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The Pathoparasitology of Nonhuman Primates: A Review

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Introduction

Most people who have had more than cursory experience in the husbandry of nonhuman primate colonies will agree that parasitism is one of the most common disease entities that affects these animals. Numerous protozoal and metazoal genera have been described as infecting the members of all major nonhuman primate groups. Many of these are considered to be nonpathogenic, or at least their detrimental effects upon the host have yet to be eludicated. A large number, however, can produce lesions that result in serious debilitation and can create opportunities for secondary infections that may be fatal. This process appears to be exacerbated by the stress of capture and confinement.

This paper discusses the anatomic features of protozoan and metazoan parasitic infections of nonhuman primates. Since the paper is essentially a review of the literature, an extensive bibliography is included for those readers who wish to pursue the subject in greater detail. All tables in the text follow a published system [290]. Major nonhuman primate groups are classified according to a published taxonomy system [621]. Prosimians include the species in the families Tupaiidae; New World monkeys include the species in the families Callitrichidae and Cebidae; Old World monkeys include the species in the family Cercopithecidae; and the great apes include the species in the families Hylobatidae and Pongidae.

The numerous articles published previously that discuss the subject of nonhuman primate parasitology as a specific entity, or as a part of an overall discussion of general parasitology, systemic pathology, or the broad topic of nonhuman primate diseases, are outlined as follows: enteric and somatic protozoa [23, 72, 90, 338, 369, 370, 433, 443, 529, 604]; hemoprotozoa [2, 123, 135, 140, 236, 320, 372, 556, 797, 874, 928]; nematodes [650]; trematodes [147, 482]; cestodes [608]; acanthocephalans [770]; arthropods [92, 207, 281, 290, 302, 395, 405, 413, 457, 938]; pentastomids [788]; general parsitology [32, 339, 416, 427, 484, 487, 497, 499, 528, 530, 531, 791, 810]; systemic pathology [21, 97, 367, 429, 542, 572, 594, 774, 820, 846]; and nonhuman primate diseases [14, 34, 35, 112, 134, 312, 373, 407, 424, 431, 438, 440, 461, 469, 508, 570, 584, 585, 742, 746, 869].

In addition, there are many papers in the literature concerning parasitological surveys and parasite checklists for specific species of nonhuman primates. These

K. Benirschke (ed.), *Primates*

are referenced as follows: rhesus monkey, *Macaca mulatta* [1, 220, 434, 435, 447, 680, 716, 741, 859, 912]; cynomolgus monkey, *Macaca fascicularis* [1, 45, 362, 393, 763, 912]; pig-tail monkey, *Macaca nemestrina* [912]; bonnet monkey, *Macaca radiata*, [912]; stump-tail monkey, *Macaca arctoides* [912]; Barbary ape, *Macaca sylvanus* [91]; Formosan rock macaque, *Macaca cyclopis* [490, 494]; African green monkey, *Cercopithecus* sp. [518]; silvered leaf monkey, *Presbytis cristatus* [3, 658, 659, 660]; chacma baboon, *Papio ursinus* [336, 578]; yellow baboon, *Papio cynocephalus* [485, 497, 616]; olive baboon, *Papio anubis*, syn. *P. doguera* [486, 488, 611]; baboons, *Papio* sp. [1, 573, 609, 610, 626, 823]; gelada baboon, *Theropithecus gelada* [493]; tamarin, *Saguinus fusciollis* [148]; owl monkey, *Aotus trivirgatus* [895]; squirrel monkey, *Saimiri sciureus* [584]; New World monkeys [121, 437, 492]; slender loris, *Loris tardigradus* [477]; tarsiers, *Tarsius bancanus* [61, 387]; chimpanzee, *Pan* sp. [284, 491, 613, 863]; and laboratory primates [712, 764, 911, 912].

References for additional reading on nonhuman primate parasitology, associated lesions, and related subjects outside the scope of this paper are as follows: pathology [290, 406, 427, 428, 742, 791]; necropsy techniques [910]; recognition of parasites [607]; comparative parasitology [127]; experimental parasitology [483, 643]; identification of parasitic eggs [421]; identification of parasites in tissue sections [128, 307, 848]; parasitic zoonoses [417, 432, 529, 931]; and parasite control [155].

Protozoan Parasites

Flagellates

The enteric flagellates and hemoflagellates described in nonhuman primates are listed in Table 45.1. All of the enteric flagellates are considered nonpathogenic except *Giardia lamblia*, which has been reported to cause diarrhea in monkeys [290, 742]. The ability of *Giardia* to cause disease is controversial, however, and the factors that govern its pathogenicity are not completely understood [529]. The majority of the hemoflagellates reported from nonhuman primates are also considered to be nonpathogenic. An exception is *Trypanosoma cruzi*, which is an important pathogen and is the cause of Chagas disease in humans. Many species of wild and domestic animals have been found to be infected with this parasite, including nonhuman primates, and it is speculated that most mammals are susceptible. In addition, since it is an important pathogen of humans, it has potentially serious public health implications and will be discussed in detail below.

South American Trypanosomiasis (Chagas Disease)

The cause of this disease, the hemoflagellate *Trypanosoma cruzi*, is distributed throughout South and Central America with extension into the southern and southwestern regions of the United States. Dr. Chagas made the original description of this parasite in *Callithrix penicillata* from Brazil [23, 109]. He also reported the first case of natural infection in the squirrel monkey [110]. Natural *T. cruzi* infection has been reported from numerous New World primate species (squirrel monkeys, marmosets, spider monkeys, cebus monkeys, and uakaris) [6, 20, 25, 32, 88, 159, 160, 212, 219, 290, 511, 559, 812]. *Trypanosoma cruzi* also

TABLE 45.1. Parasitic flagellates described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Hemoflagellates						
Trypanosoma cruzi	Blood, RE cells, muscle, heart, other tissues		X	X	X	6, 20, 23, 25, 32, 88, 109, 110, 159, 160, 212, 219, 290, 511, 559, 560, 782, 789, 812
Trypanosoma sanmartini	Blood		X			23, 212, 290, 324
Trypanosoma minasense	Blood		X			23, 109, 159, 161, 212, 290, 559, 560, 728
Trypanosoma rangeli	Blood		X			23, 212, 290, 342, 343, 528
Trypanosoma saimirii	Blood		X			23, 162, 212, 290, 728
Trypanosoma diasi	Blood		X			23, 162, 163, 212, 290
Trypanosoma lambrechti	Blood		X			23, 166, 212, 559, 560
Trypanosoma primatum	Blood				X	23, 72, 218, 290, 713, 778, 779, 936
Trypanosoma brucei	Blood			X		23
Trypanosoma perodictici	Blood	X				23, 37, 40, 41, 72, 388, 713, 779
Trypanosoma irangiense	Blood	X				23, 39, 41, 388
Trypanosoma sp.	Blood	X				136, 205, 213, 354, 357, 496, 620, 933
Enteric flagellates						
Trichomitus wenyoni	Cecum, colon			X		90, 290, 898
Trichomonas buccalis	Mouth			X		371, 742
Trichomonas tenax	Mouth			X	X	90, 290, 371, 391, 529
Trichomonas foetus	Intestine	X				61
Trichomonas hominis	Intestine				X	491, 494
Trichomonas sp.	Intestine	X	X			168, 387, 895
Pentatrichomonas hominis	Cecum, colon		X	X	X	290, 369, 371, 529, 680
Enteromonas hominis	Cecum			X	X	90, 290, 491, 530, 712
Retortamonas intestinalis	Cecum			X	X	90, 290, 529, 530

(Continued)

TABLE 45.1. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Chilomastix mesnili	Cecum, colon		X	X	X	90, 290, 369, 371, 485, 491, 530, 609, 654, 680, 712
Chilomastix tarsii	Intestine	X				61, 387, 685
Chilomastix sp.	Intestine		X			885
Hexamita pitheci	Cecum, colon			X	X	156, 290, 529
Hexamita sp.	Cecum			X	X	90, 290, 369, 896
Giardia lamblia	Anterior sm. intestine		X	X	X	90, 290, 491, 529, 530, 742
Giardia sp.	Intestine		X			168, 895

has been reported from Old World monkeys that originated in Asia (*Macaca* sp. and rhesus monkeys) [23, 290, 789, 791] and great apes (gibbon) [782]. Despite the reports in nonhuman primates other than New World monkeys, the question of whether or not *T. cruzi* exists outside the Western Hemisphere remains unanswered. A review of some of the earlier reports of this parasite in Asian monkeys concluded that infection was most probably acquired after the animals were in captivity [23]. Two additional cases have been reported involving rhesus monkeys that were members of colonies which were part of long-term research projects. Infection in these monkeys was considered also to have occurred during captivity, especially since they were housed outdoors in an endemic area of the United States [132, 436].

Two forms of *T. cruzi* are found in susceptible animals. The trypomastigote (trypanosomal) form occurs in the blood. The amastigote (leishmanial) form is found in groups in the cells of skeletal and cardiac muscle, the reticuloendothelial system, and other tissues. The life cycle is indirect with species of several genera of triatomid bugs belonging to the family *Reduviidae* (cone-nose bugs, assassin bugs, or kissing bugs) as biological vectors. For a detailed description of the morphology and life cycle of this parasite, the reader is referred to the following references: 290, 528, 530, and 791.

Infection with *T. cruzi* causes rather nonspecific clinical signs [290]. Generalized edema without necrosis or hemorrhage is said to be common. Anemia, hepatosplenomegaly, and lymphadenitis also can occur [290, 791]. Depression, anorexia, weight loss, and dehydration were seen in the case involving the gibbon [782]. Electrocardiographic patterns consistent with that of right bundle branch block are reported from the cebus monkey [88]. Also, a case of intrauterine death from congenital trypanosomiasis in marmosets has been reported [549].

The lesion mentioned most frequently in all of the naturally occurring cases of *T. cruzi* infection in nonhuman primates is myocarditis, which results in the destruction of myocardial fibers [88, 290, 511, 782]. Histopathologically, the infected myocardium contains numerous, randomly scattered, cystic structures of varying sizes, occupying individual myocardial fibers. These structures are pseudocysts that contain many individual circular to oval-shaped organisms from

1.5 to 4 μ in diameter, with a central nucleus and a prominent bar-shaped structure (kinetoplast). The kinetoplast is an intracellular organelle that is diagnostic for this parasite and aids in differentiating it from the intracellular forms of sarcocystis and toxoplasmosis. These organisms can also be seen filling the cytoplasm of large mononuclear reticuloendothelial (RE) cells, some of which can be multinucleated and have the characteristics of giant cells. Degenerating pseudocysts elicit a focal mononuclear cell inflammatory response, and there may be mild dystrophic mineralization of individual myocardial fibers in some of these areas [436].

The diagnosis of *T. cruzi* can be made by demonstrating and identifying the parasites in blood or other body fluids or in histological sections. Thin and thick blood smears should be stained with Giemsa preparations and examined for the presence of trypanosomes. Animal inoculation and xenodiagnosis can also be used to allow the organism to complete its life cycle. Xenodiagnosis involves the examination of known vectors for trypanosomes after they have been allowed to feed on suspected hosts [23, 290].

Because *T. cruzi* can cause serious disease in humans, all people involved with the care and use of nonhuman primates should exercise extreme care to avoid exposure either by accidental inoculation with trypanosomes or by contamination of mucous membranes or skin with infected material. This should be especially true for those persons working with New World monkeys or with any nonhuman primate maintained in an endemic area in the United States [23, 290].

Sarcodines (Ameba)

The parasitic sarcodines described in nonhuman primates are listed in Table 45.2. All are considered nonpathogenic except *Dientamoeba*, which sometimes can be pathogenic, and *Entamoeba histolytica*, which can cause severe enteric disease in humans and nonhuman primates. *Entamoeba histolytica* will be discussed in detail.

Amebiasis

The cause of this disease, *Entamoeba histolytica*, has a world-wide distribution and has been reported in New World monkeys (spider monkeys, cebus monkeys, woolly monkeys, howler monkeys, squirrel monkeys, and marmosets), Old World monkeys (rhesus monkeys, pig-tailed macaques, bonnet macaques, cynomolgus monkeys, colobus monkeys, proboscis monkeys, African green monkeys, baboons, and langurs), and the great apes (gibbons, orangutans, and chimpanzees) [10, 38, 51, 92, 172, 207, 222, 285, 290, 299, 329, 330, 369, 376, 423, 437, 445, 486, 491–494, 544, 545, 588, 599, 601, 661, 712, 742, 746, 747, 769, 791, 863, 929]. Infection is said to be common in Old World monkeys but uncommon or rare in New World monkeys obtained from their natural habitat [200, 742, 791, 869]. Young monkeys and New World monkeys are reported to sustain more severe lesions from infection with this parasite [46, 90, 207, 223, 704, 742, 869].

The morphology of this parasite has been discussed [90, 182, 290, 389, 529, 530, 748, 791]. Two races of *E. histolytica* are recognized, separated on the basis of size. The pathogenic race is the larger form; the trophozoites measure 20–30

TABLE 45.2. Parasitic sarcodines described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Ameba						
Entamoeba histolytica	Cecum, colon		X	X	X	3, 10, 38, 90, 168, 172, 207, 222, 285, 290, 300, 329, 330, 339, 369, 485, 486, 487, 491, 493, 494, 529, 530, 588, 599, 601, 610, 611, 616, 654, 661, 665, 684, 686, 687, 712, 742, 769, 791, 829, 863, 868, 869, 903, 939
Entamoeba hartmanni	Cecum, colon			X	X	57, 90, 290, 485, 491, 494, 529, 530, 611, 616
Entamoeba coli	Cecum, colon			X	X	90, 168, 290, 485, 486, 487, 491, 493, 494, 529, 530, 578, 616, 654, 680, 712, 741, 863
Entamoeba chattoni	Cecum, colon			X		90, 290, 444, 445, 485, 491, 529, 530, 616
Entamoeba gingivalis	Mouth			X	X	90, 290, 371, 529, 530, 742
Entamoeba polecki	Cecum, colon				X	491, 494, 611
Entamoeba sp.	Cecum, colon		X	X	X	486, 487, 491, 494, 573, 578, 895
Iodamoeba buetschlii	Cecum, colon		X	X	X	90, 207, 290, 369, 371, 485, 486, 487, 493, 494, 529, 530, 553, 611, 616, 712, 897
Iodamoeba wallacei	Intestine			X		719
Iodamoeba sp.	Cecum, colon		X	X		493, 494, 573, 578, 895
Endolimax nana	Cecum, colon		X	X	X	90, 207, 290, 369, 371, 485, 486, 487, 493, 494, 529, 530, 553, 611, 616, 654, 712, 741

TABLE 45.2. (Continued).

Parasite Genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Endolimax sp.	Cecum, colon		X			895
Dientamoeba fragilis	Cecum, colon			X		90, 290, 371, 467, 486, 487, 529, 530

μm in diameter. The smaller nonpathogenic race has trophozoites that measure 12–15 μm. Only the pathogenic organisms ingest red blood cells; the presence of erythrocytes within trophozoites is helpful in distinguishing pathogenic from nonpathogenic amebae. Cyst forms are 10–20 μm in diameter and contain four nuclei and rodlike chromatin bodies.

These organisms reproduce by binary fission [90, 290, 530]. Prior to producing the cyst form, the amebae become round and small. A cyst wall is formed; the nucleus divides twice, resulting in four small nuclei. These nuclei divide upon rupture of the cyst wall. Thus, each original organism separates into eight small amebae. Each of these in turn develops into a trophozoite.

Infection with *E. histolytica* produces mild clinical signs or none at all. There is a great variability in virulence among strains of organisms [90, 290, 529, 791]. Pathogenicity is affected by the host species infected, the nutritional status of the host, environmental factors, and the bacterial flora present in the gastrointestinal tract [90, 290, 529, 567, 791]. *Entamoeba histolytica* usually lives in the intestinal lumen where it is nonpathogenic [529]. Only when it invades the mucosa does it become pathogenic and may lead to amebic dysentery [529]. Clinically, affected animals show the following signs: lethargy, weakness, dehydration, anorexia, vomiting, and severe diarrhea that may or may not be hemorrhagic or catarrhal [90, 223, 290, 379, 385, 461, 530, 588, 665, 704, 829, 868, 869, 890]. The gross and microscopic lesions associated with amebiasis in nonhuman primates have been described [57, 60, 90, 290, 300, 461, 588, 665, 704, 742, 791, 829, 869]. At necropsy, a mild to severe necroulcerative colitis can be seen. *Entamoeba histolytica* trophozoites can be found in wet smears from material from the colon of clinically ill animals or from the colonic contents overlying the lesions seen at necropsy [461]. Histologically, the colonic mucosa is necrotic and ulcerated down to the level of the muscularis mucosae; typical flask-shaped ulcers may be seen. These ulcers can be as small as a few millimeters or may become large and confluent and involve extensive areas of the colon. Trophozoites may be seen in or adjacent to the ulcers. Often, the host response is minimal unless secondary bacterial invasion has occurred [290, 461, 704, 791]. Extensive hemorrhage with neutrophilic and mononuclear inflammatory cell infiltrates has been described in lesions from New World monkeys [461]. Gastric amebiasis and death resulting from infection with *E. histolytica* has been reported in the silver leaf monkey, douc langurs, proboscis monkey, and colobus monkey [299, 330, 544, 545, 601, 661]. Some trophozoites may enter lymphatic channels or even the venules of the mesenteric vasculature. Most are filtered by the regional lymph nodes; a few, however, may be carried to distant parts of the

body where they can produce the so-called amebic abscesses, particularly in the liver, lungs, or central nervous system [290, 300, 588, 665, 791]. Fatal amebiasis with abscess formation has been reported in a baboon [300], a chimpanzee [588], an orangutan [665], a group of spider monkeys [10], douc langurs [299], and several colobus monkeys [299, 544, 545].

The diagnosis of amebiasis depends upon the microscopic recognition of the causative organisms in the feces or in intimate association with typical lesions. These organisms are also common as nonpathogenic commensals in the digestive tract of nonhuman primates. Their presence in the feces of animals with clinical signs is not definitive evidence that protozoa are the cause of the gastrointestinal disease [290, 530, 791]. Wet-mount preparations may be used to examine the feces for trophozoites. This requires a fresh sample that must be placed immediately in a saline or buffer solution and examined while the preparation is still warm. The movement of the organisms can be seen. *Entamoeba histolytica* makes the most obvious kinds of progressive movement of all the intestinal amebae. Smears may be stained with Lugol's iodine solution, which identifies the nuclei and stains glycogen. Smears also may be fixed in Schaudinn's fluid and stained with Heidenhain's iron hematoxylin [90, 791]. Nuclear morphology can be used to distinguish pathogenic *E. histolytica* from nonpathogenic species [146].

Entamoeba histolytica causes amebic dysentery in humans; therefore, this organism in nonhuman primates poses a serious potential public health problem. The disease has been transmitted from laboratory primates to humans [48, 329]. These primates should be considered potential sources of infection, and proper care should be exercised in all phases of their management.

Sporozoans and Neosporans

The parasitic sporozoans and neosporans described from nonhuman primates are listed in Table 45.3. Infection with the coccidian parasites is considered essentially innocuous; there are no known lesions or diseases associated with their presence in the nonhuman primate gastrointestinal tract. Since *Cryptosporidium* sp. has been described recently in the gastrointestinal tract of monkeys, it will be discussed in greater detail. In addition, the malarial parasites, toxoplasmasids (sarcocystis and toxoplasma), piroplasmids (babesia and entopolypoides), and neosporins (encephalitozoon) will be discussed in detail.

Cryptosporidiosis

This disease, caused by the coccidian parasite *Cryptosporidium*, has been reported in the digestive tract of the rhesus monkey [138, 472], cynomolgus monkey, and bonnet monkey [906]. In one study, the organisms were seen in the epithelium of the common bile duct, the intrahepatic and pancreatic ducts, and the gallbladder of one monkey [472]. Histologic changes consisted of epithelial hyperplasia and mucosal inflammation. In another study, cryptosporidial organisms were found in the small-intestinal epithelium of seven rhesus monkeys and in both the small- and large-intestinal epithelium of one infant monkey. There were no lesions associated with the organisms in any of the monkeys except the infant. In this infant, the changes were characterized by atrophy of villi associated with many parasites in the brush border of the epithelium lining the villi and intestinal crypts [138].

