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Preface

As new research and clinical experience broaden our knowledge of parasites, the need for a concise, clinically oriented parasitology textbook becomes necessary. The first edition of this book provides a complete guide that covers all the essentials of veterinary parasitology for use in a busy practice setting and crowded curriculum. Its composition has taken into consideration the increasing sophistication of veterinary parasitology, and the difficulty of adequately covering many emerging topics.

For students, this book provides a solid foundation for exploring the various aspects of parasitic diseases, including clinical features, laboratory findings, differential diagnosis, and therapeutic options. All topics covered in this book are relevant to parasitologists, zoologists, biologists and veterinarians in general practice who examine many patients in the first instance. Many details of less common parasitic diseases have been omitted *deliberately*, with emphasis placed on epidemiology, choice of logical diagnostic methods, proven treatment and effective prevention strategies. For practitioners, this book provides concise yet substantial guidelines on the diagnosis and treatment of a range of important parasitic diseases to facilitate the care of the patients, and can be used as a resource for continuing veterinary education.

A logical building-block approach supplies what students and veterinarians need to know in an easy-to-use, memorable format. Many illustrations are included both for the information of the student and general practitioner, but also for use in client education. Material is presented in a progressive manner, from basic principles and concepts in parasitology to systematic description of major parasitic diseases affecting livestock and companion animals, ending with principles of parasitic diseases diagnosis and control.

For purposes of readability, references are omitted from the text, but each chapter ends with an updated list of relevant books, review articles, and selected research papers for readers who wish to pursue specific topics.

Authored by elite clinical and basic researchers at the forefront of veterinary and medical parasitology, the book presents a powerful and comprehensive synthesis of current research and clinical practices on veterinary parasitology.

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Editors' Note

Therapeutics is an ever-changing field. Readers of this book are advised to check the most current product information provided by the manufacturer of each drug to verify the recommended dose, the method and duration of administration, and adverse effects. It is the responsibility of attending practitioners to be familiar with the laws governing drugs in their practice areas. Both clients and clinicians should be cognizant of and take steps to reduce drug residues in food animals. Neither the publisher nor the editors assume any liability of any injury and/or damage to persons or property with the use of material(s) contained in this book. The mention of trade names or commercial products in this book is soley for the purpose of specific information and does not imply recommendation or endorsement by the publisher or authors.

Section I

Nature and Characteristics of Parasitism

Introductory Parasitology

1

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We tend to think of parasites as a nuisance, but they are in fact very serious disease-causing agents. Despite advances of veterinary medicine, parasitic diseases have remained a major cause of morbidity, mortality and economic losses worldwide. With the increasing burden of parasites on human and animal suffering, study of 'parasitology' has become an important and rapidly growing discipline of science. Veterinarians' awareness of parasitic diseases is undoubtedly more critical now than at any time in the history of veterinary medical practice. This chapter provides a short introduction to parasites and their unique properties.

What is a parasite?

In simple terms, parasite is an organism that is metabolically and physiologically dependent on another organism. Parasite exploits the host for development and survivability during one or more stages of its life cycle. All parasites are eukaryotic, but some are unicellular and others are multicellular. They range in size from tiny protozoa as small as 1–2 μm in diameter (= the size of many bacteria) to arthropods or tapeworms that can measure several metres in length. In some cases, two or more parasites can occur in the same host and this phenomenon is known as polyparasitism.

Types of parasites

Based on site of infection, parasites can be divided into ecto- and endoparasites. External or ectoparasites feed or live on the body surface of the host. They either suck the blood and lymph or feed upon feather, hair, skin and its secretions. Most of ectoparasites are arthropods, i.e. invertebrates with jointed legs and hard external skeletons, e.g. lice, ticks, fleas, bugs, flies and mosquitoes. Internal or endo-parasites live inside the host. Based on the site of infection, they can be divided further: for example, enteric parasites such as *Ascaris* spp. that occupy the digestive tract; haemoparasites such as *Babesia* are found in blood and blood forming organs; venereal parasites such as *Trichomonas* in cattle and *Trypanosoma equiperdum* in equines cause infections of the reproductive organs.

