

### Abstract

Protozoan zoonoses could be defined as “those protozoan diseases which are naturally transmitted between (other) vertebrate animals and man”. Diseases such as toxoplasmosis and cryptosporidiosis are worldwide in occurrence. *Toxoplasma gondii*, *Cryptosporidium parvum* and *Sarcocystis suihominis* are the most significant coccidian parasites affecting animals and man. Immunocompromised persons are always at higher risk of being infected with zoonotic parasites such as *C. parvum*, *T. gondii*, etc. Cryptosporidiosis is an emerging water-borne protozoan disease of public health significance. The parasite *Sarcocystis suihominis* is prevalent in pigs in Asian countries such as India and China. African trypanosomiasis, Chagas disease, leishmaniasis and zoonotic babesiosis are the important vector borne protozoan zoonotic diseases. African trypanosomiasis is still a priority zoonosis for the people in sub-Saharan Africa. The wild rodent *P. leucopus* acts as an important reservoir for *B. microti* human infections. Chagas disease is an important medical and economic concern in Latin America. Leishmaniasis has been reported from more than 80 countries.

## 2.1 African Trypanosomiasis

**Order:** Kinetoplastorida  
**Family:** Trypanosomatidae

name for the disease in humans (human African trypanosomiasis).

### 2.1.1 Common Name/Synonyms

Nagana (meaning powerless/useless) disease (Winkle 2005) is the common name for the disease in animals (African animal trypanosomiasis) and sleeping sickness is the common

### 2.1.2 History

The parasite is present and infecting the people in sub-Saharan Africa since many centuries in the past (Steverding 2008; Cox 2004). *T. brucei* was first discovered by David Bruce (1855–1931, Scottish microbiologist and pathologist) as the cause of cattle trypanosomiasis (cattle nagana) in 1895 (Bruce 1895). After 6 years, trypanosomes were observed in the human blood for the first

time in 1901 (Forde 1902). The parasite species were later identified and proposed as *Trypanosoma gambiense* (now *T. b. gambiense*) (Dutton 1902). The second trypanosome species pathogenic to human beings, *T. rhodesiense* (now *T. b. rhodesiense*), was later recovered in 1910 (Stephens and Fantham 1910).

### 2.1.3 Epidemiology

The disease is particularly endemic in Africa. Human epidemics due to severe sleeping sickness occurred in Africa in the twentieth century (Steverding 2008). *T. brucei gambiense* is present in central and western Africa and *T. brucei rhodesiense* is present in eastern Africa.

### 2.1.4 Etiology

Two subspecies of *T. brucei*, *T. brucei rhodesiense* and *T. brucei gambiense* (Bales 1991; Acha and Szyfres 2006) are responsible for human African trypanosomiasis (sleeping sickness). *T. brucei brucei*, the third subspecies only causes infection in animals. The chronic and epidemic form of sleeping sickness generally occurs due to *T. b. gambiense*, whereas *T. b. rhodesiense* infection is responsible for the acute form of the disease.

### 2.1.5 Reservoir

Human beings act as important reservoirs of *T. b. gambiense*. Wild and domestic animals could also act as important parasite reservoirs for human trypanosomiasis (WHO 2006; Njiokou et al. 2006; Simo et al. 2006; Steverding 2008).

### 2.1.6 Transmission

The bite of an infected tsetse fly belonging to genus *Glossina* transmits the infection from one host to the other (Fig. 2.1).

### 2.1.7 Clinical Signs in Man

The bite of an infected fly leads to eruption of red sores at the region of the bite. Symptoms such as fever, swollen lymph glands, headaches and irritability and aching muscles and joints, could arrive within a few weeks.

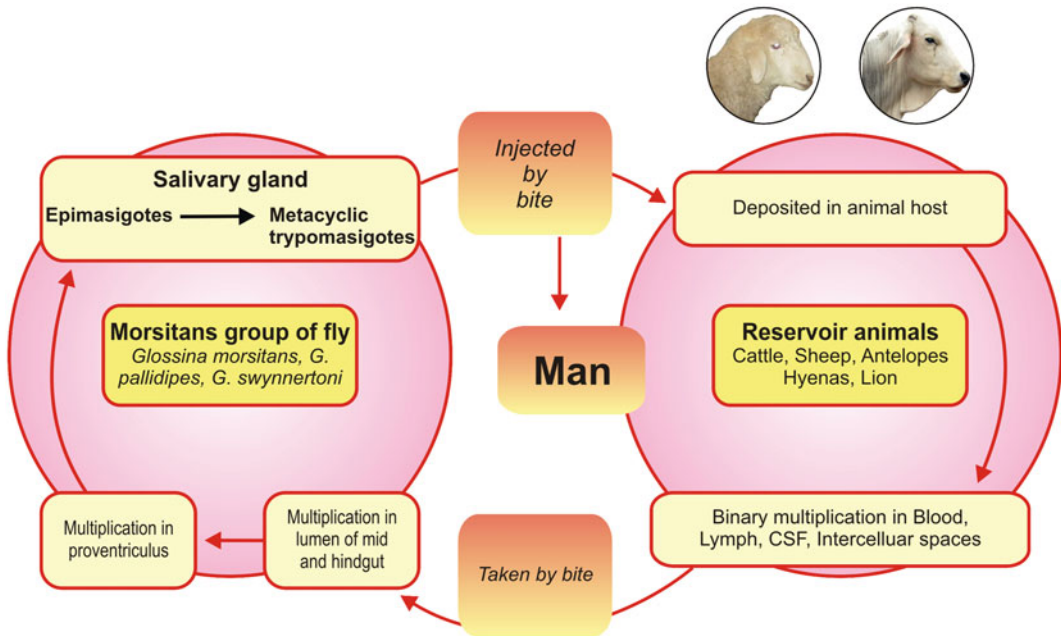
The disease progresses through two distinct stages. During the initial stage or haemolymphatic phase, the parasite infects the blood and lymph system (WHO 2006). The corresponding symptoms include fever, headache, joint pain and itching. In the later stages (neurological phase), the involvement of nervous system leads to symptoms such as changes in personality, alteration of the biological clock (the circadian rhythm), confusion, slurred speech, seizures and difficulty in walking and talking (WHO 2006). This is generally characterised by the presence of trypanosomes in the cerebrospinal fluid (WHO 2006). Death may occur in untreated patients within months (in case of *T. b. rhodesiense* infections) or within years (in case of *T. b. gambiense* infections) depending upon the species involved.

### 2.1.8 Clinical Signs in Animals

Infections due to *T. b. rhodesiense* are generally asymptomatic in domestic (cattle and sheep), wild and laboratory animals (Acha and Szyfres 2006). The second parasite species, *T. b. gambiense* has been occasionally isolated from laboratory animals with no evidence that it causes disease (Acha and Szyfres 2006).

### 2.1.9 Diagnosis

The disease may be diagnosed from clinical signs; demonstration of the parasite from lymph gland and cerebrospinal fluids, bone marrow and blood; mice inoculation and culture in special media such as glucose, lactoalbumin or GLSH. Serological tests such as card agglutination, ELISA, indirect hemagglutination may also be used but they are of limited value.



**Fig. 2.1** Life cycle of *T.b. rhodesiense*

### 2.1.10 Control

The control of the disease includes reduction in the reservoir host and vector population, chemotherapy and chemophylaxis.

## 2.2 Amoebiasis

**Subphylum:** Sarcodina  
**Order:** Amoebozoa  
**Family:** Entamoebidae

### 2.2.1 Common Name/Synonyms

The disease is known as amoebiasis, amoebic dysentery and entamoebiasis.

### 2.2.2 History

The amoebae were first found in faecal samples by Feder Losch in 1875 in Saint Petersburg, but he did not regard them as a cause for dysentery (Pinilla et al. 2008). Later, *E. histolytica* were pointed out

to be a species complex by Emile Brumpt in 1925, comprising two morphologically similar species, *E. dysenteriae* and *E. dispar* found in symptomatic and asymptomatic carriers, respectively (Pinilla et al. 2008). In 1993, Diamond and Clark also agreed to Brumpt's original 1925 hypothesis, with similar conclusions (Pinilla et al. 2008). This hypothesis was accepted by the World Health Organisation in 1997 (Pinilla et al. 2008).

### 2.2.3 Etiology

*E. histolytica* and *E. polecki* are of zoonotic importance. Knowledge regarding *E. dispar* is quite limited. Most of the asymptomatic amoebic infections occur due to *E. dispar*, although *E. histolytica* asymptomatic colonisation is also not uncommon (Braga et al. 1996; Haque et al. 1997; Cantellano and Palomo 2000).

