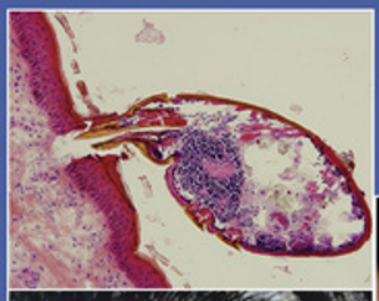
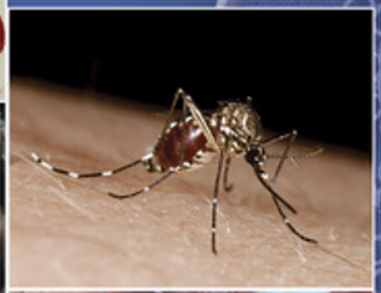
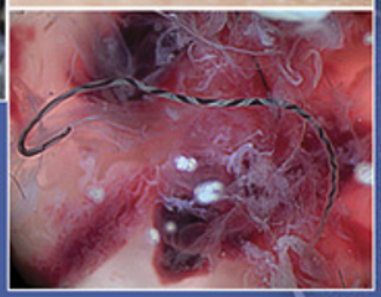
Principles of Veterinary Parasitology







Dennis Jacobs
Mark Fox
Lynda Gibbons
Carlos Hermosilla

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About the Authors

Dennis Jacobs qualified from the University of Glasgow Veterinary School in 1964 and studied for his PhD in Glasgow and Copenhagen. He is Emeritus Professor of Veterinary Parasitology at The Royal Veterinary College (University of London) and a Fellow of the Royal College of Veterinary Surgeons, the Royal College of Pathologists and the Higher Education Academy. He has been Visiting Professor at the University of the West Indies, Trinidad; St George's University Medical School, Grenada; Ross University School of Veterinary Medicine, St Kitts; the South China Agricultural University, Guangzhou, Peoples Republic of China; and at the University of Melbourne, Australia. He was Vice-president of the World Association for the Advancement of Veterinary Parasitology from 2003 to 2007 and Secretary of the European Veterinary Parasitology College from its foundation in 2003 to 2009. He has published one book and co-edited another, written several book chapters and more than 150 peer-reviewed scientific papers and reviews.

Mark Fox graduated from The Royal Veterinary College (University of London) in 1977 and, after a period in small animal practice, studied for a PhD at the same college where he is currently Professor of Veterinary Parasitology. He is a Member of the Royal College of Veterinary Surgeons, Diplomat and Board Member of the European Veterinary Parasitology College, Fellow of the Higher Education Academy and a past Honorary Secretary of the Association for Veterinary Teaching and Research Work and British Veterinary Association Council member. He established Masters degree courses in Wild Animal Biology and Wild Animal Health with the Institute of Zoology (London Zoo) and was jointly-awarded the BVA William Hunting medal in 2005 for research on coccidial infection in wild birds of prey. His current research interests focus on the epidemiology of parasite infections in both domestic and wild animals.

Lynda Gibbons graduated with a BSc Honours degree from the University of Leicester in 1969 and studied for a PhD at the London School of Hygiene and Tropical Medicine, University of London. She is a Chartered Biologist and Fellow of the Royal Society of Biology and was a council member of the Systematics Association from 1993 to 1995. She was head of the Animal Helminthology Biosystematics Unit of the CAB International Institute of Parasitology from 1992 to 1997 and an attached senior lecturer at The Royal Veterinary College until 2010. She was awarded the Elsdon-Dew medal in 1993 by the Parasitological Society of Southern Africa and the Betts prize by The Royal Veterinary College in 2006. She has run training programmes and courses for overseas students and developed a joint RVC/FAO on-line programme on faecal diagnosis of helminth infection. She has published two books, a book chapter and 78 scientific papers.

Carlos Hermosilla entered the Faculty of Veterinary Medicine at the University Austral of Chile in 1984 and achieved the degree of DVM in 1989. He obtained his doctoral degree (Dr med. vet.) in 1998 at the Justus Liebig University, Giessen, Germany where he is currently Professor of Veterinary Parasitology. From January 2008 to March 2011 he held the position of Senior Lecturer for Veterinary Parasitology at The Royal Veterinary College, London, United Kingdom and since 2009 has held a position as Visiting Professor of the University Austral of Chile. In 2009 he finished his habilitation thesis (Dr habil.) in Parasitology. In the last 14 years he has mainly been involved in the field of coccidian parasites (*Eimeria bovis*, *E. ninakohlyakimovae*, *E. arloingi*, *Neospora caninum*, *Toxoplasma gondii*, *Besnoitia besnoiti*). He has so far published more than 80 papers in international scientific journals.

Foreword

Parasites are not only scientifically fascinating but when they infect humans or animals they present sophisticated and highly evolved targets that are difficult to control even in the technically advanced world in which we live. Moreover parasitic diseases of domestic animals (in contrast to those of humans) are a real and present danger to the health and welfare of animals throughout the globe, in rich and in poor countries, in temperate as well as in tropical climates. The nature of parasitic diseases of livestock, whilst occasionally acute and lethal, is frequently chronic and endemic leading to the continual detriment of welfare and productivity. This is critical given the rapidly expanding global population and the equally rapidly expanding demand for meat and dairy products. Of current importance, the effects of parasitism on morbidity, mortality and productivity exacerbate the greenhouse gas emissions from ruminants, and the successful control of parasitism mitigates such emissions. Apart from affecting the production of food, some parasites of animals infect humans and are of considerable public health importance. So a new, up to date textbook on this subject is to be welcomed.

This book fills an important niche. It is unashamedly written for students, in its broadest sense, of the subject. These are, of course, mainly those studying to become veterinary clinicians, veterinary nurses or following other veterinary-related courses. But it is also ideal for other types of "learner" such as the qualified professional pursuing continuing education. The authors are highly experienced and knowledgeable university teachers, and it shows. The approach is clinically relevant and highly practical. The text anticipates the misunderstandings and errors that learners can easily make. For example, it makes clear that humans get infected with hydatid disease only from the ingestion of eggs (excreted by dogs) and not from accidental ingestion of hydatid cysts in meat! There is an admirable use of apt analogies to clarify concepts and frequent use of text boxes to expand, explain or expound particular issues or historical examples – for example the history of sheep scab in the UK. As experienced teachers ourselves, we recognise the care with which terms sometimes taken for granted are explained, e.g. formulation in the context of drugs. And there is even a pronunciation guide on the associated website.

The lay out, we suspect, may owe some debt to Angus Dunn's wonderful and out of print book *Veterinary Helminthology* (Professor Jacobs was a PhD student with Dunn when he was writing his classic). After an initial chapter describing basic concepts, the rest is divided into two broad sections. The first part deals with the subject matter taxonomically, although always with clinical relevance in mind; the second then approaches the subject from the perspective of the animal host species or group, and the organ(s) affected and associated syndromes, which is how parasitic diseases are presented to the clinician. In the appropriate places there are sections specifically dealing with ectoparasiticides, anthelmintics and antiprotozoal drugs. The discussions of treatments and control are suitably detailed for the target audience and their rationales are thoroughly explained; it is a lot easier to remember when one does what, if one understands the underlying reasons!