Based on their life cycle, parasites can be divided into facultative or obligatory parasites. Facultative parasites can live freely and complete their life cycle without the need of a host and only under certain conditions; they enter the body of the host and produce infection, e.g. *Strongyloides* worms or free-living amoebae. In contrast, obligatory parasites must enter their host to complete their life cycle, e.g. *Plasmodium* spp. Obligatory parasites can be further divided into monoxenus or heteroxenous groups according to the number of hosts needed to complete their life cycles. Monoxenus parasites (*Ascaris*, *Eimeria*) need one host to complete their life cycle while heteroxenous parasites (*Fasciola*) need two or more hosts for their development.

Parasites may exist in one of the following forms:

- Permanent parasites spend most of their life cycle in association with their hosts, e.g. *Entamoeba histolytica*, liver flukes, *Taenia* spp.
- Temporary parasites visit their hosts occasionally and at intermittent times for taking their meal, e.g. mosquitoes, bugs.
- Periodic or seasonal parasites are found on the body of their hosts during a certain time of the year, e.g. *Oestrus ovis*, mosquitoes.
- Incidental parasites are found in hosts other than their normal hosts, e.g. *Dipylidium caninum*.
- Erratic parasites are found in their normal hosts but in unusual organs or tissues in which they are not adapted to live, e.g. *Heterophyes*, *Fasciola* spp.
- Specific parasites have adapted to live in a specific host and within a certain part of the body, e.g. *Taenia saginata* in small intestine of humans while its larval stage (*Cysticecus bovis*) is found in the musculature of cattle.

Classification of parasites

Scientific nomenclature assigns each parasite two names; the genus name is the first name and is always capitalized, followed by species name that is not capitalized. Both names are underlined or italicized. Normally, after a scientific name has been mentioned once, it can be abbreviated with the initial of the genus followed by the species name. The taxonomic classification scheme places parasites within a phylum, class, order, family, genus and species (Table 1.1).

Figure 6.6 Acute fluke damage in an ovine liver. Note mottled necrotic regions and extensive haemorrhage. (Image reproduced by kind permission, R. Reichel, VLA © 2010 Crown copyright)

Figure 6.8 Classic 'pipestem fibrosis' in a chronically infected bovine liver. (Image reproduced by kind permission, N. Sargison, RDSVS.)

Figure 6.7 Adult fluke emerging from distended hyperplastic bile ducts in a chronically infected ovine liver. (Image reproduced by kind permission from the Moredun Research Institute ©)

Figure 6.9 Severe anaemia in a chronically infected ewe. (Image reproduced by kind permission, R. Reichel, VLA. © 2010 Crown copyright)

Subacute fluke disease

In this form of fascioliasis, infection is acquired over a more prolonged period, there is damage to the liver tissue and also the presence of adult flukes in the bile ducts. Infected animals tend to show rapid weight loss and poor body condition during mid and late winter. Deaths often occur later in the year than for acute fascioliasis, typically around late November-February.

Chronic fluke disease

This is the most common and widespread form of fluke disease in both sheep and cattle and can be associated with either 'summer' (after the main peak of shedding) or 'winter' infection of snails. Disease is, therefore, often seen in late winter/spring or early-summer. It is associated with a prolonged intake of low to moderate numbers of metacercariae from herbage and results in a progressive loss of body condition associated with the accumulation of adult flukes in the bile ducts of the liver. Anaemia is often severe in undernourished sheep and they may also exhibit submandibular oedema ('bottle jaw'), an accumulation of fluid caused by low blood protein (or hypoalbuminaemia) (Fig. 6.10). Deaths are uncommon in well nourished sheep but chronic fascioliasis is often exacerbated by poor nutrition.

