B. B. Singh Dhaliwal
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Parasitic Zoonoses

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Preface

The book "Parasitic Zoonoses" is intended for higher undergraduate and graduate students of zoonoses and public health, veterinary parasitology, parasite epidemiology; public health workers; public health veterinarians; field veterinarians; medical professionals and all others interested in the subject. The main objective of writing this book is to provide source of information on zoonotic parasites.

The book describes definition of zoonoses and parasitic zoonoses. Parasitic zoonoses are the combination of two subjects, i.e., parasitology and zoonoses where knowledge of both the fields comes into play for the study of zoonotic parasites. Parasitic zoonoses are the most diversified group of zoonotic diseases representing all the classes mentioned in zoonotic diseases classification. They have been classified and discussed based on etiological agents, transmission cycle, reservoir hosts and principal host involved along with examples. Important topics such as food borne, vector borne and occupation-related parasitic zoonoses have also been covered in the introduction. We have also discussed factors responsible for emergence of zoonotic parasites viz. climatic change associated with global warming, increased vector populations, world tourism, the demand for livestock food products, changing socio-economic conditions, poverty, lack of safe drinking water, the large number of stray animals, changed cooking practices, defecating outdoors, poor personal hygiene and the high population density in the tropics.

More than 15 protozoa and 50 other parasitic diseases are zoonotic in nature and all these diseases have been discussed in detail. Each disease has been divided in different sections viz. synonyms/common names, etiology, epidemiology, life cycle, mode of transmission, clinical signs in man and animals, diagnosis and their prevention and control. Life cycle charts and coloured photographs of these parasites have been included wherever required. An alphabetical bibliography for every disease has also been included so that readers have access to further information.

There are few diseases which are rare in occurrence or their zoonotic potential is still being questioned but they have also been given some space to update the knowledge of the readers. We hope this book will prove beneficial to the students and others interested in the subject.

B. B. Singh Dhaliwal
P. D. Juyal

Acknowledgments

First of all with great humility, we dedicatedly accord our sincere gratitude to Almighty God for His benign blessing hand and bestowing a creative and healthy environment throughout this work.

We feel humbled and acknowledge our indebtedness to the authors of the research articles whose work has been consulted and cited in this book. We also thank authors of several source books from where we took guidance and inspiration for this publication.

We particularly value the mentorship provided by Dr. Alvin A. Gajadhar and are thankful to professional and technical staff with Canadian Food Inspection Agency's Centre for Food Borne and Animal Parasitology for incessant encouragement and invaluable advice which enabled us to accomplish the hard task of completing this text book and making it worth presenting.

We are thankful to faculty members of the School of Public Health and Zoonoses and Department of Veterinary Parasitology, Guru Angad Dev Veterinary and Animal Sciences University (GADVASU), Ludhiana (India) for their ever-willing help and scholastic guidance throughout the writing of this book. We also thank authorities of GADVASU, Ludhiana, for providing excellent academic atmosphere to pen this book.

Mere words of acknowledgement will never express the sense of regards and indebtedness towards our families who are the source of our strength and inspiration. Their unconditional love, patience, compromises and sacrifice can never be forgotten.

The cooperation and help of Springer India Pvt. Ltd. to bring out this book is highly appreciated. The authors welcome valuable suggestions from the readers so as to improve the contents of the book in revised editions if any published in the future.

B. B. Singh Dhaliwal
P. D. Juyal

Contents

Introduction 1

Abstract

Parasitism is basically association between two species: Parasite (first species) and the host (second species). Parasitic diseases cause significant problems in the developing world. At present, there are more than 15 protozoa and 50 other parasitic diseases that are zoonotic in nature. Zoonotic parasites are important due to their human and animal health, food safety and economic concerns. Emergence and re-emergence of many zoonotic parasites have been reported across the globe. Contaminated water and food significantly increase the transmission of these parasites. Factors influencing prevalence of these parasites include resurgence in vector population, climate change coupled with global warming, international food trade, poverty and lack of safe drinking water in non-industrialised countries, etc. Most of the animals which live in close contact with man could harbour and transmit zoonotic parasites to human beings. Livestock, pets, domiciliated, wild animals, fish and some other animals; all of them could transmit zoonotic parasites. An update on current status of zoonotic parasites has been provided.

Parasitism is basically association between two species: Parasite (first species) and the host (second species). In this association, parasite lives on the host or its tissue for certain periods of time. This association can be of different types, viz. symbiosis, commensalism, mutualism, phoresis, predation or parasitism. Such host parasite relationship could lead to pathological disorders and disease in the host. In animals and man, many important diseases occur due to parasites. Parasitic diseases cause significant problems particularly in the developing world. Approximately 370 parasitic species (300 helminths and 70 protozoa) have been found in human beings (Ashford and Crewe 1998; Cox 2002). Out of these, some parasites cause important parasitic diseases in man. The parasites are generally classified under the categories: Protozoa, helminths and arthropods (Fig. 1.1).

At present, there are more than 15 protozoa and 50 other parasitic diseases that are zoonotic in nature. Zoonotic parasites are important due to their human and animal health, food safety and economic concerns.

As per World Health Organization (Acha and Szyfres 2006), zoonoses are defined as "those diseases and infections (the agents of) which are naturally transmitted between (other) vertebrate animals and man". Many new zoonotic diseases have been recognised in the recent past to further

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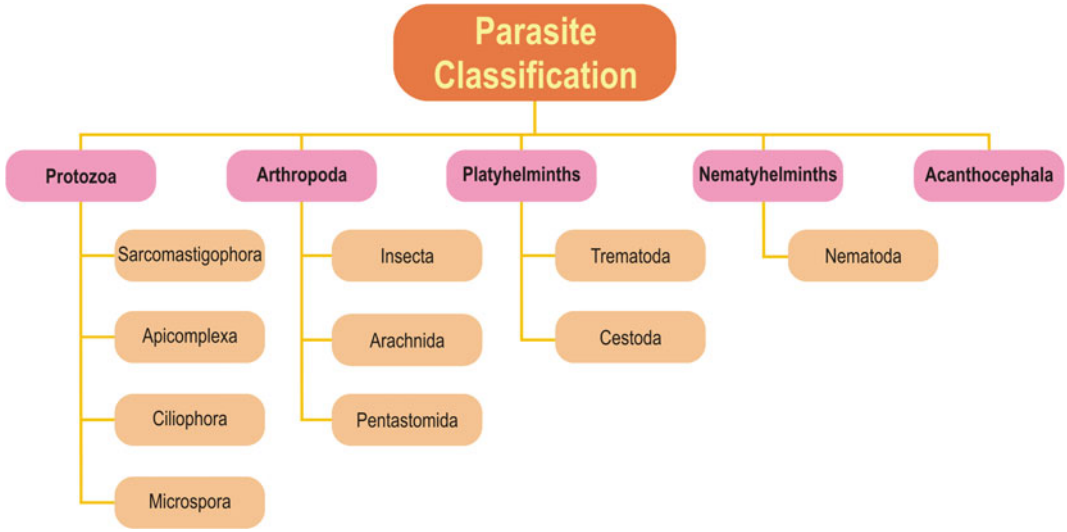

Fig. 1.1 Classification of parasites

expand the existing list of zoonotic diseases. Zoonotic parasitology is the study of phenomenon of parasites which are zoonotic in nature. In other words, parasitic zoonoses are the combination of two subjects: Parasitology and zoonoses where knowledge of both the fields comes into play for study of zoonotic parasites.

Parasitic zoonotic diseases are prevalent worldwide. First reports, emergence and re-emergence of zoonotic parasites, have been recorded in human and animal populations in the past decades (Singh et al. 2010). Important factors that influence the occurrence of zoonotic parasites, particularly in the developing world, include climate change and global warming, vector populations, poverty, lack of safe drinking water, presence of stray animals, defecating outdoors, poor personal hygiene and the high population density (Singh et al. 2011).

Parasitic zoonoses can be classified into different categories as in case of zoonoses (Schwabe 1984). Parasitic zoonoses are the most diversified group of zoonotic diseases representing all classes of zoonotic diseases classified on different bases.

1.1 Classification of Parasitic Zoonoses

1.1.1 Based on Etiological Agents

1. Protozoonoses: cryptosporidiosis, giardiasis, toxoplasmosis, etc.
2. Trematode zoonoses: clonorchiosis, paragonimiasis, etc.
3. Cestode zoonoses: hydatidosis, taeniosis, cysticercosis, etc.
4. Nematode zoonoses: larva migrans, trichinosis, zoonotic ancylostomiasis, etc.
5. Arthropod zoonoses: myiases, zoonotic scabies, etc.

1.1.2 Based on Transmission Cycle

The parasitic zoonotic diseases can be classified depending upon the life cycle of the zoonotic pathogens.

1. **Direct parasitic zoonoses**: The parasite is transmitted by direct contact or indirectly

through food, e.g. scabies, trichinellosis, giardiasis, etc.

2. **Cyclozoonoses**: The diseases caused by zoonotic parasites require two or more vertebrate hosts to complete life cycle of the parasite. Cyclozoonoses are further classified under two categories:
   (a) *Obligatory cyclozoonoses*: Human beings must act as host to complete life cycle of the parasite, e.g. *Taenia solium* and *Taenia saginata* taeniosis.
   (b) *Non-obligatory cyclozoonoses*: Human beings are not required to complete lifecycle of the parasite but could act as accidental host, e.g. human hydatidosis.
3. **Metazoonoses**: The diseases caused by those zoonotic parasites which require both vertebrate and invertebrate species to complete life cycle of the parasite. Metazoonoses are further classified under four categories:
   (a) *Metazoonoses type I*: The diseases caused by those zoonotic parasites which require one invertebrate and one vertebrate host, e.g. *Dipylidium caninum* dipylidiasis.
   (b) *Metazoonoses type II*: The diseases caused by those zoonotic parasites which require two invertebrate and one vertebrate hosts, e.g. paragonimiasis.
   (c) *Metazoonoses type III*: The diseases caused by those zoonotic parasites which require one invertebrate and two vertebrate hosts, e.g. clonorchiosis.
   (d) *Metazoonoses type IV*: The diseases caused by those zoonotic parasites which require transovarian transmission, e.g. *Babesia divergens* babesiosis.
4. **Saprozoonoses**: The diseases caused by those zoonotic parasites which require non-animate material in addition to their hosts for completion of life cycle. Saprozoonoses are further classified under three categories:
   (a) *Saproanthropozoonoses*: Animals principally act as reservoir host and the etiological agents are transmitted from animals to man through inanimate material, e.g. cutaneous larva migrans, myiasis.
   (b) *Saproamphixenoses:* Both animals and humans could act as reservoir hosts and the etiological agents are transmitted through inanimate objects, e.g. probably micronemosis.
   (c) *Saprometanthropozoonoses:* Saprometanthropozoonoses require vertebrate host, invertebrate host and inanimate substance for transmission of disease, e.g. fasciolosis.

1.1.3 Based on Reservoir Hosts

1. *Anthropozoonoses*: The diseases in which animals act as reservoir hosts and humans become accidentally infected, e.g. hydatidosis, visceral larvae migrans.
2. *Zooanthroponoses*: These diseases are normally present in humans but could be transmitted to animals, e.g. amoebosis.
3. *Amphixenosis*: The diseases in which both man and animals could act as reservoir hosts, e.g. clonorchiosis.

1.1.4 Based on Principal Host Involved

1. Cattle-related parasitic zoonoses: *T. saginata* taeniosis, cryptosporidiosis.
2. Sheep-related parasitic zoonoses: fasciolosis, hydatidosis.
3. Pig-related parasitic zoonoses: ascariasis, swine taeniosis.
4. Fish-related parasitic zoonoses: diphyllobothriosis.
5. Dog-related parasitic zoonoses: ancylostomiasis, echinococcosis.
6. Cat-related parasitic zoonoses: toxoplasmosis.
7. Racoon-related parasitic zoonoses: baylisascariosis.

1.2 Animal-Related Parasitic Zoonoses

Most of the animals which are in close contact with man could harbour and transmit zoonotic parasites to human beings. Livestock, pets, domiciliated, wild animals, fish and some other animals, all of them could transmit zoonotic parasites (Parija 1990).

1.2.1 Livestock-Related Parasitic Zoonoses

Livestock is an essential component of human activities particularly for dairy farmers, veterinarians and other related occupations. The food of animal origin, viz. meat, milk and their products are essential components of human diet. The human and livestock populations live in close association with each other. This close association and dependence for food can result in transmission of zoonotic parasites when proper hygienic measures are not taken. These diseases can be transmitted either through food, contaminated water or by vector population.

Livestock transmitted parasitic zoonoses can be classified either on the basis of species involved or on the basis of mode of their transmission (Figs. 1.2, 1.3, 1.4, 1.5, 1.6, 1.7).

Based on the animal species involved, they can be classified into:

1. Cattle-related parasitic zoonoses: *B. divergens* babesiosis, cryptosporidiosis, sarcocystosis, fasciolosis, *T. saginata* taeniosis.
2. Pig-related parasitic zoonoses: balantidiasis, sarcocystosis, toxoplasmosis, gastrodiscoidosis, *T. solium* taeniosis, ascariosis, gnathostomosis, trichinosis.
3. Sheep-related parasitic zoonoses: toxoplasmosis, fasciolosis.
4. Goat related parasitic zoonoses: toxoplasmosis.

1.2.2 Wildlife-Related Parasitic Zoonoses

Human beings, particularly hunters, forest workers, agricultural workers, army personnel, tourists and other related occupational workers, may be exposed to wild animal-related parasitic zoonoses. The wild animal reservoir hosts of some diseases may also enter human and domestic animal habitations leading to their transmission and occurrence in human beings.

Examples of wildlife-related parasitic zoonoses

Opossums (*Didelphis albiventris*, *Didelphis marsupialis*): *Trypanosoma cruzi* trypanosomosis.

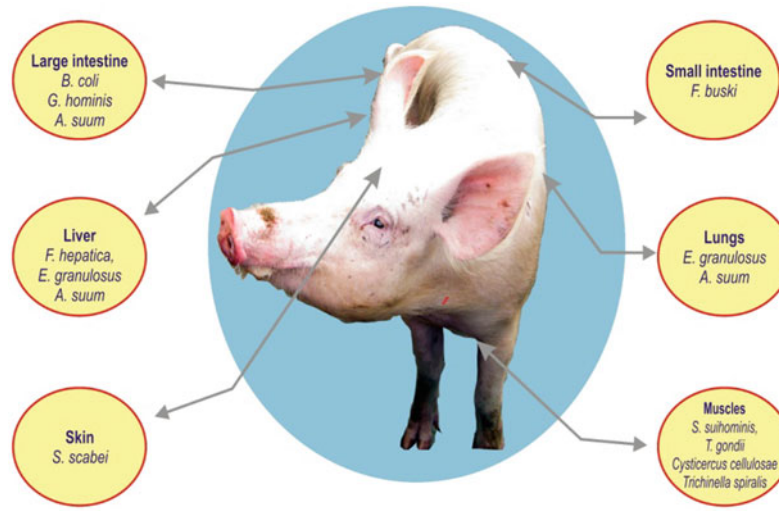

Fig. 1.2 Zoonotic parasites of pig

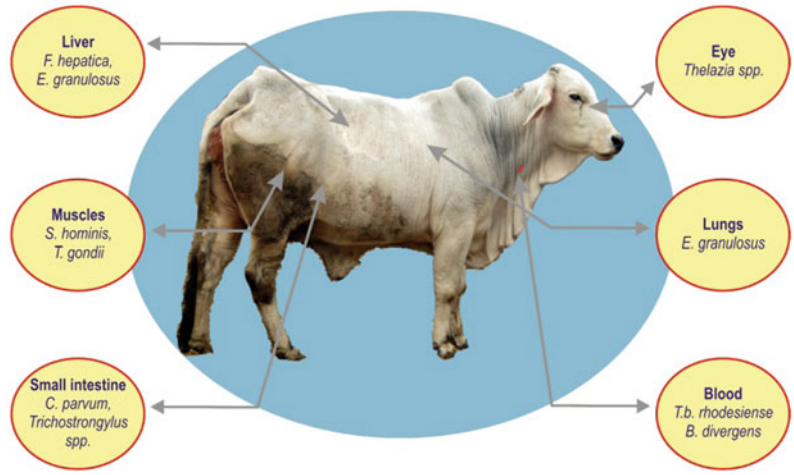

Fig. 1.3 Zoonotic parasites of cattle

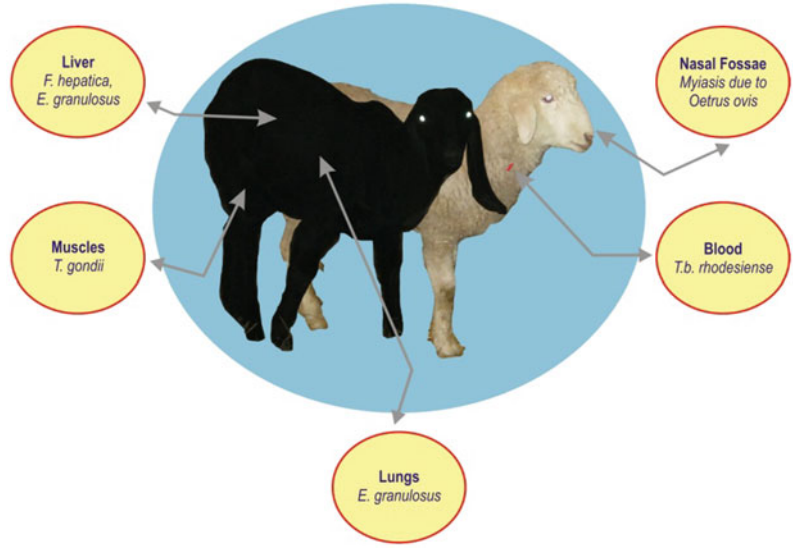

Fig. 1.4 Zoonotic parasites of sheep and goat

Monkeys: malaria of simian origin.
Wild cats: toxoplasmosis.
Wild felids: baylisascariosis.
Wild boar: trichinosis.
Racoons: baylisascariosis.

1.2.3 Domiciliated Animals

Domiciliated animals live in close association with human habitats. They may be partly dependent on man for food and shelter. Resultantly, they can always transmit zoonotic parasitic diseases.

Examples of Domiciliated animal-related parasitic zoonoses

Rodents: *Babesia microti* babesiosis, leishmaniosis, angiostrongylosis.

Reptile/amphibian borne: sparganosis, gnathostomosis.

1.2.4 Pet Animal-Related Parasitic Zoonoses

Pet animals are kept by man for companionship, security and enjoyment. Dogs and cats are the most common and popular pets kept worldwide.

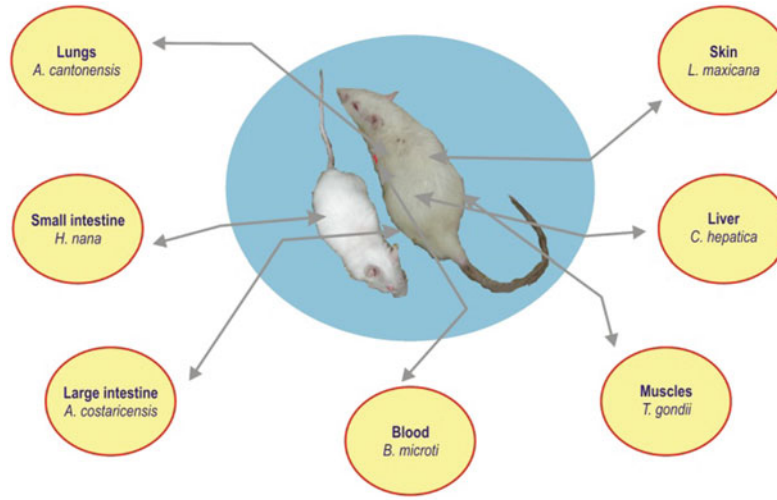

Fig. 1.5 Zoonotic parasites of rodents

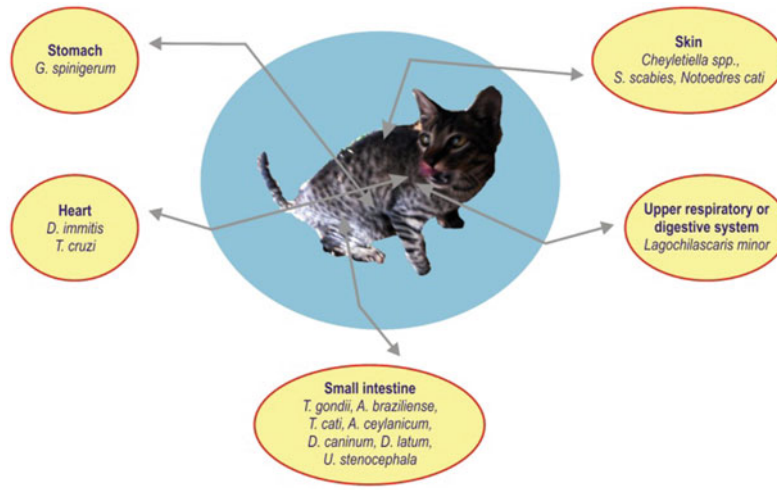

Fig. 1.6 Zoonotic parasites of cat

Poor animal hygiene may result in transmission of such diseases to the human population.

Examples of Pet animal-related parasitic zoonoses

Dogs: *T. cruzi* trypanosomosis, leishmaniosis, diplydiasis, echinococcosis, cutaneous larva migrans, gnathostomosis, toxocariasis.

Cats: *T. cruzi* trypanosomosis, toxoplasmosis, diplydiasis, cutaneous larvae migrans, gnathostomosis.

1.2.5 Fish, Shellfish and Mollusc-Borne Parasitic Zoonoses

Fish are aquatic vertebrates and are essential component of human diet. Fish is an important source of protein for man. Ingestion of contaminated raw fish may transmit important zoonotic infections.

Examples of Fish, shellfish, crab and mollusc-borne parasitic zoonoses

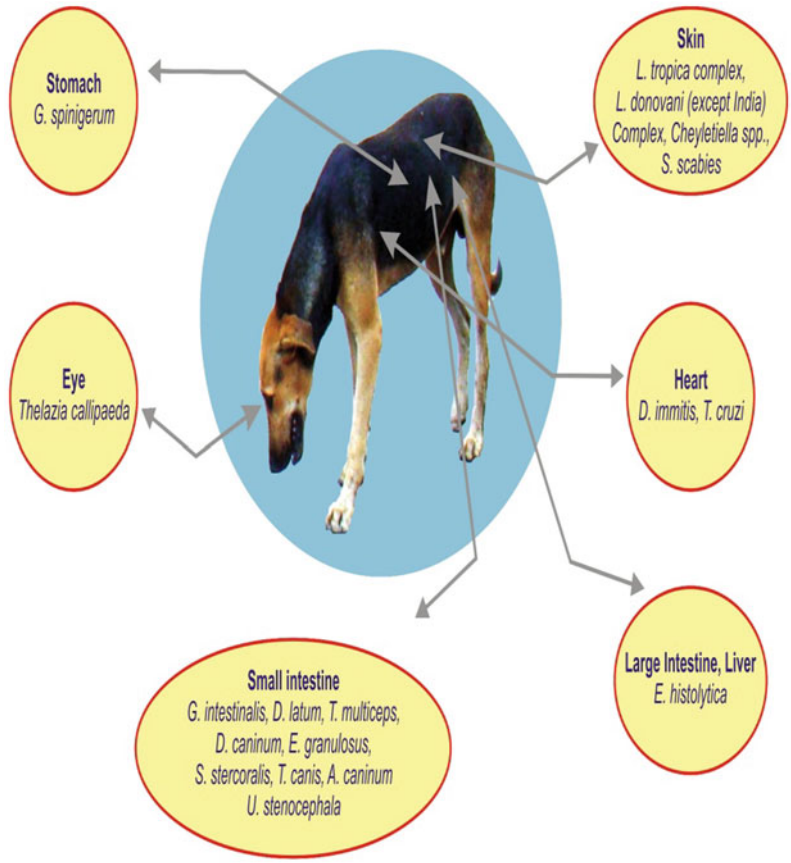

Fig. 1.7 Zoonotic parasites of dog

Angiostrongylosis, paragonimiasis, anisakiosis, clonorchiosis, diphyllobothriosis, echinostomosis, *Heterophyes heterophyes* infections, *Haplorchis* spp. infections, *Matagonimus yokogamai* infections (Figs. 1.8, 1.9).

1.3 Occupational Parasitic Zoonoses

Occupational parasitic zoonoses are the zoonotic parasitic diseases which may occur due to the occupational environment in persons related with specific occupation. There is increased risk of contacting such diseases in persons working in specific occupational areas. In addition to occupational zoonoses, immunocompromised persons are always at higher risk of being infected with zoonotic parasites such as *Cryptosporidium parvum, Toxoplasma gondii*, etc. There is also a strong correlation between occurrence of zoonotic babesiosis among splenectomized individuals (Fig. 1.10).

1.3.1 Prevention and Control

- To generate better awareness regarding transmission and prevention of occupational zoonoses among related occupational groups.
- Monitoring and surveillance of occupationally related zoonotic parasites among risk groups.
- Control of such disease in their animal hosts.

1.4 Food Borne Parasitic Zoonoses

The food animals, viz. cattle, buffalo, sheep, goat and pigs consume fodder crops and convert them into animal protein. The meat from these animals is an important source of protein and nutrients (Gracey and Collins 1992). Fish and

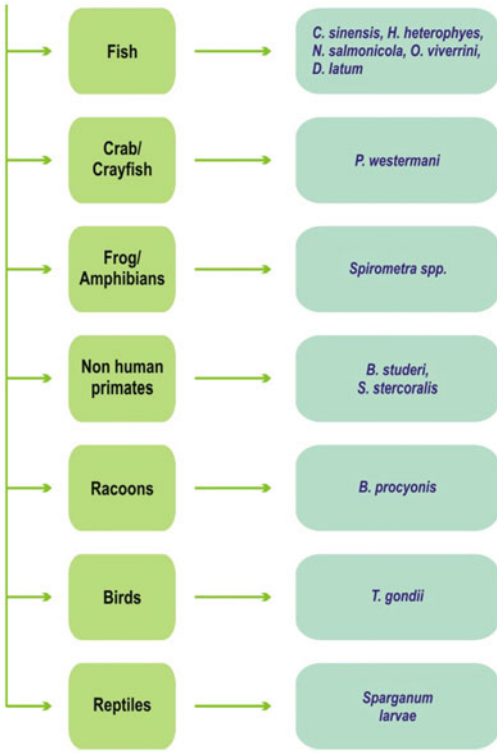

Fig. 1.8 Zoonotic parasites of some other animals

other sea foods also make a vital contribution to provide essential nourishment for human population. The meat of wild animals is also used as a part of human diet in many parts of the world. Poultry are another important meat producing species. Besides fish and meat, milk and raw vegetables also form important components of human food. Food and water are the two essential components of human diet. The food of animal origin is a rich source of proteins, energy, vitamins and minerals. The food especially of animal origin is essential for growth and development of human beings.

However, presence of zoonotic parasites in such food and contaminated water could lead to their occurrence in man and animals. Transmission of zoonoses of parasitic origin through food has public health as well as socio-economic significance (Slifko et al. 2000). Transmission of zoonotic parasites through contaminated water, food, plants, vegetables and soil assumes great importance (Slifko et al. 2000; Zhou et al. 2008). Food security is an important issue due to increasing human populations, income levels, changing food preferences and urbanisation. The demand for food of animal origin is increasing worldwide.

Food and water act as an important route of transmission for parasites from one host to another. Food and water are also important for transmission of parasites which require more than one host to complete their life cycle

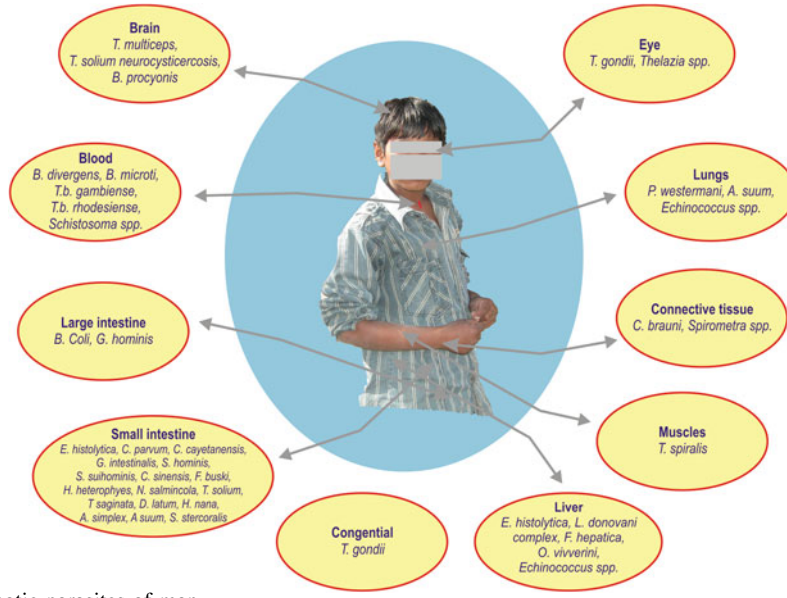

Fig. 1.9 Zoonotic parasites of man

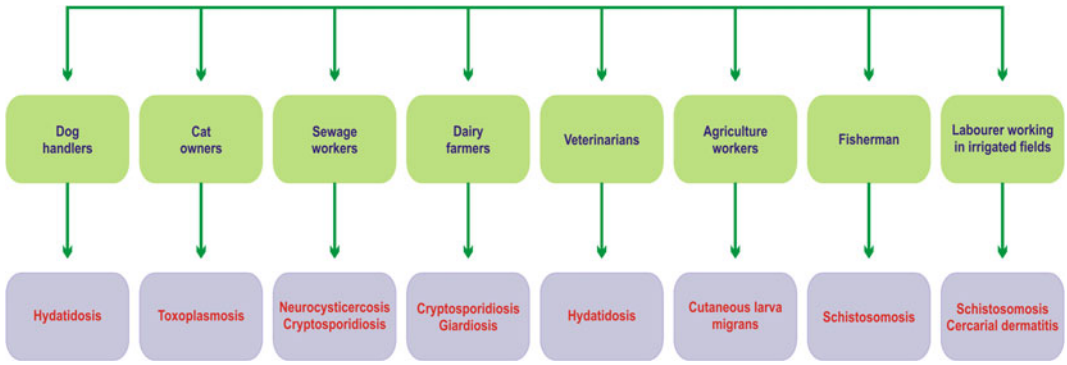

Fig. 1.10 Occupational parasitic zoonoses

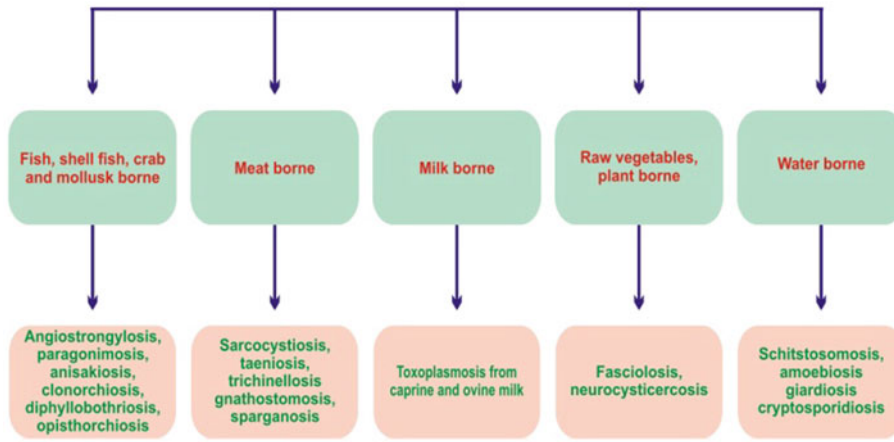

Fig. 1.11 Food borne parasitic zoonoses

(Bhatia 1991). The important parasites transmitted through food and water includes *T. solium, Echinococcus granulosus*, *T. gondii*, etc. Parasites exploit behavioural patterns of their hosts for further transmission among animals and humans (Gajadhar et al. 2006). Further, parasite life cycle stages such as eggs and oocysts have the ability to resist and survive adverse environmental conditions (Gajadhar et al. 2006). Many factors have lead to an increased risk for the occurrence of food borne zoonotic parasites. Climate change and increased food trade have manifold increased the risk of food borne parasitic zoonoses. Increased demand for animal food due to rapid urbanisation, increase in human population and high income indicates need for more emphasis on food safety issues (Gajadhar et al. 2006). Many emerging zoonotic parasites are food borne in nature (WHO 2002) (Fig. 1.11).

Prevention and control strategies should include use of advanced serological and molecular diagnostic techniques, continuous monitoring of zoonotic parasites, health education, social and economic development particularly in the developing world; and prompt treatment of cases (Dorny et al. 2009). Many important parasites shed their eggs in the faeces which serve as an important source of infection leading to contamination of environment particularly water and food supplies (Slifko et al. 2000). One or more life cycle stages of the parasite can transmit the infection. For example, ingestion of only infectious oocysts leads to

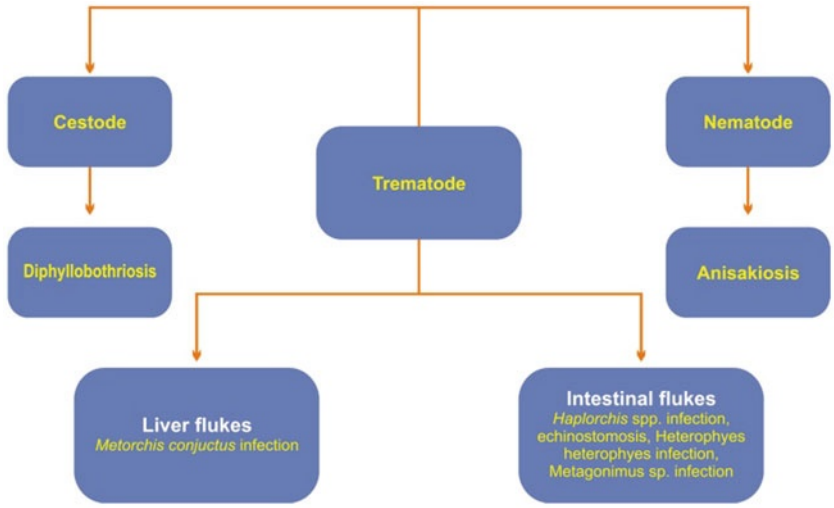

Fig. 1.12 Fish-shellfish and mollusc-borne parasitic zoonoses

cryptosporidiosis, whereas both oocysts and tissue cysts of *T. gondii* can cause infection in man (Slifko et al. 2000).