This book is a valuable addition to the literature on veterinary parasitology. Although three of the authors are based in the UK, all the authors have extensive international experience and the book reflects this with comprehensive cover of all the major parasitic diseases of domestic animals worldwide. It will be of use to students of the subject throughout the world.

Professor the Lord Trees and
Professor Diana J.L. Williams

Preface

Between us, the authors of this textbook have accumulated a century's worth of teaching experience. This has culminated in a set of undergraduate course-notes crafted to match the learning requirements of our British and American students. Always sensitive to feedback, we have, over the years, progressively honed content and eliminated ambiguity, thereby providing a solid foundation for the present more ambitious enterprise, intended as a 'student-friendly' introduction to Veterinary Parasitology.

Our teaching has been enhanced by ideas avidly gathered from many national and international sources, including visits to other institutions and attendances at meetings such as those organized by the World Association for the Advancement of Veterinary Parasitology (Eckert, 2013). We are privileged to have had the opportunity to gain inspiration from so many gifted colleagues and we thank them for sharing their knowledge and expertise.

A number of friends have contributed more directly to the evolution of our course-notes. In particular, we wish to thank Dr Manice Stallbaumer, Professor Mike Taylor, Professor Phillip Duffus and Dr Rachel Lawrence for their various inputs. Professor Taylor's authoritative book *Veterinary Parasitology, Third Edition* (Taylor, Coop and Wall, 2007) has been an invaluable reference work during the preparation of our text.

An ever-expanding knowledge-base has lead progressive universities to reappraise veterinary education. A comprehensive knowledge of every component discipline is no longer a feasible aspiration for the student nor is it a realistic expectation for examination boards. Modern approaches encourage students to become problem-solvers by instilling an understanding of basic principles rather than 'drowning' their intellect in a mass of detail. Factual information is of course important to support 'professional day 1 competencies' but it should be carefully selected and restricted to that actually required to meet defined educational and professional objectives.

Another trend in veterinary education is the adoption of 'integrated' curricula which aim to unify component strands of expertise needed for clinical practice. The downside of this otherwise commendable approach is that it tends to fragment the presentation of discipline-based subjects, dispersing information throughout 'systems' modules based on alimentary, respiratory and other body functions, or between 'species' modules focussed on equine, ruminant and small animal medicine. This makes underlying concepts and inter-relationships in Veterinary Parasitology and other disciplines more difficult to appreciate, to the detriment of understanding and clinical application.

The aims of this textbook are therefore to provide a guide to learning that:

- **i)** is straightforward, easy to comprehend and informative without being encyclopaedic;
- **ii)** is useful for students whether engaged in traditional or integrated modular educational systems;
- **iii)** provides knowledge relevant for the immediate needs of the veterinary student uncluttered by unnecessarily detailed or advanced information;
- **iv)** supports learning and enhances understanding by clearly illustrating conceptual relationships between parasitic organisms, their biology and the diseases they cause.

The scope of the original course-notes has been broadened to encompass a wider geographical coverage. In this regard, teaching experience gained by the authors in Europe, South America, the Caribbean, South-East Asia and Australasia has proved a valuable asset.

Finally, attention is drawn to a quotation from a poem by the Scottish bard, Robert Burns, that some believe to be a satire on 18th-century medicine (Nicolson, 2010): 'Some books are lies frae end to end'. Or, in modern parlance: 'Textbooks … can vary in their quality and will almost always include some form of bias, reflecting the authors' experience, opinion and interpretation of the evidence' (Dean, 2013). We have endeavoured to keep our text on a sound footing but, in accord with the 21st-century emphasis on evidence-based medicine, the reader is encouraged to use this book as a springboard for independent enquiry, to delve deeper and to challenge our assertions.

Dennis Jacobs, Mark Fox,
Lynda Gibbons, Carlos Hermosilla
The Royal Veterinary College (University of London)
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List of abbreviations

AAD	Amino-acetonitrile derivative
ACh	Acetylcholine
AIDS	Acquired immunodeficiency syndrome
BZD	Benzimidazole
Ca	Calcium
CFT	Complement fixation test
CNS	Central nervous system
CO_2	Carbon dioxide
DNA	Deoxyribonucleic acid
e.p.g.	Eggs per gram of faeces
EL_3	Early parasitic third stage larva
ELISA	Enzyme-linked immunosorbent assay
EPM	Protozoal myeloencephalitis
ERP	Egg reappearance period
FAD	Flea allergic dermatitis
Fe	Iron
HIV	Human immunodeficiency virus
HWD	Canine heartworm disease
IDI	Insect Development Inhibitor
IFAT	Indirect fluorescence antibody test
IgM, IgG, IgE, IgA, etc.	Different classes of immunoglobulin
IGR	Insect Growth Regulator
IL-2, IFN-γ, etc.	Names given to different cytokines
KOH	Potassium hydroxide
L_1, L_2 etc	First stage larva, second stage etc.
Mf	Microfilariae
MHC	Major histocompatability complex
ML	Macrocyclic lactone
Na	Sodium
nAChR	Nicotinic acetylcholine receptor binding site
NK	Natural killer cell
NLM	Neural larva migrans
NO	Nitric oxide
o.p.g.	Oocysts per gram of faeces
OLM	Ocular larva migrans
OP	Organophosphate
PABA	Para-amino benzoic acid
PCR	Polymerase chain reaction
PGE	Parasitic gastroenteritis
PP	Period of persistency
PPP	Prepatent period
RBC	Red blood cell
RNA	Ribonucleic acid
SEM	Scanning electron micrograph
SP	Synthetic pyrethroid
sp., spp.	species (singular and plural)
Th1, Th2	Two categories of helper T-lymphocytes
UV	Ultraviolet
VLM	Visceral larva migrans
$ZnSO_4$	Zinc sulphate
3D	Three-dimensional

About the companion website

This book is accompanied by a companion website:

www.wiley.com/go/jacobs/principles-veterinary-parasitology

The website includes:

- Glossary
- Parasites listed by host and body system
- Pronunciation guide
- Parasite recognition: fleas, flies, worms and worm eggs
- Revision questions and answers
- Further reading list: books, articles and websites
- Powerpoint files of all diagrams for downloading

CHAPTER 1
Veterinary Parasitology: basic concepts

1.1 Introduction

The primary aim of this book is to provide a 'student-friendly' introduction to Veterinary Parasitology for those aspiring to become veterinarians, veterinary nurses or veterinary scientists. It also offers an accessible resource for those already qualified and wishing to refresh or expand their general knowledge of the topic. Others engaged in the many and varied facets of animal health and veterinary public health will also find information relevant to their interests.

This first chapter explores the nature of parasitism while Chapters 2–7 examine clinically relevant relationships and interactions between the parasite, its host and the environment. Finally, Chapters 8 and 9 recognise that, in the real world, veterinarians and animal health workers are not usually presented with a parasite as such, but with a problem concerning some bodily dysfunction affecting a flock, herd or individual.