### 2.2.4 Epidemiology

Amoebiasis due to *Entamoeba histolytica* has a worldwide distribution (WHO 1969, WHO 1985).

Most of the infections have been reported in the developing countries of Central and South America, Africa and Asia.

### 2.2.5 Reservoir

Man is an important reservoir of *E. histolytica*.

### 2.2.6 Transmission

Food and water contaminated with faecal matter serve as important sources of infection. Additionally, flies could act as vectors and carry the cysts (Acha and Szyfres 2006).

### 2.2.7 Life Cycle

The life cycle involves trophozoites which are present in host's large intestine and cysts which are shed in the faeces of the host. Humans become infected by ingesting cysts, through contaminated food or water.

### 2.2.8 Clinical Signs in Man

According to WHO, 50 million cases of colitis and liver abscess and 100,000 deaths result annually from infection by this organism (WHO 1995).

In most cases, the infection remains asymptomatic as the parasite lives in the intestine and does not cause disease, or causes mild disease. Symptoms of this form include loose or watery stools, abdominal discomfort and stomach cramps. Sometimes, a severe form amoebic dysentery could develop leading to stomach pain, bloody stools and fever. In rare cases, the parasite reaches the liver and forms abscesses.

### 2.2.9 Clinical Signs in Animals

The disease due to *E. histolytica* could occur in non-human primates. The parasite has also been

reported from dogs, rats, cats, swine and cattle (Levine 1985). *E. polecki* has been commonly reported in swine (Pakandl 1994).

### 2.2.10 Diagnosis

The disease may be diagnosed from direct demonstration of the parasite (trophozoites and cysts) from faeces, or using specific trichrome or iron hematoxylin staining techniques (Fotedar et al. 2007). The advanced diagnostic tests include identification of the parasite based on detection of *E. histolytica*-specific antigen and DNA in stool and other clinical samples. Molecular diagnostic tests, such as conventional and real-time PCR, have also been developed for the detection and differentiation of *E. histolytica* and *E. dispar* in recent years.

### 2.2.11 Control

The control of the disease includes improved personal hygiene and avoiding contamination of food and water with infected faeces.

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## 2.3 Babesiosis

**Class:** Piroplasmida

**Family:** Babesiidae

### 2.3.1 Common Name/Synonyms

The disease is known as babesiosis, piroplasmosis.

### 2.3.2 History

*Babesia divergens* was first described and named *Piroplasma divergens* in 1911 (M'Fadyean and Stockman 1911). Later, the parasite *B. divergens* was identified as the causative agent of the disease in splenectomised humans (Fitzpatrick et al. 1968).

The first human case was reported from Croatia (then Yugoslavia) in 1956. *B. divergens* (cattle parasite) was believed to be the cause for this infection. This parasite is mainly responsible for zoonotic babesiosis in Europe (Zintl et al. 2003). Another authenticated case due to *B. microti* has also been recorded from Europe (Hildebrandt et al. 2007).

### 2.3.3 Etiology

*B. microti* (Gorenflot et al. 1998; Homer et al. 2000; Kjemtrup and Conrad 2000; Telford et al. 1993; Telford and Spielman 1997) and *B. divergens* (Beattie et al. 2002; Herwaldt et al. 1996; Olmeda et al. 1997) are the two important species responsible for zoonotic babesiosis.

### 2.3.4 Epidemiology

In Europe, most cases of human babesiosis are caused by *Babesia divergens* (Genchi 2007). Epidemiological surveys have revealed the presence of *B. divergens* throughout Europe and may extend beyond Europe into North Africa (Zintl et al. 2003). Human babesiosis due to *B. microti* was reported in the USA in 1969 (Western et al. 1970). Thirty-one human cases of *Babesia* infections have been reported from splenectomised individuals in Europe (Gorenflot et al. 1998; Marsaudon et al. 1995; Hunfeld et al. 2002).

### 2.3.5 Reservoir

Infected rodents and animals can serve as important reservoirs for other animals. The wild rodent *P. leucopus* acts as an important reservoir for *B. microti* human infections.

### 2.3.6 Transmission

*Ixodes ricinus* transmits *Babesia divergens* and *I. scapularis* transmits *B. microti* (Hunfeld and Brade 2004). The adult *I. ricinus* female

acquires the infection from the infected host and can transmit transovarially to larvae (Becker et al. 2009; Zintl et al. 2003) (Fig. 2.2).

### 2.3.7 Clinical Signs in Man

In immunocompromised patients, acute illness due to *B. divergens* appears suddenly, with haemoglobinuria as important symptom (Telford et al. 1993). Persistent high fever (40–41 °C), chills, intense sweats, headaches, myalgia and lumbar and abdominal pain are the other important symptoms. Vomiting and diarrhoea may also occur in a few cases (Gorenflot et al. 1998). Haemolysis could lead to jaundice (Hunfeld et al. 2008). Anoxia and toxic waste products could result in further complications such as respiratory, cardiac, renal or hepatic failure.

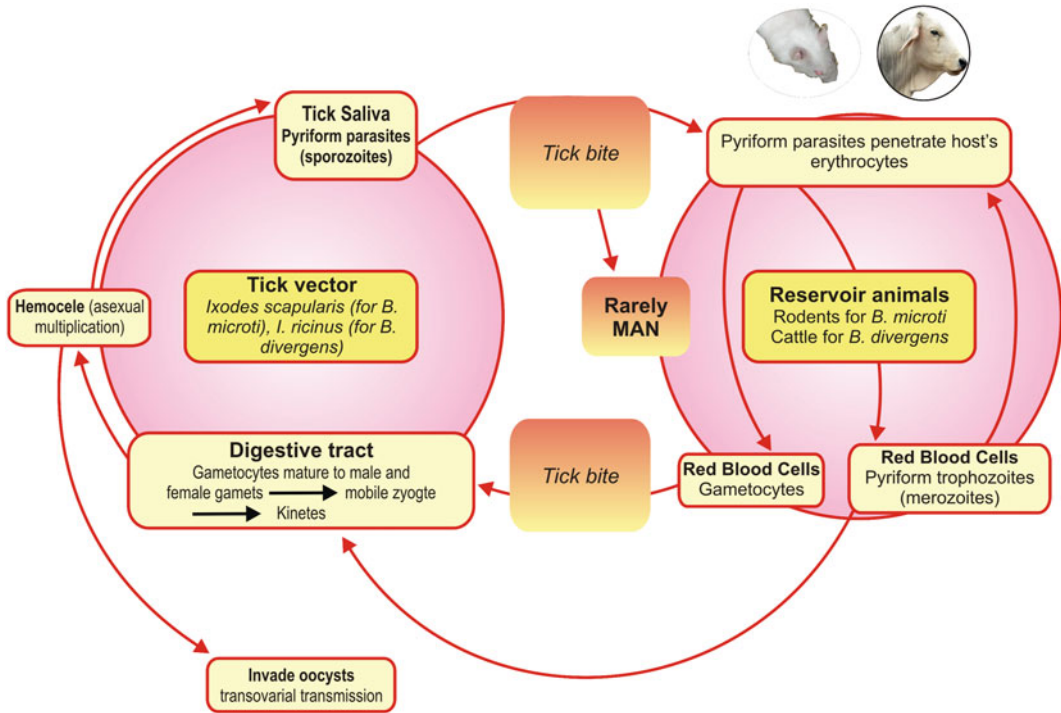
*B. microti* might result in acute and mild infections in immunocompromised and immunocompetent individuals, respectively. Asymptomatic infections are also not uncommon. The reported case of an immunocompromised individual showing symptoms such as fever and chest pain was successfully treated (Hildebrandt et al. 2007). In immunocompromised humans, this parasite may cause medical emergencies due to rapid fulmination and parasitemias that may exceed 70 % (Zintl et al. 2003).

### 2.3.8 Clinical Signs in Animals

In severe cases, there may be fever, anaemia, anorexia, depression and increased respiratory and heart rate. The mucous membranes become pale and may be jaundiced (Christensson 1989; Collins et al. 1970; Gray and Murphy 1985; Sherlock et al. 2000). Erythrocyte destruction leads to haemoglobinuria. Death generally occurs due to cardiac failure, hepatic or renal insufficiency (Collins et al. 1970).