1.4.1 Fish, Shellfish and Mollusc-Borne Parasitic Zoonoses

Fish and related zoonoses can be classified into trematode, cestode and nematode zoonoses. Trematode diseases can be further sub-classified into liver and intestinal flukes. As per WHO (1995), fish-borne trematodes infect more than 18 million people and globally more than half a billion people are at risk from these diseases (Chai et al. 2005) (Fig. 1.12).

1.4.1.1 Prevention and Control

- Avoid eating raw or improperly cooked fish.
- Interruption in the transmission cycle of the parasite.
- Health education and environmental sanitation.
- Use of clean water in slaughtering operations to avoid contamination with other water-borne zoonotic parasites.

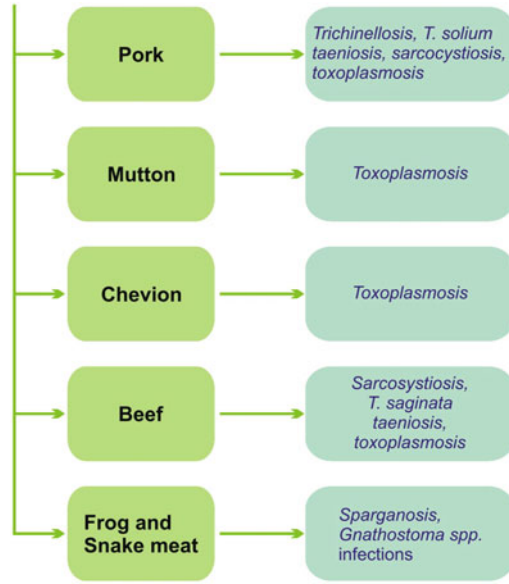

Fig. 1.13 Meat-borne parasitic zoonoses

1.4.2 Meat-Borne Parasitic Zoonoses

Many important parasitic zoonotic diseases are meat-borne in nature, e.g. toxoplasmosis, taeniosis, trichinellosis, etc. Most of the food producing animals (except poultry), viz. cattle,

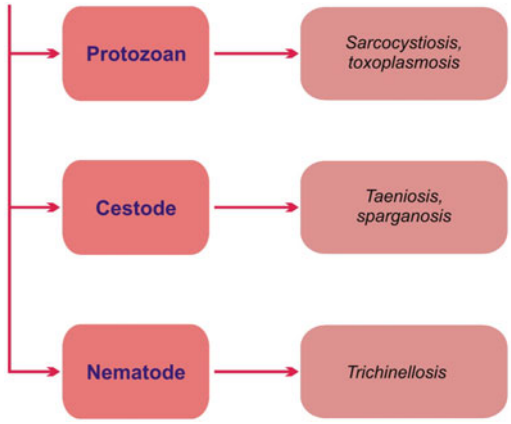

Fig. 1.14 Meat-borne parasitic zoonoses

buffalo, pig, sheep and goat could transmit zoonotic parasites. Meat-borne parasitic diseases can be further classified either on the basis of animal host involved or into protozoan, trematode, cestode and nematode meat-borne parasitic zoonoses (Figs. 1.13, 1.14).

1.4.2.1 Prevention and Control

- Avoid consumption of raw or under cooked meat.
- Provide safe food and water to livestock.

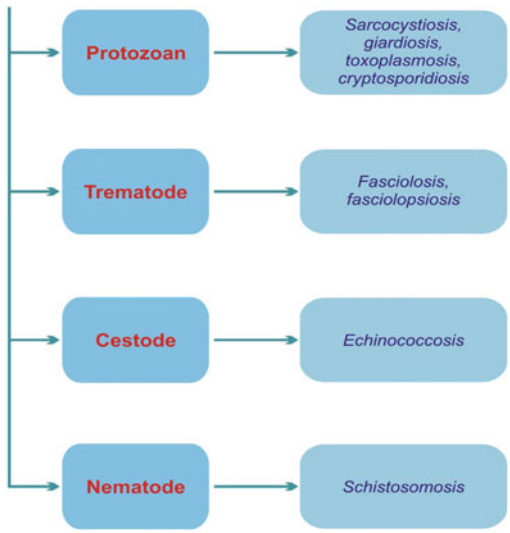

Fig. 1.15 Water-borne parasitic zoonoses

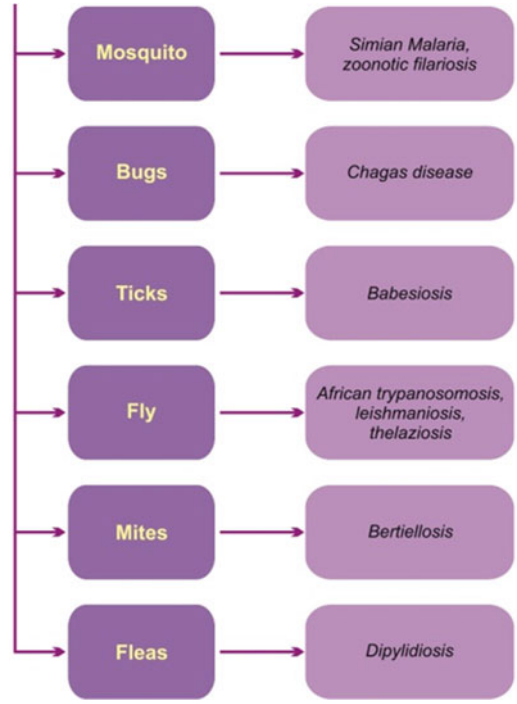

Fig. 1.16 Vector-borne parasitic zoonoses

- Use of best hygienic practices during slaughtering operations to avoid contamination with other water-borne zoonotic parasites.

1.4.3 Water-Borne Parasitic Zoonoses

These parasites are prevalent worldwide and pose a significant threat particularly in the developing world (Gajadhar and Allen 2004). The non-availability of clean and hygienic drinking water is an important risk in the developing world. The parasite stages that can be transmitted through water range from unicellular amoebae to complex metazoans such as trematodes and cestodes (Gajadhar and Allen 2004). The life cycle stages of these parasites are well adapted for their survival in the environment and further dissemination by water (Gajadhar and Allen 2004). Many important zoonotic parasites such as *C. parvum*, *T. gondii*, could be transmitted through contaminated water. Transport hosts such as swine can also

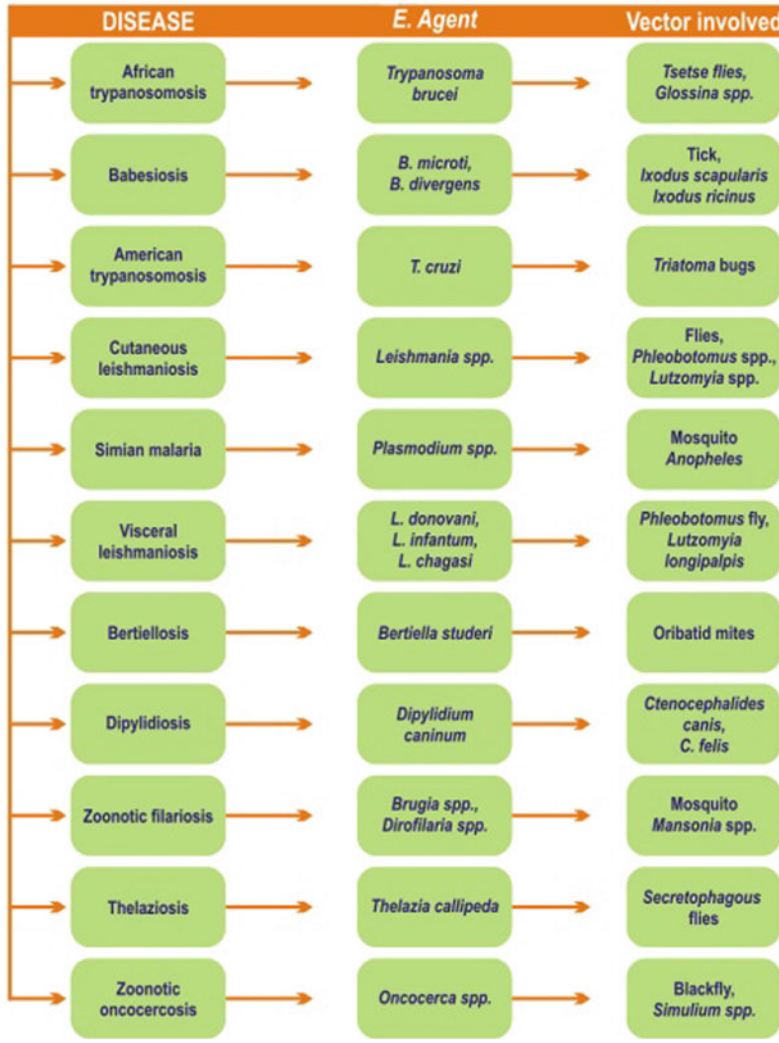

Fig. 1.17 Classification of vector-borne parasitic zoonoses as per Colwell et al. (2011)

play an important role in collection and concentration of resistant exogenous stages (Gajadhar and Allen 2004) of many parasites. Important water-borne parasitic zoonoses can be classified through protozoa, trematode and cestode zoonoses, but protozoan zoonoses are more common and significant (Fig. 1.15).

1.4.3.1 Prevention and Control

- Use properly filtered water for drinking purpose.
- Routine monitoring of drinking water.
- Regular deworming of reservoir and animal hosts.
- Avoid surface contamination of swimming pools.

1.4.4 Milk-Borne Parasitic Zoonoses

Although milk is considered as safer food in relation to zoonotic parasites, but milk of caprine and ovine can cause toxoplasmosis in man if the animal is infected with the disease and raw milk is consumed. Additionally, external faecal

contamination during or after milking could pose additional risks for transmission of some important zoonotic parasites.

1.4.5 Raw Vegetables/Plant-Borne Parasitic Zoonoses

The role of raw vegetables/salad in transmission of zoonotic parasites gets attention particularly in cases of neurocysticercosis in humans where human sewage has been used for irrigation purposes for farmland used to grow vegetables and it has been found to be an important risk for transmission of the disease. Fasciolosis is another important disease which can be transmitted through ingestion of contaminated plants. The contamination of vegetables could also help in transmission of parasites such as *Ascaris suum* and *Toxocara* species (Slifko et al. 2000).

1.4.6 Reptile, Amphibian-Borne Parasitic Zoonoses

Sparganosis and *Gnathostomosis* are two important cestode and nematode diseases, respectively which can occur in human beings due to consumption of raw or under cooked reptiles or amphibians.

1.4.7 Poultry-Related Parasitic Zoonoses

No zoonotic parasites are present in poultry meat and it is generally free from zoonotic parasites except that clean water should be used in slaughtering operations. However, free ranging chicken could serve as important reservoir for toxoplasmosis. A recent study has demonstrated that poultry may serve as a minor reservoir for fish borne trematodes (Anh et al. 2010).

1.5 Soil-Transmitted Parasitic Zoonoses

Soil-transmitted zoonotic parasites include helminth infections, viz. ascariosis due to *Ascaris lumbricoides*, hookworm infections due to *Ancylostoma* spp. and toxocarosis due to *Toxocara canis* and *Toxocara cati* under moist conditions. The parasite eggs are generally found in soil of tropical and sub-tropical countries (Bethony et al. 2006). These parasites may lead to anaemia and growth retardation.

1.5.1 Prevention and Control

- Periodic deworming with anthelmintic drugs.
- To generate awareness regarding soil-transmitted helminths in school children.
- Improved personal hygiene.
- Control of disease in animal hosts.

1.6 Vector-Borne Parasitic Zoonoses

Vector-borne parasitic zoonoses (VBPZ) are the diseases in which zoonotic parasites are transmitted from an infected animal to human beings by an arthropod or other vector. Climate change and global warming could affect occurrence, intensity and seasonality of many vector-borne zoonotic parasites. VBPZ are important as they affect human and animal health (WHO 2004) and cause economic losses (Bram et al. 2001). Factors responsible for emergence of VBPZ include climate change (Lindgren and Gustafson 2001; Rosenthal 2009), increased tourism, animal transport and international food trade (Weijden et al. 2007), drug resistance in parasite and vector (Takken and Knols 2007), and changes in land-use pattern (Colwell et al. 2011).

VBPZ can be classified either on the basis of vector involved or into protozoa, cestode and nematode vector-borne zoonoses. Under VBPZ,

protozoan-related zoonoses are most important and significant.

Depending upon the vector involved, VBPZ can be classified into fly, mosquito, mites, bugs, flea and tick-borne parasitic zoonoses (Figs. 1.16, 1.17).

1.6.1 Prevention and Control

1. Use of physical barriers such as mosquito nets.
2. Biological control of vector population.
3. Destroy or modify vector breeding grounds.
4. Use of chemicals and herbal insecticides as repellant/killer, etc.
5. Reduction in the reservoir hosts of infection.
6. Personal prevention strategies.

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Protozoonoses

2

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: Kinestoplastorida
Family: Trypanosomatidae

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

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

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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.

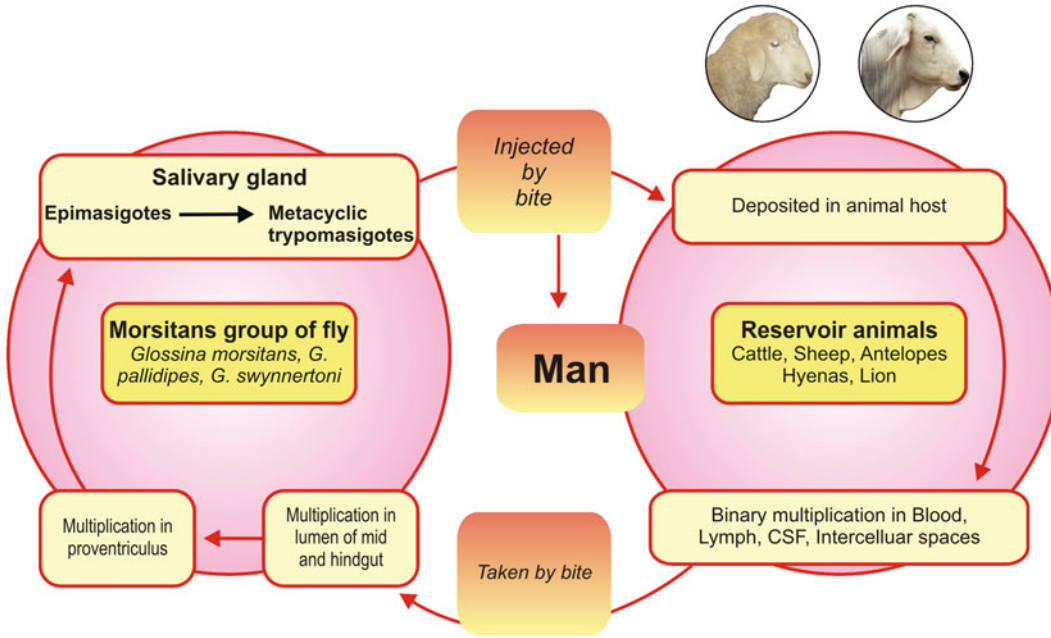

Fig. 2.1 Life cycle of *T.b. rhodesience*

2.1.10 Control

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

2.2 Amoebiasis

Subphylum: Sarcodina
Order: Amoebidorida
Family: Endamoebidae

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. dysenteríae* 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.

2.3 Babesiosis

Class: Piroplasmida
Family: Babesiidae

2.3.1 Common Name/Synonyms

The disease is known as babesiasis, 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

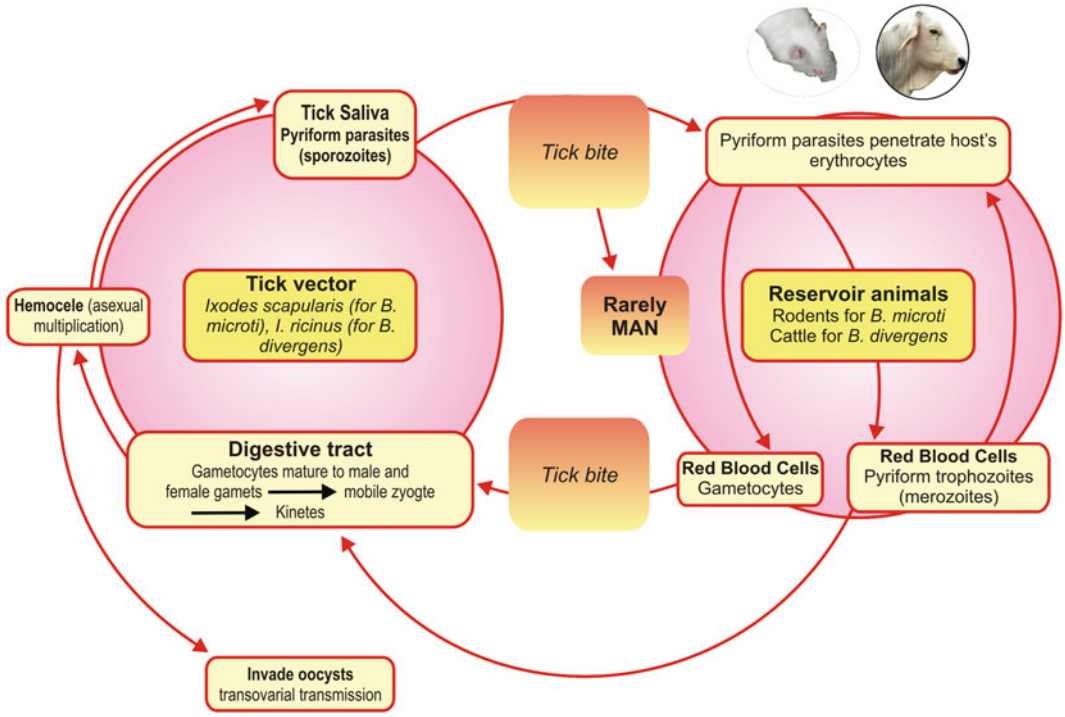

Fig. 2.2 Life cycle of *B. microti* /*B. divergens*

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 (Arean 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

2.5 Chagas Disease

Order: Kinestoplastorida
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).

2.6 Cryptosporidiosis

Subclass: Coccidea
Family: Cryptosporidiidae

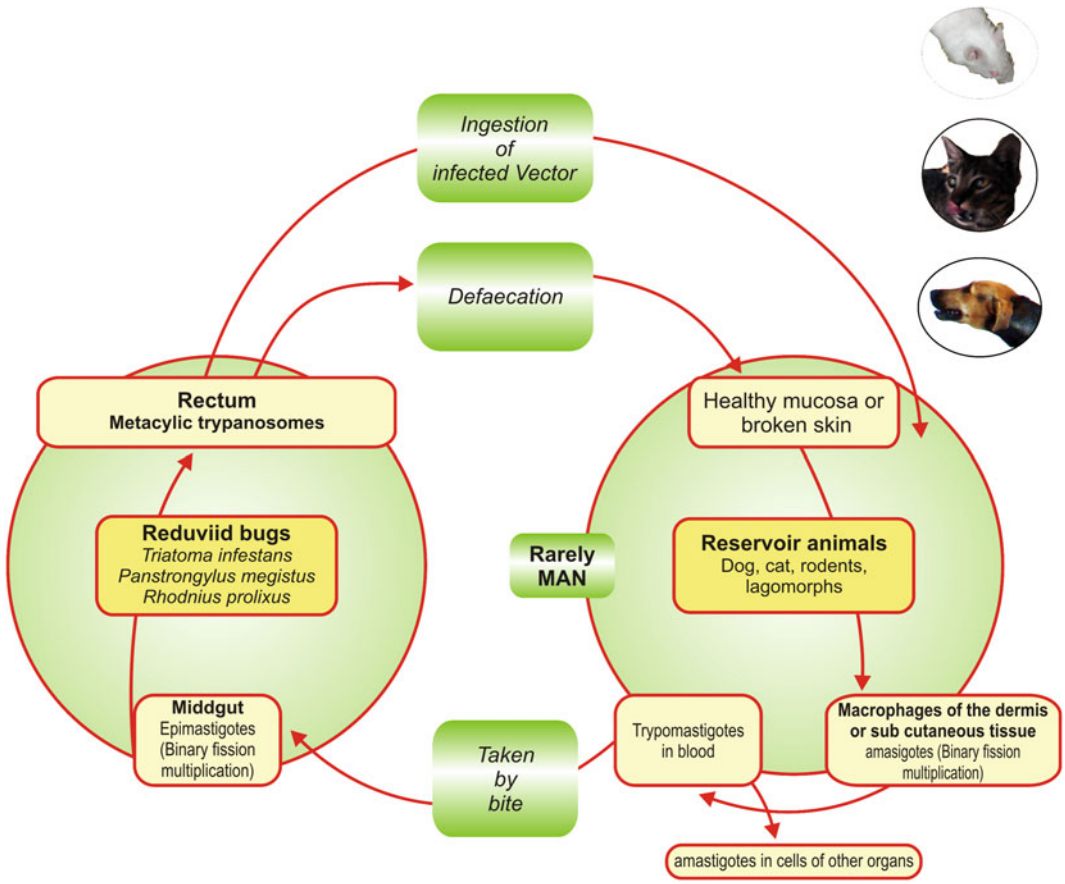

Fig. 2.3 Life cycle of *T. cruzi*

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.

2.7 Giardiosis

Class: Zoomastigophora
Order: Diplomonadida
Family: Diplomonadida

2.7.1 Common Name/Synonyms

Giardia enteritis

Fig. 2.4 *Cryptosporidium* oocysts from a Sheather's floation 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)

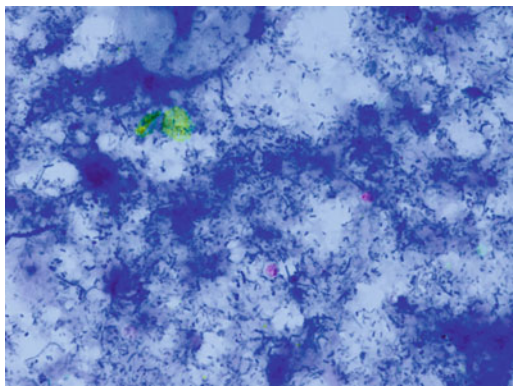

Fig. 2.5 *Cryptosporidium* oocysts from modified Ziehl-Neelson's staining of faecal samples

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). Giardiosis 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 subclinical (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.

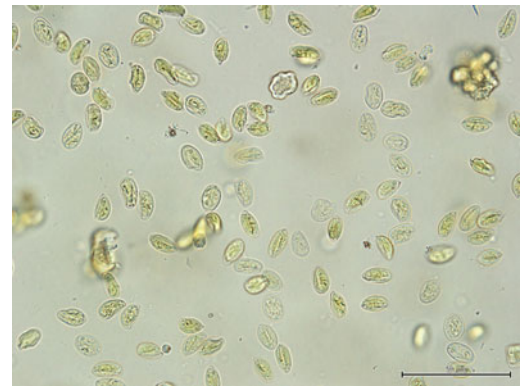

Fig. 2.6 *Giardia* cysts from a zinc sulphate flotation of faecal samples (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

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, dumdum 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 leishmaniosis (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.

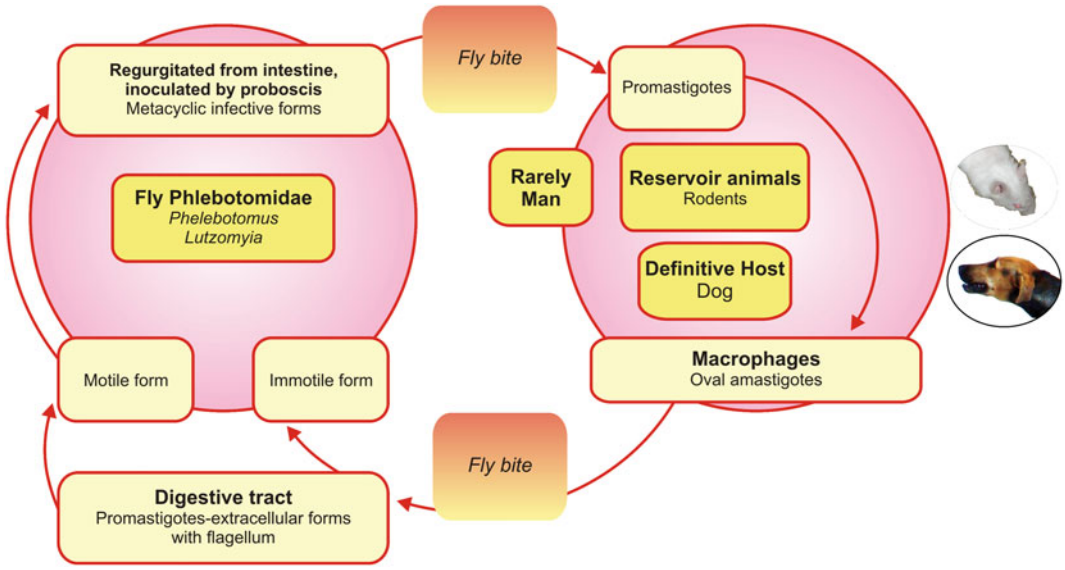

Fig. 2.7 Life cycle of *Leishmania* spp. causing cutaneous leishmaniasis

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

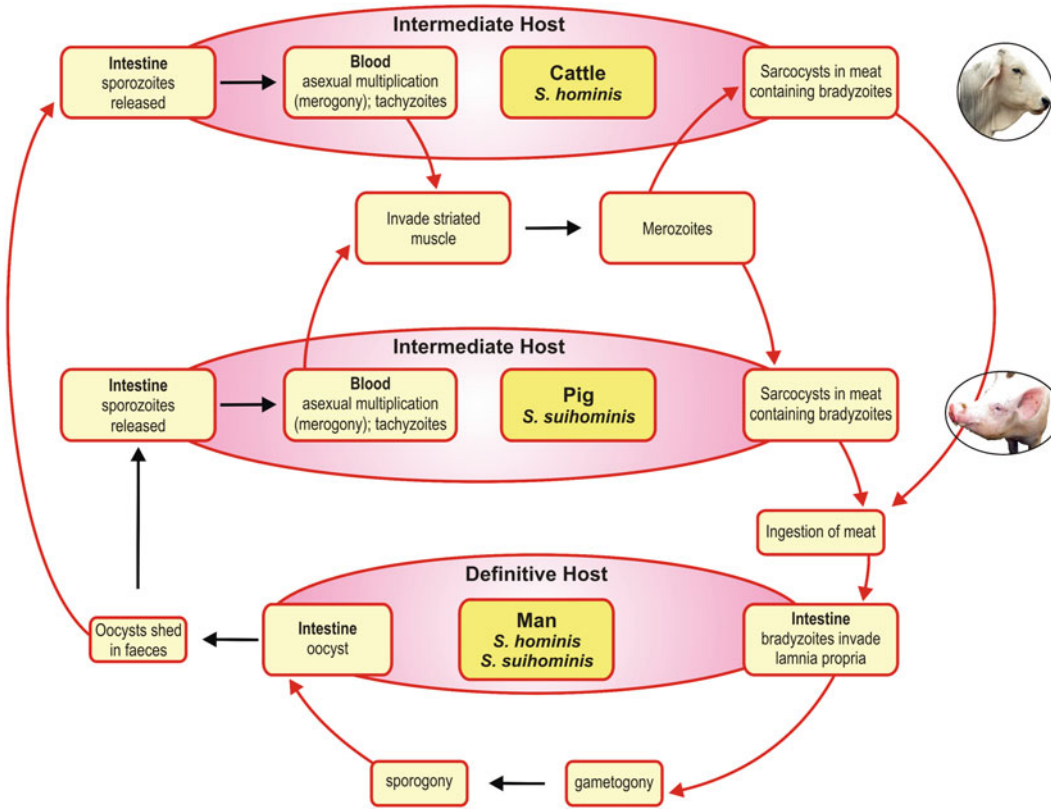

Fig. 2.8 Life cycle of *S. hominis* and *S. suihominis*

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).

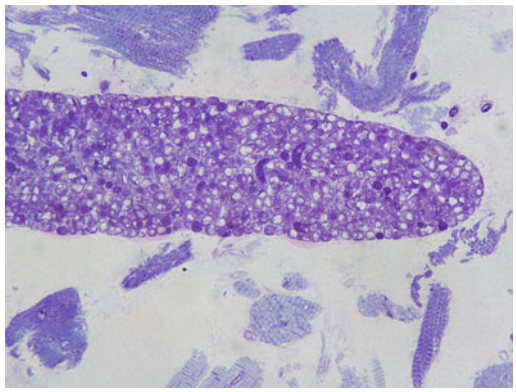

Fig. 2.9 Transmission electron microscopy of *S. cruzi*, thin walled *S. cruzi* showing zoites in the granulomata and hair-like projections on the cyst wall (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection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

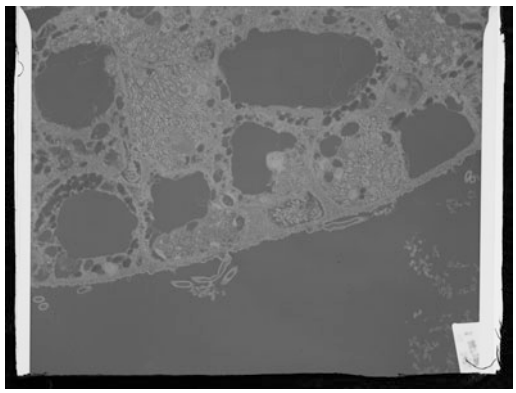

Fig. 2.10 Ultrastructure of *Sarcocyst* wall showing villar projections (VP) arising from the *S. cruzi* cyst wall. No microtubules are seen in VP and a zone of ground substance (GS) can also be seen (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and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

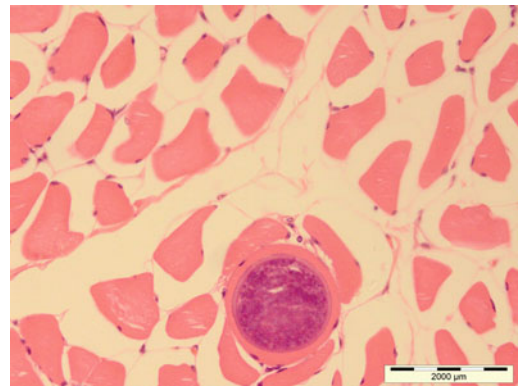

Fig. 2.11 Thick-walled *Sarcocystis* cyst in diaphragmatic muscles of pig (H & E stain, 40X)

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.

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

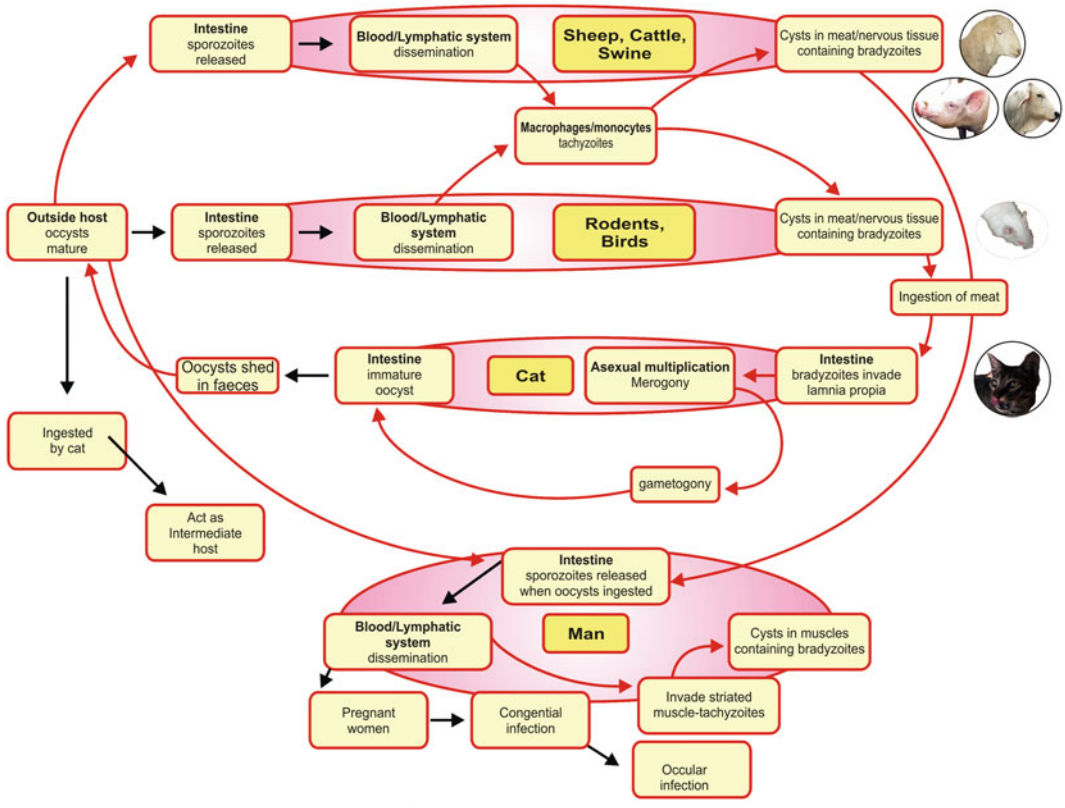

Fig. 2.12 Life cycle of *Toxoplasma gondii*

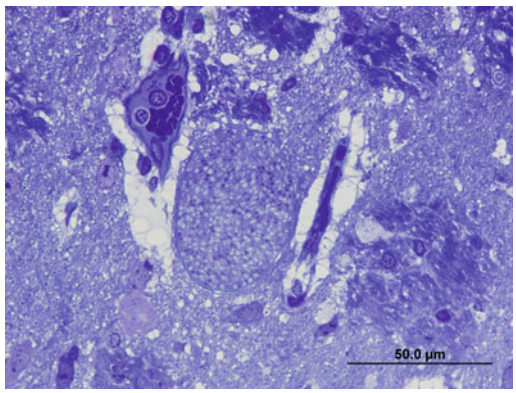

Fig. 2.13 *Toxoplasma* cyst, toludine stained (100 X) (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

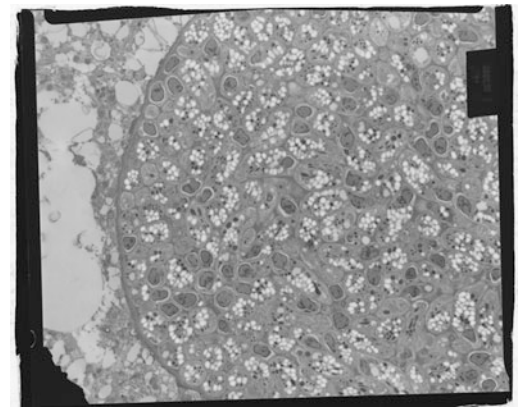

Fig. 2.14 Transmission electron microscopy showing ultrastructure of *Toxoplasma* cyst (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

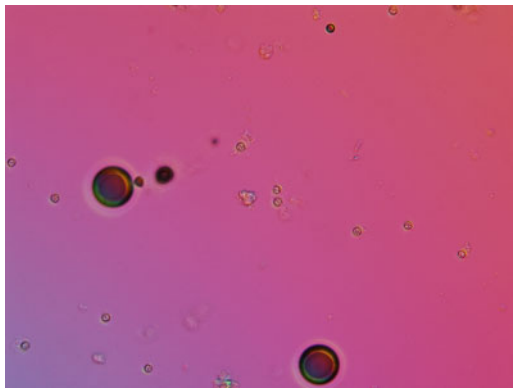

Fig. 2.15 *Cyclospora cayetanensis* from Sheather's flotation of faecal sample (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Centre for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

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 bieneusi*, *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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Trematode Zoonoses

3

Abstract

Trematode zoonoses could be defined as "those trematode diseases which are naturally transmitted between (other) vertebrate animals and man". Many neglected trematode zoonotic diseases are being considered as important human infections. The penetration of human skin by cercariae of avian schistosomes leads to cercarial dermatitis. *Metagonimus yokogawai* and *Heterophes heterophes* are the important heterophyids which could cause zoonotic disease. Pigs appear to be natural definitive host for *Gastrodiscus hominis*, which causes disease in human beings. Fasciolosis is mainly important in livestock but human cases have been reported from 42 countries. The parasite *Fasciolopsis buski* is prevalent in many Southeast Asian countries. Accidental ingestion of ants infected with *Dicrocoelium dendriticum* could cause human disease. Among all the liver flukes, *Clonorchis sinensis* is the most important fish-borne zoonoses prevalent in East Asia. The parasite *Paragonimus westermani* is found in Asia, Africa and South America. In Africa, the public health burden of schistosomiasis, caused by *Schistosoma mansoni*, *S. hematobium* and *S. intercalatum* is enormous.

