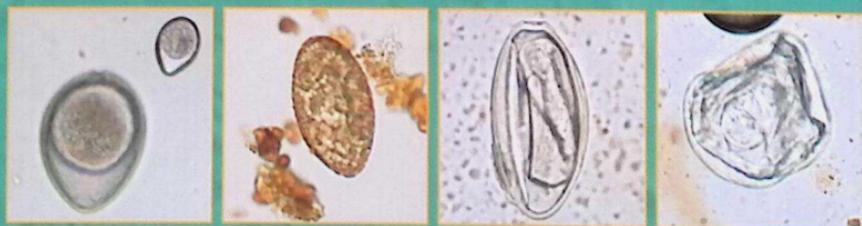


9<sup>th</sup> Edition

# Veterinary Clinical Parasitology

Anne M. Zajac | Gary A. Conboy  
Susan E. Little | Mason V. Reichard



WILEY Blackwell

# **Veterinary Clinical Parasitology**

**Ninth Edition**

**Anne M. Zajac**

**Gary A. Conboy**

**Susan E. Little**

**Mason V. Reichard**

Under the auspices of the **AAV**   
**American Association of Veterinary Parasitologists**

**WILEY Blackwell**

This edition first published 2021  
© 2021 John Wiley & Sons, Inc.

*Edition History*

This edition first published 2012 © 2012 by John Wiley & Sons Inc.  
First, Second editions, 1948, 1955 © Iowa State College Press  
Third, fourth, Fifth, Sixth editions, 1961, 1970, 1978, 1994 © Iowa State University Press  
Seventh edition, 2006 © Blackwell Publishing  
Eighth edition, 2012 © John Wiley & Sons Inc.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Anne M. Zajac, Gary A. Conboy, Susan E. Little, Mason V. Reichard to be identified as the authors of this work has been asserted in accordance with law.

*Registered Office*

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

*Editorial Office*

111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at [www.wiley.com](http://www.wiley.com).

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

*Limit of Liability/Disclaimer of Warranty*

The contents of this work are intended to further general scientific research, understanding, and discussion only and are not intended and should not be relied upon as recommending or promoting scientific method, diagnosis, or treatment by physicians for any particular patient. In view of ongoing research, equipment modifications, changes in governmental regulations, and the constant flow of information relating to the use of medicines, equipment, and devices, the reader is urged to review and evaluate the information provided in the package insert or instructions for each medicine, equipment, or device for, among other things, any changes in the instructions or indication of usage and for added warnings and precautions. While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

*Library of Congress Cataloging-in-Publication data applied for*

ISBN: 9781119300779 (Paperback)

Cover Design: Wiley

Cover Image: Courtesy of Anne M. Zajac

Set in 10/12pt Times New Roman by SPi Global, Pondicherry, India

10 9 8 7 6 5 4 3 2 1

# CONTENTS

Preface	<i>ix</i>
Acknowledgments	<i>xi</i>
Authors	<i>xv</i>
About the Companion Website	<i>xvii</i>

## CHAPTER 1 Fecal Examination for the Diagnosis of Parasitism 1

Collection of Fecal Samples	1
Storage and Shipment of Fecal Samples	2
Fecal Exam Procedures	2
Fecal Flotation	3
Additional Procedures for Fecal Examination	12
Quality Control for Fecal Exam Procedures	15
Use of the Microscope	16
Microscope Calibration	16
Pseudoparasites and Spurious Parasites	19
Identification of Nematode Larvae Recovered with Fecal Flotation or Baermann Procedures	24
Techniques for Evaluation of Strongyloid Nematodes in Grazing Animals	29
Fecal Culture	29
Identification of Ruminant and Camelid Third-Stage Larvae	30
Identification of Third-Stage Larvae of Equine Strongyles	35
Fecal Egg Count Reduction Test (FECRT)	36
Hoyer's Solution	39
Lactophenol	40
Parasites of Domestic Animals	41
Dogs and Cats	42
Ruminants and Camelids	96
Horses	126
Swine	140
Birds	154
Rodents and Rabbits	174
Reptiles	182

## CHAPTER 2 Detection of Protozoan and Helminth Parasites in the Urinary, Reproductive, and Integumentary Systems and in the Eye 191

Techniques for Parasite Recovery	191
Parasites of the Urinary System	191
Parasites of the Reproductive Tract	192
Helminth Parasites of the Integumentary System	192
Parasite Detection in Urinary and Other Systems	193

<b>CHAPTER 3</b>	<b>Detection of Parasites in the Blood</b>	<b>207</b>
	Immunologic and Molecular Detection of Blood Parasites	207
	Microscopic Examination of Blood for Protozoan Parasites	207
	Giemsa Stain	208
	Microscopic Examination of Blood for Nematode Parasites	209
	Tests for Canine Heartworm Microfilariae in Blood Samples	210
	Blood Parasites of Dogs and Cats	213
	Blood Parasites of Livestock and Horses	228
	Blood Parasites of Birds	234
<b>CHAPTER 4</b>	<b>Immunodiagnostic and Molecular Diagnostic Tests in Veterinary Parasitology</b>	<b>239</b>
	Immunodiagnostic Methods in Parasitology	239
	Molecular Diagnostic Methods in Parasitology	243
<b>CHAPTER 5</b>	<b>Diagnosis of Arthropod Parasites</b>	<b>247</b>
	Subclass Acari (Mites and Ticks)	247
	Mite Identification	247
	Tick Identification	278
	Class Insecta	300
	Lice (Order Phthiraptera)	300
	Fleas (Order Siphonaptera)	314
	Flies (Order Diptera)	322
	Other Insects	342
<b>CHAPTER 6</b>	<b>Parasites of Fish</b>	<b>347</b>
	Techniques for Recovery of Ectoparasites	347
	Skin Biopsy (Mucus Smear)	348
	Fin Biopsy (Fin Snip)	348
	Gill Biopsy (Gill Snip)	348
	Recovery of Endoparasites	349
	Parasites of Fish	350
<b>CHAPTER 7</b>	<b>Treatment of Veterinary Parasites</b>	<b>371</b>
	Introduction	371
	Anthelmintics	371
	Specific Anthelmintics	372
	Ectoparasiticides	375
	Protozoal Treatment	378
	Non-Traditional Treatments	379

## CHAPTER 8 Diagnostic Dilemmas 381

Diagnostic Dilemma 1	381
Diagnostic Dilemma 2	382
Diagnostic Dilemma 3	383
Diagnostic Dilemma 4	383
Diagnostic Dilemma 5	384
Diagnostic Dilemma 6	385
Diagnostic Dilemma 7	385
Bibliography	387
Index	391



## PREFACE

The first edition of *Veterinary Clinical Parasitology* was published in 1948 and has been used since that time by students, veterinary practitioners and others as an aid in the diagnosis of parasitic infections. Since 1994, it has been published under the auspices of the American Association of Veterinary Parasitology (AAVP) with the proceeds going to support student travel to the AAVP annual meeting. This sponsorship has not only supported development of more than one generation of veterinary parasitologists, but has also involved a community of parasitologists in the production of the book. The relationship with AAVP continues with the 9th edition. Also, as with past editions, this edition focuses on morphologic identification of parasites, which continues to be widely used in veterinary medicine, increasingly in combination with molecular or immunologic techniques.

There are also some new features in the 9th edition. We have added a chapter summarizing information on modern parasiticides. Additionally, preceding the photographs of parasites for each common domestic animal in Chapter 1 there is a table listing current U.S. label-approved treatments for many parasitic infections. We have also added another new chapter called Diagnostic Dilemmas. In this chapter we present challenging clinical scenarios and diagnostic results and allow the reader to make a diagnosis. A discussion of each of these scenarios is available on the accompanying website of the book.

The 9th edition also marks changes in the authorship of *Veterinary Clinical Parasitology*. This is the final edition that will be authored by Dr. Anne Zajac (author since the 6th edition) and Dr. Gary Conboy (author since the 7th edition). Drs. Susan Little and Mason Reichard, who joined as authors for this edition, will guide the book going forward and continue its association with AAVP.

Anne M. Zajac  
Gary A. Conboy  
Susan E. Little  
Mason V. Reichard

## ACKNOWLEDGMENTS

As ever, we are very grateful to the members of the American Association of Veterinary Parasitologists (AAVP) and others who have provided material for *Veterinary Clinical Parasitology*. Since the early 1990s appeals for photographs distributed through the AAVP listserv have brought responses from around the world.

We would also like to particularly acknowledge the extensive contributions to this edition of three of our veterinary parasitology colleagues, Dr. Mani Lejeune, Cornell University; Dr. Yoko Nagamori, Zoetis Corporation (formerly of Oklahoma State University); and Dr. Heather Walden, University of Florida, who graciously offered their magnificent photo collections for our use.

Finally, as Anne Zajac and Gary Conboy step away from authorship of *Veterinary Clinical Parasitology* they would like to gratefully acknowledge technical staff who, over many years, have assisted in identification and preparation of clinical samples utilized for photographs in this book. Those individuals include Susan King, Rosemary Cornett, John McInturff, Alex Fox and Diamond McClendon at Virginia Tech and Nicole Murphy, Robert Maloney and Janet Saunders at the University of Prince Edward Island.

Although the source of each figure is credited in the figure legend (with the exception of photos provided by chapter authors), we would also like to list all the contributors here with our deepest thanks:

- Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota,  
Minneapolis, MN
- Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton  
Rouge, LA
- Dr. Byron Blagburn, College of Veterinary Medicine, Auburn University, Auburn, AL
- Dr. Katie Boes, Virginia-Maryland College of Veterinary Medicine, Virginia Tech,  
Blacksburg, VA
- Dr. Dwight Bowman, College of Veterinary Medicine, Cornell University, Ithaca, NY
- Dr. Erin Burton, University of Minnesota College of Veterinary Medicine, St. Paul,  
MN
- Dr. Lyle Buss, Entomology and Nematology Department, University of Florida,  
Gainesville, FL
- Dr. Katie Clow, Ontario Veterinary College, University of Guelph, Guelph, Ontario,  
Canada
- Dr. George Conder, Pfizer Corporation, Kalamazoo, MI (retired)
- Dr. Kathryn Duncan, College of Veterinary Medicine, Oklahoma State University,  
Stillwater, OK
- Dr. Hany M. Elsheikha, School of Veterinary Medicine and Science, University of  
Nottingham, Loughborough, UK
- Dr. James Flowers, College of Veterinary Medicine, North Carolina State University,  
Raleigh, NC
- Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon,  
Saskatchewan, Canada

- Mr. James Gathany, Centers for Disease Control and Prevention, Atlanta, GA  
Dr. Ellis C. Greiner, College of Veterinary Medicine, University of Florida,  
Gainesville, FL (retired)  
Ms. Parna Ghosh, College of Veterinary Medicine, Oklahoma State University,  
Stillwater, OK  
Dr. Larry Hammell, Atlantic Veterinary College, University of Prince Edward Island,  
Charlottetown, PEI, Canada  
Dr. Bruce Hammerberg, College of Veterinary Medicine, North Carolina State  
University, Raleigh, NC  
Dr. Patricia Holman, College of Veterinary Medicine and Biomedical Sciences, Texas  
A&M University, College Station, TX  
Dr. Jennifer Ketzis, School of Veterinary Medicine, Ross University, St. Kitts, WI  
Dr. Manigandan LeJeune, Animal Health Diagnostic Center, Cornell University,  
Ithaca, NY  
Dr. David Lindsay, Virginia-Maryland College of Veterinary Medicine, Virginia Tech,  
Blacksburg, VA  
Ms. Megan Lineberry, College of Veterinary Medicine, Oklahoma State University,  
Stillwater, OK  
Dr. Aaron Lucas, Taylorsville Veterinary Clinic, Mt. Airy, MD  
Dr. Eugene Lyons, Department of Veterinary Science, University of Kentucky,  
Lexington, KY  
Dr. Charles Mackenzie, College of Veterinary Medicine, Michigan State University,  
East Lansing, MI  
Dr. Gil Myers, Myers Parasitological Service, Magnolia, TN  
Dr. Yoko Nagamori, Zoetis Corp., Stillwater, OK  
Dr. Stephen Jones, Lakeside Animal Hospital, Moncks Corner, SC  
Dr. Thomas Nolan, School of Veterinary Medicine, University of Pennsylvania,  
Philadelphia, PA  
Dr. Christopher Paddock, Centers for Disease Control and Prevention, Atlanta, GA  
Dr. Fernando Paiva, Universidade Federal de Mato Grosso do Sul, Campo Grande,  
MS, Brazil  
Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph,  
Ontario, Canada  
Dr. Sally Pope, Faculty of Veterinary Science, University of Sydney, Sydney, New  
South Wales, Australia  
Dr. Steffan Rehbein, Merial GmbH, Rohrdorf, Germany  
Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University,  
Manhattan, KS  
Dr. Meriam Saleh, Virginia-Maryland College of Veterinary Medicine, Virginia Tech,  
Blacksburg VA  
Dr. Nick Sangster, Charles Sturt University, Wagga Wagga, New South Wales,  
Australia  
Dr. Philip Scholl, Porto Alegre, RS, Brazil  
Dr. Stephen Smith, Virginia-Maryland College of Veterinary Medicine, Virginia Tech,  
Blacksburg, VA  
Dr. Karen F. Snowden, College of Veterinary Medicine and Biomedical Sciences,  
Texas A&M University, College Station, TX  
Dr. T. Bonner Stewart, School of Veterinary Medicine, Louisiana State University,  
Baton Rouge, LA

## ACKNOWLEDGMENTS

xiii

- Dr. Bert Stromberg, College of Veterinary Medicine, University of Minnesota,  
Minneapolis, MN
- Ms. Kellee Sundstrom, College of Veterinary Medicine, Oklahoma State University,  
Stillwater, OK
- Dr. Donald B. Thomas, U.S. Department of Agriculture Subtropical Agriculture  
Research Laboratory, Weslaco, TX
- Dr. Donato Traversa, Department of Comparative Biomedical Sciences, Faculty of  
Veterinary Medicine, Teramo, Italy
- Mr. Chris Tucker, Department of Animal Science, University of Arkansas,  
Fayetteville, AR
- Dr. Isabelle Verzberger-Epshtein, NRC Institute of Nutrisciences and Health,  
Charlottetown, PEI, Canada
- Mr. Martin Visser, Merial GmbH, Rohrdorf, Germany
- Dr. Heather Walden, College of Veterinary Medicine, University of Florida,  
Gainesville, FL
- Dr. Jerry Weintraub, Agriculture Canada, Lethbridge, Alberta, Canada
- Dr. Jeffrey F. Williams, Vanson HaloSource Inc., Redmond, WA
- Dr. Roy P. E. Yanong, Tropical Aquaculture Laboratory, University of Florida,  
Ruskin, FL
- Dr. Tom Yazwinski, Department of Animal Science, University of Arkansas,  
Fayetteville, AR
- Dr. Gary Zimmerman, Zimmerman Research, West Montana, Livingston, MT
- Dr. Kurt Zimmerman, Virginia-Maryland College of Veterinary Medicine, Virginia  
Tech, Blacksburg, VA

## AUTHORS

**Anne M. Zajac, DVM, PhD, Dip. ACVM-Parasitology**  
Department of Biomedical Sciences and Pathobiology  
Virginia-Maryland Regional College of Veterinary Medicine  
Virginia Tech  
Blacksburg, VA 24061

**Gary A. Conboy, DVM, PhD, Dip. ACVM-Parasitology**  
Department of Pathobiology and Microbiology  
Atlantic Veterinary College  
University of Prince Edward Island  
Charlottetown, Prince Edward Island C1A 4P3  
Canada

**Susan E. Little, DVM, PhD, Dip. ACVM-Parasitology**  
Department of Veterinary Pathobiology  
College of Veterinary Medicine  
Oklahoma State University  
Stillwater, OK 74078

**Mason V. Reichard, MS, PhD**  
Department of Veterinary Pathobiology  
College of Veterinary Medicine  
Oklahoma State University  
Stillwater, OK 74078

## ABOUT THE COMPANION WEBSITE

This book is accompanied by a companion website:

[www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

- The website includes PowerPoints of all figures from the book for downloading.
- Chapter 8 Diagnostic Dilemma answers.

# Fecal Examination for the Diagnosis of Parasitism

The fecal examination for diagnosis of parasitic infections is one of the most common laboratory procedures performed in veterinary practice. Relatively inexpensive and noninvasive, fecal examination can reveal the presence of parasites in several body systems. Parasites inhabiting the digestive system produce eggs, larvae, or cysts that leave the body of the host by way of the feces. Occasionally, even adult helminth parasites may be seen in feces, especially when the host has enteritis. Parasitic worm eggs or larvae from the respiratory system are usually coughed into the pharynx and swallowed, and they too appear in feces. Mange or scab mites may be licked or nibbled from the skin, thus accounting for their appearance in the feces. Many parasitic forms seen in feces have characteristic morphologic features that, when combined with knowledge of the host, are diagnostic for a particular species of parasite. On the other hand, certain parasites produce similar eggs or oocysts, and cannot be identified to the species level (e.g., many of the strongylid-type eggs from livestock). Fecal examination may also reveal to a limited extent the status of digestion, as shown by the presence of undigested muscle, starch, or fat droplets.

## COLLECTION OF FECAL SAMPLES

Fecal exams should be conducted on fresh fecal material. If fecal samples are submitted to the laboratory after being in the environment for hours or days, fragile protozoan trophozoites will have died and disappeared. The eggs of some nematodes can hatch within a few days in warm weather, and identification of nematode larvae is far more difficult than recognizing the familiar eggs of common species. Also, free-living nematodes rapidly invade a fecal sample on the ground, and differentiation of hatched parasite larvae from these free-living species can be time-consuming and difficult.

Owners of small animals should be instructed to collect at least several grams of feces immediately after observing defecation. This will ensure the proper identification of the sample with the client's pet (i.e., a sample from a stray animal will not be collected) and that feces rather than vomitus or other material is collected. The limited amount of feces recovered from the rectum on a thermometer or fecal loop should not

be relied on for routine parasitologic examination, since many infections that produce only small numbers of eggs will be missed. Owners should be instructed to store fecal samples in the refrigerator if the sample will not be submitted for examination for more than an hour or two after collection.

Feces should be collected directly from the rectum of large animals. This is particularly important when identification of individual animals is needed. Rectal samples are also needed when the sample is to be examined for lungworm larvae or cultured for identification of third-stage larvae, since contaminating free-living nematodes and hatched first-stage larvae of gastrointestinal nematodes may be confused with lungworm larvae. If rectal samples are unavailable, owners should be asked to collect feces immediately after observing defecation. The process of development and hatching of common strongylid eggs can be slowed by refrigeration. Development is also reduced when air is excluded from the sample by placing the collected feces in a plastic bag and evacuating or pressing out the air before sealing the bag.

### STORAGE AND SHIPMENT OF FECAL SAMPLES

If collected feces cannot be examined within a few hours, the sample should be refrigerated until it can be tested. Feces should not be frozen, because freezing can distort parasite eggs. If a sample needs to be evaluated for the presence of protozoan trophozoites like *Giardia* and trichomonads, it should be examined within 30 minutes after collection. The trophozoite is the active, feeding form of the parasite and is not adapted to environmental survival; it dies soon after being passed in the feces.

Increasingly, veterinary practitioners in the United States are using reference laboratories for routine diagnostic tests for parasite infection. Specific laboratory instructions for age, storage and transportation of samples to commercial labs should be followed. In general, when fresh fecal material is submitted to another laboratory for examination, it should be packaged with cold packs. In some cases, preservation of samples may be preferred. Helminth eggs can be preserved with a volume of 5%–10% buffered formalin equal to that of the sample. Formalin fixation also inactivates many other infectious organisms that may be present. Special fixatives, such as polyvinyl alcohol (PVA), are required to preserve protozoan trophozoites and are not routinely used in veterinary practices.

Slides prepared from flotation tests do not travel well, even if the coverslip is ringed with nail polish, since hyperosmotic flotation solutions will usually make parasite eggs or larvae unrecognizable within hours of preparing the slide. However, slides from flotation tests can be preserved for several hours to several days by placing them in a refrigerator in a covered container containing moist paper towels to maintain high humidity. It is best to place applicator sticks under the slide to prevent it from becoming too wet.

### FECAL EXAM PROCEDURES

Before performing specific tests on the fecal sample, its general appearance should be noted; consistency, color, and the presence of blood or mucus may all be indicative of specific parasitic infections. Hookworm disease in dogs, for example, commonly produces dark, tarry feces, whereas diarrhetic feces caused by whipworms may contain excess mucus and frank blood. The presence of adult parasites or tapeworm segments should also be noted.

## Fecal Flotation

The technique most commonly used in veterinary medicine for examination of feces is the fecal flotation test. This procedure concentrates parasite eggs and cysts while separating them from much of the sample debris. Fecal flotation is based on the principle that parasite material present in the feces is less dense than the fluid flotation medium and thus will float to the top of the container, where it can be collected for microscopic evaluation. Flotation tests are easy and inexpensive to perform, but in busy practices the choice of flotation solution and test procedure often does not receive much consideration, despite the substantial effect these choices can have on the sensitivity of flotation exams.

### Choice and Preparation of Flotation Solutions

Many different substances can be used to make flotation solutions. The higher the specific gravity (SPG) of the flotation solution, the greater the variety of parasite eggs that will float. Additionally, studies have shown that fecal flotation tests recover only a portion of each type of parasite egg/cyst in a sample because of variation in individual eggs, binding to debris, and so on. As SPG increases the portion recovered increases, which is an important consideration when the number of eggs in the sample is low. However, as SPG increases, more debris will also float, and the risk of damage to eggs from the hyperosmotic solution also increases. These factors limit the range of useful flotation solutions to SPG ranging from approximately 1.18 to 1.3. Both salt and sugar flotation solutions are commonly used in veterinary parasitology and provide flotation for common parasites with lower specific gravities than the flotation solution (Table 1.1).

Salt solutions are widely used in flotation procedures. A common flotation solution used in the United States is a commercially available sodium nitrate solution (SPG 1.20). This solution will float common helminth eggs and protozoan cysts. The commercial solution is not a saturated solution of sodium nitrate (SPG 1.33). Slides prepared with any salt solution need to be examined relatively quickly after they are prepared because crystals form as slides dry and parasites may be damaged, making them more difficult to identify.

Zinc sulfate ( $ZnSO_4$ ) solution at a SPG of 1.18–1.2 is another salt flotation solution. It is preferred at SPG 1.18 for recovery of *Giardia*, but recovers a higher proportion of

**Table 1.1. Approximate specific gravity of some common helminth eggs**

Species	Specific gravity
<i>Ancylostoma caninum</i>	1.06 <sup>1</sup>
<i>Toxocara canis</i>	1.09 <sup>1</sup>
<i>Toxocara cati</i>	1.10 <sup>1</sup>
<i>Trichuris vulpis</i>	1.15 <sup>2</sup>
<i>Taenia</i> sp.	1.23 <sup>1</sup>
<i>Physaloptera</i>	1.24 <sup>1</sup>
<i>Parascaris</i> spp.	1.09 <sup>2</sup>
Equine strongyles	1.05 <sup>2</sup>
<i>Anoplocephala perfoliata</i>	1.06 <sup>2</sup>

Source: From <sup>1</sup>David E., and Lindquist W. 1982. Determination of the specific gravity of certain helminth eggs using sucrose density gradient centrifugation. *J. Parasitol.* 68:916–919; <sup>2</sup>Norris J, Steuer A, et al. 2018. Determination of the specific gravity of eggs of equine strongylids, *Parascaris* spp., and *Anoplocephala perfoliata*. *Vet. Parasitol.* 260:45–48.

other parasites at SPG 1.2 and is probably used more frequently at this SPG. It is commercially available and when water is added to the purchased salt solution as directed, the resulting SPG is 1.2.

When detection of *Giardia* is required, a 33% zinc sulfate solution (SPG 1.18) is recommended because it does not cause the same rapid collapse of cysts seen with other flotation solutions and they are more easily recognized in flotation preparations.

Saturated solutions of sodium chloride (SPG 1.20) and magnesium sulfate (Epsom salts, SPG 1.32) are less widely used but can be easily prepared, are inexpensive, and are effective in floating common helminth eggs and protozoan cysts.

None of these salt flotation solutions will reliably float most trematode eggs, some tapeworm eggs, and very dense nematode eggs or larvae.

Another common solution used in routine flotation exams is Sheather's sugar solution (SPG 1.25). Because of its relatively higher SPG, Sheather's solution is also more efficient in recovering helminth eggs than common salt solutions, especially tapeworm and more dense nematode eggs. In addition, it does not distort eggs as rapidly as the salt solutions. Sheather's solution is specifically recommended for recovery of *Cryptosporidium* oocysts in fecal samples, but it does not appear to be as effective as 33% ZnSO<sub>4</sub> solution for detection of *Giardia*. Sheather's solution is inexpensive and easy to prepare and is also commercially available in the United States, but it is more viscous and sticky than salt solutions. The advantages and disadvantages of these solutions are shown in Table 1.2 and instructions for preparing them are given below.

Although the SPG of flotation solutions is not often measured in practices, it can be easily determined with an inexpensive hydrometer from a scientific supply company. A hydrometer will last indefinitely and should be considered part of quality control for the veterinary practice laboratory.

Table 1.2. Comparison of commonly used flotation solutions

Flotation solution	Specific gravity	Advantages	Disadvantages
Sodium nitrate (NaNO <sub>3</sub> ) Commercial product	1.18-1.2	Floats common helminth and protozoa eggs and cysts	Does not float most fluke and some tapeworm and nematode eggs
Saturated NaNO <sub>3</sub>	1.33		
Zinc sulfate (ZnSO <sub>4</sub> )	1.18-1.2	Floats common helminth and protozoa eggs and cysts; preferred for <i>Giardia</i> and some lungworm larvae	When used at SPG 1.18 recovers lower proportion of common helminth eggs; does not float most fluke and some tapeworm and nematode eggs
Saturated sodium chloride (NaCl)	1.2	Floats common helminth and protozoa eggs and cysts	Does not float most fluke and some tapeworm and nematode eggs
Saturated magnesium sulfate (Epsom salts)	1.32	Floats common helminth and protozoa eggs and cysts; higher SPG recovers parasites more efficiently	Higher SPG will float more debris; does not float most fluke and some tapeworms and nematode eggs
Sheather's sugar solution	1.20-1.28	Floats common helminth and protozoa eggs and cysts; higher SPG recovers parasites more efficiently; preferred for <i>Cryptosporidium</i> oocysts; generally less damaging than salt solutions	Does not float most fluke and some tapeworm and nematode eggs; creates sticky surfaces

**33% ZINC SULFATE SOLUTION (SPG 1.18)**

1. Combine 330 g zinc sulfate with water to reach a volume of 1000 mL.
2. Additional water or zinc sulfate can be added to produce an SPG of 1.18. If zinc sulfate solution is used with formalinized feces, the SPG should be increased to 1.20 and a SPG of 1.2 is often preferred for general use.
3. Check the SPG with a hydrometer.

**SATURATED SODIUM CHLORIDE (NaCl, SPG 1.2) OR MAGNESIUM SULFATE SOLUTION (MgSO<sub>4</sub>, SPG 1.32)**

1. Add salt to warm tap water until no more salt goes into solution and the excess settles at the bottom of the container.
2. To ensure that the solution is fully saturated, it should be allowed to stand overnight at room temperature. If remaining salt crystals dissolve overnight, more can be added to ensure that the solution is saturated. Table salt contains an anticaking compound that does not dissolve and should not be confused with residual sodium chloride crystals. Pickling salt does not contain this compound.
3. Check the SPG with a hydrometer, recognizing that the SPG of saturated solutions will vary slightly with environmental temperature.

**SHEATHER'S SUGAR SOLUTION (SPG 1.2–1.25)**

1. Combine 355 mL (12 fl oz) of water and 454 g (1 lb) of granulated sugar (sucrose). Corn syrup and dextrose are not suitable substitutes.
2. Dissolve the sugar in the water by stirring over low or indirect heat (e.g., the top half of a double boiler). If the container is placed on a high direct heat source, the sugar may caramelize instead of dissolving in the water.
3. After the sugar is dissolved and the solution has cooled to room temperature, add 6 mL formaldehyde USP to prevent microbial growth (30 mL of 10% formalin can also be used, with the volume of water reduced to 330 mL).
4. Check the SPG with a hydrometer.

**Flotation Procedures**

No matter how the flotation procedure is performed, the principle is the same. After mixing the flotation solution and the fecal sample together, the less dense material eventually floats to the top. This process can occur either by letting the mixture sit on the benchtop for a specified time (passive flotation) or by centrifuging the mixture. Centrifugation makes the flotation occur more rapidly and efficiently, regardless of the flotation solution used. Many practitioners like to use convenient commercial flotation kits that provide a container for collection of the sample and performing the test. However, the convenience of the kits is offset by the loss of sensitivity in the fecal exam procedure. A smaller amount of feces is used and the test cannot be centrifuged.

The increased sensitivity of the centrifugation procedure is particularly important in infections where the diagnostic form of the parasite may be present in low numbers (e.g., *Trichuris* and *Giardia* infections in dogs and cats). Centrifugation is also necessary when using 33% ZnSO<sub>4</sub> or sugar solution because of the slightly lower SPG of ZnSO<sub>4</sub> solution and the high viscosity of sugar solution, both of which retard the flotation

process. A veterinary practice that does not centrifuge flotation tests and relies on the traditional benchtop technique substantially reduces the sensitivity of its fecal exams.

### CENTRIFUGAL FECAL FLOTATION PROCEDURE

This is the best technique for the standard fecal flotation test regardless of the flotation solution used. It is particularly important to use this procedure with Sheather's sugar and 33%  $ZnSO_4$  flotation solutions to ensure that the flotation is effective:

1. Mix 3–5 g (about 1 teaspoonful) of feces with a small amount of flotation solution in a paper or plastic cup. Cat feces and small ruminant pellets, which are sometimes too hard to break up easily, can be ground with a mortar and pestle or allowed to soak in water until they become softer.

If the sample appears to contain a large amount of fat or mucus, an initial water wash is performed, and water should be used in Step 1. The water wash may be eliminated for most fecal samples of normal appearance.

2. Strain the mixture of feces and flotation solution (or feces and water if a water wash is performed) through a double layer of cheesecloth or gauze. A tea strainer can also be used.
3. Pour the mixture into a 15-mL centrifuge tube. If the rotor on the centrifuge is not angled (i.e., if the tubes hang straight when not spinning), the centrifuge tube can be filled with flotation solution until a reverse meniscus is formed and a coverslip is added (Fig. 1.1). The tube is spun with the coverslip. The centrifugal force generated by the centrifuge will hold the coverslip in place. If the centrifuge has an angled rotor, fill the tube to approximately 10–12 mL (amount that will prevent spilling) and place in the centrifuge.
4. Spin the mixture in a benchtop centrifuge for about 5 minutes at approximately  $500\text{--}650 \times g$  ( $650 \times g$  is 2500 rpm on a 4-in. rotor), regardless of whether the feces have been mixed with water or with the flotation solution. If a specific  $g$  force and speed setting cannot be determined, spinning the tube at the same speed used to separate serum from blood cells is sufficient.

If the initial spin is a water wash, the supernatant should be discarded, the sediment resuspended with flotation solution, and Steps 3 and 4 repeated.

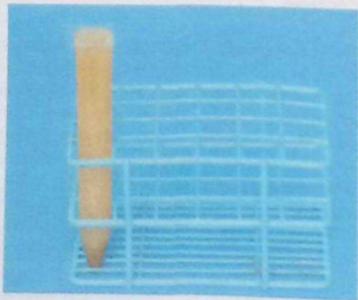


Fig. 1.1 Centrifuge tube filled with flotation solution to the top and coverslip placed in contact with the fluid column.

5. Allow the centrifuge to stop without using the brake. The slight jerking that results from the use of the brake may dislodge parasites from the surface layer. If preferred, the tube can be allowed to sit for an additional 5–10 minutes to maximize recovery of parasite material that may not have completed flotation through the liquid column to the surface.
6. Following centrifugation, there are several ways to harvest the surface layer of fluid containing parasite eggs. If the tube has been spun with the coverslip in place, lift the coverslip off the tube and quickly place it on a microscope slide. When the tube is spun without the coverslip, remove the tube from the centrifuge after spinning and place in a test tube rack. Fill the tube with additional flotation solution to form a reverse meniscus. Place a coverslip on the tube and allow it to sit for an additional 5–10 minutes before removing the coverslip and placing it on a slide.

Alternatively, after the centrifuge comes to a stop, gently touch the surface of the fluid in the tube with a glass rod, microbiologic loop, or base of a small glass tube and then quickly touch the rod to a microscope slide to transfer the drop or two of adhering fluid. This procedure will be less efficient than allowing the tube to stand with a coverslip in place.

#### BENCHTOP (SIMPLE OR PASSIVE) FLOTATION PROCEDURE

When a centrifugal flotation procedure cannot be performed, sodium nitrate and saturated salt solutions can be used in a benchtop flotation test, although a number of studies have shown that this procedure will detect significantly fewer parasite infections than the centrifugal flotation. This technique is not recommended for 33% ZnSO<sub>4</sub> or sugar flotation tests:

1. Mix several grams (a teaspoonful) of feces with the flotation solution in a cup.
2. Strain the mixture through cheesecloth or a tea strainer.
3. Pour into a test tube, pill vial, or container provided in a commercial kit. Add enough mixture or additional flotation fluid until there is a reverse meniscus on the top of the container. Place a coverslip on the fluid drop at the top.
4. Allow the flotation to stand for at least 10 minutes, remove the coverslip, place it on a slide, and examine. If the test is allowed to stand for too long, the salt may crystallize on the edges of the coverslip so that it will not lie flat on the slide.

#### FLOTATION SLIDES

Fecal flotation slides should be scanned using the 10× objective lens of the microscope (since most microscopes also have an eyepiece magnification of 10×, using the 10× objective gives a total magnification of 100×). Although most helminth eggs can be detected with the 4× objective, protozoan parasites are easily missed and this low power should not be used for scanning. The 40× lens should be used when there is uncertainty about the identity of structures on the slide and for scanning slides for *Cryptosporidium* oocysts. In some practices, to save expense, coverslips are not used. However, slides without coverslips dry out faster, do not have a flat plane of focus, and cannot be examined with the 40× lens, with the result that some parasites will be identified incorrectly or missed entirely.

## EGG-COUNTING PROCEDURES (QUANTITATIVE FECAL EXAMS)

Egg-counting techniques are also flotation tests and have several uses in food animals and horses. They can be used to assess the degree to which individual animals or groups are contributing to pasture contaminations with parasites, to assess efficacy of drug treatment (discussed in a later section in this chapter) and, in some cases, to determine relative levels of individual susceptibility to parasite infection.

Egg counts are of limited value in making judgments about the clinical condition of individual animals because many factors affect egg production, including parasite species, individual host immunity, and stage of infection. Also, counts performed on combined samples from a number of animals may not accurately reflect parasitism within that herd.

The easiest quantitative test to perform is the modified McMaster test. This test requires the use of special reusable slides (Fig. 1.2), which can be purchased from several suppliers (including Chalex Corporation, Wallowa, OR [[www.vetslides.com](http://www.vetslides.com)]; Focal Point, [www.mcmaster.co.za](http://www.mcmaster.co.za); JA Whitlock & Co., Eastwood, New South Wales, Australia [[www.whitlock.com.au](http://www.whitlock.com.au)]). The capacity of the counting chamber and the number of chambers counted per sample affect the detection level of the test. Saturated salt solutions are usually used as the flotation solution in this test.

As it is most commonly used, the modified McMaster test has a detection level of 25 or 50 eggs per gram (EPG) of feces. This level is acceptable in many situations since parasite control programs do not usually require detection of lower egg numbers. However, the accuracy of the McMaster test is reduced when egg counts are at the lower limits of detection. When egg counts of less than 200 EPG are expected in a group of animals being evaluated (e.g., in adult cattle or camelids) or when it is important to detect low numbers of eggs more accurately (e.g., in fecal egg count reduction tests [FECRTs]), a modification of the test is appropriate. The sensitivity and precision of the McMaster test can be improved by using slides that allow examination of larger quantities of the egg/flotation solution mixture, or by counting additional aliquots of the sample.

Alternatively, other procedures with lower detection limits can be used including the Wisconsin sugar flotation test (double centrifugation procedure) or a modified Stoll egg-counting test. These procedures permit detection of fewer than 10 EPG of fecal material but are more time-consuming to perform. Another procedure, the mini-FLOTAC



Fig. 1.2 McMaster slide used in the modified McMaster procedure for quantitative egg counts.

test, has been developed in Europe. Several studies have demonstrated greater precision and accuracy of the mini-FLOTAC compared to McMaster and Wisconsin tests but at this time it has limited availability and has been used in the United States primarily in research.

Another commercially available test for determining equine fecal egg counts is Parasight (Lexington, KY [www.parasightsystem.com]). This system provides counts of equine *strongyle* and *Parascaris* spp. eggs. An equine sample is mixed with reagents and fluorescence imaging is used with a software counting algorithm to produce a fecal egg count.

Regardless of the procedure used to count parasites, the most important element is consistency. Each step of the procedure should be performed in the same way for every sample.

#### *Modified McMaster test.*

1. For ruminants, combine 4 g of fecal material with 56 mL of flotation solution to yield a total volume of 60 mL. The test can also be performed with 2 g of feces and 28 mL of flotation solution when only small amounts of feces are available. For horses, it is standard in the United States to use 4 g of feces and 26 mL of flotation solution. See Note 1 below for modified calculations. Portable electronic scales that weigh in 0.1-g increments are widely available and inexpensive. If a method of weighing feces is not available, adding manure to the measured flotation solution until the final desired volume is reached can also be used (e.g., add manure to 26 mL of fluid to a total volume of 30 mL). This method would be more accurate with horse or cattle manure compared to the pelleted manure of small ruminants or camelids.
2. Mix well and strain through cheesecloth or a tea strainer. The mixture does not have to be strained, but it will be much easier to read the slide if large pieces of debris are removed. An alternative to straining is to use a filter pipette to transfer material to the counting chamber (a pipette with 12 mesh/cm wire mesh at the end is available from JA Whitlock & Co. [www.whitlock.com.au]).
3. Immediately fill each chamber of the McMaster slide with the mixture using a pipette or syringe. The entire chamber must be filled, not just the area under the grid. If large air bubbles are present, remove the fluid and refill.
4. Allow the slide to sit for at least 5 minutes before examining to allow the flotation process to occur. There has been limited investigation of the maximum amount of time slides can be allowed to sit before reading and there is no standard recommendation. Allowing slides to sit for an hour does not seem to alter results.
5. Look at the slide with the 10 $\times$  lens, focusing on the top layer, which contains the air bubbles. At this level, the lines of the grid will also be in focus. Count eggs, oocysts, and any other parasite stages, in each lane of both chambers. Each type of parasite should be counted separately. In some cases, eggs can be identified to genus or perhaps to species (e.g., *Strongyloides*, *Trichuris*, and *Nematodirus*), whereas others must be counted as a category of parasites (coccidia, strongylid eggs).

To determine the number of parasite EPG of feces, add the counts for both chambers for each parasite. The most commonly used McMaster slides in the United States are calibrated so that the number of eggs counted in a single chamber represents the number present in 0.15 mL of fecal mixture. If both chambers are counted and the results are added, the total represents the number of eggs present in 0.3 mL, which, for

example, is 1/200th of a total volume of 60 mL; therefore, the number of eggs counted must be multiplied by 200. However, if a total of 4 g of feces was used in the test, the result must be divided by 4 to yield EPG of feces. Multiplying by 200 and dividing by 4 is equivalent to multiplying the number of eggs counted by 50. Therefore, each egg observed represents 50 EPG in the final count. The same level of detection can be achieved by using 2 g of fecal material and 28 mL of flotation solution. The smaller amount of feces may be preferred when evaluating small lambs or kids.

#### Additional Notes

1. If a detection level of 25 EPG is desired, the McMaster test should be performed with 4 g of feces and 26 mL of flotation solution (results are then multiplied by 100 and divided by 4 as described earlier). Other combinations of manure and flotation solution can also be used with appropriate calculations using the formula

$$\text{eggs/g} = [\text{no. eggs counted} \times (T/V)] / F$$

where  $T$  = total volume of feces/flotation solution mixture,  $V$  = volume of aliquot examined in slide, and  $F$  = grams of feces used.

2. If pelleted feces are very hard, 2–5 mL of water can be added first and left to soften manure for at least an hour. The flotation solution is then added to the softened manure (flotation solution volume reduced by the amount of water used to soften feces).
3. A variety of methods can be used to homogenize feces and liquid. As alternatives to mixing with a tongue blade or other utensil, some laboratories use a mechanical or handheld kitchen mixer to homogenize feces and water/flotation solution. Shaking manure and fluid in a jar with glass beads has also been used, but as previously stated, whatever method is used, it should be consistent across samples.
4. In another procedure for the McMaster test, feces is mixed initially with water, strained, and centrifuged, and the supernatant is discarded. Flotation solution is added to resuspend the sediment and mixed, and the mixture is used to fill the counting chambers. For example, 3 g of feces is mixed with 42 mL of water, strained and the fluid used to fill a 15-mL centrifuge tube, which is then centrifuged at  $300\text{--}650 \times g$  for 2 minutes. The supernatant is then discarded and flotation solution is added to partially fill the tube, which is either shaken or stirred to resuspend the sediment. Additional flotation solution is added to fill the tube, and the counting chamber is filled. If the eggs in a volume of 0.3 mL are counted, the number of eggs seen is multiplied by 50 to give the number of eggs per g. The EPG can also be calculated using the formula described in Note 1. This procedure is the most effective for reducing debris but increases the time required to perform each test.

#### Mini-FLOTAC

The Mini-FLOTAC device and the Fill-FLOTAC device, which are used together in the mini-FLOTAC procedure, are available in North America from the University of Georgia. The test should be performed as described in the brochure provided with the test device. This procedure is generally more time-consuming than the basic modified McMaster procedure but is more accurate and precise.

*Wisconsin, Cornell-Wisconsin egg-counting test (double centrifugation procedure).*

The Wisconsin egg-counting test or double centrifugation flotation test is used to quantify eggs when low EPG are expected. Unlike the McMaster test, where eggs are counted in an aliquot of the mixture of feces and flotation solution, the Wisconsin test preparation collects eggs from the entire fecal sample/flotation solution mixture. Because it is time-consuming and difficult to accurately count numerous eggs on a slide with no grid this test is not suited to many circumstances where a quantitative count is needed. Any flotation solution can be used in this test:

1. Combine 1–5 g feces and 12–15 mL of water in a cup. Mix and strain into another cup, rinsing first cup with 2–3 mL of water and straining, pressing the liquid through. Pour into a 15 mL centrifuge tube.
2. Centrifuge (properly balanced) at  $300\text{--}650 \times g$  for 5–10 minutes.
3. After spinning, discard the supernatant and resuspend the pellet in flotation solution.
4. Either spin the tube with a coverslip in place or allow additional incubation after spinning as described for the centrifugal flotation procedure.
5. Remove the coverslip and place on a glass slide.
6. Examine with the  $10\times$  objective lens.
7. Count and record the number of each type of parasite egg/cyst seen, systematically scanning the slide and counting eggs or cysts of each parasite species or group separately. Care must be taken to ensure that each microscope field on the coverslip is examined once but only once so that no eggs are missed or counted twice.

This technique allows the quantification of less than 1 EPG of feces.

Additional Notes

1. If desired, the initial water wash can be omitted and the sample can be mixed directly with flotation solution and then centrifuged.
2. Alternatively, 22 mL of flotation solution is mixed with 5 g of feces, and the resulting mixture is divided between two tubes. This modification increases the accuracy of the procedure.

*Modified Stoll test.*

There are many modifications for the Stoll test, depending on the level of detection desired. Like the McMaster test, the modified Stoll egg-counting procedure is based on determining the number of eggs present in an aliquot of the prepared feces/flotation solution mixture. Any of the fecal flotation solutions can be used in this procedure.

1. Combine 5 g of feces and 20 mL of water in a cup.
2. Mix into a slurry and transfer 1 mL of the mixture to a centrifuge tube. If the mixture is not strained, a widemouthed or mesh-covered filter pipette is needed to transfer the mixture.
3. Fill the tube with flotation solution.

4. Place the tube in the centrifuge and add flotation solution until a slight inverse meniscus is formed.
5. Place a coverslip on top of the tube. It should contact the mixture without causing any to overflow.
6. Centrifuge (properly balanced) at  $300\text{--}650 \times g$  for 10 minutes.
7. Remove the coverslip and place on a glass slide.
8. Examine with the  $10\times$  objective lens.
9. Count and record the number of each type of parasite egg/cyst seen, systematically scanning the slide and counting eggs or cysts of each parasite species or group separately. Care must be taken to ensure that each microscope field on the coverslip is examined once but only once so that no eggs are missed or counted twice.

In this test, all the eggs present in 1 mL of the feces/flotation solution mixture were counted following centrifugation. This represents 1/25 of the volume of the mixture so the number of eggs counted multiplied by 25 represents the total number of eggs present, but this must be divided by 5 (grams of feces used) to yield EPG. In this situation, the multiplication factor is 5 and the minimum sensitivity of the test is 5 EPG. If the volume of fluid added to feces in the first step is 45 mL, the final multiplying factor would be 10.

For additional information on quantitative egg-counting procedures, see references by Verocai et al. (2020), Neilsen and Reinemeyer (2018), Taylor et al. (2015), Coles et al. (2006), and the Ministry of Agriculture, Fisheries and Foods (1986).

### Additional Procedures for Fecal Examination

The following procedures are used for identification of specific parasitic infections.

#### *Direct Smear and Stained Fecal Smears*

The direct smear is used to identify protozoan trophozoites (*Giardia*, trichomonads, amoebae, etc.) or other structures that float poorly or are readily distorted by flotation solutions. Because very little fecal material is used, the sensitivity of this test is low. It is not recommended for routine fecal examinations:

1. Mix a very small amount of feces with a drop of saline on a microscope slide to produce a layer through which newsprint can be read. Saline should be used because water will destroy protozoan trophozoites.
2. Use a coverslip to push large particles of debris to the side and place the coverslip on the slide. Examine with  $10\times$  and  $40\times$  magnification. The  $100\times$  lens (oil immersion) cannot be used effectively with fecal smears.
3. If the fecal layer is too thick, it will be impossible to see small, colorless protozoa moving in the field. Movement is the principal characteristic that allows recognition of trophozoites in fresh fecal smears. A drop of Lugol's iodine will enhance the internal structures of protozoan cysts but will also kill trophozoites present. To maximize the use of this test, it is best to look at an unstained smear before adding iodine.

Fecal smears can also be stained for identification of intestinal protozoa. Several stains can be used for identification of *Cryptosporidium*, including Ziehl-Neelsen.

Kinyoun, carbol-fuchsin, and Giemsa stains. Trichrome stain is widely used in human medicine for detection of *Giardia* cysts. In general, however, stains are not used extensively in veterinary practices and are not required for the identification of parasitic organisms. For details on performing these stains, a standard text on human parasitologic diagnosis should be consulted.

### **Fecal Sedimentation**

A sedimentation procedure is used to isolate eggs of flukes, acanthocephalans, and some tapeworms and nematodes whose eggs do not float readily in common flotation solutions. In the simple sedimentation test, tap water is combined with feces and allowed to settle briefly before the supernatant is removed. This allows the removal of fine particulate material, but unlike the flotation exam, sedimentation tests have only limited concentrating ability. Fat and mucus can be removed from the fecal sample if a centrifugal sedimentation exam is performed using ethyl acetate. Unfortunately, ethyl acetate is toxic and very flammable. It should be stored in a flameproof cabinet and used only in well-ventilated areas. An alternative to ethyl acetate is Hemo-De, available through Fischer Scientific ([www.fischerscientific.com](http://www.fischerscientific.com)), which is generally regarded as a safe compound and appears to give equivalent results in a centrifugal sedimentation procedure (see Neimester et al. 1987).

The Flukefinder® is a commercially available apparatus for performing sedimentation tests in the laboratory. It utilizes several screens to rapidly remove fecal debris. This device is very useful in practices conducting routine fecal examinations for flukes. Information on the Flukefinder can be obtained at [www.flukefinder.com](http://www.flukefinder.com).

### **SIMPLE SEDIMENTATION TEST**

1. Mix about 100 mL of water with about 10 g of feces, strain, and place in a beaker or other container.
2. Allow mixture to sit for 1 hour and then decant the supernatant.
3. Add more water, mix, and repeat the sedimentation procedure.
4. Stir remaining mixture and place a few drops on a microscope slide. If desired, add one drop of 0.1% methylene blue. The methylene blue will stain the background debris blue but will not stain fluke eggs, which will stand out with a yellowish brown color.
5. Coverslip and scan the slide using the 10× objective lens.

A smaller amount of feces and water can be used, placed in a test tube, and left to sit for 3–5 minutes between decantation steps. Addition of a drop of dishwashing soap to the water used in the test helps to free eggs from surrounding debris.

### **CENTRIFUGAL SEDIMENTATION TEST**

1. Mix 1 g of feces with about 10 mL of 10% buffered formalin or water. Pour mixture into a 15-mL centrifuge tube (with cap) until it is one-half to three-quarters full.
2. Add ethyl acetate (see earlier discussion on safety) or Hemo-De until the tube is almost full. Because organic solvents may dissolve some plastic centrifuge tubes, it is recommended that glass or polypropylene tubes be used for performing this test.
3. Cap and shake the tube approximately 50 times.

4. Centrifuge for 3–5 minutes at about  $500 \times g$  (as for centrifugal flotation procedure).
5. When the tube is removed from the centrifuge, it will have three layers: (1) an upper layer containing ethyl acetate, fat, and debris; (2) a middle layer containing formalin or water and fine particulate matter; and (3) a bottom layer of sediment. Using an applicator stick, loosen the top debris plug that sticks to the sides of the tube, then decant the supernatant, leaving only the bottom sediment.
6. Resuspend the sediment in a few drops of water or formalin, place one or two drops of the sediment on a slide, coverslip, and examine with the  $10\times$  microscope objective.

### Baermann Test

The Baermann test is used to isolate larvae from fecal samples and is employed most often to diagnose lungworm infections. *It is very important that the fecal sample be fresh.* If feces of a grazing animal are being examined and an old sample is used, strongylid or *Strongyloides* eggs may have hatched, or free-living nematodes may have invaded the sample, making nematode identification much more difficult. In small-animal samples, hookworm eggs may hatch very quickly and can be confused with lungworm or *Strongyloides* larvae. Coprophagy and hunting can also result in larvae of spurious parasites being present in Baermann test preparations. For discussion of identification of nematode larvae see the section in this chapter: "Identification of nematode larvae recovered with fecal flotation or Baermann procedures."

A further consideration for using a Baermann test in diagnosis is that metastrongyloid lungworms typically show erratic larval shedding patterns. Dramatic day-to-day variation in larval shedding increases the chance of false negative Baermann fecal examination results. Therefore, a single negative Baermann result is weak evidence for ruling-out lungworm infection in an animal showing signs of respiratory disease. Detection sensitivity is increased by doing multiple (at least 3) Baermann examinations.

A Baermann test requires equipment to hold the fecal sample in water so that larvae can migrate out and be collected. This can now be most easily accomplished with the use of a plastic wine glass with a hollow stem. In the absence of disposable wine glasses, the original Baermann apparatus can be used. This consists of a funnel clamped to a metal stand. A short piece of tubing with a clamp is attached to the end of the funnel. Larvae in feces placed either in the bowl of the wine glass or in the funnel migrate out of the sample and fall down into the hollow stem or the tubing above the clamp, where they can be easily collected (Fig. 1.3):

1. Place at least 10 g of feces in a piece of double-layer cheesecloth. Gather the cheesecloth around the sample so that it is fully enclosed. Use a rubber band to fasten the cheesecloth, and pass through the rubber band two applicator sticks, a pencil or other object that will rest on the edges of the glass or funnel and suspend the sample. Alternatively, place the sample on a suspended piece of wire mesh or sieve.
2. Fill the funnel or wine glass with lukewarm water. Make sure that the corners of the cheesecloth do not hang over the edge of the funnel or glass, because they will act as wicks for the water.
3. Allow the sample to sit for at least 8 hours, preferably overnight.
4. If using the disposable plastic glass, remove the fecal sample and collect the material at the bottom of the hollow stem using a Pasteur or transfer pipette or syringe.

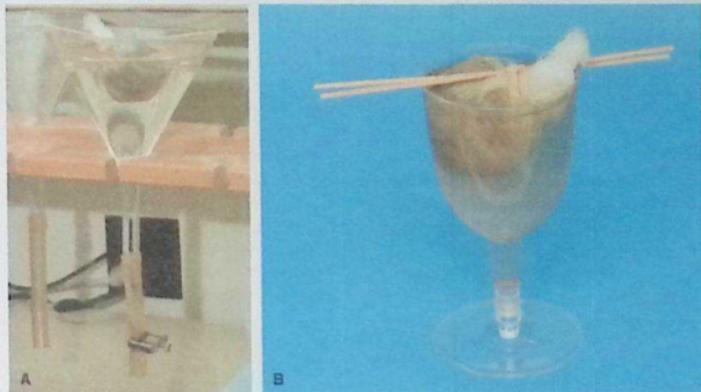


Fig. 13 (A) The traditional Baermann apparatus consisting of a suspended funnel with clamped tubing attached. For diagnostic testing of fecal samples, it is much more convenient to perform the Baermann exam with a disposable plastic wine glass (B).

- Transfer some of the fluid to a microscope slide, coverslip, and examine with the 4 $\times$  or 10 $\times$  objective lens.
- If using the funnel, release the clamp and collect the first 10 mL of fluid into a centrifuge tube. Spin as for a flotation exam, discard the supernatant, and examine the sediment. Alternatively, the very steady handed can carefully loosen the clamp and collect the first three or four drops onto a microscope slide.

### ***Immunologic and Molecular Methods of Parasite Diagnosis***

Immunologic methods have been important in the diagnosis of blood and tissue parasites for many years, and they are now being used increasingly for identification of specific parasites in fecal samples. Molecular diagnostic methods are also now being applied to detection of parasites in fecal samples. Although these techniques cannot currently replace morphologic exam of feces as a routine screening procedure for all parasites, they are useful for specific diagnosis of protozoan and helminth parasites that are detected in feces. For the discussion of these procedures, see Chapter 4.

### **QUALITY CONTROL FOR FECAL EXAM PROCEDURES**

Although the concept of quality control is not often applied to fecal exams, attention to both equipment and training will help ensure that fecal exams are consistently done correctly:

- Keep microscopes in good repair. Objective lenses and eyepieces should be routinely cleaned with lens cleaner and lens paper. Have microscopes professionally cleaned and checked every few years.

2. Check the SPG of flotation solutions with a hydrometer when first prepared to ensure that they will recover parasites effectively. If a batch of solution is used over an extended period, SPG should be checked at least monthly.
3. Use an ocular micrometer (see section on microscope calibration later in this chapter) to measure structures seen on fecal exams. If possible, recalibrate the microscope at regular intervals.
4. Make sure that personnel performing the fecal exams are adequately trained. It is not unusual for untrained assistants to be given rudimentary instruction on performing flotation tests and then be assigned to do them. Under these circumstances, it is hardly surprising that air bubbles are identified as coccidia and that smaller parasites are missed entirely.
5. As a check on the diagnostic accuracy of in-clinic fecal exams, periodically submit duplicate portions of fecal samples to a diagnostic laboratory. Both negative and positive samples should be submitted.

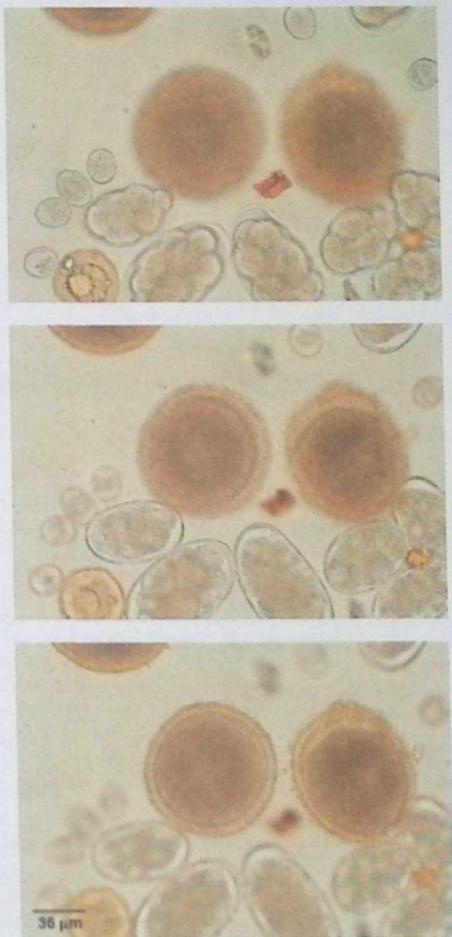
### USE OF THE MICROSCOPE

There are several points to remember in using the compound microscope to examine preparations for parasites:

1. Use the 10 $\times$  objective lens of the microscope for scanning slides. This will provide a total magnification of 100 $\times$  since most microscope eyepieces contain an additional 10 $\times$  lens. Start in one corner and systematically scan the entire slide. The 40 $\times$  objective lens (400 $\times$  total magnification with eyepiece) is useful for closer examination or for looking for very small organisms such as *Giardia* or *Cryptosporidium*. The 100 $\times$  (oil immersion) lens should not be used for flotation preparations. Not only is it likely that flotation solution will contact the lens and possibly damage it, but the pressure of the lens on the coverslip will create currents in the fluid on the slide, keeping everything in motion and making examination of structures difficult.
2. Most parasite eggs and larvae have little or no color and do not stand out well, so it is important to maximize the contrast between the parasites and their backgrounds. If a microscope has a substage condenser it can be used to increase contrast by adjustment of the condenser diaphragm or placement of the condenser in a low position. Even when a substage condenser is not available, reducing the intensity of light projected on the slide is generally advisable, either by decreasing the microscope rheostat setting or by reducing the aperture of the iris diaphragm. A higher power used for close examination will, of course, require an increased amount of light.
3. When reading a slide, it is helpful to focus up and down with the fine focus to change somewhat the plane of focus. Frequently, worm eggs will be at a slightly different level than protozoan cysts or oocysts, and a small manipulation of the fine focus may make structures more readily visible (Figs. 1.4–1.6).

### Microscope Calibration

The ability to measure the size of parasitic organisms and structures is very helpful when identifying unusual parasites or where different organisms are similar in appearance but differ in size. For measurements, a micrometer disc, also known as a reticle



**Fig 14-16** The importance of small changes in the microscope focus can be seen in these three photos of the same field in a canine fecal flotation test. In each case, a slight manipulation of the fine focus brings a different parasite into clearer view (first *Cystoisospora* oocysts, then hookworm eggs, and finally *Toxocara* eggs) since each egg or oocyst type may be present on a slightly different level.

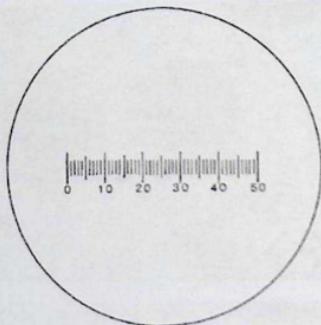


Fig. 17 A typical ocular micrometer of 50 divisions. The divisions have no meaning until calibrated against a stage micrometer.

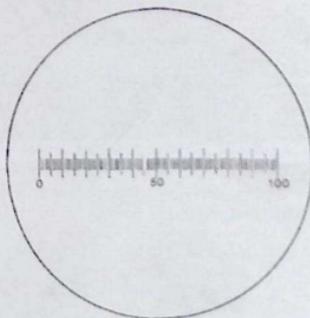


Fig. 18 A typical stage micrometer of 1 mm total length. Each division represents 10  $\mu\text{m}$ .

(Fig. 1.7), is inserted into the ocular tube of the microscope and calibrated against a known reference in the form of a stage micrometer (Fig. 1.8). Each objective lens of the microscope must be individually calibrated with the ocular lens/micrometer combination to be used, and the calibrations should be posted close to the microscope for easy reference. The calibration will be accurate only for that particular microscope ocular and objective combination. Even if each lens is not calibrated with the stage micrometer, the ocular grid will provide a consistent reference against which to compare objects seen in fecal samples. These ocular micrometer discs and stage micrometers are not expensive and can be purchased from scientific catalogs that include microscope equipment.

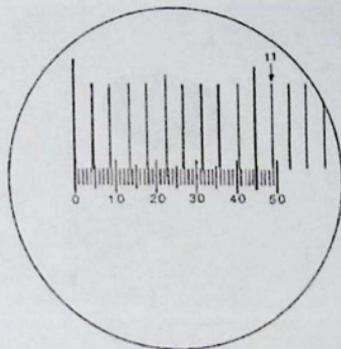


Fig. 1.9 Appearance at  $40\times$  of an ocular micrometer being calibrated with a  $10\ \mu\text{m}/\text{division}$  stage micrometer. Note the conjunction of line 11 of the stage micrometer with line 49 of the ocular micrometer.

Calibration of the  $40\times$  objective illustrates the procedure for calibration of the micrometer:

1. To calibrate the  $40\times$  objective, place the stage micrometer on the stage of the microscope and focus until the lines are sharp. In the example (Fig. 1.9), the stage micrometer is 1 mm ( $1000\ \mu\text{m}$ ) long and is divided into 100 parts; thus, each small division of the stage micrometer represents  $10\ \mu\text{m}$ .
2. Superimpose any convenient numbered line of the ocular micrometer (usually the 0 mark) on a convenient line of the stage micrometer (the first large line in the example). The field should now resemble Figure 1.9.
3. Find the two lines that are exactly superimposed. In the example, line 49 of the ocular micrometer falls exactly on line 11 of the stage micrometer. Thus, 49 divisions of the unknown ocular micrometer represent 11 divisions, each  $10\ \mu\text{m}$  in length, for a total of  $110\ \mu\text{m}$ . To complete the calibration, divide  $110\ \mu\text{m}$  by 49 divisions, resulting in a calibration factor of  $2.24\ \mu\text{m}$  per division for the ocular micrometer in the example.
4. Repeat this procedure for each objective lens to be calibrated on the microscope.

To use the calibrated microscope, superimpose the ocular micrometer scale on an egg or cyst and count the number of divisions subtended by the specimen, for example, 12. Multiply 12 by the calibration factor ( $2.24$  for the  $40\times$  lens in the example;  $12 \times 2.24 = 26.88\ \mu\text{m}$ , the size of the object measured).

## PSEUDOPARASITES AND SPURIOUS PARASITES

Fecal samples may contain deceptive "pseudoparasites" and "spurious parasites." Pseudoparasites are ingested objects that resemble parasite forms; these include pollen grains, plant hairs, grain mites, mold spores, and a variety of harmless plant and animal debris (Figs. 1.10–1.15). "Spurious parasites" are parasite eggs or cysts from one species of host that may be found in the feces of a scavenger or predator host as the result of coprophagy or predation (Figs. 1.16–1.18). One of the best ways to avoid



Fig. 1.10 Examples of pseudoparasites. (A) Pine pollen is a common pseudoparasite found in fecal samples of many animals (400 $\times$ ). (B) Adult free-living nematodes are also commonly found in fecal samples collected from the ground. These nematodes can rapidly invade fecal material. The presence of adults and variation in size and morphology (indicating different stages of the life cycle) are helpful in distinguishing these worms from parasitic larvae.

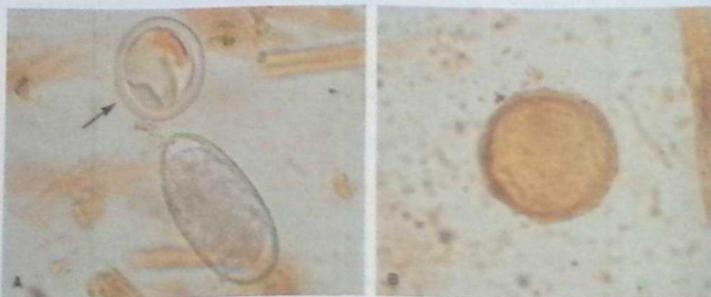


Fig. 1.11 Examples of pseudoparasites. (A) In this ovine fecal sample, both a strongylid egg and a pseudoparasite (arrow) are present. Characteristics helpful in the recognition of pseudoparasites are lack of clear internal structure and discontinuities in the outer layer. (B) Pseudoparasite, probably a pollen grain (400 $\times$ ).



**Fig. 112** Examples of pseudoparasites. (A) Insect hair from the feces of an insectivorous bird. Insect and plant hairs may be confused with worms but have no internal structure. (B) This artifact in ruminant feces appears to have structures resembling the hooks of a tapeworm embryo, but there is no distinct embryo and the outer layer is poorly defined with projections that are variable in size and shape (40 $\times$ ).



**Fig. 113** Examples of pseudoparasites. (A) Plant hairs and other fibrous material can resemble nematode larvae. They can be present in a variety of shapes and colors, but can usually be easily differentiated from nematodes because they lack clear internal structures like a digestive tract. Also, while one end is tapered, the other end often looks as though it has been broken off another structure. (B) This photo shows *Saccharomyces guttulatus*, a nonpathogenic yeast common in rabbits and seen occasionally in dogs (400 $\times$ ).

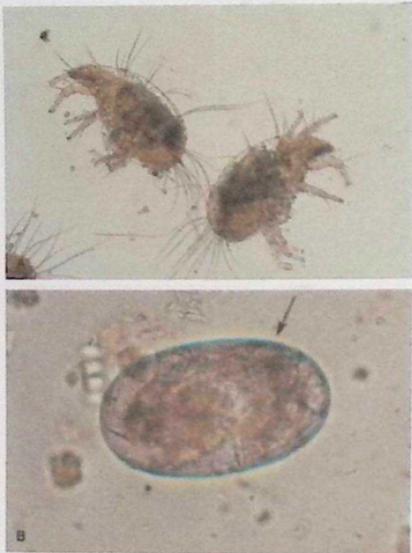


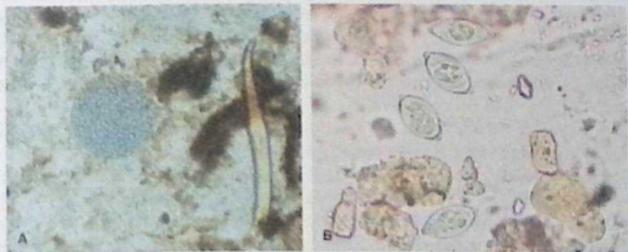
Fig. 114 Examples of pseudoparasites. (A) Free-living mites that contaminate animal feed can be found in fecal flotation procedures. Unlike many parasitic mites, free-living species lack specialized structures on their legs (suckers etc.) for adhering to the host. (B) Eggs from free-living mites will also float in flotation solution. They are usually very large ( $>100\ \mu\text{m}$ ). Developing legs of the mite can sometimes be seen inside the egg (arrows).



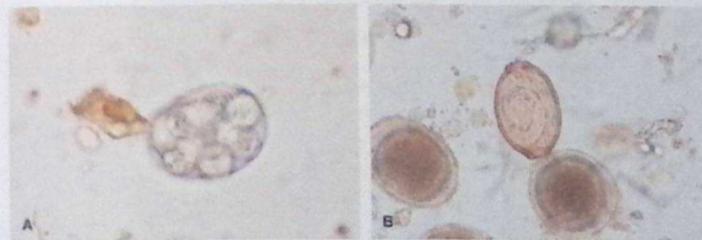
Fig. 115 Examples of pseudoparasites. Among the most common pseudoparasites found in feces are insect larvae, which may still be alive and moving when presented. Insect larvae may be ingested in food or, in the case of fly maggots, like the one shown here, eggs that are laid on the feces hatch rapidly in hot weather. Spiracles are present on the posterior (right) end of the segmented larva.



**Fig. 1.16** Spurious parasites are parasite eggs or cysts from another host that are acquired through predation or coprophagy and have merely passed through the digestive tract of the animal being tested. (A) Tapeworm egg found in a fecal sample from a calf. Although the configuration of hooks inside this egg clearly identifies it as a tapeworm, it is most likely a rodent or bird tapeworm egg. (B) Spurious parasites are common in samples from dogs that ingest fecal material. Eggs of livestock strongylid species can be found in feces of manure-eating dogs. Ruminant and equine strongylid eggs look like canine hookworm eggs but are larger and ruminant coccidia can usually be differentiated from dog and cat species based on size and shape.



**Fig. 1.17** Examples of spurious parasites. (A) Large cyst of *Monocystis*, a protozoan parasite of earthworms found in the feces of a snake that feeds on earthworms (100 $\times$ ). (B) Individual *Monocystis* oocysts that have been freed from a large cyst like the one shown in Figure 1.17A. These individual *Monocystis* oocysts are common pseudoparasites (400 $\times$ ). Photo B courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University.



**Fig. 1.18** Examples of spurious parasites. (A) *Adelina* sp. oocyst in a canine fecal sample. The oocysts of this genus are coccidia of insects and oligocheates and contain eight sporocysts. (B) Feline fecal sample containing two eggs from a feline parasite (*Toxocara*) and a single egg of a spurious parasite, *Trichosomoides*, a rodent parasite that is present as a result of hunting activity. Photos courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University.

misidentifying these pseudo- and spurious parasites is to appreciate the variety of parasites that normally infect a host species. If a fecal sample contains a possible pseudo-parasite or spurious parasite, it is best to repeat the examination with another sample collected at a later time. To limit opportunities for coprophagy or predation leading to ingestion of additional pseudo- and spurious parasites, small animals (dogs and cats) should be confined or leash walked only for 2–3 days prior to collection of the second sample.

### IDENTIFICATION OF NEMATODE LARVAE RECOVERED WITH FECAL FLOTATION OR BAERMANN PROCEDURES

Nematode larvae are passed in the feces of animals infected with various species of lungworms (*Aelurostrongylus abstrusus*, *Angiostrongylus vasorum*, *Crenosoma vulpis*, *Dictyocaulus* spp., *Filaroides hirthei*, *Muellerius capillaris*, *Oslersia osleri*, *Protostrongylus* spp., and others) or the intestinal threadworm, *Strongyloides stercoralis*. Accurate identification of nematode larvae detected on fecal flotation or Baermann tests tends to be a challenge for the veterinary laboratory diagnostician. In many cases where larvae are detected on fecal flotation, the damage due to the effects of high SPG flotation media obscures the larval morphology to the point that identification is not possible (Fig. 1.19). Therefore, the Baermann technique is the preferred method to recover first-stage nematode larvae from feces except in the case of *O. osleri* or *F. hirthei* infection in dogs (Figs. 1.94, 1.95). The Baermann technique is effective in recovering larvae that are vigorous and able to move out of the fecal matter. The larvae present in feces of dogs infected with *Oslersia* and *Filaroides* are sluggish and unable to migrate out of the feces. Therefore, zinc sulfate centrifugal flotation is the recommended method for the detection of larvae in the feces of dogs infected with these lungworms.

A further complication in larval identification may occur when there is a loss of sample integrity due to improper collection. Fecal samples that are not collected immediately after deposit on the ground may be invaded by free-living soil or plant parasitic nematodes. The challenge of sorting out these nematodes from the true parasitic ones is



Fig. 1.19 Larva detected on fecal flotation from a dog infected with lungworm. The larva is damaged due to the osmotic pressure of the high specific gravity flotation fluid. Loss of morphologic detail to this degree prevents specific identification.

beyond the training and experience of most veterinary laboratory diagnosticians. In addition, hookworm, strongyle, or trichostrongyle eggs, if present in feces, can develop and hatch in a short time under warm conditions, resulting in the detection of larvae that will be difficult to distinguish from those parasites normally passed as larvae in the feces. In small-animal practice where pet owners collect the fecal sample, the clients must be given guidance as to the requirements for a proper fecal sample. In the case of dogs, clients should be instructed to collect the fecal sample immediately after deposit and place it in an airtight, leakproof container. If submission to the veterinarian cannot occur within several hours of collection, the sample should be refrigerated at 4°C. In the case of cats, the litter pan should be cleaned and the next fecal sample observed in the pan should be collected and handled as above. Ruminant or horse samples should be collected from the rectum. If clients are collecting samples from the ground, they should be instructed to avoid collection of the portion of manure in direct contact with the soil. Lastly, a further complication in test evaluation can be the presence of spurious parasites acquired through predation or coprophagy.

The first question in the decision tree when evaluating nematode larvae is: parasite or free-living? Parasite larvae range in size from 150 to 400  $\mu\text{m}$  and their simple anatomy consists of a mouth opening leading to a buccal tube, esophagus, intestine, and anus. There may also be a discernable genital primordium. Free-living/soil/plant nematodes often occur in multiple life stages (from egg to adult), and size measurements are highly variable. The presence of adult female (eggs in the uterus, vaginal opening—Fig. 1.20) or adult male (spicules—Fig. 1.21) worms or the presence of an oral stylet in the buccal tube (Fig. 1.22) indicates that the sample may have been invaded by free-living nematodes. Unfortunately, *Strongyloides* spp. have a free-living generation that will develop if the sample is incubated and therefore are also a possibility when adult stages are recovered in a fecal sample. Detection of adult worms in the sample is an indication that the animal should be resampled and a fresh fecal sample should be submitted.

Another source of potential confusion in identifying larvae in feces is particularly common in coprophagic dogs, and can also occur in dogs or cats that are allowed to

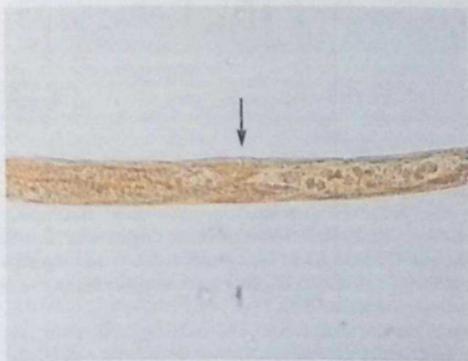


Fig. 1.20 Vaginal opening (arrow) of a free-living adult female nematode recovered from the feces of a dog. The feces were left on the ground long enough prior to collection to allow free-living soil nematodes to invade the sample.



Fig. 121 Tail of an adult male free-living nematode recovered from an improperly collected fecal sample of a dog. Note the chitinized spicules (arrow) at the cloacal opening.

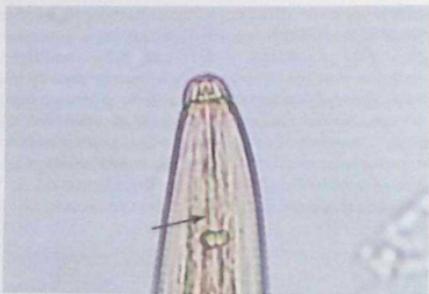


Fig. 122 Anterior end of a plant parasitic nematode recovered from an improperly collected fecal sample of a dog. Note the oral stylet (arrow) in the buccal chamber. The stylet is a daggerlike structure used in the feeding process to pierce plant roots. No parasitic first-stage larvae have this structure.

hunt. First-stage larvae present in feces or a prey animal will pass through the gastrointestinal tract intact. Depending on the timing of ingestion, the larvae may still be vigorously motile when recovered as a spurious parasite on Baermann examination. Reports of *Aelurostrongylus abstrusus* infection in the dog have all been based on detection of L1 in feces and are most likely false positive due to the ingestion of cat feces. Familiarity with common lungworms of other species will be helpful in recognizing the possibility of spurious parasitism.

Detection of larvae on microscopic examination of a slide prepared from a Baermann test is facilitated by the eye-catching vigorous motion of the larvae. However, once detected, a careful evaluation of the morphologic features is not possible in actively motile larvae. Therefore, it is necessary to kill them in a way that does not damage the morphology. Larvae are best killed by adding a drop of dilute Lugol's iodine (the color of weak tea) to the edge of the coverslip. The iodine will be slowly drawn across the

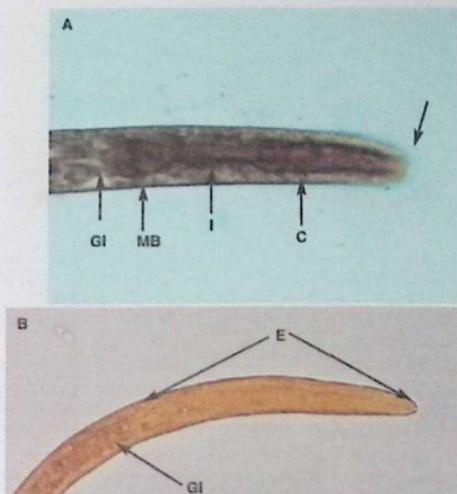


Fig. 1.23 (A) Anterior end of a first-stage larva of *Strongyloides stercoralis* recovered from the feces of a dog. This larva has been killed and stained with dilute iodine. The rhabditiform esophagus is well defined and obvious. Note the short buccal tube (arrow) and the rhabditiform esophagus made up of the corpus (C), isthmus (I), and muscular bulb (MB). Also note the distinct border demarcating the end of the esophagus and the start of the intestine (GI). (B) Anterior end of an iodine-stained first-stage larva of *Crenosoma vulpis* recovered from the feces of a dog. Note the poorly defined esophagus (E). It is difficult to discern the demarcation between the end of the esophagus and the start of the intestine (GI).

coverslip resulting in the death of the larvae. Alternatively, the larvae can be heat killed by passing the coverslip over the flame of a Bunsen burner to effect (several to many times). Larvae recovered on fecal flotation may or may not be already dead. Fecal flotation slides should be viewed as quickly as possible since the larval damage due to osmotic pressure will progressively worsen over time. Differentiation of the various parasitic nematode first-stage larvae is based on overall size and the morphology of the esophagus and tail. First-stage larvae of intestinal parasites (i.e., *S. stercoralis* or hookworm–strongyle–trichostrongyle eggs that have hatched) can be differentiated from the numerous lungworm larvae based on the presence of a distinct rhabditiform esophagus (Figs. 1.23A, 1.24, and 1.26). The rhabditiform esophagus consists of an anterior corpus that narrows to an isthmus and then ends in a muscular bulb. The rhabditiform esophagus is sharply delineated and well defined with an obvious sharp demarcation between the end of the esophagus and the beginning of the intestine. The overall length of the rhabditiform esophagus is less than 25% of the total length of the larvae (Fig. 1.24). In contrast, the esophagus of the lungworm larvae tends to be less well defined and longer, making up about 33%–50% of the total length of the larvae (Figs. 1.23B and 1.25). The relatively short buccal tube differentiates the larvae of *Strongyloides* (Fig. 1.23A) from those of hookworms–strongyles–trichostrongyles, which have a long buccal tube (Fig. 1.26).

Differentiation of the various lungworm larvae is based on tail morphology. In dogs, larvae with a straight tail and lacking a rhabditiform esophagus are *Crenosoma vulpis* (see

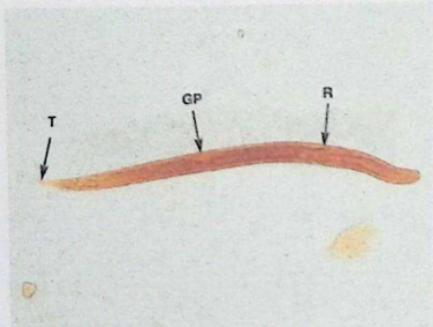


Fig. 1.24 *Strongyloides stercoralis* first-stage larvae recovered from the feces of a dog (Lugol's iodine stained and killed). Note the rhabditiform esophagus (R), prominent genital primordium (GP), and the straight tail (T). The rhabditiform esophagus makes up about 25% of the total length of the larvae.

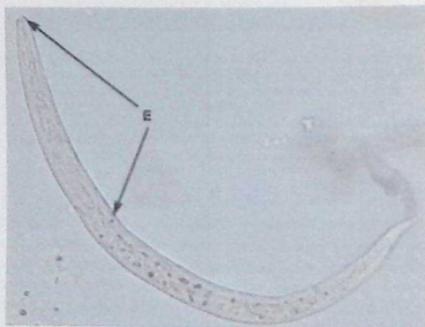


Fig. 1.25 *Cyrenosoma vulpis* first-stage larva recovered from the feces of a dog. Note the indistinct poorly defined esophagus (E). The esophagus makes up about 33%–50% of the total length of the larvae in metastrongyloid lungworms.

Figs. 1.25 and 1.92). Larvae that have a kinked S-shaped tail but lack a dorsal spine are either *Ostlerus osleri* or *Filaroides hirthi* (see Figs. 1.94 and 1.95). Larvae with a kinked tail and a dorsal spine are *Angiostrongylus vasorum* (see Fig. 1.93). In cats, larvae with a kinked tail and a dorsal spine are *Aelurostrongylus abstrusus* (see Figs. 1.90 and 1.91).

There should be only a single species of nematode lungworm larva, *Dictyocaulus viviparus* (see Fig. 1.151), recovered in properly collected fresh feces of cattle. The larvae have an abundance of visible food granules and a straight tail. The same situation occurs with the horse, although infection with *Dictyocaulus arnfieldi* (see Fig. 1.176, 1.177) is patent in donkeys, but only rarely in horses. In small ruminants, there are two larvae with

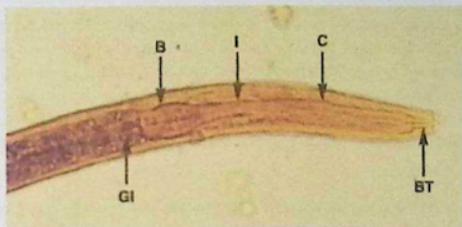


Fig. 1.126 Anterior end of a first-stage larva of a hookworm, *Uncinaria stenocephala*, recovered from an improperly collected fecal sample of a dog. This larva has been killed and stained with dilute iodine. As with *Strongyloides*, note the corpus (C), isthmus (I), and muscular bulb (B) of the rhabditiform esophagus and the intestine (GI). In contrast to *Strongyloides*, note the long buccal tube (BT).

straight tails, one with an abundance of visible food granules (*Dictyocaulus filaria*) (see Fig. 1.152) and the other without (*Protostrongylus rufescens*) (see Figs. 1.149 and 1.150). Another lungworm, *Muellerius capillaris*, produces larvae with a kinked tail and dorsal spine (see Figs. 1.146 and 1.147). *Cystocaulus* and *Neostrongylus* are small ruminant lungworms that are found in parts of Europe and Asia. The tails of their larvae have additional spines that can be used to differentiate them from *Muellerius* larvae.

## TECHNIQUES FOR EVALUATION OF STRONGYLID NEMATODES IN GRAZING ANIMALS

Grazing animals are infected with a variety of species of strongylid nematodes, which produce eggs that are not easily differentiated. In veterinary practices, it is usually unnecessary to identify individual species because treatment and control are generally directed to the entire group of nematodes rather than to a single species. If identification of the strongylid genera present in an animal or group of animals is needed, the simplest method for identification is culture of eggs to the third larval stage. In ruminants, these larvae can then be identified to parasite genus. In horses, this technique can be used to differentiate large and small strongyle larvae and identify some genera specifically. Currently, researchers are developing protocols for identification of parasite genera using molecular techniques (polymerase chain reaction [PCR]), and these procedures are now becoming commercially available.

### Fecal Culture

1. Fresh feces from cattle or horses should be thoroughly mixed and moistened with water if dry. Feces should not be wet, only moist. Larvae do not survive well in very wet fecal material. If feces are very soft or liquid, peat moss or vermiculite can be added to create a more suitable consistency. Sheep and goat pellets can be cultured as they are, without breaking them up. Rectal fecal samples are preferred for culture to prevent contamination with free-living nematodes.
2. Place feces in a cup or jar in a layer several centimeters deep. The container should have a loose cover that does not prevent air circulation but will deter flies and reduce

desiccation. The culture can be kept at room temperature for 10–20 days or at 27°C for 7 days. Daily stirring of the culture will inhibit mold growth and circulate oxygen for the developing larvae. Additional water can be added if feces begin to dry out.

- Following the culture period, harvest larvae with the Baermann test described previously. Alternative containers and methods for harvest of larvae can be found in Bowman (2014), Taylor et al. (2015), and other textbooks of veterinary parasitology.

### Identification of Ruminant and Camelid Third-Stage Larvae

To identify larvae, place a drop or two of liquid containing larvae from the Baermann procedure on a microscope slide. Add an equal amount of Lugol's iodine. The iodine will kill and stain the larvae so that they can be examined closely.

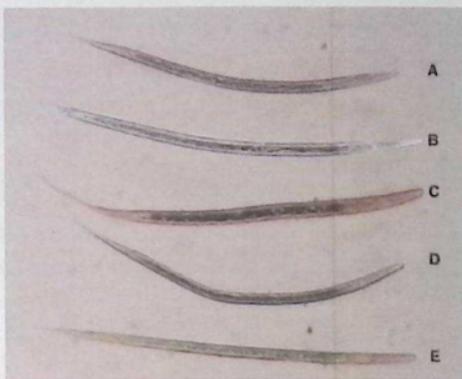
Larvae recovered from ruminant fecal material can be most easily identified by a combination of morphology and size. The shape of the head and the shape of the tail and of the sheath extending beyond the tail at the posterior end of the larva are important characteristics, and both should be evaluated on each larva before an identification is made. The sheath is the retained cuticle of the second larval stage and provides larvae with increased protection from environmental conditions. Measurements of total larval and sheath length are helpful as well (Table 1.3), but sizes often overlap between genera and size characteristics can be affected by culture conditions and age of larvae. Consequently, measurements alone should not be used to identify larvae.

**Table 1.3. Morphologic characteristics of infective third-stage strongylid larvae of domestic ruminants**

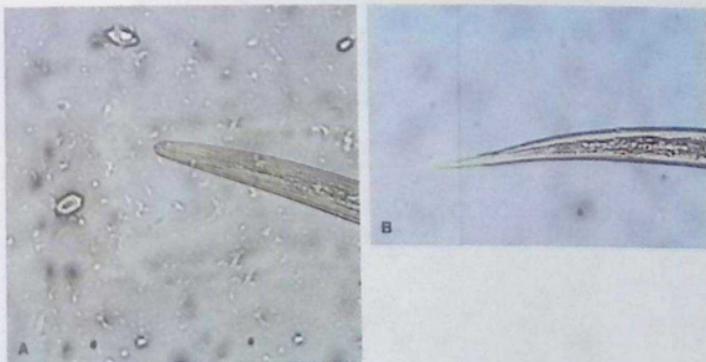
Genus	Overall length ( $\mu\text{m}$ )	Anus to tip of sheath ( $\mu\text{m}$ )	End of tail to tip of sheath ( $\mu\text{m}$ )	Other characteristics
<i>Trichostrongylus</i>				
Sheep	622–796	76–118	21–40	Head rounded; tail of sheath short; tail may have one or two tuberosities
Cattle	619–762	83–107	25–39	
<i>Ostertagia</i>				
Sheep	797–910	92–130	30–60	Head squared; tail of sheath shorter in sheep
Cattle	784–928	126–170	55–75	
<i>Haemonchus</i>				
Sheep	650–751	119–146	65–78	Head rounded; sheath tail medium length, offset
Cattle	749–866	158–193	87–119	
<i>Cooperia</i>				
Sheep	711–924	97–150	35–82	Head squared with two refractile oval bodies at anterior end of the esophagus; medium-length sheath tail tapering to fine point
Cattle	666–976	109–190	47–111	
<i>Nematodirus</i>				
Sheep	922–1118	310–350	250–290	Broad, rounded head; intestine with eight cells; tail notched and lobed; long thin sheath tail
Cattle	1095–1142	296–347	207–266	
<i>Bunostomum</i>				
Sheep	514–678	153–183	85–115	Small larva with rounded head; long thin sheath tail
Cattle	500–583	129–158	59–83	
<i>Oesophagostomum</i>				
Sheep	771–923	193–235	125–160	Rounded head; long thin sheath tail; 16–24 triangular intestinal cells
Cattle	726–857	209–257	134–182	
<i>Chabertia</i>				
Sheep	710–789	175–220	110–150	Rounded head; long thin sheath tail; 24–32 rectangular intestinal cells

Sources: Bowman (2014) and Ministry of Agriculture, Fisheries and Food (1986).

