolia	1998–99	Abattoir	H, D, T	30	27 (90.0%)	Squeezing	—	—	NS	H (100%), D (61.1%), T (90.0%)	**640**
New Zealand	1983–84	Abattoir	E, D	500	498 (99.6%)	E: 97.4%, D: 51.8%	—	LM, TEM, SEM	*S. cruzi* in 98.0%, thick-walled cysts in 79.8%	Bioassay in cat and human volunteer; only cat shed sporocysts	**127**

(*Continued*)

Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
New Zealand	1985	Abattoir	E, D, T, H	100	64 (64.0%)	—	—	Gross	—	Macro sarcocysts in 64. No microcysts investigated	**1200**
Nigeria, Zaria	2011	Abattoir	E, D	200	85 (42.5%)	Pepsin digestion	—	LM	*S. cruzi* in 99.0%, *S. hominis* in 4.0%	E (88.2%), D (65.9%)	**1300**
Peru	NS	Abattoir	H, E	85	H: 76 (89.5%), E: 2 (2.4%)	Trichi-noscopy	—	LM	NS	—	**170**
Philippines, Manila	1994	Abattoir	E, H, M, D	608	99 (16.3%)	Muscle squash	—	LM	NS	—	**219**
Philippines, Manila	NS	Abattoir	E, H, muscle from limbs and cervix	370 (3–11 yr)	40 (10.8%)	Muscle squash	—	LM, TEM (unsuc-cessful)	*S. cruzi*, *S. hominis*	—	**221**
South Korea	1988	Abattoir	H	1442	419 (29.1%)	Trysin digestion	—	LM	Apparently *S. cruzi*	—	**896**
South Korea	1991	Abattoir	H	330	144 (43.6%)	Trysin digestion	—	LM	*S. cruzi*	Bioassay in dog and cat (300 g meat)	**1372**
Sudan	NS	Abattoir	E, H, M, D	180	176 (97.8%)	Pepsin digestion	—	–	NS	E: 56.7% H: 95,6% M: 64.4% D: 63.9%	**868**
Thailand	NS	Abattoir	E	328	326 (99.4%)	Pepsin digestion	—	Gross, histology	*S. cruzi*, *S. hominis*, *S. hirsuta*	Macroscopic: 2 (0.61%) Microscopic: 57 (17.38%)	**1233**

(*Continued*)

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Table 7.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Muscle of Cattle

Country	Year	Source	Tissue	No. Tested	Total Positive	Method			Species Identified	Remarks	Reference
						Digestion/ Squeezing	PCR	LM, SEM, TEM			
Turkey	1985–86	Abattoir	D	1546	1416 (91.5%)	Not clear (in Turkish)	—	Not clear (in Turkish)	*S. cruzi*, *S. hominis*, *S. hirsuta*	No macroscopic cysts	**1343**
Uruguay	NS	Abattoir	H, E, T	15	15 (100.0%)	—	—	Histology	*S. cruzi* 15 (100%) of 15, *S. hirsuta* 2 (13.3%) of 15	Bioassay in dog and cat	**634**
USA, Texas	NS	Retail meat store	D	4	4 (100%)	Pepsin digestion	—	—	NS	—	**132**
USA, Uruguay	2004–05	Retail meat store	Sirloin	110	60 (54.5%)	—	54.5%: 41 sequenced	LM: 44 (40.0%)	Only *S. cruzi*	—	**1427**
Venezuela	2008	Abattoir	H, myocardium of fetuses	35	34 cows (97%), 0 fetuses (0.0%)	Muscle squash, histology	—	LM	NS	Myocarditis and pericarditis was detected in fetuses; presence of schizonts was not reported	**1523**
Vietnam	2003	Abattoir	D, E, T, muscle of neck	101	64 (63.3%)	Muscle squash: 25.0%	RFLP–PCR: 63%	LM	*S. cruzi*, *S. hominis*, *S. hirsuta* *S.* sp (1 *S. sibirica*-like sarcocyst)	—	**905**

D = diaphragm; E = esophagus; H = heart; M = skeletal muscle; T = tongue; NS = not stated.

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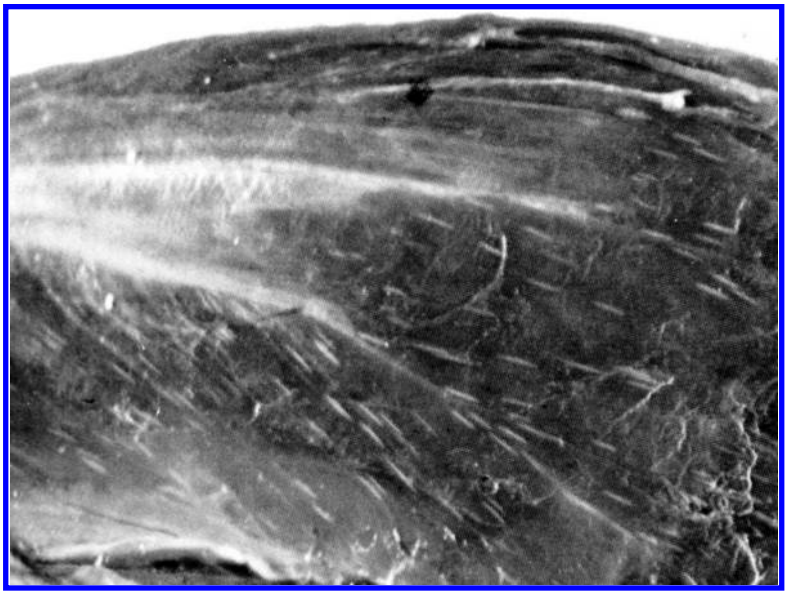


Figure 7.3 Macroscopic sarcocysts (arrow), probably *S. hirsuta*, in beef. (From Collins, G.H., 1980. *Sarcocystis and the Meat Industry*. Monograph, Massey University, New Zealand, 1–15. With permission.)

7.2.4 Other Aspects

There are numerous surveys in which *S. hirsuta* has been detected, in most of cases, as mixed infections (Table 7.2). Although this species does not represent a zoonotic risk, its frequent macroscopic character has resulted in economic loss by condemnation of carcasses during meat inspections.[397] *S. hirsuta* has been detected in meats and meat products destined for human comsumption.[771,1221,1382,1776]

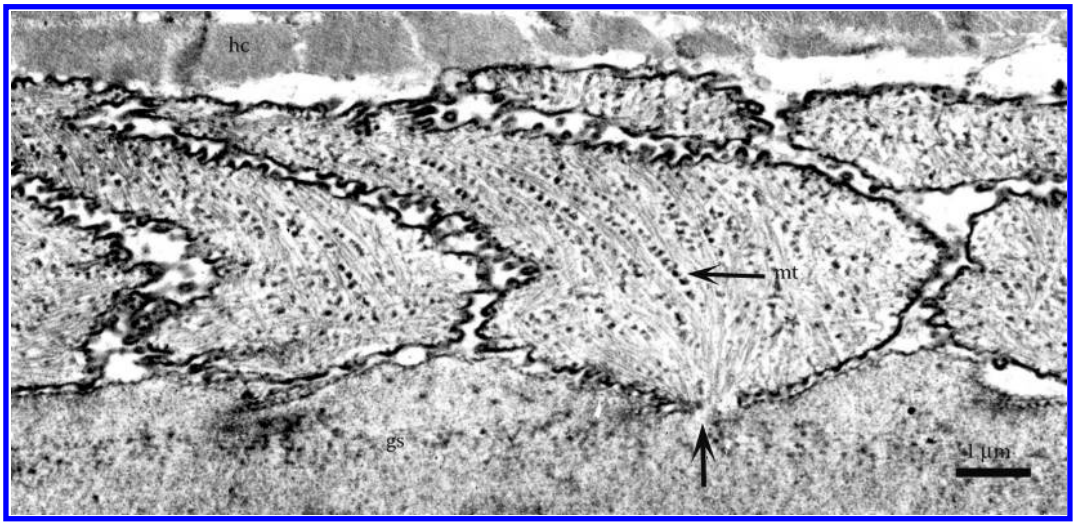


Figure 7.4 TEM of type 28 sarcocyst wall of *S. hirsuta*. Note conical VP, constricted at the base (arrow) and fine microtubules (mt).

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7.3 *SARCOCYSTIS HOMINIS* (RAILLIET AND LUCET 1891) DUBEY 1976

7.3.1 Distribution

Argentina, Belgium, Brazil, Canada, England, Germany, Italy, Japan, United States, and Vietnam.

7.3.2 Definitive Hosts

Humans (*Homo sapiens*), rhesus monkey (*Macaca mulatta*), cynomolgus monkey (*Macaca fascicularis*), baboon (*Papio cynocephalus*), and possibly chimpanzee (*Pan troglodytes*).

7.3.3 Structure and Life Cycle

The sarcocyst wall is up to 6 µm thick and appears radially striated because of numerous vp, which are up to 7 µm long and 0.7 µm wide, type 10b[1135] (Figures 7.1c, 7.2b, 7.5 and 7.6). The villar

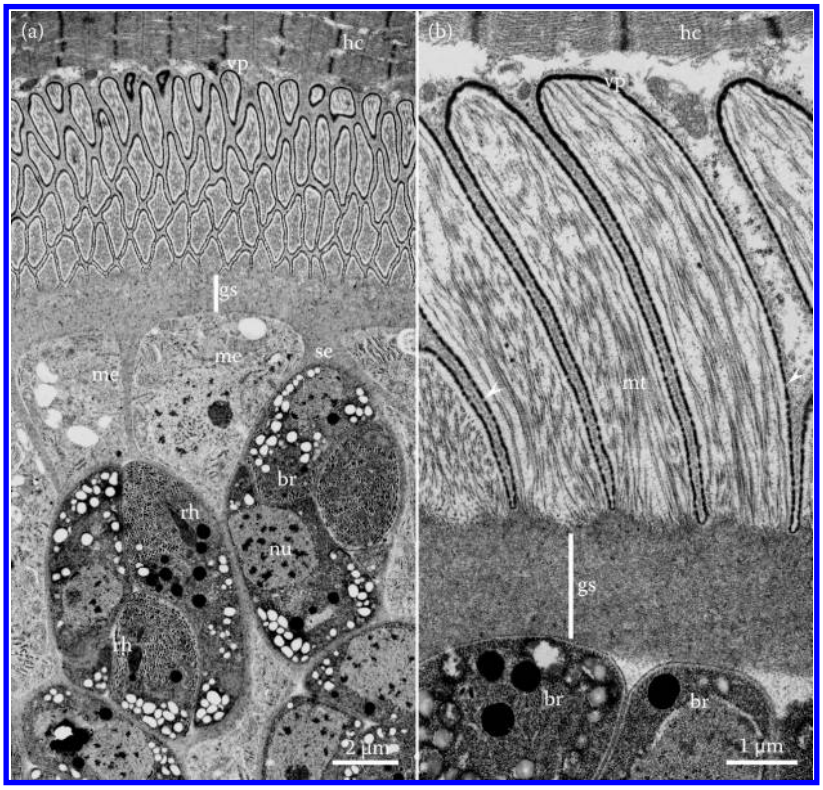


Figure 7.5 TEM of *S. hominis* sarcocyst in tongue of 2 calves after oral inoculation with sporocysts from the feces of a human volunteer who ate raw beef. Note elongated vp with thin microtubules mt. The parasitophorous vacuolar membrane is lined by electron dense layer that is thinned out at places (arrowheads). The gs is thicker at the point of origination of se. (a) 111 days p.i. and (b) 222 days p.i. The vp are of the same length in both sarcocysts but thinner at day 111 p.i. (Adapted from Dubey, J.P., Fayer, R., Speer, C.A., 1988. *J. Parasitol.* 74, 875–879.)

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Figure 7.6 TEM of *S. hominis* sarcocyst in tongue of a calf. The vp are cut at an angle. The pvm in vp is lined by electron dense layer that is thinned out at places (arrow). The mt are filamentous. The gs is thicker at the point of origination of se. Also note bradyzoites with a co, 2 rh, few dg, numerous am, a nu, an elongated mc, and leaflet–like structures (Lf) between bradyzoites.

core contains numerous microtubules that run from the base to the apex. Bradyzoites are 7–9 µm long and are arranged in packets (Figure 7.6).[1135]

S. hominis is only mildly pathogenic.[391] Calves inoculated with 1 million sporocysts develop mild anemia, but survive.[391]

A novel *S. hominis*-like parasite was detected in 18.5% of the cattle from northwestern Italy.[331] Hook-like structures were observed at the base and at the apex of the vp; further studies will be needed to confirm its identity.

7.4 *SARCOCYSTIS ROMMELI* DUBEY, MORÉ, VAN WILPE, CALERO-BERNAL, VERMA, SCHARES

This species was recently named[2392]; it was initially reported as *S. sinensis,* which is now regarded as *nomen nudum*.[211,471,701,1218,1221,1858]

7.4.1 Distribution

China, Vietnam, Germany, Hungary, Austria, Argentina.

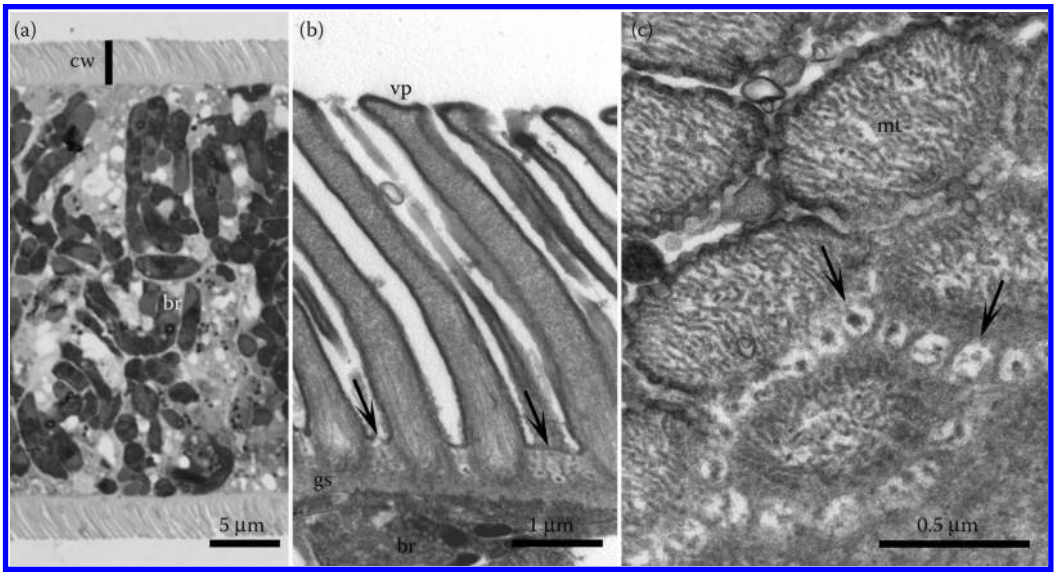


Figure 7.7 *Sarcocystis rommeli* from cattle. (a) Part of thick-walled sarcocyst in loin sample from Argentina. (b) TEM of the sarcocyst wall. Finger-like villar protrusions with microtubules bent at an angle. Note the thin ground substance (0.5 µm). Sarcocysts collected from a minced beef sample from Germany. (c) TEM image of the base of the villar protrusions. Note the presence of numerous microtubules (mt) and vesicles (arrowheads). Sarcocysts collected from a cattle loin sample from Argentina. Bar in (a) = 40 µm, (b) = 1.5 µm, (c) = 0.5 µm. (Courtesy of G. Moré.)

7.4.2 Definitive Host

Unknown, but not humans, dog, racoon dog, nor red fox.

7.4.3 Structure and Life Cycle

Sarcocysts microscopic, sarcocyst wall 4.5–5.2 µm thick. Vp 4–5 µm long, wavy, bent at an angle with mt extending from the tip to the middle of the gs, with vesicles at the base. The gs thin (<1 µm thick). Bradyzoites 10–12 µm long (Figure 7.7a–c).

7.4.4 Molecular studies

Based on 18S rRNA and cox1 gene sequences,[701,1143,1218,1221,2392] *S. rommeli* is molecularly different than *S. cruzi*, *S. hirsuta*, and *S. hominis*.

7.5 OTHER UNCONFIRMED SPECIES

Novak et al.[1284] described a species of *Sarcocystis* from cattle in Alma-Ata, former USSR. Sarcocysts were macroscopic, up to 20 mm long. By LM, cyst wall was up to 5 µm thick. By TEM, the PVM had mushroom-like protrusions. Odening[1317] named this parasite as *S. novaki*. In our opinion, this is not a valid species.

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7.6 PREVALENCE OF SARCOCYSTS IN CATTLE

Based on examination of tissues obtained at abattoirs, most cattle in the United States and throughout the world are infected with *Sarcocystis.* Recent surveys indicate that *S. cruzi* is the most prevalent species (Table 7.2), and this species is easily recognized in histologic sections, whereas *S. hirsuta* is difficult to distinguish from *S. hominis* microscopically. However, the sarcocyst walls of *S. hirsuta* and *S. hominis* can be differentiated ultrastructurally. The vp of the sarcocyst wall of *S. hirsuta* are approximately 8 µm long, constricted at their base, expanded laterally in the midregion, and tapered distally. The vp core contains numerous filaments and rows of electron-dense granules oriented parallel to the longitudinal axis of the protrusion. The sarcocyst wall appears only approximately 4 µm thick, because the distal region of each protrusion is folded over neighboring protrusions. The villar protrusions of *S. hominis* measure 5.7×0.6 and 7×1.4 µm at 111 and 222 DPI, respectively, are cylinder shaped and contain scattered granules and filaments that extend from the base to the tip of the projection.[391]

7.7 EOSINOPHILIC MYOSITIS

Eosinophilic myositis was discussed in Chapter 1 (Figures 1.5 and 1.6). After the acceptance that *Sarcocystis* spp. was the etiological agent of this process,[309,652,730,1472] several studies were focused on identifying which species of the genus were involved in the process. In this sense, while analyzing cattle carcasses affected by EM, Jensen et al.[907] detected, for the first time, *S. hominis* infecting cattle in the United States. Lesions were also associated with *S. cruzi* and *S. hirsuta*. Several studies report that *S. cruzi* is the main cause of EM,[526,654,725,1511] but *S. hominis* is the principal agent.[12,1778,1840] It is hypothesized that degeneration or rupture of the sarcocysts is the trigger for the development of EM.[652,1777]

7.8 SARCOCYSTOSIS-LIKE ENCEPHALITIS IN CATTLE

Isolated cases of fatal encephalitis were reported from Canada,[389] Germany,[1718] England,[751] and the United States (Dubey, unpublished). In both published instances, affected cattle developed neural signs before death. The case from Canada was in an 18-month-old steer. The steer became ataxic, recumbent, and blind. Grossly, a 1-cm-diameter, gray discolored area was seen in the cerebellum. Microscopically, the grossly visible lesion consisted of focal malacia, vasculitis, and infiltration of mononuclear cells, mainly macrophages (Figure 7.8). Numerous immature and mature schizonts and extracellular merozoites were seen; these stages resembled *S. cruzi* in structure. However, such lesions have never been seen in experimentally infected cattle. No clinical infection in the brain stem was reported by Singh and Parihar[1616] in 12 bullocks from India.

7.9 FOOD SAFETY

S. hominis is a well-known health risk for humans[210,807,1382]; the European Food Safety Authority is concerned about the risk associated with the presence of *Sarcocystis* sarcocysts in beef and stimulates efforts on its control.[1727] Generally, carcass condemnation by meat inspection services is based on gross examination and is associated with nonzoonotic species *S. hirsuta.*[397] This effort is not enough, and complementary assays based on microscopical examination[150,1427] are needed; related to this, *S. hominis*-infected meat and meat products had been detected in high percentages in Belgium, Brazil, Italy, Germany, and Japan.[331,675,1221,1337,1382,1776]

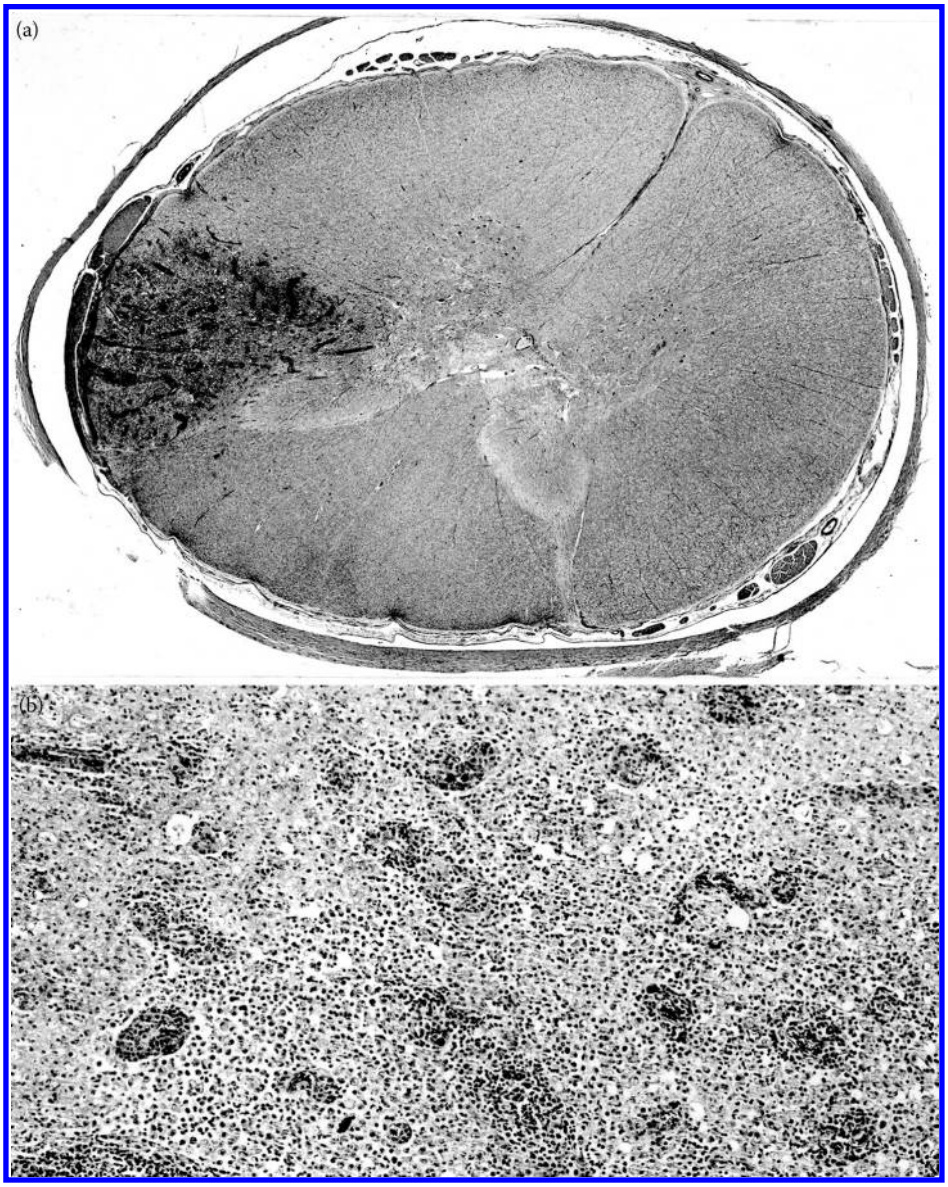


Figure 7.8 Encephalomyelitis due to a *Sarcocystis*-like organism in a naturally infected calf. H and E stain. (a) An area of necrosis in the spinal cord. (b) High-power magnification of (a), showing severe vasculitis and infiltration of macrophages.

BIBLIOGRAPHY

Complementary information pertinent to the subject matter of this chapter may be found in the following references:

Eosinophilic myositis: **12, 139, 151, 309, 526, 652, 654, 724, 725, 730, 907, 1472, 1511, 1777, 1778, 1840, 1943, 2014**.

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2047–2050, 2057, 2087, 2118, 2135, 2163, 2164, 2171, 2172, 2202, 2233, 2234, 2269, 2273, 2307, 2318, 2329, 2350, 2356, 2370.

Food safety/zoonosis: **150, 210, 211, 397, 675, 771, 807, 1221, 1337, 1382, 1427, 1727, 1776, 1943, 1947, 1982, 1984, 2022, 2042, 2043, 2304**.

Miscellaneous: **31, 54, 55, 56, 58, 125, 154, 177, 255, 594, 874, 1228, 1271, 1362, 1516, 1545, 1547, 1920, 1935, 1938, 1950, 1965, 2017, 2076, 2090, 2105, 2136, 2137, 2138, 2148, 2162, 2166, 2186, 2188, 2217, 2220, 2258, 2259, 2260, 2315, 2339, 2351, 2364, 2366**.

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Prevalence/epidemiology: **37, 54, 116, 126, 127, 132, 139, 150, 170, 219, 221, 275, 277, 318, 323, 331, 332, 340, 471, 564, 634, 640, 738, 747, 764, 773, 857, 868, 872, 884, 896, 905, 928, 1029, 1143, 1185, 1193, 1200, 1213, 1215–1217, 1221, 1223, 1233, 1241, 1282, 1283, 1300, 1335, 1337, 1343, 1372, 1417, 1427, 1446, 1449, 1521–1524, 1529, 1541, 1542, 1546, 1551, 1553, 1601, 1616, 1776, 1784, 1786, 1787, 1852 1916, 1930, 1931, 1933, 1944, 1964, 1965, 1967, 1968, 1973, 1978, 2003, 2012, 2014, 2017, 2036, 2040, 2056, 2061, 2064, 2070, 2075, 2081, 2101, 2119, 2126, 2151, 2190, 2215, 2226, 2231, 2242, 2243, 2249, 2253, 2265, 2297, 2302, 2306, 2308, 2325, 2338, 2354, 2357, 2358, 2360, 2373**.

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CHAPTER 8

Sarcocystosis in Sheep (*Ovis aries*)

There are 4 species of *Sarcocystis* in sheep (Figure 8.1). *S. tenella* and *S. arieticanis* are transmitted via canids; *S. gigantea* and *S. medusiformis* are transmitted via cats. *Sarcocystis* species are prevalent in sheep worldwide (Table 8.1).

8.1 *SARCOCYSTIS TENELLA* (RAILLIET 1886) MOULÉ 1886

8.1.1 Definitive Hosts

Dog (*Canis familiaris*), Coyote (*Canis latrans*), and Red fox (*Vulpes vulpes*).

8.1.2 Structure and Life Cycle

Sarcocysts are microscopic, up to 700 µm long, and are found in striated muscles, including heart and tongue, and in the CNS.[367] The sarcocyst wall is 1–3 µm thick and has vp up to 3.5 µm long and up to 0.5 µm wide (Figure 8.1a).[367,1796] There are no microtubules in the villi, but plaques are present (Figure 8.2a).

Information on the life cycle is summarized in Table 8.2. There are 2 well-documented generations of schizogony in the blood vessels; a possible third generation was reported in 2 lambs at 36 DPI in macrophages in visceral lymph nodes and Küpffer cells.[1288] Schizonts were small (7.4 × 5.1 µm) and contained 6–9 merozoites. Differences in size, parasitemia, and a number of phases of schizogony reported by various authors[367,804,818,1036,1037,1245,1288] might reflect the techniques used, the number of stages measured, or the possibility that *S. arieticanis* was present as a contaminant.

Sarcocysts first seen at 35 DPI had a 1-µm-wide sarcocyst wall that appeared nonstriated until over 52 DPI, when the wall was up to 2 µm thick and appeared cross-striated, type 14. At 75 DPI, sarcocysts were up to 700 µm long and contained both metrocytes and bradyzoites.[367,1170]

8.1.3 Pathogenicity

S. tenella can cause anorexia, weight loss, fever, anemia, loss of wool, abortion, premature birth, neural signs, myositis, and death, depending on the number of sporocysts ingested.[3,390,551,672,867,1243,1245,1250,1254,1258,1261,1393] Doses of 1000–100,000 sporocysts have been tested for their effect on lambs. Data using doses lower than 50,000 sporocysts are reviewed.

8.1.3.1 Clinical Disease

In Australia, 288 (179 inoculated, 109 uninoculated) 2- to 4-week-old *Sarcocystis*-free lambs were examined at 1 and 4 months PI.[1250,1261] At low doses of 1000–5000 sporocysts, as few as

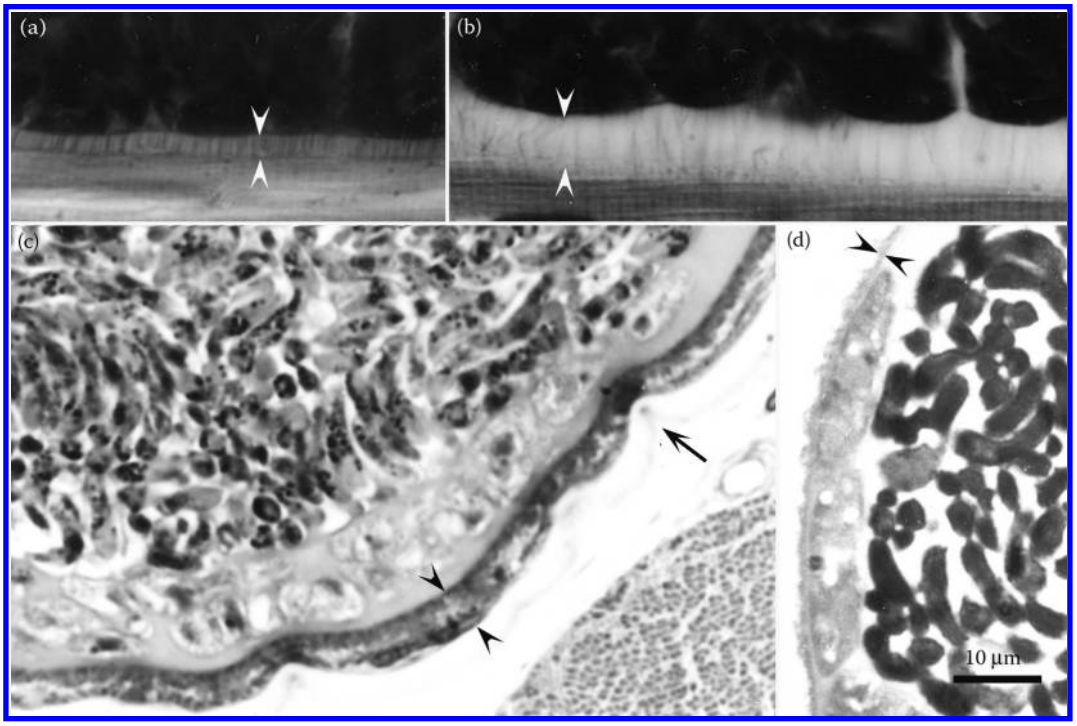


Figure 8.1 A comparison of sarcocyst walls of the 4 species of *Sarcocystis* in sheep. Arrowheads point to the tips of villi or outer margin of the sarcocyst wall. (a) *S. tenella* with thick vp. (b) *S. arieticanis* with long, hirsute protrusions. (c) *S. gigantea* with thin sarcocyst wall (arrow). A connective tissue capsule (arrowhead) surrounds the sarcocyst wall. (d) *S. medusiformis* with thin sarcocyst wall with small protrusions. (a and b) H and E stain. (d) Toluidene blue stain. Bar applies to all parts.

1000 sporocysts reduced hematocrits at 1 month PI, but both hematocrits and weights at 4 months were the same as in uninoculated lambs (Table 8.3). Ingestion of 10,000 sporocysts significantly depressed wool-growth in lambs at 1 month of age, but not at 5 months of age.[1258]

In Germany, ingestion of 25,000 sporocysts affected the health and growth of lambs.[551] The mean carcass weight and mean daily weight of 40 infected lambs in 4 groups of 10 lambs each, based on identical weights, was compared with those of 40 noninfected control lambs; all lambs were reared and managed on a commercial farm. Three infected lambs died and 1 lamb was condemned at slaughter because of carcass appearance. The carcass weights of the 4 groups of the inoculated lambs were 27%, 15.9%, 18.5%, and 6.5% less than the weights of the uninfected controls.[551]

8.1.3.2 Effect on Parturition and Reproductivity

S. tenella can cause abortion in sheep.[1037] Of 12 ewes bred 83–85 days earlier, 4 were each fed with 50,000, 100,000, or 500,000 sporocysts. Of those fed 50,000 sporocysts, 1 was not pregnant, 1 aborted 33 DPI, and 2 lambed normally; all survived the acute phase of sarcocystosis. Of those fed 100,000 sporocysts, 1 aborted 40 DPI, 1 lambed normally, and 2 ewes carrying dead fetuses became moribund and were euthanatized. Of those fed 500,000 sporocysts, 3 died 27 and 39 DPI, and 1 became moribund and was euthanatized 39 DPI; 2 of these ewes aborted 28 and 30 DPI and 2 others had dead fetuses in utero. *Sarcocystis* was not found in tissue sections of any fetus or lamb.[1037]

In another study to determine the effects of sporocyst dosage on reproduction, 20 *Sarcocystis*-free ewes were allotted to 5 groups, A–E (Table 8.4).[1254] Ewes in Group A (6 ewes) were grazed on

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Table 8.1 Reports of *Sarcocystis* Prevalence in Sheep

Country	Year	Source	No. Tested	Tissue	No. Positive	Species	Method				Reference
							Histology	Digestion	PCR	Bioassay	
Australia, South	1974–75	Abattoir	864	E, H, D, M	805 (93.2%) microscopic, 54 (6.7%) macroscopic	*S. gigantea*, *S. medusiformis*	Gross, trichinoscopy	—	—	—	**1290**
Australia, North	NS	Abattoir	51 lambs	H	47 (92.1%)	*S. tenella*, *S. arieticanis*	Histology	Pepsin digestion	—	—	**1405**
Australia, Perth	1989–92	Abattoir	146	H, D (200 g)	138 (95.2%)	*S. tenella* 9.8%, *S. arieticanis* 48.8%, mixed 41.5%	Yes	Pepsin digestion	—	—	**1544**
Austria	1991–92	Abattoir	500	E	313 (62.6%)	*S. tenella* 25.6%, *S. arieticanis* 24.0%, *S. gigantea* 2.4%	Gross, trichinoscopy	Trypsin digestion	—	—	**834**
Brazil	NS	Abattoir	602	H, D, Brain	2 (of 22 serologically positive for *Toxoplasma*)	*S. tenella*	—	Pepsin digestion	PCR-RFLP	—	**276**
Czech Republic	NS	Abattoir	342 lambs	Blood, muscle NS	35.7% microcysts, 94.4% antibodies by IFAT	NS	NS	NS	—	—	**1699**
Czech Republic	1979–80	NS	100	E	92 (92.0%)	*S. tenella* in all positive animals, *S. gigantea* in 37.0%	Gross, trichinoscopy	Pepsin digestion	—	—	**764**
Czech Republic	1985–88	Abattoir	1014 different ages	E, D	611 (60.3%)	*S. gigantea* in 3 sheep	—	Trypsin digestion	—	—	**1698**
Ethiopia, Northwest	NS	Abattoir	208	E, D, H, M	195 (93.7%)	NS	Histology	—	—	—	**1835**

(*Continued*)

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Table 8.1 (*Continued*) Reports of *Sarcocystis* Prevalence in Sheep

Country	Year	Source	No. Tested	Tissue	No. Positive	Species	Method				Reference
							Histology	Digestion	PCR	Bioassay	
Germany, Bavaria	1978	Abattoir	500	E, D	427 (85.4%)	*S. tenella* in 63.%, and *S. arieticanis* 84.8% of 166 sheep	Gross examination, trichinoscopy	Trypsin digestion	—	—	**117**
India, Uttar Pradesh	1990	Abattoir	32	E, D	23 (71.8%)	*S. tenella* (56.5%), *S. arieticanis* (26.0%)	Gross examination, muscle squash	—	—	—	**1528**
India, Orissa	NS	Abattoir	102	E, T, H, D, M	83 (81, 3%); H: 90.0%, T: 85.8%, E: 90.2%, D: 81.4%, M: 78.4%	*S. tenella*	Muscle squash	Pepsin digestion	—	—	**1207**
India, Hissar	NS	Veterinary laboratory and abattoir	32	D, H	26 (81.2%)	NS	—	Pepsin digestion	—	—	**760**
India, Andhra Pradesh	1961–82	Veterinary laboratory	3335	NS	1295 (37.3%)	NS	Histology	—	—	—	**1448**
India, Andhra Pradesh	NS	Abattoir	197	T, E, H, D, M	174 (88.3%)	*S. tenella*	—	Pepsin digestion	—	—	**1784**
Iraq	1992–96	Abattoir	605	E, H, D, M	587 (97.0%), macroscopic cyst in 25 (4.1%)	NS	Gross examination, muscle squash	Pepsin digestion	—	—	**1030**
Iraq, Duhok	NS	Abattoir	175	E	25 (14.3%)	*S. gigantea*	Gross examination	—	—	—	**24**
Iran, North Khorasan	2012–13	Abattoir	40	H, M	40 (100%)	*S. tenella*, *S. gigantea*	Gross examination	Pepsin digestion	PCR-RFLP, sequencing	—	**58**
Iran	NS	Abattoir	110	E	78 (70.9%)	NS	—	Muscle squash	—	—	**1449**
Iran, Ardabil	2004–05	Abattoir	2110	E, D, M	716 (33.9%)	NS	Gross examination	—	—	—	**294**

(*Continued*)

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Table 8.1 (*Continued*) Reports of *Sarcocystis* Prevalence in Sheep

Country	Year	Source	No. Tested	Tissue	No. Positive	Species	Method			Bioassay	Reference
							Histology	Digestion	PCR		
Iran, Fars	1992–94	Abattoir	1362	E, D, T, H, M	786 (57.7%)	*S. tenella*, *S. arieticanis*, *S. medusiformis*, *S. gigantea*	Gross examination, histology	Pepsin digestion	—	Dog, cat, hamster and Guinea pig	**1338**
Iran, North Khorasan	2013	Abattoir	32	H, D, M	32 (100.0%)	NS	—	Pepsin digestion	—	—	**1524**
Iran, Khouzestan and Lourestan	NS	Abattoir	NS	E, D, M and macroscopic cysts	NS	*S. gigantea*	Gross examination	Pepsin digestion	PCR-RFLP	—	**775**
Iran, Urmia	2011-12	Abattoir	638	E, D, M	235 (36.8%)	*S. medusiformis, S. gigantea*	Gross examination	Pepsin digestion	RFLP-PCR	—	**2393**
Japan, Saitama	1994–96	Abattoir	100	H, D-50 g	45 (45.0%)	12% *S. arieticanis* 45% *S. tenella*	Histology, TEM	—	—	Yes, dogs shed sporocysts	**1517**
Japan, Saitama	NS	Abattoir	16	D, masseter, M	2 (12.5%)	*S. mihoensis* in both cases	LM, TEM, SEM	—	—	Dog and cat; only dogs shed sporocysts	**1519**
Jordan	1986–88	Abattoir	620	E, D	Macroscopic: 70 (11.3%), microscopic E: 44.1%, D: 50.1%.	*S. tenella, S. gigantea, S. medusiformis*	Gross examination, trichinoscopy	—	—	—	**13**
Mongolia	1998–99	Abattoir	777	D, H, T	753 (96.9%); D: 93.2%, H: 94.2%, T: 100.0%	NS	Trichinoscopy	—	—	—	**640**
Nigeria, North	NS	Abattoir	400	D, E	36 (9.0%)	No macroscopic, *S. tenella*	Gross examination, dissection	Pepsin digestion	—	—	**984**

(*Continued*)

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Table 8.1 (*Continued*) Reports of *Sarcocystis* Prevalence in Sheep

Country	Year	Source	No. Tested	Tissue	No. Positive	Species	Method			Bioassay	Reference
							Histology	Digestion	PCR		
Peru, Lima	NS	Abattoir	134	H, E	122 (91.0%); E: 39.6%, H: 91.0%	NS	Trichinoscopy	—	—	—	**170**
Romania			48	E, H, D	44 (91.7%); E: 81.6%, H: 79.9%, D: 68.8%	*S. tenella*	Gross examination, trichinoscopy	—	—	Dog, cat. Only dog shed sporocysts	**1753**
Saudi Arabia	2002–03	Abattoir	3050	T, E, D, H, M	2552 (83.7%)	"*S. moulei*"	Histology, TEM	—	—	Dog, cat. Only cat shed sporocysts	**26**
Saudi Arabia	2011	Abattoir	3250	E, T, D, H, M	2790 (85.8%); E: 79.0%, T: 68.7%, D: 81.3%, H: 46.8%, M: 67.7%.	*S. arieticanis*, *S. gigantea*	Gross examination, histology	—	—	Dog, cat. Dog shed sporocysts	**23**
Senegal, Dakar	NS	Abattoir	100	E	82 (82.0%)	*S. tenella*	Histology	Homogenization	—	—	**1786**
Slovakia	NS	Abattoir	134-Sample A 30-sample B	E, H, D, T	134 (100%) in sample A 30 (100%) in sample B	*S. tenella*-96.3%, *S. arieticanis* 3.7%, *S. gigantea* 5.2% No macrocysts in sample B	—	Trypsin digestion	—	—	**1557**
Spain, Zaragoza	1978–80	Abattoir	770	E	486 (63.1%) by trichinoscopy, 756 (98.2%) by digestion, 416 (54.0%) by gross examination	NS	Gross examination, trichinoscopy	Pepsin digestion	—	—	**1530**

(*Continued*)

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Table 8.1 (*Continued*) Reports of *Sarcocystis* Prevalence in Sheep

Country	Year	Source	No. Tested	Tissue	No. Positive	Species	Method			Bioassay	Reference
							Histology	Digestion	PCR		
Spain, Center and South	2009–10	Abattoir	5720	Carcasses	712 (12.4%)	*S. gigantea* and *S. medusiformis*	Gross examination	—	—	—	**1123**
Spain, North West	NS	Abattoir	100	E	36 (78.3%) by digestion, 54 (54.4%) macroscopic cysts by gross examination	NS	Gross examination, trichinoscopy	Pepsin digestion	—	—	**1384**
Sudan	NS	Abattoir	128	E, H, M, D	123 (96.1%); E: 46.1%, H: 84.4%, M: 89.8%, D: 66.4%	NS	—	Pepsin digestion	—	—	**868**
Turkey, Kirikkale	2005–06	Abattoir	112	E, D, M	66 (58.9%)	*S. tenella* (47.3%), *S. arieticanis* (1.23%)	Gross examination, histology	—	—	—	**1345**
USA, Maryland	NS	Abattoir	86	D, E	84 (97.7%)	NS	Dissection	Pepsin digestion	—	—	**878**
USA, Detroit, Michigan	1973	Abattoir	789 sheep, 306 lambs	E (20 g)	10.8% of 306 lambs 75.3% of 789 adult	NS	Gross examination, histology	Pepsin digestion	—	—	**1586**
USA, North-West and Texas	1983	Abattoir	512 ewes	T, E, D	430 (84.4%); T: 82.1%, E: 44.4%, D: 51.7%	84.0% *S. tenella*, 3.5% *S. arieticanis*, *S. gigantea* (% NS)	Gross examination, histology, TEM	—	—	Dog, cat, both shed sporocysts	**392**

E = esophagus, H = heart, D = diaphragm, T = tongue, M = skeletal muscle.

NS = not specified.

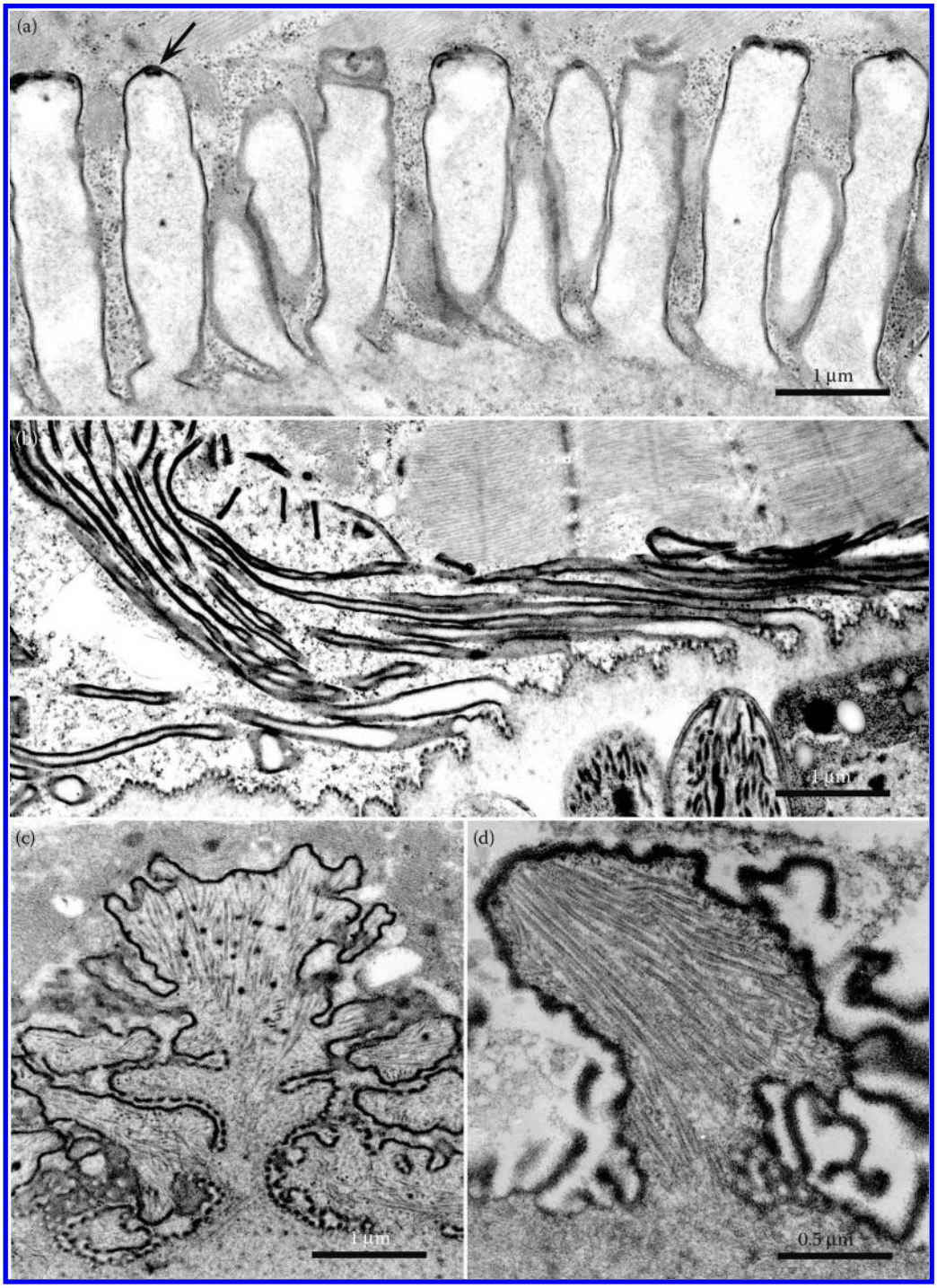


Figure 8.2 TEM showing a comparison of villar protrusions on the sarcocyst wall of the 4 species of *Sarcocystis* in sheep. (a) *S. tenella* with finger-like vp without microtubules. Unusual plaque structures are visible (arrow) at the tip of the vp. (b) *S. arieticanis* with long filamentous vp tapering at the distal end. (c) *S. gigantea* with cauliflower-like vp (arrow). (d) *S. medusiformis* with short, broad villi and numerous convoluted filaments arising from the main villus.

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Table 8.2 Comparison of Developmental Stages of *S. arieticanis*, *S. tenella* in Sheep

	S. arieticanis[a]	*S. tenella*[a]	*S. tenella*[b]
	First-Generation Schizonts		
Location	Mesenteric and mesenteric lymph node arteries	Arterioles and arteries throughout the body	Arterioles
Duration (DPI)	14–19	9–21	6–19
Peak development (DPI)	Unknown	16	Unknown
Size of meronts (μm)	45–80 × 35–50	29–45 × 24–32	13.4–35.5 × 12.2–15.1
No. of merozoites	100	Up to 168	18–28
Size of merozoites (μm)	6.8–7.5 × 2.3–3.0	7 × 1.5	Unknown
	Second-Generation Schizonts		
Location	Capillaries	Capillaries	Capillaries
Duration (DPI)	26–31	16–40	21–34
Peak development (DPI)	Unknown	25	Unknown
Size of meronts (μm)	Unknown	10.5–42 × 7–17.5	15.2 × 10.6 (12.0–27.4 × 7.5–15.3)
No. of merozoites	Unknown	Up to 54	18–38
Size of merozoites (μm)	6–7.5 × 2.3–3	6 × 15	Unknown
	Parasitemia		
Duration (DPI)	Unknown	14–16, 25–32	Unknown
Intraleukocytic multiplication	Yes	Yes	Unknown
Sarcocysts	Microscopic	Microscopic	Microscopic
First seen (DPI)	31	35	41
Maturation time (DPI)	70	75	Unknown
Location	Heart	CNS, heart	Heart
	Sarcocyst Wall		
Light microscopy	Thin	Thick	–
TEM	Hair-like protrusions of variable shape,[c] type 7	Palisade-like and cylindrical with smooth surface, type 14	–
Bradyzoites	4.6–6.4 × 1.9–2.3 μm	6.6–7.8 × 1.1–2.2 μm	–
References	**787, 817, 1565**	**367, 818, 1565**	**1288**

[a] In conventionally reared lambs.
[b] In SPF lambs.
[c] Snake-like 0.5–1.2 × 0.07–0.21 μm and trapezoidal 0.87–1.30 × 0.14–0.24 μm in size.[787]

a 0.23-hectare field 1 day after it had been sprayed with 1 million *S. tenella* sporocysts. In Group B, 3 ewes were fed 60,000 sporocysts each; in Group C, 3 ewes were fed 10,000 sporocysts each; and in Group D, 4 ewes were fed with 2500 sporocysts each. In Group E, 4 ewes were not given sporocysts. All 3 ewes fed with 60,000 sporocysts became severely ill and were euthanized 48, 50, and 84 DPI. No others became ill or died. All lambs were euthanized soon after birth, and their tissues were examined histologically. 2 of the 3 lambs born to ewes in Group B were very weak and were estimated to be 1–3 weeks premature. 1 lamb from an ewe in Group A was stunted and also appeared premature; focal areas of leukoencephalomalacia were found in the cerebrum and midbrain; the accompanying placenta had small foci of necrosis. Lesions were not seen in other lambs. *Sarcocystis* was not seen in tissues of any lambs.

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Table 8.3 Comparison of Hematocrits and Weights of Lambs Infected with 1000–10,000 *S. tenella* Sporocysts and of Uninoculated Controls

			Hematocrits (%)		Weights (kg)		
			1 Month	4 Months	0	1 Month	4 Months
Expt. No.	No. of Lambs	Dose	PI	PI	Day	PI	PI
1[a]	43	0	36	37	9	22	32
	38	5000	28	35	11	22	28
	37	10,000	25	34	10	20	27
2[b]	32	0	36	38	17	25	30
	32	1000	32	35	18	27	31
3[b]	34	0	37	37	13	20	28
	35	2500	31	35	12	19	27
	37	5000	29	35	12	19	26

Source: Data from Munday, B.L., 1979. *Vet. Parasitol.* 5, 129–135; Munday, B.L., 1986. *Vet. Parasitol.* 21, 21–24.
[a] Number of sarcocysts per cubic millimeter were <1, 117, and 102, respectively, in 3 groups.
[b] Sarcocysts were not found in control lambs, but were found in inoculated lambs.

Table 8.4 Effect of *S. tenella* Sporocysts on Lambing Weights

				Lamb Weights (kg) and Number of Lambs	
Group	No. of Sporocysts	No. of Ewes	No. of Sarcocysts/ mm^3 [a]	Singles	Twins
A	Grazed infected paddock	6	333	2.25 (2)	3.21 (8)
B	60,000	3	1250	2.38 (3)	None known
C	10,000	3	530	3.95 (2)	2.5 (2)
D	2500	4	315	4.0 (3)	2.38 (2)
E	None	4	6	3.6 (3)	3.25 (2)

Source: Data from Munday, B.L., 1981. *Vet. Parasitol.* 9, 17–26.
[a] Biopsied 1 day before and 90 DPI. No sarcocysts were seen in preinoculation biopsies.