To fulfil the aims of this book, the emphasis throughout has a clinical bias. Academic information is restricted to that necessary to gain a broad understanding of the pathogenesis, epidemiology, diagnosis and control of the commonest parasitic diseases. Key words are defined in the text or, if printed in a blue typeface, explained in a nearby 'Help box'. A glossary is provided on the website that accompanies this book.

Wherever possible, concepts are described in straightforward language, and unnecessary jargon or detail is avoided. Further aids to learning are provided in 'Help boxes', while

Principles of Veterinary Parasitology, First Edition. By Dennis Jacobs, Mark Fox, Lynda Gibbons and Carlos Hermosilla.

Companion website: www.wiley.com/go/jacobs/principles-veterinary-parasitology

'Extra Information Boxes' offer additional insights for more advanced readers. Cross-references within the book are given in the format (see Section 9.2.3), (see Table 9.10) etc. These are to assist readers who may wish to follow up on particular points, but they can otherwise be ignored.

The emphasis with regard to parasite identification and the diagnosis of associated disease is on 'how it's done' rather than 'how to do it'. Latin names and taxonomic relationships are introduced only where these provide a useful foundation for comprehension, learning or further reading. The number of parasites that might be encountered in veterinary practice is so great that to mention them all would transform this 'guide to learning' into an encyclopaedia, which would defeat the purpose of the book. Selected examples are therefore given to provide an understanding of underlying principles and to illustrate the range and diversity that exists within the wonderful world of Veterinary Parasitology.

Help box 1.1

Definition of some key technical terms

Aetiology/ aetiological agent: the cause or origin of a disease.
Biotic potential: an expression of the rate at which a parasitic species can multiply. It depends on the number of offspring produced ('fecundity') and the number of generations each year ('generation time').
Endemic: a term used to describe a population or area within which a pathogen is established, replicating and being transmitted between hosts.
Epidemiology: the science that describes and explains patterns of disease in the host population (i.e. the distribution and determinants of disease).
Eukaryote: an organism with a cytoskeleton and complex subcellular structures enclosed within membranes (including a nucleus containing chromosomes). Examples: protozoa and metazoa.
Incidence: the number of new cases of infection per unit time.
Pathogen/pathogenicity/pathogenesis: an organism that causes disease / the severity of the damage caused / the mechanism of the disease process.
Prevalence: proportion of host population infected at a point in time.
Prokaryote: an organism without a nucleus or other membrane-bound subcellular structures; DNA in circular plasmid. Example: bacteria.
Species: the basic unit of biodiversity. Although everyone knows what a species is, there is no exact definition as boundaries are often blurred. Two commonly cited definitions are: 'a group of organisms capable of interbreeding and producing fertile off-spring' and 'a separately evolving lineage that forms a single gene-pool'.
Taxonomic: relating to the laws and science of describing, identifying, naming and classifying organisms.

1.1.1 What is Veterinary Parasitology?

Animal disease can have noninfectious or infectious origins. Noninfectious diseases result from genetic defect, physiological abnormality, structural dysfunction or external factors such as injury, radiation or poisoning. In contrast, infectious diseases are associated with invasive self-replicating agents that have evolved to occupy an animal body as their ecological niche in just the same way as a koala bear has become adapted for life in a particular species of *Eucalyptus* tree.

By convention, the study of infectious agents is divided into Microbiology, which embraces noncellular and prokaryotic organisms, like viruses and bacteria, and Parasitology, which is concerned with eukaryotic life-forms. Fungi are an anomaly in this scheme as, although they are eukaryotes, they are traditionally taught as part of Microbiology in most veterinary schools and so have been omitted from this book.

Veterinary Parasitology is a composite of three distinct disciplines, each with its own set of host–parasite interactions, clinical considerations and vocabulary. The three topics that make up the bulk of Veterinary Parasitology are:

a – Veterinary entomology: the study of parasitic arthropods, including insects, ticks and mites (see Chapters 2 and 3);

b – Veterinary protozoology: a subject that embraces the wide range of single-celled eukaryotic organisms that comprise the parasitic protozoa (see Chapter 4);

c – Veterinary helminthology: which covers three main groups of parasitic worms – trematodes (flukes), cestodes (tapeworms) and nematodes (roundworms), as well as some minor groups such as the thorny-headed worms (see Chapters 5–7).

1.2 Parasitism and parasites

1.2.1 Parasitism

Parasitism is part of a spectrum of intimate zoological relationships between unrelated organisms which includes:

a – Commensalism: two species living together for the benefit of one or both, but without detriment to either

party, and without any metabolic dependence (e.g. cattle egrets and cattle).

b – Symbiosis: two species living together, each dependent on the other for their mutual well-being and survival (e.g. cellulose-digesting organisms in the caecum of a horse).

c – Parasitism: two species living together, where one of the pair (the parasite) is living at the expense of the other (the host).

d – Parasitoidism: two species living together as in parasitism except that the host invariably dies (or is at least rendered incapable of functioning) once the parasitoid has extracted the sustenance it needs for that stage of its development. Familiar examples include parasitoidal wasps used in horticulture that lay their eggs on or in other insects to provide a food-source for their larvae.

Parasitism implies nutritional dependence on the host for at least part of the life-cycle. It also involves a high degree of specialised adaptation as the animal body is not a passive ecological niche (like a rotten tree-trunk harbouring beetles, for example) but is responsive and hostile to foreign invasion. A parasite must be able to overcome host defences and evade immunological attack. Mechanisms must also be in place to ensure transfer of infection, both geographically from host to host ('horizontal transmission') and temporally from generation to generation ('vertical transmission'). This often entails an intricate integration of the life-cycle of the parasite with that of its host.

Parasites can themselves be victims or beneficiaries of invading organisms. Fleas, for example, are exploited by larval stages of both tapeworms and nematodes, while the canine heartworm, *Dirofilaria*, is metabolically dependent on a symbiotic bacterium, *Wohlbachia*.

1.2.2 Classification

The unwise student could approach every parasitic infection as a separate entity, but this would be an enormous task and a very inefficient approach to learning. It would soon become apparent that similarities exist between some diseases and this would prompt the question: 'what are the common factors?' So, classification is an inherent attribute of human curiosity. It has been noted already that Veterinary Parasitology embraces at least three types of arthropod, several types of protozoa and at least three types of parasitic worm, and so the value of classifying aetiological agents of disease is already becoming apparent.

Taxonomy is a powerful and essential component of biological understanding, although, from a clinician's viewpoint, it is a tool rather than an end in itself. Knowledge of the relationship between parasites often allows similarities in life-cycle, epidemiology, pathogenesis and drug susceptibility to be predicted. Thus, if used intelligently, classification provides a valuable framework for learning and reduces considerably the amount that has to be committed to memory. The classification in this book is kept at the simplest level compatible with this objective.