### 2.3.9 Diagnosis

The preliminary diagnosis can be done from clinical signs such as high fever with



**Fig. 2.2** Life cycle of *B. microti* / *B. divergens*

haemoglobinuria. Definitive diagnosis can be done with the detection of the parasite in the erythrocytes. Single round or oval forms are generally seen in both species. Advanced diagnostics include serological tests such as IFAT and PCR analysis followed by sequencing of the PCR product. Inoculation of infected material into splenectomised calves or gerbils is another sensitive method for detection of the parasite (Gray et al. 1989; Joyner and Davies 1967; L'Hostis et al. 1995).

For detecting *B. divergens* in ticks, microscopic examination of Giemsa-stained smears of gut or other tick tissues (Gern and Brossard 1986; Koch 1906), xenodiagnosis and PCR (Duh et al. 2001) are of great diagnostic value.

### 2.3.10 Control

Avoid contact with ticks as the infection occurs through tick bites. Asplenic and immunocompromised individuals are at the greatest risk and

should take extra care particularly when visiting endemic areas.

## 2.4 Balantidiosis

**Phylum:** Ciliophora  
**Order:** Trichostomatorida  
**Family:** Balantidiidae

### 2.4.1 Common Name/Synonyms

Balantidiosis is also known as balantidiasis, balantidial or ciliary dysentery.

### 2.4.2 Etiology

The disease occurs due to *Balantidium coli*. It is the largest ciliate protozoan and can infect swine, primates, man and rarely rats, guinea pigs and dogs.

### 2.4.3 History

Malmsten reported *B. coli* from two patients with severe diarrhoea in 1857 (Kean et al. 1978).

### 2.4.4 Epidemiology

The disease is endemic and prevalent worldwide, mostly prevalent in temperate and tropical regions (Arian and Koppisch 1956). *B. coli* can be found in many primates (Nakauchi 1999).

### 2.4.5 Transmission

Pigs and rat are important sources of infection for human beings (Esteban et al. 1998). Man-to-man transmission could occur due to poor personal/environmental hygiene (Giacometti et al. 1997). Non-human primates could also transmit the infection. Humans generally act as asymptomatic carriers of *B. coli* (Esteban et al. 1998). Contaminated water or food containing cysts serve as an important source of infection for human beings.

### 2.4.6 Life Cycle

The cysts shed in the faeces by infected hosts cause infection in the susceptible host through faecal oral route after ingestion of cyst contaminated water or food.

### 2.4.7 Clinical Signs in Man

Balantidiosis at times may resemble intestinal amoebiasis. The acute form is recognised by rapid onset of diarrhoea or dysentery (Castro et al. 1983) and other signs like abdominal colic, nausea and vomiting. In the chronic form, there are episodes of intermittent diarrhoea alternating with normal bowel movements or constipation. Other signs like headache, anorexia, weight loss or muscular weakness could also be present.

### 2.4.8 Clinical Signs in Animals

The parasite is generally non-pathogenic or causes asymptomatic disease in swine or primates.

### 2.4.9 Diagnosis

The disease may be diagnosed from direct demonstration of the parasite (trophozoites and cysts) from faeces or intestinal scrapings, in wet mounts of fresh faeces. Trophozoites are usually present in diarrhoeic stools and cysts in solid stools.

### 2.4.10 Control

The control of the disease includes avoiding contamination of food and water with infected faeces and treatment of infected cases

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## 2.5 Chagas Disease

**Order:** Kinetoplastorida

**Family:** Trypanosomatidae

### 2.5.1 Common Name/Synonyms

The disease is also known as American trypanosomiasis or Chagas mazza disease.

### 2.5.2 Etiology

American trypanosomiasis/chagas disease is caused by the protozoan *Trypanosoma cruzi*. The parasite multiplication generally occurs inside the heart and smooth muscle cells.

### 2.5.3 Epidemiology

Chagas disease is an important medical and economic concern in Latin America (Miles et al. 2003). According to a survey, more than 10



million people act as carriers of *Trypanosoma cruzi* (Collier et al. 1997).

## 2.5.4 Transmission

Many species of triatomine bug are known (Carcavallo et al. 1998) to carry *T. cruzi*. The parasite is transmitted by many species of triatomine bugs, due to deposition of faeces by infected vector on the mucous membranes or abraded skin (Miles et al. 2003). Triatomine bugs do not fly to hosts to take a blood meal as in the case of tsetse fly vectors of African trypanosomiasis (Fig. 2.3). The infection occurs when some species of triatomine bugs colonise houses in large numbers leading to feeding from humans, domestic mammals and chickens. The chickens are not susceptible to infection, but could serve as an important source of blood meal (Miles et al. 2003).

## 2.5.5 Reservoir

Infected mammals such as armadillo, carnivorous animals (dog and cat) and rodents could serve as reservoirs of infection.

## 2.5.6 Clinical Signs in Man

Chagas disease is an important cause for heart disease in Latin America (Tanowitz et al. 1992, Amorin 1979). As per estimates, approximately 50,000 deaths have been found associated with the infection (Espinosa et al. 1985, Garcia-Zapata and Marsden 1986).

### 2.5.6.1 Acute Clinical Chagas Disease

The disease may remain asymptomatic or present clinical signs. Inflammatory lesion which is also known as a chagoma may develop at the site of entry of the *T. cruzi* (Tanowitz et al. 1992). It is generally followed by fever, myalgia, cephalalgia and unilateral eyelid swelling (Acha and Szyfres 2006).

### 2.5.6.2 Chronic Chagas Cardiomyopathy

Chronic chagas disease may lead to symptoms such as arrhythmias, thromboembolic events (Oliveira et al. 1983) or congestive heart failure (Tanowitz et al. 1992).

## 2.5.7 Clinical Signs in Animals

The disease is generally asymptomatic among wild animals. Symptoms such as fever, palpebral edema, hepatomegaly and alterations in nervous system could occur in dogs (Acha and Szyfres 2006).

## 2.5.8 Diagnosis

The disease may be diagnosed from clinical signs, demonstration of the parasite from blood, serological testing, laboratory animal inoculations and molecular techniques such as PCR.

## 2.5.9 Control

Combined international efforts have led to significant reductions in the adverse impact of chagas disease in the recent years in the Americas (Dias et al. 2002). Use of pesticides such as organochlorines (dieldrin and gamma-BHC) and other synthetic pyrethroids have effectively controlled vector populations (Dias et al. 2002). Successful implementation of control measures for chronic chagas disease have led to the moving of age-specific mortality from the classical 35–55 years to age groups higher than 60 years (Dias 2000).

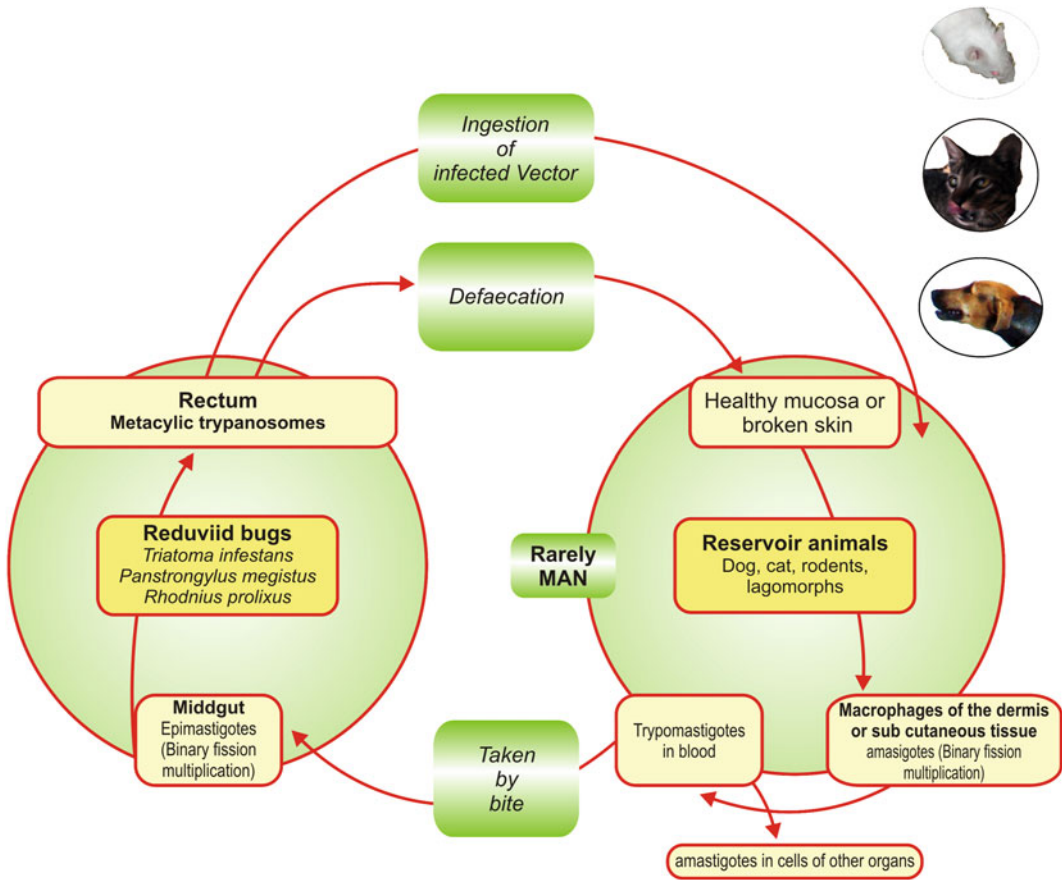
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## 2.6 Cryptosporidiosis