8.1.4 Immunity and Protection

Oral inoculation of low numbers of *S. tenella* sporocysts induces protective immunity in sheep.[612] In 20 *Sarcocystis*-free 1-year-old sheep divided into 5 groups of 4 sheep each, sheep in each group were fed 0, 5, 50, 500, or 5000 sporocysts and were challenged with 50,000 homologous sporocysts apiece 13 weeks later.[612] The sheep were euthanized 10 weeks after the challenge infection. In the sheep fed 50 or 500 sporocysts, there were fewer sarcocysts, lower body temperatures, and higher hematocrits than in the sheep fed 0 or 5 sporocysts. The sheep fed 5000 sporocysts became ill.[612]

Protection was reported in sheep inoculated with 150,000 *S. tenella* sporocysts attenuated with ultraviolet irradiation (UV-30 to UV-60 lamp for 30 min) weekly for 3 weeks and then challenged with 15,000 untreated sporocysts.[3] Clinical signs were milder in vaccinated *versus* unvaccinated infected sheep later challenged with 15,000 sporocysts.

Prophylactically administered anticoccidial drugs reduce clinical sarcocystosis.[1040,1041] Amprolium® or Salinomycin® was administered to sheep from 1 day before until 29 days after 100,000 or 1 million *S. tenella* sporocysts were fed. Amprolium premix® (50 or 100 mg/kg body weight) and Salinomycin (1 or 2 mg/kg) were mixed in the daily grain ration. Both drugs reduced clinical sarcocystosis, but did not prevent the completion of the life cycle for some parasites. Then, 63 days after sporocysts were fed, lambs given Salinomycin were challenged with 1 million *S. tenella* sporocysts, and were found to have developed a protective immunity.[1041]

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8.2 *SARCOCYSTIS ARIETICANIS* HEYDORN 1985

8.2.1 Definitive Host

Dog (*Canis familiaris*).

8.2.2 Structure and Life Cycle

Sarcocysts are found in striated muscles, but not in the CNS. They are up to 900 μm long, thin-walled (<1 μm), and have hirsute projections 5–9 μm long (Figure 8.1b). The vp lack microtubules and, thus, are folded over the sarcocyst wall (Figure 8.2b), type 7b.[23,392,787,819,940,1557]

Salient life cycle features are summarized in Table 8.2.

8.2.3 Pathogenicity

S. arieticanis is less pathogenic than *S. tenella*.[817] A group of 5 sheep fed 50,000, 1 million, 10 million, or 30 million sporocysts developed 2 peaks of fever. The first peak of fever occurred 14–16 DPI, and the second peak was 26–31 DPI.[817] Sheep fed 50,000 or 1 million sporocysts survived the acute phase of sarcocystosis. Sheep fed 2, 10, or 30 million sporocysts died between 16 and 31 DPI.

8.3 *SARCOCYSTIS GIGANTEA* (RAILLIET 1886) ASHFORD 1977

8.3.1 Definitive Host

Domestic Cat (*Felis catus*).

8.3.2 Structure and Life Cycle

S. gigantea sarcocysts are found primarily in the muscles of the esophagus, larynx (Figures 1.5b and 8.3), and tongue, and, to a lesser extent, in the diaphragm and the rest of the

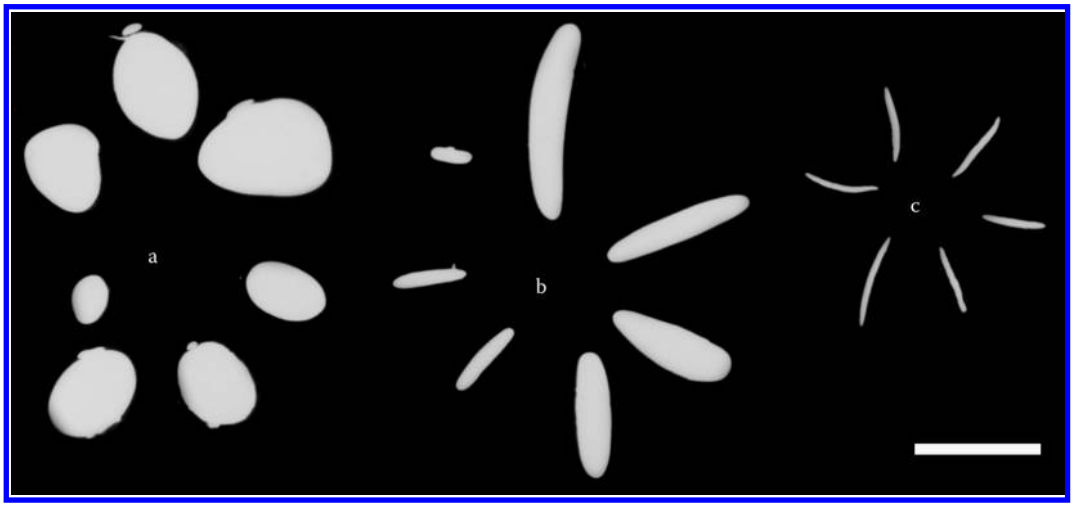


Figure 8.3 Macroscopic sarcocysts in naturally infected sheep: (a) fat *S. gigantea* sarcocysts from the esophagus, (b) slender *S. gigantea* from the diaphragm, and (c) slender *S. medusiformis* from the abdomen. (Courtesy of S. More.)

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carcass.[388,712,1258,1259,1572] Sarcocysts have not been found in the heart or the CNS. Macroscopic sarcocysts are found mainly in old sheep. Such sarcocysts are up to 1 cm long, dull white, round, oval, or pear-shaped, sometimes resembling rice grains (Figures 1.5b and 8.3). The sarcocyst wall is thin (<2 μm), smooth, and often surrounded by a PAS-positive connective tissue secondary cyst wall (Figure 8.1c). Ultrastructurally, the sarcocyst wall has cauliflower-like vp, type 21[1260] (Figure 8.2c). The septa are thin (<1 μm wide). In older sarcocysts, live bradyzoites are located peripherally, and the centers of the sarcocysts are often empty.

Little is known of the endogenous development of *S. gigantea* in sheep. Only 1 generation of schizogony is known.[1298] First-generation schizonts were found 7 and 14 DPI, but not 21, 28, and 35 DPI. They were 21–44 × 5–12 μm and were located in capillaries and arterioles in the lung, kidney, and brain.[1298] A sarcocyst was first seen 40 DPI; it measured 71.5 × 8 μm and contained only metrocytes. Bradyzoites were first seen 119 DPI.[1260] Between 10 and 14 months, PI sarcocysts grew up to 1 mm.[388,1260] At 47 months, PI sarcocysts were up to 7.5 × 5 mm. Sarcocysts first became infectious between 230 and 265 DPI. Sarcocysts grew for 4 years or more, and bradyzoites were not highly infectious to cats.[231,388,613,614,674,1259] Cats excreted sporocysts after a prepatent period of 9–11 days.[745]

8.3.3 Pathogenicity

S. gigantea is mildly pathogenic for sheep. Other than fever, lambs inoculated with 1 million sporocysts did not develop clinical signs.[388,1260] The only economic losses reported were due to carcass condemnation after sanitary inspections.[241]

8.4 *SARCOCYSTIS MEDUSIFORMIS* COLLINS, ATKINSON, CHARLESTON 1979

8.4.1 Definitive Host

Cat (*Felis catus*).

8.4.2 Structure and Life Cycle

S. medusiformis sarcocysts are up to 8 mm long and 0.2 mm wide and are found primarily in the diaphragm, abdominal muscles, and the carcass (Figure 8.3). The sarcocyst wall is thin (<2 μm) (Figure 8.1d), and there is no secondary wall.[1211] Ultrastructurally, the vp are trapezoidal. Serpentine filaments arise from the villar surfaces[240] (Figure 8.2d), type 20.

The endogenous development up to 188 DPI is not known. Immature sarcocysts were found in sheep at between 188 and 1132 DPI.[1299] Sarcocysts at 188 and 260 DPI were microscopic; at 443 DPI they were 2–3.5 mm long; at 765 and 1132 DPI they were 4–5 mm long. Cats fed these sarcocysts did not shed sporocysts; therefore, maturation time is unknown.

Cats excreted sporocysts with a prepatent period of 15–30 days.[240,1156]

8.5 REPORTS OF UNUSUAL/RARE SPECIES OF *SARCOCYSTIS* IN SHEEP

8.5.1 *Sarcocystis mihoensis* Saito, Shibata, Kubo, Itagaki 1997

This species was reported only once in 2 of 16 sheep from a farm in Miho, Ibaraki Prefecture, Japan.[1519] Sarcocysts were detected in skeletal muscles. They were up to 2.1 mm long and up to 300 μm wide. The sarcocyst wall was 10–12 μm thick and contained palisade-like vp. Ultrastructurally, the vp were unique type 39 (Figure 1.56a), wide at base, and gradually tapered

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towards the free end. The microtubules in vp were electron-dense with a mushroom-shaped end. 2 mongrel 6-month-old dogs fed infected muscles excreted sporocysts that were 15–16 × 8–9 µm in size; 2 cats fed the same material did not excrete oocysts.

8.5.2 *Sarcocystis gracilis* Rátz 1909

S. gracilis-like sarcocysts were found in 1 of 50 sheep in Italy.[677] Sarcocysts were 800 µm long and had a 5-µm-thick cyst wall. By TEM, the sarcocyst wall had 3.0–4.5 µm long villar protrusions with type 10 cyst wall characteristic of *S. gracilis* from roe deer (see Chapter 18).

8.5.3 *Sarcocystis microps* Wang, Wei, Wang, Li, Zhang, Dong, Xiao 1988

Oval-shaped sarcocysts were found in myocardium of 21 sheep from Qinghai, China.[1807] Sarcocysts were 150–300 × 50–80 µm. By TEM, the cyst wall was 1.13–1.4 µm, with 0.6–0.69 µm long T-shape vp. Bradyzoites were 8.5 × 3 µm. Also, bioassay in dog and cat was done, only dogs shed sporocyts measuring 14 × 9.1 µm.

8.6 CLINICAL SARCOCYSTOSIS IN NATURALLY INFECTED SHEEP

Scott[1572,1573] spent his career studying sarcocystosis in sheep in Wyoming, United States, and although his efforts were hampered because the life cycle was unknown, he established strong circumstantial evidence that heavy infections retard the growth of sheep. Reports of clinical sarcocystosis in sheep are summarized in Table 8.5.

There are 3 reports of neonatal sarcocystosis in naturally infected sheep. In Brazil, *Sarcocystis*-associated mortality was reported in sheep.[1390] In a flock of Corriedale sheep, 8 ewes aborted or had stillborn lambs. One stillborn lamb was necropsied. Encephalitis and myocarditis were associated with intravascular schizonts in these organs and in kidneys.

A similar case of *Sarcocystis*-associated mortality was observed in a stillborn lamb born in Denmark.[17] The predominant lesions were encephalitis and placentitis associated with schizonts. The encephalitis was characterized by multiple inflammatory foci throughout the brain, predominantly in the cerebrum. Foci of necrosis were surrounded by a zone of microglia and mononuclear cells. Immature and mature schizonts were seen in vascular endothelium, sometimes occluding the lumen (Figure 8.4). Placental lesions consisted of multiple foci of necrosis, inflammation, and mild calcification; numerous schizonts and merozoites were seen within the lesions.

In Australia, neonatal sarcocystosis was found in a 3-week-old lamb with generalized gliosis, and immature and mature schizonts were seen in the brain.[386]

There are several reports of sarcocystosis in weaned and adult sheep. In the United States, debilitation and lymphadenopathy have been found in association with a *Sarcocystis*-like organism.[1324] A 3-year-old ewe had been debilitated for 2 months and for 2 weeks had enlarged prescapular lymph nodes. Microscopically, the lymph node enlargement was found to be due to proliferation of connective tissue. Multiple foci of degeneration and calcified myofibers were seen in the heart, along with sarcocysts and schizonts. Schizonts were also seen in endothelial cells of lymphatic and blood vessels in lymph nodes. Mild arteritis and schizonts were seen in the adventitia. The schizonts were PAS-negative and contained merozoites 5–6 × 1.5–2 µm. Although it was stated that merozoites had rhoptries, none were visible in 3 illustrations. Rhoptries were absent from *Sarcocystis* merozoites (see Chapter 1). In Brazil, schizont-like structures were found in the brain of an adult ataxic sheep.[274]

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Table 8.5 Clinical Sarcocystosis in Naturally-Infected Sheep

Country	No., Age	History/Clinical Signs	Diagnosis	Reference
Australia	2, 5–6-month-old lambs	Neurological	Encephalitis, schizonts in brain	**396, 784**
	3-week-old lamb	Neurological	Encephalitis, schizonts in brain	**386**
Brazil	1 adult	Sudden death	Encephalitis, schizonts in brain	**274**
	1 stillborn	8 sheep aborted	Encephalitis, myocarditis, schizonts in several organs	**1390**
Bulgaria	1 sheep	Neurological	Encephalitis, sarcocysts in brain	**89**
Canada	4 adult ewes	Neurological	Encephalitis, sarcocysts in brain	**793**
Denmark	1 stillborn	Abortion	Encephalitis, pneumonia, schizonts	**17**
France	3-month-old lamb	Neurological	Meningitis	**746**
Germany	2 sheep submitted to inhalational anesthetic	Sudden death	Myocarditis	**148**
Hungary	6 adult ewes	Neurological	Encephalitis, sarcocysts in brain	**772**
Iran	2, 18- and 21-month-old sheep	Heart failure, atrioventricular block	Myocarditis, sarcocysts in heart	**1466**
Ireland	2 lambs	Neurological	No encephalitis, schizonts in spinal cord	**1154**
Spain	6 adult sheep	Edema, ataxia	Sarcocysts in muscle	**1608**
Turkey	1, 10-month-old lamb	Neurological	Encephalitis, sarcocysts in brain	**1861**
	10 sheep	Neurological	Encephalitis, schizonts in brain	**1346**
United Kingdom	1 adult	Neurological	Encephalitis, schizonts in brain	**1882**
	2 young lambs	Neurological	Encephalitis, schizonts and sarcocysts in brain	**1222**
	9 lambs	Neurological	Encephalitis, schizonts in brain	**899, 901, 1689**
	3 lambs	Neurological	Encephalitis, schizonts in brain	**1297**
	3 lambs	Neurological	Encephalitis, schizonts in brain	**1534**
	Several lambs within 3 different flocks	Neurological	Encephalitis, schizonts in brain	**160**
	1, 18-month ram	Heart failure	Myocarditis, sarcocysts in heart	**1575**
	1 lamb	Pneumonia, ascite	Schizonts in lung	**1563**
	20, 2–5 years ewes	Neurological	Sarcocysts in muscles	**900**
USA	3-year-old ewe	Neurological	Encephalitis, schizonts in brain	**1324**
	3 lambs	Neurological	Encephalitis, schizonts in brain, sarcocysts in muscles	**609**

Severe encephalomyelitis has been reported in sheep from Australia, Bulgaria, Canada, England, Hungary, Turkey, and the United States, and it is not clear if these were caused by *S. tenella* or another unknown species of *Sarcocystis*, because schizonts were located extravascularly in neural cells.[396,784] In Australia, severe encephalomyelitis was found in two 5- or 6-month-old lambs associated with an unidentified *T. gondii*-like organism.[784] Both lambs had noncoordination of the limbs, and flaccid paralysis. Both lambs were necropsied; their brains and spinal cords were fixed in formalin and studied histologically. Schizonts were seen in many cells, predominantly in white matter of the spinal cord. Merozoites had typical apicomplexan structures, including rhoptries. Reevaluation of the electronmicrographs indicated that dense granules were likely misidentified as rhoptries. A

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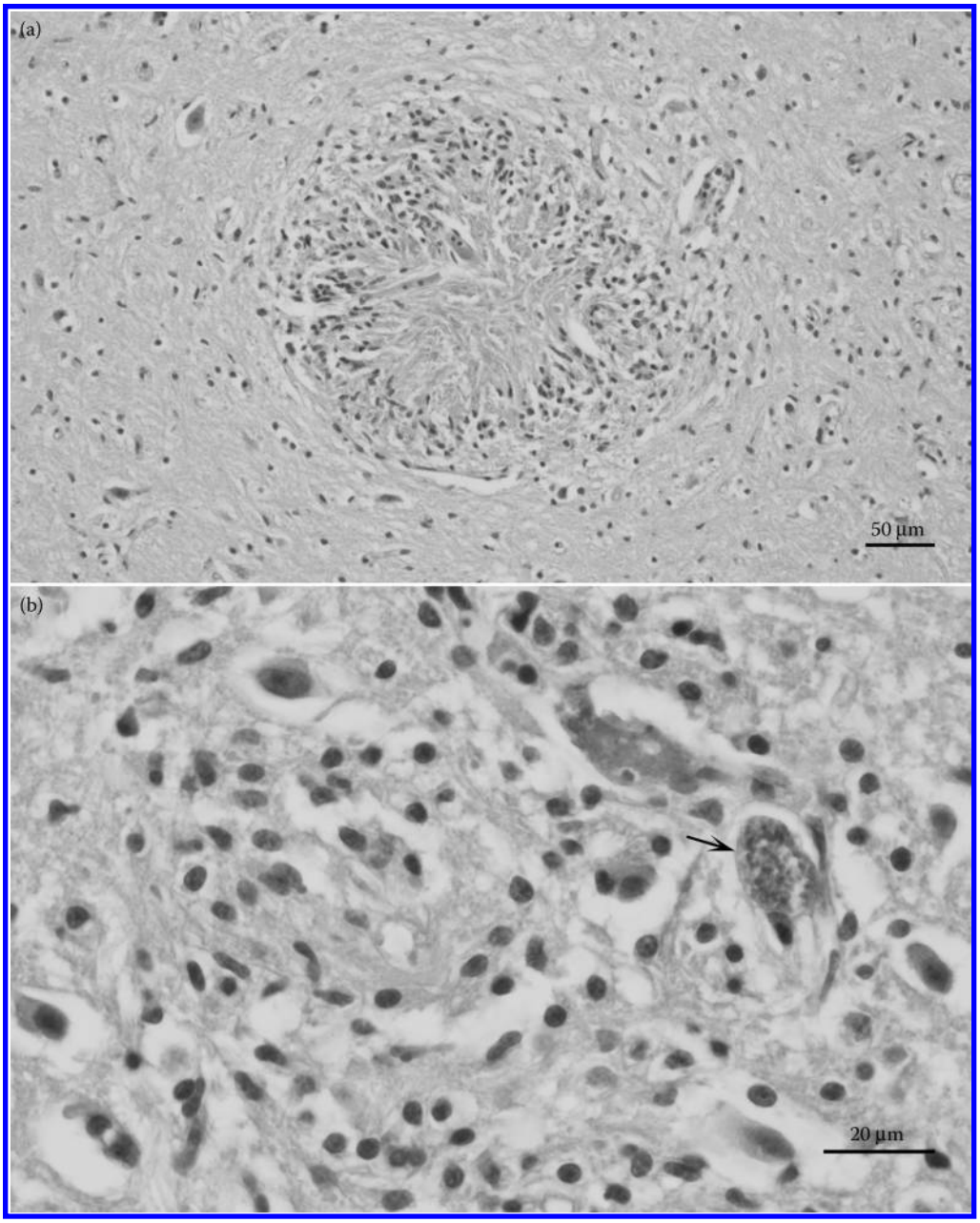


Figure 8.4 Encephalitis in the cerebellum of a stillborn ovine fetus with sarcocystosis. H and E stain. (a) Central necrosis surrounded by inflammatory cells. (b) A schizont in an inflammatory focus. (Adapted from Agerholm, J.S., Dubey, J.P., 2014. *Reprod. Dom. Anim*. 49, e60–e63.)

mature *S. tenella* sarcocyst was found in the brain of 1 of the lambs. Muscles were not examined. One of these cases was restudied ultrastructurally[396]; schizonts were located directly in host-cell cytoplasm, merozoites were formed by endopolygeny, and merozoites lacked rhoptries. An almost identical *Sarcocystis* infection was reported in 2 young lambs in England.[1222] Schizonts and mature sarcocysts were identified in the brains of both lambs.

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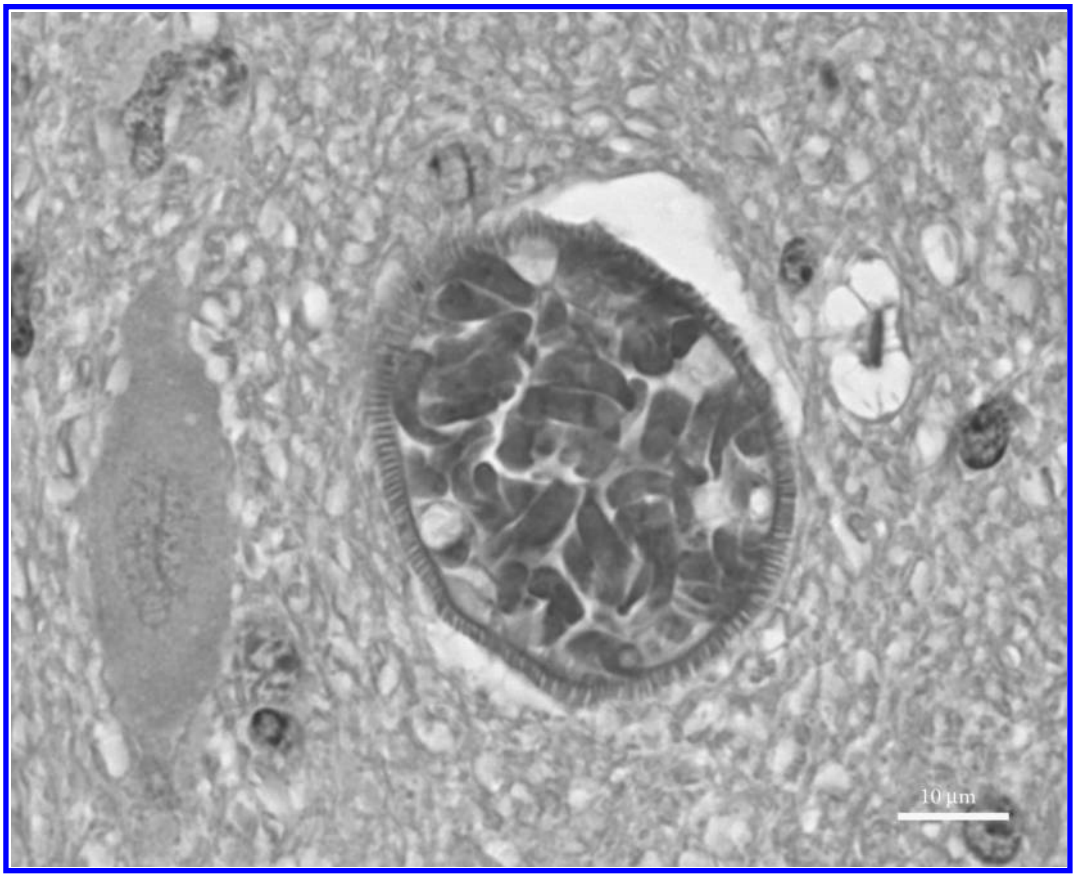


Figure 8.5 *S. tenella* sarcocyst in the cerebrum of a lamb with encephalitis. (Courtesy of Andrew Pregerine.)

On a farm in Indiana, United States, with 300 sheep, an outbreak of clinical sarcocystosis was reported in 5-month-old lambs.[609] Over a period of 4 weeks, 32 of 48 lambs in 1 pen were affected, and 22 died. The affected lambs were anorectic, lacked coordination, had stiff limbs, and exhibited trembling and general weakness. Three lambs were examined at necropsy; 2 had been euthanized and the third had died. Meningoencephalomyelitis and myositis were the predominant lesions in all 3 lambs. Necrosis and glial nodules were found throughout the brain and spinal cord. One immature schizont was found in the pons of 1 lamb. Numerous immature and mature *S. tenella*, sarcocysts similar to that shown in Figure 8.5, were seen in the muscles, particularly in the heart.

Two episodes of sarcocystosis occurred in a group of 1510-month-old Bluefaced Leicester rams in England[1297]; 12 of the 15 rams had high antibody titers to *Sarcocystis* antigen in an ELISA. In the first episode, 7 of 8 homegrown rams became ill, while seven purchased rams remained healthy. Paresis was the main clinical sign. Two mildly affected rams recovered. Three severely affected rams were euthanized and examined at necropsy. The predominant lesions in all 3 rams were edema, gliosis, and nonsuppurative myelitis. Extravascular schizonts were seen in the spinal cords of 2 of the 3 rams. Sarcocysts of *S. tenella* and *S. arieticanis* were detected in all 3 rams. In the second episode, in another flock of sheep in England, 15 ten-month-old lambs developed acute illness.[1534] Three lambs examined at necropsy had encephalitis, but schizonts were not seen. Numerous *S. tenella* sarcocysts were present in muscles. Similar outbreaks were observed in Black-faced sheep, less than 1 year old, from 3 unrelated flocks in England.[160] In each flock, sheep were

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recumbent, and from each flock 2 to 4 sheep were necropsied. All had myeloencephalitis, and schizonts were detected in the spinal cord or brain from at least 1 sheep. These schizonts reacted with a *Sarcocystis*-labeled antibody, and were negative for *Toxoplasma* and *Neospora*. A heavy infection with sarcocysts was seen in the muscles of all sheep examined.[160]

Neurological signs were observed in 10 of 350 sheep from a flock in Turkey.[1346] All 10 were examined at necropsy and had encephalitis. Schizonts were present in encephalitic lesions, and sarcocysts were seen in the muscles. Prophylactic treatment with Amprolium in another report 1 of 170 sheep flock in Izmir, Turkey became ataxic. Intravascular schizonts and mature sarcocysts were seen in sections of the brain.[1861]

In another unusual case, sarcocystosis associated with pneumonitis was reported in a lamb from a flock of 150 5- to 7 month-old sheep in England.[1563] The lamb had respiratory distress. The pathology report indicated pneumonitis and myocarditis with demonstrable *Sarcocystis* merozoies. Additionally, sarcocysts were seen in the heart. PCR-based on the DNA from the paraffin-embedded block of lung indicated *S. tenella*.

Severe *Sarcocystis*-associated myositis was reported in sheep in England. Heavy infection with sarcocysts was thought to be the cause of congestive heart failure in an 18-month-old Blue-faced Leicestershire ram.[1575] Clinical myositis was found in 20 of 600 ewes, associated with heavy infection with sarcocysts.[900] Four ewes were examined at necropsy. Muscle degeneration and myositis were found in all 4 ewes. Inadvertent ingestion of monensin sodium was considered to have aggravated myodegeneration.

In Germany, *Sarcocystis*-associated myocarditis was suspected as a contributory factor in mortality in sheep that failed to recover from inhalation anesthesia.[148]

In Hungary, muscle weakness, ataxia, loss of fleece, and 6.5%–10.8% mortality was reported in a flock of 210 Suffolk sheep.[772] Six affected sheep were examined at necropsy. Schizonts were seen in the heart and the brain. Sarcocysts of *S. arieticanis* were also identified in the hearts.

In Bulgaria, encephalitis was reported in a sheep, in association with *Sarcocystis* in the brain.[89]

Encephalitis associated with *S. capracanis* merozoites was reported from England,[622] but this report was questioned because of the mistaken identity of bradyzoites with merozoites in CSF.[469] In another case, *Sarcocystis* zoite-like structures, measuring 10.5–11.5 × 4–5 μm were seen in cerebrospinal fluid smears of 2 sheep with chronic coenurosis in Italy[1883]; it appears that these were probably bradyzoites released from intramuscular cysts while obtaining the CSF, because they were too large to be merozoites.

A case of laryngeal hemiplegia in an 18-month-old ram with respiratory distress and poor body condition associated with *Sarcocystis* spp. was reported in Spain.[1505] Histopathological examination of the laryngeal nerve revealed demyelination, degenerate axons, and atrophy. Multiple *Sarcocystis* spp. microcysts were found in the larynx musculature.

Whole carcasses or parts of adult sheep are condemned because of the presence of macroscopic sarcocysts of *S. gigantea* and *S. medusiformis*.[241] Carcasses are also condemned for eosinophilic myositis in lambs and in adult sheep.[563,907]

8.7 MOLECULAR STUDIES

Initially, *S. gigantea* was genetically characterized based on isoenzyme markers[616]; patterns were identical in all isolates from North American and Australasian origin. Genetic diversity seems to be very low for *S. gigantea*; RFLP–PCR detected null variation in the ITS-1 region among cysts within the same animal, and from different animals and countries (namely, Germany and Australia).[903]

Specific oligonuclotide probes for 18S rRNA of *S. tenella* were useful for diagnosis of acute sarcocystiosis.[840] A PCR based on 18S rRNA fragments was able to differentiate *S. tenella*, *S.*

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arieticanis, and *S. gigantea*.[1736] Other methodologies like RAPD–PCR,[910] nested-PCR,[788] or RFLP–PCR[58,276,775,1858] can diagnose *Sarcocystis* spp. infecting ovine livestock.

Based on the 18S rRNA sequencing, established phylogenetic relationships were established among ovine species (except *S. medusiformis*), and other major *Sarcocystis* spp.[843] Data from the mitochondrial *cox1* gene also contributed to this phylogeny.[701]

Little is known about genetic diversity of *Sarcocystis* spp. in sheep. The pioneering study of Jeffries et al.[903] exposed null divergence. Further studies targeting new markers for 18S rRNA or *cox1*[701] hopefully will provide data that demonstrates the real genetic variability in *Sarcocystis* spp. in sheep.

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Miscellaneous: **47, 57, 65, 214, 216, 240, 242, 243, 1899, 1902, 1904, 1912, 1945, 1970, 1976, 2015**.

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CHAPTER 9

Sarcocystosis in Goats (*Capra hircus*)

There are 3 species of *Sarcocystis* in domestic goats: *Sarcocystis capracanis*, *Sarcocystis hircicanis* (Figure 9.1), and *Sarcocystis moulei*.

9.1 *SARCOCYSTIS CAPRACANIS* FISCHER 1979

9.1.1 Definitive Hosts

Dog (*Canis familiaris*), coyote (*Canis latrans*), red fox (*Vulpes vulpes*), and crab-eating fox (*Cerdocyon thous*).

9.1.2 Structure and Life Cycle

Sarcocysts are up to 1000 µm long and 100 µm wide. The sarcocyst wall is up to 3 µm thick with radial striations and finger-like vp, type 14 (Figure 9.1a and c). Sarcocysts are found in virtually all skeletal muscles, in the CNS, and in the heart.[382] Information on the life cycle is summarized in Table 9.1 and Figure 9.2. There are 2 generations of schizogony in blood vessels.

9.1.3 Pathogenicity

S. capracanis is the most pathogenic species of *Sarcocystis* in goats.[239,359,815] It can cause fever, weakness, anorexia, weight loss, tremors, irritability, abortion, and death, depending on the number of sporocysts ingested. As few as 5000 sporocysts cause clinical disease, and 100,000 sporocysts are generally fatal.[359] Goats that recover from acute sarcocystosis remain unthrifty, have a dull, dry hair coat, and are predisposed to other infections.[372] Dosing with 1 million sporocysts cause severe acute sarcocytosis and goats die between 18 and 21 days.

9.1.4 Protective Immunity

Goats immunized once with 100 or 1000 sporocysts develop subclinical infections and become refractive to lethal challenge with large numbers of *S. capracanis* sporocysts.[358,372,373,381] This protective immunity persisted at least 274 DPI. Immunization with 1000 sporocysts provided better protection than with 100, and ingestion of 10 sporocysts induced no protection.[381]

9.1.5 Clinical Disease in Naturally Infected Goats

In Victoria, Australia, 8 of 38 Saanen goats aborted or gave birth to stillborn kids over a period of 3 months.[1094] 1 stillborn goat was necropsied. The predominant lesion was encephalitis characterized

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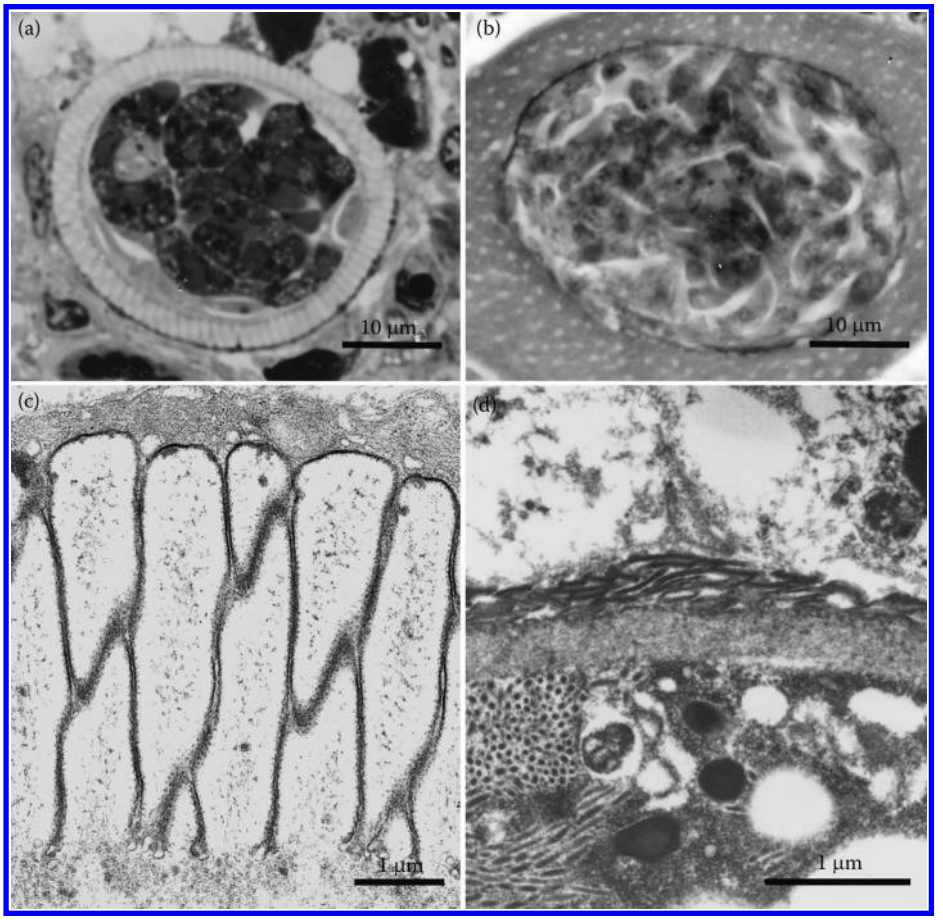


Figure 9.1 Sarcocyst walls of *S. capracanis* (a and c) and *S. hircicanis* (b and d) in skeletal muscles of goats. Arrows point to thick villi in *S. capracanis* and hirsute protrusions in *S. hircicanis*. (a) Toluidine blue stain, (b) H and E stain.

by necrosis, perivascular cuffing, and nonsuppurative encephalitis. Schizonts were seen in the blood vessels in the brain. Occasional schizonts were also identified in the lungs and kidneys.[1094]

9.2 *SARCOCYSTIS HIRCICANIS* HEYDORN AND UNTERHOLZNER 1983

9.2.1 Definitive Host

Dog (*Canis familiaris*).

9.2.2 Structure and Life Cycle

Sarcocysts are up to 2.5 mm long. The sarcocyst wall is thin (<1 µm) and has hirsute protrusions (Figure 9.1b and d).[815] The vp are of the type 7a, long, filamentous, without tubules, and thus folded over the cyst wall; they were up to 5 µm long.[1514] Little is known of the pathogenicity of this species. Of 2 goats fed 10 million sporocysts, 1 died 43 DPI and the other was euthanatized while moribund 35 DPI.

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Table 9.1 Comparison of Developmental Stages of *Sarcocystis* spp. in Goats

	S. capracanis	*S. hircicanis*	*S. moulei*
	First-generation Schizonts		Unknown
Location	Gut-associated arteries	Arterioles, intestinal lymph nodes, liver	—
Duration (DPI)	15–17	17–18	—
Peak development (DPI)	10–12	—	—
Size of meronts (μm)	26.1 × 17.4 (sections)	—	—
No. of merozoites	Up to 80	Up to 100 or more	—
Size of merozoites (μm)	5.5–7.1 × 2.5 (sections)	6–7.5 × 2.5–3.0 (smear)	—
			—
	Second-generation Schizonts		
Location	Capillary endothelium of all organs	Capillary endothelium of several organs	—
Duration (DPI)	14–24	28–35	—
Peak development (DPI)	19	—	—
Size of meronts (μm)	18.8 × 10.1	—	—
No. of merozoites	4–36	—	—
Size of merozoites (μm)	5.5 × 1.5 (sections)	6–7.5 × 2.5–3.0 (smear)	—
			—
	Parasitemia		
Duration (DPI)	17–24	—	—
Intraleukocytic	Yes	—	—
	Sarcocysts		
Size (μm)	Up to 1000	Up to 2500	Up to 1000
Earliest seen (DPI)	30	43	—
Maturation time (DPI)	64	84	—
Wall (μm)	2–3 thick, striated	<1, thin, hairy projections	3–4, cauliflower-like
Location	Striated muscle, CNS	Striated muscles	Striated muscles
Main references	**382, 815, 2013**	**816**	**7, 820**

— = unknown.

9.3 *SARCOCYSTIS MOULEI* NEVEU-LEMAIRE 1912

9.3.1 Definitive Host

Cat (*Felis catus*).

9.3.2 Structure and Life Cycle

Sarcocysts are dull white, ovoid, up to 17 mm long, and up to 7 mm wide, and typically have been found in the esophagus.[7,69] The sarcocyst wall is branched, cauliflower-like (type 21), and contains 13–15 μm long bradyzoites.[7,820]

Two sizes of macroscopic sarcocysts were found in domestic goats from Iraq.[69] Fat sarcocysts had thick rounded ends and were found only in the esophageal muscle. Bradyzoites in smears were 14.1 × 4.5 (11–17 × 3–6) μm in size. The thin sarcocysts were small, slender, and were also found in diaphragm and skeletal muscles. The contained bradyzoites were 12.2 × 2.9 (9–13 × 2–4) μm in size. However, it is uncertain if both types of sarcocysts represent the same

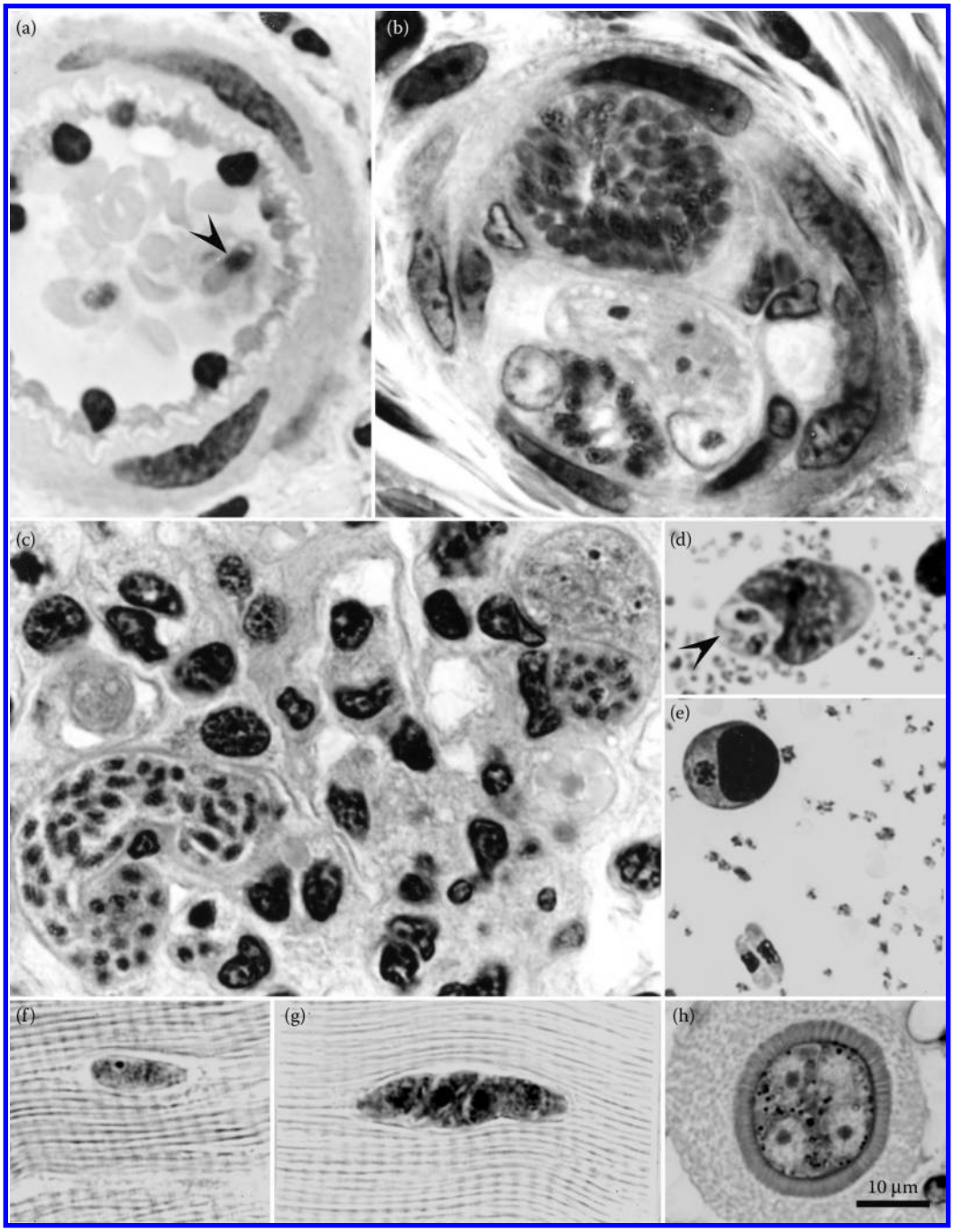


Figure 9.2 Developmental stages of *S. capracanis* in tissues of experimentally infected goats. (a) One sporozite (arrowhead) in the lumen of an artery at 5 DPI. (b) Three developing first-generation schizonts in an artery. The vascular lumen is occluded by hypertrophied endothelial cells and parasites at 10 DPI. (c) Second-generation schizonts and merozoites in renal glomerulus at 20 DPI. (d) Dividing merozoite in a monocyte-like cell in peripheral blood at 20 DPI. (e) An intracellular merozoite and 2 extracellular merozoites in peripheral blood at 20 DPI. (f) Binucleate sarcocyst at 35 DPI. (g) Sarcocyst with four nuclei at 35 DPI. (h) Sarcocyst with well-developed wall at 68 DPI. Bar applies to all parts. (From Dubey, J.P. et al., 1984. *Int. Goat Sheep Res*. 2, 252–265. With permission.)

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Table 9.2 Prevalence of *Sarcocystis* spp. in Domestic Goats

Country	Province	Year	Source	No	No. Positive	Species	Method	Bioassay	Reference
Brazil	Bahia	NS	Abattoir	250	E: 198 (79.2%)	*S. capracanis*	Gross examination, pepsin digestion	Dog and cat	**68**
Chile	Región Metropolitana	NS	Abattoir	266	244 (91.7%)	—	IFAT	—	**720**
Egypt	Cairo	2009–10	Abattoir	680	540 (79.4%)	*S. capracanis*	Gross examination, muscle squash, histology, TEM	Dog and cat	**1226**
India	Bihar	1985	Abattoir	288	146 (50.69%)	*S. hircicanis*, *S. capracanis*	Dissection	—	**1665, 1668**
India	Madhya Pradesh	1986–87	NS	76	51 (67.1%)	NS	Muscle squash	—	**1615**
India	Uttar Pradesh	1988–90	Abattoir	675	495 (73.33%)	*S. hircicanis*, *S. capracanis*	Muscle squash, pepsin digestion	—	**1589**
India	Maharashtra	1987–88	Not for human consumption	35	35 (100%)	*S. capracanis*	Muscle squash	—	**1593, 1595**
India	Maharashtra	1989	Abattoir	372	209 (56.2%)	*S. hircicanis*, *S. capracanis*	NS	—	**1801**
India	Uttar Pradesh	NS	NS	170	111 (65.3%)	*S. capracanis*	Muscle squash, pepsin digestion	—	**920**
India	Uttar Pradesh	1988	Abattoir	310	118 (58.4%); D: 60.0%, E: 58.9%, H: 55.1%	NS	Gross examination, pepsin digestion	—	**1525**
India	Jabalpur	NS	Abattoir	790	528 (66.9%)	*S. capracanis*	Gross examination, homogenization	—	**19**
India	Tripura	NS	Abattoir	80	23 (28.7%)	NS	Muscle squash	—	**1507**
India	Madhya Pradesh	NS	Abattoir	750	NS	*S. capracanis* and *S. hircicanis*	Isolation by washing with saline	Dog	**1592**
India	Akola	1994–95	Abattoir	100	25(25.0%)	All macroscopic	Gross examination, histology	—	**323**

(*Continued*)

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Table 9.2 (*Continued*) Prevalence of *Sarcocystis* spp. in Domestic Goats

Country	Province	Year	Source	No	No. Positive	Species	Method	Bioassay	Reference
India	Andhra Pradesh	NS	NS	111	80 (72.1%)	*S. capracanis*	Pepsin digestion	—	**1784**
India	Bengaluru	NS	Abattoir	100	72 (72.0)% E: 69.4%, H: 36.1%, D: 62.5%, T: 12.5%	72.0% *S. capracanis*; 21.0% *S. hircicanis*	Muscle squash, pepsin digestion	—	**278**
India	Orissa	NS	Abattoir	120	92 (76.6%); H: 76.6%, T: 85.8%, E: 80.4%, D: 76.6%, M: 68.3%	—	Gross examination, pepsin digestion	—	**1207**
India	Rajasthan	1989	Abattoir	703	480 (68.28%)	*S. capracanis*-like	Muscle squash	—	**1618**
India	Nagpur, Maharashtra	1996–97	NS	800	277 (34.62%)	NS	Muscle squash	—	**916**
India	Punjab	NS	Abattoir	45	9 (20.0%)	*S. capracanis*	Muscle squash, histology	—	**48**
Iran	Shiraz	NS	Abattoir	169	168 (99.4%)	Macroscopic in 28 (16.6%)	Pepsin digestion	—	**1599**
Iran	Tehran, Ghazvin	NS	Abattoir	NS	NS	All macroscopic: *S. moulei*	Muscle squash, pepsin digestion, PCR, TEM	—	**1229**
Iraq	Baghdad	1992–96	NS	826	278 (33.6%)	NS	Pepsin digestion: 97.4%; IFAT: 91.0%; muscle squash: 89.7%	Dog	**1030**
Iraq	North	1991–92	Abattoir	826	Macroscopic: 278 (33.6%); microscopic: 805 (97.4%)	*S. caprifelis*, *S. capracanis*	Gross examination, muscle squash, pepsin digestion, histology	Dog	**69**

(*Continued*)

Table 9.2 (*Continued*) Prevalence of *Sarcocystis* spp. in Domestic Goats

Country	Province	Year	Source	No	No. Positive	Species	Method	Bioassay	Reference
Japan	Niigata, Kanagawa	1986–89	Abattoir	15	2 (13.3%)	*S. capracanis*	Typsin digestion, histology, TEM	—	**873**
Japan	Saitama	1995	Abattoir	21	10 (47.6%)	*S. capracanis*, *S. hircicanis*	Dissection, histology, SEM	Dog	**1514**
Jordan	Central	1986–88	Abattoir	1261	Macrocysts in 46 (11.7%) of 393; microcysts in 711 (56.4%) of 1261	*S. capracanis*, *S. hircicanis*, *S. moulei*	Gross examination, trichinoscopy, IHA	—	**13**
Malaysia	Selangor	2014	Abattoir	105	55 (52.3%) by LM; 95 (90.0%) by PCR	*S. capracanis*	Muscle squash, histology, PCR	—	**1400**
New Zealand	Hawke's Bay	1977	Feral goats	60	17 (28.3%)	NS	Pepsin digestion, histology	—	**237**
Peru	Lima	NS	Abattoir	63	33 (52.4%); E: 76.2%; H: 52.4%	NS	Trichinoscopy	—	**170**
Philippines	Several	NS	NS	26	11 (42.3%)	*S. capracanis*	Gross examination, histology, TEM	—	**226**
Saudi Arabia	NS	NS	NS	NS	77.0%	NS	Gross examination, electron microscopy. Uncomplete	Cat and presumably dog	**26**
Senegal	Dakar	NS	Abattoir	75	66 (88.0%)	*S. capracanis*	Pepsin digestion, TEM	—	**1786**
Sudan	Several	NS	Abattoir	116	101 (87.1%); E: 32.8%; H: 35.3%; M: 53.4%; D: 50.0%	NS	Pepsin digestion, histology	—	**868**

D = diaphragm; E = esophagus; H = heart; M = skeletal muscle; T = tongue.
IFAT = immunofluorescent antibody test; IHA = indirect hemagglutination; LM = light microscopy.

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Sarcocystis species. The 18S rRNA gene of fat and thin macrocysts collected from the esophagus and other striated muscle of goats in Iran was sequenced, and it was found that both sizes of cysts were the same species.[7] Results matched with previously sequences for *S. moulei* reported in international databases.

Reports on macroscopic sarcocysts in goats[1231,1274,1275] were reviewed, and part of the life cycle of *S. moulei* was described.[820] In experimentally infected goats, sarcocysts were not macroscopic and were not infectious to cats at 6 months PI. At 19 months PI sarcocysts were up to 2×1 mm in size and infective to cats. At 86 months PI sarcocysts reached a size of $7–13 \times 5–8$ mm, and remained infective for cats. Cats fed sarcocysts shed sporocysts with a prepatent period of 10 days. Sporocysts were $11.6–13.1 \times 8.7–9.4$ μm.[820]

Review of all evidence[491,554,820] indicates that there is only 1 species of *Sarcocystis*, *S. moulei*, with macroscopic sarcocysts and transmitted by cats.

9.4 NATURAL INFECTIONS IN GOATS

To investigate the presence of *Sarcocystis* spp. in goat carcasses, gross examination is useful in many cases, but artificial digestion methods showed higher sensitivity yielding very high rates of prevalence of *Sarcocystis* spp. Table 9.2 summarizes surveys carried out on goats.

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Experimental infections: **39, 199, 239, 320, 321, 717, 718, 798, 820, 921, 986, 1349–1353, 1613, 1614, 1617, 1619, 1802, 1959, 1960, 2117, 2205, 2214, 2219, 2266, 2352**.

Life cycle and ultrastructure: **7, 39, 199, 334, 382, 815, 816, 820, 1226, 1514, 1617, 1618, 1896, 2305**.

Miscellaneous: **24, 733, 846, 1094, 1229, 1336, 1608, 1614, 1735**.

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CHAPTER 10

Sarcocystosis in Water Buffalo (*Bubalus bubalis*)

10.1 INTRODUCTION

There are 4 valid species of *Sarcocystis* in water buffalo (Table 10.1, Figure 10.1).[471] Endogenous development of schizonts and sarcocysts of any of the 4 species in buffalo is unknown or uncertain. None of the 4 species are considered pathogenic for buffalo, and there is no clinical report of acute sarcocystosis in buffalo.

Sarcocystis levinei (Dissanaike and Kan 1978) Huong, Dubey and Uggla 1997
Sarcocystis fusiformis (Railliet 1897) Bernard and Bauche 1912: The sarcocyst of this species was redescribed[474]
Sarcocystis buffalonis Huong, Dubey, Nikkila and Uggla 1997
Sarcocystis dubeyi Huong and Uggla 1999

10.2 PREVALENCE

Natural infections by *Sarcocystis* spp. in water buffalo are frequent; data are summarized in Table 10.2.

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Table 10.1 Species of *Sarcocystis* in Water Buffalo (*Bubalus bubalis*)

Character	*Sarcocystis fusiformis*	*Sarcocystis levinei*	*Sarcocystis buffalonis*	*Sarcocystis dubeyi*
Sarcocyst	Macroscopic	Microscopic	Macroscopic	Microscopic
Length	Up to 25 mm	Microscopic, <1 mm	Up to 8 mm	Up to 600 μm
Cyst wall	Thin, 2–5.2 μm	Thin	Thick, 3–7.7 μm	Thick
Cyst wall type—TEM	Type 21b, vp 6 μm long, with common stalk-like branches of a dead tree Microfilaments in vp	Type 7a, vp 0.5 μm at base, 0.08 μm at tip	Type 28, vp 12 μm long, narrow at base, expanded laterally, tapered distally. Microfilaments in vp	Type 10c, pallisade, bent at 45°, 4.5–8 μm long and 50–300 nm wide
Metrocytes	9.0–17.5 × 5.0–8.5 μm	Large	NS	NS
Bradyzoites	14.5–19.0 × 4.0–4.5 μm	9–12 × 2.5–4.0 μm	10.3–12.8 × 2.6 × 3.2 μm	8–11 × 2–2.5 μm
Definitive host	Cat	Dog	Cat	Unknown (not dog or cat)
Prepatent period—days	8–14	16–18	10	Unknown
Sporocyst (μm)	NS by Dissanaike and Kan (1978)[1654]	9.5–10.5 × 14.0–16.5 μm	7.5–9.5 × 12.5–13.5 μm	Unknown
Main references	**327,474**	**327,862**	**863**	**865**

NS = not stated.

