Parasitic Diseases

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INTRODUCTION

In the past, when nonhuman primates (NHP), including marmosets, were routinely captured in the wild and shipped to research institutions and zoological collections around the world, parasite infections and infestations were common. Over time, however, as wild populations declined and source country attitudes toward these faunal treasures changed, animals used in research were more often bred in captivity. Standard operating procedures, sanitation, and lack of intermediate hosts greatly reduced the incidence of parasite infection and infestation, particularly for indirectly transmitted parasites [1]. Now, except for a few directly transmitted parasites, infection and infestation are uncommon. However, the occasional movement of animals from the wild into captive populations, particularly in zoological collections, means that veterinarians and biologists working with NHP, including marmosets, should remain aware of these organisms and the conditions caused by them.

This chapter describes the parasites found in or on the common marmoset (*Callithrix jacchus*) and closely related species. The parasite taxonomy used in this chapter is based on current usage. Parasitologists sometimes disagree on specific aspects of parasite classification. Thus, the observant reader will occasionally note that the classification schemes used by various authors may differ. To retain consistency within the current literature, the organization of this chapter will largely follow the structure used by Strait et al. [2] and will serve as an aid to allow the reader to quickly find information on a parasite of interest. It is not intended to be the final word on parasite taxonomy or nomenclature.

PROTOZOAN PARASITES

Protozoan parasites have historically been among the most common parasites found in captive marmosets.

For example, older survey reports list the following genera of protozoa identified in marmosets: *Balantidium*, *Eimeria*, *Entamoeba*, *Giardia*, *Isospora*, *Trichomonas*, and *Trypanosoma* [3,4]. Most of these reports were from marmosets that had been captured in the wild and brought into captivity. In this regard, the transition to using captive-bred animals, along with improved housing, sanitation, diagnostics, and treatments, has greatly reduced the prevalence of infection with these organisms.

Flagellates

Marmosets may become infected with flagellated protozoan parasites. For this chapter, discussion has been limited to *Giardia* sp. and *Pentatrichomonas hominis*.

Enteric Flagellates
Giardia sp.

Etiology *Giardia intestinalis* (synonyms *Giardia lamblia*, *Giardia duodenalis*) infects many mammalian hosts, including marmosets. Parasites of the genus *Giardia* are the only parasites known to contain a median body, which appears as a "claw hammer" under light microscopy. The presence of this organelle facilitates identification of the genus. Trophozoites are piriform and bilaterally symmetrical, measure 9–21 μm long by 5–15 μm wide, and bear a sucking disc on the anteroventral side (Fig. 17.1A). The dorsal side is convex. There are two anterior nuclei, two slender axostyles, and four pairs of flagella. The infectious cyst stage is oval in shape. Cysts typically measure 8–12 μm long and 7–10 μm wide [5] (Fig. 17.1B). Following binary fission, cysts contain two daughter trophozoites, thereby doubling the number of internal organelles [2,6].

Clinical Signs As with other animals, infection with *Giardia* sp. is most often asymptomatic and self-limiting
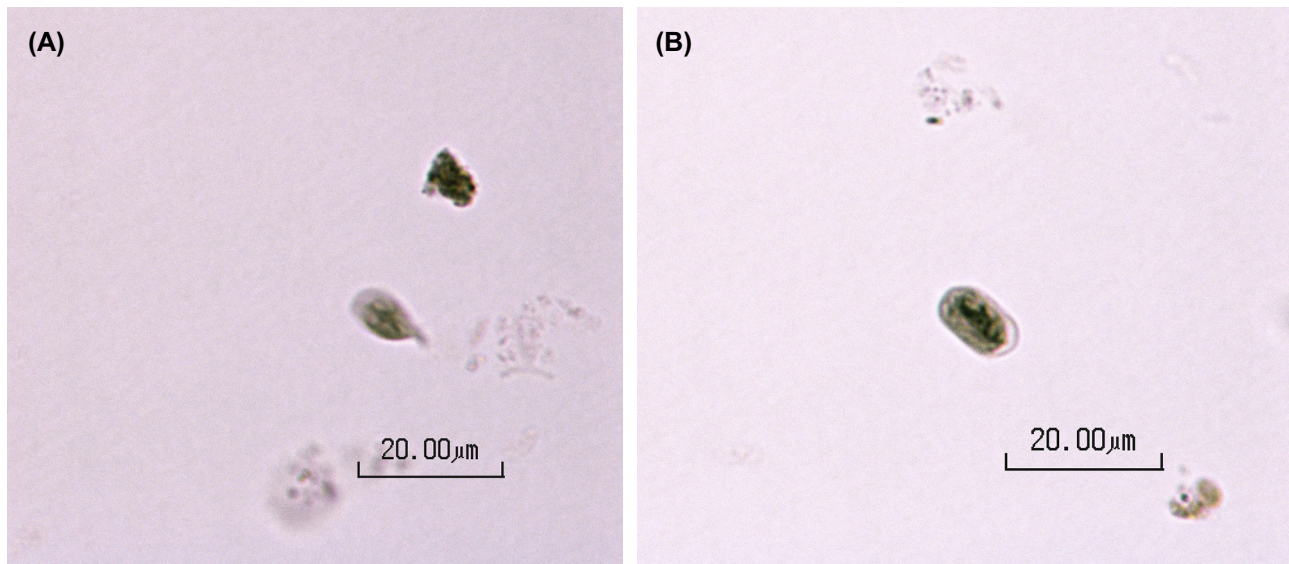
FIGURE 17.1   (A) *Giardia* sp. trophozoite. 600× magnification. (B) *Giardia* sp. cyst. 600× magnification.

in marmosets. However, a chronic carrier state such as that found in some companion animals and humans may be more common than previously recognized [7,8]. Occasionally, clinically affected animals may develop malodorous, fatty stool to watery diarrhea, flatulence, abdominal cramping and distension, anorexia, and weight loss [5,7,9].

Epizootiology   Even in well-managed marmoset colonies, giardiasis remains a challenge [1]. Factors that have been associated with *Giardia* sp. infection include situations where large numbers of animals are assembled, exposure to fouled water sources, and in some reports, age [8]. In one survey, 1 of 25 (4%) captive marmosets greater than 1 year of age, and 5 of 25 (20%) captive marmosets less than 1 year of age, were found to be infected, though none of these animals showed clinical signs of infection. In this case, prevalence rates between groups were not significantly different [5].

Pathology   *Giardia* sp. causes small intestinal malabsorption of fats and sugars through CD8+ lymphocyte-induced shortening of brush border microvilli [10]. Host−parasite ligand−receptor interactions facilitate attachment to intestinal epithelial cells [11].