3.1 Cercarial Dermatitis

Subclass: Digenea
Family: Schistosomatidae

3.1.1 Common Name/Synonyms

Schistosome dermatitis, Swimmer's itch, rice farmer's itch or clam digger's itch

Cercarial dermatitis occurs due to penetration of human skin generally with cercariae of avian schistosome parasites. The disease occurs across the globe mostly near freshwater habitats. Swimmer's itch is a common non-communicable water-borne disease and has been recently regarded as an emerging infection (De Gentile et al. 1996; Kolarova 2007). Cercarial dermatitis is an occupational zoonosis in certain populations.

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3.1.2 Etiology

The disease primarily occurs due to larvae of 20 species of Schistosome genera such as Austrobilharzia, Dendritobilharzia, Gigantobilharzia, Trichobilharzia and Orientobilharzia.

3.1.3 Epidemiology

Cercarial dermatitis is trematode zoonosis and worldwide in occurrence (Farahnak and Essalat 2003). The disease has been reported from many countries in Europe and America (De Gentile et al. 1996; Bastert et al. 1998; Folster-Holst et al. 2001; Kolarova et al. 1999; Chamot et al. 1998; Lindblade 1998). The parasite has also been detected in snails in Germany (Loy and Haas 2001). The cases have been reported in North America, Canada, Europe, Africa, the Far East (Hoeffler 1977; Mulvihill and Burnett 1990) and Taiwan (Wang and Chang 2008). Several outbreaks of cercarial dermatitis have also been reported in Quebec, Canada, Germany, France, Thailand and along the Huaihe River of China (Levesque et al. 2002; Kullallavanijaya and Wongwaisayawan 1993; Verbrugge et al. 2004; Li et al. 1998).

The schistosome larvae develop in freshwater snails leading to association of the disease in lakes or ponds where cercariae of bird schistosomes are usually reported as the causative agent of the disease (Horák et al. 2002; Kolarova 2007). Few outbreaks from salt or brackish waters have also been reported (Horák et al. 2002; Kolarova 2007).

3.1.4 Reservoir

The cercaria's normal hosts are birds, rodents and various other small mammals and human beings act as an accidental host (Wang and Chang 2008).

3.1.5 Transmission

Humans become infected when cercariae present in water bodies attach and enter through the skin of the human accidental host.

3.1.6 Life Cycle

The adult parasites live in the mesenteric blood vessels of birds or mammals and produce eggs which are released in faeces of the host. The eggs released through faeces into the water bodies hatch and release miracidia. The miracidia penetrate an appropriate species of intermediate snail host. In snail they further develop into multiple sporocysts. Numerous fork-tailed cercariae develop in the snail host through asexual multiplication (Bastert et al. 1998; Folster-Holst et al. 2001; Farahnak and Essalat 2003). The cercariae are released in the water where they penetrate a vertebrate definitive or accidental (Brant et al. 2010) host's skin using oral suckers and release a proteolytic enzyme which facilitates their penetration (Wang and Chang 2008).

3.1.7 Clinical Signs in Man

The clinical disease was first reported by Fujii in 1887 (Oda 1973). The schistosome larvae (cercariae) were identified as the causative agent by Cort in 1928 in the USA. This followed the reporting of the causative agents from United Kingdom (Matheson 1930) and France (Brumpt 1931). Christensen and Green (1928) used the word swimmers' itch for the disease. Clinical signs in man include a strong maculo-papulovesicular skin eruption and intensive itching affecting areas exposed to water (Horák et al. 2002; Kolarova 2007).

The cercaria of non-human schistosomiasis could occasionally penetrate the epidermis, but are unable to reach the bloodstream and to induce systemic infection (Haas and van de Roemer 1998; Wang and Chang 2008). These parasites induce Type I hypersensitivity reaction and finally die due to histolysis (Wang and Chang 2008). The residual cercarial protein could induce Type IV hypersensitivity reaction and diffused skin eruptions (Kourilova et al. 2004).

3.1.8 Clinical Signs in Animals

Although cercarial dermatitis is mainly related to human infections, symptoms have been reported in animals such as rabbits and dogs (Herber 1938; Augustine and Weller 1949; Olivier 1953).

3.1.9 Diagnosis

History of exposure to the contaminated freshwater and clinical signs give preliminary indication for the disease (Scrimgeour and Daar 2000). The parasite could be detected by microscopic observation of schistosome larvae from water snails (Kolarova 2007).

3.1.10 Control

Use of personnel protective measures could help prevent the disease in human beings.

3.2 Dicrocoeliasis

Subclass: Digenea
Order: Plagiorchiida
Family: Dicrocoeliidae

3.2.1 Common Name/Synonyms

Lancet liver fluke disease

3.2.2 Etiology

The disease occurs due to *D. dendriticum* and *D. hospes*. The parasite resides in the bile ducts of sheep, cattle and other mammals including man.

3.2.3 Epidemiology

The parasite is distributed in Europe (Mediterranean area), Asia (Middle East, Indonesia, Japan, Malaysia, People's Republic of China), former Soviet Union, North Africa, South America and some foci in North America (Jeandron et al. 2011; Le Bailly and Bouchet 2010). Although the infection is common in livestock, it has been rarely reported in human beings (Jeandron et al. 2011). Human infections by *D. dendriticum*, a zoonotic liver fluke are not common and rare in occurrence. Most reports concern autochthonous populations in Europe, Middle East and immigrants from North Africa (Gualdieri et al. 2011; Ashrafi 2010). *D. dendriticum* has also been reported in humans from Canada (Schweiger and Kuhn 2008). Samalia et al. (2009) reported the disease in a 7-year-old child presented with recurrent right flank subcutaneous nodules containing a liver fluke on excision, and tissue histology showed characteristic brown operculated ova of *D. dendriticum*. El-Shafie et al. (2011) reported zoonotic dicrocoeliasis among a farmer's family and his domestic animals. The human infection is mainly asymptomatic or subclinical, but clinical cases of human dicrocoeliasis have also been reported from different parts of the world, including Iran (Ashrafi 2010) and Turkey (Cengiz et al. 2010). Cases of dicrocoeliasis in patients infected with the human immunodeficiency virus (HIV) have also been reported (Drabick et al. 1988; Zali et al. 2004). *D. dendriticum* eggs have also been found in the stool of a patient with Crohn's disease (Schweiger and Kuhn 2008). In Egypt, Helmy and Al-Mathal (2003) found that 121/1196 patients were positive for *D. dendriticum*.

3.2.4 Reservoir

D. dendriticum is a small lancet fluke infecting domestic and wild ruminants (e.g., buffalo, cattle, goat, sheep and deer), rabbits, and, occasionally, dogs, horses, pigs and humans. The parasite is found in the small bile duct and gall bladder of the mammalian host (Jeandron et al. 2011).

3.2.5 Transmission

Humans become infected after accidental ingestion of infected ants (containing the metacercariae)

present on fruits, vegetables, grass or in drinking water (Haridy et al. 2003).

3.2.6 Life Cycle

Life cycle of the parasite involves two intermediate hosts' viz. land snails and ants (Fig. 3.1).

3.2.7 Clinical Signs in Man and Animals

The disease is generally asymptomatic in human beings. The clinical signs and pathology is generally similar to other liver trematodes such as *Clonorchis sinensis, Fasciola hepatica,* and *Opisthorchis viverrini*, and may include chronic diarrhoea, constipation, right upper abdominal pain, vomiting, weight loss and fatigue (Jeandron et al. 2011). Hepatomegaly, peripheral eosinophilia, biliary obstruction and cholangitis could also occur (Rana et al. 2007; Jeandron et al. 2011). Acute urticaria (Singh et al. 2008) has also been reported.

3.2.8 Diagnosis

The disease is difficult to diagnose from clinical signs. The disease can be diagnosed by identification of the parasite eggs in the faecal samples, while a living or dead fluke could be rarely seen.

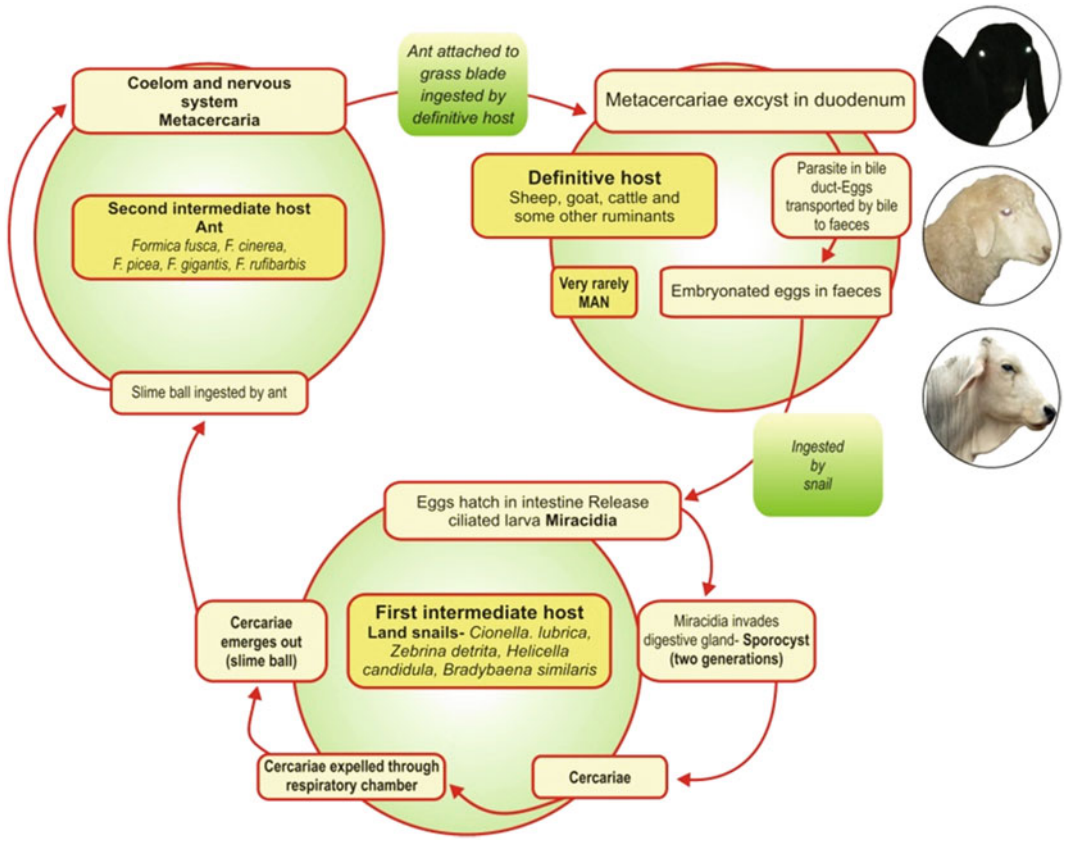

Fig. 3.1 Life cycle of *Dicrocoelium dendriticum*

3.2.9 Control

Control of intermediate host population viz. snail and ants, health education can help in the prevention of the disease.

3.3 Echinostomiasis

Subclass: Digenea
Family: Echinostomatidae

3.3.1 Common Name/Synonyms

Echinostomatidiasis

3.3.2 Etiology

These are intestinal parasites which can infect birds and mammals. There are 15 species which can infect humans (Chai et al. 2005a, b; Yamashita 1964; Huffman and Fried 1990). *Echinostoma hortense*, *Echinochasmus japonicus*, *E. echinatum* are important species from 11 fish-borne echinostomes (Chai et al. 2005a, b; Yu and Mott 1994; Chai and Lee 2002; Carney 1991; Ujiie 1963).

3.3.3 Epidemiology

Human infections have been reported from Asia, Western Pacific and Africa (Chai et al. 2005a, b; Yu and Mott 1994; Haseeb and Eveland 2002). *E. hortense* has been reported from humans and rodents in the Republic of Korea (Chai and Lee 2002) and China (Yu and Mott 1994; Yu et al. 1994). The parasite is endemic in Southeast Asia and the Far East, i.e. mainland China, Taiwan, India, Korea, Malaysia, Philippines and Indonesia (Fried et al. 2004; Bandyopathy and Nandy 1986).

3.3.4 Life Cycle

Freshwater snail act as first intermediate host where cercariae develop and may encyst as metacercariae in another snail, mollusk, tadpole or freshwater fish (second intermediate hosts) (Acha and Szyfres 2006).

3.3.5 Transmission

Ingestion of raw or undercooked second intermediate host (fish) can cause infection in human beings.

3.3.6 Clinical Signs in Man

The symptoms are generally related to the level of parasitemia (Fried et al. 2004; Bandyopathy and Nandy 1986; Chai et al. 1994). Clinical signs are generally not evident but ulcerations and bleeding in the stomach or duodenum may occur in *E. hortense* infection (Chai et al. 1994). In light to moderate infections, anaemia, headache, dizziness, slight stomach ache, gastric pain and loose stools have been reported (Chattopadyay et al. 1990; Bundy et al. 1991). Heavy infections are associated with eosinophilia, abdominal pain, profuse watery diarrhoea, anaemia, oedema and anorexia (Chattopadyay et al. 1990). Severe ulcerative lesions with bleeding in the duodenum have also been reported in some cases (Chai et al. 1994).

3.3.7 Clinical Signs in Animals

The experimental infections in rats have lead to decrease in villus/crypt ratio, villous atrophy, crypt hyperplasia and inflammation of the stroma (Chai et al. 2005a, b; Chai and Lee 2002).

3.3.8 Diagnosis

The disease can be diagnosed by identification of characteristic operculate, non-embryonated, ellipsoidal, yellow to yellow–brown eggs and by the morphology of the adult parasite (Fried et al. 2004; Bandyopathy and Nandy 1986). In rodents, the infections can be diagnosed on the basis of serological tests (Graczyk and Fried 1994, 1995; Graczyk 1997).

3.3.9 Treatment

Praziquantel @ 10–20 mg/kg in a single oral regimen can be used to treat the infections (Chai and Lee 2002).

3.3.10 Control and Prevention

The human disease can be controlled by interrupting the life cycle of the parasite (Graczyk and Fried 1998). Avoiding consumption of raw or undercooked meat of molluscs, fish, crustaceans and amphibians can help prevent the disease in humans (Graczyk and Fried 1998).

3.4 Fascioliasis

Subclass: Digenea
Superfamily: Fasciolidae

3.4.1 Common Name/Synonyms

Sheep liver fluke disease

3.4.2 Etiology

The disease occurs due to liver fluke *F. hepatica* and *F. gigantica.*

3.4.3 Epidemiology

The fascioliasis is mainly important in livestock (Malek 1980; Boray 1982). Human cases have been reported from 42 countries (Chen and Mott 1990; Esteban et al. 1998; Mas-Coma et al. 1999a, b) and up to 2.4 million people are believed to be infected with this parasite (Rim 1994; WHO 1995a). *F. hepatica* mainly occurs in Europe, Asia, Africa, Americas and Oceania. *F. gigantica* has been reported in Asia and Africa (Ashrafi et al. 2006). Human fascioliasis is becoming an important public health problem these days (Chen and Mott 1990; WHO 1995b; Mas-Coma et al. 1999a, b). Health problems due to human fascioliasis have been reported in many countries such as Bolivia, Peru, Chile, Ecuador, Cuba, Egypt, Portugal, France and Spain and Iran (Mas-Coma et al. 2004, 2005a, b).

3.4.4 Transmission

Sheep and cattle mainly act as reservoir hosts for *F. hepatica*. The infection in human beings can occur directly through drinking contaminated water or through ingestion of contaminated freshwater plants or vegetables (Chen and Mott 1990; Mas-Coma et al. 1999a; Cadel 1996).

3.4.5 Life Cycle

The parasite needs two hosts to complete their life cycle. The definitive hosts mainly include herbivorous animals such as sheep, cattle and human beings. In the definitive hosts, eggs pass through bile duct and are excreted in the faeces. Eggs develop outside the host to miracidial stage. Miracidia enters the freshwater snails. The snails of the family Lymnaeidae act as intermediate hosts. Miracidia develop to rediae in the sporocyst which further develop to cercariae which swim in water and attaches to freshwater aquatic plants. Encystation of cercaria occurs and humans and other definitive hosts become

infected after ingestion of infective metacercariae (Mas-Coma et al. 2005a, b) (Fig. 3.2).

3.4.6 Clinical Signs in Animals

There is development of haemorrhagic tracts, swelling in the liver. Fibrosis of the liver could result in bile duct blockage and inflammation which can result in death during chronic phase of the disease. The occurrence of submandibular oedema (Bottle jaw) is a typical symptom noticed in fasciolosis.

3.4.7 Clinical Signs in Man

Hepatic involvement results in inflammation and swelling of the liver. Additional symptoms include abdominal pain, anaemia, fever, cholecystitis and cholelithiasis.

3.4.8 Diagnosis

The disease can be diagnosed through clinical signs, identification of the eggs, during PM examination and by immunological tests.

3.4.9 Control

Prevention and control of animal fasciolosis, vector population can help control the disease in human beings (Roberts and Suhardono 1996; Torgerson and Claxton 1999; Spithill et al. 1999). Avoid ingestion of contaminated plants in endemic areas.

3.5 Fasciolopsiasis

Subclass: Digenea
Family: Fasciolidae

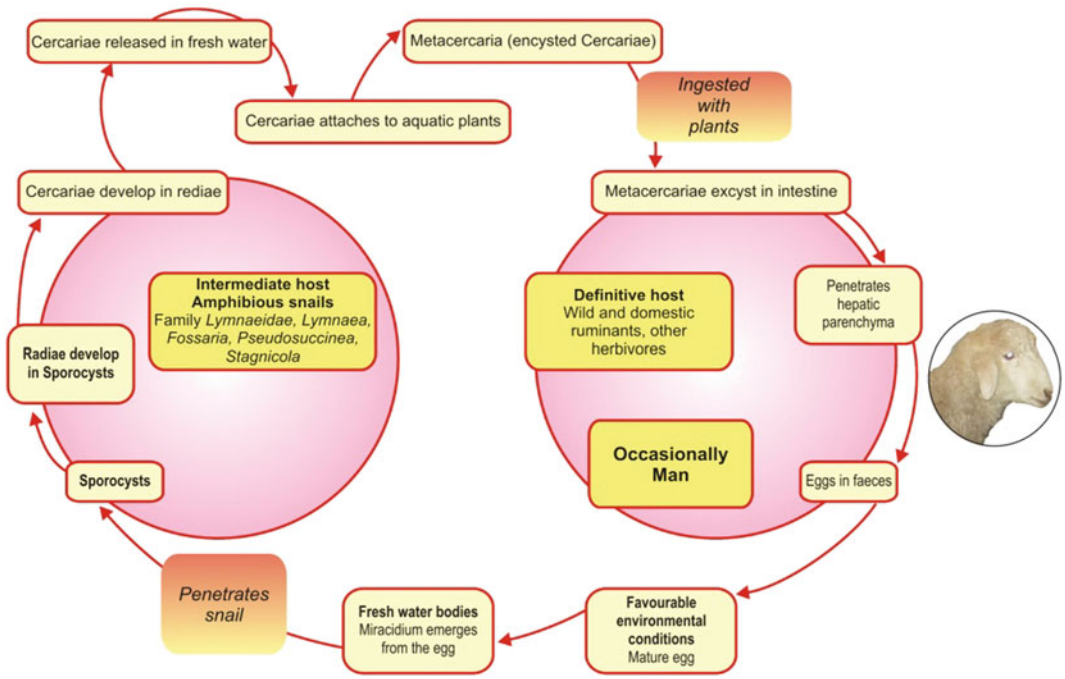

Fig. 3.2 Life cycle of *Fasciola hepatica*

3.5.1 Etiology

The disease occurs due to *F. buski,* which is the largest fluke affecting human being (Kuntz and Lo 1967).

3.5.2 Epidemiology

The parasite is prevalent in many countries of south and East Asia including Thailand, China, Taiwan, Vietnam, Bangladesh and India (Mas-Coma et al. 2005a, b; Lo and Lee 1996; Wiwanitkit et al. 2002; Gilman et al. 1982; Le et al. 2004). Fasciolopsiasis has been reviewed in detail by Mas-Coma et al. (2005a, b).

3.5.3 Reservoir

Pigs act as important reservoirs of infection (Rim 1982a, b, c).

3.5.4 Transmission

Human beings become infected through ingestion of metacercariae by drinking contaminated water or due to consumption of infected plants. Fasciolopsiasis generally occurs near freshwater habitats with stagnant or slow moving waters (Mas-Coma et al. 2005a, b). Cultivation of freshwater plants and pollution with human and animal (mainly pig) excreta are important factors for the transmission of the disease (Mas-Coma et al. 2005a, b).

3.5.5 Life Cycle

The adult parasite is present in the intestine of the human and pig host. The parasite inhabits duodenum and jejunum but could be found in other parts of the intestinal tract in heavy infections. Eggs released in the faeces embryonate in water in 3–7 weeks (Weng et al. 1989). After hatching, miracidia infect snails of the genera *Segmentina*, *Hippeutis* and *Gyraulus* which shed cercariae after 4–6 weeks (Graczyk et al. 2000). Cercariae encyst on the surface of freshwater plants (Weng et al. 1989). Ingestion of metacercariae lead to infection in human and pig hosts.

3.5.6 Disease in Man

High morbidity occurs due to fasciolopsiasis in endemic areas (Graczyk et al. 2001; Weng et al. 1989) and the disease may become fatal in heavy infections (Graczyk et al. 2001 and Weng et al. 1989). In light infections, anaemia, headache, dizziness, stomach ache, gastric pain and loose stools (Gilman et al. 1982) may be seen. Heavy infections could result in severe epigastric and abdominal pain, diarrhoea or bowel obstruction, nausea, acute ileus, anasarca and marked eosinophilia and leucocytosis (Gilman et al. 1982). Malnutrition (Graczyk et al. 2001; Weng et al. 1989) is an important factor associated with heavy infections.

3.5.7 Diagnosis

The disease may be diagnosed by faecal sample examination for presence of eggs and adult parasite (Mas-Coma et al. 2005a, b). Molecular techniques could also be used in future.

3.5.8 Control

Control can be achieved by proper diagnosis, treatment of people and livestock, preventing reinfection and instituting modern pig farming and avoiding consumption of freshwater plants (Mas-Coma et al. 2005a, b).

3.6 Gastrodisciasis

Subclass: Digenea
Family: Paramphistomatidae

3.6.1 Common Name/Synonyms

Gastrodiscoidosis, Amphistomiasis

3.6.2 Etiology

Gastrodiscoides species are approximately 8–14 mm in length (Fried et al. 2004). *Gastrodiscoides hominis* causes disease in human beings. The parasite has a pharynx, tandem, lobed testes, a post-testicular ovary, an ascending uterus and a ventral genital pore (Kumar 1999).

3.6.3 Epidemiology

The parasite has been reported primarily from India but cases have been reported from Bangladesh and East Asian countries (Acha and Szyfres 2006).

3.6.4 Transmission

The transmission of the disease occurs when raw or undercooked vegetation, crustaceans, molluscs or amphibians contaminated with encysted metacercariae are consumed.

3.6.5 Clinical Signs in Man and Animals

Swine appears to be natural definitive host. The disease condition associated with *G. hominis* is also referred to as gastrodisciasis. The flukes excyst from metacercariae and descend in the intestinal tract to reach the cecum and colon where they mature and live as adults (Kumar 1999). The parasite infects humans and hogs, e.g. hogs in Africa (Goldsmid 1975; Hira 1983) and India (Murty and Reddy 1980). Disease is generally inapparent but could cause mucoid diarrhoea (Strickland 1991).

3.6.6 Diagnosis

Morphological identification of trematode fluke and detection of parasite eggs in faecal samples can help in diagnosis of the disease.

3.7 Heterophyiasis

Subclass: Digenea
Family: Heterophyidae

3.7.1 Common Name/Synonyms

Heterophyes infection

3.7.2 Etiology

Heterophyids are minute intestinal flukes belonging to the family Heterophyidae (Chai et al. 2005a, b). These parasites infect birds and mammals (Chai et al. 2005a, b). Although 35 heterophyid species can infect man (Chai et al. 2005a, b), but from zoonotic point of view, *Metagonimus yokogawai* and *Heterophyes heterophyes* are the important species (Yu and Mott 1994). Studies have been carried out from Philippines (Belizario et al. 2001) and Thailand on *Haplorchis taichui* (Waikagul 1991; Sukontason et al. 2001) and from Korea on several species including *Heterophyes nocens* and *Metagonimus* species (Chai and Lee 1991; Chai and Lee 1990, 2002; Chai et al. 2005a, b). Aquatic snails and fresh or brackish-water fish acts as first and second intermediate hosts, respectively.

3.7.3 Metagonimiasis

The genus *Metagonimus* is identified by a laterally deviated ventral sucker and absence of ventrogenital apparatus or genital sucker (Chai and Lee 2002).

3.7.3.1 Etiology

The disease occurs due to *M. yokogawai*, *Metagonimus takahashii*, *M. minutus* and *Metagonimus miyatai.*

3.7.3.2 Transmission

The molluscan freshwater snail acts as an intermediate host (Chai and Lee 2002). The species

has rather broad fish host specificity, but the sweet fish *Plecoglossus altivelis* is an important fish host in Korea and Japan (Chai et al. 2005a, b).

3.7.3.3 Epidemiology

The disease is endemic in Korea (Chai et al. 1977; Seo et al. 1981; Chai and Lee 2002; Chai et al. 1993). The human infections have also been recorded from Siberia, Europe, China, Japan, Russia and Taiwan (Chai et al. 2005a, b; Yu and Mott 1994; Ito et al. 1991).

3.7.3.4 Clinical Signs in Man

The parasite affects the mucosa of the middle part of the small intestine (Chai 1979) and could lead to villous atrophy, crypt hyperplasia and inflammatory reactions (Chai 1979). Gastrointestinal problems such as epigastric pain, diarrhoea, anorexia and fatigue could occur in mild infections. Heavy infections may lead to abdominal cramps, malabsorption and weight loss.

3.7.3.5 Diagnosis

The disease may be diagnosed by identification of the parasite eggs by faecal examination.

3.7.3.6 Treatment

Praziquantel @ dose of 10–20 mg/kg orally may be used for treatment of the infection (Chai and Lee 2002).

Other *Metagonimus* species

Other *Metagonimus* species infecting humans include *M. takahashii Suzuki* (Chai and Lee 2002) and *M. miyatai* (Saito et al. 1997; Chai and Lee 2002) which can be differentiated from *M. yokogawai* using PCR-based-restriction fragment length polymorphism (PCR–RFLP) patterns, karyotypes, simple sequence repeat-PCR (SSR–PCR) patterns (Chai and Lee 2002) and amplification of mitochondrial cytochrome c oxidase subunit I and 28S ribosomal DNA sequences (Lee et al. 2004).

3.7.4 Heterophyiasis Due to *Heterophyes* Species

The genus *Heterophyes* is characterised by the median location of the ventral sucker and the presence of a genital sucker armed with gonotyl (Chai and Lee 2002).

3.7.4.1 Etiology

The disease occurs due to *Heterophyiasis heterophyes* (v. Siebold, 1852, Stiles and Hassall, 1900), *Heterophyes dispar* and *H. nocens* (syn. *Heterophyes katsuradai*), (Yu and Mott 1994; Chai et al. 1986; Chai and Lee 2002). The adult flukes measure 1.0–1.7 mm long, 0.3–0.4 mm wide, and possess large median ventral sucker and a genital sucker with gonotyl armed with 60–90 multidigitate spines (Beaver et al. 1984).

3.7.4.2 Epidemiology

The adult parasites are found in fish-eating birds and mammals in addition to humans (Radomyos et al. 1994; Kumar 1999). The parasite is fragmented in distribution in parts of the world (Chai et al. 2005a, b; Yu and Mott 1994). Human disease has been recorded from Egypt, Japan (Kagei et al. 1980) and Republic of Korea (Chai et al. 1986; Chai and Lee 2002).

3.7.4.3 Transmission

Consumption of raw, undercooked or inadequately processed freshwater, brackish or marine fishes (Radomyos et al. 1994) and mullet *Mugil cephalus* (Yu and Mott 1994) are important sources of infection.

3.7.4.4 Life Cycle

Dogs, cats, foxes, jackals and other fish-eating mammals act as important reservoirs (Radomyos et al. 1994). *Pirenella conica* and *Cerilbida cengulata* are the important gastropod first intermediate hosts of these heterophyids. The second intermediate hosts include brackish-water fishes in the genus *Mugil* (mullets), *Aphanius* and *Acanthogobius.*

3.7.4.5 Clinical Signs in Animals and Man

The disease is generally asymptomatic but high parasitemia could lead to irritation of intestinal mucosa, increased mucus, chronic diarrhoea, colic and nausea (Acha and Szyfres 2006). Aberrant eggs may produce granulomatous foci in myocardium and brain (Acha and Szyfres 2006).

3.7.4.6 Diagnosis

Morphological identification of adult fluke and detection of parasite eggs in faecal samples can help in diagnosis (Radomyos et al. 1994).

3.7.4.7 Prevention and control

Control measures include avoidance of eating raw or undercooked fish, personnel hygiene measures, treatment of infected people, environmental sanitation, proper excreta disposal and disease awareness.

3.7.5 Haplorchiasis

Haplorchis species have a single testis and ventrogenital sucker complex with gonotyl and chitinous spines (Chai et al. 2005a, b; Yamaguti 1958). Human disease can occur due to *H. taichui*, *Haplorchis pumilio*, *Haplorchis yokogawai*, *Haplorchiasis pleurolophocerca* and *Haplorchis vanissimus* (Yu and Mott 1994; Chai et al. 2005a, b); the first three parasites are the most important (Chai et al. 2005a, b). The *Haplorchis* species are found throughout Asia (Velasquez 1982) among humans. *H. taichui* Chen 1936 was first reported from birds and mammals from central Taiwan (Faust and Nishigori 1926). *H. pumilio* is distributed in Asia (Velasquez 1982; Yu and Mott 1994) in birds and mammals. *H. yokogawai* human infections have also been reported from many Asian countries, Australia and Egypt (Velasquez 1982; Yu and Mott 1994).

3.8 Nanophyetus

Subclass: Digenea
Family: Troglotrematidae

3.8.1 Common Name/Synonyms

Salmon poisoning (in animals), Elokomin fluke fever

3.8.2 Etiology

The disease occurs due to trematode fluke *Nanophyetus salmincola* Chapin, 1927 (syn. *Troglotrema salmincola* Witenberg 1932) belonging to the family Nanophyetidae (Chai et al. 2005a, b). *Nanophyetus schikhobalowi*, is another species described from natives of far-eastern Siberia and is regarded as a subspecies *N. salmincola schikhobalowi* (Millemann and Knapp 1970; Kumar 1999).

3.8.3 Epidemiology

Eastburn et al. (1987) recorded 10 cases of *Nanophyetus* from North West America. Ferrel et al. (1974) also detected cases of *Nanophyetus salmincola* in kippered salmon in America. The disease is endemic in the far-eastern part of Russia (Yu and Mott 1994). In USA, the prevalence in local ethnic minorities could reach up to 60 % (Eastburn et al. 1987).

3.8.4 Life Cycle

The snail *Oxytrema silicula* acts as the first intermediate host in USA and *Semisulcospira cancellata* and *S. laevigata* in Siberia. The salmonid (trout, salmon) and non-salmonid fish (Millemann and Knapp 1970) act as second intermediate hosts. The non-embryonated eggs are released in the faeces of the mammalian host. Miracidia develop within the egg in about 4 months and hatches and penetrates in the first intermediate host. Cercariae develop and emerge from the snail to penetrate skin and gills of salmonid fish. This is followed by encystation in the muscles and connective tissues of second intermediate host (Fried et al. 2004). The adult parasite develops in the intestine of definitive

host after consumption of infected fish (Schell 1985). The adult resides in the small intestine of mammals (Schell 1985).

3.8.5 Reservoir

The fishes *Oncorhynchus* species, *Salmo* species, *Cottus perplexus*, *Dicamptodin ensatus* and *Salvelinus fortinalis* transmit the infection to humans in the United States (Fried et al. 2004).