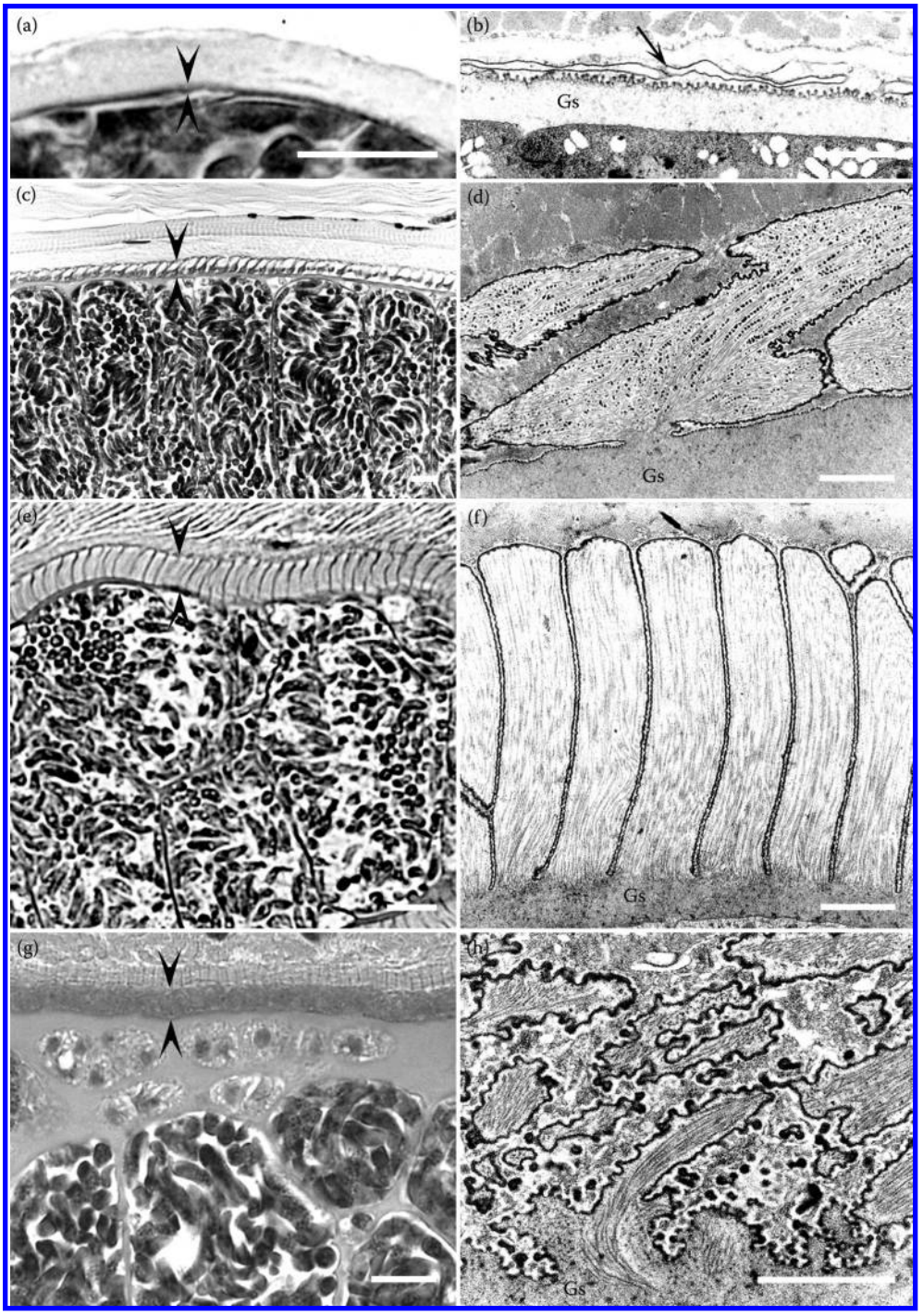


Figure 10.1 Sarcocyst walls of *S. levinei* (a, b), *S. buffalonis* (c, d), *S. dubeyi* (e, f), and *S. fusiformis* (g, h) from the water buffalo from Vietnam. (a, c, e, g), light micrographs, bar = 10 μm. (b, d, f, h) transmission electron micrographs, bar = 2 μm. Arrowheads point to the thickness of the sarcocyst walls. The vp (arrow) in *S. levinei* are slender, and folded over the sarcocyst wall, in *S. buffalonis*, have a narrow stalk, expanded laterally, in *S. dubeyi* they are elongated, and in *S. fusiformis* they are highly branched. (Courtesy of Arvid Uggla and Lam T. Huong.)

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Table 10.2 Prevalence of *Sarcocystis* Sarcocysts in Water Buffaloes (*B. bubalis*)

Country	Province	Year	No. Tested	No. Positive (%) and Species Involved	Method (Tissue)	Reference
Argentina	Northeast	NS	500	254 (50.8%)	IFAT	**971**
China	Yunnan	1988	50	*S. fusiformis*: (80%) *S. levinei*: (75%) *S.* sp: (58%)	Muscle squash	**1886**
Egypt	Cairo	NS	130	*S. fusiformis*: 65 (50.0%)	Muscle examination (O)	**488**
	El-Gharbia	2009	*35*	11 (31.4%), *S. dubeyi*	Histology, TEM	**827**
	Beni-Suef	NS	379	299 (78.9%), *S. fusiformis* and *S. levinei*	Gross examination, histology, pepsin digestion	**487**
	Alexandria	NS	300	203 (67.6%)	ELISA	**43**
	El-Gharbia	2011–12	528	*S. fusiformis*: 280 (85.4%) *S. buffalonis*: 65 (12.3%) Mixed infection: 358 (67.8%)	Histology, PCR, and sequencing	**492**
	Assiut	NS	90	23 (25.5%) by digestion and 85 (94.4%) by ELISA. *S. fusiformis*, *S. cruzi*, *S. levinei* and *S. hominis*	Histology, SEM, digestion, ELISA	**1190**
India	Mumbai	NS	50792	*S. fusiformis*: 7776 (15.31%)	Gross examination	**312**
	West Bengal (Kolkata)	1987	530	276 (52.08%) *S. fusiformis*: 200 (37.7%) *S. levinei*: 76 (14.3%)	Gross examination, pepsin digestion (H, T, D, O)	**106**
Iraq	Baghdad	1992–96	580	Macroscopic: 91 (15.6%) Microscopic: 481 (82.9%) by pepsin digestion and 464 (80.0%) by IFAT	Muscle examination, IFAT, pepsin digestion, bioassay in dogs (O, T, H)	**1030**
Iran	Khuzestan	NS	100	*S. levinei* and *S. dubeyi*: 83 (83.0%) 3 carcasses with macroscopic cysts (*S. buffalonis*)	Histology	**1339**
	Khuzestan	2009	NS	Macroscopic and microscopic sarcocysts of *S. fusiformis*: NS (NS)	Muscle examination, PCR-RFLP	**1340**

(*Continued*)

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Table 10.2 (*Continued*) Prevalence of *Sarcocystis* Sarcocysts in Water Buffaloes (*B. bubalis*)

Country	Province	Year	No. Tested	No. Positive (%) and Species Involved	Method (Tissue)	Reference
Italy	Northern	1994–95	249	82 (32.9%), *S. fusiformis*, *S. levinei*	Muscle squash, H	**163**
Malaysia	Selangor	2011–12	18	12 (66.7%), *S. levinei* and *S. cruzi*	Muscle squash, H	**1029**
Philippines	Luzon, Visayas, Mindanao	NS	142	92 (64.8%), *S. fusiformis*, *S. levinei*	Histology, TEM	**220, 222, 223**
Romania	Bucarest	NS	NS	*S. fusiformis*	Bioassay in dog and cats	**1429**
Sri Lanka	Colombo	1999	123	93 (75.6%)	ELISA	**932**
Thailand	Ratchaburi	NS	211	81 (38.4%), *S. cruzi*	Histology, digestion	**1233**
Turkey	Elazig	1985–86	183	Microscopic cyst (*S. dubeyi*): 174 (95.1%) Macroscopic cysts (*S. fusiformis*): 48 (26.2%)	Muscle examination, histology	**1343**
	Ankara	1993–94	125	*S. fusiformis*: 49 (39.2%) *S. levinei*: 92 (73.6%) *S.* sp.: 39 (31.2%) Mixed: 58 (46.4%)	Trypsin digestion, bioassay (O and others)	**481**
Vietnam	Ho Chi Minh City	1996–97	502	396 (79%), of these *S. levinei* in 74%, *S. fusiformis* in 41%, *S. buffalonis* in 33%, and *S. dubeyi* in 12%; 8% were infected with all 4 species	Gross examination, histopathology	**864**
	Son La	2003	30	*S. fusiformis*, *S. cruzi*, *S. hominis* and *S. hirsuta*	Histology, PCR, TEM	**905**

D = diaphragm; H = heart; O = oesophagus; T = tongue; NS = not specified.

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Biochemistry: **754–758, 955, 1527, 1948, 1949, 1974, 2240, 2271**.

Molecular biology: **57, 492, 701, 864, 1056, 1340, 1846, 1856–1858**.

Epidemiology: **43, 106, 222, 223, 294, 312, 327, 481, 487, 932, 971, 1029, 1030, 1190, 1221, 1233, 1339, 1343, 1452, 1476, 1893, 1903, 1916, 1937, 1952, 1969, 1973, 1975, 2024, 2025, 2059, 2070, 2071, 2114, 2116, 2173, 2243, 2248, 2278, 2342, 2373**.

Experimental infections: **211, 213, 488, 881, 1059, 1429, 1809, 1811, 1847, 1849, 1893, 1951, 1971, 1972, 1974, 1979, 2026–2028, 2030, 2031, 2062, 2063, 2326, 2327**.

Morphology and speciation: **5, 163, 220, 326, 327, 394, 471, 474, 481, 651, 862, 863, 865, 937, 1058, 1369, 1438, 1808, 1810, 1846, 1848, 1867, 1871, 1878, 1886, 1887, 1889, 1890, 1903, 1973, 1975, 2029, 2059, 2065, 2130, 2272, 2343, 2344**.

Miscellaneous: **213, 326, 488**.

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CHAPTER 11

Sarcocystosis in Horses, Mules, and Donkeys (*Equus* spp.)

11.1 INTRODUCTION

There is considerable confusion concerning the validity of different species of *Sarcocystis* in horses and other equids.[833,1306,1481] Early records of *Sarcocystis* infections in horses have been reviewed.[1481] The name *Sarcocystis bertrami* was first proposed as a new species in the horse[329] in a book. Sarcocysts were 9–10 mm long and were found in the esophagus and leg muscles. No other information was given. In retrospect, it is impossible to determine the validity of the species because there are no archived specimens.

Dogs fed naturally infected esophagus from horses in Germany excreted sporocysts.[1481] The species was named *Sarcocystis equicanis* following the nomenclature proposed.[803] The sarcocysts were thin walled (Figure 11.1b).

The life cycle of a species of *Sarcocystis* in horses in the United States was determined and it was named *Sarcocystis fayeri* based on morphological features.[351] Sarcocysts were thick walled. The name *S. bertrami* was used[1136] for the parasite originally described as *S. equicanis*[1481] and its life cycle was further described.

Based on limited observations, the parasite in the muscles of a donkey was named *Sarcocystis asinus*.[650] The validity of *S. asinus* has been questioned,[1048,1306] and we regard it as invalid. Experimental evidence indicated the similarity of the *Sarcocystis* species in horses and donkeys.[1131] Tissues from 20 naturally infected horses were fed to a dog, and those of 10 donkeys were fed to another dog. Both dogs excreted sporocysts, but cats and raccoons fed the same infected tissues did not excrete sporocysts. Experimental infections were conducted with 4 ponies raised in captivity using the sporocysts derived from the infected donkeys (inoculum A) and sporocysts derived from the inocula from horses (inoculum B). A foal fed 100,000 sporocysts of inoculum A developed 2 peaks of fever on days 10 and 11, and 19–21 DPI. Sarcocysts were detected histologically in muscles of the foal when killed 138 DPI; sarcocysts contained only metrocytes. 3 foals were fed inoculum B. Two foals fed 100,000 sporocysts of inoculum B developed 2 peaks of fever between 10 and 21 DPI; these foals were killed 21, and 197 DPI. Mature sarcocysts were found in the foal killed 197 DPI, but no parasites were detected in the foal killed 21 DPI. The fourth foal fed only 10,000 sporocysts did not develop fever; mature sarcocysts were found in its muscles when killed 212 DPI. A dog fed muscles of the experimentally infected foal excreted sporocysts. Sporocysts were structurally similar in 3 dogs that excreted sporocysts; the sporocysts were 12.2–13.8 × 9.2–9.9 μm in size and the prepatent period was 9–10 days.[1131]

Review of the literature indicates there are 2 distinct species of *Sarcocystis*, the thick-walled species (*S. fayeri*) with an unique type 11a cyst wall, and the thin-walled species (*S. equicanis*/*S. bertrami*) with a type 11c cyst wall[552,1853,1306] (Figure 11.1). The identity of the original *S. bertrami* will never be known because there are no archived specimens. Therefore, to avoid further confusion, only 2 species are identified and discussed, *S. bertrami* and *S. fayeri*.

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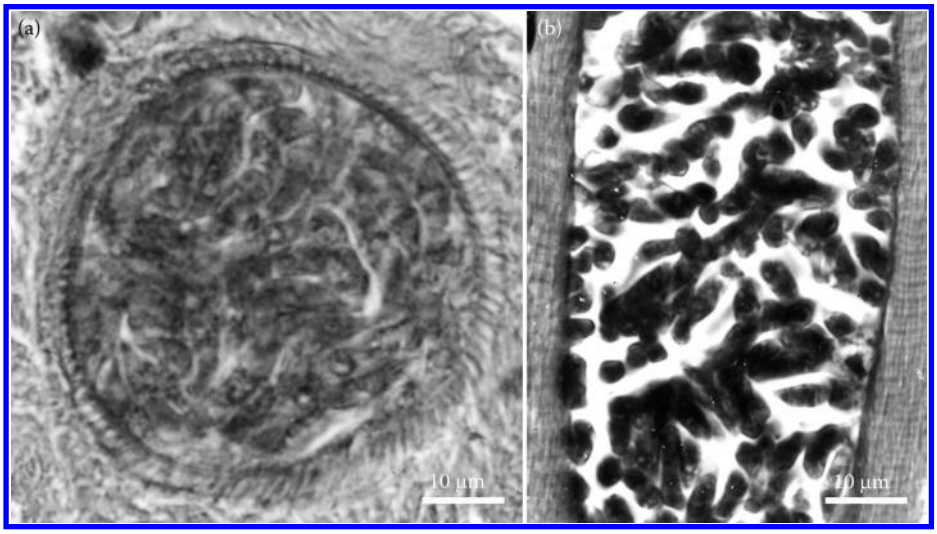


Figure 11.1 Sarcocysts of *S. fayeri* and *S. bertrami* in skeletal muscles. (a) *S. fayeri* from a horse in the United States. (b) *S. bertrami* from a horse in Germany. Note the thick, striated wall in *S. fayeri* and thin wall in *S. bertrami*. H and E stain.

11.2 *SARCOCYSTIS BERTRAMI* DOFLEIN 1901 (SYN. *S. EQUICANIS* ROMMEL AND GEISEL 1975)

Sarcocysts are up to 15 mm long.[53,552,833] By LM the thickness of the wall is variable but generally thin. By TEM, the cyst wall has villar protrusions that are up to 11 µm long and less than 0.5 µm wide. The vp are folded on the cyst wall giving it a thin-walled appearance, type 11c.[708,1306] Bradyzoites are 8–10 µm long and 2.5–3.5 µm wide.

The dog, but not the cat, raccoon or human, is the definitive host.[833] The prepatent period is 8–10 days. Sporocysts were 15–16.3 × 8.8–11.3 µm[552] and slightly smaller.[1131]

In experiments conducted in Germany, only mild clinical signs were noted in ponies. In 1 experiment, 5 ponies were each fed 200,000 sporocysts.[1562] One pony was febrile on day 12 and all ponies had fever 20–22 DPI; these 2 peaks of fever probably indicate the 2 schizogonic phases of the parasite. Three of the 5 ponies had neurological signs, apathy, and anorexia between 51 and 70 DPI. Muscle enzymes (creatinine kinase) values were elevated between 63 and 147 DPI. Sarcocysts were 2 mm long at 378 DPI and up to 9 mm long at 1040 DPI.[1136]

In another experiment, 2 ponies, each fed 100,000 sporocysts, remained asymptomatic[552]; these ponies were killed 167 and 189 DPI. Both thick-walled (*S. fayeri*) and thin-walled (*S. bertrami/S. equicanis*) sarcocysts were seen.

11.3 *SARCOCYSTIS FAYERI* DUBEY, STREITEL, STROMBERG, AND TOUSSANT 1977

Sarcocysts were found primarily in skeletal muscles and rarely in the heart. Sarcocysts in histologic sections of naturally infected horses were up to 990 µm long and 136 µm wide with radially striated cyst walls 1–3 µm thick.[351,1752] In experimentally infected ponies sarcocysts were up to 1 mm long at 79 DPI.[1539] The cyst wall has vp 2.2–3.1 µm long, with microtubules that extend from tip of vp to the zoite plasmalemma (Figure 3.10b), type 11a (Figures 11.1a and 11.2). Bradyzoites are

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Figure 11.2 Sarcocysts of *S. fayeri* in the horse. TEM of cross section showing microtubules of the vp extending into the interior of the sarcocyst. Bar = 5 μm. Inset shows a cyst seen by light microscopy. H and E stain. Bar = 10 μm. (From Tinling, S.P. et al. 1980. *J. Parasitol.* 66, 458–465. With permission.)

12–16 μm long in sections and 15–20 μm in smears. The dog (but not the cat) excreted sporocysts, 11–13 × 7–8.5 μm, with a prepatent period of 12–15 days.

Endogenous development was studied in 1 horse and 12 ponies. In the first experiment, 8 ponies and 1 horse were fed 10,000 to 10 million sporocysts and were necropsied 10–156 DPI.[585] Two generations of schizonts were found in arteries or capillaries of the heart, brain, and kidney between 10 and 25 DPI. Immature sarcocysts were seen from 55 DPI, and some sarcocysts at 77 DPI had

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Figure 11.3 A depressed pony with hair loss, 6 months after inoculation with *S. fayeri*. (From Fayer, R. et al. 1983. *Vet. Rec*. 113, 216–217. With permission.)

bradyzoites and were infectious for dogs. At 156 DPI, the sarcocysts were up to 338 µm long in histologic sections. In the second experiment, 3 ponies were fed 100,000 to 10 million sporocysts and necropsied 79 DPI;[1539] only immature sarcocysts were found in muscles.

The *Sarcocystis* species of equids are only mildly pathogenic. In 1 study, ponies fed 1 million sporocysts of *S. fayeri* developed mild anemia and fever. A horse fed 10 million sporocysts developed a stiff gait, but was otherwise clinically normal.[1651] There are indications that pathogenicity may vary among different isolates of *Sarcocystis* or among horses. For example, a pony fed 2 million sporocysts of a Texas isolate of *Sarcocystis* became lethargic, stiff-legged, tired quickly, and lost hair on its body, especially on its head and neck by 150 DPI (Figure 11.3). This pony had severe myositis associated with sarcocysts and developed an autoimmune anemia. In another study, 3 ponies fed 100,000 to 10 million sporocysts of the Texas isolate remained asymptomatic.[1539] Two cases of remarkable granulomatous and eosinophilic myositis associated with *S. fayeri* have been diagnosed in USA. At gross examination, disseminated granulomas were very patent. In 1 horse, refractory muscle pain and recurrent esophageal dysphagia were observed.[2394]

Recently, several outbreaks of food poisoning were reported after consumption of raw horse meat in Japan.[781,934] This has been related to a 15-kDa protein present in *S. fayeri* sarcocysts.[934] Freezing of horse meat seems to control the toxicity of *S. fayeri*[781] associated with raw meat consumption.

11.4 PREVALENCE OF NATURAL INFECTIONS AND CLINICAL DISEASE

Sarcocysts have been found in horses, mules, and donkeys (Table 11.1). All but 2[625,750] were based on samples collected from slaughterhouses and thus do not represent prevalence in well-kept

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Table 11.1 Prevalence of Sarcocysts in Equids

Location	Year	Tissues	Methods	Results	Species	Reference
Austria	1979–81	E	Histology, bioassay in dog, cat and human	Sarcocysts in 89 (32.4%) of 275 horses and 1 of 2 donkeys. Only dog shed sporocysts	*S. bertrami*	**833**
Belgium	NS	M, biopsy	Histology	Sarcocysts in 12 (13.2%) of 91 horses with chronic muscle problems	NS	**625**
Egypt, Giza	NS	H	Histology, TEM	Sarcocysts in 18 (90.0%) of 20 donkeys	NS, similar to those described in horses	**824**
Egypt, Giza	NS	E, D, H	Bioassay in dog	After prepatent period of 11 days, 4 dogs, fed a pool of donkey tissues (500 g), shed sporocysts	*S. bertrami*	**1873**
Germany, Munich	NS	E	Trypsin digestion, histology, bioassay in dog and cat	Bradyzoites in 21 (23.3%) of 90 horses. 3 dogs, but not 6 cats, shed sporocysts	*S. equicanis*	**1481**
Germany, Munich	1978–79	E, H, D	Trypsin digestion, histology, bioassay in dog	Bradyzoites in 31(15.5%) of 200 horses. No bradyzoites in heart digests Dogs shed sporocysts	*S. equicanis*, *S. fayeri*	**552**
Great Britain	1981	E	Trypsin digestion, muscle squash, TEM	Sarcocysts in 245 (62.2%) of 394 horses, and 1 of 2 donkeys	Sarcocysts thin walled, *S. bertrami*	**485**
Great Britain and Ireland	NS	Postural, propulsive and respiratory muscles	Histology	Sarcocysts in 3 (4.0%) of 74 horses	NS	**750**
India, Madras	NS	E, H, D, M	Muscle squash, histology	Sarcocysts in 1 pony; no. tested not stated	NS	**15**
India, Punjab	NS	E, D	Muscle squash, bioassay in dog	Sarcocysts in 1 mare; no. tested not stated. Dog shed sporocysts 7 DPI	NS	**922**

(*Continued*)

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Table 11.1 (*Continued*) Prevalence of Sarcocysts in Equids

Location	Year	Tissues	Methods	Results	Species	Reference
India, Punjab	NS	E, D, T	Muscle squash, histology	Sarcocysts in 1 mare; no. tested not stated	NS	**924**
Japan	1981–93	E, D, T, H, M	Trypsin digestion, histology, SEM, TEM	Thin-walled sarcocysts in 6 (6.4%) of 93 horses	*S. equicanis*	**1853**
Mongolia	1998–99	D, H, T	Trichinoscopy	Sarcocysts in 3 of 4 horses	NS	**640**
Mongolia, Ulaan Baatar	1998	D, H, T	Trichinoscopy	Sarcocysts in 40 (93.0%) of 43 horses	NS	**641**
Morocco	NS	E	Trichinoscopy, histology	Sarcocysts in 55 (46.2%) of 119 horses, 9 (50.0%) of 18 mules, 9 (21.9%) of 41 donkeys	NS	**965**
Turkey, Ankara	NS	E, D	Gross examination, muscle squash, trypsin digestion	Sarcocystis in 22 (68.8%) of 32 horses, and 60 (84.5%) of 71 donkeys	*S. equicanis*, *S. fayeri*, and mixed infections in both hosts	**1344**
USA: Ohio, Indiana and Wisconsin	1976	E, D, H	Trypsin digestion, histology, bioassay	Sarcocysts in H and E sections of 4 esophagi, and 2 hearts of 97 horses. Bradyzoites found in digestion of 17 (17.5%) of 97 horses 10 dogs, but not 10 cats, shed sporocysts	*S. fayeri*	**351**

E = esophagus; D = diaphragm; H = heart; T = tongue; M = skeletal muscle.
NS= not stated.

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companion horses. In 36 thoroughbred horses, both types of myocytes (dark, light) were infected.[750] Infections are more prevalent in older horses.[485,1481] The finding of mature sarcocysts in the heart of a 3-day-old foal from Canada[269] and in a 6-week-old foal in Britain[485] indicates transplacental infection.

Clinical myositis has been reported to be associated with the presence of sarcocysts in histologic sections of horses in Germany,[631,1556] in a mule in Israel,[1702] in a malnourished horse in Canada,[178] in an ataxic horse in California,[1752] and in a horse with pronounced muscle wasting in Colorado,[1760] both in the United States. In a controlled study of 91 horses with clinical myositis in Belgium, sarcocysts were found in 12 muscle biopsies, but there was no association between the presence of sarcocysts and clinical myositis.[625] Eosinophilic myositis in a horse from New Zealand is illustrated in Figure 11.4.

There are only few reports of *Sarcocystis* in wild Equidae. These include *Sarcocystis neurona* infection in a Grant's zebra (*Equus burchelli bohmi*) from the United States[1117] (see Chapter 3), and a report by Odening et al.[1306] who detected TEM wall type 11 sarcocysts in 5 species of wild equids (Przewalski's feral horse, *Equus caballus przewalskii*; Chapman's plain zebra, *Equus burchellii chapmani*; Damara plain zebra, *Equus burchellii antiquorum*; kulan, *Equus onager kulan*; and kiang, *Equus kiang holdereri*) in zoological gardens in Germany. A dog fed

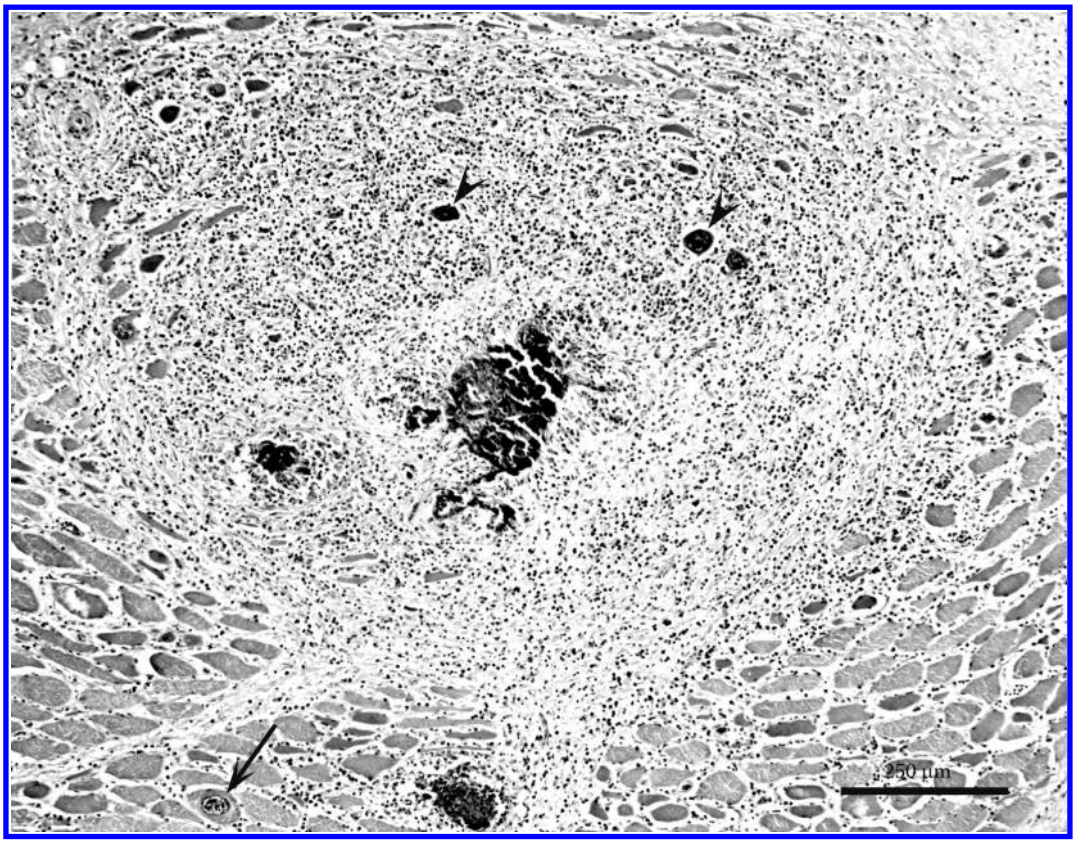


Figure 11.4 Eosinophilic myositis in skeletal muscle of a naturally infected horse. The central mineralized area is surrounded by numerous eosinophils and fibroblasts. A few giant cells (arrowheads) are present. Cross section of a sarcocyst (arrow) is in the lower left area. H and E stain. (Courtesy of late W. J. Hartley.)

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Sarcocystis species from mountain zebra (*Equus zebra hartmannae*) from South Africa excreted sporocysts.[2145]

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CHAPTER 12

Sarcocystosis in Camels (*Camelus dromedarius* and *Camelus bactrianus*)

12.1 INTRODUCTION

In Egypt, Mason[1125] first reported sarcocysts in striated muscles, including the heart, of a camel. He saw 2 types of sarcocysts, 1 with a striated wall 1–2 μm thick, and the other with a smooth, thin wall (<1 μm). Mason thought that they were both different stages of the same parasite and named them *Sarcocystis cameli*. Therefore, in retrospect, it is not possible to decide which type of sarcocyst is *S. cameli*. Because the thick-walled striated sarcocyst has been found repeatedly, Dubey et al.[393] called it *S. cameli* (Figure 12.1) and did not name the thin-walled species, which was subsequently named *Sarcocystis ippeni* by Odening.[1314] Another unnamed species of *Sarcocystis* with hirsute protrusions on the sarcocyst wall was seen occasionally in *Camelus bactrianus* born in the zoo, but additional details were not provided.[1314]

Macroscopic sarcocysts (up to 15 mm long) were reported in camels from Russia[990] and Egypt[2399] but structural details are missing. Surveys of *Sarcocystis* in camels have been summarized in Table 12.1.

The presence of thick and thin-walled sarcocysts was confirmed in camels from Saudi Arabia,[568] Somalia,[770] and Sudan.[876] Ishag et al.[875] found sporocysts of 2 sizes (13.2–13.6 × 6.5–9.5 and 16.0 × 9.9–11.5 μm) in feces of dogs fed camel meat; the larger sporocyst was called *Sarcocystis camelocanis*.

Thick-walled sarcocysts from camels were transmitted to dogs and named *S. camelicanis* without any explanation for this new name.[11] It is evident from data in Table 12.2, that the size of sporocysts is variable and species should not be based on sporocyst size alone. To add to this confusion, another new species from the camel was named *Sarcocystis miescheri*, based on finding oocysts in feces of dogs fed naturally infected camel meat.[1106] Illustrations provided by the authors resemble *Cystoisospora ohioensis* oocysts measuring 20.8–26.7 × 18.5–20.7 μm with a thick wall containing 2 sporoblasts and bearing no resemblance to other species of *Sarcocystis*. The bradyzoites measuring 21.5–32.8 × 7.7–17.7 μm appear to be artifacts misidentified as bradyzoites.

Recently, Dubey et al.[2391] reviewed all reports of sarcocystosis in camels and concluded that there are 2 structurally distinct species of *Sarcocystis* (both with microscopic sarcocysts), *S. cameli*, and *S. ippeni*. The status of species forming macroscopic sarcocysts is unclear.

The first molecular characterization of *S. cameli* was reported by Motamedi et al.[1230] who developed a restriction map for the species. This information will be useful for further speciation studies.

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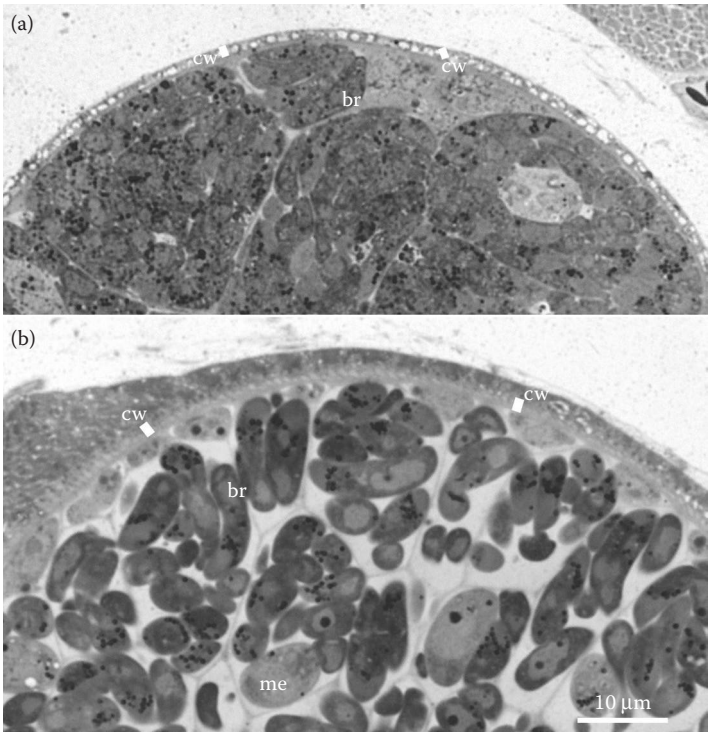


Figure 12.1 LM of *Sarcocystis* from camels; they are difficult to speciate based on light microscopy. Toluidine blue stain. (a) *S. cameli* sarcocyst. Vp are very thin and barely visible, and whitish areas are probably degenerated host tissue (hc) between vp. (b) *S. ippeni* based on tringular vp. (a, b) Note pale metrocytes (me) and banana shaped bradyzoites (br).

12.2 *SARCOCYSTIS CAMELI* (MASON 1910) AMENDED DUBEY, HILALI, VAN WILPE, CALERO-BERNAL, VERMA, AND ABBAS 2015 (SYN. *S. CAMELICANIS, S. CAMELOCANIS, S. MIESCHERI*)

12.2.1 Distribution

Egypt, Iran, Sudan, Afghanistan, Morocco, and the former USSR.

12.2.2 Definitive Host

Most likely dog.

12.2.3 Structure and Life Cycle

The sarcocysts found in striated muscles including the tongue, esophagus, and heart are microscopic. By LM, the sarcocyst wall appears thin (Figure 12.1a). The vp are conical, sloping to straight, and 3 µm long and 0.5 µm wide at the base, with knob-like structures on villar pvm, type 9j (Figure 12.2a). The bradyzoites are 14–15 × 3–4 µm long in histologic sections.

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Table 12.1 Prevalence of *Sarcocystis* Sarcocysts in Camels

Country	Year	No	Method	No. Positive	% Positive	Thick Walled	Thin Walled	Bioassay in Dog and Cat	Reference
Afghanistan	1984	192	Muscle squash, histology	118	61.4	Yes	–	NS	**966**
Egypt	NS	112	Muscle squash, histology	41	36.6	Yes	–	Dog and cat	**822**
	NS	13	Histology	3	23.1	NS	NS	NS	**530**
	2008	180	Muscle squash, histology, trypsin digestion	116	64.0	Yes	–	Dog and cat	**11**
	2009–10	156	Muscle squash	66	42.3	Yes	–	Dog	**1106**
	NS	180	Gross examination, trypsin digestion	120	66.6	–	Yes	Dog and cat	**2399**
Ethiopia	1998–99	121	Histology	55	45.5	–	Yes	NS	**1836**
India	NS	1	Histology	1	–	NS	NS	NS	**1447**
Iran	NS	400	Muscle squash	209	52.3	NS	NS	NS	**1600**
	2002–05	250	Histology	209	83.6	–	Yes	No	**1772**
	2009	130	Pepsin digestion	67	51.5	NS	NS	NS	**774**
Iraq	1992–96	36	Pepsin digestion	33	91.6	NS	NS	No	**1030**
Jordan	NS	110	Muscle squash	24	21.8	Yes	Yes	NS	**1031**
Mongolia	1998–99	5	Muscle squash	5	100.0	NS	NS	NS	**640**
Saudi Arabia	1992–93	103	Trypsin digestion	91	88.3	Yes	Yes, more common	Dog and cat	**568**
	NS	40	Histology	31	77.5	–	Yes	–	**2401**
	2002–03	624	Trypsin digestion	399	64.0	–	Yes	NS	**25**
Somalia	1987	200	Trypsin digestion	165	82.5	Yes	Yes	NS	**770**
			Histology	120	60.0				
Sudan	NS	100	Pepsin digestion	81	81.0	NS	NS	NS	**868**
	NS	NS	Bioassay	NS	NS	Yes	Yes	Dog	**876**
Former USSR (Russia)	NS	NS	Unknown	6	NS	Yes	Yes	Dog	**990**

NS = not stated.

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Table 12.2 Shedding of Sporocysts in Feces of Dogs Fed Camel Meat

Country	Prepatent Period (Days)	Size of Sporocysts (μm)	Reference
Egypt	Not stated	12 × 9	**822**
Egypt	10–11	12.0–14.0 × 8.9–11.3	**825**
Egypt	11	13.7–15.6 × 7.8–10.7 ($n = 50$)	**11**
Egypt	13–15	10.1–13.9 × 8.59–9.94 (type A) 8.7–14.3 × 11.5–10.0 (type B)	**1106**
Egypt	11	13.1–15.2 × 7.4–10.1	**2399**
Russia	Not stated	16.4 × 8.3	**990**
Saudi Arabia	9–10	10.7–14.3 × 8.3–10.7 ($n = 20$)	**568, 826**
Sudan	9–13	13.2–13.6 × 6.5–9.5 (type A) 16.0 × 9.9–11.5 (type B)	**876**

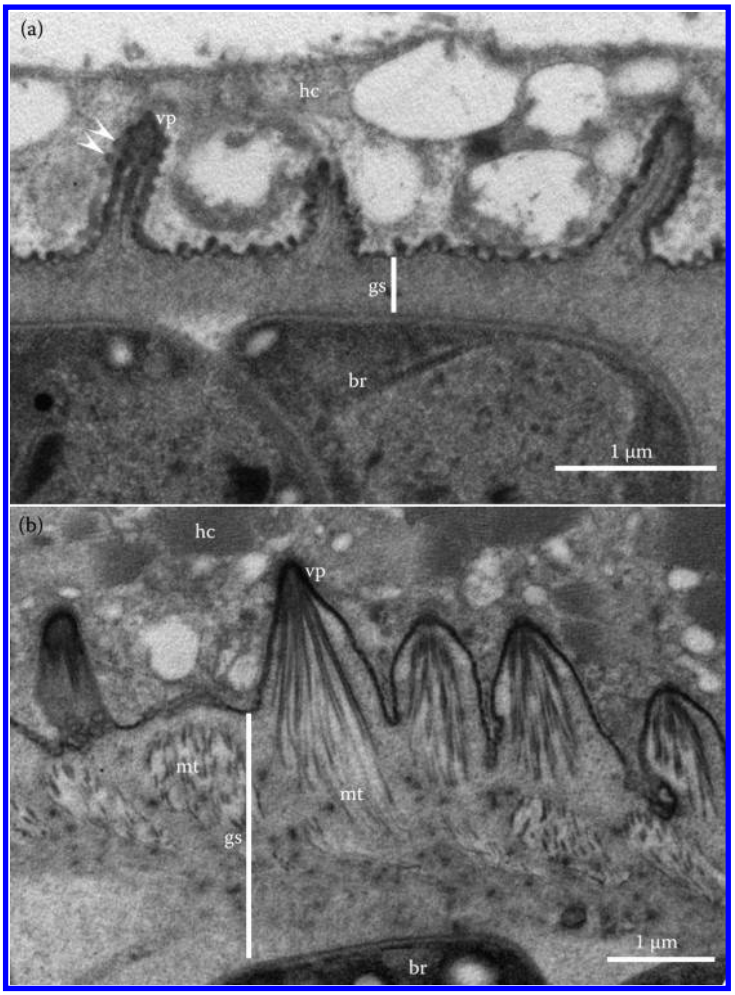


Figure 12.2 (a) TEM of *S. cameli* sarcocyst walls. The vp are interspersed with vacuolated (degenerated) hc. Note corrugations (arrowheads) at the villar tips and prominant mt. (b) TEM of *S. ippeni* sarcocyst walls. Note the vp are cut at different angles with prominent mt in conical vp. The gs is mostly electron lucent and not well demarcated. The mt in vp are more electron dense towards the villar tips and blunt tip of vp.

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12.3 *SARCOCYSTIS IPPENI* (ODENING 1997) AMENDED DUBEY, HILALI, VAN WILPE, CALERO-BERNAL, VERMA, AND ABBAS 2015

12.3.1 Structure and Life Cycle

Sarcocysts are microscopic (Figure 12.1b). By TEM, sarcocyst wall has unique type 32 vp with an electron dense knob (Figure 12.2b). The vp are approximately 1.0 μm long, 1.2 μm wide at the base, and 0.25 μm at the tip; microtubules in vp originate at midpoint of gs and continue up to tip, criss-crossed, smooth and without granules or dense areas. The total thickness of the sarcocyst wall (from the base of gs to vp tip) is 2.3–3.0 μm. Bradyzoites are 12.0–13.5 × 2.0–3.0 μm in size.[2391]

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CHAPTER 13

Sarcocystosis in South American Camelids (Alpaca, Guanaco, Llama, and Vicugna/Vicuña)

13.1 INTRODUCTION

A sarcocyst observed in a llama (*Lama glama*) was named *Sarcocystis aucheniae* but no other detail was provided.[149] In Argentina, sarcocysts found in a guanaco, *Lama guanicoe*, were named *Sarcocystis tilopodi* based on having found it in a different species of *Lama*.[1435] In Chile, macroscopic and microscopic sarcocysts were found in *L. guanicoe* which transmitted infection to dogs.[722] Four dogs fed sarcocysts excreted sporocysts but 4 cats fed the same infected meat did not excrete sporocysts. Additionally, 1 dog fed isolated macrocysts also excreted sporocysts. Based on these results, the author suggested that the parasite in *L. glama* should be named *S. lamacanis* and the parasite in the species in *L. guanicoe* should be called *S. guanicoe-canis*.[1435] In Germany, both microscopic and macroscopic sarcocysts were found in *L. glama*.[1561] 1 dog and 1 cat were fed isolated macroscopic sarcocysts; only the dog excreted oocysts. Moreover, another cat fed infected meat also did not excrete sporocysts. These experiments confirmed the findings that sarcocysts from llamas are transmissible to dogs but not cats.[722] The idea that 2 species of *Sarcocystis* (*S. aucheniae* and " *Sarcocystis lamacenis*," possibly *S. lamacanis*) infect alpacas was supported by others.[1043,1127]

Critical examination of the taxonomy of the *Sarcocystis* species led to the conclusion that there is only 1 valid name, *S. aucheniae*, and that there is no valid reason to separate species infecting llamas and alpacas (*Vicugna pacos*).[1317] Because the sarcocyst that Brumpt first recognized was macroscopic it seems reasonable to designate macroscopic species as *S. aucheniae.* A new name can be proposed for the microscopic species when its structure is described. Some authors have called the microscopic sarcocyst *S. lamacanis*,[649] others have called it *S. lamacensis*.[1490] There is no morphological description of the microcyst sarcocyst of the llama. The closest description is an illustration of the microcyst in a histologic section of the heart of a llama in figure 1 from Reference **393** reproduced here (Figure 13.1). Therefore, the names *S. guanicoe-canis, S. lamacanis, S. lamacensis* should be *nomina dubia*.

In the first morphological description of the macroscopic sarcocyst in llamas, the parasitized myocyte is located in a vacuole.[1561] By light microscopy, the sarcocyst wall is thin. By TEM, the sarcocyst wall is type 21b with highly branched cauliflower-like vp. The dog, and not the cat, is the definitive host for *S. aucheniae*.[164] Macroscopic sarcocysts, appearing like rice grains, 3–5 mm long, were isolated from the meat of 3 naturally infected llamas in the Andean flatlands region of Argentina. DNA was obtained from each of 6 clean sarcocysts that were 17.3–33.1 mg and contained more than 10 million bradyzoites that were 17.6 µm long. Full-length 18S rRNA gene sequences from individual sarcocysts had 99.1%–100% similarity, indicating a cloned parasite. In comparison, the Australian isolate of *S. aucheniae* from *V. pacos* had 96% similarity. Whether these differences were related to methods used to compare the Australian or Argentinian isolates or were related to different *Sarcocystis* species could not be determined. Based on short sequences published for the "micro-cyst producing species

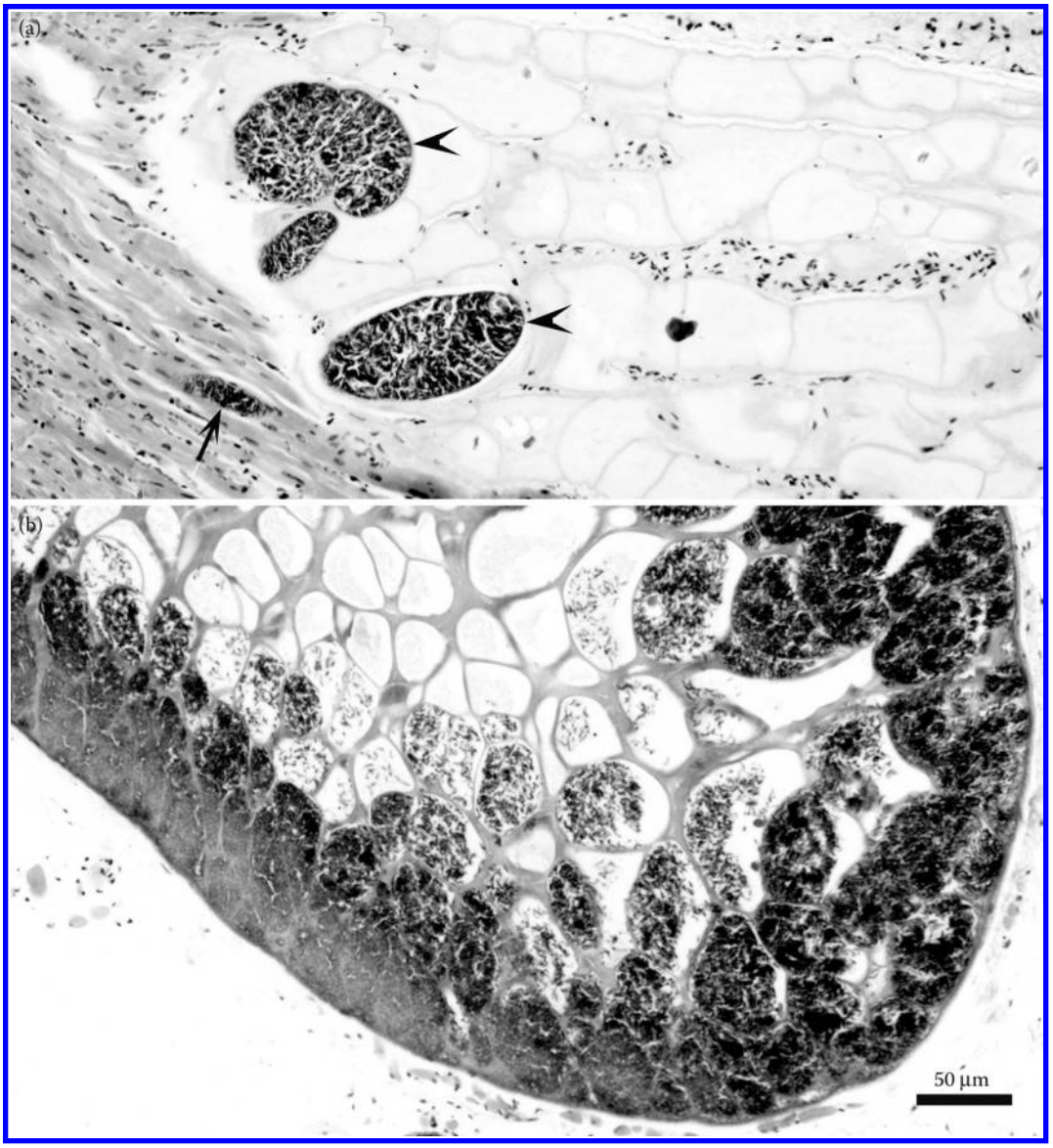


Figure 13.1 Sarcocysts in tissues of llamas (*Lama glama*). (a) Microscopic sarcocysts of an unnamed species in myocytes (arrow) and in Purkinje fibers (arrowheads) in the myocardium. (b) A macroscopic *S. aucheniae* sarcocyst in the esophagus. Note the prominent septa, zoites at the periphery, and empty areas in the center without zoites. H and E stain.

S lamacanis" 18S rRNA gene,[1162,1163] it was published and concluded that there were enough phylogenetic differences such that *S. aucheniae* should be considered a separate species.

13.2 CLINICAL AND SUBCLINICAL SARCOCYSTOSIS IN LLAMA AND ALPACA

Llamas are important to the economy of several South American countries. Little is known of clinical sarcocystosis in llamas. Severe myositis was diagnosed in a 6-year old female llama

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imported to the United States from Peru.[1010] Necrosis, hemorrhage, and eosinophilic myositis were associated with macroscopic sarcocysts; schizonts were not found. The llama aborted an 8-month-old fetus but parasites were not found in the placenta or in the fetal tissues.

Severe myositis was reported in an alpaca born in Australia.[649] This alpaca had numerous nodules and subcutaneous abscesses on head and neck. Examination of needle biopsy tissue revealed infiltration with leukocytes, predominantly eosinophils. Because of its deteriorating condition the alpaca was euthanized. At necropsy multiple caseating, pale white streaks were found in the head and neck area. Histologically, the lesions resembled eosinophilic myositis similar to that discussed in Chapter 1. Sarcocysts were found in the lesions. The authors indicated that the sarcocysts were ultrastructurally distinct from those reported in llamas but details were insufficient.

Sarcocysts are common in asymptomatic llamas and other similar camelidae (Table 13.1)[129,710,1043] and result in the down grading of carcasses at meat inspection. In an extensive survey of llamas in local abattoirs from 2006 to 2011 data, 1196 slaughtered llamas in Bolivia were analyzed with respect to *Sarcocystis* infection,[1490] and the following conclusions were drawn: (1) Yearly prevalence of sarcocysts varied from 23.4% to 50.3% and infection rates were higher in females and in older animals. (2) Prevalence in younger llamas (2.4 years) was 19.9% (96 of 482) versus 90.4% (160 of 177) in animals older than 4.5 years. (3) Down grading of *Sarcocystis*-carcasses resulted in between 13% and 20% economic loss to the farmer.

13.3 OTHER ASPECTS ON SARCOCYSTOSIS IN SOUTH AMERICAN CAMELIDS

13.3.1 Experimental Infections

Host specificity is discussed for the *Sarcocystis* spp. mentioned in the literature; thus, experimental infections have been carried out in different hosts: 1 investigation erroneously searched for sarcocysts in dogs, mice, and rats after feeding with guanaco meat but did not search for sporocysts in feces.[1435] Dogs, cats, rats, and mice were fed guanaco meat containing sarcocysts, but only dogs excreted sporocysts.[722] This study also tested the negative effect of temperature (cooking and freezing) on the infectivity of such sarcocysts for dogs. In Peru, macrocysts (considered *S. aucheniae*) of 2 different sizes (>5 and 1–3 mm) were fed to dogs.[254] Dogs that ingested the smaller sarcocysts excreted more sporocysts, possibly due to higher viability of bradyzoites in younger cysts.

Sixty-three young alpacas (5 months old) were infected with sporocysts of "*S. lamacanis*" obtained from dogs fed myocardium of alpacas containing *Sarcocystis* microcysts.[200] After feeding 1000, 2500, and 5000 sporocysts, 92.3% of the alpacas fed 5000 sporocysts died between 30 and 90 days later. However, there were no data on lesions or the stage of *Sarcocystis* (schizonts/sarcocysts) and no strong correlation between infection and loss of weight or wool production.