Diagnosis

Confirmation of disease is usually through post-mortem examination of livers. In the live animal, the presence of the classic gold-coloured, operculate fluke eggs in sheep/cattle faeces is taken as evidence of a fluke infection. However, egg counting is only indicative of a patent or adult infection (immature fluke do not lay eggs). Moreover, fluke egg counting is not an accurate indication of actual fluke burden, because egg-laying adult fluke reside in the bile ducts, so eggs only appear sporadically in the faeces. Blood enzyme profiles, looking specifically for liver/bile duct damage, may be of use in chronically infected animals. Also, a number of fluke-specific ELISA tests are commercially available.

Figure 6.10 'Bottle jaw' in a chronically infected ewe. (Image reproduced by kind permission, R. Reichel, VLA © 2010 Crown copyright.)

These detect anti-fluke antibodies in blood and/or milk and indicate that an animal has been exposed to fluke infection. These are useful as survey tools; however, their diagnostic potential is questionable because anti-fluke antibodies can persist well after an animal has been successfully treated for fluke. More recently, an ELISA test capable of detecting minute traces of antigens released by the fluke into the host's faeces (i.e. coproantigens), has become available. This has the potential to discriminate between current and previous infection and, because the readout from the test is essentially quantitative, may also provide an indication of the efficacy of treatment. Acute fascioliasis should be differentiated from haemonchosis, infectious hepatitis, eperythrozoonosis, anthrax, and enterotoxaemia. Chronic fascioliasis should be differentiated from nutritional deficiencies of copper, cobalt, Johne's disease, and other internal parasitisms, including parasitic gastroenteritis (particularly haemonchosis in sheep and ostertagiasis in cattle.

Treatment

Control of liver fluke infections in livestock relies heavily on the strategic use of flukicidal drugs. There is a wide selection of such products on the market. All are capable of killing the adult fluke in the bile ducts but vary in their ability to kill the juvenile fluke in the liver (Fig. 6.11). They are usually formulated as single product flukicides but are also available as combination fluke and worm drenches. More recently, pour-on products have been developed for use in cattle. The emergence of parasite populations that are resistant to products used for their control is an inevitable consequence of repeated treatment and selection pressure. Where resistance or lack of efficacy in fluke has been reported to date, it has concerned triclabendazole, the active ingredient in a number of leading flukicides and the drug of choice to treat acute fluke outbreaks. Triclabendazole-resistant fluke have been reported in the west of Scotland and southwest Wales. The extent of triclabendazole resistance in the UK is currently unknown but probably low at this time.

Control

On some farms it may be possible to drain localized wet areas, particularly in the early spring, to reduce the snail populations. Molluscicidal treatment of snails, whilst common practice in the past, is now deemed environmentally unacceptable. Fencing off localized snail habitats may be practical in some circumstances, especially during high-risk periods. Avoiding grazing the wettest areas in autumn/winter will reduce the intake of metacercariae and lessen the incidence of disease. Avoidance or drainage of snail habitats; strategic anthelmintic dosing programmes.

Public health implications

Whilst being primarily a parasite of livestock, *F. hepatica* is also a major parasite of public health importance. As many as 17 million