Help box 1.2

Classification

The animal kingdom is divided into some 35 phyla (singular 'phylum'), which in turn are subdivided successively into Class, Order, Family, Genus and Species, with a species being the basic replicating entity. Subclass, Suborder and Superfamily groupings are also useful in some contexts. Relationships are deduced from morphological, biological and, more recently, molecular evidence and so taxonomic charts (and, confusingly, parasite names on occasion) have to be revised as knowledge accumulates. This can lead to discrepancies between different information sources.

Nomenclature

The identity of every organism is defined by using a combination of its genus and species names. Thus, the protozoan parasite that causes redwater fever in northern European cattle is *Babesia divergens*, while the related species *Babesia bovis* and *Babesia bigemina* cause similar diseases in warmer regions. By international agreement, the ending -osis is placed on a parasite name to indicate the disease caused by that parasite, e.g. babesiosis. By tradition, the ending -iasis is sometimes preferred in human medicine and may occasionally be found in veterinary publications.

It is sometimes useful in Veterinary Parasitology to refer to the common characteristics of a larger grouping of parasites such as a family, which always has a technical name ending in -idae (e.g. the Ixodidae, which is anglicised as 'ixodid ticks'), or even a superfamily with the suffix -oidea (e.g. the Trichostrongyloidea, which becomes 'trichostrongyloid worms').

Help box 1.3

Writing parasite names

When writing parasite names, the genus name always starts with a capital letter while the species name is lower case throughout. The convention in parasitology as in all biological disciplines is to italicise these. The first time a parasite is mentioned in a text, the full name is used, but thereafter the genus name is abbreviated, e.g. *Babesia divergens* becomes *B. divergens*. The word 'species' can be abbreviated to sp. (singular) or spp. (plural), so '*Babesia* sp.' means an unnamed *Babesia* species, while '*Babesia* spp.' refers to more than one species in that genus.

Why use Latin names?

Latin names are universal, whatever language is being used for communication. Local names can be parochial (for example, babesiosis is known as 'Red-water fever' in the UK but as 'Texas fever' in the USA) or ambiguous ('sand-fly' for example refers to phlebotomine sand-flies in most countries, but is the colloquial term for biting midges in some others).

Pronouncing Latin names

There is no right or wrong way to pronounce a Latin scientific name. Some are tongue-twisters and with these it helps to know how the word can be broken down into syllables. Some of the most troublesome Latin names are listed in the Pronunciation Guide on the website that accompanies this book.

1.2.3 Host–parasite relationships

Parasites and their hosts have evolved together over many millions of years. Every host is vulnerable to infection by several, if not many, parasitic species. Thus, there are many more parasitic species on this planet than host species! It is not surprising, therefore, that a great diversity of host–parasite relationships exists. These are often amazingly intricate and are part of the fascination of parasitology, as will become apparent when the life-cycles of individual parasites are described in later chapters.

Parasites

Parasites can be broadly categorised according to their location on or in the body of their host:

a – Ectoparasites: live or feed on the surface of the host, or embed themselves into superficial or adjacent underlying tissues. Ectoparasites engage in host–parasite associations ranging from flies that land fleetingly to feed on secretions from the eyes, nose or other orifices to mites that spend nearly their whole lives in skin tunnels.

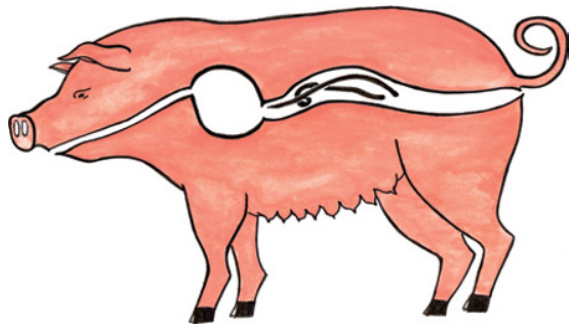

Figure 1.1 Gastrointestinal parasites such as the worms depicted here in black are technically 'outside' of any body tissue.

b – Endoparasites: live within the body of the host. Parasites may be found in every body tissue except, perhaps, bone and keratin. Those free in the lumen of the gastrointestinal tract are, technically speaking, lying outside of any host tissue (see Figure 1.1), but they are nevertheless included in this category.

A fundamental distinction that influences both the pathogenesis of infection and options for control is the relationship of the parasite to the tissue it inhabits:

a – Extracellular parasites: these live on or within host tissues but do not penetrate into host cells. Examples include almost all metazoan and also many protozoan parasites.

b – Intracellular parasites: these live inside a host cell modifying its genomic expression to cater for their needs, e.g. many protozoan parasites and at least one nematode genus (*Trichinella*).

Parasites can also be differentiated on the basis of their reproductive behaviour in the final host (see Figure 1.2). This distinction is useful as it points towards fundamental biological differences that influence pathogenesis, epidemiology, control and treatment:

a – Microparasites: these multiply within their host. Consequently, each organism that enters the body is capable of initiating a massive infection if not checked by host defences or by chemotherapy. This category includes the parasitic protozoa (as well as microorganisms such as bacteria).

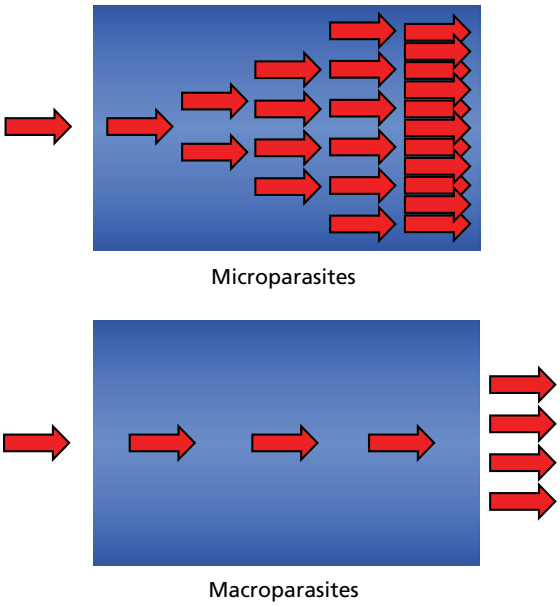

Figure 1.2 Microparasites (above) multiply their numbers within the host; whereas the number of mature macroparasites (below) never exceeds the number that invaded the host (with a few exceptions).

b – Macroparasites: these do not generally increase in number while they are on or within the final host. They may produce eggs or larvae but these are dispersed into the environment. Thus, the number of mature parasites on or in the final host never exceeds the number of infective units that originally invaded the body. This category includes arthropods and helminths, although there are a few species that break the general rule by multiplying on or in the host (for example: lice, mites and a few nematodes, e.g. some *Strongyloides* species).

c – Microcarnivores: these visit the host transiently to feed but leave again before undergoing any development or producing offspring. Many parasitic arthropods, such as mosquitoes, can be included in this designation.

With such a diverse spectrum of host–parasite associations, there are inevitably some organisms that do not fit conveniently into these broad groupings.