Five dogs were infected with 400 macrocysts obtained from naturally infected alpacas.[1866] Large numbers of oocysts of *S. aucheniae* were retrieved from the mucosa intestinalis of all dogs. Also, asexual stages of *S. aucheniae* have been cultured in different cell lines.[1769]

Experimental infections of alpacas with "*S. lamacanis*" in Peru studied the possible influence of sarcocysts located in the myocardium on electrocardiographical parameters.[129] After histological examination, sarcocysts were detected only in myocites and not in Purkinje cells.

13.3.2 Food Safety

The importance of gastrointestinal symptoms resulting from the ingestion of infected meat, due to the toxin present in the sarcocysts was highlighted.[1043] The effects of *S. aucheniae* sarcocystine toxin and its immunogenic activity were tested in rabbits.[215] Several experiments have been carried out on the control of toxic risks derived from ingesting meats containing *Sarcocystis* sarcocysts.

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Table 13.1 Prevalence of *Sarcocystis* Sarcocysts in South American Camelids

Country	Year	Host	No	Method	No. Positive	% Positive	Microscopic cysts	Macroscopic cysts	Bioassay in dog and cat	Species	References
Argentina	1966–68	Guanaco[a]	119	Gross examination, squeezing, histology	48	40.3%	NS	Yes	Dogs, mice, rats	*S. tilopodi*	**1435**
Argentina, Patagonia	2000	Guanaco[a]	12	Gross examination, histology	8	66.7	No	Yes (T, H, M)	–	NS	**90**
Argentina, Jujuy	2005–07	Llama[b]	308	IFAT	295	96.0% (92.5%: *S. cruzi* antigen; 77.0%: *S. aucheniae* antigen)	–	–	–	NS	**1214**
Bolivia	2006–11	Llama	1196	Gross examination	408	34.1%	Macroscopic	No	–	–	**1490**
Peru	NS	Alpaca[c]	200	Gross examination, histology	200	100.0%	Yes (E: 99.5%; M: 95.5%; neck: 87.5%)	Yes (E: 16.5%)	–	NS	**748**
Peru	NS	Alpaca[c]	15	ELISA, PCR	15	100.0%	Yes	Yes	–	By sequencing of 18S rRNA gene: *S. aucheniae* and *S. lamacanis*	**1162**
Peru, Junin	NS	Alpaca[c]	941	ELISA	844	89.7%	–	–	–	–	**169**

Hosts: [a] *Lama guanicoe*, [b] *Lama glama*, [c] *Vicugna pacos*.
Tissues: E = esophagus, H = heart, M = skeletal muscle, T = tongue. NS = not stated.

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Four different physical treatments for *S. aucheniae* infected meat included boiling, baking, frying, and freezing.[710] Treated meats were not toxic or infectious for rabbits and dogs, respectively. Different methods of curing and smoking failed to detoxify alpaca meat.[1792]

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Molecular biology: **164, 1162, 1163**.
Morphology and speciation: **149, 393, 1317, 1435, 1561**.
Pathology and experimental infections: **129, 200, 254, 649, 722, 1010, 1477, 1769, 1866**.

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CHAPTER 14

Sarcocystosis in Dogs (*Canis familiaris*)

14.1 MUSCULAR SARCOCYSTOSIS

Until 2005, there were few reports of sarcocysts in dogs and these were considered incidental (Table 14.1). Recently, severe myositis was diagnosed in 4 dogs and 2 species, *Sarcocystis caninum*, and *Sarcocystis svanai* were named.[476]

14.1.1 *Sarcocystis caninum* Dubey, Sykes, Shelton, Sharp, Verma, Calero-Bernal, Viviano, Sundar, Khan, and Grigg 2015

14.1.1.1 Structure and Life Cycle

Sarcocysts are microscopic, up to 1.2 mm long; the sarcocyst wall is 1–2 μm thick and is type 9c (Figure 14.1a). By TEM, vp are approximately 1 μm long, lacking microtubules. Bradyzoites are 6.0–7.5 × 2–3 μm in size. The life cycle is unknown.

14.1.2 *Sarcocystis svanai* Dubey, Sykes, Shelton, Sharp, Verma, Calero-Bernal, Viviano, Sundar, Khan, and Grigg 2015

14.1.2.1 Structure and Life Cycle

The sarcocyst wall is <0.5 μm thick and type 1a. Bradyzoites contain up to 4 rhoptries (Figure 14.1b).

14.2 CLINICAL DISEASE

Four (case numbers 5–8, Table 14.1) unrelated dogs (from Canada and the United States) had fever, apathy, anorexia, muscle weakness, ataxia, and elevated liver and muscle enzymes. One dog had dysphagia. Initially, the dogs were suspected to have neosporosis. Examination of muscle biopsies revealed severe myositis associated with numerous sarcocysts (Figure 14.2). Treatment with clindamycin was seemingly effective.

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Table 14.1 Details of Dogs with Muscular Sarcocystosis

Case No.	Year	Location	Breed	Age	Sex	Main Signs	Location of Sarcocysts	TEM	References
1	1964–65	Madhya Pradesh, India	Not known	Not known	Not known	Not known	Esophagus	No	**1509**
2	Not stated	Georgia, USA	Doberman pinscher	2 yr	Female	Adenocarcinoma	Heart	No	**828**
3	Not stated	Alabama, USA	Not known	4 yr	Male	Ataxia	Leg, biceps	Yes, irregularly spaced vp	**107**
4	1991	Kenya	German shepherd	Adult	Female	Adenocarcinoma	Heart	No	**159**
5	2003	British Columbia, Canada	Labrador cross	5 yr	Male	[a]	Shoulder, biceps	Yes, *S. caninum*	**196, 476**
6	2010	Cañon City, Colorado, USA	Golden retriever	11 yr	Male	[b]	Digital flexor	Yes, *S. caninum* + *S. svanai*	**476, 1701**
7	2010	Rural Montana, USA	Rottweiler	4 yr	Female	[c]	Biceps, triceps		**476, 1701**
8	2012	British Columbia, Canada	Rhodesian ridgeback	1.5 yr	Female	[d]	Quadriceps	Yes, *S. caninum*	**476**

[a] Lethargy, anorexia, fever, ataxia, inability to walk, dysphagia.
[b] Lethargy, ptyalism, panting, anorexia, ataxia, diarrhea then stiff gait and generalized muscle pain 28 days after the onset of illness.
[c] Lethargy, ptyalism, panting, anorexia, diarrhea then stiff gait and generalized muscle pain 28 days after the onset of illness.
[d] Anorexia, diarrhea, head tremors, fever, muscle weakness, ataxia.

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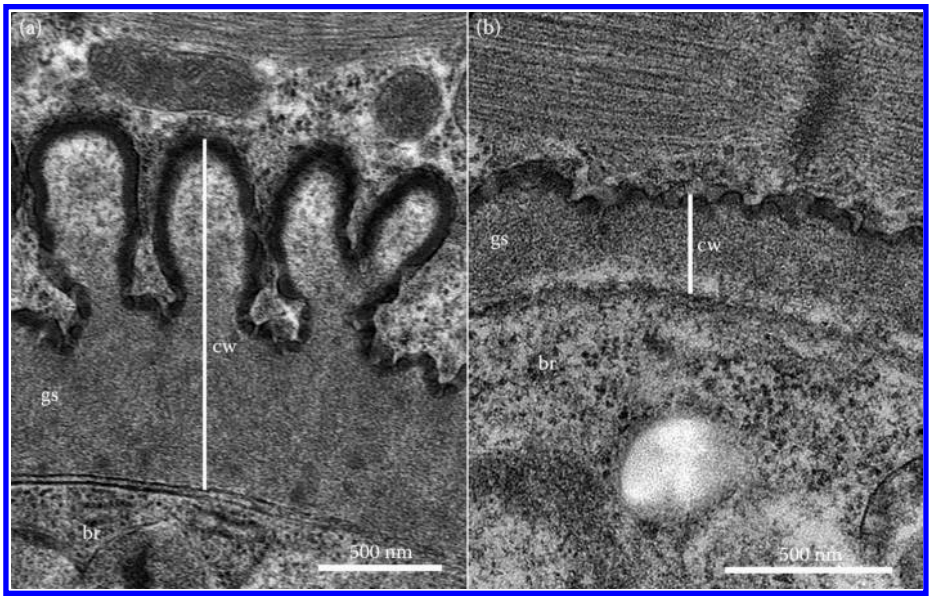


Figure 14.1 Comparison of the sarcocyst walls of two species of *Sarcocystis*. (a) *S. caninum* and (b) *S. svanai*. The cw of *S. caninum* is more than twice as thick as that of *S. svanai.* (Adapted from Dubey, J.P. et al., 2015. *J. Eukaryot. Microbiol*. 62, 307–317.)

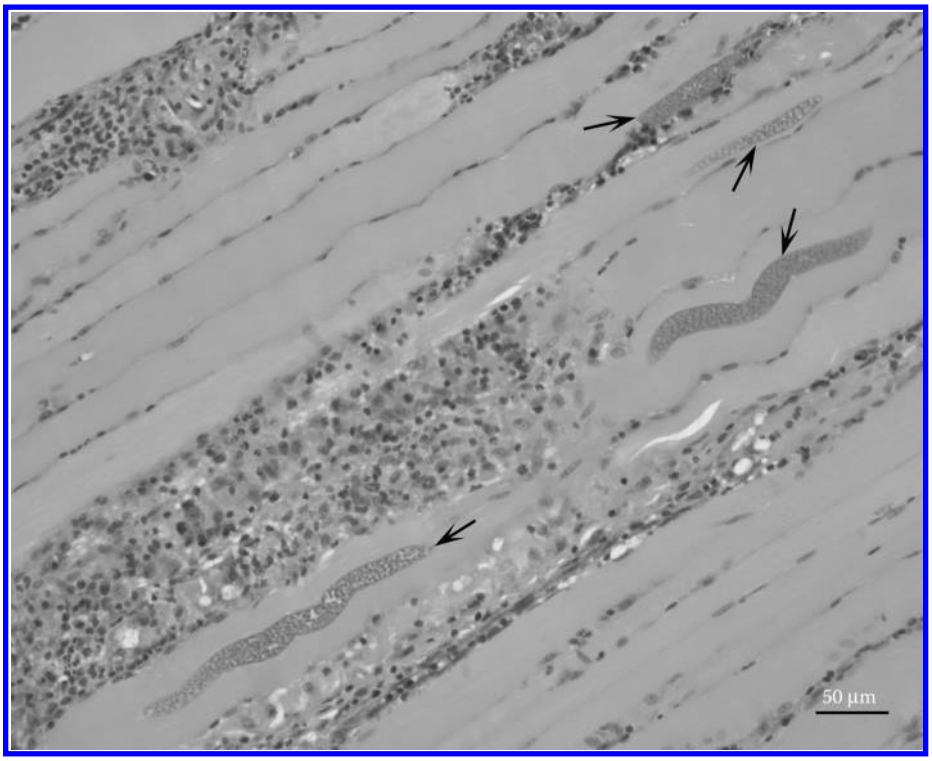


Figure 14.2 Severe myositis in a naturally infected dog. Note 4 elongated immature sarcocysts (arrows). H and E stain. (Adapted from Chapman, J., Mense, M., Dubey, J.P., 2005. *J. Parasitol*. 91, 187–190.)

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Table 14.2 ***Sarcocystis*** **Species Sporocysts in Feces of Dogs**

Species	Intermediate Host	Size of Sporocysts (μm)
S. arieticanis	Sheep	15–16.5 × 9.8–10.5
S. tenella	Sheep	14–15 × 9–10.5
S. mihoensis	Sheep	15–16 × 8–9
S. cruzi	Cattle	14.5–17 × 9–11
S. capracanis	Goat	12–16 × 9–11
S. hircicanis	Goat	15.0–17.3 × 10.5–11.3
S. bertrami	Horse	11–14.4 × 8–10.1
S. equicanis	Horse	15–16.3 × 8.8–11.3
S. fayeri	Horse	11–13 × 7.0–8.5
S. miescheriana	Pig	12.7 × 10.1 (12.2–13.2 × 9.8–10.4)
S. poephagicanis	Yak	15.4–27.8 × 14.2–25.2 (oocysts)
S. alceslatrans	Moose	14–17 × 8.5–11.5
S. wapiti	Wapiti-elk	15.7–17.4 × 9.6–11.9
S. sybillensis	Wapiti-elk	15–17 × 10.5–12.0
S. cervicanis	Wapiti-elk	15.5–16.5 × 10.5–11.3
S. grueneri	Reindeer and wapiti-elk	12.4–15.7 × 9.2–11.2
S. capreolicanis	Roe deer	12.0–18.0 × 9.0–11.4
S. hemionilatrantis	Mule deer	14–16 × 9.5–11
S. odocoileocanis	White-tailed deer	13.2–15.7 × 8.8–12.1
S. levinei	Water buffalo	9.5–10.5 × 14.0–16.5
S. cameli	Camel	12.0–14.0 × 8.9–11.3
S. gracilis (?)	Roe deer	12–18 × 9–11.4
S. sp.	Grant's gazelle	10.8–15.6 × 8.4–12.0
S. sp.	Chicken	11–12.2 × 8.5–10
S. sp.	Pheasant	13–15 × 9–11
S. sp.	Fallow deer	15.4 × 8.8
S. sp. (*S. aucheniae*)	Llama	14.6 × 10.6

14.3 INTESTINAL SARCOCYSTOSIS

The dog is a major definitive host for many *Sarcocystis* species (Table 14.2). Sexual stages of those species have been tested in experimental infections by feeding parasitized tissues to coccidia-free dogs (and other canids).[11,383,392,585] *Sarcocystis* sporocysts are excreted in the feces in high number and generally for a sustained period; several reports have shown the role of domestic and stray dogs in excreting sporocysts.[80,451,880,1060,1265,1281,1506,1543,1570,1594,1612,1686,1783]

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CHAPTER 15

Sarcocystosis in Cats (*Felis catus*)

Cats are both intermediate and definitive hosts for *Sarcocystis*.

15.1 MUSCULAR SARCOCYSTOSIS

There are several reports of sarcocysts in cats, all except 1 were from the United States (Table 15.1). The presence of sarcocysts in cats is considered incidental. Only 1 morphologic type, *Sarcocystis felis*, has been found.

15.1.1 *Sarcocystis felis* Dubey, Hamir, Kirkpatrick, Todd, and Rupprecht 1992

15.1.1.1 Structure and Life Cycle

Sarcocysts were up to 22 mm long and up to 150 µm wide.[407] Viewed by LM, the sarcocyst wall has minute protrusions with a hobnail appearance (Figure 15.1). Ultrastructurally, vp are up to

Table 15.1 Reports of Muscular Sarcocystosis in Cats

Country	No. of Cats	Remarks	Location of Sarcocysts	TEM	References
USA, Illinois	4	4 of 10 asymptomatic cats killed after termination of unrelated study	Sarcocysts in skeletal muscle of 3, and heart of 1	Yes	**561**
USA	3	5-, 5-, and 10-yr-old, hemangioendothelioma, cardiomyopathy, adenocarcinoma	Skeletal muscle, heart, abdominal muscle	Yes	**963**
USA, Texas	1	1.5-yr-old, lymphosarcoma	Sarcocysts in muscles	Yes	**486**
USA, Georgia	2	5-yr-old, lymphosarcoma, 5-yr-old leukopenia	Sarcocysts in skeletal muscle of 1 and heart of the other	Yes	**828**
USA	2	Stray cats	Sarcocysts in leg muscles of 2 cats	Yes	**607**
Chile, Valdivia	1	Domestic cats	Sarcocysts in 1 of 24 diaphragms	No	**1757**
USA, Florida	5	5 of 50 cats from animal shelter	Sarcocysts in sections of muscles of 5, up to 20 mm long. Molecular characterization	Yes	**684**
USA	3	4 yr with neurological signs, 2 yr with undefined illness, 3 yr with sudden death	Skeletal muscle, heart. Molecular characterization	Yes	**521, 519**

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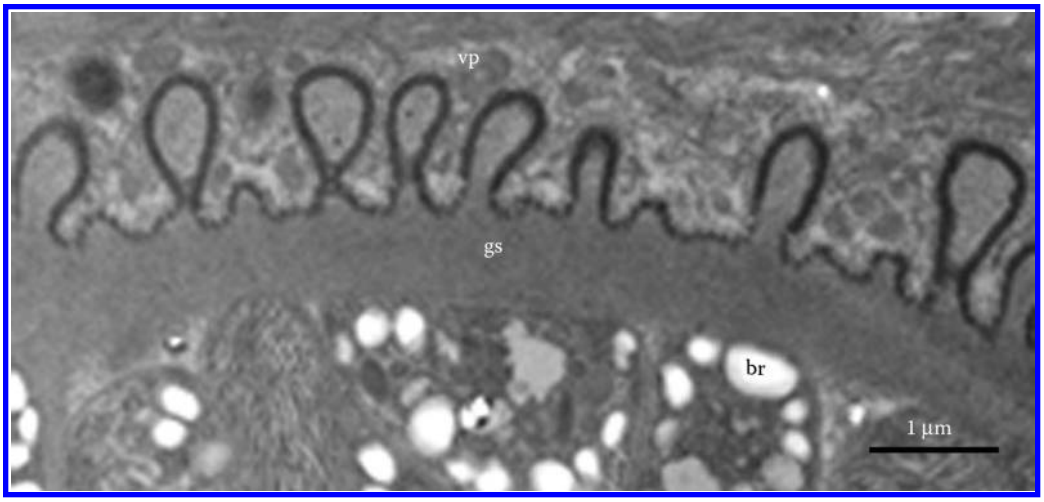


Figure 15.1 TEM of a mature *S. felis* sarcocyst in skeletal muscle of a naturally infected domestic cat. Note pleomorphic vp, thick, smooth gs and bradyzoites with amylopectin granules (br).

1.5 µm long, spaced unevenly on the sarcocyst wall. The vp are lined with a thick electron-dense layer, microtubules are absent from the vp and the ground substance layer, type 9c. Bradyzoites are slender 7–10 µm long and have numerous micronemes.[407]

The life cycle is unknown. Cats fed infected muscle did not excrete sporocysts.

15.2 INTESTINAL SARCOCYSTOSIS

Cats are definitive hosts for numerous *Sarcocystis* species (Table 15.2). There are a few surveys on the prevalence of sporocyts excreted by domestic or stray cats,[80,1021,1060,1800] nevertheless the rates

Table 15.2 Cats as Definitive Hosts for *Sarcocystis* Species

Species	Intermediate Host	Size of Sporocysts (µm)
S. gigantea	Sheep	10.5–14.0 × 8.0–9.7
S. medusiformis	Sheep	10.3–13.0 × 7.3–8.8
S. moulei	Goat	11.6–13.1 × 8.7–9.4
S. hirsuta	Cattle	11–14 × 7–9
S. fusiformis	Water buffalo	11.5–14 × 9–10
S. sp.	Grant's gazelle	12.6–18.0 × 8.4–12.0
S. muris	House mouse	8.7–11.7 × 7.5–9.0
S. cuniculi	Rabbit	11.6–14.5 × 8.7–10
S. cymruensis	Rat	10.5 × 7.9
S. sp.	Chicken	11.2–12.2 × 8.5–9.3
S. leporum	Cottontail rabbit	13–16.7 × 9.3–11.1
S. odoi	White-tailed deer	11–15 × 9–11
S. porcifelis	Pig	13.2–13.5 × 7.2–8.0
S. poephagi	Yak	20.8–25.7 × 15.9–22.4

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seem to be highly related to the high prevalence of sarcocysts found in domestic livestock (as shown in other chapters in this book).

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80, 407, 486, 519, 521, 561, 607, 684, 828, 963, 1021, 1060, 1757, 1800, 1905, 1923, 1956, 1958, 1962, 2139, 2156, 2158–2160, 2201, 2228, 2255, 2256, 2259, 2367.

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CHAPTER 16

Sarcocystosis in Chickens (*Gallus gallus*)

16.1 INTRODUCTION

There is considerable confusion concerning the species of *Sarcocystis* in chickens. The first reports of *Sarcocystis* infection in chickens were from Hungary, and the parasite was named *Sarcocystis horvathi*.[1450,1451] Sarcocysts were up to 1 mm long, the sarcocyst wall 1.8–2.7 μm thick, but the bradyzoites were not described.

In Bulgaria, *Sarcocystis* was found in chickens and a new name, *Sarcocystis gallinarum*, was proposed.[977] Sarcocysts were up to 10 mm long, bradyzoites were banana shaped, 7–13 μm long and 2–4.5 μm wide. The name was synonymized with *S. horvathi*.[1048]

In Russia, sarcocysts from naturally infected chickens with *S. horvathi* and from ducks infected with *Sarcocystis rileyi* were transmitted to dogs and cats.[715] There were 4 groups of dogs and cats: (1 and 3) 3 dogs 1.5–2 years of age; (2 and 4) 3 kittens 1.5–2 months of age. Each had 2 infected and 1 control animal. Groups 1 and 2 were fed 250–300 g of infected chicken meat and groups 3 and 4 were fed infected duck meat. Both dogs and cats fed infected meat excreted sporocysts with a prepatent period of 10 or 11 days; the patent period was 23 days for dogs, and 19 days for cats. The sporocysts were 13.3–15.4 × 10.6 μm. The author did not indicate whether dogs or cats were positive with *S. horvathi* or *S rileyi* or if both were positive with both species and whether dogs and cats had never ingested meat before the experiment.

Sarcocysts were seen in histological sections of 35 (45.5%) of 78 chickens from Papua New Guinea and in 2 chickens from Tasmania, Australia.[1248] Sarcocysts were up to 2 mm long and had a 1.5 μm thick striated wall. The ultrastructure of sarcocysts in muscles from a fowl from Tasmania and another fowl from Papua New Guinea were also described.[1173] The vp on the sarcocyst wall were 1.5 μm long and contained criss-crossing microtubules. Bradyzoites were not described. A dog fed infected muscles from a fowl excreted sporocysts but the sporocysts were not infectious for chickens. Thus, dogs could not be proven to be the definitive host,[1248] or the species found in the fowl was not infectious for domesticated chickens.

In Germany, sarcocysts were found in 45 of 241 free-range chickens but were not found in any of 207 battery raised chickens.[1822] Two types of sarcocysts were recognized based on the shape of bradyzoites. The banana-shaped bradyzoite sarcocysts were considered *S. horvathi* described in 1909[1451]; dog, cat, polecat, marten, and goshawk fed infected chicken muscle did not excrete sporocysts. They found a second type of sarcocyst containing lancet-shaped bradyzoites that were 12–15 × 2.5–3.0 μm in size. Both dogs and cats fed these sarcocysts shed sporocysts.[1822] The sporocysts were 11.6 × 9.2 μm in size. They fed 90 chickens with 1000–10,000 sporocysts (either dog or cat) and killed chickens at intervals for histological study. Schizonts were not found in chickens killed 1–15 DPI. Sarcocysts were detected starting 16 DPI. This seems very early compared with

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other species. At 40 DPI sarcocysts were immature, but they were mature at 71 DPI. Both dogs and cats fed chickens 88 DPI excreted sporocysts.[1822]

Reports of undefined species have been published more recently. In the Czech Republic, sarcocysts were found in 3 (10.0%) of 30 hens; and a dog fed infected tissues did not excrete sporocysts.[1380,1381] In China, sarcocysts were found in 6 (2.1%) of 284 free-range chickens from 3 shops in Kunming.[1109] Sarcocysts were found in most muscles of the carcass but not in the heart. Sarcocysts contained lancet-shaped bradyzoites (14.3 × 2.9 µm), and the cyst wall was identical to that from sarcocysts from Australia and Papua New Guinea.[1173] Four of 6 dogs that were 2 months old and 5 of 8 cats excreted sporocysts after consuming naturally infected muscles of chickens; sporocysts were 13.0 × 8.6 µm in size. Sporocysts (500,000; source not stated) were fed to 5 chickens that were 2 weeks old, and the chickens were killed at 20, 33, 63, 116, and 128 DPI; 3 chickens were controls but results were not given. Sarcocysts were not observed at 20 DPI. Sarcocysts at 33 DPI were immature. Mature sarcocysts were found in chickens at 63, 116, and 128 DPI.[1109]

The species with lancet-shaped bradyzoites[1822] was named *Sarcocystis wenzeli*.[1314]

In China, *S. wenzeli* was found in 17 (8.9%) of 191 free-range chickens from Yunnan and the morphological description of the species was added.[212]

In Azerbaijan, sarcocysts were found in 24 (27.9%) of 86 hens.[1182] Macroscopic sarcocysts found in pectoral and esophageal muscles were considered to be *S. horvathi*.

It appears that there are at least 2 named species, *S. horvathi* and *S. wenzeli* in chickens, with cats and dogs as definitive hosts. However, it is uncertain if both cats and dogs are hosts for the same parasite. In contrast to the foregoing reports, *Sarcocystis* sporocysts were not found in the feces of cats fed breast muscles from over 2000 chickens from grocery stores in the United States[458] and hearts from more than 1000 free-range chickens from different countries[465] although the muscles were not examined microscopically for sarcocysts or bradyzoites.

16.2 *SARCOCYSTIS WENZELI* (WENZEL, ERBER, BOCH, SCHELLNER 1982) ODENING 1997

16.2.1 Distribution

Australia, People's Republic of China, Papua New Guinea, Czech Republic, Germany.

16.2.2 Definitive Hosts

Dog, cat.

16.2.3 Structure and Life Cycle

Sarcocysts up to 3.1 mm long, thread-like, sarcocyst wall up to 3.5 µm thick, vp up to 1.9 µm long with prominent criss-crossing microtubules extending the entire length of the vp and embedded in the ground substance, villar tips with thick edl, type 9 k (Figure 16.1). Bradyzoites are lanceolate and measure 12.2–17.7 × 1.8–2.9 µm.[212] Sarcocysts in experimentally infected chickens mature by 63 DPI.[1109]

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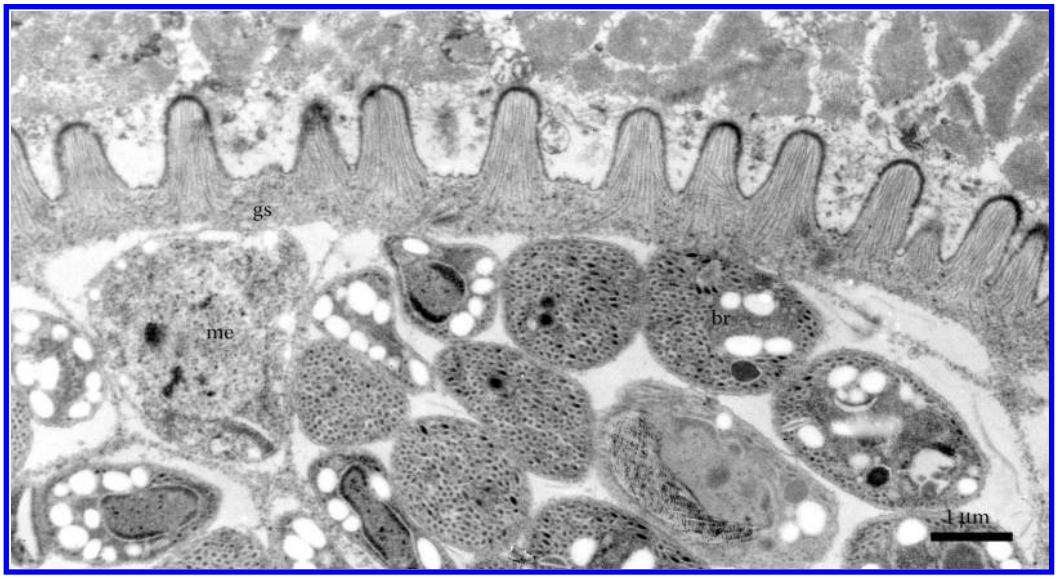


Figure 16.1 TEM of sarcocyst of *S. wenzeli.* Note stubby vp with criss-crossing microtubules. (From Mehlhorn, H., Hartley, W.J., Heydorn, A.O., 1976. *Protistologica* 12, 451–467. With permission.)

16.3 *SARCOCYSTIS HORVATHI* RÁTZ 1908

16.3.1 Distribution

Hungary, Bulgaria, Russia, Germany, Papua New Guinea, Australia, USA.

16.3.2 Structure and Life Cycle

Sarcocysts up to 980 µm long with indistinct septa. The sarcocyst wall is striated, up to 2.5–3.0 µm thick. Bradyzoites are banana shaped, 9.0–12.5 × 2.5–3.0 µm in size. No other details were found.

16.4 CLINICAL DISEASE IN NATURALLY INFECTED CHICKENS

Severe myositis was seen in chickens from Papua New Guinea and Australia[1248]; affected chickens had muscular weakness and adopted a duck-sitting posture. Numerous immature and mature sarcocysts were found in lesions of nonsuppurative myositis.

Severe encephalitis (Figure 16.2) was seen in free-range adult chickens in Mississippi, USA where many chickens had died with neurological signs.[1266] Three chickens were examined at necropsy, and all 3 had severe encephalitis associated *Sarcocystis* schizonts in various stages of development. The schizonts appeared to be in neural cells. Retrospectively, schizonts did not react to the *S. neurona* antibody (Dubey, J.P., unpublished data).

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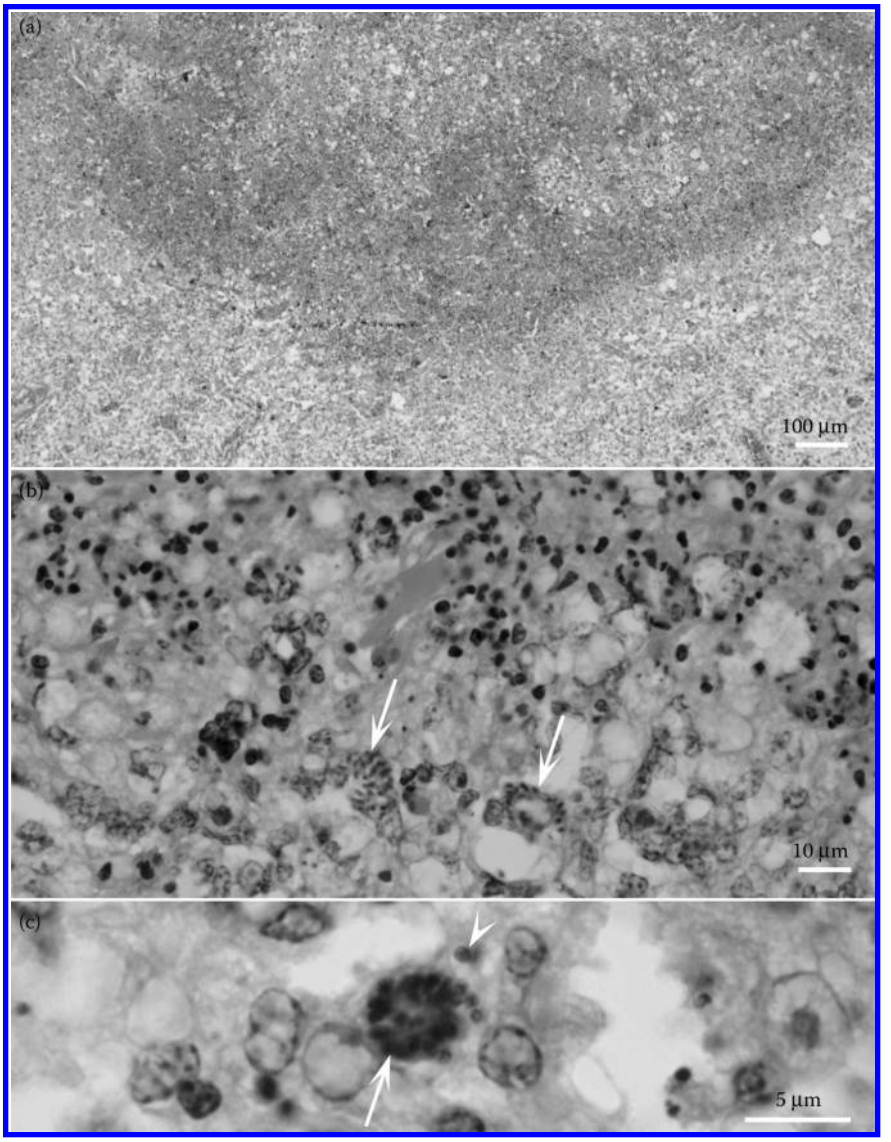


Figure 16.2 *Sarcocystis*-associated encephalitis in the cerebrum of a chicken. H and E stain. (a) Focal area of necrosis and perivascular cuffing. Numerous schizonts are present but not visible at this magnification. (b) Higher magnification of the edge of the lesion, showing schizonts (arrows). (c) Higher magnification to show a schizont (arrow) and individual merozoites (arrowhead). (Adapted from Mutalib, A. et al., 1995. *Avian Dis*. 39, 436–440.)

Myositis associated with undiagnosed *Sarcocystis/Leucocytozoon*-like parasite has been reported from Malaysia.[2203]

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212, 458, 465, 715, 977, 1048, 1109, 1173, 1182, 1248, 1266, 1314, 1380, 1381, 1450, 1451, 1822, 1916, 2372.

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CHAPTER 17

Sarcocystosis in Other Avian Species

17.1 INTRODUCTION

There are numerous species of *Sarcocystis* in birds. In this chapter, they are arbitrarily discussed in 6 groups (A–F): groups A–E, species with birds (other than raptorial) as intermediate hosts; and group F, species with raptors as definitive hosts. Group A, constituting species with an unusually wide host range and marked pathogenicity; group B constituting *Sarcocystis* species with a known definitive host; group C, *Sarcocystis* species with an unknown life cycle; group D, species with uncertain taxonomic status; group E, sarcocysts in raptorial birds; and group F, species with raptors as definitive hosts.

17.2 GROUP A

There are 2 species, *Sarcocystis falcatula* and *Sarcocystis calchasi* (Table 17.1). These species are biologically and morphologically distinct.

17.2.1 *Sarcocystis falcatula* Stiles 1893

There is considerable uncertainty concerning this parasite. It was first named for the sarcocysts in muscles of the rose-breasted grosbeck (*Pheucticus ludovicianus*). Box et al.[137] redescribed it. Sarcocysts are thick-walled, type 11b (Figure 17.1). The opossum (*Didelphis virginiana*) is the definitive host[133] and several species of birds act as intermediate hosts. Subsequently, 2 other species of South American opossums (*Didelphis albiventris* and *Didelphis marsupialis*) were found to be its natural definitive hosts.[418,441] The type specimens of *S. falcatula* deposited by Stiles have been re-examined by us, and there is no way to determine whether the parasite Box described is the same as described by Stiles. It is most likely that *S. falcatula* constitutes a group of several species. Recently, a new species *Sarcocystis lindsayi* was recognized with *D. albiventris* and *D. aurita* as definitive hosts.[433,1670] The natural intermediate host/hosts for *S. lindsayi* is unknown; budgerigars (*Melopsittacus undulatus*) were found experimentally to be an intermediate host. Budgerigars fed sporocysts from feces of opossum that died of acute sarcocystosis, similar to infection with *S. falcatula*. Another *S. falcatula*-like parasite was recognized in the carmine bee-eater (*Merops nubicus*) in the United States.[436] *In vitro* culture, life cycle studies with cloned parasites, and molecular assays will be needed to resolve the speciation issue.

Numerous outbreaks of acute sarcocystosis have been reported in passerine and psittacine birds in captivity in the Americas (Table 17.1). These outbreaks were presumed to be as *S. falcatula* infections. Some affected birds die acutely without prior signs. Depression and dyspnea are the most

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Table 17.1 Comparison of 2 Most Pathogenic Avian Species of *Sarcocystis*, *S. falcatula*, and *S. calchasi*

Character	*S. falcatula* (STILES 1893) BOX, MEIER, AND SMITH 1984	*S. calchasi* OLIAS, GRUBER, HAFEZ, HEYDORN, MEHLHORN AND LIERZ 2010
	Intermediate Hosts	
Natural	Numerous passeriformes, psittaciformes, columbiformes, strigiformes, and falconiformes including great horned owl (*Bubo virginianus*), bald eagles (*Haliaeetus leucocephalus*), golden eagle (*Aquila chrysaetos*), brown-headed cowbird (*Molothrus ater*). Luzon bleeding-heart pigeon (*Gallicolumba luzonica*), Pinon's imperial pigeon (*Ducula pinon*), nutmeg imperial pigeon (*Ducula bicolor bicolor*), Patagonian conure (*Cyanoliseus patagonus*), grackle (*Quiscalus quiscula*) and many other new and old world avian species[262,436,484,711,1305,1354,1630,1631,1691,1795]	Domestic pigeon (*Columbia livia* f. *dom.*), psittaciformes in captivity as Alexander parrots (*Polytelis alexandrae*), long-billed corella (*Cacatua tenuirostris*) and rose-breasted cockatoos (*Eolophus roseicapilla*)
Type host	Rose-breasted grosbeck (*P. ludovicianus*)	Domestic pigeon
Experimental	Canary (*S. canaria*), zebra finch (*Poephila guttata*), budgerigar (*M. undulatus*), domestic pigeon (*Columbia livia* f. *dom.*), Guinea fowl (*Numida meleagris*), house sparrows (*Passer domesticus*)	Domestic pigeon, cockatiel (*Nymphicus hollandicus*), great tit (*Parus major*)
Distribution	Americas	Europe, North America
Original	North America	Europe
	Schizonts	
Persistence	Extended period, schizonts and sarcocysts in the same animal	Extended period, schizonts and sarcocysts in the same animal
Duration	Extended	7–12 days, experimental
Main locale	Lung, liver	Liver
	Sarcocysts	
Size	Up to 3.2 mm long	Up to 2.0 mm long
Wall type	Striated, TEM type 11b	Smooth, wavy, TEM type 1a
Bradyzoites	6.8×2.1 (4.8–8.4 $\times$ 1.2–3.6) μm (muscle digest)	Lancet-shaped, 7.5×1.5 μm (fixed smears, Giemsa)
Molecular data	18S, ITS-1	18S, 28S, ITS-1
	Definitive Hosts	
Natural	Opossums (*D. virginiana, D. albiventris, D. marsupialis*)	Northern goshawk (*Accipiter g. gentilis*), European sparrowhawk (*Accipiter nisus*)
Experimental		Northern goshawk
Sporocysts size	11.2×7.4 (9.6–12.0 $\times$ 6.0–8.0) μm	11.9×7.9 μm
Reports of outbreaks and clinical cases	101, 122, 228, 229, 436, 484, 711, 831, 879, 1032, 1189, 1354, 1631, 1691, 1795, 1841, 1842	1325, 1328, 1329, 1473, 1841, 1843
General references	131, 133, 134, 135, 262, 412, 414, 418, 421, 441, 792, 1070, 1088, 1116, 1173, 1273, 1305, 1409, 1626, 1627, 1629, 1630, 1631, 1660, 1932, 2209	1099, 1326, 1327 ,1330, 1331, 1332, 1696, 2395

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Figure 17.1 TEM of *S. falcatula* sarcocyst in skeletal muscle of a budgerigar (*M. undulatus*). Note the elongated vp on the sarcocyst wall. Few microtubules are present in the vp, and they extend into the gs (arrowheads). The bradyzoites (br) are juxtaposed with the gs. (Adapted from rom Box, E.D., Meier, J.L., Smith, J.H., 1984. *J. Protozool*. 31, 521–524; Courtesy of late Edith Box.)

prominent signs. At post mortem, edema and congestion of the lungs and enlargement of the spleen and liver are the most prominent findings. Microscopically, lesions are associated with intravascular schizonts, often occluding capillaries (Figure 17.2).

The host range of *S. falcatula* is wide; in other taxonomic groups, there is a report (other than those reported in Table 17.4 in this chapter) of severe encephalitis in a free-ranging great horned owl (*Bubo virginianus*) from Minnesota, USA.[1841] Pathogenic *Sarcocystis* species add a new concern in conservation.

17.2.2 *Sarcocystis calchasi* Olias, Gruber, Hafez, Heydorn, Mehlhorn, and Lierz 2010

Unlike *S. falcatula*, *S. calchasi* sarcocysts are thin-walled, type 1a (Figure 17.3). They cause neurological signs in pigeons and psittacine birds (Table 17.1). The affected birds have signs of depression, torticollis, opisthotonus, trembling, paralysis, and polyuria.[1325] Necrotic and granulomatous meningoencephalitis is the predominant lesion associated with schizogonic development of the parasite in neural cells.

17.3 GROUP B

There are 6 species in this group (Table 17.2).

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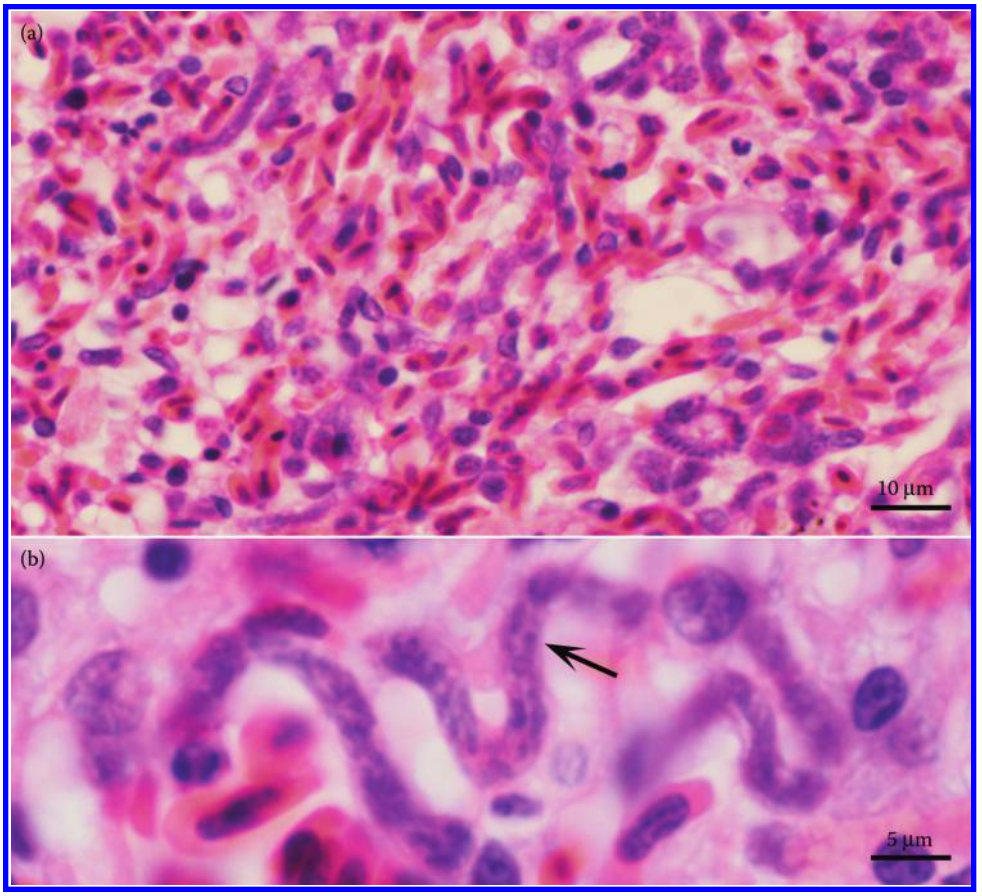


Figure 17.2 *S. falcatula*. (a) Section of a lung of a budgerigar fed sporocysts from *D. albiventris* from Argentina. Note severe parasitization of pulmonary blood vessels. H and E stain. (b) Higher magnification to show serpentine schizonts (arrow).

17.4 GROUP C

Sarcocyst morphology has been described for 23 species (Table 17.3).

Unnamed sarcocysts have been found in the straw-necked ibis (*Threskiornis spinicollis* [*Carphibis spinicollis*]),[437] coot (*Fulica atra*),[1822] wild turkey (*Meleagris gallopavo*),[1728] Northern gannet (*Morus bassanus*),[1647] American woodcock (*Scolopax minor*),[829] goldeneye (*Bucephala clangula*)[1004] and in several species of wading birds (Ciconiiformes).[1646] Seven avian families including Anatidae, Icteridae, and Corvidae in Canada were surveyed.[341] Sarcocysts were found in 11 host species but parasites were not named. The same year, in Australia, *Sarcocystis* was detected in 44 species representing 25 families (e.g., Pelecanidae, Anatidae, Falconidae, Rallidae, Corvidae, etc.).[1251] Thin- and thick-walled cysts were recorded, but final identity was not provided.

17.5 GROUP D

These species are considered invalid/species *inquirendae* as there are no differential morphological descriptions of the sarcocyst. Namely, *Sarcocystis ammodrami* (Splendore, 1907) Babudieri,

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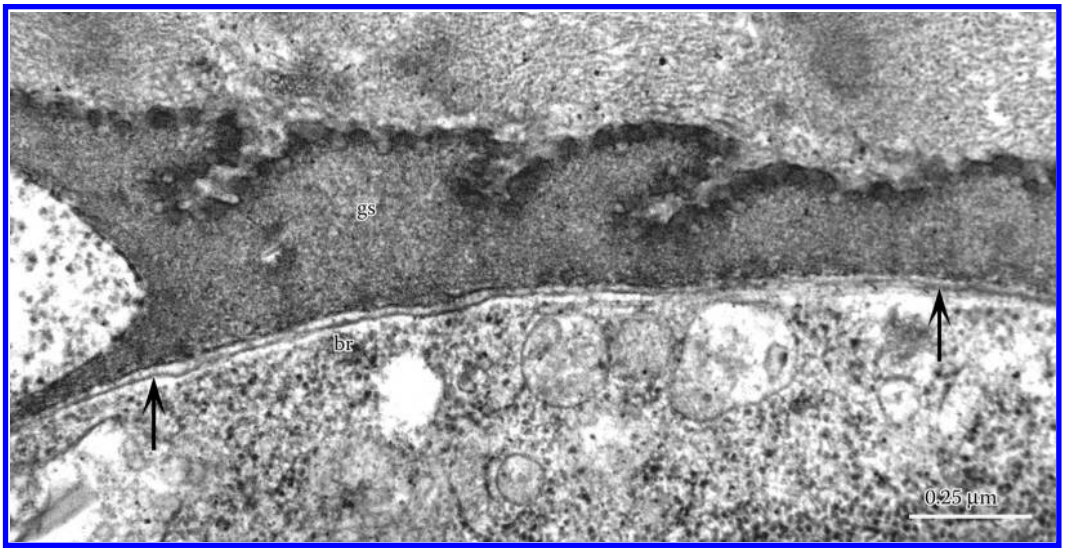


Figure 17.3 TEM of *S. calchasi* sarcocyst in skeletal muscle of a pigeon. Note the thin sarcocyst wall, without villi, and microtubules. Arrows point to the bradyzoite (br) pellicle. (Courtesy of A.G. Armién, Diagnostic Ultrastructural Pathology Service, College of Veterinary Medicine, University of Minnesota.)

1932 from the Fringillid bird (*Pheucticus ludovicianus*)[1049]; *Sarcocystis aramidis* Spendore, 1907 from the Rallid bird (*Aramides saracura*)[1049]; *Sarcocystis jacarinae* Barreto, 1940 from the Fringillid bird (*Volatinia jacarina*)[1049]; *Sarcocystis oliverioi* Pessõa, 1935 from the Psittacid bird (*Forpus passerinus*)[1391]; *Sarcocystis turdi* Brumpt, 1913 from the European blackbird (*Turdus merula*), *S. nontenella* Eble, 1961 from the common buzzard (*Buteo buteo*)[1049] and *Sarcocystis debonei* (Vogelsang, 1929) Duszynski and Box 1978 from the cowbird (*Molothrus ater*).[482]

There are 3 additional species that need validation, *Sarcocystis garzettae* Odening 1997 from the little egret (*Egretta garzetta*), *Sarcocystis kaiserae* Odening 1997 from the laughing dove (*Streptopelia senegalensis*), and *Sarcocystis spaldingae* Odening 1997 from Ciconiiformes (*Ardea Herodias*, *Egretta caerulea*, etc.).[1314,1317]

17.6 GROUP E

This group includes sarcocysts in the muscle of raptors. Sarcocysts are apparently common in raptors.[179] *Sarcocystis*-like bradyzoites were found in digests of 52 (45.6%) of 114 samples of muscles from 11 species of raptorial birds.[1069] Sarcocysts were identified in the histological sections of muscles of 28 (37.8%) of 74; 12 of 21 red-tailed hawks (*Buteo jamaicensis*), 2 of 5 red-shouldered hawks (*Buteo lineatus*), 4 of 4 Cooper's hawks (*Accipiter cooperi*), 2 of 3 sharp-shinned hawks (*Accipiter striatus*), 1 of 2 American kestrels (*Falco sparverius*), 2 of 4 turkey vultures (*Cathartes aura*), 1 of 2 black vultures (*Coragyps atratus*), 1 of 10 barred owls (*Strix varia*), and 3 of 8 screech owls (*Megascops* spp.).[1069]

Two types of sarcocysts with thin and thick walls have been reported in bald eagles (*Haliaeetus leucocephalus*). Thin-walled microscopic sarcocysts were found in a female bald eagle in Alabama.[261] Both thin- (variety A) and thick-walled (variety B) sarcocysts were seen in histologic sections of muscles of a female bald eagle from Missouri.[1333] Variety A sarcocysts were microscopic, had a thin

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Table 17.2 ***Sarcocystis*** **Species with Birds as Intermediate Host and Known Definitive Hosts**

Species	Intermediate Host	Sarcocysts: Size (μm)	Sarcocysts: TEM Wall Type	Sarcocysts: Bradyzoites (μm)	Schizonts	Definitive Host	Molecular Data	Location	References
S. albifronsi Kutkiené, Prakas, Sruoga, and Butkauskas 2012	White-fronted goose (*Anser albifons*)	Microscopic, ribbon-like, up to 4 mm long, 750 μm wide	Thick, up to 2.3 μm long vp, type 9a	Lanceolate, 10.0–13.5 × 1.5–2.5	–	Arctic fox (*Alopex lagopus*), sporocysts 10.0–12.8 × 6.8–8.6 μm	18S, 28S, ITS-1	Lithuania	**158, 1002, 1003, 1008**
S. alectorivulpes Pak, Sklyarova, and Pak 1989	Chukar partridge (*A. chukar*)	Macroscopic. Thick, striated wall	–	Cigar-shaped, 2.1–4.2 × 9.8–14.0	No data	Foxes (*Vulpes vulpes, Alopex corsac*)	No data	Kazakhstan	**1284, 1357**
S. lindsayi Dubey, Rosenthal, and Speer 2001	Experimental budgerigar (*Melopsittacus undulatus*)	Up to 600 μm, up to 2 μm	Thick, 2.0 × 0.3 μm vp like, type 11d	6 × 1.5 μm	In cell culture, merozoites 4.8–6.4 × 1.6–2.4	*D. albiventris, D. aurita*; sporocysts 12 × 7 μm	ITS-1	Brazil	**425, 433, 1669, 1670**
S. peckai (Pecka 1988) Odening 1997	Pheasant (*Phasianus colchicus*)	–	–	Lancet-shaped, 14–16 × 2–3	–	Dog	No data	Former Czechoslo-vakia	**1314, 1381, 1822**
S. rileyi (Stiles 1893) Dubey, Cawthorn, Speer, and Wobeser 2003	Shoveler duck (*Anas clypeata*), mallard duck (*Anas platyrhynchos*), common eider (*Somateria mollissima*), Eurasian wigeon (*Anas penelope*), common teal (*Anas crecca*)[a]	Macroscopic, up to 12 mm, smooth	7.5 μm long vp, type 23	Slender, 10.7–13.0 × 2.5–5.1	–	Skunk (*Mephitis mephitis*), sporocysts 10–14 × 5.5–9.5 μm. Molecular identification in feces of red fox (*Vulpes vulpes*) and raccoon dog (*Nyctereutes procyonoides*) from Europe	18S, 28S, ITS-1, *cox1*	North America, Lithuania, Finland, Norway, other countries[b]	**452, 466, 703, 1007, 1421, 2398**
S. sp. Wenzel, Erber, Boch, and Schellner 1982	Pheasant (*Phasianus colchicus*)	Up to 2 mm. Thick, striated wall, with 3–4 μm long finger-like vp	–	Lanceolate, 12.5–17 × 2–3	No data	Dog, sporocysts 13–15 × 9–11	No data	Czech Republic, Germany	**191, 1380, 1822**

[a] Cornwell[257] and Chabreck[194] reported *S. rileyi*-like in Anatidae as redhead (*Aythya americana*), canvasback (*Aythya valisineria*), and common goldeneye (*Bucephala clangula*) and reviewed other previous reports in the England, Mexico, Panama, and the United States.