**Subclass:** Coccidea

**Family:** Cryptosporidiidae





**Fig. 2.3** Life cycle of *T. cruzi*

### 2.6.1 Common Name/Synonyms

The disease is also known as *Cryptosporidium* infection.

### 2.6.2 History

*Cryptosporidium* is recognised as an important human parasite since 1976 (Current and Garcia 1991, Fraser et al. 1997). *C. parvum* has been responsible for several water-borne outbreaks (Hayes et al. 1989, MacKenzie et al. 1994). Since 1982, cryptosporidiosis has been increasingly recognised as a cause for severe and life-threatening diarrhoea in AIDS patients (Current 1983).

### 2.6.3 Epidemiology

Cryptosporidiosis is an emerging water-borne protozoan disease of public health significance worldwide. *Cryptosporidium parvum* has the ability to cause gastrointestinal illness in a wide variety of mammals, including humans, cattle, sheep, goat, pig and horses across the globe (Fayer 1997). *C. parvum* has been associated with neonatal calf diarrhoea (Nydam et al. 2001; Singh et al. 2006) and 1- to 3-week-old calves are most susceptible (Leek and Fayer 1984; Singh et al. 2006). The parasite has also been reported in cattle over 2 years of age (Henriksen and Krogh 1985). *C. parvum* is identified as a common cause for diarrhoea in immunocompetent individuals. In immunodeficient individuals,

the parasite may lead to life-threatening chronic diarrhoea. In Acquired Immuno Deficiency Syndrome (AIDS) endemic areas in the developing world, the parasite poses a significant public health problem (Casemore and Wright 1997; Griffiths 1998; O'Donoghue 1995). Infection rates are predicted to be highest in developing countries and in children (Fayer 1997).

### 2.6.4 Transmission

The disease occurs through the faecal oral route when oocysts excreted by infected man/animals contaminate food and water. The contaminated food and water when ingested leads to infection in other susceptible hosts.

### 2.6.5 Reservoir

Neonatal calves are an important source of infection to man.

### 2.6.6 Clinical Signs in Animals

The disease in calves is characterised by weight loss and watery diarrhoea. But it is necessary to distinguish the infection from many other causes of calf diarrhoea. Calves 7- to 21-days old seem to be most susceptible to this infection (Aiello 1998; OIE 2005).

### 2.6.7 Clinical Signs in Man

Clinical signs mainly include watery diarrhoea, stomach cramps and slight fever. Immuno compromised individuals are not able to clear the parasite (Angus 1983). In immunodeficient human beings, *Cryptosporidium* could lead to life-threatening chronic diarrhoea.

### 2.6.8 Diagnosis

The disease can be diagnosed by identifying the oocyst stage of the parasite in host faeces (Garcia et al. 1983; Casemore et al. 1985) or in histological sections taken during necropsy (Current 1985). Detection of the parasite in the faeces can be done by using acid fast or immunofluorescence staining of unconcentrated faecal smears. Appropriate concentration methods could be used when small numbers of oocysts are present. The parasite can be detected in mucosal scrapings of fresh intestine. Concentration techniques such as faecal flotation in sucrose or zinc sulphate solutions could be used. Polymerase chain reaction (PCR) could be used to detect cryptosporidiosis in water supplies or asymptomatic carriers. The World Organisation for Animal Health (OIE) recommends modified Ziehl-Neelson (mZn) acid fast staining and sheather's sucrose floatation method for detection of oocysts from faeces (OIE 2005) (Figs. 2.4, 2.5).

### 2.6.9 Control

Prevention and control measures include use of boiled, filtered or bottled water. Maintain good personal hygiene and wash hands thoroughly after any contact with stools. Persons with weakened immune system must take extra precautions.

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## 2.7 Giardiasis

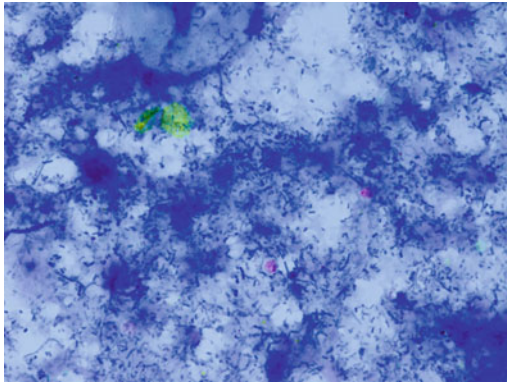
**Class:** Zoomastigophora  
**Order:** Diplomonadida  
**Family:** Diplomonadida

### 2.7.1 Common Name/Synonyms

*Giardia* enteritis



**Fig. 2.4** *Cryptosporidium* oocysts from a Sheather's flotation of faecal samples (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)



**Fig. 2.5** *Cryptosporidium* oocysts from modified Ziehl-Neelson's staining of faecal samples

### 2.7.2 Etiological Agent

*Giardia intestinalis* (syn. *duodenalis* or *lamblia*) is a water-borne pathogen and can infect all mammals. The presence of zoonotic genotypes (assemblages A and B) in animals such as dogs and cattle is of public health significance (Hamnes et al. 2006). Cattle are susceptible to infection with three genotypes of *G. intestinalis*, the zoonotic genotypes of assemblages A and B (Lalle et al. 2005) and the hoofed livestock genotype of assemblage E (Thompson 2004).

### 2.7.3 Epidemiology

The intestinal flagellate *Giardia intestinalis* is globally distributed and commonly infects many animal species and man (Xiao 1994; Kulda and Nohykova 1995). Giardiasis is worldwide and occurs in both developed and developing countries (Fraser et al. 1997; Gilman et al. 1988; Islam et al. 1983; Addiss et al. 1991; Rauch et al. 1990; White et al. 1989; Birkhead and Vogt 1989; Singh et al. 2008).

### 2.7.4 Reservoir

Humans act as the main reservoir of infection for humans. The parasite is prevalent in bovine populations (Buret et al. 1990; Iburg et al. 1996; Olson et al. 1997) which could serve as a source of contamination to water supplies as *Giardia* cyst travels through the environment (Barwick et al. 2003). It has been suggested that the few outbreaks in humans might have resulted due to contamination of drinking water by dairy pasture runoff (Degerli et al. 2005). There is evidence suggesting it to be a zoonosis (Buret et al. 1990). Thus farmers, veterinarians and technicians working in close contact with infected animals may be at risk of contacting the disease. Since infected calves excrete high numbers of *Giardia* cysts, they are considered to be the most important source of contamination for their species (Xiao and Herd 1994; Xiao et al. 1993). Although *Giardia* is more prevalent in calves, adult animals serve as reservoirs of infection (McAllister et al. 2005).

### 2.7.5 Transmission

Infection takes place through faecal oral route. Cysts of *G. intestinalis*, shed by faeces, are ingested orally via water, milk and feed (Goz et al. 2006).

### 2.7.6 Clinical Signs in Man

The disease is generally asymptomatic or sub-clinical (Farthing 1996). The important symptoms include diarrhoea, bloating and abdominal pain. Recurring diarrhoea and flatulence might persist in some patients.

### 2.7.7 Clinical Signs in Animals

The severity of *G. intestinalis* infections varies from asymptomatic to clinical disease (Xiao et al. 1993; Quilez et al. 1996). The symptoms in calves include mucoid and fatty stool, weight loss and growth retardation (Dwight 1999; Xiao and Herd 1994). An infected animal sheds microscopic cysts with the faeces and intermittent cyst shedding can continue for several weeks in calves (Xiao and Herd 1994).