3.8.6 Transmission

Consumption of raw or undercooked infected fish is an important source of infection.

3.8.7 Clinical Signs in Man

Clinical signs include gastrointestinal symptoms such as peripheral blood eosinophilia, increased frequency of bowel movements or diarrhoea, abdominal discomfort, nausea, vomition, weight loss and fatigue (Eastburn et al. 1987).

3.8.8 Clinical Signs in Animals

The parasite can infect different mammals including dogs, cat, raccoon and foxes. The parasite also affects three species of birds on the Pacific coast of North America, Canada and Eastern Siberia (Chai et al. 2005a, b; Millemann and Knapp 1970; Beaver et al. 1984). In dogs, foxes and coyotes, this fluke is particularly important as it acts as the vector of a rickettsia, *Neorickettsia helmintheca*, which causes a serious and often fatal systemic infection known as 'salmon poisoning', which has not been reported in humans (Chai et al. 2005a, b). The mortality due to rickettsia in dogs may reach 90 % (Schell 1985).

3.8.9 Diagnosis

The disease can be diagnosed by detection of eggs typical for *N. salmincola* from stool samples. Eggs of the parasite are operculate and not embryonated.

3.8.10 Control

Avoid eating raw or undercooked fish.

3.9 Other Liver Flukes

Subclass: Digenea
Family: Opisthorchiidae

These liver flukes belong to the family Opisthorchiidae. These include clonorchiasis, opisthorchiasis and *Metorchis* infections. These parasites generally have similar life cycles and symptoms (Chai et al. 2005a, b). As per WHO, 17 million people are infected with liver flukes (WHO 1995a, b, c).

3.9.1 Clonorchiasis

3.9.1.1 Common Name/Synonyms

Chinese Liver fluke disease

3.9.1.2 Etiology

The disease occurs due to *C. sinensis.* Among all the liver flukes, *C. sinensis* is the most important fish-borne zoonosis prevalent in East Asia (Chai et al. 2005a, b; Rim 1990; Chen et al. 1994; Hong 2003; Yoshida 2012).

3.9.1.3 Epidemiology

This parasite causes serious disease in many parts of the world (Chai et al. 2005a, b) particularly in China, Japan, Korea and countries of Southeast Asia (Shekhar et al. 1995).

3.9.1.4 Reservoir

The cats, dogs and pigs are believed to act as reservoirs but their role as reservoirs has not been well studied (Chen et al. 1994; WHO 1995a, b, c).

3.9.1.5 Transmission

The definitive hosts become infected through consumption of raw or undercooked infected fish.

3.9.1.6 Life Cycle

The parasite lives in the biliary tract of definitive host (man and fish-eating animals) and sheds eggs through bile duct and intestines (Chai et al. 2005a, b). Freshwater snails (*Parafossarulus manchouricus* and other hydrobid snails) ingest these eggs. The miracidia hatches in the snail and develop into sporocysts (Rim 1982a). Rediae are formed within the sporocyst and cercariae further develop within the rediae (Chai et al. 2005a, b; Kaewkes 2003). In water, free swimming cercaria penetrates beneath the fish's scales and is transformed into an encysted metacercaria (Chai et al. 2005a, b). Many species of the freshwater fishes and shrimp can act as the second intermediate host (Chen et al. 1994; Park et al. 2004). The metacercariae develop into the adult in the definitive host (Rim 1986). Humans and fish-eating mammals and birds can act as definitive hosts (Fig. 3.3).

3.9.1.7 Clinical Signs in Animals and Man

Most important symptoms include calculi formation, cholangitis, cholecystitis and cholangiohepatitis. Infection of pancreas could lead to pancreatitis or obstruction of pancreatic duct. Chronic infections can also lead to cholangiocarcinoma (Flavell 1981; Chai et al. 2005a, b).

3.9.1.8 Diagnosis

The disease can be diagnosed by multiple stool examinations for identification of parasite eggs in faeces. The eggs may also be diagnosed from duodenal aspirates. Specialised techniques such as cellophane thick smear (Chai et al. 1982) or Kato-Katz techniques (Hong et al. 2003) may be used. Serological tests such as ELISA may also be done (Choi et al. 2003).

3.9.1.9 Control

The prevention and control relies on avoiding consumption of raw or undercooked fish, vector control and prompt diagnosis and treatment of the infection.

3.9.2 Opisthorchiasis

Four species of the parasite viz. *O. viverrini, Opisthorchis felineus, O. noverca* and *O. guayaquilensis* could infect humans (Kaewkes 2003). Out of these, *O. viverrini* is prevalent in Southeast Asia (Chai et al. 2005a, b; Shekhar et al. 1995) and particularly causes serious disease (Chai et al. 2005a, b; Rim 1982b; Kaewkes 2003). *O. felineus* is a parasite of dogs, cats, foxes and pigs in Europe and the Asiatic parts of Russia, Turkey and Siberia (Chai et al. 2005a, b; Beaver et al. 1984; Rim 1982b). The life cycles, symptoms and diagnosis are similar to that of *C. sinensis.*

3.9.3 Metorchiosis (Canadian Liver Fluke)

The disease occurs due to *Metorchis conjunctus.*

The parasite is found in carnivorous animals in Canada and USA (MacLean et al. 1996). Human infections have also been reported in Canada (Yamaguti 1958; MacLean et al. 1996; Behr et al. 1998). Mode of transmission, symptoms are similar as for other liver fluke infections (Rim 1982b; Beaver et al. 1984). Carcinoma of the infected parts may lead to death in some cases.

3.10 Paragonimiasis

Subclass: Digenea
Family: Troglotrematidae

3.10.1 Common Name/Synonyms

Lung fluke disease, pulmonary distomatosis, endemic hemoptysis and oriental hemoptysis.

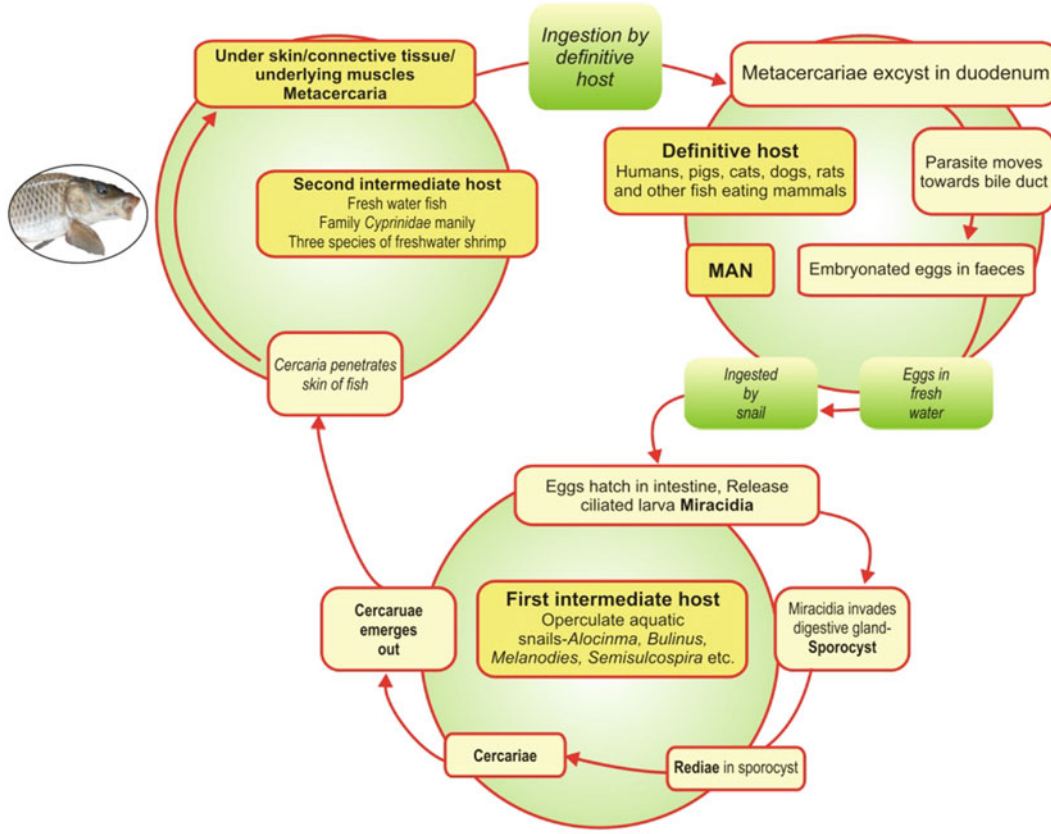

Fig. 3.3 Life cycle of *Clonorchis sinensis*

3.10.2 Etiology

The disease primarily occurs due to *Paragonimus westermani*, lung fluke belonging to the family Troglotrematidae. *Paragonimus skrjabini* is another species found in China but few parasites develop into adult in man (Liu et al. 2008).

3.10.3 Epidemiology

The parasite is found in Asia, Africa and South America. The disease has been recorded from many countries such as China, Japan, Liberia, Nigeria and Venezuela (Obara 2004; Liu et al. 2008).

3.10.4 Reservoir

The disease could occur in carnivorous animals such as tiger, lion, dogs, cats, pigs and non-human primates.

3.10.5 Transmission

Human beings become infected after consumption of infected crabs or crayfish (Singh 1986).

3.10.6 Life Cycle

Humans and carnivores animals can act as definitive hosts for *P. westermani*. Intermediate hosts include snails (importantly species

Semisulcospira, *Melanopides*) and crabs or crayfish (Velez 2003). In humans and carnivores animals, lungs are important site of predilection and harbour the adult parasite (Liu et al. 2008; Guan 2005). The eggs are coughed up from the respiratory system and passed in faeces. Contamination of freshwater with faeces of the definitive host leads to embryonation and hatching of the eggs. Free swimming miracidia enters the first intermediate host. Miracidia develops to sporocyst to redia to cercaria in the first intermediate host. Crabs or cray fish ingests the snail and cercariae encysts and develops into metacercariae in the vessels of the gills, liver and muscles of the cephalothorax. Humans and carnivores animals become infected after the ingestion of raw or undercooked infected crustacean host. Metacercariae excyst in intestinal tract and reach through abdominal cavity to diaphragm to pleural cavity and finally to the lungs (Obara 2004; Guan 2005) (Fig. 3.4).

3.10.7 Clinical Signs in Animals

The involvement of respiratory system may lead to formation of granulomata and abscess cavities.

3.10.8 Clinical Signs in Man

The respiratory system infections could result in symptoms such as chest pain, cough, blood-stained sputum and dyspnea. If gastrointestinal

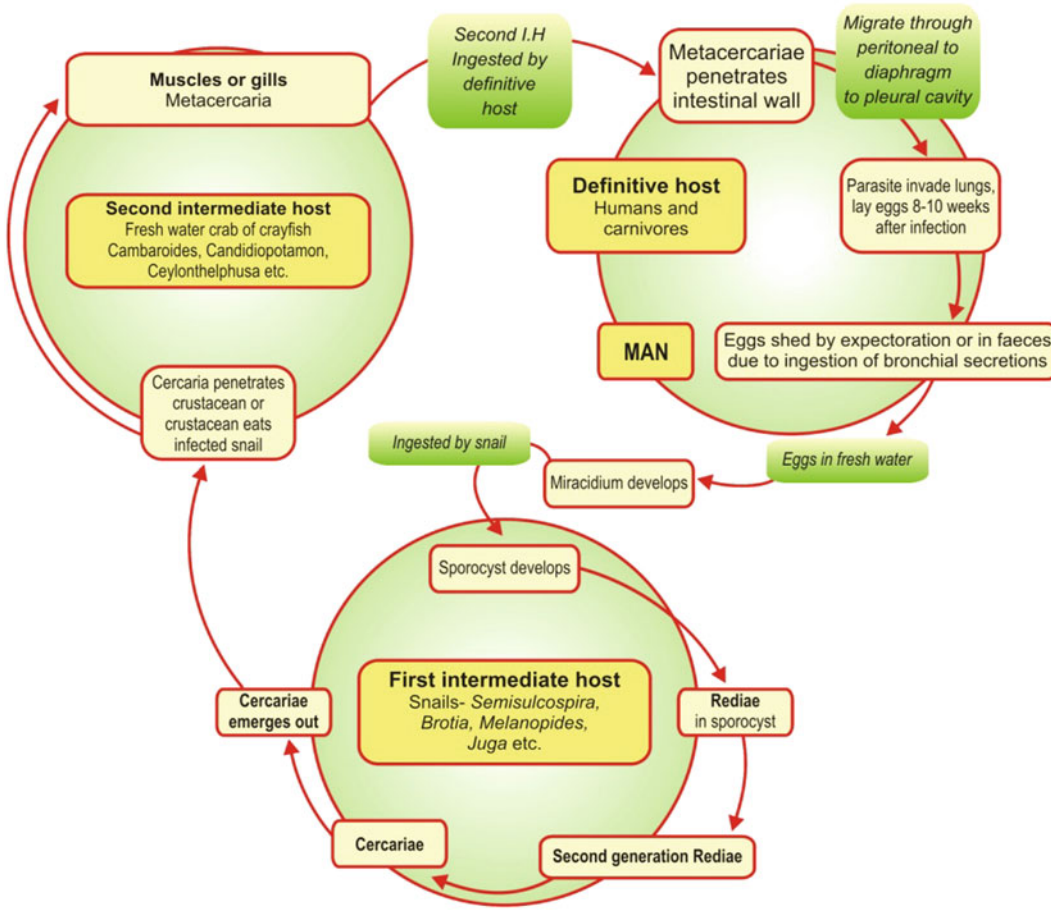

Fig. 3.4 Life cycle of *Paragonimus westermani*

system is infected, symptoms include abdominal pain, or diarrhoea and eggs can be seen in the stools (Waikagul 1989). Extrapulmonary paragonimiasis mostly result in involvement of central nervous system (Huang 2000).

3.10.9 Diagnosis

The disease may be diagnosed from clinical signs, detection of eggs from sputum or faeces and CT, MRI or serological techniques.

3.10.10 Control

Control measures include avoiding consumption of raw or undercooked crab or cray fish, control of intermediate host (snail) populations and avoiding contamination of surface water with faeces of definitive host (Liu et al. 2008).

3.11 Schistosomiasis

Subclass: Digenea
Order: Strigeidida
Family: Schistosomatidae

3.11.1 Common Name/Synonyms

Bilharziasis, snail fever

3.11.2 Etiology

The disease occurs due to *Schistosoma haematobium* (urinary schistosomiasis), *S. mansoni, S. japonicum, S. mekongi* and *S. malayi,* (gastrointestinal and hepatosplenic schistosomiasis) and *S. intercalatum,* which also affects the GI tract but with lower morbidity (Almeda et al. 1994; Corachan 2002).

3.11.3 Epidemiology

More than 300 million people are believed to be infected worldwide, the parasite being more common in children aged between 5 to 15 years (WHO 1993; Standley et al. 2009; Fenwick et al. 2009). Schistosomiasis has been reported in most African countries, limited areas of South America, Caribbean, Middle East and Asia (Corachan 2002; Standley et al. 2012; Hotez et al. 2008). *Schistosoma japonicum* is the causative agent of schistosomiasis in China, Philippines and in small pockets of Indonesia (Gray et al. 2008; Ross et al. 2001, 2002). In Africa, the public health burden of schistosomiasis, caused by *S. mansoni*, *S. haematobium* and *S. intercalatum/guineensis*, is enormous (Hotez and Fenwick 2009). The losses occurring due to human schistosomiasis caused by *S. mansoni*, *S. haematobium* and *S. japonicum* have been estimated to be 1.7–4.5 million disability adjusted life years (McManus et al. 2010) (DALYs). The true burden was found to be substantially greater than previous estimates (Finkelstein et al. 2008; Jia et al. 2007; King et al. 2005; Van der Werf et al. 2003). In China, zoonotic schistosomiasis japonica, also called "snail fever," is a significant health risk for human and livestock populations (McManus et al. 2010, 2009; Ross et al. 2001; Utzinger et al. 2005; Wang et al. 2006; Zhou et al. 2005, 2007).

3.11.4 Reservoir

The zoonotic nature of *S. japonicum* further complicates parasite transmission and control. The parasite has ability to infect more than 40 species of wild and domestic animals (WHO 1993). Earlier studies indicated public health importance of rats, dogs, pigs, sheep and goats, cattle and water buffalo hosts (Johansen et al. 2000; WHO 1993). In China, however, water buffaloes (*Bubalus bubalis*) could play a significant role in the transmission of *S. japonicum* (Johansen et al. 2000; WHO 1993; Gray et al. 2008).

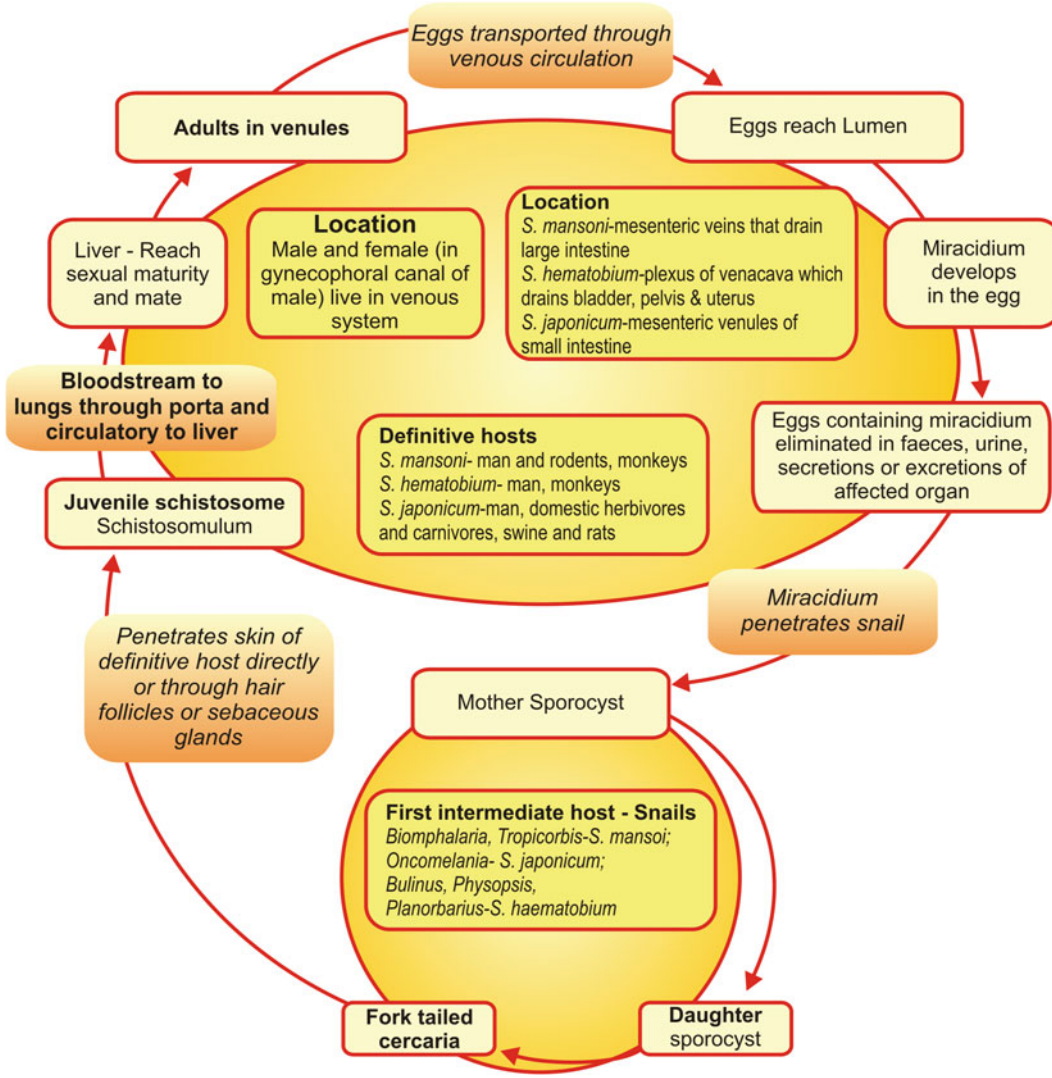

Fig. 3.5 Life cycle of *Schistosoma species*

3.11.5 Transmission

Human beings become infected after exposure to freshwater containing cercariae released by the infected snails (Corachan 2002). Snails act as the intermediate host for the parasite (Corachan 2002).

3.11.6 Life Cycle

Freshwater infected with free-swimming cercariae is important source of infection. The cercariae penetrate the skin of humans and many other animals, including water buffaloes, sheep, pigs, cattle, rodents and dogs, which can act as

important reservoirs for human infection (Ross et al. 2001; Zhou et al. 2007) (Fig. 3.5).

3.11.7 Clinical Signs in Man

The disease is generally asymptomatic in human beings. In infections due to *S. japonicum, S. mansoni;* early lesions occur in small intestine followed by portal hypertension due to *Schistosoma* eggs retained in the portal spaces, ascitis and spleenomegaly. Clinical symptoms due to *S. haematobium* schistosomiasis include haematuria, proteinuria and leukocyturia accompanied by dysuria and nocturia (Corachan 2002). Chronic infections could lead to obstructive uropathy and hydronephrosis (Corachan 2002). *S. japonicum* infection may lead to fever, headache and lethargy, portal hypertension, ascites and hepatosplenomegaly, which can cause premature death (Ross et al. 2002).

Rare CNS involvement due to embolization of eggs to the brain and spinal cord may occur in *S. japonicum* and *S. mansoni* infections (Alfred 1996). Early infection in childhood can cause growth retardation and anaemia (Ross et al. 2002).

3.11.8 Clinical Signs in Animals

The disease is common in animals. The symptoms generally are similar to as that in man.

3.11.9 Diagnosis

The disease can be diagnosed by detection of eggs in human stool samples, tissue biopsy examinations or ultrasonography (Corachan 2002), clinical signs, serological or molecular techniques. The eggs of *S. japonicum* are round and have a shell with reduced lateral spine (McManus et al. 2010).

3.11.10 Control

Control measures include health education, control of snails, water hygiene and treatment of infected cases.

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Cestode Zoonoses

4

Abstract

Cestode Zoonoses could be defined as "those cestode diseases which are naturally transmitted between (other) vertebrate animals and man." The zoonotic cestodes belonging to family *Taeniidae* are of paramount importance in the developing world. Most of the food producing animals viz. cattle, buffalo, sheep, goat and pigs along with some other mammals act as intermediate hosts for *Echinococcus granulosus.* Human beings could become infected after accidental consumption of *Echinococcus* species eggs shed in the faeces of the definitive carnivorous host/animal. Taeniasis is a true zoonotic infection (Euzoonoses) where pig and cattle act as intermediate host for *Taenia solium* and *Taenia saginata*, respectively and human beings act as definitive/final hosts. Neurocysticercosis is an important public health issue and is responsible for neurological disorders worldwide. Besides important animal and human health concern; the economic losses arising due to these infections are enormous. Coenurosis is a rare zoonosis and more than 100 human cases have been reported across the globe. Other important cestodes include parasites such as *Diphyllobothrium latum* and *Spirometra mansoni*. Different epidemiological trends have been reported across the globe for diphyllobothriasis. The parasite *Dipylidium caninum* can cause disease in domestic dogs, cats, some wild carnivores and occasionally man. Sparganosis is a rare cestode zoonosis that can infect man. The parasite has been recorded across the globe but is commonly found in eastern Asia. The important cestodes transmitted through food and water includes *T. solium, E. granulosus* and *D. latum.*

B. B. S. Dhaliwal and P. D. Juyal, *Parasitic Zoonoses*,
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4.1 Coenurosis

Order: Cyclophyllidea
Family: Taeniidae

4.1.1 Common Name/Synonyms

Gid, Sturdy, Vertigo

4.1.2 Etiology

The intermediate stages (larval forms) of the parasites *Taenia multiceps*, *Taenia serialis* and *T. brauni* viz. *Coenurus cerebralis, C. serialis*, *C. brauni*, respectively are responsible for the disease in intermediate hosts including man.

4.1.3 Epidemiology

Coenurosis is a rare zoonosis and more than 100 human cases have been reported across the globe (Scala and Varcasia 2006). Human coenurosis is rare in occurrence but cases have been reported from Africa, Asia, Europe and America (Johnstone and Jones 1950; Templeton 1971; Ing et al. 1998; Scala and Varcasia 2006; Benifla et al. 2007; El-on et al. 2008).

4.1.4 Life Cycle

The human disease occurs due to the larval form of *T. multiceps*. The canids (dogs, foxes, coyotes) act as the definitive host for *T. multiceps* and the herbivorous animals primarily sheep, act as intermediate hosts. For *T. serialis*, lagomorphs and rodents act as intermediate hosts. Wild rodents act as intermediate hosts for *T. brauni*. Humans could act as accidental intermediate hosts after ingestion of eggs of the parasite present in the faeces of the definitive host (Fig. 4.1).

4.1.5 Clinical Signs in Man

In human beings, the cysts (larval forms) have been found in CNS, eye and other tissues and symptoms vary depending upon the location and size of the cyst.

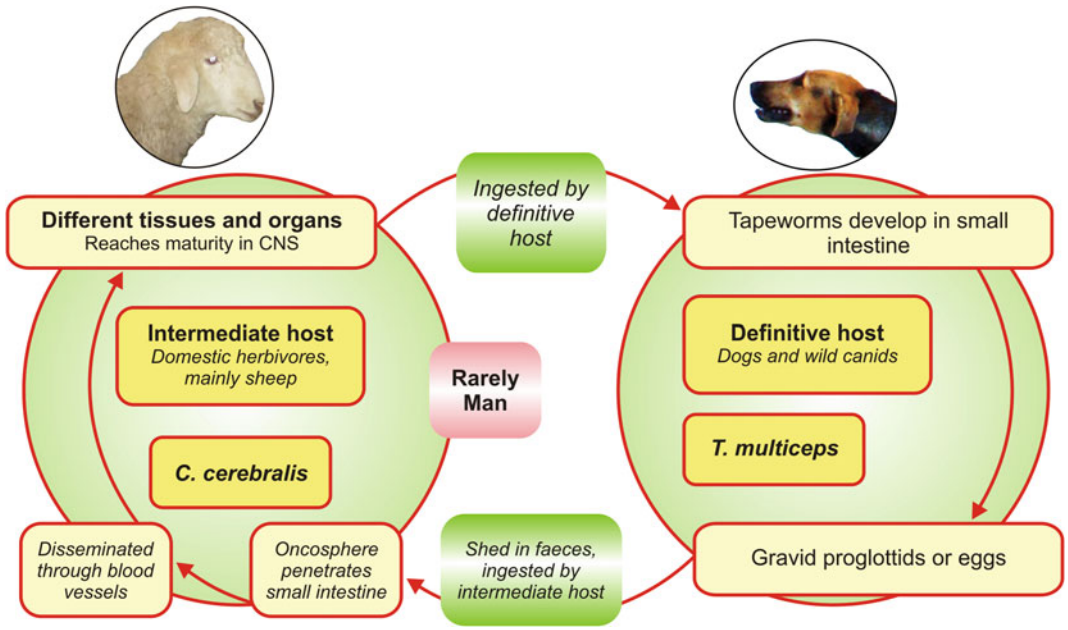

Fig. 4.1 Life cycle of Taenia multiceps

4.1.6 Clinical Signs in Animals

In sheep, the parasite affects nervous system and cysts are found in CNS resulting in nervous symptoms which may lead to death of the animal after few weeks. The disease can also occur in goat, cattle and horses.

4.1.7 Diagnosis

The disease can be diagnosed in humans using computed tomography and magnetic resonance imaging. In definitive host, disease can be diagnosed by identification of the parasite (Figs. 4.2 and 4.3).

4.1.8 Control

Control of stray dogs, avoiding free access of these stray animals to slaughter waste, deworming of the definitive host and improved personnel hygiene can help prevent the disease in human beings.

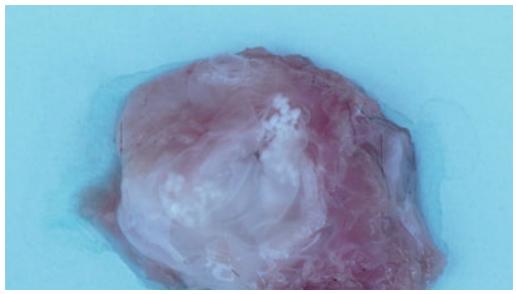

Fig. 4.2 *Coenrus* cyst in thigh muscle of goat

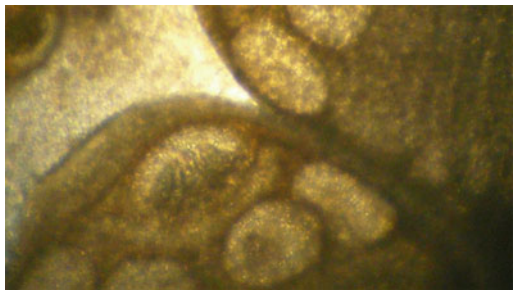

Fig. 4.3 Borax carmine stained multiple scolices of *T. multiceps*

4.2 Diphyllobothriasis

Subclass: Cestoda
Order: Pseudophyllidea

4.2.1 Common Name/Synonyms

Fish tapeworm infection, Bothriocephaliasis

4.2.2 Etiology

The disease occurs due to the tapeworm of the species *Diphyllobothrium*. *Diphyllobothrium latum* is the most important species responsible for human zoonosis followed by *D. dendriticum*, *Diphyllobothrium dalliae*, *Diphyllobothrium Klebanovskii, Diphyllobothrium nihonkaiense* and few other species (Scholz et al. 2009).

4.2.3 Epidemiology

The parasite is found in cold water areas of Europe, Asia, North and South America and has also been reported from Japan, Russia, Korea and many other countries (Dick et al. 2001). There is a decline in prevalence of the disease from North America, Asia and Europe (Scholz et al. 2009) and reemergence from countries such as Russia, South Korea, Japan and South America has been reported (Scholz et al. 2009). Among humans,
D. latum and *D. dendriticum* have been reported from holarctic region, and from circumpolar areas and South America, respectively (Torres et al. 2004). Other species such as *D. nihonkaiense, D. klebanovskii,* and *Diphyllobothrium pacificum* have been recorded in Japan, eastern Eurasia and along the Pacific Coast of South America (Chai et al. 2005). These parasites have also been reported from Middle East and Malaysia (Abo-Shehada and Ziyadeh 1991;

Rohela et al. 2002), Republic of Korea (Lee et al. 2001). Domestic animals, especially dogs (Adams and Rausch 1997; Torres et al. 2004) and faecal contamination of waterbodies (Lloyd 1998; Cross 2001; Torres et al. 2004; Dupouy-Camet and Peduzzi 2004) also help to complete life cycle of the parasite (Dick et al. 2001). Although decline in human disease has been observed, but the parasite is prevalent in the other hosts and is a permanent risk for human infection (Dick et al. 2001; Chai et al. 2005).

4.2.4 Transmission

In man, the disease occurs due to consumption of raw or undercooked fish containing the plerocercoid stage of the parasite.

4.2.5 Life Cycle

The eggs are released into the faeces of the definitive host (man and other fish eating mammals). The eggs hatch in the water to form coracidium (Von Bonsdorff 1977). The coracidium is ingested by copepod which acts as the first intermediate host (Eguchi 1973; Magath 1937; Torres et al. 2007). Procercoid develops in the first intermediate host (Chervy 2002). Fish (second intermediate host) become infected after the ingestion of infected copepod (first intermediate host). The plerocercoids develop from procercoid in the tissues of the fish (Chervy 2002). Ingestion of raw or improperly cooked fish containing plerocercoids (larval stages) lead to the infection in carnivore mammals/fish-eating birds/human beings (Bylund and Andersen 1994). The parasite requires three hosts for completion (Rausch and Adams 2000) of its life cycle. Parasite could develop at different tissues in the fish depending upon the species of the parasite involved (Dick et al. 2001) (Fig. 4.4).

4.2.6 Clinical Signs in Man

Most of the times, disease remains asymptomatic or cause mild symptoms such as abdominal discomfort and diarrhoea. Clinically, the parasite may cause megaloblastic anaemia due to deficiency of vitamin B12.

4.2.7 Clinical Signs in Animals

The disease normally remains asymptomatic in dogs and cats but very severe infection could lead to death.

4.2.8 Diagnosis

The disease can be diagnosed by identification of the eggs of the parasite (ovoid shape, operculum on narrow pole, 35–80 by 25–65 μm), or using PCR.

4.2.9 Control

The disease can be controlled by breaking the life cycle of the parasite. Avoid contamination of water bodies. Prompt treatment of infected cases and consumption of properly cooked fish could help prevent the infection in human beings. Sewage to be discharged in water bodies must be properly treated before discharge.