[b] Bulgaria, Germany, Poland, Russia,[715,931,933] and in other European countries.[1421]

Table 17.3 ***Sarcocystis*** **Species with Birds as Intermediate Host and with Unknown Definitive Hosts**

Species	Intermediate Host	Sarcocysts			Molecular Data	Country-Location	References
		Size (μm)	Wall Type	Bradyzoites (μm)			
S. anasi Kutkiené, Prakas, Sruoga, and Butkauskas 2012	Mallard duck (*Anas platyrhynchos*)	Microscopic, up to 5 mm long	Thick, palisade-like vp, type 9a	Blunt ends, 13–16.1 × 1.8–2.5	18S, 28S, ITS-1	Lithuania	**1002, 1004, 1008**
S. colii Fantham 1913	Red-faced African mouse bird (*Colius erythromelon*)	Macroscopic, 2.5 mm	No data	5–7 in smears	No data	Origin unknown, Liverpool museum, UK	**565**
S. columbae Olias, Olias, Liez, Mehlhorn, and Gruber 2010	Wood pigeon (*Columba palumbus*)	Microscopic, ribbon-like, up to 7 mm long	Thin, type 1a	Banana-shaped, 6.3–7.3	18S, 28S, ITS-1	Germany, Lithuania	**1326, 1416**
S. cornixi Kutkiené, Prakas, Sruoga, and Butkauskas 2009	Hooded crow (*Corvus cornix*)	Microscopic, ribbon-shaped, up to 6 mm	2.1 μm thick, stump-like vp, type 1 g	Banana-shaped, 6.1–7.9 × 1.4–1.8	18S, 28S, ITS-1	Lithuania	**1005**
S. corvusi Prakas, Kutkiené, Butkauskas, Sruoga, and Žalakevičius 2013	Jackdaw (*Corvus monedula*)	Microscopic, thick thread-like, up to 6 mm long	Thin, type 1d	Lancet-shaped, 5.9–7.3	18S, 28S, ITS-1	Lithuania	**1419**
S. chloropusae El-Morsey, El-Seify, Desouky, Abdel-Aziz, Sakai, and Yanai 2015	Common moorhen (*Gallinula chloropus*)	Microscopic, up to 650 μm	2.0–4.5 μm thick wall, type 10	6–12 × 1–2	18S, 28S, ITS-1	Egypt	**489, 490**
S. kaiserai (Kaiser 1983) Odening 1997	Laughing dove (*Streptopelia senegalensis)*	No data	Type 9j	No data	No data	South Africa	**926, 927, 1314**
S. kirmsei Garnham, Duggan, and Sinden 1979	Hill mynah (*Gracula religiosa*), Siamese fire-backed pheasant (*Lophura diardi*)	Cyst in brain, round to oval, up to 1100 μm. 1–4 μm thick wall with spines	–	7–10 × 1.9–2.6	No data	Thailand, Panama, Germany	**664, 895**

(*Continued*)

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Table 17.3 (*Continued*) *Sarcocystis* Species with Birds as Intermediate Host and with Unknown Definitive Hosts

Species	Intermediate Host	Sarcocysts			Molecular Data	Country-Location	References
		Size (μm)	Wall Type	Bradyzoites (μm)			
S. lari Prakas, Kutkiené, Butkauskas, Sruoga, and Žalakevičius 2014	Black-baked gull (*Larus marinus*)	Microscopic, thread-like, up to 6 mm long	Thin, type 1d	Lancet-shaped, 6.3–7.9 × 1.2–1.5	18S, 28S, ITS-1	Lithuania	**1420**
S. phoeniconaii (Murata 1986) Gobel, Erber, and Grimm 1996	Lesser flamingo (*Phoeniconaias minor*)	Macroscopic, rice grain-like, up to 7 mm	Thick, vp up to 7 μm long, type 9l	Lancet-shaped, 15–20 × 1.8–2.5	No data	Germany, Tanzania	**47, 709, 1319**
S. ramphastosi Dubey, Lane, and Van Wilpe 2004	Keel-billed toucan (*Ramphastos sulfuratus*)	Macroscopic, up to 3 mm long	Thin, smooth, vp up to 6.5 μm, type 9e	4.0–4.5 × 1.3–1.6	18S	South Africa, Costa Rica	**457, 463**
S. setophagae Crawley 1914	Redstart (*Setophaga ruticilla*)	Macroscopic 2.5 mm	No data	4–5	No data	USA	**260**
S. sulfuratusi Dubey, Lane, and Van Wilpe 2004	Keel-billed toucan (*Ramphastos sulfuratus*)	Microscopic, up to 900 μm	Thin, smooth, vp up to 4.3 μm long, type 9f	5.0–7.0 × 1.5–2.2	No data	South Africa (Bird was imported from Central America)	**457**
S. turdusi Kutkiene, Prakas, Butkauskas, and Sruoga 2012	Common blackbird (*Turdus merula*)	Microscopic, ribbon-like, up to 7 mm long	Thick, vp branched, similar to type 18b	Orange segment-shaped, 5.5–7.2 × 1.2–1.5	18S, 28S, ITS-1	Lithuania	**1009**
S. wobeseri Kutkiené, Prakas, Sruoga, and Butkauskas 2010	Barnacle goose (*Branta leucopsis*), mallard duck (*Anas platyrhynchos*), herring gull (*Larus argenticus*)	Microscopic, ribbon-like, up to 6 mm long	Thin, type 1d	Banana-shaped, 6.4–7.9	18S, 28S, ITS-1	Lithuania	**1834, 1002, 1006, 1415**
S. sp. Wenzel, Erber, Boch, and Schellner 1982	Coot (*Fulica atra*)	Microscopic, up to 360 μm long. Smooth, not clear	–	Banana-shaped, 7.5–8 × 1.5–2	No data, dog, cat not hosts	Germany	**1822**

(*Continued*)

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Table 17.3 (*Continued*) *Sarcocystis* Species with Birds as Intermediate Host and with Unknown Definitive Hosts

Species	Intermediate Host	Sarcocysts			Molecular Data	Country-Location	References
		Size (μm)	Wall Type	Bradyzoites (μm)			
S. sp. Teglas, Little, Latimer, and Dubey 1998	Turkey (*Meleagris gallopavo*)	Sarcocyst in cross section 60 μm diameter. 5 μm thick sarcocyst wall with leaf-like vp with a stalk	–	NS	No data	USA	**1728**
S. sp. Dubey, Johnson, Bermudez, Suedmeyer, and Fritz 2001	Straw-necked ibis (*Carphibis spinicollis*)	Microscopic, up to 350 μm long	2 μm thick, striated, vp 4.5 μm long, type 11d	4.5–5.0 × 1.5	No data	USA	**437**
S. sp. Spalding, Yowell, Lindsay, Greiner, and Dame 2002	Northern gannet (*Morus bassanus*)	NS	Palisade-like, type 11d	NS	ITS-1 (for schizonts in brain, not done in sarcocysts)	USA	**1647**
S. sp. Dubey and Morales 2006	Buffon's macaw (*Ara ambigua*)	Up to 950 μm long	Vp up to 4 μm long, folded over, type 11c	4.0–5.9 × 0.8–2.8	No data	Costa Rica	**1989**
S. sp. Hiller, Sidor, De Guise, and Barclay 2007	American woodcock (*Scolopax minor*)	Microscopic, up to 125 μm	Thin, 2 μm long finger-like vp, type 9b	7 × 2	No data	USA	**829**
S. sp. Kutkiené, Sruoga, and Butkauskas 2008	Goldeneye (*Bucephala clangula*)	Microscopic, up to 1.4 mm	Irregular surface, wavy, type 1a	Banana-shaped, 8.5–7.0	No data	Lithuania	**1002, 1004**

NS = not stated

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sarcocyst wall with spines, and contained bradyzoites that measured 5 × 1 μm. Variety B sarcocysts had a 2 μm thick striated cyst wall, and were immature.

Only *Sarcocystis otus* has been proposed as a species name for *Sarcocystis* in raptors. Three different types of sarcocysts were detected in the Eurasian buzzard (*B. buteo*) and long-eared owl (*Asio otus*) from Germany; the third species (proposed as *S. otus*) had hirsute vp and 5.1–2.9 × 1.9–2.8 μm long bradyzoites.[979]

17.7 GROUP F

In addition to raptors acting as definitive hosts for species of *Sarcocystis* in rodents (see Chapter 21), the sizes of oocysts and sporocysts detected in 9 species within the orders Accipitriformes and Strigiformes from the Berlin–Brandenburg area in Germany were reviewed.[1302] *Sarcocystis* sporocysts were found in 1 of 2 turkey vultures from Alabama, USA, but sporocysts were not infectious for cotton rats and mice.[1066] Sporocysts were found in an Indian pariah kite (*Milvus migrans*) that were not infective to rats and mice.[1591] Molecular methods distinguished 8 different genetic sequences of *Sarcocystis* spp. present in fecal matter from 4 species of hawks in Georgia, USA.[1851] Intermediate hosts need to be defined. In addition, dissemination of *Sarcocystis* sporocysts is also possible in aegagropiles of diurnal and nocturnal birds.[1531]

Three species in this group need additional comments:

Sarcocystis accipitris[192] was proposed as a new species between goshawk (*Accipiter gentilis*, Accipitriformes) and Passeriformes, experimentally canaries (*Serinus canaria*) acting as intermediate hosts. The sizes of the sporocysts in definitive hosts are 15–17 × 13–15 μm. Under experimental conditions, canaries developed sarcocysts up to 900 μm long, thin-walled (<1 μm) and with small

Table 17.4 Neurological Sarcocystosis in Birds in Groups B–F

Host	Country	Clinical Signs	Main Lesion	Immunostaining	References
Northern gannet (*Morus bassanus*)[a]	USA, Florida	Neurologic	Meningoencephalitis	Not reacted against *S. neurona*	**1647**
Bald eagle (*Haliaeetus leucocephalus*)[b]	USA, Missouri	Neurologic	Meningoencephalitis	*S. neurona*	**1333**
Northern goshawk (*Accipiter gentilis atricapillus*)[b]	USA, Minnesota	Paralysis	Encephalitis	*S. cruzi*	**21**
Golden eagle (*Aquila chrysaetos*)[a]	USA, Virginia	Neurological, blindness	Necrotizing encephalitis	*S. cruzi*	**403**
Straw-necked ibis (*Threskiornis spinicollis*)[b]	USA, Missouri	Neurological	Encephalitis	*S. falcatula, S. neurona, S. cruzi*	**437**
Turkey (*Meleagris gallopavo*)[a]	USA, West Virginia	Ataxia, lethargy	Myocarditis, encephalitis	*S. cruzi*	**1728**
Capercaillie (*Tetrao urogallus*)[a]	Sweden	53 dead	Meningoencephalitis	*S. cruzi*	**763**
	Finland	Found dead	Myocarditis, encephalitis, other organs affected	*S. cruzi, S. neurona*	**413**

[a] Caught in wild.
[b] In captivity.

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Figure 17.4 Severe myocarditis in a naturally infected capercaillie from Finland. (a) Massive mononuclear cell infiltration. (b) Degenerating merozoites (arrows) among inflammatory cells. (Adapted from Dubey, J.P., Rudbäck, E., Topper, M.J., 1998. *J. Parasitol.* 84, 104–108.)

(5–7 × 1.5–2.0 µm) crescent-shaped bradyzoites. TEM information has not been reported. This is similar to *Sarcocystis* sp. reported by Svobodová[1696] in a great tit, *Parus major*, with a goshawk as definitive host, and is also similar to *S. calchasi*.

Sarcocystis alectoributeonis completes its life cycle between the common buzzard (*B. buteo*, Accipitriformes) as the definitive host and the chukar partridge (*Alectoris chukar*, Galliformes) as the intermediate host.[1357] It was originally detected in Kazakhstan. Sarcocysts are macroscopic, thin-walled (0.5–1.0 µm) and smooth. TEM type was not reported. Bradyzoites are 1.2–2.1 × 7.0–8.4 µm. Sporocysts shed by definitive hosts are 8.4–10.5 × 11.2–14.7 µm.

The life cycle of *Sarcocystis ovalis* is completed between the magpie (*Pica pica*, Passeriformes) as the definitive host,[699] this is not a raptor but an opportunistic scavenger, and the moose (*Alces alces*) as the intermediate host. Information related to this species is summarized in Chapter 18.

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17.8 CLINICAL OBSERVATIONS OF SARCOCYSTOSIS IN BIRDS IN GROUPS B–F

Mature schizonts and free merozoites of *Sarcocystis* as well as *Chlamydia* sp. were found in pulmonary lesions in an immature wild red-tailed hawk (*B. jamaicensis*) in Louisiana, USA that died following respiratory distress.[1199]

Neurological sarcocystosis has been reported in the eagle, ibis, wild turkey, gannet, and goshawk from the United States (Table 17.4). Numerous *Sarcocystis* schizonts and merozoites were found in the lesions. The 4 raptors listed in Table 17.4 had been caught in the wild but were in captivity for different reasons. Therefore, it is uncertain if they acquired sarcocystosis in the wild or in captivity. The wild turkey was killed because it was ill. Grossly, there were pale areas on the myocardium suggestive of necrosis. Schizonts were seen in areas of myocardial necrosis and in areas of encephalitis.[1728]

Systemic sarcocystosis has been reported in a capercaillie (*Tetrao urogallus*) in Finland,[413] and severe encephalitis has been seen in these galliform birds in Sweden.[763] In the bird from Finland, severe lesions were found in the heart, spleen, lung, brain, and other organs. Mortality in capercaillie has been recognized in Scandinavia since 1947, but the disease was misdiagnosed as toxoplasmosis.[763] Among the 87 capercaillie examined at post mortem, nonsuppurative encephalitis was diagnosed in 53 birds in association with *Sarcocystis*; lesions and protozoa were confined to the brain. Until now, only schizonts, no sarcocysts, of *Sarcocystis* have been found in capercaillie. Most schizonts were extravascular and up to 55 μm long (Figures 17.4).

BIBLIOGRAPHY

Group A: **101, 122, 131, 133, 134, 135, 137, 228, 229, 261, 262, 412, 414, 418, 421, 433, 436, 441, 484, 510, 711, 792, 831, 879, 1032, 1070, 1088, 1099, 1116, 1189, 1273, 1305, 1325–1332, 1354, 1409, 1473, 1626, 1627, 1629–1631, 1660, 1670, 1691, 1696, 1795, 1841–1843, 1932, 2209.**

Group B: **158, 171, 179, 191, 194, 413, 425, 433, 452, 466, 703, 715, 763, 931, 933, 1003, 1007, 1008, 1357, 1380, 1421, 1669, 1670, 2182, 2328**.

Group C: **47, 155, 257, 259, 260, 341, 342, 437, 457, 463, 489, 490, 565, 570, 599, 664, 709, 829, 845, 895, 926, 927, 931, 1002, 1004–1009, 1126, 1173, 1182, 1212, 1251, 1314, 1319, 1326, 1380, 1381, 1415, 1416, 1419, 1420, 1646, 1647, 1728, 1822, 1834, 1989, 2382**.

Group D: **393, 482, 1049, 1314, 1317, 1391, 1917, 1929, 1957, 1981, 1999, 2073, 2187, 2204, 2237, 2238, 2244, 2251, 2279, 2299, 2316, 2333, 2341, 2365**.

Group E: **179, 261, 979, 1069, 1199, 1333**.

Group F: **21**, **192, 403, 699, 1066, 1302, 1333, 1357, 1531, 1591, 1696, 1851, 2141, 2146, 2147, 2150, 2347, 2353**.

Miscellaneous: **122, 192, 794, 1917, 1981.**

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CHAPTER 18

Sarcocystosis in Wild Ruminants and Other Large Animals

18.1 *SARCOCYSTIS* INFECTIONS IN CERVIDS

Sarcocysts are ubiquitous in cervids, and numerous species of *Sarcocystis* in cervids have been named. Whether *Sarcocystis* species are host-specific for cervid species is not known. Cross transmission in animals raised in captivity and/or DNA sequence data will be necessary to prove or disprove host specificity.[285,354,380,586,704,858] Available information on wild Cervidae, other large animals, and marine mammals are summarized in Tables 18.1 through 18.3, but even morphological information is incomplete for most species. Examples of species detected but considered *inquirendae* are given in Table 18.4.

18.1.1 *Sarcocystis hemionilatrantis* Hudkins and Kistner 1977

Among all cervids, the life cycle is known for only *S. hemionilatrantis* which has been studied in fawns of mule deer (*Odocoileus hemionus*) raised in captivity. The definitive hosts include the dog (*Canis familiaris*)[379,858] and the coyote (*Canis latrans*).[858] In histologic sections, the sarcocysts are microscopic, measuring up to 525 μm long. The sarcocyst wall is 1–2 μm thick (Figure 1.10f). The vp are 1.8–2.4 μm long and 1.9–2.8 μm wide and are highly convoluted and branched at the base, type 17 (Figure 1.41a). The bradyzoites seen in tissue sections are 13.3–16 × 2.6–3.2 μm. The sporocysts are 14–16 × 9.5–11.0 μm.

S. hemionilatrantis has at least 3 generations of schizogony.[379] First- and second-generation schizonts were identified in arteries and capillaries of the lung, heart, spleen, and several other organs. First-generation schizonts at 14 DPI were 14–39 × 14–25 μm and contained around 100 nuclei. The second-generation schizonts, seen 24–39 DPI, were 14–32 × 10–20 μm and contained 20–35 nuclei. Merozoites were seen in peripheral blood 24 DPI; 1 merozoite was binucleated, suggesting multiplication in the bloodstream.

A terminal generation of schizonts, characteristic of *S. hemionilatrantis*, was found in macrophages in the muscles (Figure 1.20). Schizonts were 10–28 × 7–14 μm and contained up to 40 nuclei.[379,970]

The sarcocysts at 63 DPI measured up to 350 μm long and were immature; the sarcocyst wall was thin (<1 μm) and not striated. Mature sarcocysts, containing bradyzoites, were seen 90 DPI, measured up to 525 μm long, and were striated.[379]

Experimentally, *S. hemionilatrantis* is pathogenic for mule deer.[970] Of 12 animals fed 50,000, 250,000, or 1 million sporocysts (4 deer for each dosage), all became anorectic and uncoordinated. 9 died between 29 and 65 DPI, and 3 were euthanatized 41, 63, and 90 DPI.[970]

The lesions and clinical signs were similar to those in cattle inoculated with 150,000 or more *S. cruzi* sporocysts, but the clinical disease was more protracted in mule deer than in cattle.

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Table 18.1 ***Sarcocystis*** **Species in Wild Cervids**

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall SEM	Sarcocyst: Wall TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
			Moose (*Alces alces*)				
S. alces Dahlgren and Gjerde 2008	Spindle-shaped, up to 5.5 mm long, smooth with no visible vp	Square-platform-like vp	No data	No data	Red fox (*Vulpes vulpes*), arctic fox (*V. lagopus*)	18S, *cox*1	**282, 286, 702**
S. alceslatrans Dubey 1980	7 mm, thread-like, smooth, thin wall	Hair-like, 10–12 μm vp	Type 7b	12.6–15.8 × 3.0–3.5 μm	Coyote (*C. latrans*), dog, 14.4–15.8 × 10.8–11.5	18S, *cox*1	**244, 245, 355, 702, 999, 1576**
S. hjorti (Dahlgren and Gjerde 2008) Dahlgren and Gjerde 2010	Up to 2 mm long, thin-walled, hair-like vp 10 μm long	—	No data	No data	Red fox (*Vulpes vulpes*), arctic fox (*V. lagopus*)	18S, *cox*1	**282, 285, 286, 702**
S. ovalis Dahlgren and Gjerde 2008	Ovoid, up to 1.5 mm, vp 13 μm long, secondary encapsulation	Tongue-like vp in rows	No data	No data	Magpie (*Pica pica*), 12.5 × 7.9	18S, *cox*1	**282, 699, 702**
S. scandinavica Dahlgren and Gjerde 2008	Spindle-shaped, 1 mm long, 10 μm long finger-like vp	No data	No data	No data	Unknown	18S, *cox*1	**282, 702**
S. silva (Type D, Dahlgren and Gjerde 2008) Gjerde 2012	Sac-like, rounded end, smooth, no visible vp	No data	No data	No data	Unknown	18S, *cox*1	**282, 700, 702**
S. taeniata Gjerde 2014	Sac-like, 1.1 mm, thin smooth wall	Ribbon-like vp in parallel rows, 2 μm, sparse	No data	No data	Unknown	18S, *cox*1	**702**
			European Roe Deer (*Capreolus capreolus*)				

(*Continued*)

Table 18.1 (*Continued*) *Sarcocystis* Species in Wild Cervids

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
S. capreolicanis Erber, Boch, and Barth 1978	Thread-like, up to 1.7 mm, thin, hair-like vp	Hair-like, up to 9.5 μm long vp, aligned in parallel rows	Thread or tube-like vp, type 6	No data	Dog, red fox, (not felids, raccoon, ferret)	18S	**532, 548, 700, 1533, 1577**
S. gracilis Rátz 1908	Up to 2.5 mm, hirsute vp	Densely packed, knob-like or cube-like vp, up to 0.5 μm long	—	15.6–12.5 × 3.9–3.1	Dog, red fox, silver fox (*V. vulpes*) and blue fox (*V. lagopus*), 15 × 10	18S	**284, 548, 700, 1577, 1683**
S. oviformis Dahlgren and Gjerde 2009	Ovoid, up to 600 μm long	Tongue-like vp, 10 μm long, pointed distal tip	No data	No data	Unknown	18S	**284, 700**
S. silva Gjerde 2012	Cigar-shaped, thick striated, microscopic, 0.6 mm long, upright vp 8 μm long	No data	Tightly packed finger-like, up to 7 long, granules and microfilaments, type 10	15.6–11.7 × 3.9–3.1	Unknown	18S	**532, 548, 700, 1577**
			Siberian Roe Deer (*Capreolus pygargus*)				
S. sibirica Machul'skii 1947	Macroscopic, up to 7.5 mm, thin wall	No data	Vp with stalk, 0.4 wide tip, type 27	8.6–11.5 × 2.1–2.6	Unknown	No data	**1089, 1692**
			Sika Deer (*Cervus nippon centralis*)				
S. sp. Saito, Itagaki, Shibata, and Itagaki 1995	Up to 600 mm thick hairy 6-10 μm thick wall	No data	Hair-like vp with microtubules, type 8b	12.9 × 5.0	Raccoon dog (*Nycterectes procyonides*), domestic dog, 16 × 11.5	No data	**1515**
			Fallow Deer (*Dama dama*)				

(*Continued*)

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Table 18.1 (*Continued*) *Sarcocystis* Species in Wild Cervids

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall SEM	Sarcocyst: Wall TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
S. jorrini Hernández-Rodríguez, Acosta, and Navarrete 1992	Macroscopic, up to 1.4 mm	No data	Secondary cyst wall, vp 2–10 long, type 15	8.6–13 × 3–5	Unknown	No data	**799**
S. sp. Poli, Mancianti, Marconcini, Nigro, and Colagreco 1988	Up to 360 μm	No data	Secondary absent, 20–30 nm thick, sheet-like vp, 2–3 μm long, type 8a	13.2–21.0 × 3.3–5.5	Dog, not cat, 15.4 × 8.8	No data	**1404**
S. cf. *hofmanni* Wesemeier and Sedlaczek 1995	Probably, *S. jorrini* [799]						**1823**
S. cf. *grueneri* Wesemeier and Sedlaczek 1995	Probably, *S.* sp [1404]						**1823**
White-Tailed Deer (*Odocoileus virginianus*)							
S. odoi Dubey and Lozier 1983	1050 × 260 μm long, 6.5–12 μm thick wall	No data	Vp 5–10 thick, type 10a	9.5–12.5 × 2.5–3.5	Cat, 11–15 × 9–11	No data	**378**
S. odocoileocanis Crum, Fayer, and Prestwood 1981	Up to 620 μm long, 2–3 thick, striated	No data	Vp up to 4 long, convoluted invaginations at the base, type 17	9.5–14 × 2.8–4.6	Dog, cat, red fox, coyote, gray fox (*Urocyon cinereoargenteus*), wolf	No data	**46, 266, 378**
S. sp. Dubey and Lozier 1983	7–11 μm thick wall	No data	Vp 3.2–4.4, type 15	11–14.5 × 2.4–3.4	Unknown	No data	**46, 378**
S. sp. type II Atkinson, Wright, Telford, Mclaughlin, Forrester, Roelke, and Mccown 1993	77.6 μm wide, thick wall, striated	No data	Two types IIA and IIB, both with narrow vp, similar to type 36	No data	Unknown	No data	**46**

(*Continued*)

Table 18.1 (*Continued*) *Sarcocystis* Species in Wild Cervids

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
			Mule Deer (*Odocoileus hemionus*)				
S. americana Dubey and Speer 1986	Microscopic, up to 1500, 2–10 thick, hairy	No data	No data	No data	Unknown	No data	**383, 385**
S. hemioni Dubey and Speer 1986	Microscopic, up to 800 μm, wall 5.0–8.5 thick	No data	Vp 3.6–7.1 × 06–1.3, type 10d	10–13 × 2–4	Unknown	No data	**383, 385**
S. hemionilatrantis Hudkins and Kistner 1977	Up to 535 μm, 1–2 μm thick wall	No data	Tee-shaped vp, type 17	13.3–16.0 × 2.6–3.2	Dog, coyote, 14–16 × 9.5–11.0	No data	**383, 858**
S. youngi Dubey and Speer 1986	Microscopic, up to 800, wall 4–8 thick	No data	Vp 8.1–9.0 × 3.5–5.6, type 16	10.7–15.8 × 3.2–3.6	Unknown	No data	**383, 385**
			Reindeer (*Rangifer tarandus tarandus*)				
S. grueneri Yakimoff and Sokoloff 1934	Rounded ends, sack-like, 580 × 140	Thin strip-like vp	Ribbon-like up to 4.6 × 0.5 × 0.05, type 8a	13.9–16.5 × 2.4–4.8	Fox (silver and blue), raccoon, dog	No data	**281, 283, 685, 690, 698**
S. hardangeri Gjerde 1984	Ovoid, macroscopic, up to 2 mm	Prominent, slanting tongue-like vp	Tongue-like 25 × 5 × 1, type 26	11.2–12.9 × 2.8–3.5	Unknown	18S rRNA	**281, 283, 686, 698**
S. rangi Gjerde 1984	Thread-like up to 16 mm	Long hair-like vp	Hair-like up to 12.6 × 0.3–0.6, type 7a	12.4–16.5 × 2.4–4.6	Blue fox (*Alopex lagopus*)	18S	**281, 283, 687, 697, 693, 698**
S. rangiferi Gjerde 1984 (also found in red deer)	Cigar-like shaped 400 × 2106	Upright finger-like 7 μm long vp	Villiform 13.2 × 6.7, type 15	10.4–13.0 × 2.9–4.2	Unknown	18S, ITS-1, *cox*1	**281, 283, 685, 692, 698, 1682**

(*Continued*)

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Table 18.1 (*Continued*) *Sarcocystis* Species in Wild Cervids

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall SEM	Sarcocyst: Wall TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
S. tarandi Gjerde 1984	Spindle shaped 1000 × 80	Vp upright, polygonal to oval	Villiform 9.2 × 2.2, type 10e	9.7–13.0 × 2.5–4.0	Unknown	18S	**281, 283, 685, 696, 698**
S. tarandivulpes Gjerde 1984	Spindle shaped up to 3 mm	Short knob-like vp interconnected by microfolds	Knob-like 0.6–1.2 type 17	—	Fox (blue and silver), raccoon (*Nyctereutes procyonides*), dog, 13.6 × 9.8	18S	**281, 283, 688, 695, 698**
Red Deer (*Cervus elaphus hippelaphus* and *hispanicus*)							
S. cervicanis Hernández-Rodríguez, Navarrete, and Martínez-Gómez 1981	150 to 200 μm long, wall smooth and thin (0.5 μm)	No data	Hair-like vp up to 1.4 μm long and 32 nm wide and bifurcate at the tip, type 8a	15–17 × 4–6	Dog, 15.1–17.1 × 10.3–11.9	No data	**797**
S. elongata (Gjerde 1984) Gjerde (2014)	Up to 2.3 mm, slender and spindle-shaped, striated, thick walled, 7–8 × 1.5–2.0 μm	Upright finger-like hexagonal vp, 7–8 μm long, hexagonal	No data from this host	No data	Unknown	18S, ITS-1, *cox*1	**285, 704**
S. hardangeri Gjerde 1984 (also found in reindeer)	Macroscopic, ovoid up to 1.9 mm, fibrous covering	Loosely packed flattened protrusions 10–18 × 2	No data from this host	No data	Unknown	18S, ITS-1	**285, 704**
S. hjorti Dahlgren and Gjerde 2010 (also found in moose)	Slender and spindle-shaped, up to 3.1 mm long	Slender, thin hair-like protrusions 10–12 μm, narrow at base, tapered	No data	No data	Unknown	18S, ITS-1, *cox*1	**285, 704**

(*Continued*)

Table 18.1 (*Continued*) *Sarcocystis* Species in Wild Cervids

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
S. ovalis Dahlgren and Gjerde 2008	Macroscopic, ovoid up to 1.9 mm, fibrous covering	Loosely packed flattened tongue shaped protrusions 10–18 × 2	No data from this host	No data	Unknown	18S, ITS-1, *cox*1	**282, 285, 704**
S. tarandi Gjerde 1984 (also found in reindeer)	Spindle shaped, up to 2.3 mm, tapering pointed ends	Polygonal 6 μm long vp, aligned in rows	Villiform vp 9.2 × 2.2, type 10e	No data	Unknown	18S, ITS-1, *cox*1	**280, 285, 1477**
S. truncata (Gjerde 1984) Gjerde (2014)	Cigar-shaped to ellipsoidal, up to 1.7 mm	Upright 7 × 3 vp, hexagonal base, minute pits on tips	No data from this host	No data	Unknown	18S, ITS-1, *cox*1	**285, 704**
Wapiti or North American Elk (*Cervus elaphus canadensis*)							
S. sybillensis Dubey, Jolley, and Thorne 1983	Up to 637 μm long, wall up to 8 μm thick and hairy	No data	Up to 6.1 μm long vp, 0.3 μm wide at the base and 0.1 μm at the apex, have a central core, and the villar tips bifurcate, type 12	9.5–12 × 2.5–4	Dog, 15–17 × 10.5–12	No data	**380**
S. wapiti Speer and Dubey 1982	Up to 837 μm long. The sarcocyst wall is thin (<1 μm)	No data	Stubby, finger-like vp, folded over the PVM, slender invaginations, type 2	14–18 × 1.5–4	Coyote, dog, 15.7–17.4 × 9.6–11.9	No data	**1651**
S. sp. Stolte, Bockhardt, and Odening 1996	Thick wall, striated	Ear-like vp	Unknown	No data	Unknown	No data	**1680**

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Table 18.2 ***Sarcocystis*** **Species in Other Wild Ruminants and Large Animals**

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
African Buffalo (*Syncerus caffer*)							
S. cafferi Dubey, Lane, Wilpe, Suleman, Reininghaus, Verma, Rosenthal and Mtshali 2014	Macrocysts, up to 12 mm long, cashew nut-like	Mesh-like structure with irregular shaped vp	3.6 μm thick walled, highly branched vp, up to 3 μm long, type 23b	12.1 × 2.7 μm	Unknown	18S, *cox*1	**473, 1434**
Springbok (*Antidorcas marsupialis*)							
S. gazellae Balfour 1913 (originally described from Grant's gazelle, *Gazella rufifrons*)	Macroscopic, up to 2.8 mm, smooth	No data	Vp finger-like, up to 11 μm wide, type 14	12–16 × 5–6	Unknown	No data	**62, 1320**
S. woodhousei (Dogiel 1915) Wenyon 1926	850 μm long, microscopic	No data	Tooth-like vp 0.9–7.0 × 1.3–4.5 μm, type 29a	10–12 × 3.0–3.4	Unknown	No data	**1320**
S. sp. Odening, Rudolph, Quandt, Bengis, Bockhardt, and Viertel 1998 Also described in *Aepyceros melampus*	Microscopic, 1.6 mm long, smooth	No data	Hair-like vp, 0.1–0.6 μm wide, type 7a	10.5–17.0 × 3.2–4.5	Unknown	No data	**1320**
Impala (*Aepyceros melampus*)							
S. melampi Odening, Rudolph, Quandt, Bengis, Bockhardt, and Viertel 1998	Microscopic, 1.6 mm	No data	Wart-like vp, flat, 4.6–7.0 μm wide, 1.0–2.5 μm high, type 40	11.0–13.5 × 3.2–3.4	Unknown	No data	**1320**
Defassa Waterbuck (*Kobus ellipsiprymnus defassa*)							
S. nelsoni Mandour and Keymer 1970	5 mm in esophagus, microscopic in heart, 4–5 μm, thin wall	No data	No data	No data	Unknown	No data	**1105**

(*Continued*)

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Table 18.2 (*Continued*) *Sarcocystis* Species in Other Wild Ruminants and Large Animals

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
			Saiga (*Saiga tatarica*)				
S. saiga Pak, Sklyarova, and Dymkova 1991	Thread-like, up to 3 mm long, striated, 4.2–6.5 μm long vp	No data	No data	11.2–14.0 × 4.2–5.6	Unknown	No data	**1359**
			Greater Kudu (*Tragelaphus strepsiceros*)				
S. cf. *hominis*	Microscopic, 2.5 mm	No data	Tombstone- or cone-like vp, type 15	12–15 × 2.6–3.0	Unknown	No data	**1320**
			Mongolian Gazelle (*Procapra gutturosa*)				
S. danzani Odening, Stolte, Lux, and Bockhardt 1996	Microscopic, 1.5 mm long	Molar-like vp, with a hollow from the top view	Molar tooth-like vp, 1.2–2.2 wide, type 29	11–16 × 3–4	Unknown	No data	**1313**
S. mongolica Machul'skii 1947	Macroscopic, secondary wall, rice grain like, up to 5.1 mm long	Visible small invaginations	Unusual small invaginations, type 1c	16–21 × 3–6	Unknown	No data	**1089, 1313**
S. sp. Odening, Stolte, Lux, and Bockhardt 1996	Microscopic, up to 3.8 mm long, thin wall	Hair-like vp	Thin wall (<0.5 μm), 8–15 μm long hair-like vp, type 7a	12.5–14.5 × 3.0–3.5	Unknown	No data	**1313, 1680**
			Addax (*Addax nasomaculatus*)				
S. cf. *medusiformis* Stolte, Odening, and Bockhardt 1996	Up to 2.8 mm long	No data	Scale-like or trapezoidal vp, 0.8–2.6 μm long, type 20	14.0–17.8 × 2.8–4.0	Unknown	No data	**1679**
			Blackbuck (*Antilope cervicapra*)				
S. cf. *capracanis* Stolte, Odening, and Bockhardt 1996	Up to 1.0 mm long	No data	Palisade-like texture with finger-like vp, 3.4–4.4 μm long, type 14	13.5 × 4.5	Unknown	No data	**1679**

(*Continued*)

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Table 18.2 (*Continued*) *Sarcocystis* Species in Other Wild Ruminants and Large Animals

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
Common Eland (*Taurotragus oryx*)							
S. cf. *hominis* Stolte, Odening, and Bockhardt 1996	Up to 800 μm long	No data	Tombstone- or cone-like vp, 6.8–8.2 μm long, type 15	No data	Unknown	No data	**1679**
Nilgai (*Boselaphus tragocamelus*)							
S. cf. *cruzi* (also found in eland and blackbuck)	Up to 3.5 μm long	No data	Long hair-like villar protrusions, 13 μm, type 7a	15.0–17.8 × 3.0–4.2	Unknown	No data	**1679**
Mountain Goat (*Oreamnos americanus*)							
S. sp. Foreyt 1989	Microscopic, up to 248 μm long	No data	Mushroom-shape vp, type 24	No data	Not canids, felids, bears, several Mustelidae, pigtail macaque, hawk, and owl	No data	**619**
Siberian Ibex (*Capra sibirica*)							
S. orientalis Machul'skii and Miskaryan 1958	Macroscopic, up to 7.5 mm long	No data	No data	14.1–9.2 × 6.2–2.3	Unknown	No data	**1089**
Chamois (*Rupicapra rupicapra*)							
S. cornagliai Odening, Stolte, and Bockhardt 1996	Microscopic, up to 700 μm long	No data	Thin wall (<0.77 μm), mushroom-shaped, type 24	11.5–13.5 × 3–4	Unknown	No data	**1311**
S. sp. Odening, Stolte, and Bockhardt 1996	Microscopic, up to 1.9 mm log	No data	0.2–1.3 μm thick wall, finger-shaped vp, type 14	12.0–12.5 × 3.1–4.3	Unknown	No data	**1311**

(*Continued*)

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Table 18.2 (*Continued*) *Sarcocystis* Species in Other Wild Ruminants and Large Animals

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
Alpine Ibex or Steinbock (*Capra ibex*)							
S. arieticanis-like (type III) Cornaglia, Giaccherino, and Peracino 1998	No data	No data	Discontinuous elongate digitations, similar to type 7a	No data	No data	No data	**253**
S. capracanis-like (type II) Cornaglia, Giaccherino, and Peracino 1998	No data	No data	Villus-like projections in palisade position, similar to type 14	No data	No data	No data	**253**
S. cornagliai-like (type I) Cornaglia, Giaccherino, and Peracino 1998	No data	No data	Sessile and polyp-shaped (mushroom) vp, similar to type 24	No data	No data	No data	**253, 103**
Hispanic Ibex (*Capra pyrenaica hispanica*)							
S. capricornis-like (type IV) Cornaglia, Giaccherino, and Peracino 1998	No data	No data	Finger-like projections with indented edges, fire flame-like, similar to type 41	No data	No data	No data	**253**
Himalayan Blue Sheep (*Pseudois nayaur*)							
S. pseudois (Syn. *S.* cf. *capracanis*) Novak, Fedossenko, and Orazalinova 1987) Odening, Stolte, and Brockhardt 1996	Up to 2.2 mm long, thick wall, striated	No data	1.0–2.4 μm thick wall, 2.0–2.7 μm long irregular finger-like vp, type 14	12.5–13.5 × 3.3–5.0	Unknown	18S	**608, 1284, 1309, 1317**
Himalayan Goral (*Naemorhedus goral*)							
S. sp. Agrawal, Chauhan, and Ahluwalia 1982	Macroscopic, up to 5 mm long, 2 mm wide. 3.7–5.6 μm thick wall	No data	No data	7.5–13.1 × 1.8–2.8	Unknown	No data	**1898**

(*Continued*)

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Table 18.2 (*Continued*) *Sarcocystis* Species in Other Wild Ruminants and Large Animals

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (µm)	Molecular Markers Studied	Reference
Japanese Serow (*Capricornis crispus*)							
S. capricornis Odening, Stolte, and Bockhardt 1996	Up to 4.8 mm long, striated	No data	3.3–6.4 µm thick wall, T-shaped or short, thick nails with angular head vp, 2.5–3.5 µm long, type 27	14–17 × 3–4	Unknown	No data	**1309**
Mouse Deer (*Moschiola meminna*)							
S. sp. Kannangara 1970	Up to 345 µm long, thick, striated	No data	No data	7.2–4.3 × 2.4–1.7	No data	No data	**941**
Bighorn Sheep (*Ovis canadensis*)							
S. ferovis Dubey 1983	Microscopic, up to 780 µm, thin <1 µm, smooth	No data	Flattened and mushroom-like vp, type 3a	10–14 × 3–3.5	Coyote 13–15 × 9–11 (not transmissible to sheep, goat, cattle)	No data	**368**
European Mouflon (*Ovis ammon musimon*)							
S. tenella Raillet 1886	(See Chapter 8)						**1277, 1308**
S. arieticanis Heydorn 1985	(See Chapter 8)						**1277, 1308**
Hippopotamus (*Hippopotamus amphibius*)							
S. africana Odening, Quandt, Bengis, Stolte, and Bockhardt 1997	Microscopic, round to oval, up to 720 µm wide	No data	Rectangular or semicircular vp, 2.5–5.0 × 3.5–5.0, type 15b	7.0–11.0 × 2.6–3.7	Not known	No data	**1316**
S. hippopotami Odening, Quandt, Bengis, Stolte, and Bockhardt 1997	Macroscopic, up to 10 mm, fibrous capsule	Fibrous capsule visible	Thumb-like vp, 1.2–2.4 × 0.7–1.8, type 33	15.6 × 2.2	Not known	No data	**1316**

(*Continued*)

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Table 18.2 (*Continued*) *Sarcocystis* Species in Other Wild Ruminants and Large Animals

Species	Sarcocyst: Shape, Size	Sarcocyst: Wall: SEM	Sarcocyst: Wall: TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Markers Studied	Reference
			Warthog (*Phacochoerus aethiopicus*)				
S. dubeyella Stolte, Odening, Quandt, Bengis, and Bockhardt 1998	Macrocysts, up to 12 mm, fibrous capsule	Indented margins	Rectangular vp, 2–10 μm long, 2.8–11.0 μm wide, type 30	12–19 × 2.5–3.5	Unknown	No data	**1684**
S. phacochoeri Stolte, Odening, Quandt, Bengis, and Bockhardt 1998	Macrocysts, up to 4 mm, thin cyst wall	Hollow present on one side of the vp	Molar-like vp, 1.6–3.3 × 1.7–3.3, type 29b	12–16 × 2.0–3.5	Unknown	No data	**1684**
			Yak (*Poephagus grunniens*)				
S. poephagi Wei, Chang, Duong, Wang, and Xia 1985	Macroscopic, filiform, 4 mm, thick wall 7.4 μm, secondary cyst wall	Tree-bark-like surface	Thick	No data	Cat, oocysts were 13.7 × 9.4	No data	**1815, 1817**
S. poephagicanis Wei, Chang, Duong, Wang and Xia 1985	Macroscopic, oval, 0.28 mm long, thin wall, secondary cyst wall	Honeycomb-like surface, smooth	Thin, spines	No data	Dog, oocysts were 14.6 × 10.6	No data	**1815, 1817**
			Giraffe (*Giraffa camelopardalis*)				
S. camelopardalis Bengis, Odening, Stolte, Quandt, and Bockhadt 1998	Microscopic, <1 mm long, 120 μm wide	No data	Strap-like vp, 2–3 μm long, type 36	9–14 × 2–4	Unknown	No data	**91**
S. giraffae Bengis, Odening, Stolte, Quandt, and Bockhadt 1998	Macroscopic, slim, up to 11 mm long, collagen capsule	No data	Finger-shaped vp, 6–13 μm long, type 34	14–18 × 2.5–3.5	Unknown	No data	**91**
S. klaseriensis Bengis, Odening, Stolte, Quandt, and Bockhadt 1998	Microscopic, filiform, <1 mm long, 105 μm wide	No data	Finger-like vp, 4–7 μm long, type 35	9–14 × 2.6–4.0	Unknown	No data	**91**

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Table 18.3 ***Sarcocystis*** **Species in Marine Mammals**

Intermediate Host	Species	Shape, Size	TEM	Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular Assays	References
Sea lion (*Zalophus californianus*)	*S. hueti* (Moulé, 1888) Labbé 1899	Up to 4 mm long	No data	4–5	No data	No data	**1013, 1232, 1317**
	S. sp. Mense, Dubey, and Homer 1992	Up to 100 μm wide	Thin, 0.5–1.2 μm, sloping-to-straight 1.5 μm vp, folded down the surface, type 11e.	No data	No data	No data	**1184**
Harbor seal (*Phoca vitulina richardsi*)	*S. richardi* Hadwen 1922	Macroscopic, up to 2 cm long	No data	10 × 2.5	No data	No data	**766**
Antarctic leopard seal (*Hydrurga leptonyx*)	*S. hydrurgae* Odening and Zipper 1986	Up to 13 mm long	Thin, type 1e	6.1 × 2.2	No data	No data	**2195**
Bearded seal (*Erignathus barbatus*)	*S.* sp. Bishop 1979	Location in tongue. No data	No data	No data	No data	No data	**105**
Ringed seal (*Pusa hispida*)	*S.* sp. Migaki and Albert 1980	Up to 550 μm long	Thin wall (0.8–1.0 μm)	Comma-shaped, 12–10 × 3–2	No data	No data	**1192**
Northern fur seal (*Callorhinus ursinus*)	*S.* sp. Brown, Smith, and Keyes 1974	Numerous cysts in several muscles. No data	No data	No data	No data	No data	**147**
Whale (*Balaenoptera borealis*)	*S. balaenopterialis* Akao (1970)	Thread-like, macroscopic, 10–20 cm long, 0.5 mm wide	Thin, 2–3 μm, type 1a	7.7 × 2.3	No data	No data	**22**
Beluga whale (*Delphinapterus leucas*)	*S.* sp. De Guise, Lagacé, Girard, and Béland 1993	Microscopic, up to 280 μm wide, >1 mm long	Thin, type 1a	No data	No data	No data	**310**
Sperm whale (*Physeter catodon*)	S. sp. Munday and Mason 1978	2.5 mm long × 2.5 mm wide	Thin and smooth, type 1a	No data	No data	No data	**1249**
Atlantic white-sided dolphin (*Lagenorhyncus acutus*)	*S.* sp. De Guise, Lagacé, Girard, and Béland 1993	Microscopic, up to 115 μm wide	Thin	No data	No data	No data	**310, 562**
Delphinus sp.	*S.* sp. Munday and Mason 1978	750 × 80 μm	Thin	No data	No data	No data	**1249**
Striped dolphin (*Stenella coeruleoalba*)	*S.* sp. Dailey and Stroud (1978)	No data	No data	No data	No data	No data	**287**
Sea otter (*Enhydra lutris*)	*S.* sp. Dubey, Lindsay, Rosenthal, and Thomas 2003	Microscopic, 110 μm long	Thin, 0.5–0.7 μm, type 1a	5.0–5.7 × 1.6–1.9	No data	rpoB	**453**

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Data on natural epizootiology of *Sarcocystis* infections in mule deer indicated that *S. hemionilatrantis* affects the growth of fawns and, coupled with winter conditions, probably predisposes infected deer to predation.[384]

18.2 *SARCOCYSTIS* INFECTIONS IN OTHER WILD RUMINANTS AND OTHER LARGE ANIMALS

Numerous *Sarcocystis* species have been named from animals in zoological gardens, wild ruminants, herbivores, and several omnivores. Some are summarized in Tables 18.1 and 18.2. It is likely that wild goats and feral sheep share species that are also present in domestic animals. This is the case in mouflon (*S. tenella*)[1308] and wood bison (*S. cruzi*).[162] Some *Sarcocystis* species of wild animals are host specific, such as *S. ferovis* in bighorn sheep (Table 18.2) which is morphologically different from any species in domestic animals and is not transmissible to ox, sheep, or goats.[368] Another structurally distinct, unnamed, and enigmatic species was described from the North American mountain goat (*Oreamnos americanus*).[619] It was found in 24 (43%) of 56 goats from Washington state, USA. None of the 4 coyotes, 8 dogs, 4 domestic cats, 3 bears, 2 raccoons, 2 martens, 2 fishers, 3 skunks, 5 minks, 5 ferrets, 1 pigtail macaque, 2 red tailed hawks, or 1 great horned owl fed infected meat excreted sporocysts.[619] A structurally similar sarcocyst, *Sarcocystis*

Table 18.4 Species *inquirenda*/Uncertain Status

Species	Intermediate host	Morphology	References
S. ruandae Chiwy and Colback 1926	Unnamed antelope	Microscopic cysts, striated wall, 9–12 × 1–5 μm banana-shaped bradyzoites	**216**
S. sp. Dubey 1980	Pronghorn (*Antilocapra americana*)	Microscopic cysts, 127–297 × 63–95 μm, thin walled and smooth	**355**
S. bubalis Diegel 1915	Coke's hartebeest (*Alceslaphus cokei*)	Up to 2 mm long, striated wall	**1105**
S. woodhousei Diegel 1915	Grant's gazelle (*Nanger granti*)	Up to 1.5 mm long, with rods or rhombic prisms, striated wall	**1105**
S. gusevi (Blyth 1841) Krylov and Saponzhnikov 1965	Arkhar (*Ovis ammon polii*)	Only description of bradyzoites was provided, they were lunate- or bean-shape, 10.8–14.4 × 4.8–8.4 μm	**981**
S. sp. Krylov and Saponzhnikov 1965	Bactrian deer (*Cervus elaphus bactrianus*)	No description of sarcocysts. Bean shaped, and orange slice-shaped 10.8–15.6 × 4.8–8.4 μm bradyzoites	**981**
S. sp. Rioseco, Cubillos, Gonzalez and Diaz 1976	Pudu (*Pudu pudu*)	Microscopic	**2252**
S. sp. Samuel and Gray 1974	Muskox (*Ovibos moschatus*)	Microscopic	**2281**
S. sp. Gangadharan, Valsala, Nair and Rajan 1992	Sambar deer (*Cervus unicolor*)	Microscopic	**660**
S. sp. Shrivastav, Sharma, Chaudhry, and Malik 1999	Barasingha or hard-ground swamp deer (*Cervus duvauceli branderi*)	Microscopic	**1605**

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cornagliai was described from the European chamois (*Rupicapra rupicapra*)[1311]; it was concluded that sarcocysts in the North American mountain goat were morphologically identical to *S. cornagliai*. Another example of the complexity of the epidemiology of *Sarcocystis* genus in wildlife was the finding of sarcocysts resembling *S. gracilis* of roe deer (Cervidae) in muscles of dwarf zebu (Bovidae) born and housed in a German zoo.[1312]

18.3 *SARCOCYSTIS* INFECTIONS IN MARINE MAMMALS

Information concerning *Sarcocystis* infections in marine mammals is limited. Several species have been proposed and a high prevalence was found.[562] Further studies should focus on detailed descriptions of the ultrastructure. Information regarding *Sarcocystis* spp. in marine mammals is summarized in Table 18.3.

18.4 *SARCOCYSTIS* SPP. WITH UNCERTAIN STATUS

Several species detected in wild animals are considered *inquirendae* based on poor descriptions or because there is only one report.[1317] Several examples are given in Table 18.4.

BIBLIOGRAPHY

Taxonomy and prevalence of *Sarcocystis* species in wild ruminants and other large animals can be found in the following papers:

Alpine ibex and Hispanic ibex: **103, 253, 1311**.

Antelopes: **62, 1105, 1205, 1320, 1679**.

Fallow deer: **337, 338, 542, 799, 1404, 1455, 1823**.

Mule deer: **379, 383, 384, 385, 858, 859**.

Sika, sambar, Barasingha deer: **121, 660, 1454, 1515, 1605**;

Red deer (wapiti): **61, 121, 238, 285, 337, 338, 704, 796, 797, 1000, 1102, 1664, 1676, 1763, 1824, 1961, 1997, 2100, 2132, 2212, 2291, 2322**.

Roe deer: **97, 110, 284, 336, 339, 531, 532, 534, 538, 548, 700, 969, 998, 1085, 1102, 1386, 1533, 1577, 1664, 1682, 1683, 1692, 1763, 1997, 2054, 2088, 2212, 2268, 2322**.

Reindeer (caribou): **280, 281**, **283, 685–698, 953, 1682, 2083, 2349**.

White-tailed deer: **46, 266, 267, 480, 533, 1995, 1996, 2079**.

Elk: **380, 620, 1014, 2125**.

European mouflon: **1277, 1308, 1664, 2266**.

Moose: **244, 245, 282, 284–286, 699, 700, 702, 704, 999, 1477, 2080**.

Other wild ruminants: **103, 110, 253, 368, 529, 619, 660, 894, 929, 1277, 1308, 1309, 1311, 1312, 1342, 1389, 1434, 1446, 1815, 1816, 1915, 1980, 1995, 2074, 2086, 2128, 2146, 2147, 2180, 2197–2199, 2206, 2207, 2224, 2225, 2235, 2266, 2338, 2340**.

Marine mammals: **22, 105, 108, 147, 206, 265, 287, 310, 453, 562, 766, 930, 1184, 1192, 1249, 1317, 1588, 2193–2196, 2208**.

Miscelanea: **62, 91, 432, 1028, 1102, 1314, 1315, 1317, 1321, 1418, 1664, 1680, 1898, 1915**.