Diagnosis   Traditionally, diagnosis of *Giardia* sp. infection has been accomplished by light microscopic observation of motile trophozoites or cysts in direct fecal smears by trichrome, fluorescent antibody, or other staining of fecal smears, and more recently, by detection of fecal *Giardia* antigen [2,5] or PCR assay [12]. When traditional methods are employed, multiple fecal samples may be required to diagnose infection due to intermittent shedding [2,5].

Differential Diagnosis   A thorough search for other causes of illness along with careful assessment of clinical presentation and response to treatment should all be considered prior to diagnosing clinical giardiasis in an animal shedding cysts [2]. In one comprehensive survey involving 13,385 captive nonhuman primates from several Old and New World species, not including *C. jacchus*, other potential causes of acute diarrhea included bacteria such as *Shigella* sp., *Salmonella* sp., *Campylobacter* sp., and enteropathogenic *Escherichia coli*. Possible viral causes included rotavirus and coronavirus, whereas parasitic causes included *Entamoeba histolytica* and *Strongyloides* sp. [13].

Prevention and Control   Prevention of infection with *Giardia* sp. is centered in strict adherence to sanitation practices, including protecting feed and water sources from contamination with infectious cysts. *Giardia* sp. infection in marmosets has been successfully eliminated with tinidazole (150 mg/kg PO followed after 4 days by 77 mg/kg PO, each treatment given once) [7]. Other effective treatments utilized in a variety of nonhuman primates include metronidazole (30−50 mg/kg day PO divided tid for 5−10 days), albendazole (25 mg/kg PO bid for 3−5 days), quinacrine (10 mg/kg/day PO divided tid for 5 days), furazolidone (1.5 mg/kg sid PO for 7 days), and others [2,7].

Research Complications   In addition to alterations of the gastrointestinal system associated with the clinical signs of *Giardia* sp. infection, infection of marmosets

with *Giardia* sp. may resemble those reported in other host species and include alteration of the lymphoreticular system. For example, infection with *Giardia* sp. has been shown to reduce mouse immune responsiveness to sheep erythrocytes and increase the severity of concurrent infections with other pathogens in athymic mice [14]. Because *Giardia* sp. remains a potential zoonosis, scientific and animal care staff should exercise strict personal hygiene when working with potentially infected marmosets [7].

Pentatrichomonas hominis

Etiology In addition to *Giardia* sp., common marmosets have been found infected with other enteric flagellates, including *Pentatrichomonas hominis*, in the large intestine [15]. Trophozoites of *P. hominis* measure 8–20 µm long by 3–14 µm wide and bear 3–5 anterior flagella. There is no cyst stage [16].

Clinical Signs *P. hominis* infection is considered asymptomatic. In one study, presence or absence of *P. hominis* in fecal samples was not associated with diarrhea [15].

Epizootiology In one study, 58 of 88 (66%) laboratory-bred marmosets were found to be shedding the trichomonad, *P. hominis*. The presence or absence of *P. hominis* in fecal samples was not associated with the age or sex of the host [15].

Pathology, Diagnosis, Prevention, and Control *P. hominis* is considered a nonpathogenic inhabitant of the large intestine. Trophozoites of *P. hominis* may be observed in fresh smears of both normal and diarrheic fecal samples [16]. Because *P. hominis* is nonpathogenic, efforts to eliminate or prevent infection are not usually undertaken.

Research Complications Research complications have not been identified with *P. hominis* infection.

Hemoflagellates

Common marmosets serve as hosts for at least two species of trypanosomes, *Trypanosoma cruzi* and *T. minasense*.

Trypanosoma cruzi

Etiology *Trypanosoma cruzi*, the cause of Chagas disease in humans, infects captive and free-ranging marmosets in Central and South America [16a,16b]. Two forms of the organism are found in marmosets. Early in the infection, the trypomastigote form is found in the blood. Trypomastigotes measure 16–20 µm long, with a pointed posterior end and a curved body. The kinetoplast is subterminal and larger than in other species infecting marmosets. The trypomastigote bears an undulating membrane and a long flagellum [6]. Later in the infection, the nonmotile amastigote stage is found in pseudocysts in skeletal and cardiac muscle and the reticuloendothelial system. In central nervous system infections, organisms may be found in neuroglial cells [2,6].

Clinical Signs Infection of marmosets with *T. cruzi* is most often asymptomatic and may only be detected at necropsy [16]. When present, clinical signs may include generalized edema, anemia, weight loss, and other nonspecific signs. In addition, hepatosplenomegaly and lymphadenitis may develop. Intrauterine fetal death has also been reported [2,17].

Epizootiology Triatomid bugs in the family Reduviidae serve as intermediate hosts and vectors. Other arthropod hosts capable of being infected experimentally include bed bugs, sheep keds, and soft ticks in the genus *Ornithodoros*. These therefore represent additional potential vectors [6]. However, triatomes are clearly most important in transmitting the organism, either through defecation of parasite-laden feces, with subsequent trypomastigote penetration of mucus membranes or entry into the feeding site, or via ingestion of infected triatomes by susceptible hosts. Infection is common in South and Central America. Because of the presence of suitable arthropod vectors in the extreme southern United States, *T. cruzi* has become established in some captive nonhuman primate colonies in Texas, Louisiana, and Georgia. Several species of wild animals serve as reservoirs of the parasite, making eradication of infection difficult once established in a nonhuman primate colony [16,16a].

Pathology As in human infection, the hallmark of *T. cruzi* infection of marmosets is myocarditis. Myocardial fibers become damaged by pseudocysts containing circular- to oval-shaped organisms, the amastigote stage, 1.5–4 µm in diameter. Amastigotes have a central nucleus and a prominent bar-shaped structure, the kinetoplast. Degenerating pseudocysts elicit the host inflammatory response [2].

Diagnosis Traditionally, infection with *T. cruzi* has been diagnosed by finding trypomastigote forms following Giemsa staining of blood smears or blood cultures [16]. The trypomastigote form of *T. cruzi* is smaller and thus easily distinguished from that of *T. minasense*, a nonpathogenic trypanosome also found in marmosets. In addition to direct microscopic examination of blood smears or blood cultures, infection can be diagnosed using serological tests, including ELISA and older complement fixation assays [2]. More recently, quantitative

PCR assay has been used to detect organisms in blood smears (Minuzzi-Souza et al., 2016).

Differential Diagnosis   A complete discussion of the differential diagnoses for nonspecific clinical signs is beyond the scope of this chapter. Marmosets exhibiting clinical signs of weight loss, anemia, and generalized edema should receive a complete clinical evaluation.

Prevention and Control   Prevention of infection requires exclusion of the arthropod host. There is no effective treatment for *T. cruzi* infection in humans or in NHP, including marmosets. Affected animals should be treated symptomatically. Because Chagas disease occurs in humans, preventing the establishment of *T. cruzi* infection in NHP colonies is critical.