### 2.7.8 Diagnosis

Faecal samples can be examined by wet mount (native lugol) and Wheatley's trichrome (Modification of Gomori Trichrome) staining technique (Garcia 2001; Wheatley 1951). If the number of oocysts in faecal material is fairly low or absent, then sucrose floatation should be followed by staining techniques. For trichrome staining, moderately thick faecal smears should be prepared and immediately fixed in Schaudinn's fixative for a minimum of 30 min. Microscopic examination should be done at magnification(s) of 40X and 100X. Morphometric studies of *Giardia* cysts and trophozoites can also be carried out (Fig. 2.6).

### 2.7.9 Prevention and Control

Avoid contamination of drinking water with faecal matter.



**Fig. 2.6** *Giardia* cysts from a zinc sulphate flotation of faecal samples (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

## 2.8 Leishmaniasis

**Class:** Zoomastigophora

**Family:** Trypanosomatidae

**Genus:** *Leishmania*

Leishmaniasis is a complex group of diseases endemic in many parts of the world. Leishmaniasis could occur due to more than 20 *Leishmania* species. The parasite is transmitted to human beings by many species of phlebotomine sand flies (Chappuis et al. 2007; Herwaldt 1999; Pearson and Sousa 1996).

Leishmaniasis has been reported from 88 countries. Ninety percent of visceral leishmaniasis cases have been reported from five countries: India, Bangladesh, Nepal, Sudan and Brazil (NICD 2006). Some workers believe Indian kala-azar to be anthroponotic due to absence of animal reservoirs.

Leishmaniasis occurs in four clinical forms: Cutaneous leishmaniasis; muco-cutaneous leishmaniasis; visceral leishmaniasis and post-kala-azar dermal leishmaniasis.

## 2.8.1 Visceral Leishmaniasis

### 2.8.1.1 Common Name/Synonyms

Black fever, Kala-azar, dum dum fever, post-kala-azar dermal leishmaniasis.

### 2.8.1.2 Etiology

The disease occurs due to *Leishmania donovani* complex—*L. donovani* sensu stricto in Indian subcontinent, East Africa and the *L. infantum* in North Africa, Europe and Latin America (Lukes 2007; Chappuis et al. 2007; Mauricio et al. 2000). The disease occurs due to *L. chagasi* in the American continent. *L. donovani* causes anthroponotic, whereas *L. infantum* is responsible for zoonotic visceral leishmaniasis. The disease could become fatal if not properly treated.

### 2.8.1.3 Reservoir

Dogs principally act as reservoir hosts for spread of zoonotic visceral leishmaniasis (VL) which mainly occurs due to transmission of *L. Infantum* (Chappuis et al. 2007; Alvar et al. 2004).

### 2.8.1.4 Transmission

The transmission of the parasite occurs due to bite of sand flies belonging to either *Phlebotomus* or *Lutzomyia* spp.

### 2.8.1.5 Epidemiology

The parasite *L. infantum* mostly infects children and immunosuppressed individuals, whereas *L. donovani* could infect all age groups (Chappuis et al. 2007). Estimated 500,000 new cases and more than 50,000 deaths occur due to visceral leishmaniasis every year (Chappuis et al. 2007; Desjeux 2004a, b).

From a total of approximately 500,000 visceral leishmaniasis cases occurring annually across the globe, more than 23,000 cases are reported from India. The present foci of the disease in India are Bihar, West Bengal, Uttar

Pradesh and Jharkhand (Sutherst 1993). Sporadic cases have also been reported from Gujarat (west India) (Munshi et al. 1972), Tamil Nadu and Kerala (south India) and sub-Himalayan parts of north India including Uttar Pradesh, Himachal Pradesh and Jammu and Kashmir (Kesavan et al. 2003). It was a common belief that western India has become a VL free zone, but recent studies (Sharma et al. 2007) have reported that the population of Gujarat state is again at risk of kala-azar after about 20 years.

The disease is a serious problem in Bihar, West Bengal and eastern Uttar Pradesh states of India where there is under-reporting of kala-azar and post-kala-azar dermal leishmaniasis in women and children 0–9 years of age (Bora 1999). The parasite and the disease were first reported in India in the 1820s in undivided Bengal. The disease almost disappeared in the 1950s but resurgence was reported in the 1970s in north Bihar (NICD 2006). Recent outbreaks of VL in India and the epidemic of human immunodeficiency virus (HIV) makes VL a re-emerging problem in India. Outbreaks and epidemics have been associated with climate change, urban development, deforestation and human migration. The prevalence of HIV seropositivity in VL patients was found to be 5.7 % (6/104) at a tertiary care centre in northern India (Mathur et al. 2006). Four of the six (67 %) VL/HIV co-infected patients had a chronic/relapsing course, not responding to antileishmanial treatment.

The first indigenous case of visceral leishmaniasis in a 7-year-old girl from central India (Dey et al. 2007) indicated that more than one genetic variant of *L. donovani* is circulating in different parts of India. The co-existence of malaria and kala-azar poses difficulties in differential diagnosis and results in lower reporting of the disease. Examination of *Leishmania*-stained blood smears of 450 asymptomatic healthy individuals residing in an endemic village in Bihar (Sharma et al. 2000), showed the presence of *Leishmania* amastigotes in six persons (1.3 %).



### 2.8.1.6 Clinical Signs in Man

The incubation period of the disease is between 2 and 6 months. The symptoms mainly result due to systemic infections. Symptoms include loss of appetite, fever, fatigue, weakness and weight loss. Due to invasion of the parasite in the blood and reticuloendothelial system, enlargement of lymph nodes, spleen and liver could occur. The feet, hands, abdomen and skin may take a grey hue particularly in Indian patients.

### 2.8.1.7 Clinical Signs in Animals

The disease can occur in dogs and could cause cutaneous or systemic lesions. Cutaneous symptoms include alopecia and inflammation leading to formation of nodules, scabs and ulcers. Systemic symptoms include fever, anaemia, splenomegaly and lymphadenopathy.

### 2.8.1.8 Diagnosis

- Microscopic examination of blood, lymph nodes, bone marrow or spleen aspirates for the identification of amastigote form of the parasite.
- Serological tests.
- Molecular techniques such as Polymerase chain reaction.

### 2.8.1.9 Control

The control measures for visceral leishmaniasis include control of the disease in reservoir and vector, use of insecticide impregnated materials and active detection and treatment of cases (Chappuis et al. 2007; Boelaert 2000; Davies et al. 2003).

## 2.8.2 Cutaneous Leishmaniasis

### 2.8.2.1 Common Name/Synonyms

Delhi sore, Oriental sore, Baghdad ulcer, Uta, Buba.

### 2.8.2.2 Etiology

Cutaneous Leishmaniasis can occur due to many *Leishmania* species and sand flies transmit the infection to human beings and animals (Reithinger et al. 2007). Most *Leishmania* species could cause cutaneous leishmaniasis in human beings.

*L. braziliensis* complex, *L. mexicana* complex, *L. lainsoni*, *L. naiffi* and *L. lindenbergi* cause the disease in the new world. *L. tropica* complex (*L. tropica*, *L. major* and *L. aethiopica*) species cause the disease in old world. Some strains of *L. infantum* can also cause cutaneous leishmaniasis. With the exception of *L. tropica*, all the other organisms are zoonotic in nature.

### 2.8.2.3 Epidemiology

Cutaneous leishmaniasis endemic in the tropics and neotropics (Reithinger et al. 2007). Cutaneous leishmaniasis is endemic in more than 70 countries worldwide. Ninety percent of cases occur in countries such as Afghanistan, Pakistan, Saudi Arabia, Algeria, Brazil, Peru and Syria (Desjeux 2004a, b). The disease is spreading to new areas as a result of urbanisation and deforestation (Desjeux 2001). In India, initially the disease was reported from hot dry northwestern region and was endemic in western Thar desert of Rajasthan (NICD 2006). Sporadic cases were also reported from Punjab, Delhi, Haryana and Gujarat (Shahi 1941; Lysenko 1971). New foci of infection have been reported from different parts of India (Bora et al. 1996; Sharma et al. 2003). Kerala remained free from cutaneous leishmaniasis (CL) since 1988, many cases have been recorded afterwards (Bora et al. 1996; Kumaresan and Kumar 2007). This is an important public health issue in view of a newly recognised reservoir area of CL in South India.