TABLE 45.3. Parasitic sporozoans and neosporans described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Coccidian parasites						
Eimeria galago	Intestine	X				90, 290, 678
Eimeria lemuris	Intestine	X				90, 290, 678
Eimeria otolicni	Intestine	X				90, 290, 678
Eimeria pachylepyron	Intestine	X				139
Eimeria tupaiae	Intestine	X				604
Eimeria modesta	Intestine	X				604
Eimeria ferruginea	Intestine	X				604
Eimeria sp.	Intestine	X				168, 387
Isopora arctopitheci	Intestine		X			90, 290, 377, 378, 727
Isopora callimico	Intestine		X			217, 400
Isopora papionis	Intestine			X		576, 578
Isopora sp.	Intestine	X			X	90, 290, 485, 678, 722
Cryptosporidium sp.	Intestine			X		138, 472, 791, 906
Klossiella sp.	Kidney	X				97, 776
Haemogregarina cynomolgi	Blood			X		54, 290, 505, 525, 899
Piroplasmasid parasites						
Babesia pitheci	Erythrocytes			X		23, 290, 315, 385, 386, 449, 738, 837
Babesia sp.	Erythrocytes	X				72
Entopolypoides macaci	Erythrocytes			X		23, 269, 290, 332, 363, 485, 564, 565, 596
Theileria cellii	Erythrocytes			X		98, 99, 290
Malarial parasites						
Plasmodium cynomolgi	Erythrocytes			X		70, 143, 180, 236, 239, 243, 290, 316, 415, 563, 602, 702, 772, 773
Plasmodium knowlesi	Erythrocytes			X		80, 126, 243, 290, 318, 466, 502, 773, 797, 925
Plasmodium inui syn. (*P. shortti*)	Erythrocytes			X		70, 71, 186, 236, 237, 290, 353, 399, 415, 502, 795, 796, 816
Plasmodium coatneyi	Erythrocytes			X		236, 240, 241, 244, 290, 561, 658, 660, 662, 881
Plasmodium fieldi	Erythrocytes			X		236, 242, 290, 374, 881
Plasmodium gonderi	Erythrocytes			X		68, 290, 322, 733

TABLE 45.3. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Plasmodium fragile	Erythrocytes			X		180, 290, 703
Plasmodium siminovale	Erythrocytes			X		180, 181, 290
Plasmodium sp.	Erythrocytes			X		236, 290
Plasmodium brasilianum	Erythrocytes		X			165, 206, 211, 290, 337, 690, 835
Plasmodium simium	Erythrocytes		X			164, 165, 290, 291, 317
Plasmodium pitheci	Erythrocytes				X	290, 329, 353
Plasmodium malariae syn (*P. rodhaini*)	Erythrocytes				X	68, 69, 290, 321, 730, 731
Plasmodium reichenowi	Erythrocytes				X	65, 66, 71, 290, 321, 329, 714, 801
Plasmodium schweitzi	Erythrocytes				X	67, 70, 81, 290, 732
Plasmodium hylobati	Erythrocytes				X	71, 141, 290, 329, 729
Plasmodium eylesi	Erythrocytes				X	290, 882
Plasmodium jefferyi	Erythrocytes				X	290, 883, 884
Plasmodium youngi	Erythrocytes				X	244, 290
Plasmodium silvaticum	Erythrocytes				X	325, 542
Plasmodium girardi	Erythrocytes	X				84, 290, 319, 320
Plasmodium lemuris	Erythrocytes	X				290, 404
Plasmodium foleyi	Erythrocytes	X				319, 320
Hepatocystis kochi syn. (*H. simiae*)	Erythrocytes			X		2, 68, 290, 313, 314, 323, 452, 487, 488, 518, 524, 578, 586, 610, 652, 674, 823, 867
Hepatocystis semnopitheci	Erythrocytes			X		237, 238, 290, 465, 681, 714
Hepatocystis taiwanensis	Erythrocytes			X		70, 290
Hepatocystis foleyi	Erythrocytes	X				70, 84, 290
Hepatocystis sp.	Erythrocytes				X	174, 175, 573, 594
Sergentella anthropopitheci	Blood				X	173, 290

TABLE 45.3. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Toxoplasmasids						
Sarcocystis kortei	Striated muscle, oral cavity, heart, tongue, esophagus			X		23, 192, 290, 349, 361, 471, 555, 638
Sarcocystis nesbitti	Striated muscle, oral cavity, heart, tongue, esophagus			X		23, 290, 555
Sarcocystis sp.	Striated muscle, oral cavity, heart, tongue, esophagus	X	X	X		26, 168, 290, 369, 376, 573, 578, 623, 697, 834, 932
Toxoplasma gondii	Brain, lungs, liver, heart, kidney, lymph-nodes, blood, other tissues	X	X	X	X	12, 23, 36, 37, 81, 82, 119, 178, 183, 290, 381, 418, 470, 527, 578, 579, 581, 628, 633, 706, 707, 708, 709, 726, 783, 817, 831, 842, 858, 913, 934, 935
Neosporan parasites						
Encephalitozoon cuniculi	Brain, kidneys, heart, lungs, adrenals, other tissues		X			15, 79, 656
Microsporidian sp.	Intestine		X			781
Haplosporids						
Pneumocystis carinii	Lungs		X		X	117, 543, 679, 721, 820

A recent report documents severe intestinal disease in four juvenile macaques resulting from infection with *Cryptosporidium* sp. [906]. Two of the animals died naturally and the other two were humanely killed as a consequence of the illness, despite extensive therapy. Clinical signs in these young monkeys consisted of depression, dehydration, weight loss, and intractible diarrhea [906]. At necropsy, the animals were considered to be underweight for their age and dehydrated. The intestines were distended with gas and liquid. The mesenteric lymph nodes were enlarged in three of the four monkeys [906]. Histopathologically, the lesions in the small intestine consisted of blunting and fusion of villi, variation in height of the intestinal epithelium, necrosis of individual epithelial cells, and an increased mitotic index in the crypts. Variable numbers of organisms with both light microscopic and ultrastructural morphologic features consistent with *Cryptosporidium* sp. were seen adherent to enterocytes along the tips and side of villi, as well as within the crypts [906].

These cases indicate that cryptosporidial infection in young macaques can be a severe and potentially fatal disease. The cryptosporidia were associated with ultrastructural changes in the enterocytes that can result in malabsorption, as well as fluid loss in the infected host [906].

Malaria

This disease, caused by parasites in the family *Plasmodiidae*, genus *Plasmodium*, affects both humans and animals [32, 71, 290, 742, 791, 874]. Malaria is one of the most important hemoprotozoal parasitic diseases of primates in the tropical and semitropical regions of the world. Spontaneous infection is universal among nonhuman primates with the exception of a few species: rhesus monkey, tamarins and marmosets (*Callithrix* sp. and *Saguinus* sp.), and owl monkeys. The malaria parasites that infect the anthropoid apes are a different group of Plasmodia than those that afflict monkey species. They are also homologous to the malaria parasites of humans, and they are considered to be indistinguishable morphologically. Cross-infection to humans has been documented. In the natural host, these organisms do not produce severe disease—there are no outward signs of disease seen and usually no fever. There may be a slight anemia associated with a low-grade parasitemia in some animals, but they appear outwardly normal. In the aberrant host, infection with malarial parasites produces severe disease and debilitation that often leads to death.

Malaria parasites can be classified on the basis of the host infected—human, monkey, or anthropoid ape—or on the basis of the type of cyclic fever produced. Thus, quotidian malaria has a 24-hour cycle, tertian malaria cycles every 48 hours, and quartan malaria has a 72-hour cycle. The periodicity of the cyclic fever is determined by the length of time the organisms parasitize the host erythrocytes.

The life cycle of malaria parasites has been reviewed in detail [135, 140, 236, 290, 530, 556, 791, 874]. It is indirect with numerous mosquitoes in the genus *Anopheles* serving as biological vectors. Basically, the life cycle consists of two major phases: the sexual or sporogonic phase in the mosquito vector, and the asexual or shizogonic/gametogonic phase in the vertebrate host. The schizogonic phase is further divided into the exoerythrocytic or liver phase and the erythrocytic or blood phase.

The clinical signs reported in nonhuman primates infected with malaria consist of hepatosplenomegaly, fever, depression, listlessness, anorexia, and weight loss. Thrombocytopenia, leukopenia, progressive anemia, and reticulocytosis also have been reported. Diarrhea may be an accompanying symptom [290, 791, 816, 874]. Hematocrit, hemoglobin, mean corpuscular volume, and erythrocyte values are reported to be lower in infected nonhuman primates [186]. In general, young animals exhibit more severe symptoms than older animals [874]. Fever in infected nonhuman primates is less severe than in their human counterparts. The onset of fever coincides with the rupture of the parasitized erythrocytes and the release of toxic metabolic products into the bloodstream. Depending on the species of *Plasmodium* involved, this event can occur at 24-, 48-, or 72-hour intervals. Usually the natural host of a species of Plasmodia is asymptomatic.

Histopathology consists of functional and structural changes in the liver and pigment (hemazoin) deposition in the liver, spleen, and bone marrow. Hemor-

rhages in the brain, splenic rupture, and lower nephron (tubular) necrosis of the kidney have been reported [290, 542, 737, 791, 874].

The diagnosis of malaria depends on the demonstration and identification of the organisms in erythrocytes in thin or thick blood smears stained with Giemsa's or Wright-Giemsa stains [290, 791].

Malaria in most nonhuman primates is generally not fatal. However, it may cause debilitation, and overt disease can be precipitated by stress, concurrent disease, splenectomy, or immunosuppression. Infected primates may also serve as sources of infection for humans provided the required mosquito vectors are present. All people actively working with or caring for nonhuman primates should be alert to the possible existence and potential liabilities of malarial infection.

The Malaria of Old World Monkeys

Plasmodium knowlesi. This is the only known quotidian (24-hour) malarial parasite. It is distributed geographically throughout Southeast Asia, and the natural hosts include the cynomolgus monkey, leaf monkey (*Presbytes melalophus*), and the pig-tailed macaque.

Plasmodium knowlesi produces a virulent infection in the rhesus monkey that is almost always fatal and resembles acute *P. falciparum* infection of humans [70, 80, 126, 236, 243, 290, 318, 466, 502, 542, 773, 797, 874, 925].

Plasmodium cynomolgi. This is a tertian malarial parasite with a geographic distribution including the East Indies, Southeast Asia, and the Philippine Islands. The natural hosts include a wide variety of *Macaca* species including the cynomolgus monkey, Toque monkey, pig-tailed macaque, bonnet macaque, Formosan rock macaque (*Macaca cyclopis*), and several species of leaf monkeys (*Presbytes cristatus* and *P. entellus*). Infection in the rhesus monkey with this organism is not as severe as with *P. knowlesi* and usually consists of low-grade parasitemia of long duration. This organism is similar to *P. vivax* of humans and is also transmissible to humans [70, 143, 180, 236, 243, 290, 316, 542, 563, 602, 702, 773, 874].

Plasmodium gonderi. This tertian malarial parasite is distributed throughout west Africa and tropical central Africa and is the only simian *Plasmodium* found in Africa. Natural hosts include mangabeys (*Cercocebus* sp.) and drills (*Mandrillus* sp.). This parasite produces a high, chronic parasitemia in the rhesus monkey. Baboons (*Papio* sp.) and guenons (*Cercopithecus* sp.) are also susceptible. Humans have proven to be susceptible in experimental studies [68, 135, 290, 322, 337, 542, 733, 797, 874].

Plasmodium fieldi. *Plasmodium fieldi* is a tertian malarial parasite found on the Malay Peninsula. Natural hosts include the Asian species of *Macaca* including the cynomolgus monkey and the pig-tailed macaque. Infection with *P. fieldi* produces a severe disease in the rhesus monkey that is often fatal. Humans seem to be resistant to infection with this organism [242, 290, 374, 542, 874, 881].

Plasmodium fragile. This is also a tertian malarial parasite with a geographic range throughout southern India and on the island of Sri Lanka. Natural hosts include the Toque monkey (*Macaca sinica*) and the bonnet macaque. Infection in

the rhesus monkey produces severe disease that often kills the host [135, 180, 290, 542, 703, 874].

Plasmodium siminovale. This tertian malarial parasite is found on the island of Sri Lanka. The natural host is the Toque monkey. Even though the parasitemia is not particularly severe, a pronounced anemia has been reported as accompanying this infection. This parasite is considered to be similar to *P. ovale* of humans [181, 290, 542, 874].

Plasmodium coatneyi. This species of malarial parasite causes a mild tertian malaria in susceptible hosts. Its geographic distribution includes the Malay Peninsula and the Philippine Islands. The natural nonhuman primate host is the cynomolgus monkey. It is quite closely related morphologically to *P. knowlesi*, and the infection in the rhesus monkey is similar to *P. knowlesi*, producing a severe and often fatal disease accompanied by severe anemia [240, 241, 244, 263, 290, 542, 874, 881].

Plasmodium inui. *Plasmodium inui* is a mildly pathogenic species that produces a quartan malaria in susceptible hosts. It is very widespread throughout Southeast Asia and extends from India to the Philippine Islands. The natural hosts include the Asian species of *Macaca*, cynomolgus monkey, and pig-tailed macaque. It has also been reported from members of the genus *Presbytis* and the Celebes black ape (*Cynopithecus niger*). This infection is frequently encountered in Asian monkeys and can persist for at least several years even in animals removed from endemic areas. The parasite is considered to be homologous to *P. malariae* of humans, and humans are susceptible to infection with this organism [70, 71, 236, 237, 290, 353, 399, 502, 542, 773, 795, 796, 874].

Plasmodium shorti. This quartan malarial parasite is found in India and on the island of Sri Lanka. Natural nonhuman primate hosts include the Toque monkey and the bonnet macaque. This organism has been transmitted experimentally to humans [70, 236, 290, 795].

The Malarial Parasites of New World Monkeys

Plasmodium simium. This is a tertian malarial parasite with a geographic distribution in the region of southern Brazil. The natural nonhuman primate hosts are howler monkey(*Alouatta* sp.) and woolly spider monkeys (*Brachyteles arachnoides*). The organism is similar to *P. vivax* of humans, and infection with this parasite has been reported in humans [135, 164, 290, 291, 317, 542, 874].

Plasmodium brazilianum. *Plasmodium brazilianum* is sometimes a markedly pathogenic species and is the commonest malarial parasite of New World monkeys. The natural nonhuman primate hosts include howler monkeys, spider monkeys (*Ateles* sp.), woolly spider monkeys, uakaris (*Cacajao* sp.), titis (*Callicecus* sp.), bearded sakis (*Chiroptes* sp.), capuchin monkeys (*Cebus* sp.), woolly monkeys (*Langothrix* sp.), and squirrel monkeys (*Saimiri* sp.) [2, 135, 211, 290, 337, 542, 690, 835, 874]. The geographic distribution ranges from Mexico throughout Central America and into South America down to Peru. *Plasmodium brazilianum* causes quartan malaria that can produce severe symptoms, and even adult monkeys have been known to die from this infection [542, 835]. Usually it is seen as an infection at equilibrium having a low parasitemia that may persist for several years [874]. This organism is considered to be the same as *P. malariae* of

humans, and humans are susceptible to experimental infection. This species may actually be *P. malariae* introduced into the New World by early explorers and modified through numerous passages in wild monkeys [206, 290].

The Malaria of Anthropoid Apes

Plasmodium pitheci. This is a tertian malarial parasite that was originally described in 1907 from Borneo in an orangutan (*Pongo* sp.) [353, 542]. Orangutans are the only nonhuman primates in which this parasite has been reported, and there are few details about the organism and the disease it produces. It has proved to be noninfectious for gibbons and monkeys in limited experimental studies.

Plasmodium rodhaini. *Plasmodium rodhaini* is found in west to central tropical Africa where it causes quartan malaria in its natural nonhuman primate hosts, chimpanzees (*Pan* sp.) and gorillas (*Gorilla* sp.). There is no morphological difference between this organism and *Plasmodium malariae* of humans, and these two parasites are considered to be synonymous. This is the only malarial parasite that occurs as a natural infection in humans and nonhuman primates to any great extent. Infection is easily transmitted from humans to chimpanzees and vice versa [68, 69, 290, 321, 542, 730, 731, 874].

Plasmodium reichenowi. This mildly pathogenic species occurs in west, central, and east tropical Africa. The natural nonhuman primate hosts include chimpanzees and gorillas. *Plasmodium reichenowi* causes a mild quartan malaria in these species. This organism is very similar to *Plasmodium falciparum* of humans. There is only a slight morphological difference between the two organisms, but attempts to transmit *P. reichenowi* to humans have been unsuccessful [65, 66, 68, 70, 71, 290, 321, 542, 714, 801, 874].

Plasmodium schwetzi. *Plasmodium schwetzi* is a mildly pathogenic plasmodium species found in west Africa. The natural nonhuman primate hosts are chimpanzees and gorillas, and this parasite causes a mild tertian malaria in these species. The disease is often subclinical and not obvious unless the animal if splenectomized. *Plasmodium schwetzi* is very similar to *P. vivax* of humans. This organism can infect humans and has been transmitted from chimpanzees to humans via mosquitoes. The disease in humans consists of a mild febrile period followed by a spontaneous cure [67, 70, 71, 81, 290, 542, 732, 874].

Plasmodium hylobati, Plasmodium eylesi, Plasmodium jefferyi, Plasmodium youngi. These four closely related parasites are found in the East Indies. They produce a quartan malaria in gibbons (*Hylobates* sp.), which are the natural nonhuman primate hosts for these parasites. *Plasmodium hylobati*, *P. youngi*, and *P. eylesi* are reported to be pathogenic. A febrile response associated with the parasitemia has been seen in gibbons infected with *P. hylobati* and *P. youngi*. Details of the clinical disease and pathology have not been reported [141, 244, 290, 542, 729, 874, 882–884].

Hepatocystosis

This disease is caused by parasites classified in the genus *Hepatocystis* in the family *Haemoproteidae*. The species reported to occur in nonhuman primates are listed in Table 45.3.

Hepatocystis kochi was formerly classified in the family *Plasmodiidae* as *Plasmodium kochi* and currently has several other synonyms including *H. joyeuxi*, *H. cercopitheci*, *H. bovilliezi*, and *H. simiae* [290]. These sporozoan parasites are distributed in India to the East Indies subcontinent and throughout the African continent south of the Sahara Desert. Nonhuman primates reported to be affected with *Hepatocystis* sp. include Old World monkeys (African green monkeys, guenons, mangabeys, baboons, patas monkeys, colobus monkeys, Formosan rock macaques, other *Macaca* species, and leaf monkeys) and great apes (gibbons) [2, 174, 175, 290, 313, 323, 487, 542, 557, 794, 867, 874, 884].

The incidence of *Hepatocystis* can exceed that of malaria [542] and has been reported from 42% to 56% in nonhuman primates obtained from west central Africa [68, 290, 524] and from 40% to 75% in species from east central Africa [290, 586, 674, 823, 867].

The life cycle of *Hepatocystis* is indirect with midges (*Culicoides* sp.) serving as the biological vector [290, 323, 542, 791]. *Hepatocystis kochi* is the only species in which the life cycle is completely known [313, 314, 530, 586]. The life cycle resembles that of *Plasmodium* sp. with the major exception that asexual schizogony does not take place in the host's erythrocytes. Schizogony in the liver produces grossly visible cysts called merocysts [290, 542, 874].

Hepatocystis infection in the nonhuman primate produces no cyclic fever or waves of parasites in the blood as occurs with malaria. Parasitemias are usually not too great, and evidently there is no adverse effect to the monkey's health.

Grossly, infected nonhuman primates have numerous, randomly scattered, grayish-white, translucent foci on the surface of the liver that correspond to the mature merocysts [290, 542, 867]. Histopathologically, there is no tissue reaction in the liver until the merocysts are formed. After the cyst develops, there is usually a neutrophilic exudate surrounding it. Following rupture of the cyst and release of the merozoites, a chronic granulomatous inflammatory reaction ensues with the infiltration of lymphocytes and macrophages. Healing results in fibrosis in and around the area where the cyst was located. These appear as white foci grossly [290, 542, 867].

Diagnosis is based on demonstration and identification of the parasite in thick blood smears or finding the typical hepatic lesions at necropsy and/or on histologic sections [290, 674, 867].

There are no public health considerations with this parasite since *Hepatocystis* sp. are not known to infect humans [290].

Toxoplasmosis

The cause of this disease, *Toxoplasma gondii*, is a cosmopolitan protozoan parasite. Spontaneous infection with *T. gondii* has been reported in New World monkeys (squirrel monkeys, spider monkeys, sakis, owl monkeys, uakaris, marmosets, tamarins, woolly monkeys, titi monkeys, howler monkeys, woolly monkeys, and cebus monkeys), Old World monkeys (rhesus monkeys, stump-tail macaques, cynomolgus monkeys, and baboons), great apes (chimpanzees), and prosimians (Malayan tree shrew, ring-tailed lemur, ruffed lemur, and slow loris) [12, 23, 36, 37, 81, 119, 178, 183, 290, 381, 418, 470, 527, 579, 581, 628, 633, 706–709, 726, 783, 817, 831, 842, 858, 913, 934, 935]. New World monkeys are reported to be more susceptible to this disease [23, 381, 429, 581, 742, 791,

913]; marmosets are very susceptible and may die within 5–6 days after contacting the disease [37]. There is some question as to whether or not the infection reported in the baboon (*Papio cynocephalus*) and the chimpanzee was acquired naturally since both animals had been inoculated intracerebrally with material from guinea pigs and rabbits shortly before death [23, 742]. Also, there is some doubt about the validity of the diagnosis regarding two fatal cases of toxoplasmosis in lemurs (*Lemur catta*) from Japan [23]. The diagnosis was based on the microscopic demonstration of the parasites, and the published photomicrographs are reported not to be entirely convincing [23].