Relative proportions of parasite genera in larval cultures cannot be used to predict numbers of adult worms in the gastrointestinal tract. For example, *Haemonchus contortus* is highly prolific and may dominate in small ruminant fecal cultures, even when adult parasites of other genera are present in substantial numbers. Figures 1.27–1.37 show morphologic characteristics of common third-stage larvae of small ruminants and cattle.



**Fig. 1.27** Third-stage larvae of common small ruminant strongylid genera collected from fecal culture. This photo shows the relative size relationships among the larvae. The following photographs show the details and the anterior and posterior ends of the individual larvae. *Nematodirus* spp. larvae are usually not encountered in cultures and are not illustrated here. They are bigger in total length and have a longer tail sheath than other larvae. (A) *Trichostrongylus*, (B) *Teladorsagia*, (C) *Oesophagostomum*, (D) *Haemonchus*, and (E) *Cooperia*. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.



**Fig. 1.28** *Trichostrongylus* larva from sheep, head (A) and tail sheath (B). The head of *Trichostrongylus* larvae is tapered and the tail sheath is short. The tail may end in one or two tuberosities. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.



Fig. 1.89 Ovine *Teladorsagia* head (A) and tail sheath (B). *Teladorsagia* can easily be confused with *Trichostrongylus*, but *Teladorsagia* is generally larger and the head is squared, not tapered. The sheath of the tail of *Teladorsagia* is short. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

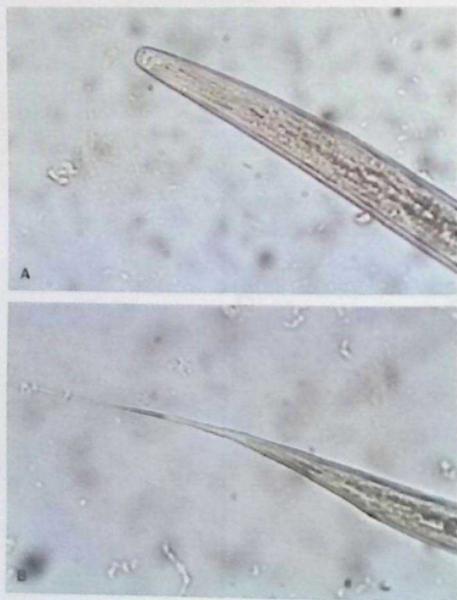


Fig. 1.80 *Oesophagostomum/Chabertia* head (A) and tail sheath (B). The larvae of these two genera are not easily distinguishable, but they are not difficult to differentiate from other genera. The tail sheath is long and filamentous, and the head is broad and rounded. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

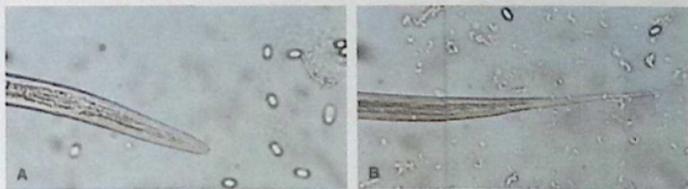


Fig. 131 *Haemonchus* head (A) and tail sheath (B). The larvae of *Haemonchus* have the most narrowly rounded head of the common larvae. The tail sheath is medium in length and often has a slight kink at the end of the tail. Photo courtesy of Dr. Tom Yarwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

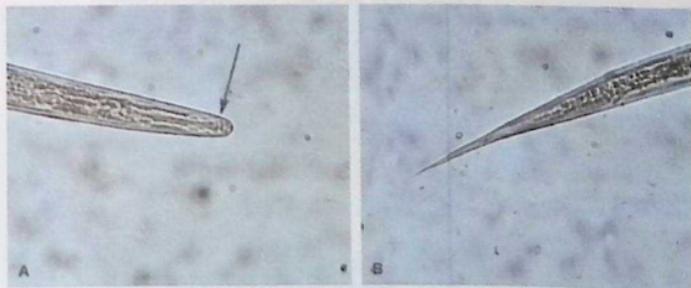


Fig. 132 *Cooperia* from a sheep, head (A) and tail sheath (B). *Cooperia* third-stage larvae are distinguished by a pair of refractile bodies (arrow) present in a squared head. These bodies are difficult to photograph but easy to appreciate under the microscope. The sheath of the tail is medium in length and tapering or finely pointed. Photo courtesy of Dr. Tom Yarwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

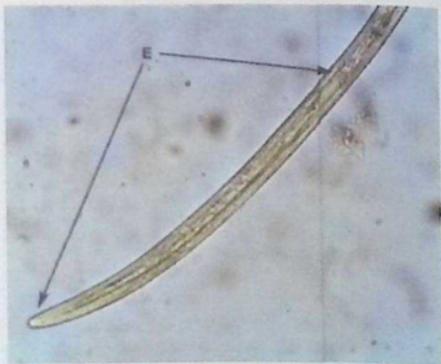


Fig. 133 *Strongyloides papillosus* is a nematode of ruminants that is unrelated to the important strongylid nematodes. Infective third-stage larvae of *Strongyloides* may be present in larval cultures. They do not have a sheath and the esophagus is very long (E). Additionally, free-living nematodes may be numerous in cultures contaminated with soil. For information on identifying free-living nematodes, see the section on identifying larval nematodes in fecal samples. Photo courtesy of Dr. Tom Yarwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.



Fig. 134 Third-stage *Cooperia* larvae from cattle. Several species of *Cooperia* infect ruminants, and the length of the tail sheath is variable. *Cooperia oncophora* (A) produces larvae with a longer tail sheath than other species of the genus (B). Photo courtesy of Dr. Tom Yarwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

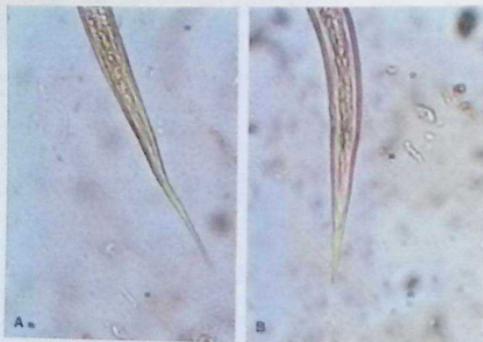


Fig. 135 Tail sheath of *Ostertagia* (A) and *Trichostrongylus* (B). Both genera have a short tail sheath, but *Ostertagia* has a blunter head. Photo courtesy of Dr. Tom Yarwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

Additional information on identification of ruminant third-stage larvae can be found at the website of the RVC/FAO Guide to Veterinary Diagnostic Parasitology: [www.rvc.ac.uk/review/Parasitology/Index/Index.htm](http://www.rvc.ac.uk/review/Parasitology/Index/Index.htm) and in van Wyk and Mayhew (2013).

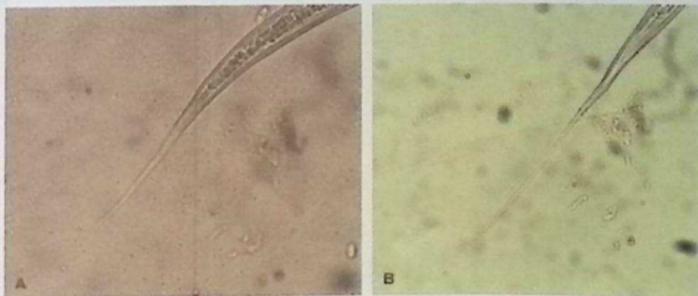


Fig. 136 Tail sheath of *Haemonchus* (A) and *Oesophagostomum* (B) from cattle. The tail sheaths of the parasites occurring in cattle are similar to those in sheep. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.



Fig. 137 Tail sheath of *Bunastomum*. Species of this ruminant hookworm infect both cattle and sheep. The third-stage larva is smaller than those of other genera and has a thin tail sheath. Photo courtesy of Dr. Tom Yazwinski and Mr. Chris Tucker, Department of Animal Science, University of Arkansas, Fayetteville, AR.

### Identification of Third-Stage Larvae of Equine Strongyles

Horses are infected with over 30 species of strongylid parasites, but only a few can be identified on the basis of the third-stage larva. Most of the small strongyle species can only be identified as cyathostomin parasites from the infective larval stage (Fig. 1.38 and Table 1.4). The posterior portion of the sheath of horse strongyle larvae is very long and filamentous, making these larvae easily recognizable as infective parasite larvae. The number of intestinal cells in these larvae is variable and is useful in identification.

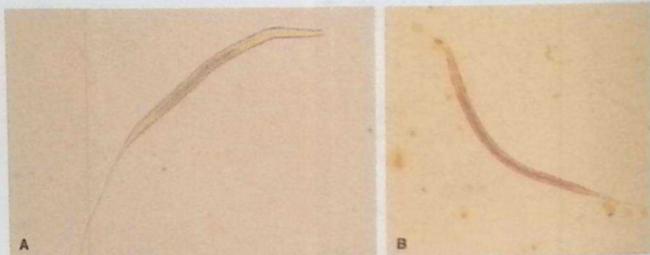


Fig. 1.38 Infective third-stage larvae of both large and small equine strongyles have a very long filamentous extension of the sheath. (A) The larvae of small strongyles (cyathostomes) have eight intestinal cells, which can be easily counted in the larva shown here. (B) Larvae of large strongyle species have more than eight intestinal cells, like this *Strongylus vulgaris* larva with at least 28 cells.

Table 1.4. Morphologic characteristics of infective third-stage strongylid larvae of horses

Genus	Characteristics
<i>Strongyloides</i>	Sheath absent; esophagus almost half the length of the body
<i>Trichostrongylus axei</i>	Tail of sheath short, not filamentous
Most small strongyles (Cyathostominae)	Long filamentous sheath; eight triangular intestinal cells
<i>Gyaloccephalus</i> (small strongyle)	Long filamentous sheath; 12 rectangular intestinal cells
<i>Oesophagodontus</i> (small strongyle)	Large larva; long filamentous sheath; 16 triangular intestinal cells
<i>Posterisostomum</i> (small strongyle)	Long filamentous sheath; 16 roughly rectangular intestinal cells
<i>Strongylus equinus</i> (large strongyle)	Long, thin larva with filamentous sheath; 16 poorly defined rectangular intestinal cells
<i>Triodontophorus</i> (large strongyle)	Medium-length and broad larva with filamentous sheath; 18–20 well-defined rectangular intestinal cells
<i>Strongylus edentatus</i> (large strongyle)	Smaller larvae with filamentous sheath; 18–20 poorly defined and elongated intestinal cells
<i>Strongylus vulgaris</i> (large strongyle)	Large larvae with filamentous sheath; short esophagus; 28–32 well-defined, rectangular intestinal cells

Source: Adapted from Ministry of Agriculture, Fisheries and Food (1986).

### Fecal Egg Count Reduction Test (FECRT)

One of the principal uses of quantitative egg counts is the evaluation of drug efficacy. Anthelmintic resistance in strongylid nematodes of horses, small ruminants, and cattle is rapidly increasing worldwide. The only technique for evaluating drug efficacy that can be conducted by veterinary practitioners in all host species is the FECRT. In this procedure, the percentage reduction in strongylid fecal egg counts following treatment is calculated to evaluate the efficacy of the product. This test has also been used to evaluate resistance to anthelmintics in equine *Parascaris* spp. infections.

The World Association for the Advancement of Veterinary Parasitology (WAAVP) is an international organization that assembles expert opinion and has issued recommendations for testing and evaluating antiparasiticide efficacy. Updated recommendations for evaluating drug resistance are expected in the near future. Some general

principles for conducting FECRT are presented here. For additional details in conducting these tests several recent publications can be consulted: Kaplan R, M. 2020. Biology, epidemiology, diagnosis, and management of anthelmintic resistance in gastrointestinal nematodes of livestock. *Vet. Clin. North Am. Food Anim. Pract.* 36:17–30, and American Association of Equine Practitioners. *Internal Parasite Control Guidelines*, updated 2019; <https://aaep.org/guidelines/parasite-control-guidelines>.

Although there is some variation in protocols for conducting FECRT, the following general principles are presented followed by comments specific to host species.

### ***Test Groups and Selection of Animals***

An accurate FECRT requires adequate animal numbers. In cases where owners wish to test very small flocks or herds, it is important to be cautious in interpretation of results. For ruminants 15 animals are preferred, but at least 10 should be used for each drug to be tested. For horses, where herd sizes are often low, at least six horses per group is recommended. Where only one or two animals are available an impression of drug efficacy can be obtained following treatment, but it should not be considered a reliable or accurate FECRT. In very large herds, 10% of the total population is adequate.

All animals used in the FECRT should have adequate egg counts to allow reductions to be determined accurately. For sheep, lambs of 3–6 months of age and cattle less than 16 months are the best candidates for a FECRT. Goats of all ages can generally be used. It is best if animals used in the test are similar in age and management. Fecal egg counts in adult cattle usually are too low to be used in an FECRT. It will be helpful to perform the FECRT at the time of year when fecal egg counts are expected to be the highest, based on epidemiology of the parasites in a region.

As a general guideline, the modified McMaster test can be used when the average FEC is 500 EPG with a group size of 10 or 250 EPG with a group size of 20. If average egg counts fall below this level, an alternative test for quantifying parasite eggs must be used to provide an accurate picture of drug efficacy. Alternatives include decreasing the detection limit of the modified McMaster test by using larger sample aliquots (e.g., using larger counting chambers available from several companies,) or using other tests with a lower detection limit (mini-FLOTAC, Stoll, or Wisconsin tests; see section on "Egg-Counting Procedures [Quantitative Fecal Exams]").

An alternative procedure for conducting a FECRT using composite fecal samples is described in Kaplan (2020).

### ***Test Drugs and Collection of Posttreatment Samples***

Once the test group or groups of animals have been established with a pretreatment fecal egg count, each animal should be individually weighed and treated with the test drug at the manufacturer's recommended dose.

On farms where individual animals cannot be weighed, the fecal egg count reduction (FECR) can be approximated by treating all animals with the drug dose for the estimated heaviest animal in the group. The results, however, will not be entirely accurate because some animals will be receiving more than the recommended dose, which may be temporarily effective against worms resistant to the recommended dose. This will lead to an overestimation of drug efficacy.

The optimum time following treatment for collection of posttreatment samples varies with the test drug because of variable effects on larval stages and temporary sterilizing effects on adult parasites. For ruminants, the following intervals are recommended:

- benzimidazoles, levamisole, or pyrantel 10–14 days,
- ivermectin and other avermectins 14–17 days,
- moxidectin 17–21 days.

For convenience in both ruminants and horses, a standard period of 14 days before collection of the posttreatment samples is often recommended.

### **Interpretation of Results**

The % FECR is calculated using arithmetic group means in the following formula:

$$\%FECR = \left[ \frac{\text{pretreatment FEC} - \text{posttreatment FEC}}{\text{pretreatment FEC}} \right] \times 100$$

When small groups of animals are used, individuals with particularly high fecal egg counts can dramatically skew the FECR when group means are used in the calculation. In these cases (e.g., in small groups of horses), it is best to calculate individual FECR using the above formula and then average the individual FECR to obtain a group average.

The FECRT can also be conducted by comparing mean posttreatment EPG in a group of untreated animals with that of a group of treated animals. This procedure is used less often than the comparison of pre- and posttreatment samples of individuals in the same group of animals because more animals are needed and pretreatment egg counts must be similar in the two groups. The formula for calculating FECR is altered accordingly when treatment and control groups are used:

$$\%FECR = \left[ \frac{\text{control FEC} - \text{treatment FEC}}{\text{control FEC}} \right] \times 100$$

If no resistance is present, and the test is administered correctly, anthelmintics in ruminants can reliably decrease fecal egg counts by >95%. For horses, reductions in fecal egg counts when no resistance is present should be >95% for benzimidazoles, >98% for ivermectin and moxidectin, and >90% for pyrantel.

The FECR obtained when testing a group of animals gives an indication of drug efficacy, but is not equivalent to the proportion of the adult worm population that is removed by treatment. For example, if the percentage reduction is 10%, a large proportion of worms in the animals are probably resistant. If the reduction is 80%, the proportion of resistant worms is much smaller, but still significant. To further evaluate which worm genera are resistant in ruminants, the nematode eggs present in both pre- and posttreatment fecal samples can be identified by larval culture (see earlier) or PCR. In horses, drug resistance is most often present in small strongyles (cyathostomins), which cannot be readily differentiated to species based on morphology.

### **IDENTIFICATION OF ADULT WORMS**

Tapeworm segments and adult gastrointestinal nematodes may occasionally be passed in feces and presented for identification by concerned owners. Segments of

common tapeworms can usually be readily identified to the level of genus by shape and identification of eggs in the segments (see Figs. 1.99, 1.102, 1.105, 1.106, and 1.154 for photographs of common tapeworm segments), but nematode parasites may be more difficult to identify. When preserving nematodes for further identification, it is helpful to place them first in tap water and refrigerate the container for several hours. This will relax the worms and make them easier to examine. After relaxation, the worms can be placed in 70% ethanol or 10% buffered formalin. Ethanol, but not formalin, allows later identification with molecular assays. While not optimum preservatives for all helminths, these chemicals are readily available to most veterinarians.

The most common nematodes presented by pet owners are the ascarids (roundworms; see Fig. 1.72). These large, stout-bodied worms are common in feces and vomitus of kittens and puppies. Horse owners might also see equine ascarids that are up to 50 cm (about 20 in.) in length. Smaller nematodes present in equine manure may be the large and small strongyles or pinworms (*Oxyuris*). Larval or adult horse strongyles may be red or cream in color and no more than about 2–4 cm in length (see Fig. 1.168). *Oxyuris*, the equine pinworm, can reach 15 cm, and the females have distinctive long, thin tails (see Fig. 1.174). Nematodes are most likely to be seen in diarrheic feces or following treatment.

Specific identification of adult nematodes is usually based on morphologic variations of the outer layer, or cuticle, of the worms. Microscopic examination of the mouthparts and accessory sexual structures may be required. To enhance visualization of these structures, the worm can be mounted in a clearing solution, which dissolves the soft tissue, leaving only the cuticle. If the worm is large, the areas of diagnostic importance (usually the anterior and posterior ends) can be cut off and mounted in a few drops of the clearing solution. Procedures for making Hoyer's and lactophenol solutions are given below, but they are not usually prepared in veterinary practices because they require either controlled or hazardous substances. Both are commercially available.

Depending on the parasite species, accurate worm identification may require the evaluation of subtle morphologic characteristics that will be unfamiliar to most practicing veterinarians. When specific parasite identification is needed, worms should be submitted to a parasitologist for examination.

### Hoyer's Solution

Hoyer's solution also provides a permanent mounting medium for specimens, although the clearing process will continue until eventually internal structures will no longer be visible:

- 30 g gum arabic;
- 16 mL glycerol;
- 200 g chloral hydrate;
- 50 mL distilled water.

Dissolve the gum arabic in water with gentle heat. Add the chloral hydrate, then the glycerol.

**Lactophenol**

- 20 mL glycerin, pure;
- 10 mL lactic acid;
- 10 mL phenol crystals, melted;
- 10 mL distilled water.

Combine all ingredients.

## PARASITES OF DOMESTIC ANIMALS

The following photographs in this chapter illustrate the diagnostic stages found in feces of a wide variety of both common and some uncommon parasites of major domestic species. Because an appreciation of relative sizes of parasite eggs, cysts, and oocysts, is very helpful in identification, a line drawing precedes sections showing parasites of common mammalian hosts (Figs. 1.39, 1.40, 1.121, 1.122, 1.162, 1.180). Generally, photographs of eggs and cysts were taken using the high-dry (40 $\times$ ) objective, although some photographs using the 10 $\times$  objective are included to show relative sizes of eggs and cysts.

The figures in which each parasite appears are listed after the name. They may include figures in other sections where more than one parasite is illustrated.

An effort has been made to minimize taxonomic information while still permitting an appreciation of the larger groups to which each individual species belongs. For more specific taxonomic information, a textbook of veterinary parasitology should be consulted.

Also at the beginning of the sections for dogs and cats, ruminants, horses, and swine are tables showing common U.S. label approved products for treatment of a number of parasitic infections. Label dose and withdrawal information should always be consulted before treatment of animals with parasiticides.

## Dogs and Cats

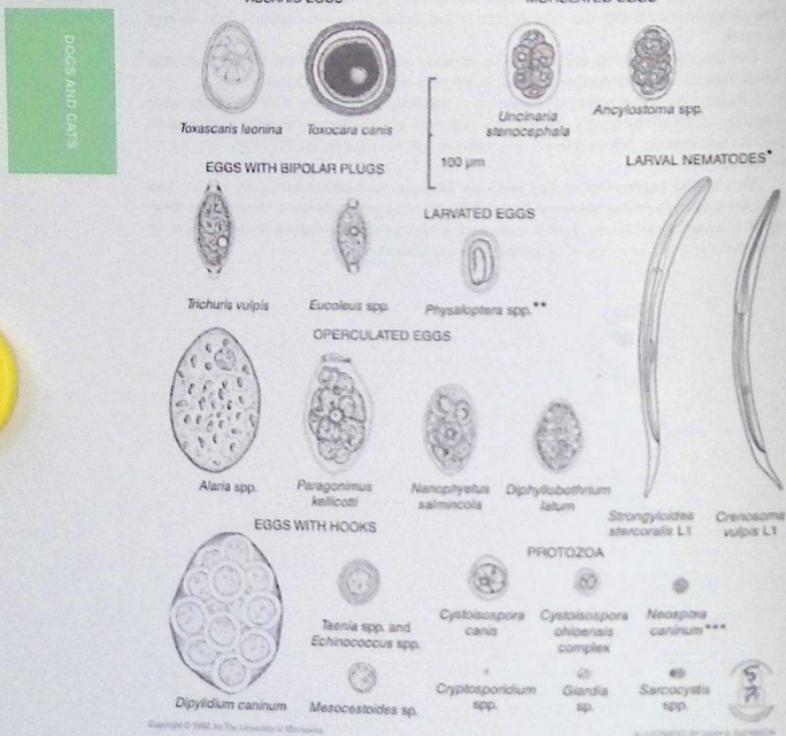
Helminth Eggs, Larvae and Protozoan Cysts  
found in freshly voided feces of the  
Dog, Wolf, Coyote and Fox

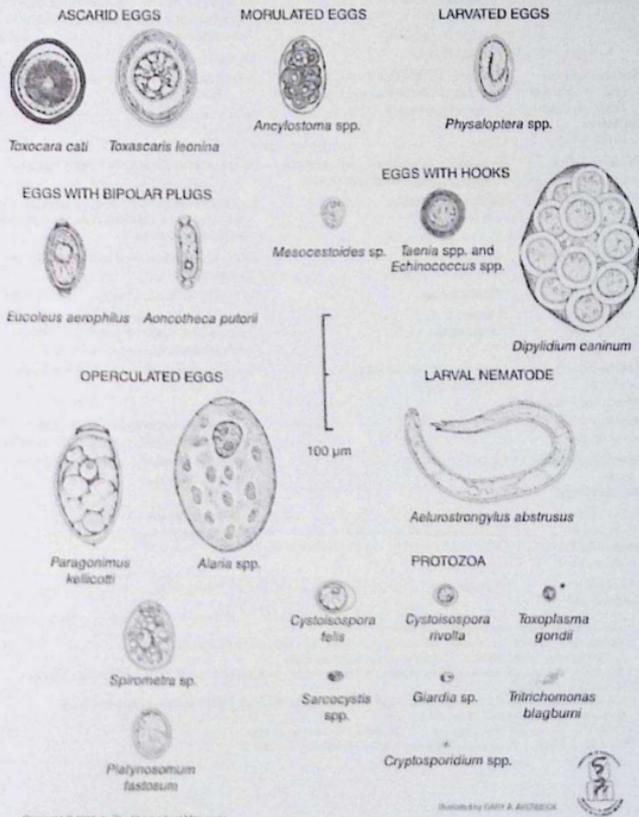
Fig. 135 Parasites found in canine feces. Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

\*Differentiate from other larvae that could be present in canine feces, including *Filaroides*, *Ostertus* and *Angiostrongylus*.

\*\**Spirocerca lupi*, which is common in many parts of the world, has a larvated egg similar in appearance to *Physaloptera*, but more elongated.

\*\*\*Oocysts of *Hammondia heylorni* are similar in appearance to *Neospora*.

## Helminth Eggs, Larvae and Protozoan Cysts found in freshly voided feces of Cats



Copyright © 2002, by The University of Minnesota

Illustration © GARY A. AVERBECK

Fig. 140 Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

\*Oocysts of *Homonidus hammondi* and *Besnoitia* spp. are similar in appearance to *Toxoplasma* oocysts.

Table 15. Representative treatments for selected parasites of dogs

Parasite	Effective treatments	Dose and administration route
<i>Cystoisospora</i> spp.	*Ponazuril, †toltrazuril	10–30 mg/kg orally q 24 h × 1–3 d
	*Sulfadimethoxine	Administer according to label directions
<i>Giardia</i> sp.	*Fenbendazole	30 mg/kg q 24 h × 3 d (combined with praziquantel and pyrantel); avoid in pregnant animals
	*Fenbendazole	50 mg/kg orally q 24 h × 3 d
<i>Ancylostoma</i> spp.	Fenbendazole, fenbendazole, †milbemycin oxime, transdermal moxidectin, pyrantel pamoate	Administer according to label directions
<i>Toxascaris leontina</i>		
<i>Toxocara canis</i>		
<i>Uncinaria stenocephala</i>		
<i>Trichuris vulpis</i>	Fenbendazole, fenbendazole, milbemycin oxime, transdermal moxidectin	Administer according to label directions
<i>Eucoleus</i> spp.	*Milbemycin oxime, †transdermal moxidectin	Extralabel use of label dose of transdermal moxidectin or elevated dose (2 mg/kg) of milbemycin oxime
<i>Physaloptera</i> spp.	*Pyrantel pamoate	20 mg/kg, repeat q 14 d until clinical signs resolve
<i>Spirocerca lupi</i>	*Doramectin	0.4 mg/kg subcutaneously q 7 d × 12 weeks
<i>Strongyloides stercoralis</i>	*Fenbendazole *Ivermectin	50 mg/kg fenbendazole orally q 24 h × 5 d, repeat in 4 weeks; 0.2 mg/kg ivermectin subcutaneously, repeat in 2 weeks
<i>Dipylidium caninum</i>	*Epsiprantel, †fenbendazole, praziquantel	Administer according to label directions
<i>Taenia</i> spp.		
<i>Echinococcus</i> spp.		
<i>Mesocostoides</i> spp.	*Praziquantel	Effective against intestinal stages when administered according to label directions
<i>Alaria</i> spp.		
<i>Diphyllobothrium latum</i>	*Praziquantel	Administer elevated dose (25 mg/kg) for 2 consecutive days
<i>Spirometra</i> spp.		
<i>Heterobilharzia americana</i>	*Fenbendazole	50 mg/kg orally for 10–14 days
<i>Paragonimus kellicotti</i>	*Praziquantel	25 mg/kg every 8 hours for 2–3 days
<i>Nanophyetus salmicosla</i>	*Praziquantel	20–30 mg/kg once

\* Extralabel use supported by published data.

† Label-approved for treating dogs with bacterial enteritis associated with coccidiosis.

\* Longer courses of treatment may be necessary in some patients.

\* Monthly products are label-approved against *Ancylostoma* spp., *Toxocara canis*, and *Toxascaris leontina*, but not

*Uncinaria stenocephala*.

\* Extralabel use of high-dose cattle products can be fatal in dogs, establish MDR1 status prior to treatment.

\* Not label-approved against *Echinococcus* spp.

\* Only effective against *Taenia* spp., not *D. caninum* or *Echinococcus* spp.

Additional information on parasite treatments can be found in Chapter 7.

**Table 1.6. Representative treatments for selected parasites of cats**

Parasite	Effective treatments	Dose, route, and regimens
<i>Cystoisospora</i> spp.	*Ponazuril, toltrazuril *Sulfadimethoxine	10–30 mg/kg orally q 24 h × 1–3 d Administer according to label directions
<i>Trichostrongylus axei</i>	*Ronidazole	30–50 mg/kg orally q 12 h × 14 d; use with caution due to safety concerns
<i>Giardia</i> sp.	*Fenbendazole	30 mg/kg × 3 d (combined with praziquantel and pyrantel); avoid in pregnant animals 50 mg/kg orally q 24 h × 3 d
<i>Ancylostoma</i> spp. <i>Toxocara cati</i>	*Fenbendazole Emodepside, eprinomectin, ivermectin, milbemycin oxime, pyrantel pamoate, transdermal moxidectin, selamectin	Administer according to label directions
<i>Strongyloides</i> spp.	*Fenbendazole *Ivermectin	50 mg/kg fenbendazole orally q 24 h × 5 d, repeat in 4 weeks; 0.2 mg/kg ivermectin subcutaneously, repeat in 2 weeks
<i>Aelurostrongylus abstrusus</i>	*Transdermal moxidectin	Administer according to label directions
<i>Dipylidium caninum</i> <i>Taenia</i> spp. <i>Echinococcus</i> spp.	*Epsiprantel, praziquantel	Administer according to label directions
<i>Mesocystoides</i> spp. <i>Alaria</i> spp.	*Praziquantel	Effective against intestinal stages when administered according to label directions
<i>Diphyllobothrium latum</i>	*Praziquantel	Administer elevated dose (25 mg/kg) for 2 consecutive days
<i>Spirometra</i> spp.		
<i>Paragonimus kellicotti</i>	*Fenbendazole *Praziquantel	50 mg/kg orally for 10–14 days 25 mg/kg every 8 hours for 2–3 days
<i>Platystrongylus concinnum</i>	*Praziquantel	25 mg/kg every 8 hours for 2–3 days

\* Extralabel use supported by published data.

† Longer courses of treatment may be necessary in some patients.

‡ Monthly product is only label-approved against *Ancylostoma* spp., not *Toxocara cati*.

§ Extralabel use of high-dose cattle products can be fatal in cats; use with particular caution in young or debilitated patients.

¶ Not label-approved against *Echinococcus* spp.

Additional information on parasite treatments can be found in Chapter 7.

### Protozoan Parasites

**Parasite:** *Cystoisospora (Isospora) spp.* (Figs. 1.4–1.6, 1.41–1.45, 1.47, 1.70, 1.78, 1.101)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia). Several host-specific species are found in the dog (*C. canis*, *C. ohioensis*, *C. neurivolta*, *C. burrowsi*) and cat (*C. felis*, *C. rivolta*).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine, cecum, and colon.

**Life Cycle:** Cats and dogs are infected by ingestion of sporulated oocysts or infected transport hosts (often rodents, but also including rabbits, ruminants, birds, and other prey animals). Following development in the final host, oocysts are passed in feces and undergo sporulation in the environment.

**Laboratory Diagnosis:** Oocysts are detected by fecal flotation examination. Oocysts have smooth, clear cyst walls, are elliptical in shape, and contain a single, round cell (sporoblast) when freshly passed. The oocysts of *C. ohioensis*, *C. burrowsi*, and *C. neurivolta* are not morphologically distinguishable and are referred to as the *C. ohioensis* complex.

Size:	<i>C. canis</i> , <i>C. felis</i>	38–51 × 27–39 μm
	Other <i>Cystoisospora</i> spp.	17–27 × 15–24 μm

**Clinical Importance:** These are the organisms typically referred to as “coccidia” of dogs and cats, although other parasites also fall into this taxonomic group. Oocysts can be found in the feces of many clinically normal young dogs and cats. Clinical coccidiosis most often occurs in puppies and kittens, often in association with weaning, change of owner, or other stress factors. Signs include diarrhea, abdominal pain, anorexia, and weight loss. In severe cases, bloody diarrhea and anemia may occur. Respiratory and neurologic signs have also been reported in some animals. Clinical disease has been difficult to reproduce in experimental infections.

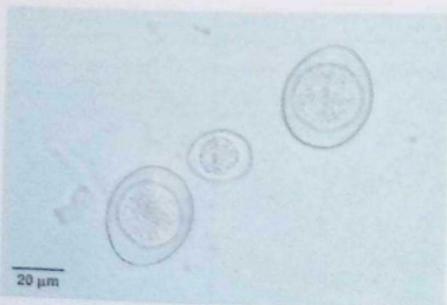


Fig. 1.41 Dog and cat coccidia species produce oocysts of different sizes. This figure shows *C. canis* (larger oocysts) and an oocyst of the *C. ohioensis* complex (smaller oocysts). Photo courtesy of Dr. David Lindsay, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.



Fig. 142 *Cystoisospora* oocysts usually require a minimum of 1–2 days to become infective for the next host (sporulated). In warm conditions, oocysts undergo the first cell division soon after being passed in the feces. In the two-cell stage, they may be mistaken for sporulated oocysts. In this fecal sample, a sporulated oocyst (arrow) is adjacent to one in the two-cell stage.



Fig. 143 A sporulated *Cystoisospora* oocyst contains two sporocysts, each containing four sporozoites. The two sporocysts can be seen in this oocyst, although only two of the four sporozoites can be visualized in each sporocyst. A large, round residual body is also present in each sporocyst.



Fig. 144 *Cystoisospora* oocyst and two iodine-stained *Giardia* cysts (arrows) in a canine fecal sample. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

Parasite: *Toxoplasma gondii*, *Neospora caninum* (Fig. 1.47)

Taxonomy: Protozoa (coccidia).

Geographic Distribution: Worldwide.

Location in Host: Intestine and other tissues of cats and other felids (*Toxoplasma*) and dogs and other canids (*Neospora*).

Life Cycle: *Toxoplasma* is transmitted to cats by ingestion of cysts containing bradyzoites in tissues of intermediate hosts. Prenatal and transmammary transmission as well as direct transmission through ingestion of sporulated oocysts can also occur. Transmission of *Neospora* in dogs appears to be similar to *Toxoplasma* transmission.

Laboratory Diagnosis: Oocysts are detected in feces by centrifugal or simple flotation techniques. However, very few oocysts of *Neospora* appear to be produced in infected dogs. Immunodiagnostic tests are available to identify current and past exposure to *Toxoplasma* in cats but are usually not positive until after fecal passage of oocysts has ceased. Dogs can also be tested for antibody to *Neospora*. The small, spherical-shaped oocysts of the two genera are morphologically identical, have a clear smooth cyst wall, and contain a single round sporoblast. Some other coccidia genera, including *Hammondia*, produce similar oocysts, which precludes definitive identification of *Toxoplasma* or *Neospora* on the basis of oocyst presence alone.

Size: 11–14 × 9–11 μm

Clinical Importance: *Toxoplasma* infections in cats are generally well tolerated. Clinical disease (ocular, respiratory, etc.) can occur in cats, especially young or immunosuppressed animals. Toxoplasmosis is an important zoonotic disease with especially serious consequences in pregnant women and the immunosuppressed. Congenital *Neospora* infection can result in severe central nervous system disease in dogs. *Neospora* infection is also an important cause of abortion in the bovine intermediate host.



Fig. 145 *Cystoisospora canis* oocysts in this canine fecal sample (arrows) are similar to the larger *Toxascaris leonina* egg (also Fig. 1.65), but the oocysts are smaller and lack the membranous appearance of the inside of the shell seen in *Toxascaris* eggs.



Fig. 146 *Eimeria* spp. oocysts are sometimes seen in dog and cat feces. *Eimeria* does not infect these hosts, but oocysts consumed as a result of predation or coprophagy will pass unharmed through the gastrointestinal tract and may be misidentified as *Cystoisospora*. Many (but not all) *Eimeria* oocysts have a knob at one end called the micropyle cap (arrow), whereas *Cystoisospora* spp. lack a cap. If this cap is present, an oocyst in dog or cat feces can be identified as a "spurious parasite."

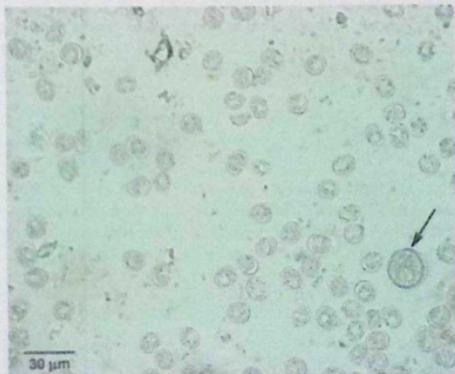


Fig. 147 *Neospora* and *Toxoplasma* oocysts are similar to common *Cystoisospora* spp., but they are smaller. The oocysts of these two coccidia genera are similar in appearance and also cannot be distinguished from oocysts of *Hammondia*, another coccidia genus of small animals. This photo of a felina fecal sample also shows an oocyst of *C. risolia* (arrow).

**Parasite:** *Sarcocystis* spp. (Figs. 1.48 and 1.49)

**Taxonomy:** Protozoa (coccidia). A number of species infect dogs or cats, each with a specific intermediate host.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs and cats.

**Life Cycle:** Cat and dog definitive hosts are infected by ingesting intermediate host tissue containing sarcocysts. Sexual reproduction in dogs or cats leads to formation of oocysts that sporulate while still in the intestinal tract.

**Laboratory Diagnosis:** Oocysts form within the gastrointestinal tract of dogs and cats. The oocyst wall breaks down in the gut, and small, ellipsoidal sporulated sporocysts are released in the feces. They are detected by centrifugal or simple flotation techniques.

**Size:** 7–22 × 3–15  $\mu$ m

**Clinical Importance:** *Sarcocystis* is generally nonpathogenic in the definitive host, although some species can cause severe disease in the intermediate host (cattle, sheep, pigs, horses).

**Parasite:** *Cryptosporidium* spp. (Fig. 1.50)

**Taxonomy:** Protozoa (coccidia). *Cryptosporidium felis* and *C. canis* appear to be the primary species infecting cats and dogs, respectively.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** These parasites have a direct life cycle. Cats and dogs are infected following ingestion of oocysts, which are infective as soon as they are passed in the feces. Following asexual and sexual multiplication of the organism in the intestine, oocysts are produced and exit the host in the feces.

**Laboratory Diagnosis:** Small oocysts in the feces are detected by use of acid-fast or other stains of fecal smears, Sheather's sugar flotation test, fecal antigen tests, or molecular diagnostic procedures. Oocysts of *C. parvum* and *C. canis* are morphologically indistinguishable, while *C. felis* oocysts are smaller than those of the other two species.

<b>Size:</b>	<i>C. felis</i>	3.5–5 $\mu$ m in diameter
	<i>C. parvum</i> , <i>C. canis</i>	7 × 5 $\mu$ m

**Clinical Importance:** Cryptosporidiosis has been reported as an uncommon cause of chronic diarrhea in cats. Affected cats are often immunosuppressed by other causes. Although implicated in rare instances, *Cryptosporidium* infections in dogs and cats do not appear to be a significant source of zoonotic exposure for humans.



Fig. 148 *Sarcocystis* sporocysts are smaller than typical coccidia oocysts and have a smooth, clear cyst wall. Each sporocyst contains four banana-shaped sporozoites. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

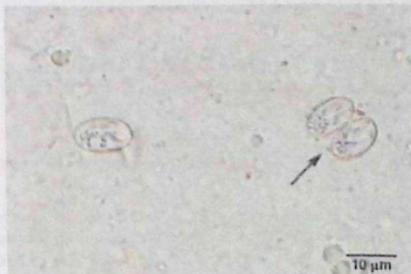


Fig. 149 *Sarcocystis* sporulates in the intestines and the oocyst wall usually ruptures before exiting the body so that only sporocysts are seen. Rarely, intact *Sarcocystis* oocysts are present (arrow). The 2 sporocysts appear to be surrounded by a thin membrane. Figure courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

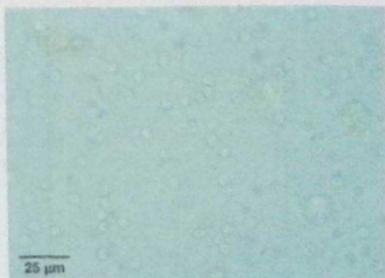


Fig. 150 *Cryptosporidium* sp. in a sugar flotation preparation. The oocysts of *C. canis* and *C. felis* are difficult to distinguish by microscopic techniques; *C. felis* oocysts are slightly smaller in size. *Cryptosporidium* oocysts can also be detected with acid-fast stains and immunodiagnostic tests.

**Parasite:** Trichomonads (Figs. 1.51 and 1.52)

**Taxonomy:** Protozoa (flagellate). Species identification of these organisms in dogs and cats is currently under investigation. *Tritrichomonas blagburni* has recently been identified as a species of feline trichomonad.

**Geographic Distribution:** Probably worldwide.

**Location in Host:** Large intestine of cats and dogs.

**Life Cycle:** Very little is known about transmission. Infection is probably by direct contact since no environmentally resistant cyst stage is known to occur.

**Laboratory Diagnosis:** The presence of trophozoites can be detected in direct saline smears of fresh feces. Flotation solutions will destroy trophozoites. Trichomonad organisms can be confused with *Giardia* but have an undulating membrane and lack the facelike appearance of *Giardia*. For detection of *T. blagburni* in the United States, the InPouch™ TF-Feline culture system, similar to the method used for bovine *T. foetus* infections in cattle (see Chapter 2), can be used. Commercial PCR tests are also available.

Size:  $6-11 \times 3-4 \mu\text{m}$

**Clinical Importance:** Cases of chronic diarrhea in cats have been associated with feline *T. blagburni* infection; however, most trichomonad infections are generally considered to be of limited pathogenicity.

**Parasite:** *Giardia duodenalis* (= *G. intestinalis*, *G. lamblia*, *G. canis*, *G. cati*, etc.) (Figs. 1.44, 1.53-1.59)

**Taxonomy:** Protozoa (flagellate). Species number and nomenclature are under investigation. Molecular analysis is used to allocate isolates into assemblages. Most isolates from dogs belong to Assemblages C and D, and most cat isolates to Assemblage F.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs, cats, many other animals, and humans.

**Life Cycle:** Dogs and cats are infected by ingesting cysts in the environment. Trophozoites are stimulated under certain conditions to encyst and are passed from the host in feces.

**Laboratory Diagnosis:** The preferred flotation procedure for cyst detection is centrifugal flotation with 33% ZnSO<sub>4</sub> solution; other flotation solutions may cause rapid distortion. To improve sensitivity of testing, centrifugal flotation combined with a *Giardia* fecal antigen test is recommended. Trophozoites are infrequently seen in a direct saline smear of fresh diarrheic feces or duodenoscopic aspirates. Examination for trophozoites should occur within 30 minutes of collection or refrigeration. Commercial PCR and indirect fluorescent antibody (IFA) tests are also available.

• Size:	Cyst	$9-13 \times 7-9 \mu\text{m}$
	Trophozoite	$12-17 \times 7-10 \mu\text{m}$

**Clinical Importance:** *Giardia* is a common parasite of small animals. Many infections are asymptomatic, but acute, chronic, or intermittent diarrhea may occur, particularly in young dogs and cats. Although a low-risk zoonosis, cats and dogs are infrequently infected with assemblages associated with humans.

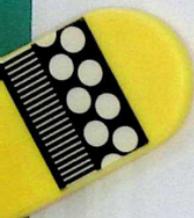




Fig. 1.51 Trichomonad parasites in dogs and cats have a distinctive undulating membrane. In fresh saline smears, they are most easily confused with *Giardia* trophozoites, but trichomonads lack the face-like appearance and the concave ventral surface of *Giardia*. In addition, the movement of *Giardia* trophozoites is usually described as "leaflike," while trichomonads movement is more jerky.

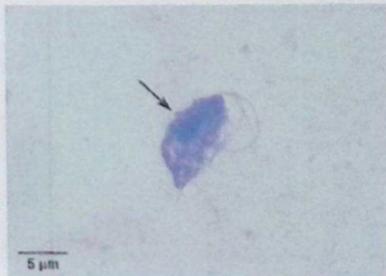


Fig. 1.52 Stained fecal smear of a trichomonad organism showing the anterior flagella and part of the undulating membrane (arrow).

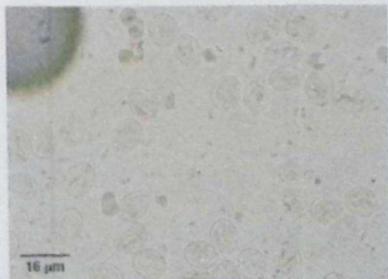


Fig. 1.53 *Giardia* cysts recovered with 33%  $ZnSO_4$  centrifugal flotation. Cysts are elliptical with a thin, smooth cyst wall and contain two to four nuclei, two slender, linear intracytoplasmic flagella, and two thick, comma-shaped median bodies.

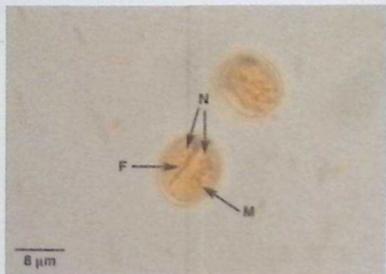


Fig. 154 A drop of Lugol's iodine may be added to a flotation preparation to stain *Giardia* cysts and make internal structures more prominent. Two nuclei (N), intraflagellarium (F), and median bodies (M) can be seen in the cyst shown here.



Fig. 155 *Giardia* cysts undergo osmotic damage when exposed to high specific gravity. With time, increasing numbers of cysts appear vacuolated, with a characteristic half-moon shape. Plant pollen and yeast cells that mimic *Giardia* cysts do not undergo this same artifact change.

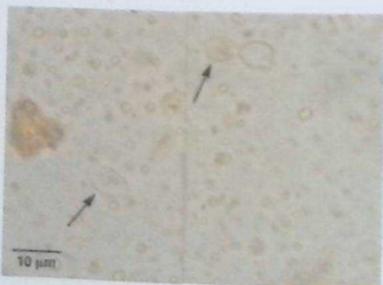


Fig. 156 The structures most often confused with *Giardia* cysts are yeast (arrows), which may be found in diarrheic feces in large numbers. Yeast are commonly slightly smaller than *Giardia* cysts and lack the complex internal structure seen in *Giardia* cysts.



Fig. 157 *Giardia* trophozoites in a direct saline smear. Trophozoites are bilaterally symmetrical and pyriform shaped with two nuclei, eight flagella, two rodlike median bodies, and a ventral, concave, adhesive disk that gives them a clown face.

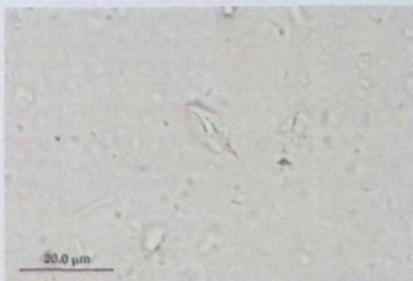


Fig. 158 Unstained *Giardia* trophozoite in a direct saline smear. The concave sucking disc is distinctive on the lateral view. Live trophozoites have a characteristic wobbling motion when swimming (often described as looking like a falling leaf) and are easily kept in the microscopic field of view.



Fig. 159 *Giardia* trophozoite (arrow) associated with a portion of mucosa in a duodenal aspirate. Aspirates should be examined within 30 minutes of collection, before the fragile trophozoites die.

## Helminth Parasites

**Parasite:** *Ancylostoma* spp., *Uncinaria stenocephala* (Figs. 1.4–1.6, 1.60, 1.61, 1.70, 1.71, 1.76)

Common name: Hookworm.

**Taxonomy:** Nematodes (order Strongylida).

**Geographic Distribution:**

*Ancylostoma caninum* (dogs) and *A. tubaeformae* (cats): worldwide.

*A. braziliense* (dogs and cats): tropical and subtropical distribution; in the United States found primarily in the Gulf Coast region.

*A. ceylanicum* (dogs and cats): various parts of Asia.

*Uncinaria stenocephala* (dogs, rarely cats): primarily cooler northern temperate regions, including the northern United States, Canada, and Europe.

**Location in Host:** Small intestine of dogs and cats and wild canids and felids.

**Life Cycle:** Transmission of *A. caninum* to dogs occurs by transmammary transmission and direct skin penetration by infective larvae, ingestion of infective larvae from the environment or in paratenic hosts. Transmission of *U. stenocephala* occurs by ingestion of infective larvae or paratenic hosts, direct skin penetration is rare. Cats can be infected with hookworms either by skin penetration, by ingestion of infective larvae, or in paratenic hosts (rodents). Adult hookworms produce eggs that exit the host in the feces.

**Laboratory Diagnosis:** Eggs are detected using centrifugal or simple flotation techniques. *Ancylostoma* and *Uncinaria* eggs are morphologically identical, with an elliptical shape and smooth shell wall containing a grape-like cluster of cells (morula). However, they differ in size.

Size:	<i>Ancylostoma</i> spp.	52–79 × 28–58 μm
	<i>U. stenocephala</i>	71–92 × 35–58 μm

**Clinical Importance:** *Ancylostoma caninum* is common in North America. In heavy infections, particularly in puppies, the blood-feeding behavior of hookworms can cause fatal anemia. Peracute, acute, and chronic disease syndromes may occur. *Ancylostoma braziliense* and *A. caninum* may cause cutaneous larva migrans and, rarely, eosinophilic enteritis in humans. *Ancylostoma* infections in cats are less common than in dogs. Many feline infections are subclinical, but heavy infections causing anemia and weight loss can be fatal. *Uncinaria stenocephala* is less pathogenic than *A. caninum*.



Fig. 160 Although *Ancylostoma* is the most common genus of hookworm in the United States, slightly larger *Uncinaria* eggs may also be seen in canine feces, and mixed infections can occur, as shown here.



Fig. 161 *Ancylostoma tubaeforme* larvated and undeveloped eggs from a cat. The egg on the right contains a developed first-stage larva; the egg on the left is undifferentiated. Hookworm eggs exposed to warm temperatures for several hours rapidly develop to the larvated stage and hatch.

**Parasite:** *Mammomonogamus* spp. (Figs. 1.62, 1.76)

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Caribbean, Asia.

**Location in Host:** Nares and nasopharynx primarily.

**Life Cycle:** The life cycle has not been completely described. Infection probably follows ingestion of infective larvae.

**Laboratory Diagnosis:** Centrifugal fecal flotation with solutions of  $\geq 1.25$  SPG will detect eggs. Mucus surrounding eggs in nasal discharge samples may inhibit flotation. Mucus can be removed by pretreatment with 5% potassium hydroxide followed by repeated sedimentation in water.

Size:  $90\text{--}133 \times 54\text{--}88 \mu\text{m}$

**Clinical Significance:** Asymptomatic infection in cats. Human cases have been reported.

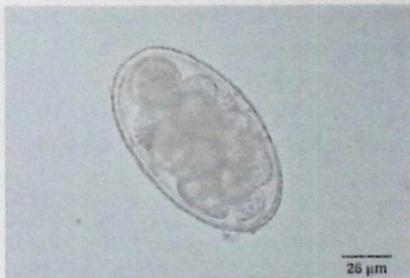


Fig. 162 Egg of *Mammonomonogamus* from a cat. This egg is most likely to be seen in cats from Caribbean islands or parts of Asia. The shell wall has subtle striations and is slightly thicker than that of a hookworm egg. Photo courtesy of Dr. Jennifer Ketzis, School of Veterinary Medicine, Ross University, St. Kitts, WI.

**Parasite:** *Toxocara* spp. (Figs. 1.4–1.6, 1.18, 1.63–1.65, 1.67–1.74, 1.101, 1.103, 1.109)

Common name: Roundworm.

**Taxonomy:** Nematode (order Ascaridida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs (*T. canis*) and cats (*T. cati*).

**Life Cycle:** Single-celled eggs pass from the host in the feces and develop to the infective stage in the environment. Dogs acquire infections of *T. canis* by transplacental and transmammmary transmission or by the ingestion of larvated eggs or paratenic hosts (rodents). Cats acquire *T. cati* infection by ingestion of larvated eggs or paratenic hosts. Transmammmary transmission may also occur in some circumstances.

**Laboratory Diagnosis:** Eggs are detected using centrifugal or simple flotation examination techniques. *Toxocara* eggs have a dark, round, single-celled embryo contained in a thick shell wall. Eggs of the two species may be difficult to differentiate. *Toxocara canis* tends to be subspherical, and *T. cati* tends to be elliptical in shape.

Size:	<i>T. canis</i>	85–90 × 75 μm
	<i>T. cati</i>	71–75 × 61–65 μm

**Clinical Importance:** *Toxocara* is an important pathogen in puppies and kittens. Stillbirths, neonatal deaths (*T. canis*), or chronic ill-thrift (*T. canis*, *T. cati*) can occur in infected animals. Adult dogs and cats are much less likely to have symptomatic infections. Additionally, both species have zoonotic importance as causes of visceral and ocular larva migrans, particularly in children.

**Parasite:** *Toxascaris leonina* (Figs. 1.45, 1.65, 1.73, 1.74)

Common name: Roundworm.

**Taxonomy:** Nematode (order Ascaridida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs, cats, wild canids and felids.

**Life Cycle:** The life cycle is similar to that of *Toxocara* spp., although there is no transmammmary or transplacental infection. Dogs and cats are infected following ingestion of larvated eggs or animals with encysted larvae in their tissues (rodents, rabbits).

**Laboratory Diagnosis:** Eggs are detected using centrifugal or simple flotation fecal examination techniques. *Toxascaris* eggs are elliptical with a thick, smooth outer shell wall containing a light-colored, single-celled embryo. The internal surface of the shell wall appears rough or wavy due to the vitelline membrane.

Size: 75–85 × 60–75 μm

**Clinical Importance:** *Toxascaris* is much less common in dogs and cats than *Toxocara* and is considered to be of minor clinical significance.

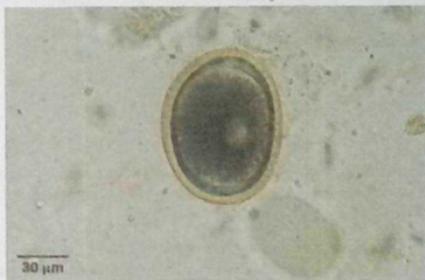


Fig. 1.63 *Toxocara* eggs are typical ascarid eggs with a thick shell. They contain a single cell when first passed in host feces.



Fig. 1.64 When the microscope is focused on the surface of a *Toxocara* egg, the rough, pitted shell-wall surface has a golf-ball-like appearance.



Fig. 1.65 *Toxascaris leonina* egg and *Toxocara cati* eggs. Note the dark single-cell embryo and rough mammillated (pitted) outer shell-wall surface of the *Toxocara* egg contrasted to the lighter appearance of the embryo and to the smooth outer shell-wall surface of the *Toxascaris* egg.

**Parasite:** *Baylisascaris procyonis* (Figs. 1.66–1.68, 1.74)

Common name: Raccoon roundworm.

**Taxonomy:** Nematode (order Ascaridida).

**Geographic Distribution:** Parts of North America and Europe.

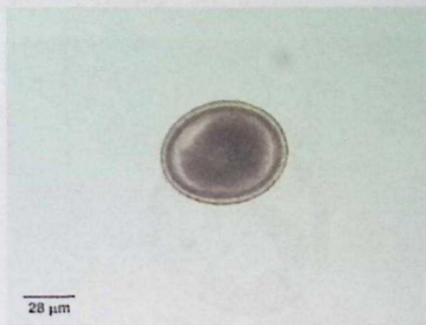
**Location in Host:** Small intestine of raccoons and occasionally dogs.

**Life Cycle:** Raccoons are infected by ingestion of infective eggs or paratenic hosts (rodents, rabbits, birds). Routes of infection in dogs are presumed to be the same. Dogs may also pass *Baylisascaris* eggs following coprophagy.

**Laboratory Diagnosis:** Eggs are detected by simple or centrifugal fecal flotation examination. Eggs are thick walled, elliptical in shape, and contain a single, large, rounded embryo. The eggs are often covered with a brown proteinaceous substance and have a fine granular shell-wall surface.

Size: 63–75 × 53–60 μm

**Clinical Importance:** Infection is well tolerated in the raccoon definitive host. Severe central nervous system or ocular disease can result when birds, rabbits, rodents, marsupials, and humans ingest infective *B. procyonis* eggs. Infections in dogs may result in either patent adult worms in the small intestine or larval tissue migration causing central nervous system disease.



**Fig. 1.66** *Baylisascaris procyonis* egg in feces of a naturally infected dog. Usually, *Baylisascaris* eggs are brown due to the presence of a protein coat (see Fig. 1.67). When lacking the protein coat, *Baylisascaris* eggs are easily misidentified as *Toxocara* eggs. *Baylisascaris* eggs can be differentiated from those of *Toxocara* based on their smaller size and granular shell-wall surface rather than the pitted surface of *Toxocara*.



**Fig. 1.67** *Toxocara canis* egg and *Baylisascaris procyonis* egg (arrow). Eggs of *B. procyonis* appear in the feces of dogs due to either patent infections or coprophagy. The larger *Toxocara* egg ( $85\text{--}90 \times 75 \mu\text{m}$ ) has a rough, pitted, outer shell-wall surface. The *B. procyonis* egg is smaller, has a finely granular shell-wall surface, and may be brown in color. *Baylisascaris* eggs are easily misidentified as *Toxocara* in canine fecal exams. Mistakes can be minimized with the use of an ocular micrometer to measure egg size.



**Fig. 1.68** *Toxocara* (Ta), *Toxascaris* (Ts) and *Baylisascaris* (B) eggs in a canine fecal sample. *Toxascaris* eggs have a smooth outer layer on the egg shell in contrast to the other two species.



Fig. 1.66 *Toxocara* and other ascarids typically require several weeks of development in the environment before an infective larva forms, although initial larval development can occur in a few days in hot weather.



Fig. 1.70 Canine fecal sample containing *Toxocara canis* (T) and *Ancylostoma* (A) eggs and a *Cystoisospora* oocyst (C).

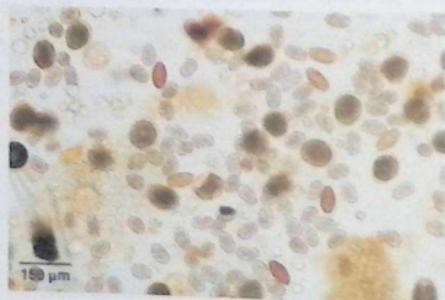


Fig. 1.71 *Toxocara*, *Ancylostoma*, and *Trichuris* eggs in a canine fecal sample. These are the most common intestinal helminths encountered in dogs. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

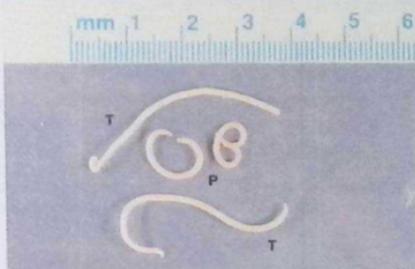


Fig. 172. Ascarids are often passed in the feces or vomitus of dogs and cats, particularly in young or recently dewormed animals. They are the only large, thick-bodied nematodes commonly seen by owners. Rarely, the stomach worm *Physaloptera* is present in vomitus. It usually assumes a C shape when passed out of the host. Two specimens each of *Toxocara* (T) and *Physaloptera* (P) are shown here.

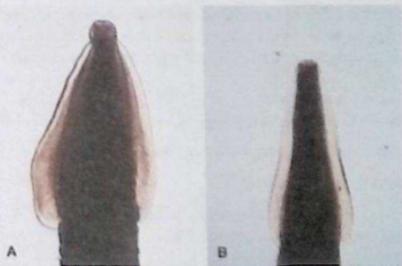


Fig. 173. The morphology of the anterior end of small animal ascarids can be used to differentiate species. The anterior end of adult *Toxocara cati* has an "arrowhead" appearance due to the cervical alae (winglike expansions of the cuticle). Adult specimens of *Toxocara cati* (A) recovered from the feces or vomitus of cats can be differentiated from *Toxascaris leonina* (B), which has less prominent cervical alae.



Fig. 174. The cervical alae of adult *Toxocara canis* (A) are similar in appearance to those of *Toxascaris leonina*. In contrast to the other ascarids infecting dogs (*Toxocara*, *Toxascaris*), adult *Baylisascaris* (B) lack visible cervical alae.

**Parasite:** *Trichuris vulpis* (Figs. 1.71, 1.75, 1.76, 1.78, 1.79, 1.116)

Common name: Whipworm.

**Taxonomy:** Nematode (order Enoplida).

**Geographic Distribution:** Worldwide. *Trichuris felis (serrata, campanula)* occurs in cats in South America, Caribbean, semi-tropical United States (Florida), Australia.

**Location in Host:** Cecum and large intestine.

**Life Cycle:** Dogs are infected by ingesting infective eggs in the environment. Eggs are produced by adult worms in the large bowel and, after leaving the host in the feces, develop to the infective stage in the environment.

**Laboratory Diagnosis:** Eggs are best detected by centrifugal flotation and less effectively by simple flotation examination of feces. Eggs are typically symmetrical about the bipolar plugs, barrel-shaped, and brown. The shell-wall surface is smooth.

Size: 72–90 × 32–40 μm

**Clinical Importance:** Heavy infection in dogs can cause weight loss, unthriftiness, and profuse diarrhea that may be bloody. Resultant electrolyte imbalance can mimic endocrine disease. Infection with feline whipworm is considered rare, although on some Caribbean islands prevalence is high. Clinical signs are often absent.

**Parasite:** *Eucoleus (= Capillaria) aerophilus, E. boehmi* (Figs. 1.77–1.81, 1.101)

Common name: Fox lungworm (*E. aerophilus*).

**Taxonomy:** Nematode (order Enoplida).

**Geographic Distribution:** *Eucoleus aerophilus* is found worldwide; *E. boehmi* has been reported from North and South America and Europe.

**Location in Host:** Trachea, bronchi, and bronchioles of dogs, cats, and foxes (*E. aerophilus*). Epithelium of nasal turbinates and sinuses of dogs and wild canids (*E. boehmi*).

**Life Cycle:** The definitive host is probably infected by ingestion of eggs containing infective larvae, although an earthworm intermediate host may be involved.

**Laboratory Diagnosis:** Eggs are detected by fecal flotation tests or in tracheal or nasal mucus samples. The eggs are clear to golden (*E. boehmi*) or brownish green (*E. aerophilus*), are bipolar plugged, tend to be asymmetrical in shape, and contain a multicelled embryo.

Size:	<i>E. aerophilus</i>	58–79 × 29–40 μm
	<i>E. boehmi</i>	54–60 × 30–35 μm

**Clinical Importance:** *Eucoleus aerophilus* infections in dogs and cats are usually subclinical; in some cases, chronic cough occurs. It is an important pathogen of farmed foxes. *Eucoleus boehmi* infections are usually subclinical. Clinical signs include sneezing and a mucopurulent nasal discharge that may contain blood.



Fig. 175 The egg of *Trichuris vulpis* with its prominent bipolar plugs is one of the most common found in canine feces. Capillariid species also produce eggs with polar plugs. Also shown in this photo are *Monocystis* cysts (M), a spurious protozoan parasite of earthworms. Although cysts are similar in shape to whipworm eggs, they are far too small to be confused with helminth eggs. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

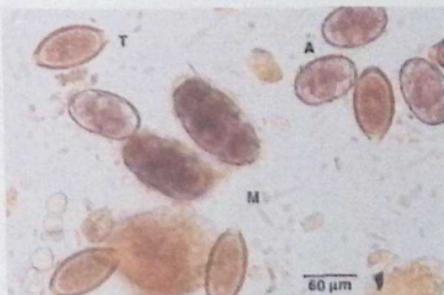


Fig. 176 Feline fecal sample containing eggs of *Ancylostoma* (A), *Mammomonogamus* (M), and *Trichuris* (T). While feline *Trichuris* eggs are similar to those of *T. vulpis*, they are generally smaller. Photo courtesy of Dr. Jennifer Ketzis, School of Veterinary Medicine, Ross University, St. Kitts, WI.



Fig. 177 *Eucolus* eggs are passed in the undifferentiated one- or two-celled stage. *Eucolus boehmi* (right) and *E. aerophilus* (left) eggs are similar in appearance, although in fresh feces, eggs of *E. boehmi* already contain a morula (cluster of cells) that does not completely fill the interior of the egg. Differences in the eggshell can also be used to differentiate the species (see Figs. 1.80 and 1.81). Photo courtesy of Dr. Robert Rudley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

**Parasite:** *Aonchotheca* (= *Capillaria*) *putorii* (Fig. 1.82)

**Taxonomy:** Nematode (order Enoplida).

**Geographic Distribution:** North America, Europe, and New Zealand,

**Location in Host:** Small intestine and stomach of cats, raccoons, foxes, and wild felids and mustelids.

**Life Cycle:** Definitive hosts are infected following ingestion of larvated eggs. Adults develop in the gastrointestinal tract and produce eggs that are passed in the feces.

**Laboratory Diagnosis:** Eggs are detected by centrifugal or simple flotation tests. The yellow-gray eggs are asymmetrical about the bipolar plugs. The sides of the eggs tend to be parallel. The shell-wall surface has a network of deep longitudinal ridges.

Size: 56–72 × 23–32 μm

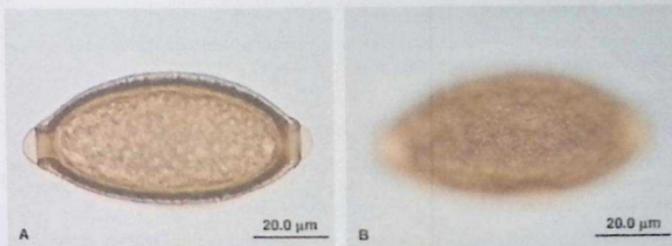
**Clinical Importance:** Infections in cats are usually subclinical and are uncommon in North America. Gastritis with vomiting can occur. Hemorrhagic enteritis can occur in mink.



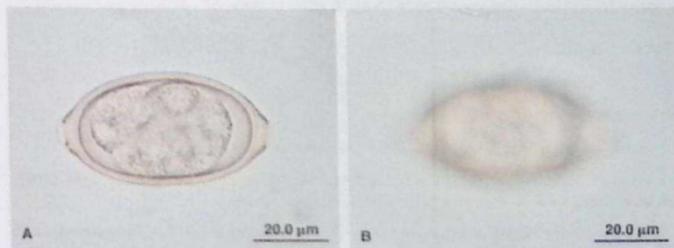
Fig. 1.78 The only canine parasite eggs that could be mistaken for whipworm eggs belong to the capillariid parasites *Euceleus* and *Aonchotheca*. *Trichuris vulpis* eggs (T) are larger, have a smooth-walled shell, and are usually browner than capillariid eggs (E). In cats, *Trichuris* eggs are smaller, and other characteristics should be used to differentiate them from capillariids. *Cytosospora* oocysts are also present in this sample.



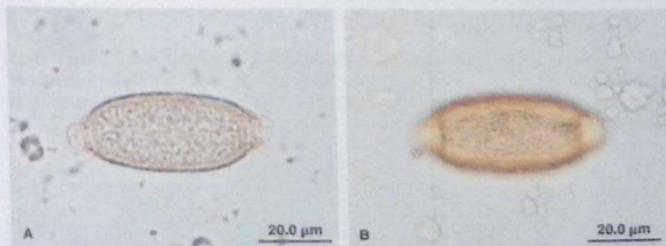
Fig. 1.79 Portion of a *Trichuris vulpis* egg (A) demonstrating the ridges seen in the bipolar plug (arrow). Capillariid eggs (B) lack ridges on the plugs. Capillariid eggs are also smaller than whipworm eggs and their bipolar plugs often appear asymmetrical.



**Fig. 180** Examination of the surface of the shell wall of small-animal capillarid eggs can be used in making a specific identification. The images on this page were taken with an oil immersion lens (100 $\times$ ). However, even with the high-dry objective (40 $\times$ ) of the microscope differences in the surface of the egg can be seen. The surface of the *Eucoleus aerophilus* egg (A) has a network of interconnecting ridges (B).



**Fig. 181** In addition to having a morula that does not fill the interior of the egg, the surface of *Eucoleus boehmi* eggs (A) is pitted, resulting in a stippled appearance (B).



**Fig. 182** Eggs of *Aonchotheca putorii* are similar to those of other capillarids (A), but in contrast to the ridges of the *Eucoleus aerophilus* egg, the shell surface of *A. putorii* consists of a network of deep longitudinal ridges (B).

**Parasite:** *Physaloptera* spp. (Figs. 1.72, 1.83, 1.84)

**Taxonomy:** Nematode (order Spirurida). Several species have been described (*P. praeputialis*, *P. felidis*, *P. pseudopraeputialis*, *P. rara*, *P. canis*).

**Geographic Distribution:** Worldwide.

**Location in Host:** Stomach of dogs, cats, and various wild animals.

**Life Cycle:** Cockroach, beetle, or cricket intermediate hosts ingest eggs shed in feces of wild animals, dogs or cats. The definitive host is infected by ingesting the insect intermediate host or a paratenic host (reptiles and possibly other animals).

**Laboratory Diagnosis:** Eggs of this group of nematodes are not reliably detected by fecal flotation due to their density. *Physaloptera* eggs are best detected by fecal sedimentation. The eggs are clear and elliptical, have a smooth shell wall, and contain a larva coiled inside.

**Size:** 42–53 × 29–35 μm

**Clinical Importance:** Infections may result in clinical signs of vomiting and anorexia.

**Parasite:** *Spirocera lupi* (Fig. 1.85)

**Common name:** Esophageal worm.

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** Worldwide but primarily in warmer regions.

**Location in Host:** Adults are found in the wall of the esophagus, stomach, and, rarely, aorta of dogs, wild canids, and various other wild animals.

**Life Cycle:** Dung beetle intermediate hosts ingest eggs in feces. The definitive host is infected by ingesting the insect intermediate host or a paratenic host (rodents, other mammals, birds, reptiles).

**Laboratory Diagnosis:** Eggs are best detected by fecal sedimentation or (less reliably) by fecal flotation. The eggs are narrow, ellipsoidal, and cylindrical; have a smooth, clear shell wall; and contain a fully developed larva coiled inside.

**Size:** 30–38 × 11–15 μm

**Clinical Importance:** Infections are often subclinical. The most common clinical signs are dysphagia and regurgitation, but aortic stenosis, aneurysm, esophageal rupture, or obstruction, cachexia, and esophageal sarcomas may occur. *Spirocera* infection is uncommon in the United States.



**Fig. 183** *Physaloptera* eggs do not float consistently in routine flotation exams. The eggs are larger and less elongated than those of *Spirocerca* (Fig. 1.85). *Physaloptera* eggs are smaller and have a thicker shell than larvated hookworm eggs, with which they might be confused. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.



**Fig. 184** Adult *Physaloptera* may be seen with gastroscopy in cases where routine fecal flotation does not detect the parasite eggs. Several worms can be seen on the surface of this canine stomach. Photo courtesy of Dr. Michael Leib, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.



**Fig. 185** *Spirocerca* eggs do not float consistently in common flotation solutions. These larvated eggs are more elongated than *Physaloptera* eggs. Photo courtesy of Dr. Isabelle Verzberger-Epshten, NRC Institute for NutriSciences and Health, Charlottetown, PEI, Canada.

Parasite: *Strongyloides stercoralis* (Figs. 1.23, 1.24, 1.86–1.89)

Common name: Intestinal threadworm.

Taxonomy: Nematode (order Rhabditida).

Geographic Distribution: Worldwide.

Location in Host: Adult females live in the canine small intestine.

Life Cycle: *Strongyloides* parthenogenetic females in the small intestine release larvae that may develop into infective parasitic larvae or, alternatively, may undergo a single free-living cycle of maturation and reproduction before infective parasitic larvae are formed. Infection of the host from the environment is primarily through skin penetration. Transmammary transmission may also occur if the dog is newly infected during lactation.

Laboratory Diagnosis: First-stage larvae may be identified in fresh feces using a Baermann test. *Strongyloides* larvae do not have the modifications of the tail seen in most lungworm larvae. They closely resemble hatched hookworm larvae or free-living nematodes that may be present in fecal samples that have been allowed to sit for a period of time prior to collection (see section on identification of nematode larvae collected by Baermann exam, which is at the beginning of this chapter). If identification of the first-stage larvae is uncertain, larvae in the feces can be cultured for a few days, and the third-stage larvae can be identified.

Size: 150–390  $\mu\text{m}$  in fresh feces. *Strongyloides* larvae can grow quickly in the environment and their size increases before the molt to the second larval stage. This results in a large size range for the larvae. In addition, the size range may be increased by the mistaken identification and measurement of larger second-stage larvae.

Clinical Importance: Infections may be subclinical, but heavy infection can produce respiratory signs from migrating larvae as well as enteritis associated with adults. *Strongyloides stercoralis* also infects humans and may produce severe and even fatal infections in immunocompromised humans. The degree to which canine strains infect humans is unclear, but because of the seriousness of some human cases, infection in dogs should be considered a zoonosis.



Fig. 1.86 *Strongyloides* larvae must be differentiated from hatched hookworm larvae and free-living nematodes. Photo courtesy of Dr. Yoko Naganori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

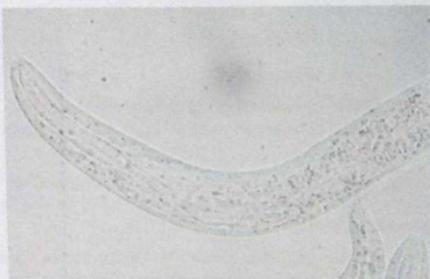


Fig. 1.87 First-stage larvae of *Strongyloides stercoralis* have a well-defined rhabditiform esophagus, a characteristic shared by first-stage hookworm larvae and free-living nematodes, but not seen in canine lungworm larvae. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

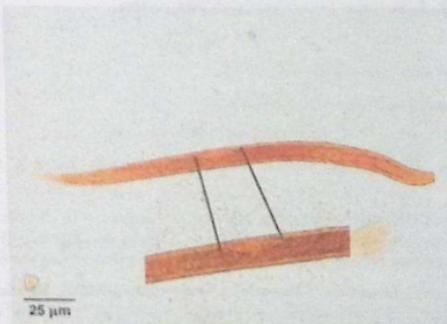


Fig. 1.88 Iodine-stained *Strongyloides* first-stage larva. The prominent genital rudiment (*inset*) and a straight tail lacking accessory spines are helpful in identifying first-stage *Strongyloides* larvae.

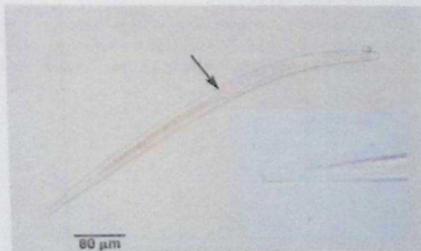


Fig. 1.89 To confirm identification of *S. stercoralis*, the fecal sample can be cultured for 2–4 days and examined for third-stage larvae, which have a distinctively long esophagus (*arrow indicates junction of esophagus and intestine*) and a double-pronged tail tip (*inset*). Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Aelurostrongylus abstrusus* (Figs. 1.90 and 1.91)

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Lung parenchyma (terminal respiratory bronchioles, alveolar ducts) of cats. Reports of canine infection based on fecal examination are most likely spurious parasitism due to coprophagy.

**Life Cycle:** First-stage larvae are released in the airways, coughed up, swallowed, and passed out in the feces. Cats are infected by ingesting a snail or slug intermediate host or paratenic hosts (rodents, birds).

**Laboratory Diagnosis:** First-stage larvae are detected in feces using the Baermann technique (most reliable) or by  $ZnSO_4$  centrifugal flotation. The first-stage larval tail has a severe kink (S-shaped curve) and a dorsal spine.

**Size:** 360–400 × 15–20  $\mu m$

**Clinical Importance:** Infrequently diagnosed; infected animals may suffer signs of chronic cough and anorexia. Heavy infection may be fatal.

**Parasite:** *Crenosoma vulpis* (Figs. 1.25, 1.92)

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Northeastern North America and Europe.

**Location in Host:** Bronchioles, bronchi, and trachea of dogs, foxes, and various wild carnivores.

**Life Cycle:** Canid definitive hosts are infected by ingestion of slug/terrestrial snail intermediate hosts containing third-stage larvae. Larvae produced in the respiratory system by adult worms are coughed up, swallowed, and passed from the host in feces.

**Laboratory Diagnosis:** Detection of first-stage larvae in feces using the Baermann technique (most reliable) or by  $ZnSO_4$  centrifugal fecal flotation. First-stage larvae tend to assume a C shape when they are killed by gentle heat or iodine. The terminus of the tail has a slight deflection but does not show the kink and spine seen in other nematode lungworms.

**Size:** 264–340 × 16–22  $\mu m$

**Clinical Importance:** *Crenosoma vulpis* infection in dogs produces a nonfatal chronic cough. Canine infection is generally rare in North America, except in the Atlantic Canadian provinces, where crenosomosis is a frequent cause of chronic respiratory disease.



Fig. 1.90 *Aclurostrongylus* larva in a feline fecal sample. Larvae have the distinctive S-shaped kink at the end of the tail that is typical of many members of this group of lungworms. These larvae also show a subterminal spine (Fig. 1.91).

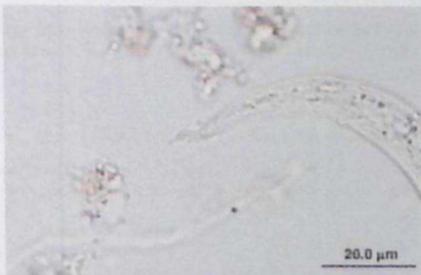


Fig. 1.91 Detail of the tail of *Aclurostrongylus abstrusus* larva showing its characteristic subterminal spine that would be absent in other larvae found in feline feces. For further information on identification of lungworm larvae, see the section in this chapter "Identification of Nematode Larvae Recovered with Fecal Flotation or Baermann Procedures."

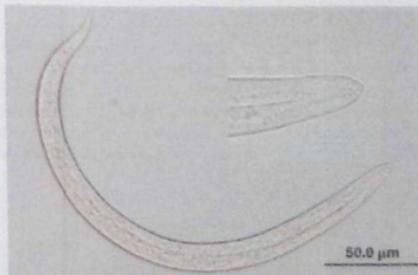


Fig. 1.92 The tail of *Crenosoma* larvae has a slight deflection but lacks a definite kink or dorsal spine, allowing it to be differentiated from other lungworms. At the anterior end is a small cephalic button (inset). Larvae of *Crenosoma* can be differentiated from those of *Strongyloides* based on the morphology of the esophagus (Fig. 1.25).

**Parasite:** *Angiostrongylus vasorum* (Fig. 1.93)

Common name: French heartworm.

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Canada, Europe, South America, Africa. There is a single report of *A. vasorum* in a fox in the eastern United States based on histopathologic findings, but its endemicity has not been established.

**Location in Host:** Pulmonary arteries, right ventricle of dogs and foxes.

**Life Cycle:** First-stage larvae are passed in canine feces, and slugs, snails, and frogs act as intermediate hosts of the parasite. Frogs and birds also serve as paratenic hosts. Dogs and foxes are infected when they ingest intermediate hosts or paratenic hosts.

**Laboratory Diagnosis:** First-stage larvae in fresh feces are detected using the Baermann technique (most reliable) or fecal flotation. The larvae have a cephalic button on the anterior end, and there is a severe kink (S-shaped curve) in the tail, which has a dorsal spine.

Size: 340–399 × 13–17 μm

**Clinical Importance:** The parasite is a serious pathogen of dogs, causing potentially fatal cardiopulmonary disease. Ocular and central nervous system disease and bleeding disorders have also been reported.

**Parasite:** *Oslerus* (= *Filaroides*) *osleri* (Figs. 1.94 and 1.95)

**Taxonomy:** Nematode (order Strongylida). Two other closely related species (*Filaroides hirtili*, *F. milksi*) occur in the respiratory system of dogs but are rare and even more rarely cause disease.

**Geographic Distribution:** Worldwide.

**Location in Host:** Luminal nodules in the tracheal bifurcation in dogs, coyotes, wolves, dingoes, and foxes.

**Life Cycle:** Infection follows ingestion of first-stage larvae from sputum or vomitus of an infected dog or other canid. This life cycle varies from that of other strongylid lungworms because the first larval stage is infective for the definitive host. Typically, the third larval stage is the infective form.

**Laboratory Diagnosis:** Diagnosis is best achieved by visual observation of the nodules on tracheal endoscopic examination. First-stage larvae may be detected in feces by ZnSO<sub>4</sub> centrifugal flotation or in transtracheal wash samples. The Baermann technique is not the method of choice because larvae passed in the feces are usually moribund or dead. The tail of the first-stage larva has an S-shaped sinus wave curve but lacks a dorsal spine. The first-stage larvae of *O. osleri* are indistinguishable from those of *F. hirtili*.

Size:	Larvae recovered from feces	232–266 μm
	Larvae recovered from trachea	325–378 μm

**Clinical Importance:** This is an uncommon infection in dogs in North America. Younger animals tend to be more severely affected than older ones. Respiratory distress, chronic cough, and weight loss can occur. Heavily infected animals may die.

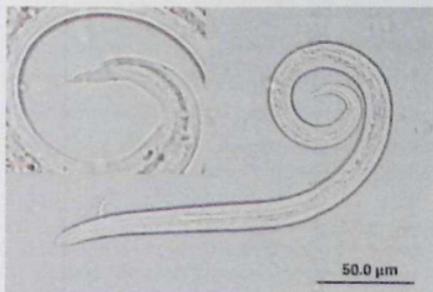


Fig. 1.93 *Angiostrongylus vasorum* first-stage larva. This species is uncommon in North America. Larvae can be differentiated from those of *Filaroides* and *Ostleria* in canine feces by the presence of the subterminal dorsal spine. The inset shows the S-shaped curve and the dorsal spine. The tail of *Achrostrongylus abstrusus* (Fig. 1.91) is similar in appearance.



Fig. 1.94 First-stage *Ostleria* larva in dog feces. The larvae have a kinked tail, but the accessory spine seen in *Achrostrongylus* and *Angiostrongylus* larvae is not present. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 1.95 First-stage larva of *Filaroides hirshi*, which is similar in appearance to *Ostleria ostleri* and *Filaroides milksi*. The inset shows the tail with a kink, but not an accessory spine.

**Parasite:** *Ollulanus tricuspis* (Fig. 1.96)

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Europe, North America, parts of South America, Australia.

**Location in Host:** Adult worms are found in the stomach of cats and other felids.

**Life Cycle:** Adult female worms in the stomach produce third-stage larvae that primarily leave the host in vomitus. Infection of cats occurs through ingestion of these larvae in vomitus.

**Laboratory Diagnosis:** Infection is diagnosed by identification of larvae or the small adults in vomitus, using the Baermann test. Rarely, stages of the parasite may be seen in feces, but they are usually digested before reaching the environment.

Size:	Third-stage larvae	500 $\mu\text{m}$
	Adults	700–1000 $\mu\text{m}$

**Clinical Importance:** Infection can cause chronic gastritis and vomiting in cats. Colony and feral cats are most often infected.



**FIG. 106** Adult male *Ollulanus tricuspis* in a fecal sample. Larvae and adult worms are only rarely present in feces. Vomitus should be examined to diagnose infection. These worms can easily be differentiated from ascarids that may be vomited up by cats. The ascarid worms are several inches in length, while *Ollulanus* adults only reach a maximum length of 1 mm. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

**Parasite:** *Dipylidium caninum* (Figs. 1.97–1.99, 1.105, 1.106)

Common name: Double-pored or cucumber seed or flea tapeworm.

**Taxonomy:** Cestode.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs and cats.

**Life Cycle:** Animals acquire infection through the ingestion of larval cysticercoids contained in fleas or, less frequently, in chewing lice (*Trichodectes*, *Felicola*). Arthropod intermediate hosts become infected by the ingestion of egg packets/segments.

**Laboratory Diagnosis:** Tapeworm segments in the perianal area or in feces are often observed by owners. Specific diagnosis is made by identification of egg packets recovered from segments. Occasionally, eggs and/or egg packets are detected on fecal flotation examinations, but flotation is very insensitive and most infections will be missed.

Size:	Egg packets	120–200 $\mu\text{m}$ (contain an average of 25–30 eggs)
	Eggs	35–60 $\mu\text{m}$

**Clinical Importance:** Infections of this common tapeworm are generally subclinical; however, the passage of segments from the rectum may induce anal pruritus. *Dipylidium caninum* is zoonotic, with young children at greatest risk of acquiring infections from ingesting the infected flea or louse intermediate host.

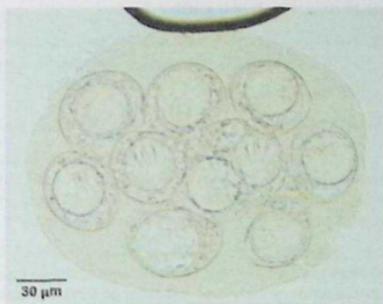


Fig. 197 *Dipylidium caninum* eggs, each containing a hexacanth embryo, occur in packets of about 25–30 eggs. The hooks are readily visible inside the eggs shown here. (Conboy, Gary. *Cestodes of Dogs and Cats in North America*. Veterinary Clinics of North America: Small Animal Practice. Elsevier, 2009.)

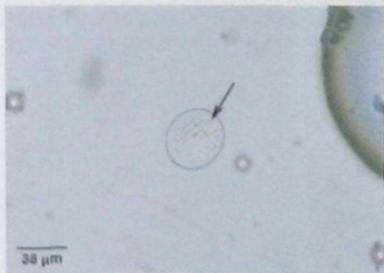


Fig. 198 Occasionally, *Dipylidium* eggs are released from the packets and may be detected individually on fecal flotation. Note the clear, thin shell wall of the egg and the refractile hooks (arrow) of the embryo.



Fig. 199 *Dipylidium caninum* segments on the surface of a canine fecal sample. Photo courtesy of Dr. Kathryn Duncan, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Taenia* spp. (Figs. 1.100–1.103, 1.105, 1.106)

**Taxonomy:** Cestode. Numerous species infect small animals, including *T. taeniaeformis* in cats and *T. pisiformis*, *T.* (= *Multiceps*) *multiceps*, *T. hydatigena*, *T. ovis* in dogs.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs, cats, and various wild carnivores.

**Life Cycle:** Carnivores acquire infections through the ingestion of the immature metacestode stage (morphologic forms include cysticerci, coenuri, and strobilocerci) in the tissues of prey animals. Prey animals become infected with the metacestode through the ingestion of food contaminated with eggs passed in carnivore feces.

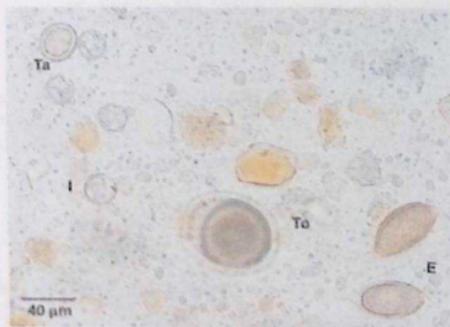
**Laboratory Diagnosis:** Eggs are detected when free in the feces by flotation techniques. Generally, however, eggs are passed from the host contained in tapeworm segments. Therefore, fecal flotation tends to be a poor indicator of infection status.

**Size:** 25–40  $\mu\text{m}$  in diameter

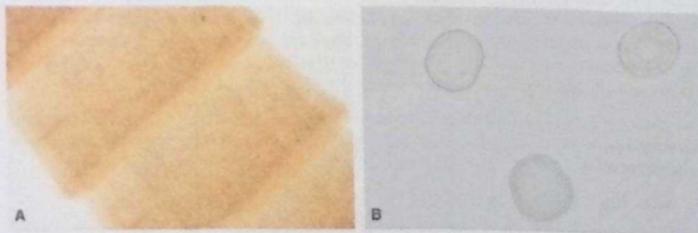
**Clinical Importance:** Infections in the definitive host are generally subclinical; however, the passage of segments from the rectum may induce anal pruritus. *Taenia taeniaeformis* (small-rodent intermediate host) and *T. pisiformis* (rabbit intermediate host) are common species infecting pet cats and dogs, respectively. Metacestode infection in the intermediate hosts can cause disease (*T. multiceps*) or meat condemnation (*T. ovis*).