### 2.8.2.4 Reservoir

Many domestic animals could serve as potential reservoirs (Davies et al. 2000; Reithinger et al. 2003). Both dogs and rodents serve as the zoonotic reservoirs for cutaneous leishmaniasis in

the Thar desert (Ahuja et al. 2006). In dogs, an incidence of 6.8 and 6.12 % was recorded during 1985 (Nirban 1985) and 1999 (Chhangani 1993), respectively, in Bikaner. Natural *Leishmania* infections are found in a variety of non-human mammal hosts (e.g. rodents, edentates, marsupials and carnivores). So far, only a few reservoir hosts for the main *Leishmania* spp (i.e. *L. infantum*, *L. amazonensis*, *L. peruviana*, *L. mexicana*, *L. panamensis*, *L. guyanensis*, *L. major* and *L. aethiopica*) have been reported (Davies et al. 2000; Ashford 1996).

### 2.8.2.5 Transmission

The transmission of the parasite occurs due to the bite of sand flies belonging to either *Phlebotomus* (in Asia, Europe, North Africa and middle east) or *Lutzomyia* species (from southern USA to northern Argentina) (Killick-Kendrick 1999) (Fig. 2.7).

### 2.8.2.6 Clinical Signs in Man

Several *Leishmania* spp. can cause cutaneous leishmaniasis in human beings, although most infections generally remain asymptomatic (Murray et al. 2005). Initially, a small erythema develops after the bite of an infected sand fly.

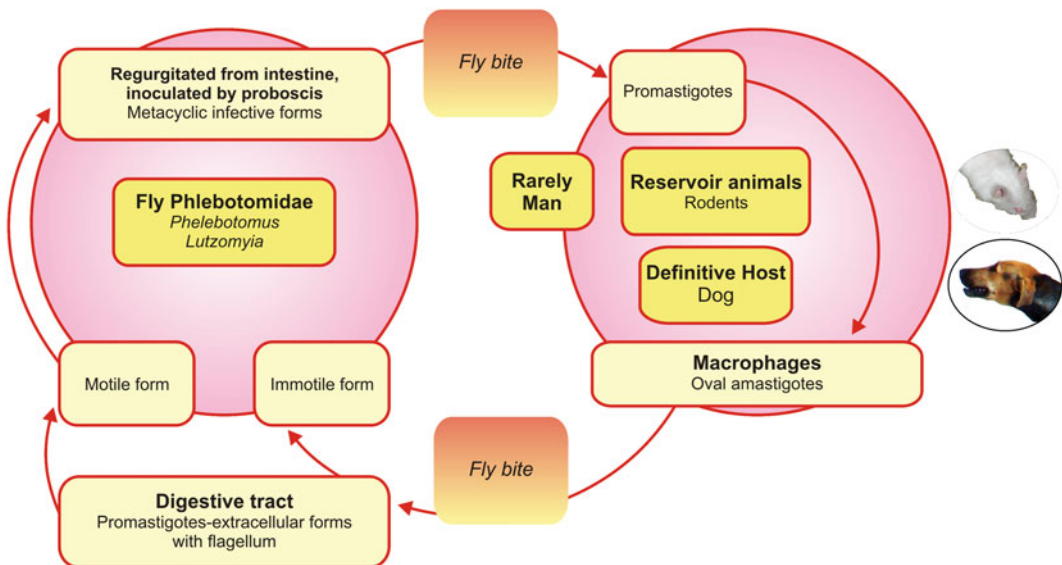
The erythema further develops into a papule, then a nodule which ulcerates over a period of 2 weeks to 6 months and becomes the lesion characteristic of localised cutaneous leishmaniasis (Peters and Killick-Kendrick 1987). In disseminated cutaneous leishmaniasis, non-ulcerative nodules disseminate from the initial site of infection and could cover the entire human body (Peters and Killick-Kendrick 1987). Mucosal involvement could also occur in *L. braziliensis* infections and could lead to life-threatening mucosal leishmaniasis in some patients (Reithinger et al. 2007).

### 2.8.2.7 Clinical Signs in Animals

The disease could occur in dogs which can show both cutaneous and visceral manifestations (Acha and Szyfres 2006).

### 2.8.2.8 Diagnosis

- Microscopic examination in the lesions for identification of amastigote form of the parasite.
- Isolation of the organism by culture in suitable media.
- Immunological tests.



**Fig. 2.7** Life cycle of *Leishmania* spp. causing cutaneous leishmaniasis



### 2.8.2.9 Control

The control measures for cutaneous leishmaniasis include control of infection in reservoir and vector and the use of insecticides.

## 2.9 Sarcocystosis

**Subclass:** Coccidia  
**Order:** Eucoccidiorida  
**Family:** Sarcocystidae

### 2.9.1 Common Name/Synonyms

Sarcosporidiosis

### 2.9.2 Etiological Agent

Sarcocystosis is a protozoan disease of almost all species of domestic animals caused by *Sarcocystis* sp. Cattle and pig act as the main sources of human disease. Three species of *Sarcocystis* have been recognised in cattle, of these, *S. cruzi* has the dog as its definitive host, *S. hirsuta* the cat and *S. hominis* the man. Pig harbours three species of *Sarcocystis* namely, *S. miescheriana*, *S. porcifelis*, and *S. suihominis* with their definitive hosts as dog, cat and man, respectively. Sarcocysts have been commonly found in retailed foods and can remain viable for several days under refrigeration conditions.

### 2.9.3 Life Cycle

Human beings act as definitive host for *S. hominis*, *S. suihominis* and get infected by eating raw or undercooked beef and pork, respectively. Meat with visible sarcocysts is unacceptable to the consumer and the condemnation of carcasses leads to considerable losses to the meat industry. In intermediate hosts, signs are evident at the time when vascular endothelium is parasitised by schizonts. Later, the schizonts disappear within about a month and lead to formation of cysts in the muscles (Fig. 2.8).

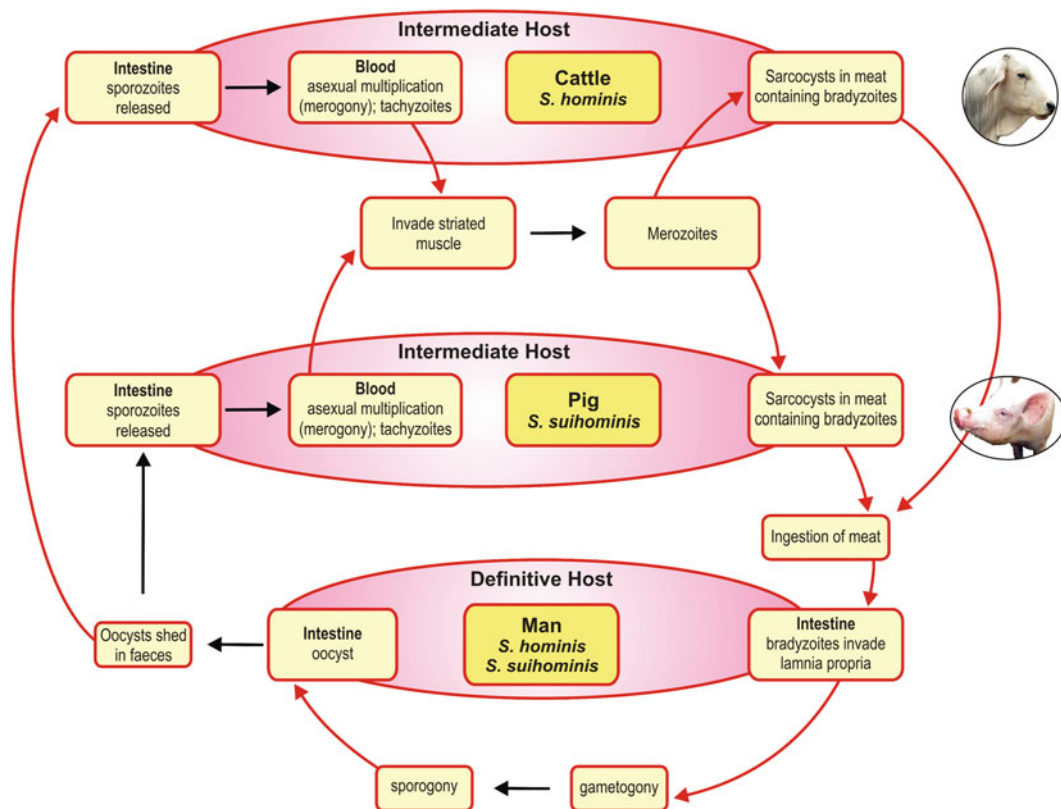
### 2.9.4 Epidemiology

Previous studies indicate high incidence of sarcocystosis all over the world (Carvalho 1993, Dubey et al. 1989, Foggin 1980; Shi and Zhao 1987; Stalheim et al. 1980; Singh et al. 2003, 2004; Avapal et al. 2002, 2004; Juyal et al. 1982; Juyal 1991; Juyal and Bhatia 1989). Studies using conventional techniques have reported the presence of *S. suihominis* in India (Banerjee et al. 1994; Bhatia 1991). *Sarcocystis* species has been reported from many countries. In India, *S. cruzi* has been reported from Uttranchal, Madhya Pradesh, Bihar and Assam, whereas *S. hominis* has been reported from Uttar Pradesh and Madhya Pradesh states only (Devi et al. 1998; Jain and Shah 1988; Pandit et al. 1994; Saleque and Bhatia 1991; Singh 2001; 2002). *Sarcocystis* species mainly infect skeletal and cardiac muscles. *Sarcocystis* species has been found in the muscles of oesophagus, thigh, cardiac, diaphragm, eye, tongue and tail.