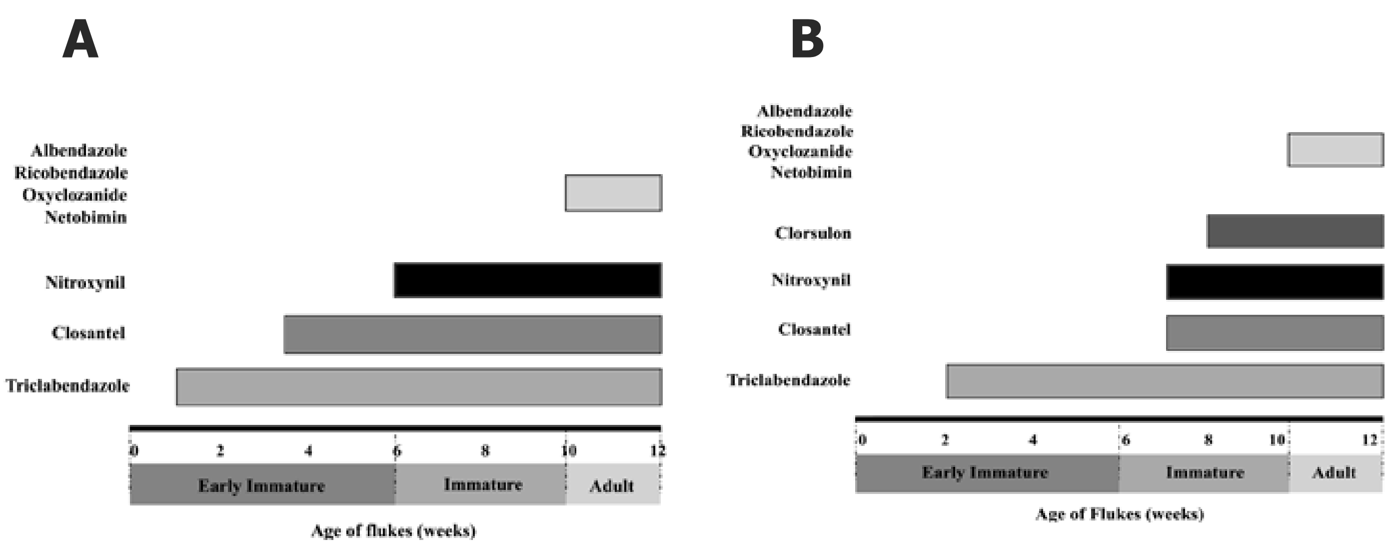

Figure 6.11 Spectrum of activity of major fasciolicides in (A) sheep and (B) cattle. (Image reproduced by kind permission from the Moredun Research Institute ©).

people are thought to be infected with fluke in over 40 different countries, mostly in the Middle East, South-East Asia and South America. Endemic fascioliasis in humans requires the combined presence of the snail intermediate host, domestic grazing livestock, appropriate climatic conditions and the suitable dietary habits of at risk human subjects. Humans become infected by ingesting metacercarial cysts on aquatic vegetation, such as watercress and water chestnuts. Pathogenesis depends on the number of cysts ingested and is similar to that reported in other animals.

Paramphistomosis

Aetiology

Rumen fluke disease (paramphistomosis) is caused by *Paramphistomum cervi*, *P.* microbothrioides and related flukes. Paramphistomes in their early stage are located in the small intestine and abomasum, from where they move to the rumen to finally establish as adults.

Epidemiology and geographical distribution

Paramphistomum spp. have a worldwide distribution and are considered to be important parasites of a number of ruminant species, particularly in tropical and subtropical areas. Geographical distribution, seasonality, and disease risk are determined by the occurrence of intermediate molluscan hosts (planorbid and bulinid snails). *Paramphistomum* infection requires the coexistence of favourable temperature and humidity and the presence of intermediate hosts. Paramphistomosis has been described in low and easily flooded lands, rice growing areas and natural grass pastures with slow running water, as well as in areas of lakes and marshlands. Snails reproduce during the warm and rainy months, when their number increases and they become easily infected with *Paramphistomum* miracidia.

Life cycle and pathogenesis

Within the intermediate host, the external phase in the life cycle of *Paramphistomum* spp. is similar to that of *F. hepatica*. Within the definitive host, when the metacercariae are ingested and reach the anterior part of the small intestine, the immature flukes are shed and remain attached to the intestinal wall, feeding on cellular detritus. Once they have developed sufficiently, they migrate towards the rumen, where the parasites will reach the adult stage, remaining there and living on ruminal fluid.