**Fig. 1.100** *Taenia* eggs are brown with a thick shell wall (embryophore) and contain a hexacanth embryo (six hooks). Three-four refractile hooks are visible in the egg on the lower right of the photo. Note the radial striations in the wall of the egg.



**Fig. 1.101** Eggs of *Eucoleus* (E), *Toxocara* (To), and *Taenia* (Ta) and oocysts of *Cystoisospora* (I) in a feline fecal sample. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.



**Fig. 1.102** Tapeworm segments from an animal (A) can usually be easily identified as such by squashing the segment between two slides and identifying the eggs. Sometimes, however, segments are passed that contain no eggs. These can still be identified as segments by observing the numerous small, rounded, transparent bodies, called "calcareous corpuscles," which produce the stippled effect (A) and are shown at higher magnification in (B). Photo (B) courtesy of Dr. Manigandan LeJeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**Parasite:** *Echinococcus* spp. (Figs. 1.103, 1.105, 1.106)

**Common name:** Dwarf dog or fox tapeworm.

**Taxonomy:** Cestode. A number of species are found worldwide infecting domestic and wild canids and felids. *Echinococcus granulosus*, *E. canadensis* and *E. multilocularis* have the widest distribution and all infect dogs and wild canids. Cats can be infrequently infected with *E. multilocularis*.

**Geographic Distribution:** *Echinococcus granulosus* and *E. canadensis* are found worldwide; *E. multilocularis* occurs in the United States, Canada, and in parts of Europe and Asia.

**Location in Host:** Small intestine.

**Life Cycle:** Carnivores acquire infections through the ingestion of larvae (metacystodes) in the tissues of prey animals. Prey animals become infected with the metacystode through the ingestion of eggs passed in carnivore feces.

**Laboratory Diagnosis:** Like the eggs of *Taenia*, *Echinococcus* eggs have a thick shell wall with radial striations (embryophore). The six hooks of the hexacanth embryo allow it to be distinguished from pollen grains or other debris. The eggs of *Taenia* and *Echinococcus* are morphologically identical.

**Size:** 25–40  $\mu\text{m}$  in diameter

**Clinical Importance:** Adult parasite infections in the definitive host are subclinical. *Echinococcus* spp. are important due to their zoonotic potential. Human infection with the metacystode stage can cause serious disease and death. Rarely, the metacystode stage can develop in dogs that ingest eggs. Resulting disease is similar to that seen in humans.

**Parasite:** *Mesocestoides* spp. (Figs. 1.104–1.106)

**Taxonomy:** Cestode. Species include *M. canislagopodis*, *M. lineatus*, *M. literatus*, *M. vogae*.

**Geographic Distribution:** Worldwide (except Australia).

**Location in Host:** Small intestine of dogs, cats, various wild mammals, and birds.

**Life Cycle:** The life cycle is not completely known. Dogs and cats acquire infections through ingestion of tetrathyridia contained in the tissues of various reptile, amphibian, bird, and mammal intermediate hosts. Eggs are passed in motile segments in the feces of infected dogs and cats. The first intermediate host and the form of the first larval stage of *Mesocestoides* are unknown.

**Laboratory Diagnosis:** Club-shaped segments are passed in the feces. Eggs are contained in a round parauterine organ at the broad end of the segment. Eggs are rarely found free in the feces of definitive hosts but would, presumably, be detected by fecal flotation.

**Size:** Eggs 30–40  $\mu\text{m}$  in diameter

**Clinical Importance:** Infection of the definitive host with the adult tapeworm is usually subclinical. Fatal peritonitis due to large numbers of tetrathyridia or acephalic metacystodes has been reported in dogs acting as hosts to the larval stages.

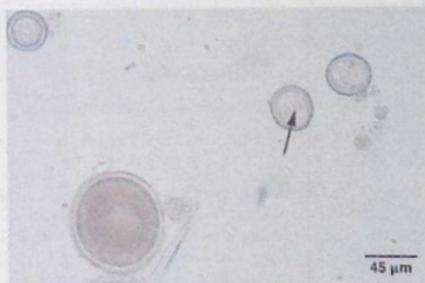


Fig. 1.103 Embryonic hooks are visible in the two *Taenia* or *Echinococcus* eggs in this photo (arrow). Hooks can be used to differentiate tapeworm eggs from similar artifacts like pollen grains. The eggs of *Taenia* and *Echinococcus* are morphologically identical. A *Toxocara* egg is also present.



Fig. 1.104 *Mesocostoides* eggs have a thin, clear, smooth shell wall and contain a hexacanth embryo. The hooks of the embryo are readily visible.

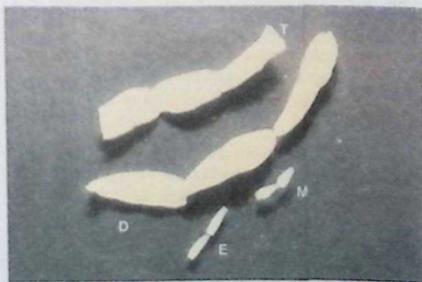


Fig. 1.105 Mature tapeworm segments passed in the feces may be observed by owners and presented for identification. The size and shape of these segments are quite characteristic: *Taenia* segments (T) are square to rectangular in shape. *Dipylidium* segments (D) are more barrel-shaped, and *Mesocostoides* has club-shaped segments (M). *Echinococcus* (E) segments are small and are often overlooked. Identification can be confirmed by examining eggs from the segments.

**Parasite:** *Diphyllobothrium* (= *Dibothriocephalus*) *latum* (Fig. 1.107)

Common name: Broad fish tapeworm.

**Taxonomy:** Cestode. Dogs and cats can also be infected with *D. dendriticum*.

**Geographic Distribution:** Northern Hemisphere and South America.

**Location in Host:** Small intestine of dogs, cats, pigs, humans, and various other fish-eating mammals.

**Life Cycle:** Eggs passed in the feces of the final host hatch coracidia, which are ingested by freshwater copepods (first intermediate hosts). Fish eat the copepods containing the next larval stage (procercoids), which develop into the plerocercoids (infective stage) in the fish. Predatory fish can acquire plerocercoids through ingestion of infected smaller fish. Mammalian definitive hosts acquire infections through the ingestion of plerocercoids contained in the tissues of fish.

**Laboratory Diagnosis:** The eggs can be detected in feces using a sedimentation technique. Lengths of reproductively spent segments are occasionally passed in the feces.

Size: Eggs 58–76 × 40–51 μm

**Clinical Importance:** Uncommon in pets in North America. Infections are generally subclinical in dogs and cats. Dogs and cats do not serve as direct sources of infection for humans. Human infection with this tapeworm may lead to the development of vitamin B<sub>12</sub> deficiency.

**Parasite:** *Spirometra* spp. (Fig. 1.108)

Common name: Zipper tapeworm.

**Taxonomy:** Cestode.

**Geographic Distribution:** *Spirometra mansonioides* occurs in North and South America, and *S. erinacei* occurs in Asia and Europe. Other species have been reported in Africa and Asia.

**Location in Host:** Small intestine of cats, dogs, and wild animals.

**Life Cycle:** Dogs and cats acquire infections by the ingestion of frogs, snakes, rodents, or birds containing plerocercoids (known as spargana). Eggs passed in the feces of dogs and cats hatch coracidia, which are eaten by freshwater copepods and develop into procercoids. The second intermediate hosts (frogs, snakes, etc.) acquire plerocercoids by feeding on the copepods.

**Laboratory Diagnosis:** The yellow-brown eggs can be detected in feces using a sedimentation technique but are also often recovered in flotation procedures. Lengths of reproductively spent segments are occasionally passed in the feces.

Size: Eggs 65–70 × 35–37 μm

**Clinical Importance:** Infections in the definitive hosts are usually subclinical, although vomiting has been reported. Cats can also serve as paratenic hosts, with plerocercoids surviving in various tissues (sparganosis) and causing clinical signs depending on location.



Fig. 3.106 Within hours of passing from the host, tapeworm segments lose their motility and dry up. Clients may find dried tapeworm segments in resting areas of dogs and cats. The dried segments still retain their characteristic shape. Shown in this photo are *Taenia* (T), *Echinococcus* (E), *Dipylidium* (D), and *Monostephanus* (M) segments.



Fig. 3.107 Unlike common tapeworms, *Dipyllobothrium* eggs lack hooks and resemble trematode eggs. They are light brown and operculate (arrow). They contain an undifferentiated embryo surrounded by yolk cells that completely fill the space within the eggshell. A pore in the shell wall at the pole opposite to the operculum is often visible due to the slight bit of protein protruding from it.



Fig. 3.108 The operculate eggs of *Spirometra* also resemble trematode eggs. They contain an undifferentiated embryo and yolk cells that completely fill the space within the eggshell. The eggs are asymmetrical about the long axis. The operculum in this egg is at the lower end.

**Parasite:** *Alaria* spp. (Figs. 1.109 and 1.110)

**Taxonomy:** Trematode.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of dogs, cats, and various wild carnivores.

**Life Cycle:** Eggs are passed in the feces of the mammalian host. Following larval development in a snail intermediate host, a second intermediate host (frog) is infected. Infection of dogs and cats occurs by ingestion of frogs or various paratenic hosts harboring the larval stage (mesocercaria). Transmammary transmission has been reported in cats.

**Laboratory Diagnosis:** The most reliable method is detection of eggs by sedimentation examination of feces, although sometimes eggs may be detected on fecal flotation.

Size: 98–134 × 62–68 μm

**Clinical Importance:** Infections are generally nonpathogenic in dogs and cats. *Alaria* is a potentially serious zoonotic risk to humans through the ingestion of raw or improperly cooked frogs containing mesocercaria.

**Parasite:** *Paragonimus kellicotti* (Fig. 1.111)

**Taxonomy:** Trematode.

**Geographic Distribution:** North America. Other species of *Paragonimus* that infect domestic animals, humans, and wildlife occur in South and Central America, Africa, and Asia.

**Location in Host:** Lung parenchyma of cats, dogs, pigs, goats, minks, and various other wild mammals.

**Life Cycle:** Snails are infected by miracidia that emerge from eggs released in the feces of definitive hosts. Crayfish serve as the second intermediate host. Dogs and cats acquire infection by ingesting the metacercaria in the tissues of crayfish or paratenic hosts.

**Laboratory Diagnosis:** A sedimentation technique (recommended) or fecal flotation (less reliable) can be used to detect the yellow-brown, operculate eggs, which have a thickened ridge in the shell wall along the line of the operculum. Cysts may be evident on radiographs.

Size: 75–118 × 42–67 μm

**Clinical Importance:** Infection may be subclinical or cause eosinophilic bronchitis and granulomatous pneumonia, resulting in chronic cough and lethargy. In some cases pneumothorax may develop due to rupture of cysts. Infections can be fatal.

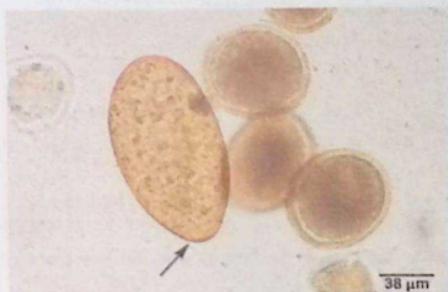


Fig. 1.108 *Alaria* eggs are large, operculate, yellow-brown in color and contain an undifferentiated embryo surrounded by yolk cells. The operculum in this egg is difficult to see but is marked by a slight discontinuity in the shell (arrow). *Toxocara* eggs are also present. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1.110 Sometimes *Alaria* spp. eggs are detected using the centrifugal flotation technique; however, egg morphology may be altered: the eggs appear collapsed or folded due to the osmotic pressure associated with the high specific gravity of the flotation solution.



Fig. 1.111 *Paragonimus* eggs have an undifferentiated embryo when passed in the feces. The yellow-brown, operculate eggs can be identified by the characteristic thickened ridge in the shell wall along the line of the operculum (arrow). Collapsed eggs may be seen in flotation preparations.

**Parasite:** *Nanophyetus salmincola* (Fig. 1.112)

Common name: Salmon poisoning fluke.

**Taxonomy:** Trematode.

**Geographic Distribution:** Pacific Northwest region of North America.

**Location in Host:** Small intestine of dogs, cats, and various other piscivorous carnivores.

**Life Cycle:** Dogs and cats are infected by the ingestion of metacercariae in the tissues of salmonid fish (second intermediate hosts). Snails serve as the first intermediate host.

**Laboratory Diagnosis:** Eggs can be detected by sedimentation examination of feces.

Size: 72–97 × 35–55 μm

**Clinical Importance:** *Nanophyetus salmincola* serves as a vector for the causal agent of salmon poisoning disease (*Neorickettsia helminthoeca*) and Elokomin fluke fever (*Neorickettsia* sp.). Salmon poisoning disease is extremely pathogenic in dogs.

**Parasite:** *Heterobilharzia americana* (Figs. 1.113 and 1.114)

**Taxonomy:** Trematode.

**Geographic Distribution:** Southeast and south-central United States.

**Location in Host:** Mesenteric and hepatic portal veins of dogs and various wildlife species.

**Life Cycle:** Eggs released in the feces of dogs produce ciliated larvae (miracidia) that develop in a snail intermediate host. Cercariae that are released from the snail intermediate host infect dogs and wildlife through direct skin penetration.

**Laboratory Diagnosis:** Eggs can be detected by sedimentation examination of feces in saline (or 5% formol-saline). It is important to use saline in the procedure because eggs are stimulated to hatch when they contact water. The free-swimming miracidia can be observed by placing the sediment in water after performing the sedimentation procedure with saline.

Size: Eggs 74–113 × 60–80 μm

**Clinical Importance:** Infection with *H. americana* in dogs is uncommon in most areas. Infection can cause chronic diarrhea, anorexia, and emaciation. *Heterobilharzia* also has zoonotic importance as one of the causal agents of cercarial dermatitis (swimmer's itch) in humans.



Fig. 1.112 The operculated eggs of *Nanophyetus* contain an undifferentiated embryo surrounded by yolk cells.



Fig. 1.113 The large, elliptical eggs of *Heterobilharzia americana* have a smooth, thin shell wall and contain a fully formed miracidium. The shell wall of the egg lacks an operculum. Photo courtesy of Dr. Bruce Hammerberg and Dr. James Flowers, College of Veterinary Medicine, North Carolina State University, Raleigh, NC.

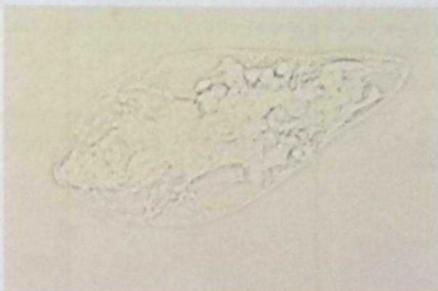


Fig. 1.114 On contact with freshwater, *H. americana* eggs hatch, releasing a ciliated miracidium stage. To prevent hatching of the eggs, a sedimentation procedure for diagnosis should be performed using saline instead of water. Photo courtesy of Dr. Bruce Hammerberg and Dr. James Flowers, College of Veterinary Medicine, North Carolina State University, Raleigh, NC.

**Parasite:** *Platynosomum concinnum* (also known as *P. fastosum*, *P. illiciens*) (Figs. 1.115, 1.116)

**Common name:** Lizard poisoning fluke.

**Taxonomy:** Trematode.

**Geographic Distribution:** Southeastern United States, South America, Caribbean, West Africa. Other flukes that may be found in the bile or pancreatic ducts in North America include *Eurytrema procyonis*, *Metorchis conjunctus* and *Parametorchis* spp.

**Location in Host:** Gall bladder and bile ducts of cats.

**Life Cycle:** Adult worms produce eggs that are passed in the feces of cats. The life cycle is complex, involving snail, crustacean, and amphibian or reptile intermediate hosts. Cats are infected following ingestion of lizards or amphibians containing larvae.

**Laboratory Diagnosis:** A sedimentation procedure is most effective for recovering the relatively small, operculate eggs of *Platynosomum*.

**Size:** 34–50 × 20–35 μm

**Clinical Significance:** Light infections are asymptomatic. Heavily infected cats may show signs of weight loss and hepatomegaly that can be severe and fatal.

**Parasite:** *Cryptocotyle lingua* (Fig. 1.117)

**Taxonomy:** Trematode.

**Geographic Distribution:** Northern temperate marine coastal regions of the world.

**Location in Host:** Intestine.

**Life Cycle:** Snails are infected by larval stages emerging from eggs released in the feces of infected definitive hosts. Fish serve as the second intermediate host. Dogs, cats, and birds become infected by ingesting metacercariae, visible as small black spots on the skin of fish.

**Laboratory Diagnosis:** A sedimentation technique (recommended) or fecal flotation (less reliable) can be used to detect these small, undifferentiated, yellow-brown, operculate eggs. The eggs are elliptical-shaped with one end narrower than the other. The operculum occurs at the narrower end and is difficult to see.

**Size:** 32–50 × 18–25 μm

**Clinical Significance:** Clinical disease due to infection with this intestinal fluke appears to be rare. Enteritis may occur in cases of heavy infections. Cats appear to be less susceptible to infection.



Fig. 1.115 The small brown eggs of *Platynosomum* have an operculum at one end, contain a fully formed miracidium, and may not be seen with routine flotation procedures. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.

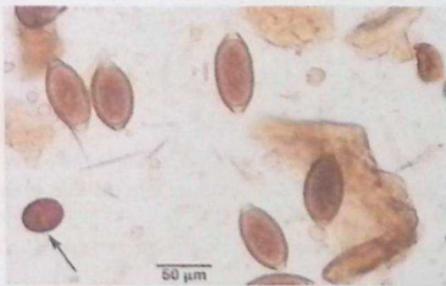


Fig. 1.116 *Platynosomum* (arrow) and *Trichuris* eggs in the feces of a cat. Both parasites are unlikely to be encountered in the United States outside the southeast. Photo courtesy of Dr. Jennifer Ketzis, School of Veterinary Medicine, Ross University, St. Kitts, WI.

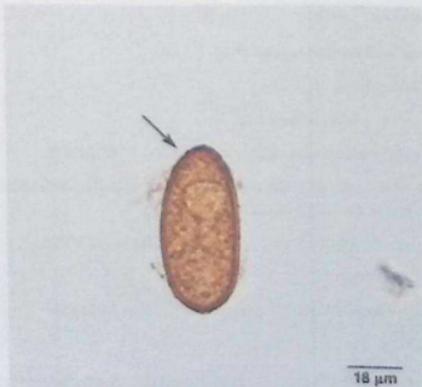


Fig. 1.117 *Cryptocotyle lingua* egg detected on fecal sedimentation of feces from a dog. The operculum of *Cryptocotyle* is difficult to see; it occurs on the narrower pole of the egg (arrow). The eggs are undifferentiated when passed in the feces.

**Parasite:** *Metorchis* spp., *Eurytrema* spp. (Fig. 1.118)

**Taxonomy:** Trematode.

**Geographic Distribution:** Europe, Asia, North America.

**Location in Host:** Bile ducts and gallbladder (*Metorchis*), pancreatic ducts (*Eurytrema*) of wild carnivores, rarely dogs and cats.

**Life Cycle:** Snails are infected by larval stages emerging from eggs in feces of definitive hosts that become infected following ingestion of a second intermediate host (fish in the case of *Metorchis* and probably an arthropod for *Eurytrema*).

**Laboratory Diagnosis:** A sedimentation technique would be most effective.

Size: *Metorchis* 24–30 × 13–16 μm, *Eurytrema* 45–53 × 29–36 μm

**Clinical Significance:** Infection in dogs and cats is rare, but may be associated with disease of infected organ.

**Parasite:** *Linguatula* spp. (Fig. 1.119)

**Taxonomy:** Pentastomid.

**Geographic Distribution:** Most infections from Africa and Asia.

**Location in Host:** Nasal passages, frontal sinuses of canids and, less often, felids.

**Life Cycle:** Eggs shed in nasal discharge or feces of infected hosts. Herbivores are intermediate hosts with larvae in tissues infecting the final host when ingested.

**Laboratory Diagnosis:** Centrifugal fecal flotation with solutions of ≥1.25 SG.

Size: 90–133 × 54–88 μm

**Clinical Significance:** In dogs infection may be asymptomatic or produce increased nasal discharge and sneezing. Humans can serve as intermediate hosts or, rarely, definitive hosts.

**Parasite:** *Macracanthorhynchus ingens* (Fig. 1.120)

**Taxonomy:** Acanthocephala.

**Geographic Distribution:** North America.

**Location in Host:** Intestine of raccoons, black bears, rarely dogs.

**Life Cycle:** Eggs shed in feces of infected hosts. Larvae infect definitive hosts when milpide intermediate hosts carrying larvae are ingested.

**Laboratory Diagnosis:** A sedimentation test would be most effective for detecting eggs.

Size: 90–110 × 50–65 μm

**Clinical Significance:** Dogs are rarely infected with this parasite.



Fig. 1.118 *Metorchis* is another genus of flukes present in wild animals that may occasionally infect dogs and cats.



Fig. 1.119 Large eggs of *Linguatula* may be seen in dogs in North America with a history of travel to an endemic area. Careful examination at high power should reveal the presence of several hooks, in the parasite embryo (arrow and inset). Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1.120 *Macracanthorhynchus ingens* eggs. Acanthocephalan eggs contain a larva with spines, which may be seen inside eggs. Dogs are also rarely infected with another acanthocephalan parasite, *Oncicola canis*. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.

## Ruminants and Camelids

Helminth Eggs, Larvae and Protozoan Cysts  
found in freshly voided feces of

## Cattle

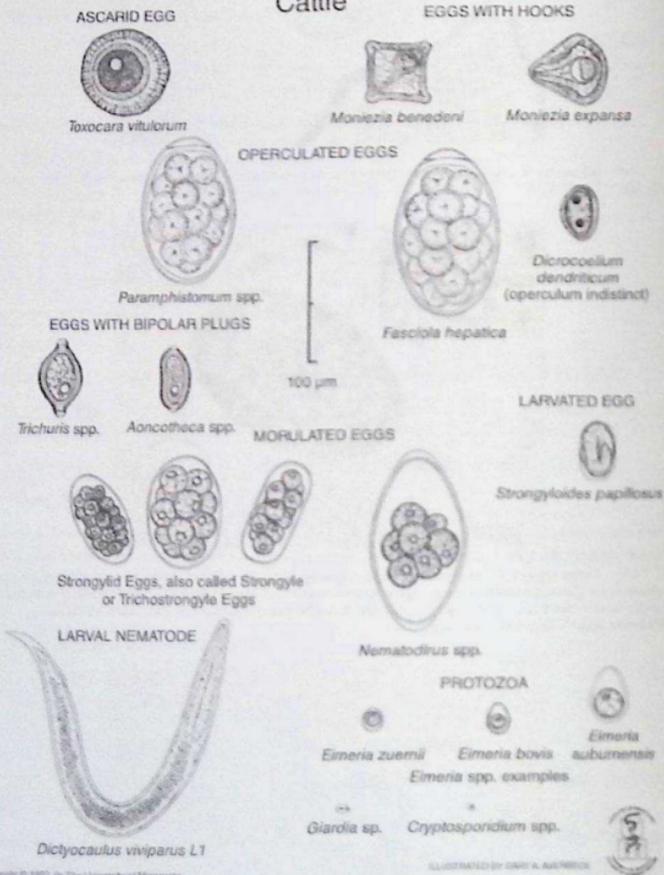


FIG. 1121 Parasites found in bovine feces. Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

## Helminth Eggs, Larvae and Protozoan Cysts found in freshly voided feces of Sheep, Goats and Camelids

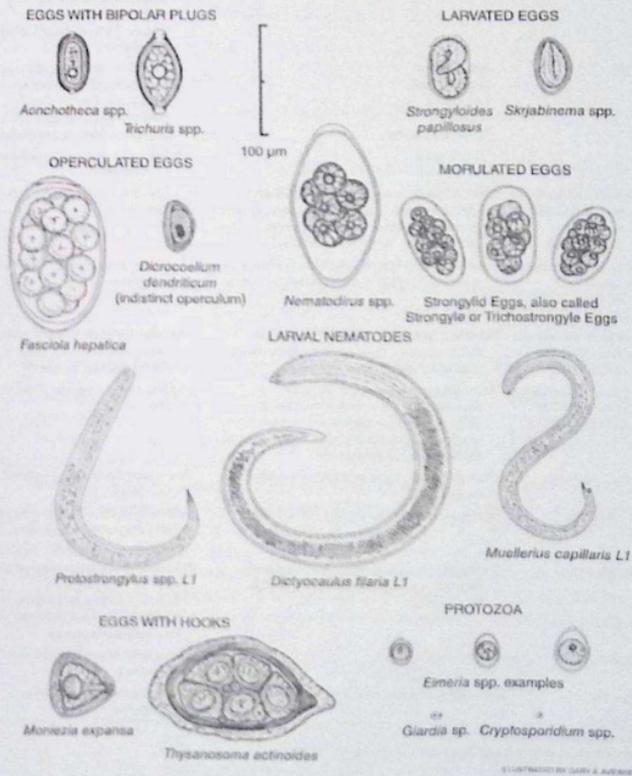


Fig. 1.192. Parasites found in feces of sheep and goats. Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

**Table 1.7. Representative treatments for selected parasites of ruminants and camelids**

Parasite	Effective treatments	Dose, route, and regimen
<i>Cryptosporidium parvum</i>	*Halofuginone lactate	Administer according to label directions. Begin within 48 h of birth for prevention, within 24 h of diarrhea for treatment
<i>Eimeria</i> spp.	Amprolium Decoquinatol Lasalocid Monensin Sulfamethazine *Toltrazuril	Administer according to label directions for prevention or treatment. Use in small ruminants/camelids may require modification of dose  Administer according to label directions
Gastrointestinal nematodes of pastured and feedlot cattle (see label for specific indications)	Albendazole, †closantel, doramectin, eprinomectin, †febantel, fenbendazole, ivermectin, levamisole, moxantel tartrate, moxidectin, †netobimol, oxfendazole	Administer according to label directions
Gastrointestinal nematodes of dairy cattle >20 months of age (see label for specific indications)	Eprinomectin (pour-on only), fenbendazole, moxantel tartrate, moxidectin (pour-on only)	Administer according to label directions
Gastrointestinal nematodes of goats (see label for specific indications)	Albendazole, †closantel, †doramectin, fenbendazole, †ivermectin, †levamisole, moxantel tartrate, †moxidectin	Administer according to label directions; elevated dose †aten required for goats
Gastrointestinal nematodes of sheep (see label for specific indications)	Albendazole, †closantel, †derquantel-abamectin, †doramectin, †febantel, †fenbendazole, ivermectin, levamisole, †mcbendazole, †monepantel, moxidectin, †netobimol, †oxfendazole	Administer according to label directions
<i>Dictyocaulus viviparus</i>	Fenbendazole, eprinomectin, ivermectin, moxidectin	Administer according to label directions
<i>Muellerius capillaris</i>	†Fenbendazole, †ivermectin, †moxidectin	Effective when administered at label-approved or elevated dose
<i>Toxocara vitulorum</i>	†Doramectin (injectable), †fenbendazole, †ivermectin (injectable), †moxidectin (injectable)	Effective when administered at label-approved dose. Efficacy demonstrated for injectable but not pour-on macrocyclic lactams
<i>Moniezia</i> spp.	Albendazole	Administer according to label directions
<i>Fasciola hepatica</i>	Albendazole, closulon, †closantel	Administer according to label directions

† Not label-approved in the United States.

Additional information on parasite treatments can be found in Chapter 7.

### Protozoan Parasites

**Parasite:** *Eimeria* spp. (Figs. 1.46, 1.123–1.130, 1.133, 1.136, 1.145)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia).

**Geographic Distribution:** Worldwide.

**Location in Host:** Many host-specific species of *Eimeria* infect the intestinal tract of domestic ruminants and camelids.

**Life Cycle:** Fecal oocysts sporulate in the environment and infect intestinal cells following ingestion. Asexual and sexual reproduction are followed by the production of oocysts that exit the host in manure. Sporulated oocysts can survive for long periods under favorable environmental conditions.

**Laboratory Diagnosis:** Oocysts are found by fecal flotation techniques. Species identification is difficult and may require microscopic exam of sporulated (infective) oocysts. Although the number of oocysts in feces has been used as an indicator of clinical disease, high numbers of oocysts can also be present in the absence of clinical signs.

**Size:** Approximately 12–45  $\mu\text{m}$  in length (oocyst), depending on species

**Clinical Importance:** Most ruminants become infected with coccidia at an early age, and low-level infection persists through adulthood. While infection is often subclinical, coccidiosis is a common cause of diarrhea in young ruminants. Signs range from mild diarrhea to severe, bloody diarrhea.

Not all species of *Eimeria* are equally pathogenic. Of 12 species of common bovine *Eimeria*, clinical disease is usually associated with *E. bovis*, *E. zuernii*, or, less commonly, *E. alabamensis*. Similarly, *E. bakuensis*, *E. ahsata*, and *E. ovinoidalis* are pathogenic ovine *Eimeria* species. In goats, *E. airongi*, *E. caprina*, *E. ninakohlyakimovae*, and *E. christenseni* have been associated with clinical disease. Fewer species of coccidia occur in camelids than in ruminants. They all have been reported to cause clinical disease, although in the United States, *E. macusaniensis* is considered most pathogenic.



**Fig. 1.123** Ruminants and camelids are infected with a variety of *Eimeria* spp. The oocysts of most of these species are colorless, have a thin wall, and are oval or round. When seen in fresh feces, oocysts contain a single cell.

Table 1.6. Common *Eimeria* species of cattle

Name	Average size ( $\mu\text{m}$ )	Range ( $\mu\text{m}$ )	Length:width (range)	Notes
<i>Eimeria alabamensis</i>	19 x 13	13-25 x 11-17	Not established	• No micropyle • <b>Pyriform</b> (sub-ellipsoidal or sub-cylindrical) • Colorless to greyish lavender • Wall - thin & delicate • Mildly pathogenic
<i>Eimeria autumnensis</i>	38 x 23	32-46 x 19-28	L:W 1.67 (1.32-2.08)	• Flat micropyle • <b>Elongate ovoid</b> (sub-ellipsoidal to notably tapered) • Oocyst wall typically smooth rarely rough (mammillations) • Yellowish brown • Mildly pathogenic
<i>Eimeria bovis</i>	28 x 20	23-34 x 17-23	L:W 1.37 (1.1-1.8)	• Flat micropyle • <b>Stoutly egg-shaped/ovoid</b> (tapered towards micropylar/blunted narrow end) • Yellowish • Most pathogenic
<i>Eimeria brasiliensis</i>	38 x 27	31-49 x 21-33	Not established	• Micropyle • Polar cap (may be collapsed) • <b>Ovoidal</b> • Colorless to yellowish or pinkish • Occasionally plaques on wall • Nonpathogenic
<i>Eimeria baklanovensis</i>	47.4 x 33	43-51 x 30-35	L:W 1.4 (1.3-1.8)	• Micropyle • <b>Piriform</b> • <b>Brown radially striated/speckled wall</b> • Mildly pathogenic
<i>Eimeria canadensis</i>	33 x 23	28-38 x 20-29	L:W 1.39 (1.2-1.6)	• Micropyle • <b>Ellipsoidal</b> but varies from cylindrical to stoutly ellipsoidal • Colorless to pale yellow • Nonpathogenic • Oocyst wall 1 $\mu\text{m}$ in middle, thins as it tapers
<i>Eimeria cylindrica</i>	23 x 14	16-30 x 12-17	L:W 1.67 (1.3-2.0)	• No micropyle • <b>Cylindrical</b> (vary from ellipsoidal to narrow cylinder) • Colorless • Mildly pathogenic
<i>Eimeria ellipsoidalis</i>	17 x 13	12-27 x 10-18	L:W 1.30 (1.0-1.6)	• No micropyle • Predominantly <b>ellipsoidal</b> (also spherical to subspherical) • Colorless • Mildly pathogenic
<i>Eimeria pellita</i>	40 x 28	36-41 x 26-30	Not established	• Flat micropyle • <b>Ovoid</b> • Brown • Thick <b>velvety walls</b> • Nonpathogenic
<i>Eimeria subspherica</i>	11 x 10	9-14 x 8-13	Not established	• No micropyle • <b>Subspherical</b> but vary from spherical to bluntly ellipsoidal • Thin oocyst wall • Colorless • <b>More fragile appearance</b> compared to <i>E. ellipsoidalis</i> or <i>E. zuernei</i> • Nonpathogenic
<i>Eimeria wyomingensis</i>	39.9 x 28.3	36-44 x 26-30	Not established	• Micropyle • <b>Ovoid</b> • Yellowish-brown • Nonpathogenic
<i>Eimeria zuernei</i>	17.8 x 15.6	15-22 x 13-18	L:W 1.14 (1.0-1.4)	• No micropyle • <b>Spherical to bluntly ellipsoidal</b> • Colorless • Most pathogenic

Information compiled by Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY. Source: Bowman, D.D. Georgia Parasitology for Veterinarians 4th edition, 2021, Elsevier.

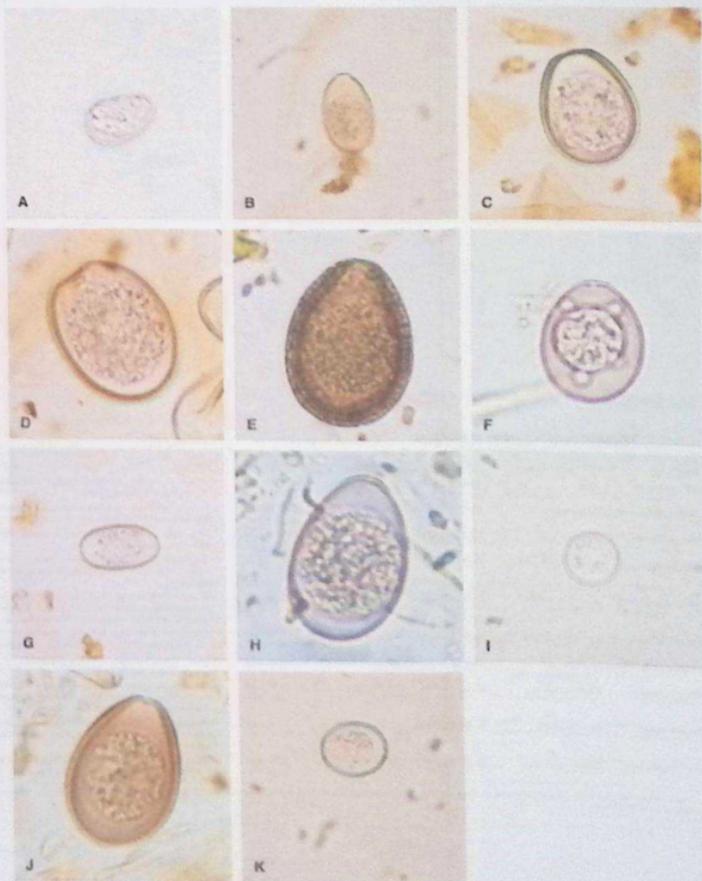


FIG. 1.124 Bovine *Eimeria* oocysts (relative sizes not accurate; see Table 1.8): (A) *E. alabamensis*, (B) *E. anabrenensis*, (C) *E. bovis*, (D) *E. branthensis*, (E) *E. bulidanimensis*, (F) *E. canadensis*, (G) *E. cylindricalellipsoidalis*, (H) *E. pellita*, (I) *E. subspheerica*, (J) *E. ussomingensis*, (K) *E. zierenii*. (A, F, H, I) Courtesy of Dr. Aaron Lucas, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA. Other photos courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

Table 1.11. Common *Eimeria* species of sheep

Name	Oval equatorless	Average Size (µm)	Range (µm)	Length:width (range)	Notes
<i>Eimeria abarata</i>	N/A	33-48 × 20-26	29-48 × 17-30	L:W 1.52 (3.3-4.8)	* Macropyle + Polar cap - dome shaped + Ellipsoidal to oval * Faint stain + Very pathogenic.
<i>Eimeria baharum</i>	<i>Eimeria abarata</i>	27-31 × 20-21	23-36 × 16-24	L:W 1.41 (3.3-4.8)	* Macropyle + Polar cap - dome to rounded-shaped - faint to ellipsoid (generally strongly white). Yellow-brown - Pathogenic
<i>Eimeria crandalli</i>	<i>Eimeria hirci</i>	22-23 × 18-19	15-28 × 14-22	L:W 1.13 (3.0-4.35)	* Macropyle + Polar cap + Spherical to broadly ellipsoidal * Colorless + May cause diarrhoea
<i>Eimeria fauci</i>	<i>Eimeria ophryocyba</i>	26-32 × 20-23	24-37 × 18-28	Not established	* Macropyle + 'Cap-shaped' toward posterior + Oval to spindle * Light stained + Oocyst wall with fine striated line + Mildly pathogenic.
<i>Eimeria grandiosa</i>	<i>Eimeria jobbhoyi</i>	28-32 × 21-24	22-37 × 17-26	Not established	* Macropyle + Polar cap - sometimes readily delineated - Oocyst wall lightly colored with thick outer layer - Broad shallow to elliptical to broadly ellipsoidal + Nonpathogenic.
<i>Eimeria intricata</i>	<i>Eimeria hirci</i>	46-51 × 32-38	39-58 × 27-47	L:W 1.47 (3.3-4.8)	* Macropyle + Polar cap + Ellipsoidal - Oocyst wall 1.5-2.0 µm thick - Ellipsoid, single, brown, striated + Mildly pathogenic
<i>Eimeria orientalis</i>	<i>Eimeria nassabadi</i> / <i>Eimeria nassabi</i>	23 × 15	16-28 × 14-23	L:W 1.07 (3.0-4.7)	* Macropyle barely seen + Oocyst is ellipsoidal * Colorless + Very pathogenic
<i>Eimeria pallida</i>	<i>Eimeria pallida</i>	14-15 × 10-11	12-30 × 8-13	L:W 1.43 (3.2-3.7)	* Macropyle not seen + Ellipsoidal + Oocyst wall thin. Colorless to yellowish with a single dark refraction line on outer edge
<i>Eimeria parva</i>	<i>Eimeria alpeji</i>	15-18 × 13.5-15	12-23 × 10-19	L:W 1.18 (3.0-4.7)	* Macropyle not seen + Roundish - Oocyst wall pale yellow to light brown with two dark refraction lines on each side of inner line + Mildly pathogenic.
<i>Eimeria parvula</i>	<i>Eimeria parvula</i>	21-26 × 15-19	18-28 × 10-21	L:W 1.29 (3.3-4.3)	* Macropyle + Polar cap + Ellipsoidal/polyspherical to oval * Case-shaped pits on multipolar like a bubble + Non pathogenic

Information compiled by Dr. Manojendra Lakshmi, Animal Health Diagnostic Center, Cornell University, Ithaca, NY. Source: Robinson, D.D. *Sheep Parasitology for the Practitioner* (1st edition 2011, Elsevier).

Table 3.10. Common *Eimeria* species of goats

Name	Sheep equivalent	Average Size (µm)	Range (µm)	Length: width (range)	Notes
<i>Eimeria arloingi</i>	<i>Eimeria infantum</i>	20 × 21	21–35 × 10–20	L:W 1.5–1.6 (1.1–2.1)	• Microcystle • Polar cap • <b>Elongate ellipsoidal</b> • 2 layers (outer colorless, inner brownish yellow) • Pathogenic
<i>Eimeria ahenorhiza</i>	<i>Eimeria faecalis</i>	20–33 × 23–24	24–37 × 10–20	L:W 1.2–1.4 (1.1–1.8)	• Microcystle • 'Cup-shaped' apical projection • <b>Ovoid (egg-shaped)</b> • Oocyst wall colorless • A small knob associated with microcystle on inside of oocyst • Mildly pathogenic
<i>Eimeria caprina</i>	N/A	31 × 23	27–40 × 19–20	L:W 1.4 (1.2–2.1)	• Microcystle • <b>Ellipsoid to oval</b> • 2 layers (outer brownish yellow, inner colorless) • Nonpathogenic
<i>Eimeria caprinae</i>	N/A	30 × 24	26–34 × 21–20	L:W 1.3 (1.1–1.5)	• Microcystle • <b>Ellipsoidal to dispherical</b> • 2 layers (outer colorless, inner brownish yellow)
<i>Eimeria chelonensis</i>	N/A	30–41 × 25–28	31–44 × 22–31	L:W 1.5–2.6 (1.2–1.8)	• Microcystle • <b>Ovoid</b> • 2 layered (outer colorless to pale yellow, inner brownish yellow & wrinkled at microcystle end) • Pathogenic
<i>Eimeria berberis</i>	<i>Eimeria intricata</i>	45 × 37	41–50 × 34–37	Not established	• Microcystle • Polar cap • <b>Ellipsoid</b> • Oocyst wall 3 layered • Opaque, rough, becom. striated • Mildly pathogenic
<i>Eimeria hirci</i>	<i>Eimeria mundialis</i>	21–23 × 16–18	19–20 × 14–21	L:W 1.2–1.3 (1.1–1.7)	• Microcystle • Polar cap • <b>Spherical to broadly ellipsoidal</b> • 2 layers (outer colorless, inner brownish yellow) • May cause diarrhea
<i>Eimeria jahrogi</i>	<i>Eimeria parvula</i>	31–33 × 20–28	26–37 × 18–20	L:W 1.4–1.5 (1.2–1.7)	• Microcystle • Polar cap prominent (usually divided) • <b>Broad shouldered oval</b> like periorbitally ellipsoidal • Oocyst wall lightly colored with thick brownish yellow inner layer • Nonpathogenic
<i>Eimeria woodhullakumarai</i>	<i>Eimeria erinoidalis</i>	24–28 × 18–21	20–28 × 6–24	L:W 1.2–1.3 (1.0–1.5)	• Microcystle barely seen • <b>Ovoid to ellipsoidal</b> • Colorless • Very pathogenic
<i>Eimeria pallida</i>	<i>Eimeria pallida</i>	14 × 11	13–18 × 10–14	L:W 1.2 (1.2–1.8)	• Microcystle not seen • <b>Ellipsoidal</b> • Oocyst wall thin, colorless, two layers with a single dark refraction line on inner edge • Nonpathogenic • Narrower than <i>Eimeria aljezi</i>
<i>Eimeria aljezi</i>	<i>Eimeria parva</i>	17–20 × 14–19	15–23 × 10–22	L:W 1.1–1.2 (1.1–2.1)	• Microcystle not seen • <b>Rounded</b> • Oocyst wall pale yellow, two layers with two dark refraction lines on each side of inner layer • Mildly pathogenic • Less common in goats
<i>Eimeria parvula</i>	<i>Eimeria parvula</i>	20 × 20	21–31 × 15–25	L:W 1.3 (1.2–1.7)	• Microcystle • Polar cap • <b>Ellipsoidal to subpherical to ovoid</b> , greenish yellow • <b>Cone-shaped</b> pits on well-defined line • <b>binuclei</b> • Non pathogenic

Information compiled by Dr. Macgregor Laird, Animal Health Diagnostic Center, Cornell University, Ithaca NY, Ithaca, D.D. Georgia Parasitology for Veterinarians 11th edition, 2013, Elsevier



Fig. 1.125 Small ruminants are infected with a variety of coccidia species, although few are highly pathogenic. Sheep and goats are infected by different *Eimeria* spp. (E), although oocysts from the two host species are often indistinguishable. Slightly out of focus in this photo are an egg of *Nematodirus* (N) and a typical strongylid egg (S).

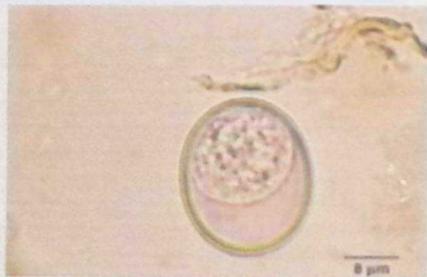


Fig. 1.126 The oocyst shown here is *E. ninakoblyukimovae* from a goat. A similar oocyst is produced by *E. ovinodalis* in sheep. Both species can be pathogenic in their respective hosts. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY



Fig. 1.127 Many, but not all, *Eimeria* species have a cap that covers the micropyle, which can be seen at the upper end of this oocyst. The micropyle cap (or polar cap), if present, is helpful in identifying the genus in dogs that have eaten feces of other animal species. The photo shows the oocyst of *Eimeria intricata*, a parasite of sheep. The oocyst is brown and larger than other ovine *Eimeria*.



Fig. 1128 In this bovine fecal sample, several fully sporulated *Eimeria* oocysts can be seen. In some cases, examination of the sporulated oocyst is required for confirmation of identification of the *Eimeria* species. In many clinical cases, specific identification is not performed. Photo courtesy of Dr. Aaron Lucas, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

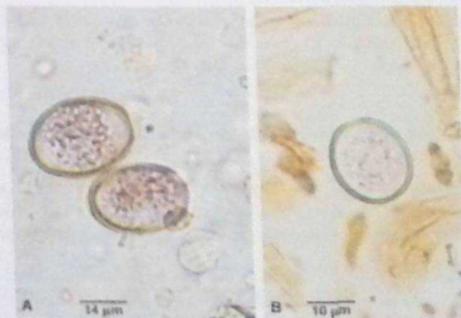


Fig. 1129 *Eimeria blanae* (A) and the smaller *E. panamensis* (B) are coccidia of New World camelids. Photos courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

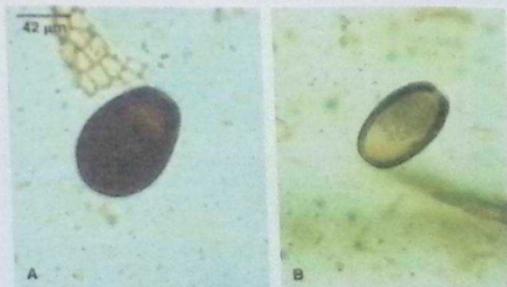


Fig. 1130 New World camelids are also infected with two species of *Eimeria* that produce large brown oocysts, which can be confused: *E. macusaniensis* (A) and *E. vituensis* (B). The oocysts of *E. macusaniensis* are pear-shaped, while those of *E. vituensis* are more elongated and elliptical in shape.

**Parasite:** *Cryptosporidium* spp. (Figs. 1.131 and 1.132)

**Taxonomy:** Protozoa (coccidia). Ruminants are infected with several *Cryptosporidium* species, including *C. parvum*, *C. andersoni*, *C. bovis*, *C. ubiquitum*, and *C. xiaoi*.

**Geographic Distribution:** Worldwide.

**Location in Host:** *Cryptosporidium parvum* is an intestinal parasite of ruminants, camelids, and other mammals. Other species parasitize the small intestine or stomach (abomasum).

**Life Cycle:** Ruminants are infected by ingestion of oocysts. Oocysts are infective as soon as they are passed in manure and are very resistant to environmental conditions.

**Laboratory Diagnosis:** The small oocysts of *Cryptosporidium* spp. can be detected with centrifugal flotation exam using Sheather's sugar solution. Fecal smears can also be stained with acid-fast stains or examined by immunodiagnostic techniques. Species identification requires molecular analysis.

**Size:** 4–8  $\mu\text{m}$  in diameter depending on species

**Clinical Importance:** Infections may be subclinical or cause diarrhea of varying severity, especially in young animals. *Cryptosporidium parvum* is a widely reported zoonotic species. Other *Cryptosporidium* species may also be zoonotic.

**Parasite:** *Giardia duodenalis*, also identified as *G. lamblia*, *G. intestinalis*, *G. bovis*, etc. (Figs. 1.53–1.59, 1.133)

**Taxonomy:** Protozoa (flagellate). The taxonomy of *Giardia* species is currently undergoing revision. Isolates of the parasite are currently assigned to assemblages based on genetic analysis. Isolates of hoofed stock typically belong to Assemblage E.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of ruminants and camelids.

**Life Cycle:** *Giardia* cysts passed in the feces infect other animals when ingested in the environment. Following excystation, trophozoites inhabit the small intestine.

**Laboratory Diagnosis:** Cysts of *Giardia* can be found in fecal samples using centrifugal flotation procedures (33% ZnSO<sub>4</sub> flotation solution preferred). Trichrome-stained fecal smears and immunodiagnostic tests can also be used.

**Size:** Cysts 9–13  $\times$  7–9  $\mu\text{m}$   
Trophozoites 12–17  $\times$  7–10  $\mu\text{m}$

**Clinical Importance:** Many animals are infected, particularly when young. Clinical disease is uncommon, but there are some reports of parasite impact on growth in young animals.





Fig. 1.131 The small oocysts of *Cryptosporidium* are best seen using the high-dry objective (40 $\times$ ) on the microscope. They are highly refractile and often appear to have a single black dot in the center.



Fig. 1.132 *Cryptosporidium* oocysts can also be detected in acid fast-stained fecal smears. Photo source: CDC/ Public Health Image Library (<https://phil.cdc.gov/Details.aspx?pid=7829>).



Fig. 1.133 This calf fecal sample contains a larvated *Strongyloides* egg (S) as well as numerous collapsed *Giardia* cysts (G). Several small *Eimeria* oocysts (E) are also present. Most coccidia oocysts are larger than *Giardia* cysts.

**Parasite:** *Buxtonella sulcata* (Fig. 1.134).

**Taxonomy:** Protozoa (ciliate).

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum of ruminants.

**Life Cycle:** The life cycle has not been fully described. Trophozoites are present in the cecum and cysts pass out of the host in the manure.

**Laboratory Diagnosis:** Cysts may be seen in manure samples examined by sedimentation test procedures and rarely by fecal flotation procedures.

**Size:** Cyst 40–60  $\mu\text{m}$

**Clinical Significance:** No clinical significance has been reported.

### Helminth Parasites

**Parasite:** Strongylid Parasites of Ruminants and Camelids (Figs. 1.16, 1.128, 1.135–1.140, 1.145)

**Common name:** Various, including brown stomach worm, barber pole worm, hookworm, nodular worm, strongyles, trichostrongyles.

**Taxonomy:** Nematodes (order Strongylida). Numerous genera belong to this group, including *Ostertagia*, *Haemonchus*, *Cooperia*, *Trichostrongylus*, *Teladorsagia*, *Mecistocirrus*, *Oesophagostomum*, *Bunostomum*, *Chabertia*, *Camelostrongylus*, and *Lananema*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Gastrointestinal tract of ruminant and camelid hosts. The genera infecting these hosts are largely the same, although species may vary.

**Life Cycle:** Adult worms in the gastrointestinal tract produce eggs that develop in manure in the environment. Infective larvae are released onto pasture, where they are ingested by grazing hosts.

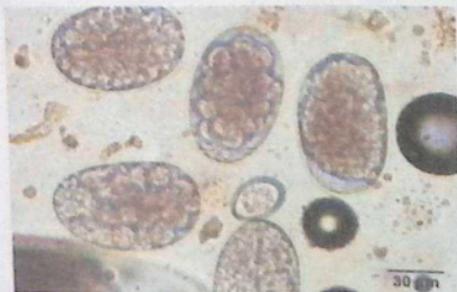
**Laboratory Diagnosis:** Eggs are detected by routine or quantitative fecal flotation procedures and are similar in appearance. For diagnosis of genera, culture of feces and identification of infective third-stage larvae may be performed. Quantitative egg counts are useful in designing and evaluating parasite control programs. Molecular testing to identify parasite genus is also available.

**Size:** Approximately 65–100  $\times$  34–50  $\mu\text{m}$ , depending on species

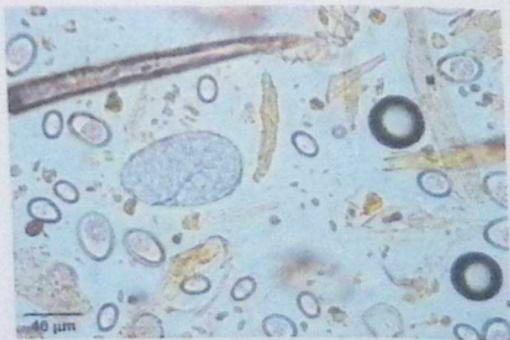
**Clinical Importance:** Virtually all grazing animals are infected with strongylid parasites, and many infections are asymptomatic. Young, nonimmune animals are most susceptible to subclinical and clinical disease, which may include diarrhea, anemia, hypoproteinemia, reduced growth, and death in severe cases. The species of greatest importance vary with host and region.



**Fig. 1134** Cyst of *Buxtonella sulcata*. These cysts may be seen in bovine fecal samples examined by sedimentation test and infrequently in bovine fecal flotation test preparations. The bean-shaped macronucleus characteristic of ciliates can be seen (arrow). Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



**Fig. 1135** The strongylid (also referred to as strongyle or trichostrongyle) egg is the helminth egg seen most often in ruminant and camelid feces. In fresh feces, eggs are thin shelled and oval in shape and contain a grapelike cluster of cells (morula). Development to the first larval stage occurs in the egg. Identification of eggs to a specific genus or species is not generally considered reliable because of substantial overlap in the sizes and shapes of eggs from different strongylid species.



**Fig. 1136** Once strongylid eggs are exposed to oxygen and adequate temperature, development to the first larval stage begins. In warm conditions, partially (as seen in this photo) or fully formed larvae may be seen when samples are collected from the ground. In older samples, the eggs will hatch releasing the larvae, which cannot be easily identified. *Emerita* oocysts are also present in this sample, representing several species, based on morphologic differences.

**Parasite:** *Nematodirus* spp. (Figs. 1.128, 1.137, 1.138)

**Common name:** Thread-necked worm.

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Several species are found in the small intestine of ruminants and camelids.

**Life Cycle:** Unlike most other strongylids, larvae develop to the infective stage within the egg. Ruminants are infected when they ingest the hatched infective larvae.

**Laboratory Diagnosis:** Large eggs present in routine or quantitative fecal flotation exams.

**Size:** 152–260 × 67–120 μm, depending on species (a similar egg is produced by *Marshallagia marshalli*, a parasite of sheep in the western United States)

**Clinical Importance:** Most species of *Nematodirus* do not usually cause clinical disease. *Nematodirus battus*, however, is an important cause of lamb diarrhea in some parts of the world. *Nematodirus* infections may cause disease in young camelids.

**Parasite:** *Strongyloides papillosus* (Figs. 1.133, 1.139)

**Taxonomy:** Nematode (order Rhabditida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of ruminants and camelids.

**Life Cycle:** Eggs shed in the feces hatch, releasing first-stage larvae. After a period of free-living development in the environment, infective third-stage larvae are produced that infect the host by ingestion or penetration of the skin. Transmammary infection also occurs.

**Laboratory Diagnosis:** Eggs are detected by routine flotation techniques. They are smaller than strongylid eggs and contain a larva when passed in the feces.

**Size:** 40–60 × 32–40 μm

**Clinical Importance:** Infection usually has no clinical significance, although very heavy infection may produce severe diarrhea in young animals.





Fig. 1.137 Egg of *Nematodirus* sp. This is one of the few strongylid eggs of ruminants that can be easily identified specifically because of its large size and two to eight distinctive, large cells inside the freshly passed egg. The eggs of *Marshallagia* spp. are similar in size to those of *Nematodirus*. *Marshallagia* spp. are found worldwide, but this genus is less common than *Nematodirus*. A much smaller typical strongylid egg is also present in the photo.



Fig. 1.138 Ovine fecal sample containing strongylid eggs (S), and a *Nematodirus battus* egg (N), which is usually browner in color than the eggs of other *Nematodirus* species.



Fig. 1.139 *Strongyloides* eggs may be confused with strongylid eggs. The two egg types can be easily distinguished because *Strongyloides* eggs are smaller and contain a fully formed larva when passed in the feces (arrow). Eggs are primarily seen in samples from immature animals. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Trichuris* spp. (Figs. 1.140–1.142)

Common name: Whipworm.

**Taxonomy:** Nematode (order Enoplida). Several species (*T. ovis*, *T. discolor*, etc.) occur in ruminants.

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum and colon of ruminants and camelids.

**Life Cycle:** Eggs produced by adults in the large intestine are passed in the feces. After a minimum of 3 weeks in the environment, eggs reach the infective stage and can infect a host when ingested.

**Laboratory Diagnosis:** Identification of brown, bipolar-plugged eggs in fecal flotation preparations.

Size: 70–80 × 30–42 μm

**Clinical Importance:** Eggs of *Trichuris* are often found in ruminant fecal samples. Clinical disease (diarrhea) is rare and associated with heavy infection.

**Parasite:** *Aonchotheca* (= *Capillaria*) spp. (Figs. 1.141–1.143)

**Taxonomy:** Nematode (order Enoplida). These parasites (*A. bovis* in cattle, *A. longipes* in sheep) were formerly included in the genus *Capillaria*. Camelids are also infected with *Aonchotheca* and at least one other capillariid species.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of ruminants and camelids.

**Life Cycle:** Parasite eggs are shed from the host in manure. Infection follows ingestion of infective eggs in the environment.

**Laboratory Diagnosis:** Eggs with bipolar plugs are detected by fecal flotation procedures. Although they are similar to *Trichuris* (whipworm) eggs, *Aonchotheca* spp. eggs are smaller.

Size: 45–50 × 22–25 μm

**Clinical Importance:** *Aonchotheca* infection in ruminants is considered clinically insignificant.



Fig. 1.140 *Trichuris* eggs are common in ruminant feces. They have a thick, brown shell and polar plug at each end. In this photo from an ovine fecal sample a *Trichuris* egg and a strongylid egg are present.



Fig. 1.141 This bipolar *Aonchotheca* (*Capillaria*) egg can be confused with *Trichuris* eggs but is smaller and less brown.



Fig. 1.142 Ruminant fecal sample containing both *Trichuris* (T) and *Aonchotheca* (A) eggs.

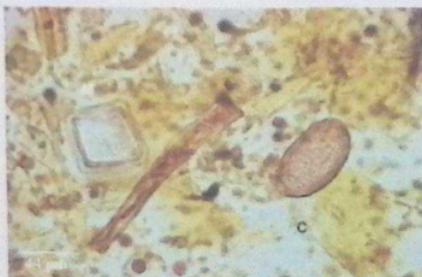


Fig. 1.143 Camelids can be infected with *Aonchotheca* sp. but are also parasitized by another capillid, which produces a distinctively larger egg with asymmetric bipolar plugs. Neither species appears to have clinical significance. A large capillid egg (C) is shown with a *Moniezia* egg (M).

Parasite: *Toxocara (Neoscaris) vitulorum* (Fig. 1.144)

Common name: Roundworm.

Taxonomy: Nematode (order Ascaridida).

Geographic Distribution: Worldwide, but rare in cattle in North America.

Location in Host: Small intestine of cattle and bison.

Life Cycle: Cattle are infected following the ingestion of larvated eggs in the environment. Larvae migrate into tissues and form a somatic reservoir that is activated in pregnancy. Egg-producing adult infections occur primarily in calves as a result of transmammary transmission.

Laboratory Diagnosis: Detection of typical ascarid-type eggs with flotation procedures.

Size: 75–95 × 60–75 μm

Clinical Importance: Infection with adult worms occurs in calves less than 6 months of age. Small to moderate infection may be tolerated without signs of disease, but diarrhea, weight loss, and death can occur in heavy infection.

Parasite: *Skirjabinema* spp. (Fig. 1.145)

Common name: Pinworm.

Taxonomy: Nematode (order Oxyurida).

Geographic Distribution: Worldwide.

Location in Host: Cecum of sheep, goats, some wild ruminant species, and some camelids.

Life Cycle: Female worms deposit eggs on the perianal skin. Eggs fall off the host, become dispersed in the environment, and are eaten by other animals.

Laboratory Diagnosis: Eggs are rarely seen in routine fecal exams as they are not deposited in feces.

Size: 47–63 × 27–36 μm

Clinical Importance: Clinically insignificant.





Fig. 1144 *Toxocara vitellorum* eggs have the thick shell typical of ascarids. *Toxocara* is rarely seen in cattle in North America. Photo courtesy of Dr. Gil Myers, Myers Parasitological Service, Magnolia, TN and Dr. Eugene Lyons, Department of Veterinary Science, University of Kentucky, Lexington, KY.



Fig. 1145 *Strjabonema* (Sk), the ruminant pinworm. Pinworm eggs often appear flattened on one side. These eggs are rarely found in fecal exams because they are not passed in the feces. A strongylid egg (S) and *Eimeria* (E) oocyst are also present. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Dictyocaulus* spp. (Figs. 1.151 and 1.152)

Common name: Lungworm.

**Taxonomy:** Nematode (order Strongylida). Species include *D. viviparus* (cattle, camelids), *D. filaria* (sheep, goat, camelids), *D. cameli* (camel).

**Geographic Distribution:** Worldwide.

**Location in Host:** Trachea, bronchi, and bronchioles.

**Life Cycle:** First-stage larvae are passed in the feces of the host. Infective third-stage larvae develop on pasture and are ingested during grazing. Larvae migrate from the intestine to the respiratory tract and become mature.

**Laboratory Diagnosis:** The Baermann test is used to detect first-stage larvae in fresh feces. Some larvated eggs may also be present in fresh feces.

Size:	<i>D. viviparus</i>	300–360 $\mu\text{m}$
	<i>D. filaria</i>	550–580 $\mu\text{m}$

**Clinical Importance:** Heavy infections may cause severe respiratory signs, especially in cattle. Disease is usually seen in young animals before immunity develops.

**Parasite:** *Moniezia* spp. (Figs. 1.143, 1.153, 1.154)

Common name: Tapeworm.

**Taxonomy:** Cestode. Species include *M. benedeni* and *M. expansa*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of ruminants and camelids.

**Life Cycle:** Tapeworm eggs are shed in segments from the host. Ruminants are infected following ingestion of the intermediate host (free-living pasture mites) containing the tapeworm larvae.

**Laboratory Diagnosis:** Eggs may be found in fecal flotation tests, but infection is usually recognized when owners see tapeworm segments on the animal or in the environment.

Size: 65–75  $\mu\text{m}$  in diameter

**Clinical Importance:** In general, little clinical importance, although there are anecdotal reports that heavy infection may cause reduced growth in young animals.



Fig. 3.151 *Dictyocaulus viviparus* first-stage larva. Intestinal cells contain characteristic dark food granules. *Dictyocaulus filaria* larvae have a small knob at the anterior end that is not present in *D. viviparus* larvae. Photo courtesy of Dr. Mangandan Lejcune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 3.152 Although *Dictyocaulus* larvae are most often seen in fecal samples, unhatched eggs may also be found in feces and samples collected from the trachea. The dark food granules are evident even in this unhatched *Dictyocaulus* larva. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 3.153 Eggs of *Moniezia* are often square or triangular, unlike the more common round or oval shape of other parasite eggs. The presence of the embryo with its six hooks clearly identifies these structures as tapeworm eggs. In this egg, four hooks are visible.

**Parasite:** *Dictyocaulus* spp. (Figs. 1.151 and 1.152)

Common name: Lungworm.

**Taxonomy:** Nematode (order Strongylida). Species include *D. viviparus* (cattle, camelids), *D. filaria* (sheep, goat, camelids), *D. cameli* (camel).

**Geographic Distribution:** Worldwide.

**Location in Host:** Trachea, bronchi, and bronchioles.

**Life Cycle:** First-stage larvae are passed in the feces of the host. Infective third-stage larvae develop on pasture and are ingested during grazing. Larvae migrate from the intestine to the respiratory tract and become mature.

**Laboratory Diagnosis:** The Baermann test is used to detect first-stage larvae in fresh feces. Some larvated eggs may also be present in fresh feces.

Size:	<i>D. viviparus</i>	300–360 $\mu\text{m}$
	<i>D. filaria</i>	550–580 $\mu\text{m}$

**Clinical Importance:** Heavy infections may cause severe respiratory signs, especially in cattle. Disease is usually seen in young animals before immunity develops.

**Parasite:** *Moniezia* spp. (Figs. 1.143, 1.153, 1.154)

Common name: Tapeworm.

**Taxonomy:** Cestode. Species include *M. benedeni* and *M. expansa*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of ruminants and camelids.

**Life Cycle:** Tapeworm eggs are shed in segments from the host. Ruminants are infected following ingestion of the intermediate host (free-living pasture mites) containing the tapeworm larvae.

**Laboratory Diagnosis:** Eggs may be found in fecal flotation tests, but infection is usually recognized when owners see tapeworm segments on the animal or in the environment.

Size: 65–75  $\mu\text{m}$  in diameter

**Clinical Importance:** In general, little clinical importance, although there are anecdotal reports that heavy infection may cause reduced growth in young animals.



Fig. 1381 *Dictyocaulus viviparus* first-stage larva. Intestinal cells contain characteristic dark food granules. *Dictyocaulus filaria* larvae have a small knob at the anterior end that is not present in *D. viviparus* larvae. Photo courtesy of Dr. Manigandan Lejune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 1382 Although *Dictyocaulus* larvae are most often seen in fecal samples, unhatched eggs may also be found in feces and samples collected from the trachea. The dark food granules are evident even in this unhatched *Dictyocaulus* larva. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 1383 Eggs of *Moniezia* are often square or triangular, unlike the more common round or oval shape of other parasitic eggs. The presence of the embryo with its six hooks clearly identifies these structures as tapeworm eggs. In this egg, four hooks are visible.

**Parasite:** *Thysanosoma, Stilesia* (Fig. 1.155)

**Taxonomy:** Cestodes.

**Geographic Distribution:** *Stilesia* is found in Europe, Africa, and Asia, while *Thysanosoma* is confined to North and South America. In the United States, its distribution appears to be limited to the western states.

**Location in Host:** Bile ducts of ruminants, especially sheep, and camelids.

**Life Cycle:** Although these tapeworms have not been extensively studied, it is thought that their intermediate hosts may be oribatid mites and psocid insects. Like *Moniezia*, the definitive host is infected following ingestion of the intermediate host.

**Laboratory Diagnosis:** Tapeworm segments are passed in the feces and eggs may be found in fecal flotation tests.

**Size:** Approximately  $30 \times 20 \mu\text{m}$

**Clinical Importance:** These tapeworms have no economic importance unless they are present in large enough numbers to cause liver condemnation.

**Parasite:** *Fasciola hepatica* (Figs. 1.156–1.158)

**Common name:** Liver fluke.

**Taxonomy:** Trematode.

**Geographic Distribution:** Worldwide. A similar species, *F. gigantica*, is also found in Africa, Asia, and Hawaii.

**Location in Host:** Adults in the bile ducts of cattle, sheep, goats, camelids, and a variety of other animals, including dogs, horses, and humans.

**Life Cycle:** Miracidia hatch from the eggs and invade an appropriate snail host. Cercariae emerging from the snail encyst on vegetation and are ingested by host animals. Larvae leave the gastrointestinal tract and migrate through the liver to reach the bile ducts.

**Laboratory Diagnosis:** Large brown eggs are detected using a sedimentation procedure. Eggs may be difficult to detect and not indicative of the level of infection in a herd. A commercially available apparatus, the Flukefinder, simplifies the sedimentation procedure (see the section "Fecal Sedimentation").

**Size:**  $130\text{--}150 \times 63\text{--}90 \mu\text{m}$

**Clinical Importance:** *Fasciola* infections in ruminants may cause significant production losses. Sheep are particularly susceptible, and heavy infection may be fatal. Chronically infected animals can develop anemia and unthriftiness. A similar parasite, *Fascioloides magna*, is the liver fluke of white tailed deer. Migration of *F. magna* larvae in other ruminants, especially small ruminants, may cause hepatic disease.



Fig. 1.154 Owners may be alarmed by the presence of *Moniezia* segments in the feces of their animals, although tapeworms have little clinical significance. Tapeworm segments are seen most often in the manure of young animals. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 1.155 Packet of *Thyxanorona* eggs from a sheep. The eggs lack the pyriform apparatus seen in *Moniezia* eggs. Hooks can be seen in some of the eggs. The entire packet of eggs is  $124 \times 62 \mu\text{m}$ . Photo courtesy of Dr. Ellis C. Greiner, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1.156 *Fasciola hepatica* egg. These large eggs have an operculum (arrow) and look similar to eggs of rumen flukes, although *Paramphistomum* eggs are slightly larger (about  $160 \mu\text{m}$ ) and less brown in color.

**Parasite:** *Paramphistomum* spp. (Figs. 1.157 and 1.158)

Common name: Rumen fluke.

**Taxonomy:** Trematode. Other genera belonging to this family include *Cotylophoron* and *Calicophoron*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Adult flukes in the rumen of cattle, sheep, other ruminants, and camels.

**Life Cycle:** Eggs passed in the feces of the host animal hatch in water, liberating miracidia, which infect snails. Following development in the snail, cercariae are released, which encyst on vegetation. Definitive hosts are infected by ingesting fluke metacercariae while grazing.

**Laboratory Diagnosis:** Eggs of paramphistomes are similar to those of *Fasciola*. They are best recovered using a sedimentation procedure, but examination of fecal material will not detect immature flukes, which are the most pathogenic stage of infection.

**Size:** Approximately  $114\text{--}175 \times 65\text{--}100 \mu\text{m}$ , depending on species.

**Clinical Importance:** Clinical disease is rare in North America. In other parts of the world, larval paramphistomes in the duodenum and upper ileum are reported to cause enteritis leading to diarrhea, emaciation, and death in severe cases.

**Parasite:** *Dicrocoelium dendriticum* (Fig. 1.159)

**Taxonomy:** Trematode.

**Geographic Distribution:** Europe, Asia, sporadic occurrence in North America.

**Location in Host:** Bile ducts of domestic and wild ruminants, pigs, dogs, horses, rabbits.

**Life Cycle:** Larvae in eggs are ingested by snails. Ants act as the second intermediate host, and the final host is infected while grazing. Larval flukes enter bile ducts directly and do not migrate through the liver.

**Laboratory Diagnosis:** A sedimentation test will detect the small, brown, operculate eggs in the feces.

**Size:**  $38\text{--}45 \times 22\text{--}30 \mu\text{m}$

**Clinical Importance:** In heavy infections, extensive cirrhosis of the liver can develop, leading to anemia and weight loss.



Fig. 1.157 *Fasciola hepatica* (F) and *Paramphistomum* (P) eggs. The two eggs are very similar but the browner color of *Fasciola* is easily seen in this photo. The operculum of each egg is indicated by an arrow. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1.158 Egg of *Fascioloides magna*, the liver fluke of white-tailed deer. These eggs are similar in size and appearance to those of *F. hepatica*. Because patent infections do not develop in domestic livestock, eggs will not be present in their feces.



Fig. 1.159 *Dicrocoelium dendriticum* eggs contain a fully formed miracidium, tend to be flattened on one side, and are smaller than eggs of *Fasciola* and the paramphistome flukes.

**Parasite:** *Eurytrema* spp. including *E. coelomaticum*, *E. pancreaticum* (Fig. 1.160)

**Taxonomy:** Trematode.

**Geographic Distribution:** Asia, parts of South America.

**Location in Host:** Adult flukes are found in the pancreatic ducts of small ruminants, cattle, camels, pigs, and occasionally humans. Flukes are also occasionally found in the bile ducts and small intestine.

**Life Cycle:** The eggs produced by adult flukes leave the host in manure. Snails are the first intermediate host. A grasshopper or cricket second intermediate host transmits the infection when ingested by the final host.

**Laboratory Diagnosis:** A sedimentation test will detect the small, brown eggs in the feces.

Size: 44–48 × 23–36 µm

**Clinical Importance:** Many infections are subclinical. Heavy worm burdens can cause fibrosis of the ducts and pancreatic atrophy, resulting in weight loss and poor condition.

**Parasite:** *Schistosoma* spp. (Fig. 1.161)

**Taxonomy:** Trematode. Several species infect ruminants, camels, horses, and pigs, including *S. bovis*, *S. mattheei*, and *S. japonicum*.

**Geographic Distribution:** Africa, Asia.

**Location in Host:** Most important species are found in the portal mesenteric veins of the host.

**Life Cycle:** Eggs in host feces hatch in water, releasing the miracidia, which enter the snail intermediate host. Cercariae produced by multiplication within the snail are released and penetrate the skin of the definitive host. There is no second intermediate host in the life cycle.

**Laboratory Diagnosis:** A saline sedimentation procedure is used to detect eggs in manure. Fecal examination is most useful in early infection because egg production declines as infection progresses. Eggs do not have an operculum and most are spindle-shaped. In some species, a spine is present on one end of the egg.

Size: 130–280 × 38–85 µm, depending on species

**Clinical Importance:** Disease results from the host reaction to the presence of parasite eggs in tissue. Clinical signs may occur in heavy infections, including diarrhea, anemia, and wasting.



Fig. 1.160 The eggs of *Eurytrema* are similar in appearance to those of *Dicrocoelium*. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.

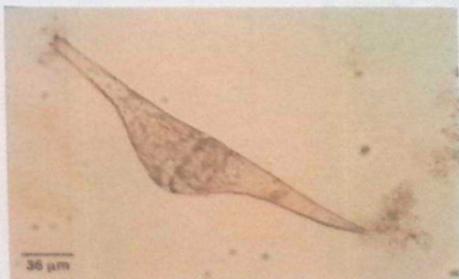


Fig. 1.161 Ruminant schistosomes typically produce a spindle-shaped egg. This egg of *Schistosoma spindale*, an Asian species, has a spine at one end. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.

## Horses

In comparison to dogs, cats, and ruminants, the diversity of parasite eggs and cysts frequently encountered in equine feces is much reduced. The most common finding in equine samples is the strongylid egg. Horses are infected with many strongylid species, although individual species cannot be determined by morphologic characteristics of eggs alone. The parasites shown in this section can also infect other equid species. Parasites illustrated in other sections may not be extensively covered here and references are given to figures elsewhere in the book.

### Helminth Eggs, Larvae and Protozoan Cysts found in freshly voided feces of Horses

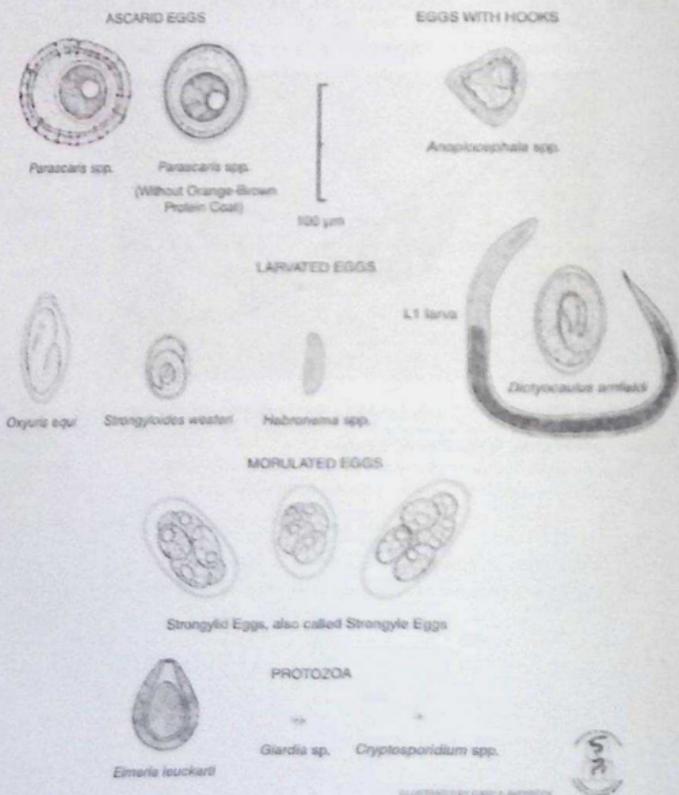


Fig. 1.10 Parasites found in fecal samples of horses. Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

Table 1.11. Representative treatments for selected parasites of horses

Parasite	Effective treatments	Dose, route, and regimen
<i>Eimeria leuckarti</i> <i>Giardia</i> sp. <i>Cryptosporidium</i> spp.	None indicated	NA, rarely associated with clinical disease
<i>Strongylus</i> spp. (large strongyles)	Fenbendazole, ivermectin, moxidectin, oxbendazole, pyrantel pamoate, pyrantel tartrate	Administer according to label directions
Cyathostomins (small strongyles)	*Fenbendazole, ivermectin, moxidectin, †oxibendazole, †pyrantel pamoate, †pyrantel tartrate	Administer according to label directions
<i>Parascaris equorum</i>	Fenbendazole, †ivermectin, †moxidectin, oxbendazole, †pyrantel pamoate, pyrantel tartrate	Administer according to label directions
<i>Oxyuris equi</i>	Fenbendazole, †ivermectin, †moxidectin, oxbendazole, pyrantel pamoate, pyrantel tartrate	Administer according to label directions
<i>Trichostrongylus axei</i>	Ivermectin, moxidectin	Administer according to label directions
<i>Strongyloides westeri</i>	Ivermectin, oxbendazole	Administer according to label directions
<i>Habronema muscae</i> <i>Habronema microstoma</i> <i>Draconia megastoma</i>	Ivermectin, †moxidectin	Administer according to label directions
<i>Dictyocaulus arnfieldi</i>	Ivermectin	Administer according to label directions
<i>Anoplocephala perfoliata</i>	Praziquantel  Pyrantel pamoate	Administer according to label directions; equine formulations available in combination with macrocyclic lactones  Administer according to label directions for tapeworm treatment

\* Resistance or

† suboptimal efficacy to this treatment has been reported.

‡ Not label-approved against *H. microstoma* or *D. megastoma*.

NA, not applicable.

Additional information on parasitic treatments can be found in Chapter 7.

### Protozoan Parasites

**Parasite:** *Eimeria leuckarti* (Fig. 1.163)

**Taxonomy:** Protozoa (coccidia).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of horses and donkeys.

**Life Cycle:** Oocysts leave the host in the manure. Sporulation occurs in the environment, and new hosts are infected by ingestion of infective oocysts.

**Laboratory Diagnosis:** Infection is diagnosed by finding the large, deep-brown oocysts in the feces. A sedimentation procedure has been recommended, but oocysts can also be seen with flotation procedures.

**Size:** 80–88 × 55–59 μm

**Clinical Importance:** Infection appears to have little clinical significance in horses, although rare cases of diarrhea have been reported. Infections are seen only in young animals.

**Parasite:** *Giardia duodenalis*, *Cryptosporidium* spp. (Figs. 1.164 and 1.165)

**Taxonomy:** Protozoa (*Giardia*, flagellate; *Cryptosporidium*, coccidia).

**Geographic Location:** Worldwide.

**Location in Host:** Small intestine.

**Laboratory Diagnosis:** As in other hosts, *Giardia* cysts are most easily detected with 33% ZnSO<sub>4</sub> centrifugal flotation, and *Cryptosporidium* oocysts with Sheather's sugar centrifugal flotation. Both organisms can also be found in fecal smears with appropriate stains and by non-host-specific immunodiagnostic or molecular tests.

**Clinical Importance:** Both infections occur most frequently in young animals but are rarely associated with clinical disease.



Fig. 1.163 The large size and deep-brown color of the oocysts of *E. leuckarti* make them very distinctive. They are seen in feces of young horses.



Fig. 1.164 Fecal flotation test containing iodine-stained *Giardia* cysts.



Fig. 1.165 *Cryptosporidium* oocysts in a sugar flotation preparation.

### Helminth Parasites

**Parasite:** Equine Strongylid Parasites (Figs. 1.166–1.168, 1.171, 1.179)

**Common name:** Various, including bloodworm, small and large strongyle.

**Taxonomy:** Nematode (order Strongylida). Numerous genera belong to this group, including the large strongyles (e.g., *Strongylus vulgaris*), the small strongyles (cyathostomins), and *Trichostrongylus axei*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum and colon (with the exception of *T. axei*, a parasite of the stomach).

**Life Cycle:** Eggs released by adult worms in the large bowel develop in feces in the environment. Infective larvae on pasture are ingested by grazing horses. Large and small strongyles undergo a period of development in the intestinal wall (small strongyles) or in extra-intestinal tissue (large strongyles) before maturing in the bowel lumen.

**Laboratory Diagnosis:** Eggs are detected on routine fecal flotation. Eggs are similar in appearance and are not routinely identified specifically. Quantitative egg counts are used in targeted selective treatment programs and testing for drug efficacy.

**Size:** Variable, with considerable overlap among species; eggs approximately 60–120 × 35–60 μm

**Clinical Importance:** Virtually all grazing horses are infected with strongylid parasites. Many low to moderate infections are subclinical, although they may cause reduced weight gain and performance. Young, nonimmune animals are most susceptible to clinical disease, which may include diarrhea, colic, and hypoproteinemia.



Fig. 1.166 Equine strongylid (strongyle) eggs in fresh fecal samples are typical of the order, with a thin shell surrounding a central group of cells (morula). In warm weather, larvae may form within 1–2 days. The two eggs shown here demonstrate the range of sizes in this group of worms, but in most cases there is too much overlap to identify parasite species. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

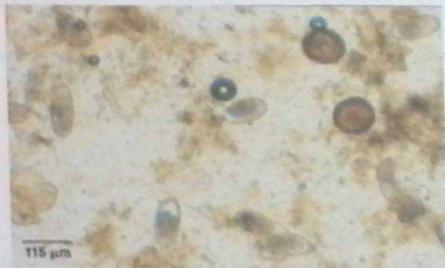


Fig. 1.167 Several equine strongylid eggs are present in this fecal sample. Two *Parasarcis* (roundworm) eggs are also present.



Fig. 1.168 Equine small strongylid larvae (arrows) in manure. These small (less than 2 cm) red larvae may be present in large numbers in manure of horses with acute larval cyathostomiasis.

**Parasite:** *Parascaris* spp. (Figs. 1.167, 1.169–1.171)

Common name: Roundworm.

**Taxonomy:** Nematode (order Ascaridida). This parasite genus was previously generally identified as *P. equorum*, but investigators have recently found that *P. univalens* is a common species.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of horses and other equids.

**Life Cycle:** Infective larvae develop in eggs passed in the feces of horses. Infection occurs by ingestion of larvated eggs. Larvae migrate through the liver and lungs of the host before returning to the small intestine to mature.

**Laboratory Diagnosis:** Flotation procedures will detect the typical thick-shelled ascarid eggs.

Size: 90–100  $\mu\text{m}$  in diameter

Following treatment with some anthelmintics, adult ascarids may be passed in manure. These worms will be much larger than any other equine helminths, with females reaching 50 cm in length.

**Clinical Importance:** Adult worms are common in young horses, infrequent in adults. Heavy infections can cause respiratory signs (from migrating larvae), ill-thrift, colic, diarrhea, and intestinal obstruction that may be fatal.



Fig. 1.169 *Parascaris* eggs are typical, thick-shelled ascarid eggs containing a single cell when passed in the feces. Figure 1.167 shows *Parascaris* eggs at a lower magnification.

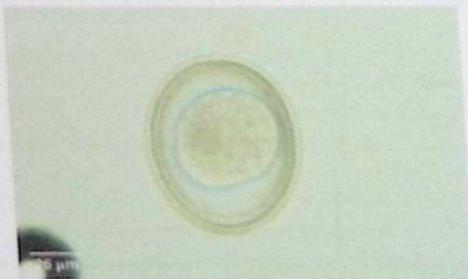


Fig. 1.170 *Parascaris* eggs may lose the rough, proteinaceous coat on the eggshell, but they can still be identified as ascarid eggs by the thick shell and single cell inside the freshly passed egg.



Fig. 1.171 Larvated strongylid egg and *Parascaris* egg that has undergone the first cell division. The fecal sample containing these eggs was fresh when collected but was not examined for some time, allowing development to occur.

**Parasite:** *Strongyloides westeri* (Fig. 1.172)

Common name: Threadworm.

**Taxonomy:** Nematode (order Rhabditida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** Patent infections develop primarily by transmammary infection of foals. Larvated eggs passed in the feces of foals lead to the development of infective larvae that can penetrate the skin or be ingested. In adult horses, larvae migrate to tissues and form a somatic larval reservoir.

**Laboratory Diagnosis:** Small, larvated eggs are detected by flotation procedures.

Size: 40–52 × 32–40 μm

**Clinical Importance:** Clinical disease occurs only in foals. Heavy burdens can produce severe diarrhea and dehydration. Respiratory signs may develop associated with larval migration.

**Parasite:** *Oxyuris equi* (Figs. 1.173 and 1.174)

Common name: Pinworm.

**Taxonomy:** Nematode (order Oxyurida). *Probstmayria vivipara* is a less common pinworm of horses.

**Geographic Distribution:** Worldwide.

**Location in Host:** Large intestine of horses and other equids.

**Life Cycle:** Horses ingest infective eggs. Adult female worms migrate to the perianal region and lay clusters of sticky eggs. These eventually are rubbed off the horse and contaminate the environment.

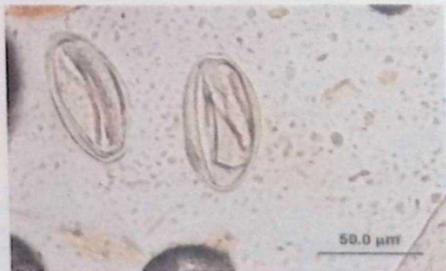
**Laboratory Diagnosis:** Because eggs are attached to hairs in the perianal region, they are not often seen in flotation tests. A more successful procedure for recovering eggs is the "Scotch tape test." A piece of clear adhesive tape is touched to the skin in the perianal area and then taped onto a microscope slide and examined. Pinworm eggs can easily be seen through the tape.

Size: 85–95 × 40–45 μm

**Clinical Importance:** Egg-laying activities of the female worms produce intense pruritus. Horses bite at and rub the perineal region, leading to a "rat-tailed" appearance and possible secondary trauma.



**Fig. 1.172** Egg of *Strongylides westeri*, the horse threadworm. These eggs are usually seen only in the feces of young horses. *Strongylides* eggs are already larvated when passed in the feces, and they are smaller than strongylid eggs. Figure 1.139 shows both *Strongylodes* and strongylid eggs of ruminants, which have a similar size relationship to the species found in horses. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



**Fig. 1.173** *Oxyuris* eggs are asymmetrical with a single polar plug. Eggs embryonate rapidly and may be seen with a larva inside.



**Fig. 1.174** Adult pinworms are occasionally seen in the feces of horses. Adult female *Oxyuris* can reach a maximum size of 10 cm and can be recognized by their thin, pointed tails. Male worms are much smaller. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**Parasite:** *Habronema microstoma*, *H. muscae*, *Draschia megastoma* (Fig. 1.175)

**Taxonomy:** Nematodes (order Spirurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Stomach of horses and donkeys.

**Life Cycle:** Adult worms are found in the stomach; *D. megastoma* in tumor-like masses near the margo plicatus. Larvated eggs are passed in the feces and are ingested by fly larvae intermediate hosts. Infective worm larvae deposited by adult flies around the lips of horses make their way into the mouth and to the stomach.

**Laboratory Diagnosis:** The larvated eggs passed in the feces are too dense to float in most flotation solutions. A sedimentation procedure is recommended for detection.

**Size:** 40–80 × 10–20 µm

**Clinical Importance:** Gastritis resulting in poor growth may develop. If flies deposit third-stage larvae on a wound, a condition known as “summer sore” can occur, in which larvae survive and prevent wound healing.

**Parasite:** *Dictyocaulus arnfieldi* (Figs. 1.152, 1.176 and 1.177)

Common name: Lungworm.

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Bronchi and bronchioles of horses and donkeys.

**Life Cycle:** Adult worms in the respiratory tract produce larvated eggs that hatch before or soon after leaving the host. Eggs and/or larvae are coughed up, swallowed, and passed in the feces. Larvae develop to the infective third stage in the environment and are ingested by grazing horses.

**Laboratory Diagnosis:** Larvated eggs may be detected in feces with flotation tests. However, eggs hatch rapidly or even before leaving the host, so the Baermann test for the first-stage larvae is the preferred technique for diagnosis. While lungworms readily mature in donkeys, they may not mature in horses, making diagnosis more difficult.

**Size:** Eggs 74–96 × 46–58 µm

Larvae 420–480 µm

**Clinical Importance:** Bronchitis and pneumonia may develop. Infections appear to be tolerated better by donkeys than by horses.



Fig. 1.175 Larvated egg of *Habronema* sp. The larva is surrounded by a thin shell. *Draschia* sp. eggs are similar. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 1.176 The only parasitic nematode larvae that would be expected in the fresh feces of horses are those of *Dictyocaulus*. See also Figure 1.152 for *Dictyocaulus* eggs that may be seen in tracheal fluid samples. Photo courtesy of Dr. Craig Reinemeyer, East Tennessee Clinical Research, Knoxville, TN.