Hosts

Some parasites require just one host to complete their developmental cycle and produce progeny. Others utilise two or more animals. Hosts can be exploited in different ways and the following terminology is used to differentiate between these:

a – Final (or definitive) host: a term used to identify the host in which sexual reproduction of the parasite takes place.

b – Intermediate host: this is a host in which only immature stages grow and develop. Asexual replication may occur (but not sexual reproduction).

c – Transport and paratenic hosts: no parasitic development of any kind takes place in these and they are not a necessary part of the life-cycle. The parasite takes advantage of another animal by using it as a vehicle to increase its chances of reaching its next essential host. The word 'paratenic' implies an intimate relationship in which the parasite becomes embedded within the tissues of its host. The corresponding association with a transport host is more casual and often passive in nature. The two terms are sometimes used interchangeably with less precision.

d – Reservoir host: as the name suggests, this depicts a host population that acts as a source of infection for other animals.

e – Vector: this is a vague term for an insect, tick or other creature that carries (transmits) a disease-causing organism from one host to another.

Life-cycles are described as being:

a – Indirect (or heteroxenous): if an intermediate host is involved; or

b – Direct (or homoxenous): if there is no intermediate host.

Zoonoses

Parasitic zoonoses are diseases of mankind associated with animal parasites (see Section 9.3). They can be classified according to the various biological pathways that lead to human infection (see Figure 1.3):

a – Direct zoonoses: direct transfer from animal to human, e.g. *Cheyletiella* mites from an infested lap-dog.

b – Cyclozoonoses: where humans infect animals and vice versa in strict rotation, e.g. the beef tapeworm.

c – Metazoonoses: these involve a vector as intermediary, e.g. phlebotomine sandflies carrying *Leishmania* from dogs to humans.

d – Saprozoonoses: indirect transfer via the environment, e.g. children playing on ground contaminated with *Toxocara* eggs from a dog or fox.

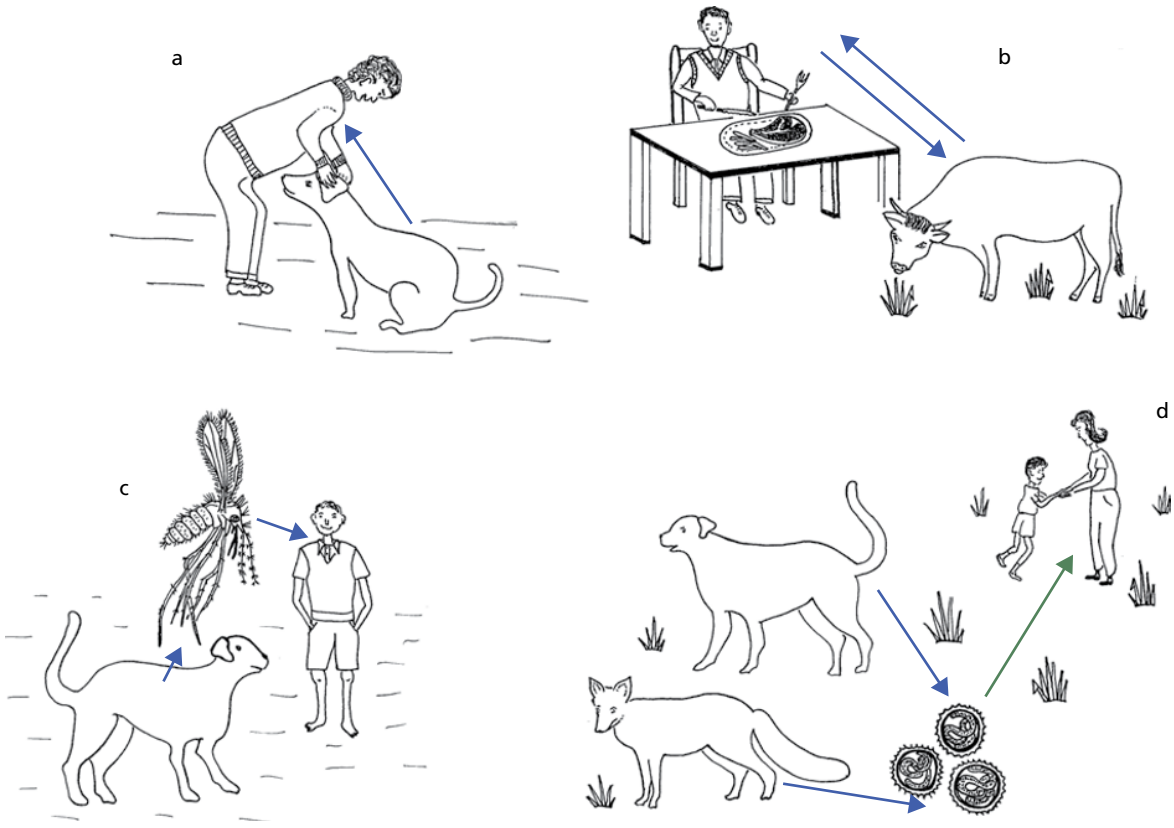

Figure 1.3 Ecological relationships that expose humans to zoonotic parasites: a – direct zoonoses; b – cyclozoonoses; c – metazoonoses; d – saprozoonoses (further explanation in text which uses same lettering as shown above). Sandfly redrawn after Mönnig from Lapage, 1962 with permission of Wolters Kluwer Health - Lippincott, Williams & Wilkins.

1.3 Host–parasite interactions

Hosts rarely gain any benefit from the presence of parasites and are often harmed by them. Defence mechanisms have therefore evolved which, if totally effective, would have extinguished parasitism as a lifestyle. But the continued existence of an abundance of parasites indicates that successful counter-strategies have arisen through natural selection. These in turn have driven the development of further protective measures and so the cycle known as the 'parasitic arms-race' continues. Coevolution has resulted in host–parasite interactions of such complexity that they can be reviewed only at a superficial level in an introductory text such as this.

1.3.1 Host defences

Hosts have evolved many behavioural and other strategies to reduce the risk of succumbing to parasitism. Herbivores, for example, will not eat the lush grass close to a faecal deposit where the greatest concentration of infective worm larvae occurs (the 'zone of repugnance'). The most powerful form of defence, however, is the immune system. This comprises a battery of chemical and cellular weaponry used to combat invasive organisms. Immune reactions may completely or partially disable the attacker or they may alleviate the clinical consequences of infection.

Ideally, immunity should protect against reinfection after the invading parasites have been eliminated. This is called 'sterile immunity'. It can last for a lifetime but often wanes with time. Sometimes, however, such protection persists

only as long as a few parasites survive to continually boost the immune processes. This is known as 'premunity'.

In some cases, parasite evasion has gained an evolutionary advantage that renders host immunity relatively ineffective, so the host remains vulnerable despite being repeatedly exposed to infection (e.g. sheep with liver fluke). Some immune reactions directed at a parasite can produce collateral damage to host tissues. Hypersensitivity and allergy are well-known examples.

Innate and acquired immunity

Vertebrates have evolved two separate but closely linked systems to provide protection against invasive pathogens. These are known as innate and acquired immune responses.