The morphology of *T. gondii* has been reviewed previously [290, 303, 426, 530]. Toxoplasma tachyzoites (formerly called trophozoites) are crescent- or banana-shaped structures that measure 4–8 μ by 2–4 μ. One end is pointed, and the other is rounded and contains a centrally located nucleus. Tachyzoites can be found in various cells throughout the host and also in blood and peritoneal fluid. Initially, they occupy vacuoles in the host cells (current preferred terminology, group stage or colony) [303, 791]. As they multiply, a cyst forms around them. The encysted forms are known as bradyzoites (formerly called merozoites). The oocysts seen in the intestinal epithelial cells and feces of the cat measure 10 $\times$ 12 μ and are the smallest of the three common cat coccidia.

For an in-depth discussion of the life cycle of this interesting parasite, the reader is referred to the following references: 290, 302, 303, 426. Briefly, the life cycle consists of an enteroepithelial phase, which occurs only in the definitive host, and a extraintestinal or tissue phase, which occurs in all susceptible species (intermediate hosts). Asexual reproduction (endodyogony, endopolygony, and schizogony) and sexual reproduction (gametogony), which leads to the production of oocysts, occur in the intestinal epithelium of various domestic and feral members of the family *Felidae*. Unsporulated occysts are shed by these animals, the only known definitive hosts, and sporogeny occurs in the feces. In the extraintestinal cycle multiplication of tachyzoites occurs by endodyogony in all other tissues of the intermediate hosts, which include a wide variety of domestic and wild animals (including cats), birds, and some nonmammalian species [290, 302, 303, 791].

Infection with *Toxoplasma* can occur via transplacental transmission, consumption of tissue cysts, and consumption of oocysts. The organisms also can be spread mechanically and by insect vectors, such as cockroaches [290, 302, 303, 791, 879].

Nonspecific clinical signs reported in nonhuman primates infected with *T. gondii* include emesis, depression, diarrhea, anorexia, fever, cough, weakness, ocular and nasal discharges, pale mucous membranes, leukopenia, dyspnea, premature birth, abortion, and death [290, 429, 791]. Neurologic signs include circling, incoordination, paresis, and terminal convulsions [290, 429]. At necropsy, the most frequently observed abnormalities reported are cardiomegaly, myocardial necrosis, hemorrhagic lymphadenopathy, pulmonary edema, hepatic congestion, hepatocellular necrosis, splenomegaly, and splenic hyperplasia [36].

Histopathologically, focal hepatic necrosis, focal to diffuse necrotic lymphadenitis and splenitis, segmental intestinal lesions, interstitial and fibrinous pneumonia, and focal myocarditis have been reported. Necrotic foci and extracellular and/or intercellular tachyzoites are frequently found in conjunction with the

inflammatory lesions. Lesions seen in the central nervous system include gliosis, focal hemorrhage, microscopic infarcts, and cellular degeneration [290, 429, 791]. *Toxoplasma gondii* cysts and free organisms have been noted in capillary endothelial cells and in the brain tissue, frequently with associated perivascular cuffing and cellular necrosis [429, 470, 581].

Diagnosis of toxoplasmosis depends on the demonstration and identification of the causative organism in smear preparations or in histopathologic sections, or by animal inoculation [23, 290, 429, 530]. Laboratory tests include the indirect fluorescent antibody test, the Sabin-Feldman dye test, and the hemagglutination test. Isolation of *T. gondii* itself is most reliable but is time consuming and expensive. Recognition of oocysts in fecal samples of cats is important for the prevention and control of both animal and human toxoplasmosis [290, 429, 791]. The following references should be consulted for information about preparing fecal samples and the morphological differences between the common feline coccidia oocysts [189, 191].

Because toxoplasmosis can occur in humans, reasonable care should be taken to prevent infection in those personnel responsible for the care and use of nonhuman primates. Feces from *Felidae* should be removed frequently (within 24 hours) and preferably incinerated or disposed of in some other way that will prevent contact of vectors and fomites with sporulated oocysts [290].

Sarcocystosis

This disease is caused by coccidian parasites commonly classified in the genus *Sarcocystis*. The cystic phase of this parasite has been described in skeletal muscle fibers and occasionally in cardiac or smooth muscle fibers in a wide variety of animals throughout the world [23, 225, 290, 461, 530, 532]. These cysts are common in the skeletal muscle of the tongue or the esophageal muscle of many nonhuman primates. *Sarcocystis kortei* and *S. nesbitti* have been described in the rhesus monkey, and other unnamed species have been reported in both Old and New World monkeys [23, 192, 270, 290, 361, 376, 452, 471, 534, 550, 555, 623, 638, 697, 791, 823].

It is now known that *Sarcocystis* has an obligatory two-host life cycle. The reader is referred to the definitive works published recently outlining the intricacies of the life cycle of this unique and interesting parasite [190, 302, 303, 743].

Lesions associated with spontaneous infections in nonhuman primates are rare [225, 290, 461, 791]. Inflammation characterized by infiltrates of lymphocytes, plasma cells, and eosinophils is associated with degeneration of the cysts within the muscle fibers. With time, there is a proliferation of fibrous connective tissue and resulting scar formation [791, 840]

Babesiosis

Two organisms are associated with this disease in nonhuman primates. *Babesia pitheci* has been reported from Old World monkeys (mangabeys, guenons, macaques, and baboons) [23, 290, 315, 385, 386, 449, 738, 837] and New World monkeys (marmosets) [385] . Its distribution and incidence in nature is unknown [290, 542]. The complete life cycle is unknown, but ticks are thought to be the biological vectors [23, 290, 542]. This babesial parasite is considered to be only slightly pathogenic in normal intact monkeys but can result in severe anemia and

death after splenectomy. Marked poikilocytosis and anisocytosis are associated with the anemia [23, 290, 315, 449, 542, 837]. *Babesia pitheci* organisms are pyriform in shape and measure 2–6 μm long. Round, elliptical, oval, lanceolate, and ameboid stages have been also observed in peripheral blood smears [23, 290, 542].

The second babesia-like organism, *Entopolypoides macaci*, is a mildly pathogenic hemosporozoal parasite that has been described in Old World monkeys (cynomolgus monkeys, rhesus monkeys, baboons, and guenons) and great apes (chimpanzees) [32, 269, 290, 332, 363, 564, 565, 596, 791]. This organism does not have true pyriform stages, but early ring-shaped stages and ameboid stages with polypoid projections of cytoplasm similar to the true *Babesia* species have been seen. *Entopolypoides macaci* is smaller than *Babesia* and *Plasmodium* species parasites and is morphologically distinct [332, 542]. Parasitized erythrocytes are not enlarged, and pigment is not formed [542].

Fever, monocytosis, and anemia have been reported in parasitized nonhuman primates; however, infection with *E. macaci* appears to have little effect on the host [290, 542, 565]. Chronic, latent infections are known to occur, and splenectomy or immunosuppression will result in recurrence of the parasitemia and a marked increase in the intensity of hemolytic anemia and icterus. Under these conditions, the disease may be fatal [791]. There are indications that this organism is common in nonhuman primates [542].

Diagnosis of these two organisms depends on the demonstration and identification of the parasites within the host's erythrocytes [290, 791].

There is no public health significance associated with either *B. pitheci* or *E. macaci* infections because neither parasite has been reported in humans [290].

Encephalitozoonosis

The cause of this disease, *Encephalitozoon cuniculi*, is an obligate intracellular protozoan parasite that has been reported in a wide variety of vertebrate and invertebrate species [791]. Only a few cases have been reported in nonhuman primates, all in New World monkeys. Two of these cases involved squirrel monkeys, and one involved an unidentified microsporidian parasite in a dusky titi monkey (*Callicebus moloch*) [15, 79, 781].

Encephalitozoon cuniculi is a small, oval parasite that measures approximately 2.5 × 1.5μm. Division occurs by binary fission and produces two spores per sporont. The organisms can be distinguished from Toxoplasma and other parasites by their location, size, and positive staining characteristics with the gram stain and various silver impregnation methods. A coiled polar filament with four to five coils is a distinctive ultrastructural feature of *E. cuniculi* [791]. The life cycle of this parasite is not known completely at this time [791].

The signs described in nonhuman primates infected with *E. cuniculi* consist of nervous symptoms displayed by a 2-month-old squirrel monkey for approximately 1 month prior to death [79]. Lesions in this animal were focal granulomatous meningoencephalitis, hepatitis, and nephritis. Characteristic *E. cuniculi* organisms were seen by both light and electron microscopy. Also, a granulomatous encephalitis caused by *E. cuniculi* infection has been reported in a newborn squirrel monkey [15]. In the case involving the dusky titi, gram-positive, acid-fast microsporidial organisms having a polar filament with as many as seven coils

were found in the jejunal epithelium. It was felt that this organism was one that normally infects arthropods rather than *E. cuniculi* and that the monkey became infected through ingestion of the arthropods that it was able to capture in its outdoor environment. No host response to the presence of the organisms in the intestinal epithelium was reported [781]. *Encephalitozoon cuniculi* infection also has been reported as the cause of death in two infant squirrel monkeys [656].

Diagnosis of *E. cuniculi* can be made by finding the parasites associated with the typical lesions during histopathological examination of the tissues or by demonstration of the organisms in the urine. Currently, an immunofluorescence test that detects antibodies against the organisms and an intradermal test have both proved reliable [429, 791].

The public health importance of this organism is unknown at this time. There is one report of a natural *E. cuniculi* infection in a human; however, its validity has been questioned by some authors [791]. Nevertheless, personnel working or caring for nonhuman primates should follow accepted personal hygiene practices, and because urinary excretion has been proposed as a possible mode of transmission, excrement from nonhuman primates should be handled with caution. Also, care should be taken to ensure that captive nonhuman primates are protected from exposure to species known to be carriers of this organism [290].

Ciliates

The parasitic ciliates described from nonhuman primates are listed in Table 45.4. *Balantidium coli* is the only species that has been associated with lesions of the intestinal tract.

Balantidiasis

The cause of this disease, *Balantidium coli*, has a world-wide distribution and has been reported in a number of nonhuman primate species including New World monkeys (howler monkeys, spider monkeys, and cebus monkeys), Old World

Table 45.4. Parasitic ciliates described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Balantidium coli	Cecum, colon		X	X	X	38, 90, 290, 331, 349, 368, 459, 461, 485, 486, 487, 491, 494, 529, 599, 611, 616, 694, 712, 791, 839, 863, 905, 929
Balantidium sp.	Cecum, colon			X		573, 578
Troglodytella abrassarti	Cecum, colon				X	82, 157, 284, 290, 463, 491, 529, 599, 624, 863
Troglodytella gorillae	Cecum, colon				X	90, 290

monkeys (rhesus monkeys, cynomolgus monkeys, and baboons), and great apes (orangutans, chimpanzees, and gorillas) [38, 90, 92, 285, 290, 294, 331, 349, 368–371, 437, 443, 459, 461, 487, 570, 599, 680, 687, 694, 723, 791, 839, 863, 905, 929]. The organism is usually nonpathogenic and is a common inhabitant of the cecum of nonhuman primates [90, 92, 290, 361, 461, 494, 611, 694, 712, 863]. Some have been reported to be symptomless carriers [137, 877].

Balantidium coli trophozoites are large, ovoid structures with a heavily ciliated outer surface [18, 89, 290, 351, 473, 530, 790, 791, 899]. This form measures 30–150 × 25–120 μm. Internal structures consist of a macronucleus and micronucleus, two contractile vacuoles, and numerous food vacuoles. Cyst forms are spherical to ovoid and measure 40–60 μm in diameter. Reproduction occurs by conjugation or by transverse binary fission. Infection occurs through ingestion of trophozoites or cysts [90, 290, 530, 791].

Infection with *B. coli* can cause severe ulcerative enterocolitis that can be fatal in great apes [53, 130, 285, 459, 742]. Signs of clinically ill animals are weight loss, anorexia, muscle weakness, lethargy, watery diarrhea, tenesmus, and rectal prolapse [90, 369, 598, 839]. At necropsy, lesions may resemble those seen in amebiasis and may consist primarily of an ulcerative colitis [90, 461, 791]. Histologically, the ulcers may be large and may extend down to the muscularis mucosae [290, 791]. There may be an accompanying lymphocytic infiltrate and, in time, coagulation necrosis and hemorrhage [290, 791]. Typical large *B. coli* organisms can be seen in masses associated with lesions in the tissues or in capillaries, lymphatics, or regional lymph nodes [90, 290, 791, 877].

Diagnosis depends on identification of the characteristic *B. coli* organisms associated with the typical colonic lesions [290, 461, 791]. Their presence as secondary invaders to a primary disease caused by other microorganisms should always be considered and must be ruled out [290].

Balantidium coli may cause diarrhea in humans; therefore, care should be taken in handling captive nonhuman primates to avoid infection [290].

Metazoan Parasites

Nematodes

The parasitic nematode genera described from nonhuman primates are listed in Table 45.5. Because nematodiasis is such a common occurrence in nonhuman primates, the majority of the genera listed will be discussed in detail.

Rhabditoids

Strongyloidiasis

This disease results from infection by the parasitic members of the genus *Strongyloides*. These small nematodes are prevalent in most tropical and subtropical areas, but their occurrence in the temperate zones is sporadic. Several species have been reported to affect nonhuman primates: *Strongyloides cebus* has been found in New World monkeys (cebus monkeys, woolly monkeys, spider monkeys, squirrel monkeys, and marmosets) [207, 290, 437, 461, 492, 541, 634, 650, 752, 791], *Strongyloides fulleborni* in Old World monkeys and great apes (rhesus monkeys, cynomolgus monkeys, guenons, baboons, and chimpanzees) [290, 345,

TABLE 45.5. Parasitic nematodes described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Strongyloidids						
Strongyloides fulleborni	Intestines			X	X	3, 284, 290, 345, 349, 360, 650, 741, 742, 764, 791, 863, 878, 921, 922, 929
Strongyloides cebus	Intestines		X			148, 290, 461, 541, 650, 791
Strongyloides stercoralis	Intestines				X	176, 290, 531, 541, 650, 791, 810, 922
Strongyloides papillosus	Intestines			X	X	290, 541
Strongyloides sp.			X	X	X	448, 570, 578, 654, 658, 905, 922
Pelodera strongyloides	Skin lesions			X		530, 742
Ancylostomatids						
Ancylostoma duodenale	Sm. intestine				X	38, 116, 290, 626, 650, 791, 929
Necator americanus	Sm. intestine		X	X	X	38, 75, 185, 280, 290, 486, 487, 491, 609, 650, 723, 742, 791, 841, 929
Globocephalus simiae	Sm. intestine			X		290, 916
Characostomum asimilium	Sm. intestine	X		X		290, 919
Necator sp.	Sm. intestine	X				387
Strongylids						
Oesophagostomum apiostomum	Colon, mesentery			X		3, 276, 290, 349, 350, 530, 650, 658, 742, 791, 921
Oesophagostomum bifurum	Colon			X	X	47, 276, 290, 339, 345, 485, 486, 487, 488, 493, 494, 530, 578, 609, 650, 742, 791, 863
Oesophagostomum aculeatum	Colon			X		276, 290, 362, 530, 650, 712, 742, 791, 836
Oesophagostomum stephanostomum	Colon			X	X	276, 280, 290, 491, 530, 609, 650, 712, 742, 791, 863
Oesophagostomum blanchardi	Colon				X	290, 650, 791, 919

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Oesophagostomum sp.	Colon			X		1, 335, 488, 491, 573, 618, 654, 658, 660, 662, 922
Ternidens deminutus	Cecum, colon			X	X	9, 290, 335, 339, 349, 530, 578, 609, 626, 650, 712, 742, 764, 791, 836
Ternidens sp.	Cecum, colon			X		494
Trichostrongylids						
Molineus torulosus	Sm. intestine		X			201, 207, 290, 461, 509, 791, 926
Molineus vexillarius	Sm. intestine, stomach		X			148, 168, 201, 207, 290, 461, 689, 791
Molineus elegans	Sm. intestine		X			201, 207, 290, 461, 791
Molineus vogelianus	Sm. intestine	X				201, 290
Pithecostrongylus alatus	Intestine			X	X	290, 798, 851, 919
Trichostrongylus colubriformis	Sm. intestine			X	X	276, 290, 493, 509, 530, 578, 712, 810, 862
Graphidioides berlai	Intestine		X			290, 461, 919
Nematodirus weinbergi	Sm. intestine				X	290, 699, 919
Longistriata dubia	Sm. intestine		X			148, 207, 290, 461, 689
Nochtia nochi	Stomach			X		1, 58, 290, 339, 362, 508, 650, 742, 784, 791, 803, 851, 922
Tupaiostrongylus liei	Sm. intestine	X				203, 216
Tupaiostrongylus major	Sm. intestine	X				215
Tupaiostrongylus minor	Sm. intestine	X				215
Anoplostrongylus liei	Intestine	X				216
Hepatojarakus malayae	Intestine	X				216
Nycteridostrongylus petersi	Intestine, lungs	X				216
Trichostrongylus sp.	Sm. intestine			X		485, 486, 487, 491, 494, 616, 658

(Continued)

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Metastrongylids						
Filaroides barretoi	Lungs		X			207, 290, 339, 584, 742
Filaroides gordius	Lungs		X			207, 290, 339, 742
Filaroides cebus	Lungs		X			63
Filaroides sp.	Lungs		X			148, 290
Filariopsis arator	Lungs		X			290, 919
Filariopsis asper	Lungs		X			207, 461, 865
Angiostrongylus costaricensis	Mesenteric arteries		X			802, 846
Angiostrongylus malaysiensis		X				535
Atractidids						
Probstmayria nainitalensis	Rectum			X		17
Probstmayria gombensis	Intestine				X	283, 284
Probstmayria gorillae	Intestine				X	284, 476
Probstmayria simiae	Intestine				X	284, 558
Ascaridids						
Ascaris lumbricoides	Sm. intestine			X	X	209, 290, 509, 530, 650, 676, 712, 723, 742, 791, 922
Ascaris sp.	Intestine			X		491
Subulurids						
Subulura distans	Stomach, sm. intestine	X		X		290, 346, 609, 626, 742, 922
Subulura malayensis	Colon			X		290, 922
Subulura jacchi	Sm. intestine		X			148, 168, 290, 554, 689, 742, 841, 922
Subulura perarmata	Cecum, colon	X				61
Subulura indica	Lg. intestine, cecum	X				477
Oxyurids						
Enterobius vermicularis	Lg. intestine				X	96, 131, 290, 362, 409, 618, 650, 718, 723, 742
Enterobius bipapillata	Lg. intestine			X	X	290, 409, 650, 723, 919
Enterobius brevicauda	Lg. intestine			X		290, 409, 485, 650, 759, 919
Enterobius anthropopitheci	Lg. intestine				X	209, 328, 409, 491, 650, 712, 723, 742
Enterobius buckleyi	Lg. intestine				X	290, 409, 650, 759, 919

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Enterobius lerouxi	Lg. intestine				X	290, 409, 650, 759, 919
Enterobius pitheci	Lg. intestine			X		409
Enterobius parallela	Lg. intestine			X		409
Enterobius zakiri	Lg. intestine			X		409
Enterobius microon	Lg. intestine		X			290, 650, 919
Enterobius sp.	Lg. intestine	X		X	X	61, 387, 390, 448, 485, 658, 660, 662, 712, 922
Buckleyenterobius dentata	Lg. intestine			X		409
Trypanoxyuris trypanuris	Lg. intestine		X			409
Trypanoxyuris (Buckleyentero-bius) atelis	Lg. intestine		X			85, 96, 290, 409, 759, 919
Trypanoxyuris (Buckleyen-terobius) duplicidens	Lg. intestine		X			85, 409, 290, 919
Trypanoxyuris (Buckleyen-terobius) lagothricis	Lg. intestine		X			85, 409, 290, 919
Trypanoxyuris (Enterobius) interlabiata	Lg. intestine		X			290, 409, 650, 759, 919
Trypanoxyuris minuta	Lg. intestine		X			290, 409, 410, 461, 650, 683, 841, 922
Trypanoxyuris sceleratus	Lg. intestine		X			207, 290, 409, 412, 461, 650
Trypanoxyuris brachytelesi	Lg. intestine		X			409
Trypanoxyuris callithricis	Lg. intestine		X			409
Trypanoxyuris tamarini	Lg. intestine		X			148, 168, 290, 410, 412, 450, 461, 689
Trypanoxyuris oedipi	Lg. intestine		X			410
Trypanoxyuris goeldii	Lg. intestine		X			410
Enterobius lemuris	Lg. intestine	X				409
Lemuricola nycticebi	Lg. intestine	X				411
Lemuricola malaysensis	Lg. intestine	X				28, 208, 411
Lemuricola contagiosus	Lg. intestine	X				411

(Continued)