Research Complications   Specific research complications involving marmosets infected with *T. cruzi* have not been reported. However, infected animals would be unsuitable for cardiovascular, lymphoreticular, and perhaps other research uses.

Trypanosoma minasense

Etiology   *Trypanosoma minasense* (synonyms *T. advieri, T. brirnonti, T. devei, T. escomeli, T. florestali, T. manguinhense, T. mycetae*) is a nonpathogenic trypanosome commonly found in free-ranging marmosets in Central and South America [16–18].

Epizootiology   Similar to *T. cruzi*, triatomid bugs in the family Reduviidae serve as intermediate hosts and vectors of *T. minasense*. Natural infection with *T. minasense* has not been reported in laboratory-bred marmosets.

Clinical Signs and Pathology   Infection of marmosets with *T. minasense* is asymptomatic as the parasite is nonpathogenic in marmosets. Infection may represent an incidental finding during examination of thin or thick blood films, either ante- or postmortem.

Diagnosis, Prevention, and Control   Infection with *T. minasense* may be diagnosed by finding trypomastigote forms following Giemsa staining of blood smears or blood cultures [16]. The trypomastigote form is larger than that of *T. cruzi* and measures 29–46 μm long. The kinetoplast is smaller and is located more anteriorly than that of *T. cruzi* [17a]. Prevention of infection requires exclusion of the arthropod host. There is no need or means to effectively treat *T. minasense* infection in marmosets. *T. minasense* is not considered to be zoonotic.

Research Complications   Specific research complications involving marmosets infected with *T. minasense* have not been reported.

Sarcodines: Amoebae

There is scant literature on infections of common marmosets with amoebae. Though rarely reported, it is assumed that, like so many other New World monkeys, marmosets are susceptible to infection with several species of amoebae [16]. Perhaps most important among these is *Entamoeba histolytica*.
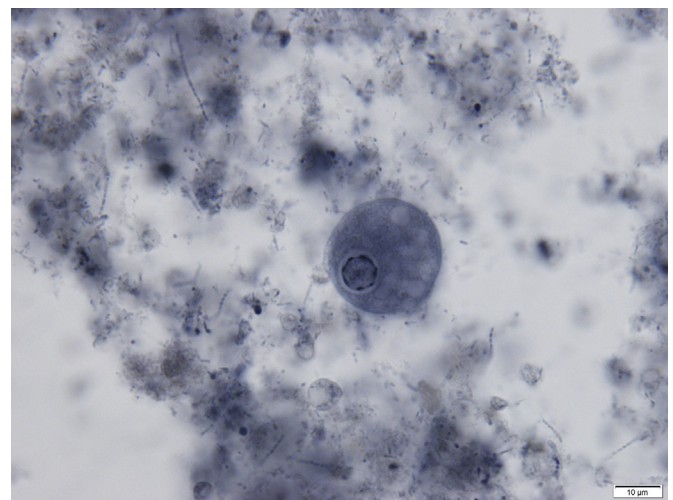
Entamoeba histolytica
Etiology

Entamoeba histolytica may infect virtually all species of NHP [19]. Distribution is worldwide. However, compared to Old World monkeys, New World monkeys are less commonly, if not rarely infected. The trophozoite form of *E. histolytica* measures 20–30 μm in diameter (Fig. 17.2). Trophozoites may be observed to contain phagocytized red blood cells. Cysts form in the large intestine and pass out in the fecal stream. Mature cysts contain four nuclei and measure 10–20 μm in diameter. Nuclear morphology is distinctive for the parasite species and serves as a diagnostic aid [16]. When stained with hematoxylin, *E. histolytica* has a small, central endosome and a ring of small peripheral granules interspersed with chromatin granules [6].

Clinical Signs

Many infections with *E. histolytica* are asymptomatic. When present, clinical signs include lethargy, anorexia,

FIGURE 17.2 *Entamoeba histolytica* precystic trophozoite. Fine chromatin granules can be seen lining the nuclear membrane, giving a beaded appearance. Food vacuoles are present in the cytosol. 630× magnification.

weight loss, weakness, dehydration, vomiting, and severe diarrhea, which may be hemorrhagic or catarrhal [16,20,21].

Epizootiology

Cysts of *E. histolytica* are passed in the feces. Prior to encysting, trophozoites round up and become smaller, eliminate food vacuoles, form a cyst wall, and undergo nuclear division twice, resulting in an organism with four distinct nuclei. Following ingestion of mature cysts by a suitable host, the trophozoite will emerge from the cyst and both the nuclei and cytoplasm will divide, resulting in eight daughter trophozoites [6]. New World monkeys, including marmosets, are less commonly infected with *E. histolytica* than are Old World monkeys. However, when infected, clinical disease is generally worse than in Old World monkeys. Other factors influencing severity of disease include the age and nutritional status of the host, with younger animals being more severely affected. In addition, environmental conditions that increase host animal stress and the intestinal microbiota of the host may play a role in disease severity [6,16,22].

Pathology

Early infection with *E. histolytica* is localized to the large intestinal mucosa. Pathologic findings include necro-ulcerative colitis with characteristic flask-shaped lesions. Multiple small lesions may coalesce. Later, ulcerative extension through all layers of the large intestine may result in peritonitis. Pathogen invasion of the vascular or lymphatic system results in dissemination to other organs, most commonly the regional lymph nodes, liver, lungs, and central nervous system [6,20].

Diagnosis

E. histolytica infection can be diagnosed in direct fecal smears, where one can readily observe the motile trophozoites. Alternatively, fecal films fixed in Schaudinn's fluid [6] can be stained with periodic acid Schiff. Other stains, including trichrome, Giemsa, or iron hematoxylin, can be used to evaluate nuclear morphology to allow differentiation of *E. histolytica* from other, nonpathogenic amoebae [6]. Molecular methods of diagnosis and identification have also been developed, but these are not yet in widespread use [22a].

Differential Diagnosis

A complete analysis of the differential diagnoses for diarrhea is beyond the scope of this chapter. The mere finding of *E. histolytica* cysts in diarrheic feces does not prove causality. Other infectious causes should be considered in the clinically affected marmoset. Perhaps most prominent among them are pathogenic bacteria, or bacterial dysbiosis [6].

Prevention and Control

Prevention of infection with *E. histolytica* is grounded in good sanitation practices and maintenance of water supply systems. Routine chlorination procedures do not kill the cyst form. In addition, facility operators should prevent exposure of marmosets to Old World NHP species, a practice that serves many purposes and is uniformly accepted in well-run animal facilities. Because many people are asymptomatic shedders of *E. histolytica*, regular fecal screening of animal facility employees, with treatment of infected personnel, should be standard operating procedure. Persons working with marmosets should practice excellent personal hygiene to prevent transmission of pathogens between themselves and the animals under their care [6,22]. Newly arrived marmosets should be checked for *E. histolytica* infection several times during the first 2 months. Infected marmosets should be treated with metronidazole, whether or not clinical signs of disease are present [2,20].