### 2.9.5 Clinical Signs in Animals

The disease is also of economic importance, as it causes both direct and indirect economic losses. Direct losses arise through losses incurred in detaining or condemning carcasses and indirect losses arise from delayed growth and production losses on the farm. In heavy infections of intermediate host (cattle), important symptoms include anorexia, pyrexia (42 °C or more), anaemia, cachexia, enlarged palpable lymph nodes, excessive salivation, nervousness, lameness and hair loss on the extremities.

Hair loss is noticed especially at the end of the tail and complete loss of the switch gives the animals a “rat tail” appearance. The cystic stage of sarcocystosis is virtually non-pathogenic. Pathological changes observed at necropsy include eosinophilic myositis (Jensen et al. 1986), generalised lymphadenopathy, erosions and ulcerations in the oral cavity and oesophagus and severe laminitis. Histologically, there is lymphadenitis, myositis, myocarditis and



**Fig. 2.8** Life cycle of *S. hominis* and *S. suis hominis*

glomerulonephritis. Schizonts of *Sarcocystis* species may be found in the endothelial cells of blood vessels in many organs. Abortions may occur in breeding stocks.

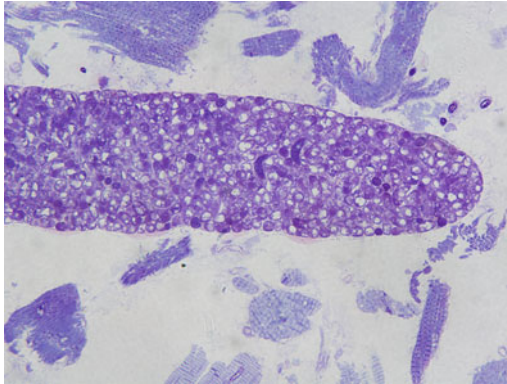
### 2.9.6 Clinical Signs in Man

Human sarcocystosis is reviewed under two sections: Intestinal sarcocystosis, where man acts as a definitive host and muscular sarcocystosis, where man acts as an intermediate host for various *Sarcocystis* species. Intestinal sarcocystosis is mainly characterised by abdominal pain and diarrhoea. The muscular form of the disease is characterised by pain, swelling, stiffness and displacement of myofibrils in the affected muscles.

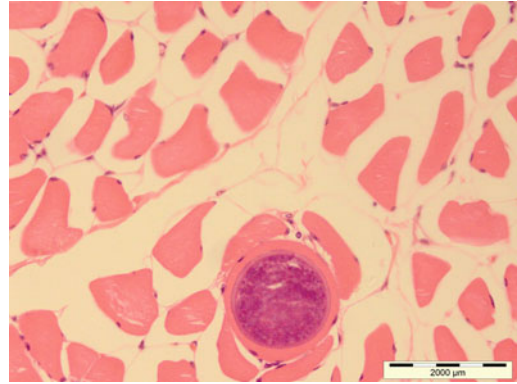
### 2.9.7 Diagnosis

Samples of the animal's muscle tissues like thigh, cardiac, jaw, tail, diaphragm, neck, eye muscles, oesophagus and brain should be collected in polythene bags containing ice at 4 °C. They should be first examined grossly and then subjected to pepsin digestion method (Jacob et al. 1960) after removing fat and connective tissue. The diagnosis from muscles can be done using the following methods: (Figs. 2.9, 2.10, 2.11)

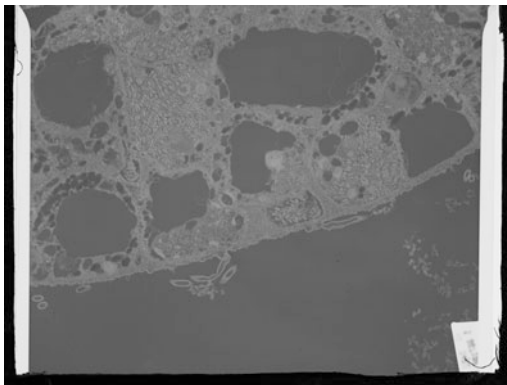
- Isolation of intact microsarcocysts from muscles (Juyal et al. 1989).
- Pepsin digestion technique.
- Histopathological examination (Luna 1968).
- Serological tests like ELISA, etc.
- Electron microscopic studies (Mehlhorn et al. 1976).



**Fig. 2.9** Transmission electron microscopy of *S. cruzi*, thin walled *S. cruzi* showing zoites in the granulomata and hair-like projections on the cyst wall (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)



**Fig. 2.11** Thick-walled *Sarcocystis* cyst in diaphragmatic muscles of pig (H & E stain, 40X)



**Fig. 2.10** Ultrastructure of *Sarcocyst* wall showing villar projections (VP) arising from the *S. cruzi* cyst wall. No microtubules are seen in VP and a zone of ground substance (GS) can also be seen (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

**Control:** Avoid consumption of raw or undercooked pork or beef.

## 2.10 Toxoplasmosis

**Subclass:** Coccidia  
**Order:** Eucoccidiorida  
**Family:** Sarcocystidae

### 2.10.1 Synonyms

Congenital toxoplasmosis.

### 2.10.2 Etiology

Toxoplasmosis is a coccidial disease which occurs due to the parasite *Toxoplasma gondii*.

### 2.10.3 Reservoir

*T. gondii* is a protozoan parasite of flies with many intermediate hosts, e.g. sheep, goat, pig, cattle, etc., including man (Dubey and Beattie 1988).

### 2.10.4 Epidemiology

Toxoplasmosis in man and sheep is prevalent worldwide including India (Dubey and Beattie 1988; Dubey 1987; Chhabra and Mahajan 1982). In the first national serological prevalence of *T. gondii* in India (Dhumne et al. 2007), 23,094 serum samples were tested for *T. gondii* IgG and IgM antibodies using a solid-phase immunocapture ELISA. Antibodies IgG and IgM were found in 24.3 and 2 % samples, respectively. The lowest seroprevalences were reported from the northern

parts of the country, with the highest in the south. The data probably indicate the effects of significantly drier conditions and their related negative impact on the survivability of *T. gondii* oocysts in northern India. It has also been observed that the seroprevalence of *T. gondii* in humans in India is low compared to that of western countries (Dubey and Beattie 1988). Many other workers have also reviewed human toxoplasmosis in India (Parija 2004; Dubey 1987).

### 2.10.5 Transmission

Human beings can become infected either after eating raw or undercooked contaminated meat infected with cysts of the parasite or from cat faeces infected with *T. gondii* oocysts. The infection can further be transmitted from an infected mother to the foetus through the placenta. Although rare, organ transplant from infected persons could also lead to infection. Transplacental transmission may also take place. Persons with weakened immune systems are at high risk of being infected. Cat acts as the definitive host and shed oocysts in the faeces. The sporulated oocysts can be ingested by man, animals and mice (Fig. 2.12).