Pathological findings

The adult parasites in the rumen appear to cause relatively little pathology, unless in heavy infections. The major pathological effects are seen in the intestinal phase of the infection, where the immature fluke become attached to the ileum and duodenum, causing severe erosion of the duodenal mucosa. Large numbers of small, flesh-coloured flukes can be seen attached to the ruminal mucosa (Fig. 6.12).

Figure 6.12 Paramphistomes on the surface of a bovine rumen. (Image reproduced by kind permission H. Carty, SAC VIS.)

Clinical features

Enteritis, fetid diarrhoea, anaemia, protein loss, which generates a generalized oedema (hydrothorax, hydropericardium, ascites, lung oedema).

Diagnosis

Demonstration of immature flukes in faeces. Faecal sedimentation to detect eggs. Eggs are similar in shape to those of *F. hepatica* but slightly larger, and transparent in aspect. Paramphistomosis should be differentiated from nutritional deficiency of copper, infection with intestinal roundworms, infectious enteritides, usually accompanied by fever, Johne's disease in adult animals, but this is a much more chronic disease, and poisonings, including many weeds, inorganic arsenic and lead.

Treatment

Oxyclozanide.

Control

Avoidance or drainage of snail habitats; anthelmintic treatments to prevent contamination of pastures with eggs.

Public health implications

None known, it would appear that the adults cannot develop in non-ruminant hosts.

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Control

As mites may survive in the environment for a limited time, the treatment of the animals has to be accompanied with cleaning and disinfection of the stable and objects in contact with cattle.

Public health implications

Symptoms in humans may occur: in exceptional cases the mites may cause inflammatory reactions on the skin which will disappear, as soon as the contact with infested animals stops.

Tick infestation

Overview

The ticks found on domesticated animals are not host specific, although they do have host preferences, and their distribution is subject to environmental conditions. The species, and their host ranges, are listed in Table 9.2. Ticks are identified as being soft or hard ticks. The hard ticks are generally classified as one-, two-, or three-host ticks. Some ticks may complete the cycle in a relatively short period (*Rhipicephalus* spp.), whereas other ticks (*Dermacentor* spp.) require 2 years, with 1 year between each stage before they reattach to a host.

Ixodidiosis

Aetiology

Ticks may act as causative agent of disease or may be carriers and vectors of pathogens like viruses, bacteria, protozoa and even helminths. In Europe, cattle are mostly affected by ticks of the widespread genera *Ixodes* (Fig. 9.6), *Haemaphysalis*, *Dermacentor* and *Rhipicephalus* (Fig. 9.7). Many tick species can affect livestock. *Ixodes ricinus* is the most frequent representative and will be discussed in more detail.

Figure 9.6 Dorsal view of a female *Ixodes ricinus* tick.

Figure 9.7 Ventral view of an engorged *Rhipicephalus* sp. female tick.

Table 9.2 Common tick species of livestock

Cattle	Sheep	Pigs
Ornithodoros spp.[1]	*Ornithodoros* spp.[1]	*Ornithodoros* spp.[1]
Otobius spp.[1]	*Otobius* spp.[1]	
Rhipicephalus spp.[2]	*Rhipicephalus* spp.[2]	*Rhipicephalus* spp.[2]
Ixodes spp.[2]	*Ixodes* spp.[2]	*Ixodes* spp.[2]
Hyalomma spp.[2]	*Hyalomma* spp.[2]	*Hyalomma* spp.[2]
Amblyomma spp.[2]	*Amblyomma* spp.[2]	*Dermacentor* spp.[2]
Dermacentor spp.[2]	*Dermacentor* spp.[2]	
Haemaphysalis spp.[2]	*Haemaphysalis* spp.[2]	
Boophilus spp.[3]	*Boophilus* spp.[3]	

[1]Soft tick belong to the family Argasidae, the rest are hard ticks belonging to the family Ixodidae.

[2]Three-host life cycle.