Fig. 1.177 Larvae of *Dictyocaulus arnfieldi*, the equid lungworm, have a small terminal projection at the posterior end (arrow) that can be seen with higher magnification.

**Parasite:** *Anoplocephala perfoliata*, *A. magna*, and *Paranoplocephala mamillana* (Figs. 1.178 and 1.179)

Common name: Tapeworm.

**Taxonomy:** Cestodes.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of horses and donkeys.

**Life Cycle:** Eggs passed in the feces are ingested by free-living pasture mites. Horses are infected during grazing when they ingest mites containing tapeworm cysticeroid larvae.

**Laboratory Diagnosis:** Flotation procedures for detection of eggs in fecal samples are used, but false-negative results are common. A test for detection of antibodies to *Anoplocephala* infection is available.

Size:	<i>A. perfoliata</i>	65–80 $\mu\text{m}$ in diameter
	Other species	50–60 $\mu\text{m}$

**Clinical Importance:** Most tapeworm infections are asymptomatic. Disease has been associated with *A. perfoliata*. These parasites cluster at the ileo-cecal junction, where heavy infection can cause ulceration leading to perforation or intussusception.

Horses may also be infected with some of the trematode (flake) parasites that affect ruminants. For information on these parasites, see Figures 1.156 and 1.161.



Fig. 3.178 The egg of the equine tapeworm *Anoplocephala* is similar in appearance to that of the ruminant tapeworm *Moniezia*. The eggs of both genera are often irregularly shaped. A pyriform apparatus surrounds the embryo, which has six hooks (hexacanth).

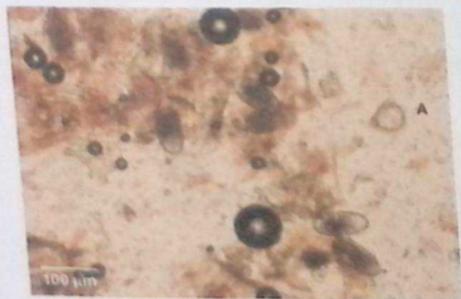


Fig. 3.179 *Anoplocephala* egg (A) and several equine strongylid eggs.

## Swine

## Helminth Eggs, and Protozoan Cysts found in freshly voided feces of Pigs

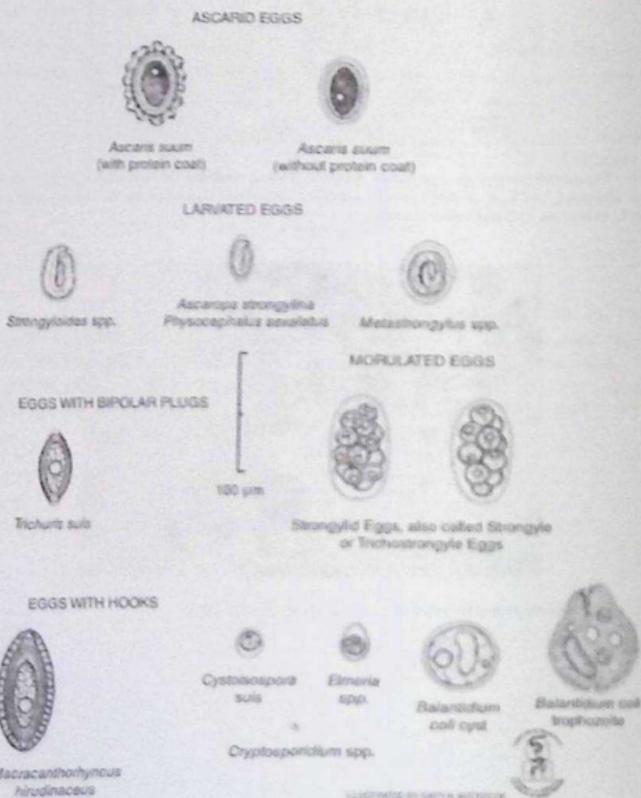


Fig. 1.18C Parasites found in fecal samples of pigs. Figure courtesy of Dr. Bert Stromberg and Mr. Gary Averbeck, College of Veterinary Medicine, University of Minnesota, Minneapolis, MN.

Table 1.12. Representative treatments for selected parasites of swine

Parasite	Effective treatments	Dose, route, and regimen
<i>Cystoisospora suis</i>	*Toltrazuril	20 mg/kg PO, once
<i>Ascaris suum</i>	Dichlorvos, doramectin, fenbendazole, †hygromycin B, ivermectin, levamisole, piperazine, pyrantel tartrate	Administer according to label directions
<i>Trichuris suis</i>	Dichlorvos, fenbendazole, †hygromycin B	Administer according to label directions
<i>Oesophagostomum</i> spp.	Dichlorvos, doramectin, fenbendazole, †hygromycin B, ivermectin, levamisole, pyrantel tartrate	Administer according to label directions
<i>Metastrongylus</i> spp.	Doramectin, fenbendazole, ivermectin, levamisole	Administer according to label directions
<i>Strongyloides ransomi</i>	Doramectin, ivermectin, levamisole	Administer according to label directions
<i>Stephanurus dentatus</i>	Doramectin, fenbendazole, ivermectin, levamisole	Administer according to label directions
<i>Hyostrongylus rubidas</i>	Doramectin, fenbendazole, ivermectin	Administer according to label directions
<i>Macracanthorhynchus hirudinaceus</i>	†Ivermectin	Administer according to label directions

\* Not label-approved for use in pigs in the United States.

† Label-approved as an aid in the control of infection.

‡ Reported effective when administered according to label directions.

Additional information on parasite treatments can be found in Chapter 7.

### Protozoan Parasites

**Parasite:** *Cystoisospora (Isospora) suis* (Fig. 1.181)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** Oocysts are shed in the feces and can sporulate rapidly in warm weather. Transmission occurs in the neonatal period with sows serving as a source of oocysts. Neonates are infected when they ingest sporulated (infective) oocysts.

**Laboratory Diagnosis:** *Cystoisospora* oocysts are detected in feces with flotation procedures. However, oocysts are shed at such a low rate in asymptomatic carriers that detection is very difficult. In clinically affected neonatal pigs, disease usually develops before oocysts are shed, and intestinal mucosal impression smears at necropsy are more effective for diagnosis.

**Size:** Oocysts are nearly spherical and approximately  $18 \times 20 \mu\text{m}$

**Clinical Importance:** *Cystoisospora suis* can produce neonatal diarrhea in pigs (5–10 days of age) that may be severe and cause death.

**Parasite:** *Eimeria* spp. (Figs. 1.182 and 1.183)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia). Eight species of porcine *Eimeria* have been described, including *E. scabra*, *E. debbieki*, *E. porci*, and *E. spinosa*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Intestinal tract; location depends on species and stage of development.

**Life Cycle:** Oocysts are shed in the feces and sporulate rapidly in warm weather. Pigs are infected when they ingest sporulated (infective) oocysts.

**Laboratory Diagnosis:** *Eimeria* oocysts are detected in feces with flotation procedures.

**Size:** Oocysts vary with species, with a range of  $11\text{--}35 \times 9\text{--}20 \mu\text{m}$

**Clinical Importance:** *Eimeria* spp. infections are common in swine but rarely produce clinical disease.

Swine may also be infected with *Cryptosporidium parvum* and *Giardia duodenalis*. These two protozoan parasites appear to have little clinical significance but may have zoonotic importance. For further information, see sections related to these parasites in the small animal and ruminant sections.

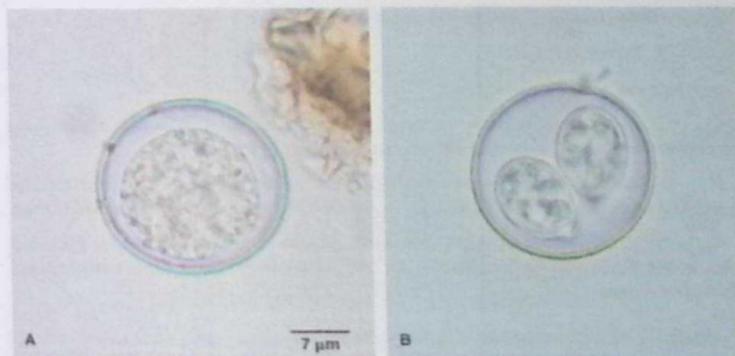


Fig. 1.181 Unsporulated (A) and sporulated (B) oocyst of *Cystoisospora suis*. Photos courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 1.182 Oocysts of *E. porci* (P) and *E. scabra* (S), two of the eight *Eimeria* species described from pigs. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 1.183 Numerous *Eimeria* oocysts in a porcine fecal sample. Although common in pigs, *Eimeria* spp. infections have little clinical importance. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Balantidium* (= *Neobalantidium*) *coli* (Figs. 1.184–1.186)

**Taxonomy:** Protozoa (ciliate).

**Geographic Distribution:** Worldwide.

**Location in Host:** Large intestine of swine; may also occasionally infect humans and other primates, camels, dogs, and other animals.

**Life Cycle:** Infection follows ingestion of cysts shed into the environment from infected swine. The only other stage of the life cycle is the motile trophozoite in the intestinal tract.

**Laboratory Diagnosis:** Motile trophozoites can be seen in direct fecal smears. Flotation tests are more sensitive for detecting cysts. The kidney-bean-shaped macronucleus is a distinctive feature.

Size:	Trophozoites	50–150 × 40–65 μm
	Cysts	40–60 μm

**Clinical Importance:** *Balantidium* infection is generally asymptomatic in swine, although bloody diarrhea may occur in some hosts.

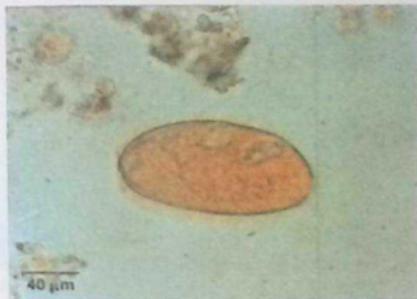


Fig. 1184 *Babantidium coli* trophozoite in a direct saline smear. The cilia covering trophozoites can be seen as a halo surrounding the organism. Trophozoites are usually destroyed by fecal flotation procedures. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.



Fig. 1185 The kidney-bean-shaped macronucleus, characteristic of ciliates, is present in this unstained *Babantidium* cyst in a porcine fecal sample (arrow). The macronucleus is usually easily seen in stained specimens. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 1186 Hyperosmotic flotation solutions can cause distortion of *Babantidium coli* cysts. This cyst, found in a ZnSO<sub>4</sub> centrifugal fecal flotation preparation, is partially collapsed. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

### Helminth Parasites

**Parasite:** *Ascaris suum* (Fig. 1.187)

Common name: Roundworm.

**Taxonomy:** Nematode (order Ascaridida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** Pigs are infected when they ingest infective eggs in the environment. Following migration through the liver and lungs, adults develop in the small intestine. Like other ascarids, *A. suum* has very resistant eggs that can survive for years in the environment.

**Laboratory Diagnosis:** Eggs can be detected in feces with routine flotation procedures.

Size: 50–70 × 40–60 μm

**Clinical Importance:** *Ascaris suum* is a common and important parasite of swine, even in confinement systems. Larval migration through liver and lung may cause liver condemnation and predispose pigs to bacterial or viral pneumonia. Adult worms in the small intestine may cause reduced growth. Larvae can also migrate and cause disease in other animals and humans.

**Parasite:** *Hyostrongylus rubidus*, *Oesophagostomum* spp. (Figs. 1.188, 1.191)

Common name: Red stomach worm (*Hyostrongylus*), nodular worm (*Oesophagostomum*).

**Taxonomy:** Nematodes (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Stomach (*Hyostrongylus*) and large intestine (*Oesophagostomum*) of wild and domestic swine.

**Life Cycle:** These parasites have a direct life cycle; eggs are shed in the feces and hatch in the environment. Swine are infected following ingestion of third-stage larvae.

**Laboratory Diagnosis:** Typical strongylid eggs are detected in fecal samples by flotation techniques. Eggs of *Hyostrongylus* and *Oesophagostomum* cannot be distinguished from each other or from less common strongylid parasites of swine, including *Trichostrongylus axei* and *Globocephalus*.

Size: 69–85 × 39–45 μm

**Clinical Importance:** These parasites are common in pastured swine. *Hyostrongylus* may cause ulcerative gastritis, resulting in anemia and reduced production. Host response to *Oesophagostomum* larvae in the wall of the intestinal tract leads to the development of nodules that, in heavy infections, can lead to enteritis and reduced production.



Fig. 1167 Like other ascarid eggs, those of *Ascaris suum* contain a single cell surrounded by a thick shell when first passed in the feces. In some cases, the rough, brown outer layer of the shell may be absent, as seen in two of the eggs in this figure. *Ascaris suum* eggs are very similar in appearance to those of the human ascarid, *A. lumbricoides*.



Fig. 1168 *Hyostromylin* and *Oesophagostomum* are the most common and important strongylid parasites of swine and produce indistinguishable eggs typical of this group of nematodes. Fecal culture and identification of third-stage larvae are necessary to identify parasite genus.

**Parasite:** *Trichuris suis* (Fig. 1.189)

Common name: Whipworm.

**Taxonomy:** Nematode (order Enoplida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Large intestine of wild and domestic pigs.

**Life Cycle:** *Trichuris* spp. have a direct life cycle. Pigs are infected following ingestion of infective eggs in the environment. Eggs leave the host in manure and are able to survive for long periods in the environment.

**Laboratory Diagnosis:** Eggs can be detected in routine fecal flotation tests.

Size: 50–60 × 21–25 µm

**Clinical Importance:** This common helminth infection of swine may cause diarrhea and dehydration. Severe infections can produce bloody diarrhea.

**Parasite:** *Strongyloides ransomi* (Figs. 1.190 and 1.191)

Common name: Threadworm.

**Taxonomy:** Nematode (order Rhabditida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** Larvated eggs are passed in the feces and hatch in the environment. After a free-living period in the environment, third-stage larvae are produced that infect pigs through either ingestion or skin penetration. Sows carrying larvae in the tissues transmit the parasite through the milk to their litters.

**Laboratory Diagnosis:** Eggs are detected in fecal samples using flotation techniques. The thin-shelled egg contains a larva in fresh fecal samples.

Size: 45–55 × 26–35 µm

**Clinical Importance:** *Strongyloides* causes diarrhea in pigs as young as 10 days of age. Severe infections may be fatal.

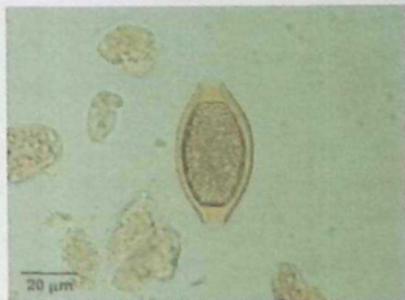


Fig. 1.188 Like other members of the genus, eggs of *Trichouris suis*, the swine whipworm, are football-shaped with a polar plug at each end.



Fig. 1.190 Larvated *Strongyloides* eggs are usually present only in the feces of young pigs. Photo courtesy of Merial.



Fig. 1.181 The small *Strongyloides ransomi* egg (Sr) pictured here could be confused with strongylid eggs, but strongylid eggs (S) are larger and do not contain a larva in fresh fecal samples. The strongylid eggs in this photo are from *Oesophagostomum dentatum*. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** *Metastrongylus* spp. (Fig. 1.192)

Common name: Lungworm.

**Taxonomy:** Nematode (order Strongylida). Several species similar in life cycle and pathogenicity have been described.

**Geographic Distribution:** Worldwide.

**Location in Host:** Bronchi and bronchioles of domestic and wild pigs.

**Life Cycle:** Eggs containing a larva are passed in the feces of pigs. When earthworm intermediate hosts ingest the eggs, they hatch and develop to infective larvae. Swine are infected by ingestion of earthworms containing third-stage larvae.

**Laboratory Diagnosis:** Larvated eggs are detected in feces by flotation procedures.

Size: 51–63 × 33–42 μm

**Clinical Importance:** These parasites are uncommon in intensively raised swine. Heavy infections, especially in young pigs, can cause clinical respiratory disease.

**Parasite:** *Physocephalus sexalatus*, *Ascarops strongylina* (Figs. 1.193 and 1.194)

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Stomach of domestic and wild pigs.

**Life Cycle:** Eggs containing larvae are passed in the feces, where they are ingested by beetle intermediate hosts. Pigs are infected following ingestion of larvae in the intermediate host.

**Laboratory Diagnosis:** A sedimentation procedure is recommended for detecting spirurid parasite eggs. The ellipsoidal eggs of both species are indistinguishable and are larvated when passed in the feces.

Size: 39–45 × 17–26 μm

**Clinical Importance:** Spirurid parasites are uncommon in intensively raised swine. Most infections are asymptomatic, but heavy infections may produce gastritis, leading to weight loss or failure to gain.



Fig. 1192 In fresh fecal samples, *Metastrongylus* eggs contain a larva and have a thick shell with a rough surface. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1193 Egg of *Physicocephalus*. Photo courtesy of Merial.

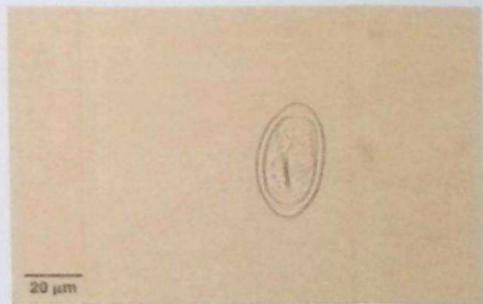


Fig. 1194 *Ascarops* egg. Spirurid eggs are unlikely to be seen if standard flotation procedures are used. Their larvated eggs have a thick shell (easily appreciated in the *Physicocephalus* egg), unlike the thin-shelled eggs of *Strongyloides* or *Metastrongylus*. Photo courtesy of Merial.

**Parasite:** *Fasciola* spp., *Eurytrema pancreaticum*, *Dicrocoelium dentriticum*, *Schistosoma* spp. (Figs. 1.156, 1.159, 1.160, 1.195)

Although swine are not considered to be the primary host for most trematodes, pigs may be infected with trematode parasites (*Fasciola* spp., *E. pancreaticum*, *D. dentriticum*, *Schistosoma* spp.) that also infect ruminants. Trematode infections of domestic swine would only be seen in pastured pigs because the complicated life cycle of flukes prevents transmission in confinement operations.

**Parasite:** *Macracanthorhynchus hirudinaceus* (Fig. 1.196)

Common name: Thorny-headed worm.

**Taxonomy:** Acanthocephalan.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine.

**Life Cycle:** Parasite eggs are passed in manure and ingested by beetle intermediate hosts. Swine are infected when they ingest infective larvae (cystacanths) in beetles.

**Laboratory Diagnosis:** Eggs are not consistently recovered by flotation procedures. A sedimentation procedure should also be performed.

Size: Variable, 67–110 × 40–65 μm

**Clinical Importance:** Acanthocephalan parasites have a proboscis covered with hooks. Attachment of the proboscis causes damage to the intestinal wall. Clinical signs range from none to diarrhea and weight loss. *Macracanthorhynchus* is unlikely to be present in total-confinement systems.



Fig. 1.195 *Fasciola* eggs. Trematode infections are rarely encountered in swine in North America, but are more common in other parts of the world where pigs range freely and infections also occur in other domestic animals. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.



Fig. 1.196 *Macracanthorhynchus hirudinaceus* egg. The embryo (acanthor) is surrounded by several membranes and contains hooks at one end that are often visible (*arrow*). Common tapeworm eggs also contain hooks; but thorny-headed worm eggs have a more complex membranous structure and an elongated embryo. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.

## Birds

### Protozoan Parasites

**Parasite:** *Eimeria* spp., *Isospora* spp. (Figs 1.197–1.201, 1.211)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia). *Eimeria* spp. are common in poultry and other Galliformes and Columbiformes. *Isospora* spp. are more common in Passeriformes, Psittaciformes, and Piciformes. Species of coccidia are host specific. *Caryospora*, *Sarcocystis*, and *Atoxoplasma* are other coccidia genera that infect some birds.

**Geographic Distribution:** Worldwide.

**Location in Host:** Primarily in the gastrointestinal tract.

**Life Cycle:** These parasites have a typical coccidia life cycle. Birds ingest infective oocysts from the environment. Sexual and asexual multiplication most often occurs within cells of the intestinal tract (some species are found in other organs). Development culminates in the production of oocysts, which are passed in the feces.

**Laboratory Diagnosis:** *Eimeria* oocysts are detected with routine flotation procedures. Sporulation of oocysts may be required for species identification.

Size: Approximately 10–45  $\mu\text{m}$  in length (oocysts), depending on species

**Clinical Importance:** Coccidia are common parasites of domestic and wild birds. Many infections are asymptomatic, but under some circumstances coccidia may cause severe diarrhea and death. *Eimeria* spp. are among the most important pathogens in modern poultry confinement operations.

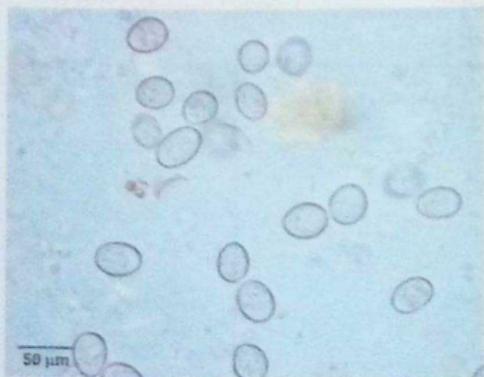
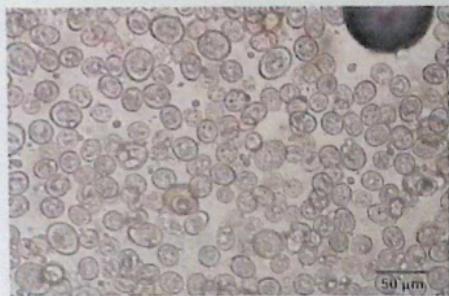


Fig. 1.197 *Eimeria* spp. oocysts from a chicken. *Eimeria* species are common parasites of wild and domestic birds. Individual birds may be infected with multiple coccidia species.

**Fig. 1.108** After reaching the environment, *Eimeria* oocysts sporulate; the length of time required for this process is determined by temperature but may take only a few days. Birds may be simultaneously infected with multiple *Eimeria* species. This chicken sample shows oocysts of different sizes representing several different species. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



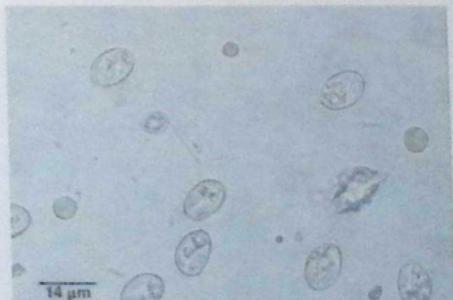
**Fig. 1.109** *Isospora* spp. can also be found in birds, as seen in this fecal sample from a zebra finch. Sporulated *Isospora* spp. oocysts contain two sporocysts. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



**Fig. 1.200** *Caryospora* oocyst. The sporulated oocyst of this genus contains a single sporocyst with eight sporozoites. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.



**Fig. 1.201** *Sarcocystis* is another coccidia genus that may be found in the feces of carnivorous birds that act as the definitive host of the parasite. Oocysts sporulate in the host, and small sporocysts are passed in the feces. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



**Parasite:** *Cryptosporidium* spp. (Fig. 1.202)

**Taxonomy:** Protozoa (coccidia). Avian species of this genus include *C. baileyi* and *C. meleagridis*. The latter species has been found to be infective for humans and some other mammals, as well as birds.

**Geographic Distribution:** Worldwide.

**Location in Host:** Gastrointestinal, respiratory, and/or urinary tracts depending on host.

**Life Cycle:** Infection of the bird host follows ingestion of the infective oocyst. Development and reproduction occur in the epithelial cells of the gastrointestinal and respiratory tract primarily. In some bird species, the urinary tract is affected.

**Laboratory Diagnosis:** Oocysts can be detected with the Sheather's sugar flotation procedure. Acid-fast or other staining procedures of fecal smears can also be used, as well as fecal immunodiagnostic tests.

Size: 4–6  $\mu\text{m}$  in diameter

**Clinical Importance:** Depending on the body systems affected, birds may show diarrhea, coughing, sneezing, and dyspnea or renal disease. Severe infection may cause death. *Cryptosporidium meleagridis* can infect humans.

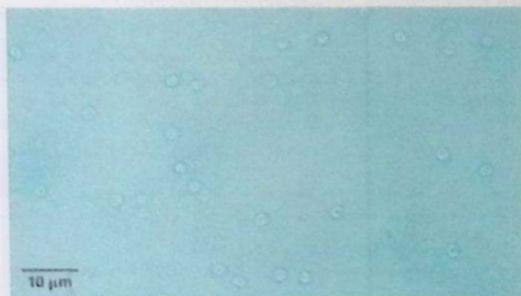


Fig. 1262 *Cryptosporidium* spp. oocysts in birds are similar in appearance to those of mammalian species. Slides should be examined using the 40 $\times$  lens of the microscope.

**Parasite:** Gastrointestinal Flagellates (Figs. 1.203 and 1.204; see also Figs. 1.53–1.59 for illustrations of *Giardia*)

**Taxonomy:** Protozoa (flagellates). Several genera are found in the avian digestive tract, including *Trichomonas*, *Cochlosoma*, *Histomonas*, *Giardia*, *Spiromucleus* (= *Hexamita*), and *Chilomastix*.

**Geographic Distribution:** Worldwide.

**Location in Host:** *Trichomonas gallinae* is found in the upper digestive system; *Histomonas* in the cecum and liver; and *Cochlosoma*, *Giardia*, and *Spiromucleus* in the intestines.

**Life Cycle:** Most avian flagellates have only a trophozoite stage. They are transmitted from bird to bird by direct contact and contaminated food or water. Feeding of young birds by adults can also transmit *T. gallinae*, while *Histomonas* can be carried in the eggs of the cecal roundworm, *Heterakis*. *Giardia* trophozoites encyst in the intestinal tract. Cysts passed in the feces are ingested by birds. *Chilomastix* also forms cysts.

**Laboratory Diagnosis:** *Trichomonas* trophozoites have anterior flagella and an undulating membrane. They can usually be detected in smears made from exudates or lesions in the oral cavity, esophagus, and crop. *Giardia* cysts can be recovered by  $ZnSO_4$  centrifugal flotation. *Chilomastix* cysts are rarely found in feces. *Giardia*, *Spiromucleus*, and *Cochlosoma* trophozoites may be seen in very fresh fecal smears. *Spiromucleus* has no sucking disk, unlike *Giardia* and *Cochlosoma*. *Histomonas* is unlikely to be detected in fecal smears.

Size:	<i>Trichomonas</i>	8–14 $\mu$ m in length, depending on species
	<i>Giardia</i> trophozoites	10–20 $\times$ 5–15 $\mu$ m
	<i>Giardia</i> cysts	10–14 $\times$ 8–10 $\mu$ m
	<i>Cochlosoma</i>	6–10 $\times$ 4–6.5 $\mu$ m
	<i>Spiromucleus</i>	5–12 $\times$ 2–7 $\mu$ m
	<i>Chilomastix</i>	6–24 $\times$ 3–10 $\mu$ m

**Clinical Importance:** Pigeons and raptors are particularly susceptible to trichomoniasis of the upper digestive tract. Affected birds may show depression and weakness with characteristic plaques and accumulation of cheesy material in the mouth, esophagus, and crop. Severe infections may be fatal. Although many *Giardia* infections are asymptomatic, the parasite can cause diarrhea, depression, and debilitation, particularly in young psittacines. Feather picking associated with infection has also been described. *Histomonas* is the cause of blackhead in turkeys. Other flagellates may also produce diarrhea, but many infections are asymptomatic. None of these avian flagellates has zoonotic importance.



Fig. 1203 *Trichomonas* spp. in birds have an undulating membrane and several anterior flagella. They can be seen in smears made from lesions in the upper gastrointestinal tract. The presence of caseous lesions in the esophagus and crop is also helpful in diagnosis.

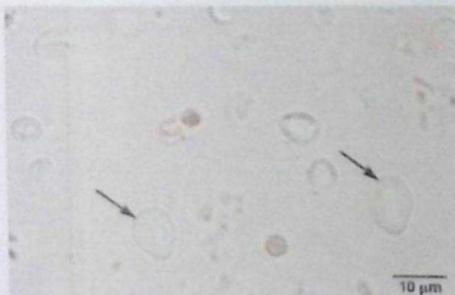


Fig. 1204 *Chilomastix* is a genus of intestinal flagellate found in birds, pigs, and primates. Infections are usually considered of no pathogenic importance, but the lemon-shaped parasite cysts (arrow) may be seen in fecal samples. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

### Helminth Parasites

**Parasite:** *Capillaria* spp., *Eucoleus* spp. (Figs. 1.205–1.207, 1.213)

**Taxonomy:** Nematode (order Enoplida). A variety of species can be found in domestic and wild birds.

**Geographic Distribution:** Worldwide.

**Location in Host:** Various locations in the gastrointestinal tract, depending on species.

**Life Cycle:** Eggs are passed in the feces of the host and become infective in the environment. Some species, such as *C. obsignata*, have a direct life cycle, while others have been shown to use an earthworm intermediate host.

**Laboratory Diagnosis:** Bipolar-plugged eggs can be detected in the feces with flotation techniques.

**Size:** Approximately 45–70  $\mu\text{m}$  in length, depending on species

**Clinical Importance:** Capillarid parasites can cause severe inflammation wherever species occur in the digestive tract, including the esophagus, crop, and intestines. Heavy infections may be fatal.



Fig. 1.205 Eggs of *Capillaria* spp. are very common in the feces of domestic and wild birds. The bipolar-plugged eggs have a thick shell and are typically yellowish. This *Capillaria* egg was present in a fecal sample from a superb starling. *Capillaria* spp. are uncommon in poultry maintained in total-confinement systems. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

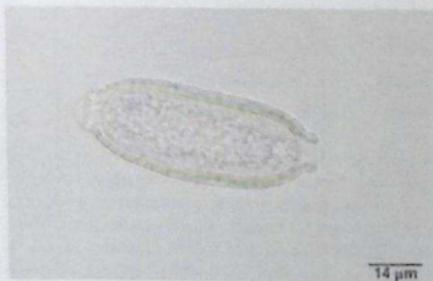


Fig. 1.206 Egg of *Eucileus dipae* from a sharp-shinned hawk. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

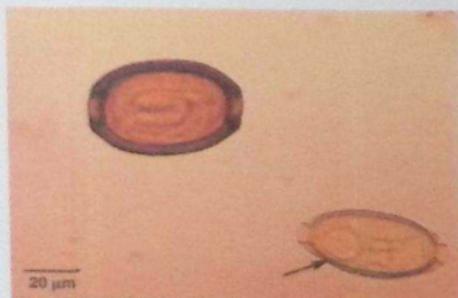


Fig. 1.207 *Capillaria* sp. egg in an owl (arrow). The larger, browner egg belongs to *Trichosomoides* sp., a parasite of the bladder of rats. Because parasite eggs of prey are sometimes found in predator feces, it is important to appreciate the normal array of parasites found in a host so spurious parasites can be correctly identified. Photo courtesy of Dr. Stephen Smith, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

**Parasite:** Avian Ascarids (Figs. 1. 208–1.210)

Common name: Roundworm, cecal worm (*Heterakis*).

**Taxonomy:** Nematodes (order Ascaridida). *Ascaridia* spp. and *Heterakis* spp. are common in poultry and many wild bird hosts. *Subulura* spp. occur less commonly in poultry and various other species of birds. *Porrocaecum* spp. and *Contracecum* spp. are also found in a variety of bird hosts.

**Geographic Distribution:** Worldwide.

**Location in Host:** Intestinal tract; *Heterakis* spp. and *Subulura* spp. parasitize the ceca of birds.

**Life Cycle:** Eggs are passed in the feces of the bird host and develop to the infective stage in the environment. Birds become infected when they ingest infective eggs. Earthworms may act as transport hosts.

**Laboratory Diagnosis:** Thick-shelled eggs are detected with flotation procedures. Eggs of *Ascaridia* and *Heterakis* are similar in size and morphology and may be difficult to differentiate. Eggs of *Subulura* are larvated.

Size:	<i>Ascaridia</i>	77–94 × 43–55 μm
	<i>Heterakis</i>	66–79 × 41–48 μm
	<i>Subulura</i>	51–86 × 45–76 μm

**Clinical Importance:** Larvae of *Ascaridia* appear to be the most pathogenic stage and may cause enteritis in the prepatent period. Heavy burdens of adult worms can cause enteritis and intestinal obstruction. *Heterakis* is primarily important in poultry as a vector of *Histomonas meleagridis* (blackhead), which can be a serious disease of turkeys. The protozoan is carried from bird to bird in the eggs and larvae of *Heterakis*. Fatal infections of *Heterakis isolonche* due to nodular typhlitis have been reported in pheasants.



Fig. 1208 A smooth, thick shell is seen in both *Ascaridia* spp. and *Heterakis* spp. eggs. This sample from a chicken shows two larger *Ascaridia* eggs and a single smaller *Heterakis* egg. The eggs of these two genera may be difficult to distinguish. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1209 Another ascarid genus found in ducks and wild birds is *Porroecium*, which has eggs with a rough shell similar in appearance to that of many mammalian ascarid species. Some *Porroecium* eggs have polar plugs. (A) was found in feces from a Harris hawk, and (B) was from a great horned owl.

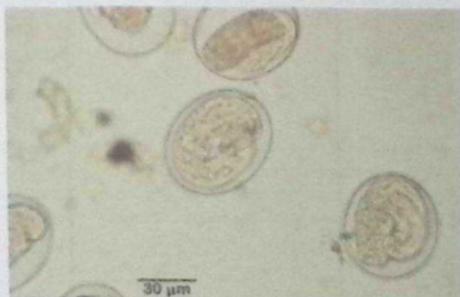


Fig. 1210 *Subulura brumpti* eggs in the feces of a chicken. Unlike most ascarids, the relatively thick-walled eggs of subulurids are larvated when passed in the feces, but species identification may be difficult. Photo courtesy of Dr. Erin Burton, University of Minnesota College of Veterinary Medicine, St. Paul, MN.

**Parasite:** *Trichostrongylus tenuis*, *Amidostomum* spp., and Other Avian Strongylids (Fig. 1.211)

**Taxonomy:** Nematodes (order Strongylida). *Trichostrongylus tenuis* and *Amidostomum* spp. occur in domestic and game birds.

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum and intestines of game birds, poultry, and wild birds.

**Life Cycle:** Eggs are passed from the host in the feces. First-stage larvae hatch from the eggs, develop to the infective stage in the environment, and develop to the adult stage when ingested by the avian host.

**Laboratory Diagnosis:** Typical thin-shelled strongylid eggs can be detected with flotation procedures.

Size:	Varies with species	
	<i>T. tenuis</i>	65–75 × 35–42 μm
	<i>Amidostomum anseri</i>	85–110 × 50–82 μm

**Clinical Importance:** Heavy infections of *T. tenuis* can produce severe enteritis with resulting hemorrhagic diarrhea, weight loss, and death.

**Parasite:** *Syngamus* spp. (Figs. 1.212 and 1.213)

Common name: Gapeworm.

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Trachea and bronchi of numerous domestic and wild birds. Species of a similar genus, *Cyathostoma*, are found in some aquatic birds and birds of prey.

**Life Cycle:** Eggs are produced by females in the trachea and are coughed up, swallowed, and passed out of the host in the feces. Infective larvae may be eaten directly from the environment by the avian host, or they may be ingested by an earthworm or molluscan transport host that is eaten, in turn, by a bird.

**Laboratory Diagnosis:** Ellipsoidal, bipolar eggs are seen in the feces with flotation procedures.

Size: 80–110 × 40–50 μm

**Clinical Importance:** Young birds are most severely affected. Large numbers of parasites and exudate obstruct the airways and can suffocate the host. The parasite's common name originates from the gaping and gasping of infected birds as they attempt to breathe. *Syngamus* is uncommon in total-confinement poultry systems.



Fig. 1211 Strongylid egg and coccidia oocysts in an avian fecal sample.



Fig. 1212 *Syngamus* sp. egg from a crow. This is a common parasite of wild birds and poultry kept outside. Unlike most strongylid eggs, *Syngamus* eggs have bipolar plugs.



Fig. 1213 Like *Syngamus*, eggs of *Capillaria* and *Eucoileus* spp. have bipolar plugs. However, the eggs of *Syngamus* are larger and contain several well-defined cells (morula) when passed in the feces. In this specimen from a pheasant, an egg of both *Syngamus* (S) and *Capillaria* (C) is present. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**Parasite:** *Dispharynx*, *Echinuria*, *Tetrameres*, *Cheilosporira* (*Acuaria*), *Serratospiculum*, and Others (Figs. 1.214–1.216)

**Taxonomy:** Nematodes (order Spirurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Species are found throughout the digestive tract. *Serratospiculum* parasitizes the respiratory tract of some wild birds.

**Life Cycle:** Adults (except *Serratospiculum*) are located in the digestive tract. Eggs are passed in the feces and are ingested by various arthropod intermediate hosts. Birds are infected by ingesting an intermediate host carrying infective larvae.

**Laboratory Diagnosis:** Flotation and sedimentation techniques can be used to detect the relatively small, larvated eggs in feces.

**Size:** Approximately  $30\text{--}55 \times 20\text{--}35 \mu\text{m}$ , depending on species

**Clinical Importance:** Most infections with this group of parasites are not highly pathogenic. However, *Tetrameres* and *Dispharynx* in large numbers in the proventriculus may cause weight loss and reduced production. In poultry, members of this group of parasites are rare in total-confinement management systems.

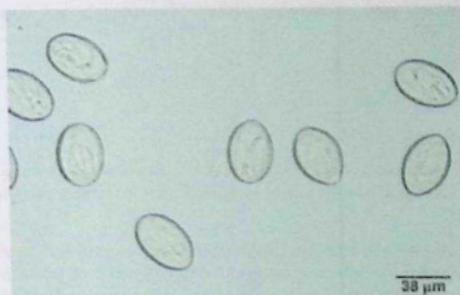


Fig. 1214 Spirurid egg in the feces of a double-crested cormorant. Larvae are visible within the eggs.

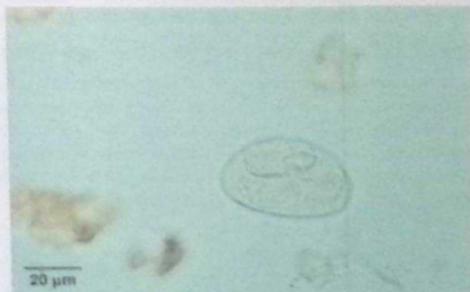


Fig. 1215 Spirurid egg in the feces of a red-tailed hawk. The larva within the egg is clearly visible. Photo courtesy of Dr. Stephen Smith, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

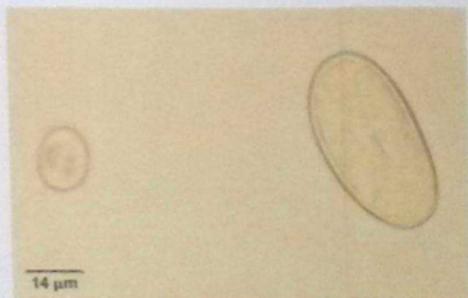


Fig. 1216 *Tetrameres* egg in the feces of a homing pigeon. Like other spirurid eggs, this *Tetrameres* egg is larvated when passed in the feces, but further specific identification is difficult. Also pictured is a coccidia oocyst. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

**Parasite:** *Echinostoma* spp., *Echinoparyphium*, *Prosthogonimus* spp., and Others (Figs. 1.217 and 1.218)

**Common name:** Fluke.

**Taxonomy:** Trematode.

**Geographic Distribution:** Worldwide.

**Location in Host:** Domestic and wild birds are parasitized by many fluke species. Adults can be found in various body systems, including the intestines and respiratory and reproductive tracts.

**Life Cycle:** Adult worms produce eggs that leave the host principally via the digestive tract. The first intermediate host is a mollusk, and a variety of animals act as second intermediate host depending on the fluke species (with the exception of schistosomes, which do not require a second intermediate host).

**Laboratory Diagnosis:** Fluke eggs in feces are best detected with a sedimentation technique because of their higher density. Fluke eggs are typically operculated and brown.

**Size:** Highly variable with species, approximately 20–100  $\mu\text{m}$  in length

**Clinical Importance:** Many fluke infections are of low pathogenicity. Some genera of importance in domestic poultry are *Echinostoma* and *Echinoparyphium*, which may cause enteritis, and *Prosthogonimus*, a parasite of the oviduct, which can cause abnormal egg production and peritonitis. Because of their complex life cycles, flukes will be seen only in birds with access to intermediate hosts and will not occur in poultry confinement operations.

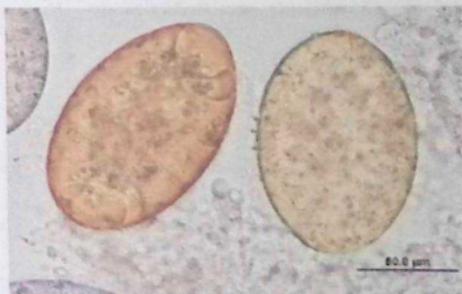


Fig. 1217 Fluke eggs in avian feces. The operculum is not readily visible in these trematode eggs.



Fig. 1218 The developing miracidium can be seen inside this avian trematode egg found in feces from a bird of prey.

**Parasite:** *Davainea* spp., *Choanotaenia* spp., *Railletina* spp., *Hymenolepis* spp., and Others (Figs. 1.219, 1.220)

Common name: Tapeworm.

**Taxonomy:** Cestode.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of a wide variety of domestic and wild birds.

**Life Cycle:** Eggs are passed in the feces. Insects are the most common intermediate hosts, but other invertebrates may also be used by some species (e.g., the intermediate host of *Davainea* is a mollusk).

**Laboratory Diagnosis:** Although proglottids are intermittently passed in the feces, detection of eggs by fecal exam is unreliable, and diagnosis is usually made at necropsy.

**Size:** Varies with species; individual eggs of many species are approximately 50–80  $\mu\text{m}$ ; some species have large egg packets

**Clinical Importance:** Many tapeworm infections are asymptomatic. However, some are serious pathogens; for example, *Davainea proglottina* and *Railletina echinobothrida* can cause severe enteritis and death in domestic poultry. Tapeworms are common in unconfined poultry with access to the intermediate hosts.



Fig. 1219. *Raillietina* sp. eggs from a chicken. The embryonic hooks are clearly visible, and this characteristic is very helpful in recognizing tapeworm eggs. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

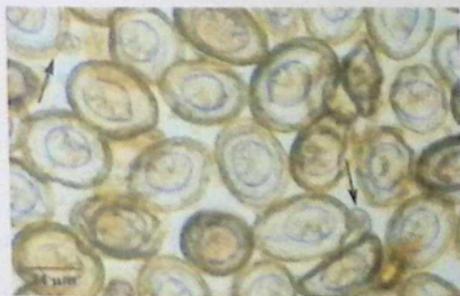


Fig. 1220. Tapeworm eggs from a magpie. Scattered among the cestode eggs are occasional coccidia oocysts (arrows). Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.

**Parasite:** *Polymorphus* spp., *Filicollis* spp., and Others (Figs. 1.221 and 1.222)

Common name: Thorny-headed worm.

**Taxonomy:** Acanthocephalan.

**Geographic Distribution:** Worldwide.

**Location in Host:** Digestive system.

**Life Cycle:** Eggs are passed in the feces of the host. Infective larvae develop in arthropod intermediate hosts and infect birds when they are ingested.

**Laboratory Diagnosis:** Eggs are not consistently recovered by flotation procedures. A sedimentation procedure should also be performed.

**Size:** Approximately 50–100  $\mu\text{m}$  in length, depending on species

**Clinical Importance:** Thorny-headed worms are primarily parasites of free-ranging and wild birds because of their complex life cycles. Heavy infections may cause diarrhea and debilitation.



Fig. 1221 Egg of a thorny-headed worm (*Centrorhynchus*) from the feces of a barred owl. Acanthocephalan eggs have several internal layers surrounding the larva (acanthor). Photo courtesy of Dr. Ellis C. Greiner, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1222 Acanthocephalan (*Plagiosthynchus* sp.) eggs in the feces of an eastern blue bird. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

## Rodents and Rabbits

### Protozoan Parasites

**Parasite:** *Eimeria* spp. (Figs. 1.223–1.225)

Common name: Coccidia.

**Taxonomy:** Protozoa (coccidia).

**Geographic Distribution:** Worldwide.

**Location in Host:** A variety of species parasitize the intestinal tract of rodents and rabbits. *Eimeria stiedae* is found in the bile ducts of rabbits.

**Life Cycle:** Oocysts passed in the feces can sporulate quickly and infect the host when ingested. Asexual and sexual reproduction occurs in cells of the gastrointestinal tract.

**Laboratory Diagnosis:** Oocysts can be detected in feces by centrifugal or simple flotation techniques. Oocysts are elliptical to spherical-shaped.

**Size:** Approximately  $10\text{--}45 \times 10\text{--}30 \mu\text{m}$ , depending on species

**Clinical Importance:** Some species are nonpathogenic. Pathogenic intestinal coccidia can cause anorexia, weight loss, profuse diarrhea, and death. *Eimeria stiedae* infection of the rabbit liver can cause anorexia, diarrhea, distended abdomen, and death.

Rodents may also be infected with species of *Giardia* and *Cryptosporidium* (see Figs. 1.50 and 1.53–1.59).

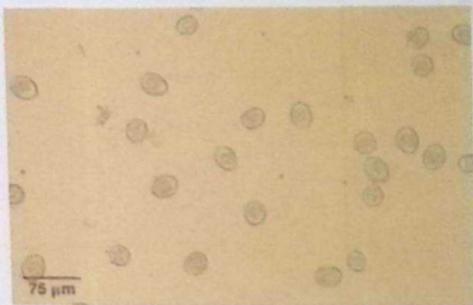


Fig. 1223 *Eimeria* oocysts have smooth, clear cyst walls and contain a single round cell when freshly passed. The oocysts in this figure are *Eimeria nutschulzi*, a parasite of rats. Photo courtesy of Dr. George Conder, Pfizer Veterinary Medicine Pharmaceuticals Clinical Development, Pfizer, Inc., Kalamazoo, MI.

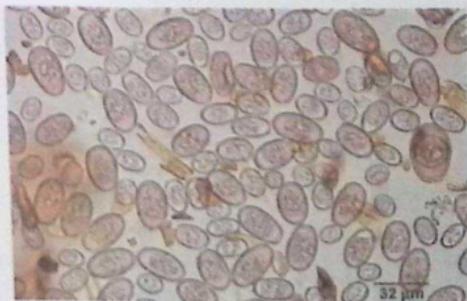


Fig. 1224 Rodent and rabbit hosts may be infected with multiple species of coccidia. Oocysts of more than one species of rabbit *Eimeria* were detected in this sample. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 1225 Once in the environment, oocysts undergo sporulation to the infective stage. Sporulated oocysts of *Eimeria* contain four sporocysts, each containing two sporozoites (individual organisms). The sporocysts can clearly be seen inside this rabbit *Eimeria* sp. oocyst. Surrounding the oocyst are several elongated nonpathogenic yeast organisms that are very common in rabbit feces.

### Helminth Parasites

**Parasite:** *Syphacia obvelata* (Fig. 1.226)

Common name: Pinworm.

**Taxonomy:** Nematode (order Oxyurida). *i*

**Geographic Distribution:** Worldwide.

**Location in Host:** Large intestine of mice and gerbils.

**Life Cycle:** Eggs passed in the feces quickly become infective. Rodents develop infection following ingestion of larvated eggs.

**Laboratory Diagnosis:** When present in the feces, eggs can be detected by routine flotation techniques. Eggs are normally found on the skin in the perineal region of infected animals.

Size: 100–142 × 30–40 μm

**Clinical Importance:** Infections are typically subclinical.

**Parasite:** *Aspicularis tetraptera* (Fig. 1.227)

Common name: Pinworm.

**Taxonomy:** Nematode (order Oxyurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Large intestine of mice.

**Life Cycle:** The life cycle is similar to that of *Syphacia obvelata*. Adult pinworms develop following ingestion of infective eggs.

**Laboratory Diagnosis:** Eggs can be detected in feces by centrifugal or simple flotation techniques. The eggs are ellipsoidal with a distinctive double shell wall and contain an undifferentiated embryo in fresh feces.

Size: 70–98 × 29–50 μm

**Clinical Importance:** Infections are usually subclinical.



Fig. 1326 *Siphacia* egg in feces from a gerbil. Eggs of *Siphacia* have a smooth, clear shell wall; are flat on one side; and contain an undifferentiated morula in fresh fecal samples.



Fig. 1327 *Aspicularis* eggs have narrowed poles and a smooth, clear, double shell wall. Pinworms are common in rodents.

**Parasite:** *Passalurus ambiguus* (Fig. 1.228)

Common name: Pinworm.

**Taxonomy:** Nematode (order Oxyurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum of rabbits.

**Life Cycle:** Eggs are passed in the feces of infected rabbits. The eggs become infective within a short period and infect the next host when ingested.

**Laboratory Diagnosis:** Eggs can be detected in feces by flotation techniques. The eggs have a smooth, clear shell wall that is flat on one side.

Size: 95–103 × 43 µm

**Clinical Importance:** Infections are usually subclinical.

**Parasite:** *Paraspidodera uncinata*, *Heterakis spumosa* (Fig. 1.229)

**Taxonomy:** Nematodes (order Ascaridida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Cecum of guinea pigs (*Paraspidodera*) and rats (*Heterakis*).

**Life Cycle:** Eggs passed in the feces develop to the infective stage and infect the host when they are ingested.

**Laboratory Diagnosis:** Fecal flotation procedures can be used to recover these thick-shelled eggs.

Size: *Heterakis* 55–60 × 40–55 µm

*Paraspidodera* 43 × 31 µm

**Clinical Importance:** Infection is usually subclinical. These parasites are seen primarily in wild rats and guinea pigs raised on dirt.

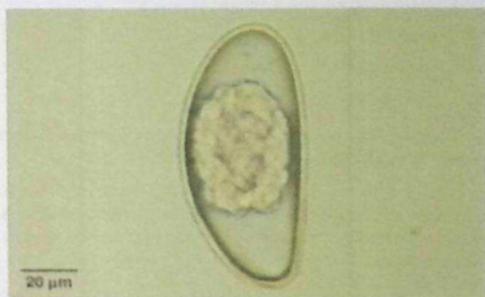


Fig. 1228 *Passalurus ambiguus* eggs have an operculum-like structure at one end (not clearly visible in this example), and the eggs contain an undifferentiated morula in fresh fecal samples.



Fig. 1229 Specimens of *Passopillulella uncinata*, the cecal worm of guinea pigs. The eggs produced by this cecal nematode of rodents are similar to those of the avian cecal worm, *Heterakis* (Fig. 1.194). Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

**Parasite:** *Heligmosomoides polygyrus*, *Nippostrongylus braziliensis*, *Obeliscoides cuniculi*, *Graphidium strigosum* (Fig. 1.230)

**Taxonomy:** Nematodes (order Strongylida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Stomach (*Obeliscoides*, *Graphidium*) of rabbits; small intestine of rats (*Nippostrongylus*) and mice (*Heligmosomoides*).

**Life Cycle:** Eggs passed in the feces develop into first-stage larvae, which hatch and continue development in the environment. In most cases, the definitive host is infected by ingesting third-stage larvae, but larvae of *Nippostrongylus braziliensis* usually penetrate the skin of the final host.

**Laboratory Diagnosis:** Fecal flotation procedures will recover the typical strongylid eggs produced by these parasites.

Size: 52–106 × 28–58 µm (eggs), depending on species

**Clinical Importance:** Parasites belonging to this group of nematodes are unlikely to occur in caged rabbits and rodents but are common in wild animals. Infection is usually subclinical.

**Parasite:** *Hymenolepis* spp. (Figs. 1.231 and 1.232)

Common name: Dwarf tapeworm of humans (*Hymenolepis nana*).

**Taxonomy:** Cestode. The most common species in domestic rodents is *Hymenolepis* (= *Vampirolepis*) *nana*. Other species infecting rodents are *H. diminuta* and *H. microstoma*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of rodents (mouse, rat, hamster). Humans and other primates also serve as hosts for *H. nana*.

**Life Cycle:** Eggs passed in the feces of definitive hosts are ingested by beetle intermediate hosts. Rodents and humans are infected following ingestion of the intermediate host containing cysticercoids. Infection with adult *H. nana* can also follow ingestion of the egg.

**Laboratory Diagnosis:** Eggs with six embryonic hooks are detected by either centrifugal or simple fecal flotation examination.

Size: *H. nana* 40–45 × 34–37 µm

*H. diminuta* 60–88 × 52–81 µm

**Clinical Importance:** Infections are usually subclinical. Heavy infections, particularly in young animals, can result in poor growth and rarely intestinal impaction and death.



Fig. 1.230 Strongylid eggs in the feces of rabbits and rodents are similar to those produced by species in other common domestic hosts. These eggs were observed in the feces of a rabbit. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

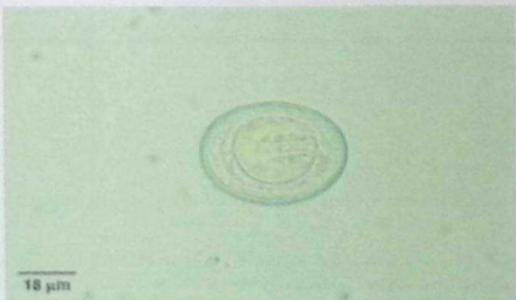


Fig. 1.231 *Hymenolepis nana* infects rodents and primates. Eggs are elliptical in shape with a smooth, clear shell wall and contain an embryo with six hooks. There are two knoblike protrusions at each end of the embryo. Like eggs of other common tapeworms, hooks are visible within the embryo.



Fig. 1.232 Other species of *Hymenolepis* found in rodents have larger, rounder eggs than *H. nana*. Hymenolepid eggs are seen occasionally in fecal samples of dogs and cats that have recently eaten rodents.

**Parasite:** *Cittotaenia* spp. (Fig. 1.233)

Common name: Rabbit tapeworm.

**Taxonomy:** Cestode.

**Geographic Distribution:** Worldwide.

**Location in Host:** Small intestine of rabbits and hares.

**Life Cycle:** Eggs are ingested by free-living oribatid mites and develop into the cysticeroid larval stage. Rabbits are infected when they ingest the infected mites.

**Laboratory Diagnosis:** Fecal flotation procedures will recover *Cittotaenia* eggs.

Size: 64  $\mu$ m in diameter

**Clinical Importance:** Heavy infections may cause weight loss.

## Reptiles

**Parasite:** *Entamoeba*, *Cryptosporidium*, *Eimeria*, *Isospora*, *Caryospora*, other protozoa (Figs. 1.234–1.238)

**Taxonomy:** Protozoa.

**Geographic Distribution:** Worldwide.

**Location in Host:** Gastrointestinal tract.

**Life Cycle:** Reptiles are infected by ingestion of cysts or oocysts that are passed in host feces.

**Laboratory Diagnosis:** *Eimeria*, *Caryospora*, and *Cryptosporidium* oocysts and *Nyctotherus* cysts can be detected by fecal flotation techniques (the centrifugal flotation procedure with Sheather's sugar solution is recommended for *Cryptosporidium*). Wet mounts of feces or colonic washings can be examined for *Entamoeba* trophozoites and cysts and *Nyctotherus* trophozoites. Fecal smears may also be stained with Wright's stain or Giemsa stain.

**Clinical Importance:** *Cryptosporidium serpentis* may cause chronic hypertrophic gastritis in snakes, associated with weight loss and regurgitation. *Entamoeba invadens* can cause bloody diarrhea and hepatitis in snakes and some tortoise and lizard hosts. Weight loss and enteritis may accompany infection with *Eimeria* (all reptiles) and *Caryospora* (primarily snakes). *Nyctotherus* and several other intestinal protozoan organisms are considered commensal in most hosts but may become pathogenic if the host is immunosuppressed.



Fig. 1.233 *Cytospora* eggs are quite similar in appearance to *Hymenolepis* eggs. The hooks in the tapeworm embryo are usually easily seen.

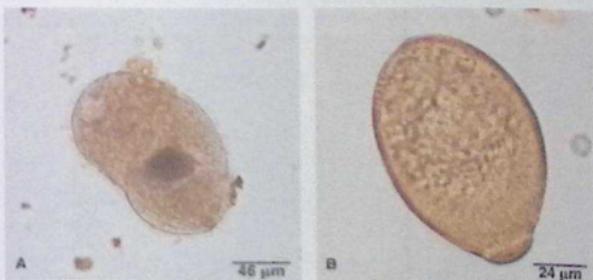


Fig. 1.234 Trophozoite (A) and cyst (B) of the commensal ciliate *Nyctotherus* in feces from an iguana. The trophozoites can be seen in fecal smears; cysts can be recovered with flotation procedures. Photos courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

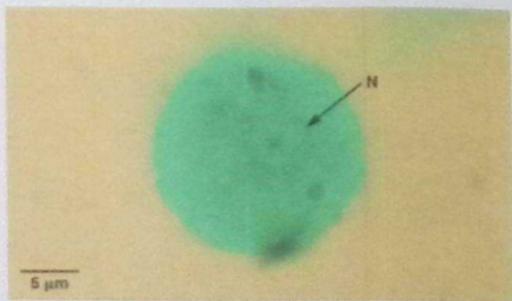


Fig. 1.235 *Entamoeba invadens* trophozoite in a stained fecal smear from a Burmese python. A single nucleus (N) is seen in the trophozoite, and they are approximately 16  $\mu\text{m}$  in size. Trophozoite movement can be seen in a wet mount of fresh feces. Photo courtesy of Dr. Ellis C. Greiner, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1236 Cyst of *Entamoeba invadens* in an iodine-stained smear of reptile feces. Two of the four macronuclei (N) present in the cyst can be seen (arrow). Cysts are 11–20 μm. Photo courtesy of Dr. Thomas Nolan, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA.



Fig. 1237 Sporulated *Caryospora* oocyst in the feces of a loggerhead turtle. Oocysts of prey coccidia species may be found in the feces of carnivorous reptiles. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1238 Coccidia in the feces of a bearded dragon. *Isospora* (I) oocysts in the 2-cell stage and a sporulated *Eimeria*-like (E) oocyst are present. A large oxyurid egg is also present. Photo courtesy of Dr. Yoito Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**Parasite:** Reptile Helminths (Figs. 1.238–1.254)

**Taxonomy:** Nematodes, cestodes (tapeworms), trematodes (flukes), acanthocephalans (thorny-headed worms), and pentastomid parasites.

**Geographic Distribution:** Worldwide.

**Location in Host:** Helminth parasites can be found in a variety of body systems, although those detected by fecal exam are primarily gastrointestinal or respiratory system parasites.

**Life Cycle:** Life cycle varies widely depending on the species. Some nematodes have a direct life cycle. Other nematodes and the remaining helminth groups all require at least one intermediate host.

**Laboratory Diagnosis:** Eggs are detected in feces by flotation or sedimentation procedures.

**Clinical Importance:** As in other hosts, low levels of helminth infection are usually well tolerated by reptiles. Heavy infections may result in clinical disease, especially in young or immunosuppressed animals. The complex life cycles of most flukes, tapeworms, and thorny-headed worms make these parasites uncommon in reptiles bred in captivity.



Fig. 1.238 Oxyurid (pinworm) eggs are frequently encountered in reptile feces. These eggs are usually elongated and may appear flat on one side. The eggs shown here are from a bearded dragon.

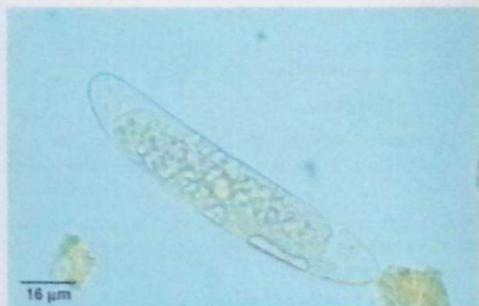


Fig. 1.240 Pinworm egg from a turtle.



Fig. 1.241 Reptiles may be infected with the same groups of helminth parasites that infect other vertebrates. The photo shows examples of reptile capillaria and ascarid eggs.



Fig. 1.242 This *Capillaria serpentina* egg from a turtle has a distinctive rough coat. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

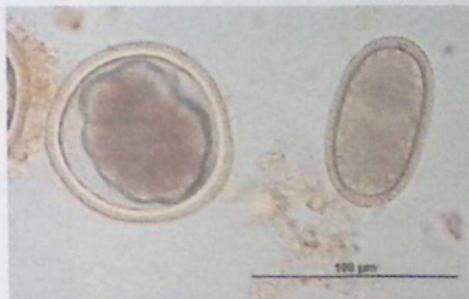


Fig. 1243 Eggs of the ascarids *Hexametra* and *Spinicada* in the feces of a chameleon.



Fig. 1244 *Kalicephalus* sp. egg from a python. This is one of the genera of strongylid nematodes parasitizing reptiles. Eggs of this nematode group are thin shelled. In fresh feces, reptile strongylid eggs may be larvated or contain a morula (cluster of cells), like the one shown here. Photo courtesy of Dr. Elia C. Greiner, College of Veterinary Medicine, University of Florida, Gainesville, FL.

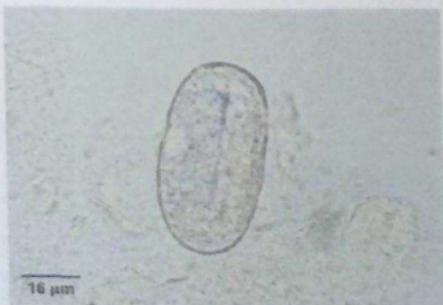


Fig. 1245 Rhabditid nematodes like *Rhabdias* and *Strongyloides* occur in reptiles and amphibians. This egg was present in the feces of a rat snake. The larvated eggs are smaller than strongylid eggs.



Fig. 1.246 *Rhabdias* is a common lungworm of frogs. First-stage larvae are passed in the feces.



Fig. 1.247 Tapeworm egg from a water moccasin. This egg is surrounded by a gelatinous layer with one hook of the hexacanth embryo visible (arrow).



Fig. 1.248 Tapeworm egg from an anole. This egg is also surrounded by a clear gelatinous layer.



Fig. 1248 Fluke (trematode) infections are common in wild reptiles, especially those associated with water. The operculum is clearly visible (arrow) in this fluke egg from an anole.



Fig. 1250 Flukes are also common in amphibians. These eggs of the lung fluke *Haematoloechus* were found in the feces of an American bullfrog. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1251 Spirorchid egg detected on sedimentation of feces from a wood turtle. Spirorchids are blood flukes infecting various species of turtles. This large egg lacks an operculum and contains a fully formed miracidium. Note the two black eye spots in this miracidium.

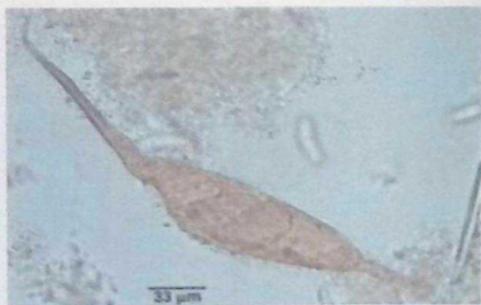


Fig. 1252 Another spirochid fluke is *Hapalostroma*. This egg from a green sea turtle has a long filament at each end. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 1253 Acanthocephalans (thorny-headed worms) are also common in wild reptiles. This egg was found in mammalian feces but shows the complex layered shell that would also be seen in the eggs of thorny-headed worms of snakes.

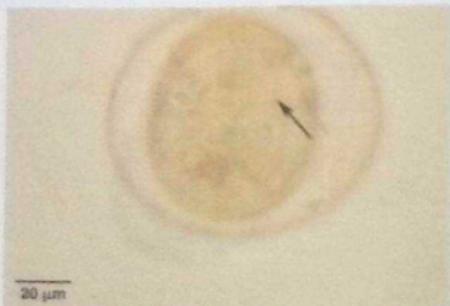


Fig. 1254 Pentastomid egg in feces from a Boelen's python. This is an unusual group that shows some arthropod characteristics. Adults parasitize the respiratory tract. Large eggs (over 100 μm) in feces are often surrounded by a capsule. Larvae within the eggs have legs bearing hooklets (arrow) and could be mistaken for mite eggs. An intermediate host is required for completion of the life cycle. Photo courtesy of Dr. Ellis C. Greiner, College of Veterinary Medicine, University of Florida, Gainesville, FL.

# Detection of Protozoan and Helminth Parasites in the Urinary, Reproductive, and Integumentary Systems and in the Eye

## TECHNIQUES FOR PARASITE RECOVERY

### Parasites of the Urinary System

Several organisms parasitize the urinary tract, and their eggs and cysts can be detected by routine urine sedimentation. Samples collected by cystocentesis are preferred because voided urine samples could be contaminated with fecal material containing parasite eggs or larvae from the intestinal or respiratory systems.

#### *Urine Sedimentation*

Centrifuge 5–10 mL of urine in a conical-tip centrifuge tube for 5 minutes at 1500–2000 rpm (approximately  $100 \times g$ ).

1. Decant the supernatant fluid, leaving 0.5 mL. Resuspend the sediment.
2. Transfer a drop of sediment to a slide, add a coverslip, and examine.

## Parasites of the Reproductive Tract

Organisms that are primary parasites of the reproductive tract are not important pathogens of common domestic species in North America, with the exception of *Tritrichomonas foetus*. In South America, West Asia, and parts of Africa, *Trypanosoma equiperdum* causes serious disease in horses and is transmitted venereally. However, this parasite is difficult to recover from tissue fluids or blood, and immunodiagnostic tests are usually used for confirmation of infection. Several other parasites can affect reproduction as a part of general systematic effects, including *Neospora*, *Toxoplasma*, and *Sarcocystis*. These parasites also would not be recovered and identified in the live animal.

*Tritrichomonas foetus* is a parasite of cattle found in the uterus and vagina of cows and the prepuce of bulls. Diagnosis of infection is usually by detection of the organism in preputial samples from bulls, although vaginal or cervical secretions from cows can also be tested. A number of U.S. states require testing of bulls for *T. foetus* before interstate movement. A commercial kit is available that provides a plastic pouch containing a medium into which the sample can be inoculated either for culture and microscope examination or for use as a transport medium when the sample is submitted for PCR (InPouch<sup>®</sup> TF; Biomed Diagnostics, Inc., White City, OR, [www.biomeddiagnostics.com/](http://www.biomeddiagnostics.com/)).

### *Bovine Preputial Sample Collection*

- 1 Attach a dry 21-in. infusion pipette to a 20-cc syringe and insert into the prepuce of the bull. The tip of the pipette is scraped back and forth across the epithelium while suction is applied.
- 2 Examine the collected sample microscopically immediately if desired and then inoculate into Ringer's solution (or the commercial InPouch<sup>®</sup>) and refrigerate for transport to the laboratory for culture.
- 3 Samples not placed directly into nutritive medium should not be held for more than 48 hours in Ringer's or other balanced salt solution.

## Helminth Parasites of the Integumentary System

Several filarial nematode parasites produce microfilariae that are found in the subcutaneous tissue. These larvae can be detected in fresh skin biopsies or on examination of fixed and stained biopsy tissue sections. For recovery of microfilariae from fresh biopsies, the biopsy is macerated and allowed to incubate in saline for several hours at room temperature. Following incubation, the saline is examined microscopically for the presence of microfilariae. Free-living nematodes that occasionally invade the skin may also be seen in fresh or fixed and stained skin biopsies.

## Parasite Detection in Urinary and Other Systems

### Urinary System Parasites

**PARASITE:** *Diocotophyme renale* (Fig. 2.1)

Common name: Giant kidney worm.

**TAXONOMY:** Nematode (order Enoplida).

**Geographic Distribution:** North America and Europe.

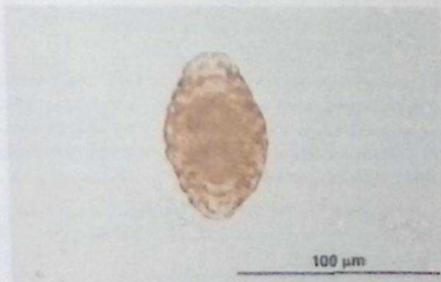
**Location in Host:** Kidney and occasionally peritoneal cavity of dogs, mink, and other domestic and wild animals.

**Life Cycle:** Adult worms in the kidney produce eggs that are passed in the urine. The first intermediate host is an annelid worm; the second intermediate host is a fish. The final host becomes infected when it eats a fish containing infective parasite larvae.

**Laboratory Diagnosis:** Eggs with a thick, rough shell are detected in the urine.

Size: 60–80 × 39–46 μm

**Clinical Importance:** The spectacular adult worms (females may reach 100 cm in length) enter the renal pelvis and eventually destroy the kidney, leaving only the capsule. Typically, only one kidney (usually the right) is affected. Most infections are asymptomatic, despite the loss of a kidney, although hematuria and dysuria may occur. *Diocotophyme renale* is a rare finding in dogs in North America.



**FIG. 2.1** *Diocotophyme* eggs are larger than those of *Pearsonema* and have a thicker eggshell with a rougher surface.

**PARASITE:** *Pearsonema* (= *Capillaria*) *plica*, *P. feliscati* (Figs. 2.2 and 2.3)

**Taxonomy:** Nematode (order Enoplida). These parasites were previously included in the genus *Capillaria*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Adult worms in the bladder of dogs and foxes (*P. plica*) and cats (*P. feliscati*).

**Life Cycle:** Parasite eggs passed in the urine become infective in the environment. Although the life cycle is not known with certainty, the final host is probably infected by ingesting an earthworm intermediate host or a transport host, such as a bird.

**Laboratory Diagnosis:** Typical capillarid eggs are detected in urine sediment. Cystocentesis is preferred for collecting urine samples since fecal material containing similar eggs of other capillarid species may contaminate voided urine samples.

**Size:** 51–65 × 24–32 μm

**Clinical Importance:** Many infections are asymptomatic, although infected animals may develop cystitis.

**PARASITE:** *Stephanurus dentatus* (Fig. 2.4)

**Common name:** Kidney worm.

**Taxonomy:** Nematode (order Strongylida).

**Geographic Distribution:** Tropical and subtropical regions.

**Location in Host:** Adults are found in the wall of the ureters and the pelvis of the kidney as well as in the peritoneal fat of swine.

**Life Cycle:** Parasite eggs leave the host via the urine. First-stage larvae hatch from the eggs and develop to the infective third stage. The pig final host is infected by ingestion of infective larvae or an earthworm transport host or by skin penetration by infective larvae. Once in the host, larvae migrate through the liver before moving to the perirenal tissues, where development is completed.

**Laboratory Diagnosis:** Diagnosis is made by detection of eggs from urine sedimentation tests or from clinical signs. Disease may be present before the infection is patent (prepatent period is 4–6 months).

**Size:** 90–114 × 53–65 μm

**Clinical Importance:** Damage associated with larval migration through the liver is an important component of disease caused by the parasite. Pigs may show reduced growth or weight loss and general loss of condition. *Stephanurus dentatus* is unlikely to occur in modern swine confinement systems.

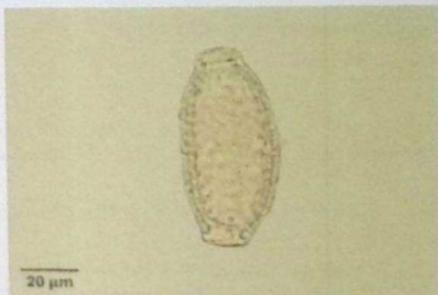


Fig. 2.2 *Pearsonema feliscati* egg in urine sediment. Methylene blue stain is often added to these preparations, which will stain the eggs purple. Like other capillarids, *P. feliscati* eggs are elongated and bipolar plugged.

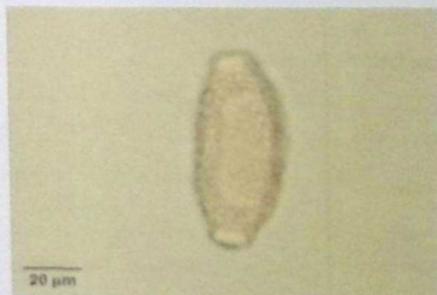


Fig. 2.3 If the microscope is focused on the shell wall of the *Pearsonema* egg, a thick globular pattern of ridges can be seen.



Fig. 2.4 *Stephanurus dentatus* adults produce typical strongylid eggs that can be seen in urine. However, disease may develop before the infection becomes patent. Photo courtesy of Dr. T. Bonner Stewart, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

**PARASITE:** *Trichosomoides crassicauda* (Figs. 2.5 and 1.207)

**Taxonomy:** Nematode (order Enoplida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Adult female worms are found in the wall of the bladder of wild and laboratory rats. Male worms exist as hyperparasites in the reproductive tract of the females.

**Life Cycle:** Eggs leave the host in the urine. Infection occurs when eggs are ingested by the host.

**Laboratory Diagnosis:** Infection is often detected by identification of adults in the bladder or during histologic examination of bladder sections, but diagnosis may also be made by finding eggs in the urine. *Trichosomoides* eggs are brown with bipolar plugs.

**Size:** 60–70 × 30–35 μm

**Clinical Importance:** Infection is typically subclinical but is undesirable in laboratory rats.

### **Reproductive System Parasites**

**PARASITE:** *Tritrichomonas foetus* (Fig. 2.6)

**Taxonomy:** Protozoa (flagellate).

**Geographic Distribution:** Worldwide, but uncommon where artificial insemination is widely practiced.

**Location in Host:** Preputial cavity of bulls, uterus and vagina of cows.

**Life Cycle:** Organisms are transmitted during breeding. The trophozoite in the reproductive tract is the only form of the organism.

**Laboratory Diagnosis:** Organisms are detected in vaginal or uterine discharges or in preputial scrapings. The prepuce is the site most frequently sampled. Organisms may be present in small numbers, and culture is recommended using Diamond's medium or the commercial InPouch® system available in North America. *Tritrichomonas* is easily recognized by its undulating membrane and three anterior flagella. A polymerase chain reaction (PCR) test is also used and may be required before movement of bulls into some U.S. states.

**Size:** 10–25 × 3–15 μm

**Clinical Importance:** The presence of *T. foetus* in a cattle herd produces chronic abortion and infertility and can have a serious economic impact on production.



Fig. 2.5 The eggs of *Trichostrongylus* have bipolar plugs and are embryonated when passed in the urine. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

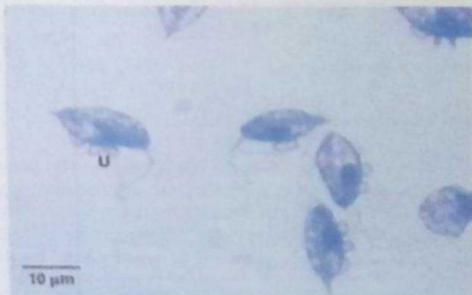


Fig. 2.6 *Trichostrongylus foetus* organisms from culture. The undulating membrane (U) and anterior flagella can be seen in several of the organisms. In culture, living organisms move in a jerky motion, and the rippling undulating membrane can be seen with the 40 $\times$  objective. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.

### Parasites of Other Systems (Excluding Arthropods)

**PARASITE:** *Onchocerca* spp., *Stephanofilaria* spp. (Figs. 2.7 and 2.8)

**Taxonomy:** Nematodes (order Spirurida).

**Geographic Distribution:** *Onchocerca cervicalis* and *O. gutturosa* are found worldwide. Other species have more limited distribution, primarily in Africa but also in the Middle East and Asia. *Stephanofilaria* spp. are found worldwide.

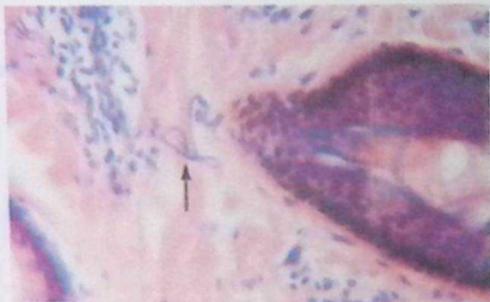
**Location in Host:** *Onchocerca cervicalis* is found in the equine nuchal ligament. Other species are bovine parasites. *Onchocerca gutturosa* parasitizes the nuchal and gastrosplenic ligament, while *O. gibsoni* is found in subcutaneous and intermuscular nodules and *O. armillata* is found in the wall of the thoracic aorta. *Stephanofilaria* spp. live in the subcutaneous tissue of the ventrum and other areas in cattle.

**Life Cycle:** Depending on the parasite species, *Culicoides* (midges), *Simulium* (black flies), *Haematobia* (horn flies), and *Musca* species act as the intermediate host and transmit the parasite during feeding.

**Laboratory Diagnosis:** Diagnosis is made by skin biopsy of affected areas and examination for microfilariae following saline incubation or histopathologic examination. Examination of tissues from lesions caused by *Stephanofilaria* will reveal both adults and microfilariae.

Size: <i>Onchocerca</i> microfilariae	approximately 200 to >300 $\mu\text{m}$ in length, depending on species
<i>Stephanofilaria</i> microfilariae	approximately 50 $\mu\text{m}$

**Clinical Importance:** *Onchocerca* spp. are not generally considered to be highly pathogenic parasites, although equine *O. cervicalis* infection can be associated with dermatitis in horses, and some of the bovine species cause skin or connective tissue lesions that must be trimmed at slaughter. In North America, localized ventral midline dermatitis caused by *Stephanofilaria* is relatively common in adult cattle but has no clinical significance.



**Fig. 27** Microfilariae of *Onchocerca gutturosa* (arrow) in bovine skin. Diagnosis can be made more rapidly by incubating skin biopsies in saline and examining the fluid several hours later for moving microfilariae that have migrated out of the skin. Photo courtesy of Dr. Fernando Paiva, Universidade Federal de Mato Grosso do Sul, Campo Grande, MS, Brazil.



**Fig. 28** Horn flies (*Haematobia irritans*) feeding on a *Stephanofilaria stilesi* lesion on the ventrum of a bovine host. The flies are the intermediate host of the parasite and ingest microfilariae in the lesion during feeding. *Stephanofilaria stilesi* is common, but clinically unimportant, in North America. Photo courtesy of Dr. Jeffrey F. Williams, Vasso HaloSource, Inc., Redmond, WA.

**PARASITE:** *Onchocerca lupi* (Fig. 2.9)

**Taxonomy:** Nematodes (order Spirurida).

**Geographic Distribution:** Europe, North America.

**Location in Host:** Adults usually occur in small subconjunctival nodules or cysts in other tissues around the eye in dogs and, more rarely, cats.

**Life Cycle:** A black fly (Fam. Simuliidae) ingests microfilariae during a blood meal. Larvae develop to the infective third larval stage in the fly and are deposited on another host during feeding.

**Laboratory Diagnosis:** Diagnosis is made by detection of nodules around the eye or skin biopsy of affected areas and examination for microfilariae following saline incubation or histopathologic examination.

Size: microfilariae  $110\text{--}120 \times 5\text{--}7 \mu\text{m}$

**Clinical Importance:** *Onchocerca lupi* may cause a variety of signs associated with its location, including exophthalmos, periocular swelling and discharge. Human cases have also been reported.

**PARASITE:** *Dracunculus insignis* (Fig. 2.10)

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** North America. Infection with the important human parasite, *Dracunculus medinensis*, has recently been described in dogs and cats in Africa.

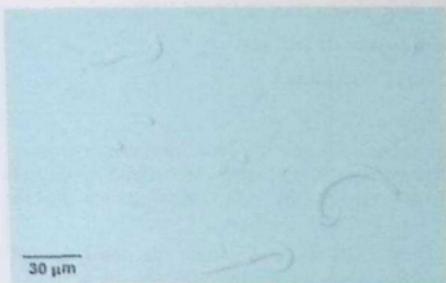
**Location in Host:** Adults occur in the subcutaneous tissue, usually on the limbs, of raccoons and other wild animals. Dogs are occasionally infected. Infection in cats is rare.

**Life Cycle:** The female worm causes an ulcer on the skin of a limb. When the limb is placed in water, the female extrudes a portion of the uterus, which ruptures, releasing first-stage larvae into the water. Larvae are ingested by a copepod, *Cyclops*, which acts as the intermediate host. The definitive host becomes infected by drinking water containing the infected intermediate host.

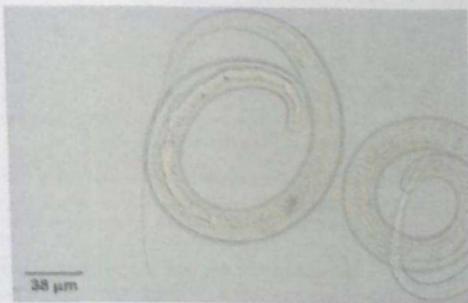
**Laboratory Diagnosis:** Diagnosis is usually made by examination of the skin lesion and removal of the worm.

Size: First-stage larva  $596\text{--}857 \mu\text{m}$

**Clinical Importance:** *Dracunculus* can cause a chronic ulcer that may develop secondary bacterial infection.



**Fig. 2.8** Microfilariae of *Onchocerca lupi* from the uterus of an adult worm. They are present in skin biopsies of infected animals and are the smallest microfilariae found in dogs. Photo courtesy of Dr. Gui Verocai, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX.



**Fig. 2.9** First-stage larvae of *Dracunculus insignis*. These distinctive larvae with their long, thin tails can be teased from the uterus of a worm removed from a skin ulcer and confirm the diagnosis of *Dracunculus* infection.

**PARASITE:** *Onchocerca lupi* (Fig. 2.9)

**Taxonomy:** Nematodes (order Spirurida).

**Geographic Distribution:** Europe, North America.

**Location in Host:** Adults usually occur in small subconjunctival nodules or cysts in other tissues around the eye in dogs and, more rarely, cats.

**Life Cycle:** A black fly (Fam. Simuliidae) ingests microfilariae during a blood meal. Larvae develop to the infective third larval stage in the fly and are deposited on another host during feeding.

**Laboratory Diagnosis:** Diagnosis is made by detection of nodules around the eye or skin biopsy of affected areas and examination for microfilariae following saline incubation or histopathologic examination.

**Size:** microfilariae 110–120 × 5–7 µm

**Clinical Importance:** *Onchocerca lupi* may cause a variety of signs associated with its location, including exophthalmos, periorbital swelling and discharge. Human cases have also been reported.

**PARASITE:** *Dracunculus insignis* (Fig. 2.10)

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** North America. Infection with the important human parasite, *Dracunculus medinensis*, has recently been described in dogs and cats in Africa.

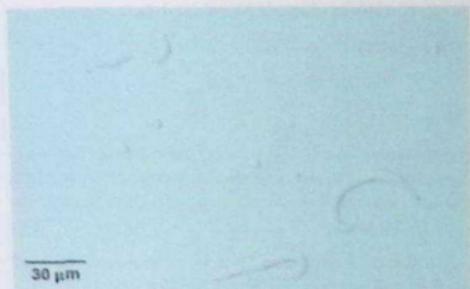
**Location in Host:** Adults occur in the subcutaneous tissue, usually on the limbs, of raccoons and other wild animals. Dogs are occasionally infected. Infection in cats is rare.

**Life Cycle:** The female worm causes an ulcer on the skin of a limb. When the limb is placed in water, the female extrudes a portion of the uterus, which ruptures, releasing first-stage larvae into the water. Larvae are ingested by a copepod, *Cyclops*, which acts as the intermediate host. The definitive host becomes infected by drinking water containing the infected intermediate host.

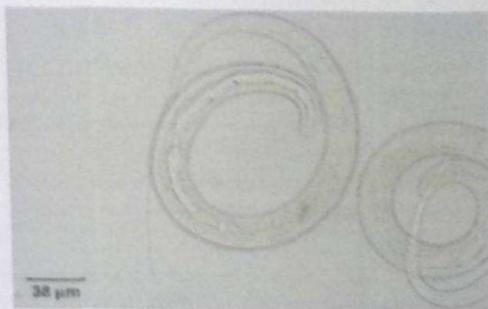
**Laboratory Diagnosis:** Diagnosis is usually made by examination of the skin lesion and removal of the worm.

**Size:** First-stage larva 596–857 µm

**Clinical Importance:** *Dracunculus* can cause a chronic ulcer that may develop secondary bacterial infection.



**FIG. 2.28** Microfilariae of *Onchocerca lupi* from the uterus of an adult worm. They are present in skin biopsies of infected animals and are the smallest microfilariae found in dogs. Photo courtesy of Dr. Gui Verocai, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX.



**FIG. 2.29** First-stage larvae of *Dracunculus insignis*. These distinctive larvae with their long, thin tails can be teased from the uterus of a worm removed from a skin ulcer and confirm the diagnosis of *Dracunculus* infection.

**PARASITE:** *Pelodera strongyloides* (Figs. 2.11 and 2.12)

**Taxonomy:** Nematode (order Rhabditida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Skin.

**Life Cycle:** *Pelodera strongyloides* is a free-living nematode that may invade the skin of animals. It is usually seen as a pathogen where animals are confined to areas with moist bedding high in organic material that will support growth and development of the nematodes.

**Laboratory Diagnosis:** Parasite infection can be diagnosed by finding larvae with a rhabditiform esophagus in skin scrapings from affected areas.

**Clinical Importance:** *Pelodera* is an uncommon cause of dermatitis in a variety of animal species.

**PARASITE:** *Thelazia* spp. (Fig. 2.13)

Common name: Eye worm.

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Conjunctival sac and lacrimal duct of cattle, horses, dogs.

**Life Cycle:** Muscid flies act as intermediate hosts and ingest first stage larvae released by adult worms into the tears. Development to the third larval stage occurs in the fly, which deposits the infective larvae on the host during feeding.

**Laboratory Diagnosis:** Laboratory diagnosis is unnecessary since the worms can be seen during examination of the eyes of the host.

Size: Adult worms 1–2 cm

**Clinical Importance:** Worms may cause tearing and conjunctivitis, but many chronic cases are asymptomatic. Human cases of infection are occasionally reported.

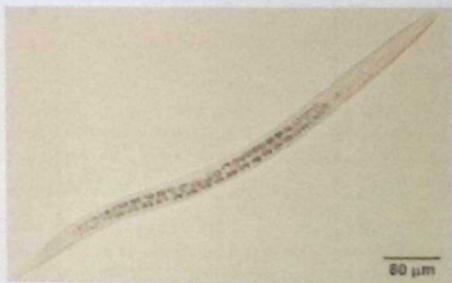


Fig. 2.77 Third-stage larva of *Pinodera strongyloides* recovered from a canine skin lesion. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 2.78 (A) Anterior end of *Pinodera* third-stage larva. The bulb of the rhabditiform esophagus (R) and long buccal tube lined with cuticle (C) are indicated. (B) *Pinodera* dermatitis in a dog. Larvae are recovered in skin scrapings. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 2.88 *Thelazia* in the eye of a cow. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**PARASITE:** *Besnoitia* spp. (Figs. 2.14 and 2.15)

Common name: Elephant skin disease (cattle).

**Taxonomy:** Protozoa (coccidia).

**Geographic Distribution:** *Besnoitia besnoiti* (cattle, goats) is found worldwide but is of greatest importance in Africa. *Besnoitia bennetti* (horses and donkeys) occurs primarily in Africa, southern Europe, and South America but is also found in North America.

**Location in Host:** Cysts are seen in the subcutaneous tissue and scleral conjunctiva of livestock.

**Life Cycle:** The definitive host of this coccidian parasite is the cat. Oocysts are shed in cat feces and are ingested by the intermediate host (cattle, horses, etc.). Parasites multiply in the endothelial cells and finally form large cysts in the subcutaneous tissue of intermediate hosts.

**Laboratory Diagnosis:** *Besnoitia* spp. often form cysts ("pearls") in the scleral conjunctiva that can easily be seen with the naked eye. Diagnosis can also be made by examining stained sections of skin biopsies for the presence of subcutaneous cysts.