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CHAPTER 19

Sarcocystosis in Wild Terrestrial Carnivores

19.1 INTRODUCTION

As seen in previous chapters wild carnivores serve as definitive hosts for numerous *Sarcocystis* species.[286,374,383] Prevalence ranges from 4% to 92%.[94,246,364,504,953,1122,1837] Carnivores can also act as intermediate hosts harboring sarcocysts (Table 19.1).

19.1.1 Prevalence and Clinical Disease

The prevalence of sarcocysts in the muscles of wild carnivores might be higher than previous estimates. Muscle infections have been reported in more than 21 terrestrial species. Sarcocysts were reported in 6 of 21 wild dogs (*Lycaon pictus*) from Kenya,[159] and the prevalence in raccoons (*Procyon lotor*) ranges between 47.5% and 66% in different U.S. states.[776,964,1633] Sarcocysts have been found in 4 of 5 feral skunks (*Mephitis mephitis*) from Oregon,[446] in 80.4% of wolverines (*Gulo gulo*) from Canada,[467] in 83% of fishers (*Martes pennanti*) from Pennsylvania,[1025] and in 2 coyotes from Oklahoma.[268]

The first record of *Sarcocystis* in bears reported a prevalence of 11% in black bears (*Ursus americanus*) from southeastern and northeastern United States.[265] Several cases of infection have been reported in North Carolina, Oregon, Florida, and Pennsylvania.[206,415,462,621] Sarcocysts were also reported in brown bears (*Ursus arctos*) from Alaska.[461]

Among wild Felidae the prevalence of *Sarcocystis* is very high. A prevalence of 50% was found in Florida bobcats (*Felis rufus floridanus*),[29] and infections were found in 11 of 14 Florida panthers and 4 of 4 cougars (*Felis concolor*).[732] Infection was reported in a young leopard (*Panthera pardus*)[1644] and in 2 lions (*Panthera leo*), both from India.[100] Sarcocysts were detected in a lion from Kenya and in 4 lions from Namibia,[410,962] as well as 7 of 10 cheetahs (*Acinonyx jubatus*) in a breeding colony in Oregon, USA.[142]

Clinical sarcocystosis has been described in carnivores. Numerous reports attributed to *S. neurona* and *S. canis* infections have been summarized, respectively, in Chapter 3. A case of generalized eosinophilic myositis was reported in a raccoon from Pennsylvania, and although numerous sarcocysts were present, *Sarcocystis* could not be confirmed as the cause.[776] In a case of meningoencephalitis in a young male Japanese raccoon dog (*Nyctereutes procyonoides viverrinus*), *Sarcocystis* schizonts were detected in the brain, causing severe encephalitis specially in the cerebellum.[983] The protozoa reacted weakly with *S. cruzi* antiserum, and the analysis of the partial 18S rRNA gene sequence revealed its close relationship with a *Sarcocystis* sp. isolated from a white-fronted goose (*Anser albifrons*) presumably *S. albifronsi*, with a high degree of similarity (99.3%–99.7%) to *S. neurona* and *S. canis*.

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Table 19.1 ***Sarcocystis*** **Species in Wild Terrestrial Carnivores**

Species	Sarcocyst: Shape, Size	Sarcocyst: SEM	Sarcocyst: TEM, Wall	Sarcocyst: Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular	References
Raccoon (*Procyon lotor*)[a]							
S. kirkpatricki Synder, Sanderson, Toivio-Kinnucan, and Blagburn 1990	Microscopic, up to 801 μm long, 2–3 μm thick, striated	No data	2–3 μm thick, striated, type 11b	5.2–3.9 × 2.0–1.2	Not dog or cat	No data	**1633**
S. sp. 1 Stolte, Odening, Walter, and Bockhardt 1996	Microscopic, 99.5 μm wide, only 1 cyst seen, palisade-like wall	No data	4.6–6.9 μm thick wall, vp 5.8–7.4 μm long, type 17	No data	Unknown	No data	**1681**
S. sp. 2 Stolte, Odening, Walter, and Bockhardt 1996	2 sarcocysts detected, 1.8 and 1.9 mm	No data	Thin, type 7a	14.0–17.1 × 3.4–3.9	Unknown	No data	**1681**
S. sp. 3 (sf. *S. sebeki*) Stolte, Odening, Walter, and Bockhardt 1996	Up to 18 mm, thin wall, smooth	No data	Thin, smooth, type 1a	6.6–8.5 × 1.3–1.8	Unknown	No data	**1681**
Arctic Fox (*Vulpes lagopus*)							
S. arctica Gjerde and Schulze 2014	Up to 12 mm, serrated, knob-like protrusions on wall	No data	No data	No data	Unknown	18S, ITS-1, cox1	**705**
S. lutrae Gjerde and Josefsen 2015	Up to 970 μm long, thin wall (0.5 μm)	No data	No data	No data	Unknown	18S, 28S, ITS-1, cytb, coxI	**706**
Red Fox (*Vulpes vulpes)*							
S. sp. Pak 1991	Up to 3.9 mm long, 2.0–2.8 μm, thick wall, smooth	No data	No data	2.8–1.4 × 9.8–8.4	Unknown	No data	**1359**
Corsac Fox (*Alopex corsac*)							
S. corsaci Pak 1979	Up to 8.2 mm, cyst wall with two layers. Wall 2.8–2.1 μm thick	No data	-	2.8–1.4 × 8.4–7.0	No data	No data	**1360**

(*Continued*)

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Table 19.1 (*Continued*) *Sarcocystis* Species in Wild Terrestrial Carnivores

Species	Sarcocyst: Shape, Size	Sarcocyst: SEM	Sarcocyst: TEM, Wall	Sarcocyst: Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular	Reference
Jackal (*Canis melomelas*)							
S. sp. 1 Wesemeier, Odening, Walter, and Bockhardt 1995	Up to 393 μm, thick wall	No data	Up to 6.7 μm palisade-like vp, type 10f	No data	Unknown	No data	**1825**
S. sp. 2 Wesemeier, Odening, Walter, and Bockhardt 1995	Up to 695 μm, thin wall	No data	Flat, mushroom-like vp, type 3b	No data	Unknown	No data	**1825**
Coyote (*Canis latrans*)							
S. sp. Cummings, Kocan, Barker, and Dubey 2000	Microscopic, 99 μm, thin cyst wall	No data	Type 1b	5.0 long	Unknown	No data	**268**
Wolverine (*Gulo gulo)*							
S. kalvikus Dubey, Reichard, Torretti, Garvon, Sundar, and Grigg 2010	Macroscopic, 900 μm, smooth, thin wall	No data	Type 1a	5 × 1	Unknown	18S, ITS-1	**467**
S. kitikmeotensis Dubey, Reichard, Torretti, Garvon, Sundar, and Grigg 2010	Microscopic, 1100, thick wall	No data	vp 1 μm long, type 9 h	9–13 × 2–3	Unknown	18S, ITS-1	**467**
Wild Dog (*Lycaon pictus*)							
S. sp. Bwangamoi, Ngatia, and Richardson 1993	Microscopic, up to 830 μm, 3 μm thick wall	No data	No data	No data	Unknown	No data	**159**
European Badger (*Meles meles)*							
S. hofmanni Odening, Stolte, Walter, and Bockhardt 1994	Microscopic, up to 1.2 mm long	No data	vp up to 6.6 μm long, finger-like, type 10b	8.3–9.9 × 2.6–3.6	Unknown	No data	**1303, 1304**
S. melis Odening, Stolte, Walter, and Bockhardt 1994	2–5 mm long, thin wall	No data	Thin, 1f	8.2-6.9 × 1.7-0.8	Unknown	No data	**1303, 1304**

(*Continued*)

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Table 19.1 (*Continued*) *Sarcocystis* Species in Wild Terrestrial Carnivores

Species	Sarcocyst: Shape, Size	Sarcocyst: SEM	Sarcocyst: TEM, Wall	Sarcocyst: Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular	Reference
S. cf. *sebeki* Odening, Stolte, Walter, and Bockhardt 1994	Up to 9 mm long	No data	Thin, wavy, type 1a	6.1–7.2 × 1.6–2.2	Unknown	No data	**1303, 1304**
S. cf. *gracilis* Odening, Stolte, Walter, Bockhardt, and Jakob 1994	Up to 1.2 mm long	No data	Thin, 1.7 μm thick wall, type 1g	13.2–11.0 × 3.5–2.7	Unknown	No data	**1304**
Japanese Badger (*Meles meles anakuma*)							
S. sp. Kubo, Okano, Ito, Tsubota, Sakai, and Yanai 2009	Up to 380 μm long	No data	2.3–1.8 μm thick, type 12	No data	Unknown	No data	**982**
Otter (*Lutra lutra*)							
S. lutrae Gjerde and Josefsen 2015	Up to 970 μm long, thin wall (0.5 μm)	No data	No data	No data	Unknown	18S, 28S, ITS-1, cytb, coxI	**706**
S. sp. Wahlström, Nikkila, and Uggla 1999	Up to 2.3 mm long, thin wall	No data	Thin 2 μm, type 1b	No data	Unknown	No data	**1803**
Skunk (*Mephitis mephitis*)[a]							
S. erdmanae (Erdman 1978) Odening 1997	Microscopic, up to 300 μm long	No data	No data	No data	Dog, 12 × 10	No data	**555, 1314**
S. mephitisi Dubey, Hamir, and Topper 2002	Microscopic, up to 400 μm long, >6 μm thick wall	No data	vp up to 5 long, constricted at the base, leaf-like, type 9 m.	11 × 3.2	Unknown	No data	**446**
Mink (*Mustela vison*)							
S. sp. (most likely *S. neurona*) Ramos-Vara, Dubey, Watson, Winn-Elliot, Patterson, and Yamini 1997	Microscopic, up to 400 μm long, thick wall	No data	vp up to 2 long, constricted at the base, leaf-like, type 11b	4.5–5.0	Unknown	No data	**1445**

(*Continued*)

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Table 19.1 (*Continued*) *Sarcocystis* Species in Wild Terrestrial Carnivores

Species	Sarcocyst: Shape, Size	Sarcocyst: SEM	Sarcocyst: TEM, Wall	Sarcocyst: Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular	Reference
Steppe Polecat (*Mustela eversmanii*)							
S. eversmanni Pak, Sklyarova, and Dymkova 1991	No data	No data	No data	2.8–3.0 × 7.0–8.4	Unknown	No data	**1314, 1359**
Common European Weasel (*Mustela nivalis*)							
S. sp. Tadros and Laarman 1979	Macroscopic, up to several mm long, smooth	No data	No data	9 × 2.5	Possibly tawny owl (*Strix aluco*)	No data	**1711**
Fisher (*Martes pennanti*)							
S. sp. Larkin, Gabriel, Gerhold, Yabsley, Wester, Humphreys, Beckstead, and Dubey 2011	Microscopic, 1.6 μm long vp	No data	No data	No data	Unknown	18S	**1025**
Black Bear (*Ursus americanus*)[b]							
S. ursusi Dubey, Humphreys, and Fritz 2008	Microscopic, thin wall, <1 μm, serrations	No data	vp up to 3.8 μm long, type 9g.	4.8–6.0 × 1.4–1.8	Unknown	No data	**415, 462**
S. sp. Dubey, Humphreys, and Fritz 2008	Microscopic, 700 μm long, 2 μm palisade-like wall, thick wall	No data	No data	No data	Unknown	No data	**462**
S. sp. Cheadle, Cunningham, and Greiner 2002 (From Florida black bear, *Ursus americanus floridanus*)	Microscopic, 181 μm long, thin wall	No data	No data	No data	Unknown	No data	**206**

(*Continued*)

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Table 19.1 (*Continued*) *Sarcocystis* Species in Wild Terrestrial Carnivores

Species	Sarcocyst: Shape, Size	Sarcocyst: SEM	Sarcocyst: TEM, Wall	Sarcocyst: Bradyzoites	Definitive Host, Sporocysts (μm)	Molecular	Reference
Brown Bear (*Ursus arctos*)[b]							
S. arctosi Dubey, Rosenthal, Sundar, Velmurugan, and Beckman 2007	Microscopic, 75 μm long, thin wall, 1 μm	No data	Type 1a	5.6–6.8 × 0.7–1.8	Unknown	No data	**461**
Wild Felids, Namely Bobcat (*Felis rufus*)[a,c], Cheetah (*A. jubatus*)[c], Lion (*P. leo*)[c], Florida Panther and Cougar (*Felis concolor*)[c]							
S. felis Dubey, Hamir, Kirkpatrick,Todd, and Rupprecht 1992	2.1 mm, cyst wall minute striations	No data	vp hob-nail, no microtubules	7.0–10.0 × 1.5	Unknown	No data	**407**
Leopard (*Panthera pardus*)							
S. sp. Somvanshi, Koul, and Biswas 1987	Thin wall	No data	No data	No data	No data	No data	**1644**

[a] Intermediate host for *S. neurona.*[204,446,1788]
[b] Intermediate host for *S. canis*.[305, 663, 1874]
[c] Wild Felidae as hosts for *S. felis*: bobcat,[29,407,732,1788] cheetah,[142] lion,[410,962] Florida panther and cougar.[732]

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19.2 *SARCOCYSTIS* SPECIES IN WILD CARNIVORES

In a survey of 10 wild carnivore species in Japan, 4 were found to be positive, namely, Japanese raccoon dog (*Nyctereutes procyonoides viverrinus*), Japanese red fox (*Vulpes vulpes japonica*), Japanese martens (*Martes melampus melampus*), and Japanese badger (*Meles meles anakuma*).[982] Two types of sarcocysts were detected; 1 with a thin wall and minute undulations (presumably TEM type 1) was common in canids and martens, and type 2, with a thick wall and finger-like villous projections containing microtubules, was detected in Japanese badgers (Table 19.1).

The species present in wild Felidae comprising lions, cheetahs, and bobcats, among others was named *Sarcocystis felis*[407] (Table 19.1). This species has been reported in the domestic cat[521,684] (see Chapter 15).

Data on *Sarcocystis* species infecting other frequently observed terrestrial carnivores as foxes, badgers, or coyotes are summarized in Table 19.1.

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29, 94, 100, 103, 142, 153, 159, 204, 206, 246, 265, 268, 286, 305, 364, 374, 383, 407, 410, 415, 446, 461, 462, 467, 504, 521, 546, 555, 621, 663, 684, 705, 706, 732, 776, 933, 953, 962, 964, 982, 983, 1025, 1122, 1187, 1303, 1304, 1314, 1359, 1360, 1445, 1633, 1644, 1681, 1711, 1788, 1803, 1825, 1837, 1874, 1894, 1905, 1907, 1939, 1940, 1958, 1996, 2002, 2035, 2045, 2102, 2168, 2181.

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CHAPTER 20

Sarcocystosis in Marsupials

20.1 INTRODUCTION

Opossums act as definitive and intermediate hosts for *Sarcocystis* species.

20.2 AMERICAN OPOSSUMS AS DEFINITIVE HOSTS FOR *SARCOCYSTIS* SPECIES

The North American opossum (*Didelphis virginiana*) and the 3 species of South American opossums (*Didelphis albiventris*, *Didelphis marsupialis,* and *Didelphis aurita*) are hosts for at least 4 species of *Sarcocystis*: *S. neurona* (discussed in Chapter 3), *S. falcatula,* and *S. lindsayi* (Chapter 17), and *S. speeri* (discussed here, Table 20.1).

20.2.1 Prevalence of *Sarcocystis* Sporocysts in Opossums

Sporocysts are common in opossums and the estimated prevalence varies with methods employed. Because sporocysts are trapped in the lamina propria, they are excreted in feces irregularly and sometimes none are detectable in feces although millions are present in the intestines. Based on the examination of intestinal digests, sporocysts were found in as many as 54.4% of

Table 20.1 American Opossums as Definitive Hosts for *Sarcocystis* Species

Opossum Species (*Didelphis* spp.)	*Sarcocystis* Species (Sporocysts)[a]	Place of Report	Main Reference
D. albiventris (White-eared opossum)	*S. falcatula*	Argentina	**418**
	S. lindsayi	Brazil	**433**
	S. neurona	Brazil	**442**
	S. speeri	Argentina	**426**
D. aurita[b] (Big-eared opossum)	*S. lindsayi*	Brazil	**1670**
D. marsupialis (Common opossum)	*S. falcatula*	Brazil	**441**
	S. neurona	Brazil	**442**
	S. speeri	Brazil	**427**
D. virginiana (Virginia opossum)	*S. falcatula*	USA	**137**
	S. neurona	USA	**601**
	S. speeri	USA	**416**

[a] KO mice are intermediate host for *S. speeri* under experimental conditions. Its natural intermediate host is unknown.

[b] Monteiro et al.[1209] reported DNA of *S. neurona*- and *S. falcatula*-like sporocysts in *D. aurita* and *D. albiventris* from Brazil.

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opossums (see Table 3.3). Identification of *Sarcocystis* species in opossum feces is difficult and expensive. Several methods are available for species identification, including morphology of sporocysts, molecular, and bioassays (see Chapter 2). Among these methods, sporocyst morphology is unreliable and DNA of not all *Sarococystis* species of opossums has been characterized for definitive identification. Currently, bioassay is the most definitive method for sporocyst identification. Bioassay in KO (knockout) mice and budgerigars can distinguish *S. falcatula* from *S. neurona* and *S. speeri*; *S. falcatula* is not infective to KO mice and *S. neurona* and *S. speeri* are not infective to budgerigars.[421] As indicated in Chapter 3, *S. neurona* does not produce sarcocysts in KO mice whereas *S. speeri* does (Figure 20.1). Based on the bioassays in KO mice and budgerigars, of the 44 opossums examined the prevalences were *S. speeri* 18.8%, *S. falcatula* 43.1%, and *S. neurona* 31.8%.[421] It has been suggested this diversity of *Sarcocystis* species in the intestine of opossums may enable sexual recombination contributing to their already allelic variability.[1209]

20.2.2 *Sarcocystis speeri* Dubey and Lindsay 1999

Opossums (*D. virginiana, D. albiventris,* and *D. marsupialis*) are definitive hosts and a parasite has been found in the United States, Argentina, and Brazil.[414,416,423,426,427] The parasite has been transmitted from the South American opossum to the North American opossum via infections induced in the KO mouse experimental intermediate host.[428] Sporocysts from experimentally infected opossums are 12–15 × 8–10 µm in size. The natural intermediate host for *S. speeri* remains unknown.

Sarcocysts of *S. speeri* in skeletal muscles of KO mice are up to 5 mm long and filiform (Figures 1.18 and 20.1). By LM, the sarcocyst wall is thin (<1 µm thick); by TEM, the cyst wall is up to 1.8 µm thick and has characteristic steeple-shaped vp surmounted by a spire, type 38 (Figure 1.52).[416,1432]

Schizonts are found in many organs, including liver, brain, and uterus of KO mice. Schizonts have also been grown in cell cultures seeded with merozoites from the liver of a KO mouse infected with an isolate from *D. albiventris* from Argentina. Merozoites from cell culture are not infective to KO mice, thus, *S. speeri* has obligatory 2-host life cycle.[423]

The endogenous development of *S. speeri* in tissues of KO mice is illustrated in Figure 20.1 to facilitate further studies using this parasite as laboratory model for *Sarcocystis.*

20.3 AMERICAN OPOSSUMS AS INTERMEDIATE HOSTS FOR *SARCOCYSTIS* SPECIES

Opossums serve as intermediate hosts for 6 *Sarcocystis* species (Table 20.2). There are 2 prevalence surveys involving *Didelphis virginiana* in which 24 of 240 opossums were found infected with *S. greineri*[59]; and, in at least 3 of 137 cases there was coinfection with *S. neurona*,[516] but overall prevalence was not clear.

20.4 SARCOCYSTS OF *SARCOCYSTIS* SPECIES IN OTHER MARSUPIALS

In a vast survey of Australian mammals, sarcocysts were found in the muscle of individuals from 8 different families (Dasyuridae, Peramelidae, Phalangeridae, Burramyidae, Petauridae, Macropodidae, Phascolarctidae, and Vombatidae) including infections in Bennett's wallaby (*Macropus rufogriseus*), swamp wallaby (*Wallabia bicolor*), pademelon (*Thylogale billardierii*), eastern grey kangaroo (*Macropus giganteus*), red kangaroo (*Macropus rufus*), and Tasmanian devil (*Sarcophilus harrisii*).[1249] Two types of sarcocysts were observed: type A, thick walls and/or large

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Table 20.2 ***Sarcocystis*** **Species in Marsupials**

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm or Stated)	Sarcocysts: Wall Thickness (μm) and TEM Type	Sarcocysts: Bradyzoites (μm)	Definitive Host	Molecular Data	References
Common opossum (*Didelphis marsupialis*)[a]	*S. didelphidis* Scorza, Torrealba, and Dagert 1957	Macroscopic, 935 μm long, 5.2 μm long vp	–	6.5 × 1.5	Unknown	No data	**1571**
	S. garnhami Mandour 1965 Four-eyed opossum (*Philander opossum*) is also a host for this species	Macroscopic, 3.3 mm long, spiny cyst wall	8–6 μm long vp, type 9 m	5.3–6.8 × 1.3–1.9	Unknown	No data	**1103, 1567, 1596**
Virginia opossum (*Didelphis virginiana*)[a]	*S. greineri* Cheadle 2001	Macroscopic, up to 6 mm long	4.0–2.8 μm long vp, stumpy, type 9b	6.2–9.0 × 1.8–3.0	Unknown, not dog, cat nor opossum	No data	**201**
	S. inghami Elsheikha, Fitzgerald, Mansfield, and Saeed 2003	Microscopic, up to 700 μm long	7 μm thick wall, type 9n	9.7–12.0 × 2.5–3.0	Unknown	ITS-1	**509, 516**
	S. sp. Scholtyseck, Entzeroth, and Chobotar 1982	Microscopic, 140 μm long	7–3.4 μm long, type 9 m	7.0–10.0 × 2.5–3.0	Unknown	No data	**1567**
Murine opossum (*Marmosa murina*)	*S. marmosae* Shaw and Lainson 1969	Macroscopic, up to 2 mm. Spiny, rose-thorn 11.5–13.0 μm long vp	–	6.2–9.0 × 1.8–3.0	Unknown	No data	**1596**
Wallabies (*Macrops rufogriseus, Petrogale assimilis*) and pademelon (*Thylogale billardierii*)	*S. mucosa* (Blanchard 1885) Labbe 1889	Macroscopic, ovoid, up to 2 mm diameter, secondary cyst wall	Thin, type 13	8.5 × 2	Unknown	No data	**1293, 893**
Bettong or rat kangaroo (*Bettongia* sp.)	*S. bettongiae* Bourne 1932	No data	No data	No data	Unknown	No data	**128**

[a] Common and Virginia opossum are also hosts for *S. neurona*, *S. falcatula*, *S. speeri*, and *S. lindsayi*.

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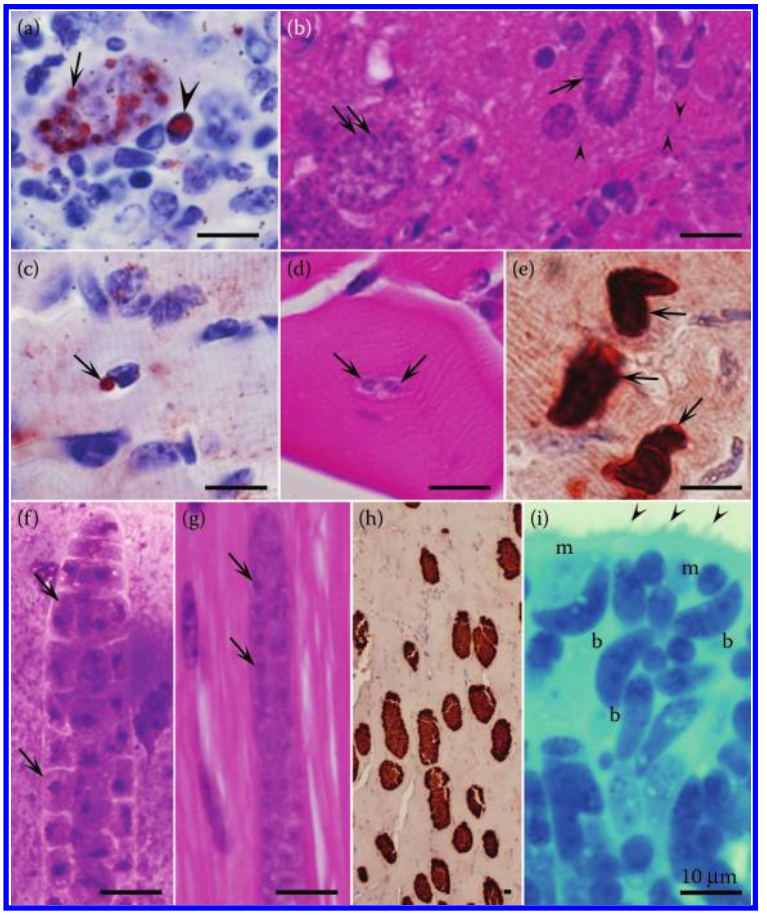


Figure 20.1 Development of *S. speeri* in experimentally infected KO mice. Schizonts (a, b) in the liver, and sarcocysts (c–i) in skeletal muscles of KO mice fed sporocysts. (b, d, and g) stained with H and E, I stained with Toluidine blue. (a) Immunohistochemical (IHC) staining with anti-*S. speeri* merozoite antibodies. (c, e, and h) IHC staining with anti-*S. speeri* bradyzoite antibodies. (f) Smear stained with Giemsa, the rest are tissue sections. (a) Immature schizont (arrow), and a uninucleate merozoite/schizont (arrowhead) at 11 DPI. (b) A schizont with peripherally arranged developing merozoites (arrow), a mature schizont (double arrows), and numerous individual merozoites (arrowheads) scattered in liver parenchyma at 11 DPI. (c) Myocyte containing 1 metrocyte inside a vacuole at 14 DPI. (d) A myocyte with 2 metrocytes (arrows), each in a separate vacuole. (e) Three immature sarcocysts. Day 17 PI. (f, g) Immature sacrocysts with metrocytes (arrows) at 25 DPI. The metrocytes in the smear are much bigger than metrocytes in tissue section. (h) Numerous sarcocysts at 52 DPI. (i) Mature sarcocyst. Note the villar protrusions (arrowheads) on the sacrocyst wall, few metrocytes (me), and longitudinally cut bradyzoites (br) at 222 DPI.

zoites and/or trabeculation, and type B, thin wall with small zoites. The prevalence of macroscopic sarcocysts of *Sarcocystis mucosa* was found to be 13% and 16% for Bennett's wallabies and pademelon, respectively.[893]

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Opossums: **59, 134, 201, 202, 416, 421, 423, 426–428, 433, 509, 516, 1103, 1209, 1432, 1492, 1567, 1571, 1596, 1670, 1722, 1942**.

Other marsupials: **128, 893, 941, 1249, 1293**.

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CHAPTER 21

Sarcocystosis in Rodents, Lagomorphs, and Other Small Mammals

This chapter summarizes *Sarcocystis* species detected in rodents, namely mice (Table 21.1), rats (Table 21.2), voles (Table 21.3), and in lagomorphs/leporids (Table 21.4), and other small mammals (Table 21.5). In most small animals the clinical significance of infection with *Sarcocystis* spp. is unknown. Three species with rodents as intermediate hosts, *S. muris*, *S. cymruensis* (Syn. *S. rodentifelis*), and *S. singaporensis*, are described in detail because of their interest for researchers.

21.1 *SARCOCYSTIS MURIS* (RAILLIET 1886) LABBÉ 1899

Sarcocystis muris is one of the first species named in the genus.[1191] The house mouse (*Mus musculus*) is the intermediate host, and the domestic cat and the ferret (*Putorius putorius furo*) are definitive hosts[1484,1503] (Table 21.1). In Egypt, sporocysts from a naturally infected cat were infectious for immunocompetent and immunocompromised mice.[27] The definitive host was required to complete the life cycle even in the case of immunodeficient mice, although theoretically *S. muris* possesses both diheteroxenous and dihomoxenous life cycles enabling transmission by cannibalism among mice.[974]

S. muris (Figure 1.1) does not cause clinical disease and is not zoonotic. The main interest in this species is its usefulness as a biological model for research on cyst forming coccidia.[1486] Many studies have been done on *in vitro* cultivation and immunology of *S. muris* infection (see additional references summarized in the Bibliography of this chapter).

Asexual stages of *S. muris* were found only in *Mus musculus*. Schizonts were seen in hepatocytes 11–17 DPI, but details of schizogony are unknown.[1503] Sarcocysts in muscles were first seen at 28 DPI, and first attained infectivity for cats at 76 DPI. As sarcocysts grew in size, the structure of the sarcocyst wall (type 1a) did not change.[1597] Mice remained asymptomatic, except for an abnormal gait, despite observations that 80% of their muscles were infected (Figures 1.1, 21.1, and 21.2). As indicated in Table 21.1, *S. muris* sarcocysts are thread-like, and several centimeters long. Bradyzoites in stained smears are 14–16 × 4–6 µm in size. Sporocysts are 8.7–11.7 × 7.5–9.0 µm.[1503]

Most international research on *S. muris* from 1973 to 2010 was performed using the Costa Rican isolate of *S. muris*, originally obtained by feeding a naturally infected house mouse in Costa Rica to a kitten.[1597] Both outbred and inbred *Mus musculus* are susceptible to infection, but results are inconsistent. After testing oral doses of 1–100,000 sporocysts, either as a single dose or as repeated doses, the most consistent results were obtained using 50 sporocysts.[1486] Infections were produced more consistently in immunosuppressed or immunodeficient mice (including nude, SCID, KO). In 2010, *S. muris* was isolated from the feces of a naturally infected *Felis catus* in Egypt.[27] Attempts to find schizonts in KO or Swiss mice inoculated orally with the Egyptian isolate of

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Table 21.1 ***Sarcocystis*** **Species in Mice**

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and Type	Definitive Host	References
House mouse (*Mus musculus*)	*S. atheridis* Šlapeta, Modrý, and Koudela 1999	Macroscopic, 30 × 0.9 mm	Thin wall, type 1b	Nitsche's bush viper (*Atheris nitschei*), 10.0–11.0 × 7.0–8.5	**1620, 1623**
	S. crotali Entzeroth, Chobotar, and Scholtyseck 1985	Up to 4 mm long	Smooth, type 1a	Mojave rattlesnake (*Crotalus scutulatus*)	**540**
	S. dispersa Černá, Kolářova, and Šulc 1978	Microscopic	Smooth, <1 μm, type 18a	Barn owl (*Tyto alba*), masked owl (*Tyto novaehollandiae*)	**187, 1585**
	S. muris (Railliet 1886) Labbe 1899	Several centimeters	Smooth, <1 μm, type 1a	Cat (*Felis catus*), ferret (*Putorius putorius furo*)	**1484, 1503, 1597**
	S. muriviperae Matuschka, Heydorn, Mehlhorn, Abd-al-Aal, Diesing, and Biehler 1987	Up to 8 mm, thick wall	3.5 μm long vp, type 18b	Palestinian viper (*Vipera palaestinae*) and Caspian whipsnake (*Coluber jugularis*)	**1142, 1363**
	S. scotti Levine and Tadros 1980	No data	No data	Tawny owl (*Strix aluco*)	**1048, 1247, 1715, 1716**
	S. sebeki Tadros and Laarman 1976	Several centimeters, smooth	<1 μm, type 1b	Tawny owl (*Strix aluco*)	**1705, 1708, 1712**
Deer mouse (*Peromyscus maniculatus*)	*S. espinosai* (Espinosa, Sterner, and Blixt 1988) Odening 1997	Microscopic, thin wall	<1 μm, type 1h	Northern saw-whet owl (*Aegolius acadicus*)	**558**
	S. idahoensis Bledsoe 1980	Up to 9.4 mm long	Thin wall (<1 μm)	Gopher snake (*Pituophis melanoleucus*)	**111, 112, 113, 375**
	S. peromysci Dubey 1983	Up to 1.8 mm	Thick wall (2–5.5 μm), hairy vp	Unknown	**375**

Table 21.2 ***Sarcocystis*** **Species in Rats**

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and Type	Definitive Host	References
Rice rat (*Oryzomys capito*)	*S. azevedo* Shaw and Lainson 1969	Up to 2.7 mm long	1–1.8 μm thick wall, striated	Unknown	**1596**
	S. oryzomyos Shaw and Lainson 1969	Up to 24.7 mm long	3–3.2 μm thick wall, striated	Unknown	**1596**
Cotton rat (*Sigmodon hispidus*)	*S. sigmodontis* Dubey and Sheffield 1988	More than 1 cm long	Thin wall, vp up to 1 μm, type 4	Unknown	**1996**
Moon rat (*Echinosorex gymnurus*)	*S. booliati* Dissanaike and Poopalachelvam 1975	Up to 5 mm, thin wall	<0.5 μm long vp, type 1b	Unknown	**325, 935**
Multimammate rat (*Mastomys natalensis*)	*S. disrumpens* Häfner and Matuschka 1984	Macroscopic, up to 2.5 cm	Thin, smooth, type 1b	Vipers (*Bitis* spp.) (see Table 22.2)	**768, 769, 1138**
Experimentally: rodents of genera *Mesocricetus, Phodopus, Gerbillus, Meriones,* and *Mus*	*S. hoarensis* Matuschka and Häfner 1984	Macroscopic, up to 2.5 mm, smooth	Secondary cyst wall, type 42	Vipers (*Bitis* spp.) (see Table 22.2)	**1133, 1141**
Indonesian rats (*Bunomys chrysocomus, Bunomys fratrorum, Paruromys dominator*)	*S. sulawesiensis* O'Donoghue, Watts, and Dixon 1987	Up to 120 μm long, smooth	<1 μm thick, type 5	Unknown	**1292**
Spiny rat (*Proechimyos guyannensis*)	*S. proechimyos* Shaw and Lainson 1969	Up to 3.9 mm	3.5–4 μm thick wall, striated	Unknown	**1596**
Norway rat (*Rattus norvegicus*)	*S. cymruensis* Ashford 1978 (Syn. *S. rodentifelis*) Grikienienė, Arnastauskienė, and Kutkienė 1993 Also in Oriental house rat (*Rattus flavipectus*). Experimental hosts: laboratory mouse, bank vole	Macroscopic, up to 50 mm, thin wall, smooth	Thin <1 μm thick, smooth, type 1c	Cat (*Felis catus*)	**41, 741, 854**
	S. murinotechis Munday and Mason 1980	Up to 300 μm	Thick, 6 μm long vp, type 9b	Tiger snake (*Notechis ater*) (see Table 23.2)	**1252**
	S. singaporensis Zaman and Colley (1975) 1976	1 mm long, striated, and thick wall	3–5 μm thick, type 19	Python (*Python reticulatus*)	**1868, 1870**

(*Continued*)

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Table 21.2 (*Continued*) *Sarcocystis* Species in Rats

Intermediate Host	*Sarcocystis* Species	Sarcocysts		Definitive Host	References
		Maximum Length (μm, or Stated)	Wall Thickness (μm) and Type		
	S. villivillosi Beaver and Maleckar 1981	1.1 mm long	1.5 μm thick, type 22	Python (*Python reticulatus*)	**85**
	S. zamani Beaver and Maleckar 1981	2 mm long	1–3 μm thick, type 18c	Python (*Python reticulatus*)	**85**
	S. zuoi Hu, Ma, and Li 2005	Up to 2.3 mm, striated wall	8.5–9.6 μm long vp, type 17	King ratsnake (*Elaphe carinata*)	**853, 855**
	S. dispersa-like Munday 1983	Microscopic	Smooth, <1 μm, type 18	Masked owl (*Tyto novaehollandiae*)	**1256**
Malaysian house rats (*Rattus rattus diardii*)	*S.* sp.	Microscopic	5.7 μm long finger-like and hexagonal in section vp, type 19	Unknown	**936**
Wood rat (*Neotoma micropus*)	*S. neotomafelis* Galavíz-Silva, Mercado-Hernández, Ramírez-Bon, Arredondo-Cantú, and Lazcano-Villareal 1991	Macroscopic, up to 2 mm long	2–4 μm thick, type 1c	Domestic cat (*Felis catus*)	**658**
Bush rat (*Rattus fuscipes*)	*S.* sp. type B Rzepczyk and Scholtyseck 1976	Macroscopic, up to 1 mm long, thin wall	Type 1b	Unknown	**1504**
Bandicoot (*Bandicota indica*)	*S.* sp. Kannangara 1970	Up to 440 μm long, smooth wall	No data	Unknown	**941**

Table 21.3 ***Sarcocystis*** **Species in Voles**

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and type	Definitive Host	References
Common European vole (*Microtus arvalis*)	*S. cernae* Levine 1977	Microscopic, up to 900 μm, smooth	<1 μm thick	Kestrel (*Falco tinnunculus*)	**182, 188, 185, 187, 1047**
Common European vole (*Microtus arvalis*), short-tailed vole (*Microtus agrestis*)	*S. putorii* (Railliet and Lucet 1891) Tadros and Laarman 1978	Macroscopic, several mm, smooth	Thin, bristly, type 9b	Ferret (*Mustela putorius* var. *furo*), common European weasel (*Mustela nivalis*), stoat (*Mustela nivalis, Mustela erminea*), mink (*Mustela lutreola*)	**1703, 1704, 1708, 1710, 1716**
European voles (*Microtus arvalis, Microtus oconomus, Microtus guentheri, Clethrionomys glareolus*)	*S. clethrionomyelaphis* Matuschka 1986	Up to 4.5 mm long, striated	4–5 μm long vp, type 9	Aesculapian snake (*Zamenis longissimus*) and others under experimental conditions.	**830, 1135, 1178**
	S. dirumpens Häfner and Matuschka 1984	Up to 25 mm, smooth	3–5 μm thick wall	Vipers (*Bitis arietans, Bitis gabonica, Bitis caudalis, Bitis nasicornis*)	**768**
	S. muriviperae Matuschka, Heydorn, Mehlhorn, Abd-al-Aal, Diesing, and Biehler 1987	Up to 8 mm long	3.5 μm long vp, type 18b	Palestinian viper (*Vipera palestinae*)	**1142**
Meadow vole (*Microtus pennsylvanicus*) and long-tailed vole (*Microtus longicaudus*)	*S. jaypeedubeyi* (Dubey 1983) Modry, Votypka, and Svobodova 2004	Macroscopic, 1.2 mm, thick wall	1.8–3.3 μm thick wall, type 9o	Unknown	**370, 1204**
	S. montanaensis Dubey 1983	Up to 648 mm, thin wall	<1 μm thick, smooth, type 1a	Unknown	**370**
Prairie vole (*Microtus ochrogaster*)	*S. montanaensis*-like Lindsay, Upton, Blagburn, Toivio-Kinnucan, McAllister, and Trauth 1991	Up to 800 μm	<1 μm thick, smooth, type 1a	Southern copperhead (*Agkistrodon contortrix*)	**1065**
Vole (*Microtus savii*)	*S. pitymysi* Splendore 1918	No data	No data	No data	**1317**
Large oriental vole (*Eothenomys miletus*)	*S. eothenomysi* Hu, Liu, Yang, Esch, Guo, and Zou 2014	Up to 3 mm long, smooth	1–2 μm thick, striated, type 18a	Unknown	**856**

(*Continued*)

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Table 21.3 (*Continued*) *Sarcocystis* Species in Voles

		Sarcocysts			
Intermediate Host	***Sarcocystis* Species**	**Maximum Length (μm, or Stated)**	**Wall Thickness (μm) and type**	**Definitive Host**	**References**
Japanese vole (*Clethrionomys rufocanus bedfordiae*)	*S. clethrionomysi* Ohbayashi and Kitamura 1959	Up to 1.5 mm long	3–7 μm thick wall	Unknown	**1322**
Voles (*Arvicola sapidus*, *Clethrionomys rufocanus*, *Clethrionomys rutilus*)	*S. glareoli* (Erhardová 1955) Odening 1997	Up to 400 μm	Type 1a	Common buzzard (*Buteo buteo*) and rough-legged buzzard (*Buteo lagopus*)	**1314, 1317**
Northern mole vole (*Ellobius talpinus*)	*S. talpini* Pak, Sklyarova, and Dymkova 1991	Up to 3.6 mm long	1.0–1.4 μm thick wall	Unknown	**1359**
European water vole (*Arvicola terrestris*)	*S. terrestri* Pak, Sklyarova, and Dymkova 1991	Up to 3.0 mm long	Thin wall (0.3–0.4 μm), hirsute	Unknown	**1359**

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Table 21.4 *Sarcocystis* Species in Lagomorphs

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and Type	Definitive Host	References
European rabbit (*Oryctolagus cuniculus*)	*S. cuniculorum* (Brumpt 1913) Odening, Wesemeier, and Bockhardt 1996	Up to 4.5 mm	8–11 μm, striated, type 10b	Cat (*Felis catus*)	**525, 1305, 1310, 1706**
Cottontail rabbit (*Sylvilagus floridanus, S. nuttalli, S. pallistris*)	*S. leporum* Crawley 1914	Macroscopic, 2 mm	5–6 μm, striated, type 10b	Cat (*Felis catus*), raccoon (*Procyon lotor*)	**260, 525, 581**
European hare (*Lepus europaeus*)	*S.* cf. *cuniculi* Brumpt 1913	Up to 2.2 mm	10.3-8.6 μm, packed long slim-looking vp, type 10b	Unknown	**1305**
	S. sp. 1 Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	800 μm	2.7-0.9 μm, hair-like vp, type 7a	Unknown	**1305**
	S. sp. 2 Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	4.3 mm	4.0-3.2 μm, cauliflower-like vp, type 26	Unknown	**1305**
	S. cf. *bertrami* Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	Up to 4.0 mm	4.4-1.9 μm, elongated leaflet-like vp, type 11b	Unknown	**1305**
	S. sp. 1 Odening, Wesemeier, and Bockhardt 1996	Oval to cigar-shaped, up to 835 μm	No vp, type 1a	Unknown	**1310**
	S. sp. 2 Odening, Wesemeier, and Bockhardt 1996	Elliptical, up to 385 μm	Stub-like vp, type 29	Unknown	**1310**
Pika (*Ochotona* spp.)	*S. ochotonae* Odening, Aue, Ochs, and Stolte 1998	Up to 0.6 mm long Sarcocysts in brain	Thin wall, type 1a	Unknown	**1318**
Alpine pika (*Ochotona alpina*)	*S. galuzoi* Levit, Orlov, and Dymkova 1984	Macroscopic, up to 2 mm long. Smooth	3–5 μm thick wall	Unknown	**1050, 1317**
Daurian pika (*Ochotona daurica*)	*S. dogeli* Machul'skii 1947	Fat macrocysts	–	Unknown	**1317**

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Table 21.5 ***Sarcocystis*** **Species in other Small Mammals**

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and Type	Definitive Host	References
Striped hamster (*Cricetulus griseus*)	*S. cricetuli* Patton and Hindle 1926	Macroscopic, 1.5 mm	No data	Unknown	**1378**
Large gerbil (*Rhombomys opimus*)	*S. fedoseenkoi* (Fedoseenko and Romanova 1983) Odening 1997	No data	70 nm thick, type 9	Unknown	**598, 1314, 1317**
Gerbil (*Gerbillus gerbillus, Gerbillus perpallidus, Psammomys obesus, Pachyuromys duprasi*)	*S. gerbilliechis* Jäkel 1995	Macroscopic, 11.7 × 0.19 mm	Smooth, type 1b	Arabian saw-scaled viper (*Echis coloratus*), 12.3–13.3 × 10.7–11.4	**885**
Guinea pig (*Cavia porcellus*)	*S. caviae* Almeida 1928	No data	No data	Unknown	**1048**
Porcupine (*Erethizon dorsatum*)	*S. sehi* Dubey, Hamir, Brown, and Rupprecht 1992	Up to 2.3 mm long	Thin wall (<1.2 μm), type 1e	Unknown	**406**
9-banded armadillo (*Dasypus novemcinctus*)[a]	*S. dasypi* Howells, Carvalho, Mello, and Rangel 1975	Up to 1.53 mm, moderately thick wall	3.7–4.3 μm, spinose, type 11	Unknown	**851**
	S. diminuta Howells, Carvalho, Mello, and Rangel 1975	Up to 224 μm, thick wall	2.6–3.5 μm, hair-like projections, type 11	Unknown	**851**
	S. sp. Lindsay, McKown, Upton, McAllister, Toivio-Kinnucan, Veatch, and blagburn 1996	Only one cyst. No data	Vp narrow at the base and expand as they grow outwardly. Unclassified	Unknown	**1067**
Marmot (*Marmota baibacina*)	*S. baibacinacanis* syn. *S. baibacina* Umbetaliev 1979	Up to 17 mm	No data	Dog (*Canis familiaris*), fox (*Vulpes vulpes*), wolf (*Canis lupus*)	**1767**
Collared anteater (*Tamandua tetradactyla*)	*S. tamanduae* Artigas and Oria 1932	No data	No data	Unknown	**38, 1048**
Richardson's ground squirrel (*Spermophilus richardsonii*)	*S. bozemanensis* Dubey 1983	300 μm, thin-walled	Smooth, <1 μm, type 1a	Unknown	**369**

(*Continued*)

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Table 21.5 (*Continued*) *Sarcocystis* Species in other Small Mammals

Intermediate Host	*Sarcocystis* Species	Sarcocysts: Maximum Length (μm, or Stated)	Sarcocysts: Wall Thickness (μm) and Type	Definitive Host	References
	S. campestris Cawthorm, Wobeser, and Gajadhar 1983 Also in 13-lined ground squirrel (*Spermophilus tridecemlineatus*)	Macroscopic, 4 mm, thick-walled, 3.5–6.4 μm	3.5 μm long vp, type 9a	American badger (*Taxidea taxus*)	**172, 369, 418**
Red squirrel (*Tamiasciurus hudsonicus*)	*S.* sp. Entzeroth, Chobotar, and Scholtyseck 1983	Microscopic, total length not known	<1 μm thick wall, type 1b	Unknown	**535**
Yellow suslik (*Spermophilus fulvus*)	*S. citellibuteonis* Pak, Pak, and Sklyarova 1989	Up to 10 mm, thin wall	1.0-0.7 μm long vp	Common buzzard (*Buteo buteo*)	**1355,1358**
	S. citellivulpes Pak, Perminora, and Yeshtokina 1979	Up to 8 mm, thick wall, striated	1.4–3.5 μm long vp	Red fox (*Vulpes vulpes*) and Corsac fox (*V. corsac*)	**1356**
Varying lemming (*Dicrostonyx richardsoni*)	*S. rauschorum* Cawthorn, Gajadhar, and Brooks 1984	Up to 122 μm long, thin wall, smooth	<1 μm thick, type 1h	Snowy owl (*Bubo scandiacus* syn. *Nyctea scandiaca*)	**173, 174, 175, 636, 1671**
Treeshrew (*Tupaia belangeri chinensis*)	*S. tupaia* Xiang, Rosenthal, He, Wang, Wang, Song, Shen, Li, and Yang 2010	Thread-like, up to 10 mm long	Smooth, type 1a	Unknown	**1845**
Greater white-toothed shrew (*Crocidura russula*)	*S. russuli* Pak, Sklyarova, and Dymkova 1991	Up to 6.0 mm long	Thin wall (0.5–1.0 μm)	Unknown	**1359**
Chipmunk (*Eutamias asiaticus*)	*S. eutamias* Tanabe and Okinami 1940	Up to 530 μm long	Not striated	Unknown	**1721**
Eastern chipmunk (*Tamias striatus*)	*S.* sp. Entzeroth, Scholtyseck, and Chobotar 1983	Microscopic, up to 75.6 μm long	Thin wall, type 1b	Unknown	**536**

[a] Nine-banded armadillo is also intermediate host for *Sarcocystis neurona*.[205,1723]

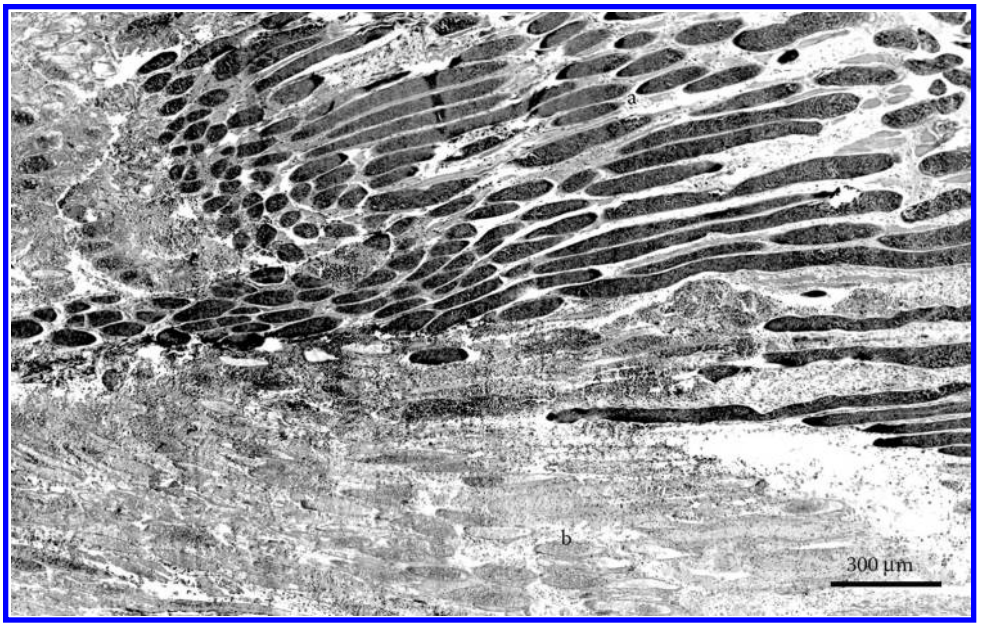


Figure 21.1 *Sarcocystis muris*-associated myositis in a mouse. Note the numerous intact (a) and degenerating (b) sarcocysts. H and E stain.

S. muris failed, although the mice had hepatitis (unpublished). The Costa Rican and the Egyptian isolates of *S. muris* are no longer available.

21.2 *SARCOCYSTIS CYMRUENSIS* ASHFORD 1978 (SYN. *S. RODENTIFELIS* GRIKIENIENÉ, ARNASTAUSKIENÉ, AND KUTKIENÉ 1993)

It has been proposed that the parasite from the bank vole and the Norway rat is capable of circulating among some rodents without the participation of the definitive host (domestic cat), based on a series of observations.[34,739,740,741,992] This species exhibited low specificity, able to infect multiple families such as Cricetidae and Muridae. Of principal interest was the possibility of transmission by cannibalism and transplacental transmission.[993,996] This second idea was almost discarded after comprehensive research[1001]; no sarcocysts were detected in 83 offspring born from experimentally infected rats. Norway rats are both definitive and intermediate hosts for *S. rodentifelis* under laboratory conditions[742]; a similar situation has been detected for *S. muris* and *S. cymruensis*. Thus, based on indistinguishable morphological and biological characteristics we have synonymized *S. rodentifelis* with *S. cymruensis* (Table 21.2).