[3]One-host life cycle. In recent literature *Boophilus* has been re-named to *Rhipicephalus*.

Epidemiology and geographical distribution

Ixodes ricinus can be found in shady habitats with sufficient humidity. They prefer coppice, bush and underbrush. Larvae normally climb up to 30 cm off the ground while nymphs and adults may reach up to 1 m. *I. ricinus* ideally develops at temperatures between 17–20°C and a relative humidity of 80–95%. It is therefore mostly active in the mild climate of spring and autumn. Most relevant pathological findings in Europe are caused by tick-transmitted agents: babesiosis, caused by *B. divergens*, and anaplasmosis (tick-borne fever), caused by *Anaplasma phagocytophilum*. Other diseases can occur (see Chapter 10 for more details).

Life cycle and pathogenesis

The adult female drops to the ground after the blood meal, produces one to several thousand eggs and dies. The tick development includes one larval (three pairs of legs) and one nymph stage (four pairs of legs) before the adult stage is reached. Most European ticks change the host for each stage, which extends the life cycle to several years, due to host finding and climatic conditions. While larvae mainly infest small mammals, birds and reptiles, the later stages (nymphs and adults) prefer larger mammals, including humans. The full life cycle in middle Europe takes 2 years but may be considerably extended under unfavourable climatic conditions.

Pathological findings

Local cutaneous reactions vary in severity with the tick and its secretions, and host resistance. Gross lesions consist of focal erosions, erythema, and crusted ulcers with alopecia and nodules. Microscopic lesions include epidermal and dermal necrosis, and perivascular to diffuse inflammation at the margins of the necrotic area. The exudate is composed of eosinophils, macrophages and lymphocytes. Cutaneous basophil hypersensitivity likely contributes to the reactions induced by tick bites.

Clinical features

Ticks localize mainly on head and ears as well as on the perineum and the inner part of fore and hind legs. Attachment may result in thickening of skin (hyperkeratosis) and local skin inflammation with ulcers. In case of high infestation anaemia, weight loss and reduced milk production may be observed. *Ixodes ricinus* is transmitter of infective agents such as *Trypanosoma*, *Theileria*, *Babesia*, *Dipetalonema* larvae, the tick-borne encephalitis virus (TBE) and *Borrelia burgdorferi*, which causes borreliosis (Lyme disease) in humans. In most cases tick infestation in Europe does hardly cause clinical signs and is often not noted. Problems may occur in the case of local inflammation and by secondary bacterial infection of the penetration site. Severe diseases may be due to tick-transmitted pathogens.

Diagnosis

Ticks (Fig. 9.8) can be easily identified on animals, mainly on the favoured spots like udder, inguinal region, head, ears and cervix. For species differentiation the ticks can be sent to specialized labs. Ticks can be easily identified on the animals, especially engorged adult females. Mating often occurs on the host while the female is sucking blood. The male has a scutum that covers his whole back. *Ixodes ricinus* can be easily identified by the inverted U-shaped perianal groove.

Figure 9.8 Ticks can be easily identified on cattle. High infection loads may cause skin irritation, pruritus and inflammation.

Treatment

Systematic treatment of cattle against ticks is rare in Europe. A decision on required treatments has to be based on the severity of clinical signs and production losses and on the expenses and risks (costs, residues etc.) of treatment. Acaricides can be applied by pour on, spray on, washing or injection. Registered active classes are pyrethroids and macrocyclic lactones. The latter are also used for anthelmintic treatment. It is described that cattle dewormed with macrocyclic lactones have less tick infestation when put on pasture.

Control

Control of ticks should target the infested animals and the animal's surrounding environment. The former is more challenging because most ticks of veterinary significance use more than one host other than the infested animal to complete their life cycle. Reducing exposure to ticks by being informed about endemic species in the local area and avoiding periods when most ticks are active may reduce the animal and the animal owner's risk of exposure.