**Clinical Importance:** Many cases are asymptomatic. However, an acute febrile illness can develop, followed by thickening and wrinkling of skin that makes the hide unsuitable for leather production. Affected animals may recover slowly. Although *Besnoitia* occurs in North America, its extent is unknown, and it rarely seems to cause clinical disease.



Fig. 2.14 Extensive skin thickening and wrinkling caused by *Bessonia bennetti*. This disease is most prevalent in Africa. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 2.15 Scleral "pearls," or cysts, of *Bessonia bennetti* in the eye of a donkey in North America. Photo courtesy of Dr. Hany M. Elsheikha and Dr. Charles Mackenzie, College of Veterinary Medicine, Michigan State University, East Lansing, MI.



# Detection of Parasites in the Blood

Various pathogenic and nonpathogenic protozoa and nematodes may be detected in blood samples from domestic animals. Most of these parasites are ingested by arthropod vectors during feeding and are present in the blood of their vertebrate hosts as a normal part of their life cycles.

## IMMUNOLOGIC AND MOLECULAR DETECTION OF BLOOD PARASITES

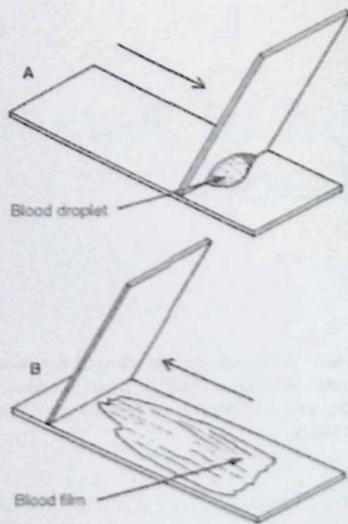
Although the focus of this book is the morphologic diagnosis of parasitism, it is important to recognize that immunologic tests are widely used in conjunction with or in place of microscopic examination of blood smears for some blood-borne parasites, and the use of these tests can be expected to increase in the future. Immunologic and molecular tests offer increased sensitivity compared with morphologic techniques in many cases and are especially valuable in some chronic hemoprotozoan infections and in canine and feline heartworm infection. In both cases, many infections are undetectable by routine microscopic tests. The commercial tests used most widely are the indirect fluorescent antibody (IFA) test, the enzyme-linked immunosorbent assay (ELISA), and polymerase chain reaction (PCR). For additional discussion of the basis and use of immunodiagnostic and molecular diagnostic procedures in veterinary parasitology, see Chapter 4.

## MICROSCOPIC EXAMINATION OF BLOOD FOR PROTOZOAN PARASITES

Most hemoprotozoan parasites are intracellular in erythrocytes or white blood cells and may cause anemia. A routine thin blood smear is therefore useful both for assessing erythrocyte abnormalities and for detecting the presence of parasites. Parasites are most likely to be detected in blood smears during acute infection. Once infections become

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard.

© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)



**Fig. 3.1** Technique for making a blood smear. (A) Bring a spreader slide back at an angle until it touches the drop of blood; wait until the drop flows laterally. (B) Draw the spreader slide away from the drop, maintaining an angle. The blood will spread into a smooth, thin film.

chronic, immunologic or molecular diagnostic techniques are usually more sensitive as parasitemias can drop below detectable limits by light microscopy.

For microscopic examination of blood smears for hemoprotezoa, Giemsa stain is most effective, but Wright's stain can also be used in most cases. Commercial stain kits used in many veterinary practices (an example is Dip Quick Stain, Jorgensen Laboratories, Loveland, CO, [www.jorvet.com](http://www.jorvet.com)) will also stain hemoprotezoa when used as directed, but the stain will be of poorer quality. The following procedure can be used for Giemsa stain.

To prepare a thin blood smear, place a drop of blood on one end of a microscope slide and draw the blood into a thin film as shown in Figure 3.1.

### Giemsa Stain

- 1 Air-dry the blood film, protecting it from flies and other insects if it is not to be stained immediately.
- 2 Fix in absolute methanol for 5 minutes and air-dry.
- 3 Dilute stock Giemsa stain 1 : 20 with distilled water and flood the film (or place slide in staining jar). Fresh stain should be prepared at least every 2 days.
- 4 Stain for 30 minutes.
- 5 Wash stain away gently with tap water.
- 6 Air-dry; parasite cytoplasm will stain blue, and nuclei will stain magenta.

Table 3.1. Average diameters of erythrocytes

Animal	Erythrocyte diameter ( $\mu\text{m}$ )
Horse	5.5
Cattle	5.8
Sheep	4.5
Goat	3.2
Dog	7.0
Cat	5.8
Chicken	$7.0 \times 12.0$

Source: Measurements from Weiss and Wardrop (2010).

The stained blood film can be scanned using the  $40\times$  objective of the microscope with use of the oil immersion lens for greater detail when suspected parasites are found.

The dimensions of blood parasites are best determined by means of an ocular micrometer (see Chapter 1). A micrometer is highly recommended for accurate measurement of parasites in blood and fecal samples. If a micrometer is not available, the size of the parasite on a blood film may be approximated by comparison with the dimensions of host erythrocytes (Table 3.1).

## MICROSCOPIC EXAMINATION OF BLOOD FOR NEMATODE PARASITES

Many species of parasitic worms enter the bloodstream of the host to reach certain organs, where they develop to maturity. These parasites usually stay in the blood only minutes or hours; thus, they are seldom seen in blood samples taken for diagnostic purposes. There are some filarial nematodes, however, whose larvae (e.g., microfilariae) are normally found in the peripheral blood. The microfilarial stage of these parasite species remains in the circulation until ingestion by the bloodsucking intermediate host. Microfilaria testing is often performed for detection of canine heartworm infection. The following discussion of techniques for microfilaria detection is directed specifically to *Dirofilaria immitis* testing. However, all species of microfilariae in the blood could be detected by the same microscopic techniques.

Although the techniques for microscopic detection of heartworm microfilariae are presented below, the ELISA antigen test for diagnosis of canine heartworm infection is a commonly used screening test. Antigen tests are significantly more sensitive than microfilaria tests because many heartworm infections are amicrofilaricemic (occult infections). The absence of microfilariae may be due to low or single-sex worm burdens or immune clearance of microfilariae. Moreover, some heartworm preventives are microfilaricidal and may render infected dogs amicrofilaricemic after one or several months of administration.

The American Heartworm Society and the Companion Animal Parasite Council currently recommend annual testing using an antigen test and a microfilaria test to identify *D. immitis* infection in dogs. Dogs testing positive on an antigen test should always be tested for microfilariae to determine if microfilaricidal treatment is necessary. Antigen tests currently available in the United States are available in ELISA and immunochromatographic formats. Differences in sensitivity among these tests have been found experimentally and are particularly evident when only a few adult worms are present. False-negative antigen results can occur, especially when immune complexes have formed, precluding detection. Pre-treatment of the serum or plasma to disrupt

immune complexes using heat or chemicals prior to performing the antigen test releases the antigen and allows detection. Because specialized equipment is required, sending a sample to a diagnostic laboratory is recommended when blocked antigen is suspected. Available tests are considered highly specific although false-positive results have been reported both before and after treatment to reveal blocked antigen.

Diagnosis of heartworm infection in cats is more difficult than in dogs. Several of the antigen tests can be used in cats, but false-negative results are common because of immune complex formation as well as the low worm burdens usually found in cats. Similarly, cats are rarely microfilaremic and may develop disease before the adult stage, detectable by antigen testing, is present. To improve the sensitivity of heartworm detection in cats, heartworm antibody tests have been developed. These tests can detect infection earlier than antigen tests but may only indicate exposure to the parasite rather than active infection. Care should be taken in interpreting a feline antibody test, and the results of that test alone should not be used to establish a diagnosis of heartworm infection. In a cat showing clinical signs consistent with heartworm infection, both antigen and antibody tests should be performed as part of the diagnostic workup.

For current recommendations of the American Heartworm Society and the Companion Animal Parasite Council relating to diagnosis and treatment of heartworm infection in dogs and cats, consult the websites of the two organizations: [www.heartwormsociety.org](http://www.heartwormsociety.org) and [www.capevet.org](http://www.capevet.org).

### Tests for Canine Heartworm Microfilariae in Blood Samples

The following techniques can be used to detect microfilariae in blood samples. The canine heartworm, *Dirofilaria immitis*, is found throughout the world. In North America, dogs may also be infected with *Acanthocheilonema* (= *Dipetalonema*) *reconditum* or, rarely, with *Dirofilaria striata*, a parasite of wild felids in North and South America. In parts of Europe, Asia, and Africa, *Dirofilaria repens* and *Acanthocheilonema dracunculoides* parasitize dogs. When a microfilaria test is used for heartworm diagnosis, the microfilariae of other species must be differentiated from those of *D. immitis*.

Staining characteristics can be used in discriminating among species, but are not usually performed in veterinary practices. Measurement of total length, width, and the shape of the head can also aid microfilaria identification (Table 3.2). Sizes should be determined with an eyepiece micrometer (see Chapter 1 for micrometer calibration procedure). The standard measurements of microfilariae in Table 3.2 were determined with formalin-fixed specimens; use of other fixatives or lysing solutions may alter the size of the organisms. Similarly, storage of microfilariae in blood samples for more than 3 days may cause *D. immitis* microfilariae to shrink in length to the size of *A. reconditum*.

#### Wet Mount

The wet mount is the simplest and most rapid of the procedures for microfilariae detection. It is not a very sensitive technique but can be used in conjunction with an adult heartworm antigen test to determine if microfilariae are present or to evaluate the pattern of movement of microfilariae when attempting to differentiate between *Dirofilaria* and *Acanthocheilonema*.

Place one drop of anticoagulated venous blood onto a clean microscope slide and coverslip.

**Table 3.2. Characteristics of *Dirofilaria* spp. and other microfilariae found in canine blood based on formalin-fixed specimens**

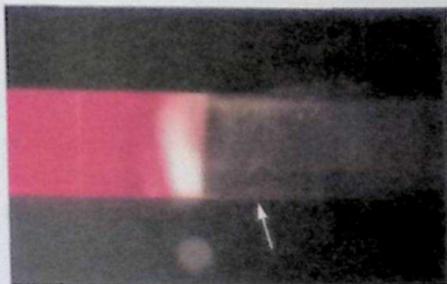
	<i>Dirofilaria immitis</i>	<i>Dirofilaria repens</i>	<i>Dirofilaria striata</i>	<i>Acanthocheiloneema reconditum</i>	<i>Acanthocheiloneema dracunculoides</i>
Length (µm)	295–325	268–360	360–385	250–288	189–230
Width (µm)	5–7.5	5–8	5–6	4.5–5.5	5–6
Head	Tapered	Blunt	Tapered	Blunt	Blunt
Tail	Straight	Variable—straight or hooked	Curved	Variable—hooked (30%) or curved	Sharp and extended
Body shape	Straight		S-shaped	Curved	
Motion (live)	Stationary		Stationary	Progressive	
Relative number	Few to many		Few	Few	
Location of adult	Pulmonary arteries, right heart	Subcutaneous intramuscular tissues	Subcutaneous intramuscular tissues	Subcutaneous tissues	Peritoneum
Geographic location	Worldwide	Europe, Africa, Asia	North and South America	Africa, Europe, North America	Africa, Europe, India

- Examine the coverslip area under low magnification (10×) of the microscope. Look for undulating movements of larvae, which may retain motility for as long as 24 hours.

### Hematocrit Test

This technique is only slightly more sensitive than the wet mount:

- Draw fresh whole blood into a microhematocrit tube.
- Spin for 3 minutes in a hematocrit tube centrifuge.
- Examine the plasma portion of the separated blood, while still in the tube, under low magnification (10×). Moving microfilariae will be present in the plasma above the buffy coat (Fig. 3.2).



**Fig. 3.2** Results of a hematocrit test using a blood sample containing *D. immitis* microfilariae. The microfilariae can be seen as a hazy layer to the right of the buffy coat layer (arrow). Closer microscopic examination would show individual moving microfilariae.

The wet mount and microhematocrit techniques may not detect infections with only small numbers of microfilariae. Therefore, if a microfilariae test is used as a screening procedure for heartworm infection, one of the following concentration techniques should be used.

### **Modified Knott's Test**

The modified Knott's technique is the preferred concentration method for the detection and identification of microfilariae in blood:

1. Draw a sample of blood into a syringe containing anticoagulant such as EDTA or heparin.
2. Mix 1 mL of the blood with 9 mL of a 2% formalin solution. If not well mixed, the red cells will not be thoroughly lysed by the hypotonic formalin solution, making the test much more difficult to read. Microfilariae, but not red cells, will be fixed by 2% formalin. If 10% formalin is used (the concentration used for fixation of tissues), red cells will also be fixed and not lysed.
3. Centrifuge the mixture at 1200 rpm for 5 minutes (or as for fecal flotation procedures) and discard the supernatant.
4. Add one drop of 0.1% methylene blue to the sediment, mix well, and transfer the entire stained sediment to a microscope slide using a Pasteur pipette.
5. Examine using the 10 $\times$  microscope objective. Microfilariae will be fixed in an extended position with nuclei stained blue.

An alternative procedure using the same amount of blood is the filter test, which traps microfilariae on a filter that is examined with the microscope. This technique can be performed more quickly than the modified Knott's test, but microfilariae are not easily measured for identification. Materials for performing the filter test were sold as a kit (Difil-Test<sup>®</sup>), which is no longer available. Components of the test can be purchased individually if desired.

### **Filter Test**

1. Mix 1 mL of blood with 9 mL lysing solution (2% formalin) in a syringe.
2. Attach the syringe to a filter holder containing a transparent 25 mm filter with a 5- $\mu$ m pore size and empty the syringe.
3. Refill syringe with water and pass it through the filter to wash away remaining small debris.
4. Refill syringe with air, reattach to the filter apparatus, and express.
5. Unscrew the filter assembly, remove the filter with forceps, and place the filter on a microscope slide.
6. Add one drop of 0.1% methylene blue, coverslip, and examine at 10 $\times$ .

## BLOOD PARASITES OF DOGS AND CATS

**Parasite:** *Hepatozoon* spp. (Fig. 3.3)

**Taxonomy:** Protozoa (hemogregarine).

**Geographic Distribution:** *Hepatozoon canis* occurs worldwide, while the distribution of *Hepatozoon americanum* appears to be limited to the southeastern United States.

**Location in Host:** Gamonts are found in polymorphonuclear leukocytes (*H. americanum*, *H. canis*) and meronts in skeletal muscle (*H. americanum*) or various organs (*H. canis*) of dogs, cats, and various wild carnivores.

**Life Cycle:** Ticks acquire infection during feeding. Dogs become infected by ingesting infected ticks. *Hepatozoon americanum* is transmitted by *Amblyomma maculatum* (the Gulf Coast tick), and the vector of *H. canis* is *Rhipicephalus sanguineus* (the brown dog tick).

**Laboratory Diagnosis:** Sausage-shaped *Hepatozoon* gamonts can be detected in polymorphonuclear leukocytes in Wright- or Geimsa-stained blood smears. Although this method of diagnosis readily reveals *H. canis*, *H. americanum* is rarely found on blood smears, and a molecular diagnostic test may be necessary. Morphologic diagnosis of this species generally occurs by the detection of meronts in skeletal muscle biopsies or on histopathology after necropsy.

**Size:** Gamonts  $8-12 \times 3-6 \mu\text{m}$

**Clinical Importance:** *Hepatozoon americanum* can cause severe disease, with fever, depression, joint pain, myositis, periosteal bone proliferation, and chronic wasting. *Hepatozoon canis* infections are usually subclinical.



**Fig. 3.3** *Hepatozoon* gamont in a polymorphonucleocyte. The parasite is sausage-shaped with a centrally compact nucleus that stains only faintly in this specimen (arrow). *Hepatozoon americanum* is rarely detected in blood films, and muscle biopsies are a more sensitive means of diagnosis.

**PARASITE:** Large (e.g., *Babesia canis*) and small (e.g., *B. gibsoni*) *Babesia* spp (Figs. 3.4 and 3.5) *Babesia* spp.

**TAXONOMY:** Protozoa (piroplasm). *Babesia* spp. are divided into large (>4  $\mu\text{m}$ ) and small (<3  $\mu\text{m}$ ). Large species include *B. canis vogeli*, *B. canis rossii*, *B. canis canis*, *Babesia* sp. (Coco), and an unnamed British isolate. Small *Babesia* include *B. gibsoni*, *B. conradae*, and *B. vulpes*.

**Geographic Distribution:** *Babesia* mostly occurs in the tropical and subtropical regions of the world. *B. canis vogeli* is found worldwide, *B. canis rossii* in Africa, *B. canis canis* in Europe, and *Babesia* sp. (Coco) has been reported sporadically in immunocompromised dogs in various U.S. states. *Babesia gibsoni* is widely distributed throughout most of the world, *B. conradae* in dogs from California and Oklahoma, and *B. vulpes* infects a variety of wild canids (primarily foxes) and occasionally domestic dogs in Europe, North America, and western Asia.

**Location in Host:** Canine red blood cells. *Babesia* spp. have been described in cats but are not widely distributed and do not appear to be present in North America.

**Life Cycle:** Ticks are definitive hosts for *Babesia* spp. In North America, dogs acquire *B. canis vogeli* from the brown dog tick, *Rhipicephalus sanguineus*. Other tick vectors include *Dermacentor reticulatus* in Europe and *Haemaphysalis leachi* in Africa. A definitive tick vector for *B. gibsoni* has not been demonstrated in North America and transmission is thought to occur primarily or only through the transfer of blood contaminated with piroplasms. Dog fighting increases the risk of infection with *B. gibsoni*.

**Laboratory Diagnosis:** Piroplasms can be detected in erythrocytes on Wright- or Giemsa-stained blood smears. Immunologic and molecular diagnostic tests can also be used and are a more sensitive diagnostic technique in chronic infections. *Babesia canis vogeli* is pear-shaped and usually occurs in pairs; *B. gibsoni* is round to oval-shaped.

Size:	Large <i>Babesia</i> spp.	4–5 $\mu\text{m}$
	Small <i>Babesia</i> spp.	1–3 $\mu\text{m}$

**Clinical Importance:** Severity of clinical disease may range from mild to life threatening. Anemia, hemolytic crisis, and multi-organ failure can occur. North American and European strains appear to be less pathogenic than those infecting dogs in Africa and Asia.

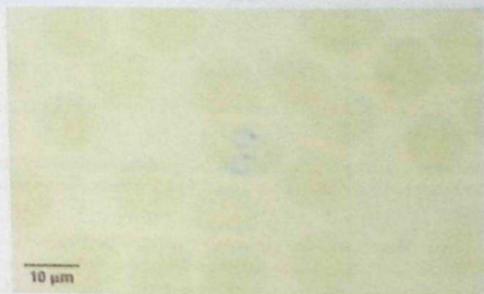


Fig. 5.4 Erythrocyte containing pear-shaped *Babesia canis* piroplasm. The parasite is usually found in pairs, as many as eight piroplasms may be found in a single red blood cell.

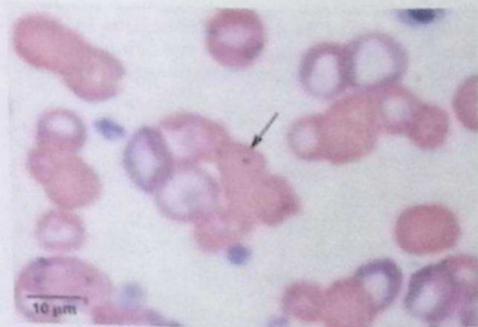


Fig. 5.5 *Babesia gibsoni* (arrow) is a smaller organism than *B. canis*. The pear shape seen so clearly with *B. canis* is much less distinct with *B. gibsoni*. Photo courtesy of Dr. Kurt Zimmerman, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

**PARASITE:** *Cytauxzoon felis* (Figs. 3.6 and 3.7)

**TAXONOMY:** Protozoa (piroplasm).

**Geographic Distribution:** Southern United States.

**Location in Host:** Merozoites occur in red blood cells, and schizonts occur in histiocytes of bobcats and cats.

**Life Cycle:** Transmission occurs through the blood-feeding activities of the tick vectors. *Amblyomma americanum* and *Dermacentor variabilis* have been shown to transmit infections experimentally.

**Laboratory Diagnosis:** Merozoites are detected in red blood cells (1–4 merozoites/erythrocytes) in Wright- or Giemsa-stained blood smears. Schizonts are detected in mononuclear cells in spleen, lymph nodes, and bone-marrow aspirates. PCR diagnostic tests are also available.

Size: 1–2  $\mu\text{m}$

**Clinical Importance:** *Cytauxzoon felis* is highly pathogenic in cats. Infections are usually fatal; cats die within a few days of the onset of clinical signs. In the last 20 years, more cats have been found to survive cytauxzoonosis and treatment should be initiated if possible. Anemia, depression, high fever, icterus, hepatomegaly, and splenomegaly occur.

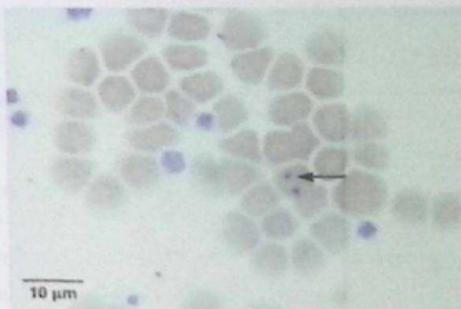


Fig. 8.6 Small *Cytauxzoon felis* merozoites in infected erythrocytes (arrow) have a dark nucleus and a light-blue cytoplasm on Wright- or Giemsa-stained blood smears. Photo courtesy of Dr. Karen F. Snowden, College of Veterinary Medicine, Texas A&M University, College Station, TX.

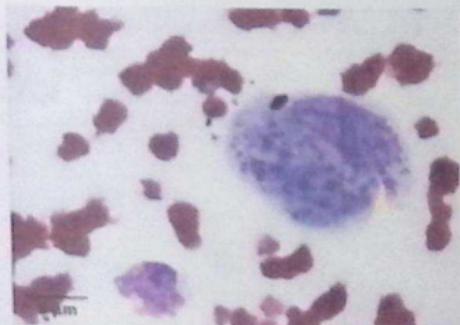


Fig. 8.7 Mononuclear cell containing a *Cytauxzoon felis* schizont. Photo courtesy of Dr. Kurt Zimmerman, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

**PARASITE:** *Leishmania* spp. (Figs. 3.8 and 3.9)

Common name: Visceral and cutaneous leishmaniasis.

**TAXONOMY:** Protozoa (hemoflagellate). Species include *L. donovani*, *L. tropica*, *L. infantum*, *L. chagasi*, *L. braziliensis*, *L. mexicana*.

**Geographic Distribution:** Worldwide.

**Location in Host:** Amastigotes occur in macrophages and cells of the reticuloendothelial system of various organs (skin, spleen, liver, bone marrow, lymph nodes).

**Life Cycle:** Blood-feeding sand flies (*Lutzomyia*, *Phlebotomus*) serve as vector.

**Laboratory Diagnosis:** Diagnosis occurs by detection of amastigotes in macrophages in stained smears made from needle aspirate biopsies of lymph node, bone marrow, or spleen or in impression smears of skin lesions. Amastigotes are rarely seen in stained peripheral blood smears. Serologic and polymerase chain reaction (PCR) techniques are also used in diagnosis.

Size: 2.5–5.0 × 1.5–2.0 μm

**Clinical Importance:** Infection in dogs is often subclinical. However, disease may develop involving various visceral organs and skin, resulting in cutaneous lesions, lethargy, progressive weight loss, and anorexia that may end in death. Infection with *L. infantum* is common in foxhounds in the United States. Cats are rarely infected. Leishmaniasis is a serious, potentially fatal disease in humans. Dogs serve as an important reservoir host of the parasite in some parts of the world.

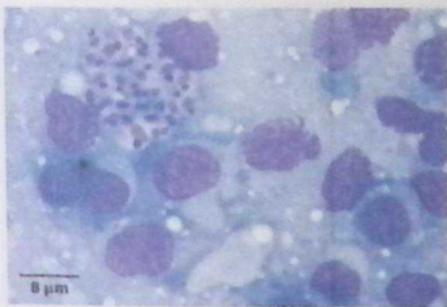


Fig. 5.8 *Leishmania* sp. amastigotes in a lymph node impression smear. Photo courtesy of Dr. Karen F. Snowden, College of Veterinary Medicine, Texas A&M University, College Station, TX.

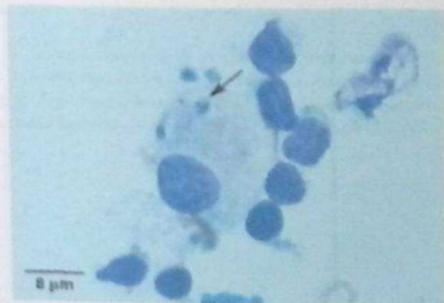


Fig. 5.9 *Leishmania* amastigote (arrow) in a macrophage from a canine lymph node. The small round kinetoplast can be seen adjacent to the nucleus in this amastigote. Photo courtesy of Dr. Bernard Feldman, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.

**PARASITE:** *Trypanosoma cruzi* (Fig. 3.10)

Common name: Chagas disease.

**TAXONOMY:** Protozoa (hemoflagellate). Dogs and cats can also be infected with *T. brucei*, *T. congolense*, and *T. evansi*, trypanosome species more commonly associated with large animals (see below).

**Geographic Distribution:** North and South America.

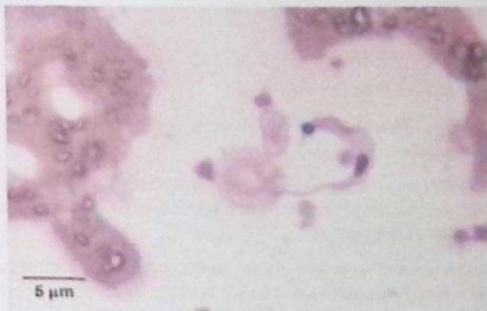
**Location in Host:** Trypomastigotes occur in the blood; amastigotes and epimastigotes occur in skeletal muscle, reticuloendothelial cells, and various other tissues of humans, dogs, cats, and many wildlife mammalian species.

**Life Cycle:** Triatomids (kissing or assassin bugs) pass trypomastigotes in the feces during blood feeding on the vertebrate definitive host. Parasites enter the definitive host through mucous membranes or through the triatomid bite-wound site.

**Laboratory Diagnosis:** Trypomastigotes are detected on Wright- or Giemsa-stained blood smears early in infection. Diagnosis in chronic or light infections may require serologic tests, culture, or xenodiagnosis.

Size: 16–20  $\mu\text{m}$

**Clinical Importance:** Infection with *T. cruzi* is highly pathogenic, causing acute and chronic cardiac disease. Lymphadenopathy, pale mucous membranes, lethargy, ascites, hepatomegaly, splenomegaly, anorexia, diarrhea, and neurologic signs may be seen. In North America infection is most common in the south central United States, particularly in Texas.



**FIGURE 3.10** Stained trypanomastigote of *Trypanosoma cruzi* in a blood smear from an infected dog. The organisms often assume a C shape in blood smears. The dark-staining kinetoplast can easily be seen in this specimen. Photo courtesy of Dr. Karen F. Snowden, College of Veterinary Medicine, Texas A&M University, College Station, TX.

**PARASITE:** *Dirofilaria immitis* (Figs. 3.11–3.13)

Common name: Heartworm.

**TAXONOMY:** Nematode (order Spirurida).

**Geographic Distribution:** Worldwide. *Dirofilaria striata* is a rare parasite of dogs in the southeastern United States.

**Location in Host:** Adult worms are found in the pulmonary arteries and right ventricle of dogs, wild canids, and ferrets. Cats are much less likely than dogs to become infected, and feline infections are rarely patent.

**Life Cycle:** Mosquitoes serve as intermediate hosts, acquiring microfilariae and transmitting infective third-stage larvae while blood feeding. The prepatent period in dogs is about 6–9 months.

**Laboratory Diagnosis:** The most sensitive technique for heartworm diagnosis is detection of antigen using one of the various commercial antigen kits for use with serum, plasma, and/or whole blood. Less sensitive is testing for microfilariae in blood samples using the Knott's test or a filter test, which is equal in ability to detect microfilariae. However, the Knott's test should be used for specific identification of microfilariae (on the basis of size and morphology).

Size: 295–325 × 5–7.5 μm

**Clinical Importance:** Heartworm infection is highly pathogenic and is an important medical health issue in both canine and feline medicine. Chronic heartworm infection in dogs can lead to fatal right-sided congestive heart failure. Caval syndrome occurs in some dogs with heavy worm burdens (>100) that, without prompt surgical removal, leads to a rapidly fatal hemolytic crisis. Heartworm infection in cats can be subclinical or result in severe chronic disease (respiratory or vomiting/gastrointestinal) or cats may die acutely.



Fig. 3.17 Microfilariae of *Dirofilaria immitis* recovered from a blood sample using the modified Knott's technique. Photo courtesy of Dr. Thomas Nolan, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA.

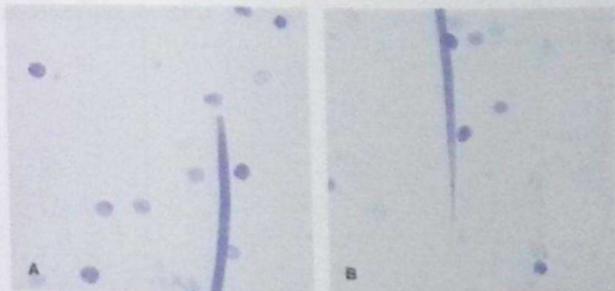


Fig. 3.18 Microfilariae of *Dirofilaria immitis* have gently tapered heads (A) and relatively straight tails (B).

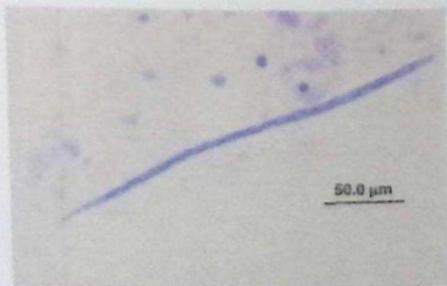


Fig. 3.19 Microfilariae are at an earlier developmental stage than first-stage larvae. Dark-staining cell nuclei can be seen in this *D. immitis* microfilaria. Photo courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Acanthocheilonema* (= *Dipetalonema*) *reconditum* (Figs. 3.14 and 3.15)

**Taxonomy:** Nematode (order Spirurida).

**Geographic Distribution:** United States, South America, Africa, southern Europe, Asia.

**Location in Host:** Subcutaneous tissues of dogs and various wild canids.

**Life Cycle:** Dogs acquire infections from fleas (*Ctenocephalides*, *Pulex*) and lice (*Linognathus*, *Heterodoxus*). Arthropods ingest microfilariae in the blood (*Ctenocephalides*, *Pulex*, *Linognathus*) or skin (*Heterodoxus*) of infected canids.

**Laboratory Diagnosis:** Diagnosis is by detection of microfilariae as for *D. immitis*. Canine heartworm antigen tests do not give a positive reaction in the presence of *Acanthocheilonema* infection. Microfilariae of *A. reconditum* have a blunt anterior end, and the tails of some individuals may form a small hook or U shape, usually referred to as a "buttonhook."

**Size:** 250–288 × 4.5–5.5 μm

**Clinical Importance:** Infections with *A. reconditum* are subclinical. The accurate diagnosis of *A. reconditum* infections in dogs is important in order to prevent misdiagnoses of heartworm infection.

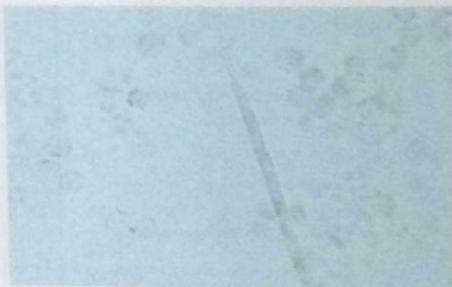


Fig. 3.14 Microfilariae of *Acanthocheilomena* (*Dipetalonema*) *reconditum* in a modified Knott's test. The anterior end is blunter than that of *D. immitis*.



Fig. 3.15 The tails of some individual microfilariae of *Acanthocheilomena reconditum* and *Dirofilaria repens* may form a buttonhook shape when fixed. See Table 3.2 for a comparison of morphologic characteristics of microfilariae found in dogs.

**PARASITE:** *Dirofilaria repens* (Figs. 3.15–3.17)

**TAXONOMY:** Nematode (Spirurida)

**Geographic Distribution:** Various countries in Europe, Asia, and Africa. Introduction into North America is considered a possibility.

**Location in Host:** Subcutaneous tissues of canids, felids, and various other carnivores.

**Life Cycle:** Transmission occurs by introduction of infective third-stage larvae through the bite of culicid mosquito intermediate hosts. After a prepatent period of 6–8 months, microfilariae are produced and circulate in the blood of infected hosts.

**Laboratory Diagnosis:** Microfilariae are detected in circulating blood by the modified Knott's test or by the use of filter tests. PCR tests have also been used. When detected by the modified Knott's examination, the *D. repens* microfilariae have a relatively blunt anterior end, and the tail may show the buttonhook preservation artifact as occurs with *A. reconditum*. The larger *D. repens* microfilariae may be differentiated from *D. immitis* and *A. reconditum* based on size (see Table 3.2). *Dirofilaria repens* can produce false-positive canine heartworm antigen tests, both before and after treatment to disrupt immune complexes.

**Size:** 268–360 × 5–8 μm

**Clinical Importance:** Infection in dogs and cats is usually subclinical. Infection can result in dermatitis with focal alopecia, pruritus, erythema, and crusting. Human infection can also occur.

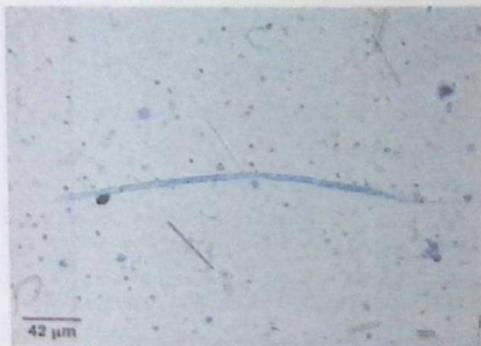


Fig. 3.13 *Dirofilaria repens* microfilaria. This species is expanding its range in Europe, and microfilariae can be difficult to distinguish from those of *Dirofilaria immitis*.

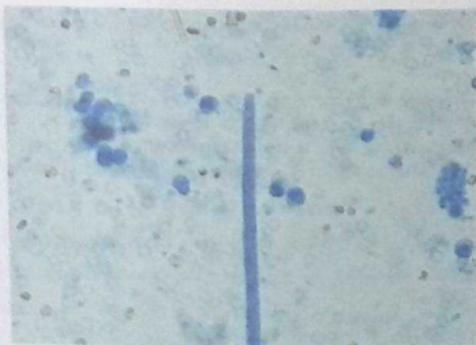


Fig. 3.14 The head of *Dirofilaria repens* is blunt in comparison to the tapering head of *D. immitis* (Fig. 3.12).

## BLOOD PARASITES OF LIVESTOCK AND HORSES

**PARASITE:** *Babesia* spp. of ruminants (Fig. 3.18)

Common name: Redwater, Texas cattle fever, tick fever.

**Taxonomy:** Protozoa (piroplasm).

**Geographic Distribution:** Worldwide, particularly in tropical regions.

**Location in Host:** Red blood cells of cattle (at least six species, including *B. bovis*, *B. divergens*, *B. bigemina*, *B. major*) and sheep and goats (*B. motasi*, *B. ovis*).

**Life Cycle:** A variety of tick genera, including *Ixodes* and *Rhipicephalus*, transmit *Babesia* spp. to ruminants. Ticks acquire the parasite during feeding.

**Laboratory Diagnosis:** In acute infection, blood smears stained with Giemsa or Wright's stain can be examined for parasites in red blood cells. In chronic infection, parasites are difficult to find in peripheral blood, and therefore, antibody tests, including IFA and ELISA tests, are used for diagnosis. *Babesia* typically appears as pairs of organisms in red blood cells, although erythrocytes may also contain single organisms.

**Size:** 1.5–4.5 × 0.4–2.0 μm, depending on species (*B. bigemina*: 4.5 × 2.5 μm; *B. bovis*: 2.4 × 1.5 μm)

**Clinical Importance:** In susceptible animals, infection can lead to the development of anemia, hemoglobinuria, and fever, with death often occurring during the acute phase of infection. Unlike many parasitic diseases, young animals are less likely to develop disease than adults. Bovine babesiosis has been eradicated from the United States.

**PARASITE:** *Babesia caballi*, *Theileria (Babesia) equi* (Figs. 3.19 and 3.20)

**Taxonomy:** Protozoa (piroplasm). *Theileria equi* was previously named *Babesia equi*.

**Geographic Distribution:** Equine piroplasmosis is endemic in Central and South America, Africa, southern Europe, and parts of Asia.

**Location in Host:** Equine erythrocytes. Infection is acquired from ticks in the genera *Rhipicephalus*, *Hyalomma*, *Amblyomma* and *Dermacentor*.

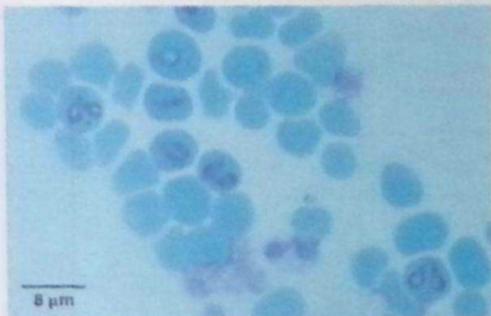
**Life Cycle:** Similar to ruminant *Babesia* spp.

**Laboratory Diagnosis:** Blood smears are examined in acute infection. Chronic carriers are unlikely to show parasites in the peripheral blood. ELISA and IFA tests are used for detecting antibody to parasites in chronic infection.

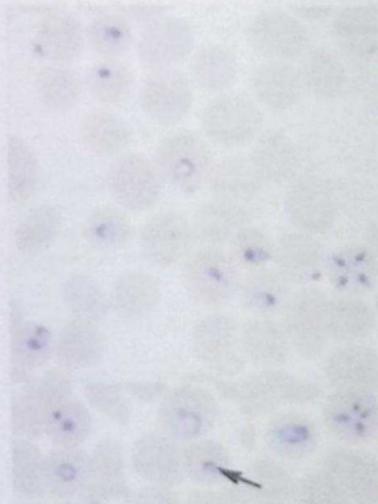
**Size:**

<i>T. equi</i>	2 μm
<i>B. caballi</i>	2.5–4 μm

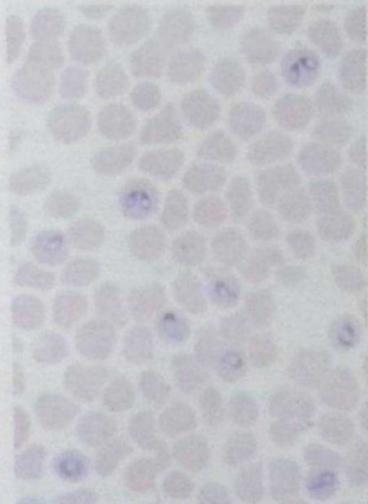
**Clinical Importance:** Equine piroplasmosis can cause anemia, hemoglobinuria, and edema. *Babesia caballi* may cause incoordination and paralysis.



**FIG. 3.17** *Babesia bigemina* can be seen in this bovine blood smear. The teardrop-shaped organisms are present in pairs in several erythrocytes. *Babesia trautmanni*, a cause of porcine babesiosis in parts of Europe and Africa, has a similar morphology. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.



**FIG. 3.18** Composite photo showing *Theileria equi* in equine red blood cells. The definitive diagnostic tetrad form is seen in the top right. Also shown in the photo are the small rings (arrow) that are most commonly seen in low parasitemia (carrier state). Photo courtesy of Dr. Patricia Holman, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX.



**FIG. 3.19** Composite photo of *Babesia caballi*. The definitive diagnostic form is the joined pair form, in contrast to the tetrad form of *Theileria equi* (Fig. 3.18). Also shown are other forms of the organism. The different large forms of the two species demonstrate the difficulty of distinguishing *B. caballi* from *T. equi* in the absence of paired puriforms in the former and tetrads in the latter. Photo courtesy of Dr. Patricia Holman, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX.

**PARASITE:** *Theileria* spp. (Figs. 3.21 and 3.22)

Common name: East Coast fever, corridor disease, African Coast fever, oriental theileriosis.

**Taxonomy:** Protozoa (piroplasm).

**Geographic Distribution:** *Theileria parva* in East Central and South Africa and *T. annulata* in North Africa and southern Europe. *Theileria orientalis* has been described worldwide and has recently been detected in the United States.

**Location in Host:** Bovine erythrocytes and lymph nodes.

**Life Cycle:** *Rhipicephalus* (*T. parva*), *Hyalomma* (*T. annulata*), and *Haemaphysalis* (*T. orientalis*) ticks are infected when they ingest host red blood cells. Following development in the tick, sporozoites are passed to cattle during feeding and enter lymphocytes, where schizogony occurs, releasing merozoites that infect red blood cells.

**Laboratory Diagnosis:** Schizonts can be seen in smears of lymph node biopsies and, in the case of *T. annulata* and *T. orientalis*, infected red blood cells may be seen in a blood smear. *Theileria parva* is unlikely to be present in blood smears except in advanced cases. IFA and ELISA tests are available, but may not detect acute infection and are of greater use in assessing host response in recovered animals. PCR can also be performed on blood samples, but may be negative in chronic infections.

Size:	Piroplasms in red blood cells	1.5–2.0 × 0.5–1.0 μm
	Schizonts in lymphocytes	approximately 8 μm

**Clinical Importance:** In African theileriosis, susceptible animals develop fever, lymphadenopathy, depression, and nasal discharge; there is high mortality in nonimmune animals. Chronic disease signs are variable, including diarrhea and reduced production. *Theileria orientalis* is less pathogenic, but has been associated with anemia and mortality in Australia and New Zealand.

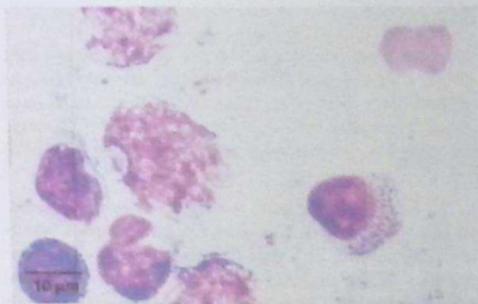
**PARASITE:** *Trypanosoma* spp. (Figs. 3.23–3.28)

Common name: Nagana, sleeping sickness, surra, dourine.

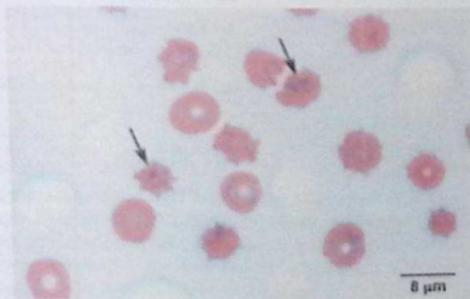
**Taxonomy:** Protozoa (flagellate).

**Geographic Distribution:** Clinically important livestock species, including *T. congolense*, *T. brucei brucei*, *T. simiae*, *T. vivax*, and *T. evansi*, are all found in Africa. *Trypanosoma evansi* and *T. vivax* are also found in South America and parts of Asia. *Trypanosoma theileri* (cattle only) and *T. melophagium* (sheep only) occur worldwide. Another species, *T. equiperdum* (horses), is found in tropical and subtropical regions.

**Location in Host:** Bloodstream of ruminants, horses, swine, and other domestic animals. *Trypanosoma brucei* can also be found in other tissues, including the heart and central nervous system and *T. equiperdum* is found in the urethra of stallions and vagina of mares.



**Fig. 3.21** *Theileria parva* multinucleated schizont in a lymphocyte. The species of *Theileria* are difficult to differentiate morphologically. Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.



**Fig. 3.22** *Theileria orientalis* in a bovine blood smear (arrows). Photo courtesy of Dr. Katie Boes, Virginia-Maryland College of Veterinary Medicine, Virginia Tech, Blacksburg, VA.



**Fig. 3.23** *Trypanosoma brucei* and *T. congolense* are found in domestic mammals in Africa. *Trypanosoma brucei* has a prominent undulating membrane (U) and a kinetoplast that is located subterminally (K). Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.

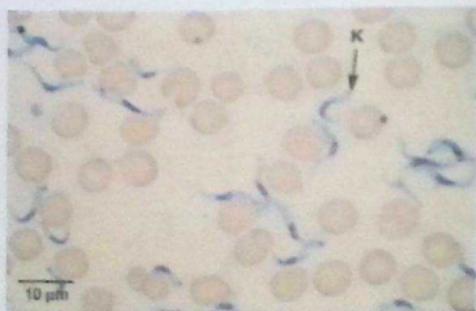
*Trypanosoma* (continued)

**Life Cycle:** Trypanosomes are transmitted to the host by biting flies. The vector of *T. congolense*, *T. vivax*, *T. brucei*, and *T. simiae* is the tsetse fly (*Glossina*) in Africa. In other areas, tabanid and other biting flies transmit *T. vivax* and *T. evansi*. Tabanid flies also transmit *T. theileri* in cattle, while the sheep ked (*Melophagus ovinus*) vectors *T. melophagium*. The exception to fly transmission is *T. equiperdum*, which is transmitted venereally.

**Laboratory Diagnosis:** In acute infection, most trypanosome species can usually be detected in stained smears of blood. Species of trypanosomes can be differentiated based on size, presence or absence of a free flagellum, location and size of the kinetoplast, and characteristics of the undulating membrane. An ELISA antigen test and DNA probes have been developed for detection of African trypanosomiasis in cattle. *Trypanosoma equiperdum* infection is diagnosed by a complement fixation test.

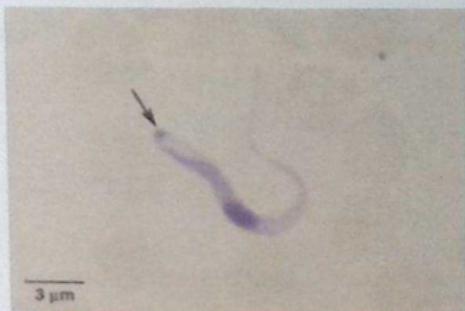
Size:	<i>T. vivax</i>	20–26 $\mu\text{m}$
	<i>T. brucei</i>	12–35 $\mu\text{m}$
	<i>T. congolense</i>	9–18 $\mu\text{m}$
	<i>T. evansi</i>	15–35 $\mu\text{m}$
	<i>T. theileri</i>	60–70 $\mu\text{m}$ , may be up to 120 $\mu\text{m}$
	<i>T. melophagium</i>	50–60 $\mu\text{m}$
	<i>T. simiae</i>	13–18 $\mu\text{m}$

**Clinical Importance:** The principal clinical signs of trypanosomiasis are anemia accompanied by fever, edema, and loss of condition. Mortality may be high, especially if other disease agents are also present. *Trypanosoma equiperdum* produces the disease known as dourine in horses, which is marked by genital and ventral edema, abortion, nervous system disease, and emaciation. *Trypanosoma melophagium* and *T. theileri* are widespread nonpathogenic species that may occasionally be seen in blood smears.

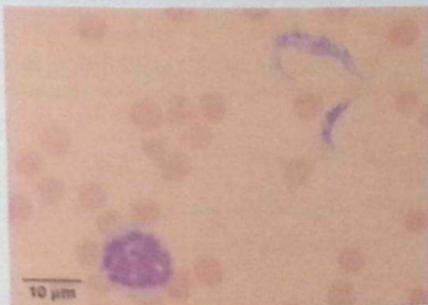


**Fig. 2.26** *Trypanosoma congolense* has a subterminal kinetoplast that is on the margin of the trypanosome (K). Its undulating membrane is not distinctive. Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.

**Fig. 2.25** *Trypanosoma vivax* is found in Africa and other parts of the world. Its kinetoplast is at the end of the organism (arrow), and its undulating membrane is not distinctive. Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.



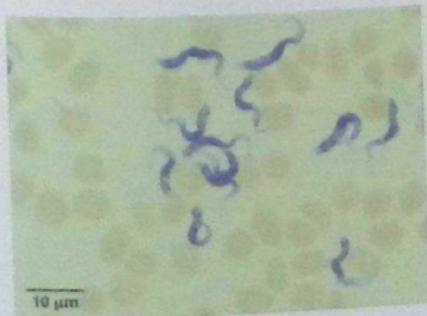
**Fig. 2.26** *Trypanosoma theileri* is found worldwide in cattle, and a similar parasite, *T. melophagium*, occurs in sheep. Parasites are occasionally seen in blood smears but rarely are of clinical importance. These species can be distinguished from the pathogenic trypanosome species by their large size (50 µm or more). This blood smear from a calf demonstrates the wide size variation that can occur with *T. theileri*.



**Fig. 2.27** *Trypanosoma evansi* is an important pathogen of horses and camels in parts of Africa, Asia, and Latin America. It is difficult to distinguish from *T. brucei* microscopically. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



**Fig. 2.28** *Trypanosoma simiae* is a parasite of African warthogs that is transmitted by the tsetse fly to domestic pigs and camels. Swine may also be infected with other trypanosome species. Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.



**PARASITE:** *Setaria* spp. (Fig. 3.29)

**TAXONOMY:** Nematode (order Spirurida).

**Geographic Distribution:** Worldwide.

**Location in Host:** Adult worms are found primarily in the peritoneal cavity of ruminants and equids.

**Life Cycle:** Microfilariae in the blood are ingested by mosquitoes. The infective third larval stage develops in the mosquito; transmission to the definitive host occurs during feeding.

**Laboratory Diagnosis:** Detection of sheathed microfilariae in blood smears.

**Size:** Approximately 200–300  $\mu\text{m}$  in length.

**Clinical Importance:** *Setaria* has no clinical importance, with the exception of rare cases of abnormal migration of parasites in the nervous system.

## BLOOD PARASITES OF BIRDS

**PARASITE:** *Leucocytozoon* spp. (Figs. 3.30 and 3.31)

**TAXONOMY:** Protozoa (hemosporozoa).

**Geographic Distribution:** Important species include *L. simondi*, which is found worldwide in domestic and wild ducks and geese; *L. smithi* in North American and European domestic and wild turkeys; and *L. caudleyi* in chickens in Asia.

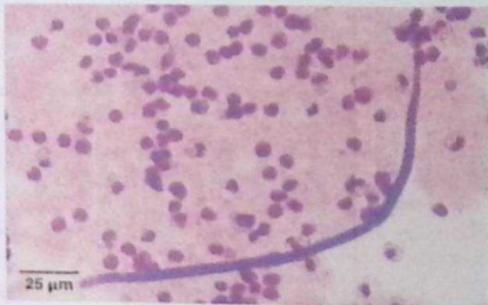
**Location in Host:** Gamonts (microgamonts and macrogamonts) occur in leukocytes and erythrocytes.

**Life Cycle:** Black flies (*Simulium* spp. and other simuliids) transmit infections to birds during blood feeding.

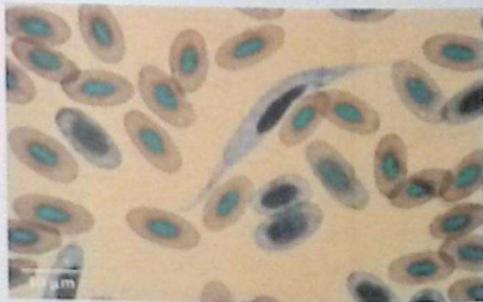
**Laboratory Diagnosis:** Gamonts are detected in white and red blood cells on Wright- or Giemsa-stained blood smears. Meronts are detected in stained tissue sections.

**Size:** Gamonts 14–22  $\mu\text{m}$

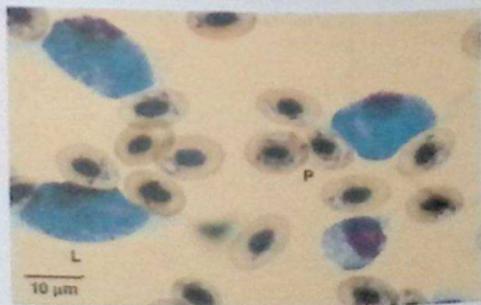
**Clinical Importance:** *Leucocytozoon* is pathogenic in ducks, geese, and turkeys, especially in younger birds. Clinical signs vary somewhat with species but can include lethargy, emaciation, and acute or chronic fatalities.



**FIG. 2.28** Microfilariae of *Setaria* spp. may occasionally be seen in ruminant or equine blood samples but have no clinical significance. A clear sheath can often be seen projecting beyond the end of the larva, although it is not evident in this specimen. Photo courtesy of Dr. Jeffrey F. Williams, Vansoo HaloSource, Inc., Redmond, WA.



**FIG. 2.29** Host leukocytes and erythrocytes containing the sausage-shaped *Leucocytozoon* gamonts appear elongated, with the remnants of the nucleus pushed to one side and the cytoplasm extending beyond the parasite and forming "horns." Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.



**FIG. 2.31** Birds may be infected with more than one species of protozoa. Both *Leucocytozoon* (L) and *Plasmodium* (P) are present in this hawk. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

**PARASITE:** *Haemoproteus* spp. (Fig. 3.32)

**Taxonomy:** Protozoa (hemosporozoa).

**Geographic Distribution:** Worldwide (except *H. meleagridis*, which occurs only in North America).

**Location in Host:** Gamonts (microgamonts and macrogamonts) occur in erythrocytes of pigeons and doves (*H. columbae*, *H. sacharovi*), wild and domestic turkeys (*H. meleagridis*), and wild and domestic ducks and geese (*H. nettionis*).

**Life Cycle:** Birds acquire infections from blood-feeding hippoboscid flies, midges (*Culicoides*), and deer flies (*Chrysops* spp.).

**Laboratory Diagnosis:** Gamonts are detected in red blood cells on Wright- or Giemsa-stained blood smears. The gamonts of *Haemoproteus* may vary in size and contain pigment granules. They appear morphologically identical to those of *Plasmodium* spp.

**Size:** Approximately 7  $\mu\text{m}$

**Clinical Importance:** Infections are usually subclinical.

**PARASITE:** *Plasmodium* spp. (Figs. 3.31 and 3.33)

**Common name:** Malaria.

**Taxonomy:** Protozoa (hemosporozoa).

**Geographic Distribution:** Worldwide.

**Location in Host:** Erythrocytes and various other tissues in a wide variety of birds.

**Life Cycle:** Mosquitoes transmit *Plasmodium* during feeding.

**Laboratory Diagnosis:** Gamonts, merozoites, and meronts are detected in red blood cells on Wright- or Giemsa-stained blood smears. Gamonts of *Plasmodium* appear morphologically identical to those of *Haemoproteus* spp.

**Size:** Gamonts 7–8  $\mu\text{m}$

**Clinical Importance:** Most species are nonpathogenic. Exceptions are *P. cathemerium* and *P. matutinum* in canaries; *P. gallinaceum* and *P. fuxtanucleare* in chickens; *P. hermani* in turkeys; and *P. relictum* in pigeons. Birds infected with these species may become anemic, with high fatality rates possible.



Fig. 32 Gamonts of *Haemoproteus* in a Swainson's hawk. The gamonts are often crescent-shaped and wrapped around the nucleus of the host erythrocyte. Pigment granules can be seen inside the gamont. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

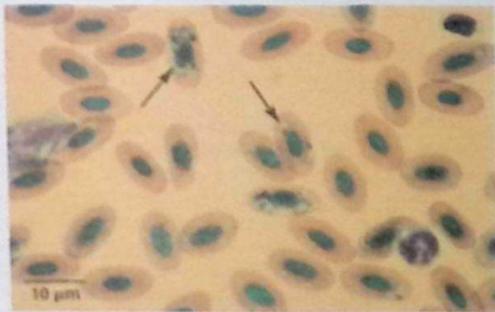


Fig. 33 The appearance of multiple stages of the parasite (signet-ring stage, meronts, and gamonts) in infected erythrocytes differentiates *Plasmodium* infection from *Haemoproteus* (in which only gamonts are found). In this sample from a cockatoo, several stages of the parasite are present (arrows). Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.



# Immunodiagnostic and Molecular Diagnostic Tests in Veterinary Parasitology

Karen F. Snowden

## IMMUNODIAGNOSTIC METHODS IN PARASITOLOGY

Immunodiagnostic methods for a range of parasitologic infections are selectively available at fee-for-service diagnostic laboratories and as point-of-care tests that can be conducted in a clinical setting.

There are two basic approaches in designing an immunologic test. **Antigen detection** tests identify specific parasite-associated compounds in blood, serum or fecal suspensions that indicate the presence of the organism in the host. Alternatively, **antibody detection** tests show the host immune response to a parasite through the production of specific antibodies. In order to have a positive test result, it is assumed that the host animal is immunologically competent to react to the pathogen and that a sufficient time of exposure has occurred for the animal to produce detectable antibodies.

There are a variety of test formats for immunodiagnostic tests. The enzyme-linked immunosorbent assay (ELISA) is designed with a series of wells in a plate or tray with an end result indicated colorimetrically (Fig. 4.1). The lateral flow immunochromatographic assay uses similar principles and reagents in a cassette format, and works by capillary action with a series of reagents moving along a membrane with the end result indicated as a colored dot or line on the membrane (Fig. 4.2). Both of these test formats can be designed as antigen or as antibody detection assays, and both test formats have been developed for use with blood, serum, or feces. Most ELISA tests are designed for processing sample batches in a diagnostic lab setting. One benefit of this test format is that the intensity of color generated in the reaction is measurable using a spectrophotometer and is generally proportional to the antigen/antibody that is being detected. Therefore the ELISA may be used as a quantitative measure in a carefully calibrated test. The immunochromatographic tests are designed to give

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard.

© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

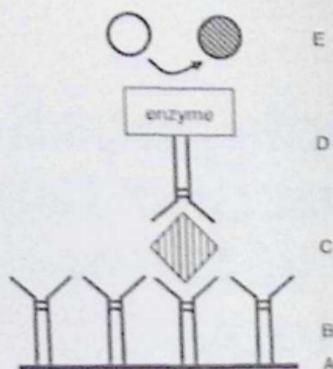


Fig. 4.1 Schematic ELISA antigen detection procedures: (A) test surface: polystyrene well; (B) parasite-specific capture antibody (may be monoclonal or polyclonal); (C) parasite antigen in serum of the animal patient; (D) detecting reagent: parasite-specific antibody labeled with an enzyme; (E) visualizing step: if enzyme is present, it acts on soluble substrate to produce color, which can be evaluated visually or measured spectrophotometrically.

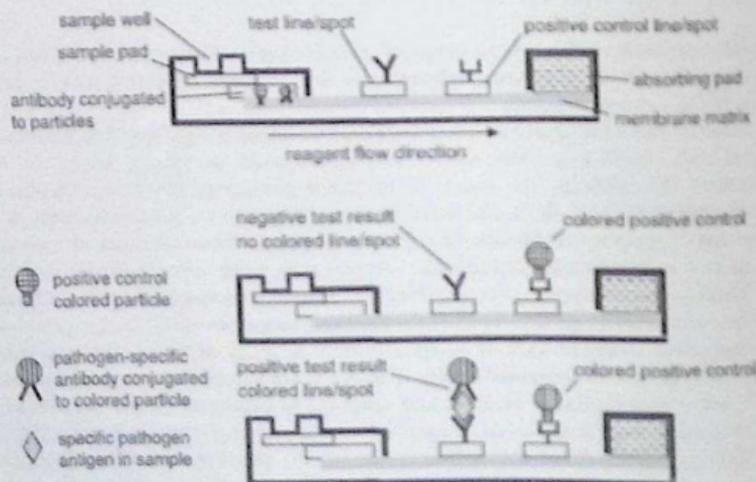
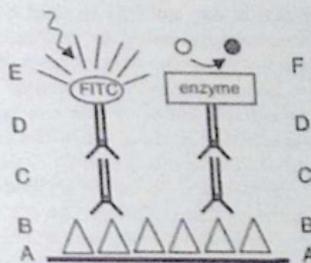


Fig. 4.2 Schematic of antigen detection using an immunochromatographic lateral flow test.

positive/negative results and are not generally designed to be quantitative; therefore the intensity of the colored dot/line is not necessarily proportional to the amount of antigen/antibody detected.

Immunochromatographic tests and selected ELISA assays are available as point-of-care tests that can be performed in a clinical setting on one or several samples in a relatively rapid time frame. Two of the most widely used parasitologic immunochromatographic or ELISA tests in companion animal medicine are the heartworm antigen test, which detects antigens primarily produced by adult female *Dirofilaria immitis* and



**Fig. 4.3** Comparison of common antibody detection procedures: indirect fluorescent antibody (IFA, left) test and enzyme-linked immunosorbent assay (ELISA, right). A, test surface: glass slide (IFA) or polystyrene well (ELISA); B, parasite antigen: whole parasite, such as cultured tachyzoites and promastigotes (IFA), or soluble parasite antigen, which can be a crude homogenate, a purified protein, or a recombinant protein (ELISA); C, serum of the animal patient, which may contain parasite-specific antibodies; D, detecting reagent for host-specific antibody: host-specific antibody labeled with a fluorochrome such as fluorescein (IFA) or host-specific antibody labeled with an enzyme (ELISA); E, visualizing step: specific UV wavelength from microscope causes fluorescein (FITC) to emit yellow-green fluorescence (IFA); F, visualizing step: if enzyme is present, it acts on soluble substrate to produce color, which can be evaluated visually or measured spectrophotometrically.

detected in blood, serum, or plasma, and the fecal antigen test for *Giardia duodenalis*. Since these types of assays are convenient, generally inexpensive, and relatively easy to perform in a clinical setting, it is likely that more point-of-care tests for additional parasites may become commercially available in the future.

Another commonly used format is the indirect immunofluorescent assay (IFA), which is an **antibody detection** test designed for use with serum or plasma (Fig. 4.3). These tests are routinely performed in the diagnostic lab setting because a compound microscope equipped with appropriate barrier filters and a UV light source is needed to conduct the test. The test result is typically expressed as a "titer" and the IgG antibody isotype is usually the immunoglobulin that is detected in the test. The titer value is the reciprocal of the highest dilution of serum/plasma where the test remains positive. Different test formats (ELISA vs. IFA most commonly) have different thresholds of antibody detection. Therefore, the positive/negative cutoff value for each standardized assay should be provided by the laboratory performing the test in order to adequately interpret the meaning of the antibody titer as positive/negative or high/low. For example, the cutoff value for an IFA test might be a 1 : 10 dilution, while a similar test in an ELISA format might have a 1 : 100 dilution cutoff value as positive.

Other less common immunodiagnostic test formats include direct or indirect hemagglutination (HA or IHA), complement-fixation tests (CF) and western blot tests for antibodies that react to selected parasites/pathogens. These tests are conducted at fee-for-service labs since reagents and equipment to conduct these assays are not routinely available in a clinical setting.

It is important to have an understanding of the life cycle and pathogenicity of a specific parasite in order to interpret antibody titers in a specific test. A helpful memory tool is the acronym, "PIE," which represents Protected, Infected, or Exposed. For some pathogens (usually viruses, but parasites in a few cases), having a high antibody titer is considered protective. For instance, if a cat has a significant antibody titer against

*Toxoplasma gondii*, that animal is very unlikely to shed oocysts, and it is a low-risk animal as a pet for an immunocompromised or pregnant owner. However, having antibodies against a parasite is not protective in most cases. With most parasites, having a positive titer may indicate current infection or previous exposure without current active infection. Having a measurable antibody titer can be of significant diagnostic value in confirming a parasite infection as the cause of clinical disease. For example, if a dog has chronic dilated cardiac disease with a titer to *Trypanosoma cruzi*, a probable diagnosis is Chagas disease. Alternatively, having an antibody titer may indicate exposure, but not necessarily an active parasite infection. An example of this situation is a domestic cat with an antibody titer to *Dirofilaria immitis*. The cat can produce detectable antibodies to the parasite after exposure to larvae from an infected mosquito bite, but those parasites do not necessarily develop successfully to adult worms causing a patent infection. In summary, interpretation of serologic results is diagnostically helpful in the context of understanding the role that antibodies play in the host-pathogen relationship for each parasite.

In a clinical setting, the veterinarian must interpret the diagnostic test results in the context of the clinical data available for an individual patient or on a group basis for a herd/flock. The diagnostic accuracy of immunodiagnostic tests has traditionally been framed in the context of **sensitivity** and **specificity**). Sensitivity is defined as the probability that an animal that has the infection/disease will test positive, and a sensitive test indicates few false negative results in an infected population (Table 4.1). Specificity is defined as the probability that an animal that does not have the disease will have a negative test result, and a specific test yields few false positive test results in an infection-free population. Typically, commercial point-of-care tests will make sensitivity and specificity data available for each type of test/kit, which provides overall information on the accuracy of the test. However, the use of these terms may have limited applications in a clinical setting when dealing with a problematic or confusing test result for an individual patient.

Other terms that may aid in test result interpretation are **positive and negative predictive values**. These values correspond to the probability that an individual truly is or is not infected, given a positive or negative test result (Table 4.1). These values differ from sensitivity and specificity since predictive values are dependent on the prevalence of infection/disease in the population of interest. The positive predictive values increase as the prevalence of infection increases in the tested population. Conversely, negative predictive values increase as the prevalence of infection decreases in the tested population.

**Table 4.1** Calculation of the diagnostic accuracy of a test (sensitivity, specificity, predictive values)

Test result	True infection status		Total
	Infection present	Infection absent	
positive	a	b	a + b
negative	c	d	c + d
total	a + c	b + d	a + b + c + d

$$\text{sensitivity} = a / (a + c)$$

$$\text{specificity} = d / (b + d)$$

$$\text{positive predictive value} = a / (a + b)$$

$$\text{negative predictive value} = d / (c + d)$$

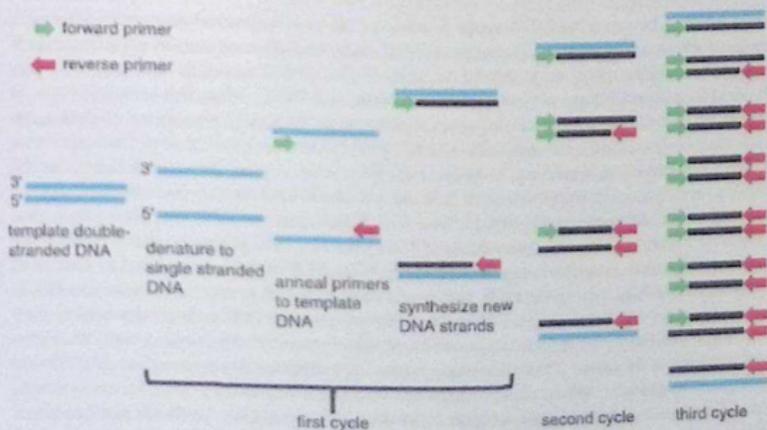
Source: Modified from table in Timoit et al., 2018.

## MOLECULAR DIAGNOSTIC METHODS IN PARASITOLOGY

The advent of molecular diagnostic methods has resulted in an expanding selection of sensitive and specific tests for a range of infectious organisms and genetic diseases. Experimental molecular diagnostic tests for parasitic infections are frequently reported in the scientific literature, and an increasing number of specific tests are commercially available to detect parasitic pathogens. Generally, molecular diagnostic tests are able to detect pathogen DNA at very low concentrations when compared with typical antigen detection immunodiagnostic tests. The high sensitivity of these molecular tests makes them especially attractive in cases of low parasite burden.

Molecular diagnostic assays detect DNA (or sometimes RNA) from a specific parasite/pathogen generally indicating active infection. Assays can be performed with many types of samples including blood, urine, tissues, feces, and other body fluids as well as environmental samples. Regardless of the sample matrix, the first step in a protocol is to isolate the nucleic acid for testing. The polymerase chain reaction (PCR) technique is a widely used molecular technique that amplifies and detects a specific short piece of target nucleic acid (such as from a parasite) (Fig. 4.4). In a well-validated assay, the test may be considered positive if the appropriately sized DNA amplicon is visualized by gel electrophoresis. In other cases, in order to confirm the identity of the parasite/pathogen, the amplified DNA fragment must be further analyzed by nucleic acid sequencing for comparison with previously determined target gene sequences from a known pathogen.

Currently, the most popular version for molecular diagnostic tests is the quantitative PCR (qPCR, sometimes called "real-time" or rtPCR), which is a rapid test that is sensitive and reliable for the detection of molecular targets. The technique is quantitative



**Fig. 4.4** Schematic of a conventional PCR assay. PCR is a process used to selectively amplify a targeted section of double-stranded DNA in a sequence of repeated steps. Double-stranded template DNA is first heated to denature to single-stranded DNA, and then cooled so that pathogen-specific short DNA primers can anneal to their complementary targets on the template DNA. By adding dNTP nucleotides and a DNA polymerase enzyme, new strands of DNA are synthesized. By repeating the cycles of heating and cooling the target DNA segment is amplified in an exponential manner.

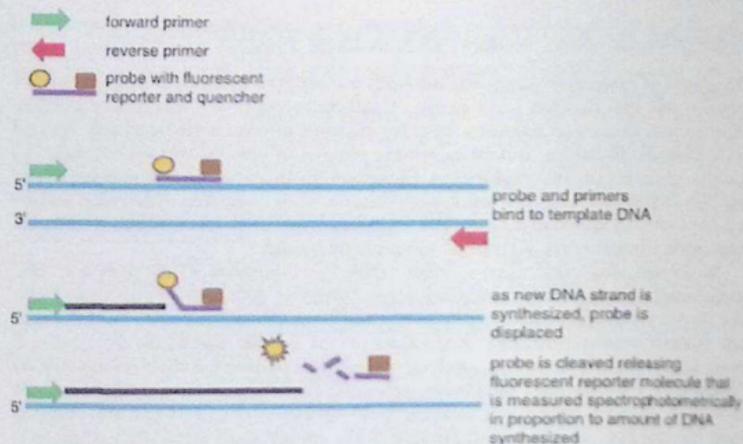


Fig. 4.5 Simplified illustration of how a qPCR assay differs from a conventional PCR assay. The qPCR uses primers to amplify a targeted section of template DNA similar to conventional PCR (see Fig. 4.4) but it also adds a sequence-specific DNA probe with a fluorescent reporter molecule and a quencher molecule that bind to the single-stranded DNA template. As the new strand of DNA is synthesized, the probe is cleaved releasing the fluorescent reporter molecule. The fluorescence intensity is measured spectrophotometrically at each cycle indicating the amount of DNA synthesized. Since a larger amount of template DNA in the starting sample will result in greater fluorescence in fewer cycles, the method is called "real-time" PCR or "quantitative" PCR.

and real-time because the DNA amplification product is measured repeatedly after each cycle of DNA amplification through the detection and quantification of a fluorescent reporter molecule (Fig. 4.5). An advantage of the qPCR assay is that it can be performed in a shorter time period than a conventional PCR. Also, the technique can be designed to simultaneously detect several pathogens by using a mixture of pathogen-specific primers along with sequence-specific probes that are labeled with fluorochromes of various detection wavelengths (Fig. 4.6). The accuracy of the test depends on the quality and quantity of DNA or RNA extracted as well as the presence/absence of inhibitors that can negatively impact the assay. A detractor to PCR methodology is that very small samples of a few microliters of fluid or a few milligrams of tissue or feces are processed for this assay, so it is possible that parasite DNA is not included in the tested sample. Therefore, a positive PCR test result indicates the presence of parasite DNA, while a negative test does not rule out possible infection with the target pathogen.

A parasitologic molecular diagnostic test widely used at this time detects the venereal protozoan of cattle, *Tritrichomonas foetus*, in preputial swabs or specialized short-term *in vitro* cultures. Since this parasite has become a regulatory issue in many states, the validation and comparison of molecular and *in vitro* culture methods has become a topic of interest to many diagnostic laboratories, and to veterinary practitioners and their clients as well. New test offerings, particularly in companion animal medicine, are also becoming available from an expanding number of commercial labs. Illustrating the potential for multi-pathogen molecular tests, canine vector-borne disease agent panels

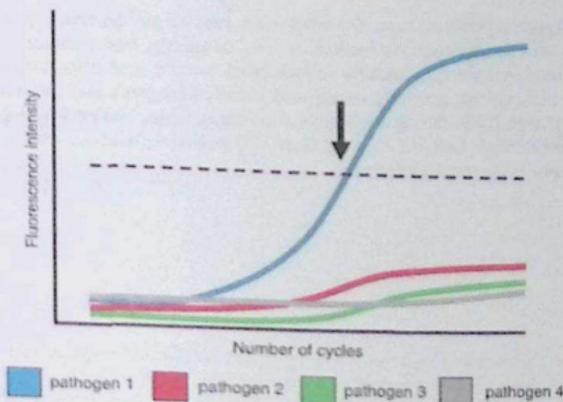


Fig. 4.5 Simple schematic of fluorescence plot of results from a multiplex qPCR assay that potentially detects four pathogens in a single test. For each pathogen, specific primers and probes labeled with fluorescent reporter molecules that emit light at different wavelengths are included in a single assay (see Fig. 4.5). If pathogen template DNA is present in the test sample, then an increasing amount of fluorescence at a specific wavelength is detected as the number of heating/cooling cycles increases. Based on previous standardization of the test with known pathogen DNA, a sample (blue pathogen 1) is considered positive if the fluorescence intensity reaches a minimum threshold at the completion of a pre-determined number of cycles (arrow). If no pathogen template DNA is included in the test sample, then no new DNA is synthesized and no fluorescence is produced for those pathogens, and the test is considered negative (pathogens 2, 3, and 4).

are offered by a number of commercial and university-associated diagnostic laboratories on a fee-for-service basis. These panels may include *Ehrlichia* spp., *Anaplasma* spp., *Rickettsia* spp., *Trypanosoma cruzi*, *Leishmania* spp., and sometimes hemoprotozoan parasites such as *Babesia canis*, *B. gibsoni*, *Cytauxzoon felis*, or *Hepatozoon* spp. Similarly, diarrheal disease molecular panels for a variety of animal hosts that are readily available commercially sometimes include *Giardia duodenalis* or *Cryptosporidium* spp. In the future, it is likely that molecular tests for additional parasites will be available as individual assays or as part of clinically relevant panel screens from commercial laboratories based on clinical demands and the need to develop better diagnostic techniques for problematic parasitic pathogens. At this time, molecular diagnostic tests are conducted in fee-for-service labs or research labs due to the expense of the necessary equipment and the technical expertise used in conducting the tests. However, it is likely that in the foreseeable future, simplified benchtop PCR equipment and pathogen-specific reagent kits will be available for diagnostic use in a clinical setting.

As with any diagnostic testing, false positive or false negative results may occur for any individual immunodiagnostic or molecular diagnostic test. To minimize the likelihood of errors and to ensure accurate results it is important that all involved personnel are well trained and are monitored for consistent performance in following procedures and/or performing assays. When sending out samples for testing remember to: (1) collect samples in appropriate, accurately labeled containers, (2) store/ship/submit samples at the correct temperature using packaging containment appropriate for biological samples, (3) include appropriate submission forms with adequate clinical

history and patient information, (4) when test results are received, promptly enter information into the appropriate medical record. Similarly, best practices for point-of-care tests should include: (1) carefully collect, label, handle, and store samples correctly, (2) only use nonexpired, properly stored and handled reagents and kit/assay contents, (3) follow a standard written protocol for collecting samples and performing each assay according to the manufacturer's instructions, (4) promptly and accurately record test results in appropriate medical records.

# Diagnosis of Arthropod Parasites

The phylum Arthropoda contains many parasitic species, including ticks and mites (class Arachnida, subclass Acari) and insects (class Insecta). Crustaceans that parasitize aquatic animals also belong to this phylum (see Chapter 6). Arthropods are characterized by jointed appendages in the adult, and sometimes immature, stages as well as the presence of exoskeletons. This chapter presents a selection of arthropods that are common or important in domestic animals; we are grateful to Dr. Ellis Greiner, University of Florida (retired), who provided the original text.

## SUBCLASS ACARI (MITES AND TICKS)

Mites and ticks are divided into two parts: the gnathosoma, which bears the mouthparts (pedipalps, chelicerae), and the idiosoma, where the jointed appendages (legs) and reproductive structures are found. Upon larval emergence from the egg, mites and ticks develop through simple metamorphosis to subsequent nymphal and adult stages. Larvae of ticks and mites have six legs; nymphs and adults have eight legs. Nymphal ticks and mites usually closely resemble the adults but are smaller and lack a genital opening.

### Mite Identification

The majority of parasitic mites are microscopic, rarely exceeding 1 mm in length. Female mites are usually larger than males. All stages are covered by a soft integument. Respiration may occur directly across the integument (astigmata) or through spiracular openings (stigmata) associated with tracheal ducts; stigmata are often used in identification. Scales, spines, or setae (hairs) on the body and claws or suckers on the legs (Fig. 5.1) are also used in identifying the organisms. Many common sarcoptiform (round-bodied) mites resemble one another, but they can be differentiated on the basis of the characteristics outlined in Table 5.1.



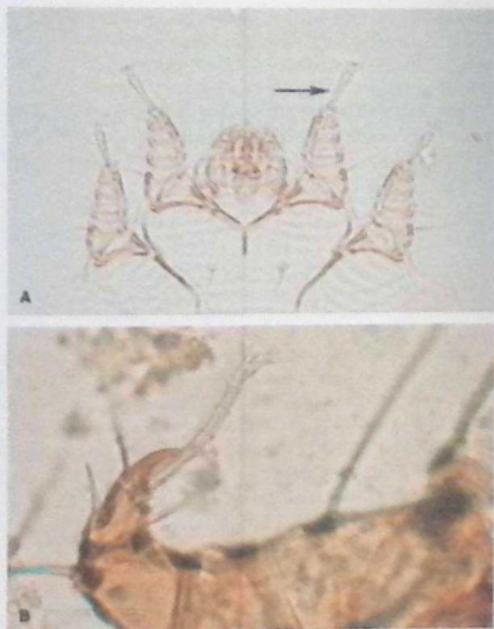


Fig. 5.1 Important characteristics for identification of a number of common mites are length and segmentation of the stalk (pedicle) connecting a terminal sucker to the leg. In *Sarcoptes scabiei* (A) the stalk is long and unjointed (arrow) whereas *Psoroptes* spp. mites have a long, jointed pedicle (B).

Table 5.1 Microscopic characteristics of some mites important in veterinary medicine

Genus	Egg-laying female	Leg characteristics		Anus
		Male		
<i>Sarcoptes</i>	Suckers on long, unsegmented stalks on legs 1, 2; many pointed scales on dorsum	Suckers on long unsegmented stalks on legs 1, 2, 4; few pointed scales on dorsum		Terminal
<i>Notoedres</i>	Suckers as above; many prominent rounded scales on dorsum	Suckers as above; few rounded scales on dorsum		Dorsal
<i>Knoemidokoptes</i>	No suckers	Suckers on unsegmented stalks on legs 1, 2, 3, 4		Terminal
<i>Psoroptes</i>	Suckers on long, segmented stalks on legs 1, 2, 4	Suckers on long, segmented stalks on legs 1, 2, 3		Terminal
<i>Chorioptes</i>	Suckers on short, unsegmented stalks on legs 1, 2, 4	Suckers on short, unsegmented stalks on legs 1, 2, 3, 4; legs 4 rudimentary		Terminal
<i>Otodectes</i>	Suckers on short, unsegmented stalks on legs 1, 2; legs 4 rudimentary	Suckers on short, unsegmented stalks on legs 1, 2, 3, 4		Terminal

Most common mite infestations are diagnosed by deep or superficial skin scrapings. For a deep skin scraping, a dulled, rounded scalpel blade (#10) is coated with mineral oil. The site selected for scraping should be at the periphery of a lesion or the predilection site of the suspected parasite. The blade should be scraped back and forth over the skin until capillary bleeding is evident (a shallower scraping can be done for surface-dwelling mites). For collection of *Demodex*, the follicle mite, a fold of skin should be gently compressed between the fingers to express the mites before scraping. The debris collected on the scalpel blade is then placed on a microscope slide, a coverslip applied, and the material examined using the 10 $\times$  microscope objective. Several slides may need to be examined before mites are found, especially in cases of *Sarcoptes* infestation.

To recover surface mites, such as *Cheyletiella* spp., a superficial scraping that does not cause bleeding is made with a scalpel blade coated in mineral oil. Alternatively, scurf can be combed from animals and examined directly, or clear tape can be used to collect material. The tape is pressed to the hair coat in an affected area and then placed on a microscope slide, trapping skin debris and mites against the slide and allowing microscopic examination.

If mites are shipped to a veterinary diagnostic laboratory for identification, they should be stored in 70% alcohol. Storing skin scrapings and mites dry may prevent successful identification.

**PARASITE:** *Sarcoptes scabiei* (Figs. 5.2–5.5)

Common name: Itch mite or scabies mite.

**TAXONOMY:** Mite (family Sarcoptidae).

**HOST:** Host-specific varieties of *Sarcoptes scabiei* are found on a wide range of hosts including dogs, pigs, humans, ruminants, horses, rodents, and camelids. Traditionally, each mite is referred to according to the host on which it was found (*S. scabiei* var *canis*, *S. scabiei* var *suis*, *S. scabiei* var *hominis*, etc.).

**Geographic Distribution:** Worldwide.

**Location on Host:** On dogs, the margins of the ear, lateral elbows, and lateral hocks are most commonly affected. In pigs, the ears, neck, and back are infested. Over time, large portions of the skin can be involved.

**Life Cycle:** Transmission of *Sarcoptes scabiei* occurs following direct contact with an infested animal or fomites (e.g., clippers). Mites burrow deep into the epidermis, depositing eggs and feces. The life cycle from egg to adult requires approximately 3 weeks to complete.

**Laboratory Diagnosis:** To confirm the diagnosis, examine multiple, deep skin scrapings from the margins of affected areas. When mites are not detected, patients may be treated presumptively and the diagnosis confirmed upon resolution of pruritus. Fecal examination is also useful in revealing mites and mite eggs ingested during grooming. In pigs, *S. scabiei* is found in hyperkeratotic crusts on the margins of the pinnae. On the farm, this crusted material can be removed and crumbled over dark paper to reveal the tiny, motile mites. Digesting crusts with 10% sodium hydroxide may also reveal mites.

**Size:** Females approximately 400  $\mu\text{m}$ ; males approximately 250  $\mu\text{m}$

**Clinical Importance:** Mange caused by *Sarcoptes scabiei* is extremely pruritic and often accompanied by alopecia, hyperkeratosis, and dermal thickening. In North America, infestations occur most commonly in dogs and pigs; mites from animals readily transfer to humans and cause self-limiting pruritus.



**Fig. 5.2** Pruritic mite infestations may stimulate intense grooming by the host, resulting in the presence of both mites and eggs (not shown) in the feces.

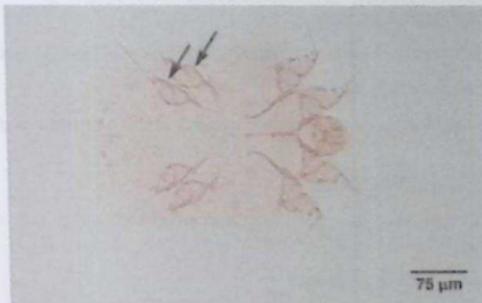


Fig. 5.3 *Sarcoptes* and related mites are typically round bodied. The third and fourth pairs of legs (arrows) are short and often do not project beyond the margin of the body. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 5.4 *Sarcoptes scabiei* var. *canis* causes "scabies" or "sarcoptic mange" in dogs. Lesions commonly occur on the face and along the margin of the ear as well as on the lateral elbows and hocks of infested dogs. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 5.5 Sarcoptic mange in an alpaca. In chronic sarcoptic mange, affected skin is hairless, thickened, and wrinkled. These nonspecific changes also occur in other chronic skin diseases. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**PARASITE:** *Notoedres* spp. (Fig. 5.6)

Common name: Feline mange mite, Ear mange mite (rodents).

**Taxonomy:** Mite (family Sarcoptidae).

**Host:** *Notoedres cati* occurs on cats. Other species occur on bats, rodents, and other small mammals.

**Geographic Distribution:** Worldwide.

**Location on Host:** The head is usually infested first, but mites may spread to other regions of the body.

**Life Cycle:** Similar to *Sarcoptes scabiei*.

**Laboratory Diagnosis:** Mites are observed in deep skin scrapings.

**Size:** *Notoedres cati* approximately 200–225  $\mu\text{m}$

**Clinical Importance:** Feline notoedric mange is usually confined to the head and neck. Infestations are rarely seen in North America.

**PARASITE:** *Knemidokoptes* spp. (Figs. 5.7 and 5.8)

Common name: Scaly leg or scaly face mite.

**Taxonomy:** Mite (family Knemidokoptidae).

**Host:** Birds, including domestic poultry and pet birds.

**Geographic Distribution:** Worldwide.

**Location on Host:** Nonfeathered portions of the body, including feet, legs, and face.

**Life Cycle:** Like *Sarcoptes scabiei*, transmission occurs by direct contact with infested birds or fomites, and all stages of the mite are found on the host.

**Laboratory Diagnosis:** Mites can be found in skin scrapings collected from the periphery of lesions. Typically, the exudative lesions produced by the mites contain numerous small holes, giving them a honeycombed appearance.

**Size:** Approximately 400  $\mu\text{m}$

**Clinical Importance:** *Knemidokoptes* species burrow under the scales on the legs or nonfeathered portions of the face, inducing a serous exudate that hardens into crusts. These proliferative lesions eventually may cause trauma and disfigurement leading to the death of the host.

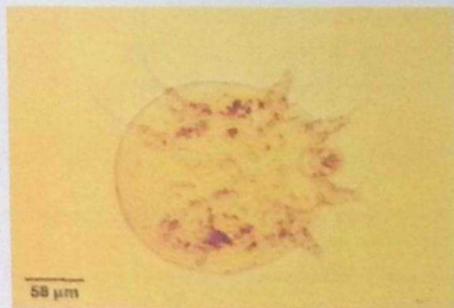


Fig. 5.6 *Notoedres* mites are similar in appearance to *Sarcoptes*. However, the anus of *Notoedres* is located on the dorsal surface rather than the ventral. *Notoedres* also has scalloped scales on the dorsum rather than the sawtooth scales on *Sarcoptes*. The suckers on the front legs of both *Notoedres* and *Sarcoptes* are attached to the legs by long, unjointed stalks.



Fig. 5.7 *Knemidokoptes* is a round-bodied mite, generally similar in appearance to sarcoptiform mites.



Fig. 5.8 Budgerigar with a deformed beak resulting from the proliferative lesion produced by *Knemidokoptes* infestation. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**PARASITE:** *Trixacarus* spp. (Fig. 5.9)

**TAXONOMY:** Mite (family Sarcoptidae).

**Host:** Guinea pigs (*Trixacarus caviae*) and rats (*T. diversus*).

**Geographic Distribution:** Europe and North America.

**Location on Host:** Lesions begin on the head, neck, and back but can spread to other areas.

**Life Cycle:** Similar to *Sarcoptes scabiei*. Mites are readily transferred from the dam to young animals in the neonatal period.

**Laboratory Diagnosis:** Mites are identified in skin scrapings.

**Size:** Approximately 200  $\mu$ m

**Clinical Importance:** *Trixacarus* is the sarcoptic mange mite of guinea pigs. Infestation is associated with pruritus, alopecia, and hyperkeratosis and can become a serious problem in guinea pig colonies. Humans in contact with infested guinea pigs may develop transient lesions.

**PARASITE:** *Chorioptes bovis* (Figs. 5.10 and 5.11)

Common name: Foot mange, leg mange, itchy heel.