21.3 *SARCOCYSTIS SINGAPORENSIS* ZAMAN AND COLLEY (1975) 1976

The rats (*Rattus norvegicus*) are the intermediate hosts and the pythons (*Python reticulatus*) are the definitive hosts (Table 21.2).[85,140,1868,1870] The ultrastructural development of *S. singaporensis* sarcocysts in rats has been described.[1367,1368] 2 subpopulations (type 1 and 2) of merozoites were detected in lungs from acutely infected rats.[888] Developmental stages in the definitive host (*Python reticulatus*) have been described.[1365]

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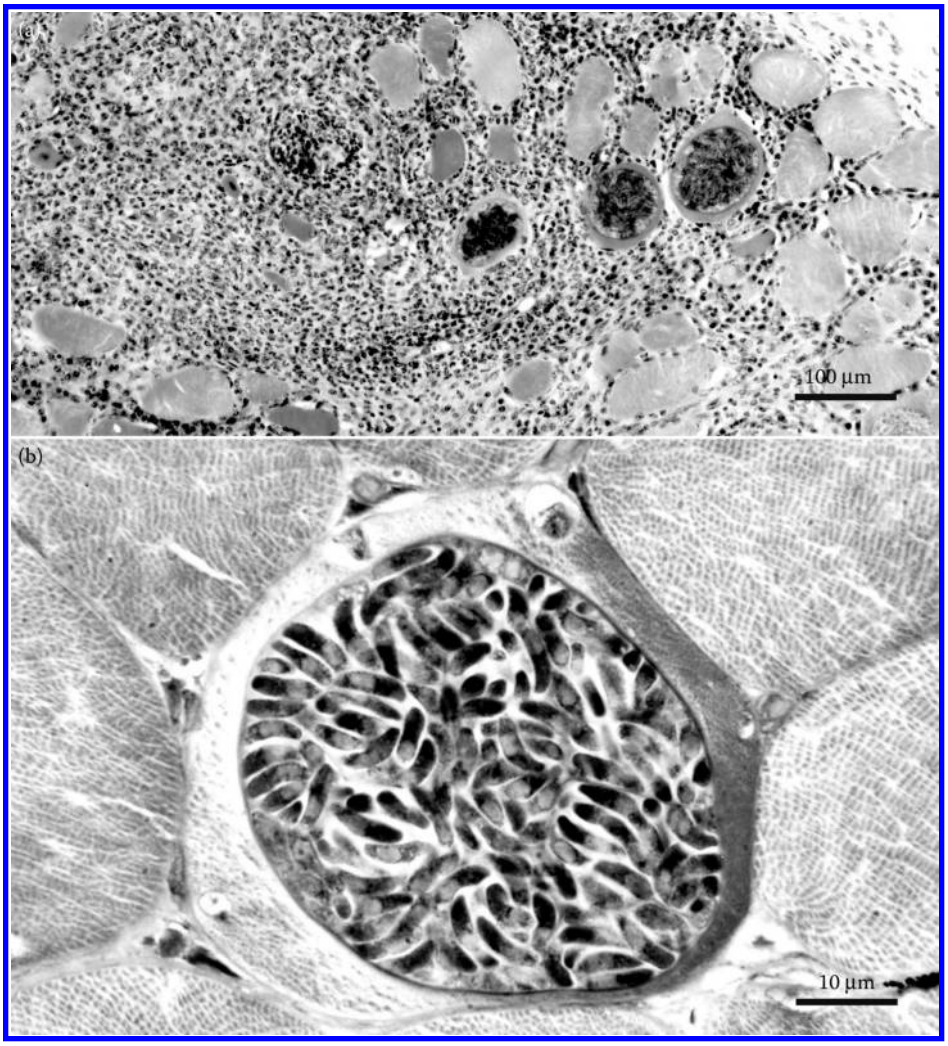


Figure 21.2 *S. muris* infection in skeletal muscle of a mouse. H and E stain. (a) Severe inflammation around degenerating sarcocysts. (b) An intact sarcocyst within a myocyte without host reaction.

Unlike other rodent *Sarcocystis* species, schizogony of *S. singaporensis* occurs in blood vessels.[85,140,1368] The dose and the strain of the parasite might affect the organ parasitized. In rats fed 200,000 sporocysts in the United States,[85] schizonts were seen only in the liver at 7 DPI but at 10 days schizonts were also present in the muscles, including cardiomyocytes, but never in the liver. Two generations of schizonts, first at 6 DPI and the second at 16 DPI, were found in rats infected in Germany.[140] In rats euthanized at 10 days after feeding them 40 million sporocysts, schizonts were seen in brain, kidneys, lungs, and the heart. Heavy infection in these rats facilitated the detailed description of schizogonic development of the parasite, including an occasional parasitization of erythrocytes and blood vessel occlusion due to hypertrophied parasitized endothelial cells.[1368]

The use of *S. singaporensis* has been proposed for the control of wild populations of rodents. It is known that natural infections in wild rats do not induce protective immunity, but a significant immune response can be induced experimentally in laboratory rats using about 1000 sporocysts.[886] A specific immunoglobulin subclass response suggests the existence of an immune evasion strategy

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for *S. singaporensis*,[890] and a natural selection for intermediate degrees of virulence of parasite strains has been confirmed.[892] The use of *S. singaporensis* as a biocontrol agent was preceded by studies on host specificity and pathogenicity.[886] Under controlled conditions,[889] bait-pellets containing high numbers of sporocysts resulted in mortalities ranging from 58% to 92% in 3 species of rodents (*Rattus norvegicus*, *Rattus tiomanicus*, and *Bandicota indica*). Using species-specific monoclonal antibodies against merozoites of *S. singaporensis* in lungs, the parasitic etiology of mortality was confirmed.[889,891]

Highly variable 18S rDNA sequences were found in *S. singaporensis* from pythons,[1622] which also seem to have a phylogenetic close relationship with *Sarcocystis atheridis* from the African viper and *Sarcocystis* sp. from the American rattlesnake. *Sarcocystis* sporocysts from the green python (*Morelia viridis*) have 95% of identity with *S. singaporensis* 18S rRNA gene sequences.[1219] Further molecular investigations will be needed in order to confirm the identity of such genotypes and the possibilities of additional hosts for *S. singaporensis*.

21.4 *SARCOCYSTIS* SPECIES WITH SARCOCYSTS IN BRAIN (PREVIOUSLY CALLED *FRENKELIA*)

21.4.1 Introduction

In 1934, Findlay and Middleton[605] reported large parasitic cysts in the brains of voles in Wales, United Kingdom, and they called it *Toxoplasma microti*. In 1953, a similar organism was found in a vole in Montana, USA (Figure 21.3) and was called "M" organism.[630] A similar parasite was described in a bank vole and the name *Toxoplasma glareolus* was proposed.[556] A new genus, *Frenkelia* was also proposed for these organisms.[102] When the life cycle of a similar parasite was completed it was named *Frenkelia clethrionomyobuteonis* (Figure 1.3).[1483] With the development of molecular tools during the last 3 decades, several investigators indicated that the genus *Frenkelia* is a synonym of *Sarcocystis*.[76,1236,1317,1799]

21.4.2 *Sarcocystis microti* (Findlay and Middleton 1934) Modrý, Votypka, and Svobodová 2004

21.4.2.1 Distribution

Europe and North America.

21.4.2.2 Intermediate Hosts

Microtus agrestis,[605] *Microtus arvalis*,[976] *Apodemus sylvaticus*,[976] *Apodemus flavicollis*,[976] *Apodemus agrarius*,[976] *Mesocricetus auratus*,[976] *Rattus norvegicus*,[1483] *Mus musculus*,[976] *Mastomys natalensis*,[1482] *Cricetus cricetus*,[1482] *Chinchilla laniger*,[424,1180,1482] *Oryctolagus cuniculus*,[1482] and probably *Lemmus lemmus*,[528] *Microtus modestus*,[630] *Ondatra zibethica*,[944] *Rattus* sp.,[786] and *Erethizon dorsatum*.[951]

21.4.2.3 Definitive Hosts

The common buzzard (*Buteo buteo*) is the definitive host but remains unknown for *S. microti* in North America; hawks and owls are suspected.[794,1063] European workers[1716] found that *F. microti* can cycle through the American red-tailed hawk, *Buteo borealis*.

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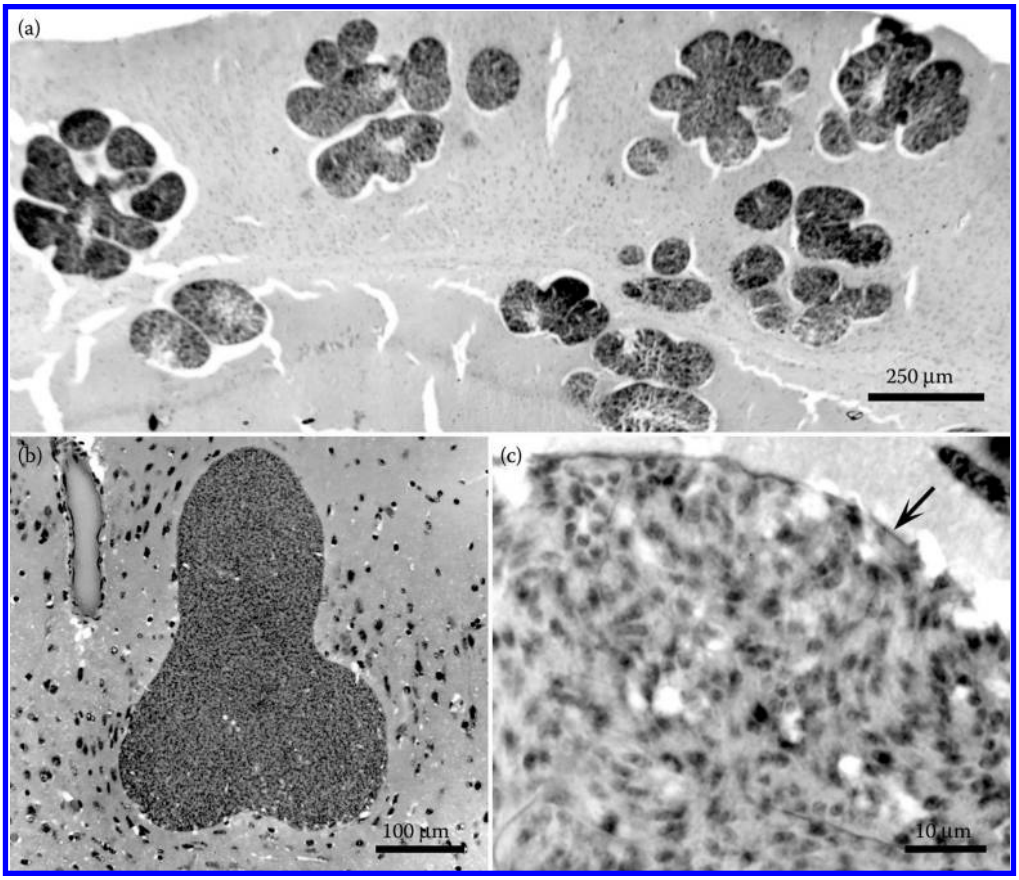


Figure 21.3 *Sarcocystis microti*-like cysts from rodents in the United States. (a) Lobulated cysts in the brain of *Microtus modestus.* (Courtesy of late J. K. Frenkel.) (b and c) A cyst in the brain of a chinchilla; arrow points to the thin cyst wall. (Adapted from Dubey, J.P. et al., 2000. *J. Parasitol.* 86, 1149–1150.)

21.4.2.4 Structure

The sarcocysts are thin-walled, lobulated, measure up to 1 mm in diameter, and are located in the brain (Figure 21.3). The sarcocyst wall is type 1a. Cysts were first observed in the brain 23 DPI.[669]

The sporocysts are 12–15 × 9–12 (mean 12 × 10) µm.[976] The schizonts are in the liver parenchymal cells 6 and 7 DPI.

21.4.3 *Sarcocystis glareoli* Erhardová 1955

21.4.3.1 Distribution

Europe and Japan.[639]

21.4.3.2 Intermediate Hosts

The European bank vole (*Clethrionomys glareolus*) is its intermediate host.[528,556,1482]

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21.4.3.3 Definitive Host

The common buzzard (*Buteo buteo*).[975]

The sporocysts are 11.3–13.8 × 7.8–10.0 (mean 12.5 × 8.8) µm.[1482,1483] The schizonts are in the liver parenchymal cells between 5 and 8 DPI; merozoites are 7.6 × 2.2 µm.[975] The cysts are found in the brain, beginning 18 DPI. They are microscopic,[952,1717] and up to 400 µm at 120 DPI.[975] Congenital transmission is suspected, but not proven.[975,1709]

21.5 PREVALENCES OF SARCOCYSTS IN RODENTS, LAGOMORPHS, AND OTHER SMALL MAMMALS

Small animals, and specially rodents, are the intermediate hosts for numerous species of *Sarcocystis* involving snakes, raptors, and carnivorous mammals as definitive hosts. Thus, the prevalence of infections in those animals is high. Infection rates of 88% for *S. dasypi* and 21% for *S. diminuta* have been found in armadillos (*Dasypus novemcinctus*) from 4 southern states in the United States.[1067] Similar infection rates (*S. dasypi*, 60.3% and *S. diminuta*, 9.5%) were found in Florida.[313] Mixed infections were also recorded in these surveys. In lagomorphs, macroscopic and microscopic sarcocysts were found in 54.1% of Eastern cottontails from Pennsylvania (*Sylvilagus floridanus*).[258] Risk factors such as the presence of cats in the environment are important; the absence of such definitive hosts seems to be the cause of the null presence of sarcocysts in 555 farmed rabbits from Lithuania.[991] The prevalence of thick-walled sarcocysts in rabbits (*Oryctolagus cuniculus*) from Australia was 21.1%.[1249]

In Richardson's ground squirrels (*Spermophilus richardsonii*) from Montana 11.1% had sarcocysts.[369] In the same state, 40.8% and later 25.5% of deer mice (*Peromyscus maniculatus*) were infected.[112,375] In woodrats (*Neotoma microtus*) from Mexico, 28.7% were infected.[658] Infection with *Sarcocystis* has also been reported in 25% of woodrats from Texas.[197,198] In large oriental voles (*Eothenomys miletus*) from China, 25% were infected with *Sarcocystis*.[856]

Wide surveys involving several host species had been carried out. The presence of sarcocysts in the brain and muscle was detected in 13 species of micromammals from different habitats in Italy.[252] Species of *Sarcocystis* were not stated. *Sarcocystis* infection was absent in 59 common moles (*Talpa europaea*) from Lithuania.[1872] Previously, in the same country, infection was reported in an Insectivora, the common shrew (*Sorex araneus*), and in 3 additional species of rodents.[743] Factors such as habitat or host weight influenced the prevalences of *Sarcocystis* in populations of small mammals and buzzards (*Buteo buteo*) in the Czech Republic.[1697]

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CHAPTER 22

Sarcocystosis in Poikilothermic Animals

22.1 REPTILES

Reptiles can act as intermediate or definitive hosts for numerous *Sarcocystis* species. Information is summarized in Tables 22.1 and 22.2, respectively.

22.1.1 Reptiles as Intermediate Hosts for *Sarcocystis*

There are 23 *Sarcocystis* species in which reptiles serve as intermediate hosts (Table 22.1). Few definitive hosts are known, but 7 snakes play this role, especially for species parasitizing lizards and geckoes (*S. stenodactylicolubris, S. gongyli, S. chalcidicolubris, S. acanthocolubri, S. ameivamastigodryasi, S. lacerate, S. podarcicolubris*).

S. gallotiae has an unusual life cycle.[1139] It can complete its life cycle in 1 host, the Gran Canaria giant lizard *Gallotia galloti*. Its sarcocysts are found in the tails of the lizards. Cannabalism is common in these reptiles, and sporocysts are produced in the intestines of lizards after eating the tails of other lizards.[1139] This particular dihomoxenous life cycle has been tested for some other *Sarcocystis* species of lizards.[67]

None of the species of *Sarcocystis* are known to cause clinical illness in reptiles. Lainson and Shaw[1015] reported granulomatous myositis in several naturally infected tortoises; lesions seemed to be associated with cyst rupture.

Epidemiological surveys on *Sarcocystis* in reptiles are scarce. In Australia, sarcocysts were reported in the muscles of 2 goannas (1 *Varanus gouldii* and 1 *V. varius*) and in 1 metallic skink (*Niveoscincus metallicus* syn. *Leiolopisma metallica*).[1251] No morphological details were given. Only 3 reports were found of sarcocysts in snakes from different locations (Table 22.1). *S. pythonis* was found in the Australian carpet snake (*Morelia spilota* syn. *Python spilotes*) in Australia.[1744] Sarcocysts in muscle were microscopic, 1.1 mm long, and thin-walled. *S. atractaspidis* was named from 2 Ogaden burrowing asps (*Atractaspis leucomelas*) from Kenya.[1370] Sarcocysts were macroscopic and were not reported in striated muscle but in mesentery and lungs. No illustrations accompanied the article, so this species should be regarded as *species inquirendae*. A massive number of microscopic sarcocysts were observed in striated muscle, not in the heart, of a wild caught female South American rattlesnake (*Crotalus durissus terrificus*) of unknown origin but confiscated in Tennessee.[1474] The sarcocyst wall was type 1h; the 18S rRNA gene sequence was closely related to that of *S. mucosa* from Australian marsupials. The authors hypothesize that an opossum is a final host.

22.1.1.1 Species Inquirendae

The following species need further revision, because there are no differential morphological descriptions of the sarcocysts and no DNA data: (1) *S. atractaspidis* Parenzan, 1947 from the

Table 22.1 Reptiles as Intermediate Hosts for *Sarcocystis*

Species	Intermediate Host	Size of Sarcocyst (μm)	Size of Bradyzoites (μm)	Sarcocyst Wall	Definitive Host, Sporocysts (μm)	Molecular Data	Reference
S. acanthocolubri Morsy, Bashtar, Abdel-Ghaffar, Mehlhorn, Quraishy, Al-Ghamdi, Koura, and Maher 2012	Lizards (*Acanthodactylus boskianus, A. scutellatus, A. pardalis*)	Macroscopic, 0.95 × 10.1 mm	4–7	2–5.8 μm thick, finger-like and stalkless vp, type 9d	Colubrid snake (*Spalerosophis diadema*), 10.6 × 9.1	No data	**1227**
S. ameivamastigodryasi Lainson and Paperna 2000	Giant ameiva lizard (*Ameiva ameiva*)	1000–2000 × 80	7.2 × 2.5	Similar to type 1b	Colubrid snake (*Mastigodryas bifossatus*), 10–7.5 × 11.2–8.7	No data	**1018**
S. atractaspidis Parenzan 1947	Ogaden burrowing asp (*Atractaspis leucomelas*)	7 × 1 mm	Piriform, 15–25	No data	Unknown	No data	**1370**
S. bunopusi Abdel-Baki, Abdel-Haleem, and Al-Quraishy 2012	Rock gecko (*Bunopus tuberculatus*)	42–45 × 22–25	5–7 × 1.5–2.0	Smooth, thin, type 1a	Unknown	No data	**4**
S. chalcidicolubris Matuschka 1987	Ocellated skink (*Chalcides ocellatus*)	Microscopic, 124–275 × 27–53	10–12 × 2	Thin, irregular, type 1e	Spotted whip snake (*Hemorrhois ravergieri*), 9.4–11.7 × 8.2–9.9	No data	**1137**
S. chamaeleonis Frank 1966	Chameleon (*Chamaeleo fischeri*)	500–1000 × 15,000	10–13 × 2	Thin (<1 μm), short vp	Unknown	No data	**623**
S. dugesii Matuschka and Mehlhorn 1984	Madeira wall lizard (*Lacerta dugesii*)	40–125 × 220–605	12–16 × ?	Thick, 4 μm long vp, type 17	Autophagy, 10.2–10.7 × 8.7–9.2	No data	**1132, 1144**
S. gallotiae Matuschka and Mehlhorn 1984	Western Canaries lizard (*Gallotia galloti*)	43–140 × 110–370	16 × 12	Thick, 7 μm long vp, type 37a	Unknown	18S	**1132, 1621**
S. gongyli Trinci 1911	Ocellated skink (*Chalcides ocellatus*)	60–100 × 200–900	4.1–5.3 × 0.8–2.0	Striated, 2–6 μm thick, leaf-like 1.9–3.0 μm long vp, type 9d	Diadem snake (*Spalerosophis diadema*), 9.1 × 10.6	No data	**8, 81**

(*Continued*)

Table 22.1 (*Continued*) Reptiles as Intermediate Hosts for *Sarcocystis*

Species	Intermediate Host	Size of Sarcocyst (μm)	Size of Bradyzoites (μm)	Sarcocyst Wall	Definitive Host, Sporocysts (μm)	Molecular Data	Reference
S. kinosterni (Lainson and Shaw 1971) 1972	Scorpion mud turtle (*Kinosternon scorpioides*)	170–230 × 8000	18.4 × 1.7	Smooth, thin-walled (<1 μm)	Unknown	No data	**1015, 1017**
S. lacertae Babudieri 1932	Eurasian wall lizard (*Podarcis muralis*)	Macroscopic, 1000 × 1800- 2000	Banana-shaped, 6.5–7.3 × 1.5–2.0	Smooth, 2.5–3.2 μm, type 37a	Smooth snake (*Coronella austriaca*), 9.0–10.0 × 7.0–8.0	18S	**53, 1621, 1798**
S. lacertautae (Syn. *S. utae*) Ball (1944) Lainson and Shaw 1971	Side-blotched lizard (*Uta stansburiana*)	120 × 950	5.5–7 × 1.5–2	Smooth, thin (<1 μm)	Unknown	No data	**1015**
S. mitrani Abdel-Ghaffar and AL-Johany 2002	Eastern skink or sand-fish lizard (*Scincus mitranus*)	Macroscopic, 0.05–0.3 × 0.5–1.8	5.2 × 2.0	Thick, vp up to 3.1 μm long vp, serrated pvm, microtubules in center of vp, type 9d	Unknown	No data	**9**
S. platydactyli Bertram 1892	Common wall gecko (*Tarentola mauritanica*)	Macroscopic, 400 × 2000	5–6 × 1.5–2	Striated, thick wall, 7–10 μm	Unknown	No data	**1138**
S. platydactyliscelopori (Ball 1944) Odening 1998	San Joaquín fence lizard (*Sceloporus occidentalis biseriatus*)	600 × 180	5.2–6 × 1.5–2	Thick wall (2–8 μm)	Unknown	No data	**63, 1317, 1015**
S. *podarcicolubris* Matuschka 1981	Eurasian wall lizards (*Podarcis* spp., *Lacerta* spp., *Algyroides nigropunctatus*	90–290 × 430–1300	7.7–10 × 2	Striated, type 9m	Whip snakes (*Coluber viridiflavus, C. gemonensis, C. jugularis, C. najadum, Macropodon cucullatus*), 6.9 × 9.6	No data	**1130, 1132, 1134**
S. *pythonis* Tiegs 1931	Python (*Morelia spilota*)	1.1 mm	Crescent-shaped, 4–7 × ?	Thin	Unknown	No data	**1744**
S. scelopori Ball 1944 (Lainson and Shaw 1971)	Western fence lizard (*Sceloporus occidentalis*)	180 × 600 thickness	5.2–6.0 × 1.5–2.0	Striated, 2–8 μm thick wall	Unknown	No data	**63, 1015**

(*Continued*)

Table 22.1 (*Continued*) Reptiles as Intermediate Hosts for *Sarcocystis*

Species	Intermediate Host	Size of Sarcocyst (μm)	Size of Bradyzoites (μm)	Sarcocyst Wall	Definitive Host, Sporocysts (μm)	Molecular Data	Reference
S. schneideri Bashtar, Al Aal, Maarouf, Morsy, and Quraishy 2014	Barber skink (*Eumeces schneideri schneideri*)	Microscopic, 250–900 × 50–100	Banana-shaped, 3–5 × 1.5–2.5	Thin wall (0.28 μm), type 1b	Unknown	No data	**82**
S. simonyi Bannert 1992	Hierro giant lizard (*Gallotia simonyi*)	Microscopic, 400–720 × 130–220	5.5–6.3 × 2–3	Thin-walled, vp up to 2 μm long, unusual spine-like shape 37a	Autophagy, 7.8–9.4 × 6.2–7.8	No data	**66, 67**
S. stehlinii Matuschka and Bannert 1989	Gran Canarian giant lizard (*Gallotia stehlini*)	90–400 × 60–160	6–7 × 1.5–2.3	Thick, 11.8–14.7 μm long vp, type 7c	Dihomoxenous, 8.2–9.4 × 5.9–7.0	No data	**67, 1145**
S. stenodactylicolubris Modrý, Koudela, and Šlapeta 2000	Fan-fingered gecko (*Ptyodactylus guttatus*) (Also: Jordan short-fingered gecko, *Stenodactylus grandiceps*)	175–200 × 35–50	5–6 × 1.5	1–2 μm thick wall, spine-like and lobulated vp, type 4	Dahl's whip snake (*Coluber najadum*), 9–10 × 7–8 (Also: *Coluber rogersi*)	No data	**1203**
S. turcicii Abdel-Ghaffar, Bashtar, Al-Quraishy, Al Nasr, and Mehlhorn 2009	Mediterranean house gecko (*Hemidactylus turcicus*)	250–800 × 70–90	5–7	Thick, serrated, up to 4 μm long vp, without stalk and folded, type 9d	Unknown	No data	**10**
S. sp. Roberts, Wellehan, Weisman, Rush, Childress, and Sibley 2014	South American rattlesnake (*Crotalus durissus terrificus*)	? × 50–150	Unknown	Thin, type 1h	Unknown	18S	**1474**

Table 22.2 Species of *Sarcocystis* Utilizing Reptiles as Definitive Hosts

Sarcocystis Species	Intermediate Host	Schizonts	Sarcocysts Length, Cyst Wall	Bradyzoites	Maturation Time	Clinical Disease in IH	Definitive Host, Sporocysts (μm)	Molecular Data	References
S. atheridis Šlapeta, Modrý, and Koudela 1999	House mouse (*Mus musculus*) and Barbary striped mouse (*Lemniscomys barbarus*)	Liver	Macroscopic, 30 × 0.9 mm, thin wall, type 1b	6–8 × 1–1.5	61–63 dpi	Yes	Nitsche's bush viper (*Atheris nitschei*), 10.0–11.0 × 7.0–8.5	18S, 28S	**1620, 1623**
S. clethrionomyelaphis Matuschka 1986	Voles (*Microtus arvalis*, *M. oeconomus*, *M. guentheri*, *Clethrionomys glareolus*)	Hepatocytes, 7–9 dpi	Up to 4.5 mm, 3 μm long vp, smooth, type 9	10–12 × 2 μm	68 dpi	Yes	Aesculapian snake (*Zamenis longissimus*, and others in the genus), 8.2–9.4 × 11.1–12.3	No data	**1135, 1178**
S. crotali Entzeroth, Chobotar, and Scholtyseck 1985	House mouse (*Mus musculus*)	Unknown	Up to 4 mm, thin, smooth type 1a	Unknown	Up to 67 dpi	No data	Mojave's rattlesnake (*Crotalus scutulatus scutulatus*), 7.6–10.4 × 10.4–11.7	No data	**540**
S. dirumpens (Hoare 1933) Häfner and Matuschka 1984	Natal multimammate rat (*Mastomys natalensis*) and others (*Gerbillus perpallidus, Meriones unguiculatus, Mesocricetus auratus, Phodopus sungorus, Mus musculus*)	No data	Macroscopic, up to 2.5 cm long, smooth, type 1b	8–9 × 2	Over 60 dpi	No	Snakes of genus *Bitis* (*B. arietans, B. caudalis, B. gabonica, B. nasicornis*), 10.6–11.6 × 7.9–8.3	No data	**768**
S. gerbilliechis Jäkel 1995	Gerbils (*Gerbillus gerbillus, G. perpallidus, Psammomys obesus, Pachyuromys duprasi*)	Endothelium, several organs	Macroscopic, 11.7 × 0.19 mm, smooth, type 1b	8.1 × 2.2	60 dpi	Yes	Arabian sawscaled viper (*Echis coloratus*), 12.3–13.3 × 10.7–11.4	No data	**885**

(*Continued*)

Table 22.2 (*Continued*) Species of *Sarcocystis* Utilizing Reptiles as Definitive Hosts

Sarcocystis Species	Intermediate Host	Schizonts	Sarcocysts Length, Cyst Wall	Bradyzoites	Maturation Time	Clinical Disease in IH	Definitive Host, Sporocysts (μm)	Molecular Data	References
S. hoarensis Matuschka, Mehlhorn, and Abd-al-Aal 1987	Natal multimammate rat (*Mastomys natalensis*) and others (*Gerbillus perpallidus, Meriones unguiculatus, Mesocricetus auratus, Phodopus sungorus, Mus musculus*)	No data	Macroscopic, up to 2.5 mm long, secondary cyst wall, type 42	11–12 × 2	No data	No data	Snakes of genus *Bitis* (*B. arietans, B. caudalis, B. gabonica, B. nasicornis*), 11.7–13.1 × 7.8–9.6	No data	**1129, 1133, 1141**
S. idahoensis Bledsoe 1980	Deer mouse (*Peromyscus maniculatus*)	Hepatocytes	Up to 9.4 mm, thin wall (<l μm)	Crescent-shaped, 2.0 × 6–7	50 dpi	Yes	Gopher snake (*Pituophis melanoleucus*), 11–12 × 13–14	No data	**111, 112, 113, 375**
S. murinotechis Munday and Mason 1980	Norway rat (*Rattus norvegicus*) and other rats of genus *Rattus*, *Pseudomys* and *Mastacomys*	Endothelium, several organs	Up to 300 μm long, thick-walled, 6 μm long vp, type 9b	No data	90 dpi	Yes	Tiger snake (*Notechis ater*), 10.5–11.5 × 7–8	No data	**1252**
S. muriviperae Matuschka, Heydorn, Mehlhorn, Abd-al-Aal, Diesing, and Biehler 1987	House mouse (*Mus musculus*)	Hepatocytes	Up to 8 mm long, 3.5 μm long vp, type 18b	No data	36 dpi	Yes	Palestinian viper (*Vipera palaestinae*) and Caspian whip snake (*Coluber jugularis*), 8.8–10.5 × 11.7–12.9	No data	**1142, 1363, 1364**

(*Continued*)

Table 22.2 (*Continued*) Species of *Sarcocystis* Utilizing Reptiles as Definitive Hosts

Sarcocystis Species	Intermediate Host	Schizonts	Sarcocysts Length, Cyst Wall	Bradyzoites	Maturation Time	Clinical Disease in IH	Definitive Host, Sporocysts (μm)	Molecular Data	References
S. nesbitti Mandour 1969	See Chapter 5						Possibly reticulated python (*Braghammerus reticulatus* syn. *Python reticulatus*) and monocled cobra (*Naja kaouthia*)	18S	**1033** For other references, see Chapters 4 and 5
S. singaporensis Zaman and Colley (1975) 1976	Norway rat (*Rattus norvegicus*), bandicoot rats (*Neosokia indica*), roof rats (*R. rattus frugivorous*), Nile grass rats (*Arvicanthis niloticus*)	Endothelium, several organs	Up to 4.4 mm long, striated and thick wall, 3–5 μm thick, type 19	4.5–6.0 × 1.0–1.5	60 dpi	Yes	Python (*Python reticulatus*), 8.4–10.2 × 7.2–8.0	18S	**886, 1033, 1365, 1868, 1870**
S. villivillosi Beaver and Maleckar 1981	Norway rat (*Rattus norvegicus*)	Endothelium, several organs	Up to 1.1 mm long, 1.5 μm thick wall, striated, type 22	Fusiform, 5 × 1	60 dpi	No	Pythons (*Python reticulatus, P. timorensis, P. sebae, Aspidites melanocephalus*), size of sporocysts could not be stated	No data	**85, 767**
S. zamani Beaver and Maleckar 1981	Norway rat (*Rattus norvegicus*)	Endothelium, several organs	Up to 2 mm long, 1–3 μm thick wall, smooth, type 18c	Banana-shaped, 10–12 × 1.5	60 dpi	No	Python (*Python reticulatus*), size of sporocysts could not be stated	No data	**85**
S. zuoi Hu, Ma, and Li 2005	Norway rat (*Rattus norvegicus*)	No data	Up to 2.3 mm, striated wall, 8.5–9.6 μm long vp, type 17	7–10 × 2.0–2.5	No data	No data	King ratsnake (*Elaphe carinata*), 10.8 × 8.0	18S	**853, 855, 1033**

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Ogaden burrowing asp (*Atractaspis leucomelas*).[1370] In this case, the author provided information: up to 7 mm long, piriform, and 15–25 μm long bradyzoites. This species was considered a *nomina dubia*[1138] cited that (2) *S. pythonis* Tiegs 1931,[1744] (3) *S. gallotiae* Matuschka and Mehlhorn 1984,[1132] (4) *S. scelopori* Ball 1944, (Lainson and Shaw 1971),[63,1015] and (5) *S. lacertautae* (Syn. *S. utae*) Ball, 1944, Lainson and Shaw 1971.[63,1015]

22.1.2 Reptiles as Definitive Hosts for *Sarcocystis*

Snakes serve as the definitive host for 13 species of *Sarcocystis* which have mammals as intermediate hosts (Table 22.2). The importance of *S. singaporensis*, *S. nesbitti*, and other species is discussed in Chapter 21.

There are 4 additional reports of *Sarcocystis* sporocysts in the feces of snakes, whose final identity was not stated. Sporocysts (no information about size and shape was given) were found in 1 Australian copperhead snake (*Austrelaps superba*) and 4 tiger snakes (*Notechis ater*).[1251] In another case, sporocysts measuring 12.0–13.6 × 10.0–11.2 μm were detected in a Western green rat snake, *Senticolis triaspis intermedia*, from New Mexico.[1149] Oocysts measuring 9–16 × 5.5–12 μm, suspected to be *S. idahoensis*, were found in a bullsnake, *Pituophis melanoleucus sayi*, in London via Tennessee.[295] Sporocysts measuring 11.9 × 10.3 μm were detected in 2 New Mexico ridgenose rattlesnakes, *Crotalus willardi obscurus*, from Sonora, Mexico.[1150]

22.1.3 Molecular Tools for *Sarcocystis* Species in Reptiles

Initial molecular assays on *Sarcocystis* from reptiles were carried out by Doležel et al.,[330] who discussed the coevolution of *Sarcocystis* spp. in their final hosts. It was later hypothesized that despite similar 18S rRNA gene sequences for *S. gallotiae* and *S. lacertae*, their dihomoxenous and heteroxenous life cycles differ because *S. gallotiae* evolved in a snake-free environment in the Canary Islands.[1621] Evolutionary relationships were shown among *Sarcocystis* spp. in 7 endemic African tree vipers (*Atheris* spp., and *Bitis* spp.) that are all monophyletic.[1623] Bioassays in different rodent species were most successful in laboratory mice. In a captive Indian cobra (*Naja naja*) from China, ITS-1 sequencing detected a *Sarcocystis* species closely related to species with rodents as intermediate hosts (*S. muris*, *S. singaporensis,* and *S. zuoi*).[942] Based on 18S rRNA gene sequences from sporocysts excreted by different Malaysian snakes, *S. nesbitti* was detected in a monocled cobra, *Naja kaouthia*, and a reticulated python, *Braghammerus reticulatus* syn. *Python reticulatus*; *S. singaporensis* was identified in a reticulated python and a Malayan brown pit viper, *Ovophis convictus*; *S. zuoi* was identified in a Malayan keeled rat snake, *Ptyas carinata*; and an unidentified *Sarcocystis* sp. was detected in a reticulated python.[1033] Based on 18S rRNA gene sequences from sporocysts from green pythons (*Morelia viridis*), 2 different *Sarcocystis* spp. were detected with 95%–97% sequence identity with *S. singaporensis*.[1219]

22.2 FISHES

S. salvelini Fantham and Porter, 1943, was reported from the trout, *Salvelinus fontinalis*.[566] Sarcocysts were observed only once among several hundred trout examined in eastern Canada.[566] Sarcocysts up to 0.5 mm long were seen as whitish threads in the abdominal muscles. The bradyzoites were 5.2–8.8 × 1.5–2.5 μm. No other information is available.

Also in Canada, sarcocysts of an unnamed species were found in an eel (*Zoarces angularis*).[566] They were up to 1 mm long and zoites were up to 15 μm long and up to 3.5 μm wide. No other information is available. In 7 fish species examined in Australia, *Sarcocystis* was not found.[1251]

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Reptiles as definitive hosts: **85, 111, 112, 113, 295, 375, 540, 767, 768, 853, 855, 885, 886, 1129, 1133, 1135, 1138, 1141, 1142, 1149, 1150, 1178, 1251, 1252, 1363, 1364, 1365, 1620, 1868, 1870, 2270**.

Other references: **330, 566, 942, 1033, 1219, 1621, 1623, 2175**.

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CHAPTER 23

Genetics, Phylogeny, and Molecular Epidemiology of *Sarcocystis*

23.1 INTRODUCTION

Sarcocystis species, especially *S. neurona*, are morphologically and biologically related to the coccidians *T. gondii* and *N. caninum* that are economically important pathogens. For every paper indexed in PubMed (http://www.ncbi.nlm.nih.gov/pubmed) from the last 10 years referencing any species of *Sarcocystis*, more than 10 appeared that referenced *T. gondii*. As explained in Chapter 3, the vast amount of research on *T. gondii* has facilitated investigations on genetics of *S. neurona*. The progress with the genetics of *S. neurona* is summarized in Chapter 3.

23.2 GENE REGULATION OF METABOLIC PATHWAYS

The first successful genetic transfection of *S. neurona* was accomplished using electroporation of a plasmid-encoded transgene encoding a selectable marker (pyrimethamine resistance) and reporter molecules (beta-galactosidase and yellow fluorescent protein).[656] This system was not, however, subsequently embraced by a large research community. However, this achievement was built upon by an approach exploiting the dependency of such parasites on host-derived purines to construct a system to systematically study gene function through positive and negative selection. By rendering parasites deficient in an enzyme (hypoxanthine-xanthine-guanine phosphoribosyltransferase) that mediates purine salvage, a line of *S. neurona* absolutely dependent on a second enzyme was made that performed this essential metabolic function.[293] Inhibiting this second pathway renders parasites entirely dependent on functional complementation with a plasmid encoding the first, providing a means to select for parasites containing this "rescue" plasmid. By altering the plasmid to convey other genes, this system can be flexibly deployed to create and isolate parasites harboring specific genetic traits. The phenotypic effects of any such traits are thereby amenable for study, opening new vistas in our biological understanding.

The ability to manipulate and study the effects of particular genes could not have come at a more opportune moment, given newfound knowledge of the composition of the *S. neurona* genome. For example, 6 rhoptry kinases were identified in the *S. neurona* genome when surveying members of this important family of virulence factors.[1719] This is an important observation because the merozoites of *Sarcocystis*, including *S. neurona*, lack rhoptries. Until now, it has not been easy to study, in any species of *Sarcocystis,* the biological functions mediated by any such molecule; the magnitude of this missed opportunity is clear in view of experiments using *T. gondii* that have implicated such molecules as key regulators of host metabolism and immunology and essential to establishing intracellular parasitism. Differential expression of certain rhoptry kinases explain a great deal of the

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variation in virulence resulting from infection in mice and may underlie evident differences in the clinical spectrum of *S. neurona* infections in animals.

Developing insights in model systems are needed, given how difficult and expensive it is to perform clinical research in horses or sea otters (see Chapter 3). Pigeon protozoal myeloencephalitis (PPE), caused by infections with *S. calchasi* (Chapter 17), may provide a valuable basis for exploring the pathobiology of neurological sarcocystosis. In a recent PPE study, immune evasion and delayed-type hypersensitivity were identified in hosts developing extensive cerebral lesions.[1331] During the early phases of schizogony, the parasites appeared to suppress IL-12, IL-18 and interferon in their hosts. Later, as disease progressed, cytokines induced by interferon gamma and tumor necrosis factor alpha increased accompanied by increased expression of MHC-II genes and the infiltration of various T cells, B cells, and macrophages.[1331]

Species of *Sarcocystis* modulate host immunity. For example, studies[1110,1240] have resolved structures for homologous microneme proteins secreted, during host cell invasion, by *T. gondii* and *S. muris*, respectively. In *T. gondii,* the molecule plays an important role in binding glycans expressed on the surface of host cells that enable the parasite to "pull itself" into the host cell by engaging the host cell's actin/myosin motor. Because this molecule may compete with host lectins (termed galectins because they preferentially bind terminal galactose residues) that regulate cell adhesion, cell signaling, and apoptosis, Marchant et al.[1110] proposed that "proteolytic maturation of TgMIC4 provides a mechanism to liberate a soluble galectin-like lectin, which could subsequently contribute independently to parasite dissemination or down-regulation of the host immune response." Indeed, galectins and their ligands have been termed "master regulators of immune cell homeostasis."[1436] Related data from *Neospora caninum* suggest that galactose recognition by these microneme proteins may be a common strategy by which tissue-cyst forming coccidia regulate host immunity and, specifically, forestall destruction and elimination of parasitized cells by cytotoxic lymphocytes.[1864] Similarly, enzymes that degrade extracellular nucleoside triphosphates (NTPases) regulate purigenic signaling, thereby modulating immune responses,[1532] and homologous versions of 1 such gene (TgNTP1 and SnNTP1) have been identified and shown to be functional in *T. gondii* and *S. neurona*.[1876]

The function of other structural elements of species of *Sarcocystis* is suggested by experiments in the model system (*T. gondii*) and may now be subjected to direct examination in a species of *Sarcocystis.* For example, a dense granule protein (DGP) was identified that may be important in the formation and/or maintenance of the parasitophorous vacuole in which *T. gondii* lives.[1124] Sequence homology suggests that this protein (TgGRA23) shares common origins (and, perhaps, a common function) with a DGP (SmDG32) previously characterized in *S. muris.* The availability of a comprehensive catalogue of such DGPs (furnished by emerging genome projects) and the newfound ability to study the independent phenotypic effects of specified genes illustrate the opportunity that awaits those curious to understand the cell biology, molecular biology, and pathobiology of sarcocystosis. In another example, stable fluorescent fusion proteins were used to localize a protein (named TgRNG1 in *T. gondii*, SnRNG1 in *S. neurona*) to the apical polar ring, a structure which organizes microtubules at the terminus of the inner membrane complex.[1758] Beyond this apical ring, characteristic of and unique to coccidian parasites, the parasite is bounded by only 1 membrane; apical secretion of molecules vital to host cell invasion occur only through the "extreme apex."

Defining surface proteins, as has been done in *S. neurona*, in other pathogenic *Sarcocystis* species might help in developing diagnostic tests and immunoprophylaxis (see Chapter 3).[263,498,668,850] Differences in the expression of shared genes contribute to the distinctive attributes of various species of *Sarcocystis.* For example, antibodies raised to the enolase present in various stages of *T. gondii* also recognize the enolase of *S. neurona* but highlight distinct patterns of stage-specific expression in these parasites.[123] This underscores the need not only to identify which genes are (or are not) shared among various species of parasites, but to also study their differential expression and regulation as they define myriad and unique biological outcomes.

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Molecular approaches seeking to understand what makes each parasitic species unique will also undoubtedly encounter genes that are unique to the particular taxon. For example, libraries of expressed sequence tags from *S. neurona* include numerous sequences with no known homolog in *T. gondii* or elsewhere. One such protein is expressed prominently on the surface of merozoites (SnSPR1) and mediates attachment to host cells. It has no known homolog in any other parasite yet examined; in particular, it should not be confused with surface antigens collectively ascribed to the SAG family.[1877] Logically, sequencing the genomes of more species of *Sarcocystis* will be necessary in order to understand how many genes, and what kinds of genes, are truly unique to each.

Comparing the fully sequenced genomes of *S. neurona* and *T. gondii* identifies broad conservation of metabolism, GRA proteins, ROP kinases, and SRS family adhesions, with interesting differences such as the absence in *S. neurona* of certain ROP kinases especially implicated as virulence factors in *T. gondii* (i.e., ROP 5, 16, and 18). When considering other genomes, including that for *N. caninum,* another tissue cyst-forming coccidia closely related to *T. gondii,* and *Eimeria tenella,* a coccidia that completes its life cycle in a single host and does not form tissue cysts, 18% of the predicted proteins were found to be unique to *S. neurona,* another 18% were unique to tissue cyst forming coccidia, and 10% were shared with these and with more distantly related apicomplexans (belonging to *Plasmodium, Theileria,* and/or *Cryptosporidium*).[109] As genomes of additional species accumulate, we will gain a better understanding of the drivers and consequences of parasite diversification and uncover new means of understanding and managing these infections.

23.3 GENETIC MARKERS THAT HAVE BEEN EMPLOYED FOR PHYLOGENETIC AND DIAGNOSTIC PURPOSES

23.3.1 18S rDNA

For most species of *Sarcocystis,* at most 1 gene has been sequenced (the gene encoding the small subunit ribosomal RNA [18S rDNA]) and for 1 purpose (to ascribe a genetic signature so that the species might be compared to others). Ribosomal RNA has many attractive attributes, and a few drawbacks, for use in characterizing and comparing biological lineages. Advantages include the fact that it is ubiquitous, portions of the molecule are so highly conserved among related groups of organisms as to allow easy application of conserved PCR primers, and portions of the molecule are more variable, allowing distinction among related biological species and lineages. Disadvantages include the possibility that closely related species, although phenotypically distinct, may differ little or not at all in this locus, and the possibility that distinctions among versions of the gene harbored by a given lineage may confound attempts to compare 1 parasite to another. Finally, the vastly differing rates of evolution among portions of the molecule raise questions as to how to model the evolutionary process when seeking to infer phylogenetic relationships from extant variation in such sequences.

In spite of such complications, 18S remains obvious choice when first characterizing an isolate of *Sarcocystis* owing to its relative ease of amplification and, moreover, owing to the richness of the available reference set of sequences. Table 23.1 summarizes the literature employing this molecule to render diagnoses and to infer phylogenetic relationships. In spite of benefits that might have been offered by alternative loci, the history of the field dictates that new species descriptions should, whenever possible, include 18S rDNA. So too, new 18S sequences stand the greatest chance of being employed in future comparative studies. Nonetheless, science advances when hypotheses withstand independent validation, and phylogenetic hypotheses are no exception. To date, that concept has been evaluated in *Sarcocystis* primarily by subjecting the same 18S data to independent tree-building approaches. Sampling other data, however, would provide even more compelling tests of such relationships, because results from any given set of data can engender an undeserved sense of certainty.

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Table 23.1 Studies of 18S rDNA Variation in Species of *Sarcocystis*

Hosts	Parasite	Year	Reference
Mouse (*Mus musculus*)	*S. muris*	1991	**653**
Mouse (*Mus musculus*)	*S. muris*	1991	**1732**
Mouse (*Mus musculus*)	*S. muris, S. gigantea*	1992	**655**
Various	Various	1993	**144**
Cattle (*Bos taurus*), sheep (*Ovis aries*)	*S. cruzi, S. tenella, S. fusiformis, S. gigantea*	1993	**840**
Cattle (*Bos taurus*)	*S. cruzi*	1994	**1095**
Cattle (*Bos taurus*)	*Neospora caninum*	1994	**494**
Various	*Neospora caninum* and *T. gondii*	1994	**841**
Sheep (*Ovis aries*)	*S. tenella, S. arieticanis, S. gigantea,* and *T. gondii*	1994	**1736**
Horse (*Equus caballus*)	*S. neurona*	1994	**600**
Opossum (*Didelphis virginiana*)	*S. neurona*	1995	**601**
Brown-headed cowbird (*Molothrus ater*) and Moluccan cockatoo (*Cacatua moluccensis*)	*S. neurona*, *S. falcatula*	1995	**290**
Sheep (*Ovis aries*)	*S. arieticanis, S. gigantea, S. tenella*	1995	**497**
Various	Various	1995	**495**
Horse (*Equus caballus*)	*S. neurona*	1996	**1113**
Various	Various, compared to *Eimeria*	1997	**77**
Goat (*Capra hircus*)	*S. capricanis* and *S. moulei*	1997	**904**
Cattle (*Bos taurus*), dwarf zebu (*B. taurus*), and bison (*Bison bison*)	*S. hirsuta, S. cruzi, S. hominis*-like	1998	**608**
Rodents	Synonymize *Frankelia* with *Sarcocystis*	1998	**1799**
Various	Sarcocystidae include *Sarcocystis* spp. and *Isospora* spp., *Neospora* spp., and *Toxoplasma* sp.	1998	**166**
Macaque (*Macaca mulatta*)	*S.* sp.	1998	**1020**
Various ruminants	*Sarcocystis* infecting ruminants are divided between those having cats and dogs as definitive hosts	1999	**843**
Mouse (*Mus musculus*), barn owl (*Tyto alba*), and Nitsche's bush viper (*Atheris nitschei*)	*Sarcocystis* sharing a type of final host are especially closely related	1999	**330**
Various	*Hammondia hammondi* is similar to *T. gondii, Frenkelia* are *Sarcocystis*	1999	**906**
Nitsche's bush viper (*Atheris nitschei*)	*S. atheridis*	1999	**1620**
Sheep (*Ovis aries*)	*S. tenella*, *S. arieticanis*	1999	**788**
Various	Effects of 18S sequence alignment	2001	**1237**
Various	Relationship of *Lankesterella* and *Caryospora* to other Sarcocystidae	2001	**78**
Various	Dihomoxenous life cycle evolved more than once	2001	**1621**
Cattle (*Bos taurus*) and water buffalo (*Bubalus bubalis*)	*S. cruzi*	2001	**1856**
Water buffalo (*Bubalus bubalis*)	*S. hominis*-like	2001	**1857**
Cat (*Felis catus*)	*S. felis*	2003	**684**
Snakes and rodents	*S. singaporensis*	2002	**1622**
Water buffalo (*Bubalus bubalis*)	*S. cruzi*	2002	**1056**
Various	Various: PCR followed by RFLP	2002	**1858**

(*Continued*)

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Table 23.1 (*Continued*) Studies of 18S rDNA Variation in Species of *Sarcocystis*

Hosts	Parasite	Year	Reference
Vipers	Various	2003	**1623**
Opossum (*Didelphis virginiana*)	*S. neurona*, *S. inghami*	2004	**513**
Dog (*Canis familiaris*)	*S. neurona*-like	2005	**1780**
Horses and opossums (*Didelphis virginiana*)	*S. neurona*	2005	**518**
White-fronted goose (*Anser albifrons*)	*S.* sp.	2006	**1003**
Reindeer (*Rangifer tarandi*)	Various	2007	**280**
Cattle (*Bos taurus*)	*S. hominis*	2007	**1776**
Reindeer (*Rangifer tarandi*)	Various	2007, 2008	**281, 283**
Moose (*Alces alces*)	*S. alces, S. ovalis, S. scandinavica, S. alceslatrans, S. silva,* and *S.* sp.	2008	**282**
Cattle (*Bos taurus*)	*S. cruzi*	2008	**1427**
Cattle (*Bos taurus*) and water buffalo (*Bubalus bubalis*)	*S. cruzi, S. hominis, S. hirsuta, S. fusiformis*	2009	**905**
Southern sea otters (*Enhydra lutris*)	*S. neurona*	2009	**1196**
Human (*Homo hominis*), cat (*Felis catus*), and dog (*Canis familiaris*) definitive hosts	*S. hominis, S. fusiformis,* and *S. cruzi*	2009	**1844**
Raccoons (*Procyon lotor*)	*S.* sp.	2009	**909**
Racing pigeon (*Columbia livia*)	*S.* sp.	2009	**1325**
Buteo spp. buzzards and *Accipiter* spp. hawks	*S.* sp.	2009	**1851**
Hooded crow (*Corvus cornix*)	*S. cornixi*	2009	**1005**
Roe deer (*Capreolus capreolus*)	*S. gracilis, S. oviformis, S. silva*	2008, 2014	**284, 969**
Raccoon dogs (*Nyctereutes procyonoides*)	*S.* sp.	2010	**983**
Sparrow hawks (*Accipiter nisus*)	*S. calchasi*	2010	**1327**
Red deer (*Cervus elaphus)*	*S. hjorti, S. hardangeri, S. ovalis, S. rangiferi,* and *S. tarandi*	2010	**285**
Wolverine (*Gulo gulo*)	*S. kalvikus, S. kitikmeotensis*	2010	**467**
Red (*Vulpes vulpes*) and arctic fox (*V. lagopus*)	*S. alces, S. hjorti*	2010	**286**
Striped skunk (*Mephitis mephitis*)	*S. neurona*	2010	**153**
Water buffalo (*Bubalus bubalis*)	*S. cruzi*	2010	**1846**
Mallard duck (*Anas platyrhynchos*)	*S. rileyi*	2010	**466**
Corvid birds (Corvidae)	*S. ovalis*	2010	**699**
Racing pigeons (*Columbia livia*) and goshawks (*Accipiter* spp.)	*S. calchasi*	2010	**1329**
Racing pigeons (*Columbia livia*) and goshawks (*Accipiter* spp.)	*S. calchasi, S. columbae,* S. sp.	2010	**1326**
Ferret (*Mustela putorius furo*)	*S. neurona*	2010	**145**
Black bear (*Ursus americanus*)	*S. canis*-like	2011	**305**
Herring gull (*Larus argentatus*)	*S. wobeseri*	2011	**1415**
Mallard duck (*Anas platyrhynchos*)	*S. rileyi*	2011	**1007**
Wood pigeon (*Columba palumbus*)	*S. columbae*	2011	**1416**
Water buffalo (*Bubalus bubalis*)	*S. rommeli*	2011	**211**
Cattle (*Bos taurus*)	*S. hominis*-like	2011	**331**
Water buffalo (*Bubalus bubalis*)	*S. fusiformis*	2011	**1340**

(*Continued*)

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Table 23.1 (*Continued*) Studies of 18S rDNA Variation in Species of *Sarcocystis*

Hosts	Parasite	Year	Reference
Fischer *Martes pennanti*	*S.* sp.	2011	**1025**
Camel *Camelus dromedarius*	*S.* sp.	2011	**1230**
Swine (*Sus scrofa*)	*S. miescheriana*	2011	**168**
Swine (*Sus scrofa*)	*S. miescheriana*	2011	**956**
Goat (*Capra hircus*)	*S. moulei*	2010	**1229**
Rhesus monkey (*Macaca mulatta*)	*S. nesbitti*	2012	**1743**
Roe deer (*Capreolus capreolus*)	*S. capreolicanis, S. silva*	2012	**700**
Wall lizard (*Podarcis* sp.)	*S. gallotiae*	2012	**783**
Woodrat (*Neotoma micropus*)	*S. neotomafelis*	2012	**198**
Jackdaw (*Corvus monedula*)	*S. corvusi*	2013	**1419**
Various psittacine species	*S. calchasi*	2013	**1473**
Cat (*Felis catus*), house mouse (*Mus musculus*)	*S. muris*	2013	**27**
Swine (*Sus scrofa*)	*S. miescheriana*	2013	**1854**
Llama (*Llama glama*) and alpaca (*Vicugna pacos*)	*S. aucheniae*	2013	**164**
Water buffalo (*Bubalis bubalis*)	*S. cruzi*	2013	**1029**
Human (*Homo sapiens*)	*S. nesbitti*	2013	**14**
Great black-backed gull (*Larus marinus*)	*S. lari*	2014	**1420**
Dog (*Canis familiaris*)	*S. caninum, S. svanai*	2014	**476**
Oriental vole (*Eothenomys miletus*)	*S. eothenomysi*	2014	**856**
Sheep (*Ovis aries*)	*S. gigantea*	2014	**775**
Beef (*Bos taurus*)	*S.* sp.	2014	**1221**
Feral swine (*Sus scrofa*)	*S. miesheriana*	2014	**161**
African buffalo (*Syncerus caffer*)	*S. cafferi*	2014	**473**
Stellar sea lion (*Eumetopias jubatus*)	*S. canis*	2014	**1818**
Human (*Homo sapiens*)	*S. nesbitti*	2014	**1034**
Malaysian snakes (*Naja* sp., *Braghammerus* sp., *Ovophis* sp., *Ptyas* sp.).	*S. nesbitti, S. singaporensis, S. zuoi,* and *S.* sp.	2014	**1033**
Ducks (*Anas* spp.)	*S. rileyi*	2014	**1421**
Sheep (*Ovis aries*)	*S. tenella*	2014	**2402**
Eurasian otter (*Lutra lutra*)	*S. lutrae*	2015	**706**
Common moorhen (*Gallinula chloropus*)	*S. chloropusae*	2015	**490**
Wood bison (*Bison bison athabascae*)	*S. cruzi*	2015	**162**
Bobcat (*Felis rufus*)	*S. neurona*	2015	**1788**
Cattle (*Bos taurus*)	*S. cruzi, S. hominis, S.* sp.	2015	**1143**
Goat (*Capra hircus*)	*S. capracanis*	2015	**1400**
Cattle (*Bos taurus*)	*S. rommeli*	2015	**2392**
Opossum (*Didelphis* spp.)	*S. speeri*	2015	**1432**

Overreliance on the 18S marker, in particular, masks true differences between closely related lineages and confounds attempts to resolve their order of evolutionary branching. Controversies regarding the validity of distinctions between phenotypically and biologically distinctive parasites in this genus (see, e.g., *S. neurona* vs. *S. falcatula*) illustrates how 18S can fail to provide researchers sufficient resolution when attempting to differentially diagnose lineages that deserve independent

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recognition (based, e.g., on distinct host ranges).[271,290,1722] Moreover, ribosomal DNA is subjected to vastly different functional constraints in its active sites, its structure-defining and base-paired stems, and certain variable loops. Such complexities justify meaningful debate as to how best to align homologous positions and model the process of evolutionary change.[495,1237] Structural models of the ribosomal RNA can help ensure the accurate comparison of evolutionarily homologous nucleotides and have been shown to be capable of improving phylogenetic analyses of *Sarcocystis* rDNA; until that process can be validated and automated, algorithmic approaches to sequence alignment that do not take into account structural information are likely to continue to play an important role in phylogenetic inference with only limited reduction in the power to resolve well-supported relationships.