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Labatorobius scleratus	Lg. intestine		X			290, 919
Oxyuronema atelophorum	Lg. intestine		X			290, 339, 474, 919
Primasubulura jacchi	Lg. intestine		X			168
Primasubulura otolicini	Lg. intestine	X				232
Trypanoxyuris sp.	Lg. intestine		X			609, 689
Spirurids						
Chitwoodspirura serrata	Stomach, sm. intestine				X	290, 919
Spirura guianensis	Esophagus		X			148, 290, 623
Trichospirura leptostoma	Pancreas		X			26, 148, 149, 290, 461, 492, 649, 650, 806, 807
Protospirura (Mastophorus) muricola	Stomach		X	X		101, 290, 292, 488
Pterygodermatites nycticebi	Sm. intestine	X	X			536, 593, 856
Rictularia alphi	Sm. intestine		X			290, 597, 919, 922
Rictularia sp.	Sm. intestine		X			4, 591, 592, 930
Thelaziids						
Streptopharagus armatus	Stomach			X	X	290, 349, 578, 609, 836
Streptopharagus pigmentatus	Stomach			X	X	290, 339, 362, 485, 488, 494, 609, 712, 764
Streptopharagus baylisi	Stomach			X		485, 486, 487
Streptopharagus guptai	Rectum			X		809
Streptopharagus sp.	Stomach			X		485, 616
Gongylonema macrogubernaculum	Esophagus, stomach		X	X		290, 339, 461, 533, 547, 548, 609, 650, 791
Gongylonema pulchrum	Tongue, oral cavity, esophagus, stomach		X	X		290, 461, 531, 547, 548, 650, 791
Physocephalus sp.	Stomach			X		654, 421, 911
Metathelazia ascaroides	Lungs			X		187, 188, 290, 919
Thelazia callipaeda	Eyes			X		271, 290, 531, 810

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Physalopterids						
Physaloptera tumefaciens	Stomach			X		290, 362, 494, 531, 650, 764, 836, 907, 921, 922
Physaloptera dilatata	Stomach		X			168, 290, 650, 919
Physaloptera masoodi	Stomach	X				477
Physaloptera sp.	Stomach	X				612, 654, 841, 911
Abbreviata caucasica	Stomach			X	X	73, 284, 290, 419, 485, 487, 531, 578, 609, 626, 742, 919
Abbreviata poicilometra	Stomach			X		290, 756, 799, 919
Abbreviata sp.	Stomach			X		616
Onchocercids						
Dirofilaria magnilarvatum	Subcutis, peritoneal membranes			X		13, 102, 290, 500, 580, 605, 693, 762, 838, 901
Dirofilaria corynodes syn. (*D. aethiops, D. schoutedeni*)	Subcutis			X		13, 102, 280, 290, 364, 507, 537, 580, 875, 885, 886, 887, 888
Dirofilaria immitis syn. (*D. pongoi*)	Subcutis, muscle, right ventricle				X	13, 102, 290, 580, 742, 873, 885
Dirofilaria repens syn. (*D. macacae*)	Subcutis			X		605, 754, 885
Dirofilaria sp.	Blood			X		561, 777
Edesonfilaria malayensis	Peritoneal cavity			X		290, 308, 531, 605, 635, 712, 920, 924
Loa loa	Subcutis, mesenteries, eyes		X	X		194, 196, 199, 280, 290, 514, 531, 609, 626, 646, 734, 755, 852, 872, 886
Macacanema formosana	Peritracheal connective tissue			X		43, 290, 531, 768
Meningonema peruzzii	Subdural space–medulla oblongata			X		648
Brugia pahangi	Lymphatic system	X		X		13, 102, 221, 290, 531, 660, 662, 766, 767, 810

(Continued)

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Brugia malayi	Lymphatic system	X		X		13, 87, 102, 280, 290, 507, 531, 561, 605, 660, 662, 810, 900
Brugia tupaiae	Lymphatic system	X				642
Wuchereria kalimantani	Inguinal lymphnodes, testicles			X		561, 659, 660, 662
Filaria (?) *nycticebus*	Intestine	X				590, 886
Dipetalonema gracile	Peritoneal cavity		X			26, 52, 94, 148, 167, 168, 184, 210, 290, 310, 475, 508, 531, 537, 566, 568, 569, 580, 651, 742, 865, 885, 889
Dipetalonema caudispina	Peritoneal cavity		X			210, 290, 311, 580
Dipetalonema tenue	Subcutis, body cavity		X			102, 103, 290, 919
Dipetalonema barbascalensis	Peritoneal cavity		X			231
Dipetalonema petteri	Pleura and peritoneum	X				105
Protofilaria furcata	Thoracic cavity	X				114, 886
Parlitomosa zakii	Peritoneal cavity		X			210, 231, 461, 619, 886
Tetrapetalonema (Dipetalonema) *obtusus*	Periesophagal connective tissue		X			230, 290, 531, 580, 923
Tetrapetalonema (*Depetalonemia*) *marmosetae*	Subcutis, body cavity		X			210, 273, 290, 580, 742, 845, 885, 923
Tetrapetalonema (*Dipetalonema*) *tamarinae*	Peritoneal cavity		X			167, 210, 230, 290
Tetrapetalonema (*Dipetalonema*) *atelensis*	Connective tissue		X			290, 580, 742, 885, 919, 923
Tetrapetalonema (*Dipetalonema*) *parvum*	Connective tissue		X			207, 290, 580, 742, 885, 922, 923
Tetrapetalonema (*Depetalonema*) *vanhoofi*	Connective tissue		X			210, 461
Tetrapetalonema nicollei	Peritoneal cavity		X			210, 885
Tetrapetalonema dunni	Subcutis	X				603
Dipetalonema perstans	Subcutis, body cavity				X	102, 123, 197, 276, 290, 340, 348, 507

TABLE 45.5. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Dipetalonema vanhoofi	Peritoneal cavity			X	X	42, 100, 101, 290, 531, 609, 646, 669, 670, 671, 739, 742, 885, 923
Dipetalonema rodhaini	Subcutis, body cavity				X	290, 646, 671, 742, 885, 919, 923
Dipetalonema streptocerca	Subcutis, peritoneal cavity				X	122, 195, 197, 276, 290, 531, 646, 671, 742, 885, 923
Dipetalonema digitatum	Peritoneal cavity			X	X	102, 103, 114, 290, 605, 742, 758, 885, 889, 919, 923
Dipetalonema leopoldi	Subcutis				X	42
Dipetalonema gorillae	Subcutis				X	42
Tetrapetalonema papionis	Skin and skeletal muscle fascia			X		578
Dracunculids						
Dracunculus medinensis	Skin, subcutis, viscera			X		276, 290, 531, 609, 742
Onchocerca volvulus	Connective tissue				X	42, 646
Trichurids						
Trichuris trichiura	Cecum, colon		X	X	X	3, 75, 76, 276, 290, 339, 350, 461, 486, 487, 494, 650, 654, 688, 712, 742, 791, 836, 921, 922
Trichuris sp.	Cecum colon	X		X		61, 387, 390, 488, 491, 493, 494, 616, 658, 660, 662, 922
Capillaria hepatica	Liver		X	X	X	276, 290, 349, 350, 420, 507, 531, 742, 810, 854, 855
Capillaria sp.	Liver		X	X	X	922
Anatrichosomatids						
Anatrichosoma cutaneum	Nasal mucosa, skin			X		8, 74, 276, 290, 650, 712, 742, 791, 833
Anatarichosoma cynomolgi	Nasal mucosa			X		8, 129, 290, 650, 805
Anatrichosoma ocularis	Eye	X				282

349, 634, 650, 654, 712, 741, 742, 752, 764, 791, 863, 922, 929], and *Strongyloides stercoralis* and *Strongyloides* sp. in Old World monkeys (patas monkeys) and great apes (gibbons, chimpanzees, gorillas, and orangutans) [38, 171, 177, 222, 285, 290, 360, 504, 570, 650, 672, 687, 774, 791, 857, 861, 922]. Only adult females and larvae are found in the gastrointestinal tract of the host animal. Migrating larvae can be found in the lungs and other parenchymatous organs. Parasitic males have never been described [290].

The life cycle of *Strongyloides* sp. is complex and consists of both parasitic and free-living generations [116, 290]. The reader is referred to parasitology texts and referenced papers for a detailed discussion of this unique life cycle [30, 120, 272, 290, 540, 791, 811]. A variation in this life cycle, known as autoinfection, is a direct reinvasion of the host animal by filariform larvae that have developed during passage through the lower intestinal tract [171, 467]. This phenomenon results in hyperinfection of the infected host and is most responsible for sustained infections that result in clinical disease, severe damage to affected organs, and death [77, 95, 171, 290]. There is also evidence of intrauterine or transcolostral transmission [290, 589, 819]. Fatal cases of strongyloidiasis have been reported in the chimpanzee, gibbon, orangutan, patas monkey, and woolly monkey [50, 51, 171, 176, 228, 360, 422, 504, 526, 571, 636, 650, 677, 857, 861].

The disease in gibbons has been reported in detail [171]. Diarrhea, which may be hemorrhagic or mucoid, is the most common clinical sign described in infected animals [171]. Other common clinical signs are dermatitis, urticaria, anorexia, depression, listlessness, debilitation, vomiting, emaciation, reduced growth rate, dehydration, constipation, dyspnea, cough, prostration, and death [171, 274, 290, 461, 742, 791, 810]. Paralytic ileus is described in infected gibbons [171].

Gross lesions consist of catarrhal to hemorrhagic or necrotizing enterocolitis [171, 290, 461, 791]. There may be a secondary peritonitis associated with the enterocolitis [290, 428]. Pulmonary hemorrhage is the most common lesion outside the digestive tract [171, 290, 461, 791]. Histologic examination of the small intestine of the infected animal shows a multifocal erosive and ulcerative enteritis caused by adults, eggs, and rhabditiform larvae [171]. The mucosa contains numerous parasites, most of which are in intraepithelial tunnels or lumina of intestinal glands. These lesions may be infiltrated by neutrophils. Mononuclear cells and an occasional eosinophil can be seen in the lamina propria. Intestinal villi are short and blunt, and in severe infections bridging and loss of villi are seen [171]. In cases where autoinfection has occurred, changes in the small and large intestines in response to invasion by the filariform larvae range from a mild inflammatory cell response to severe, acute, or granulomatous or necrotizing enterocolitis. Larval invasion of the submucosal and serosal lymphatics results in a severe granulomatous endolymphangitis [171]. These changes are associated with various degrees of lymphatic obstruction and submucosal and serosal edema, fibrosis, or both. In the lungs, acute multifocal or diffuse hemorrhage is most common. Larval granulomas may be seen over the surface of the pleura. Filariform larvae also are seen in many tissues throughout the body, most commonly in the lymph nodes and liver [171]. Fatal strongyloidiasis has been described in lowland gorillas and chimpanzees [672].

This condition may be diagnosed by identification of typical larvae in the stool; by clinical signs; or by demonstration of parasitic adult females, eggs, and larvae at necropsy or at histologic examination [290].

Strongyloidiasis in nonhuman primate colonies is considered a potential public health problem. Infections by *S. fulleborni* that have been transmitted naturally from monkey to human have been reported [64, 290, 650, 742, 878]. Experimental infections in humans by *Strongyloides* sp. isolated from nonhuman primates also have been reported [49, 176, 272, 290, 650, 752].

Oxyurids

Oxyuriasis

This disease is caused by infection by nematodes in the family Oxyuridae. Commonly known as pinworms, these small nematode parasites inhabit the colon and cecum of nonhuman primate hosts. Genera described in nonhuman primates are *Trypanoxyuris* and *Oxyuronema* species found in New World monkeys [207, 410, 412, 461]; *Enterobius vermicularis* and other *Enterobius* species, found in Old World monkeys and great apes; *Enterobius anthropopitheci* in the chimpanzee [92, 96, 131, 222, 290, 362, 409, 570, 618, 650, 686, 687, 712, 718, 724, 742, 759, 774, 919, 922]; and several species in prosimian primates [28, 41, 106–108]. These parasites are considered cosmopolitan in geographic distribution but are more prevalent in temperate and cold climates. The life cycle is direct.

Most reports of oxyuriasis in nonhuman primates state that these infections are essentially innocuous [290, 461, 650, 791]. Clinical signs usually are limited to anal pruritis and irritation that may lead to self-mutilation, restlessness, and increased aggressiveness [131, 158, 200, 290, 461, 570, 650, 742, 774, 791]. Heavy pinworm infections are reported to be common in chimpanzees, and their coprophagic habits make constant reinfection inevitable [718]. Fatal cases of enterobiasis have been reported in chimpanzees [390, 439, 774, 775, 853], characterized by extensive ulcerative enterocolitis, peritonitis, and necrogranulomatous lymphadenitis involving the mesenteric lymphnodes. Numerous parasites with the morphologic characteristics of *Enterobius vermicularis* were associated with these lesions. There is also an early report of the death of a red spider monkey caused by an overwhelming pinworm infection [474].

Multiple intestinal polyps associated with immature male oxyurid parasites have been described in a male chimpanzee [848]. The gross and histologic characteristics of these lesions were identical to those produced by *Nochtia nochti* in the stomach and esophagus of Old World primates. It was thought that the lesion resulted from hypersensitivity to oxyurid infection in an aberrant host.

Adult oxyurids may be seen emerging from the anus. Perianal swabs or cellophane tape also can be used to recover the typical ellipsoid, asymmetrical pinworm eggs [276, 290].

Naturally infected nonhuman primates may be sources of infection in humans. Also, captive primates can acquire *E. vermicularis* infection from humans and then can act as reservoirs to reinfect them [290].

Strongylids

Oesophagostomiasis

This disease is caused by infection by nematodes in the genus *Oesophagostomum*. These parasites are known commonly as nodular worms and are considered to be the most common nematode parasite found in Old World monkeys and great apes

[42, 290, 442, 570, 645, 650, 712, 742, 791]. They have been described in baboons, mangabeys, guenons, macaques, chimpanzees, and gorillas [38, 47, 92, 285, 290, 339, 345, 350, 362, 367, 392, 438, 452, 486, 494, 578, 610, 618, 654, 712, 740, 774, 791, 618, 836, 863, 894]. They are rare in New World monkeys [207, 650, 922]. Their geographic distribution is widespread, almost universal. At least 11 different species have been proposed but not clearly defined [645, 922]. The species mentioned most frequently are *O. apiostomum*, *O. bifurcim*, *O. aculeatum*, and *O. stephanostomum* [276, 290, 531, 742, 764]. The life cycle is direct.

Infected monkeys usually are asymptomatic, and light infections usually go unrecognized [290, 742, 791]. Monkeys with severe infections may show general unthriftiness and debilitation characterized by increased weight loss and diarrhea; the mortality rate increases for this group [290, 742, 791]. Lesions seen at necropsy consist of the typical oesophagostomum nodules, which are elevated, smooth 2–4 mm in diameter, and firm. They are seen most frequently on the serosal surface of the large intestine and cecum and in the mesentery supporting these organs [92, 290, 618, 645, 742, 791, 869] but also in ectopic sites, such as the peritoneal wall, mesentery of the small intestine, omentum, kidney, liver, lungs, or diaphragm [118, 645, 650, 791]. The nodules may be black or brown if there is associated hemorrhage; older nodules usually are white because of caseation of the contents. Viable worms may be seen in relatively young nodules; usually, however, the parasite is dead and surrounded by a mass of caseous debris. Older nodules may contain foci of mineralization [290, 350, 791]. The parasite and cell detritus usually are surrounded by a mantle of chronic inflammatory cells, mainly macrophages with scattered eosinophils and lymphocytes and plasma cells. Foreign-body giant cells sometimes are present in the cellular exudate. A fibrous capsule of various degrees of thickness and maturity, depending on the age of the nodule, surrounds the centrally located necroinflammatory mass [92, 290, 618, 791]. Sometimes ulcers form in the colonic mucosa at the point where the larval penetration occurred, and a migratory tract filled with inflammatory exudate connects the nodule in the wall with the intestinal lumen [290, 349, 350, 791]. Death of a chimpanzee from septicemia caused by bacterial invasion of esophagostomum nodules in the colon has been reported [892, 893, 894]. Rupture of the nodules may result in acute or chronic peritonitis with fibrous peritoneal adhesions [645, 791]. Adhesions may restrict intestinal motility and result in obstruction or rarely in ascites [290, 742, 791].

Oesophagostomum infection can be diagnosed by identifying the eggs in the feces. A problem arises, however, because the eggs of the different *Oesophaostomum* species cannot be differentiated from one another and also are indistinguishable from those of *Ternidens* and other hookworm species. The diagnosis of oesophagostomiasis based solely on typical eggs in the feces always should be questioned. Occasionally, adults are passed and can be identified. The postmortem diagnosis is based on typical nodular lesions or identification of adults, or both [290, 791].

This parasite has been reported to infect humans and therefore should be considered to have zoonotic potential [290, 347, 546]. Appropriate care in handling nonhuman primates should be exercised.

Ternideniasis

The cause of this disease, *Ternidens deminutus*, is a strongyle that is related to the oesophagostomes and hookworms [290, 650]. These parasites inhabit the cecum and colon and have been reported in Old World monkeys (macaques, guenons, and baboons) and the great apes (gorilla and chimpanzee) [9, 290, 339, 349, 531, 609, 626, 712, 742, 764, 836, 862, 919]. The morphologic features of the adult worms and their eggs are similar to those of *Oesophagostomum* [290, 742, 836]. The life cycle is direct and also is similar to *Oesophagostomum* [290]. There is little evidence of any lesions associated with this parasite; since it is a blood sucker, however, it can cause anemia and cystic nodules in the colonic wall [290, 531, 650].

This parasite can infect humans; it causes intestinal nodules. Infected captive animals should be handled with caution [290].

ANCYLOSTOMATIDS

Ancylostomiasis and Necatoriasis

The cause of these diseases, the hookworms usually found in humans, *Ancylotoma duodenale* and *Necator americanus*, are recorded occasionally in nonhuman primates, including monkeys, mandrills, baboons, gibbons, chimpanzees, and gorillas [38, 185, 222, 280, 290, 355, 358, 538, 570, 609, 647, 687, 718, 929]. Reports of their presence in South American monkeys are rare [650]. These parasites have a direct life cycle.

Clinical signs associated with heavy hookworm infection in nonhuman primates are similar to those produced by these parasites in humans and other animals and include anemia, eosinophilia, "pot-belly," dyspnea on exertion, and a general debilitation [290, 355, 358, 742]. Necropsy findings have included a general pallor of all tissues. The mucosa of the small intestine was thickened by a chronic inflammatory reaction. Small hemorrhages were seen throughout the intestinal mucosa, and large numbers of hookworms were attached to the mucosa [358].

The diagnosis of hookworm disease is based on finding eggs in the feces or mature worms in the bowel at necropsy [290]. Since hookworm eggs are morphologically identical to those of several species of strongyles that also infect nonhuman primates, diagnosis based on the eggs alone should be viewed with caution [650].

Because humans are the normal definitive host for these parasites, infected captive primates should be handled with caution [290].

TRICHOSTRONGYLIDS

Molineiasis

This disease is caused by trichostrongyles in the genus *Molineus*. These are small, slender, pale red worms that inhabit the upper digestive tract, duodenum, and sometimes the pyloric region of the stomach of nonhuman primates. Occasionally, they may involve the pancreas and mesentery [62, 148, 168, 201, 207, 290, 461, 509, 791, 919]. They are always found lying on the mucosa, never

attached. Geographically, they are distributed throughout Central and South America, with one species occurring in Africa [201, 207, 290]. Species described in nonhuman primates include *M. vexillarius* in marmosets; *M. elegans* in squirrel monkeys, cebus monkeys, and howler monkeys; *M. vogelianus* in pottos; and *M. torulosus* in cebus monkeys, squirrel monkeys, and owl monkeys [148, 201, 207, 290, 461, 791, 926]. *Molineus torulosus* is the only species reported to be a specific pathogen [62, 290, 461, 509, 791].

The life cycle and method of transmission of these parasites are unknown. Diagnosis rests upon the identification of typical eggs in the feces or the presence of adult worms associated with typical lesions in the digestive tract [290, 531, 649].

Infection with *M. torulosus* has been reported to cause hemorrhagic or ulcerative enteritis, sometimes associated with diverticula of the intestinal wall [62, 290, 461, 509, 791]. Serosal nodules that involved the upper portion of the small intestine have been seen in capuchin monkeys [62]. These nodules communicated with the intestinal lumen through 1-mm reddish brown ulcers. Histologically, the nodules were composed of an intense granulomatous inflammatory response surrounded by a rim of proliferating fibrous connective tissue. The central portion contained a mass of nematode parasites and their eggs surrounded by eosinophilic debris. Neutrophils, histiocytes, and other chronic inflammatory cells were present adjacent to the worms. Chronic pancreatitis also was seen, with worms and eggs in inflamed pancreatic ducts [62, 791].

Nothing is known of the public health significance of this parasite [290].

Nochtiasis

This disease results from infection by trichostrongyles in the genus *Nochtia*. *Nochtia nochti* is a small, slender, bright red worm that has been described in the prepyloric region of the stomach of Asian macaques [58, 290, 508, 650, 746, 791, 803, 851]. Eggs are thin shelled and ellipsoid, typical of members of the Trichostrongyles. *Nochtia* eggs can be differentiated from those of the superfamily *Strongyloidea* because they are larger and more pointed and are embryonated when passed in the feces. Free parasites are not found in the feces or the gastrointestinal tract. The life cycle is direct [58, 290].