**TAXONOMY:** Mite (family Psoroptidae).

**Host:** Varieties of *C. bovis* are found on ruminants, horses, and rabbits.

**Geographic Distribution:** Worldwide.

**Location on Host:** *Chorioptes* are found primarily on the lower body of the host. In horses, the mites are seen more often in breeds with feathered legs. In cattle, the rear legs, base of the tail, and back of the udder are most often affected.

**Life Cycle:** Transmission is by direct contact or fomites. *Chorioptes* mites spend their entire life cycle on the skin surface. The life cycle can be completed in about 3 weeks.

**Laboratory Diagnosis:** Mites are observed in skin scrapings. *Chorioptes* has short, unsegmented stalks bearing the suckers on the legs (Table 5.1).

**Size:** Approximately 400  $\mu$ m

**Clinical Importance:** Infestation may be asymptomatic or cause only mild lesions in some animals. As mite populations increase, pruritus, alopecia, and crusting may develop.

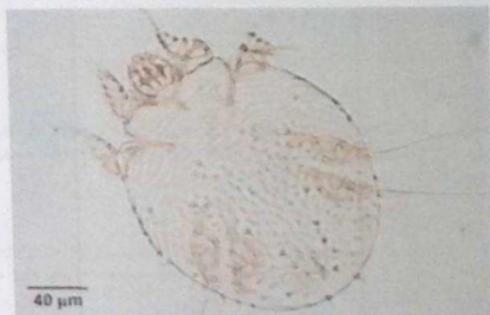


Fig. 5.9 Like other sarcoptiform mites, *Trixacarus* is a round-bodied mite with short legs. *Trixacarus curvii* causes mange in guinea pigs.



Fig. 5.10 *Chorioptes* mites are more elongated, with longer legs than the sarcoptiform mites. Suckers are evident on short, unsegmented stalks (arrows) at the end of the legs. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 5.11 Female *Chorioptes* mites with eggs present (arrows). Mite eggs are large, ~200 μm long, and may be seen retained within mites or found on fecal flotation from infested animals. Photo courtesy of Dr. Yoko Nagamori, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Psoroptes* spp. (Figs. 5.12–5.15)

Common name: Scab mite (ruminants).

**Taxonomy:** Mite (family Psoroptidae).

**Host:** *Psoroptes ovis* is the cause of psoroptic mange in ruminants; *P. ovis cuniculi* (formerly referred to as *P. cuniculi*) is found on rabbits and ruminants. *Psoroptes* spp. can also be found on horses and some wildlife hosts.

**Geographic Distribution:** Worldwide.

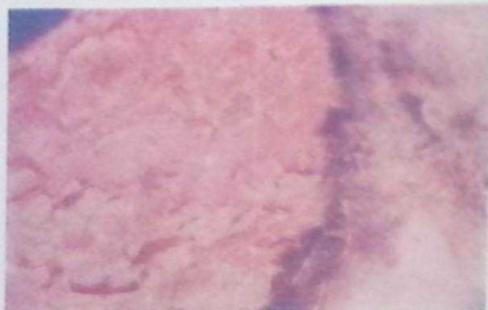
**Location on Host:** *Psoroptes ovis cuniculi* is found in the ears of rabbits, sheep, goats, and horses. Other *Psoroptes* infestations are often first detected on the dorsum of the host but may spread to other areas.

**Life Cycle:** Transmission is by direct contact or fomites. Unlike sarcoptiform mites, *Psoroptes* spp. do not burrow, and all stages are found on the skin surface. The life cycle can be completed in as little as 10 days. Mites may survive for several days off the host.

**Laboratory Diagnosis:** Superficial skin scrapings should be collected from the periphery of skin lesions. Alternatively, skin scabs can be broken apart or digested and the residue examined microscopically. Crusts from the ears can be treated similarly when infestations of *P. ovis cuniculi* are suspected. Psoroptic mites are more oval in shape and have longer legs than sarcoptiform mites.

**Size:** Approximately 750  $\mu\text{m}$

**Clinical Importance:** *Psoroptes* is a highly contagious, economically important cause of skin disease in ruminants worldwide. Infestation leads to exudative dermatitis and hair loss. In the United States, the strain of *P. ovis* affecting sheep has been eradicated, and the bovine strain has diminished in importance since the introduction of macrocyclic lactone endectocide drugs. In severe cases, *P. ovis cuniculi* lesions on rabbits may extend beyond the ears to the face, neck, and back.



**Fig. 5.12** Psoroptic mange or "scab" can be a serious infestation in ruminants. In sheep, mite activity causes an exudate that forms a crust on the surface of the skin, with the resulting loss of the fleece over affected areas.



Fig. 5.13 Psoroptic ear mange in a rabbit. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

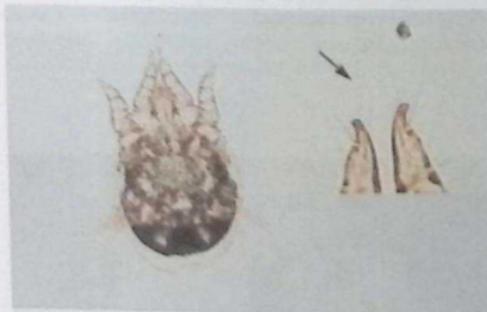


Fig. 5.14 *Psoroptes* sp. mites have a more oval shape and longer legs than round-bodied, sarcoptiform mites. This morphology is sometimes referred to as "psoropiform." Terminal suckers are connected to the legs by long, segmented stalks (arrow). Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 5.15 *Psoroptes* may be up to 800 μm in length. Shown here are specimens of *Psoroptes cuniculi* from rabbit ears. Figures 5.1B and 5.14 show a closer view of the jointed pedicle on some of the legs of *Psoroptes*. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

**PARASITE:** *Otodectes cynotis* (Figs. 5.16–5.19)

Common name: Ear mite.

**Taxonomy:** Mite (family Psoroptidae).

**Host:** Dogs, cats, and ferrets.

**Geographic Distribution:** Worldwide.

**Location on Host:** Ear canal.

**Life Cycle:** Mites complete their life cycle in the ear. Transmission occurs by direct contact or fomites. Kittens and puppies are easily infested by contact with the dam.

**Laboratory Diagnosis:** Routinely diagnosed by otoscope or microscopic examination of aural exudate collected with cotton swabs.

**Size:** Approximately 300  $\mu$ m

**Clinical Importance:** These mites are a common cause of otitis externa. Bacterial decomposition of otic secretions and exudate leads to the formation of black, waxy cerumen. Infested animals often suffer severe pruritus that may lead to self-inflicted trauma. Heavy infestations may spread outside the ear to the face, neck, and back.



Fig. 5.16 *Otodectes cynotis* infestation in a cat. The mites cause the production of black, waxy exudate in the ear canal. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



**Fig. 5.17** Mating *Otodectes cynotis* mites from a ferret. *Otodectes* is another psoroptiform mite with an oval-shaped body and long legs. They are similar in size and appearance to *Chorioptes*. In heavy infestations, it is common to find copulating mites in ear swab preparations. The short unsegmented stalks carrying the suckers can be seen in this photo.



**Fig. 5.18** Gravid female of *Otodectes cynotis* with egg present (arrow). Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



**Fig. 5.19** Male *Otodectes cynotis*. The pair of distinct circular structures evident on the posterior end (arrows) are copulatory suckers. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE:** *Demodex* spp. (Figs. 5.20–5.23)

Common name: Follicle mite, red mange.

**TAXONOMY:** Mite (family Demodicidae).

**Host:** Species of *Demodex* are host-specific and have been identified from many animals, including dogs, cats, pigs, horses, cattle, goats, sheep, laboratory animals, and humans.

**Geographic Distribution:** Worldwide.

**Location on Host:** Sebaceous glands and hair follicles.

**Life Cycle:** Mites are usually transferred from the dam to offspring in the neonatal period. All stages of the life cycle are found on the host.

**Laboratory Diagnosis:** Deep skin scrapings are required for diagnosis. Compressing a skin fold before scraping aids in expressing mites from follicles and sebaceous glands. A high proportion of eggs and immature mites on skin scraping is considered indicative of a more severe infestation. *Demodex* mites and eggs are often ingested during grooming and identified by fecal flotation.

Size: 100–400  $\mu\text{m}$ , depending on species

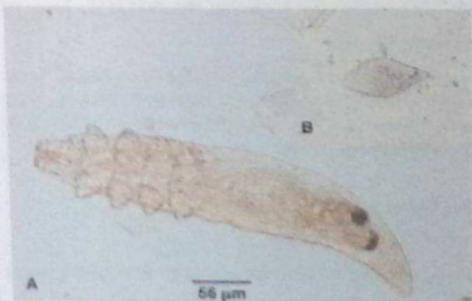
**Clinical Importance:** Most animals harbor mites but do not develop clinical disease. When the immune system fails to keep mite populations in check, proliferation can occur leading to folliculitis, furunculosis, and secondary bacterial infection. Disease is seen most often in dogs and may be localized and self-limiting or generalized, severe, and potentially fatal.



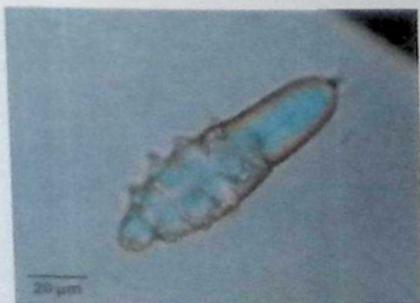
**FIG. 5.20.** *Demodex* is most often seen as a clinical problem in dogs. Lesions often appear first on the face or forelegs. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



**Fig. 5.21** In goats and cattle, clinical demodectosis is usually associated with the formation of nodular pustules. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA, and Dr. C. Williams, Langley, WA.



**Fig. 5.22** *Demodex* spp. mites (A) have a distinct, elongated appearance and are often described as looking like cigars with legs. *Demodex coris* reaches a length of ~390  $\mu\text{m}$ . Eggs (B) are spindle-shaped (~100  $\mu\text{m}$ ). Both may be present on skin scrape or found on fecal flotation from infested animals after self-grooming. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



**Fig. 5.23** Many animals are parasitized by *Demodex* spp. Shown here is *Demodex* from a gerbil. Hosts rarely show clinical signs of infestation. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

**PARASITE:** *Cheyletiella* spp. (Figs. 5.24 and 5.25)

Common name: Walking dandruff.

**Taxonomy:** Mite (family Cheyletiellidae).

**Host:** *Cheyletiella parasitovorax*, *C. yasguri*, and *C. blakei* are seen on rabbits, dogs, and cats, respectively.

**Geographic Distribution:** Worldwide.

**Location on Host:** *Cheyletiella* infestations are usually seen on the back. In cats, the head is also often affected.

**Life Cycle:** Transmission is by direct contact or fomites. *Cheyletiella* can be carried from one animal to another by fleas (*Ctenocephalides*). Mites can live up to 10 days in the environment.

**Laboratory Diagnosis:** *Cheyletiella* is a fur mite and not a skin dweller, so only superficial skin scrapings are required for diagnosis. Alternatively, if material combed from the hair is examined against a dark background, mites can be seen as moving white dots ("walking dandruff"). The distinctive feature of the mite is the large palpal claws.

**Size:** Approximately 400  $\mu\text{m}$

**Clinical Importance:** Many infested animals do not show clinical signs. Young animals are most likely to show evidence of infestation, including crusting, increased skin scurf, and pruritus. In heavy infestations, hair loss may occur. Owners may develop lesions in areas of close contact with their animals.

**PARASITE:** *Psorobia* (*Psoregates*) spp. (Fig. 5.26)

**Taxonomy:** Mite (family Cheyletiellidae).

**Host:** Mice (*Psorobia simplex*); related species (e.g. *Psoregates* spp.) are found on ruminants.

**Geographic Distribution:** *Psorobia simplex* is found on mice worldwide. *Psoregates ovis* of sheep is uncommon and occurs in Australia, New Zealand, South Africa, and South America. *Psoregates bos* of cattle is also uncommon but has been reported from Africa, Australia, Europe, and North America.

**Location on Host:** *Psorobia simplex* can be found anywhere on the body although lesions often develop in the ears and mite numbers are highest on the head and neck. Lesions of *Psoregates ovis* develop on the neck and shoulders and then spread to the flanks and rump. Cattle with *Psoregates bos* develop lesions on the dorsal head, neck, shoulders, rump, and back.

**Life Cycle:** Similar to *Sarcoptes scabiei*. Mites readily move between animals in close contact.

**Laboratory Diagnosis:** Mites are identified in skin scrapings.

**Size:** 100–200  $\mu\text{m}$ ; smaller than *Sarcoptes scabiei*

**Clinical Importance:** *Psorobia simplex* can cause ear mange as well as small, white, dermal nodules in mice. *Psoregates ovis* causes fleece damage in sheep; Merinos are particularly susceptible. Cattle with psorergatic mange present with pruritus, scaling, and alopecia.



Fig. 5.24 *Cheyletiella* is a surface mite that can be collected by brushing the hair coat or collecting material with sticky tape. In heavy infestations, mites may be found throughout the hair coat. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 5.25 *Cheyletiella* spp. are readily identified microscopically by the presence of large palpal claws (*arrows*). Small combs are present on the legs instead of the suckers seen in some other species of parasitic mites. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



Fig. 5.26 *Psorobia simplex* from a mouse. Note the rounded body, short legs, and presence of small claws or combs rather than suckers on the end of each leg. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Lynxacarus radovskyi* (Fig. 5.27)

**Taxonomy:** Mite (family Listrophoridae).

**Host:** Cats; other species infest bobcats and weasels.

**Geographic Distribution:** Australia, southern United States, Caribbean, and Hawaii.

**Location on Host:** Mites clasp the hairs of cats, primarily on the tail head, tail tip, and in the perineal area.

**Life Cycle:** The entire life cycle is spent on the host. Infestation is by direct contact.

**Laboratory Diagnosis:** Laterally flattened mites can be seen clinging to cat hairs. Eggs are attached to the hairs.

**Size:** Approximately 450  $\mu\text{m}$

**Clinical Importance:** Heavy mite infestations can affect the entire body and lead to poor condition of the hair coat. This mite is rare in North America.

**PARASITE:** *Leporacarus* (= *Listrophorus*) *gibbus* (Figs. 5.28 and 5.29)

**Common name:** Fur mite.

**Taxonomy:** Mite (family Listrophoridae). This mite formerly belonged to the genus *Listrophorus*.

**Host:** Rabbits.

**Geographic Distribution:** Worldwide.

**Location on Host:** Throughout the fur.

**Life Cycle:** Transmission is by direct contact. All stages of the life cycle are found on the host.

**Laboratory Diagnosis:** Mites are large enough to be seen as small specks on the hairs and can be collected by combing and examining hairs with a magnifying glass or microscope.

**Size:** Approximately 350–500  $\mu\text{m}$

**Clinical Importance:** Mites usually cause no clinical signs even though large numbers may be present.



Fig. 5.27 The body of the *Lymniscus* mite is laterally compressed like that of a flea and has large sternal plates that are used, along with the first two pairs of legs, to encircle the hair.



Fig. 5.28 Male *Leporacarus* mites have a brown anterior shield that projects beyond the mouthparts. Males also have distinctive adanal clasping organs.



Fig. 5.29 Adult *Leporacarus* on the hair of a rabbit. The female mites also bear an anterior shield. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

PARASITE: *Chirodiscoides caviae* (Fig. 5.30)

Common name: Fur mite.

Taxonomy: Mite (family Listrophoridae).

Host: Guinea pigs.

Geographic Distribution: Worldwide.

Location on Host: Attached to hairs.

Life Cycle: Transmission is by direct contact with an infested individual or fomite.

Laboratory Diagnosis: Mites are detected by examining hairs from the host.

Size: Approximately 350–500  $\mu\text{m}$

Clinical Importance: This mite is considered generally nonpathogenic.

PARASITE: *Mycoptes musculinus*, *Myobia musculi*, *Radfordia* spp. (Figs. 5.31 and 5.32)

Common name: Fur mite.

Taxonomy: Mites (families Listrophoridae and Myobidae).

Host: Mice and rats.

Geographic Distribution: Worldwide.

Location on Host: Hair coat.

Life Cycle: Transmission is by direct contact; all stages of the life cycle are found on the host.

Laboratory Diagnosis: Diagnosis is made by detecting mites on host hairs.

Size:	<i>Radfordia</i> and <i>Myobia</i>	approximately 400–450 $\mu\text{m}$
	<i>Mycoptes</i>	approximately 350 $\mu\text{m}$

Clinical Importance: Some infested animals tolerate large numbers of mites without clinical signs, although pruritus, erythema, hair loss, and thickened skin may occur in others. Secondary bacterial infections may develop.



Fig. 5.30 *Chirodiscoides* from a guinea pig. The first two pairs of legs are adapted for wrapping around the hair shafts of the host. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

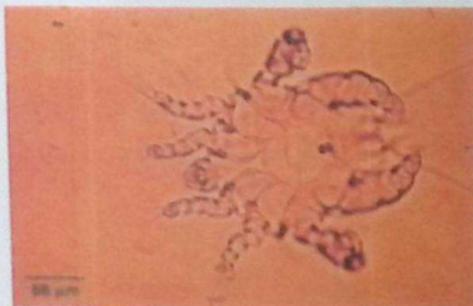


Fig. 5.31 *Myocryptes muscardinus* from the hair coat of a mouse. In males, the fourth pair of legs is enlarged and directed backward. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.



Fig. 5.32 *Radfordia* is found at the base of the hairs. The first pair of legs is modified for feeding and project forward. *Radfordia* is similar in appearance to another rodent fur mite, *Myobia musculi*. However, *Radfordia* has two claws on the second pair of legs, while *Myobia* has only one claw.

**PARASITE:** Avian Feather Mites (Figs. 5.33 and 5.34)

**Taxonomy:** Mites (numerous families and species).

**Host:** Domestic and wild birds.

**Geographic Distribution:** Worldwide.

**Location on Host:** Species specialized for different feather environments.

**Life Cycle:** Most mites live on the feather surface and feed on secretions and skin and feather debris. Quill mites live in the base of the feathers and feed on host tissue or fluids.

**Laboratory Diagnosis:** Diagnosis is made by detection and identification of mites on feathers.

**Size:** Variable with species

**Clinical Importance:** Most feather mite infestations appear to cause little damage and are usually considered of minor clinical importance. Occasionally irritation, dermatitis, and feather damage develop.



Fig. 5.33 Feather mite from a chicken. Feather mite species show great variation in morphology as a result of specialization for life in different parts of the avian feather environment. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 5.34 *Megninia*, a feather mite from a fish. Feather mites infest various species of birds, often feeding at the base of feathers. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE:** *Ornithonyssus sylviarum*, *O. bursa* (Figs. 5.35 and 5.36)

Common name: Northern fowl mite, tropical fowl mite.

**Taxonomy:** Mite (order Mesostigmata).

**Geographic Distribution:** The northern fowl mite, *O. sylviarum*, is found in temperate regions worldwide. The tropical fowl mite, *O. bursa*, is found in tropical and subtropical climates. Both species are found in the United States.

**Location on Host:** Mites and egg masses can be found on the skin among the feathers. In poultry, *O. sylviarum* often concentrates around the vent, causing a dark discoloration of the area.

**Life Cycle:** *Ornithonyssus sylviarum* spends its life on the avian host, whereas *O. bursa* spends greater periods of time off the host. Wild birds can introduce the mites into poultry facilities. Under appropriate conditions, the life cycle of *O. sylviarum* can be completed in a week.

**Laboratory Diagnosis:** Large, grossly visible mites are observed on birds or in the environment.

Size: Approximately 750  $\mu\text{m}$

**Clinical Importance:** Scabbed, matted feathers develop on infested birds. In severe cases, anemia, production loss, and death may occur. Mites can act as vectors of other avian disease agents, including those causing Newcastle disease and fowl pox. Humans in contact with mites may also develop lesions.

**PARASITE:** *Ornithonyssus bacoti* (Figs. 5.35 and 5.36), other *Ornithonyssus* spp.

Common name: Tropical rat mite.

**Taxonomy:** Mite (order Mesostigmata).

**Host:** Rodents, wild birds; occasionally other animals and humans.

**Geographic Distribution:** Worldwide.

**Location on Host:** Skin.

**Life Cycle:** Adult mites lay eggs in the environment. Mites visit the host only to feed; they spend the rest of the time in the host's bedding or nest. The life cycle can be completed in about 2 weeks.

**Laboratory Diagnosis:** Large, grossly visible mites are observed on animals or in the environment.

Size: Approximately 750  $\mu\text{m}$

**Clinical Importance:** In large numbers, this blood-feeding mite can cause anemia, debilitation, and death. Humans in contact with infested laboratory or pet rodents may develop lesions. Lesions can also develop on pets and people when rodent or bird nests associated with human dwellings are abandoned, leaving mites behind to seek other hosts.



Fig. 5.55 *Ornithonyssus* spp. belong to the mesostigmatid order of mites. These mites are quite large and have long legs in the anterior portion of the body. In *Ornithonyssus* spp., the anus (arrow) is at the anterior end of the anal plate. The anus of *Dermatonyssus* spp., a morphologically similar mite, is located in the posterior portion of the anal plate. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

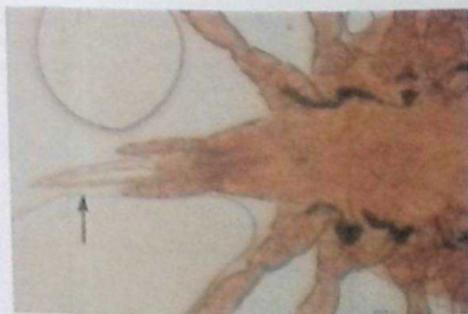


Fig. 5.56 Another characteristic used to differentiate *Ornithonyssus* from the similar genus *Dermatonyssus* is the chelicerae (arrow). The chelicerae in this *Ornithonyssus* mite are shorter than the long, whip-like chelicerae of *Dermatonyssus*.

**PARASITE:** *Dermanyssus gallinae* (Fig. 5.37)

Common name: Red poultry mite.

**Taxonomy:** Mite (order Mesostigmata).

**Host:** Wild and domestic birds.

**Geographic Distribution:** Worldwide.

**Location on Host:** Mites can occur anywhere on the body.

**Life Cycle:** Mites visit the host at night only to take blood meals. During the day, the mites are found in crevices in the environment. The life cycle can be completed in as little as 10 days. Adults can survive in the environment for several months without feeding.

**Laboratory Diagnosis:** Mite infestation may be difficult to diagnose because the mites are not on the host during the day. Close examination of the environment may reveal mites under crusts of manure on perches or in nest boxes. If infestation is suspected in caged birds, the cage can be covered with a white cloth at night. In the morning, mites will be seen as small black or dark red dots clinging to the cloth.

**Size:** Approximately 750  $\mu\text{m}$

**Clinical Importance:** Heavy infestation can cause anemia and death, particularly in hatchlings. Hens may be reluctant to sit on their nests. Other animals and humans in close proximity to infested birds or their nests may also develop lesions.

**PARASITE:** *Pneumonyssoides caninum* (Fig. 5.38)

Common name: Nasal mite.

**Taxonomy:** Mite (order Mesostigmata).

**Host:** Dogs. A similar mite, *Pneumonyssus simicola*, is found in the lungs of rhesus macaques.

**Geographic Distribution:** Worldwide.

**Location on Host:** Nasal sinuses of dogs.

**Life Cycle:** The life cycle of this mite is poorly understood, but transmission is thought to be by direct contact since mites are sometimes seen crawling on the nose.

**Laboratory Diagnosis:** Large mites grossly visible in the nasal sinuses and passages or crawling outside the nostrils.

**Size:** Approximately 1 mm

**Clinical Importance:** Infestations are usually asymptomatic but may produce sneezing, rhinitis, sinusitis, and malaise. In captive rhesus macaques, *Pneumonyssus simicola* can cause significant respiratory disease.



Fig. 5.37 *Dermomyias gallinae* infests both domestic and wild birds. The anus of this mite is present in a more posterior position on the anal plate than in *Ornithonyssus*. *Dermomyias* also has long, whip-like chelicerae (not visible in this figure). Differentiating the genera may be helpful in determining appropriate control measures because of differences in life cycles.



Fig. 5.38 *Pneumonyssoides caninum*, the nasal mite of dogs. A related mite, *Pneumonyssus simicola*, is the lung mite of several species of African monkeys. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**PARASITE:** *Ophionyssus natricis* (Figs. 5.39 and 5.40)

Common name: Snake mite.

**TAXONOMY:** Mite (order Mesostigmata).

**Geographic Distribution:** Worldwide on captive snakes.

**Location on Host:** Mites are found on the skin or under the scales of snakes and may also infest lizards.

**Life Cycle:** *Ophionyssus* nymphs and adults feed and females deposit eggs in the environment after taking a blood meal. Larvae do not feed. The entire life cycle takes about 13–19 days. Infestation occurs by direct contact with an infested snake or its environment.

**Laboratory Diagnosis:** Identification of large mites collected from snakes. Females that have taken a blood meal are dark colored. A cotton-tipped swab can be dipped in mineral oil and used to collect mites for identification.

**Size:** Females 0.6–1.3 mm

**Clinical Importance:** *Ophionyssus* infestations are common in captive snakes. Affected animals may show irritation and depression. Shedding may increase, and snakes may soak themselves more frequently in water.



**Fig. 5.29** *Ophiomyssus* mites are the most common external parasite on captive snakes and may seriously affect the health of the host. Female mites lay eggs off the host, and successful control of infestation requires environmental treatment. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.



**Fig. 5.40** *Ophiomyssus*, like other mesostigmatid mites, has claws on the tips of the legs instead of the suckers seen in many other parasitic mites (Fig. 5.1).



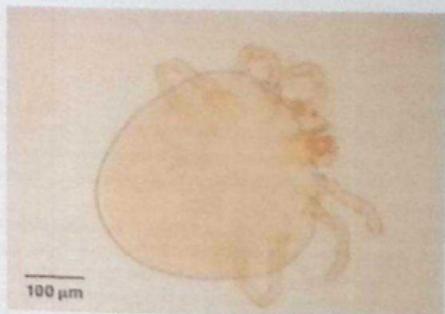


Fig. 5.41 Only the six-legged larvae of chiggers are parasitic, which is helpful in identification of the parasites. This *Blankaria* sp. chigger was removed from a bird.



Fig. 5.42 Specimen of trombiculid larvae that cause mammalian chigger infestation. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 5.43 Typical chigger lesions on the leg of a parasitologist. Chiggers may also cause pruritus and irritation on animals.



## Tick Identification

Ticks are usually larger than mites, ranging in length from 3 to 12 mm, or more in the case of engorged females. Ticks are divided into two families: Ixodidae (hard ticks) and Argasidae (soft ticks). The Ixodid (hard) ticks are of greatest importance in veterinary medicine. Various hard tick species are vectors of a number of viral, bacterial, protozoal, and nematodal animal and human pathogens. In addition, hard tick species cause tick paralysis and tick toxicosis.

All ticks pass from the egg through larval and nymphal stages before becoming adults and utilize one or more host animals during the developmental cycle. Eggs are always laid in the environment. Hard tick larvae are acquired by the host from the environment. All hard ticks undergo a single molt from the larval to the nymphal stage and a second molt from the nymph to the adult. These molts follow attachment and blood-feeding on the host that usually lasts for several days. Tick species that remain on the host during the two molting periods are known as one-host ticks. In two-host tick species, the molt to the nymphal stage occurs on the host, but the engorged nymph leaves the host, molts in the environment, and then finds a new host. In the three-host tick life cycle, both the larva and nymph leave the host to molt, attaching to a host again after each molt. In some cases, each tick stage prefers the same host species; in others, host preference may vary with the stage of the tick. In much of North America, the most important tick species are three-host ticks. Soft tick life cycles are more variable than those of the hard ticks. Many soft tick species live in the environment and visit the host only briefly to take repeated blood meals.

All stages of ticks are large enough to be grossly visible on animals, although larvae may be only a few millimeters in length and soft ticks usually do not attach for long periods. Hard ticks may attach anywhere on their hosts but are likely to be found attached in areas on the host that cannot be easily groomed, for example, the head, neck, and ears of most host species (Fig. 5.44), and also the tail of horses. Because ticks are important vectors of pathogens that may result in disease (e.g., Lyme disease, Rocky Mountain spotted fever, ehrlichiosis, cytauxzoonosis, anaplasmosis, etc.), they should be removed as quickly as possible using forceps or tweezers instead of fingers to reduce the possibility of contact with tick body fluids containing infectious organisms. The tick should be firmly grasped directly behind the point of attachment to the skin and then pulled off. Often a small portion of skin will also be pulled away.



Fig. 5.44. Ticks are often found attached on parts of the body that are difficult for the host to groom. Unidentified ticks are attached to the ear of this dog. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

Hard ticks have a hard dorsal shield called the scutum. The scutum is limited to the anterior, central region of the dorsum in females, whereas in males, the scutum extends over the entire dorsal region. The mouthparts of hard ticks are evident from the dorsal surface. The Argasid (soft) ticks have a leathery integument, which often is spinose or bumpy. The mouthparts of adult soft ticks can be seen only from the ventral aspect of the tick.

Identification of adult hard ticks to the level of genus is not difficult in a veterinary practice. One of the most useful characteristics for identifying the genus of a hard tick is the shape of the basis capituli and mouthparts. Figure 5.45 shows these characteristics on adult females; a magnifying glass or dissecting microscope is useful for looking at the basis capituli. The pigmented markings of the scutum (referred to as "ornamentation") are another useful characteristic. Ticks with ornamentation are called "ornate"; those lacking these markings are "inornate." Additionally, in some genera, the posterior margin of the body has a series of indentations, known as festoons (see adult *Dermacentor*). Features like festoons are much more difficult to appreciate on engorged female ticks. Identification is easiest with non-engorged females or males. Nymphs, like adults, have eight legs but lack the genital pore seen in adults. Larvae have six legs (Fig. 5.46), are smaller than nymphs, and may require the assistance of an expert to identify beyond genus. Figure 5.47 shows adult female ticks of the most common species in the United States. They also represent the most common tick genera found

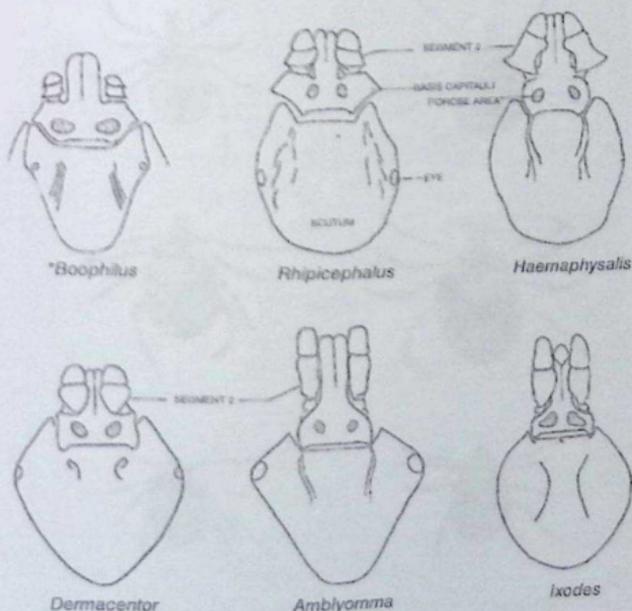


FIG. 5.45. Comparison of the basis capituli and mouthparts of females of the important Ixodid tick genera of domestic animals in North America. The scutum of the adult male hard ticks covers the entire dorsum of the parasite. \**Boophilus* ticks have now been incorporated into the genus *Rhipicephalus*. Modified from USDA APHIS Agriculture Handbook No. 485.



Fig. 5.46 Larval ticks are often called "seed ticks" because of their small size. Tick larvae have only six legs and can be distinguished from nymphs and adults each of which has eight legs. Microscopic examination will likely be necessary to count the number of legs on tick larvae. Left, engorged tick larva. Right, non-engorged or flat tick larva that has been treated with a clearing solution to highlight morphological characteristics.

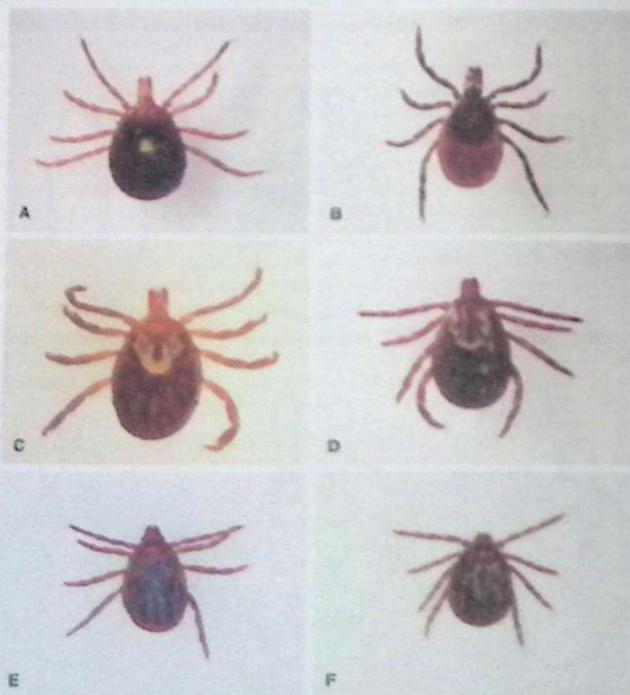


Fig. 5.47 Female ticks of highest veterinary medical importance in the United States. (A) *Amblyomma americanum*, lone star tick; (B) *Ixodes scapularis*, black-legged tick or deer tick; (C) *Amblyomma maculatum*, Gulf Coast tick; (D) *Dermacentor variabilis*, American dog tick; (E) *Rhipicephalus sanguineus*, brown dog tick; (F) *Haemaphysalis longicornis*. More information on these species can be found in the following pages. Photos courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.

PICTORIAL KEY TO GENERA OF ADULT TICKS IN UNITED STATES

By Harry D Pratt

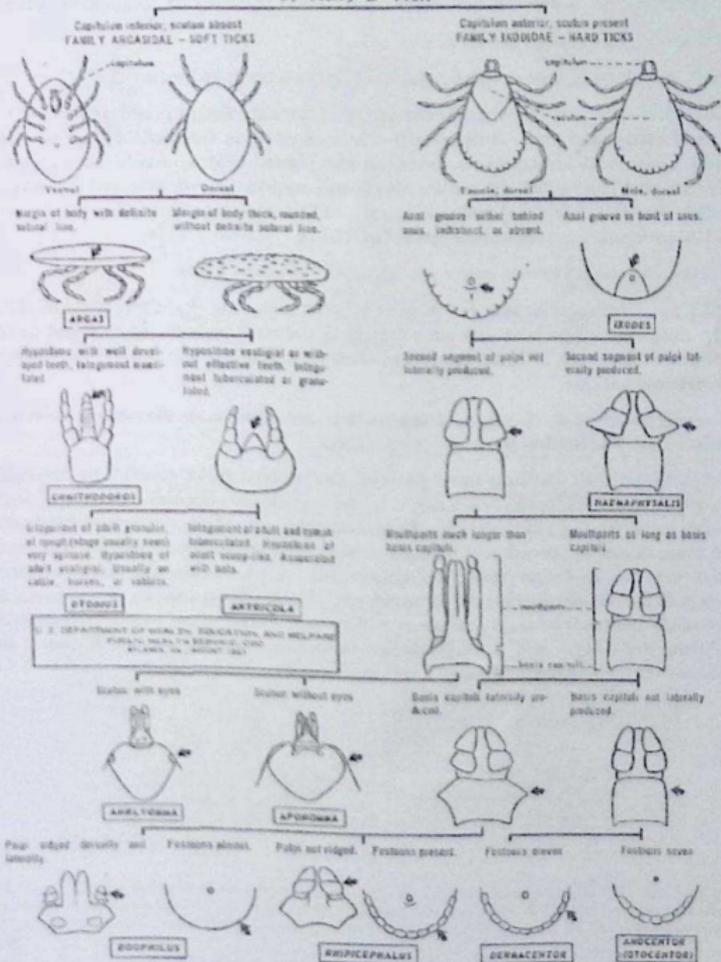


Fig. 5.48 Key to adult tick genera found in North America. Examination of ticks with low magnification should allow identification of features used in this key. *Boophilus* ticks have now been incorporated into the genus *Rhipicephalus*. Courtesy of U.S. Public Health Service, CDC.

throughout the world. If assistance is needed with specific identification of ticks, they should be preserved and submitted to a diagnostic laboratory in 70%–80% alcohol. A dichotomous key for the genera of adult ticks (Fig. 5.48) can be followed for identification of most tick specimens in North America.

**PARASITE:** *Amblyomma* spp. (Figs. 5.49–5.54; see also Fig. 5.47)

**COMMON NAMES:** Lone star tick, Gulf Coast tick, Cayenne tick, tropical bont tick.

**TAXONOMY:** Tick (family Ixodidae).

**HOST:** A wide variety of domestic and wild animals serve as hosts.

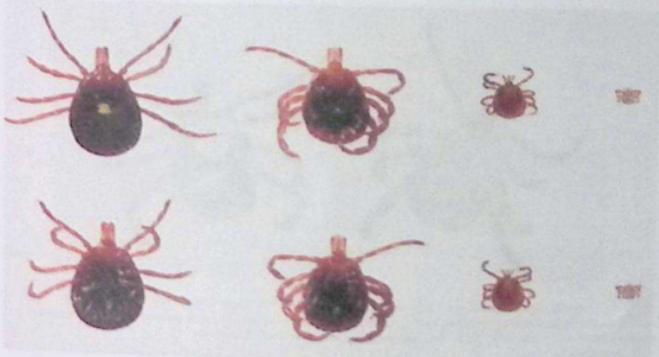
**Geographic Distribution:** Approximately 100 species are found predominantly in tropical and subtropical areas. *Amblyomma americanum* (lone star tick) and *A. maculatum* (Gulf Coast tick) are common species in the United States. *Amblyomma cajennense* (Cayenne tick) and other species are also found on a variety of wild and domestic animals in the United States, Mexico, Central and South America. *Amblyomma hebraeum* and *A. variegatum* are important species in Africa.

**Location on Host:** Various, prefer ventral aspect of many hosts.

**Life Cycle:** *Amblyomma* spp. are three-host ticks, meaning that each stage of the life cycle must find a new host following a molt in the environment. Larvae and nymphs feed on a wide variety of hosts; adults are often found on ruminants and other domestic animals and humans.

**Laboratory Diagnosis:** Long mouthparts are an important diagnostic feature of *Amblyomma*. The scutum is usually ornamented.

**Clinical Importance:** The long mouthparts of *Amblyomma* make attachment particularly painful and susceptible to secondary infection. *Amblyomma americanum* is a vector of several notable pathogens (e.g., *Ehrlichia* spp., *Francisella tularensis*, *Cytauxzoon felis*, *Phlebotomus*) that cause disease in animals and humans. *Amblyomma maculatum* transmits *Hepatozoon americanum* and *Rickettsia parkeri*. Infestations with *A. maculatum* can cause permanent damage to the ears of cattle known as "gotch ear." The lesions caused by *A. cajennense* are particularly painful. It serves as a vector of *R. rickettsia* and agents of equine piroplasmiasis. In Africa, *A. hebraeum* and *A. variegatum* transmit *Ehrlichia ruminantium*, *R. conori*, and *Nairovirus*. In the Caribbean, *A. variegatum* also transmits *E. ruminantium*.



**Fig. 5.48** Dorsal (top row) and ventral (bottom row) aspects of all motile stages of *Amblyomma americanum* (lone star tick), demonstrating the considerable variation in size of life stages. From left to right are adult female, adult male, nymph, larva. This tick is a generalist and all stages may infest medium- and large-sized animals within its range. The female of this common U.S. tick is easily recognized by the presence of the large white spot at the posterior margin of the scutum. Males do not have conspicuous ornamentation on the scutum, although with close inspection, some iridescent markings can be seen on the margin of the scutum and festoons. Photo courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.



**Fig. 5.49** Dorsal (left) and ventral (right) views of *Amblyomma americanum* nymph. Like adults, nymphs have eight legs, but are smaller and lack a genital opening. The nymphs of *A. americanum* lack the dramatic "lone star" of adult females. Photos courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.



Fig. 5.51 *Amblyomma maculatum*: (left) male; (right) female. The Gulf Coast tick is found in the southeastern United States, Mexico, and South and Central America. It feeds primarily on the head and neck of birds and mammals. Note the long mouthparts typical of this genus.



Fig. 5.52 *Amblyomma cajennense*: (left) male; female (right). The Cayenne tick is found in Texas, Mexico, Central and South America. It parasitizes a wide variety of mammals and birds. Photo courtesy of James Gathany and Christopher Paddock, Centers for Disease Control and Prevention (Public Health Image Library).



Fig. 5.53 *Amblyomma variegatum*. (left) male; (right) female. The tropical bont tick is an important vector of heartwater in cattle. This tick is also found in the Caribbean.



Fig. 5.54 *Amblyomma* spp. are common in the tropics and subtropics. They are often highly ornamented with iridescent markings like these African *Amblyomma*. Some tick genera, like *Amblyomma*, have simple eyes on the margin of the scutum (*arvosa*).

**PARASITE:** *Hyalomma* spp. (Fig. 5.55)

Common name: Bont-legged tick, camel tick, tortoise tick.

**Taxonomy:** Tick (family Ixodidae).

**Host:** Many host species, including domestic animals. One species, *H. aegyptium*, is a parasite of tortoises.

**Geographic Distribution:** Asia, Europe, North Africa.

**Location on Host:** Various.

**Life Cycle:** *Hyalomma* species are usually two-host ticks, which leave the host after nymphal and adult blood meals.

**Laboratory Diagnosis:** Ticks of the genus *Hyalomma* have eyes and long mouthparts like those of *Amblyomma*, but lack the ornamentation usually seen on *Amblyomma* spp.

**Clinical Importance:** *Hyalomma* species cause tick toxicosis and serve as vectors of *Babesia*, *Theileria*, and *Rickettsia* spp.



Fig. 5.55. *Hyalomma* spp. ticks are important disease vectors in Africa, Asia, and Australasia, although they are not found in the Western Hemisphere. They have long mouthparts, eyes, and festoons like *Amblyomma* but are not highly ornamented.

**PARASITE:** *Ixodes* spp. (Figs. 5.56–5.58; see also Figs. 5.47, 5.67)

Common name: Black-legged tick, deer tick, European sheep tick (castor bean tick), hedgehog tick, British dog tick, Australian and South African paralysis ticks.

**TAXONOMY:** Tick (family Ixodidae). *Ixodes* is the largest genus of hard ticks, containing more than 200 species. Approximately 35 species of *Ixodes* are found in North America.

**Host:** Many host species, including domestic animals and humans.

**Geographic Distribution:** Some of the most important species in domestic animals include *I. scapularis* (black-legged or deer tick) and *I. pacificus* (western black-legged tick) in North America; *I. ricinus* (sheep or castor bean tick), *I. canisuga* (British dog tick), and *I. hexagonus* (hedgehog tick) in Europe; *I. rubicundus* (South African paralysis tick); and *I. holocyclus* (Australian paralysis tick).

**Location on Host:** Various.

**Life Cycle:** *Ixodes* species are three-host ticks.

**Laboratory Diagnosis:** The most helpful characteristic for identification of *Ixodes* ticks is an anal groove that runs from the posterior margin of the body to just anterior to the anus. A magnifying glass or dissecting-type microscope may be needed to identify this feature.

**Clinical Importance:** *Ixodes* spp. in North America and Europe are vectors of *Borrelia* spp., *Babesia* spp., *Ehrlichia* spp., and *Flavivirus*. *Ixodes ricinus* transmits *Flavivirus* and *Babesia* spp. in Europe. *Ixodes* spp. can also cause dermatitis and tick worry and are major causes of tick paralysis in Australia and South Africa.



Fig. 5.56 Engorged nymph and engorged adult female *Ixodes scapularis*, the deer tick. This species is the primary vector of the agent of Lyme disease in the United States and is smaller than other common ticks (see Fig. 5.47 for comparison).



Fig. 5.57 Unfed adult female (left) and male (right) *Ixodes scapularis* viewed dorsally. The scutum of *Ixodes scapularis* is not ornamented and both legs and scutum are a dark brown-black color. Photo courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.



Fig. 5.58 A distinctive morphologic detail of the *Ixodes* ticks is the groove that runs anterior to the anus (arrow). In other tick genera, this groove is either posterior to the anus or absent. *Ixodes* ticks also have long mouthparts. Photos courtesy of Parna Ghosh, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Dermacentor* spp. (Figs. 5.59–5.62; see also Fig. 5.47)

Common name: American dog tick, Rocky Mountain wood tick, winter tick, tropical horse tick.

**Taxonomy:** Tick (family Ixodidae).

**Host:** Depending on the species, a wide variety of wild and domestic hosts can be used.

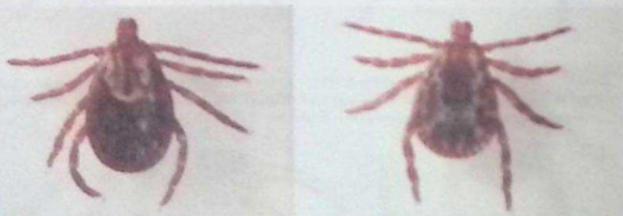
**Geographic Distribution:** Primarily Europe, Asia, and North America. *Dermacentor variabilis* (American dog tick), *D. andersoni* (Rocky Mountain wood tick), *D. albipictus* (winter or elk or horse tick), and *D. occidentalis* (Pacific Coast tick) are found in North America and parasitize a variety of animals. *Dermacentor* (= *Anocentor*) *nitens* is a parasite of equids in Florida, the Caribbean, Central and South America. In Europe, *D. reticulatus* is an important species.

**Location on Host:** Various.

**Life Cycle:** Most *Dermacentor* spp. are three-host ticks that prefer small rodents in larval and nymphal stages and larger vertebrates in the adult stage. *Dermacentor nitens* and *D. albipictus* are one-host ticks.

**Laboratory Diagnosis:** *Dermacentor* spp. are usually ornamented with relatively short mouthparts and a rectangular basis capituli.

**Clinical Importance:** *Dermacentor* spp. in the United States are the most common vectors of *Rickettsia rickettsii* and can also transmit *Anaplasma marginale* to cattle. *Dermacentor* (*Anocentor*) *nitens* is the vector of *Babesia caballi* and *Theileria equi* in the United States. In Europe, *D. reticulatus* is the vector of *Babesia* spp. to horses and dogs. Several species of *Dermacentor* are known to cause tick paralysis.



**Fig. 5.59** *Dermacentor variabilis*: female (left), male (right). Like many members of this genus, *D. variabilis* (the American dog tick) is an ornamented tick. The short mouthparts, rectangular shape of the basis capituli, and presence of festoons are used in identifying the genus. Photos courtesy of Megan Lieberry, Oklahoma State University, Stillwater, OK.



Fig. 5.60 *Dermacentor andersoni*: (left) male; (right) female; the Rocky Mountain wood tick. This tick is found in the central and western United States and Canada. Photo courtesy of James Gathany and Christopher Paddock, Centers for Disease Control and Prevention (Public Health Image Library).



Fig. 5.61 *Dermacentor albipictus*, the winter or mouse tick, is a one-host tick most active in winter. Both the brown variant (left) and variegated (right) strains of *D. albipictus* occur widely in North America. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 5.62 *Dermacentor (Anocentor) nitens* engorged female (left) and male (right). Unlike most important hard ticks, this is a one-host tick species that prefers horses as host but will attach to many mammals. It is important as a vector of equine piroplasmiasis in Central and South America, the Caribbean, Mexico, Texas, and Florida.

**PARASITE:** *Rhipicephalus* spp. (Figs. 5.63–5.66; see also Figs. 5.47, 5.67)

**Common name:** Brown dog tick or kennel tick, brown ear tick, red-legged tick, cattle fever tick, blue tick, tropical cattle tick.

**Taxonomy:** Tick (family Ixodidae). Important parasites of domestic animals include *R. sanguineus* (brown dog tick), *R. appendiculatus* (brown ear tick), and *R. evertsi* (red-legged tick). Ticks previously classified in the genus *Boophilus* are now included in *Rhipicephalus*, including the former *B. microplus* (tropical cattle tick), *B. annulatus* (cattle fever tick), and *B. decoloratus* (blue tick).

**Host:** This genus is most important in livestock and dogs.

**Geographic Distribution:** *Rhipicephalus sanguineus* is found worldwide. *Rhipicephalus appendiculatus* and *R. evertsi* are found on livestock in Africa. *Rhipicephalus* (*Boophilus*) *microplus* (tropical cattle tick) is found worldwide; *R. annulatus* (cattle fever tick) is found in the Western Hemisphere and parts of Africa; *R. decoloratus* (blue tick) is an African tick.

**Location on Host:** Various.

**Life Cycle:** *Rhipicephalus sanguineus* is a three-host tick that uses a dog host for each stage of the life cycle. *Rhipicephalus appendiculatus* is also a three-host tick, while *R. evertsi* is a two-host tick and *R. microplus*, *R. annulatus*, and *R. decoloratus* are one-host ticks. After hatching from the egg in the environment, ticks locate the host, where they remain through the subsequent nymphal and adult stages. Females leave the host to lay their eggs in the environment.

**Laboratory Diagnosis:** *Rhipicephalus* spp. have a hexagonally shaped basis capituli. The members of the genus that were formerly called *Boophilus* have ridged palps.

**Clinical Importance:** *Rhipicephalus sanguineus* transmits *Babesia canis vogeli* and *Ehrlichia canis* to dogs. *Rhipicephalus* species infesting livestock, including *R. appendiculatus*, *R. evertsi*, *R. microplus*, and *R. annulatus* are the primary vectors of bovine *Theileria* spp., *Babesia* spp., *Anaplasma* spp., and *Nairovirus* to livestock.



**Fig. 5.63** Engorged female *Rhipicephalus sanguineus* (brown dog tick). This species is not ornate. In the United States, it is most common in southern states but can also be a pest in kennels in other areas since dogs are used as hosts for every stage of the life cycle. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



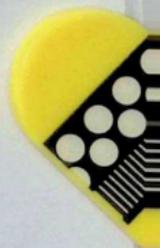
Fig. 5.64 *Rhipicephalus sanguineus* male (left) and engorged female (right). There are no markings on the scutum.



Fig. 5.65 The brown dog tick, *Rhipicephalus sanguineus*. Members of this tick genus have a basis capituli that is hexagonal in shape with flared sides (arrow; see also Fig. 5.45).



Fig. 5.66 *Rhipicephalus (Boophilus) microplus* female (left) and male (right). This is a one-host tick of cattle and the vector for bovine babesiosis.



**PARASITE:** *Haemaphysalis* spp. (Figs. 5.67–5.70; see also Fig. 5.47)

Common name: Rabbit tick, yellow dog tick, bush tick, longhorned tick.

**Taxonomy:** Tick (family Ixodidae).

**Host:** *Haemaphysalis* ticks parasitize a wide range of mammals and birds, depending on the species.

**Geographic Distribution:** Worldwide. *Haemaphysalis leporispalustris* (rabbit tick) is found in the Western Hemisphere; *H. leachi* (yellow dog tick) is found in Africa and parts of Asia; and *H. longicornis* (longhorned tick) is found primarily in Asia but was identified in the United States in 2017. Several other species parasitize livestock in Europe, Africa, Asia, and Australasia.

**Location on Host:** Various.

**Life Cycle:** *Haemaphysalis* spp. are three-host ticks and leave the host after each blood meal. Larvae and nymphs typically feed on small mammals and birds, and adult ticks feed on larger mammals. *Haemaphysalis longicornis* females are capable of reproduction through parthenogenesis. Introduction with only one or a few female ticks may result in massive infestations.

**Laboratory Diagnosis:** Ticks of this genus have festoons, and the second segment of the palps flares out on the lateral margin.

**Clinical Importance:** Large numbers of *Haemaphysalis* ticks contribute to poor condition and “tick worry.” *Haemaphysalis punctata* can transmit several species of *Babesia* and *Anaplasma* to livestock. *Haemaphysalis leachi* is a vector of canine babesiosis. *Haemaphysalis longicornis* is capable of producing severe clinical disease impacting the health and production of infested animals. *Haemaphysalis longicornis* is a vector for *Phlebovirus* and infected with various species of *Anaplasma*, *Babesia*, *Borrelia*, *Ehrlichia*, and *Rickettsia* of animals and humans. At the time of this writing, it is unknown if any exotic pathogens were introduced with *H. longicornis* into the United States.



Fig. 5.67 From left to right, engorged *Ixodes*, *Haemaphysalis*, and *Rhipicephalus* females. Photo courtesy of Dr. Nick Sangster, Charles Sturt University, Wagga Wagga, NSW, Australia and Ms. Sally Pope, Faculty of Veterinary Science, University of Sydney, Sydney, NSW, Australia.



**Fig. 5.68** *Haemaphysalis longicornis*, the longhorned tick, was recently discovered in North America. These ticks are inornate with festoons and short mouthparts. The second segment of the palps project laterally and are wider than the rectangular basis capituli. Photo courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.



**Fig. 5.69** *Haemaphysalis leporispalustris*, the rabbit tick, is found in North America and can carry the bacterial agent of tularemia, *Francisella tularensis*. Other members of this tick genus are vectors of *Babesia* and *Theileria* in Africa, Asia, and Australasia. The female tick in this photo (right) has host skin tissue attached to the mouthparts.



**Fig. 5.70** The palps of *Haemaphysalis* ticks are wider than they are long, and in most species, the second segment of the palps flares laterally (arrow).



**PARASITE:** *Otobius megnini* (Figs. 5.71–5.73)

**Common name:** Spinose ear tick.

**Taxonomy:** Tick (family Argasidae).

**Host:** Ruminants and horses primarily, also camelids and small animals.

**Geographic Distribution:** North and South America, Africa, India.

**Location on Host:** External ear canal.

**Life Cycle:** Eggs hatch in the environment. Larvae enter the ear of the host and may remain for several months until the nymphal stage is completed. There are one larval and two nymphal stages. Nymphs leave the host after feeding and molt to the adult stage, which does not feed.

**Laboratory Diagnosis:** These ticks are easily diagnosed based on host location and recognition of specimens as soft ticks. *Otobius* is covered with short spines, leading to the name spinose ear tick. Hard ticks may also attach in the ears, but they have a distinctive hard, enameled appearance compared with soft ticks like *Otobius*. Also, any adult-stage ticks found in the ears will not be *O. megnini*, since spinose ear ticks are parasitic only as larvae and nymphs.

**Clinical Importance:** Large numbers can cause severe inflammation and rupture the ear drum.



**Fig. 5.71** *Otobius*, the spinose ear tick, and *Dermacentor variator*. This picture demonstrates the difference between soft and hard ticks. The soft tick, *Otobius* (left), does not have the hard enamel-like surface of the ixodid tick, *Dermacentor* (right). The capitulum of soft ticks is also not always visible from the dorsal surface. Hard ticks are much more common than soft ticks on domestic animals in North America.



**Fig. 5.72** Partially engorged larva of *Otobius megnini*, the spinose ear tick. The integument is striated with a few bristle-like hairs. In fed larvae (above), the capitulum extends from a conical anterior projection. They are found clustered in the ears of the host. Photo courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.



**Fig. 5.73** This closer view of *Otobius megnini* nymphs shows the spines that cover the surface. The hypostome is well developed and more commonly viewed from the ventral aspect. The second nymphal stage is the one most often encountered. Photos courtesy of Megan Lineberry, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Argas* spp. (Fig. 5.74)

Common name: Fowl tick.

**Taxonomy:** Tick (family Argasidae). Important species include *Argas persicus* and *Argas reflexus*.

**Host:** Poultry and wild birds.

**Geographic Distribution:** Worldwide.

**Location on Host:** Various.

**Life Cycle:** *Argas* is a soft tick that lives in the environment, attacking birds only to feed, usually during the night. Several blood meals are taken by larval and adult ticks.

**Laboratory Diagnosis:** As their name suggests, soft ticks lack the hard "enameled" appearance of hard ticks. The mouthparts of soft ticks cannot be seen from the dorsal surface, which also helps to distinguish them from hard ticks. *Argas* spp. have a flattened body margin.

**Clinical Importance:** Heavy burdens can cause loss of production and death. *Argas* spp. act as vectors for *Borrelia anserina* and can cause fowl paralysis. They are uncommon in total-confinement poultry systems.

**PARASITE:** *Ornithodoros* spp. (Fig. 5.75)

Common name: Tsetse.

**Taxonomy:** Tick (family Argasidae). *Ornithodoros moubata* is the African tsetse; *O. hermsi* is one of the species found in the United States.

**Host:** Domestic livestock and humans.

**Geographic Distribution:** Africa, Asia, North and South America.

**Location on Host:** Various.

**Life Cycle:** These ticks are often nocturnal and are found in animal or human habitations, including dens, nests, or crevices of buildings.

**Laboratory Diagnosis:** In contrast to *Argas* spp. ticks, *Ornithodoros* spp. do not have a lateral sutural line and there is no distinct body margin.

**Clinical Importance:** Large numbers of these soft ticks can cause significant blood loss. *Ornithodoros* transmits endemic relapsing fever in humans. In the United States, *Ornithodoros* ticks are most common in the western and southwestern states.



Fig. 5.74 *Argas* sp., the fowl tick, is a soft tick. The ventral location of the mouthparts is clearly seen in this specimen. The surface of *Argas* is granulated. Photo courtesy of Dr. Dwight Bowman, College of Veterinary Medicine, Cornell University, Ithaca, NY.

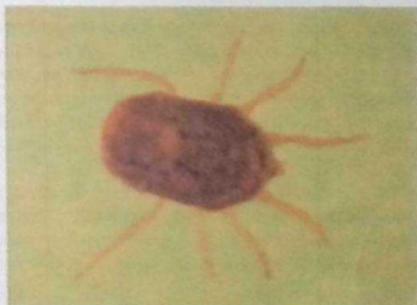


Fig. 5.75 The surface of the soft tick *Ornithodoros* is covered with mammillae (small bumps) and there is no distinct margin to the body.



## CLASS INSECTA

Like ticks and mites, insects also belong to the phylum Arthropoda. All insects have bodies composed of three parts: the head, the thorax (which bears the legs), and the abdomen. Adult insects have six legs and some have wings. Life cycles of parasitic insects may be quite simple, in which larval stages are similar in appearance to the adults, or very complex, involving transformation from a worm-like maggot through a pupal stage to the adult. The insects of greatest veterinary importance are the lice, fleas, and flies.

## Lice (Order Phthiraptera)

Lice are wingless, dorsoventrally flattened insects ranging in length from about 1 to 8 mm. They are common ectoparasites of mammals and birds. Louse infestations in domestic animals are most commonly observed in the winter and are referred to as "pediculosis," a term derived from a genus of lice important to humans. Lice are highly species-specific and the entire life cycle is completed on the host; stages only survive off the host for 2–3 days. Immature lice resemble adults but are smaller. Eggs (nits) are white or yellow in color and may be observed attached individually to hair shafts and sometimes at the base of feathers on birds. Lice and nits can usually be seen with the unaided eye although magnification aids recovery.

Lice are traditionally divided into two groups based on how they feed: anopluran (sucking lice) and mallophagan (chewing lice). Both sucking and chewing lice are found on mammals, but birds are parasitized only by chewing lice. Sucking lice (anopluran) feed on blood, move slowly compared to chewing lice, and may be seen with their head pointed downward, close to the skin surface, or actually feeding. Sucking lice are generally larger than chewing lice (Fig. 5.76) and are gray to dusky red, depending on the quantity of blood ingested (Fig. 5.77). The head of sucking lice is narrower than the thorax and has elongated protrusible piercing mouthparts. Chewing lice (mallophagan) are smaller (Fig. 5.76, 5.78) and feed on skin scurf and other organic material on the skin. Chewing lice move rapidly and have blunt heads that are wider than the thorax and mandible-like mouthparts. Chewing lice are often a yellow color. While species



Fig. 5.76 Chewing lice of domestic animals are usually smaller than sucking lice. *Bovicola bovis* (arrow) is pictured here with one of the bovine sucking lice, *Linognathus*. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.



Fig. 5.77 Sucking louse infestation on a calf. Note the reddish-brown color of these blood-feeding lice. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

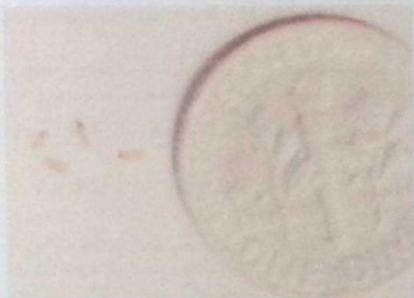


Fig. 5.78 *Bovicola ovis* is a small white or tan chewing louse that can be very difficult to detect on a heavily fleeced sheep. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 5.79 Section of bovine skin with louse eggs (nits) attached to the hairs. "Nits" refers to insect eggs adhered to the hair and is used to describe the eggs of lice or bot flies on animals. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

identification of lice is usually not required in veterinary practice, recognition of an organism as a chewing or sucking louse may be helpful when selecting treatment.

Lice or their eggs (nits, Fig. 5.79) can be recovered from animals with a fine-toothed comb or by examining hair coat brushings. A magnifying lens or dissecting microscope may be useful if lice are very small. Specimens may be transferred to saline, mineral oil, or Hoyer's solution and placed on a microscope slide with fine forceps to facilitate viewing. If shipping to a laboratory, lice should be placed in 70% ethanol.

**PARASITE:** *Haematopinus* spp. (Fig. 5.80)

Common name: Pig louse, short-nosed cattle louse, cattle tail louse.

**Taxonomy:** Insect (Order Phthiraptera, anopluran or sucking lice).

**Host:** Species important in domestic animals include *H. suis* (pigs), *H. asini* (horses), *H. eurysternus* (short-nosed sucking louse of cattle), and *H. quadripertusus* (tail louse of cattle). Sucking lice are also found on camelids (genus *Microthoractus*).

**Geographic Distribution:** Worldwide. *Haematopinus quadripertusus* is found primarily in the tropics and subtropics.

**Location on Host:** *Haematopinus* spp. are often found on the head, neck, and back of the host. *Haematopinus quadripertusus* is usually found around the tail.

**Life Cycle:** Transmission is by direct contact or fomites. Eggs are glued to the hairs of the host.

**Laboratory Diagnosis:** Members of this genus are large, about 4–5 mm in length, with the elongated heads typical of the sucking lice.

**Clinical Importance:** *Haematopinus* infestations can produce alopecia and pruritus, leading to self-inflicted trauma and production losses. Heavy infestations can produce anemia. *Haematopinus suis* is a common and important ectoparasite of swine; *H. eurysternus* is considered the most important cattle louse worldwide. Infestation of horses is uncommon in well-managed stables.

**PARASITE:** *Linognathus* spp. (Figs. 5.81 and 5.82)

Common name: Face louse and foot louse of sheep, long-nosed cattle louse.

**Taxonomy:** Insect (Order Phthiraptera, anopluran or sucking lice).

**Host:** *Linognathus pedalis* (ovine foot louse), *L. ovillus* (ovine face louse), *L. vituli* (long-nosed cattle louse), *L. africanus* (bovine African blue louse), *L. setosus* (dogs).

**Geographic Distribution:** Worldwide.

**Location on Host:** The face and foot lice of sheep are found primarily in those locations; other species are less restricted in distribution.

**Life Cycle:** Transmission is by direct contact or fomites.

**Laboratory Diagnosis:** Detection of lice and eggs on the host by gross observation.

**Clinical Importance:** As with other lice, infested animals show pruritus and dermatitis, and severe infestations can lead to production losses and anemia.



Fig. 5.60 *Haematopinus* spp. have prominent ocular points (arrow, partially obscured by antenna) and legs of equal size. *Haematopinus suis*, shown here, is the largest louse found on domesticated animals. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.



Fig. 5.61 *Linognathus* spp. have no ocular points. Unlike *Haematopinus*, the second and third pairs of legs are larger than the first pair. The louse shown here is *L. setosus*, the sucking louse of dogs. Members of this genus are usually 2–3 mm in length.



Fig. 5.62 Each sucking louse leg ends in a prominent claw. Shown are the claws of *L. africanus* from a goat. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE:** *Solenopotes capillatus* (Fig. 5.83)

Common name: Little blue cattle louse.

**Taxonomy:** Insect (Order Phthiraptera, anopluran or sucking lice).

**Host:** Cattle.

**Geographic Distribution:** Worldwide.

**Location on Host:** Usually found concentrated on the face, neck, shoulders, back, and tail.

**Life Cycle:** The entire life cycle is spent on the host with transmission by direct contact or fomites.

**Laboratory Diagnosis:** *Solenopotes capillatus* is similar in appearance to *Linognathus*, but *Solenopotes* has tubercles carrying spiracles that project from abdominal segments.

**Clinical Importance:** Large numbers of lice may cause production loss from dermatitis and anemia.

**PARASITE:** *Pediculus* spp. (Fig. 5.84), *Phthirus pubis* (Fig. 5.85)

Common name: Human head louse, body louse, crab louse.

**Taxonomy:** Insects (Order Phthiraptera, anopluran or sucking lice). *Pediculus humanus humanus* is the body louse; *Pediculus humanus capitis* is the head louse; and *Phthirus pubis* is the pubic or crab louse.

**Host:** Humans

**Geographic Distribution:** Worldwide.

**Location on Host:** Head lice are found on the scalp, while body lice live principally in clothing and visit the skin to feed. Crab lice are found in the pubic area or on other coarse body hair.

**Life Cycle:** Transmission of all human lice infestations is by direct contact or fomites. Head and crab lice glue their eggs to host hair, while body lice deposit their eggs in clothing.

**Laboratory Diagnosis:** Observation of lice and eggs (nits).

**Clinical Importance:** Humans are the only hosts of these parasites. Their veterinary importance lies in the occasional detection of a human louse on a pet and resulting confusion about who gave what to whom. In these cases, the family pet has lice only because of close contact with infested humans. Human lice do not survive or reproduce on domestic animals.



Fig. 5.83 *Solenopotes capillatus*, the little blue cattle louse, is less common than *Linognathus* spp. The projecting tubercles carrying spiracles on the abdominal segments of *Solenopotes* (arrow) are helpful in identification of this louse. Photo courtesy of Merial.

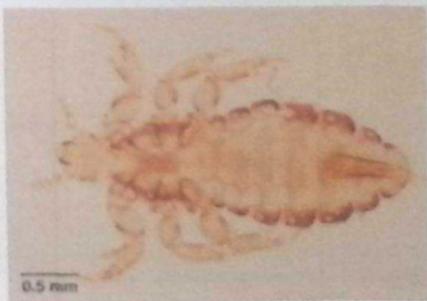


Fig. 5.84 *Pediculus humanus* has well-developed eyes, no ocular points, and three large pairs of legs.



Fig. 5.85 The human crab louse, *Pthirus pubis*, has a distinctive crab-shaped body. Photo courtesy of Dr. Brian Herrin, College of Veterinary Medicine, Kansas State University, Manhattan, KS.

**PARASITE:** *Polyplax spinulosa* (Figs. 5.86 and 5.87)

Common name: Spiny rat louse.

**Taxonomy:** Insect (Order Phthiraptera, anopluran or sucking lice)

**Host:** Rats. Other species of *Polyplax* spp. are found on mice and other rodents, and other sucking lice are found on rabbits.

**Geographic Distribution:** Worldwide.

**Location on Host:** Predilection sites variable, depending on species.

**Life Cycle:** As with other lice, transmission is by direct contact with an infested animal or fomites.

**Laboratory Diagnosis:** Detection of eggs and lice on hair and morphologic identification of lice.

**Clinical Importance:** Large numbers of lice may cause loss of condition and possibly anemia.

**PARASITE:** *Gliricola porcelli* (Fig. 5.88)

Common name: Slender guinea pig louse.

**Taxonomy:** Insect (Order Phthiraptera, mallophagan or chewing lice).

**Host:** *Gliricola porcelli* is the most common louse of guinea pigs. Two other species of chewing lice, *Gyropus ovalis* and *Trimenopon jenningsi*, are also found on guinea pigs.

**Geographic Distribution:** Worldwide.

**Location on Host:** *Gliricola porcelli* prefers the fine hair around the back legs and anus, whereas *Gyropus ovalis* is found around the head and face.

**Life Cycle:** Transmission is by direct contact.

**Laboratory Diagnosis:** Detection and identification of lice and eggs. *Gyropus ovalis* is a broader louse than *Gliricola* and has a wide head. Eggs of *Gyropus ovalis* can be found most easily around the back of the ears. *Trimenopon jenningsi* is a dark brown louse that is less common than the other two species.

**Clinical Importance:** Light infestations are usually asymptomatic. Heavier infestations may be associated with hair loss, unthriftiness, and pruritus, especially at the back of the ears. Louse infestation may not be evident until the death of the host, when lice move up to the hair tips as the body temperature declines.



Fig. 5.86 Sucking louse (*Polyplax*) species from a rat. Typical of sucking lice, the head is narrower than the thorax in this specimen.



Fig. 5.87 *Polyplax* egg glued to a rat hair. The presence of lice eggs ("nits") on the hairs is helpful in diagnosis of infestation.



Fig. 5.88 *Gliricicola porcelli*, a chewing louse of guinea pigs, is one of three species of lice infesting guinea pigs. Photo courtesy of Dr. Yoko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Bovicola* spp. (Figs. 5.89)

Common name: Chewing louse.

**TAXONOMY:** Insect (Order Phthiraptera, mallophagan or chewing lice); formerly *Damalinia* spp.

**HOST:** *Bovicola bovis* (cattle), *B. ovis* (sheep), *B. caprae* (goats), *B. breviceps* (camelids).

**Geographic Distribution:** Worldwide.

**Location on Host:** In general, preferred sites include the neck, shoulder, and back, but lice can be found anywhere on the body.

**Life Cycle:** Like the sucking lice, all stages of chewing lice are found on the host, and transmission is by direct contact with an infested animal or fomites.

**Laboratory Diagnosis:** Infested animals should be closely observed for nits and lice, although *Bovicola* spp. are only a few millimeters in length and may be difficult to see. A fine-toothed comb may be useful in recovering lice, and hair coat brushings can also be examined with a magnifying glass.

**Clinical Importance:** These common external parasites cause pruritus and dermatitis and are associated with production losses and secondary infections in heavy infestations.

**PARASITE:** *Werneckiella equi* (Figs. 5.90 and 5.91)

Common name: Chewing louse.

**TAXONOMY:** Insect (Order Phthiraptera, mallophagan or chewing lice); formerly *Damalinia equi*.

**HOST:** Horses.

**Geographic Distribution:** Worldwide.

**Location on Host:** Preferred sites include the head, mane, and base of tail.

**Life Cycle:** Transmission is by direct contact with an infested horse, shared grooming supplies, shared tack, or other fomites. All stages are found on horses.

**Laboratory Diagnosis:** Lice and associated eggs (nits) are usually identified by close visual examination. A fine-toothed comb may be useful in recovering lice, and hair coat brushings can also be examined with a magnifying glass.

**Clinical Importance:** *Werneckiella equi* is very allergenic and may cause pruritus, hyperkeratosis, and alopecia. Heavy infestations are most often seen in horses in poor condition and those rarely treated with parasite control products; weight loss and self-trauma may occur.



Fig. 5.89 Like other chewing lice, the head of *Bovicola* spp. is broader than the thorax. Pictured here is the bovine parasite *Bovicola bovis*. Photo courtesy of Dr. Robert Ridley, College of Veterinary Medicine, Kansas State University, Manhattan, KS.



Fig. 5.90 Chewing lice (*Bovicola equi*) in the hairs of a horse.



Fig. 5.91 Lesions on the shoulder and neck of a horse with a heavy burden of chewing lice. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

**PARASITE:** *Trichodectes canis* (Fig. 5.93)

**Taxonomy:** Insect (Order Phthiraptera, mallophagan or chewing lice).

**Host:** Dogs and other canids.

**Geographic Distribution:** Worldwide. Another species of chewing louse, *Heterodoxus spinigera*, may also be found on dogs in tropical and subtropical areas.

**Location on Host:** Predilection sites for *T. canis* are the head, neck, and tail, but lice will be found throughout the hair coat in heavy infestations.

**Life Cycle:** Transmission is by direct contact or fomites. Louse eggs are adhered to host hairs.

**Laboratory Diagnosis:** Recovery and identification of lice. Dogs can be infested with the chewing lice *Trichodectes* and *Heterodoxus*, as well as the sucking louse, *Linognathus setosus*.

**Clinical Importance:** Large infestations of lice cause pruritus and poor hair condition. *Trichodectes* is the most common dog louse seen in the United States and can act as an intermediate host for the tapeworm *Dipylidium caninum*.

**PARASITE:** *Felicola subrostratus* (Figs. 5.92, 5.94–5.95)

**Taxonomy:** Insect (Order Phthiraptera, mallophagan or chewing lice).

**Host:** Cats.

**Geographic Distribution:** Worldwide.

**Location on Host:** Predilection sites for *F. subrostratus* include the face, back, and ears although lice can be found anywhere on the body in heavily infested cats.

**Life Cycle:** Transmission is by direct contact or fomites. Louse eggs are adhered to host hairs.

**Laboratory Diagnosis:** *Felicola* is the only louse infesting cats, although infestations on cats may be difficult to diagnose because of the effective grooming habits of the host. Infestations are rare in pet cats but may be seen in kittens or adults in poor condition.

**Clinical Importance:** Pruritus is variable. When present, self-excoriation from scratching can lead to trauma, alopecia, and crust formation.



**Fig. 5.92** White louse eggs (nits) can be seen attached to the hairs of this infested kitten. Photo courtesy of Dr. Jeffrey F. Williams, Vansen HaloSource, Inc., Redmond, WA.



Fig. 5.83 *Trichodectes canis* is the canine chewing louse.



Fig. 5.84 The head of *Felicola subrostratus*, the feline chewing louse, is notched at the tip. Photo courtesy of Dr. Heather Walden, College of Veterinary Medicine, University of Florida, Gainesville, FL.

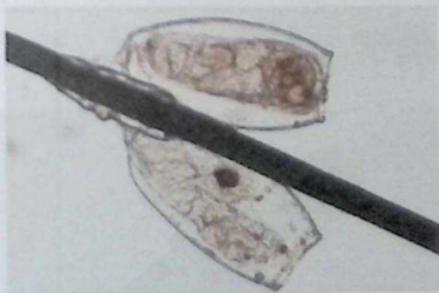


Fig. 5.85 Eggs (nits) of *Felicola subrostratus* adhered to cat hair. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE:** Avian Lice (Figs. 5.96-5.100)

**Taxonomy:** Insects (Order Phthiraptera, mallophagan or chewing lice).

**Host:** Lice are common external parasites of birds, and multiple louse species may be found occupying different niches on the same host species. All avian lice are mallophagan or chewing lice.

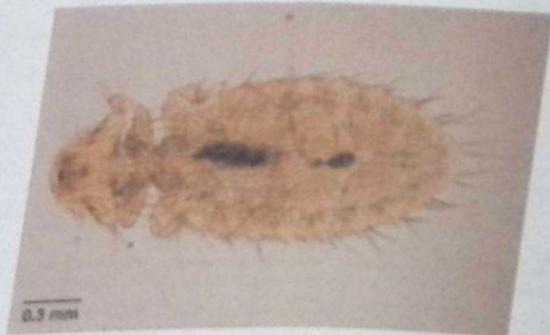
**Geographic Distribution:** Worldwide

**Location on Host:** Lice can be found on all areas of birds, with different species showing specific predilection sites.

**Life Cycle:** Similar to other lice; the entire life cycle is spent on the host, and transmission is by direct contact or fomites.

**Laboratory Diagnosis:** Detection of lice and eggs on the host with morphologic identification of lice.

**Clinical Importance:** Heavy infestations may cause loss of condition. Sick or malnourished birds often carry large numbers of lice.



5.96 There are more than 700 species of avian lice, all of which are chewing lice. Morphology of the chewing lice of birds is widely variable. Shown here is *Monopon gallinae*, the shaft louse of chickens.



*caponis*, the wing louse of poultry.

Fig. 5.96  
courtesy of E

Fig. 5.100  
Columb  
College of Veterinar

DIAGNOSIS OF ARTHROPOD PARASITES



Fig. 5.98 *Laemobothrion* sp. from an eagle. Photo courtesy of Dr. Manigandan Lejeune, Animal Diagnostic Center, Cornell University, Ithaca, NY.



*Colletes columbae*, the slender pigeon louse, on the flight feathers of a pigeon. Photo courtesy of Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



*Columbae*, the slender pigeon louse. Photo courtesy of Dr. Heather Walden, University of Florida, Gainesville, FL.

## Fleas (Order Siphonaptera)

Fleas are laterally compressed, wingless insects, typically medium to dark brown in color. Unlike many insects, they do not demonstrate clear delineations between body regions (head, thorax, and abdomen). The sucking mouthparts are usually seen protruding from the ventral aspect of the head. The three pairs of legs originate on the thorax. The enlarged third pair of legs facilitates the incredible jumping potential of these insects. In veterinary medicine, fleas are most often encountered on dogs and cats; however, they also live on a variety of other animals, including birds. Fleas are more likely than lice to move onto a different host species if the preferred host is unavailable, although they may leave after obtaining a blood meal. The adult flea is the only parasitic stage of the life cycle. The egg, larva, and pupa are found in the environment.

Fleas can be seen with the unaided eye (Fig 5.101). They are most easily collected from the host by using a quick-acting insecticide and then removing individual fleas from the hair coat or combing them out with a flea comb. On birds, fleas may be attached to the unfeathered portions of the host and can be removed with forceps. Fleas should be placed in 70% ethanol for fixation and storage. They may be cleared in 5% potassium hydroxide or mounted in Hoyer's solution to allow visualization of reproductive structures used in identification. The presence of combs on the cheek (genal comb) or at the back of the first thoracic segment (pronotal comb), the shape of the head, and host preference are important characteristics used in identification. Figure 5.102 is a key for identification of some common species in North America. In many cases, a specialist is required for specific identification. When submitting fleas for identification, it is best to submit as many individuals as possible to ensure that both sexes are represented and that structures damaged on one individual are still intact on another.



**Fig. 5.101** Puppy infested with fleas. This severe level of infestation causes anemia, especially in young animals and small breeds of dogs. Photo courtesy of Dr. Stephen Jones, Lakeside Animal Hospital, Moncks Corner, SC.

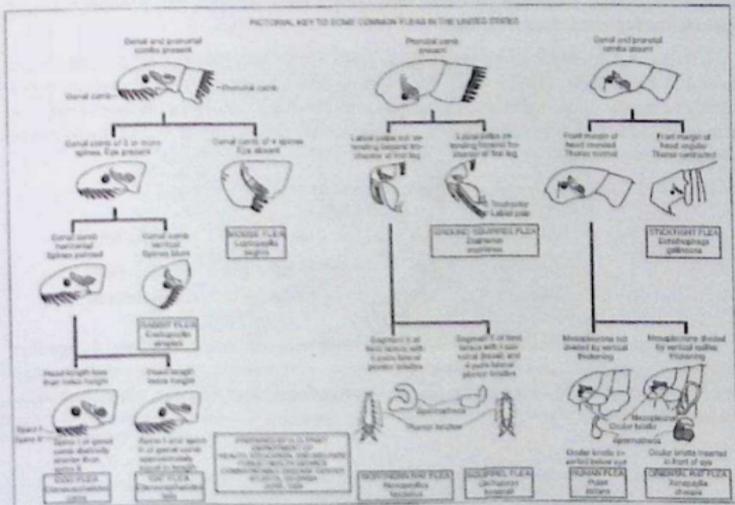


Fig. 8-100. Key to common flea species in the United States. Courtesy of U.S. Public Health Service, CDC.

**PARASITE:** *Ctenocephalides felis felis* (Figs. 5.103–5.105)

Common name: Cat flea.

**Taxonomy:** Insect (order Siphonaptera). *Ctenocephalides felis felis* is the most common flea that infests dogs and cats. A similar species, *Ctenocephalides canis*, also occurs but is less common than *C. felis felis* on both dogs and cats in the United States.

**Host:** Cats, dogs, ferrets, and numerous small and medium-sized wild mammals. Occasionally, populations may adapt to living on confined animals, such as goats or calves in a barn.

**Geographic Distribution:** Worldwide. *Ctenocephalides* is the most common flea of dogs and cats.

**Location on Host:** Fleas can be found throughout the hair coat, but predilection sites include the tail head, neck, and flanks.

**Life Cycle:** Adult cat fleas seldom leave the host. Females deposit eggs that fall off the host and hatch in the environment. Flea larvae feed on organic debris and adult flea feces and pupate in the environment. Adults are stimulated to emerge from the pupa by vibration and mechanical compression. Under optimum conditions, the life cycle can be completed in about 2 weeks.

**Laboratory Diagnosis:** Cat fleas have both pronotal and genal combs.

Size:	Adult female	approximately 2.5 mm in length
	Adult male	approximately 1 mm in length
	Larva	approximately 5 mm in length

**Clinical Importance:** Low levels of flea infestation may be mildly pruritic. Large flea populations can produce severe pruritus, alopecia, and anemia. Animals that develop flea-bite hypersensitivity may suffer severe dermatologic disease even when flea numbers are very low.



Fig. 5.105 Female (left) and male (right) *Ctenocephalides felis*. The cat flea is the most common flea found on both dogs and cats. It is difficult to differentiate from the less common *Ctenocephalides canis*. Characteristics helpful in identifying this genus are the genal and pronotal combs. Male cat fleas are smaller than females. Photos courtesy of Dr. Byron Blagburn, College of Veterinary Medicine, Auburn University, Auburn, AL.



Fig. 5.104 *Ctenocephalides* spp. eggs (arrow) are about 0.5 mm long. Larvae and pupae (covered with sand grains) of the cat flea are also shown in this figure. The surface of the pupa is sticky and becomes camouflaged with environmental debris. Photo courtesy of Dr. Byron Blagburn, College of Veterinary Medicine, Auburn University, Auburn, AL.



Fig. 5.106 Pet owners may find larvae of the cat flea, *C. felis felis*, in their homes and present them for identification. Mature larvae are about 5 mm long. The two "anal stuts" projecting from the posterior end of the body (arrow) are distinctive. Photo courtesy of Dr. Byron Blagburn, College of Veterinary Medicine, Auburn University, Auburn, AL.

**PARASITE:** *Pulex* spp. (Fig. 5.106)

Common name: Human flea.

**Taxonomy:** Insect (order Siphonaptera). *Pulex irritans* and *P. simulans* are closely related species found on dogs and cats in the New World.

**Host:** Humans, pigs, dogs, cats.

**Geographic Distribution:** Worldwide.

**Location on Host:** General distribution on the host.

**Life Cycle:** Like *C. felis felis*, only adults are found on the host; other stages are present in the environment.

**Laboratory Diagnosis:** Adult *Pulex* spp. have no genal or pronotal combs and possess an ocular bristle below the eye. Female *P. irritans* and *P. simulans* have no morphologically distinguishing features and cannot be identified to species morphologically.

**Clinical Importance:** Worldwide, *P. irritans* is found more often on swine than on humans or cats and dogs. Heavy flea infestations can produce intense irritation and pruritus. *Pulex irritans* is uncommon in North America, although it has been recorded from most states in the United States. *Pulex* spp. can serve as a vector of *Yersinia pestis*, the bubonic plague bacillus, and *Rickettsia typhi*, the agent of murine typhus.

**PARASITE:** *Echidnophaga gallinacea* (Fig. 5.107), *Ceratophyllus* spp.

Common name: Sticktight flea, European chicken flea.

**Taxonomy:** Insects (order Siphonaptera).

**Host:** *Echidnophaga gallinacea*, the sticktight flea, is found on poultry, wild birds, and occasionally on dogs, cats, and other animals. *Ceratophyllus* spp. are parasites of wild and domestic birds, especially chickens.

**Geographic Distribution:** Worldwide. *Echidnophaga* is found primarily in tropical and subtropical regions of the New World.

**Location on Host:** The sticktight flea is usually found on nonfeathered skin on the head, comb, and wattles of poultry.

**Life Cycle:** The life cycle of poultry fleas is similar to other fleas, but female *Echidnophaga* attach permanently to the head of the host. Eggs are deposited either onto the ground or into the sore created at the attachment site.

**Laboratory Diagnosis:** Identification of adult fleas. Female *Echidnophaga* can be identified by their location and attachment. This species also lacks genal and pronotal combs and has a sharply angled head. *Ceratophyllus* has a pronotal comb.

Size:	Female <i>Echidnophaga</i>	approximately 2 mm in length
	<i>Ceratophyllus</i>	approximately 4 mm in length

**Clinical Importance:** Sticktight fleas may cause severe irritation by their attachment to birds and other hosts. Heavy infestations can produce anemia. In wild birds, attachment near the eyes may lead to blindness and death. Heavy infestations of *Ceratophyllus* have been associated with anemia, restlessness, and decreased production.

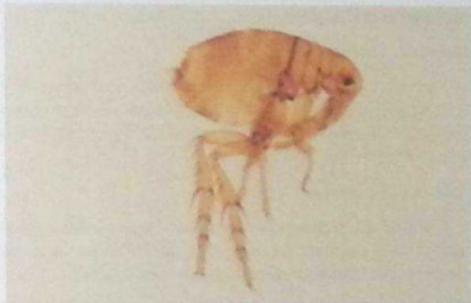


Fig. 5.126 The human flea, *Pulex irritans*, is less common on people in industrialized countries. It may also be found on companion animals and pigs. The absence of genal and pronotal combs and location of the ocular bristle below the eye are helpful in identification.



Fig. 5.127 *Echidnophaga gallinacea*, the sticktight flea, has no combs and a sharply angled head. Because this flea attaches to its host, it is most often submitted as a tick for identification.



**PARASITE:** Fleas of Rodents and Rabbits (Figs. 5.108 and 5.109)

**Taxonomy:** Insects (order Siphonaptera).

**Host:** A variety of species parasitize rodents and rabbits, including *Xenopsylla cheopis* (oriental rat flea), an important vector of bubonic plague. *Xenopsylla cheopis* will also readily feed on dogs, cats, humans, and other animals when normal rodent hosts are unavailable.

**Geographic Distribution:** Worldwide.

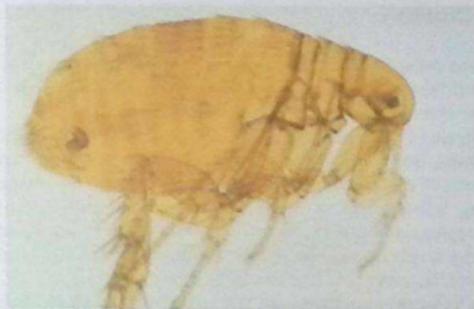
**Location on Host:** General distribution on the host. *Spilopsyllus cuniculi*, a flea of rabbits, attaches to the skin in the ears for long periods of time in a manner similar to *Echidnophaga gallinacea*, the sticktight flea of birds (see below).

**Life Cycle:** Similar to other fleas. Some species of rodent flea only visit the host to feed, unlike the common cat flea, which is resident on the host as an adult.

**Laboratory Diagnosis:** Rodent and rabbit fleas are identified based on morphologic characteristics.

**Clinical Importance:** *Xenopsylla cheopis* and some other rodent fleas are vectors of bubonic plague, caused by *Yersinia pestis*, and murine typhus, caused by *Rickettsia typhi*. Because the bacteria interfere with normal flea feeding, fleas rapidly move from host to host, spreading infection. Other rodent fleas can transmit tapeworms, trypanosomes, and myxomatosis to rabbits.





**Fig. 5.100** *Xenopsylla cheopis*, the oriental rat flea. Genal and pronotal combs are absent like *Pulex* spp.; however, the ocular bristle for *X. cheopis* originates in front of the eye. Fleas belonging to several genera are found on rodents and rabbits. Photo courtesy of Dr. Byron Biagburn, College of Veterinary Medicine, Auburn University, Auburn, AL.



**Fig. 5.101** *Cediopsylla simplex*, a rabbit flea. Photo courtesy of Dr. David Baker, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA.

## Flies (Order Diptera)

Flies belong to the order Diptera, and some species feed on animals in the larval or adult stage. Larval flies parasitizing animals are referred to as either "bots" ("grubs" and "warbles" are synonymous terms) or "maggots."