Research Complications

Infection of marmosets with *E. histolytica* would interfere with research involving the gastrointestinal system and other forms of research in which normal body condition, cognition, and activity levels were required.

Apicomplexa (Coccidians)

A small number of coccidian species have been reported from common marmosets. These include *Cryptosporidium* sp., *Isospora* sp., and *Plasmodium* sp. [2]. It is likely that other species also infect marmosets but may not have been reported. Species such as *Toxoplasma*, which may inadvertently or experimentally infect marmosets but are not considered primary parasites of marmosets, are not discussed. For this chapter, *Cryptosporidium* sp. and *Plasmodium* sp. are presented. Little is known of the *Isospora* sp. reported from marmosets, but the interested reader is referred elsewhere for an excellent review of the available information [23].

Cryptosporidium *sp.*
Etiology

Cryptosporidium sp. is known to infect nearly all species of mammals, including NHP species. The coccidian parasite inhabits an intracellular yet extracytoplasmic location within host intestinal epithelial cells. The life cycle is direct, with schizogony and gametogony occurring in the host. Sporulated, infective oocysts shed in the feces measure 1.5—6 μm in diameter [6,24].

Clinical Signs

Clinical signs consist of acute, self-limiting to chronic, profuse, watery diarrhea resulting in dehydration,

anorexia, weight loss, depression, hypothermia, and, occasionally, death [5,24]. Fever is uncommon [24].

Epizootiology

Primary factors that have been associated with *Cryptosporidium* sp. infection include immune deficiency or incompetence, young age, length of time spent in the nursery, and social interactions with other monkeys [1,5,24]. In a survey involving both young and adult marmosets, however, prevalence rates between age groups were not significantly different. In that study, 1 of 25 (4%) captive marmosets greater than 1 year of age, and 4 of 25 (16%) captive marmosets less than 1 year of age, were found to be infected, though none of these animals showed clinical signs of infection. The authors postulated that significant differences may have been uncovered with larger samples [5].

Pathology

Histopathologic changes associated with cryptosporidiosis are found in the small intestine and include mild-to-moderate villus blunting with fusion of villi, variations in height of enterocytes, enterocyte vacuolization or necrosis, and increased numbers of mitotic figures in crypts. Mesenteric lymph nodes are enlarged with reactive lymphoid hyperplasia and histiocytosis [25]. Occasionally, organisms have been found in organ extensions of the gastrointestinal system, including the bile duct, intrahepatic and intrapancreatic ducts, and the gall bladder [26].

Diagnosis

Cryptosporidiosis may be diagnosed using acid-fast or modified acid-fast staining of fixed fecal slides, though this method may have a sensitivity of only 50−70% [5,24]. Immunofluorescent staining methods have been demonstrated to be more specific and easier to read than acid-fast staining [5]. Fecal ELISA has also been used successfully [27].

Differential Diagnosis

There are many potential causes of acute, watery diarrhea in marmosets. Finding *Cryptosporidium* sp. in a fecal sample does not prove causality but necessitates a thorough search for other etiologies, including viral and bacterial pathogens. In one comprehensive survey involving 13,385 captive nonhuman primates from several Old and New World species, not including *C. jacchus*, other potential causes of acute diarrhea included viral agents such as rotavirus and coronavirus, bacteria including *Shigella* sp., *Salmonella* sp., *Campylobacter* sp., and enteropathogenic *Escherichia coli*, and parasitic causes including *Entamoeba histolytica* and *Strongyloides* sp. [13,24].

Prevention and Control

Cryptosporidial oocysts are relatively resistant to dessication, as well as to chemical disinfection, and so may survive in the cage environment for extended periods [2]. Historically, infection of marmosets with *Cryptosporidium* sp. has been refractory to treatment. In a recent report, two marmosets appeared to have been cleared of infection with an extended treatment regiment of the aminoglycoside antibiotic paromomycin (15 mg/kg PO bid for 28 days). The authors of that report acknowledged that the cessation of parasite shedding may not have been due to the treatment, but that the marmosets may have simply and coincidentally "self-cured" during the course of treatment [27]. Because *Cryptosporidium* sp. is known to infect humans, scientific and animal care staff should exercise strict personal hygiene when working with potentially infected marmosets.

Research Complications

Infection of marmosets with *Cryptosporidium* sp. would compromise research involving the gastrointestinal and immune systems, as well as any other system requiring normal hydration, appetite, and physiologic homeostasis.

TREMATODE PARASITES

Several species of trematodes have been reported from all types of marmosets. While trematode infections are common among wild marmosets, the lack of intermediate hosts has greatly reduced the prevalence of these infections in captive marmosets due to an inability to complete the indirect life cycles.

Platynosomum sp.

Etiology

Marmosets are susceptible and have been found infected with several members of the genus *Platynosomum*, including *P. amazonensis*, *P. illiciens*, and *P. marmoseti* [28] (Fig. 17.3). It should be noted, however, that the taxonomy of species of *Platynosomum* that infect marmosets is not well established. In a recent study, the authors concluded that *P. amazonensis* and *P. marmoseti* should be synonymized with *P. illiciens* [28]. In one study, eggs of *P. illiciens* measured, on average, 40.1 μm long and 26.6 μm wide [29]. Terrestrial snails serve as first intermediate hosts, whereas terrestrial isopods serve as second intermediate hosts. Small lizards may serve as paratenic hosts [30].

FIGURE 17.3 *Platynosomum* sp. 40× magnification.

Clinical Signs

Light infections are asymptomatic. Clinical signs associated with heavy infection with *Platynosomum* sp. are due to hepatobiliary dysfunction and may include anorexia, weight loss, jaundice, peripheral edema, hepatomegaly, general unthrift, and, occasionally, death [31,32].

Epizootiology

Prevalence of infection of wild marmosets can be high. In captive situations where the intermediate and paratenic hosts are available, thereby facilitating completion of the life cycle, infection rates may remain high [29,33]. Cats also serve as definitive hosts and thus may constitute an important reservoir of infection in some environments [30].

Pathology

Captive black-tufted marmosets (*Callithrix penicillata*) may carry *P. illiciens* in their biliary tree. Infection is associated with mild hepatitis and manifests with hematologic and clinical chemistry perturbations, including eosinophilia, thrombocytopenia, anemia, delayed clotting times, hypoalbuminemia, and elevated serum liver enzymes [31]. Typical findings at necropsy include portal fibrosis, ascending cholangitis, cholestasis, secondary biliary cirrhosis, and thickening of the biliary ducts with traumatic compression of surrounding tissue, resulting in hepatocellular death [31,32].