4.3 Dipylidiosis

Order: Cyclophyllidea
Family: Dilepididae

4.3.1 Common Name/Synonyms

Dog tapeworm infection

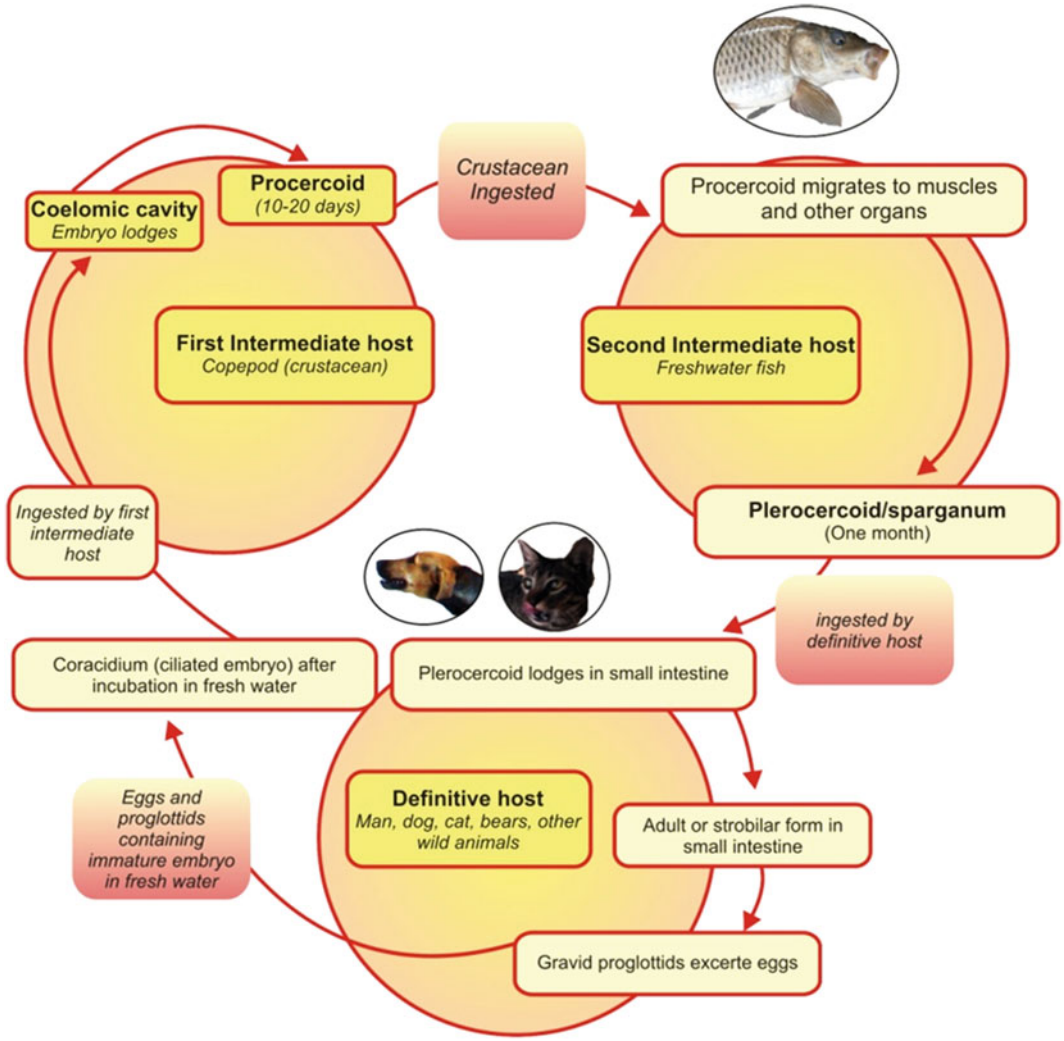

Fig. 4.4 Life cycle of Diphyllobothrium latum

4.3.2 Etiology

The disease occurs due to *Dipylidium caninum* (cyclophyllidean cestode). The parasite *D. caninum* (Linnaeus 1758, Cyclophyllidea, Dipylidiidae) can cause disease in domestic dogs, cats, some wild carnivores, and occasionally human beings (Schmidt and Roberts 2000).

4.3.3 Epidemiology

The disease occurs across the globe and human cases have been reported in many European countries, India, China, Japan, etc. The human infection mostly occurs in young children as they often play with dogs and cats (Marx 1991).

4.3.4 Life Cycle and Transmission in Man

Intermediate hosts include cat and dog (*Ctenocephalides felis* and *Ctenocephalides canis*) flea but dog lice (*Trichodectes canis* and *Heterodoxus spiniger)* and human flea (*Pulex irritans)* can also transmit the parasite (Craig and Ito 2007; Chappell and Penn 1990). Definitive hosts (dogs and cats) become infected after ingestion of the flea and lice infected with cysticercoids (Robertson and Thompson 2002). Accidental infection occurs in human beings after ingestion of the infected cat and dog flea or food contaminated with infected flea (Turner 1962). The intermediate hosts become infected after ingesting contaminated animal faeces (Fig. 4.5).

4.3.5 Clinical Signs in Man

The disease has been reported from infants and young children (Tsumura et al. 2007; Reddy 1982; Claudia et al. 2003; Reid et al. 1992). In human beings, the disease is rarely seen and often remains asymptomatic but symptoms such as abdominal pain, diarrhoea and increased irritability of anus and intestinal obstruction may be seen. Proglottids (rice grain like) are excreted in the faeces of the host.

4.3.6 Clinical Signs in Animals

The infection generally remains asymptomatic in dogs, but few typical signs such as crawling and rubbing around the anus could result due to perianal pruritus (Dantas-Torres 2008). Proglottids (rice grain like) can also be seen around perianal area (Dantas-Torres 2008).

4.3.7 Diagnosis

The disease can be diagnosed after microscopic identification of parasite eggs or observing rice grain-like proglottids in faeces. The gravid proglottids contain eggs packed in embryonic membrane. The eggs are 20–40 μm in diameter and contain a hexacanth embryo (Neira et al. 2008; Ramana et al. 2011).

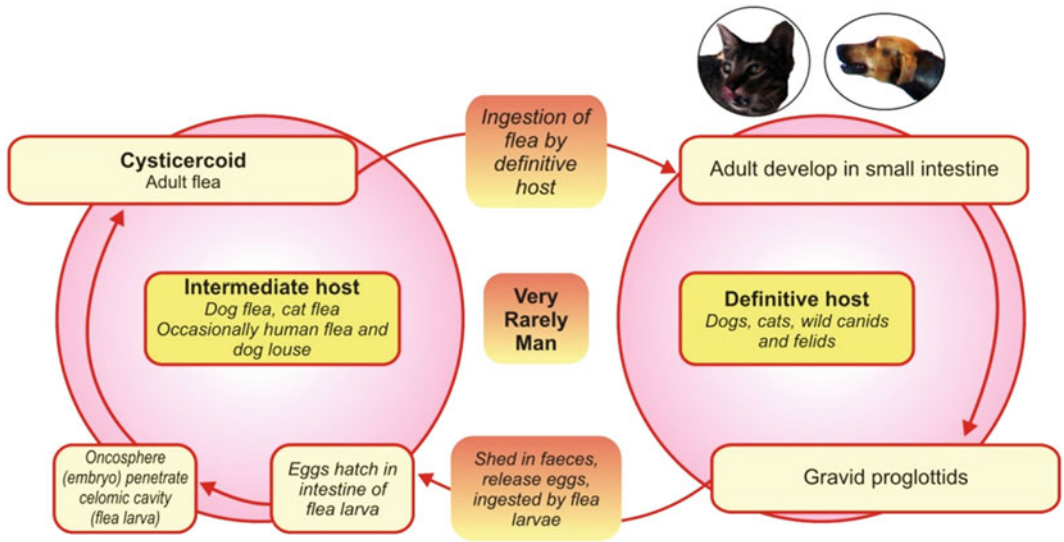

Fig. 4.5 Life cycle of *Dipylidium caninum*

4.3.8 Control

Control of the flea vectors from dog, cat and their surroundings, personnel hygiene (washing hands of children after they play with pets), proper disposal of cat and dog faeces and regular deworming of pet animals can help control the infection.

4.4 Echinococcosis

Class: Cestoda
Order: Cyclophyllidea
Family: Taeniidae

4.4.1 Common Name/Synonyms

Hydatidosis, dwarf dog tapeworm

4.4.2 Etiology

Echinococcosis is a very important cestode zoonosis. The infection occurs due to the parasites belonging to the species *Echinococcus* (Family *Taeniidae)*. *E. granulosus*, *Echinococcus multilocularis, Echinococcus oligarthrus* and *Echinococcus vogeli* (Rausch 1975 and Thompson 1995) are the species of the genus *Echinococcus* responsible for the disease. Among these, *E. granulosus* zoonosis is particularly significant due to many domestic, man-made life-cycle patterns and involvement of number of intermediate hosts (Eckert 1998; Schantz et al. 1995). Further, there are nine recognised strains of *E. granulosus* (Thompson et al. 1995). The metacestode infection is commonly known as hydatidosis.

4.4.3 Epidemiology

E. granulosus is worldwide in occurrence (Eckert et al. 2001; Singh et al. 2012a, b, Juyal et al. 2005; Bhatia and Pathak 1990) and human cases have been reported throughout the world whereas *E. multilocularis* is mainly prevalent in northern hemisphere. The other two parasites are found in central and south America (Acha and Szyfres 2006).

4.4.4 Transmission

The carnivorous animals act as definitive hosts, and shed eggs in their faeces. The ingestion of these eggs by the intermediate host animals and occasionally man leads to development of metacestode stage in their visceral organs primarily liver and lungs (WHO and OIE 2001).

4.4.5 Life Cycle

Most of the food producing animals viz. cattle, buffalo, sheep, goat and pigs along with some other mammals act as intermediate hosts for *E. granulosus* (Rausch 1986; Rausch 1995; Thompson and Allsopp 1988; WHO 1984). Dog and foxes act as definitive hosts for *E. granulosus* and *E. multilocularis,* respectively for most of the times.

E. granulosus—Definitive host: Dogs and wild canids
Intermediate host: Sheep, pig, cattle, buffalo, goat, horses, camels, other herbivorous animals and man
E. multilocularis—Definitive host: Arctic fox (*Alopex lagopus*) and red fox (*Vulpes vulpes*)
Intermediate host: Wild rodents
E. oligarthrus—Definitive host: Wild felids (Jaguars, Pumas etc.)
Intermediate host: Rodents
E. vogeli—Definitive host: Wild canid (*Speothos venaticus*)
Intermediate host: Rodent *Cuniculus paca*

Life cycle of other *Echinococcus* species is almost similar to *E. granulosus* involving their respective intermediate and definitive hosts (Fig. 4.6).

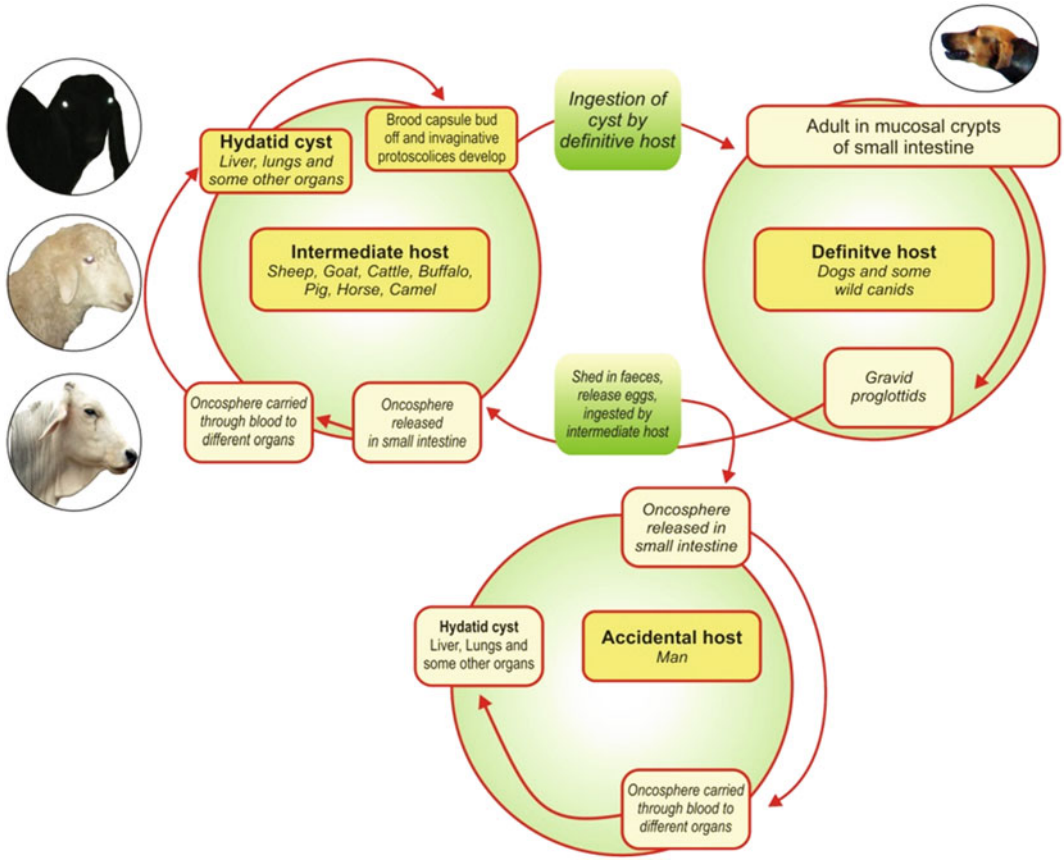

Fig. 4.6 Life cycle of *Echinococcus granulosus*

4.4.6 Clinical Signs in Man

Human beings could become infected after accidental consumption of eggs of the parasite shed in the faeces of the definitive carnivorous host/animal. Depending upon the parasite involved, three types of the disease could occur in human beings (WHO and OIE 2001).

Cystic echinococcosis (CE): The disease occurs due to metacestode of *E. granulosus*. *C. echinococcosis* is an important disease problem in many parts of the world (WHO and OIE 2001). Unilocular cysts occur in liver, lung and few other organs.

Alveolar echinococcosis (AE): The disease occurs due to metacestode of *E. multilocularis* and produces alveolar hydatidosis. Although rare, but disease is malignant and highly fatal among infected human beings.

Polycystic echinococcosis (PE): The disease occurs due to metacestode of *E. vogeli* or *E. oligarthrus*.

The clinical signs in these forms vary depending upon the type of visceral organ involved. Liver and lungs are parasitised most of the times and the related clinical signs are seen accordingly.

4.4.7 Clinical Signs in Animals

4.4.7.1 Intermediate Hosts

The symptoms generally do not appear and the domestic animals remain asymptomatic throughout their life, but the disease causes significant economic losses in most of the food producing animals at the time of slaughter due to

condemnation of infected organs. Large-sized hydatid cysts can cause symptoms in the animals due to pressure atrophy of the affected organ(s) (Schwabe 1986). The cysts are mainly found in liver and lung but can be present in spleen, heart, kidney and other organs (Orlando 1997; WHO and OIE 2001). The cysts are responsible for reduced growth and milk production especially in dairy animals.

4.4.7.2 Definitive Host

The infected carnivorous animals generally do not present any clinical symptoms.

4.4.8 Diagnosis

In intermediate host animals, the disease can be diagnosed at the time of post mortem inspection of the infected organs, radiographic techniques, ultra sonography, PCR and serological techniques. In carnivorous animals, detection of eggs using conventional parasitological techniques and later confirmation by PCR and serological techniques can be used. In human beings, radiography along with serological confirmation is routinely used for diagnosis of the parasite (Figs. 4.7, 4.8, 4.9, 4.10, 4.11, 4.12 and 4.13).

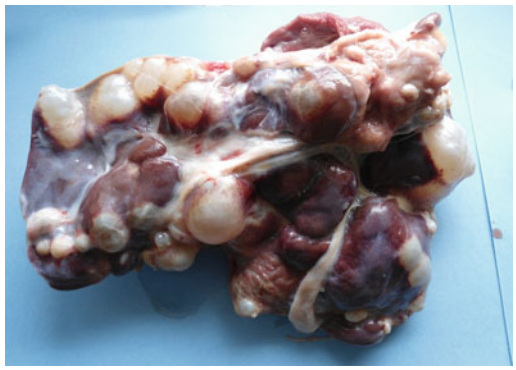

Fig. 4.7 Hydatid cysts in liver of sheep

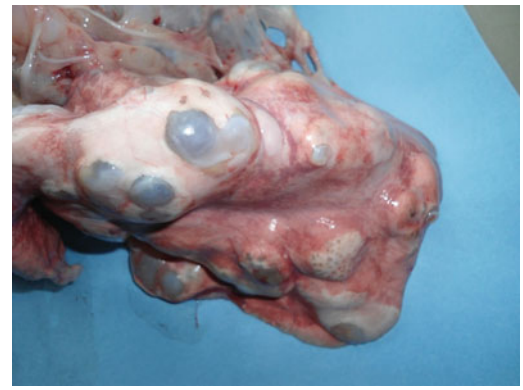

Fig. 4.8 Hydatid cysts in lungs of sheep

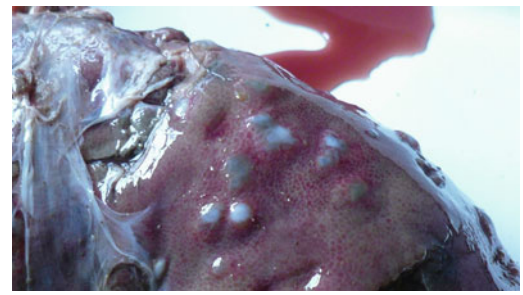

Fig. 4.9 Hydatid cysts in liver of pig

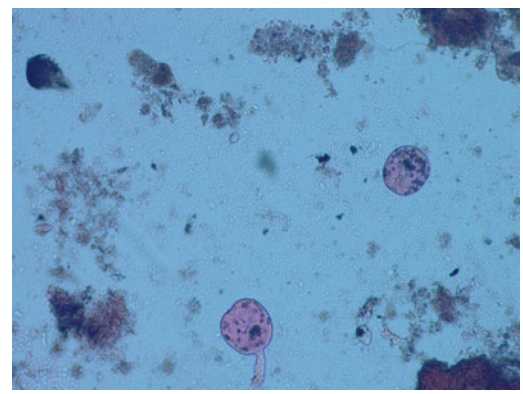

Fig. 4.10 Fully mature invaginative forms of protoscolices at 10 X

4.4.9 Control

Control of stray dogs, prevention of illegal slaughter (with free access to dogs) and treatment of infected dogs are important steps to be taken

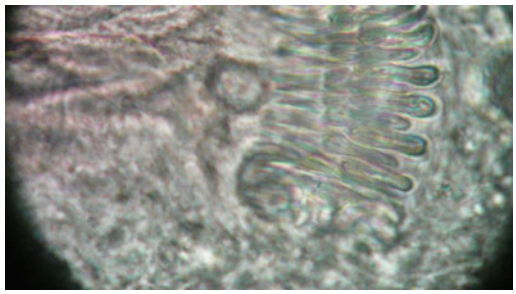

Fig. 4.11 Alternate arrangement of large and small hooks in the rostella (*E. granulosus*) at 100X

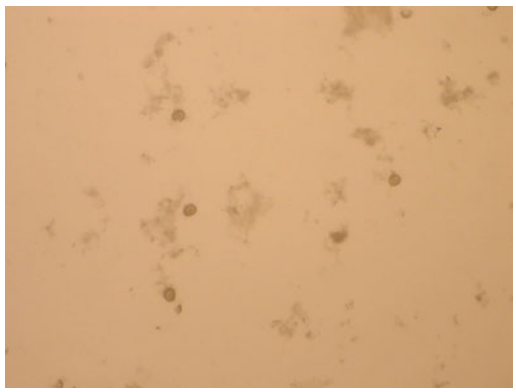

Fig. 4.12 *Taenia* spp. eggs seen at 10X

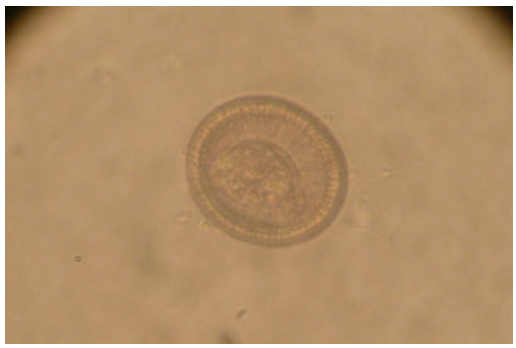

Fig. 4.13 *Taenia* spp. eggs at 100X

particularly for the control of *E. granulosus* related echinococcosis.

4.5 Sparganosis

Subclass: Cestoda
Order: Pseudophyllidea
Family: Diphyllobothriidae

4.5.1 Common Name/Synonyms

Spirometrosis, Larval diphyllobothriasis

4.5.2 Etiology

Several species of the *Spirometra*, such as *Spirometra mansoni*, *S. mansonoides* and *S. erinaceieuropaei* can cause the disease.

4.5.3 Epidemiology

Sparganosis is a rare cestode zoonosis that can infect man. The parasite has been recorded across the globe but is commonly found in eastern Asia (Viroj 2005) viz. countries such as China, Korea, Japan, and from Southeast Asia. The parasite has also been reported from a domestic cat in India (Saleque et al. 1989).

4.5.4 Life Cycle

Humans can act as accidental secondary intermediate host where plerocercoid (metacestode stage) develop and results in sparganosis (Beaver et al. 1984; Andersen 1983; Cho et al. 1990; Tesacharoen 1980). Frogs act as the second intermediate hosts. Ingestion of larvae from cyclops (copepod; first intermediate host) or frogs can lead to infection in humans, pigs and mice (Fukushima and Yamane 1999; Nithiuthai et al. 2004). Second stage larvae (spargana) during their migration in these hosts (reptiles-snakes; mammals such as pigs and human beings) cause the disease (Mueller 1974; Bearup 1953; Daly 1982; Acha and Szyfres 1987). The animals belonging to the family canidae and felidae act as definitive hosts (Fig. 4.14).

4.5.5 Transmission

The disease is transmitted to man through ingestion of infected copepods in drinking water,

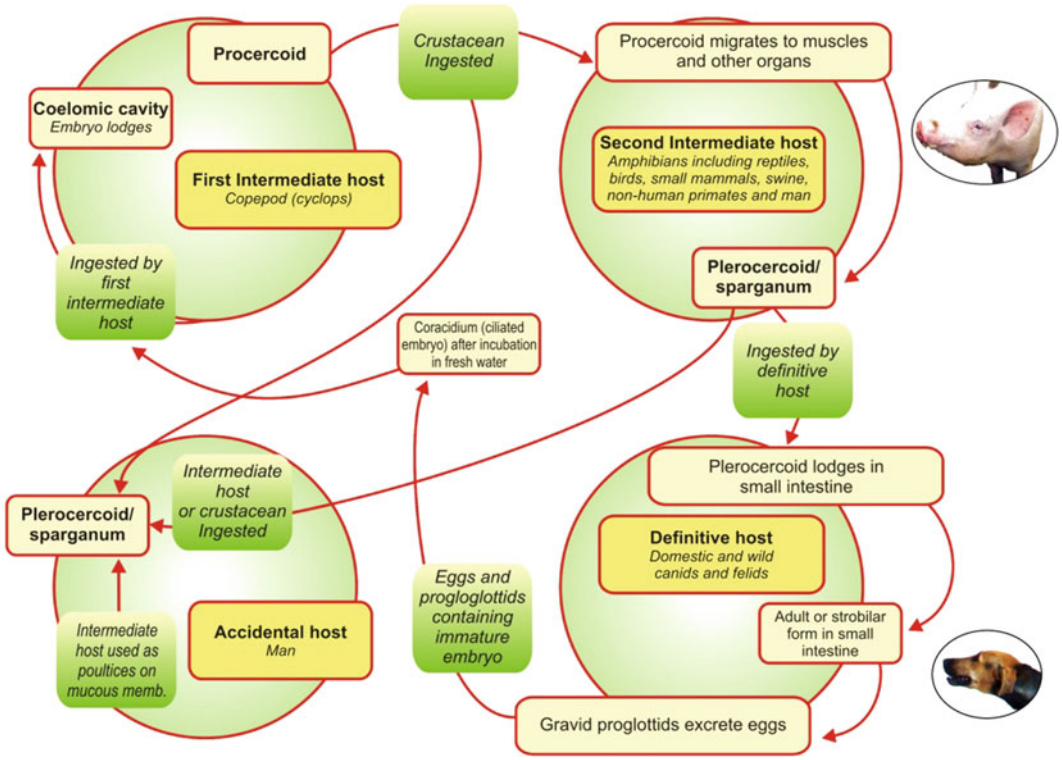

Fig. 4.14 Life cycle of *Spirometra species*

ingesting lightly cooked frogs and snakes, poultry and pork. Use of infected frog as a bandage and pressing it on the ulcers, skin and eye could lead to transmission of the parasite through mucous membranes (Mueller 1974; Acha and Szyfres 1987).

4.5.6 Clinical Signs in Man

The spargana can enter brain, eye, subcutaneous tissues, abdominal cavity, and rarely spinal cord which can result in blindness, paralysis and death (Yoon et al. 2004; Cho et al. 2000). The parasite has the ability to infect cerebral, ocular, subcutaneous and other visceral organs depending upon the migration of the parasite (Holodniy et al. 1991).

4.5.7 Clinical Signs in Animals

The infection generally remains asymptomatic in intermediate and definitive hosts but could cause weight loss and emaciation in cats.

4.5.8 Diagnosis

The disease can be diagnosed from clinical signs, identification of parasitic stages and serological tests.

4.5.9 Prevention and Control

For preventing the disease, clean drinking water and properly cooked meat of the concerned species should always be consumed.

4.6 Taeniasis and Cysticercosis

Order: Cyclophyllidea
Family: Taeniidae

Taeniasis is a true zoonotic infection (Euzoonoses) where pig and cattle act as intermediate host for *Taenia solium* and *Taenia saginata*, respectively and human beings act as definitive hosts. *Taenia asiatica* (from pigs), much related to *T. saginata* could also infect human beings (Li et al. 2007).

4.6.1 Common Name/Synonyms

T. solium taeniasis, *T. saginata* taeniasis, cysticercosis, measly pork, measly beef, neurocysticercosis (NCC)

4.6.2 Etiology

The infection occurs due to *T. solium* and *T. saginata* in pigs and cattle, respectively. The larval forms of *T. solium* and *T. saginata* are known as *Cysticercus cellulosae* and *C. bovis* in pigs and cattle, respectively.

4.6.3 Epidemiology

T. saginata, *T. solium*, and *T. asiatica* are prevalent worldwide. Taeniasis swine is an important public health issue particularly in developing countries (Pathak et al. 1984; Juyal et al. 2008) and 20 million persons are believed to be infected with *T. solium* cysticerci (Bern et al. 1999) and as per WHO, adult parasites are prevalent among 2 million people worldwide (Shandera et al. 1994; White 2000; Yanagida et al. 2012; Del Brutto 1999; Murrell 2005). Neurocysticercosis is an important public health issue and is responsible for neurological disorders worldwide (White 2000; Flisser et al. 2005). *T. asiatica* is distributed in east Asia (Anantaphruti et al. 2007).

4.6.4 Transmission

Humans and pigs act as definitive and intermediate hosts, respectively for *T. solium.* But accidental ingestion of eggs of the parasite (*T. solium*) by human beings can also lead to serious disease such as neurocysticercosis. So both the taeniasis and cysticercosis due to *T. solium* can occur in human beings. Human beings become infected after ingestion of raw or partially cooked measly pork (Singh et al. 2002). Pigs and human beings can become infected after ingestion of eggs shed by human carriers. Taeniasis infected person (*Taenia* carriers) act as important risk factor for neurocysticercosis for the other members of the family (Flisser 1994). Poor sanitary conditions and feeding of pigs on garbage and human waste are important factors responsible for the spread of the disease (Li et al. 2007; Sarti et al. 1992; Pawlowski and Murrell 2001; Burneo and Garcia 2002). Sewage farming is another important risk factor for neurocysticercosis even for the vegetarians and non-pork eaters. The consumption of measly beef could lead to *T. saginata* taeniasis in man (Fig. 4.15).

4.6.5 Clinical Signs in Man

The important symptoms due to taeniasis include abdominal pain, nausea, diarrhoea but the disease may remain asymptomatic at times (Garcia et al. 2003). But the prompt diagnosis of *T. solium* taeniasis is important so as to avoid the risk of autoinfection through faecal contamination leading to neurocysticercosis in human beings. In case of cysticercosis, cysticerci may be present in the subcutaneous tissue beneath the skin, skeletal or cardiac muscles. Rare eye infections leading to blindness (Cardenas et al. 1992) and tongue infections (Elias et al. 2005) can also occur. The cysticerci have tendency to develop in the brain tissue leading to neurocysticercosis (Garcia et al. 2003). Depending upon the location and number of cysticerci in the central nervous system, the symptoms such as seizures and headaches, vision problems, late-onset/

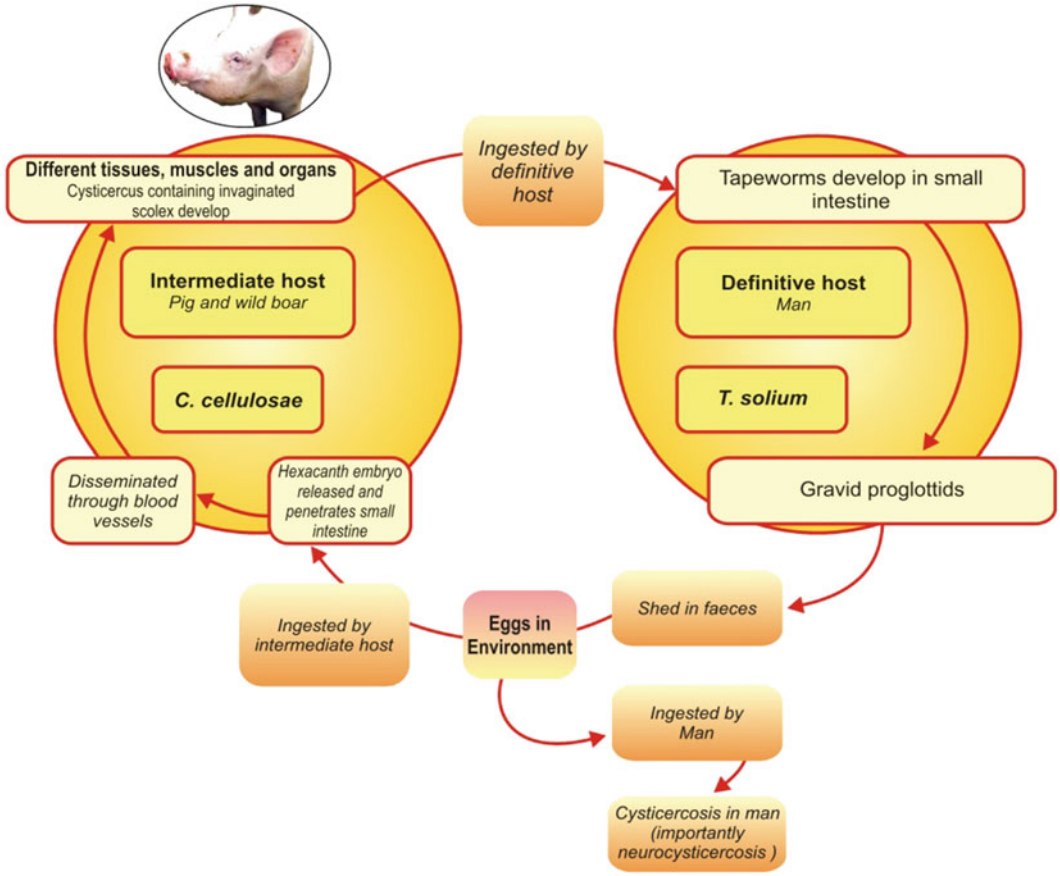

Fig. 4.15 Life Cycle of *Taenia solium*

adult/acquired epilepsy (Medina et al. 1990; White 2000) and death can occur. *T. saginata* taeniasis usually remain asymptomatic but very high parasite loads can result in abdominal pain, diarrhoea, weight loss and intestinal obstruction.

4.6.6 Clinical Signs in Animals

No symptoms are usually seen in pigs even if thousands of cysts are present (Garcia et al. 2003). Small cysts can be seen in many organs such as skeletal muscles and some visceral organs, but the disease generally remains asymptomatic. However, *T. solium* cysticercosis causes huge economic losses to the pig industry (Garcia et al. 2003).

4.6.7 Diagnosis

Detection of *Taenia* eggs from faecal samples, immunodiagnostic tools such as ELISA, copro antigen detection assays, CT and MRI can be used for diagnosis of the disease from human beings (Garcia et al. 2003; Allan et al. 2007). Cysticercosis is mainly diagnosed at the time of post mortem examination in pigs (Figs. 4.16, 4.17, 4.18 and 4.19).

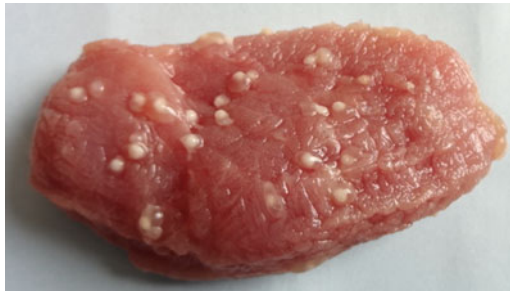

Fig. 4.16 *Cysticercus cellulosae* in striated muscles of pig

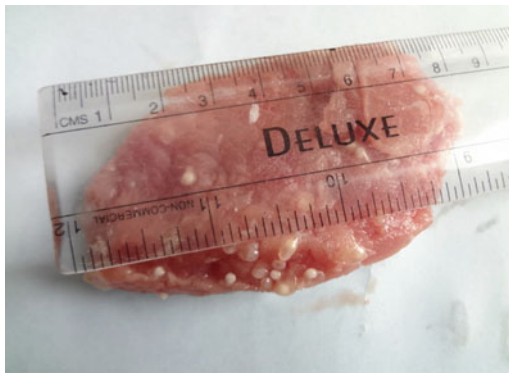

Fig. 4.17 *Cysticercus cellulosae* in striated muscles of pig

4.6.8 Control

Elimination of human carriers is highly important for prevention of neurocysticercosis (CDC 1992). The *T. solium* carriers should be treated with anthelmintics such as praziquantel (Flisser et al. 2005; Garcia 2001). Interdisciplinary approaches (Engels et al. 2003), use of anthelmintics (Roman et al. 2000) in endemic areas, avoiding free access of human faeces to pigs, education of lower socioeconomic population can help reduce the incidence of the disease (Juyal et al. 2008). Consumption of properly cooked pork, implementation of meat inspection practices and freezing of pork for appropriate time before consumption can help prevent the disease.

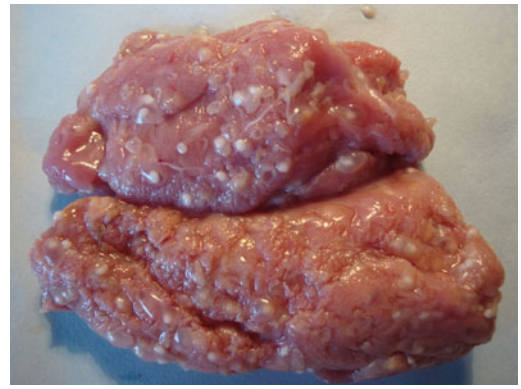

Fig. 4.18 *Cysticercus cellulosae* in striated muscles of pig

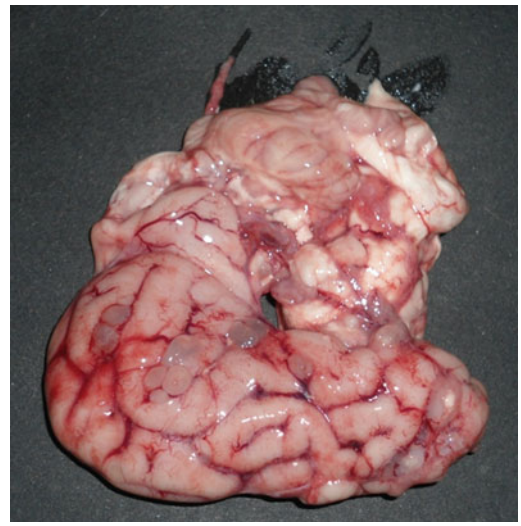

Fig. 4.19 Pig brain infected with *Cysticercus cellulosae*

4.7 Other Cestode Zoonoses

4.7.1 Bertiellosis

Bertiellea species cestode commonly occurs in chimpanzees and rodents. The two species *B. studri* and *B. macronata* have also been reported from human beings (Feldman et al. 1983; Denegri and Perez-Serrano 1997; Denegri et al. 1998). Oribatid mites act as intermediate host where cysticercoid stage occurs. Human beings can become infected after accidental

ingestion of these mites. The disease remains asymptomatic but there might be abdominal pain, diarrhoea and general fatigue in some human patients (Denegri and Perez-Serrano 1997).