Innate immunity

The innate (or nonspecific) immune response is the body's first line of defence. It functions similarly whatever the nature of the invader and whether or not the host has experienced similar attack before. It comprises a series of natural physical, chemical and cellular barriers that are either permanent features (such as the integrity of skin and mucosae or the acidity of the stomach) or that can be quickly mobilised. The latter include a variety of cell-types with different modes of attack as well as humoral factors such as complement. A spectrum of communication molecules (cytokines and chemokines) released by white blood cells (leukocytes) enables the innate immune system to interact with the acquired immune system.

Help box 1.4

Some key immunological and pathological terms

Antibodies: macromolecules (immunoglobulins) produced by the host adaptive immune system to recognise specific receptor sites on alien molecules (antigens) and to initiate or assist in their neutralisation or destruction. There are different classes of antibody that are labelled IgM, IgG, IgE etc.
Antigen: molecule presented to a host that invokes an adaptive immune response.
Apoptosis: controlled and purposeful cell death (as opposed to necrosis, which is cell death due to an acute insult or injury, and autophagy, which is related to recycling cell components).
Chemokines: a specific class of cytokines that attract cells towards each other (chemotaxis), e.g. immune cells to the site of infection.
Complement: a biochemical cascade of small plasma and membrane-bound protein molecules that assist in the destruction of some invading organisms. One such cascade is a nonspecific innate response (the 'alternative pathway') while another is antibody-dependent (the 'classical pathway').
Cytokines: signalling molecules that cells use to communicate with each other. The term includes the interleukins (with names such as IL-2 and IFN-γ) that serve to modulate immune responses.
Eosinophilia: an increase in the number of eosinophils (white blood cells with red-staining granules) in the blood.
Humoral: a word used to describe aspects of immunity mediated by macromolecules in the blood or other body fluids (as opposed to cell-mediated immunity).
Hyperplasia: greater than normal proliferation of a particular cell type or tissue.
Lymphocytes: mononuclear white blood cells. There are several types: NK (natural killer) cells involved in innate immunity; B cells that produce antibodies; T cells involved in cell-mediated immunity, including Th (helper cells) that produce cytokines and cytotoxic cells that can kill parasitized host cells. There are also memory cells which enable pathogens to be quickly recognised on reinfection.
MHC: Major Histocompatibilty Complex. Molecules that carry parasite antigen to the surface of the host cell so that it can be recognised by antigen-processing cells.
Phagocyte: A cell that engulfs and ingests foreign particles.

Acquired immunity

Acquired (also called 'adaptive' or 'specific') immune responses come into action more slowly than innate reactions as they are tailor-made to combat the particular nature of each new challenge. A quicker response occurs when an animal is subsequently re-exposed to the same pathogen as the system is already primed for that specific reaction. Acquired immunity starts with the detection of foreign molecules (antigens) and the processing of these by antigen-presenting cells. This process generates two forms of adaptive response which are strongly linked to each other:

i) a cellular response characterised by T-lymphocyte participation, and
ii) humoral immune reactions mediated by B-lymphocytes and antibody-producing plasma cells.

Extra information box 1.1

The Th1/Th2 dichotomy

Different Th-lymphocyte subpopulations have different cytokine profiles and therefore play different roles. As either the Th1 or the Th2 subpopulation tends to predominate in a particular parasitic infection, the 'Th1/Th2 dichotomy' is an important determinant in the pathogenesis of infection and in the design of vaccination strategies. Th1-mediated responses are concerned mainly with cellular immunity and lead to the activation of effector cells, such as macrophages and dendritic cells. Th2-mediated responses are primarily associated with humoral immunity, with cytokines that result in anti-inflammatory reactions accompanied by an increase of specific antibody production, in particular IgE. Mast cells and eosinophils are also activated. These contain granules which, when released onto the surface of larger organisms, are capable of initiating enzymatic digestion.

In general, antigen-presenting cells processing bacterial and protozoan antigens tend to produce IL-12 which leads to an expansion of the Th1 population, whereas antigens derived from helminths and arthropods trigger mainly IL-4 and IL-6 which stimulate Th2-cell proliferation.

Immunity to arthropods

Most parasitic arthropods are ectoparasites. The degree of contact they have with body tissues and the time they spend on the host vary greatly – from a mosquito's fleeting visit to mites that burrow into the superficial epidermis. A few, like warble fly larvae, are true endoparasites, penetrating much more deeply into the body. Thus, opportunity for host detection of arthropod antigens varies accordingly, influencing both the nature and effectiveness of the subsequent immune responses.

In cases where contact is intimate and prolonged, as with some mange mites, a cell-mediated and partially protective immunity often develops. But where the antigens presented to the host are confined to those in the saliva injected during transient feeding behaviour (e.g. biting insects), immune responses may be limited to a local hypersensitivity. Such reactions do little to discourage further flies from biting and can become very itchy (pruritic). This may be of benefit if it encourages animals to move away from infested land or to adopt a more effective grooming behaviour (e.g. in flea or louse infestations), but pruritus can also provoke excessive scratching, rubbing and biting.

Ixodid ticks are rather different as, although they are temporary parasites, they remain attached to their host for several days while taking a blood meal. This provides greater opportunity for immune attack and, over time, parasitized hosts can develop a partially effective species-specific immunity. This acts by interrupting blood-sucking processes, thereby reducing the well-being and reproductive capability of the tick.

Immunity to protozoa

Parasitic protozoa that establish in extracellular positions within the body are exposed to humoral immune responses and are thereby susceptible to destruction by membrane disruption or ingestion by phagocytes. Those that have adopted an intracellular lifestyle will be shielded from such attack (except when moving between host cells) and cellular immune mechanisms are then more likely to be effective.

Extra information box 1.2

Some immune effector mechanisms

Lysis: A complement-dependent process in which the alternative pathway is activated by parasite surface antigens leading to destruction of the parasite by membrane disruption.

Opsonisation: A process whereby a pathogen is 'labelled' with a molecule (e.g. complement factors or a specific antibody) that attracts destructive cells such as phagocytes.

Phagocytosis: Phagocytes such as neutrophils, macrophages, monocytes and dendritic cells will ingest opsonised protozoa or parasitized host cells and attempt to kill them with oxidants, nitrous oxide, etc. and to digest them with enzymes.

Immunity to helminths

In contrast to protozoa, helminths are multicellular, relatively large and have a less intimate relationship with host tissues. Generally, they are extracellular and do not multiply within the host. Consequently, it is more difficult for the host to respond effectively. This is especially true for the many helminths that live in the lumen of the gastrointestinal tract as they are not in direct contact with any body tissue (see Figure 1.1). Immune attack has to be multifaceted and is often aimed at securing the parasite's demise by long-term attrition rather than swift execution.

Expulsion of nematodes from the gastrointestinal tract is a complex two-stage process. Firstly, the mucosal lining has to become permeable to macromolecules so

that specific antibodies (e.g. IgA) can 'leak' into the lumen at the site of parasitism. During this process goblet cell hyperplasia results in excess mucus formation. This helps to dislodge some helminths while others exploit it as their primary food-source, which illustrates the complexity and fascination of host–parasite relationships.