Diagnosis

Infection with *Platynosomum* sp. can be diagnosed using standard fecal sedimentation procedures. Serial analysis of feces over three consecutive days increases diagnostic sensitivity [33]. Ultrasound examination of the liver may allow for assessment of disease severity and may facilitate diagnosis [31].

Differential Diagnosis

Marmosets displaying clinical signs indicative of hepatobiliary disease should be thoroughly examined for other causes, particularly those associated with protein loss or imbalance.

Prevention and Control

Infection with *Platynosomum* sp. can be prevented with careful exclusion of the intermediate hosts. Infected animals can be cleared of infection with praziquantel, 150 mg/kg PO administered once daily for three consecutive days [31].

Research Complications

Infection of laboratory marmosets with *Platynosomum* sp. would severely compromise research involving the hepatobiliary system and other forms of research requiring normal appetite, behavior, and physiology.

CESTODE PARASITES

Reports of cestode parasitism are surprisingly sparse in the scientific literature and so will not be discussed in detail in this chapter. Adult or larval cestodes found in various species of wild or captive marmosets include *Atriotaenia* sp. (Fig. 17.4), *Bertiella mucronata*, *Raillietina* sp., *Spirometra reptans*, and *Rodentolepis* sp. (Fig. 17.5) [3,4]. It is possible that some cestode species found in other New World monkeys might also infect the common marmoset, but that such infections simply have not yet been reported [2,16].

FIGURE 17.4 *Atriotaenia* sp, anterior end of adult cestode. 20× magnification.

FIGURE 17.5   *Rodentolepis nana* egg. 600× magnification.

NEMATODE PARASITES

Infection with nematodes has historically been common in many species of New World primates. Many, though certainly not all, of these have been found in several varieties of marmosets [2,16]. Those nematode parasitisms that have not been reported in marmosets may not have been so because marmosets are not suitable hosts and thus are not infected, or because infections have simply not yet been reported. For the latter reason, it is likely that the breadth of nematode parasites that may infect marmosets is greater than revealed in the scientific literature. For this reason, the reader should realize that marmosets may be found with parasites that are not on any of the lists of reported marmoset parasitism and so might need to consult the literature on other New World primate species for information regarding specific parasitisms. Furthermore, several nematode superfamilies or species have only infrequently been reported, or have only been reported under somewhat artificial conditions. Uncommonly reported nematode genera parasitizing wild and/or captive marmosets include *Angiostrongylus*, *Enterobius*, *Filariopsis*, *Gongylonema*, *Physaloptera*, *Primasubulura*, *Protospirura*, *Riticularia*, *Subulura*, and *Trypanoxyuris* [2,4,16,34—39]. However, relatively little is known of the biology or clinical effects of these parasites. Thus, uncommon or little-known parasitisms such as these are not covered in this chapter. The parasites presented here are those that are reasonably common, and for which there is an established body of scientific literature.

Superfamily Filarioidea

Several species of filarids have been recovered from marmosets and other New World primates [16,40].

Infection with *Dipetalonema gracile* is among the most common, and possesses many of the features common to filarial diseases in New World primates and thus will be covered in this chapter.
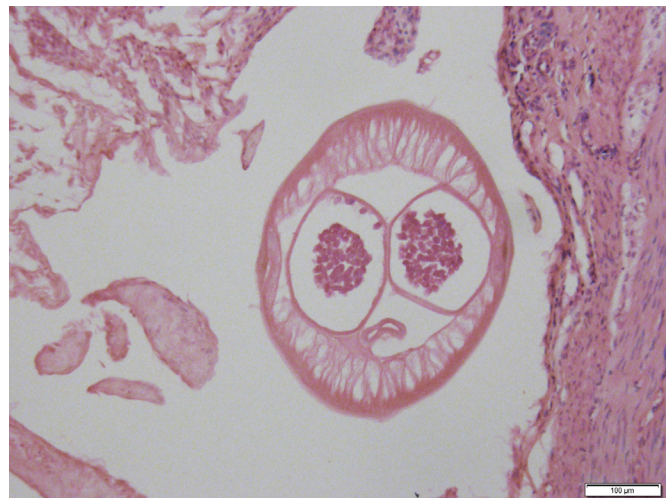
Dipetalonema gracile
Etiology

Dipetalonema gracile is known to infect several species of New World monkeys, including the common marmoset [4,41]. Adult worms inhabit the abdominal cavity, where they are often recovered incidentally at necropsy (Fig. 17.6). Adult worms are white and slender. Adult worms of both sexes may vary greatly in their overall length. Male worms measure 8—19 cm in length, whereas females measure 15—60 cm in length [42,43]. The life cycle requires a blood-sucking arthropod intermediate host, typically gnats of the genus *Culicoides* [44]. Microfilaria are found in the blood.

Epizootiology

Infection with filarial worms is common in wild monkeys regularly exposed to arthropod vectors. The incidence of infection should be lower in captive populations where vector control is practiced. Infection should not occur in laboratory colonies of marmosets.

Clinical Signs and Pathology

Clinical signs of abdominal filariasis are usually lacking. However, the presence of abdominal filarids results in parasitic peritonitis and adhesions. Mesenteric fat may contain multiple, perivascular foci of neutrophils and eosinophils [42]. It is likely that heavily infected monkeys may develop nonspecific symptoms, including malaise, anorexia, and weight loss.

FIGURE 17.6   *Dipetalonema gracile*. Cross-section of adult worm in the abdominal cavity of a common marmoset. 100× magnification.

Diagnosis

Dipetalonemiasis is most commonly diagnosed as an incidental finding at necropsy, when adult worms are found free in the abdominal cavity. However, antemortem diagnosis may be accomplished by finding microfilaria in peripheral blood using standard methods.

Differential Diagnosis

There are many potential causes of general malaise, anorexia, and weight loss in primates, including both infectious and noninfectious diseases, as well as poor husbandry. The primate veterinarian will need to perform a complete clinical workup to exclude causes other than dipetalonemiasis.

Prevention and Control

Prevention of filarial infection relies on vector control. Diethylcarbamazine, given orally at 50 mg/kg for 10 days, has been used to clear both adults and microfilariae of *D. gracile* in squirrel monkeys, and may also be successfully used to treat *D. gracile* in marmosets [45]. While ivermectin would likely be effective against microfilariae and developing larvae, it would likely be ineffective against adult worms [42]. *D. gracile* is not known to be zoonotic and thus poses no direct threat to animal caretakers.

Research Complications

Research complications of abdominal filariasis have not been published. It is expected that animals experiencing general malaise due to abdominal parasitism might not be suitable for research requiring normal appetite, weight, and physiologic balance. The presence of parasitic peritonitis would also complicate research dependent on normal abdominal tissue immune system architecture.