What should be used to augment the information provided by sequencing 18S? The publication of genome data provides a nearly limitless menu of possibilities. However, only several loci have so far been used as a basis by which to compare species, populations, or individual isolates (reviewed in Reference **1678**). Table 23.2 summarizes research characterizing variation in loci other than the small subunit rDNA gene. These include genes encoded in the apicoplast genome (its ribosomal RNA and the beta subunit of the RNA polymerase II gene), in the mitochondrial genome (cytochrome b), and the nuclear genome (the large subunit ribosomal RNA gene/28S, various microsatellite markers, and cloned fragments of anonymous DNA derived initially from RAPD DNA profiling). With some justification, the extant variation among those markers is presumed not to influence the survival of the parasites (because functionally consequential mutations likely so disfavor survival and reproduction as to lead to their hasty demise). In contrast, variation in surface antigens likely reflects not only historical subdivision but also ongoing selection for particular function, increasing its interest for those studying the host–parasite interaction but decreasing its reliability as a surrogate for genome evolution, writ-large. None of these is as easy to initially characterize as 18S rRNA, but each affords access to more rapidly evolving portions of the genome.

23.3.2 Other Nuclear Genes

Few other genes have been characterized from multiple species of *Sarcocystis.* Bands originally obtained from RAPD PCRs were cloned and sequenced in efforts to help discriminate *S. neurona* from *S. falcatula*.[1722] Limited work was done to characterize surface antigens and microneme proteins from *S. muris*.[633] The internal transcribed spacers of rDNA (ITS1 and ITS2) have been employed in several instances to discriminate among species of *Sarcocystis* that are identical in 18S rDNA (see Table 23.2). The larger subunit rDNA (28S) encompasses phylogenetically informative variation that augments information derived from the small subunit.[1235] With the publication of the *S. neurona* genome, this limited repertoire has been inundated with thousands of annotated genes, any subset of which may prove useful in understanding the identities of and relationships among species of *Sarcocystis.*

23.3.3 Plastid Genes

A defining feature of the phylum Apicomplexa is its plastid, an organelle (related to the chloroplasts of plants) derived from alpha-proteobacteria. This organelle contributes to the distinctive metabolism of these parasites and possesses a genome derived from the formerly free-living ancestor presumed to have been acquired via an ancient symbiosis. That genome encodes its own transcriptional machinery, as well as certain enzymes, in species of *Sarcocystis,* and these have been explored for their contribution to understanding relationships among diverse species. When seeking to describe phylogenetic relationships among a wide sample of Apicomplexa, a study concluded that the plastid small subunit RNA afforded more resolution than did the corresponding sequences derived from the nucleus.[1301] Similarly, the beta subunit of the plastid-encoded RNA polymerase gene (rpoB) has been used a basis to explore the diversification history of parasite species.[453,902]

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Table 23.2 Analyses of Variation in Other Molecular Markers

Host	Parasite	Locus	Year	Reference
Cattle (*Bos taurus*)	*Neospora caninum* and *T. gondii*	ITS-1, 5.8S rDNA	1996	**842**
Various	Various	RNA Polymerase B (RPOb)	1996	**902**
Sheep (*Ovis aries*)	*S. gigantea*	ITS-1	1996	**903**
Cattle (*Bos taurus*)	*Neospora caninum*	ITS-1	1997	**1675**
Various	*S. neurona, S. falcatula*	ITS-1	1999	**1116**
Various	*S. neurona, S. falcatula*	Cloned RAPD bands	1999	**1722**
Opossums *(Didelphis marsupialis and Didelphis albiventris)*	*S. falcatula*	Cloned RAPD band	2001	**441**
Various	*Sarcocystis* and *Frenkelia* should be synonymized	28S	1999	**1236**
Cattle (*Bos taurus*), wildebeest (*Connochaetes* spp.) and goats (*Capra hircus*)	Relationship of *Besnoitia* spp. to Toxoplasmatinae	18S, ITS-1	2000	**496**
Pacific harbor seal (*Phoca vitulina richardsi*)	*S. neurona*	18S, ITS-1	2001	**1194**
Opossum (*Didelphis albiventris*)	*S. lindsayi*	28S, ITS-1	2001	**433**
Opossum (*Didelphis virginiana*)	*S. neurona*	Cloned RAPD band	2001	**442**
Opossum (*Didelphis virginiana, D. albiventris,* and *D. marsupialis*)	*S. neurona*	Cloned RAPD band	2001	**1492**
Various	Various	Apicoplast rDNA	2002	**1301**
Southern sea otter (*Enhydra lutris*)	*S.* sp.	Plastid-encoded RNA polymerase	2003	**453**
African tree viper (*Atheris* spp., *Bitis* spp., *Proatheris* sp.)	*S. atheridis* and other unknown genotypes	18S, 28S	2003	**1623**
Cell cultures	Various	18S, hsp70	2006	**520**
Various	*S. neurona, S. falcatula*	Microsatellites	2006	**45, 44**
Dog (*Canis familiaris*)	Various	18S, ITS-1	2006	**460**
African gray parrot (*Psittacus erithacus*)	*S.* sp.	18S, ITS-1	2006	**459**
Pig (*Sus scrofa*) and cattle (*Bos taurus*)	*S. hominis, S. suihominis*	ITS1, ITS-2	2006	**719**
Cat (*Felis catus*)	*S. felis*	18S, ITS-1	2006	**521, 519**
Rhesus macaque (*Macaca mulatta*)	*S.* sp.	18S, ITS-1	2007	**723**
Brown-headed cowbird (*Molothrus ater*)	*S. neurona, S. falcatula*	RAPD bands	2008	**1108**
Various	*S. neurona*	Microsatellites	2008	**1693**
Cattle (*Bos taurus*)	*S. cruzi*	18S, ITS-1	2008	**1493**
Goldeneye (*Bucephala clangula*) and mallard duck (*Anas platyrhyncos*)	Various	28S	2008	**1004**
Hooded crow (*Corvus cornix*)	*S. cornixi*	18S, 28S	2009	**1005**
Australian glider (*Petaurus australis*)	*S.* sp.	18S, 28S	2009	**1879**
Racing pigeon (*Columbia livia*)	*S. calchasi*	28S, ITS-1	2009	**1325**
Southern sea otter (*Enhydra lutris*)	*S. neurona*	18S, RPOb, CO1, SnSAG 1–5, microsatellite Sn9	2010	**1821**

(*Continued*)

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Table 23.2 (*Continued*) Analyses of Variation in Other Molecular Markers

Host	Parasite	Locus	Year	Reference
Southern sea otter (*Enhydra lutris*)	*S. neurona*	ITS-1, 25/396 RAPD marker, SnSAG 2, 3, 4, microsatellites	2010	**1464**
Southern sea otter (*Enhydra lutris*)	*S. neurona*	ITS-1	2009, 2010	**1197, 1196**
Wolverine (*Gulo gulo*)	*S. kalvikus*, *S. kitikmeotensis*	18S, ITS-1	2010	**467**
Wood pigeon (*Columbia palumbus*) and sparrowhawk (*Accipiter nisus*)	*S. calchasi, S. columbae*	18S, 28S, ITS-1	2010	**1327**
Racing pigeon (*Columbia livia*) and goshawk (*Accipiter* spp.)	*S. calchasi, S. columbae,* S. sp.	18S, 28S, ITS-1	2010	**1326**
Mallard (*Anas platyrhyncos*)	*S. rileyi*	18S, 28S, ITS-1	2010	**466**
Racing pigeon (*Columbia livia*) and goshawk (*Accipiter* spp.)	*S. calchasi*	18S, 28S	2010	**1329**
Mallard (*Anas platyrhyncos*)	*S. wobeseri*	18S, 28S, ITS-1	2010	**1006**
Domestic pigeon (*Columbia livia*)	*S. calchasi*	28S, ITS-1	2011	**1843**
Herring gull (*Larus argentatus*)	*S. wobeseri*	18S, 28S, ITS-1	2011	**1415**
Accipiter hawk (*Accipiter g. gentilis, Accipiter n. nisus*)	*S. calchasi*	ITS-1	2011	**1330**
Mallard duck (*Anas platyrhynchos*)	*S. rileyi*	18S, 28S, ITS-1	2011	**1007**
Common blackbird (*Turdus merula*)	*S. turdusi*	18S, 28S, ITS-1	2012	**1009**
White-fronted goose (*Anser albifrons*) and mallard duck (*Anas platyrhynchos*)	*S. albifronsi, S. anasi*	18S, 28S, ITS-1	2012	**1008**
Jackdaw (*Corvus monedula*)	*S. corvusi*	18S, 28S, ITS-1	2013	**1419**
Various psittacine species	*S. calchasi*	18S, ITS-1	2013	**1473**
Duck (*Anas* spp.)	*S. rileyi*	18S, 28S, ITS-1	2014	**1421**
Cockatiel (*Nymphicus hollandicus*) and North American rock pigeon (*Columba livia f. dom.*)	*S. calchasi*	ITS-1	2014	**1332**
Great black-backed gull (*Larus marinus*)	*S. lari*	18S, 28S, ITS-1	2014	**1420**
Moose (*Alces alces*)	*S. alces, S. alceslatrans, S. ovalis, S. taeniata*	18S, cox1	2014	**702**
Arctic fox (*Vulpes lagopus*)	*S. arctica*	18S, 28S, ITS-1, and cox1	2014	**705**
Red deer (*Cervus elaphus*)	*S. elongata, S. truncata*	cox1	2014	**704**
Common eider (*Somateria mollissima*)	*S. rileyi*	18S, 28S, ITS-1, cox1	2014	**703**
Dog (*Canis familiaris*)	*S. caninum, S. svanai*	18S, RPOb	2014	**476**
African buffalo (*Syncerus caffer*)	*S. cafferi*	18S, cox1	2014	**473**
Eurasian otter (*Lutra lutra*)	*S. lutrae*	18S, 28S, ITS-1, cytb, cox1	2015	**706**
Common moorhen (*Gallinula chloropus*)	*S. chloropusae*	18S, 28S, ITS-1	2015	**490**
Bobcat (*Felis rufus*)	*S. neurona*	18S, ITS-1	2015	**1788**
Fox (*Vulpes vulpes*), racoon dog (*Nyctereutes procyonoides*)	*S. rileyi*	ITS-1	2015	**2398**
Opossum (*Didelphis* spp.)	*S. speeri*	18S, 28S, ITS-1,	2015	**1432**

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23.3.4 Mitochondrial Genes

In recent years, the cytochrome c oxidase subunit 1 (cox1) has been promoted as a means to differentiate among various species of *Sarcocystis*.[473,702,1477,1941] This approach mirrors broader efforts in the biological community to "barcode" species with greater resolution than that which is possible using ribosomal RNA alone. Initiated by entomologists and subsequently embraced by a broad spectrum of biologists, this essential gene (whose product mediates a step in the respiratory chain of oxidative phosphorylation in all aerobic life forms) is comprised of codons whose variation tends to be restricted to synonymous sites (that leave the resulting amino acid sequences unchanged, preserving enzymatic function). Variation in this locus has been used to examine intra- and interpopulation subdivision in metazoa, as well as descent relationships among closely related organisms; mutational saturation at the limited subset of mutable sites, however, obscures efforts to recover evolutionarily deeper relationships at such loci.

23.3.5 Surface Antigens

The surface of *Sarcocystis* parasites includes gpi-anchored glycoproteins that engender immunological responses. It has been established that a given surface antigen in the SAG family can harbor any 1 of several, markedly distinct alleles, and that, when considering the SAG family as a whole, these alleles combine to form distinctive assemblages of surface antigens.[1209] "Swapping" of alleles at these loci has evidently diversified the surfaces of parasites derived from *S. falcatula.* Although these differ one to another, the pool of alleles from which they sample appears distinct from the pool of alleles used by sympatric parasites attributed, by contrast, to *S. neurona.* These, in turn, consistently differed (but to a lesser extent) from alleles characteristic of *S. neurona* in North America. Taken together, these data illustrate a dynamic process of allele swapping and undermine variation in any given SAG locus as the basis by which to derive inferences about parasite phylogeny.

23.3.6 Microsatellites

Short nucleotide repeats can provide a rich source of allelic variation with which to characterize the structure of biological populations and have been used to some extent in examining the connections between populations of *S. neurona*.[44,45,1693 1821] These have identified only limited differentiation among candidate populations in the United States and have affirmed that infections in terrestrial and marine mammals share a common source of infection.

23.4 EPIDEMIOLOGICAL INSIGHTS DERIVED FROM MOLECULAR MARKERS

Molecular markers have contributed greatly to our understanding of the diversity and transmission of species of *Sarcocystis* and have become established as essential to future progress in this area. Three important examples illustrate the point. A molecular signature (18S rDNA) was essential to unlocking the transmission cycle responsible for EPM. Although a protozoal agent was identified in this neurological disease of horses, it was not clear how horses contracted the infection. Because no natural predator of horses exists where this disease is prevalent, there was little *a priori* basis to suspect any particular carnivore as responsible. Neither cats nor dogs (the most readily tested and abundant of carnivores) could be demonstrated as competent definitive hosts. The serendipitous discovery of oocysts bearing 18S rDNA matching the agent of EPM[601] solved the mystery, and engendered a flurry of productive research into the biology and epidemiology of this organism. In addition to linking prey species with the predators (or scavengers) that support the sexual development of species of *Sarcocystis*, molecular data have been employed to establish

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the range of intermediate hosts susceptible to infection. The case of *S. neurona* is notable for an uncommon and unexpected range of hosts (including raccoons, cats, armadillos, horses, birds, and sea otters). Without this sequence (and additional markers that provided greater specificity), there is little chance that we would understand the vast range of intermediate hosts that would be shown susceptible to infection with this agent, including a variety of domesticated and wildlife species. Certainly a strong connection between terrestrial systems and the marine aquatic environment could not have been predicted, but *S. neurona* was subsequently confirmed as a major infectious cause of mortality in Southern sea otters *Enhydra lutris*.[453] Similarly, genetic signatures have been crucial to differentiating among sporocysts excreted by a given host, for example *S. neurona* and *S. falcatula* in the feces of opossums.[1722]

A similar application of molecular epidemiology established an unexpected cycle of transmission of *S. ovalis*.[699] Here, the definitive host of a parasite of a moose (*Alces alces*) was sought. Noting that corvid birds often feed on moose carcasses, droppings were examined and found to contain sporocysts. These matched the signature sequence of 18S rDNA, and the mucosal scrapings of a magpie (*Pica pica*) were thereby demonstrated to harbor parasites bearing this same sequence.

A precise "match" between sequences, whether identified through serendipity or hypothesis testing, need not occur in order to derive epidemiologically valuable clues. A good example of this concerns successful prediction of the definitive host responsible for human infections with *S. nesbitti* (see Chapters 4 and 5). Infections in a macaque[1859] were characterized at the 18S rDNA. On the basis of the resulting phylogenetic tree, the entity was shown to share an especially close relationship to a species of *Sarcocystis* that complete their life cycle in predatory snakes.[1743] When human cases of sarcocystosis[14,877,559,560] were subsequently attributed to a parasite identical in 18S sequence, a presumptive diagnosis was provided with a presumptive transmission mode implied. Focused sampling efforts subsequently confirmed the occurrence of related sporocysts in predatory snakes,[1033,1034] and ongoing efforts are searching for such parasites in the monitor lizards that abound in the vicinity of an important ongoing outbreak. Without a phylogenetic clue as to the likely source of such sporocysts, the rapid progress in understanding the source(s) of human infection would not have been possible.

23.5 EVOLUTIONARY INFERENCES FROM MOLECULAR MARKERS

Molecular phylogeny provides the context for understanding parasite history and evolution in ways too numerous to mention. Important landmarks in our understanding of *Sarcocystis* and the evolution of tissue cyst parasites, however, deserve mention here:

a. *Sarcocystis* comprises an evolutionary ancient and diverse group. They parasitize all tetrapod groups, and likely have been diversifying over that entire period. Unlike the *Eimeria,* which show signs of early diversification in fish hosts,[1208] the ultimate origins of tissue cyst forming coccidia remains ill-defined. However, their extant diversity (over 150 named species) seems certain to grow as more hosts are examined and the tools of modern genetics are applied to their diagnosis.
b. Not all genera are created equal. As opposed to *Sarcocystis*, which signifies a broad and ecologically diverse assemblage of parasites, other generic names demarcate far less diverse biological lineages. We speak of strains of *Toxoplasma* but not of other coccidia; the paucity of known species of *Hammondia* and *Neospora* also stand in stark contrast to the diverse and deeply branching *Sarcocystis.* Together, the Toxoplasmatinae (including *T. gondii, N. caninum,* and species of *Hammondia* and *Besnoitia*) constitute a sister group to *Sarcocystis,* which itself comprises greater numbers of species and probably has existed for far longer. Molecular phylogenies also served as the basis for subsuming *Frenkelia* within *Sarcocystis* on grounds of common evolutionary heritage. These genera were initially separated by location of tissue cysts in brain (*Frenkelia*) and in muscle (*Sarcocystis*).

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c. Definitive hosts have bequeathed parasites to the prey upon which they feed. It was noted that, among parasites encysting in ruminant intermediate hosts, those cycling through cats descended from a lineage distinct from the one encompassing those cycling through dogs.[843] A reasonable conclusion was that an ancestral parasite in a feline ancestor gave rise to those now specific to each intermediate host, and the same was true for the ancestor in an ancestral canine. That pattern generally holds up when considering the deep divergences in the group's evolutionary history: the parasites of a given definitive host share an especially close evolutionary history. The successful search for definitive snake hosts of *S. nesbitti*, summarized above, also illustrates this notion. Although it is also true that related intermediate hosts can share related species of parasite, instances of host shifts are notable (e.g., *S. neurona* and *S. falcatula* infect mammals and birds but share a common definitive host). From an evolutionary perspective, feline transmitted *Sarcocystis* species will eventually disappear from livestock because the sarcocysts stage of feline transmitted species takes several months or years to mature, and by that time the livestock are slaughtered for meat.
d. Phylogenies have also illustrated the flexibility of life-history attributes. The evolutionary histories of parasites have, of necessity, evolved the means to use the same species as its definitive and intermediate host. It was shown that these species are closely related to parasites that complete their life cycles in the expected 2-host cycle, indicating that this life-cycle has originated at least twice,

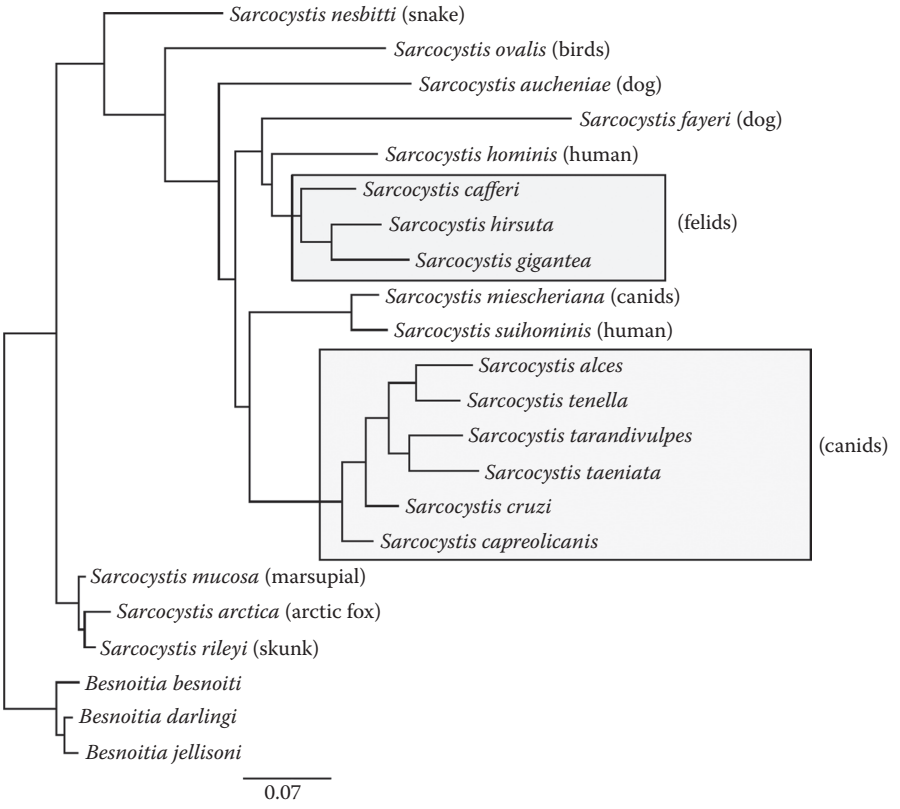


Figure 23.1 Phylogenetic relationships among species of *Sarcocystis* with known or suspected definitive hosts, reconstructed from 18S rDNA sequences aligned using MAFFT[947] under the criterion of maximum likelihood as implemented by PhyML[749] using Geneious 8.2.5. Parasites of felids appear to derive from a single common ancestor, which might have been acquired from a human or canid host. Parasites of more diverse definitive hosts (mustelids, marsupials, reptiles, and birds) represent distinct lineages.

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independently.[1623] This result is interesting in light of the major life-history departure that evidently sets apart *T. gondii* from (so far as we know) all other tissue cyst forming coccidians: its ability to circumvent (at least for a time) definitive hosts altogether.[1690]

The transmission of each species of *Sarcocystis* requires a definitive host to feed upon contaminated prey and to contaminate the environment inhabited by competent intermediate hosts. It is therefore interesting to consider the phylogenetic evidence suggesting how new parasite species have become established in new hosts. Although all infected prey species of a given predator or scavenger might conceivably be suited to becoming a reservoir of infection and although all animals imbibing water or consuming vegetation contaminated with the feces of each infected carnivore might acquire new infections, actual cycles of transmission tend to be much more circumscribed. Until the definitive host of many more parasites has been established, our view of this process will remain clouded. But even the fragmentary data now available (Figure 23.1) suggest that certain species transmitted by felid-definitive hosts had a common ancestor (presumably also transmitted by cats) and that these in turn may have derived from an ancestor in human or canine hosts. Greater evolutionary diversity characterizes species of *Sarcocystis* transmitted by other hosts (including marsupials, mustelids, reptiles, and birds), indicating that an important constraint in the elaboration of diversity in this parasite group has been imposed by adaptations to particular definitive host lineages.

BIBLIOGRAPHY

General references: **109, 123, 263, 293, 498, 656, 668, 850, 1110, 1124, 1240, 1331, 1436, 1532, 1719, 1758, 1864, 1876, 1877**.

18S rDNA: **14, 27, 77, 78, 144, 145, 153, 161, 162, 164, 166, 168, 198, 211, 271, 280–286, 290, 305, 330, 331, 466, 467, 473, 476, 490, 494, 495, 497, 513, 518, 600, 601, 608, 653, 655, 684, 699, 700, 706, 775, 783, 788, 840, 841, 843, 856, 904–906, 909, 956, 969, 983, 1003, 1005, 1007, 1020, 1025, 1029, 1033, 1034, 1056, 1095, 1113, 1196, 1221, 1229, 1230, 1237, 1325–1327, 1329, 1340, 1415, 1416, 1419–1421, 1427, 1432, 1473, 1620–1623, 1678, 1722, 1732, 1736, 1743, 1776, 1780, 1788, 1799, 1818, 1844, 1846, 1851, 1854, 1856–1858**.

Other nuclear genes: **44, 45, 433, 441, 442, 453, 459, 460, 466, 467, 473, 476, 490, 496, 519, 520, 521, 633, 702–706, 719, 723, 842, 902, 903, 1004–1009, 1108, 1116, 1194, 1196, 1197, 1235, 1236, 1301, 1325–1327, 1329, 1330, 1332, 1415, 1419–1421, 1432, 1464, 1473, 1492, 1493, 1623, 1675, 1693, 1722, 1788, 1821, 1843, 1879**.

Plastid genes: **453, 902, 1301**.

Mitochondrial genes: **473, 702, 1477, 1941**.

Surface antigens: **1209.**

Microsatellites: **44, 45, 1693, 1821**.

Epidemiological insights: **14, 453, 559, 560, 601, 699, 877, 1033, 1034, 1722, 1743, 1859**.

Evolutionary inferences: **843, 1208, 1623, 1690**.

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CHAPTER 24

Current Status of *Sarcocystis* Species

Initial revisions on species-systematic of genus *Sarcocystis* by Levine and Tadros[1048] reported 93 species in 1980; later, Levine[1049] listed 122 in 1986; and more recently, Odening[1317] listed 189 in 1998. Here, we summarize the current status in 7 categories A–G (Tables 24.1 and 24.2): **A**, species with full life cycle known, including endogenous stages; **B**, with definitive and intermediate hosts known; **C**, with definitive host unknown; **D**, partial description of sarcocysts; **E**, *species inquirendae*; **F**, species invalid; and **G**, species synonymized here.

Table 24.3 summarizes 85 unnamed *Sarcocystis* species reported mostly once in different hosts. All species are listed alphabetically in Table 24.4.

Table 24.1 List of Valid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
A	*S. arieticanis*	9
	S. bertrami	11
	S. calchasi	17
	S. capracanis	9
	S. cernae	21
	S. clethrionomyelaphis	22
	S. cruzi	7
	S. dispersa	21
	S. falcatula	17
	S. fayeri	11
	S. glareoli	21
	S. hemionilatrantis	18
	S. hircicanis	9
	S. hirsuta	7
	S. idahoensis	21, 22
	S. microti	21
	S. miescheriana	6
	S. murinotechis	21, 22
	S. muris	21
	S. muriviperae	21, 22
	S. neurona	3A
	S. rauschorum	21
	S. singaporensis	21
	S. tenella	8

(*Continued*)

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Table 24.1 (*Continued*) List of Valid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
	S. villivillosi	21, 22
	S. zamani	21
B	*S. acanthocolubri*	22
	S. accipitris	17
	S. albifronsi	17
	S. alces	18
	S. alceslatrans	18
	S. alectoributeonis	17
	S. alectorivulpes	17
	S. ameivamastigodryasi	22
	S. atheridis	21, 22
	S. aucheniae	13
	S. baibacinacanis	21
	S. buffalonis	10
	S. cameli	12
	S. campestris	21
	S. capreolicanis	18
	S. cervicanis	18
	S. chalcidicolubris	22
	S. citellibuteonis	21
	S. crotali	21, 22
	S. cuniculorum	21
	S. cymruensis	21
	S. disrumpens	21
	S. dugesii	22
	S. espinosai	21
	S. ferovis	18
	S. fusiformis	10
	S. gerbilliechis	21
	S. gigantea	8
	S. gongyli	22
	S. gracilis	18
	S. grueneri	18
	S. hjorti	18
	S. hoarensis	21
	S. hominis	4, 7
	S. ippeni	12
	S. lacertae	22
	S. leporum	21
	S. levinei	10
	S. lindsayi	17
	S. medusiformis	8
	S. moulei	9
	S. neotomafelis	21
	S. odocoileocanis	18
	S. odoi	18

(*Continued*)

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Table 24.1 (*Continued*) List of Valid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
	S. ovalis	18
	S. podarcicolubris	22
	S. poephagi	18
	S. poephagicanis	18
	S. putorii	21
	S. rangi	18
	S. rileyi	17
	S. rommeli	7
	S. sebeki	21
	S. simonyi	22
	S. stehlinii	22
	S. stenodactylicolubris	22
	S. suihominis	4, 6
	S. sybillensis	18
	S. tarandivulpes	18
	S. wapiti	18
	S. wenzeli	16
	S. zuoi	21
C	*S. africana*	18
	S. americana	18
	S. anasi	17
	S. arctica	19
	S. arctosi	19
	S. azevedoi	22
	S. balaenopteralis	18
	S. booliati	21
	S. bozemanensis	21
	S. bunopusi	21
	S. cafferi	18
	S. camelopardalis	18
	S. caninum	14
	S. canis	3B
	S. capricornis	18
	S. chamaeleonis	22
	S. chloropusae	17
	S. clethrionomysi	21
	S. columbae	17
	S. cornagliai	18
	S. cornixi	17
	S. corsaci	19
	S. corvusi	17
	S. danzani	18
	S. dasypi	21
	S. didelphidis	20
	S. diminuta	21
	S. dubeyella	18

(*Continued*)

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Table 24.1 (*Continued*) List of Valid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
	S. dubeyi	10
	S. elongata	18
	S. eothenomysi	21
	S. felis	15
	S. gallotiae	22
	S. garnhami	20
	S. gazellae	18
	S. giraffae	18
	S. greineri	20
	S. hardangeri	18
	S. hemioni	18
	S. hippopotami	18
	S. hofmanni	19
	S. horvathi	16
	S. hydrurgae	18
	S. inghami	20
	S. jaypeedubeyi	21
	S. jorrini	18
	S. kalkivus	19
	S. kinosterni	22
	S. kirkpatricki	19
	S. kirmsei	17
	S. kitikmeotensis	19
	S. klaseriensis	18
	S. kortei	5
	S. lari	17
	S. lutrae	19
	S. markusi	5
	S. marmosae	20
	S. melampi	18
	S. melis	19
	S. mephitisi	19
	S. mitrami	22
	S. mongolica	18
	S. montanaensis	21
	S. mucosa	20
	S. nesbitti	22
	S. ochotonae	21
	S. oryzomyos	21
	S. oviformis	18
	S. peromysci	21
	S. phacochoeri	18
	S. phoeniconaii	17

(*Continued*)

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Table 24.1 (*Continued*) List of Valid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
	S. proechimyos	21
	S. ramphastosi	17
	S. rangiferi	18
	S. scandinavica	18
	S. scelopori	22
	S. schneideri	22
	S. sehi	21
	S. sibirica	18
	S. sigmodontis	21
	S. silva	18
	S. speeri	20
	S. sulawesiensis	21
	S. sulfuratusi	17
	S. svanai	14
	S. taeniata	18
	S. tarandi	18
	S. truncata	18
	S. tupaia	21
	S. turcicii	22
	S. turdusi	17
	S. ursusi	19
	S. wobeseri	17
	S. woodhousei	18
	S. youngi	18
D	*S. dogeli*	21
	S. erdmanae	19
	S. eutamias	21
	S. eversmanni	19
	S. fedoseenkoi	21
	S. galuzoi	21
	S. hueti	18
	S. orientalis	18
	S. platydactyliscelopori	22
	S. pseudois	18
	S. rhombomys	21
	S. richardii	21
	S. undulati	21

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Table 24.2 List of Invalid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
E	*S. capreoli*	18
	S. colii	17
	S. ctenosauris	NR
	S. garzettae	17
	S. kaiserae	17
	S. lacertaeutae	22
	S. lampropeltis	22
	S. microps	8
	S. mihoensis	8
	S. nelsoni	18
	S. novaki	18
	S. peckai	17
	S. porcifelis	6
	S. russuli	21
	S. saiga	18
	S. salvelini	22
	S. setophagae	17
	S. spaldingae	17
	S. talpini	21
	S. terrestri	NR
	S. vulpis	19
F	*S. ammodrami*	17
	S. aramidis	17
	S. asinus	11
	S. atlanticae	22
	S. atractaspidis	22
	S. bettongiae	21
	S. bubalis	NR
	S. caviae	21
	S. cervi	18
	S. cheeli	NR
	S. citellivulpes	21
	S. cricetuli	21
	S. gusevi	18
	S. jacarinae	17
	S. miescheri	12
	S. nontenella	17
	S. oliverioi	NR
	S. otus	17
	S. pitymysi	21
	S. platydactyli	22
	S. pythonis	22
	S. roudabushi	NR
	S. scotti	NR

(*Continued*)

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Table 24.2 (*Continued*) List of Invalid *Sarcocystis* Species

Category	*Sarcocystis* Species	Chapter
	S. tamanduae	21
	S. tropicalis	NR
	S. turdi	17
G	*S. buteonis*	21
	S. cuniculi	21
	S. equicanis	11
	S. rodentifelis	21

NR = not reported in this book.

Table 24.3 List of the 85 Unnamed *Sarcocystis* Species

Sarcocystis Species	Intermediate Host	Main Reference	Chapter
Nonhuman Primates			
S. sp. Mehlhorn, Heydorn, and Janitschke 1977	Rhesus monkey (*Macaca mulatta*)	**1175**	5
S. sp. Mehlhorn, Heydorn, and Janitschke 1977	Baboon (*Papio cynocephalus*)	**1175**	5
S. sp. Mehlhorn, Heydorn, and Janitschke 1977	Tamarin (*Saguinus oedipus*)	**1175**	5
Wild Ruminants			
S. sp. Saito, Itagaki, Shibata, and Itagaki 1995	Sika deer (*Cervus nippon centralis*)	**1515**	18
S. sp. Gangadharan, Valsala, Nair, and Rajan 1992	Sambar deer (*Cervus unicolor*)	**660**	18
S. sp. Shrivastav, Sharma, Chaudhry, and Malik 1999	Barasingha or hard-ground swamp deer (*Cervus duvauceli branderi*)	**1605**	18
S. sp. Poli, Mancianti, Marconcini, Nigro, and Colagreco 1988	Fallow deer (*Dama dama*)	**1404**	18
S. cf. *hofmanni* Wesemeier and Sedlaczek 1995	Fallow deer (*Dama dama*)	**1823**	18
S. cf. *grueneri* Wesemeier and Sedlaczek 1995	Fallow deer (*Dama dama*)	**1823**	18
S. sp. Dubey and Lozier 1983	White-tailed deer (*Odocoileus virginianus*)	**378**	18
S. sp. type II Atkinson, Wright, Telford, Mclaughlin, Forrester, Roelke, and Mccown 1993	White-tailed deer (*Odocoileus virginianus*)	**46**	18
S. sp. Stolte, Bockhardt, and Odening 1996	Wapiti or North American elk (*Cervus elaphus canadensis*)	**1680**	18
S. sp. Odening, Rudolph, Quandt, Bengis, Bockhardt, and Viertel 1998	Springbok (*Antidorcas marsupialis*), Impala (*Aepyceros melampus*)	**1320**	18
S. sp. cf. *hominis* Odening, Rudolph, Quandt, Bengis, Bockhardt, and Viertel 1998	Greater kudu (*Tragelaphus strepsiceros*)	**1320**	18

(*Continued*)

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Table 24.3 (*Continued*) List of the 85 Unnamed *Sarcocystis* Species

Sarcocystis Species	Intermediate Host	Main Reference	Chapter
S. sp. cf. *medusiformis* Stolte, Odening, and Bockhardt 1996	Addax (*Addax nasomaculatus*)	**1679**	18
S. sp. Odening, Stolte, Lux, and Bockhardt 1996	Mongolian gazelle (*Procapra gutturosa*)	**1313**	18
S. sp. cf. *capracanis* Stolte, Odening, and Bockhardt 1996	Blackbuck (*Antilope cervicapra*)	**1679**	18
S. sp. cf. *hominis* Stolte, Odening, and Bockhardt 1996	Common eland (*Taurotragus oryx*)	**1679**	18
S. sp. cf. *cruzi* Stolte, Odening, and Bockhardt 1996	Nilgai (*Boselaphus tragocamelus*), Blackbuck (*Antilope cervicapra*)	**1679**	18
S. sp. Foreyt 1989	Mountain goat (*Oreamnos americanus*)	**619**	18
S. sp. Dubey 1980	Pronghorn (*Antilocapra americana*)	**355**	18
S. sp. Odening, Stolte, and Bockhardt 1996	Chamois (*Rupicapra rupicapra*)	**1311**	18
S. sp. Kannangara 1970	Mouse deer (*Moschiola meminna*)	**941**	18
S. cornagliai-like (type I) Cornaglia, Giaccherino, and Peracino 1998	Alpine ibex or steinbock (*Capra ibex*)	**253**	18
S. capracanis-like (type II) Cornaglia, Giaccherino, and Peracino 1998	Alpine ibex or steinbock (*Capra ibex*)	**253**	18
S. *arieticanis*-like (type III) Cornaglia, Giaccherino, and Peracino, 1998	Alpine ibex or steinbock (*Capra ibex*)	**253**	18
S. capricornis-like (type IV) Cornaglia, Giaccherino, and Peracino 1998	Hispanic ibex (*Capra pyrenaica hispanica*)	**253**	18
S. sp. Agrawal, Chauhan, and Ahluwalia 1982	Himalayan goral (*Naemorhedus goral*)	**1898**	18
S. sp. Rioseco, Cubillos, Gonzalez, and Diaz 1976	Pudu (*Pudu pudu*)	**2252**	18
S. sp. Samuel and Gray 1974	Musk ox (*Ovibos moschatus*)	**2281**	18
	Marine Mammals		
S. sp. Mense, Dubey, and Homer 1992	Sea lion (*Zalophus californianus*)	**1184**	18
S. sp. Bishop 1979	Bearded seal (*Erignathus barbatus*)	**105**	18
S. sp. Migaki and Albert 1980	Ringed seal (*Pusa hispida*)	**1192**	18
S. sp. Brown, Smith, and Keyes 1974	Northern fur seal (*Callorhinus ursinus*)	**147**	18
S. sp. De Guise, Lagacé, Girard, and Béland 1993	Beluga whale (*Delphinapterus leucas*)	**310**	18
S. sp. Munday and Mason 1978	Sperm whale (*Physeter catodon*)	**1249**	18
S. sp. De Guise, Lagacé, Girard, and Béland 1993	Atlantic white-sided dolphin (*Lagenorhyncus acutus*)	**310, 562**	18
S. sp. Munday and Mason 1978	*Delphinus* sp.	**1249**	18

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Table 24.3 (*Continued*) List of the 85 Unnamed *Sarcocystis* Species

Sarcocystis Species	Intermediate Host	Main Reference	Chapter
S. sp. Dailey and Stroud 1978	Striped dolphin (*Stenella coeruleoalba*)	**287**	18
S. sp. Dubey, Lindsay, Rosenthal, and Thomas 2003	Sea otter (*Enhydra lutris*)	**453**	18
	Wild Carnivores		
S. sp. 1 Stolte, Odening, Walter, and Bockhardt 1996	Raccoon (*Procyon lotor*)	**1681**	19
S. sp. 2 Stolte, Odening, Walter, and Bockhardt 1996	Raccoon (*Procyon lotor*)	**1681**	19
S. sp. 3 (sf. *S. sebeki*) Stolte, Odening, Walter, and Bockhardt 1996	Raccoon (*Procyon lotor*)	**1681**	19
S. sp. Pak 1991	Red fox (*Vulpes vulpes*)	**1359**	19
S. sp. 1 Wesemeier, Odening, Walter, and Bockhardt 1995	Jackal (*Canis melomelas*)	**1825**	19
S. sp. 2 Wesemeier, Odening, Walter, and Bockhardt 1995	Jackal (*Canis melomelas*)	**1825**	19
S. sp. Cummings, Kocan, Barker, and Dubey 2000	Coyote (*Canis latrans*)	**268**	19
S. sp. Bwangamoi, Ngatia, and Richardson 1993	Wild dog (*Lycacon pictus*)	**159**	19
S. sp. cf. *sebeki* Odening, Stolte, Walter, and Bockhardt 1994	European badger (*Meles meles*)	**1303, 1304**	19
S. sp. cf. *gracilis* Odening, Stolte, Walter, Bockhardt, and Jakob 1994	European badger (*Meles meles*)	**1304**	19
S. sp. Kubo, Okano, Ito, Tsubota, Sakai, and Yanai 2009	Japanese badger (*Meles meles anakuma*)	**982**	19
S. sp. Wahlström, Nikkila, and Uggla 1999	Otter (*Lutra lutra*)	**1803**	19
S. sp. (most likely *S. neurona*) Ramos-Vara, Dubey, Watson, Winn-Elliot, Patterson, and Yamini 1997	Mink (*Mustela vison*)	**1445**	19
S. sp. Tadros and Laarman 1979	Common European weasel (*Mustela nivalis*)	**1711**	19
S. sp. Larkin, Gabriel, Gerhold, Yabsley, Wester, Humphreys, Beckstead, and Dubey 2011	Fisher (*Martes pennanti*)	**1025**	19
S. sp. Dubey, Humphreys, and Fritz 2008	Black bear (*Ursus americanus*)	**462**	19
S. sp. Cheadle, Cunningham, and Greiner 2002	Florida black bear (*Ursus americanus floridanus*)	**206**	19
S. sp. Somvanshi, Koul, and Biswas 1987	Leopard (*Panthera pardus*)	**1644**	19
	Small Mammals		
S. dispersa-like Munday 1983	Norway rat (*Rattus norvegicus*)	**1256**	21
S. sp. cf. *singaporensis* Kan and Dissanaike 1977	Malaysian house rats (*Rattus rattus diardii*)	**936**	21
S. sp. type B Rzepczyk and Scholtyseck 1976	Bush rat (*Rattus fuscipes*)	**1504**	21

(*Continued*)

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Table 24.3 (*Continued*) List of the 85 Unnamed *Sarcocystis* Species

Sarcocystis Species	Intermediate Host	Main Reference	Chapter
S. montanaensis-like Lindsay, Upton, Blagburn, Toivio-Kinnucan, Mcallister, and Trauth 1991	Prairie vole (*Microtus achrogaster*)	**1065**	21
S. cf. *cuniculi* Brumpt 1913	European hare (*Lepus europaeus*)	**1305**	21
S. sp. 1 Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	European hare (*Lepus europaeus*)	**1305**	21
S. sp. 2 Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	European hare (*Lepus europaeus*)	**1305**	21
S. cf. *bertrami* Odening, Wesemeier, Pinkowski, Walter, Sedlaczek, and Bockhardt 1994	European hare (*Lepus europaeus*)	**1305**	21
S. sp. 1 Odening, Wesemeier, and Bockhardt 1996	European hare (*Lepus europaeus*)	**1310**	21
S. sp. 2 Odening, Wesemeier, and Bockhardt 1996	European hare (*Lepus europaeus*)	**1310**	21
S. sp. Lindsay, Mckown, Upton, Mcallister, Toivio-Kinnucan, Veatch, and Blagburn 1996	9-banded armadillo (*Dasypus novemcinctus*)	**1067**	21
S. sp. Scholtyseck, Entzeroth, and Chobotar 1982	Virginia opossum (*Didelphis virginiana*)	**1567**	21
S. sp. Entzeroth, Chobotar, and Scholtyseck 1983	Red squirrel (*Tamiasciurus hudsonicus*)	**535**	21
S. sp. Entzeroth, Scholtyseck, and Chobotar 1983	Eastern chipmunk (*Tamias striatus*)	**536**	21
S. sp. Kannangara 1970	Bandicoot (*Bandicota malabarica*)	**941**	21
	Birds		
S. sp. type A Crawley, Ernst, and Milton 1982	Bald eagle (*Haliaeetus leucocephalus*)	**261**	17
S. sp. type B Olson, Wunschmann, and Dubey 2007	Bald eagle (*Haliaeetus leucocephalus*)	**1333**	17
S. sp. Wenzel, Erber, Boch, and Schellner 1982	Pheasant (*Phasianus colchicus*)	**1822**	17
S. sp. Hiller, Sidor, De Guise, and Barclay 2007	American woodcock (*Scolopax minor*)	**829**	17
S. sp. Dubey, Johnson, Bermudez, Suedmeyer, and Fritz 2001	Straw-necked ibis (*Carphibis spinicollis*)	**437**	17
S. sp. Wenzel, Erber, Boch, and Schellner 1982	Coot (*Fulica atra*)	**1822**	17
S. sp. Teglas, Little, Latimer, and Dubey 1998	Turkey (*Meleagris gallopavo*)	**1728**	17
S. sp. Spalding, Yowell, Lindsay, Greiner, and Dame 2002	Northern gannet (*Morus bassanus*)	**1647**	17
S. sp. Dubey and Morales 2006	Buffon's macaw (*Ara ambigua*)	**1989**	17
S. sp. Kutkiené, Sruoga, and Butkauskas 2008	Goldeneye (*Bucephala clangula*)	**1004**	17

(*Continued*)

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Table 24.3 (*Continued*) List of the 85 Unnamed *Sarcocystis* Species

Sarcocystis Species	Intermediate Host	Main Reference	Chapter
	Reptiles		
S. sp. Roberts, Wellehan, Weisman, Rush, Childress, and Sibley 2014	South American rattlesnake (*Crotalus durissus terrificus*)	**1474**	22
	Fish		
S. sp. Fantham and Porter 1943	Eel (*Zoarces angularis*)	**566**	22

Table 24.4 List of Named *Sarcocystis* Species (Alphabetized)

S. acanthocolubri	*S. cervicanis*
S. accipitris	*S. chalcidicolubris*
S. africana	*S. chamaeleonis*
S. albifronsi	*S. chloropusae*
S. alces	*S. citellibuteonis*
S. alceslatrans	*S. clethrionomyelaphis*
S. alectoributeonis	*S. clethrionomysi*
S. alectorivulpes	*S. columbae*
S. ameivamastigodryasi	*S. cornagliai*
S. americana	*S. cornixi*
S. anasi	*S. corsaci*
S. arctica	*S. corvusi*
S. arctosi	*S. crotali*
S. arieticanis	*S. cruzi*
S. atheridis	*S. cuniculorum*
S. aucheniae	*S. cymruensis*
S. azevedoi	*S. danzani*
S. baibacinacanis	*S. dasypi*
S. balaenopteralis	*S. didelphidis*
S. bertrami	*S. diminuta*
S. booliati	*S. dispersa*
S. bozemanensis	*S. disrumpens*
S. buffalonis	*S. dogeli*
S. bunopusi	*S. dubeyella*
S. cafferi	*S. dubeyi*
S. calchasi	*S. dugesii*
S. cameli	*S. elongata*
S. camelopardalis	*S. eothenomysi*
S. campestris	*S. erdmanae*
S. caninum	*S. espinosai*
S. canis	*S. eutamias*
S. capracanis	*S. eversmanni*
S. capreolicanis	*S. falcatula*
S. capricornis	*S. fayeri*
S. cernae	*S. fedoseenkoi*

(*Continued*)

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Table 24.4 (*Continued*) List of Named *Sarcocystis* Species (Alphabetized)

S. felis
S. ferovis
S. fusiformis
S. gallotiae
S. galuzoi
S. garnhami
S. gazellae
S. gerbilliechis
S. gigantea
S. giraffae
S. glareoli
S. gongyli
S. gracilis
S. greineri
S. grueneri
S. hardangeri
S. hemioni
S. hemionilatrantis
S. hippopotami
S. hircicanis
S. hirsuta
S. hjorti
S. hoarensis
S. hofmanni
S. hominis
S. horvathi
S. hueti
S. hydrurgae
S. idahoensis
S. inghami
S. ippeni
S. jaypeedubeyi
S. jorrini
S. kalkivus
S. kinosterni
S. kirkpatricki
S. kirmsei
S. kitikmeotensis
S. klaseriensis
S. kortei
S. lacertae
S. lari
S. leporum
S. levinei
S. lindsayi
S. markusi
S. marmosae
S. medusiformis
S. melampi
S. melis
S. mephitisi
S. microti
S. miescheriana
S. mitrami
S. mongolica
S. montanaensis
S. moulei
S. mucosa
S. murinotechis
S. muris
S. muriviperae
S. neotomafelis
S. nesbitti
S. neurona
S. ochotonae
S. odocoileocanis
S. odoi
S. orientalis
S. otus
S. oryzomyos
S. ovalis
S. oviformis
S. peromysci
S. phacochoeri
S. phoeniconaii
S. platydactyliscelopori
S. podarcicolubris
S. poephagi
S. poephagicanis
S. proechimyos
S. pseudois
S. putorii
S. ramphastosi
S. rangi
S. rangiferi
S. rauschorum
S. rhombomys
S. richardii
S. rileyi
S. rommeli
S. scandinavica
S. scelopori
S. schneideri
S. sebeki
S. sehi
S. sibirica

(*Continued*)

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Table 24.4 (*Continued*) List of Named *Sarcocystis* Species (Alphabetized)

S. silva	*S. sigmodontis*
S. simonyi	*S. truncata*
S. singaporensis	*S. tupaia*
S. speeri	*S. turcicii*
S. stehlinii	*S. turdusi*
S. stenodactylicolubris	*S. undulati*
S. suihominis	*S. ursusi*
S. sulawesiensis	*S. villivillosi*
S. sulfuratusi	*S. wapiti*
S. svanai	*S. wenzeli*
S. sybillensis	*S. wobeseri*
S. taeniata	*S. woodhousei*
S. tarandi	*S. youngi*
S. tarandivulpes	*S. zamani*
S. tenella	*S. zuoi*

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