At necropsy, hyperemic, cauliflowerlike masses are seen protruding from the gastric mucosa at the junction of the fundus and prepyloric regions. Histologically, these masses are benign inflammatory polyps composed of hyperplastic fronds of gastric mucosa and inflammatory tissue. Adult worms and their eggs can be found deep at the base of the lesion [58, 290, 622, 650, 746, 791, 803, 851].

Diagnosis depends on identification of typical eggs in the feces of affected animals or on the finding at necropsy of characteristic gastric polyps containing the parasite [290, 791].

Nothing is known of the public health aspects of this nematode.

METASTRONGYLIDS

Angiostrongyliasis

The cause of this disease, the metastrongyle *Angiostrongylus costaricensis* normally is found in rats in South and Central America [304]. It also causes a clinical syndrome in humans, particularly in children who reside in this geographic

region. This syndrome is characterized by an inflammatory granulomatous mass that usually is located in the wall of the appendix but that can extend to the ileum, the cecum and ascending colon, and regional lymph nodes [304]. Histologically, the granulomatous mass is composed of chronic inflammatory cells and nematode eggs. Adult parasites reside in the arteries of the intestinal wall and mesentery [304].

Similar parasitic granulomas in the wall of the small intestine have been reported from two mustached marmosets (*Saguinus mystax*) [802, 846]. The histomorphologic features of these lesions were identical to those described previously for *A. costaricensis* infection in humans. In addition to the chronic inflammatory cells, the granulomas contained numerous nematode eggs and many larvae. The eggs of this particular parasite are reported to hatch within the rat or monkey host, then migrate to the gut and pass out in the feces to complete the life cycle [304]. Adult parasites with morphologic features consistent with a diagnosis of *Angiostrongylus* sp. were found in the mesenteric arteries associated with the granuloma.

On the basis of the nonhuman primate species involved, the fact that this species originated in the geographical region where this parasite has been reported to occur, the gross and histological appearance of the lesions, and the finding of adult *Angiostrongylus* sp. in the mesenteric arteries intimately involved with the granulomas, these parasites were identified as *A. costaricensis*. People who work with marmosets should be aware of the presence of this parasite and look for additional cases to document further its occurrence in South American monkeys.

Pulmonary Nematodiasis

This condition is the result of infection with the metastronglyid lungworms in the genera *Filaroides* and *Filariopsis*. These parasites are most commonly seen in New World monkeys (marmosets, squirrel monkeys, cebus monkeys, and howler monkeys) [63, 148, 207, 290, 339, 461, 623, 650, 742, 791, 820, 865, 919].

In the live state, these parasites are very slender and fragile [650]. Adults are found in the terminal brochioles, respiratory bronchioles, and pulmonary aveoli [290, 461, 650, 791, 820]. The adult female is viviparious. They produce larvae that are coughed up, swallowed by the host, and passed in the feces. The remainder of the life cycle, and whether or not any intermediate hosts are required, is not known [461, 650, 820].

Gross lesions in the pulmonary parenchyma are subtle. The lung appears normal except for the presence of varying numbers of randomly located, small, elevated, subpleural nodules, which may be hyperpigmented and cause the pleura to bulge. Histopathologically, there are varying degrees of atelectasis and foci of chronic inflammatory cells infiltrating the affected alveolar spaces and intra-alveolar septae. Most infections are considered to be subclinical in nature, and although the parasite is common in certain species of New World monkeys, there is no evidence to suggest that the presence of lungworms has been the cause of death [461, 650, 820].

Diagnosis can be made by finding and identifying the typical lungworm larvae in the feces in the intact animal. In the dead animal, the presence of characteristic gross and histopathologic pulmonary lesions associated with metastrongylid parasites is also diagnostic.

The public health significance of these parasites is unknown [290].

ASCARIDIDS

Ascariasis

This disease results from infection with members of the genus *Ascaris*, commonly known as roundworms. They are a common finding in the intestinal tract of nonhuman primates [650]. The specimens that have been recovered are reported to be indistinguishable from *Ascaris lumbricoides* in humans [19, 209, 844, 922]. Both Old World monkeys and great apes have been reported to be infected [38, 209, 222, 279, 349, 355, 468, 676, 712, 742, 813, 814, 922]. The life cycle of this parasite is direct.

Although roundworm infection in nonhuman primates is thought to be relatively innocuous and of little clinical significance [570, 650], fatal cases of ascariasis have been reported in both monkeys and great apes [349, 676, 813]. Death in the great apes was thought to be due to the presence of many worms, blockage of the bowel, and migration of the worms into the bile duct and liver.

Diagnosis of ascariasis is based on the presence of typical eggs in the feces or adults in the digestive tract at necropsy.

Since the ascarids reported in nonhuman primates are morphologically identical to *A. lumbricoides* in humans, cross-infection from infected animals to humans is possible. We could find no reports that documented such an occurrence; nevertheless, infected nonhuman primates should be considered a potential zoonotic threat and should be handled accordingly.

SPIRURIDS

Trichospiruriasis

The cause of this disease, *Trichospirura leptostoma*, is a spirurid nematode that parasitizes the pancreatic ducts of several species of New World monkeys including marmosets, squirrel monkeys, and owl monkeys. Geographic distribution is confined to Central and South America [148, 149, 290, 461, 649, 650, 806, 807]. Male and female adult parasites measure up to 15 mm and 120 mm, respectively. The eggs are medium in size and are typically spirurid in that they are thick shelled and contain a larvae [534]. The complete life cycle is unknown [290, 461].

The parasite usually is found incidentally on histological examination. Infection usually causes little tissue destruction or inflammatory reaction. Tissue response apparently varies in proportion to the number of parasites present [461, 650]. Chronic pancreatitis in association with the worms has been described in marmosets (*Callithrix* sp.) [650, 807]. Acute pancreatitis in owl monkeys consisting of a patchy granulocytic interstitial infiltrate adjacent to intralobular ducts was thought to be associated with leakage of retained pancreatic secretions. Larger ducts containing cross sections of worms also contained granulocytes [649, 650].

Nothing is known about the public health significance of this parasite.

Pterygodermatitiasis

The cause of this disease, *Pterygodermatites nycticebi*, is a spirurid nematode that has been reported from prosimians (slow loris), New World monkeys (tamarins and marmosets), and the great apes (gibbons) [536, 593, 856]. Several reports in members of the family Callitrichidae refer to this parasite by a synonym, *Ric-*

tularia nycticebi [4, 591, 592, 930]. The life cycle of *P. nycticebi* is indirect with cockroaches serving as intermediate hosts [593].

Morbidity and mortality associated with infection of *P. nycticebi* have been reported in golden lion tamarins (*Leontopithecus rosalia*) [593]. Clinical signs in heavily infected animals included extreme weakness, passage of watery diarrhea that contained the adult parasites, anemia, leukopenia, and hypoproteinemia [593]. At necropsy, masses of *P. nycticebi* parasites were found throughout the gastrointestinal tract. Histopathologically, the anterior ends of the adult worms were embedded in the mucosa of the small intestine. Larvae were seen deeper in the submucosa. In a few cases, worms were seen in the tunica muscularis and the pancreatic ducts. There was severe clubbing of the small intestinal villi and randomly located foci composed of a necrotic pseudomembrane containing spirurid eggs, numerous yeasts, and pseudohyphae consistent with *Candida* sp. [593].

Diagnosis depends upon demonstrating and identifying the characteristic spirurid eggs, adult worms, or larvae in the feces, in the gastrointestinal tract at necropsy, or in histopathological slide preparations.

Nothing is known about the public health significance of this parasite.

Methods of control should be directed against the cockroach intermediate host through reducing populations and preventing consumption by susceptible hosts.

Thelaziids

Streptopharagiasis

This disease is caused by parasitic members of the genus *Streptopharagus*. These are thelaziid nematodes that have been described in the stomach of Old World monkeys and great apes [290]. *Streptopharagus armatus* is reported in the rhesus monkey, other macaques, guenons, patas monkey, baboon, and gibbon [290, 349, 836]. *Streptopharagus pigmentatus* has been reported in the rhesus monkey, cynomolgus monkey, guenon, baboon, and gibbon [290, 339, 362, 712]. The life cycle of *S. pigmentatus* has been reviewed recently [551]. Little is known about the anatomic effects of these parasites [290]; however, there is one report of the death of a baby chimpanzee as a result of a perforated esophagus secondary to the migration of *Streptopharagus* sp. larvae [127].

Gongylonemiasis

The cause of this disease, parasites in the genus *Gongylonema*, are small filiform, thelaziid nematodes that have been reported in many nonhuman primates including both Old and New World monkeys [16, 290, 461, 533, 650, 911, 919, 922]. The species most commonly mentioned are *G. macrogubernaculum* and *G. pulchrum*. They have a cosmopolitan geographic distribution. A characteristic feature of the adults is several rows of conspicuous oval to round cuticular bosses located at the anterior extremity [650]. The life cycle is indirect, with cockroaches or dung beetles serving as intermediate hosts [209, 650].

Infection with this parasite is asymptomatic. Its presence usually is recognized only histologically and is considered to be an incidental finding. Adults are found in tunnels in the stratum malpighii of the squamous epithelium of the esophagus, lip, tongue, and other parts of the buccal cavity. They have been recovered from bronchi and the stomach. There is little or no tissue reaction [290, 461, 650].

Gongylonema pulchrum has been reported to occur in humans [290].

PHYSALOPTERIDS

Physalopteriasis

This disease is caused by infection by members of the genus *Physaloptera*. Four species of physalopterids have been reported to occur in the upper gastrointestinal tract of nonhuman primates [290]. *Physaloptera tumefaciens* is common in the stomach of Asian macaques [290, 362]. *Physaloptera dilatata* is found in the stomach of New World monkeys (titi monkeys, bearded sakis, and marmosets) [168, 207, 290, 492, 919]. *Abbreviata caucasica* has been found in the esophagus, stomach, and small intestine of the rhesus monkey, baboon, and orangutan [290, 419, 531, 626, 742, 919]. *Abbreviata poicilometra* has been found in the stomach of mangabeys and guenons [290, 756, 799, 919].

The life cycle of the physalopterids is indirect; an arthropod intermediate host is required. The entire life cycle is not completely understood, and a second intermediate or paratenic host may be necessary [290, 810]. Lesions result from the attachment of the worms to the wall of the affected organ. Gastritis, esophagitis, enteritis, erosion, and ulceration of the mucosa at the point of attachment are seen at necropsy [290]. Hyperplastic gastric lesions and perforation of the stomach wall associated with *Physaloptera* sp. infection in cynomolgus monkeys have been described [287, 746].

Diagnosis depends upon identification of the ova in the feces or the presence of adult worms attached to the mucosa of the upper digestive tract [290].

The public health aspects of these parasites are unknown.

ONCHOCERIDS

Filariasis

This condition is caused by a variety of onchocercid or filarial nematodes that are commonly encountered parasites of nonhuman primates. The adult filarids are long, slender worms that inhabit various tissue sites in the host animal outside the gastrointestinal tract [290, 461, 531, 650, 742, 791, 889]. The length of the adult worm varies, depending on the species, from a few centimeters to as much as 30 cm. Female filariae are typically much larger than the males. The female worms produce small, primitive larvae called microfilariae that circulate throughout the peripheral blood or live in the skin of the definitive host [167, 168, 290, 461, 650, 742, 889]. The life cycle for these parasites is indirect. Obligatory intermediate hosts include an extensive variety of biting and blood- or lymph-sucking insects [461, 791]. The filarial worms reported from nonhuman primates are listed in Table 45.5.

Filariasis in New World Monkeys. At least 12 different species of filarid nematodes have been described from New World monkeys (marmosets, squirrel monkeys, cebus monkeys, spider monkeys, and owl monkeys). These include four species of *Dipetalonema* and seven species of *Tetrapetalonema* [52, 148, 168, 184, 207, 210, 230, 231, 290, 310, 311, 461, 508, 580, 742, 791, 845, 889, 923]. Mixed infections in the same animal are reported to be very common, with some animals containing as many as four different species at the same time [461]. These species live in the abdominal or thoracic cavities or in the subcutaneous tissues of the definitive host. The worms that locate in the subcutaneous tissues

cause very little if any inflammatory response [461, 650]. Those species that are found in the serous cavities (*D. gracile* and *D. caudispina*) can cause a fibrinopurulent peritonitis or pleuritis with associated fibrinous adhesions that frequently results in entrapment of the worms [461, 650].

The filarid *Loa loa* has been reported from a spider monkey [872].

Filariasis in Old World Monkeys. Dirofilaria corynodes is reported to be the most prevalent filarial parasite of African Old World monkeys (vervets, mangabeys, colobus monkeys, and patas monkeys). These are large parasites that are found in the subcutaneous tissues of the trunk and lower extremities where their presence causes very little tissue reaction [650]. Two closely related species, *D. magnilarvatum* and *D. macacae*, have been reported from Asian Old World monkeys (cynomolgus monkeys) [650].

Macacanema formosana has been reported from Asian Old World monkeys (Taiwan macaque and cynomolgus monkey). This parasite commonly inhabits the peritracheal connective tissue and the diaphragm of the infected host [43, 290, 531, 650, 768].

Edesonfilaria malayensis has been described in Old World monkeys (cynomolgus monkeys and rhesus monkey) [290, 308, 531, 635, 712, 920, 924]. The adult worms usually are found free in the peritoneal cavity but have been reported from the subserosal connective tissue of the abdominal and thoracic cavities. In one report, they were associated with retroperitoneal masses composed of fibrous connective tissue and multiple foci of lymphoplasmocytic infiltrates. Numerous migratory tracts containing amorphous eosinophilic debris or adult *E. malayensis* worms were scattered throughout the masses [308]. In another report [635], six female adult *Macaca fascicularis* monkeys were found to be infected with *E. malayensis*. Clinical pathological findings in the infected animals included reduced values of hemoglobin and hematocrit, eosinophilia, elevated level of total protein, and a decreased A/G ratio. Gross lesions consisted of thickening of the connective tissues, hemorrhage, and adhesions of the serosa in the site occupied by the worms. Mechanical damage was seen occasionally in tissues adjacent to the location of the parasites such as the pancreas and iliopsoas muscle. Splenic nodules were seen in five of the six infected monkeys. Histopathologically, there was hemorrhage, fibroplasia of connective tissue, and proliferation of granulation tissue with infiltration of eosinophils, lymphocytes, and other inflammatory cells associated with the presence of worms in the tissues. The nodular lesions in the spleen consisted of a highly vascular network of large reticuloendothelial cells, reticulum fibers, eosinophils, and erythrocytes. Microfilariae were present in some of these lesions, and it was felt that the splenic nodules were most likely associated with their existence in the spleen [635].

Loa loa, normally a parasite of humans, has been reported from a variety of Old World monkeys (drills, baboons, mangabeys, and vervets) [199, 650, 755, 852]. Except for size, the worms described from both human and nonhuman primates are nearly identical morphologically. Another variation is the different circadian rhythm displayed by the microfilariae produced by the worms that infect nonhuman primates. These larvae circulate in the peripheral blood with a nocturnal periodicity [650]. Infection is usually asymptomatic, and significant lesions related to the presence of the adult *L. loa* in the subcutaneous tissues of nonhuman primate hosts has not been reported [650]. However, there has been a report

of splenic lesions in drills infected with *L. loa*. Grossly, there were multiple nodules over the surface of the spleen resulting from the presence of granulomas that arose in the red pulp. Microscopically, the nodules were composed of fibrous connective tissue and numerous multinucleated giant cells, many of which contain disintegrating microfilariae within their cytoplasm. These lesions were attributed to the destruction of microfilariae within the spleen [198].

Brugia malayi and *Brugia pahangi* have been reported from a wide variety of Asian monkeys, particularly *Macaca* species [87, 500, 650]. *Brugia malayi* is also a parasite of humans. The adult parasites are found in the lymphatic and perilymphatic tissues of their nonhuman primate hosts. Symptoms and histopathology in the lymphatic system similar to that seen in human Malayan filariasis have not been reported in infected nonhuman primates [650]. Another species, *Brugia tupaidae*, has been described from the lymphatic system of prosimians (tree shrews) [650].

Meningonema peruzzii is a relatively recently reported filarid parasite from African Old World monkeys (vervets and talapoin monkeys) [648]. These worms were found only in the subarachnoid space along the dorsum of the brain stem at the level of the medulla oblongata. Female *M. peruzzii*, unlike most other filariae, are quadridelphic. Symptoms and lesions associated with infection by this parasite were not reported [648].

Filariasis in Great Apes. *Onchocerca volvulus*, a parasite of humans, has been reported from the gorilla [42, 650]. The parasite was located in a subcutaneous fibrous nodule morphologically similar to that formed by the parasite in the human host [42, 650].

Dipetalonema streptocerca and *D. rodhaini* are two filarid parasites reported from the chimpanzee. These two parasites, along with *O. volvulus* from the gorilla, are different from other filarids in that the microfilariae produced by the female remain in the dermis rather than circulating in the peripheral blood [646, 650].

Several other filarids have been reported from the great apes including *Dirofilaria pongoi* from the heart of an orangutan [650, 873] and *Dirofilaria immitis* in the abdominal cavity of another orangutan [650, 760]. *Loa loa* also has been reported from the chimpanzee and gorilla [42, 734]. *Dipetalonema vanhoofi*, a filarid parasite of the chimpanzee, inhabits the mesenteries and the connective tissue adjacent to the gall bladder, bile duct, liver, pancreas, and kidney; and the loose connective tissues and lymphatics surrounding the hepatic blood vessels [650, 671]. They also have been described from the periadrenal connective tissue [646, 650].

Diagnosis is based on demonstration and identification of the adult worms in the body cavities or subcutaneous connective tissues, or the characteristic microfilariae in the blood [111, 290, 791].

Several filarial nematodes affect humans (*Dirofilaria*, *Onchocerca*, *Loa*), but the public health significance for the majority of these species is unknown [276, 290].

Trichurids

Trichuriasis

This disease is caused by parasites in the genus *Trichuris*. Trichurid parasites are common inhabitants of the cecum and large intestine of nonhuman primates [276,

290, 531, 570, 650, 742]. These nematodes have a world-wide distribution but are more prevalent in the tropics and subtropics [290]. Nonhuman primates reported to be affected include New World species (howler monkeys, woolly monkeys, and squirrel monkeys) [76, 208, 365, 492, 650, 814], Old World species (rhesus monkeys, cynomolgus monkeys, Japanese macaques, Formosan macaques, African green monkeys, and baboons) [75, 290, 339, 362, 487, 494, 609, 712, 741, 764, 836, 922], and great apes (gibbons and chimpanzees) [222, 290, 359, 570, 686, 689, 712, 843, 863, 893]. These parasites are morphologically identical to and indistinguishable from *T. trichiura* in humans [290, 570, 650, 742]. The life cycle is direct.

Trichuriasis in nonhuman primates usually does not cause any significant clinical problems [570]. Light infections are reported to cause no apparent lesions; heavy infections, however, have been reported to cause anorexia, a gray mucoid diarrhea, and sometimes death [290, 339, 742, 843]. Fatal whipworm infections have been reported in two chimpanzees and a gibbon. Death of one chimpanzee was attributed to a severe parasitic enteritis; the second death was thought to be the result of a secondary bacterial infection resulting from the *Trichuris* infection [843, 893]. The death of a gibbon with chronic colitis caused by an overwhelming infection with *Trichuris* and oxyurid parasites also has been reported [359].

Diagnosis depends upon the identification of the characteristic double operculated eggs in the feces or adults in the cecum [290].

Because the trichurid species that affects nonhuman primates is morphologically similar to the whipworm found in humans, cross-infection from animal to human is possible [290, 570, 742]. Appropriate care in the handling and management of infected captive nonhuman primates is recommended.

Capillariasis

This disease results from infection with the cosmopolitan trichurid parasite *Capillaria hepatica*. It has been reported in the liver of a wide variety of mammalian hosts throughout the world including New World monkeys (squirrel monkeys, cebus monkeys, and spider monkeys), Old World monkeys (rhesus monkeys), and great apes (chimpanzee) [276, 290, 349, 350, 420, 507, 531, 742, 791, 810, 854, 855].

The anterior portion of these parasites is more slender than the posterior, but it is not as pronounced as in the whipworms. The eggs have bipolar opercula, and the shell contains many small perforations giving it a striated appearance. This feature is unique and is used to distinguish ova of *C. hepatica* from those of other trichurids [290, 650, 791].

The life cycle is direct and unique. Adult worms are found only in the hepatic parenchyma, and eggs are retained within the liver until the host dies or is killed. The eggs must be liberated from the liver either by decomposition of the original host or by passage through a predator or scavenger. Ingestion of infected liver tissue produces only spurious passage of the eggs in the feces. To become infective, the eggs must undergo embryonation under aerobic conditions. Infection occurs when embryonated eggs are ingested [290, 350, 650, 791].

The liver of infected animals reveals randomly placed white or yellow patches or nodules over the surface. Histopathologically, these foci are composed of adult *C. hepatica* and masses of eggs that are surrounded and infiltrated by proliferating fibrous connective tissue, chronic inflammatory cells, and foreign body giant

cells. These lesions are ultimately converted to scar tissue, and the liver becomes cirrhotic. Fatal hepatitis has been reported in infected nonhuman primates [280, 290, 650, 742, 791].