Superfamily Rhabditoidea

Strongyloides cebus

Etiology

Strongyloides cebus is known to infect several species of New World monkeys, including marmosets. Parasitic adult female worms are very small, measuring just 2−5 mm long by 30−80 μm wide [16] (Fig. 17.7). As is the case for other members of the genus, parasitic adult male worms have not been observed. Eggs measure 40−70 μm long by 20−35 μm wide. Eggs are thin-shelled, embryonated when released by the female, and often hatch in the fecal stream, resulting in the finding of first-stage larvae, typical of some other members of the genus [16]. The life cycle is complex and direct. Free-living and parasitic populations exist in
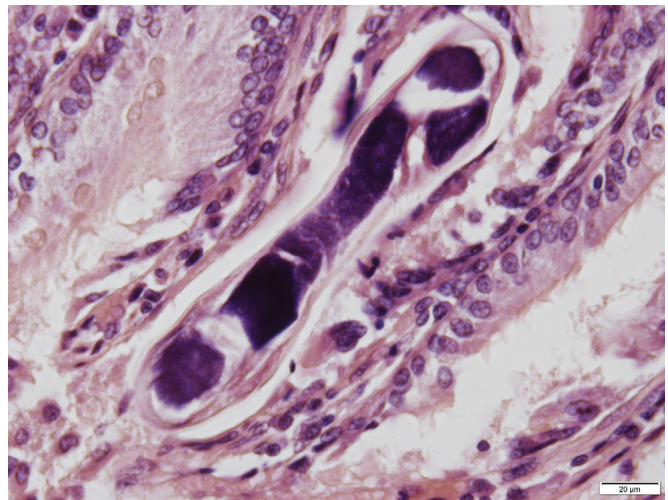
FIGURE 17.7 *Strongyloides cebus*. Longitudinal section of a worm in the intestinal mucosa. 400× magnification.

the same environment. Following infection, larvae migrate via the bloodstream to the lungs, from where they are brought up and swallowed and so arrive in the small intestine where they complete development to parthenogenic adult female worms.

Clinical Signs and Pathology

Infection with *S. cebus* is usually mild or inapparent. However, clinical signs may develop in heavy infections. These, and the accompanying pathologic changes, can be separated into three distinct phases: invasion, migratory, and intestinal [16]. Each phase is characterized by distinct clinical signs due to the specific location and activities of the worm. The invasion phase may be associated with irritation, erythema, and pruritus as larvae penetrate moist skin or oral mucosa. In the migratory phase, larvae are carried to the cardiopulmonary system and are coughed up and swallowed. Coughing may be sporadic or severe. The presence of larvae in the vasculature and lungs can induce bronchopneumonia, pulmonary hemorrhaging, and death. Finally, in the intestinal phase, worms undergo localized histotrophic migration into the intestinal epithelium where they cause acute enteritis with accompanying diarrhea, listlessness, anorexia, weight loss, reduced growth rate, and, occasionally, death [16].

Epizootiology

Strongyloides spp. are soil-dwelling nematodes. Therefore, moist, warm, and dirty conditions, such as exist in unserviced animal enclosures, facilitate the accumulation of infective forms in the environment and thus exposure of a suitable host to large numbers of infective larvae. In addition, larval development while in transit in the host intestine may result in

overwhelming infection ("hyperinfection"). Finally, larvae reaching the infective third stage on arrival at the anus may penetrate the anal or perianal skin, further increasing parasite burden. As for other members of the genus, young monkeys are at greatest risk for patent infection.

Diagnosis

Strongyloidiasis can be diagnosed antemortem by clinical signs and by routine fecal floatation procedures. If a fecal floatation procedure is performed, the technician may find both embryonated eggs and first-stage larvae. Large numbers of very small, translucent adult worms may be found at necropsy in the proximal small intestine [16].

Differential Diagnosis

If only clinical signs are considered, the differential diagnoses for diarrhea are numerous. An aid to diagnosis is found in observing the environmental conditions in which affected animals are housed. Young animals housed in moist, soiled environments are at high risk of infection. Localized damage to the skin, through chronic exposure to moisture and filth, facilitates larval penetration. Thus, the presence of moist, compromised skin on young animals, particularly at points of contact with soiled bedding, is an obvious site to examine for larval activity.

Prevention and Control

Infection with *S. cebus* can be prevented by strict husbandry practices that prevent the accumulation of moist, soiled areas within the animal enclosure. Testing and treatment of new arrivals will help keep environmental contamination to a minimum. Infection can be eliminated with ivermectin (400 μg/kg IM) diluted in sterile propylene glycol. Alternatively, moxidectin (500 μg/kg) can be applied topically [2,16].

Research Complications

Infection of marmosets with *S. cebus* could compromise research involving several body systems. Dermatitis associated with larval skin penetration could compromise skin research, though simply allowing the skin to become chronically moist and erythematous, even without parasite infection, would compromise such research. Cardiopulmonary migration of larvae could compromise research involving the cardiopulmonary and immune systems, whereas intestinal parasitism could compromise research involving this system, as well as any research dependent on normal appetite, activity, and rate of growth.

Superfamily Spiruroidea
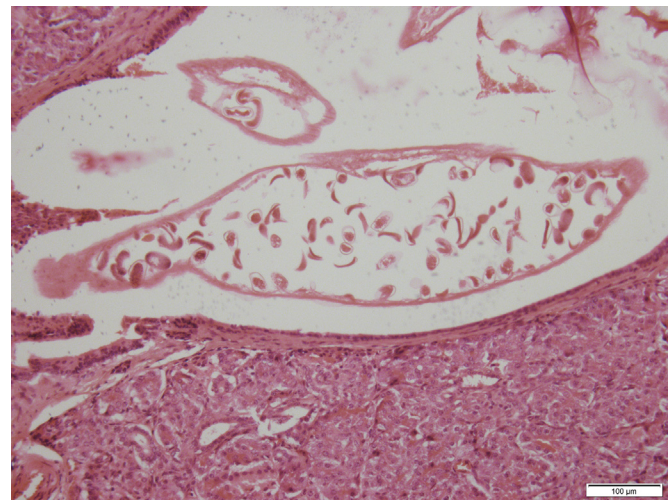
Trichospirura leptostoma
Etiology

Trichospirura leptostoma is a spirurid nematode that parasitizes the pancreatic ducts of several species of New World primates, including wild marmosets in Central and South America, and in captive imported populations [1,2,46,47]. Adult male worms measure 15 mm long, whereas female worms measure up to 120 mm long [2] (Fig. 17.8). The eggs are oval, thick-shelled, and embryonated, typical of the superfamily, and measure 40–50 μm long by 15–20 μm wide [48]. The life cycle is indirect, with cockroaches serving as intermediate hosts [49].