4.7.2 Hymenolepiosis

The disease occurs due to *Hymenolepis nana* or *H. diminuta* which have zoonotic significance (Waugh et al. 2006). Rodents act as principal host for these parasites but the parasite could infect humans, particularly children (Goswami et al. 2011). The parasite has been recorded from many parts of the world (Marangi et al. 2003) particularly from India (Watwe and Dardi 2008). The life cycle of parasite is direct with the infection through faecal oral route in humans, but rat may play role in the transmission of the disease due to contamination of human food with rat faeces. The parasite infects gastrointestinal system and remains asymptomatic but can cause abdominal pain, diarrhoea, weakness, etc.

4.7.3 Inermicapsiferosis

The infection occurs due to *Inermicapsifer madagascariensis* found in rodents in Africa (Acha and Szyfres 2006). The parasite rarely infects man through rodent-arthropod-man cycle (Acha and Szyfres 2006).

4.7.4 Mesocestoidosis

The disease occurs due to *Mesocestoides lineatus* and *M. variablis* (Acha and Szyfres 2006). The parasite has been reported from Japan, China and Korea (Eom et al. 1992). The life cycle of the parasite is not clear but dogs, cats and other carnivores may act as definitive host and coprophagous arthropod as first intermediate host and rodents, birds, amphibians, reptiles as second intermediate host (Acha and Szyfres 2006). Consumption of undercooked meat of amphibians, reptiles, etc. may cause the disease in man. The parasite infects gastrointestinal system and may cause abdominal pain, diarrhoea, weakness, etc.

4.7.5 Raillietinosis

The infection occurs due to *Raillietina celebensis* and *Raillietina demerariensis* and few other species found in rodents. Insects act as intermediate hosts where cysticercoids develop and rare human infections occur after accidental ingestion of food contaminated with infected arthropod (Acha and Szyfres 2006). The parasite generally remains asymptomatic but could affect circulatory, nervous or digestive system in man.

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Nematode Zoonoses

5

Abstract

Nematode Zoonoses could be defined as "those nematode diseases which are naturally transmitted between (other) vertebrate animals and man". Zoonotic nematodes infect millions of people worldwide. An overview of the epidemiology, transmission, clinical manifestations and diagnosis of zoonotic nematodes is presented, along with methods of prevention and control. *Angiostrongylosis* is endemic in south Asia, Pacific islands, Australia, China and Caribbean islands. Zoonotic ascariasis due to *Ascaris suum* has been widely reported in North America and some European countries. *Baylisascaris procyonis*, the intestinal roundworm of raccoons has been reported to cause severe neurologic disease in man. Subcutaneous migration of the animal hookworms could lead to cutaneous larva migrans (CLM) in man. Gnathostomiasis is a food-borne zoonosis of public health significance. Trichinellosis has been reported in most parts of the world except the deserts. The provision of safe food and water in the non-industrialised countries is of utmost importance for the prevention and control of these diseases.

5.1 Acanthocephalus

Phylum: Acanthocephala
Family: Oligacanthorhynchidae

5.1.1 Common Name/Synonyms

Macracanthorhynchus

These parasites are spiny-headed worms and at times are included in the same phylum with nematodes (Garcia 2001). They are pseudocoelomate endoparasites and have many unique morphological and biological features (Marquards et al. 2000; Garcia 2001).

5.1.2 Etiology

Acanthocephalus primarily occurs due to following species of parasites:

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Name of the parasite	Definitive host	Intermediate host	References
Macracanthorhynchus hirudinaceus Synonym: *Gigantorhynchus hirudinaceus*, *G. gigas*, *Echinorhynchus gigas*	Swine, wild boar and occasionally rodents, bovine, dog, monkey and man	Dung beetle of the family Scarabaeidae	Acha and Szyfres (2006)
Moniliformis moniliformis	Rats and small rodents	Beetles and cockroaches	
Corynosoma strumosum	Arctic fox (Alopex lagopus), dog, sea otter (Enhydra lutris) and species of cetaceans and pinnipeds	Amphipod crustacean (Pontoporeia affinis) Many species of fish could act as paratenic host	
Acanthocephalus rauschi, *A. bufonis* (*A. sinensis*)	Crustaceans	–	
Bolbosoma spp.	Cetaceans	Juvenile state found in fish	

5.1.3 Epidemiology

Acanthocephalus is not common and has been rarely reported in man. Till date, seven cases of acanthocephlus have been reported (Roberts and Janovy 2005; Berenji et al. 2007). These parasites include *Acanthocephalus bufonis*, *Corynosoma strumosum*, *Acracanthorhynchua hirudinaceus*, *Moniliformis moniliformis*, *Bolbsoma* sp. (Counselman et al. 1989; Marquards et al. 2000; Roberts and Janovy 2005), *Macracanthorhynchua ingens* (Counselman et al. 1989, Roberts and Janovy 2005) and *Acanthocephalus rauschi* (Marquards et al. 2000; Roberts and Janovy 2005). The parasite *M. moniliformis* has been reported from many parts of the world (Ikeh et al. 1992; Roberts and Janovy 2005). Human infections due to *M. moniliformis* have been reported from Africa, Asia, Australia, Europe, and America (Muller 1975; Beaver et al. 1984; Ikeh et al. 1992; Baker et al. 2000; Berenji et al. 2007). The morphological parameters of the parasite have been described in detail by different authors (Lawlor et al. 1990; Beaver et al. 1984; Roberts and Janovy 2005).

5.1.4 Transmission

Man gets the infection after consumption of raw paratenic host (Roberts and Janovy 2005) or due to incidental consumption of intermediate host (Berenji et al. 2007).

5.1.5 Life Cycle

Humans are the incidental hosts, and the worm resides in the small intestine. The definitive hosts become infected after eating the intermediate host (Ikeh et al. 1992).

5.1.6 Clinical Signs in Man and Animals

The symptoms of the disease are not well defined. High parasitemia may lead to vomition, abdominal pain, perforation of the intestine, fatigue, diarrhoea, anorexia, irritability and intermittent burning sensations around the umbilicus (Beaver et al. 1984; Counselman et al. 1989; Ikeh et al. 1992;

Marquards et al. 2000; Roberts and Janovy 2005). The penetration of proboscis and release and reattach tendency of the parasite could lead to inflammation, ulcer and haemorrhages in the definitive host (Marquards et al. 2000; Garcia 2001; Roberts and Janovy 2005). It has been observed that *M. moniliformis* infected cockroach move more slowly and travel shorter distances (Moore et al. 1994; Libersat and Moore 2000; Roberts and Janovy 2005). This might be responsible for the parasite's predation by the definitive host (Libersat and Moore 2000). The morbidity is generally high among mammals infected with acanthocephalans. However, fish may remain asymptomatic even after high intensities of worms deeply penetrated into their intestinal wall (Taraschewski 2000). The high heavy metals absorbance capacity indicates the usefulness of acanthocephalans as bioindicators (Taraschewski 2000).

5.1.7 Diagnosis

The disease can be diagnosed by identification of the thick-shelled eggs containing first larval stage in the faecal samples, while a living or dead fluke is rarely seen.

5.1.8 Control

Humans should avoid ingestion of intermediate or paratenic host.

5.2 Angiostrongylosis

Order: Strongylida
Superfamily: Metastrongyloidea

5.2.1 Common Name/Synonyms

Intestinal angiostrongyliasis, eosinophilic meningitis

5.2.2 Etiology

The rat lung worm, *Angiostrongylus cantonensis* was first time reported in China (Chen 1935) from domestic rat. This is a food-borne zoonotic nematode parasite of significant public health importance. *Angiostrongylus costaricensis* causes abdominal eosinophilic granulomas in Central and South America (Pien and Pien 1999).

5.2.3 Epidemiology

Angiostrongylosis due to *A. cantonensis* is endemic in south Asia, Pacific islands, Australia, China and Caribbean islands (Wang et al. 2008; Lv et al. 2008).

5.2.4 Transmission

Human beings become infected after consumption of intermediate (raw snails) or paratenic hosts (freshwater prawns, crabs, frogs), or vegetables contaminated with the infected larvae (Alicata 1991).

5.2.5 Life Cycle

For *A. cantonensis*, rats and molluscs act as the definitive and intermediate host for this parasite, respectively. The crustaceans (prawns and land crabs), predacious land planarians (flatworms in the genus *Platydemus*), frogs and monitor lizards (Wang et al. 2008) are the important paratenic hosts. The parasite *A. cantonensis* is present in the (pulmonary arteries) rat lungs. Larvae hatch and migrate through trachea and gastrointestinal tract to rat faeces. These first stage larvae are eaten up by snails and slugs, and further develop into infective larvae (third stage). Man and rats become infected after consuming infected snail or contaminated vegetables. Larvae migrate towards the brain in human beings and could lead to brain abscess and haemorrhage (Koo et al. 1988). The parasite *A. costaricensis* resides in mesenteric arteries of caecum of cotton rat. The

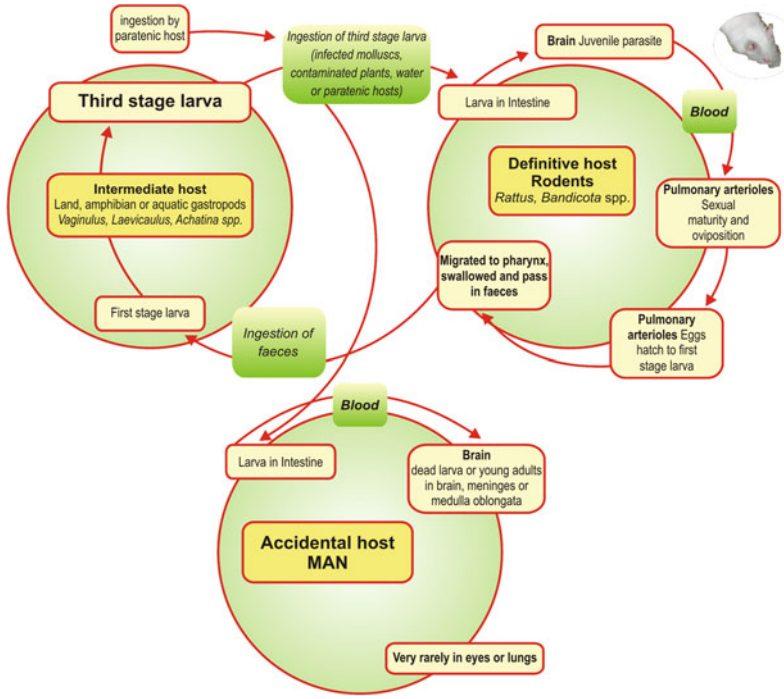

Fig. 5.1 Life cycle of *Angiostrongylus cantonensis*

first stage larvae penetrate slugs and develop to second and third stage larvae. The cycle is completed when definitive host ingest infective larvae released from slugs (Fig. 5.1).

5.2.6 Clinical Signs in Man

In *A. cantonensis* angiostrongylosis, the parasite infects brain (Cross 1978; Chotmongkol and Sawanyawisuth 2002). The other important symptoms include headache, neck stiffness, paraesthesia, vomition and nausea (Wang et al. 2008). Symptoms due to *A. costaricensis* include abdominal pain, vomition and rarely gastrointestinal bleeding (Hulbert et al. 1992; Silvera et al. 1989).

5.2.7 Clinical Signs in Animals

The infected rodents may show sneezing, coughing, fibrosis in the lungs and occasionally gastrointestinal lesions.

5.2.8 Diagnosis

The disease can be diagnosed after detection of the parasite from cerebrospinal fluid or ocular chamber (Wang et al. 2008). History, clinical signs and serological tests are also useful in the diagnosis. Diagnosis of human angiostrongylosis (*A. cantonensis*) is based on clinical features as well as laboratory findings (Eamosobhana and Yong 2009; Eamsobhana 2006).

5.2.9 Control

Prevention and control measures primarily rely upon health education, avoidance of eating raw or undercooked intermediate and paratenic hosts, unwashed vegetables and rodent control.

5.3 Anisakiosis

Order: Ascaridida
Family: Anisakidae

Anisakiosis is parasitic zoonosis which occurs due to accidental ingestion of the parasite larvae (Sakanari and Mckerrow 1989).

5.3.1 Common Name/Synonyms

Herring worm disease, Cod worm disease

5.3.2 Etiology

The disease can occur due to *Anisakis*, *Pseudoterranova* and *Contracaecum* species, but *Anisakis* is primarily important. *A. simplex* is particularly associated with gastroallergic anisakiosis (Audicana et al. 2002).

5.3.3 Epidemiology

The scientist 'Van Thiel' for first time recognized Anisakiosis in 1960 (Van Theil 1960). The disease is common in Japan (about 2000 cases/year) but cases have also been recorded from northern Europe (Scandinavia, Holland) and Pacific coast of South America (Caramello et al. 2003). Gastric anisakiosis is more common in Japan, while intestinal anisakiosis has been mainly reported from Europe (Audicana et al. 2002).

5.3.4 Transmission

Human beings become infected after eating raw or lightly cooked marinated seafood such as Japanese sushi and sashimi, Dutch salted or smoked herring, Scandinavian gravlax, Hawaiian lomi–lomi and Latin American ceviche (Chai et al. 2005).

5.3.5 Life Cycle

The adult parasites are present in stomach of marine fish (whales and dolphins). Crustaceans act as first intermediary hosts. Fish and some cuttlefish act as paratenic hosts (Caramello et al. 2003). Human beings become infected after consumption of third stage larvae present in infected salt-water fish or cephalopod *mollusc* species, including *mackerel*, *squid*, *sardine*, *horse mackerel*, *salmon* and *bonito* (Yasunaga et al. 2010; Karasawa et al. 2008). In fish, larvae generally localise on the surface of visceral organs, body cavity and occasionally on the muscles. In man, the parasites do not mature, but they cause the disease. Other definitive hosts include birds, reptiles and amphibians (Fig. 5.2).

5.3.6 Clinical Signs in Man

The symptoms could arise due to both gastric and intestinal anisakiosis. Clinical signs are related to allergic reactions and tissue damage. Presence of *Anisakis* allergens in the human food cause gastroallergic anisakiosis which may lead to urticaria, angioedema and anaphylactic shock associated with other gastrointestinal symptoms (Caramello et al. 2003; Lopez Serrano et al. 2000). Acute gastric anisakiosis occurs suddenly 12–72 h after ingestion of raw seafood and leads to severe abdominal pain (Sugimachi et al. 1985). The tissue damage by the larvae causes intestinal anisakiosis. Further changes include invasion of the gut wall, and eosinophilic granuloma, or perforation which may lead to symptoms such as fever, abdominal pain, vomition and bloody stools.

5.3.7 Clinical Signs in Animals

The parasite larvae may lead to liver atrophy, perforations of the stomach wall, visceral adhesions and muscle damage (Smith and Wootten 1978). In marine mammals, parasites have been found embedded in gastric mucosa tumours (Acha and Szyfres 2006).

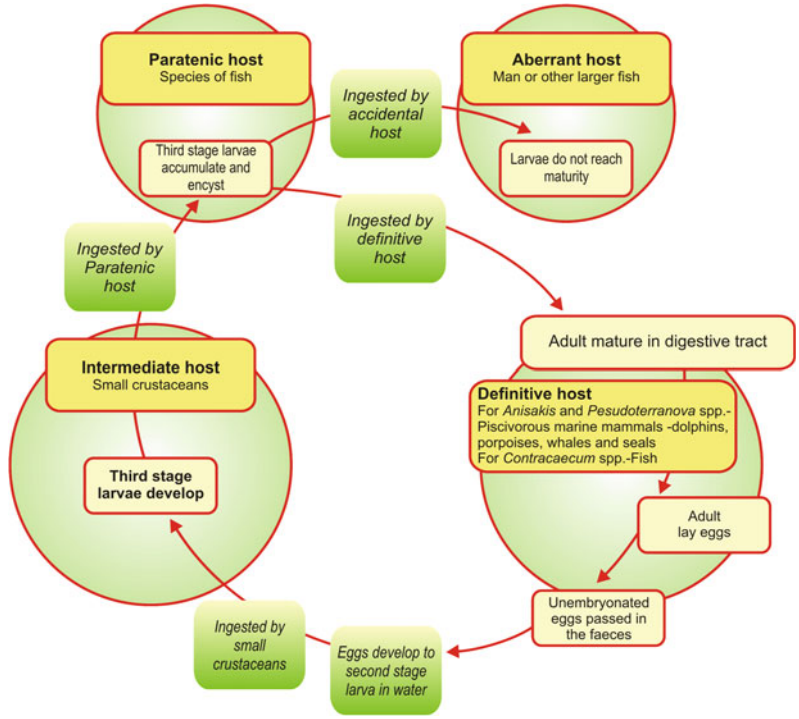

Fig. 5.2 Life cycle of Anisakis, *Pseudoterranova*, or *Contracaecum* species

5.3.8 Diagnosis

The disease can be diagnosed by upper gastrointestinal endoscopy for visualisation of the worm (McCarthy and Moore 2000) or using molecular techniques (Mattiucci et al. 2011).

5.3.9 Control

Prevention and control measures include health education; avoid eating raw or undercooked fish and heating or freezing of fish at appropriate time–temperature combinations before consumption.

5.4 Ascariosis

Order: Ascaridida
Superfamily: Ascaridoidea

5.4.1 Common Name/Synonyms

Ascaridiasis

5.4.2 Etiology

Ascariosis occurs due to *Ascaris lumbricoides* and *Ascaris suum*; two closely related species responsible for the disease in humans and pigs, respectively (Dold and Holland 2010). These species are morphologically indistinguishable with few molecular variations (Anderson et al. 1993). Albeit both the parasites are specific to their hosts (Crompton 2001), experimental studies have shown that they can infect both the hosts (Takata 1951; Galvin 1968). In persons living in low prevalence areas of human parasites, *A. suum* could cause zoonotic infections (Crewe and Smith 1971; Jaskoski 1961; Lord and Bullock 1982; Nejsum et al. 2005). The parasite *A. suum* has also been found among zoo chimpanzees (Nejsum et al. 2010).

5.4.3 Epidemiology

A. lumbricoides is a common human parasite worldwide and more than 1.2 billion people are believed to be infected globally (de Silva et al. 2003). *A. lumbricoides* has been commonly reported from sub-Saharan Africa, Americas, China and East Asia (WHO 2006). *A. suum* is another parasite prevalent across the globe and infects pigs with high prevalence rates (Roepstorff et at. 1998; Nansen and Roepstorff 1999). Zoonotic ascariosis due to *A. suum* has been reported in North America, Denmark (Anderson 1995; Nejsum et al. 2005) and the United Kingdom (Nejsum et al. 2010). Chimpanzees in the Copenhagen Zoo were found infected with *A. suum* from pigs by molecular analysis (Nejsum et al. 2005).

5.4.4 Transmission

Human beings become infected after accidental ingestion of *Ascaris* eggs through contaminated food, water or soil.

5.4.5 Life Cycle

The disease is transmitted via faecal-oral route. After ingestion of infective eggs, L3 larvae hatches in the intestine (Geenen et al. 1999). This is followed by movement of larvae from small to large intestine and further penetration of the mucosa (Murrell et al. 1997). Later, the larvae reach the liver via blood circulation and shed L2 cuticle and finally migrates to the lungs (Roepstorff et al. 1997). The larvae penetrate alveolar space and move towards pharynx where they are swallowed. The larvae again return to small intestine where they mature and reach sexual maturity (Pilitt et al. 1981; Roepstorff et al. 1997). The eggs are shed in faeces and develop to infective larvae under favourable conditions.

5.4.6 Clinical Signs in Man and Animals

Clinical symptoms may range from asymptomatic to severe coughing and wheezing. The lower age groups are usually affected. *Ascaris* pneumonia could result due to damage caused by larvae. Heavy worm loads may lead to intestinal blockage and obstruction in children. Further complications include intussusception, perforation and gangrene of the bowel. In endemic areas, biliary and pancreatic disease could lead to biliary colic, acute cholecystitis, acute cholangitis, acute pancreatitis and hepatic abscess.

5.4.7 Diagnosis

The disease can be diagnosed by detection of the parasitic eggs during the intestinal phase of the disease. Clinical signs such as laboured breathing, stunting etc. can also aid in the diagnosis. Serological, molecular (Zhu et al. 1999) techniques can provide some help during the migratory phase of the larvae.

5.4.8 Control

Prevention and control measures include improved personnel hygiene, health education, improved sanitary measures and chemotherapy (Hotez et al. 2006).

5.5 Baylisascariasis

Order: Ascaridida
Superfamily: Ascaridoidea

5.5.1 Etiology

The disease occurs due to *Baylisascaris procyonis*, the intestinal roundworm of raccoons,

which has been regarded as a cause of severe neurologic disease among humans (Sorvillo et al. 2002) since last 2–3 decades.

5.5.2 Epidemiology

This parasite is found in raccoons from North America and has also been recorded in other countries from Europe and Asia (Snyder 1987; Wirtz 1982). Fourteen confirmed human cases due to *B. procyonis* have been cited in the literature (Wise et al. 2005; Gavin et al. 2005).

5.5.3 Transmission

Human beings become infected after ingestion of infective eggs from environment contaminated with racoon faeces.

5.5.4 Life Cycle

The North American raccoon (*Procyon lotor*) acts as the definitive host for *B. procyonis*. Intermediate hosts include small birds and mammals (particularly rodents). Humans act as accidental intermediate hosts. The important risks for human infection include young children with pica or geophagia (Kazacos 2000). Raccoons become infected with *B. procyonis* through two ways: either through ingestion of infective eggs from contaminated environment or after consumption of paratenic hosts (Wise et al. 2005; Kazacos 2001). The larvae are released between eggs and they develop into male and female adult worms (Kazacos 2001). Gravid females again start releasing eggs into the faeces. The eggs undergo embryonation and become infective (Kazacos 2001). The paratenic hosts such as squirrels and rodents play an important role in the completion of the life cycle. After they ingest eggs, larvae are released which penetrate and disseminate in different organs and tissues. Larvae often migrate to the central nervous system (CNS) (Tiner 1953) and these paratenic hosts are consumed by racoons. The larvae mature in racoon to become male and female adults and shed eggs (Kazacos 1983, 2001) (Fig. 5.3).

5.5.5 Clinical Signs in Man

B. procyonis could rarely lead to fatal or neurologically devastating neural larva migrans (NLM). This results in acute eosinophilic meningoencephalitis in infants and young children (Goldberg et al. 1993; Kazacos et al. 1985; Gavin et al. 2005). Pica, exposure to infected raccoons and contaminated environments with racoon faeces are the three significant risk factors for infection (Gavin et al. 2005). The parasite may occasionally lead to ocular larva migrans (OLM) usually in healthy adults. Clinical symptoms are not fully understood but could include visceral, neural and ocular larva migrans.

5.5.6 Clinical Signs in Animals

The infection in raccoons is generally subclinical. *B. procyonis* is considered as the most common cause of clinical larva migrans in some other animals such as rodents, particularly NLM (Gavin et al. 2005). The case reports of infection in dogs and cats have also been reported (Kazacos 2001; Greve and O'Brien 1989; Averbeck et al. 1995).

5.5.7 Diagnosis

The disease can be diagnosed from clinical signs and history, brain biopsy or serological tests and identification of ellipsoidal and dark brown *B. procyonis* eggs (Kazacos 2001).

5.5.8 Control

Avoid contact with raccoons or their faeces and practice good sanitary measures.

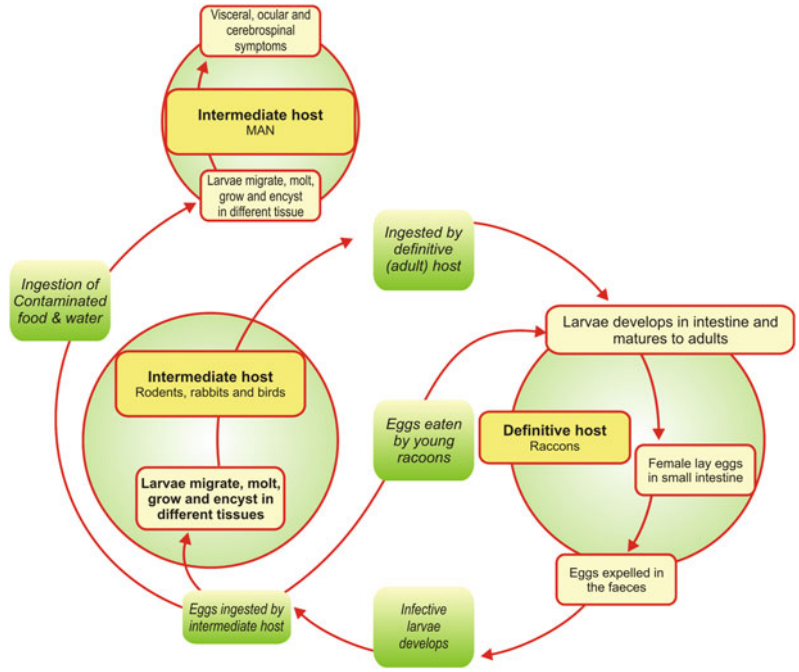

Fig. 5.3 Life cycle of *Baylisascaris procyonis*

5.6 Capillariasis

Superfamily: Trichuroidea
Family: Capillariidae

5.6.1 Etiology

The nematodes *Capillaria hepatica*, *C. aerophila* and *C. philippinensis* are responsible for hepatic, pulmonary and intestinal capillariasis in man (Cross 1992). Out of these, *C. philippinensis* related intestinal capillariasis is most important disease in human beings.

5.6.2 Epidemiology

Epidemics due to *C. philippinensis* have been reported from Philippine islands, Thailand and Taiwan (Cross 1992). In Philippines and Thailand, more than 2,000 cases have been reported. Sporadic cases have also been reported from many other countries such as Korea, Japan, Taiwan, India, Iran, Egypt, Italy, United Arab Emirates, Spain and United Kingdom (Cross 1998; El Hassan and Mikhail 1992; Austin et al. 1999). Increasing number of cases of Intestinal capillariasis are being reported from Asia (Hong and Cross 2005).

5.6.3 Reservoir

Fish-eating birds act as the reservoir hosts (Cross 1998) for intestinal capillariasis. Rodents serve as reservoir for *C. hepatica* infections.

5.6.4 Transmission

Human beings become infected after consumption of tiny freshwater and brackish-water fish (Cross 1998).

5.6.5 Life Cycle

For *C. philippinensis*, fish act as intermediate host and fish-eating birds or man are the definitive host for intestinal capillariasis (Bhaibulaya and Indra-Ngarm 1979; Cross and Basaca-Sevilla 1983). Human beings become infected after accidental consumption of the tiny fish which are normally consumed by birds.

5.6.6 Clinical Signs in Man

The symptoms of intestinal capillariasis include severe enteropathy and abdominal pain, intermittent diarrhoea, weight loss and occasionally death in human beings (Cross 1992, 1998) if the cases are not properly treated. Hepatic capillariasis could result in hepatomegaly, abdominal discomfort, diarrhoea, constipation etc. *C. aerophila* causes pulmonary capillariasis and related symptoms.

5.6.7 Clinical Signs in Animals

C. hepatica capillariasis and *C. aerophila* capillariasis can cause clinical disease in rodents and foxes, respectively.

5.6.8 Diagnosis

The disease can be diagnosed by stool examination. *Capillaria* spp. eggs should be differentiated from other closely related species such as *Trichuris* and *Trichinella*. If eggs, larvae or adult of *C. philippinensis* are not found in the stool samples, biopsy specimens could help in the diagnosis.

5.6.9 Control

Avoid eating raw or undercooked fish in endemic areas.

5.7 Cutaneous Larva Migrans

Superfamily: Ancylostomatoidea

5.7.1 Common Name/Synonyms

Creeping eruption, serpiginous eruption, larva currens (*Strongyloides* spp. infection)

5.7.2 Etiology

Cutaneous larva migrans (CLM) occurs due to subcutaneous migration of the nematode larvae, especially animal hookworms. The etiological agents include *Ancylostoma braziliense*, *Ancylostoma caninum*, *Ancylostoma ceylanicum*, *A. tubaeforme*, *Uncinaria stenocephala* or *Bunostomum phlebotomum*. *A. braziliense* is mostly responsible for the occurrence of disease in human beings (Caumes 2000). Many other parasites such as *Strongyloides papillosus*, *S. westeri*, *S. stercoralis*, *S. procyonis* and *S. myopotami* can occasionally cause a similar disease but they move more rapidly than animal hookworms. Moving *Gnathostoma spinigerum* and *Dirofilaria repens* could also cause a similar type of dermatitis.

5.7.3 Epidemiology

A. braziliense is endemic in tropical and subtropical areas, whereas *A. caninum* and *B. phlebotomum* more commonly occur in temperate areas. Human cases have been reported across the globe from many countries (Shinkar et al. 2005; Yoshida et al. 1973). Travellers visiting endemic areas are at particular risk of the infection.

5.7.4 Reservoir

Carnivorous animals act as reservoir for the infection.

5.7.5 Transmission

Human beings become infected due to penetration of the parasite larvae present in the soil contaminated by infected animal faeces.

5.7.6 Life Cycle

In carnivorous animals, the larvae present in the soil penetrate the skin and migrate through the tissue to reach small intestine of the host where they mature into adults. In human beings, although larva penetrates and migrates through skin, but it do not develop further and cause the disease.

5.7.7 Clinical Signs in Man

These migrating larvae produce erythematous, serpiginous, pruritic, cutaneous track also known as creeping eruption (Caumes et al. 2002; Beaver 1956; Bryceson and Hay 1998; Lucchina and Wilson 1999) due to percutaneous penetration and subsequent migration of larvae of many nematode parasites (Knight 2003).

5.7.8 Clinical Signs in Animals

The disease occurs in carnivorous (dogs and cat) animals. The symptoms include diarrhoea, anaemia and allergic dermatitis.

5.7.9 Diagnosis

The disease can be diagnosed by clinical symptoms, successful treatment or by histopathological confirmation (Purdy et al. 2011).

5.7.10 Control

For prevention and control, one should not walk barefoot and use a mattress while lying in sandy areas particularly in disease endemic tropical and subtropical areas.

5.8 Dioctophymosis

Superfamily: Dioctophymatoidea
Family: Dioctophymidae

5.8.1 Common Name/Synonyms

Dioctophymiasis

5.8.2 Etiology

Dioctophymosis occurs due to *Dioctophyma renale*, a large nematode also known as the giant kidney worm (Soulsby 1978; Bowman 1999).

5.8.3 Epidemiology

The parasite has been reported across the globe barring Africa and Oceania (Soulsby 1978; Freitas 1980; Kommers et al. 1999). The parasite basically infects domestic and wild fish-eating carnivores (Soulsby 1978; Maxie 1993; Bowman 1999; Nakagawa et al. 2007) but has the ability to cause the disease in human beings (Sun et al. 1986; Acha and Szyfres 2006). Among domestic animals, the parasite is more commonly found in dogs than cats, horses and cattle (Maxie 1993; Nakagawa et al. 2007). The adult parasite is usually found in kidney but can also occur in abdominal cavity, uterus, ovary, mammary gland, urethra, subcutaneous tissues of the inguinal region and mesenteric lymph nodes (Freitas 1980; Maxie 1993).

5.8.4 Reservoir

The mink (*Mustela vison*) is considered to be the main reservoir and definite host (Bowman 1999), and humans and dogs are thought to be terminal or accidental hosts (Acha and Szyfres 2006; Nakagawa et al. 2007).

5.8.5 Transmission

Human beings become infected after eating infected raw fish, frogs or by drinking water contaminated with the mud-worm infected by *D. renale* in its larval form.

5.8.6 Life Cycle

The life cycle of the parasite involves different kind of hosts (Soulsby 1978; Freitas 1980; Bowman 1999). The eggs of the parasite are passed in the urine and are ingested by intermediate hosts viz. mud-worms (aquatic oligochaetes). The nematode develop into third stage larva (Freitas 1980) in mud worms and then these are eaten by fishes or frogs which act as paratenic hosts and the larvae become ingested in tissue without further development (Bowman 1999). Wild fish eating carnivores act as the definite hosts and become infected after consuming infected intermediate or paratenic host (Soulsby 1978; Bowman 1999). The larva migrates to the kidney in the definitive host (Freitas 1980; Bowman 1999; Nakagawa et al. 2007).

5.8.7 Clinical Signs in Animals

In dogs, most cases generally remain asymptomatic (Soulsby 1978; Maxie 1993) or the clinical signs are not conclusive for the definitive diagnosis of the disease. Right kidney and abdominal cavity are more commonly affected in case of dogs (Freitas 1980; Maxie 1993; Bowman 1999; Kommers et al. 1999). The infection could lead to renal parenchyma atrophy, dilation of the renal pelvis and urethral obstruction (Soulsby 1978; Maxie 1993). Other signs such as renal failure, haematuria, abdominal distention and peritonitis may also be observed.

5.8.8 Clinical Signs in Man

The parasitic infection could lead to similar kind of symptoms in man.

5.8.9 Diagnosis

The disease can be diagnosed using imaging techniques such as radiology (Nakagawa et al. 2007), sonography (Soler et al. 2008) and detection of parasite ova or eggs (Birchard and Sherding 1994) in the urine. Postmortem-based detection of the parasite is another method routinely employed for detection of the parasite (Pereira et al. 2006).

5.8.10 Control

Avoid consumption of raw or undercooked fish and frog or water contaminated with infected mud worms.

5.9 Dracunculiasis

Class: Secerentea
Suborder: Camallinida
Superfamily: Dracunculoidea

5.9.1 Common Name/Synonyms

Guinea worm disease, Dracontiasis

5.9.2 Etiology

The disease occurs due to guinea worm *Dracunculus medinensis*, one of the longest tissue nematode which can infect human beings.