Public health implications

Ticks can infect humans and transmit pathogens. Of most relevance are *Borrelia burgdorferi* (Lyme disease), Flaviviridae (Tick Borne Encephalitis) and *Ehrlichia*. *Babesia divergens* is worth mentioning as it may cause clinical signs in splenectomized and immunosuppressed patients, which may be misdiagnosed as malaria. Infestation of domestic animals with ticks should alert their human owners that they too are at risk of tick exposure and thus potential exposure to tick-borne diseases (see Chapter 10). Humans in contact with tick-infested animals should inspect themselves for similar infestation and remove ticks as soon as possible before engorgement occurs and before the transmission of infectious agents to humans occur.

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Glossary

Abomasum: The fourth and final compartment of the ruminant's stomach.

Acanthella: Acanthocephalan larva following the acanthor and prior to the cystacanth.

Acanthor: Acanthocephalan larva with bladelike hooks and develops inside the egg capsules.

Acetabulum: Ventral muscular sucker or holdfast of digenetic trematodes; a sucker on a tapeworm scolex.

Acoelomate: A condition in which a body cavity is lacking, as in the members of the phylum Platyhelminthes, where the organs lie embedded in parenchyma.

Ala (-ae): Wing-like projection such as the cuticular expansions in certain nematodes.

Alveolus (-i): The air sac in the lung where gaseous exchange occurs.

Ametabolous: A type of metamorphosis in insects in which there is no external change as they proceed through a series of molts to the adult; *ametabola* refers to the taxonomic group.

Amphid: Anterior sensory structures of nematodes.

Anaphylaxis: A strong hypersensitivity reaction in which the individual may collapse, stop breathing, and die.

Anapolysis: The process in which terminal, gravid proglottids are not shed in certain tapeworms.

Anisogamete: Morphologically different male and female gametes.

Anterior station: Protozoan development in the anterior part of an insect vector, for example, Salivaria of the genus *Trypanosoma*; transmission takes place by biting.

Anthroponoses: Human diseases that are transmissible to animals.

Antibody: Serum protein (immunoglobulin) synthesized by lymphoid cells in response to an antigenic stimulus.

Antigen: Any substance that can stimulate an immune response.

Antigenic mimicry: Acquisition of or production of host antigens by a parasite so that it is not recognized as non-self, as in *Schistosoma*.

Apical complex: Organelles characteristic of members of the phylum Apicomplexa. It encompasses polar rings, subpellicular microtubules, conoid, rhoptries, and micronemes.

Apolysis: The process in which terminal, gravid proglottids are detached and shed by certain tapeworms.

Autoinfection: A process in which the progeny of a parasite reinfect the host without passing out of it, for example, *Taenia solium*.

Axostyle: A longitudinal rod-like or tube-like structure in members of the protozoan order Trichomonadida; probably serves as a cytoskeleton.

Biramous: Divided into two branches; typical of the terminal segments of the legs of Crustacea.

Bladder worm: Infective stage of *Taenia* tapeworm (Cysticercus). The name refers to the fluid filled bladder which surrounds the larval scolex (*Taenia*) or scolices (*Coenurus*). Bladder worm is also a common name of *Capillaria plica*, found in the dog urinary bladder.

Bothrium (-ia): Shallow, sucking groove on the scolex of tapeworms of the order Psuedophyllidea.

Bots: Larvae of several fly species, particularly *Gastrophilus* (horse bot), *Oestrus* (sheep bot), and *Dermatobia* and *Hypoderma* (affect cattle and other species).

Bottle jaw: Fluid accumulation under the lower jaw (submandibular oedema).

Bradyzoite: Slow-growing zoite or meront of the pseudocyst of *Toxoplasma* and related cyst-forming coccidian protozoa.

Buccal capsule: Mouth cavity of a nematode.

Bursa (copulatory bursa): A cuticular copulatory structure at the posterior end of males of the order Strongylida. It is useful in nematode taxonomy and species identification.

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