### 2.10.6 Clinical Signs in Man

In healthy persons, the disease is generally asymptomatic. The symptoms generally appear 7–14 days after the exposure. The parasite can infect many vital organs such as brain, eye, liver, lung and heart. Symptoms generally include headache, fever, enlarged lymph nodes, particularly of the neck and sore throat, and muscle pain. In congenital toxoplasmosis, eye infection could lead to blindness. Congenital toxoplasmosis could lead to mental retardation, multiple organ failure, hydrocephalus or foetal death (Hohlfeld et al. 1989). *Toxoplasmic* encephalitis (TE) is the most frequent cause for focal nervous system disorder complicating AIDS. There have been various reports of TE from India (Chaddha

et al. 1999). Although *Toxoplasma* infection does not cause repeated foetal losses, this is the most common indication for investigation of toxoplasmosis in India.

### 2.10.7 Clinical Signs in Animals

The parasite remains asymptomatic most of the time. The disease is particularly important in sheep and goat where it may lead to abortions in pregnant animals. Congenital infections can also occur with young ones having problems in muscle coordination.

### 2.10.8 Diagnosis

In intermediate hosts, the disease can be diagnosed by molecular and serological tests, mice inoculation, histological and other tests such as MRI, CT scan, etc. Giemsa-stained microscopic examination of impression smears can also be done. In cat (definitive host), faecal examination using flotation techniques or serological tests can be performed (Figs. 2.13, 2.14).

### 2.10.9 Control

For prevention of the disease, consume properly cooked meat. Persons involved in slaughtering should handle raw meat carefully and wash their hands properly. Children should avoid access to cat faeces, particularly in playgrounds and parks. Pregnant women should not handle cat litter.

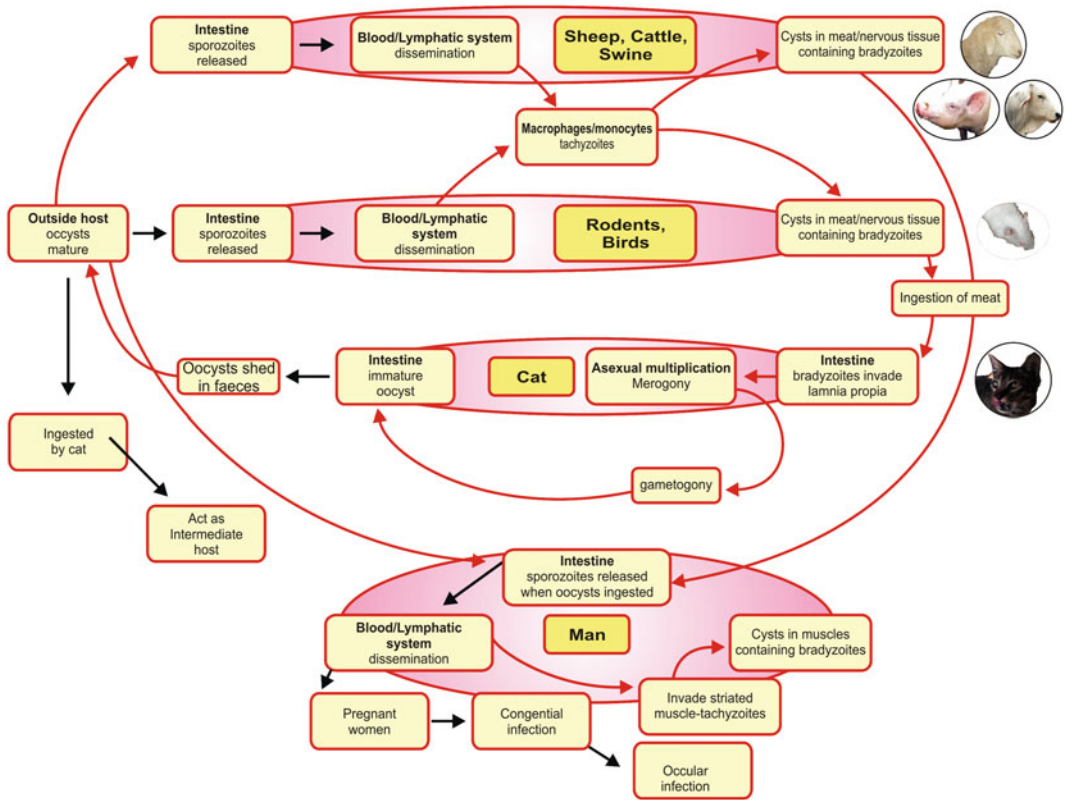
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## 2.11 Other Protozoan Zoonotic Diseases

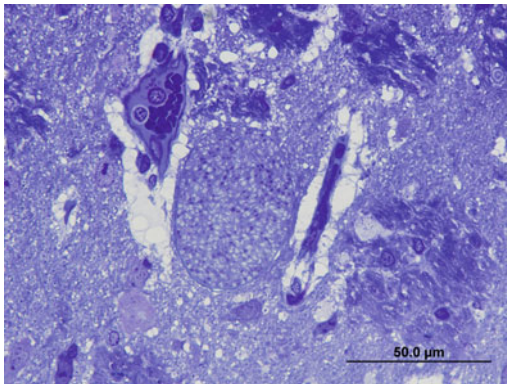
### 2.11.1 Cyclosporiasis

Cyclosporiasis occurs due to coccidium parasite *Cyclospora cayetanensis*. The human disease is worldwide in occurrence. The occurrence of the disease in animals is unknown. The parasite

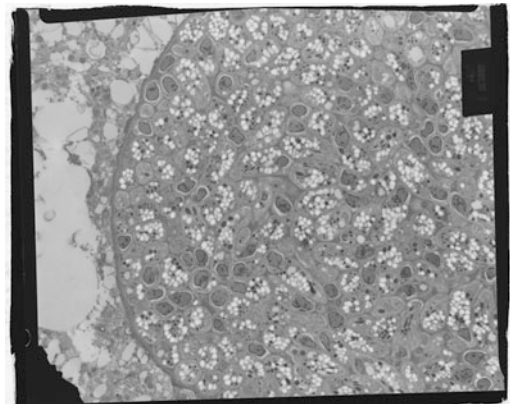




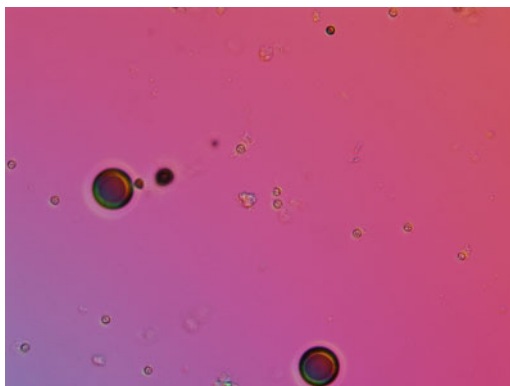
**Fig. 2.12** Life cycle of *Toxoplasma gondii*



**Fig. 2.13** *Toxoplasma* cyst, toluidine stained (100 X) (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)



**Fig. 2.14** Transmission electron microscopy showing ultrastructure of *Toxoplasma* cyst (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)



**Fig. 2.15** *Cyclospora cayetanensis* from Sheather's flotation of faecal sample (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

could cause watery diarrhoea in human subjects. The double-walled oocysts can be detected in faeces using oocyst concentration and staining techniques (Fig. 2.15).

### 2.11.2 Infections Caused by Free-Living Amoebae

The free-living amoebae belonging to the genus *Naegleria*, *Acanthamoeba* and *Balamuthia* could cause the disease in man and some other mammals. The parasite *Naegleria fowleri* could cause amoebic meningoencephalitis in man. *Acanthamoeba* spp. could cause granulomatous amoebic encephalitis in man. The important sources of infection include contaminated water and soil.

### 2.11.3 Malaria in Non-human Primates

The disease occurs due to parasites belonging to the genus *Plasmodium* (Phylum Apicomplexa) which affect non-human primates. Mosquitoes belonging to the genus *Anopheles* act as vectors of the disease. The infection due to plasmodia of non-human primates has been rarely recorded in human beings.

### 2.11.4 Microsporidiosis

The disease primarily occurs due to *Enterocytozoon bienersi*, *Encephalitozoon intestinalis*, *E. cuniculi* and some other species. Microsporidiosis is an important disease in immunodeficient individuals. The important symptoms in man include diarrhoea and watery stools. Contamination of water with infected faeces and stools could serve as a source of infection for other persons.

### 2.11.5 Atypical Human Trypanosomiasis

Although the zoonotic potential of trypanosomiasis in humans due to animal trypanosomes is not certain, 19 (*T. lewisi*-8, *T. evansi*-5, *T. brucei*-4, *T. vivax*-1, *T. congolense*-1) atypical human trypanosomiasis cases have been reported in the literature (Joshi 2013).

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