Clinical Signs

Light infections with *T. leptostoma* are asymptomatic. Heavy infections result in signs of acute to chronic pancreatitis, pancreatic insufficiency, secondary malnutrition, emaciation in spite of normal appetite, anemia, and, occasionally, biliary disease. Affected monkeys develop increased fecal volume or diarrhea due to pancreatic inflammation and destruction, and jaundice due to biliary outflow obstruction [2,48,50]. Some have suggested *T. leptostoma* as one of the several possible causes of marmoset wasting syndrome [1,48].

Epizootiology

The incidence of infection with *T. leptostoma* is dependent on availability of the cockroach intermediate host. Colony environments infested with cockroaches are prime locations for high levels of infection. The introduction of an infected animal shedding parasite eggs

FIGURE 17.8 *Trichospirura leptostoma*. Oblique sections of adult worms in the pancreatic duct of a common marmoset. 100× magnification.

in a facility infested with cockroaches will establish the parasite. Monkeys are fond of catching and eating cockroaches, whereas the roaches are attracted to the plentiful food and water universally found in and around primate caging. Infections are long-lived. In one report, marmosets remained infected for at least 11 months in captivity [47].

Pathology

Infection with *T. leptostoma* results in chronic pancreatitis, with eventual replacement of functional pancreatic parenchyma with fibrotic exocrine tissue [46,47,50,51]. Pathologic changes are proportional to worm burden.

Diagnosis

Infection with *T. leptostoma* is often only made during histopathologic examination of the pancreas, and that with some difficulty [51]. Antemortem, embryonated eggs may be found in fecal preparations as early as 8—9 weeks, and for up to 2.5 years, postinfection [49], though the excretion of eggs is irregular in both timing and quantity [48]. Thus, fecal examinations, repeated at intervals of a few days, may be necessary to make the diagnosis [50]. Formalin—ethyl acetate sedimentation is the method of choice for fecal examination [50].

Differential Diagnosis

Differential diagnoses include other causes of pancreatitis and pancreatic insufficiency. In one report, trichospiruriasis was considered to be a cause of wasting marmoset disease, suggesting that the syndrome should be considered among the differential diagnoses [48].

Prevention and Control

Prevention of *T. leptostoma* depends on exclusion of the cockroach vector and treatment of infected animals [50]. Egg shedding has been greatly reduced with fenbendazole (50 mg/kg PO sid for 14 days). It could not be determined that worms had actually been killed, only that egg shedding ceased in most animals [50].

Research Complications

Infection of marmosets with *T. leptostoma* would compromise research involving the pancreatic and possibly the hepatobiliary systems. No publications are available on this topic.

ACANTHOCEPHALAN PARASITES

Marmosets have been found infected with several species of acanthocephalan parasites, including *Prosthenorchis elegans*, *P. lenti*, *P. sigmoides*, *P. spirula*, and others [4]. Based on literature reviews, it appears that among these, *Prosthenorchis elegans* is most commonly encountered and so will be discussed here [16].

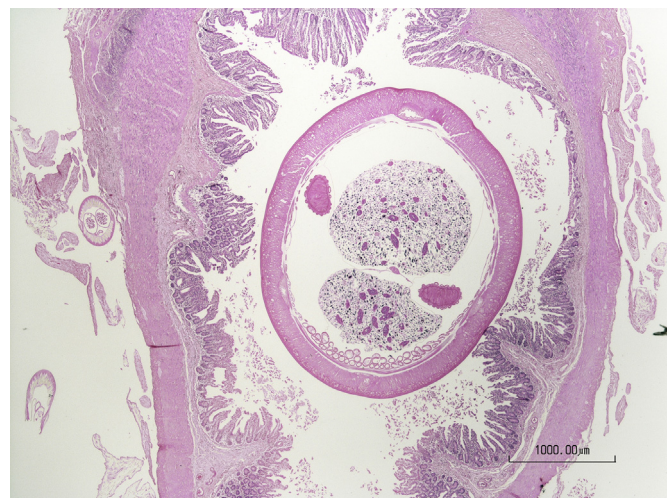## Prosthenorchis elegans

Etiology

Prosthenorchis elegans inhabits the lower intestinal tract of many species of South and Central American primates, including marmosets [4,16,39,52]. Adult male *P. elegans* measure 20—30 mm in length, whereas adult female worms measure 30—50 mm long (Fig. 17.9). The eggs measure 42—53 μm wide and 65—81 μm long [16]. The German cockroach, *Blattella germanica*, serves as an intermediate host. Roaches become infected after ingestion of the parasite egg containing an acanthor larva and passed in the feces of the marmoset definitive host. Marmosets become infected when they ingest roaches harboring the infective cystacanth stage within its coelomic cavity [53].

Clinical Signs

Clinical signs and pathologic lesions are caused by the parasite embedding its spiny proboscis into the intestinal mucosa of the primate definitive host and may be proportional in severity to the worm burden. Clinical signs include watery diarrhea, anorexia, weakness, lethargy, and, if intestinal perforation occurs, death [16,53,54].

Epizootiology

Quite dramatic outbreaks of acanthocephaliasis have been initiated by the entry of infected, egg-shedding animals into a naïve population [53]. Because all acanthocephalans undergo an indirect life cycle, the presence

FIGURE 17.9 *Prosthenorchis elegans*. Cross-section of adult worm in the intestine of a common marmoset. An adult *Dipetalonema gracile* in cross-section can be seen intraperitoneally. 20× magnification.

and availability of the intermediate host is an essential component in establishing and maintaining the parasitic life cycle [52]. While the natural range of the parasite is South and Central America, Old World primates are similarly susceptible to infection and thus should be protected from spread of roach vectors from New World to Old World primate colonies.

Pathology

Lesions caused by *P. elegans* include distal small bowel enteritis, colitis, and typhlitis, and intestinal wall perforation with subsequent peritonitis. Grossly, abscesses and granulomata may be observed at the sites of parasite attachment, and white nodules may be seen on the serosal surface. On histologic section, the worm's proboscis can be observed embedded to the level of the submucosal tissue, with surrounding inflammation, ulceration, necrosis, and red blood cell extravasation. Affected animals may become anemic and develop hypoalbuminemia. Occasionally, intussusception or obstruction of the bowel occurs in the region of the ileocecal junction [16,52,53].

Diagnosis

Diagnosis of *P. elegans* infection is based on finding acanthocephalan eggs in the feces of diarrheic animals or adult worms embedded in the lower bowel mucosa. The latter may be found at necropsy or proctoscopically [2]. The infective cystacanth can also be found in the coelomic cavity of infected roach intermediate hosts, though this is rarely done and is not necessary to make a diagnosis [53]. If a fecal examination is performed, formal–ether sedimentation rather than fecal floatation is the method of choice [2].