Extra information box 1.3

Immune effector mechanisms against helminths in the gastrointestinal tract

Immune protection against gastrointestinal helminths is largely orchestrated by Th2-cells situated in the Peyer's Patches (prominent thickenings of the gut wall). When activated by excretory/secretory (ES) helminth antigens, these cells produce a range of cytokines and chemokines which stimulate IgE production and eosinophilia, together with hyperplasia of mast and goblet cells. The IgE triggers mast cells to release granules containing vasoactive amines and histamine. These substances not only damage helminths directly but also increase gut permeability (permitting an outflow of specific antibodies). They also increase smooth muscle contractions in the gut wall (which helps to dislodge weakened parasites from their predilection sites).

Many gastrointestinal helminths migrate through body tissues en route to their predilection site and may consequently elicit different sets of immune responses during their parasitic life-cycle. They are likely to have reached the gut before acquired immunity to the tissue-stage becomes functional, but the activation of these adaptive responses will help protect the host against future invasion by the same species. Thus, there is an important difference between immunity that protects against reinfection and immunity that eliminates or ameliorates an existing infection.

Extra information box 1.4

Immune effector mechanisms against helminths within host tissues

Protection against tissue-dwelling helminths is predominantly of a cellular nature, reflecting their more intimate contact with their host. They are particularly prone to destruction by an antibody-dependent cell-mediated mechanism. IgE antibodies formed against surface antigens enable host cells such as eosinophils, neutrophils, macrophages and platelets to attach to the parasite and flatten out to ensure tight adhesion. The cells then secrete cationic proteins that are highly toxic to the helminth.

1.3.2 Parasite evasion of immunity

The survival of parasitic species is dependent on being able to escape the immune responses of its host. Such evasion strategies are multifaceted and can be divided into several main groups:

a – Sequestration: making it as difficult as possible for immune processes to reach the parasite. There are two main ways of doing this:

- **i)** by adopting a relatively inaccessible predilection site, e.g. within particular cell types or organs (such as the CNS or within the lumen of the gastrointestinal tract);
- **ii)** by generating a protective capsule, membrane or cyst wall.

b – Masking or changing surface antigens – examples include:

- **i)** incorporation of host molecules onto the surface of the parasite;
- **ii)** synthesis of parasite antigens which mimic host molecules;
- **iii)** antigen variance – periodic changes of surface antigens, thereby rendering previous host adaptive responses ineffective. Some parasites have stage specific antigens that serve the same purpose.

c – Disturbance of immunological effector mechanisms – examples include:

- **i)** surface shedding to remove adhering immune cells or specific antibodies bound to parasite antigen;
- **ii)** enzymatic digestion of antibodies;
- **iii)** inhibition of oxidative products synthesised by leukocytes;
- **iv)** reducing MHC-expression on the surface of infected cells, thereby inhibiting antigen presentation to the immune system.

d – Modulation of the host immune response – this can be achieved in various ways, for example:

- **i)** induction of multiple clones of T- and B-cells that produce nonspecific antibodies (polyclonal activation), thereby disabling the host's ability to manufacture in sufficient quantity the specific antibodies needed to combat the invading parasite;
- **ii)** induction of immune complexes in the blood and cleavage of antibody/ complement factors, both of which result in severe immune suppression.

e – Influencing apoptosis:

i) release of pro-apoptotic factors that shorten the life of leukocytes that might threaten the parasite;
ii) synthesis of anti-apoptotic factors by an intracellular protozoan parasite to prolong the life-span of its host cell.

f – Arrested development and hypobiosis: Some parasites are able to pause their development at a strategic point in their parasitic life-cycle. This waiting phase (termed 'arrested development') is used to synchronise parasitic development with host or environmental events (e.g. parturition or the onset of a favourable season of the year). There are various biological advantages to be gained from this (see for example Section 6.3.1). During this process, parasites often 'hide' from targeted host immune responses by slowing or shutting down vulnerable metabolic processes ('hypobiosis').

1.4 Parasitic disease

1.4.1 The host–parasite balance

In nature, the coevolution of host defence mechanisms and parasite evasion strategies has resulted in an uneasy equilibrium whereby there is no undue threat to the continued existence of either at a population level, although the well-being or survival of individuals (host or parasite) may be compromised. The parasite needs to feed and reproduce, yet it faces extinction should infection jeopardise the survival of the host population. In a stable ecosystem, a well-adapted parasitic species is one that survives in the host long enough to replicate but provokes no more than tolerable damage to the host population.

Disease generally indicates a disturbance of this ecological balance. This may be caused by naturally occurring factors, such as unusual weather conditions, but is often due to human intervention. Compare, for example, zebra roaming the African savannah carrying large worm burdens seemingly without ill-effect, with the vulnerability of horses confined to small paddocks.

The host–parasite relationship can be perturbed in two ways:

a – Increased host susceptibility – for example, if animals are:

i) stressed, debilitated or immunocompromised;
ii) exposed to parasites with which they have not coevolved (e.g. European cattle placed in a tropical environment);
iii) not allowed to express natural behaviour (e.g. restrained so they cannot groom to remove ectoparasites);
iv) selectively bred for production traits at the expense of natural ability to resist infection (innate or acquired);
v) inbred (e.g. some canine blood lines are particularly vulnerable to demodectic mange).

b – Increased parasite numbers – exposure to host-seeking (infective) life-cycle stages may increase, for example, if:

i) host stocking density is increased, thereby increasing the output of parasite eggs / larvae etc. per unit area (or per kg forage);
ii) parasitized animals are introduced into a previously clean area (e.g. through livestock movements, global trade etc.), thereby infecting susceptible local livestock, potential wild-life reservoirs or vectors;
iii) short-term weather patterns or longer-term trends such as global warming produce conditions more favourable for the development of preparasitic life-cycle stages;
iv) there is a surge in the population of intermediate hosts or vectors, or an increase in the number infected or their accessibility;
v) the parasite population becomes resistant to anti-parasitic medication.

As host defences and parasite immune evasion are both contributory elements to a stable host–parasite relationship, the total elimination of a parasite from the host population can have unintended consequences. For example, without the immuno-modulatory effect of parasites, the human immune system can go into 'overdrive' in some individuals. This may, at least in part, account for the recent increase in allergies and immune-mediated diseases recorded in affluent societies (see Section 7.1.6).