Neither eggs or adult parasites will be found in the feces; therefore, diagnosis depends on demonstration and identification of the typical eggs and/or worms through liver biopsy or at necropsy [290, 791].

This parasite is pathogenic for humans, but because of the unusual life cycle of *C. hepatica*, infective nonhuman primates do not constitute a public health menace for persons caring for or working with them [276, 290].

ANATRICHOSOMATIDS

Anatrichosomiasis

This condition is the result of infection with the anatrichosomatid parasites *Anatrichosoma cutaneum* or *Anatrichosoma cynomolgi*. These two species have been described from both Asian and African Old World nonhuman primates (rhesus monkeys, cynomolgus monkeys, patas monkeys, vervets, talapoin monkeys, mangabeys, and baboons) [8, 129, 142, 276, 290, 446, 585, 644, 712, 742, 805, 833] and great apes (gibbons) [74].

The adult worms are small and slender. The eggs are large, barrel shaped, have bipolar opercula, and unlike *Trichuris* and *Capillaria* contain a larva [290, 650].

The entire life cycle and method of transmission are not known, but the cycle is thought probably to be direct. The female worms migrate through the stratified layers of squamous epithelium forming tunnels in which the embryonated eggs are deposited [8, 290, 644, 650]. These tunnels are composed of epithelial cells and maintain their integrity. They are sloughed with the superficial keratin layers of the squamous epithelium and accumulate on the mucosal surface of the nares. Eggs are excreted from the host in the nasal secretions and less often in the feces [8, 290, 644, 650].

The original report of this parasite in nonhuman primates was from skin lesions on the extremities. Grossly, these lesions had the appearance of white, serpentine tracks on the palms and/or soles of the hands and feet [650, 833]. Since then they have been reported only from the stratified squamous epithelium of the external nares. Infection of the nares does not produce serious disease and is usually subclinical but is considered to be common in susceptible animals. Histopathologically, the affected epithelium is diffusely hyperplastic and parakeratotic, and there is a mild inflammatory infiltrate composed of leukocytes and plasma cells in the underlying lamina propria [8, 290, 644, 650, 791].

Diagnosis in the living animal can be made through the use of nasal mucosal scrapings or swabs that will reveal the characteristic eggs. In the dead animal, finding of the parasite in microscopic slides of the mucosa is considered to be diagnostic [8, 290, 650, 791].

This parasite has been reported in humans, where it causes a type of creeping eruption. Even though infection in humans is considered to be uncommon, those personnel who work with and care for nonhuman primates should handle those species known to be infected or susceptible to infection with proper caution [276, 290, 650].

A new species, *Anatrichosoma ocularis*, has been reported recently from the eye of a tree shrew (*Tupaia glis*) [282].

Trematodes

The parasitic trematodes described from nonhuman primates are listed in Table 45.6. The species most frequently mentioned in the literature are discussed below.

Trematodiasis

This disease in nonhuman primates can be caused by infection with a number of species of trematodes. Several of the more commonly encountered species will be discussed in detail.

Gastrodiscoidiasis

Gastrodiscoides hominis is a small, orange-red fluke that attaches to the mucosa of the cecum and colon [290, 339, 380, 791]. The parasite is distributed throughout the tropical orient and has been described in various *Macaca* species that range throughout this geographic area [276, 290, 339, 362, 380, 403, 570, 695, 712, 764, 791, 903, 940]. The life cycle is indirect with a snail serving as the intermediate host [276, 290, 791]. Diagnosis can be made by identifying the characteristic eggs in the feces or by finding the typical adult flukes in the lumen of the cecum or colon at necropsy [290, 791].

Infection usually is asymptomatic when the parasites are present in small numbers. Heavy infections produce a mucoid diarrhea and mild chronic colitis. Attachment of the flukes to the intestinal mucosa results in focal lesions characterized by hyperemia, loss of surface epithelium, and necrosis. Neutrophilic infiltrates may be associated with these lesions. The submucosa may be sclerotic because of proliferation of fibrous connective tissue and a lymphoplasmacytic cell infiltrate [276, 290, 296, 380, 403, 791].

This parasite has been reported to cause a mild diarrhea in humans, but because of the obligatory snail intermediate host in the life cycle, infected captive monkeys are not a direct health hazard for humans [276, 290].

Watsoniasis

Watsonius watsoni, *W. deschieni*, and *W. macaci* have been reported to inhabit the intestinal tract of several Old World primate species (guenons, baboons, and cynomolgus monkeys) [276, 280, 290, 339, 362, 609, 742].

Adult trematodes of this genus are translucent, orange, and pear-shaped. The complete life cycle is not known but probably involves a snail intermediate host and is thought to be similar to that of *Fasciola hepatica* [276, 290, 742].

Watsonius watsoni and *W. deschieni* have been reported to be associated with diarrhea, severe enteritis, and death in monkeys [290, 339, 742]. Little else is known about the anatomic effects of these species [290].

Diagnosis can be made from the characteristic eggs in the feces or adults in the intestine at necropsy [290].

The public health considerations for these flukes are the same as described for *G. hominis*.

TABLE 45.6. Parasitic trematodes described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Plagiorchids						
Plagiorchis multiglandularis	Intestine			X		482
Lecithodendriids						
Novetrema nycticebi	Intestine	X				482
Odeningotrema apidion	Intestine	X				482
Odeningotrema bivesicularis	Intestine	X				482
Phaneropsolus bonnei	Intestine	X		X		482
Phaneropsolus lakdivensis	Intestine	X				147, 482
Phaneropsolus longipenis	Intestine	X			X	147, 482
Phaneropsolus orbicularis	Intestine		X			147, 148, 290, 461, 482, 689, 841, 917
Phaneropsolus oviformis	Intestine	X		X		147, 290, 482
Phaneropsolus simiae	Intestine			X		482
Phaneropsolus aspinosus	Intestine			X		657
Primatotrema macacae	Intestine			X		147, 290, 482
Dicrocoeliids						
Athesmia foxi	Bile ducts		X			26, 59, 147, 235, 275, 280, 290, 311, 333, 461, 482, 689, 742
Athesmia heterolecithodes	Bile ducts		X			482
Brodenia laciniata	Bile ducts, pancreas			X		147, 290, 482, 486, 487
Brodenia serrata	Pancreas			X		147, 290, 482
Concinnum brumpti syn. (*Eurytrema brumpti*)	Bile duct, pancreas				X	147, 280, 290, 482, 694, 701, 791, 828, 922
Controrchis biliophilus	Gall bladder, bile ducts		X			147, 461, 482
Dicrocoelium colobusicola	Bile ducts			X		147, 482
Dicrocoelium lanceatum	Bile ducts			X	X	147, 367, 482, 922
Dicrocoelium macaci	Bile ducts			X	X	147, 280, 290, 356
Euparadistomum cercopitheci	Gall bladder			X		482

TABLE 45.6. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Eurytrema pancreaticum	Pancreatic ducts			X		482
Eurytrema satoi	Bile ducts, pancreas			X	X	147, 280, 290, 482
Leipertrema rewelli	Pancreas				X	147, 290, 482, 760
Platynosomum amazonensis	Gall bladder, bile ducts		X			147, 148, 290, 461, 482
Platynosomum marmoseti	Gall bladder, bile ducts		X			147, 148, 290, 461, 482
Zonorchis goliath	Bile ducts		X			461, 482
Zonorchis microcebi	Bile ducts		X			482
Fasciolids						
Fasciola hepatica	Liver			X		147, 290, 339, 362, 482
Fasciolopsis buski	Duodenum, stomach			X		147, 276, 290, 361, 482
Opisthorchids						
Chonorchis sinensis	Bile ducts			X		482, 490
Opisthorchis felineus	Bile and pancreatic ducts			X		482
Heterophyids						
Haplorchis pumilio	Intestine			X		482
Haplorchis yokogawai	Intestine			X		147, 482
Metagonimus yokogawai	Intestine			X		147, 482
Pygidiopsis summa	Intestine			X		482
Microphallidids						
Spelotrema brevicaeca	Intestine			X		482
Echinostomatids						
Artyfechinostomum sp.	Intestine			X		147, 290, 482
Echinostoma aphylactum	Sm. intestine		X			147, 461, 482, 841
Echinostoma ilocanum	Intestine			X		48, 147, 276, 290, 482
Reptiliotrema primata	Intestine			X		147, 290, 482
Notocotylids						
Ogmocotyle ailuri	Sm. intestine			X		482
Ogmocotyle indica	Sm. intestine			X		147, 290, 339, 482, 490, 927

(Continued)

TABLE 45.6. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Troglotrematids						
Paragonimus westermani	Lungs, pleural cavity, diaphragm, body cavity, brain			X		147, 276, 288, 290, 362, 606, 742, 761, 810
Paragonimus africanus	Lungs			X		744
Achillurbainia sp.	Parotid gld.	X				655
Schistosomatids						
Schistosoma bovis	Mesenteric and abdominal veins			X		482
Schistosoma haematobium	Mesenteric, visceral, and abdominal veins			X	X	147, 170, 227, 276, 290, 482, 625, 627, 698
Schistosoma japonicum	Mesenteric and portal veins			X	X	147, 401, 482
Schistosoma mansoni	Mesenteric and abdominal veins		X	X	X	124, 147, 227, 276, 278, 280, 290, 367, 402, 482, 486, 487, 498, 507, 587, 698, 717, 822, 832
Schistosoma mattheei	Mesenteric and abdominal veins			X		49, 276, 482, 578, 609, 823
Schistosoma sp.	Mesenteric and abdominal veins			X	X	482
Diplostomatids						
Diplostomid mesocercariae	Visceral and pulmonary cysts		X	X		482
Neodiplostomum tamarini	Intestine		X			147, 148, 193, 290, 461, 482
Paramphistomids						
Chiorchis noci	Intestine			X		147, 290, 482
Gastrodiscoides hominis	Cecum, colon			X		147, 276, 290, 339, 362, 380, 403, 482, 660, 662, 658, 695, 712, 764, 791, 904
Watsonius deschiensi	Intestine			X		147, 276, 290, 482, 609, 742, 791
Watsonius watsoni	Intestine			X		147, 276, 280, 290, 339, 362, 482, 609, 742, 791
Watsonius macaci	Intestine			X		147, 276, 290, 362, 482, 791

Paragonimiasis

This disease, caused by the oriental lung fluke *Paragonimus westermanii*, has been reported in the cynomolgus monkey. Infection is directly associated with this animal's ingestion of infected raw crabs or crayfish as part of its dietary regimen [276, 288, 290, 362, 606, 742, 761, 810].

The adult flukes have a brown, plump, ovoid body with scalelike spines. The eggs are oval shaped, golden-brown in color, and have a partly flattened operculum at one end. The life cycle is indirect with snails and crabs or crayfish serving as intermediate hosts [276, 290, 791].

Adult flukes are found primarily in the lung but sometimes occur in ectopic sites such as the brain, liver, and other organs. The clinical signs reported in infected animals include coughing, wheezing, bloody or rusty-tinged sputum, moist rales, and progressive emaciation [290, 791].

At necropsy, lesions consist of focal areas of emphysema and soft, dark red to brown cysts that measure 2–3 cm in diameter and are randomly located throughout the pulmonary parenchyma. These cysts may be elevated above the lung surface, and pleural adhesions can sometimes be present. Two or more flukes occupy each cyst [290, 482, 791].

Histopathologically, the presence of the flukes provokes a leukocytic infiltration, and there is usually a mature fibrous capsule around the parasites that in turn are surrounded by a purulent exudate containing blood and groups of typical-appearing fluke eggs. Hemorrhage into the cyst often occurs, and this may lead to hemoptysis. Additional lesions described include hyperplasia of bronchial epithelium and submucosal glands and focal areas of inflammation in the lung parenchyma associated with groups of fluke eggs [290, 482, 791].

The diagnosis depends on the demonstration and identification of the typical eggs in the feces or the adult flukes in the pulmonary tissue at necropsy [290, 482, 791].

Paragonimus westermanii can affect humans; however, because of the obligatory molluscan and crustacean intermediate hosts in the life cycle, infected captive nonhuman primates are not a direct health hazard for humans [290, 482].

Schistosomiasis

Several species of schistosomatid flukes have been reported to infect nonhuman primates naturally. These include *Schistosoma mansoni* in New World monkeys (squirrel monkeys) [482, 832], Old World monkeys (mangabeys, patas monkeys, guenons, and baboons) [124, 227, 276, 278, 280, 290, 482, 487, 498, 587, 698, 791, 822], and the great apes (chimpanzees) [367, 402, 482, 717, 791]; *Schistosoma haematobium* in Old World monkeys (mangabeys, guenons, and baboons) [227, 276, 290, 482, 625, 627, 698, 791] and the great apes (chimpanzees) [170, 290, 482]; and *Schistosoma mattheei* in Old World monkeys (baboons) [49, 276, 482, 578, 609, 791, 823]. Although schistosomatids are considered to be extremely serious pathogens for humans, they are of little consequence in captive nonhuman primates and are usually found incidentally at necropsy [290, 487].

In the schistosomatids, both male and female forms are present and differ in appearance. They are usually found together in constant copulation with the long, slender female in the sex canal of the short, muscular male. The egg of *S. mansoni* is elongated-ovoid in shape, rounded at both ends, and bears a lateral

spine. The egg of *S. haematobium* is also elongated-ovoid in shape, rounded at the anterior end, and bears a posterior terminal spine. Adult *Schistosoma mansoni* and *Schistosoma mattheei* inhabit the mesenteric veins, whereas *Schistosoma haematobium* adults are found in the pelvic or portal veins of susceptible hosts [290, 791].

The life cycle is indirect with snails serving as intermediate hosts [290, 791].

The reported clinical signs include pyrexia, hemorrhagic diarrhea or hematuria, and ascites [276, 290, 482, 791]. The principal pathologic effects are caused by the presence of eggs in the tissues [276, 290, 428, 482]. The eggs may be found almost anywhere in the abdominal or pleural cavities. The most frequently encountered lesion is thickening of the intestinal or urinary bladder walls caused by chronic inflammation. Microgranulomas surrounding typical schistosome eggs are also very common in the liver, brain, spleen, wall of the gastrointestinal tract and urinary bladder, and other organs. Continued insult can lead to stenosis of portions of the gastrointestinal tract, urinary bladder and other parts of the urogenital system, and cirrhosis of the liver [171, 276, 290, 428, 482, 698].

Diagnosis is made based on the finding and identification of the characteristic eggs in the feces or urine, the presence of adult schistosomes in the blood vessels at necropsy, or the finding of the typical lesions during histopathological examination of appropriate tissues [290, 482, 791].

Infected captive nonhuman primates are not of direct public health significant to humans because of the requirement for an obligatory molluscan intermediate host. However, because schistosomiasis is such an important and serious disease in humans, excreta from nonhuman primates should be decontaminated before disposal [290].

Athesmiasis

The cause of this disease, *Athesmia foxi*, is considered to be a moderately pathogenic fluke that inhabits the bile ducts of susceptible nonhuman primate species. It is a common finding in nonhuman primates obtained from South America and has been reported in a variety of New World monkeys (cebus monkeys, squirrel monkeys, tamarins, and titi monkeys) [59, 147, 235, 275, 280, 290, 311, 333, 461, 689, 742, 824].

The adult flukes are long and slender and measure 8.5 × 0.7 mm. Eggs are ovoid and golden-brown in color; they have a thick shell and are operculated [290, 791, 824].

The life cycle is indirect with a mollusk serving as a required intermediate host. However, because the method of infection of the vertebrate host is unknown, our knowledge about the life cycle of this particular fluke is incomplete [290, 791].

Infections in nonhuman primates are usually asymptomatic and most often considered an incidental finding. Aside from causing a moderate to marked distension of affected ducts, these parasites cause very little damage and do not invoke much of a host inflammatory response. Heavy infections can result in hyperplasia of the biliary epithelium and fibroplasia around eggs and the ducts. Extremely severe infections can result in a pronounced thickening of the bile ducts with resultant pressure and trauma to adjacent hepatic parenchyma leading to fatty degeneration of affected hepatocytes [59, 235, 290, 311, 461, 481, 742, 791, 824].

Diagnosis depends on the demonstration and identification of the adult flukes in the bile duct either at necropsy or on histopathological examination of liver sections, or by demonstration and identification of the characteristic eggs in the feces [290, 791].

Nonhuman primates infected with *A. foxi* do not pose any public health problems for humans. This parasite has not been reported from humans. Infected nonhuman primates are not a direct hazard to humans because of the need of a required molluscan intermediate host to complete the life cycle [280, 290].

Cestodes

Cestodiasis

This condition results from infection by one of any of the numerous tapeworm genera that have been described in the intestinal tract of nonhuman primates, including prosimians, New and Old World monkeys, and great apes [151, 202, 204, 222, 280, 290, 362, 365, 461, 486, 487, 578, 608, 654, 687, 712, 771, 774, 825, 826, 832, 836, 862, 908, 918]. Cestode genera and the primate group they parasitize are listed in Table 45.7. Life cycles for all the genera listed, except one, are indirect and require an arthropod intermediate host for completion of the cycle. *Hymenolepis nana* can complete its life cycle either through direct or indirect means [290, 461].

Although these parasites may be present in large numbers, clinical disease or enteric lesions are seldom associated with tapeworm infection. Diagnosis depends on the identification of characteristic eggs in the feces, passing of proglottids of adult worms, or the recovery of adult worms at necropsy [290].

Some tapeworm genera (*Hymenolepis*, *Raillietina*, *Bertiella*) rarely affect humans. Proper precautions in handling captive nonhuman primates, good personal hygiene by the caretakers, and care in disposing of bedding and feces of infected animals should be stressed in order to rule out accidental transfer of infection to humans [290].

Larval Cestodiasis

Nonhuman primates may serve as intermediate hosts for several species of tapeworm parasites and thus develop various larval forms of these parasites in their somatic tissues. The larval cestode species and the primate group they parasitize are listed in Table 45.7. The cestode larvae are classified as solid and bladder forms. The solid larvae are represented by the sparganum. The bladder larvae consist of cysticercus, coenurus, hydatid, and tetrathyridium.

Sparganosis

This term denotes infection with the elongate, nonspecific plerocercoid larvae of cestodes in the order Pseudophylloidea [461, 791]. The adult tapeworms belong to the genera *Diphylobothrium* and *Spirometra*, which are intestinal parasites of various carnivores, birds, and reptiles [608].

Spargana have been described in New World monkeys (squirrel monkeys and marmosets) [148, 204, 207, 290, 461, 608], Old World monkeys (rhesus monkeys, cynomolgus monkeys, vervets, baboons, and talapoin monkeys) [290, 495, 600, 609, 610], and prosimians (tree shrew) [771]. These larvae are solid with a

TABLE 45.7. Parasitic cestodes described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Anoplocephalids						
Bertiella studeri	Sm. intestine			X	X	32, 276, 290, 362, 461, 485, 486, 487, 494, 578, 608, 712, 791, 825, 836, 862, 908, 922
Bertiella mucronata	Sm. intestine		X		X	32, 151, 204, 280, 290, 608, 682, 689
Bertiella fallax	Sm. intestine		X			204, 280, 290, 608
Bertiella satyri	Sm. intestine		X	X		113
Bertiella okabei	Sm. intestine			X		765
Bertiella sp.	Sm. intestine			X	X	151, 480, 491, 570, 616, 654, 660, 764
Anaplocephala sp.	Sm. intestine				X	608, 753
Parabertiella sp.	Sm. intestine			X		3, 658, 660, 662
Moniezia rugosa	Sm. intestine		X			204, 280, 290, 461, 608
Thysanotaenia sp.	Sm. intestine	X				608, 918
Tupaiataenia quentini	Sm. intestine	X				771
Intermicapsifer sp.	Sm. intestine			X		539, 608
Atriotaenia megastoma	Sm. intestine	X	X			148, 168, 204, 290, 311, 461, 608, 623
Matheovataenia brasiliensis	Sm. intestine		X			478
Matheovataenia curzsilvai	Sm. intestine			X		582
Matheovataenia sp.	Sm. intestine		X			31, 461, 608
Davaineids						
Rallietina alouattae	Sm. intestine		X			204, 290, 608, 742
Rallietina demerariensis	Sm. intestine		X			32, 204, 290, 482, 841
Rallietina sp.	Sm. intestine	X	X			168, 290, 461, 608, 623
Paratriotaeniids						
Paratriotaenia oedipomidatus	Sm. intestine		X			207, 290, 461, 608, 689, 826
Dilepidids						
Dilepis sp.	Sm. intestine			X		608
Choanotaenia infundibulum	Sm. intestine			X		425

TABLE 45.7. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Hymenolepidids						
Hymenolepis nana	Sm. intestine		X	X	X	38, 276, 290, 349, 461, 584, 608, 742, 764, 791, 808, 880
Hymenolepis diminuta	Sm. intestine	X		X		276, 290, 349, 461, 608, 742, 764, 771, 791, 880
Hymenolepis cebidarum	Sm. intestine		X			204, 290, 623
Hymenolepis sp.	Sm. intestine	X			X	61, 387, 570, 764, 922
Vampirolepis sp.	Sm. intestine		X			461, 608
Mesocestoidids						
Mesocestoides sp. (*Tetrathyridium*)	Larva: peritoneal cavity			X	X	245, 290, 339, 608, 609, 712, 715, 751, 911
Taeniids						
Taenia crocutae (*Cysticercus*)	Larva: skeletal muscle			X		578
Taenia hydatigena (*Cysticercus tenuicollis*)	Larva: liver, peritoneal cavity			X		32, 280, 290, 339, 488, 506, 507, 608, 609, 810
Taenia solium (*Cysticercus cellulosae*)	Larva: brain, heart, muscle, subcutis			X	X	32, 280, 290, 507, 608, 609, 742, 745, 870, 876
Multiceps serialis (*Coenurus serialis*)	Larva: subcutis			X		32, 226, 290, 742
Multiceps brauni (*coenurus*)	Larva: subcutis, pleural and abdominal cavities, brain			X		32, 248, 290
Echinococcus granulosus (Hydatid cyst)	Larva: liver, lungs, peritoneal cavity	X	X	X	X	7, 32, 44, 55, 152, 179, 224, 276, 290, 309, 366, 397, 398, 408, 461, 501, 614, 615, 640, 663, 664, 691, 780, 810, 830
Diphyllobothriids						
Diphyllobothrium erinacei (sparganum)	Larva: subcutis, muscle		X	X		32, 148, 204, 280, 290, 509, 609, 742, 810

(Continued)

TABLE 45.7. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Spriometra reptans (sparganum)	Larva: subcutis		X			204, 207, 290
Spirometra sp. (sparganum)	Larva: abdominal cavity, subcutis, muscle	X		X		61, 290, 495, 578, 600, 610, 771, 911

scolex that contains a pseudosucker. They are white, ribbonlike, and of variable size and motility. They resemble the adult except they lack proglotids and mature genitalia. Spargana can vary from a few millimeters to several centimeters in length [608, 791, 847]. In nonhuman primates, spargana may be found in any part of the body: in retroperitoneal tissues, in abdominal or pleural cavities, or in subcutaneous and muscular tissues. They are commonly encased by a connective tissue capsule, and they do not incite much of an inflammatory response unless they die. These degenerating larvae may cause local inflammation and edema. Most infections in nonhuman primates are usually asymptomatic, and their presence is considered to be an incidental finding at necropsy [608, 791].