Bots are obligate parasites that develop in various internal locations in the host; adults are free-living, non-feeding, bee-like flies. Mature bot larvae are barrel-shaped and often have rows of spines on the body. The genus of a bot or maggot can be identified by examining the pattern of the spiracular plates surrounding the breathing holes (spiracles) on the posterior end (Fig. 5.110). Spiracles are best examined by positioning the bot on a small amount of modeling clay or sand so that the structures can be examined from the posterior view.

Maggots are fly larvae associated with "fly strike" or "fly blow." In most cases, they are opportunistic parasites. Adult flies attracted to wounds or hair stained with feces or blood lay their eggs on the animal, and larvae feed on debris and necrotic tissue. Maggots are more elongated than bots and are narrower at the anterior than at the posterior end. They are usually present on the surface of the body in conjunction with wounds or hair soiled with feces, urine, blood, or other organic material.

Parasitic adult flies are usually blood feeders. Hematophagy (blood-feeding) has evolved multiple times in flies and a diverse array of diptera feed on blood.

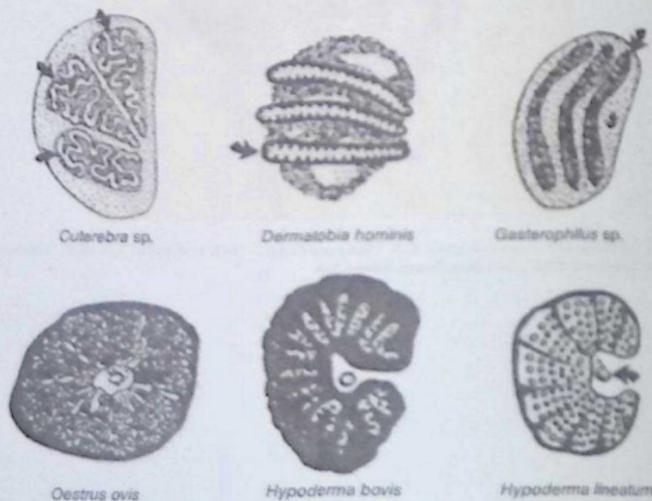


Fig. 5.110 Posterior spiracles of bot fly larvae. Row 1: *Cuterebra* spp. spiracles are each divided into several plates. *Dermatobia hominis* spiracles are sunken in a deep cavity, and *Gasterophilus* spp. spiracles are curved and in a shallow cavity. Row 2: Spiracles of *Oestrus ovis* have a central button that is completely surrounded, whereas *Hypoderma* spp. spiracles have either a narrow opening (*H. bovis*) or a wider opening (*H. lineatum*). Adapted from Pictorial Keys to Arthropods, Centers for Disease Control and Prevention, 1969.



Fig. 5.111 Paired spiracle plates on the posterior end of a bot larvae (*Gasterbra* sp.) Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



Fig. 5.112 The hairy body of adult warble (bot) flies makes them look more like bees than flies. The presence of adult warble flies, like this *Hypoderma bovis*, is distressing to potential hosts, which will actively try to avoid the flies. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA, and Dr. Jerry Weintraub, Agriculture Canada.

**PARASITE:** *Cuterebra* spp. (Figs. 5.113–5.115)

Common name: Rodent bot fly.

**Taxonomy:** Insect (order Diptera).

**Host:** Rodents and rabbits are the principal hosts. Dogs, cats, and rarely humans may also be infected.

**Geographic Distribution:** Western Hemisphere.

**Location on Host:** Larvae are found in subcutaneous cysts in various locations. In dogs and cats, they are seen most often on the head and neck.

**Life Cycle:** Adult flies lay eggs around rodent holes. Larvae crawl onto animals and enter through facial orifices. Following migration to subcutaneous sites, the *Cuterebra* larva forms a visible nodule with an external breathing hole. After completing development, the larva emerges and pupates on the ground.

**Laboratory Diagnosis:** *Cuterebra* larvae can be preliminarily identified by location on the host. Confirmation requires examination of larval spiracular plates.

**Clinical Importance:** The presence of one or two bots in most hosts is not associated with clinical problems. However, abnormal migration to the nervous system or other tissues occasionally occurs, resulting in disease.



Fig. 8.113 Bot larvae (*Cuterebra* sp.) spiracles visible through patent opening into dermal cyst in an infested cat.



Fig. 8.114 *Cuterebra* larvae are about 2.5 cm in length and covered with spines when fully developed. Spiracles are present on the posterior end (left) and paired hooks are evident on the anterior end (arrow).



Fig. 8.115 Veterinary practitioners occasionally remove young *Cuterebra* larvae from animals and, because of their small size and white color, may have difficulty recognizing them as *Cuterebra*. Starting at the left, this figure shows larvae at 6, 8, 10, and 13 days after infestation of the host. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.

**PARASITE:** *Dermatobia hominis* (Fig. 5.116)

Common name: Human bot fly.

**Taxonomy:** Insect (order Diptera).

**Host:** Cattle, humans, dogs, other domestic and wild animals.

**Geographic Distribution:** Central and South America.

**Location on Host:** Various subcutaneous sites.

**Life Cycle:** Adult female *Dermatobia* glue clusters of eggs to various muscid flies and mosquitoes. When these organisms visit a host, the *Dermatobia* larvae are deposited and enter the subcutaneous tissue, where each larva develops in a nodule with a hole in the skin through which respiration occurs. Larvae leave the host to pupate.

**Laboratory Diagnosis:** Identification of larvae is based on shape and pattern of the spiracles.

**Clinical Importance:** Although best known as a human parasite, *D. hominis* is primarily a cattle pest.



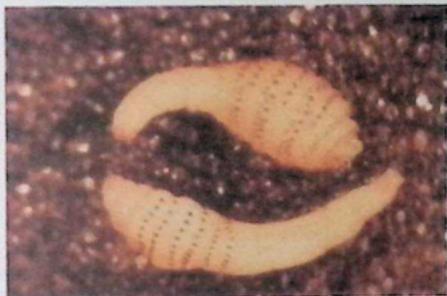


Fig. 5.116 *Dermatiobia hominis* bots are often seen in the second-instar larval stage. At this stage, they have a distinctive narrow, spineless posterior end that becomes less prominent as they continue development. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.

**PARASITE:** *Gasterophilus* spp. (Figs. 5.117–5.120)

Common name: Stomach bot.

**TAXONOMY:** Insect (order Diptera). Species include *G. intestinalis*, *G. nasalis*, *G. haemorrhoidalis*, and *G. pecorum*.

**Host:** Horses and other equids.

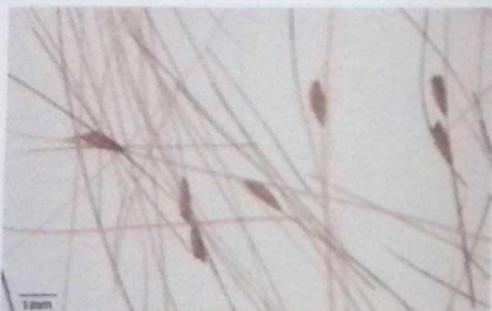
**Geographic Distribution:** Worldwide.

**Location on Host:** Equine stomach (*G. intestinalis*), small intestine (*G. nasalis*), or rectum (*G. haemorrhoidalis*). Developing stages may be found in the oral cavity.

**Life Cycle:** Most *Gasterophilus* spp. deposit eggs on the legs or face of horses. After hatching, larvae enter through the mouth and spend a period of development in the tongue and gums before moving to the stomach. After a period of 8–11 months in the stomach, bots pass out in the feces and pupate on the ground.

**Laboratory Diagnosis:** Bots are usually identified by presence in the stomach or intestine at necropsy, but they may also be seen in the feces at different stages of development following treatment of the host with macrocyclic lactone anthelmintics. They can be recognized as bots by their barrel shape and rows of spines. Eggs (nits) may be evident attached to the hairs of the legs or face.

**Clinical Importance:** Horses appear to tolerate small to moderate burdens of bots. In rare instances, humans in close contact with horses are infested and develop a transient dermatitis usually occurring on the face.



**Fig. 5.117** Eggs (nits) of *Gasterophilus* adhered to horse hair. Photo courtesy of Dr. Heather Welden, College of Veterinary Medicine, University of Florida, Gainesville, FL.



Fig. 5.118 Eggs of *Gasterophilus intestinalis*, the most common equine bot species, can be seen attached to the hairs of the forelegs. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.

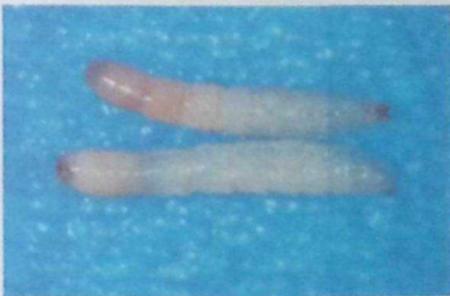


Fig. 5.119 Second-instar *Gasterophilus* larvae. Following treatment with a macrocyclic lactone anthelmintic or other effective drug, young *Gasterophilus* larvae may also be present in manure. Although the less mature larvae lack the barrel shape of mature bots, they still show the distinctive rows of spines around each segment. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.



Fig. 5.120 Equine stomach bot, *Gasterophilus*. Species can be distinguished based on patterns of spines on the body, but species identification is unnecessary for control and treatment of the parasite. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE:** *Hypoderma bovis*, *H. lineatum* (Figs. 5.121 and 5.122)

Common name: Cattle grub, ox warble.

**Taxonomy:** Insect (order Diptera).

**Host:** Cattle are the normal hosts; horses and goats are occasionally infested. Other species of *Hypoderma* parasitize deer and reindeer, and *Przhevalskiana silenus* is a similar parasite of goats in the Mediterranean region.

**Geographic Distribution:** North America, Europe, Asia.

**Location on Host:** Visible nodules with an external opening appear on the backs of infested animals.

**Life Cycle:** Adult flies deposit eggs on cattle. After hatching, larvae penetrate through the skin and migrate to sites either along the esophagus (*H. lineatum*) or in the tissue surrounding the spinal cord (*H. bovis*). After a period of development lasting several months, the larvae migrate to the host's back and form subcutaneous nodules with a breathing hole. After several more months of development, larvae emerge, pupate on the ground, and adult flies are formed.

**Laboratory Diagnosis:** Diagnosis can usually be made based on parasite location in the host and history.

**Clinical Importance:** Damage from *Hypoderma* comes from several sources. Adult fly activity associated with oviposition worries cattle and may interfere with grazing; migrating larvae cause necrotic tracks in muscle tissue and the hide is damaged for leather production. In addition, if larvae die while along the esophagus or spinal cord, serious inflammatory reactions can lead to bloat or paralysis.

**PARASITE:** *Oestrus ovis* (Fig. 5.123), *Rhinoestrus* spp.

Common name: Nasal bot.

**Taxonomy:** Insects (order Diptera).

**Host:** *Oestrus ovis* larvae are found in sheep and goats. *Rhinoestrus* larvae are found in horses. Other species parasitize deer and camels (e.g., *Cephalopina titillator*).

**Geographic Distribution:** *Oestrus ovis* is found worldwide. *Rhinoestrus* infestation occurs in Africa, Europe, and Asia.

**Location on Host:** Nasal passages and sinuses.

**Life Cycle:** Adult female flies deposit larvae in or near the nasal passages. Larvae develop in nasal passages and sinuses. When development is complete, they fall out of the nose and pupate on the ground.

**Laboratory Diagnosis:** Nasal bots are not usually presented for identification because of their location, but they are sometimes found by owners in water troughs or on the ground after they exit the host. Specific identification is made by examination of the spinacular plates.

**Clinical Importance:** Although infested animals show increased levels of nasal discharge, small or moderate numbers of bots are usually well tolerated. Sheep will attempt to avoid ovipositing female flies by keeping their muzzles near the ground or under available shelter (buildings, cars, one another, etc.). Bots occasionally wander to abnormal sites, where they may cause serious disease.



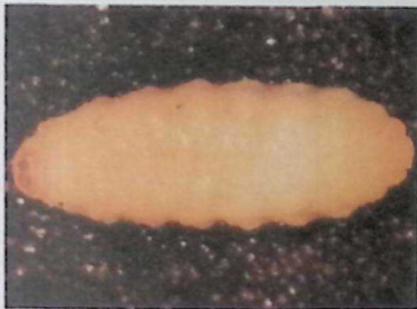


Fig. 5.121 Early third-instar cattle grub, *Hypoderma lineatum*. Grubs or warbles can be distinguished from fly maggots by their barrel-shaped bodies and typical location in the host. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.



Fig. 5.122 *Hypoderma* sp. grub emerging from its subcutaneous location on the back of the bovine host. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.



Fig. 5.123 Ovine nasal bot, *Oestrus ovis*. These bots are occasionally seen by producers when they leave the small ruminant host to pupate or following treatment. Photo courtesy of Dr. Philip Scholl, Agricultural Research Service, USDA.

**PARASITE:** Fly Strike or Blow Flies (Figs. 5.124–5.127)

**Taxonomy:** Insects (order Diptera). Many of the maggots causing fly strike belong to the families Calliphoridae (blow flies) and Sarcophagidae (flesh flies).

**Host:** Various, not host-specific.

**Geographic Distribution:** Worldwide.

**Location on Host:** Various, wherever there is blood or other body secretion that attracts female flies.

**Life Cycle:** Larvae of most of the flies in this group are not obligatory parasites. Adults are attracted by the odors of decaying organic material and deposit their eggs on carrion. Wounds or skin on animals soiled with blood or feces may also attract the flies. Larvae feed primarily on necrotic material, and when development is completed, they leave the host and pupate in the environment.

**Laboratory Diagnosis:** Recognition of maggots on animals is sufficient for diagnosis of fly strike. Specific identification of fly larvae requires examination of larval spiracles.

**Clinical Importance:** "Fly blown" animals can be seriously affected by fly larvae. The presence of large numbers of maggots may produce tissue destruction, toxemia, and even death.



Fig. 5.124 Posterior spiracles of maggots associated with fly strike. Adapted from Pictorial Keys to Arthropods, Centers for Disease Control and Prevention, 1969.



Fig. 5.125. A case of ovine "fly strike" or "fly blow" in which an animal has become infested with fly maggots. In most cases, these flies are not true parasites and would be equally attracted to carrion. Sheep are particularly susceptible to fly strike in warm weather if wool is persistently wet or becomes soiled with blood or feces. Photo courtesy of Dr. Dwight Bowman, College of Veterinary Medicine, Cornell University, Ithaca, NY.



Fig. 5.126. Posterior spiracles of *Lucilia* spp. larva. Photo courtesy of Kellee Sundstrom, Oklahoma State University, Stillwater, OK.



Fig. 5.127. *Lucilia* spp. is one of the genera of blow flies that cause facultative myiasis (fly strike). Blow flies are typically metallic blue, green, or bronze in color. *Lucilia* spp. infestations of sheep are a source of significant economic loss to the Australian sheep industry. Photo courtesy of Dr. Nick Sangster, Charles Sturt University, NSW and Ms. Sally Pope, University of Sydney, NSW, Australia.

**PARASITE:** *Cochliomyia hominivorax*, *Chrysomya bezziana* (Figs. 5.128–5.130)

Common name: Screwworm.

**Taxonomy:** Insects (order Diptera). Screwworms belong to the blow fly family (Calliphoridae). Several species of sarcophagid flies are also obligatory parasites but are of less importance.

**Host:** Wild and domestic animals, humans.

**Geographic Distribution:** *Cochliomyia hominivorax*, the New World screwworm, is found in South America. The Old World screwworm, *Chrysomya bezziana*, occurs in Africa, India, and Southeast Asia.

**Location on Host:** Various, often on body openings or the edges of wounds.

**Life Cycle:** Adult female flies deposit eggs on the host. Larvae feed invasively on living tissue. Following completion of development, larvae fall to the ground and pupate. Adults emerge and mate. The entire life cycle can be completed in as little as 24 days.

**Laboratory Diagnosis:** Examination of larval spiracles is important for larval identification. The larvae also have two posterior tracheal trunks, which look like dark lines extending anteriorly from the posterior end. Although screwworm has been eradicated from the United States, reintroduction is possible. If screwworm infestation is suspected, larvae should be collected in 70% alcohol and submitted to federal or state veterinarians for identification.

**Clinical Importance:** Screwworm infestation is a serious disease that can rapidly lead to the death of the host. The parasite was eradicated from the United States in the 1960s through a sterile-male release program. This program has now been successful in removing the fly from Mexico and Central America, with only occasional outbreaks still reported.



Fig. 8.128 Screwworm infestation on the ear of a calf. If untreated, these infestations are often fatal. Photo courtesy of Dr. Donald B. Thomas, USDA Subtropical Agriculture Research Laboratory, Weslaco, TX

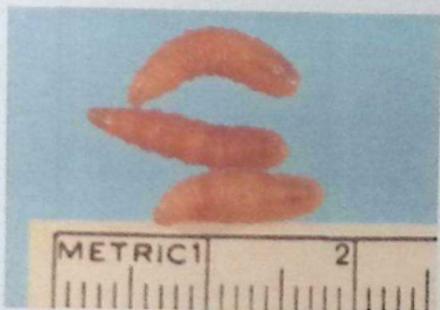


Fig. 8.129 Screwworm maggots. If screwworm infestation is suspected in the United States, larvae should be submitted in 70% alcohol to state or federal veterinarians.



Fig. 8.130 *Cochliomyia* sp. larvae showing pigmented tracheal trunks (arrow). If screwworm infestation is suspected in the United States, larvae should be submitted in 70% alcohol to state or federal veterinarians. Photo courtesy of Kellee Sundstrom, Oklahoma State University, Stillwater, OK.

**PARASITE:** Louse Flies, including *Melophagus ovinus* (Figs. 5.131–5.133)

Common name: Sheep ked, louse fly.

**Taxonomy:** Insects (order Diptera).

**Host:** Sheep and goats are parasitized by *M. ovinus*, the sheep ked. Louse flies may be found on a variety of animals. Examples include *Hippobosca variegata* on horses, cattle, and camels; *Lipoptena* spp. on wild ruminants; *Hippobosca longipennis* on dogs; and *Pseudolynchia* spp. on birds.

**Distribution:** Worldwide, although in North America, only the sheep ked and the pigeon fly (*Pseudolynchia*) are found on domestic animals.

**Location on Host:** Various.

**Life Cycle:** In some species, winged adult flies are temporary parasites while feeding; in others, louse flies lose their wings after finding a host and become permanent parasites. The sheep ked, *Melophagus*, has no functional wings and is transmitted only by direct contact of hosts. Adult female sheep keds deposit larvae that pupate immediately on the skin of the host.

**Laboratory Diagnosis:** Identification is based on adult flies. Hippoboscid flies generally have a rather flat, leathery appearance compared with other flies. Ked feces, which resemble flea feces, may be found on the host.

**Clinical Importance:** Sheep keds can cause significant damage to the skin, making it unsuitable for leather production, and can also reduce the value of the sheep fleece. Biting activity of flies causes pain and irritation.



Fig. 6.131 Typical hippoboscids (horse flies) showing the flattened appearance of the body with relatively large legs and wings. Some species of hippoboscids retain their wings, others lose their wings after finding a host, and still others are wingless throughout their development. Photo courtesy of Dr. Alvin Gajadhar, Centre for Animal Parasitology, CFIA, Saskatoon, Saskatchewan, Canada.



Fig. 6.132 Adult and pupal stages of the sheep ked, *Melophagus*. Although sometimes mistaken for ticks, these organisms are insects with six legs and three main body parts. Ked excrement, which resembles flea feces, can also be found on the host and is helpful in diagnosis. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



Fig. 6.133 Adult *Lipoptena cervi* from a moose. Alopecia is reported in moose due to massive *L. cervi* infestations. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

**PARASITE: Biting Flies (Figs. 5.134–5.141)**

**COMMON NAMES:** Horse fly, deer fly, mosquito, black fly, sand fly, tsetse fly, horn fly, stable fly, midge, and so on.

**TAXONOMY:** Insects (order Diptera). Biting flies belong to many families within the order.

**Host:** Domestic and wild animals and humans.

**Geographic Distribution:** Worldwide.

**Location on Host:** Various, although many biting flies have predilection sites.

**Life Cycle:** Like other Diptera, biting flies lay eggs that hatch into larvae. Pupation follows a period of larval development, followed by the emergence of adult flies. In some biting fly species, like the mosquito, only the females are blood feeders. In other species, adults of both sexes feed on blood.

**Laboratory Diagnosis:** Although detailed descriptions of biting flies are not within the scope of this book, some generalizations can be made that allow basic identification (see figures).

**Clinical Importance:** Biting flies have enormous importance in veterinary medicine and public health because of their role as disease vectors. In addition, the activity of biting flies causes irritation, and their bites can lead to allergic dermatitis and, in some cases of massive fly attacks, toxemia, and death.



**Fig. 5.184** Tabanid flies. These large biting flies are familiar worldwide. Horse flies (*Tabanus* spp., pictured) may reach 2.5 cm in length. Photo courtesy of Dr. Jeffrey F. Williams, Vanson HaloSource, Inc., Redmond, WA.



**Fig. 5.185** Deer flies, *Chrysops*, also belong to the tabanid group but are smaller and have distinct bands on their wings. Photo courtesy of Kellee Sundstrom, Oklahoma State University, Stillwater, OK.



**Fig. 5.186** Head and mouthparts of *Stomoxys calcitrans*, the stable fly (left), and *Haematobia irritans*, the horn fly (right). The mouthparts of *Stomoxys* project from the head at a right angle and the lateral aspect of the eye has a concave shape (arrow). Photos courtesy of Kellee Sundstrom, Oklahoma State University, Stillwater, OK.



Fig. 5.137 Horn flies, *Haematobia irritans*, are a major pest of cattle throughout the world. Horn flies often cluster on the backs and sides of cattle, heads pointing toward the ground. If disturbed, they fly a short distance into the air and rapidly settle on the host again. Photo courtesy of Kellie Sundstrom, Oklahoma State University, Stillwater, OK.



Fig. 5.138 The stable fly, *Stomoxys calcitrans*, is approximately the size of a house fly but is a major blood-feeding pest of cattle and, sometimes, domestic dogs. Photo courtesy of Dr. Lyle Buss, Entomology and Nematology Department, University of Florida, Gainesville, FL.



Fig. 5.139 *Glossina*, the tsetse fly, is the vector of trypanosomiasis in domestic animals and humans in Africa. Species reach up to 14 mm in length. Like stable flies, the mouthparts of the fly project forward from the head, but the wings of tsetse flies lie across the back like scissors. Photo courtesy of Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.



Fig. 5.542. Mosquitoes are common in many regions. Mosquito larvae develop in or near water. These delicate flies with long legs have mouthparts that are at least twice as long as the head. Photo from Agricultural Research Service, USDA.



Fig. 5.543. Calliphora (midges or no-see-ums) are very small biting flies (rarely larger than 2 mm) with patterned wings. Species are found worldwide. Photo courtesy of Kellee Sundstrom, Oklahoma State University, Stillwater, OK.

## Other Insects

**PARASITE:** Triatomine or reduviid bugs (Fig. 5.142)

Common name: Kissing bugs, conenose bugs, assassin bugs, chinchas.

**TAXONOMY:** Insect (order Hemiptera, family Reduviidae).

**Host:** Humans, wildlife, and domestic animals.

**Geographic Distribution:** Several different species of triatomine bugs are found in forested and dry areas in the southern United States, Mexico, Central America, and South America.

**Location on Host:** Hide in dark crevices during day, emerging at night to feed.

**Life Cycle:** Kissing bugs visit the host at night to feed and spend daylight hours in cracks and crevices in human or animal environments. Eggs are laid in the environment.

**Laboratory Diagnosis:** Identification of immature or adult instars of triatomine bugs from the environment around animals or people, including kennels, chicken coops, and other housing. There are more than 100 species of triatomines in the Americas; common genera include *Triatoma* and *Rhodnius*. Adults are 1.5–3 cm in length and size of most stages increases with blood feeding. Some beetles and non-triatomine reduviid bugs may be confused with kissing bugs, including wheel bugs, corsairs, and leaf-footed bugs.

**Clinical Importance:** Kissing bugs serve as vector of *Trypanosoma cruzi*, the agent of Chagas disease, to humans, dogs, and other hosts. Over 50% of kissing bugs collected in Texas are infected with *T. cruzi*. Dogs become infected when they ingest the kissing bug as well as through stercorarian transmission during bug feeding.



FIG. 5.182 Adult triatomine bug, *Triatoma sanguinipes*, an important vector of *Trypanosoma cruzi* in the southcentral United States. Photo courtesy of Megan Laneberry, Oklahoma State University, Stillwater, OK.

**PARASITE:** *Cimex lectularis*, *C. hemipterus*, *C. adjunctus* (Fig. 5.143–5.145)

Common name: Bedbug.

**Taxonomy:** Insect (order Hemiptera).

**Host:** Humans and domestic animals. Other members of the genus parasitize birds and bats.

**Geographic Distribution:** Worldwide with *C. lectularis* in temperate and *C. hemipterus* in tropical areas.

**Location on Host:** Bedbugs do not have specific predilection sites on the host.

**Life Cycle:** Five nymphal instars with the sixth stage being sexually mature, dimorphic males and females. All motile stages of bedbugs visit the host only at night to feed and spend daylight hours in cracks and crevices in human or animal environments. Eggs are laid in the environment.

**Laboratory Diagnosis:** Identification of reddish-brown, dorsoventrally flattened, wingless insects. Adults are 5–7 mm in length. In human infestations, bedbug feces may be seen in the bed.

**Clinical Importance:** Bedbugs are most important as human parasites, but they will also attack domestic animals. In the United States, bedbugs have recently become more common human parasites as the use of broad-spectrum pesticides for other insect pests has declined. The role of bedbugs as vectors for pathogens is uncertain. There is some laboratory data suggesting *Trypanosoma cruzi* may be transmitted by bedbugs. However, the epidemiological data to support vector competency and establish this transmission as a naturally occurring phenomenon is lacking.



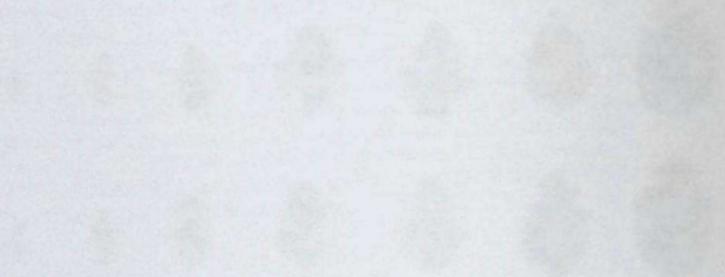
Fig. 5.140 Adult bedbug, *Cimex lectularis*. Photo courtesy of Megan Laneberry, Oklahoma State University, Stillwater, OK.



Fig. 5.144 Dorsal (top row) and ventral (bottom row) adult and nymphal bedbugs, *Cimex lectularis*. Females are slightly larger with rounded abdomens (far left). Males are smaller with a pointed abdomen (second from left). Each motile stage of *Cimex* spp. must take a blood meal before molting to the next stage. Photo courtesy of Megan Laneberry, Oklahoma State University, Stillwater, OK.



Fig. 5.145 *Cimex adjunctus*, the eastern bat bug, can be mistaken for *C. lectularis*. However, bat bugs are found in cracks and crevices of bat roosting areas, and the hairs on the thorax of a bat bug are longer than those of a bed bug. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.



# Parasites of Fish

Stephen A. Smith

Fish can serve as definitive, intermediate, or paratenic (transport) hosts in the life cycle of many species of protozoan, metazoan, and crustacean parasites. Most of these parasites can be readily identified grossly or microscopically, and as with mammalian parasites, the correct identification and an understanding of their life cycle are important in the prevention or management of an outbreak of disease due to parasites.

Protozoan parasites probably cause more disease in both ornamental and cultured fish than any other group of parasites. An example of a common protozoan disease in fish is white spot disease, or "ich," caused by *Ichthyophthirius multifiliis* in freshwater fish or by *Cryptocaryon irritans* in marine species. Other protozoan parasites that commonly occur on fish include *Tetrahymena* spp., *Trichodina* spp., *Trichophyra* spp., *Amyloodinium* spp. and *Ichthyobodo* spp.

Metazoan parasites can be found as larval or adult forms in almost every tissue of fish. Most can be grossly identified as monogeneans, digenetic trematodes, nematodes, cestodes, acanthocephalans, or crustaceans, but specific identification generally involves special staining techniques or clearing of specimens.

Examples of common fish helminths include monogeneans on the gills and skin; larval digenetic trematodes (metacercariae) in the eyes, skin, musculature, and abdominal cavity; larval cestodes and nematodes in the visceral organs and abdominal cavity; and an assortment of adult trematodes, nematodes, cestodes, and acanthocephalans in the lumen of the gastrointestinal tract. In addition, a number of arthropod parasites and leeches can be found occurring on or attached to the skin and fins of fish.

## TECHNIQUES FOR RECOVERY OF ECTOPARASITES

A variety of nonlethal techniques that include skin, fin, and gill biopsies have been developed for the diagnosis of the common external parasites of fish. Most of these biopsy techniques can be performed on live fish without the use of anesthesia, although light sedation often simplifies the procedure and makes it less stressful for the fish.



Fig. 6.1 Collection of mucus sample via a skin biopsy from the side of a fish for external-parasite examination.

### Skin Biopsy (Mucus Smear)

The skin is the primary target organ for many of the external fish parasites. Therefore, a biopsy of the skin (Fig. 6.1) is one of the most useful and common samples for diagnosing ectoparasitic problems. This biopsy is performed by gently scraping a small area on the surface of the fish with a scalpel blade or the edge of a microscope coverslip in a cranial to caudal direction. Care should be taken to use only a minimal amount of pressure to obtain this superficial scraping, since damage to the skin may result in secondary bacterial and fungal infections or osmoregulatory imbalance in the fish.

The mucus from the skin scraping should be transferred immediately to a drop of aquarium, tank, or pond water (either fresh or salt water, depending on the species of fish, but *not* city tap water or distilled water) on a glass microscope slide and a coverslip carefully applied. This wet mount should then be examined under the compound microscope for the presence of free-swimming, attached, or encysted protozoan or metazoan parasites.

### Fin Biopsy (Fin Snip)

A fin biopsy (Fig. 6.2) is obtained by snipping a small piece of tissue from the peripheral edge of one of the fins. This procedure is more traumatic to the fish than a skin biopsy, since a physical wound is produced. The fin snip should be transferred immediately to a drop of aquarium, tank, or pond water on a glass microscope slide, spread to its full extent, and a coverslip carefully applied. This wet mount should then be examined under the compound microscope for the presence of protozoan or metazoan parasites.

### Gill Biopsy (Gill Snip)

A gill biopsy (Fig. 6.3) is obtained by inserting the tip of a pair of fine scissors into the branchial cavity behind the operculum (gill cover) and cutting off the distal ends of several of the primary gill lamellae attached to the gill arch. Since only the tips of the primary lamellae are removed, minimal bleeding should occur. The gill tissue should be transferred immediately to a drop of aquarium, tank, or pond water on a glass



Fig. 6.2 Collection of a fin biopsy by clipping a small portion of the distal tip of the pectoral fin.



Fig. 6.3 Collection of a gill biopsy by lifting the operculum (gill chamber cover) and removing the distal tips of several filaments (lamellae) of the gill.

microscope slide; the individual lamellae separated; and a coverslip carefully applied. This wet mount should then be examined under the compound microscope for the presence of protozoan or metazoan parasites.

### RECOVERY OF ENDOPARASITES

Examination of fish feces for the presence of internal parasites is accomplished with the same techniques as those used for mammals and birds. A fresh fecal sample is collected with a pipette either from the bottom of the aquarium or as it hangs from the vent of the fish. If an appropriate sample cannot be acquired from the environment or if examination of a specific individual is desired, the application of gentle pressure on the sides of a netted fish often produces the desired sample. The fecal specimen is then processed by standard flotation, sedimentation, or direct smear techniques and evaluated for the presence of parasite eggs and larvae. Though specific parasite identification is generally impossible, fecal examination does provide useful information about the types of parasites (nematodes, trematodes, cestodes, acanthocephalans) that may be present in a fish.

## PARASITES OF FISH

PARASITE: *Ichthyophthirius multifiliis* (Figs. 6.4 and 6.5)

Common name: "Ich" or freshwater white spot disease.

Taxonomy: Protozoa (ciliate).

Geographic Distribution: Freshwater fish worldwide.

Location in Host: Within the surface epithelial layer of the skin, fins, and gills.

Life Cycle: This parasite has a direct life cycle, with free-swimming, ciliated tomites (theronts) in the water invading the skin, fins, and gills of fish. The tomites penetrate into the epithelial tissues and form large, feeding trophozoites (trophonts) that eventually excyst from the host and enter the water, where each develops into a cyst. The cyst stage then undergoes multiple divisions, producing numerous infective tomites that are released to the environment to infect other fishes.

Laboratory Diagnosis: This large, holotrich ciliate has a characteristic C-shaped macronucleus and is detected in wet mounts of skin biopsies and gill and fin snips.

Size:	Trophozoite in tissue	up to 1 mm in diameter
	Tomite in water	25–50 × 15–22 μm

Clinical Importance: The parasite causes small, raised, white lesions on skin, fins, and gill tissue. Penetration into and excystation out of the epithelial tissue by the parasite causes loss of integrity of external tissues and results in disruption of normal homeostatic osmoregulatory processes.

PARASITE: *Cryptocaryon irritans* (Fig. 6.6)

Common name: Marine white spot disease.

Taxonomy: Protozoa (ciliate).

Geographic Distribution: Marine and brackish water fishes worldwide.

Location in Host: Within the surface epithelial layer of the skin, fins, and gills.

Life Cycle: The life cycle is similar to that of freshwater *Ichthyophthirius multifiliis*: free-swimming, ciliated tomites in the water invade the skin, fins, and gills, penetrate into the epithelial tissues, and form large, feeding trophozoites that eventually excyst from the host into the water and develop into cysts. The cyst stage then undergoes multiple divisions, producing numerous infective tomites that are released to the environment to infect other fishes.

Laboratory Diagnosis: This holotrich ciliate, which does *not* have a C-shaped nucleus like *I. multifiliis*, is detected in wet mounts of skin biopsies and gill and fin snips.

Size:	Trophozoite in tissues	up to 1 mm in diameter
	Tomite in water	25–50 × 15–22 μm

Clinical Importance: *Cryptocaryon irritans* causes small, raised, white lesions on the skin, fins, and gill tissue. Penetration into and excystation out of host epithelial tissue by the parasite causes loss of integrity of external tissues and results in disruption of normal homeostatic osmoregulatory processes.



Fig. 6.4 *Ichthyophthirius multifiliis* trophozoite. This ciliate, which produces small, raised, white lesions on the skin, fins, and gills of fish, is commonly called "ich" or "white spot disease." This freshwater parasite varies in size, ranging from 100 to 1000  $\mu\text{m}$ , depending on the stage of maturation, available nutrition, and host species.



Fig. 6.5 Encysted trophozoite of *Ichthyophthirius multifiliis* embedded in the epithelial tissues of a gill filament of a goldfish (*Carassius auratus*).



Fig. 6.6 *Cryptocaryon irritans* trophozoite. This ciliated parasite, which has a similar life cycle to *Ichthyophthirius multifiliis*, occurs in marine fish species.

**PARASITE:** *Tetrahymena* spp. (Fig. 6.7)

**Taxonomy:** Protozoa (ciliate). Species include *T. corlissi* and *T. pyriformis*.

**Geographic Distribution:** Freshwater fish worldwide.

**Location in Host:** Trophozoites are generally found on surface epithelial layers of the skin and fins but may occasionally be found in deeper skin tissues, muscle, and abdominal organs.

**Life Cycle:** This parasite forms reproductive cysts in the freshwater environment in which two to eight infective tomites are produced.

**Laboratory Diagnosis:** Small, cylindrical- to pyriform-shaped ciliates can be found in wet mounts of skin biopsies.

Size: Trophozoite 55 × 30 μm

**Clinical Importance:** This facultative parasite can become histophagous and invade the skin, muscle, and internal organs causing osmoregulatory problems and body system dysfunction.

**PARASITE:** *Uronema* spp., including *U. marinum* (Fig. 6.8)

**Taxonomy:** Protozoa (ciliate).

**Geographic Distribution:** Marine and brackish water fishes worldwide.

**Location in Host:** Surface epithelial layers of skin and fins of marine fish, but may also invade deeper skin tissues, muscle, and abdominal organs.

**Life Cycle:** This parasite (similar to freshwater *Tetrahymena* spp.) forms reproductive cysts in the marine environment in which several infective tomites are produced.

**Laboratory Diagnosis:** Small, cylindrical- to pyriform-shaped ciliates can be found in wet mounts of skin biopsies.

Size: Trophozoite 50 × 30 μm

**Clinical Importance:** This facultative, histophagous parasite (similar to freshwater *Tetrahymena* spp.) can invade the skin, muscle, and internal organs, causing osmoregulatory problems and body system dysfunction.



Fig. 6.7 Trophozoites of *Tetrahymena* spp. in a skin biopsy of a hybrid striped bass (*Morone saxatilis* × *M. chrysops*). This small ciliate also infects many species of ornamental aquarium fish.



Fig. 6.8 Tissue section showing numerous trophozoites of *Uronema* spp. invading the deeper tissues of a summer flounder (*Paralichthys oblongus*) species. This marine organism causes pathology similar to that of *Tetrahymena* spp. of freshwater species of fish.

PARASITE: *Epistylis* spp. (Fig. 6.9)

TAXONOMY: Protozoa (ciliate). Species include *E. colisarium* and *E. lwoffi*.

Geographic Distribution: Freshwater fish worldwide.

Location in Host: Attached to surface epithelial layers of the skin and fins

Life Cycle: This sessile, peritrichous ciliate divides by binary fission along the longitudinal axis, producing two daughter cells.

Laboratory Diagnosis: This colonial parasite is detected in wet mounts of the skin and is identified by its conical or elongated cylindrical body, branched noncontractile stalk, and epistomal disk for attachment.

Size: 150–300 × 40–60 μm

Clinical Importance: The epistomal disk used for attachment to the host causes a localized skin lesion and mechanical disruption of the normal osmoregulatory process of the skin. Generally, fish develop skin lesions only in heavy infestations.

PARASITE: *Trichodina* spp. (Fig. 6.10)

TAXONOMY: Protozoa (ciliate). Related genera include *Trichodinella* spp., *Hemitrichodina* spp., *Dipartiella* spp., *Paratrachodina* spp., and *Tripartiella* spp.

Geographic Distribution: Freshwater, brackish, and marine fish worldwide.

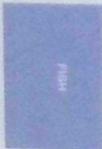
Location in Host: Generally on the external surface of the skin, fins, and gills, though a few species are parasitic in the urinary tract of fish.

Life Cycle: This peritrichous ciliate divides by binary fission, producing two daughter cells.

Laboratory Diagnosis: This flattened, circular, ciliated protozoan is detected in wet mounts of skin biopsies and gill and fin snips and is identified by its prominent internal denticular (toothlike) ring and its ventrally located concave adhesive disk.

Size: Variable, 150–300 × 40–60 μm

Clinical Importance: These organisms are usually ectocommensal but can become ectoparasitic when environmental and host conditions are suitable. This mobile scrub-brush-like parasite may cause localized to generalized skin lesions and disruption of the normal respiratory process of the gill and osmoregulatory processes of the skin. Generally, the parasite is only a problem in heavy infestations of the skin and gill.



FISH





Fig. 6.9 *Epistylis* sp. is a colonial ciliate that has a conical or elongated cylindrical body, a noncontractile stalk, and an epistomal disk for attachment to the host. The parasite generally causes only localized skin lesions.

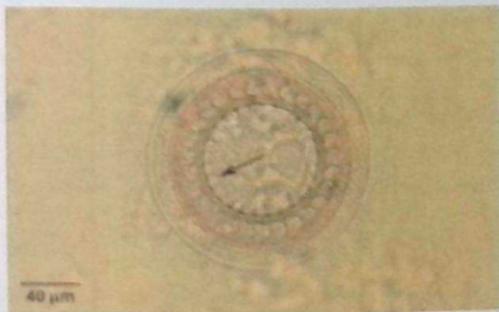


Fig. 6.10 *Trichodina* sp. from a skin biopsy of a tropical freshwater oscar (*Astronotus ocellatus*). These flattened ciliates have a prominent circular, denticular ring (arrow), upon which specific identification is based.

PARASITE: *Trichophyra* spp. (Fig. 6.11)

Taxonomy: Protozoa (ciliate).

Geographic Distribution: Freshwater fish worldwide.

Location in Host: Attached to epithelial surfaces of the skin, fins, and gills.

Life Cycle: This parasite reproduces by endogenous budding.

Laboratory Diagnosis: The parasite can be observed in wet mounts of skin biopsies and gill snips and is diagnosed by its numerous suckorial tentacles.

Size: 30–50  $\mu\text{m}$

Clinical Importance: This small, sedentary parasite is generally only a problem in heavy infestations, where this pincushion-like ciliate attaches to the skin, fin, and gill tissues and causes irritation and localized lesions that disrupt normal respiratory and osmoregulatory processes of the fish.

PARASITE: *Amyloodinium ocellatum* (Figs. 6.12 and 6.13).

Common name: Velvet disease or rust disease.

Taxonomy: Protozoa (flagellate). Other genera include *Oodinium* spp. and *Piscinoodinium* spp.

Geographic Distribution: Freshwater, marine, and brackish fish worldwide.

Location in Host: Attached to epithelial tissues of the skin, fins, and gills.

Life Cycle: The trophozoite attaches to the host via an attachment disk with filiform projections that penetrate into the epithelial tissue of the host. The trophozoite detaches from the host and forms an encysted tomat that divides and produces up to 256 free-swimming, infective dinospores that are released into the water.

Laboratory Diagnosis: Large, cylindrical dinoflagellates may be seen in wet mounts of gill snips where the organism is attached to the lamellae of the gill tissue.

Size:	Trophozoite attached to tissues	up to 150–200 $\mu\text{m}$ in length
	Dinospores in water	12–15 $\mu\text{m}$ in diameter

Clinical Importance: This ectoparasite primarily causes irritation of the gill, resulting in hyperplasia of the epithelial tissues and fusion of gill lamellae. Outbreaks may be fatal in heavily infected fish, especially in fish that are weakened or stressed by other conditions.



Fig. 6.11 *Trichophyru* sp. on a gill biopsy of a channel catfish (*Ictalurus punctatus*). This freshwater ciliate parasite has a "pincushion" appearance (arrow) and is often found on the skin and gills of pond-reared fish.



Fig. 6.12 *Amyloodinium ocellatum* on the gill tissue of a striped bass (*Morone saxatilis*). This organism is the cause of "velvet" or "rust" in ornamental marine species of fish. *Oodinium* sp. is a similar parasite in freshwater fish.



Fig. 6.13 Histopathology of *Amyloodinium ocellatum* attached to the gill tissue of a striped bass (*Morone saxatilis*) showing the basilar site of rhizoid penetration (arrow) into the epithelial cells of the gill.

**PARASITE:** *Ichthyobodo* (= *Costia*) *necator* (Fig. 6.14)

**Taxonomy:** Protozoa (flagellate).

**Geographic Distribution:** Freshwater fish worldwide.

**Location in Host:** Attached to epithelial tissues of the skin, fins, and gills.

**Life Cycle:** The attached feeding stage alternates with a free-swimming nonfeeding stage and reproduces by simple transverse division.

**Laboratory Diagnosis:** This very small flagellate with two unequal flagella extending from its posteriolateral groove is detected in wet mounts of skin biopsies and gill snips. The parasite is diagnosed by its small size and characteristic rapid erratic movements through the water.

**Size:** Trophozoite 10–20 × 5–10 μm

**Clinical Importance:** This often-overlooked parasite is extremely pathogenic in young fish and older fish with lowered resistance. *Ichthyobodo* causes irritation of the skin and gill tissue, resulting in hyperplasia of the epithelial tissues and fusion of gill lamellae.

**PARASITE:** *Myxobolus* spp., including *M. cerebralis* (Fig. 6.15)

**Taxonomy:** Cnidaria (Myxosporea). Fish are parasitized by numerous myxosporean genera and species including *Ceratomyxa* spp., *Kudoa* spp., *Sphaeromyxa* spp., *Myxidium* spp., and *Heneguya* spp.

**Geographic Distribution:** Freshwater, brackish, and marine fish worldwide.

**Location in Host:** Generally the epithelial tissues of the gills, skin, or intestine, but may also invade the muscle, cartilage, and other internal organs.

**Life Cycle:** The myxospore stage of this parasite develops in an annelid or polychaete worm to produce an actinospore stage that either penetrates or is eaten by the definitive fish host. The sporoplasm stage then develops and produces a polysporic cyst in the target tissue of the fish.

**Laboratory Diagnosis:** Variable-sized cysts found within wet mounts of various tissues contain spores with ellipsoidal, rounded, or spindle-shaped smooth valves.

**Size:** Spore 7–10 μm in diameter

**Clinical Importance:** Infection of salmonids with *M. cerebralis* may cause loss of melanophore control in the skin resulting in black-colored tails in very young fish, or skeletal deformities due to cartilage destruction in older fish. This protozoan is a significant pathogen of wild and cultured trout populations. Other myxosporean species may cause white cysts within the skin, gill, or muscle tissue.

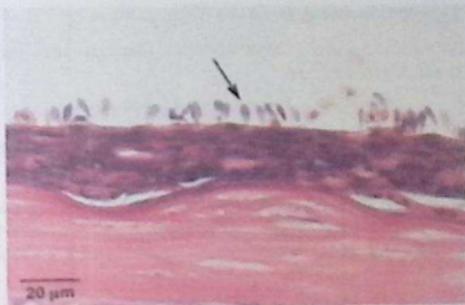


Fig. 6.14 *Ichthyobodo necator*, attached to the skin (arrow), is an extremely pathogenic flagellate parasite of freshwater fish that is often overlooked due to its small size.

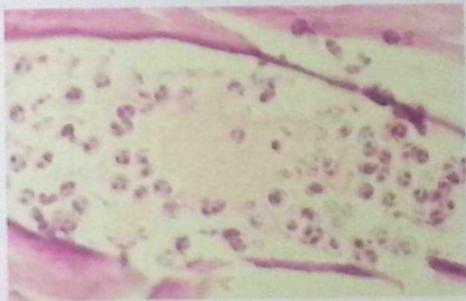


Fig. 6.15 Myxospores of *Myxosporis* sp. (a myxosporean parasite) in cartilaginous tissues of a rainbow trout (*Oncorhynchus mykiss*). Note the two polar capsules within the myxospore.

**PARASITE:** *Henneguya* spp., including *H. ictaluri* (Fig. 6.16)

**Taxonomy:** Cnidaria (Myxosporrea). Myxosporrean fish parasites include numerous genera and species; see *Myxobolus* spp.

**Geographic Distribution:** Freshwater, brackish, and marine fish worldwide.

**Location in Host:** Generally the epithelial tissues of the gills and skin, but may also invade muscle and internal organs.

**Life Cycle:** The myxospore stage of this parasite develops in an annelid or polychaete worm to produce an actinospore stage that either penetrates or is eaten by the definitive fish host. The sporoplasm stage then develops and produces a polysporic cyst in the target tissue of the fish.

**Laboratory Diagnosis:** Variable-sized cysts found within wet mounts of gill or other tissues containing spores with ellipsoidal, rounded, or spindle-shaped smooth valves, with or without caudal appendages.

**Size:** Spore  $19 \times 4.5 \mu\text{m}$ , with two caudal appendages of  $45 \mu\text{m}$  each

**Clinical Importance:** *Henneguya ictaluri* causes a hyperplastic reaction in the epithelial cells of the gills, causing severe branchitis with respiratory compromise, and is a significant pathogen of the commercial catfish industry.

**PARASITE:** *Monogeneans* (Fig. 6.17)

**Taxonomy:** Monogenea (Monopisthocotylea). Including the genera *Gyrodactylus* spp., *Dactylogyrus* spp., and *Neobenedenia* spp.

**Geographic Distribution:** Freshwater, brackish, and marine fish worldwide.

**Location in Host:** Attached to external surface of the skin, fins, and gills.

**Life Cycle:** Monogeneans complete their viviparous or ovoviviparous life cycle on one host. Larvae are morphologically similar to adults.

**Laboratory Diagnosis:** These small- to medium-sized flatworms have a posterior attachment organ, or opisthaptor, that may have hooks, anchors, clamps, or suckers. Monogeneans can be detected in wet mounts of gill snips or skin biopsies.

**Size:** Up to 5 mm in length

**Clinical Importance:** These parasitic flatworms cause local damage at the site of attachment and also through their feeding activity on the external surfaces of the fish.



Fig. 6.16 Giemsa-stained myxospores of the myxosporean parasite *Henegoyi* sp. from a ruptured cyst in the gill filament of a freshwater channel catfish (*Ictalurus punctatus*). Note the two anterior polar capsules within the spore and forked caudal appendages.

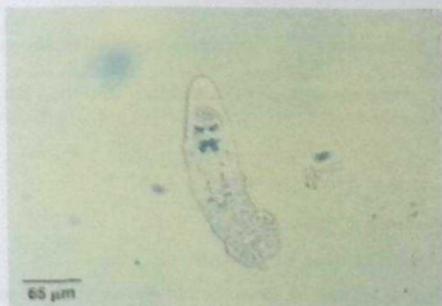


Fig. 6.17 Adult monogenean from a skin biopsy of a rainbow trout (*Oncorhynchus mykiss*). These external parasites have hooks, anchors, clamps, or suckers for attachment to the skin, fins, and gills.

PARASITE: Larval flukes (Fig. 6.18)

TAXONOMY: Trematode (Digenea).

Geographic Distribution: Freshwater, brackish, and marine fish worldwide.

Location in Host: Encysted metacercariae can be found in various tissues of the fish, including the skin, gills, muscle, eyes, brain, and visceral organs.

Life Cycle: Digentic trematodes require at least one intermediate host, commonly a snail or other mollusk, to complete their life cycle. Eggs are deposited in the water by the definitive host (mammal, bird, or fish), which hatch, and release free-swimming miracidia that enter a first intermediate snail host. The developing parasites emerge into the water from the snail as free-swimming cercariae that penetrate the tissues of a second intermediate host, commonly a fish. The parasites then become encysted metacercariae and do not develop further until ingested by the appropriate carnivorous host (e.g., fish-eating bird, fish, or mammal) where the parasite becomes a mature adult.

Laboratory Diagnosis: Larval trematodes are generally visible grossly within the fish tissue.

Size:	Metacercaria	Variable, up to several millimeters
-------	--------------	-------------------------------------

Clinical Importance: These parasites are often unsightly in tissues, but generally are of minimal significance unless large numbers of metacercariae interfere with organ function.

PARASITE: *Argulus* sp. (Fig. 6.19)

Common name: Fish louse.

TAXONOMY: Arthropod (Crustacea, Branchiura).

Geographic Distribution: Freshwater, brackish, and marine fish worldwide.

Location in Host: Generally on the external surfaces of the skin and fins.

Life Cycle: Eggs, which are laid in water, release free-swimming copepod larvae that attach to suitable fish hosts and metamorphose several times before reaching the adult stage.

Laboratory Diagnosis: These grossly visible dorsoventrally flattened, oval parasites have two prominent sucking disks, two dark-colored eyespots, and a centrally located piercing stylet.

Size: 3–5 mm

Clinical Importance: This crustacean causes damage by piercing the external tissues of the host and ingesting cellular fluids, and often causing a severe localized reaction at the site of stylet penetration. These parasitic arthropods may be important vectors in the transmission of certain viral, bacterial, and protozoan fish diseases.



Fig. 6.18 Encysted metacercariae of two different species of digenetic trematodes in the muscle of a bluegill (*Lepomis macrochirus*). The smaller organism (B) is commonly called "black spot disease" (*Neascus* sp.), and the larger, paler one (Y) is commonly called a "yellow grub" (*Clonostomum* sp.). Both are larval stages that will not complete metamorphosis until ingested by a carnivorous definitive host.



Fig. 6.19 *Argulus* sp. from a goldfish (*Carassius auratus*), commonly known as a "fish louse." This ectoparasitic crustacean can be found on the skin and fins of freshwater fish. Other species can be found on brackish and marine species of fish.

PARASITE: *Lepeophtheirus* spp., *Caligus* spp. (Fig. 6.20)

Common name: Sea louse.

Taxonomy: Arthropod (Crustacea, Copepoda).

Geographic Distribution: Marine fish worldwide.

Location in Host: Generally on the external surfaces of the skin and fins.

Life Cycle: Attached female lice release eggs into the water where the eggs hatch releasing free-swimming nauplii larvae. The copepod metamorphoses through multiple free-swimming and then attached larval stages (copepodid and chalimus) to become parasitic adults feeding on the mucus, skin, and blood of the fish.

Laboratory Diagnosis: These grossly visible parasites have a large cephalothorax that acts like a suction organ for holding the louse on the fish. Females, which are larger than males, often have two long egg sacs trailing from their body.

Size: 5-8 mm

Clinical Importance: This crustacean causes physical, enzymatic, and inflammatory damage at the site of attachment and feeding resulting in hemorrhagic ulcerative skin lesions. These parasitic arthropods may also be important vectors for certain viral fish diseases.

PARASITE: *Ergasilus* spp. (Fig. 6.21)

Common name: Gill louse.

Taxonomy: Arthropod (Crustacea, Copepoda).

Geographic Distribution: Freshwater, brackish, and marine fish worldwide.

Location in Host: Most commonly found attached to the gills, rarely the skin and fins.

Life Cycle: Females release eggs into the water that hatch and release free-swimming larvae. The copepod metamorphoses through numerous free-swimming stages to become either free-living males or attached parasitic females feeding on the mucus and skin of the fish.

Laboratory Diagnosis: These parasites have a large modified prehensile antennal segment for grasping gill filaments of the fish. Gravid females commonly have two egg sacs trailing from their body.

Size: 4-6 mm

Clinical Importance: This crustacean causes traumatic crushing damage at the site of attachment to the gill filaments of the host resulting in focal devitalized gill tissue.

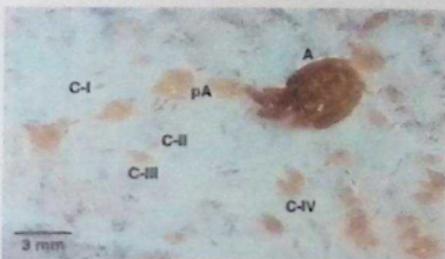


Fig. 6.20 *Lepeophtheirus salmonis*. Sea lice on the skin of an Atlantic salmon (*Salmo salar*). This copepod has several free-swimming stages known as nauplii that molt into an infectious copepodid stage. Once on the fish, the copepodid molts into parasitic chalimus stages (C-I through C-IV), then a pre-adult stage (pA) before becoming a reproductive adult (A = female adult). Photo courtesy of Dr. Larry Hammell, Atlantic Veterinary College, University of Prince Edward Island, Charlottetown, PEI, Canada.



Fig. 6.21 *Ergasilus* spp. Adult female copepod parasites removed from the oral cavity of a freshwater striped bass (*Morone saxatilis*) where they were firmly attached to the gill filaments. Each parasite has a cephalothorax with modified grasping appendages, a central abdomen, and two elongated egg sacs.

PARASITE: *Lernaea* spp. (Fig. 6.22)

Common name: Anchor worm.

Taxonomy: Arthropod (Crustacea, Copepoda).

Geographic Distribution: Freshwater fish worldwide.

Location in Host: Most commonly found attached to the skin and fins.

Life Cycle: Females release eggs into the water that hatch and release free-swimming larvae. The copepod metamorphoses through several free-swimming stages with mating occurring during the last copepodid stage. After mating, the female attaches to the skin of the fish and transforms into an unsegmented wormlike parasite. The anterior end becomes modified into an anchor-like holdfast organ buried in the skin, while the posterior end hangs free in the water.

Laboratory Diagnosis: These parasites have a large anchor-like anterior end and a long unsegmented body. Females commonly have two egg sacs trailing from their body.

Size: 4–6 mm

Clinical Importance: This crustacean induces a localized, hyperplastic inflammatory reaction at the site where the parasite is embedded in the tissue.

PARASITE: Leeches (Fig. 6.23)

Taxonomy: Phylum Annelida (subclass Hirudinea).

Geographic Distribution: Freshwater, brackish, and marine fish worldwide.

Location in Host: Attached to external tissues of the skin, fins, and gills.

Life Cycle: Leeches are hermaphroditic and reproduce by reciprocal fertilization.

Laboratory Diagnosis: These grossly visible flat to cylindrical wormlike parasites have a varying number of body divisions and generally both anterior and posterior suckers.

Size: Variable, up to several centimeters in length

Clinical Importance: Besides the physical damage caused by attachment and blood-sucking activities, leeches are vectors for a variety of fish pathogens, including viruses, bacteria, and hemoparasites.



Fig. 6.22 *Lernaeus* spp. This parasitic copepod, commonly known as an anchor worm, has several free-swimming larval stages before becoming infective to the host. After mating, the male generally dies, while the female burrows into the flesh of the fish. The anterior end of the parasite develops into an anchor-like adaptation that becomes embedded in the tissues of the fish, while the posterior end becomes modified into an unsegmented, wormlike body with two egg sacs that hang from the fish's body. Photo courtesy of Dr. Roy P. E. Yanong, Tropical Aquaculture Laboratory, University of Florida, Ruskin, FL.

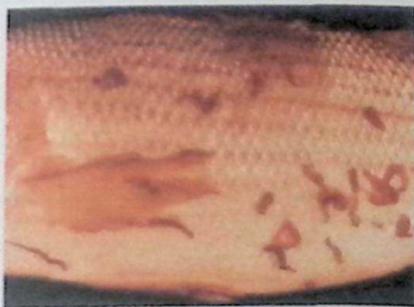


Fig. 6.23 Leeches of an unidentified species attached to the skin of a cultured hybrid striped bass (*Morone saxatilis* × *M. chrysops*). These parasites can cause anemia in fish, as well as act as vectors of hemoparasites and other pathogens.

**PARASITE:** Thorny-headed worm (Fig. 6.24)

**Taxonomy:** Phylum Acanthocephala.

**Geographic Distribution:** Freshwater, brackish, and marine fish worldwide.

**Location in Host:** Lumen of the posterior intestinal tract.

**Life Cycle:** These parasites have an indirect life cycle, with usually a crustacean intermediate host.

**Laboratory Diagnosis:** Eggs are observed on standard fecal flotation or direct smear.

**Size:** 50–65 × 30–40 μm

**Clinical Importance:** These parasites generally do not cause any clinical signs in fish. Rarely, a severe infestation will result in perforation of the intestinal tract, with subsequent peritonitis.

**PARASITE:** Nematode Parasites (Figs. 6.25 and 6.26)

**Taxonomy:** Nematode.

**Geographic Distribution:** Freshwater, brackish, and marine fish worldwide.

**Location in Host:** Lumen of the intestinal tract.

**Life Cycle:** Both direct and indirect life cycles have been reported.

**Laboratory Diagnosis:** A variety of nematode-type eggs may be observed on standard fecal flotation or direct smear.

**Size:** Variable

**Clinical Importance:** Adult nematodes generally do not cause significant clinical signs in fish. A severe infestation sometimes will result in mechanical blockage of the intestinal tract and/or chronic weight loss.



Fig. 6.24 Egg of an acanthocephalan parasite from a black crappie (*Pomoxis nigromaculatus*).



Fig. 6.25 Eggs of *Capillaria* sp. from a freshwater angelfish (*Pterophyllum scalare*).



Fig. 6.26 Unidentified nematode egg from a largemouth bass (*Micropterus salmoides*).



# Treatment of Veterinary Parasites

## INTRODUCTION

A wide variety of safe, effective parasiticides can eliminate infections and infestations or prevent development or exacerbation of parasitic disease. Despite the availability of these powerful treatments, parasite control continues to be an unmet need for many domestic animals due to lack of use or incomplete knowledge on efficacy. Choosing the parasiticide treatment most appropriate for a particular clinical situation requires careful assessment of risk factors as well as veterinary expertise regarding individual patient, herd, or flock characteristics and needs. Rarely is one treatment or a single protocol the best option for all patients, even when general concerns about the type of parasitic disease of greatest risk are similar. In this chapter we summarize information about anthelmintics, ectoparasiticides, and antiprotozoals currently available to veterinarians. Only products approved by FDA, EPA, or similar regulatory agencies and that have been confirmed to be safe and efficacious will be discussed here; availability of some approved products or formulations may be limited. Products covered in this chapter are largely restricted to those available in the United States and Canada. Veterinarians should be familiar with and consult current regulatory-approved label information prior to prescribing or recommending any specific product.

## ANTHELMINTICS

Effective control of helminths involves more than just selection of a safe, effective anthelmintic. Effective parasite management programs must incorporate likelihood of environmental contamination and thus risk of re-infection, seasonality of transmission, age and immune status of animals, and zoonotic risk of allowing infections to persist. Selection of the best anthelmintic for a given clinical situation is often based on factors other than efficacy, including safety, application route, persistence, and knowledge about resistance. Traditionally, anthelmintics were administered in liquid oral formulations either directly or via nasogastric tube. However, many anthelmintics are

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard  
© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

Table 7.1. Common administration routes for anthelmintics

Application route	Examples	Species available for
Oral		
Tablets/chewable	Milbemycin oxime +/- praziquantel Ivermectin +/- pyrantel +/- praziquantel Pyrantel/praziquantel +/- febantel	Cats, dogs Cats, dogs Cats, dogs
Paste	Fenbendazole, ivermectin, moxidectin, oxbendazole, pyrantel pamoate Fenbendazole	Horses *Cattle
Liquid	Albendazole, closantel, fenbendazole, ivermectin, levamisole, moxidectin, oxfendazole	*Cattle, *sheep, *goats
Water additive	Fenbendazole	*Pigs
Feed additive	Fenbendazole, morantel tartrate Dichlorvos, fenbendazole, ivermectin, levamisole, piperazine, pyrantel tartrate	*Cattle *Pigs
Block / mineral	Fenbendazole	*Cattle
Pellets	Fenbendazole, pyrantel tartrate	Horses
Injectable	Melarsomine dihydrochloride Moxidectin Doramectin, ivermectin Levamisole, moxidectin Eprinomectin extended release parasiticide Praziquantel	Dogs Dogs *Cattle, *pigs *Cattle *Cattle Cats, dogs
Transdermal	Moxidectin, selamectin Emodepside/praziquantel Eprinomectin/praziquantel Doramectin, eprinomectin, ivermectin, moxidectin	Cats, dogs Cats Cats *Cattle

\* Products listed not approved for use in all food animals, always consult label prior to administering.

now given with the feed, injected, or as a transdermal product that is administered topically and absorbed systemically (Table 7.1).

Anthelmintics also differ in the degree to which they persist in the animal after administration, with some products being cleared within 24 hours and others lasting and remaining efficacious as long as 6 months. Persistency is often desired to reduce the number of times animals must be treated but can affect withdrawal times (Table 7.2) and influence selection for resistance. With use of any antiparasitic drug treatment, including anthelmintics, selection for resistance will occur. Anthelmintic resistance has reduced the utility of these drugs in certain clinical situations. Accordingly, recognizing resistance when it occurs and developing appropriate strategies to protect animal health in the face of diminished anthelmintic efficacy are important clinical skills. Resistance to parasitocides is discussed further below.

## Specific Anthelmintics

Macrocyclic lactones, also referred to as avermectin/milbemycin compounds, constitute some of the safest, most effective, and most widely used anthelmintics ever developed. First marketed in the early 1980s, the class now includes **abamectin**, **doramectin**, **eprinomectin**, **ivermectin**, **milbemycin oxime**, **moxidectin**, and **selamectin**. These compounds cause flaccid paralysis by impeding glutamate-gated chloride channels; removal

Table 7.2. Examples of withdrawal times for selected anthelmintics

Anthelmintic	Species treated	*Withdrawal times
Albendazole	Beef cattle	27 d oral liquid
	Goats, sheep	7 d oral liquid
Dichlorvos	Pigs	0 d feed additive
Doramectin	Pigs	24 d injectable
	Beef cattle	35 d injectable; 45 d pour-on
	Dairy cattle	Injectable only in heifers <20 mo. of age
Eprinomectin	Beef cattle	0 d pour-on; 48 d injectable
	Dairy cattle	0 d pour-on; injectable only in heifers <20 mo. of age
Fenbendazole	Pigs	0 d feed additive
	Beef cattle	8 d oral liquid, paste; 11 d molasses block; 13 d flaked meal, free-choice mineral, pellets; 16 d protein block
	Dairy cattle	0 d oral liquid, flaked meal, free-choice mineral, paste, pellets
	Goats	6 d oral liquid
Ivermectin	Pigs	5 d feed additive; 18 d injectable
	Beef cattle	35 d injectable; 48 d pour-on
	Dairy cattle	Injectable only in heifers <20 mo. of age
	Sheep	11 d oral liquid
Levamisole	Pigs	3 d water or feed additive
	Beef cattle	2 d bolus, oral liquid, feed additive; 7 d injectable; 9 d pour-on
	Sheep	3 d oral liquid
Morantel tartrate	Beef cattle	14 d feed additive
	Dairy cattle	0 d feed additive
Moxidectin	Beef cattle	0 d pour-on; 21 d injectable
	Dairy cattle	0 d pour-on; injectable only in heifers <20 mo. of age
	Sheep	7 d oral liquid
Oxfendazole	Beef cattle	7 d oral liquid
Piperazine	Pigs	21 d water or feed additive
Pyrantel tartrate	Pigs	24 h feed additive

\* Withdrawal times listed are according to FDA-approved labels and may vary by formulation; always consult label prior to administering. Food Animal Residue Avoidance Databank (FARAD, <http://www.farad.org>) withdrawal times may differ from those on the FDA-approved label.

of gastrointestinal parasites follows via peristalsis and stages in tissues are cleared by the immune system. Although the specific efficacy of individual compounds differs and formulations influence spectrum of activity, in general, members of this class are effective against a diverse array of nematodes and arthropods. All available formulations are safe when administered according to label directions. However, extralabel use of high doses can result in toxicity, particularly in collies and other dogs that carry the MDR1 mutation and in certain exotic animals, including chelonians and some other reptiles.

Originally developed as plant fungicides, the benzimidazoles have been used as anthelmintics since the early 1960s. Key members of this class still used in veterinary medicine include albendazole, fenbendazole, flubendazole, mebendazole, oxfendazole, oxibendazole, ricobendazole, and triclabendazole as well as febantel, a pro-benzimidazole. The benzimidazoles bind tubulin, preventing formation of microtubules necessary for cellular function, including energy metabolism. Depending on the compound considered, efficacy has been demonstrated against a wide variety of nematodes as well as some plathelminths and *Giardia* sp. Although generally safe, some members of this

class, notably albendazole and febantel, are potentially teratogenic when administered early in pregnancy.

The tetrahydropyrimidines have been used as anthelmintics since the 1970s and include the popular dewormer **pyrantel**, which is available in several different salt forms, as well as **morantel tartrate**. After administration, these drugs remain in the gastrointestinal lumen and thus are not effective against tissues stages of helminths. Pyrimidines are acetylcholine agonists that lead to spastic paralysis and subsequent expulsion of parasites. Efficacy has been documented against a variety of nematodes as well as equine tapeworms. The safety profile of this class is excellent even at elevated doses.

**Levamisole** is an imidazothiazole that also acts as a nicotinic acetylcholine agonist; levamisole has a narrow safety margin and overdosing can lead to nicotinic-type symptoms, particularly when animals are dehydrated or stressed. Other anthelmintics that bind to acetylcholine receptors include **monepantel**, an amino-acetonitrile derivative, and **derquantel**, a semi-synthetic spiroindole. Both compounds lead to paralysis of gastrointestinal nematodes and are marketed for sheep in some areas. **Emodepside** is a cyclic depsipeptide anthelmintic developed for treating hookworms and ascarids in cats and dogs. The drug paralyzes nematodes upon binding to lactrophilin-like receptors. **Closantel** is a salicylanilide effective against adult and immature *Fasciola hepatica*, some nematodes, and larval stages of some arthropods. This compound is a potent oxidative phosphorylase uncoupler and toxicity has been reported in ruminants at both standard and elevated doses.

**Melarsomine dihydrochloride** is an arsenical compound with a narrow therapeutic index used to treat infection with adult *Dirofilaria immitis* in dogs. The arsenic salt reacts with sulphydryl enzymes resulting in parasite death. Injections are administered deep in the epaxial muscle and both injection site reactions and systemic reactions can occur. Reactions are most common in dogs with high numbers of *D. immitis*. Due to toxicity concerns, melarsomine dihydrochloride should not be used in dogs with reduced liver function or in cats. Less commonly used dewormers effective against nematodes include **piperazine**, which disrupts GABA neurotransmitters in ascarids leading to neuromuscular blockade; **dichlorvos**, an organophosphate labeled against gastrointestinal nematodes in swine; and **hygromycin B**, an antibiotic fed to poultry and swine that also controls intestinal nematodes.

Some anthelmintics are only effective against cestodes or trematodes, including the isoquinolones **praziquantel** and **epsiprantel**, and the benzene sulfonamide **clorsulon**. The isoquinolones act by increasing membrane permeability to calcium resulting in destruction of the tegument and parasite paralysis and have wide efficacy against a number of intestinal and tissue stages of cestodes and trematodes. Safety of these compounds is excellent; high doses can be used extralabel when necessary. Clorsulon is primarily used against *Fasciola hepatica* in cattle but availability is limited in many countries. Combination anthelmintics also are available. For example, a macrocyclic lactone, benzimidazole, or tetrahydropyrimidine with broad spectrum nematode efficacy may be combined with an isoquinolone effective against cestodes. These formulations provide efficacy against a broader spectrum of helminths and facilitate ease of use. In some areas, multiple anthelmintic groups with similar spectrums of action are combined to enhance efficacy against resistant nematodes.

Selection for resistance occurs within a population of parasites as anthelmintics are used. Examples of anthelmintic resistance include resistance to benzimidazoles in equine small strongyles and ruminant trichostrongyles; resistance to macrocyclic

lactones in *Dirofilaria immitis*, equine ascarids, and ruminant trichostrongyles; and resistance to pyrantel in canine hookworms and equine small strongyles. Strategies to delay selection for resistance focus on maximizing **refugia**, the portion of the parasite population not exposed to an anthelmintic and thus not subject to selection pressure, along with pasture and animal management strategies that reduce the need for anthelmintic use.

## ECTOPARASITICIDES

Arthropod pests and disease vectors are controlled by environmental management, animal treatment, or a combination of both strategies. As with anthelmintics, treatment decisions may be influenced by seasonal activity, production loss, and potential for zoonotic risk. Selection of an appropriate compound is influenced by persistence, route of application, safety, and resistance. Traditionally, control of arthropod pests was achieved by topical application of short-acting compounds. However, in addition to topical application, many modern systemic insecticides and acaricides may be administered orally, transdermally, or by injection, and efficacy persists for weeks to months after treatment (Table 7.3, Table 7.4). Products should only be used in accordance with regulatory requirements. In the United States, it is a violation of federal law to use EPA-registered pesticides in a manner inconsistent with the label. Pesticides approved by EPA for one host (e.g., cattle, dogs) should not be used in another host (e.g., horses, cats).

Isoxazolines have persistent efficacy against fleas, ticks, and other arthropods. First introduced in 2014, members of the class include **afoxolaner**, **fluralaner**, **lotilaner**, and **sarolaner**. The compounds are administered by the oral or transdermal route and provide systemic control against fleas, ticks, and mites in dogs and cats for 4–12 weeks following a single administration. Efficacy is achieved through inhibition of arthropod GABA/glutamate-gated chloride channels. While isoxazolines are considered safe and effective, the class has been associated with adverse reactions, including muscle tremors, ataxia, and seizures, in some pets and should not be used in patients with a history of seizures.

**Fipronil**, a phenylpyrazole developed in the mid-1990s, is a GABA chloride channel antagonist effective against fleas, lice, mites, and ticks. Although safe for dogs and cats, fipronil should not be applied to rabbits. Macrocyclic lactones (see earlier) also have efficacy against a variety of arthropods. Specific claims vary with individual formulations, but members of this class may be effective against fleas, flies, lice, mites, and ticks with injectable, oral, otic, topical, and transdermal formulations available. **Indoxacarb** is an oxadiazine insecticide that, once ingested and metabolized by insects, blocks insect voltage-gated sodium ion channels. Indoxacarb has efficacy against eggs, larvae, and adult fleas; some formulations are combined with permethrin and thus should not be applied to cats (see below).

Neonicotinoids were first developed in 1991 and are widely used to control plant pests and fleas; members of this class include **imidacloprid**, **dinotefuran**, and **nitropyran**. Neonicotinoids are agonists of insect nicotinic acetylcholine receptors (nAChR) and have rapid efficacy against fleas and other insects. **Spinosad** is a macrolide that, similar to neonicotinoids, activates nAChR but at a unique site, leading to paralysis and death of fleas and other arthropods. These compounds have high affinity for insect receptors making them very safe for use in mammals and birds. Topical neonicotinoids are often formulated in combination with pyrethroids (see below) for efficacy against ticks, as

**Table 73. Common administration routes for insecticides and acaricides used in small animals and primary arthropods targeted**

Application route	Examples	*Compounds used	Primary target for control
Environmental	Premise spray, fogger	Pyrethrins/pyrethroids	Fleas, flies, ticks on premise
		Methoprene, pyriproxyfen	Fleas (immature stages)
Topical	On-animal spray, wipe, powder	Fipronil, pyrethrins/pyrethroids	Fleas, ticks on dogs, cats
		Dimetofuran	Fleas on dogs, cats
	Shampoo	Pyrethrins/pyrethroids	Fleas, ticks on dogs, cats
		Methoprene, pyriproxyfen	Fleas (immature stages) on dogs, cats
	Dip	Pyrethroids	Fleas, ticks on dogs
	Collar	Amtraz	Ticks on dogs
		Deltamethrin	Fleas, ticks, flies on dogs
		Imidacloprid/flumethrin	Fleas, ticks on dogs, cats
	Spot-on	Methoprene, pyriproxyfen	Fleas (immature stages) on dogs, cats
			Fleas, ticks on dogs
Fipronil, pyrethroids		Fleas, ticks on dogs	
		Flies on dogs	
Spinetoram		Fleas on cats	
Systemic		Oral	Nitazopyram, spinosad
	Afoxolaner, fluralaner, lotilaner, sarolaner		Fleas, ticks on dogs
	Lufenuron		Fleas (immature stages) on dogs
	Transdermal	Moxidectin, selamectin	Fleas, mites, ticks on dogs, cats
		Fluralaner, sarolaner	Fleas, ticks on dogs, cats

\* All types of compounds listed may not be approved or safe for use in all breeds or species; always consult label prior to administering.

well as insect growth regulators like methoprene or pyriproxyfen, juvenile hormone mimics that limit development of fleas in the environment. Lufenuron and diflubenzuron are examples of insect development inhibitors that disrupt chitin synthesis and prevent development of immature fleas. While neonicotinoids have an excellent safety profile when used alone, products combined with permethrin should not be applied to cats.

Pyrethrins are natural botanical compounds first isolated from *Chrysanthemum cinerariaefolium* in the 1960s and provide short-acting control of arthropods; several generations of synthetic pyrethroids with higher potency, longer photostability, and more persistent efficacy have since been developed. Examples of pyrethroids used in veterinary medicine include betacyfluthrin, cypermethrin, cyphenothrin, deltamethrin, esfenvalerate, flumethrin, lambdacyhalothrin, and permethrin; etofenprox is a related compound with similar activity. These chemicals disrupt sodium- and potassium-mediated neurotransmission in arthropods, and formulations often include piperonyl butoxide as a synergist to inhibit arthropod oxidation of the compounds, thus increasing activity. Pyrethrins and pyrethroids are effective against both insects and ticks, with

**Table 24. Common administration routes for insecticides and acaricides used in large animals and primary arthropods targeted**

Application route	Examples	Compounds used	Primary target for control	
Environmental	Premise spray, fogger	Pyrethrins/pyrethroids, organophosphates, spinosad	Flies on premise	
	Granule	Imidacloprid, indoxacarb, pyrethroids, spinosad	Flies on premise	
Topical	Fly strip	Organophosphates, pyrethroids	Flies on premise	
	On-animal spray	Amitraz	Lice, ticks on cattle, pigs	
	On-animal spray, wipe, dust	Pyrethrins/pyrethroids, organophosphates	Flies, lice on cattle, horses	
	Back rubber	Pyrethroids, organophosphates	Flies, lice, ticks on cattle	
Systemic	Ear tag	Abamectin, pyrethroids, organophosphates	Flies, lice, ticks on cattle	
		Oral	Cyromazine, diflubenzuron	Flies (immature stages) on horses
			Cyromazine, methoprene	Flies (immature stages) on cattle
	Injectable	Macrocyclic lactones	Lice on cattle; grubs/bots in cattle, horses	
		Macrocyclic lactones	Lice, mites on cattle, pigs; grubs, in cattle	
Transdermal	Macrocyclic lactones	Flies, lice, grubs, mites on cattle		

\* All types of compounds listed may not be approved or safe for use in all breeds or species; always consult label prior to administering.

persistency of different formulations varying from as short as 1 day (pyrethrins) to as long as 6 or 8 months (deltamethrin and flumethrin/imidacloprid collars for dogs and cats and cats, respectively). Products are intended for application to the environment or directly to animals, and on-animal formulations often repel and kill insects and ticks. Pyrethrins have an excellent safety profile and can be used in all ages of animals. When label directions are followed, pyrethroids are also safe but caution must be taken to avoid applying highly concentrated formulations of some pyrethroids, most notably permethrin, to cats.

Organophosphates (e.g., **coomaphos**, **diiazinon**, **dichlorvos**, **phosmet**, **pirimiphos**, and **tetrachlorvinphos**) were first developed as insecticides in the 1930s and 1940s; carbamates (e.g., **carbaryl**, **propoxur**) followed in the 1950s. Organophosphates and carbamates inhibit acetylcholine esterase (AChE) by binding to the enzyme irreversibly or reversibly, respectively. Present use includes flea and tick collars, powders, sprays, and dips for small animals; ear tags, sprays, dusts, and rub-ons for fly, tick, mite, or lice control on cattle; and adhesive fly strips or sprays for environmental arthropod control; the compounds may be used alone or in combination with pyrethroids. Mammalian toxicity with organophosphates and carbamates results in some combination of diarrhea, urination, miosis, bronchospasm, bradycardia, emesis, lacrimation, and salivation, referred to by the mnemonic "DUMBBELS"; atropine is used to block overstimulation due to either class while 2-PAM is used only in organophosphate toxicity to reactivate inhibited AChE. Lean animals are particularly susceptible to cholinesterase inhibition, and use of these compounds should be avoided in young animals, cats, sighthounds (e.g., whippets, greyhounds), Brahman cattle, and some other cattle breeds (e.g., Charolais, Gelbvieh, Simmental). Due to human safety concerns, on-animal use of

organophosphates and carbamates is often avoided, particularly in animals likely to be handled by people (e.g., dogs, cats, horses). **Amitraz** is a formamidine approved for use in dogs, pigs, and cattle that acts as a monoamine oxidase inhibitor. Dog products are labeled against mites and ticks; food animal products are labeled against lice, mites, and ticks. Due to safety concerns, amitraz should not be used in cats or horses.

## PROTOZOAL TREATMENT

Protozoal infections of animals are treated with a variety of different drugs, many of which are not label-approved for that application. For some infections, such as coccidia, environmental sanitation to limit infection is as important as effective treatment at preventing disease. Not all compounds are readily available in all countries. Care must be taken in food animals to avoid residues that can create human health risk. Safety concerns exist with some of the antiprotozoal treatments discussed.

Some benzimidazoles (e.g., **albendazole**, **fenbendazole**) and pro-benzimidazoles (**febantel**) have efficacy against *Giardia* sp. by preventing microtubule formation. Fenbendazole is label-approved to treat dogs for giardiasis in Europe and is safe to use in pregnancy; fenbendazole is also safe in cats. Febantel is teratogenic but also effective against *Giardia* sp. Both compounds must be administered for at least 3 consecutive days with fenbendazole treatment often continued for a week or more. Albendazole may cause aplastic anemia in some canine and feline patients and, because alternatives are available, should not be used for protozoal treatment in dogs and cats. **Imidocarb** inhibits nucleic acid metabolism in some apicomplexan protozoa and has been used to treat babesiosis, hepatozoonosis, and cytauxzoonosis, although atovaquone and azithromycin combination therapy is preferred for *Babesia gibsoni* and *Cytauxzoon felis*. Pretreatment with glycopyrrolate or atropine is recommended in debilitated or young animals.

Antibiotics with efficacy against protozoa include **clindamycin**, the nitroimidazoles (e.g., **metronidazole**, **ronidazole**, **benznidazole**), and the **sulfonamides**. Clindamycin is a lincosamide antibiotic used to treat clinical toxoplasmosis in cats and dogs. Although metronidazole is somewhat effective against *Giardia* sp., fenbendazole (see earlier) is considered both safer and more effective for treating canine giardiasis; side effects reported with metronidazole include weakness, ataxia, tremors, and seizures. Ronidazole is used in cats with diarrhea due to *Tritrichomonas foetus*. Treated cats may develop neurologic signs. Benznidazole is used for treating dogs with Chagas disease due to *Trypanosoma cruzi*. The nitroimidazoles are not FDA-approved for use in any veterinary species and should not be used in food animals.

Sulfonamides are antibiotics available since the 1930s that are commonly used as coccidiostats. Common sulfonamides include **sulfadiazine**, **sulfadimethoxine**, **sulfamerazine**, **sulfamethazine**, **sulfamethoxazole**, and **sulfaquinoxaline** and act as competitive inhibitors of folic acid synthesis, impairing pathogen growth and metabolism. These compounds are often combined with potentiators such as **ormetoprim**, **pyrimethamine**, or **trimethoprim** that inhibit a subsequent step in folic acid synthesis. Sulfonamides are also used to treat equine protozoal myeloencephalitis. Adverse effects associated with this class include crystalluria and keratoconjunctivitis sicca, as well as hypersensitivity reactions, hepatic necrosis, and anemia. Resistance to sulfonamides is common among *Eimeria* spp.

A number of other compounds also are used as coccidiostats. Of these, only **amprolium** also is approved for treatment of coccidiosis in cattle and chickens in the United States. Amprolium competitively inhibits thiamine; neurotoxicity can occur due to overdose and is treated with thiamine supplementation. **Toltrazuril**, **ponazuril**, and **diclazuril** are triazine antiprotozoals that are very effective against coccidia and some other apicomplexan parasites. Toltrazuril is approved for treatment of coccidiosis in cattle and sheep in many areas of the world, while ponazuril and diclazuril are approved to treat equine protozoal myeloencephalitis in horses. Ponazuril is commonly used extralabel for coccidiosis in dogs, cats, rabbits, bearded dragons, and camelids, and for *Hepatozoon americanum* in dogs. Diclazuril is an approved coccidiostat in poultry. **Clopidol** and **decoquinat** are quinolone or quinolone-like coccidiostats effective against coccidia sporozoites. **Nicarbazin** and **robenidine** are synthetic coccidiostats used in chickens. Ionophore coccidiostats have been used since the early 1970s and include **lasalocid**, **monensin**, **narasin**, **salinomycin**, and **semduramicin**. Ionophores are primarily used as growth promoters but also have efficacy as coccidiostats by inhibiting mitochondrial function. These compounds are widely used in cattle, small ruminants, and poultry but can be fatal when inadvertently fed to horses or birds other than chickens.

### NON-TRADITIONAL TREATMENTS

Alternative parasite treatment strategies are occasionally promoted, but only products approved by FDA, EPA, or similar regulatory agencies have been confirmed to be safe and efficacious. Feeding some types of high-tannin forage and administering copper oxide wire particles (COWP) to small ruminants support control of *Haemonchus contortus*, but should be used with care to avoid adverse effects. Boric acid can be used to desiccate larval stages of fleas in the environment. Supplementing fiber and probiotics has been shown to support resolution of clinical disease in dogs with diarrhea due to giardiasis when combined with an effective antiprotozoal agent. Garlic products, herbal dewormers, pumpkin seed, papaya seed, apple cider vinegar, vitamin supplements, peppermint oil, brewer's yeast, and other home remedies have not been shown to be effective parasite control strategies.



# Diagnostic Dilemmas

Presented below are seven challenging cases in diagnostic parasitology. Case scenarios are based on information and specimens provided to the veterinary diagnostician. The answers to these diagnostic dilemmas are available on the accompanying website [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology) to this volume.

## Diagnostic Dilemma 1

A 2.5-year-old neutered male, indoor/outdoor American bulldog family pet presented in Scranton, PA for an annual wellness exam. The dog had been dewormed monthly and was currently on prevention for heartworm, intestinal helminths, fleas, and ticks. During preparation of the fecal flotation, the veterinary technician noted a small (~5 mm) organism in the fecal material and suspected it might be a worm of some kind. What is the identification of this specimen?



**FIG. 8.1** Specimen observed in the fecal material of a dog. The specimen was approximately 5 mm in length. Photo courtesy of Dr. Manigandan Lejeune, Animal Health Diagnostic Center, Cornell University, Ithaca, NY.

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard.

© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

## Diagnostic Dilemma 2

A 4-year-old mixed breed neutered male dog was presented in Jonesboro, Arkansas for annual physical examination and vaccination. The dog was not on heartworm preventive and had an intermittent cough. A blood sample was negative for heartworm antigen on a well-validated commercial test. Numerous microfilariae were found on microscopic examination of a saline wet mount. Whole blood was submitted for Knott's test, which confirmed the presence of *Dirofilaria immitis* microfilariae based on morphologic appearance, size, and nuclear staining (Figs. 8.2, 8.3).

What is the most likely explanation for a negative heartworm antigen test in the face of numerous *Dirofilaria immitis* microfilariae in this dog?

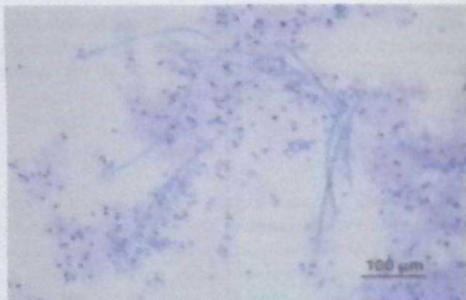


Fig. 8.2 *Dirofilaria immitis* microfilariae recovered on Knott's test from an infected dog. The identification of *D. immitis* is supported by the presence of numerous microfilariae in the sample (see Table 8.2).

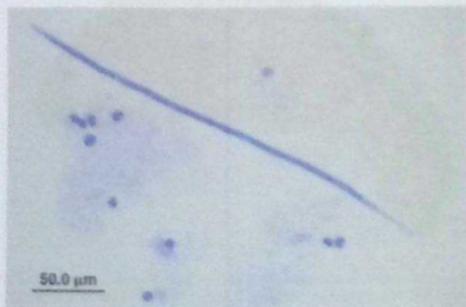


Fig. 8.3 Higher magnification view of a single *Dirofilaria immitis* microfilaria. Although individual microfilaria will shrink in formalin over time, when first prepared, *D. immitis* microfilaria recovered on a Knott's test usually measure 295–325 μm long by 5–7.5 μm wide.

### Diagnostic Dilemma 3

A horse owner with 6 pleasure horses read about anthelmintic resistance and was curious to know if the dewormer in use on the farm was still effective since it had been used exclusively for several years. The owner collected a fecal sample from each horse, dewormed them and dropped the samples off at your hospital for quantitative egg counts. A month later, the owner collected a second set of samples and had those evaluated as well. The results are shown in the table below. The minimum detection limit of the modified McMaster test used to perform the egg counts was 25 eggs/gram. How effective was the deworming treatment?