5.9.3 Epidemiology

The parasite was prevalent in southern Asia and North, West and East Africa for past two decades (Tiben and Hopkins 2006). After the end of 2007, the disease remains endemic only in few countries viz. Sudan, Ghana, Mali and Niger due to successful implementation of dracunculiasis eradication programme (Hopkins et al. 2008).

5.9.4 Reservoir

Human being acts as the reservoir of *D. medinensis*.

5.9.5 Transmission

Human beings become infected after drinking water from stagnant ponds, pools, cisterns or open wells containing fresh water copepods (Cyclops or water fleas) harbouring infective larvae of the parasite (Tiben and Hopkins 2006).

5.9.6 Life Cycle

The first stage larvae are released by female worm when they emerge from the human body. The released larvae remain in the water and are ingested by an intermediate host, copepod. Larvae develop into third stage in the copepod (Cairncross et al. 2002; Muller 1971). Human beings become infected after drinking water containing infected copepods. In human beings, gastric juices digest the vector and release larva. The larvae migrate through thoracic cavity and enter connective tissues to reach maturity. Mating occurs, female continue their development and emerge from lower part of the human body (Karam and Tayeh 2006). Blister formation occurs and female worm releases thousands of larvae after contact with cold water (Muller 1971) (Fig. 5.4).

5.9.7 Clinical Signs in Man

In human beings, the symptoms start with the formation of cutaneous blister. There is intense burning sensation and symptoms could extend to erythema, urticarial rash, intense pruritus, nausea and vomiting, diarrhoea, dyspnoea, giddiness and syncope (Muller 1971). After the blister bursts, symptoms start to disappear slowly. If the larvae are released inside the joint, it may lead to severe arthritis and ankylose.

5.9.8 Clinical Signs in Animals

Clinical manifestations are similar as in man. Few presumed zoonotic cases have been reported in Japan, Korea and Indonesia, where disease is not endemic and these infections might have occurred from animal reservoirs (Hashikura 1927; Kobayashi et al. 1986; Van Heutsz 1926). Possibly, the infection occurred after consumption of raw freshwater fishes as paratenic hosts (Cairncross et al. 2002). Emerging female worms have also been recovered occasionally from a wide range of mammals from both endemic and non-endemic areas (Cairncross et al. 2002).

5.9.9 Diagnosis

The disease can be diagnosed from the clinical and allergic symptoms or using immunodiagnostic/serological tests (Bloch et al. 1993, 1998; Bapna and Renapurkar 1996).

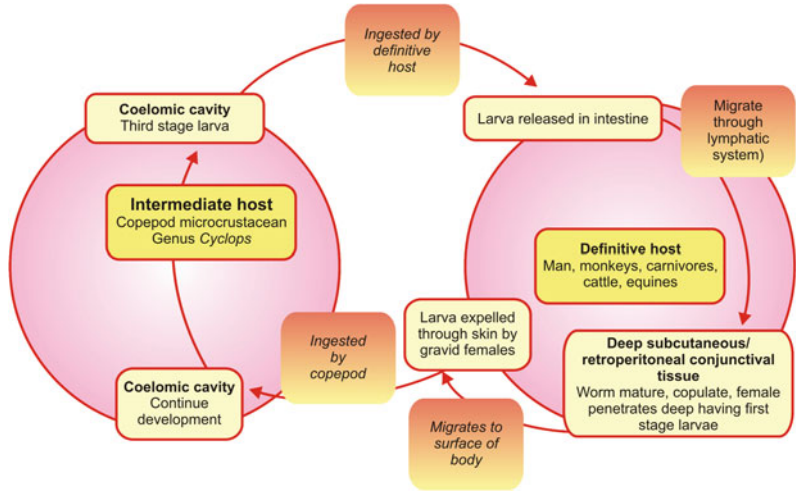

Fig. 5.4 Life cycle of *Dracunculus medinensis*

5.9.10 Control

The ponds and shallow or step wells are the important sources of the disease. The provision of clean drinking water is a must for prevention and control of the disease.

5.10 Gnathostomiasis

Superfamily: Spiruroidea
Family: Gnathostomatidae

5.10.1 Common Name/Synonyms

Gnathostomiasis, larva migrans (Gnathostoma)

5.10.2 Etiology

Gnathostomiasis is important food-borne zoonosis of public health significance. The disease occurs due to 12 species of *Gnathostoma* (Guitierrez 2000; Herman and Chiodini 2009). Out of these, 4 species have been recorded in human beings viz. *G. spinigerum* (reported from wild and domestic cats and dogs in India, China, Japan and southeast Asia), *G. hispidum* (reported from wild and domestic pigs in Europe, Asia and Australia), *G. doloresi* (reported from wild boars) and *G. nipponicum* (reported from weasels in Japan) (Guitierrez 2000; Herman and Chiodini 2009).

5.10.3 Epidemiology

The disease is endemic in Japan, Thailand and parts of Southeast Asia (Rusnak and Lucey 1993). The disease has also been reported from Central and South America, Cambodia, Laos, Myanmar, Indonesia, Philippines, Malaysia, China, Sri Lanka and India (Rusnak and Lucey 1993).

5.10.4 Reservoir

In endemic areas, cats, dogs and pigs can serve as important reservoirs of infection (Daensvang 1980; Moore et al. 2003),

5.10.5 Transmission

Human beings become infected after consumption of raw or undercooked infected freshwater fish or snakes, frogs, chickens and pigs. Ingestion of water containing infected copepod

is another important risk for the infection (Daengsvang 1981).

5.10.6 Life Cycle

The adult parasites live in the stomach wall of fish consuming carnivores and shed the eggs in their faeces. When eggs are shed in freshwater, first stage swimming larvae form which are consumed by first intermediate host small crustacean cyclops. Fish consume these hosts and infective larvae develop in fish muscle tissue as L3 larvae. In definitive host, adult parasite develops after consumption of infected fish. For *G. spinigerum*, feral cats and dogs mainly act as definitive host, but infections have also been recorded from tigers, leopards, lions, racoons, minks, opossums and otters (McCarthy and Moore 2000). Human beings accidentally become infected after consumption of infected fish. Additionally, pigs, frogs, snails, chickens and snakes can also act as intermediate hosts (McCarthy and Moore 2000) (Fig. 5.5).

5.10.7 Clinical Signs in Man

The symptoms include malaise, fever, urticaria, anorexia, nausea, vomition, diarrhoea, and epigastric pain (Rusnak and Lucey 1993). The parasite migrates through skin and subcutaneous tissue leading to painful swelling and cutaneous gnathostomiasis (Rusnak and Lucey 1993). Occasionally, parasite may migrate to other visceral organs and could lead to pulmonary, ocular, genitourinary, gastrointestinal, auditory and central nervous gnathostomiasis with fatal consequences (Rusnak and Lucey 1993).

5.10.8 Clinical Signs in Animals

In their respective definitive hosts, the parasite produces clinical symptoms. Necrotic tunnels in visceral organs (such as liver, pancreas), abdominal tissues and peritonitis can occur in cat and dogs due to *G. spinigerum* infections (Acha and Szyfres 2006). Stomach ulcers and other abdominal symptoms could also be seen in pigs infected with *G. hispidum* and *G. doloresi* (Acha and Szyfres 2006).

5.10.9 Diagnosis

Clinical signs, history and serological tests can help in diagnosis of the disease.

5.10.10 Control

Avoid eating raw or undercooked intermediate hosts such as fish, snakes, frogs, chicken etc. and drink clean water. Freezing of meat to −20 °C for 3 to 5 days can also render meat safe for consumption (Herman and Chiodini 2009).

5.11 Gongylonemosis

Order: Spirurida
Superfamily: Spiruroidea
Family: Thelaziidae

5.11.1 Common Name/Synonyms

Gongylonematosis

5.11.2 Etiology

The disease primarily occurs due to *Gongylonema pulchrum*, Molin 1857, a spiuroid nematode although many other species of *Gongylonema*, could also affect mammals and birds (Soulsby 1982; Yamaguti 1961; Cappucci et al. 1982). Ruminants, pigs and wild boar are the main definitive hosts of this parasite, but it has also been reported from nonhuman primates, carnivores and rodents. Human beings become accidentally infected after ingestion of intermediate host or drinking contaminated water.

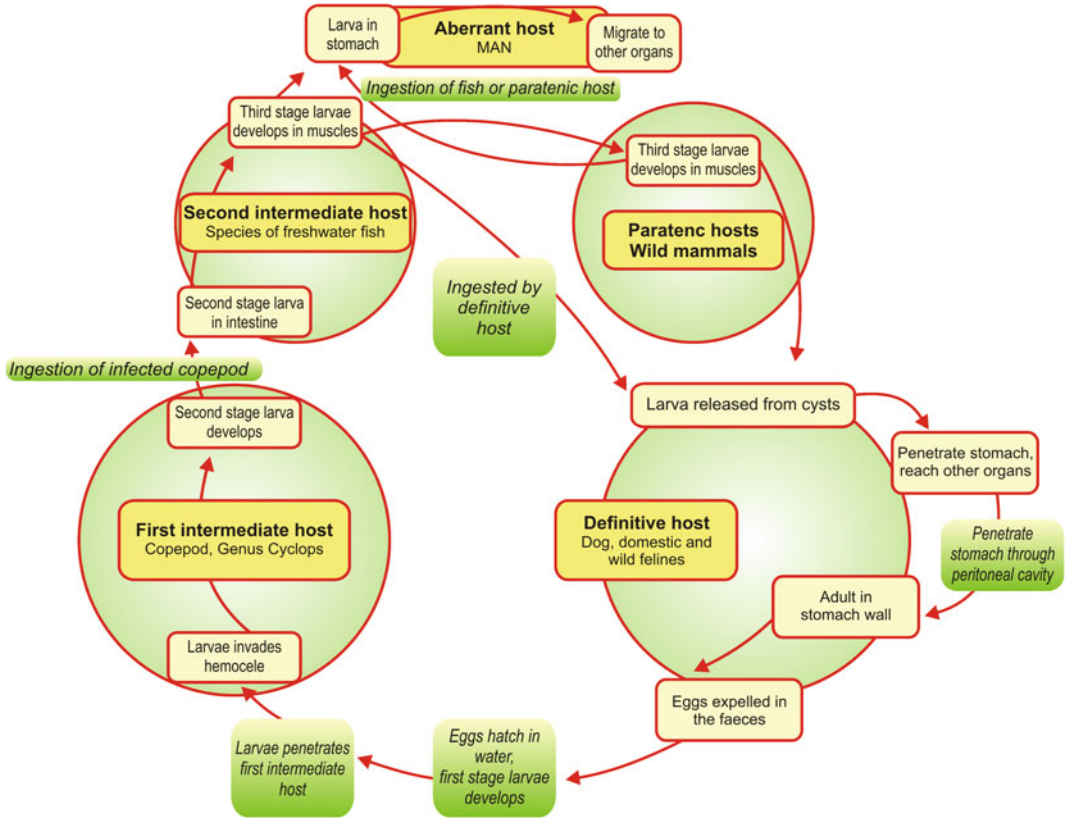

Fig. 5.5 Life cycle of *Gnathostoma spinigerum*

5.11.3 Epidemiology

Although a rare zoonosis, but the disease in humans is wide in distribution. More than 50 human cases have been reported from China, Middle East, Australia, Italy, United States, Europe, Sri Lanka, New Zealand, Japan and Southeast Asia (Wilde et al. 2001; Cappucci et al. 1982).

5.11.4 Reservoir

Existence of large number of definitive and intermediate hosts serves as source of infection.

5.11.5 Transmission

Accidental ingestion of small beetles possibly through contaminated salad and vegetables could serve as source of infection in man.

5.11.6 Life Cycle

Approximately fifty species of arthropod coprophagous insects such as dung beetles, cockroaches, etc. act as intermediate hosts for *G.pulchrum* (Cappucci et al. 1982; Illescas-Gomez et al. 1988; Soulsby 1965). Embryonated eggs are shed in the faeces of definitive

host and they are further consumed by intermediate hosts. The third stage larva develops in the intermediate host. Definitive hosts become infected after ingestion of these intermediate hosts, whereas swine mostly become infected due to coprophagia (Wilde et al. 2001; Cappucci et al. 1982).

5.11.7 Clinical Signs in Man

In human beings, sensation occurs due to the movements of the worm. The parasite can produce irritation in buccal mucosa, and occasionally may cause bloody expectoration, tarry stools, numbness of tongue, pain in chest and abdomen, vomiting, bloating, pharyngitis and stomatitis (Cappucci et al. 1982; Kudo et al. 2008). Association between oesophageal squamous cell carcinoma and gongylonemosis has also been reported (Bleier et al. 2005).

5.11.8 Clinical Signs in Animals

In animals, the lesions include mild or chronic inflammatory changes in the affected parts. The infection may also lead to occlusions of the oesophagus due to reflex action produced by irritation.

5.11.9 Diagnosis

The disease can be diagnosed by history of moving worm followed by further parasite identifications.

5.11.10 Control

Improved personnel and environmental hygiene can help to prevent the disease.

5.12 Lagochilascariosis

Order: Ascaridida
Superfamily: Ascaridoidea

5.12.1 Etiology

There are five species of genus *Lagochilascaris*: *L. minor*, *L. major*, *L. buckleyi*, *L. turgida* and *L. sprenti* (Leiper 1909; Sprent 1971). The human disease occurs due to the nematode *L. minor* (Paco et al. 1999).

5.12.2 Epidemiology

L. minor lagochilascariosis is present in Neotropical areas (from Mexico to Brazil), Uruguay (Sakamoto and Cabrera 2002). It is an emerging zoonotic disease in Brazil (Fraiha et al. 1989; Barbosa et al. 2006; Prudente et al. 2008; Freire-Filha et al. 2001). Rural and low socioeconomic status populations are at the high risk of infection in endemic areas.

5.12.3 Reservoir and Transmission of the Disease

Natural reservoirs and transmission of the disease are not clear. Ingestion of embryonated eggs released by another species might be the cause of disease in man.

5.12.4 Life Cycle

Albeit, the life cycle is not fully understood, but there is strong belief that cats act as the definitive host for this parasite. The adult worms are present in the upper portion of the

respiratory or digestive tract and eggs are passed through faeces of the cat. Synanthropic rats and other rodents ingest the infective eggs containing third stage larvae and act as the intermediate hosts for this parasite. Further, larvae hatch in the intermediate host and migrate to different organs such as liver, lungs and form cysts in the muscle and subcutaneous tissue (deMoura et al. 2012; Campos et al. 1992; Volcan et al. 1992). Humans and the canids, act as accidental hosts after consumption of raw or undercooked infected rodent meat (Campos et al. 1992). Rodents (Paco et al. 1999) capybara (*Hidrochaeris hidrochaeris*) and agouti (*Dasyprocta aguti*) are particularly important. The developmental stages could be found in cat or other accidental definitive hosts (Leão et al. 2005).

5.12.5 Disease in Man

Important clinical signs in humans include purulent subcutaneous tissue abscess in ear, neck, jaw, orbit, mastoid process, head and presence of eggs, larval and adult parasites in retropharyngeal tissue (Leiper 1909; Olle-Goig et al. 1996; Moncada et al. 1998). In rare cases, the parasite may affect pulmonary tissue or central nervous system (Rosemberg et al. 1986; Veloso et al. 1992) leading to death (Sakamoto and Cabrera 2002).

5.12.6 Disease in Animals

The symptoms in the definitive host are not much clear. Two reported cases in cat revealed presence of fistulated abscesses (deFreitas et al. 2008; Amato et al. 1990).

5.12.7 Diagnosis

The disease can be diagnosed by identification of eggs in the faeces or from identification of the parasite in affected lesions.

5.12.8 Control

No specific control measures are known for this disease.

5.13 Mammomonogamus

Order: Strongylida
Superfamily: Strongyloidea
Family: Syngamidae

5.13.1 Common Name/Synonyms

Syngamiasis

5.13.2 Etiology

The disease occurs due to *Mammomonogamus laryngeus* (*Syngamus laryngeus*). This parasite is generally present in laryngotracheal region of cattle and some other bovines (Gutierrez 1999; Anderson et al. 1980). The other parasite *M. nasicola* is present in nasal fossa of bovids. These parasites are found in the larynx of cattle (Anderson et al. 1980) and are found rarely in human beings (Beaver et al. 1984).

5.13.3 Epidemiology

More than 100 human infections due to *M. larygeus* (Gutierrez 1999; Nosanchuk et al. 1995; da Costa et al. 2005; Angheben et al. 2009) have been reported from different countries such as Caribbean Islands, South America (especially Brazil), Australia, Canada, the United States, France, United Kingdom (Cunnac et al. 1988), Philippines (Beaver et al. 1984), Thailand (Pipitogool et al. 1992), Italy (Angheben et al. 2009) and Korea (Kim et al. 1998). The parasite is mainly found in the Caribbean and South America and other cases have been reported from tourists visiting these areas (da Costa et al. 2005).

5.13.4 Reservoir

Ruminants act as reservoirs for *M. laryngeus* and *M. nasicola*.

5.13.5 Transmission

The transmission cycle of the parasite is not clearly known. It is thought that infection occurs either after ingestion of embryonated egg, hatched larvae or paratenic host such as earthworms, snails, or arthropods. Contaminated raw vegetables or drinking water could also serve as a source of infection.

5.13.6 Life Cycle

Life cycle of the parasite is not fully understood but it is assumed that it penetrates the intestinal wall and then migrates to the tracheolaryngeal region through the body (Gutierrez 1999; Severo et al. 1988). The eggs are produced by the parasite in the tracheal mucosa, which are swallowed and are then passed through the faeces.

5.13.7 Clinical Signs in Man

The symptoms primarily include chronic cough, fever and occasionally hemoptysis. Additionally, parasite may cause irritation of the larynx and asthma.

5.13.8 Clinical Signs in Animals

The disease is not symptomatic in animals. Ruminant infections have been recorded from tropical America, Africa, India, Malaysia, Vietnam and Philippines (Acha and Szyfres 2003).

5.13.9 Diagnosis

The disease can be diagnosed by brochoscopic or expectorated identification of the parasite (Gutierrez 1999). Eggs may also be seen in sputum or faeces of infected persons.

5.13.10 Control

Improved personnel hygiene can help prevent infection in human beings.

5.14 Micronemosis

Order: Rhabditida
Superfamily: Rhabditoidea

5.14.1 Etiology

The disease occurs due to *Micronema deletrix* (Synonym: *Halicephalobus gingivalis*), a small free living nematode. The parasite is most commonly found in brain but could be found in kidneys, oral and nasal cavities, lymph nodes, spinal cord and adrenal glands (Spalding et al. 1990; Rames et al. 1995).

5.14.2 Epidemiology

Micronemosis has been reported from United States (Anderson and Bemrick 1965; Rubin and Woodard 1974; Alstad et al. 1979; Simpson et al. 1988), Switzerland (Pohlenz et al. 1981) and Netherlands (Keg et al. 1984) in horses.

5.14.3 Reservoir

This parasite is found in close association with decaying humus (Anderson and Bemrick 1965;

Poinar 1983). The opportunistic parasite depends for food on bacteria, but may become independent for its development (Poinar 1983).

5.14.4 Transmission

The route of transmission is not fully understood but it is believed to be through contamination of oral or nasal wounds, due to common parasite infestation of these sites. The manure contamination of cutaneous lacerations or decubital ulcers was also found responsible for two human cases with CNS involvement (Gardiner et al. 1981; Rames et al. 1995).

5.14.5 Life Cycle

M. deletrix is a facultative parasite of man and animals as all the developmental stages have been found in the environment outside the body of human and animal hosts (Shadduck et al. 1979; Acha and Szyfres 2003).

5.14.6 Clinical Signs in Man

In humans, three cases of *M. deletrix* micronemosis responsible for parasitic meningoencephalitis have been recorded in Canada (Hoogstraten and Young 1975) and the United States (Shadduck et al. 1979; Gardiner et al. 1981).

5.14.7 Clinical Signs in Animals

Among horses, the parasite was found to cause infection in nasal passages, lung, lymph nodes, stomach, maxillae, mandible, brain, kidney, adrenal glands and femur (Gordon et al. 1990).

5.14.8 Diagnosis

The diagnosis can be done at the time of postmortem using histological examination of the affected organs.

5.14.9 Control

Improved personnel hygiene can help prevent infection in human beings.

5.15 Oesophagostomiasis

Order: Strongylida
Superfamily: Strongyloidea
Family: Trichonematidae

5.15.1 Common Name/Synonyms

Esophagostomiasis, Ternidensiasis

5.15.2 Etiology

Oesophagostomiasis occurs due to strongylid intestinal parasites *Oesophagostomum bifurcum*, *O. stephanostomum*, *O. aculeatum* and *Ternidens derminutus* of non-human primates and humans (Remfry 1978; McCarthy and Moore 2000). Out of these, *O. bifurcum* was considered a rare zoonosis. The zoonotic potential of *O. bifurcum* is still not fully understood. Recently, epidemiological, molecular and morphological studies on *O. bifurcum* from humans and various monkeys clarified that different strains of *O. bifurcum* are prevalent in humans and monkeys and they are specific to their respective hosts indicating a non-zoonotic transmission between these species (Polderman et al. 2010). However, another study carried out in western Uganda indicates that *O. bifurcum* are prevalent in chimpanzees which have potential for zoonotic transmission of these species (Krief et al. 2010).

5.15.3 Epidemiology

O. bifurcum is widely prevalent in West Africa, but a few cases have also been reported from other parts of Africa, Asia and South America (Bogers et al. 2001).

5.15.4 Reservoir

Non-human primates are thought to be the important reservoir hosts.

5.15.5 Transmission

Humans become infected after ingestion of infective larvae from soil contaminated with faeces of non-human primates.

5.15.6 Life Cycle

Humans or primates become infected after ingestion of L3 larvae. The larvae penetrate colon and some of these larvae develop and return to the bowel lumen, while others become immature worms and lead to formation of abscess in the bowl lumen. Adult mature worms mate and shed eggs in the faeces. The eggs hatch in soil and develop from L1 to L3 infective larvae (Polderman and Blotkamp 1995).

5.15.7 Clinical Signs in Man and Animals

Most of the times, infections are not symptomatic. Two forms of the disease viz. multinodular and uninodular (Dapaong tumour) can occur (Polderman and Blotkamp 1995). Clinical signs include 'Dapaong tumour', named after the capital of Togo's northernmost province. In this an inflammatory mass develop around juvenile worms in the colonic wall or abdominal cavity. Adhesions develop around the abdominal wall and this may lead to occasional obstruction of colon (Storey et al. 2000; Polderman et al. 1999). Heavy infections can lead to diarrhoea, weight loss, weakness and death in man.

5.15.8 Diagnosis

The disease can be diagnosed after parasitic identification and stool culture on stool charcoal mixture (Polderman et al. 1991), PCR–RFLP and Random Amplified Polymorphic DNA—Gel—electrophoresis.

5.15.9 Control

Improved personnel hygiene, chemotherapy of infected animals can help control the infection.

5.16 Strongyloidiasis

Order: Rhabditida
Superfamily: Rhabditoidea
Family: Strongyloididae

5.16.1 Common Name/Synonyms

Strongyloidiasis, threadworm infection

5.16.2 Etiology

The disease occurs due to the parasite *Strongyloides stercoralis and S. fuelleborni*. Role of the animal hosts dogs and cats for *S. stercoralis* zoonotic infections is not clearly understood. It is believed that they might be contributing to human infections in some parts of the world. The zoonotic potential of *S. fuelleborni* is believed to be much higher than *S. stercoralis*. It mainly infects non-human primates but can cause zoonotic disease in Africa and Southeast Asia or at least have a zoonotic origin (Ashford and Barnish 1989; Olsen et al. 2009). Other S*trongyloid* infections can also cause dermatitis in man which can be zoonotic in nature.

5.16.3 Epidemiology

S. stercoralis infections could be seen across the globe but is particularly endemic in tropical and subtropical areas viz. Southeast Asia, sub-Saharan Africa, Latin America, and the south-eastern United States (Berk et al. 1987). The parasite is believed to be affecting 100–200 million persons worldwide (Genta 1989). *S. fullerborni* infections occur in non-human primates of Africa and Asia. *S. stercoralis* infection can occur for very long periods of time due to the ability of the parasite to cause autoinfection which result in persistent chronic infections (Grove 1996). Poor sanitary conditions are important factor for *S. stercoralis* infections (Hall et al. 1994).

5.16.4 Reservoir

Humans are reservoir for *S. stercoralis* infections. For *S. fuelleborni* infections, African and Asian simians serve as important reservoirs.

5.16.5 Transmission

The disease occurs through direct skin contact with contaminated soil particularly in agricultural and recreational activities.

5.16.6 Life Cycle

The parasite has both free-living and parasitic stages. The female parasite present in the small intestine lays eggs in the intestinal mucosa. The eggs develop into larvae and are shed in the faeces. In suitable environment, larvae develop into infective filariform larvae or develop into free-living adults. The sexual reproduction of the parasite occurs in the free-living adult stage (Neva 1994). The parasite generally enters the human body through cutaneous route, enters the bloodstream and reaches through lungs finally to the gastrointestinal tract (Keiser and Nutman 2004).

5.16.7 Clinical Signs in Man

The infection primarily remains asymptomatic (Grove 1989) but can cause symptoms related to cutaneous (pruritus, urticaria), respiratory (cough, chronic bronchitis) and intestinal (abdominal pain, intermittent or persistent diarrhoea) systems. However, in immune compromised individuals, the parasite can cause a fulminant fatal disease (Keiser and Nutman 2004) viz. hyperinfection (uncontrolled multiplication) and dissemination of larvae to all the visceral organs (Olsen et al. 2009).

5.16.8 Clinical Signs in Animals

The disease in dogs and cats is not long lasting and generally infects young puppies. The symptoms are self limiting and generally resemble as in healthy human beings. Among non-human primates, mild to severe haemorrhagic diarrhoea could occur particularly in young or weakened animals (Acha and Szyfres 2003).

5.16.9 Diagnosis

The parasite can be detected using faecal concentration or culture techniques (Cheesbrough 1987). Immunological tests such as Western blot, ELISA and IFAT can also be used in the diagnosis (Silva et al. 2003; Rodrigues et al. 2007).

5.16.10 Control

Proper disposal of human faeces, treatment of infected persons and using protective measures in endemic areas can help control the disease.

5.17 Thelaziasis

Order: Spirurida
Superfamily: Spiruroidea
Family: Thelaziidae

5.17.1 Common Name/Synonyms

Oriental eye-worm, thelaziasis, eye worm infections, conjunctival spirurosis

5.17.2 Etiology

Thelazia callipaeda is a helminth responsible for causing eye infection in humans and animals such as domestic cats and dogs (Otranto and Eberhard 2011). *T. callipaeda* causes insect-borne zoonosis in man. Some other species such as *T. californiensis* and *T. rhodesii* could also cause the disease.

5.17.3 Epidemiology

Among humans, the parasite is distributed in erstwhile Soviet Union and Asian countries such as China, Korea, Japan, Indonesia, Thailand, Taiwan and India (Otranto and Eberhard 2011). More than 157 cases have been reported across the globe in addition to 100 cases reported from Japan (Koyama et al. 2000; Kitano et al. 1996; Singh and Singh 1993). Children and old person are at higher risk of being infected. The parasite is also widespread in domestic and wild animals in Asia, Italy, France and Germany (Miyazaki 1991; Otranto et al. 2003a; Kosin et al. 1989; Shen et al. 2006; Sohn et al. 2011).

5.17.4 Reservoir

Domestic and wild mammals serve as important reservoirs of disease.

5.17.5 Transmission

The disease is transmitted by secretophagous flies which deposit it in the eye orbit of human beings (Otranto and Traversa 2005) and other animals.

5.17.6 Life Cycle

The secretophagous flies play central role in transmission of this disease. These flies feed on the lachrymal secretions of man and other infected animals and thus ingest first stage larvae in the process. The third stage larva develops in the fly and is deposited back into the orbit of the eye.

5.17.7 Clinical Signs in Man

Clinical signs in man include lachrymation, epiphora, conjunctivitis, keratitis and corneal ulcers in severe cases (Anderson 1992).

5.17.8 Clinical Signs in Animals

Domestic dogs, cats and wild carnivores (e.g. foxes, wolves and wild cats) are primarily affected (Otranto et al. 2009). Symptoms are generally similar as in the human infections.

5.17.9 Diagnosis

The disease can be diagnosed after removal of the worm from the eye followed by morphological identification of the parasite (Otranto et al. 2003b).

5.17.10 Control

Avoidance of eye contact with intermediate hosts can prove beneficial for prevention of the disease.

5.18 Trichinosis

Order: Enoplida
Superfamily: Trichuroidea
Familly: Trichinellidae

5.18.1 Common Name/Synonyms

Trichinellosis, Trichiniasis, Trichinelliasis

5.18.2 Etiology

The disease occurs due to the parasites of genus *Trichinella*. At present, two clads are recognised: One that contain species which encapsulate in host muscle cell (*T. spiralis*, *T. nativa*, *T. britovi*, *T. murrelli*, *T. nelsoni and Trichinella T6*, *T8*, *T9 and T12*) and second that do not encapsulate in host muscle cell (*T. psudospiralis*, *T. papuae*, *T. zimbabwensis*). All the species present in the first clad only infect mammals, whereas in second clad, *T. pseudospiralis* infects mammals and birds; and *T. papuae*, *T. zimbabwensis* both infect mammals and reptiles (Gajadhar et al. 2009).

5.18.3 Epidemiology

The parasite causes serious zoonosis and as many as 11 million people may be infected (Dupouy-Camet 2000). Numerous outbreaks of trichinellosis have been reported in different parts of the world (Dupouy-Camet 2000). *Trichinella* infection has been reported in domestic (mainly pigs) and wild life species of 43 and 66 countries, respectively (Pozio 2007). Trichinellosis is a worldwide prevalent nematode zoonotic disease of medical, veterinary and economic significance (Dupouy-Camet 2000). *Trichinella* is one of the most wide spread parasites infecting people and other mammals across the globe in most climates, except for deserts (Dupouy-Camet 2000). Domestication of pigs some 10,000 years ago in Asia created a permanent reservoir of parasites for humans, limited by the use of fire and Mosaic laws (Dupouy-Camet 2000). Trichinellosis significantly impact national economies due to reasons of public health, socioeconomics and trade (Todd 1989; Roberts and Murrell 1993; Ortega-Pierres et al. 2000). The occurrence of significant transmission from scavenged animal carcasses suggests that environmental temperature and humidity play an important role in the transmission of *Trichinella* among wild life (Pozio 2000). Environmental contamination with *Trichinella*-infected meat scraps could lead to infection among herbivores, particularly in horses (Pozio 2000). Unhygienic slaughtering of food animals, roaming behaviour of stray pigs, lack of structured meat inspection, commonly used garbage and kitchen practices for pigs, free access of dogs and cats to slaughter houses, socioeconomic conditions, culinary habits etc. undoubtedly enhances perpetuation of the transmission cycle.

5.18.4 Reservoir

Wild boar or other animals are important reservoirs of infection.c

5.18.5 Transmission

Consumption of raw or undercooked pork or wild boar meat is important source for transmission of the disease in man.

5.18.6 Life Cycle

Trichinella spp. can complete its life cycle in a single host. After ingestion of infected meat containing first stage infective larvae, digestion of meat in the stomach releases larva which mature in small intestine in few days (Gajadhar et al. 2006) (Fig. 5.6).

The adult female starts shedding larvae. The larvae enter blood circulation and reach skeletal muscles and encapsulate and usually live there for 3–5 years or more (Gajadhar et al. 2006). The infected meat when consumed by another host restarts the life cycle of the parasite. High numbers of *Trichinella* larvae are present in diaphragm, tongue and massetar muscles (Gajadhar et al. 2006).

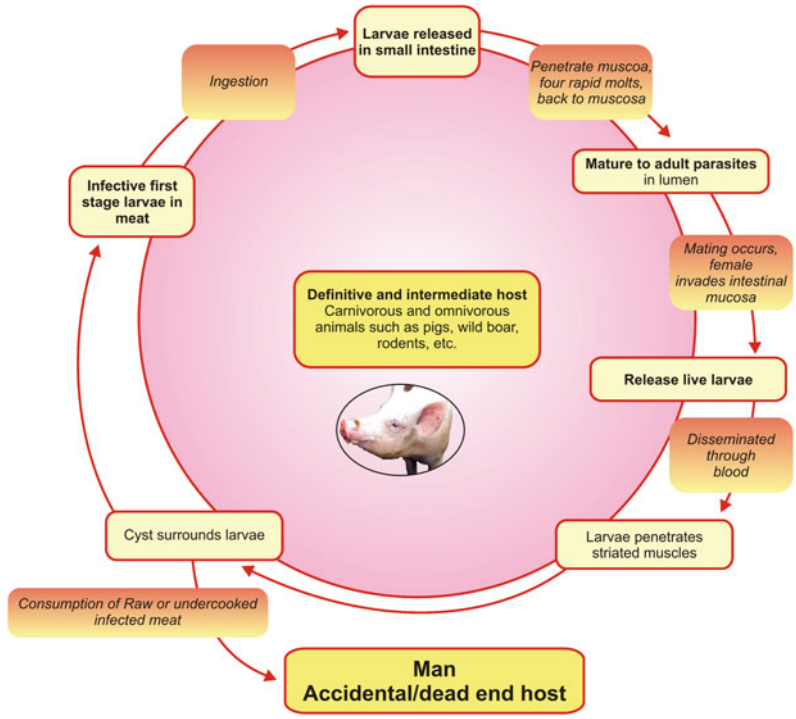

Fig. 5.6 Life cycle of *Tichinella spiralis*

5.18.7 Clinical Signs in Man

The number of larvae present, genotype involved and host immunity determine the severity of disease in man. Clinical signs vary from asymptomatic infections to diarrhoea, gastrointestinal disturbances, periorbital and facial oedema, myalgia, fever, conjunctivitis, headache and skin rash (Gajadhar et al. 2006). In extreme cases, life threatening encephalitis, endocarditis, myocarditis may also be seen (Gajadhar et al. 2006).

5.18.8 Clinical Signs in Animals

The disease is generally asymptomatic in animals. Among animals, pigs, bear, canids, rodents, skunks, raccoon, walrus, opossum and seals can act as hosts.