Diagnosis can be made in the live animal by radiography, which may reveal calcified nodules. Also, one may palpate mobile nodules in the subcutaneous tissue with localized edema. In the dead animal, diagnosis is made through demonstration and identification of the characteristic spargana larvae either grossly at necropsy or microscopically in histopathologic specimens [608, 791].

Cystricercosis

This condition is the result of infection with the larval form of various members of the family Taeniidae. Adult tapeworms of this family commonly parasitize birds and mammals [461, 608]. Cystericerci have been described in New World monkeys (squirrel monkeys and marmosets) [461], Old World monkeys (rhesus monkeys, baboons, mangabeys, patas monkeys, langurs, and vervets) [280, 290, 339, 608, 609, 742, 870, 876], great apes (gibbons and chimpanzees) [608, 745, 922], and prosimians (lemur) [86, 384, 608]. Cysticerci are oval, translucent cysts that contain a single invaginated scolex with four suckers. In those species that have them, a circle of hooks is present [461, 608, 847]. These cysts may be found in the abdominal or thoracic cavities, muscle, subcutaneous tissue, and central nervous system. Usually there is very little host inflammatory reaction to the presence of viable cysts. As the cysts enlarge, there may be compression of adjacent tissues. Dead cysts will provoke an intense chronic inflammatory reaction [461, 608, 870].

Symptoms in nonhuman primates are directly related to the tissue in which the cysticercus develops and the number present [608]. Involvement of the central nervous system can produce neurological disorders, but this appears to be less of a problem in infected nonhuman primates than in cerebral cysticercosis in the human patient [608, 870, 876].

Diagnosis depends upon the finding of the characteristic bladder-shaped structure in the tissues. Identification of the specific species involved is based on the characteristic hook size and structure [608].

Coenurosis

This condition is the result of infection with the larval form of the tapeworms *Multiceps multiceps* or *Multiceps serialis*, which are intestinal cestodes of dogs and related carnivores [290, 461]. Coenurosis has been reported in Old World monkeys (macaques, vervets, gelada baboon, and other baboons) [133, 226, 248, 290, 489, 501, 608, 700, 735, 742, 757, 860] and prosimians (lemur) [608].

The coenurus is a polycephalid larval form that produces both internal and external daughter cysts. The inner layer of the cyst wall is composed of germinal epithelium from which numerous scolices develop [290, 608, 847].

Coenuri have been described in the subcutaneous tissues, peritoneal cavity, liver, brain, and other organs of affected nonhuman primates [290, 461, 608, 757]. Clinical signs and histopathology depend on the number of coenuri present and their location. In general, infection in nonhuman primates has produced minimal symptoms and lesions [226, 501, 608, 700, 735, 860]. However, in those cases where there is involvement of the central nervous system, typical neurological symptoms are observed [608, 860].

Diagnosis can be made by radiography or the finding of a tumorlike mass in the subcutaneous tissues. Identification of the species of cestode is based on the hook structure of the scolex [290, 608].

Hydatidosis

This disease, also known as echinococcosis, is the result of infection by the larval stage of cestode parasites in the genus *Echinococcus*. The adult tapeworms are found in the intestinal tract of dogs, wolves, bush dogs, other members of the canine family, and related carnivores [290, 461, 608]. Hydatid cysts caused by *E. granulosus* have been described from a number of Old World monkeys (guenons, colobus monkey, mangabeys, mandrills, rhesus monkeys, other macaques, Celebes ape, and baboons) [7, 55, 152, 179, 200, 224, 290, 366, 397, 408, 501, 608, 614, 615, 663, 664, 691, 830], New World monkeys (marmoset) [461], great apes (chimpanzee, gorilla, and orangutan) [44, 309, 640], and prosimians (galago and lemurs) [290, 663, 780]. Recently, hydatid cysts from the tapeworm *E. vogeli* have been reported from a group of young great apes (gorillas, orangutans, and chimpanzees) [398].

Hydatid cysts are large, unilocular cysts. The inner layer of the cyst wall is composed of germinal epithelium from which numerous brood capsules develop. Multiple scolices then develop from the wall of the brood capsule. The cyst wall of *E. granulosa* is characteristically laminated and composed of a thick hyaline material [290, 461, 791, 847].

Hydatid cysts may be located in the abdominal cavity, liver, lungs, subcutis, or throughout the body [152, 179, 200, 290, 408, 461, 608, 615, 691, 791]. The size of the cyst and the amount of involvement and host reaction depends on its age and the location within the host. Abdominal distension or localized subcutaneous swellings are sometimes seen, but usually the presence of the cysts

causes no clinical signs or ill effects and they are found incidentally at necropsy [152, 290, 366, 507, 608, 791, 830].

The gross appearance of the cyst is that of a varying sized, spherical mass, usually in the liver, but it may sometimes be embedded in the lungs or be free in the abdominal cavity. Rupture of pulmonary hydatid cysts and resulting anaphylactic shock have been suggested as the cause of death in several cases of echinococcosis in nonhuman primates [7, 608, 705]. Free scolices from ruptured cysts can implant in other tissues and produce additional cysts [608].

The diagosis of hydatidosis is usually not made until after the cyst reaches considerable size. Symptoms may mimic a neoplasm. Radiographs can be helpful in detecting the presence of pulmonary or calcified hepatic cysts. However, pulmonary changes can be mistaken for tuberculosis or neoplasia. Serological tests such as the Casoni intradermal skin test or tanned cell hemagglutination test are of value in the diagnosis of hydatid disease in nonhuman primates. Specific identification is based on the finding of detached scolices, or daughter cysts, in the cyst fluid. The hook is considered characteristic for the genus. If scolices are not present, the histomorphology of the cyst wall can be used as identifying criteria [152, 290, 430, 608, 791]. Abdominal ultrasonic scanning has been used successfully in diagnosing echinococcosis in gorillas [640].

Tetrathyridiosis

This condition results from infection with the larval stage of cestode parasites in the genus *Mesocestoides*. The adult tapeworms of this genus parasitize various birds and mammals [290, 608, 791]. It has been described in Old World monkeys (rhesus monkeys, guenons, cynomolgus monkeys, and baboons) [245, 290, 339, 608, 609, 712, 715] and great apes (gibbon) [751], but its occurrence in nonhuman primates is considered to be uncommon or even rare [290].

The tetrathyridial larva is flat and has an extremely contractile body. They may be confused with spargana. The anterior end is knotlike and contains an invaginated holdfast apparatus with four suckers. Length can vary from 2 to 70 mm,

TABLE 45.8. Parasitic acanthocephalans described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Moniliformis moniliformis	Sm. intestine				X	29, 290, 461, 742, 770, 791, 909, 922
Prosthenorchis elegans	Ileum, cecum, colon		X			26, 33, 115, 167, 168, 204, 219, 290, 311, 461, 552, 623, 689, 720, 742, 770, 791, 834, 841, 915
Prosthenorchis spirula	Ileum, cecum, colon	X	X			83, 115, 204, 290, 623, 689, 742, 770, 791, 841

depending on the species of cestode and species of host. The tetrathyridum is proglotted shaped in the monkey [290, 339, 608, 791].

These larvae usually are found free in the serous cavities of the body or are found encysted in various tissues. Tetrathyridum evokes little host response and is usually considered to be an incidental finding in nonhuman primates [245, 290, 339, 608, 791].

Diagnosis depends on demonstration and identification of the characteristic larval form in the body cavities or encysted in the host tissues [290, 608, 791].

Larval cestodes in nonhuman primates are of little public health importance to humans because infection can occur only by ingestion of the larval form. Of more importance to both humans and captive nonhuman primates is the possible ingestion of eggs passed by the infected definitive host. For this reason, feces from domestic and feral canids should be handled and disposed of with extreme care. Control of these parasites can only be accomplished through programs aimed at eliminating them from the definitive host [290].

Acanthocephalans

The parasitic acanthocephalans described in the alimentary tract of nonhuman primates are listed in Table 45.8. Those species most frequently encountered in nonhuman primates are discussed below.

Acanthocephaliasis

This disease in nonhuman primates is most frequently the result of infection with acanthocephalan parasites in the genus *Prosthenorchis*. These parasites are distributed throughout Central and South America and have been reported in a variety of New World monkeys. Prosimians, Old World primates, and great apes can become infected under laboratory or captive conditions [115, 204, 290, 742, 770]. The species involved are *P. elegans*, which inhabits the cecum or colon, and *P. spirula*, which favors the terminal ileum [115, 168, 204, 207, 290, 311, 461, 552, 583, 623, 632, 720, 742, 770, 791, 827, 834, 866].

The life cycle is indirect with cockroaches and beetles acting as the intermediate hosts [229, 290, 339, 461, 631, 770, 791].

Diagnosis depends upon identification of the characteristic thick-walled eggs or, more rarely, the worm itself in the feces. Conventional fecal flotation methods are ineffective as a means of demonstrating the eggs of these worms; fecal smears or sedimentation techniques must be used. At necropsy the finding of typical "thorny-headed worms" attached to the intestinal mucosa is considered diagnostic [168, 290, 461, 770, 791].

No distinctive symptoms accompany infection with acanthocephalans. Suspected cases must be confirmed by diagnostic methods. Clinical signs vary depending upon the severity of the infection. Diarrhea, anorexia, debilitation, and death all have been associated with acanthocephaliasis in New World monkeys. In cases of massive infection, there is often cachexia caused by secondary complications and perhaps pain, sometimes of sudden onset; death follows rapidly. Most often the parasite does not contribute directly to the animal's death, but rather produces lesions that allow secondary pathogens to become estab-

TABLE 45.9. Leeches described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Limnatus africana	Nasal cavities			X		125, 290, 506
Dinobdella ferox	Nasal cavities, pharynx			X		93, 276, 290, 295, 494, 696

lished, resulting in debilitation and the ultimate demise of the host [115, 168, 290, 461, 597, 718, 770, 791, 834].

Attachment of the proboscis of these parasites to the intestinal mucosa causes a pronounced, usually severe, granulomatous inflammatory response, and the nodules formed usually can be seen from the serosal surface. The proboscis often penetrates the mucosa and invades the muscular layers of the intestinal wall. If complete penetration of the intestinal wall occurs, a fatal peritonitis results. Adult parasites sometimes are found in the abdominal cavity. Severe infections can cause mechanical blockage of the intestinal tract, intussusception, or rectal prolapse. Under these circumstances, infected animals will be depressed and pass bright red blood and scanty feces [167, 168, 204, 290, 311, 461, 583, 623, 720, 770, 791, 834].

Histologically, a chronic, active inflammatory response is seen, with ulcers of the mucosa and granuloma and abscess formation in the intestinal wall associated with penetration of the proboscis and the resulting destruction of existing tissues. A focal suppurative to fibrinopurulent serositis also may be present in areas where the parasites approach penetration or actually rupture the intestinal wall [83, 290, 461, 770, 791]. A hepatic abscess and granulomatous myositis (diaphragm) associated with migration of an unidentified acanthocephalan has been reported from an adult bushbaby (*Galago crassicaudatus*) [78].

Infection with these parasites has not been reported in humans [290].

Annelida

The species of annelids that parasitize nonhuman primates are listed in Table 45.9.

DINOBDELLAIASIS

The cause of this condition is the leech, *Dinobdella ferox*, which is distributed geographically throughout southern Asia [276, 290, 820]. It is a frequent parasite of the nasal cavities of macaques that range throughout this region of the world.

Dinobdella ferox has been reported from several Old World monkey species (rhesus monkeys and Formosan macaques) [93, 290, 295, 494, 696, 820].

The life cycle of this parasite is direct. Adults are hermaphroditic and eggs are laid in cocoons that are attached to objects at the surface of a pond. After hatching, the immature leeches stay at the water's surface. Infection of the host occurs during drinking; the leech enters the body through the oral or nasal cavities, attaches to the mucosa of the upper respiratory tract, sucks blood for periods that

TABLE 45.10. Fleas described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Ctenocephalides felis	Hair, skin	X		X		281, 290, 609, 814
Tunga penetrans	Skin			X	X	280, 281, 290, 814
Pulex irritans	Hair, skin			X		290, 931
Ctenocephalides canis	Hair, skin			X		281, 814
Echidnophaga gallinacea	Skin	X				281, 814

may last a few days or many weeks, grows and matures, detaches, and drops out through the nostrils. The adult leeches are not parasitic [93, 290, 696].

Infection with a few parasites is usually asymptomatic, but heavy infection is reported to cause restlessness, epistaxis, anemia, weakness, asphyxiation, and sometimes death [290, 696, 820]. Histopathologically, the lesions are composed of a mild, focal, chronic inflammatory infiltrate, and increased mucus production involving the nasopharyngeal mucosa [290, 696, 820]. Diagnosis is based on recognizing and identifying the parasite in its typical anatomical location within the host [290, 696].

This leech presents some public health significance because it does attack humans; however, infection under laboratory conditions is improbable. Nevertheless, precautions should be taken when removing leeches from affected monkeys [290].

Arthropods: Insecta

The parasitic genera of Siphonaptera (fleas), Diptera (flies), and Mallophaga and Anoplura (lice) described from nonhuman primates are listed in Tables 45.10, 45.11, and 45.12, respectively. The most important members of these Orders will be discussed in detail below.

TABLE 45.11. Flies described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Cuterebra sp.	Skin, subcutis		X	X		281, 290, 792
Dermatobia hominis	Skin, subcutis		X			290, 584
Cordylobia anthropophaga	Skin			X		280, 281, 290
Alouattamyia sp.	Skin		X			207
Cochliomyia hominivorax	Skin			X		595

TABLE 45.12. Lice described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Anoplurans						
Pedicinus eurygaster	Hair			X		290, 479, 491
Pedicinus obtusus	Hair			X		290, 479, 491, 609
Pedicinus patas	Hair			X		290, 479
Pedicinus hamadryas	Hair			X		290, 609
Pedicinus mjobergi	Hair			X		290, 682
Pedicinus schaeffi	Hair			X	X	281, 290, 396, 460, 814
Docophthirus acionetus	Hair	X				214, 281, 396, 814
Phthiropediculus propitheci	Hair	X				281, 396, 814
Lemurphthirus galagus	Hair	X				281, 396, 814
Pediculus lobatus pseudohumanus	Hair		X			281, 396, 814
P. l. atelophilus	Hair		X			281, 396, 814
Harrisonia uncinata	Hair		X			281, 396, 814
Gliricola pintoi	Hair		X			281, 396, 814
Pediculus humanus friedenthali	Hair				X	281, 396, 814
Phthirus pubis	Hair				X	281, 396, 814
Phithirus gorillae	Hair				X	281, 396, 814
Pediculus humanus capitis	Hair		X		X	154, 281, 396, 814
Pediculus sp.	Hair		X			207, 736
Sathrax durus	Hair	X				214
Pedicinus longiceps	Hair			X		491
Mallophagans						
Trichodectes armatus	Hair		X			281, 814
Trichodectes colobi	Hair			X		281, 814
Trichodectes mjoebergi	Hair	X				281, 814
Trichodectes semiarmatus	Hair		X			281, 814
Trichodectes sp.	Hair		X			281, 814
Trichophilopterus babakotophilus	Hair	X				218, 814
Tetragynopus aotophilus	Hair		X			281, 814
Trichopilopterus ferrisi	Hair	X				281, 396
Eutrichophilus setosus	Hair			X		277, 281

TABLE 45.12. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Aotiella aotophilus	Skin		X			207, 396
Cebidicola armatus	Skin		X			207, 396
Cebidicola semiarmatus	Skin		X			207, 396

For detailed information regarding the morphology and life cycle of these parasites, the reader is referred to standard pathology or parasitology texts [32, 290, 428, 791, 811].

Flea Infestation

There is a relative paucity of information regarding the extent of flea infestation in nonhuman primates. The available reports concern fleas that, for the most part, are natural parasites of animals other than nonhuman primates (dogs, cats, and chickens). There is no suggestion about the importance of siphonapterids in nonhuman primates or of any potential role they may play in transmission of disease to humans [281].

Tunga penetrans (stick-tight, jigger, or chigoe flea) has been reported from Old World monkeys (guenons and baboons) [280, 281, 290, 791] and great apes (gorilla) [281, 814]. These parasites frequently invade the hard skin covering the ischial callosities where the female *T. penetrans* becomes firmly attached and penetrates into the epidermis that proliferates around the parasite. The implanted female fleas elicit severe irritation and pruritus, and secondary bacterial infections can occur particularly after removal of the parasite from the site of attachment [280, 281, 290, 352, 791].

Dermal Myiasis

The larvae (bots) of several species of flies in the families *Cuterebridae* and *Calliphoridae* are reported to infect nonhuman primates [281, 290].

New World monkeys (howler monkeys) are reported to be a natural host for *Cuterebra* sp. larvae [281, 290, 792]. Infection with these parasites produces dermal cysts or swellings, containing a central pore, primarily in the cervical region. A chronic inflammatory reaction occurs around these sites and a seropurulent exudate containing the dark feces of the larva may exude through the pore. Healing of these lesions is usually rapid after emergence of the larva. Secondary bacterial infections can occur, and these may be more severe than the primary infection [290].

Diagnosis depends on demonstrating and identifying the typical larvae from the characteristic dermal cysts [290].

Although these flies affect humans in the geographical locations where they normally occur, captive nonhuman primates are not considered to be a direct human public health hazard [290].

Pediculosis

Mallophaga

The mallophagans, or biting lice, are reported to be relatively rare on nonhuman primates and apparently are unimportant in regard on zoonoses [281]. Species of biting lice have been reported from prosimians (loris, indri, and mongoose lemur), New World monkeys (woolly spider monkeys, howler monkeys, and owl monkey), and Old World monkeys (colobus monkeys) [281, 814]. There is also a single report of infestation of rhesus monkeys with *Eutrichophilus setosus*, the porcupine-biting louse. These monkeys were housed in close proximity to a cage of porcupines, resulting in cross-infestation [277, 281]. There are no reports of Mallophaga infestations involving the great apes or humans.

Anoplura

Numerous species of anoplurans, or sucking lice, have been reported from a wide variety of nonhuman primates including prosimians (tree shrew, lemurs, and galagos), Old World monkeys (macaques, langurs, green monkeys, guenons, baboons, and colobus monkeys), New World monkeys (sakis, uakaris, howler monkeys, spider monkeys, marmosets, and tamarins), and great apes (gibbons, siamangs, chimpanzees, and gorilla) [234, 281, 396, 441, 814, 904]. There has been at least one report of a black spider monkey (*Ateles paniscus*) being infested with the human head louse. The infection was thought to have been the result of contact with an infected person, indicating that the Anoplura can be shared by humans and the New World monkeys, but not the Old World monkeys [154, 281].