1.4.2 Why parasites are important

Many microbial diseases sweep through populations as dramatic and sometimes devastating epidemics. While parasites can also kill or provoke acute disease, their greatest effect is in the form of chronic, low-grade and debilitating damage. Frequently, the deleterious consequences of parasitism are not readily apparent on clinical examination and so the term 'subclinical disease' is often employed. The various ways in which parasites impact veterinary medicine can be summarised as follows:

a – Animal welfare: many parasitic infections cause pain, discomfort or are otherwise distressing to the host.

b – Agriculture: as well as obvious losses due to death and disease, subclinical disease is of significance as it prevents farm animals from attaining their full genetic potential. The constant drain on bodily resources, imposed by the need to maintain the immunological battle against parasites and to repair the physiological and structural damage they cause, can lead to reduced weight-gain or an increased food conversion ratio, or to a reduction in meat, milk or fibre (e.g. wool) yield and quality. This obviously affects agricultural production and economics. In impoverished rural communities, it deprives the human population of much needed sustenance and diminishes the animal power available to work the land and carry produce to market.

c – Veterinary public health: many parasites of animals are transmissible to humans and capable of causing disease. Parasite vectors can also transfer microbial diseases from animals to humans, e.g. ticks carrying the Lyme disease bacterium. Veterinary input is important in food hygiene to ensure that zoonotic parasites, such as the nematode *Trichinella*, are excluded from the food chain (see Section 9.3.1).

d – Aesthetic considerations: animal owners and consumers often find the sight or thought of parasites repugnant, even though there may be no immediate danger to themselves or their pets, e.g. a cat passing a tapeworm segment, or foodstuffs harbouring an innocuous parasite. Affected meat may be condemned at the abattoir, even though the parasite concerned is neither capable of infecting humans nor of causing overt disease in animals, e.g. *Taenia ovis*.

1.4.3 Pathogenic mechanisms

There are many ways in which parasites can damage tissues or adversely influence bodily functions. These include traumatic outcomes and mechanical defects, parasite-induced cellular and pathophysiological changes, together with detrimental cellular and immunological 'own-goals'. Intracellular parasites not only use their host cell as a food source but may also reprogram its genomic expression to meet their physiological requirements. A selection of the most commonly encountered pathologies is listed in Table 1.1. These and other mechanisms are described in later chapters.

Table 1.1 Some examples of how parasites damage their hosts

Type of damage	An example	More information in Section:
Space occupying lesions	Hydatid disease	5.3.4
Intestinal obstruction/perforation	Ascarid infections	7.1.3
Mechanical damage	Blowfly myiasis	2.2.6
Cell damage/necrosis by intracellular parasites	Coccidiosis	4.6.2
Fibrosis	Liver fluke disease	5.6.2
Epithelial hyperplasia: protein-losing enteropathies	Parasitic gastroenteritis	6.3.2
Malabsorption: villous atrophy	Coccidiosis	4.6.2
Plug feeding	*Strongylus vulgaris*	6.3.3
Anaemia: blood sucking	Hookworms	6.3.4
Anaemia: haemolysis	Babesiosis	4.8.1
Thrombosis	*Strongylus vulgaris*	6.3.3
Lung damage	Bovine lungworm	6.3.5
Heart malfunction	Canine heartworm	7.1.5
Immunological damage	Leishmaniosis	4.5.1
Inflammatory damage	Sheep scab	3.3.3
Neurological damage	*Sarcocystis neurona*	4.7.1
Secretion of pharmacologically active substances	Canine heartworm	7.1.5
Secretion of toxins	Some ticks	3.2.1
Abortion	Toxoplasmosis	4.7.3
Dermatitis	Flea infestation	2.2.2
Tumour formation	*Spirocerca*	7.1.5
Transmission of other pathogens	Many dipteran flies	2.2.5

1.5 Diagnostic techniques

Accurate diagnosis is an essential prerequisite for effective treatment and control. Sometimes the cause of disease may be obvious from clinical signs and history. On many occasions, however, the root of the problem may be obscure or confirmation may be required in order to rule out other possibilities. Diagnosis involves demonstrating parasitic involvement, determining the identity of the organism and, if necessary, quantifying the intensity of infection. Detection of a causal agent can be by direct observation of life-cycle stages in faeces, blood etc. or by gathering indirect evidence, such as the occurrence of specific antigens, antibodies or DNA-sequences. Sometimes, particular biochemical changes are associated with a parasitic infection (e.g. elevated serum pepsinogen concentrations in bovine ostertagiosis). Similarly, quantification can be direct (e.g. worm counts at autopsy) or it may provide an indirect indication (e.g. eggs per gram of faeces or an antibody titre).

1.5.1 Direct detection methods

Some ectoparasites, such as blowfly maggots, are easily accessible and large enough to be collected manually for identification. Others, such as parasitic mites, are too small or too deeply embedded in the skin and so brushing or scraping techniques are needed, with collected material subsequently prepared for microscopic examination.

Haematogenous parasites (i.e. carried in the blood) can be demonstrated in blood samples, which can be prepared as wet or dry smears, centrifuged or filtered as appropriate. Other endoparasites may present a greater challenge, but biopsy may be an option in specific cases. If deaths have occurred, autopsy of representative animals provides an opportunity for investigating the whole body for parasites or parasitic damage.

With living animals, however, faecal examination ('coproscopy') is probably the commonest laboratory diagnostic procedure for demonstrating the presence of endoparasites. Many parasites living in the respiratory system, liver or gastrointestinal tract have life-cycle stages that leave the animal with digestive waste. Sometimes microscopic examination of a fresh faecal smear may suffice, particularly if motile forms are present in large numbers. More often, there are only a few parasitic structures in a large faecal volume. Concentration techniques are therefore needed to assist detection.

Flotation

An appropriate amount of faeces is mixed with a larger volume of an aqueous solution (such as saturated sodium chloride, sodium nitrate or sugar) with a specific gravity that allows lighter parasitic structures, such as eggs, cysts or oocysts, to float while heavier faecal debris sinks. If known weights and volumes are used, a quantitative estimate can be made, e.g. eggs or oocysts per gram of faeces (abbreviated to e.p.g. and o.p.g., respectively). A McMaster counting chamber is often used for this purpose (see Figure 1.4). A subsample (aliquot) of the faecal suspension is pipetted into each of the two chambers on the slide. Eggs that rise and come to rest within the boundaries of the marked grids are identified microscopically and counted. As the volume of fluid beneath each square is known (0.15 ml), the e.p.g. value can easily be calculated.

Sedimentation

Some parasitic structures (e.g. trematode eggs) are too heavy to rise reliably in commonly used flotation fluids and so, in these cases, the faecal sample is mixed with a large volume of water, sieved to remove larger particles, and allowed to stand in a tall vessel. The sediment is examined after an appropriate period.

There are also centrifugation techniques that increase the speed and sensitivity of flotation and sedimentation. Some parasitic structures are more delicate than others and a technique must be selected that does not distort or destroy the object being sought.

It is sometimes necessary to 'culture' faecal samples to encourage development to life-cycle stages that are easier to identify, e.g. by hatching strongyle eggs and

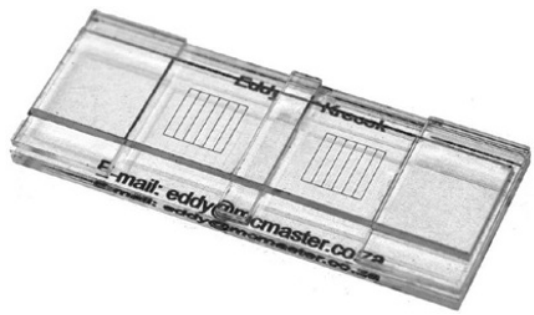

Figure 1.4 McMaster chamber (used for counting helminth eggs and/or coccidian oocysts in faecal samples). Reproduced with permission of T.E. Krecek.