5.18.9 Diagnosis

The disease can be diagnosed using trichinoscopy, *Trichinella* artificial digestion assay, serological, histological, bioassay and molecular techniques. The digestion assay is more reliable and recommended test to trichinoscopy (Figs. 5.7, 5.8).

5.18.10 Control

Proper meat inspection practices, rodent control, avoiding garbage feeding to pigs, proper carcass disposal can help control the disease. Consumption of properly cooked meat can avoid infection in man (Gajadhar et al. 2006).

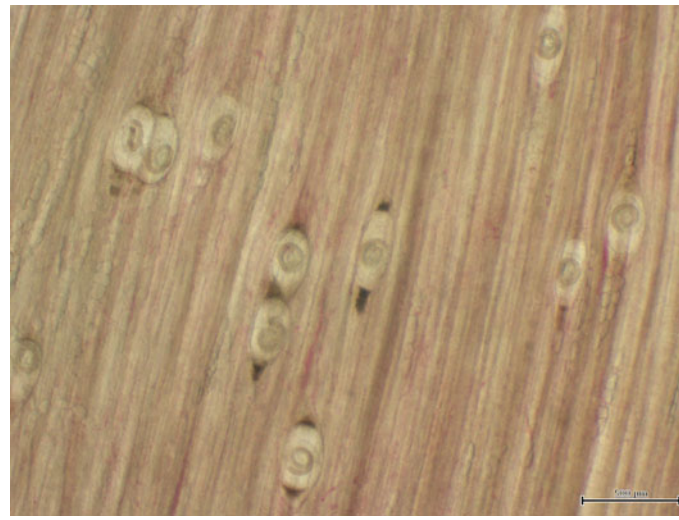

Fig. 5.7 Numerous coiled infective *Trichinella* larvae seen using trichinelloscopy (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Center for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

Fig. 5.8 Coiled infective *Trichinella* larvae recovered from horse meat using pepsin HCl digestion assay (with kind permission from Dr. Alvin A Gajadhar, Research Scientist cum Head, Center for Food-Borne and Animal Parasitology, Canadian Food Inspection Agency Saskatoon Lab, Canada)

5.19 Trichostrongylosis

Order: Strongylida
Superfamily: Trichostrongyloidea
Family: Trichostrongylidae

5.19.1 Common Name/Synonyms

Trichostrongyliasis

5.19.2 Etiology

There are several species of the genus which can infect sheep, goat and bovines. For human infections, cases due to *Trichostrongylus orientalis*, *T. axei*, *T. colubriformis*, *T. skrjabini*, *T. vitrinus*, *T. probolurus*, *T. capricola*, *T. brevis*, *T. affinis and T. calcaraus* have been recorded (Acha and Szyfres 2003).

5.19.3 Epidemiology

The disease has been reported across the globe. Sporadic human cases have been reported from Asian countries such as Thailand, South Korea, China, Laos; United States and Australia (Sato et al. 2011; Beaver et al. 1984; Boreham et al. 1995; Panasoponkul et al. 1985; Yong et al. 2007; Wall et al. 2011). Farmers and other persons involved in dairy farming are at risk of being infected particularly in the developing world and endemic areas (Adams et al. 2005). Among animals, high prevalence has been recorded in Asia and Middle East (Youn 2009; Giboda et al. 1991; Poirriez et al. 1984).

5.19.4 Reservoir

Herbivorous animals act as reservoirs for most of the *Trichostrongylus* species.

5.19.5 Transmission

Human beings become infected after consumption of contaminated food and water particularly in the places where animal faeces are used for soil fertilisation.

5.19.6 Life Cycle

The host species releases eggs in the faeces. These eggs develop into first stage larva. After feeding on organic matter, these larvae further develop into third stage larva which is infective to the host. After ingestion of third stage larva, it mature into adult and start releasing eggs in the faeces of the host.

5.19.7 Clinical Signs in Man

Disease generally remains asymptomatic but in high parasitemia, clinical symptoms may include eosinophilia, bloating diarrhoea, abdominal pain, weight loss and some other abdominal disorders.

5.19.8 Clinical Signs in Animals

The disease is responsible for production losses in animals.

5.19.9 Diagnosis

The disease can be diagnosed using identification of eggs followed by faecal culture so as to identify larvae for species differentiation.

5.19.10 Control

For prevention and control of the disease, pasture management, stock rotation and regular de-worming of animals particularly in endemic areas should be practised. For human beings, improved personnel and environmental hygiene can help to prevent the disease.

5.20 Trichuriasis of Animal Origin

Order: Enoplida
Superfamily: Trichuroidea
Family: Trichridae

5.20.1 Common Name/Synonyms

Whipworm disease, trichocephaliasis

5.20.2 Etiology

The zoonotic trichuriasis occurs due to *Trichuris vulpis* and *Trichuris suis* which can be occasionally present in intestine of man. Rarely, *T. vulpis* is responsible for causing VLM in man. *T. vulpis* primarily infects domestic and wild canids, whereas *T. suis* causes infection in domestic pigs and wild boars. *T. trichuria* is another parasite which could cause infection in man (Acha and Szyfres 2003).

5.20.3 Epidemiology

Both the parasites are distributed across the globe. Malnourished 5–6 year old children in tropical areas are at high risk of being infected (Márquez-Navarro et al. 2012).

5.20.4 Reservoir

Dogs, wild canids and swine could act as important reservoirs of infection.

5.20.5 Transmission

Trichuriasis of animal origin is a soil transmitted nematode infection. The infection is transmitted directly through faecal oral route due to contaminated soil or water.

5.20.6 Life Cycle

The animal or human host becomes infected after ingestion of infective eggs. After gastric passage, larvae are released in the small intestine from these eggs. First stage larva reaches the large intestine and after multiple moults develops into the adults.

5.20.7 Clinical Signs in Man

The disease is mostly asymptomatic but diarrhoea, dysentery, rectal prolapse, abdominal pain, vomiting, headache etc. may be seen in heavy infections.

5.20.8 Clinical Signs in Animals

The disease is generally asymptomatic but heavy infections can cause symptoms related to gastrointestinal system, such as diarrhoea and secondary disease complications.

5.20.9 Diagnosis

The disease can be diagnosed by identification of eggs in the faeces of the host.

5.20.10 Control

Improved personnel and environmental hygiene, proper disposal of excreta, hand washing, consumption of hygienic food and water can help to prevent the infection in children.

5.21 Visceral Larva Migrans

Order: Ascaridida
Superfamily: Ascaridoidea
Family: Ascarididae

Beaver et al. (1952) for the first time reported migration of *Toxocara* larvae to the liver. After that, many cases of these larvae migrating to liver, lungs and other visceral organs have been reported (Inoue et al. 2002; Roig et al. 1992; Morimatsu et al. 2006).

5.21.1 Common Name/Synonyms

Toxocariasis, Toxocarosis, Larval granulomatosis, OLM.

5.21.2 Etiology

The human disease occurs due to the larvae of *Toxocara* sp. roundworms. The human disease occurs due to *Toxocara canis* and *T. cati*. In addition to these, *T. vitulorum* has also been recognised as rare cause of this zoonosis. The zoonotic potential of *T. pteropodis* is being questioned. *T. canis* toxocariasis is considered to be more important than *T. cati* infections. For *T. canis*, dogs and some other canids act as definitive host, whereas cats act as definitive host for *T. cati*.

5.21.3 Epidemiology

The disease is distributed across the globe and eggs have been found in soil from most parts of the world. High seroprevalences have been recorded from tropical countries.

5.21.4 Reservoir

Dogs act as important reservoir for human infections.

5.21.5 Transmission

The parasite normally affects young children after accidental ingestion of infective eggs from contaminated soil, unwashed hands, contaminated food and water or through ingestion of raw or undercooked tissues (particularly liver). The infection occurs through contamination of soil and direct contact with pets is not likely to cause infection in human beings. Occurrence of cases after ingestion of meat from quails, cows and chickens (Sakakibara et al. 2002) suggests its importance as food-borne parasitic zoonosis.

5.21.6 Life Cycle

The adult parasite sheds un-embryonated eggs in the faeces of the definitive host. The eggs become infective to third stage larvae within 7–14 days in the environment under favourable conditions. The larvae enter the host through ingestion of third stage larvae present in the contaminated soil or water. The larvae hatch in the digestive system and migrate towards the liver and then disseminate to other organs, such as lungs and heart (Mok 1968; Woodruff 1970). In definitive hosts, some larvae again reach intestine through lungs and develop into adult worms and start releasing the eggs. For kittens, *T. cati* toxocariasis is not transmitted in utero but can be transmitted through milk or colostrum (Fig. 5.9).

5.21.7 Clinical Signs in Man

The disease is generally not symptomatic. Clinical symptoms in man can be discussed under four different forms viz. visceral or systemic, ocular, neural and covert toxocariasis (Acha and Szyfres 2003). In visceral form, symptoms could vary from asymptomatic to chronic eosinophilia, hepatomegaly, pneumonitis, fever and digestive disorders (Acha and Szyfres 2003). The ocular form could lead to variety of symptoms such as retinal granulomas, retinal detachment, Keratitis and can cause loss of vision and blindness. Neurological form may lead to meningoencephalitis. The covert form is not uncommon, and may produce symptoms such as malaise, abdominal pain and eosinophillia (Acha and Szyfres 2003).

5.21.8 Clinical Signs in Animals

Adult cat and dogs generally do not show symptoms for the disease. Young puppies and kittens may show symptoms such as diarrhoea, vomiting and other digestive disturbances.

5.21.9 Diagnosis

The human infections can be diagnosed based on clinical signs, ophthalmoscopic examination, eosinophilia, other clinical pathology findings, serological tests, histologically or isolation of larva (Aragane et al. 1999). The disease in animal hosts can be diagnosed by detection of the eggs.

5.21.10 Control

For prevention and control, regular de-worming of definitive hosts, especially their puppies and kittens must be done. Health education of persons in close contact with these animals should also prove helpful.

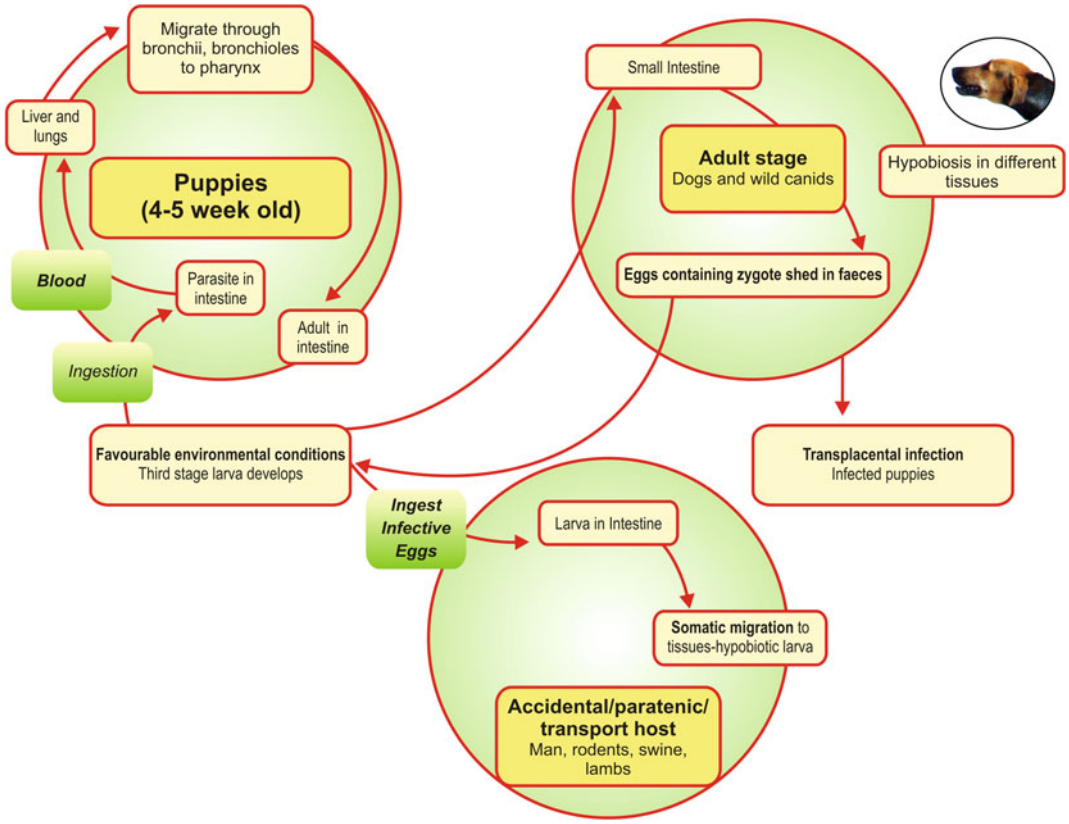

Fig. 5.9 Life cycle of *Toxocara canis*

5.22 Zoonotic Ancylostomiasis

Order: Strongylida
Superfamily: Ancylostomatoidea
Family: Ancylostomatidae

5.22.1 Common Name/Synonyms

Hookworm disease, necatoriasis, uncinariasis

5.22.2 Etiology

The disease occurs due to *A. ceylanicum* (cat nematode) and *A. caninum* (dog nematode).

5.22.3 Epidemiology

Human infections are rare but have been reported from many parts of the world. These parasites are endemic in India (Traub et al. 2007, 2008, 2004) and south East Asia (Choo et al. 2000; Setasuban et al. 1976; Yoshida et al. 1968). Detailed publications on human ancylostomiasis due to *A. ceylanicum* were published between the mid 1960s and early 1970s (Anten and Zuidema 1964; Areekul et al. 1970; Chowdhury and Schad 1972; Yoshida et al. 1968). Recent studies by Ngui et al. (2012), Traub et al. (2008) revealed using the combination of epidemiological, conventional diagnostic and molecular tools that *A. ceylanicum* is common in endemic areas particularly where

there is close contact between human and cat, dog populations. *A. caninum* is responsible for human eosinophilic enteritis (EE) and has been reported from Australia (Loukas et al. 1992; Croese et al. 1994a; Landmann and Prociv 2003), United States (Khoshoo et al. 1995), Egypt (Bahgat et al. 1999), Philippines, South America and Israel (Croese et al. 1994b). *A. ceylanicum* is able to produce patent infections in human beings both naturally and experimentally (Ngui et al. 2012; Wijers and Smit 1966; Carroll and Grove 1986). Human infections due to *A. ceylanicum* have also been reported from West New Guinea (Anten and Zuidema 1964), Philippines (Velasquez and Cabrera 1968), Taiwan (Yoshida et al. 1968), Thailand (Areekul et al. 1970; Traub et al. 2008; Jiraanankul et al. 2011), Laos (Sato et al. 2011) and India (Chowdhury and Schad 1972).

5.22.4 Reservoir

Dogs can act as reservoir hosts for human hookworm infections.

5.22.5 Transmission

Ingestion of contaminated soil or food like vegetables could transmit infection in human beings.

5.22.6 Life Cycle

The parasite *A. ceylanicum* has a direct life cycle. This parasite is present in small intestine of dogs, cats and humans. The infection occurs due to ingestion or percutaneous migration of infectious third stage larvae from the soil. The host animal passes un-embryonated eggs in the faeces after 14 days of entry into the host (Yoshida et al. 1974). The infectious larvae usually develop in 2–8 days under favourable conditions.

5.22.7 Clinical Signs in Man

A. ceylanicum can develop into adult in human beings (Tu et al. 2008; Anten and Zuidema 1964) and produces natural patent infections causing anaemia (Anten and Zuidema 1964; Velasquez and Cabrera 1968; Chowdhury and Schad 1972; Traub et al. 2008). *A. caninum* occasionally reaches adulthood in humans (Landmann and Prociv 2003; Prociv and Croese 1990; Croese et al. 1994a, b) and can cause eosinophilic enteritis (Prociv and Croese 1996).

5.22.8 Clinical Signs in Animals

Heavy infections due to *A. ceylanicum* can cause diarrhoea, bloody and mucoid stool and iron deficiency anaemia (Carroll and Grove 1984) in dogs. Migrating larva in heavy infections can affect and cause symptoms in cutaneous, respiratory and intestinal systems.

5.22.9 Diagnosis

The disease can be diagnosed by identification of parasite eggs, from clinical signs in animals such as anaemia (pale mucus membranes), rough hair coat, by use of serological tests such as ELISA, western blot and molecular techniques (Sato et al. 2010).

5.22.10 Control

Control measures for non-zoonotic human ancylostomiasis such as prophylactic and therapeutic treatment and improved sanitation for animals also work well for control of zoonotic ancylostomiasis.

5.23 Zoonotic Filariasis

Order: Spirurida
Suborder: Filariina
Superfamily: Filariroidea

5.23.1 Common Name/Synonyms

Brugia malayi filariasis

5.23.2 Etiology

Many species of animal filariasis are reported to cause disease in man (Addario 1885; Gutierrez 1990; Orihel and Ash 1995).

Zoonotic filariasis could primarily occur due to the following etiological agents:

1. *Dirofilaria* spp. viz. *D. immitis*, *D. tenuis*, *D. repens*
2. *Onchocerca* spp. viz. *O. cervicalis*, *O. gutturosa*
3. *Brugia* spp. viz. *B. malayi*, *B. pahangi*, *B. leporis*
4. Other species such as *Loaina* sp., *Meningonema* sp., and some unidentified species

5.23.3 Epidemiology

Cases due to zoonotic filariasis have been reported across the globe (Orihel and Eberhard 1998; Thomas and Mark 1998). Zoonotic filariasis has been reviewed in detail by Orihel and Eberhard (1998). Different species of *Brugia* infect animals such as tree shrews and monkeys in Southeast Asia, and raccoons and rabbits in the United States (Eberhard 1984; Orihel 1966; Orihel and Pacheco 1966). Human infections due to *D. repens* have been reviewed by Pampiglione et al. (1995) and reported 400 cases from 30 countries in Europe, Asia and Africa. Most human infections have been reported from temperate zones (Orihel and Eberhard 1998).

5.23.4 Reservoir

Monkeys, cats and wild carnivores act as reservoir for subperiodic brugiasis in Southeast Asia (Acha and Szyfres 2003). Other wild and domestic animals might also act as reservoirs for the disease.

5.23.5 Transmission

The disease is transmitted by the bite of blood sucking insects such as mosquitoes belonging to the genra *Mansonia*.

5.23.6 Life Cycle

Zoonotic filariasis is a vector borne disease and blood sucking insects act as biological vectors for transmission of the disease. The adult parasite lives in vertebrate host's tissues. The female releases embryos (microfilariae) which can be found in blood, lymph or skin. The vectors transmit the infection by feeding previously on an animal infected with a patent filaria infection (Anderson 1992). The arthropod vector ingests microfilariae present in blood, lymph or skin. The microfilariae further develop into infectious third stage larva and migrate towards the mouthparts of the arthropod. The insect vector then transmits the infection while feeding on another new vertebrate host.

5.23.7 Clinical Signs in Man

Brugia spp. infects lymphatic system and produces symptoms such as lymphadenopathy and lymphangitis. *Dirofilaria* spp. generally infects skin and subcutaneous tissue and produce related symptoms such as nodules and swellings. *D. immitis* can infect heart, pulmonary and other blood vessels and produce related symptoms.

5.23.8 Clinical Signs in Animals

The disease is generally asymptomatic but large number of parasites could pose problems such as stenosis of pulmonary vessels, obstruction in flow of blood.

5.23.9 Diagnosis

Blood smear examinations and Millipore filter concentration techniques, PCR, ELISA can be used for diagnosis of parasites present in the human blood.

5.23.10 Control

Control of vector populations can help prevent and control these infections.

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Arthropod Zoonoses

6

Abstract

Arthropod Zoonoses could be defined as "those arthropod diseases which are naturally transmitted between (other) vertebrate animals and man". Infestation of dipteran fly larva could cause myiasis in animals and man. Pentastomiasis is a zoonotic disease which can occur in human beings. The disease occurs due to *Armillifer* and *Linguatula* species (Families Porocephalidae and Linguatulidae); endoparasites of reptiles, birds and mammals. Tungiasis occurs due to the infestation of female sand flea *Tunga* (*Sarcopsylla*) *penetrans* and could result in tungiasis in dogs, cats, pigs, non-human primates and occasionally man. Tungiasis has been reported from many countries. Zoonotic scabies occurs due to *Sarcoptes scabiei* which affects many mammal and marsupial species.

6.1 Myiasis

Class: Insecta
Order: Diptera

The disease occurs due to dipteran fly larva which infests live vertebrates viz. animals and humans (Zumpt 1965). These larvae feed on the living/dead tissue/body fluids of the host or on ingested food (Francesconi and Lup 2012). The larvae can infest different locations and produce spectrum of symptoms depending upon the larvae species and the host involved (Noutsis and Millikan 1994; Francesconi and Lup 2012).

6.1.1 Etiology

Although many species can cause the disease, the important etiological agents include:

6.1.1.1 Specific Parasites

- Larvae of *Cochliomyia hominivorax* (screw worm)
- Larvae of *Chrysoma bezziana*
- Furuncular myiasis due to larva of *Cordylobia anthropophaga*
- Furuncular myiasis due to larvae of *Dermatobia hominis*
- Furuncular myiasis due to *Cuterebra* spp.
- Larvae of *Hypoderma* spp.
- Larvae of *Oestrus ovis* and *Rhinoestrus purpureus*
- Larvae of *Gasterophilus* spp.
- Furuncular myiasis due to *Wohlfahrtia* spp.

6.1.1.2 Facultative or Semi Specific Myiasis

- Due to larvae of *Lucilia sericata, Sarcophaga, Parasarcophaga, Phormia, Paraphromina* species

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6.1.1.3 Accidental Myiasis

- Due to larvae of *Musca domestica, Musca stabulans, Fannia canicularis, F. scalaris*

6.1.2 Epidemiology

The disease is worldwide in distribution. High prevalence has been recorded from poor socio-economic regions of tropical and subtropical countries (Francesconi and Lup 2012). Animals play an important role and man generally acts as an accidental host. Poor hygiene and low socioeconomic status are the important risks for the occurrence of the disease (Fernandes et al. 2009; Marquez et al. 2007).

6.1.3 Clinical Signs in Animals

Myiasis could lead to severe production losses such as reduced milk yield, inferior quality of hide, decreased weight and fertility losses (Francesconi and Lup 2012).

6.1.4 Clinical Signs in Man

Depending upon symptoms, human myiasis has been discussed under following headings.

6.1.4.1 Cutaneous Myiasis

This is most frequently encountered form of myiasis (Diaz 2009).

1. Furuncular myiasis: It occurs when dipteran larvae penetrate healthy skin. Symptoms include erythematous, furuncle-like nodule containing one or more maggots in it. It mainly occurs due to *Dermatobia hominis* and *Cordylobia anthropophaga*. Important clinical symptoms include pruritus, pain, and movement sensation (Mahal and Sperling 2012).
2. Migratory (creeping) myiasis: It occurs due to aimless migration of dipteran larva through burrows in the skin. It produces migratory patterns of the lesions. Clinical symptoms include painful and migratory swelling.
3. Wound myiasis: It results due to local tissue destruction and further invasion into deep tissues.

6.1.4.2 Cavitary Myiasis

Cavitary myiasis occurs due to infestation of body cavities by the larvae. The symptoms vary depending upon the cavity or organ affected or involved. For example in ophthalmomyiasis, important clinical symptoms include vision loss and eye pain.

6.1.5 Diagnosis

The disease can be diagnosed from clinical signs, ultrasound examination or by detection of the larva.

6.1.6 Control

Improved personnel and environmental sanitation, measures for control of flies, maggot therapy and reducing animal myiasis can help control the infection.

6.2 Pentastomiasis

Class: Pentastomida
Family: Porocephalidae

6.2.1 Common Name/Synonyms

Porocephaliasis, Tongue worm infection, golf caddy's disease, halzoun, marrara

6.2.2 Etiology

Pentastomiasis is a zoonotic disease which can infect human beings. These parasites are

endoparasites of reptiles, birds and mammals. The disease occurs due to parasites belonging to families Porocephalidae and Linguatulidae viz. *Armillifer* and *Linguatula* species. The disease can occur in two forms viz. visceral or nasopharyngeal pentastomiasis. The visceral form occurs when humans ingest infective eggs of *Armillifer armillatus* or *L. serrata* and act as intermediate host.

Nasopharyngeal pentastomiasis occurs when man ingests raw or undercooked infected intermediate host (e.g. sheep or goat) containing encysted nymphs of the parasite *L. serrata.*

6.2.3 Epidemiology

The disease is worldwide in distribution with many reports from Asia and tropical Africa. *A. armillatus* is primarily found in Africa whereas human cases due to *L. serrata* have been reported from North Africa, Europe and Middle East (Acha and Szyfres 2006; Aynimode et al. 2010). Pentastomiasis among humans in China have been found to be associated with the ingestion of raw or undercooked snakes (Min et al. 2008).

6.2.4 Transmission

Vertebrate animals including primates and humans act as intermediate hosts (Lang et al. 1987). Human beings become infected either after consumption of contaminated water or vegetables contaminated by faeces of definitive host or due to the naso bronchial secretions of infected snakes or carnivores, or by eating raw or undercooked snake or lizard (Machado et al. 2006).

6.2.5 Life Cycle

For *Armillifier* species, snakes act as definitive hosts and rodents and other wild mammals act as intermediate hosts. For *Linguatula* spp., herbivorous animals such as sheep, goat act as intermediate hosts and dogs, canids and some felids act as definitive hosts. Intermediate hosts become infected after the ingestion of the egg contained either in faeces or in the sputum of the definitive hosts. The eggs develop to larva in the intermediate host and then burrow towards the abdominal cavity. Larvae mature to nymphs and remain in the viscera in an encysted form. Definitive hosts become infected after consumption of the intermediate host containing the encysted nymphs.

6.2.6 Clinical Signs in Man

The disease is generally not symptomatic but could produce symptoms when encysted larvae enlarge through molting and cause pressure or during larval migration in vital organs. There are reports of vital organ collapse viz. lung (Stock 1946), intestinal obstruction (Cannon 1942), peritonitis (Herzog et al. 1985) and glaucoma (Lang et al. 1987). Liver is another commonly affected organ.

6.2.7 Clinical Signs in Animals

In definitive hosts, *Linguatula* infections could produce symptoms such as nasal catarrah, sneezing and epistaxis (Acha and Szyfres 2006). In general, the infections remain asymptomatic in intermediate hosts.

6.2.8 Diagnosis

In definitive hosts, the disease can be diagnosed by the identification of the characteristic thick-shelled eggs containing an embryo with rudimentary legs. Intermediate host infections are difficult to diagnose and may be diagnosed either by X-rays or histopathological examination.

6.2.9 Control

Improved personnel hygiene and avoid eating raw or undercooked infected meat for prevention of the disease.

6.3 Tungiasis

Order: Siphonaptera
Family: Tungidae

6.3.1 Synonyms

Chigoe, jigger flea infestation sand flea infestation

6.3.2 Etiology

The disease occurs due to the infestation of female sand flea *Tunga* (Sarcopsylla) *penetrans*.

6.3.3 Epidemiology

This flea could infest warm-blooded animals including dogs, cats, pigs, non-human primates and man. The disease has been reported from Central, South and Latin America, Caribbean, Africa and India (Leung et al. 2007).

6.3.4 Transmission

In endemic areas, human beings could become infected when they walk barefoot on the soil containing the fleas.

6.3.5 Life Cycle

The fleas are hematophagous (Eisele et al. 2003; Fein et al. 2001) and male dies quickly after the copulation. The female flea penetrates in the host epidermis and lay eggs for 2–3 weeks before death. The eggs fall and mature into larvae on the soil (Fein et al. 2001). The larvae develop to pupae and finally develop into adult flea. The whole cycle is completed in 18 days under favourable conditions (Leung et al. 2007).

6.3.6 Clinical Signs in Man and Animals

Clinical symptoms range from asymptomatic to pruritic, painful ulceration having papular or nodular lesion with a central dark spot. The flea commonly infests feet but could affect any part which comes in contact with the soil (Leung et al. 2007). The symptoms could be further deteriorated with secondary bacterial infections.

6.3.7 Diagnosis

The disease can be diagnosed from clinical symptoms or by identification of flea from characteristic lesions.

6.3.8 Control

Flea control measures, avoidance of walking barefoot can help prevent the infection.

6.4 Zoonotic Dermatitis

Zoonotic dermatitis occur in man primarily due to three parasites

1. *Sarcoptes scabiei* causing zoonotic scabies
2. Tick infestations due to ticks of various species
3. Other acarid parasites such as parasites of the families Cheyletiellidae, Dermanyssidae and Macronyssidae

6.4.1 Zoonotic Scabies

Phylum: Arthropoda
Class: Arachnida
Family: Sarcoptidae

6.4.1.1 Common Name/Synonyms

Acariasis, Mange, sarcoptic itch

6.4.1.2 Etiology

The disease occurs due to *Sarcoptes scabiei* which affects many mammal and marsupial species. There are many subtypes of the parasite which could be host specific. Important subtypes include *S. scabiei* var *hominis* which primarily infect humans, *S. scabiei* var. *canis*, *S. scabiei* var *bovis, S. scabiei* var. *equi*, *S. scabiei* var *suis* and *S. scabiei* var *ovis*. Humans could also become infected by many of these subtypes. In addition to these, humans can also become infected with *Notoedres cati (*notoedric mange) present in cats and *Trixacarus caviae* (guinea pig mite) affecting guinea pigs.

6.4.1.3 Epidemiology

The parasite *S. scabies* is worldwide in distribution. Human disease particularly occurs in persons belonging to poor and lower socioeconomic groups.

6.4.1.4 Transmission

Human beings become infected either through direct contact with infected animal or by indirect contact with fomites (Arlian et al. 1984).

6.4.1.5 Life Cycle

The parasite burrows tunnels in the skin and lives there. The parasite completes its life cycle on the host skin and cannot survive in the environment for long period of time. Males die after copulation. The female form tunnels in the skin and lay eggs. The larvae hatch and migrate towards the surface of the skin. Larvae further molt to nymphal and adult parasite.

6.4.1.6 Clinical Signs in Man

Human cases due to S. *scabiei* var. *canis* have been reported (Arlian et al. 1984; Weese et al. 2002). Zoonotic scabies is generally a self limiting disease in human beings. In humans, the parasite may cause intense pruritus commonly affecting arms, legs, thoracic and abdominal regions. The zoonotic infections normally do not cause burrows and produce symptoms such as dermatitis, vesicles and papules.

6.4.1.7 Clinical Signs in Animals

Clinical symptoms in animals include intense pruritus and are commonly found on legs, face and trunk (Arlian et al. 1984).

6.4.1.8 Diagnosis

The disease can be diagnosed from clinical signs and identification of the mite from the lesions.

6.4.1.9 Control

Prevention and control of the parasite in animals and using protective measures can help prevent infection in human beings.

6.4.2 Tick Infestations

Class: Arachnida
Order: Acarina

6.4.2.1 Family

Argasidae (soft ticks) and Ixodidae (hard ticks)

6.4.2.2 Etiology

Many species can cause tick infestations in man. There are approximately 12 argasid (*Argas* and *Ornithodos*), 20 ixodid (4 *Amblyomma*, 7 *Dermacentor* species, 3 *Haemaphysalis* species, 2 *Hyalomma* species and 6 *Ixodes* species) tick species which can infest man (Estrada-Pen and Jongejan 1999). The detailed reviews on tick infestations in human beings are available in the literature (Estrada-Pen and Jongejan 1999).

6.4.2.3 Epidemiology

The ticks are distributed across the globe varying with species and regions. Many human

cases have been recorded in the literature (Estrada-Pen and Jongejan 1999).

6.4.2.4 Transmission

Infected animals shed adult or larvae in the environment from where they can infect human beings.

6.4.2.5 Clinical Signs in Man

Accidental infections normally occur in human beings. Tick infestations could lead to paralyses, toxicities and allergic reactions (Estrada-Pen and Jongejan 1999; Gauci et al. 1989). Additionally, ticks can also transmit many important vector borne zoonotic diseases in animals and man (Sonenshine 1991). Paralysis due to bite of *Otobius megnini* (Peacock 1958), ear infestations (Chellappa 1973), allergic reactions (Gauci et al. 1989), irritation (Eads and Campos 1984; Uilenberg et al. 1980) and toxic reactions following the bites of *A. brumpti* and *O. moubata* (Sonenshine 1993) have been reported.

6.4.2.6 Clinical Signs in Animals

Clinical symptoms are generally similar as in man. Additionally, ticks can transmit many important vector borne zoonotic diseases in animals and man (Sonenshine 1991).

6.4.2.7 Diagnosis

The disease can be diagnosed by removal and identification of tick species involved.

6.4.2.8 Control

Improved personnel habitat and control of ticks in animals can help prevent human infections.

6.4.3 Dermatitis Due to Acarid Parasites Belonging to Families Cheyletiellidae, Dermanyssidae and Macronyssidae

Class: Arachnida
Order: Acarina

6.4.3.1 Etiology

1. *Cheyletiella parasitovorax* (rabbit parasite), *C. yasguri* (dog parasite) and *C. blakei* (cat parasite)
2. *Dermanyssus gallinae* (chicken, turkeys, pigeons, canaries, wild fowl parasite)
3. *Liponyssoides sanguineus* (small rodent parasite)
4. *Ornithonyssus bacoti* (rodents and small marsupials parasite), *O. bursa* and *O. sylvarium* (birds parasite)

6.4.3.2 Epidemiology

Most of these mites are worldwide in distribution.

6.4.3.3 Transmission

Close contact with respective animal hosts is responsible for transmission of the disease.

6.4.3.4 Life Cycle

The female mite lays eggs in burrows, nests or cracks or attached to animal hairs depending upon the host involved. These eggs hatch and release larvae which further develop to become adults.

6.4.3.5 Clinical Signs in Man

The primary sign is dermatitis and parasite may infect different body parts such as thorax, arms, thighs, etc. Bite of these mites could produce severe symptoms such as intense pruritis, allergic dermatitis, urticaria and skin irritation depending upon the species involved (Acha and Szyfres 2003).

6.4.3.6 Clinical Signs in Animals

In the animal hosts, symptoms such as dandruff, alopecia, pruritis, scaly condition, anemia and irritation could be seen varying upon the host and species involved (Acha and Szyfres 2003).

6.4.3.7 Diagnosis

Identification of causative agents using microscopic or skin scrapings can help diagnose the disease.

6.4.3.8 Control

The infected animals should be properly treated with acaricides (Acha and Szyfres 2003).

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Index

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