Fiennes [281] regards the sucking lice as interchangeable among humans, the great apes, and the New World monkeys, with the possible exception of the marmosets and tamarins. Old World monkeys are not affected by the species of Anoplura that infect humans, the great apes, and New World monkeys. There are no reports of transmission of rickettsial diseases by lice from the great apes or New World monkeys to humans, or vice versa, though in theory such transmission would seem possible [281].

Arachnida

The parasitic genera of ticks and mites described from nonhuman primates are listed in Tables 45.13 and 45.14, respectively. The most important members will be discussed in detail below.

As for the parasitic insecta, the reader is referred to standard pathology or parasitology texts for detailed information regarding the morphology and life cycle of these parasites [32, 290, 341, 428, 811].

Tick Infestation

According to Fiennes [281], the problem of ticks on captive monkeys is not important because when engorged, the ticks drop off the host, and, under the conditions of captivity, reinfection does not occur [281]. Species of ixodid ticks have been reported from numerous nonhuman primates including prosimians (bushbabies), New World monkeys (spider monkeys), and Old World monkeys (rhesus monkeys, cynomolgus monkeys, baboons, colobus monkeys, bonnet

TABLE 45.13. Ticks described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Ixodids						
Rhipicephalus sanguineus	Skin	X		X		281, 290, 394, 395, 609, 862
Rhipicephalus appendiculatus	Skin	X		X		290, 394, 395, 486, 609
Rhipicephalus puchellus	Skin			X		488
Rhipicephalus haemaphysaloides	Skin			X		850
Rhipicephalus evertsi	Skin			X		395
Rhipicephalus pravus	Skin			X		395
Rhipicephalus simus	Skin	X		X		395
Dermacentor auratus	Skin			X		850
Ixodes ceylonensus	Skin			X		850
Ixodes petauristae	Skin			X		850
Ixodes calvipalpus	Skin			X		281, 395, 814
Ixodes loricatus	Skin		X			144, 207, 281, 814
Ixodes schillingeri	Skin			X		281, 290, 394, 395, 814
Ixodes rasus	Skin			X		395, 488
Ixodes lemuris	Skin	X				395
Ixodes sp.	Skin	X				395
Amblyomma hebraeum	Skin	X		X		290, 394, 395, 578, 609
Amblyomma variegatum	Skin	X				395
Amblyomma sp.	Skin			X		395, 488, 850
Boophilus annulatus	Skin			X		281, 814
Hyalomma truncatum	Skin			X		395
Hyalomma sp.	Skin			X		395
Haemaphysalis wellingtoni	Skin			X		850
Haemaphysalis aculeata	Skin			X		850
Haemaphysalis cuspidata	Skin			X		850
Haemaphysalis kyasanurensis	Skin			X		850
Haemaphysalis minuta	Skin			X		850
Haemaphysalis leachii	Skin	X				395
Haemaphysalis lemuris	Skin	X				395

(Continued)

TABLE 45.13. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Haemaphysalis palmata	Skin			X		281, 395, 814
Haemaphysalis spinigera	Skin			X		290, 849, 850, 862
Haemaphysalis koningsbergi	Skin	X				11
Haemaphysalis hylobatis	Skin				X	11
Haemaphysalis bispinosa	Skin			X		637, 850
Haemaphysalis turturis	Skin			X		850
Haemaphysalis papuanakinneari	Skin			X		850
Haemaphysalis sp.	Skin			X		848
Argasids						
Ornithodorus talaje	Skin			X		144, 281, 290, 814
Argas reflexus	Skin			X		281, 814

macaques, langurs, and green monkeys) [281, 290, 394, 609, 814, 849, 850]. Argasid ticks have been infrequently reported from Old World monkeys (cynomolgus monkeys). In addition, the argasid tick, *Argas reflexus*, normally parasitic on pigeons and other avians, has been reported from an otherwise unidentified monkey [281, 814].

It appears that feral nonhuman primates are parasitized by ticks in most of the geographical areas in which they live and that they are infected by a variety of different species. The importance of ticks as parasites is their world-wide geographic distribution and their role as vectors of a wide variety of diseases, many of which are zoonotic. Since they can infect other animals and contaminate the premises, producing long-term difficulties in parasite control, procedures aimed at eliminating them from newly acquired animals are of primary importance [281, 791].

Most cases of tick infection are asymptomatic; however, heavy parasite loads can result in irritation, restlessness, weight loss, and anemia. Tick bites cause a local inflammatory reaction characterized by hyperemia, edema, and focal hemorrhage. Bite wounds may be involved with secondary bacterial infections [791].

Diagnosis is based on the signs and on the demonstration and identification of the specific species of tick on the host [290].

CUTANEOUS ACARIASIS

Scabies (mange)

The cause of this disease, *Sarcoptes scabiei*, the human itch mite, has been reported from Old World monkeys (cynomolgus monkeys and drills) [22, 290, 293, 305, 523, 891] and the great apes (gorillas, chimpanzees, orangutans,

TABLE 45.14. Mites described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Mesostigmates						
Pneumonyssus simicola	Lungs			X	X	1, 22, 24, 251, 254, 270, 286, 290, 301, 306, 326, 349, 393, 405, 413, 414, 451, 452, 456, 457, 522, 562, 570, 585, 629, 725, 821, 862, 871, 938
Pneumonyssus duttoni	Bronchi, trachea			X		145, 251, 253, 267, 290, 405, 413, 630
Pneumonyssus santos-diasi	Lungs			X		251, 290, 405, 413, 485, 488, 609, 938
Pneumonyssus longus	Lungs, bronchi, trachea			X	X	251, 253, 290, 405, 413
Pneumonyssus oudemansi	Lungs, bronchi, trachea			X	X	56, 249, 251, 253, 290, 405, 413
Pneumonyssus africanus	Bronchi			X		251, 290, 405, 413
Pneumonyssus mossambicencis	Lungs			X		251, 290, 405, 413, 485, 488, 578, 609, 938
Pneumonyssus congoensis	Trachea, lungs			X		233, 251, 290, 405, 413, 488, 609
Pneumonyssus rodhaini	Lungs and nasal fossae			X		246, 405, 413
Pneumonyssus vitzthumi	Lung, bronchi				X	56, 405, 413
Pneumonyssus vocalis	Laryngeal ventricles, vocal pouch			X		573, 577, 578
Pneumonyssus sp.	Lungs			X		405, 413
Rhinophaga dinolti	Nasal cavities, lungs			X		247, 290, 306, 405, 413, 653
Rhinophaga cercopitheci	Lungs, frontal sinuses			X		247, 268, 290, 405, 413, 821
Rhinophaga papionis	Lungs, nasal fossae			X		247, 268, 290, 405, 413, 488, 573, 574, 578, 609, 821
Rhinophaga pongicola	Maxillary sinuses, nasal fossae				X	250, 405, 413
Rhinophaga elongata	Nasal mucosa			X		488, 573, 574, 578
Pneumonyssoides stammeri	Large bronchiole, larynx, nasal cavities, sinuses		X			254, 290, 327, 405, 413, 871

(Continued)

TABLE 45.14. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Rhinophagus sp.	Mucosal and submucosal nasal tissues			X		450
Prostigmates						
Psorergates cercopitheci	Skin			X		27, 290, 517, 710, 793, 938, 939
Psorergates sp.	Skin			X		513, 517, 521
Demodex canis	Skin		X		X	281, 673
Demodex saimiri	Skin		X			520
Demodex sp.	Skin		X			383, 461, 668
Astigmates						
Sarcoptes scabiei	Skin			X	X	22, 281, 290, 293, 305, 334, 523, 675, 706, 718, 742, 864, 890, 891, 931
Sarcoptes pitheci	Skin			X		281, 673, 742
Prosarcoptes pitheci	Skin		X	X		257, 260, 265, 290, 673
Pithesaroptes talapoini	Skin			X		260, 265, 290
Cosarcoptes scanloni	Skin			X		265, 290, 804
Notoedres galagoensis	Skin	X				257, 259, 265, 290
Alouattalages corbeti	Skin		X			263, 290
Fonsecalges saimirii	Skin		X			148, 207, 263, 289, 290
Paracoroptes gordoni	Skin			X		257, 290, 512
Pangorillages pani	Skin				X	256, 290
Listrocarpus cosgrovei	Skin		X			148, 264, 290
Listsrocarpus hapalei	Skin		X			264, 290
Listrocarpus saimirii	Skin		X			264, 290
Listrocarpus lagothrix	Skin		X			264, 290
Rhyncoptes anastosi	Skin		X			261, 290
Rhyncoptes cebi	Skin		X			261, 290
Rhyncoptes cercopitheci	Skin			X		261, 290
Saimirioptes paradoxus	Skin		X			266, 290
Audycoptes greeri	Hair follicles		X			207, 266, 290, 515
Audycoptes lawrenci	Hair follicles		X			207, 266, 290, 515
Lemurnyssus galagoensis	Nasal cavities	X				258, 290

TABLE 45.14. (Continued).

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Mortelmansia brevis	Nasal cavities		X			148, 207, 252, 258, 290
Mortelmansia longis	Nasal cavities		X			148, 207, 252, 258, 290
Mortelmansia duboisi	Nasal cavities		X			148, 207, 258, 290, 492
Dunnalges lambrechti	Skin		X			207, 461, 515, 516
Rosalialges cruciformis	Skin		X			207, 461, 515, 516
Prosarcoptes sp.	Skin			X		804
Pithesarcoptes sp.	Skin			X		804
Kutzerocoptes sp.	Skin			X		517, 804
Trombiculidids						
Trombicula sp.	Skin			X		281, 848

gibbons, and siamiangs) [281, 290, 334, 706, 718, 742, 864, 890, 891]. A closely related species, *Prosarcoptes pitheci*, has been reported in Old World monkeys (African green monkeys and baboons) and New World monkeys in captivity (cebus monkeys) [257, 260, 265, 290, 673]. Two sarcoptiform species, *Dunnalges lambrechti* and *Rosalialges cruciformis*, have been reported from New World monkeys (marmosets and owl monkeys) [461, 515]. There appear to be no reports of *S. scabiei* infection in prosimians [281].

Signs associated with *S. scabiei* infection in nonhuman primates include intense pruritus, anorexia, weakness, weight loss, tremors, and emaciation. Gross lesions include thickening and scaling of the skin and severe alopecia. The severe itching can result in self-mutilation, with secondary hemorrhage and suppurative bacterial dermatitis [290, 334, 523, 791, 864, 891]. Death of a chimpanzee has been ascribed to a severe *S. scabiei* infection [675].

Histopathologically, the infected skin is characterized by hyperkeratosis, parakeratosis, and crusting. The epidermis contains burrows in which many parasites and eggs are seen [290, 305, 334, 742, 791].

The tentative diagnosis of scabies is based on the signs and lesions and confirmed by demonstrating and identifying the parasites and/or eggs in deep skin scrapings [290, 791].

Sarcoptes scabiei infections in nonhuman primates are transmissible to humans by direct contact [290, 334, 742, 891, 931]. Thus, infected nonhuman primates, or those suspected of being infected, should be handled with caution by those responsible for their care and management [290].

PULMONARY ACARIASIS

The cause of this condition is any one of at least ten species of lung mites in the genus *Pneumonyssus* that have been reported from the lower respiratory tract of Old World monkeys (rhesus monkeys, cynomolgus monkeys, pig-tailed macaques, patas monkeys, Celebes black apes, mangabeys, baboons, numerous members of the genus Cercopithecus, colobus monkeys, langurs, and proboscis

monkeys) and the great apes (chimpanzees, gorillas, and orangutans) [24, 56, 233, 246, 247, 249, 251, 253, 270, 290, 326, 349, 405, 413, 456, 457, 585, 629, 630, 666, 742, 938]. Also, there has been one species of lung mite in the genus *Pneumonyssoides* that has been reported from the lungs, larynx, nasal cavities, and sinuses in New World monkeys (woolly monkeys and howler monkeys) [254, 290, 405, 413, 742, 871].

The most commonly encountered member of this genus, *Pneumonyssus simicola*, is found in the lungs of essentially 100% of imported rhesus monkeys [286, 290, 301, 326, 413, 414, 453, 454, 522, 725]. Also, this mite has been seen in the lungs of infant rhesus monkeys allowed to remain with their wild-caught parents after birth [519]. Reports of *P. simicola* infection in other macaque species are less frequent [24, 251, 254, 290, 393, 821]. Despite the common occurrence in feral *M. mulatta*, *P. simicola* has not been seen in laboratory-born monkeys taken from their mothers at birth [290, 464]. A high incidence of lung mite infection with species other than *P. simicola* has been reported from baboons [451, 452, 823]. The complete life cycle of this parasite is unknown [290, 301, 454].

The infection in the rhesus monkey is usually nonsymptomatic, and clinical signs are uncommon [290, 306, 413, 414, 456, 457, 791]. There have been reports of paroxysms of sneezing and coughing, but these may be the result of associated pulmonary disease [290, 375, 413].

Gross lesions are randomly located throughout the pulmonary parenchyma and consist of varying-sized pale spots or yellowish-gray foci that are usually flat or slightly umbilicated on the surface and contain translucent areas. Those located near the surface of the lungs elevate the visceral pleura. The lesions can resemble tubercules but are soft to the touch rather than firm. Adjacent lesions may become confluent. Bullous emphysematous lesions and hemorrhagic lesions may be seen in some cases. Many animals have fine, stringlike fibrous adhesions between visceral and parietal pleural surfaces and between all of the lung lobes. Under the dissecting microscope, the lesions present as pale, white, jellylike masses that have a small opening or slit in the center. These so-called "mite houses" can contain from 1 to 20 mites. The majority of these are females, but sometimes eggs, larvae, and male mites are also present. A characteristic golden-brown to black pigment permeates the lesions and surrounding pulmonary parenchyma [290, 326, 341, 349, 413, 414, 456–458, 503, 791].

Histopathologically, lung mite lesions are characterized by a localized bronchiolitis, peribronchiolitis, focal lobular pneumonitis, alveolar collapse or consolidation, and sometimes bronchiolectasis. There is thickening of the bronchiolar wall, loss of the lining epithelium, hyperplasia of the bronchiolar smooth muscle, and formation of peribronchiolar lymphoid aggregations. A pleocellular inflammatory cell exudate consisting of neutrophils, eosinophils, lymphoplasmacytes, and macrophages infiltrates the affected bronchiolar wall. There is little or no tissue necrosis or giant cell formation [290, 326, 341, 349, 413, 414, 456–458, 503, 791].

Macrophages, whose cytoplasm is laden with a golden-brown to blackish pigment and refractile crystals, are always present in and around the lesions and throughout the lung tissues. This pigment, which is not seen in the lungs of mite-free monkeys, does not contain carbon or melanin but is iron positive and birefringent under polarized light. The exact source of the pigment is not known,

but it is felt that it probably results from the breakdown and excretion of the hosts' blood proteins by the mites [290, 326, 341, 349, 413, 414, 456–458, 503, 639, 791]. The immunological response to pulmonary acariasis has been reviewed by Kim [457].

Lung mite infection has been reported to be associated with pneumothorax [711] and pulmonary arteritis [510, 522, 914] in the rhesus monkey. There is also a report that describes extensive pleuritis and pericarditis associated with ruptured lung mite lesions [455].

Even though several earlier reports ascribe fatalities to *P. simicola* infection, it probably results in death only under conditions of massive infections. Such cases of massive infections and resultant death have been reported in the rhesus monkey, proboscis monkey, "lion macaque," pig-tailed macaque, douc langurs, and chimpanzee [14, 34, 298, 344, 357, 405, 457, 818]. The gross lesions and histopathology of lung mite infection in baboons and chimpanzees are similar to those described for rhesus monkeys [458, 570].

Diagnosis of lung mite infection in live monkeys is difficult. Thoracic x-rays or hematologic studies are of little value [290, 414, 791]. There has been some success in demonstrating lung mite larvae in tracheobronchial washings, but a negative finding is not conclusive proof that infection does not exist. Gross lesions are rather characteristic but must be differentiated from tuberculosis. Tissue sections containing the mites and/or the characteristic pigment and crystals are diagnostic [290, 414, 791]. Lung mites can be found in the feces of infected nonhuman primates [667].

There is no evidence that *P. simicola* infects humans. Therefore, there is no public health significance associated with lung mite infections in nonhuman primates [290].

Nasal Acariasis

Five species of nasal mites of the genus *Rhinophaga* have been described from the upper skull and olfactory mucosa of Old World monkeys (rhesus monkeys, baboons, *Cercopithecus* sp.) and great apes (orangutan) [249, 268, 405, 413, 450, 457, 573, 574, 653]. *Rhinophaga papinois* is found in the maxillary sinuses of the chacma baboon where it causes mucosal polyps [457, 547, 573]. In the lungs it causes pneumonitis and excessive mucus production. *Rhinophaga elongata*, also reported from the chacma baboon, is an extremely long mite that has been described in the apex of small mucosal nodules randomly distributed throughout the nasal cavity. The anterior third of the mite was embedded deeply in the nasal mucosa and in some cases in the adjacent bone. An inflammatory reaction and obstruction of the mucosal glands, which became greatly dilated, was associated with the presence of this mite [457, 573, 574]. *Rhinophaga dinolti* has been reported from the lungs and nasal cavities of the rhesus monkey. Lesions associated with the presence of this parasite in tissues have not been reported [247, 290, 306, 413, 653]. *Rhinophaga cercopitheci* has been reported from the lungs and frontal sinuses of several species of guenons (*Cercopithecus ascanius*, *C. mitis*). Lesions include pneumonitis and excessive mucus production [268, 290, 413, 821]. *Rhinophaga pongicola* has been reported from the maxillary sinuses and nasal fossae of an orangutan [250, 413].

TABLE 45.15. Pentastomids described from nonhuman primates.

Parasite genus-species	Location in host	Pro-simians	New World monkeys	Old World monkeys	Great apes	References
Linguatula serrata	Mesenteric lymphnodes, viscera		X	X		290, 493, 788
Porocephalus clavatus	Peritoneum, viscera		X			148, 290, 461, 623, 689, 750, 785, 787, 791, 800, 815
Porocephalus subulifer	Viscera	X		X		255, 290, 788, 791
Gigliolella brumpti	Mesentery	X				104, 290
Armillifer armillatus	Peritoneal cavity	X	X	X	X	153, 177, 207, 255, 280, 290, 339, 362, 382, 393, 461, 486, 641, 750, 788, 791, 815, 902, 937
Armillifer moniliformis	Viscera, peritoneal cavity			X		45, 262, 290, 788, 791
Porocephalus crotali	Peritoneal cavity		X			177, 207, 815
Nephridiacanthus sp. (juvenile)	Rectal wall			X		486, 487

LARYNGEAL ACARIASIS

A newly recognized mite, *Pneumonyssus vocalis*, has been reported from the mucosa of the laryngeal ventricles and vocal pouch of the chacma baboon where it elicits a mild local inflammatory response [457, 573, 577].

Pentastomids

The parasitic pentastomid nymphs described from nonhuman primates are listed in Table 45.15. They are discussed in detail below.

PENTASTOMIASIS

The parasites that cause this disease are considered to be highly aberrant arthropods [255, 290, 461, 785, 788]. Four genera have been described: *Linguatula*, which has a world-wide distribution; *Porocephalus*, found in both South America and Africa; *Armillifer*, which occurs in Africa, Asia, and Australia; and

TABLE 45.16. Anaplasmatids described from nonhuman primates.

Parasite Genus-species	Location in Host	Pro-simians	New World Monkeys	Old World Monkeys	Great Apes	References
Hemobartonella sp.	Erythrocyte		X			5

Gigliolella, found on the Island of Madagascar. The nymph form occurs in nonhuman primates, which serve as intermediate hosts in the pentastome life cycle [153, 177, 229, 290, 461, 788, 791, 937]. The adult forms of *Linguatula* are found in the nasal passages of dogs, other canids, domestic animals, and humans. Adults of the other two genera are found in the lungs and air sacs of various snakes [290, 461, 788, 791]. Pentastomid nymphs have been reported in a wide variety of nonhuman primates, including prosimians, New and Old World monkeys, and great apes [148, 150, 255, 262, 280, 290, 297, 355, 382, 393, 461, 493, 609, 623, 787, 788, 800, 937].

Infection with this parasite usually is asymptomatic. Dead nymphs act as foreign bodies and invoke an intense inflammatory response in the host. Fatal peritonitis has been reported in overwhelming infections with penetration of the intestinal wall by nymphs. When one infection follows another of considerable duration, there may be a lymphocytic response as a result of presensitization by the initial infection [150, 255, 290, 297, 461, 786, 787, 800, 902].

Diagnosis usually is based on an incidental finding at necropsy and hinges on the identification of the characteristic C-shaped nymph in the tissues [290]. Nymphs have been described in the lungs, liver, omentum, and serosa of the intestinal tract of nonhuman primates [290, 461, 623, 788, 791] but may be found in almost any tissue, including the brain [297, 788].

Pentastomids have been reported in humans in tropical Africa and Asia, but the parasite in captive animals is of no public health significance [290, 692, 749]. Infection of humans can occur only through the ingestion of eggs passed in the feces or in the saliva of the definitive host [290].

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