Differential Diagnosis

Differential diagnosis of chronic diarrhea would include several possible conditions, both infectious and noninfectious. A complete review of causes of diarrhea is beyond the scope of this chapter.

Prevention and Control

Prevention is based on excluding parasite egg shedders from entering the animal facility. This is accomplished through careful quarantine and screening of new arrivals or, preferably, careful screening prior to shipment. One reported outbreak began with the entry into the animal facility of a single infected gibbon [53]. Control of an outbreak will depend on eliminating the cockroach vector population [53,54]. Anthelmintic treatment of acanthocephaliasis is usually unrewarding [53]. However, some success has been reported with oral carbon tetrachloride (0.5 mg/kg) or fenbendazole (20 mg/kg daily for 7 days) [2].

Research Complications

Infection of marmosets with *P. elegans* would render the animals unusable for nearly all types of research. Studies involving the gastrointestinal system would be particularly affected.

ARTHROPOD PARASITES

Reports of natural or experimental infestation of marmosets with arthropod parasites are surprisingly uncommon. However, as was noted for nematode parasitisms, those caring for and working with marmosets should be watchful for external parasitisms, which have been reported in other species of New World primates, as many of these may also, under proper conditions, infest marmosets. Many external parasites are not highly host specific, though they certainly exhibit host preferences, and thus may infest marmosets as conditions provide opportunity. For example, marmosets may be readily infested with the common dog and cat fleas, *Ctenocephalides canis* and *C. felis*, respectively. While these infestations are unlikely to be found in wild marmosets, it is not difficult to envision conditions in which these fleas could move from domestic animals to captive marmosets, especially if housed in outdoor enclosures. Infestations with *Ctenocephalides* sp. should be prevented and managed using approaches similar to those used for domestic animals. For the purposes of this chapter, general descriptions of infestation will be presented for major groups of external parasites, without, for the most part, direct and specific reference to infestations of marmosets.

Acariasis (Mites and Ticks)

Etiology

Many species of mites and very few species of ticks have been reported from New World primates [2]. Mites found on New World primates are in the suborders Trombidiformes or Sarcoptiformes. The trombidiformes are limited to the genus *Demodex*. In contrast, several genera are represented among the sarcoptiformes, including *Dunnalges*, *Rosalialges*, and *Sarcoptes* (Fig. 17.10) [2].

Clinical Signs and Epizootiology

Clinical signs of mite infestation are greatly influenced by whether the infesting parasite is a burrowing or nonburrowing species. As with other hosts, infestation of marmosets with *Sarcoptes scabiei* may cause severe clinical signs, including intense pruritus, anorexia, weakness, and emaciation [2]. It is likely that those individuals capable of mounting a more robust
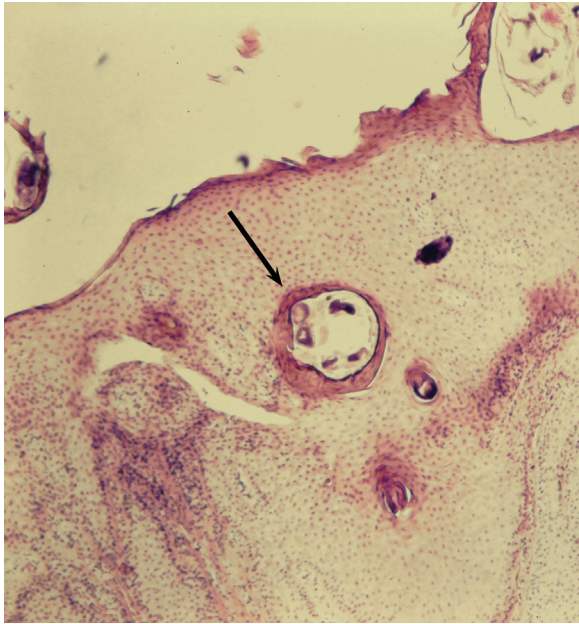
FIGURE 17.10 *Sarcoptes scabiei.* A section of an adult mite (*arrow*) can be seen in the hyperplastic epidermis of a nonhuman primate.

type I hypersensitivity reaction would show the worst signs and harbor the fewest mites, though this has not been reported. In contrast, clinical signs of infestation with nonburrowing species are uncommon. Tick infestations are usually asymptomatic in marmosets and are unlikely to be of concern in captive colonies [2].

Pathology

Mite and tick infestation may lead to dermatologic disease, including hyperkeratosis, anemia, and hypersensitivity [2]. There are no reports of disease transmission to marmosets by mites or ticks.

Diagnosis, Prevention, and Control

Diagnosis of mite or tick infestation is based on visual inspection and the examination of skin scrapings using standard procedures as described for other mammals. Marmosets should be protected from mite and tick infestation through physical barriers to entry and by exclusion of other parasitized primates from close proximity. Infested marmosets can be treated with acaricides commonly used on other veterinary species.

Research Complications

Infestation of marmosets with mites and ticks could compromise research involving the skin and immune system and could compromise research requiring animals exhibiting normal behavior.

Pentastomes

Porocephalus crotali

Etiology

New World primates representing many species, including marmosets, are susceptible to infection with the pentastome *Porocephalus crotali*. Primates, and less commonly other mammals, may serve as intermediate hosts. Adult pentastomes are found in the respiratory tracts of various snakes, which serve as definitive hosts [16]. Parasite eggs containing larvae are passed in the feces of the definitive host. Following ingestion, eggs hatch, releasing larvae, which penetrate the intestinal wall and migrate to several locations within the host, but most commonly to the mesenteric tissues, where they undergo several ecdyses [55], and encyst as the nymphal stage, which is infective to the definitive host.

Clinical Signs, Epizootiology, and Pathology

Clinical signs are usually not observed, though penetration of the intestine by large numbers of larvae may cause fatal peritonitis. Live, encysted nymphs elicit minimal inflammation and so are relatively silent in the intermediate host. In contrast, dead nymphs elicit a strong inflammatory response and can therefore induce nonspecific signs of malaise. Wild-caught monkeys are commonly infected. The replacement of wild-caught, with captive-bred monkeys has virtually eliminated this parasitism from research colonies.

Diagnosis, Prevention, and Control

Diagnosis of *P. crotali* infection is typically made at necropsy or during abdominal surgery. Infections in captive populations are easily prevented by exclusion of the snake definitive host. An effective treatment regimen to eliminate the nymphs has not been reported. However, the common use of ivermectin in captive monkeys may have contributed to the reduction in prevalence of this parasitism as ivermectin has been found to be effective against other pentastomes [56]. While *P. crotali* is a known zoonosis, persons can only become infected by consuming the parasite eggs excreted in the feces of the snake definitive host. Therefore, infected marmosets would not pose a transmission threat to humans.

Research Complications

The presence of nymphal stages in the mesenteric tissues of marmosets is not likely to interfere with most types of research involving monkeys.

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