Horse	Pretreatment egg count	Posttreatment egg count
Madison	200	<25 (none seen)
Finn	500	100
Chessie	100	<25 (none seen)
Simon	<25 (none seen)	25
Thea	350	75
Sammy	175	100

### Diagnostic Dilemma 4

A 6-year-old spayed female German shorthaired pointer used for hunting in Virginia presented for a persistent cough. Results of centrifugal fecal flotation included nematode larvae that could not be identified because of distortion by flotation solution. A Baermann test was performed and several live nematode larvae, approximately 350  $\mu\text{m}$  long, were recovered (Fig. 8.4). What is the identification of these larvae?



Fig. 8.4 Nematode larva, approximately 350  $\mu\text{m}$  long, recovered from the feces of a dog presented for chronic cough. The inset shows a higher magnification of the tail of the larva. Photo courtesy of Dr. Meriam Saleh, Virginia-Maryland College of Veterinary Medicine, Blacksburg VA.

### Diagnostic Dilemma 5

The owner of a 1-year-old intact female Chesapeake Bay retriever submitted a fecal sample from the dog after noticing small (7–10 mm) whitish oval to rectangular-shaped structures passed in the dog's feces (Figs 8.5, 8.6). The client lived on a hobby farm and the dog had freedom to roam over a fairly large area on the property.

What is the identification of these structures?

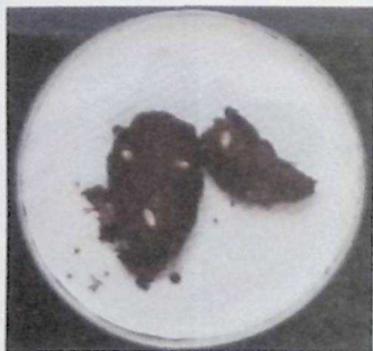


Fig. 8.5 Fecal sample from a clinically normal Chesapeake Bay retriever.

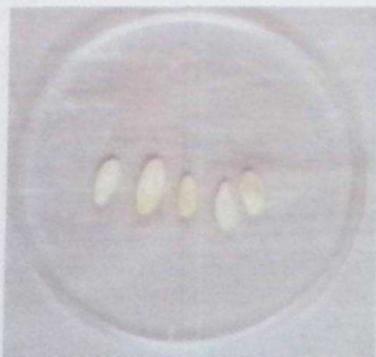


Fig. 8.6 Closer view of 7–10 mm white objects present in the fecal sample.

## Diagnostic Dilemma 6

A 2-year-old intact male, indoor-outdoor domestic shorthair cat was presented to a veterinary practice in Battle Creek, Michigan for lethargy and inappetence. An abscess was identified on the right rear leg and a bite wound secondary to fighting was suspected. The wound was cleaned and flushed and a course of antibiotics instituted. During physical examination, a partially engorged tick was identified and removed from the neck. Although not associated with the primary complaint (abscess due to fighting), the owner asked if the tick might have transmitted any infections to the cat.

The tick was examined dorsally and ventrally (Figs. 8.7, 8.8).

What tick genus and species is this? What disease agents might it transmit?



Fig. 8.7 Dorsal view of tick removed from a cat. Photo provided courtesy of Dr. Katie Clow, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.



Fig. 8.8 (A) Ventral view of tick removed from a cat. (B) Higher magnification of area outlined in black showing the presence of an anal groove (black arrows) arching anterior to the anus (white arrow). Photo provided courtesy of Dr. Katie Clow, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada.

## Diagnostic Dilemma 7

An approximately 25-year-old, castrated male Arab horse spit a worm from his mouth. The owner submitted the worm for identification. No other abnormalities were observed with this horse.

The worm was approximately 10 cm in length and appeared to be a fragment consisting of only the mid-body section of an adult female nematode (Fig. 8.9). There were many eggs (about  $30 \times 60 \mu\text{m}$ ) in the uterus of the female worm fragment (Fig. 8.10).

What further testing could be done and what is the diagnosis?



Fig. 8.9 Fragment of an adult worm expelled from the mouth of a horse. Figure courtesy of Dr. Yuko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

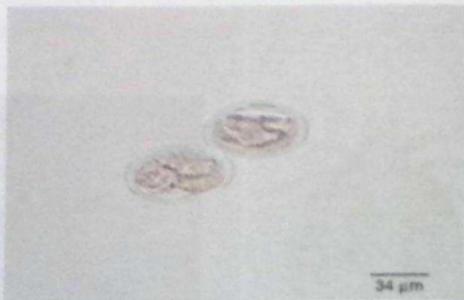


Fig. 8.10 Eggs recovered from the fragment of female worm from a horse. Figure courtesy of Dr. Yuko Nagamori, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK.

## BIBLIOGRAPHY

- American Association of Equine Practitioners. 2019. AAEP Internal Parasite Control Guidelines. Accessed May 2020. <https://aaep.org/guidelines/internal-parasite-control-guidelines>
- Atkinson, C. T., Thomas, N. J., and Hunter, D. B. 2008. *Parasitic Disease of Wild Birds*. Wiley-Blackwell, Ames, IA.
- Baker, D. G. 2007. *Flynn's Parasites of Laboratory Animals*, 2nd ed. Blackwell Publishing, Ames, IA.
- Barnard, S. M., and Upton, S. J. 1996. *A Veterinary Guide to the Parasites of Reptiles: Protozoa*. Krieger Publishing Co., Malabar, FL.
- Bowman, D. D. 2021. *Georgis' Parasitology for Veterinarians*, 11th ed. Elsevier Saunders, St. Louis, MO.
- Bowman, D. D., Hendrix, C. M., Lindsay, D. S., and Barr, S. C. 2002. *Feline Clinical Parasitology*. Iowa State University Press, Ames, IA.
- Clyde, V. L., and Patton, S. 1996. Diagnosis, treatment and control of common parasites in companion and avian birds. *Semin. Avian Exot. Pet Med.* 5:75-84.
- Coles, G. C., Bauer, C., Borgsteede, F. H. M., Geerts, S., Klei, T. R., Taylor, M. A., and Waller, P. J. 1992. World Association for the Advancement of Veterinary Parasitology (W.A.A.V.P.) methods for the detection of anthelmintic resistance in nematodes of veterinary importance. *Vet. Parasitol.* 44:35-44.
- Coles, G. C., Jackson, F., Pomroy, W. E., Prichard, R. K., von Samson-Himmelstjerna, G., Silvestre, A., Taylor, M. A., and Vercruyse, J. 2006. The detection of anthelmintic resistance in nematodes of veterinary importance. *Vet. Parasitol.* 136:167-185.
- Day, M. J. 2015. Introduction to antigen and antibody assays. *Top. Companion Anim. Med.* 30:128-131.
- Deplazes, P., Eckert, J., Mathis, A., von Samson-Himmelstjerna, G., and Zahner, H. 2016. *Parasitology in Veterinary Medicine*. Wageningen Academic Publishers, Wageningen, the Netherlands.
- Egweg, T. G., and Slocombe, J. O. D. 1982. Evaluation of the Cornell-Wisconsin centrifugation flotation technique for recovering trichostrongylid eggs from bovine feces. *Can. J. Comp. Med.* 46:133-137.
- Environmental Health Services, Centers for Disease Control and Prevention. 2019. Pictorial Keys to Arthropods, Reptiles, Birds, and Mammals of Public Health Significance. Accessed October 2019. [https://www.cdc.gov/nceh/ehs/publications/pictorial\\_keys.htm](https://www.cdc.gov/nceh/ehs/publications/pictorial_keys.htm)
- Foreyt, W. J. 1997. *Veterinary Parasitology Reference Manual*. Iowa State University Press, Ames, IA.
- Fowler, M. E. 2010. *Medicine and Surgery of Camelids*, 3rd ed. Wiley-Blackwell, Ames, IA.
- Garcia, L. S. 2016. *Diagnostic Medical Parasitology*, 6th ed. ASM Press, Washington, DC.
- Georgi, J. R., and Georgi, M. E. 1992. *Canine Clinical Parasitology*. Lea & Febiger, Philadelphia, PA.
- Greiner, E. C., and Ritchie, B. W. 1994. Parasites. In: *Avian Medicine: Principles and Application*, ed. B. W. Ritchie, G. J. Harrison, and L. R. Harrison, 1007-1029. Wingers Publishing, Lake Worth, FL.
- Hendricks, C. M. 2002. *Laboratory Procedures for Veterinary Technicians*, 4th ed. Mosby, St. Louis, MO.

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard.

© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

- Hoffman, G. L. 1999. *Parasites of North American Freshwater Fishes*. Cornell University Press, Ithaca, NY.
- Jacobs, D. E. 1986. *A Colour Atlas of Equine Parasites*. Gower Medical Publishing, London.
- Jacobson, E. R. 2007. *Infectious Diseases and Pathology of Reptiles*. CRC Press, Boca Raton, FL.
- Kaplan, R. M. 2020. Biology, epidemiology, diagnosis and management of anthelmintic resistance in gastrointestinal nematodes of livestock. *Vet. Clin. North Am. Food Anim. Pract.* 36:17-30.
- Kassai, T. 1999. *Veterinary Helminthology*. Butterworth-Heinemann, Oxford.
- Kaufmann, J. 1996. *Parasitic Infections of Domestic Animals*. Birkhäuser, Boston, MA.
- Kennedy, M. J., Mackinnon, J. D., and Higgs, G. W. 1998. *Veterinary Parasitology: Laboratory Procedures*. Alberta Agriculture, Food and Rural Development Publishing Branch, Edmonton.
- Klingenberg, R. J. 2016. *Understanding Reptile Parasites*, 2nd ed. Advanced Vivarium Systems, Irvine, CA.
- Levine, N. D. 1980. *Nematode Parasites of Domestic Animals and Man*, 2nd ed. Burgess Publishing, Minneapolis, MN.
- Little, S., Saleh, M., Wohltjen, M., and Nagamori, Y. 2018. Prime detection of *Dirofilaria immitis*: understanding the influence of blocked antigen on heartworm test performance. *Parasit. Vectors* 11:186.
- Lom J., and Dykova, I. 1992. *Protozoan Parasites of Fishes. Developments in Aquaculture and Fisheries Science, Volume 26*. Elsevier, Amsterdam.
- Ministry of Agriculture, Fisheries and Food. 1986. *Manual of Veterinary Parasitological Laboratory Techniques*. Reference Book 418. Her Majesty's Stationery Office, London.
- Mullen, G., and Durden, L. 2018. *Medical and Veterinary Entomology*, 3rd ed. Academic Press, New York.
- Neimester, R., Logan, A. L., Gerber, B., Egleton, J. H., and Kleger, B. 1987. Hemo-De as substitute for ethyl acetate in formalin-ethyl acetate concentration technique. *J. Clin. Microbiol.* 25:425-426.
- Nielsen, M. K., and Reinemeyer, C. R. 2018. *Handbook of Equine Parasite Control*, 2nd ed. Wiley Blackwell, Hoboken, NJ.
- O'Farrell, B. 2015. Lateral Flow Technology for Field-Based Applications—Basics and Advanced Developments. *Top. Companion Anim. Med.* 30:139-147.
- Owen, D. G. 1992. *Parasites of Laboratory Animals*. Royal Society of Medicine Services, London.
- Roberts, R. J. 2001. The parasitology of teleosts. In: *Fish Pathology*, 3rd ed., ed. R. J. Roberts, 254-296. W. B. Saunders, Philadelphia, PA.
- Samuel, W. M., Pybus, M. J., and Kocan, A. A. 2001. *Parasitic Diseases of Wild Mammals*, 2nd ed. Iowa State University Press, Ames, IA.
- Smith, S. A. 1996. Parasites of birds of prey: their diagnosis and treatment. *Semin. Avian Exot. Pet Med.* 5:97-105.
- Smith, S. A. 2002. Non-lethal clinical techniques used in the diagnosis of fish diseases. *J. Am. Vet. Med. Assoc.* 220:1203-1206.
- Smith, S. A., and Noga, E. J. 1992. General parasitology of fish. In: *Fish Medicine*, ed. M. K. Stoskopf, 131-148. W. B. Saunders, Philadelphia, PA.
- Smith, S. A., and Roberts, H. E. 2009. Chapter 8: Parasites of fishes. In: *Fundamentals of Ornamental Fish Health*, ed. H. E. Roberts, 102-112. Wiley-Blackwell, Hoboken, NJ.
- Sonenshine, D. E., and Roe, R. M. 2014. *Biology of Ticks*, 2nd ed. Oxford University Press, New York.
- Soulsby, E. J. L. 1965. *Textbook of Veterinary Clinical Parasitology*. F. A. Davis Co., Philadelphia, PA.
- Taira, N., Ando, Y., and Williams, J. C. 2003. *A Color Atlas of Clinical Helminthology of Domestic Animals*. Elsevier Science, Amsterdam.
- Taylor, M. A., Coop, R. L., and Wall, R. L. 2015. *Veterinary Parasitology*, 4th ed. Wiley Blackwell, Ames, IA.
- Telford, S. R. Jr. 2009. *Haemoparasites of the Reptilia*. CRC Press, Boca Raton, FL.

- Thienpont, D., Rochette, F., and Vanparijs, O. F. J. 1979. Diagnosing Helminthiasis through Coprological Examination. Janssen Research Foundation, Beerse, Belgium.
- Timsit, E., Leguillette, R., White, B. J., Larson, R. L., and Buczinski, S. 2018. Likelihood ratios: an intuitive tool for incorporating diagnostic test results into decision-making. *J. Am. Vet. Med. Assoc.* 252(11):1362-1366.
- U.S. Department of Agriculture. 1976. Ticks of Veterinary Importance. Agriculture Handbook 485. U.S. Government Printing Office, Washington, DC.
- Valkiunas, G. 2005. Avian Malaria Parasites and other Haemosporidia. CRC Press, Boca Raton, FL.
- van Wyk, J., and Mayhew, E. 2013. Morphological identification of parasitic nematode infective larvae of small ruminants and cattle: a practical lab guide. *Onderstepoort J. Vet. Res.* 80:539-553.
- Verocai, G. G., Chaudhry, U. N., and Lejeune, M. 2020. Diagnostic methods for detecting internal parasites of livestock. *Vet. Clin. North Am. Food Anim.* 36:125-143.
- Wall, R., and Shearer, D. 2001. Veterinary Ectoparasites: Biology, Pathology and Control. Blackwell Science, London.
- Weiss, D. J., and Wardrop, K. J. 2010. Schalm's Veterinary Hematology, 6th ed. Wiley-Blackwell, Ames, IA.
- Williams, R. E., Hall, R. D., Broce, A. B., and Scholl, P. J. 1985. Livestock Entomology. John Wiley & Sons, New York.
- Wilson, S. C., and Carpenter, J. W. 1996. Endoparasitic disease of reptiles. *Semin. Avian Exot. Pet Med.* 5:64-74.
- Woo, P. T. K. (ed.). 2006. Fish Diseases and Disorders, Volume 1: Protozoan and Metazoan Infections, 816. CABI Publishing, Oxfordshire, UK.



Page references given in *italics* indicate figures or tables

- abamectin, 98, 372, 377  
*Acanthocheilonema* spp.  
*A. dracunculoides*, 210, 211  
*A. reconditum*, 210, 211, 224, 225  
 acaricides. *see* ectoparasiticides  
*Acuaria* spp., 166  
*Adelina* spp., 23  
*Aelurostrongylus abstrusus*, 24, 26, 28,  
 43–45, 74, 75  
 afoxolaner, 375, 376  
 African blue louse, 302  
 African Coast fever, 230  
 African tampan, 298  
*Alaria* spp., 42–45  
 albendazole, 98, 372, 373, 373, 378  
 alpaca. *see* ruminants and camelids  
*Amblyomma* spp., 279, 281, 282, 283–285,  
 286  
*A. americanum*, 216, 280, 282, 283  
*A. cajennense*, 282, 284  
*A. hebraeum*, 282  
*A. maculatum*, 213, 280, 282, 284  
*A. variegatum*, 282, 285  
 American dog tick, 280, 290  
 American Heartworm Society, 209–210  
*Amidostomum* spp., 164  
*A. anseri*, 164  
 amitrax, 376, 377, 378  
 amprolium, 98, 379  
*Amyloodinium ocellatum*, 347, 356, 357  
*Anaplasma* spp., 245, 290, 292, 294  
 anaplasmosis, 278  
 anchor worm, 366, 367  
*Ancylostoma* spp., 42–45, 56, 57, 64, 67  
*A. braziliense*, 56  
*A. caninum*, 3, 56  
*A. ceylanicum*, 56  
*A. tubaeforme*, 56, 57  
 eggs, 17, 42, 56, 57, 64, 67  
*Angiostrongylus vasorum*, 24, 28, 76, 77  
 annelids, 193, 366, 367  
*Anocentor nitens*, 290, 291  
*Anoplocephala* spp., 126, 138, 139  
*A. magna*, 138  
*A. perfoliata*, 3, 127, 138  
 anthelmintics, 371–375, 372, 373  
 administration routes for, 372  
 anthelmintic efficacy testing (FECRT),  
 36–39  
 for dogs and cats, 44, 46  
 for horses, 127  
 persistency, 372  
 resistance to, 372, 374–375  
 for ruminants and camelids, 98  
 for swine, 141  
 withdrawal times, 373  
 antibiotics, as protozoal treatment, 378  
 antibody detection, 239, 241  
 antibody titer, 241–242  
 antigen detection, 239  
*Antricola* spp., 281  
*Aonchotheca* spp., 68, 69, 96, 97, 112, 113  
*A. bovis*, 112  
*A. longipes*, 112  
*A. putorii*, 43, 68, 69  
*Aponomma* spp., 281  
*Argas* spp., 281, 298, 299  
*A. persicus*, 298  
*A. reflexus*, 298  
*Argulus* sp., 362, 363  
 arsenicals, 374  
*Ascaridia* spp., 162, 163

*Veterinary Clinical Parasitology*, Ninth Edition. Anne M. Zajac, Gary A. Conboy, Susan E. Little, and Mason V. Reichard.

© 2021 John Wiley & Sons, Inc. Published 2021 by John Wiley & Sons, Inc.  
 Companion website: [www.wiley.com/go/zajac/parasitology](http://www.wiley.com/go/zajac/parasitology)

- Ascaris* spp.  
*A. lumbricoides*, 147  
*A. stam*, 140, 141, 146, 147  
*Ascarops strongylina*, 140, 150, 151  
*Aspicularis tetraptera*, 176, 177  
 assassin bugs, 195, 342  
*Atoxoplasma* spp., 154  
 Australian paralysis tick, 288  
 avian feather mites, 268, 269  
 avians. *see* birds
- Babesia* spp., 286, 288, 290, 292, 294, 295  
*B. bigemina*, 228, 229  
*B. bovis*, 228  
*B. caballi*, 228, 229, 290  
*B. canis*, 214, 215, 245, 292  
*B. canis canis*, 214  
*B. canis rossii*, 214  
*B. canis vogeli*, 214, 292  
*B. conradae*, 214  
*B. divergens*, 228  
*B. equi*, 228, 229  
*B. gibsoni*, 214, 215, 245  
*B. major*, 228  
*B. motasi*, 228  
*B. ovis*, 228  
*B. traubmanni*, 229  
*B. vulpes*, 214  
 in dogs/cats, 214, 215  
 in horses, 228, 229  
 PCR test for, 245  
 in ruminants, 228, 229, 292
- babesiosis  
 bovine, 228, 293  
 canine, 214, 294  
 equine, 228
- Baermann test  
 described, 14–15, 15  
 identification of nematode larvae  
 recovered from fecal samples,  
 24–29, 24–29
- Balantidium coli*, 140, 144, 145  
 barber pole worm, 108  
*Baylisascaris procyonis*, 62, 63, 65  
 bedbug, 344, 345  
 benchtop fecal flotation procedures, 7  
 benzimidazoles, 38, 373–374, 378  
 benzimidazole, 378
- Besnoitia* spp., 43, 204, 205  
*B. benetti*, 204, 205  
*B. besnoiti*, 204, 205  
 beta-cyfluthrin, 376  
 birds, 154–173, 234–237. *see also*  
 Chapter 5, Arthropod parasites  
 black fly, 200, 234, 338  
 blackhead disease, 158, 162  
 black-legged tick, 280, 288  
*Blankaartia* spp., 277  
 blood parasites, 207–238  
 birds, 211–213, 234–237  
 dogs/cats, 213–227  
 immunologic detection, 207  
 livestock/horses, 228–234  
 microscopic examination  
 for nematode parasites, 209–212  
 for protozoan parasites, 207–209
- blood smear  
 Giemsa stain, 208–209  
 technique for making, 208
- bloodworm (equine strongyles), 130  
 blow flies, 332, 333  
 blue tick, 292  
 body louse, human, 304  
 bont-legged tick, 286  
 bont tick, 282
- Boophilus* spp., 279, 281, 292  
*B. annulatus*, 292  
*B. decoloratus*, 292  
*B. microplus*, 292
- boric acid, 379
- Botrelia* spp., 288, 294  
*B. anserina*, 298
- bots, 322
- Bovicola* spp., 308, 309  
*B. bovis*, 300, 308, 309  
*B. breviceps*, 308  
*B. caprae*, 308  
*B. ovis*, 301, 308
- bovine preputial sample collection, 192  
 bovine theileriosis, 292  
 British dog tick, 288, 292  
 broad fish tapeworm, 86  
 brown dog tick, 213, 280, 292, 293  
 brown ear tick, 292  
 brown stomach worm, 108  
 bubonic plague, 320

- Bunostomum* spp., 30, 35, 108  
bush tick, 294  
*Buxtonella sulcata*, 108, 109
- Caligus* spp., 364  
*Calliphora* sp., 332  
Calliphoridae, 332  
*Camelostromylus* spp., 108  
camels/camelids. *see* ruminants and camelids  
camel tick, 286  
canines. *see* dogs  
*Capillaria* spp., 369  
  *Aonchotheca* (*Capillaria*) spp., 68, 69, 112, 113  
  *C. obsignata*, 160  
  *C. serpentina*, 186  
  *Eucoleus* (*Capillaria*) *aerophilus*, 66, 67, 69  
  in birds, 160, 161, 165  
  *Pearsonema* (*Capillaria*) *plica*, 194  
carbamates, 377-378  
carbaryl, 377  
carbol-fuchsin stain, 13  
*Caryospora* spp., 154, 155  
castor bean tick, 288  
cat flea, 315, 316, 317  
cats, 43-95, 193-195, 200-203, 209-227.  
  *see also* Chapter 5, Arthropod parasites  
  anthelmintic administration routes, 372  
  ectoparasiticide administration routes, 376  
  helminth eggs, larvae and protozoan cysts, 43  
  treatment for selected parasites, 45  
cattle. *see* ruminants and camelids  
cattle fever tick, 292  
cattle grub, 330  
cattle tail louse, 302  
Cayenne tick, 282, 284  
cecal worm, 162  
*Cediopsylla simplex*, 321  
centrifugal fecal flotation procedure, 6, 6-7  
*Centrorhynchus* spp., 173  
*Cephalopina titillator*, 330  
*Ceratomyxa* spp., 358  
*Ceratophyllus* spp., 318  
cervical alae, 65  
CF (complement-fixation) test, 241  
*Chabertia* spp., 30-32, 108  
Chagas' disease, 220, 242, 342  
*Cheilosporira* spp., 166  
*Cheyletiella* spp., 262, 263  
  *C. blakei*, 262  
  *C. parasitovorax*, 262  
  *C. yasguri*, 262  
  superficial scraping for, 249  
chickens. *see* birds  
chiggers, 276, 277  
*Chilomastix* spp., 158, 159  
chinchies, 342  
*Chirodiscoides caviae*, 266, 267  
*Choanotaenia* spp., 170  
*Choriops* spp., 248, 259  
  *C. bovis*, 254, 255  
*Chrysoma bezziana*, 334  
*Chrysops* spp., 236, 339  
*Cimex* spp., 344, 345  
  *C. adjunctus*, 344, 345  
  *C. hemipterus*, 344  
  *C. lectularis*, 344, 345  
*Cittotaenia* spp., 182, 183  
clindamycin, 378  
*Clinostomum*, 363  
clopidol, 379  
clorsulon, 98, 374  
closantel, 98, 372, 374  
coccidia  
  in birds, 154, 155  
  in dogs and cats, 46, 47  
  in horses, 128, 129  
  in rodents and rabbits 174, 175  
  in ruminants and camelids, 99-104  
  in swine 142, 143  
coccidiostats, 378-379  
*Cochliomyia* spp., 332, 335  
  *C. hominivorax*, 334  
*Cochlosoma* spp., 158  
*Columbicola columbae*, 313  
Companion Animal Parasite Council (CAPC), 209  
complement-fixation (CF) test, 241  
conenose bugs, 342  
*Contracecum* spp., 162

- Cooperia* spp., 108  
*C. oncophora*, 34  
 larvae, 30–31, 33, 34  
 copper oxide wire particles (COWP), 379  
 coprophagy and spurious parasites, 19,  
 23, 24  
 corridor disease, 230  
 coumaphos, 377  
 crab louse, 304  
*Crenosoma vulpis*, 42, 74, 75  
 identification in fecal samples, 24, 27,  
 27, 28  
 larvae, 27, 27, 28, 74, 75  
*Cryptocaryon irritans*, 347, 350, 351  
*Cryptocotyle lingua*, 92, 93  
*Cryptosporidium* spp.  
 in birds, 156, 157  
*C. andersoni*, 106  
*C. baileyi*, 156  
*C. hovis*, 106  
*C. canis*, 50, 51  
*C. felis*, 50, 51  
*C. meleagridis*, 156  
*C. parvum*, 50, 98, 106, 142  
*C. serpentis*, 182  
*C. ubiquitum*, 106  
*C. xiaoi*, 106  
 in dogs/cats, 42, 43, 50, 51  
 fecal flotation solution choice and, 4, 4  
 in horses, 126, 128, 129  
 microscopic examination, 16  
 PCR test for, 245  
 in reptiles, 182  
 in rodents, 174  
 in ruminants/camelids, 96–97, 106, 107,  
 127  
 scanning fecal flotation slides for, 7  
 stains for, 12–13  
 in swine, 140, 142  
*Ctenocephalides* spp., 224, 262  
*C. canis*, 315, 316, 317  
*C. felis felis*, 315, 316, 317, 318  
 cucumber seed tapeworm, 80  
*Culicoides* spp., 236, 341  
 culture, fecal, 29–30  
*Cuterebra* spp., 322–323, 324, 325  
*Cyathostoma* spp., 164  
 cyathostomes, 130  
*Cyclops*, 200  
 cypermethrin, 376  
 cyphenothrin, 376  
 cyromazine, 377  
*Cystocaulus* spp., 29  
*Cystosporospora* spp., 46, 47, 49, 64,  
 83, 140  
*C. burrowsi*, 46  
*C. canis*, 42, 46, 46, 49  
*C. felis*, 43, 46  
*C. neorivolta*, 46  
*C. ohioensis*, 42, 46, 46  
*C. rivolta*, 43, 46, 49  
*C. suis*, 140, 141, 142, 143  
 in cats, 45–47, 46  
 in dogs, 44, 46, 46, 47  
 oocysts, 17, 46, 46, 47, 64  
*Cytauxzoon felis*, 216, 217, 245  
  
*Dactylogyrus* spp., 360  
*Damalinia* spp., 308  
*Davainea* spp.  
 in birds, 170  
*D. proglottina*, 170  
 decoquinate, 98, 379  
 deer fly, 236, 338, 339  
 deer tick, 280, 288, 289  
 deltamethrin, 376, 376  
 demodocosis, 249, 261  
*Demodex* spp., 249, 260, 260–261  
*D. canis*, 261  
 skin scraping for, 249, 261  
*Dermacentor* spp., 228, 279, 279, 281,  
 290, 290–291, 297  
*D. albipictus*, 290, 291  
*D. andersoni*, 290, 291  
*D. nitens*, 290, 291  
*D. occidentalis*, 290  
*D. reticularis*, 214, 290  
*D. variabilis*, 216, 280, 290, 290  
*Dermatophytosis gallinae*, 271, 272, 273  
*Dermatobia hominis*, 322, 326, 327  
 derquantel, 98, 374  
 diagnostic accuracy of immunodiagnostic  
 methods, 242, 242  
 diazinon, 377  
 dichlorvos, 141, 373, 374, 377  
 diclazuril, 379  
*Dicrocoelium dendriticum*, 96, 97, 122,  
 123, 152

- Dictyocaulus* spp., 118, 119, 137  
*D. arnfieldi*, 28, 126–127, 136, 137  
*D. cameli*, 118  
*D. filaria*, 29, 97, 118, 119  
*D. viviparus*, 96, 98, 118, 119  
 identification in fecal samples, 24, 28–29
- diffubenzuron, 376, 377
- dinotefuran, 375, 376
- Dioctophyme renale*, 193, 193
- Dipartiella* spp., 354
- Dipetalonema reconditum*, 210, 224, 225
- Diphyllobothrium latum*, 42, 44–45, 86, 87
- Dipylidium* spp., 85, 87  
*D. caninum*, 42–45, 80, 81, 310  
*Trichodectes* as intermediate host for, 310
- direct hemagglutination (HA) test, 241
- direct smear  
 fecal, 12–13
- Dirofilaria* spp.  
 characteristics, 211  
*D. immitis*, 209–210, 211–212, 222, 223, 242  
 anthelmintics for, 374, 375  
 antigen test, 240–241  
 characteristics, 211  
*D. repens*, 210, 211, 225, 226, 227  
*D. striata*, 210, 211, 222  
 tests for microfilariae in blood samples, 209–212, 222–226
- Dispharynx* spp., 166
- dog flea, 315
- dogs, 42–95, 193–195, 200–203, 209–227.  
 see also Chapter 5, Arthropod parasites  
 anthelmintic administration routes, 372  
 ectoparasiticide administration routes, 376  
 helminth eggs, larvae and protozoan cysts, 42  
 treatment for selected parasites, 44
- donkeys. see horses
- doramectin, 44, 98, 141, 372, 372, 373
- double-pored tapeworm, 80
- dourine, 230
- Dracunculus*  
*D. insignis*, 200, 201  
*D. medinensis*, 200
- Draschia megastoma*, 127, 136
- dwarf dog tapeworm, 84
- dwarf tapeworm of humans, 180
- ear mange mite (rodents), 252
- ear mite, 258, 258
- East Coast fever, 230
- Echinophaga gallinacea*, 318, 319
- Echinococcus* spp., 42–45, 84, 85, 87  
*E. canadensis*, 84  
*E. granulosus*, 84  
*E. multilocularis*, 84
- Echinoparyphium* spp., 168
- Echinostoma* spp., 168
- Echinuria* spp., 166
- ectoparasite recovery techniques, in fish, 347–349  
 fin biopsy (fin snip), 348, 349  
 gill biopsy (gill snip), 348–349, 349  
 skin biopsy (mucus smear), 348, 348
- ectoparasiticides, 375–378, 376, 377  
 administration routes in large animals, 377  
 administration routes in small animals, 376  
 carbamates, 377–378  
 isoxazolines, 375  
 neonicotinoids, 375–376  
 organophosphates, 377–378  
 pyrethrins and pyrethroids, 376–377
- egg-counting (quantitative) fecal flotation procedures, 8–12  
 mini-FLOTAC test, 9, 10  
 modified McMaster test, 8, 8–9  
 modified Stoll test, 11–12  
 sensitivity of test, 8  
 Wisconsin, Cornell–Wisconsin (double centrifugation) test, 11
- Ehrlichia* spp., 278, 282, 288, 292, 294  
*E. canis*, 292  
*E. raninantium*, 282  
 PCR test for, 245
- Eimeria* spp.  
*E. ahata*, 99  
*E. aironi*, 99, 102  
*E. alabamensis*, 99, 100–101  
*E. alijevi*, 102  
*E. aspheronica*, 102  
*E. auburnensis*, 96, 100–101

- Eimeria* spp. (cont'd)
- E. bakuensis*, 99
  - E. bovis*, 96, 99, 100–101
  - E. brasiliensis*, 100–101
  - E. bukidinonensis*, 100–101
  - E. canadensis*, 100–101
  - E. caprina*, 99, 102
  - E. christenseni*, 99, 102
  - E. crandallii*, 102
  - E. cylindrical*, 100
  - E. debliki*, 142
  - E. ellipsoidalis*, 100
  - E. granulosa*, 102
  - E. hirci*, 102
  - E. intricata*, 102–104
  - E. ivitaiensis*, 105
  - E. jolchejevi*, 102
  - E. leuckartii*, 126–127, 128, 129
  - E. llamae*, 105
  - E. macusaniensis*, 99, 105
  - E. nieschulzi*, 175
  - E. ninakohlyakimovae*, 99, 102–104
  - E. ovinoidalis*, 99, 102–104
  - E. pallida*, 102
  - E. parva*, 102
  - E. pellita*, 100
  - E. porci*, 142, 143
  - E. punctata*, 102
  - E. scabra*, 142, 143
  - E. spinosa*, 142
  - E. stiedae*, 174
  - E. subspherica*, 100
  - E. wyomingensis*, 100–101
  - E. zuernii*, 96, 99, 100–101
  - in birds, 154, 154–155
  - in dog/cat feces, 49
  - in horses, 126, 128, 129
  - in rabbits, 174, 175
  - in reptiles, 182, 184
  - in rodents, 174, 175
  - in ruminants/camelids, 96–98, 98, 99, 104–105, 109, 115
  - in swine, 140, 142, 143
- elephant skin disease, 204, 205
- ELISA. *see* enzyme-linked immunosorbent assay (ELISA)
- elk tick, 290
- Elokomin fluke fever, 90
- emodepside, 45, 372, 374
- endemic relapsing fever, 298
- endoparasite recovery, in fish, 349
- Entamoeba* spp.
- E. imadensis*, 182, 183–184
  - in reptiles, 182, 183–184
- enzyme-linked immunosorbent assay (ELISA), 207, 209, 239–241, 240–241
- for *Giardia*, 52
  - for heartworm, 209
- Epistylis* spp., 354, 355
- E. coltsarum*, 354
  - E. hoeffii*, 354
- eprinomectin, 45, 98, 372, 372, 373
- epsiprantel, 44, 45, 374
- equids. *see* horses
- equine protozoal myeloencephalitis, 378, 379
- equine piroplasmosis, 282, 291
- Ergasilus* spp., 364, 365
- esfenvalerate, 376
- esophageal worm, 70
- etofenprox, 376
- Eucoleus* spp., 42–44, 66, 67–69, 83, 160, 161
- E. dupar*, 161
  - E. aerophilus*, 43, 66, 67, 69
  - E. boehmi*, 66, 67, 69
- European chicken flea, 318
- European sheep tick, 288
- Eurytrema* spp., 94
- E. coelomaticum*, 124
  - E. pancreaticum*, 124, 152
  - E. procyonis*, 92
- Eutrombicula* spp.
- E. alfreddugesi*, 276
  - E. splendens*, 276
- eye worm, 202
- face louse, 302
- Fasciola* spp.
- in bile or pancreatic duct, 92
  - F. gigantica*, 120
  - F. hepatica*, 92, 96–98, 120, 121, 374
  - in swine, 152, 153
- Fascioloides magna*, 123
- feather mites, 268, 269

- feather picking, 158
- febantel, 44, 45, 98, 372, 373, 378
- fecal antigen test, for *Giardia*, 52
- fecal egg count reduction tests (FECRTs), 36-38
- interpretation of results, 38
  - test drugs and collection of posttreatment samples, 37-38
  - test groups and selection of animals, 37
- fecal exam procedures
- Baermann test, 14-15, 15
  - direct smear, 12-13
  - fecal flotation, 3-12
  - fecal sedimentation, 13-14
    - benefits of, 13
    - centrifugal sedimentation test, 13-14
    - Flukefinder® apparatus, 13
    - simple test, 13
  - identification of adult worms, 38-39
  - immunologic and molecular methods, 15
  - microscope use, 16-19
  - pseudoparasites and spurious parasites
    - found in, 19, 20-23, 24
    - free-living nematodes, 20
    - insect larvae, 22
    - insect/plant hairs, 21
    - mites, free-living, 22
    - parasite eggs/cysts, 23
    - pollen grains, 20
    - yeast, 21
  - quality control, 15-16
  - stained smear, 12-13
  - techniques for evaluation of strongylid nematodes in grazing animals, 29-38
  - fecal culture, 29-30
  - fecal egg count reduction test (FECRT), 36-38
  - identification of horse third-stage larvae, 35-36, 36
  - identification of ruminant and camelid third-stage larvae, 30-35, 30-35
- fecal flotation procedures, 5-12
- benchtop, 7
  - centrifugal, 6, 6-7
  - passive, 7
  - principle of, 5
  - quantitative (egg-counting), 8-12
    - mini-FLOTAC test, 9, 10
    - modified McMaster test, 8, 8-10
    - modified Stoll test, 11-12
    - sensitivity of test, 8
    - Wisconsin egg-counting test (double centrifugation procedure), 11
  - simple, 7
  - slide preservation, 2
  - slides, 7
  - solution choice and preparation, 3-5, 4
    - advantages/disadvantages of common solutions, 4
    - magnesium sulfate solution, saturated, 4, 4-5
    - Sheather's sugar solution, 4, 4-5
    - sodium chloride solution, saturated, 4, 4-5
    - sodium nitrate solution, 3, 4
    - specific gravity of solutions, 3
    - zinc sulfate solution (33%), 3-5
- fecal samples
- collection of, 1-2
  - fecal culture, 29-30
  - shipment, 2
  - storage, 2
- FECRTs. *see* fecal egg count reduction tests (FECRTs)
- Felicola* spp., 80
- F. subrostratus*, 310, 311
- feline chewing louse, 311
- feline mange mite, 252
- felines. *see* cats
- fenbendazole, 44, 45, 98, 127, 141, 372, 373, 373, 378
- Filaroides* spp.
- F. hirshi*, 24, 28, 76, 77
  - F. milkst.*, 76
- Filicollis* spp., 172
- filter test, for microfilariae, 212, 222, 226
- fin biopsy (fin snip), 348, 349
- fipronil, 375, 376
- fish, 347-369
- fish louse, 362, 363
  - fixatives, 2
  - Flavivirus*, 288
  - fleas, key for identification, 315

- flea tapeworm, 80  
 flesh flies, 332  
 flotation slides, 7  
 flotation solutions, 3-5, 4  
   advantages/disadvantages of common solutions, 4  
   magnesium sulfate solution, saturated, 4, 4-5  
   Sheather's sugar solution, 4, 4-5  
   sodium chloride solution, saturated, 4-5, 6  
   sodium nitrate solution, 3, 4  
   specific gravity of solutions, 3  
   zinc sulfate solution (33%), 3-5, 4  
 flubendazole, 373  
 Flukefinder® apparatus, 13  
 flumethrin, 376, 376  
 fluralaner, 375, 376  
 fly strike/fly blow, 322, 332, 332-333  
 follicle mite, 260  
 foot louse of sheep, 302  
 foot mange, 254  
 formalin, 2, 39, 211  
 fowl paralysis, 298  
 fowl tick, 264, 266, 267  
 fox lungworm, 66  
 fox tapeworm, 84  
*Francisella tularensis*, 282, 295  
 French heartworm, 76  
 freshwater fish, 347-369  
 freshwater white spot, 350, 351  
 fur mites  
   guinea pigs, 266  
   rabbits, 264  
   rodents, 266, 267  
 gapeworm, 164  
*Gasterophilus* spp., 322, 328, 328-329  
   *G. haemorrhoidalis*, 328  
   *G. intestinalis*, 328, 329  
   *G. nasalis*, 328  
   *G. pecorari*, 328  
 genital primordium, 25, 28  
 giant kidney worm, 193  
*Giardia* spp., 52, 53-55  
   in birds, 158  
   centrifugation procedure, 5  
   cysts, 13, 42, 43, 47, 52, 53-54, 96-97, 106, 107, 126, 128, 129, 158  
   direct smear, 12  
   in dogs/cats, 42-45, 47, 52, 53-55  
   fecal flotation solution choice and, 3-4, 4  
   *G. bovis*, 106  
   *G. canis*, 52  
   *G. cati*, 52  
   *G. duodenalis*, 142  
   *G. duodenalis*, 52, 128, 129, 245  
   *G. intestinalis*, 52, 106  
   *G. lamblia*, 52, 106  
   of horses, 126-127, 128, 129  
   microscopic examination, 16  
   in rodents, 174  
   in ruminants/camelids, 96-97, 106, 107  
   in swine, 142  
   trichomonads confused with, 50, 53  
   trichrome stain for, 13  
   trophozoites, 2, 12, 52, 55, 158  
 Giemsa stain  
   for blood smears, 208-209  
   for fecal smears, 13  
 gill biopsy (gill snip), 348-349, 349  
 gill louse, 364, 365  
*Gliricicola porcelli*, 306, 307  
*Globocephalus* spp., 146  
*Glossina* spp., 232, 340  
 goats. *see* ruminants and camelids  
 gotch ear, 282  
*Graphidium strigosum*, 180  
 grazing animals, techniques for evaluation of strongylid nematodes in, 29-38  
 grubs, 322  
 Gulf Coast tick, 213, 280, 282, 284  
*Gyalocephalus* spp., 36  
*Gyrodactylus* spp., 360  
*Gyropus ovalis*, 306  
*Habronema* spp., 126, 136, 137  
   *H. microstoma*, 127, 136  
   *H. muscae*, 127, 136  
*Haemaphysalis* spp., 230, 279, 281, 294, 294-295  
   *H. leachi*, 214, 294  
   *H. leporispalustris*, 294, 295  
   *H. longicornis*, 280, 294, 295  
   *H. punctata*, 294

- Haematobia* spp., 198, 199  
*H. irritans*, 339-340
- Haematopinus* spp., 302, 303  
*H. asini*, 302  
*H. eurystermus*, 302  
*H. quadripertusus*, 302  
*H. suis*, 302, 303
- Haematoloecus*, 189
- Haemonchus* spp., 108  
*H. contortus*, non-traditional treatments for, 379  
 larvae, 31, 33, 35  
 morphological characteristics of infective third-stage larvae, 30
- Haematoproteus* spp., 236, 237  
*H. columbae*, 236  
*H. meleagridis*, 236  
*H. nettionis*, 236  
*H. sacharovi*, 236
- halofuginone lactate, 98
- Hammondia* spp., 48  
*H. hammondi*, 43
- Hapalotremia*, 190
- hard ticks. *see* ticks, hard
- harvest mite, 276
- HA (direct hemagglutination) test, 241
- head louse, human, 304
- heartwater, 285
- heartworm  
 antigen test, 209-210, 222, 240-241  
 in cats, 210, 222  
 French, 76  
 microfilariae, test for in blood samples, 209-212  
 filter test, 212, 222, 226  
 hematocrit test, 211, 211-212  
 modified Knott's test, 212, 222, 226  
 wet mount, 210-211
- hedgehog tick, 288
- Heligmosomoides polygyrus*, 180
- hematocrit test, for microfilariae, 211, 211-212
- Hemirichodina* spp., 354
- Henneguya* spp., 347, 358, 360, 361  
*H. ictaluri*, 360
- Hepatozoon* spp., 213, 213, 245  
*H. americanum*, 213, 213, 282, 379  
*H. canis*, 213
- Heterakis* spp., 162, 163, 178  
*H. isolonche*, 162  
*H. spumosa*, 178
- Heterobilharzia americana*, 44, 90, 91
- Heterodoxus* spp., 224  
*H. spinigera*, 310
- Hexanetra*, 187
- Hexamita* spp., 158
- Hippobosca* spp., 336  
*H. longipennis*, 336  
*H. variegata*, 336
- hippoboscid flies, 236, 336, 337
- Hirudinea (leeches), 366, 367
- Histomonas* spp., 158  
*H. meleagridis*, 162
- hookworms  
*Ancylostoma* spp., 56, 57, 64, 67  
 strongylid parasites, of ruminants and camelids, 108  
*Uncinaria stenocephala*, 29
- horn fly, 198, 199, 338, 339-340
- horse fly, 338, 339
- horses, 126-139, 198-199, 202-205, 228-235. *see also* Chapter 5, Arthropod parasites  
 anthelmintic administration routes, 372  
 helminth eggs, larvae and protozoan cysts, 126  
 insecticide and acaricide administration routes, 377  
 treatments for selected parasites, 127
- horse tick, 290
- Hoyer's solution, 39
- human body louse, 304
- human bot fly, 326
- human flea, 315, 318, 319
- human head louse, 304
- Hyalomma* spp., 228, 230, 286, 287  
*H. aegyptium*, 286
- hygromycin B, 141, 374
- Hymenolepis* spp., 170, 180, 181, 183  
*H. diminuta*, 180  
*H. microstoma*, 180  
*H. nana*, 180, 181
- Hyostrongylus rubidus*, 141, 146, 147
- Hypoderma* spp., 330, 331  
*H. bovis*, 322-323, 330  
*H. lineatum*, 322, 330, 331

- ich, 347, 350, 351  
*Ichthyobodo (Costia) necator*, 347, 358, 359  
*Ichthyophthirius multifiliis*, 347, 350, 351  
 IFA (indirect fluorescent antibody), 52, 207, 241, 241  
 IHA (indirect hemagglutination) test, 241  
 imidacloprid, 375, 376, 377  
 imidocarb, 378  
 immunochromatographic tests, 239–240  
 immunodiagnostic methods, 15, 207, 239–242, 240–242  
   for blood parasites, 207  
   diagnostic accuracy, 242, 242  
   enzyme-linked immunosorbent assay (ELISA), 239–241, 240–241  
   on fecal samples, 15  
   indirect fluorescent antibody (IFA), 241, 241  
 indirect fluorescent antibody (IFA) test, 52, 207, 241, 241  
 indirect hemagglutination (IHA) test, 241  
 indoxacarb, 375, 376, 377  
 InPouch™ TF-Feline culture system, 52  
 insect growth regulators, 375–376  
 insect hair, 21  
 insecticides. *see* ectoparasiticides  
 intestinal threadworm, 72  
 ionophores, 379  
*Isospora* spp.  
   in birds, 154, 155  
   in reptiles, 182, 184  
 isoxazolines, 375  
 itch mite, 250  
 itchy heel, 254  
 ivermectin, 38, 45, 98, 127, 141, 372, 372, 373  
*Ixodes* spp., 228, 279, 281, 288, 289, 294  
   *I. canisuga*, 288  
   *I. hexagonus*, 288  
   *I. holocyclus*, 288  
   *I. pacificus*, 288  
   *I. ricinus*, 288  
   *I. rubicundus*, 288  
   *I. scapularis*, 280, 288, 289  
*Kallicephalus* sp., 187  
 kennel tick, 292  
 kidney worm, 194  
 Kinyoun stain, 13  
 kissing bugs, 220, 342  
*Knemidokoptes* spp., 248, 252, 253  
 Knott's test, modified, 211–212, 222, 226  
*Kudoa* spp., 358  
 lactophenol, 39–40  
*Lamaneia* spp., 108  
 lambda-cyhalothrin, 376  
 larvae. *see also* specific parasites  
   cat parasites, 43  
   cattle parasites, 96  
   dog parasites, 42  
   identification of nematode larvae recovered with fecal flotation or Baermann procedures, 24–29  
   sheep and goat parasites, 97  
 lasalocid, 98, 379  
 leeches, 193, 366, 367  
 leg mange, 254  
 leishmaniasis, 218  
*Leishmania* spp., 218, 219  
   *L. braziliensis*, 218  
   *L. chagasi*, 218  
   *L. donovani*, 218  
   *L. infantum*, 218  
   *L. mexicana*, 218  
   *L. tropica*, 218  
   PCR test for, 245  
*Lepeophtheirus* spp., 364, 365  
   *L. salmonis*, 365  
*Leporacarus (Listrophorus) gibbus*, 264, 265  
*Lernaea* spp., 366, 367  
*Leucocytozoon* spp.  
   in birds, 234, 235  
   *L. caulleryi*, 234  
   *L. simondi*, 234  
   *L. smithi*, 234  
 levamisole, 38, 98, 141, 372, 373, 374  
 lice, detection of, 300  
*Linguatula* spp., 94, 95  
*Linognathus* spp., 224, 300, 302, 303, 304, 305  
   *L. africanus*, 302, 303  
   *L. ovis*, 302  
   *L. pedalis*, 302  
   *L. setosus*, 302, 303  
   *L. vituli*, 302  
*Lipeurus caponis*, 312

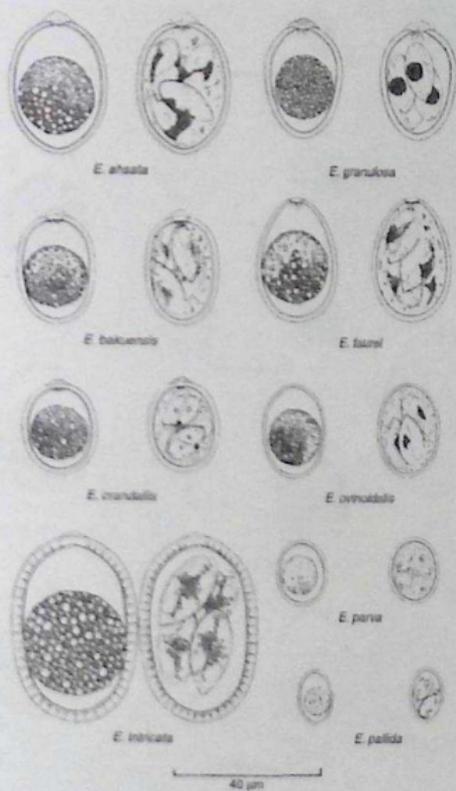


Fig. 7.78 Unsporulated and sporulated oocysts of nine species of *Eimeria* of sheep (x1000). (from Joynt LP, Norton CC, Davies SH, Watkins CV. The species of coccidia occurring in cattle and sheep in the southwest of England. *Parasitology* 56:533, 1966. Crown copyright. Reproduced with permission from the Controller of Her Britannic Majesty's Stationery Office.)

(Fig. 7.60). Cystosporidians such as small subspherical oocysts, roughly 5 µm in size. Excavates include *Entamoeba polecki* (single nucleated cysts 5–11 µm in diameter), *Iodamoeba butschlii* (cysts 8–10 µm long), *Endolimax nana* (oval cysts are 5–14 µm long), *Giardia* (cysts roughly 10 µm long), and others. The very common ciliate *N. coli* (Fig. 7.61), with its large kidney-shaped macronucleus, will most likely be seen in the cyst stage, which ranges in size from 40 to 60 µm in diameter. Other than the species of *Eimeria*, *Cystosporon*, and *Cryptosporidium*, most of these parasites will not be readily recognizable in sugar floatations owing to distortion.

There are a number of common eggs found in pig feces that include nematodes and an acanthocephalan (Fig. 7.79), and see

(Fig. 7.69). The fertile eggs of the ascariid *A. suum* have a rough, bile-stained, external protein layer, and eggs are 56 to 60 by 40 to 60 µm. Infertile *A. suum* eggs can be common and appear a little longer and thinner than the fertilized eggs; the middle wall of the shell tends to be thinner, and the central portion looks disorganized. The spirurids *Acarops* and *Physoclephas* produce thick-walled eggs; both produce larvated eggs in the 31 to 45 by 12 to 26 µm range. *Strongyloides ransomi* (Rhabditida) eggs resemble those of *S. papillorum* and are thin-shelled, larvated, and 45 to 55 by 26 to 35 µm in size (Fig. 7.70), and see (Fig. 7.69). Strongyle eggs in pig feces may represent infection with the trichostrongylid *Trichostrongylus rufus* (60–76 by 31–38 µm in size), the strongyloid species

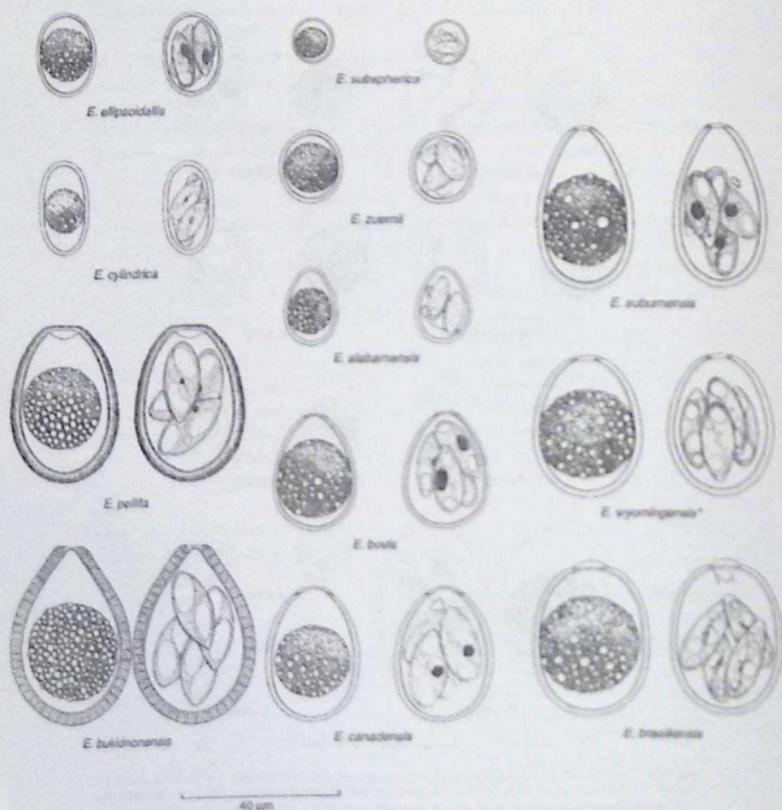


Fig. 7.80 Unsporulated and sporulated oocysts of 12 species of *Eimeria* of cattle (x1000). (From Joyner LB; Norton CC, Davies SFM, Watkins CV. The species of coccidia occurring in cattle and sheep in the southwest of England, *Parasitology* 56:536, 1966. Crown copyright. Reproduced with permission from the Controller of Her Britannic Majesty's Stationary Office.) \*Resembles *E. bukionensis*.

of *Oesophagostomum* (50–80 by 35–70 µm), or the ancylostomatids *Glabrocephala strutschalis* (52–56 by 26–35 µm), or *Necator americanus* (60–76 by 30–40 µm), but most commonly with only the first two. The metastrongyloid parasitic organism in swine is unusual compared with many in that it has an earthworm rather than a molluscan intermediate host, and unlike most metastrongyloids of domestic animals, *Metastrongylus apri*, *Metastrongylus salmi*, and *Metastrongylus pulegioides* eggs are small and subglobular and

contain a larva. Eggs of the first two species are indistinguishable and measure 43 to 57 by 38 to 41 µm in size, whereas the eggs of *M. pulegioides* are slightly larger: 57 to 64 by 39 to 45 µm in size. *Trichostrongylus axei* (Trichinelloidea) living in the mucosa of the cecum and colon produces eggs that are typical of the genus, and almost identical to the *Trichostrongylus trichuris* of humans, but much smaller than the eggs of the dog whipworm, *T. vulpis*. The eggs of *T. axei* measure 50 to 56 by 25 to 21 µm. *Macracanthorhynchus hiracinarum*

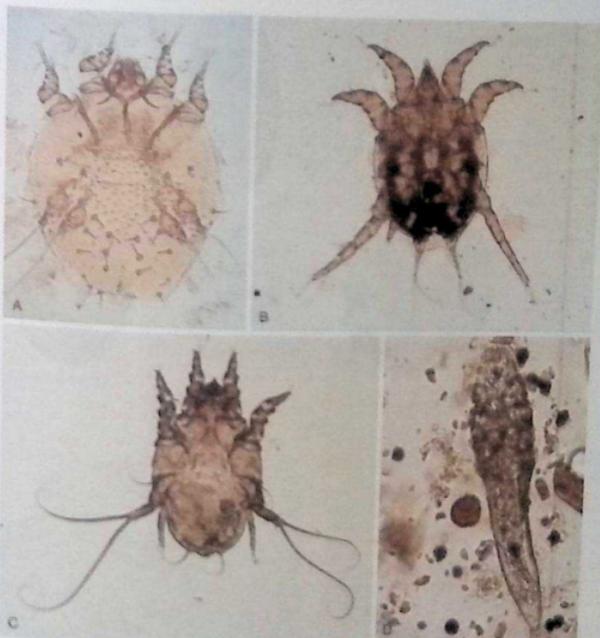


Fig. 7.81 Common mites of large animals. (A) *Sarcopptes scabiei* (x100). (B) *Phoropses* (x40). (C) *Chorioptes* (x40). (D) *Demodex bovis* (x200).

(Acanthocephala) eggs (87–110 by 40–65  $\mu\text{m}$ ) have three concentric, ellipsoidal shells surrounding the scarabur embryo.

The eggs of *S. slanskyi* (*Stenostrongyloides*) are large (90–120 by 45–70  $\mu\text{m}$ ) and morulated and are found in urine samples from infected swine. The last urine voided contains the highest concentration of eggs.

#### Examination for Trichinae

*Trichinella spiralis* infections can be diagnosed by procedures described earlier in this chapter by “squash” preparations of bits of muscle between two glass slides and scanning under low power, or by peptic digestion of tissue. The diaphragm and masseter muscles are especially likely to yield positive findings. Newly laid larvae are approximately 100  $\mu\text{m}$  in size but grow extensively (to 745–975  $\mu\text{m}$  in length) once they have settled in striated muscle. *Trichinella* larvae can be easily identified by their long stichosome esophagus (spanning almost two-thirds of their body length) and their terminal anus (Fig. 8.76). When observed in

a muscle biopsy, the presence in a thick, fibrous capsule around the coiled larva is also characteristic (see Fig. 8.77).

#### Identification of Ectoparasites

Common lice, fleas, and mites associated with piglets shown in Figs. 7.79, 7.80, and 7.81. In the case that a specimen does not match one of these illustrations (as may happen when transient or uncommon infestations are acquired from other hosts), it may be necessary to seek the advice of an expert.

Pigs not housed indoors may be affected by ticks in the same manner other production animals are, and tick identification to the genus level can often be achieved through the examination of the mouthparts and scutum of the specimen in question (Fig. 8.78). As for mites, pigs are generally host to only two *Demodex* spp., *D. phylloides* and *S. scabiei*. Both genera morphologically resemble the hosts we have already discussed; genera morphologically resemble the hosts we have already discussed: one is elongate, with mouthparts and legs all clustered in the anterior

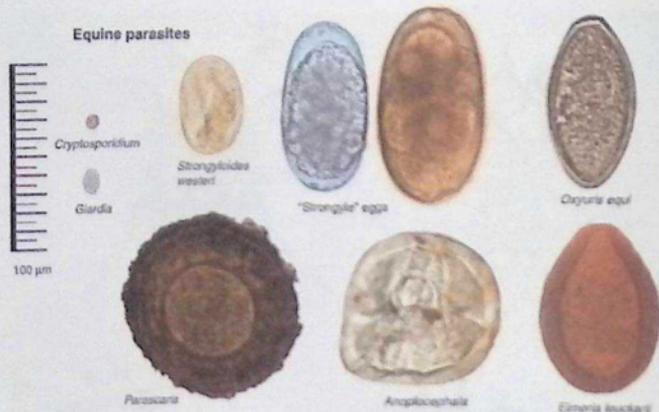


Fig. 7.82 Equine parasites in freshly voided feces from infected animals.

half, and the other is round, with long unsegmented pretarsi on its front legs and triangular thickened setae on its dorsum.

Insect parasites of pigs include the flies, *Musca* and *Stomoxys*, as well as flesh flies that may cause fly-strike. In case of the latter, the spiracular plates can give good indication of the affinities of the fly in question. *Haematopinus suis* is the only louse of pigs and is characteristic of the group in its narrow head with tarsi of equal size. Fleas that may be found in association with pigs include the human flea, *P. irritans*, which has a rounded head and lacks combs *gallinae*, and the stick-tight flea, *E. gallinae*, with its flat head.

### PARASITES OF LABORATORY ANIMALS

Many parasites lose all opportunity to complete their life histories the day their host becomes a member of a laboratory animal colony (Fig. 7.83). Although they may limit the usefulness of their immediate hosts as experimental subjects, such parasites present no continuing



Fig. 7.83 *Eimeria leuckarti* unsporulated (left) and sporulated (right) oocysts (x425).



Fig. 7.84 Commensal ciliates recovered from a horse's fecal sample (x200).

problem of control. Heartworm infection, for example, renders a dog unfit for experiments involving the circulatory or respiratory system but, in the absence of mosquitoes, must remain confined to the host in which it arrived. However, a surprising variety of arthropod, protistan, and helminth parasites do succeed in maintaining impressive populations even in reasonably hygienic laboratory animal colonies. Hair-clipping mites, mucedoniflagellates, coccidians, *Hymenolepis tapeworms*, and pinworms are particularly common.

A few of the more common parasites of rodents and rabbits are represented in Fig. 7.86.

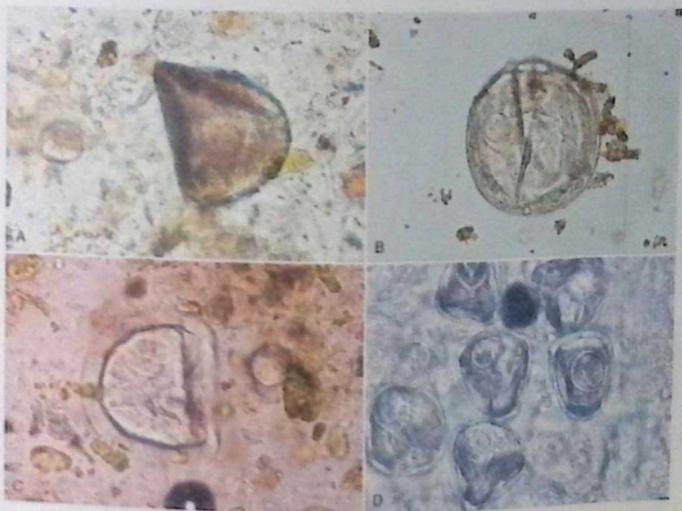


Fig. 7.85 Eggs of *Anisoplophora parvifolia* in three different preparations. (A) Egg in water. (B) Egg in a zinc sulfate centrifugal flotation. (C,D) Eggs in a centrifugal flotation, viewed with brightfield (C) and Nomarski (D) optics. The eggs of *Anisoplophora magna* and *Paranisoplophora mammalis* are similar, but the eggs of *P. mammalis* are only three-fourths as large as these.

The annotated host-parasite listings for these hosts in the section to follow are by necessity, an incomplete outline including only the common parasites of laboratory rabbits, rats, mice, guinea pigs, monkeys, and apes. It bears noting that the kinds of primate parasites to be found depend on the species and geographic origin of the monkey and on the duration and environmental conditions of its captivity. Certain parasites (e.g., *Strongyloides* and *Cercophagostomum*) flourish in captive monkeys. Others, especially those whose natural intermediate hosts are no longer available, tend to fade away. In united colonies, parasites that are not discriminating in their selection of hosts may spread to species of monkeys that, for geographic or ecologic reasons, they rarely or never infect in the wild. Such cross-infections are more likely to cause disease because of the lack of mutual adaptation of host and parasite. The annotated host-parasite listing in the section to follow, therefore represents a composite listing of the more common parasites of monkeys and apes without particular regard to natural host species' preferences or geographic origins. Many of the parasites of primates are shared with humans, and a text such as *Atlas of Human Parasitology* (Ali and Chubb, 1990) can be consulted for the identification of these parasites.

### COMMON PARASITES OF OTHER HOSTS OF INTEREST

Diagnosis of parasitic infection in zoo, wild, or exotic animals is frequently necessitated. Although it is reasonable to attempt a generic diagnosis based

on morphologic and morphometric characters of various parasite stages that may be recovered from the feces of these animals as discussed previously in this chapter, one must be aware that when working with less-studied hosts, specific identification of specimens may be a challenge even for trained parasitologists. In undertaking such a task, it is advisable to first consult the literature and make a checklist of parasites previously reported in the host in question, including any morphologic and morphometric information associated with each parasite. It is outside the scope of this text to attempt to compile this information, however. Fig. 7.85 includes a few examples of some common parasite stages routinely observed in the feces of some monodermic hosts.

### ANNOTATED HOST-ORGAN LISTINGS FOR PARASITES OF DOMESTIC AND LABORATORY ANIMALS

#### Annotated Host-Organ Listing of Parasites of Dogs

*Trichostrongylus axei* may occur in any tissue of any host as extracellular or intracellular trichostrongylids or as trichostrongylid cysts (see Figs. 3.36 and 8.28). *Neoplasma caninum* may occur in similar locations (see Figs. 3.37 and 8.29).

#### Alienatory System

##### Monh

Protozoa. *Trichomonas canisomae* (Mastigophora). Found around gam margins; nonpathogenic but may become more numerous in the presence of pyenteria.

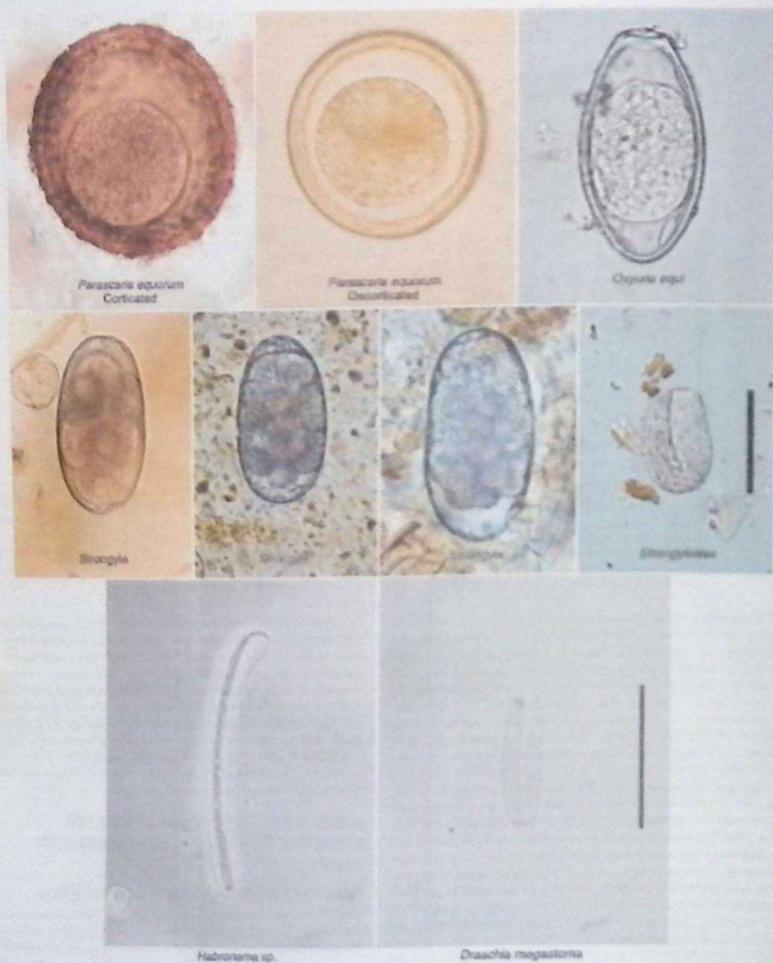


Fig. 7.86 Eggs of some nematode parasites of horses. *Parascaris equorum* and *Oxyuris equi* are the largest and sturdiest of these eggs, being close to 90 to 100  $\mu\text{m}$  in length. Strongylid eggs come in a variety of overlapping sizes and cannot be differentiated. Larvated eggs are those of *Strongyloides* *weileri* (bar 50  $\mu\text{m}$ ) or the stomach parasites, *Habronema* or *Draschia* (bar 30  $\mu\text{m}$ ).

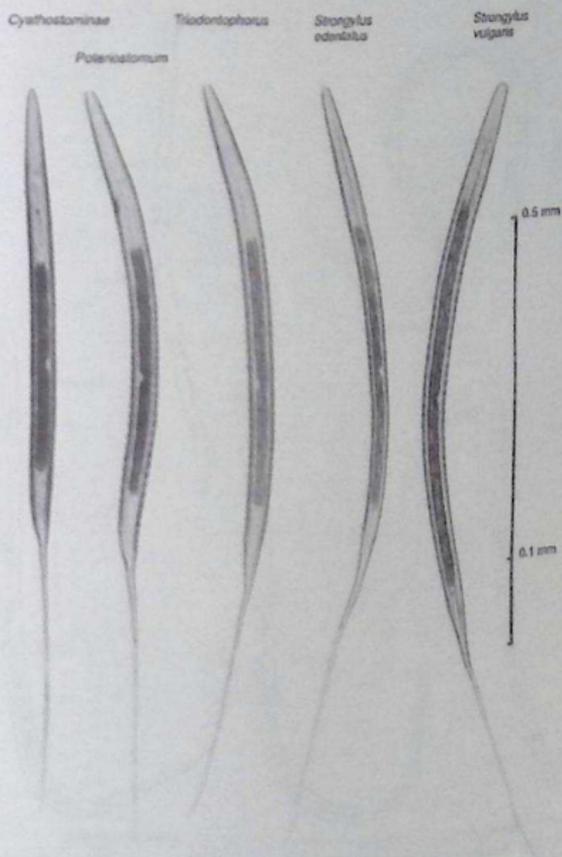


Fig. 7.87 Infective third-stage larvae of some horse strongylids. Larvae of the subfamily Cyathostominae, represented here by *Cyathostomum cationum*, have eight intestinal cells. *Gyrocampa capillata* (not shown) has 12. *Potentostomum* has 16. *Tridontophorus* has 18 but the *Tridontophorus aeneus* larvae shown here have only 16. *Strongylus edentatus* has 18 to 20, and *Strongylus vulgaris* has 32 intestinal cells. *Strongylus vulgaris* is easily distinguished from all the rest by its large size and long column of intestinal cells.

#### Esophagus and stomach

**Nematodes.** *Spirocerca lapy* (Spiruridae) (females: 54–80 mm by 1.15 mm; males: 30–54 mm by 760  $\mu$ m). Found in fibrous nodules in the wall of the esophagus and sometimes the stomach. Larvae migrate through the adventitia of the arteries and aorta to the walls of the stomach or esophagus. Adults encyst in nodules that communicate with the lumen of these two organs. Cysts may be found in other

locations as well (Figs. 7.89 and 7.90). Chronic infection is associated with dysphagia, vomiting, esophageal osteosarcoma, aortic aneurysm (rupture rare), and pulmonary osteoarthropathy.

**Physaloptera** *rara* and *Physaloptera propeptalis* (Spirurida). (*P. rara* - females: 27–41 mm by 0.9–1.1 mm; males: 25–29 mm by 800  $\mu$ m; and *P. propeptalis* - females: 15–28 mm by 1.0–1.7 mm; males: 13–45 mm by 0.7–1.3 mm) (see Figs. 4.160 and 4.161). Adult

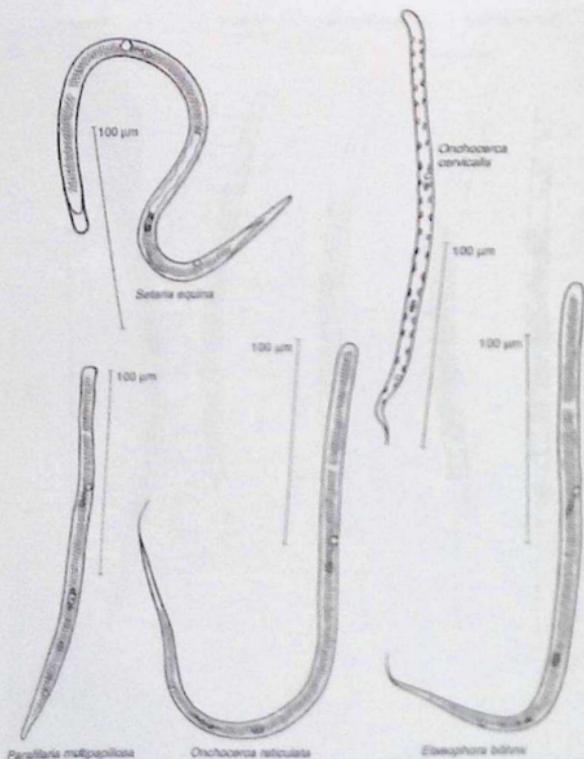


Fig. 7.88 Microfilariae of filarid parasites of horses. (Redrawn from Supperer T. *Filarosen der Pferde in Österreich*, Wiener Tierärztliche Monatschr 40(4): 193-220, 1953.)

worms are found with their anterior end firmly embedded into the gastric mucosa, often at the junction between the stomach and the small intestine; this may lead to complications with the pyloric sphincter. Infection can be asymptomatic or may be associated with vomiting and anorexia.

*Gnathostoma spinigerius* (Spirurids) (females: 9-31 mm by 1.0-2.5 mm; males: 10-25 mm by 1.0-1.9 mm). Relatively rare in North America (see Figs. 4.138 and 4.139). Adults occur in nodules in the stomach wall. Larval migration through the liver and other organs is destructive. Clinical signs have not been described. Rupture of nodules containing adult worms into the peritoneal cavity can cause a medical emergency.

#### Small intestine

Nematodes. *Tricostema ovum* (females: 5-18 cm by 2.5-3.0 mm; males: 4-10 cm by 2-2.5 mm) and *T. levis* (females: 2-10 cm by 1.8-2.4 mm; males: 2-7 cm by 1.5-2 mm) (Ascariididae) are large cream-colored worms (see Fig. 4.140). *Tricostema* has a glandular ventriculus intercalated between the muscular esophagus and the intestine (Fig. 7.86A), whereas *Tricostema* lacks this glandular structure (Fig. 7.86C). The ventriculus is visible in transilluminated fresh specimens under the stereoscopic microscope, and in fixed, cleared specimens under the compound microscope. Large, fixed specimens may be dissected to determine the presence or absence of a ventriculus. The tail of male *Tricostema* is finger-like (Fig. 7.86B), whereas the tail of male



Fig. 7.89 Porcine parasites in freshly voided feces from infected animals.

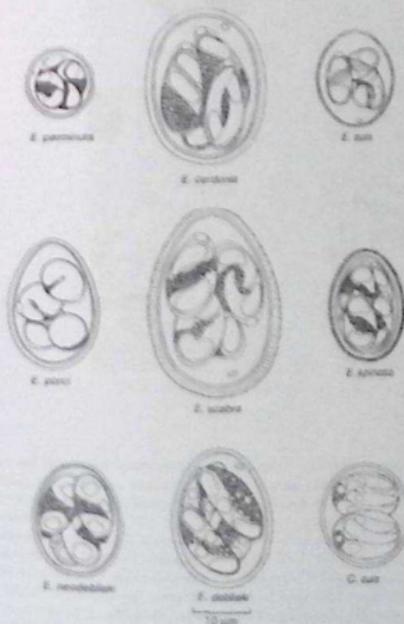


Fig. 7.80 Sporulated oocysts of eight species of *Eimeria* and one species of *Cystoisospora* from swine. (From Vetterling JM. Coccidia (Protozoa: Eimeriidae) of swine. *J Parasitol* 51(SI):909, 1965.)

*Trichostrongylus axei* tapers to a point (Fig. 7.90D). Female *Trichostrongylus axei* and *Trichostrongylus colubriformis* may be easily distinguished by comparing their eggs (Fig. 7.90).

Ascarids in the small intestine (Fig. 7.90) may cause bloating and can interfere with intestinal motility and digestion. Mucoid diarrhea, vomiting, abdominal distension, emaciation, and a failure to thrive



Fig. 7.91 Cyst of *Neobalantidium coli* and the egg of *Ascaris suum*. The cyst is about the same size as the *A. suum* egg (1–60 µm).

may all be noted as clinical signs, especially in young animals acquiring infections with *T. canis* in utero. Puppies are expected to harbor larger numbers of these adult worms than older dogs, but dogs of any age may be hosts of adult worms. Infection with *E. leuana* is less pathogenic and usually results in diarrhea and vomiting in only the worst cases.

*Baylisascaris procyonis*, the raccoon roundworm, is capable of causing patent (adult) infections in dogs (females reaching 20–22 cm; males 9–11 cm). This is a dangerous parasite because the zoonotic disease produced by the ingestion of embryonated eggs can be devastating and life-threatening to people. Although perhaps a rather rare condition, cases are regularly occurring. When fully mature, the worms tend to be larger than *T. canis* or *T. leuana*, and the eggs can be differentiated by the facts that they are smaller, have a rough external shell (see Figs. 4.154, 4.155 and 7.92), and appear darker than the eggs of the two more common dog ascaridoids. Males can be differentiated from ascarids of other genera based on the raised rugose area adjoining the cloacal opening (Fig. 7.90C). Infected dogs are typically without any clinical signs.

*Ancylostoma caninum* (females: 14–20.5 mm by 0.5–0.57 mm; males 11–15 mm by 0.34–0.39 mm), *A. hookworm* (females: 0.5–10.6 mm by 0.22–0.32 mm; males 5–7.5 mm by 0.19–0.27 mm), and *U. stenocephala* (females: 7–15 mm by 0.2–0.25 mm; males 5–9 mm by ~0.2 mm) (*Ancylostomatoidae*). Mature hookworms are found anchored to the



Fig. 7.82 Eggs of some parasites found in the feces of pigs. (Image of infecta *Ascaris* egg courtesy Dr. M. Dale Little.)



Fig. 7.83 Coenurus from a laboratory-pouched rat, *Cricetomyia gambiense*. The animal had been wild-caught for introduction to the colony.

mucosa by their buccal capsules unless the endoventer has coiled out or the host has died of an overdose of barbiturates, in which case many specimens will be found unattached. Thirdly *A. caninum* burrow deeply and destructively in the mucosa (Fig. 4.101) and the mucosal lymph nodes may be hemorrhagic as a result during the juvenile phase of severe infections. An adult *A. caninum* is colored red, whereas *A. braziliense* and *U. stenocephala* are grayish-white. The red color of *A. caninum* quickly fades on fixation, however. Specimens may be differentiated by microscopic examination of their buccal structures: *A. caninum* has three pairs of pointed teeth on the ventral border of the buccal capsule; *A. braziliense* has one pair of pointed teeth (and sometimes a very small medial tooth); and *U. stenocephala* has a pair of rounded plates instead of teeth (see Fig. 4.109). *Ancylostoma caninum* sucks much more blood than either of the other hookworm species affecting dogs. Suckling pups experience prostrate infections of this parasite owing to transmammary transmission of larvae; such infection may be fatal. Affected pups will have pale mucous membranes and can pass soft liquid stools containing partially digested blood. Dogs of

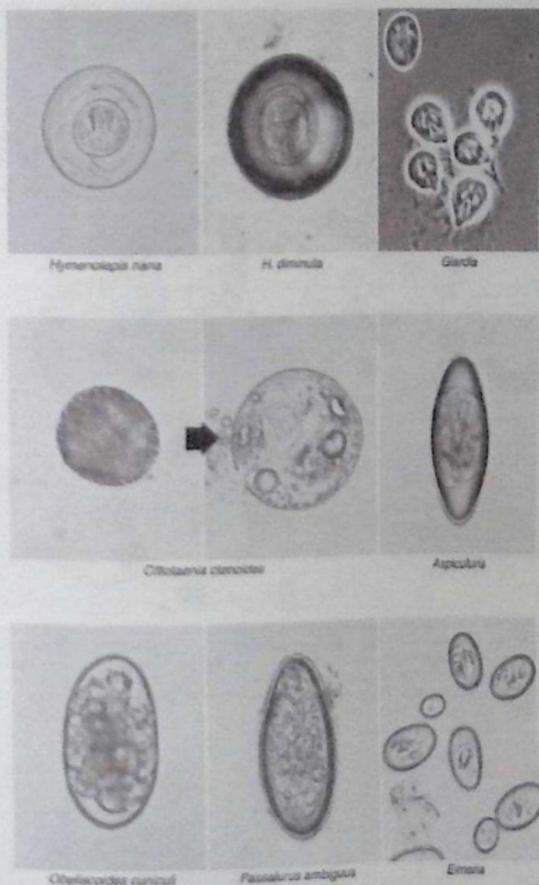


Fig. 7.94 Common parasites of laboratory mice, rats, and rabbits. For a more comprehensive listing of laboratory animal parasites by host and organ, see text. Mouse and rat: *Hymenolepis nana* and *Hymenolepis diminuta* (Hymenolepididae) are also parasites of humans. *Hymenolepis nana* infection in rodent colonies is directly infective to human beings; no intermediate host is required by this tapeworm. Various beetles and dipterids serve as intermediate hosts for *H. diminuta* and, facultatively, for *H. nana*. *Giardia* (Mastigophora trophozoites [group of five, center and cysts inset, upper left]) are common parasites of mice. Rabbit: *Cytospora ctenoites* (Aspiculariidae) eggs appear as amorphous spheres (left of arrow) until crushed by pressure on the coverslip (right of arrow), whereupon the oncosphere and pear-shaped embryophore become visible. *Obeliscoidea cuniculi* eggs are typical strongyle eggs. *Passalurus ambiguus* (Oxyuridae) are somewhat asymmetric and have a cap at one end. *Eimeria*, sporulated oocysts. Avoid mistaking *Saccharomyces guttulatus* (see Fig. 7.11) for a bona fide parasite of the rabbit. All mice except *Giardia* (x1000).

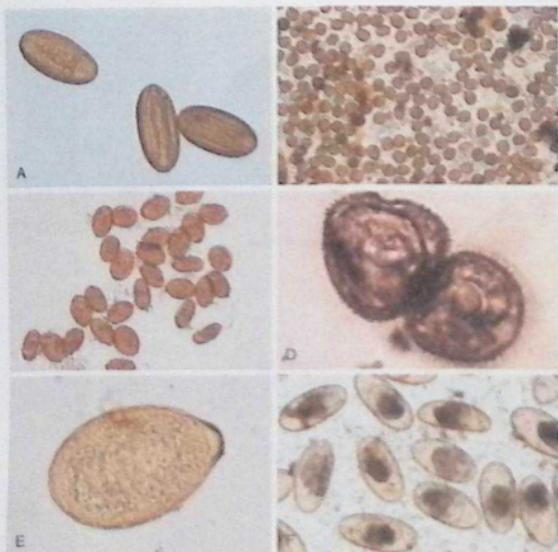


Fig. 7.86 Commonly observed fecal-borne parasites from nondomestic hosts: (A) *Cellulosa* sp. (Nematode) eggs from a black and white ruffed lemur, *Varace variegata* ( $\times 400$ ). (B) *Emerica procyonis* oocysts from a raccoon ( $\times 400$ ). (C) *Haematolochus* sp. (Trematode) eggs from an American buffing, *Lithobates catesbeianus* ( $\times 600$ ). (D) *Monocostus* sp. eggs from a porcupine ( $\times 400$ ). (E) *Mirotholus* sp. (Ciliate) cyst from an iguana ( $\times 600$ ). (F) *Tachygoneta* sp. (Crysalis) eggs from a desert tortoise, *Gopherus agassizii* ( $\times 200$ ).



Fig. 7.87 *Spirocerca lupi* ( $\times 22$ ) in a nodule in a dog. (Case described in Georg ME, Hart H, Hattick DW. *Spirocerca lupi* [Rudolph, 1906] nodule in the rectum of a dog in Connecticut. *Cornell Vet* 70:43, 1980.)



Fig. 7.87 *Spirocerca lupi* ( $\times 50$ ) sections through the region of the glandular esophagus showing the lateral chords (arrows) projecting into the pseudocoelom, and showing the nature of the intestine, with a prominent brush border and many cells with nuclei lined up in a row and uterus filled with tiny eggs.

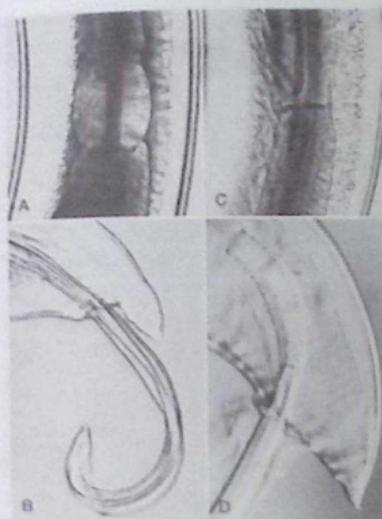


Fig. 7.88 (A and B) In *Toxocara*, a verticillus is intercalated between the esophagus and the intestine (A), and the tail (B) of the male is finger-like. (C and D) In *Toxascaris*, there is no verticillus between the esophagus and intestine (C), and the tail of the male tapers gradually (D).



Fig. 7.89 *Toxocara canis* worms in the intestine of a dog at necropsy.

any age may be infected and clinically affected by *A. caninum* if the effects of their worm burden exceed their ability to compensate for the loss of blood. *Uncinaria stenocephala*, in contrast, is not very pathogenic and clinical signs are not expected in association with its presence.

*Strongyloides stercoralis* (Rhabditioidea). The tiny, thread-like parthenogenetic parasitic female worms (1.7–2.7 mm by 30–40  $\mu$ m) (see Figs. 4.131 and 8.53) may be found in scrapings of the mucous membrane. The most notable characters for the adult females, aside from their very small size, is their very long, cylindrical esophagus and the presence of embryonated eggs in the uterus. Clinical signs vary from none to watery diarrhea that may progress to dysentery in cases in which large

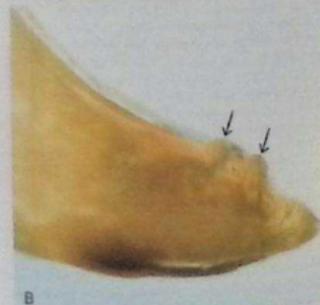


Fig. 7.100 Tail region of *Baylisascaris procyonis* male nematodes. (A) Scanning electron microscope image of the ventral aspect of the tail showing the rugose area anterior and posterior to the cloaca; this feature is not present on *Toxocara* or *Toxascaris*. (Photo courtesy Dr. Daniel Snyder). (B) Lateral aspect of the tail showing the characteristic rugose area (arrows).



Fig. 7.101 *Ancylostoma caninum* female attached to the intestinal mucosa at a feeding site.

numbers of worms are present, animals may become emaciated; hyperinfections may occur, especially in association with prednisolone immunosuppression. Transmammary transmission establishes the infection in puppies and may be an important consideration in areas of the country where the parasite is endemic and antihelminthic treatments have not been used. Infective larvae may develop within the host and lead to autoinfection by penetration of the bowel wall or the skin around the anus; these larvae are carried to the lungs and can potentiate respiratory signs.

*Trichostrongylus axei* (Trichostrongylidae) (females 3–4 mm by 60  $\mu$ m; males 1.4–1.6 mm by 40  $\mu$ m). The small adults are found threaded through the mucosa of the duodenum and produce “penetrates” that enter the intestinal mucosa (see Figs. 4.181 and 4.182). Vomiting or mild diarrhea may result from these initial stages of infection. Pre-larvae may sometimes be recovered in feces.

Cestodes. *Taenia pisiformis*, *T. hydatigena*, *T. ovis*, *T. multiceps*, and *T. serialis* (Taeniidae). Members of the genus *Taenia* are large tapeworms ranging in size from a few centimeters to several meters in length. These worms are comprised of hundreds to thousands of segments (proglottids), which individually measure 2–7 mm by 8–14 mm in size. A simple way to narrow down species identification of taenids involves studying the morphology and morphometry of the taenid species expected in a host, coupled with rostellar hook morphology and morphometry will help narrow down species identification. For example, Fig. 7.183 depicts the rostellar hooks of a *Taenia* specimen matching in size those expected for *T. pisiformis* (Table 7.8). PCR identification may also be used for taenid species confirmation. Adult tapeworms (Fig. 7.183) see also Figs. 4.38 to 4.40, and 4.42) typically cause no significant signs but may incite owner disgust.

*Echinococcus granulosus* (2–11 mm long with up to seven segments), *E. multilocularis* (1.2–4.5 mm long with up to six segments), and other species (Taeniidae). Adult tapeworms (see Fig. 4.49) cause no significant signs. If *Echinococcus* is to be expected, the intestine collected during necropsy must be frozen at  $-80^{\circ}\text{C}$  for 5 days before examination and parasite collection. This is done to inactivate eggs that are otherwise zoonotic in humans and can lead to establishment of larval (hydatid) tapeworm infections.

*Dipylidium caninum* (20–70 cm), *Dipylidium* (4–12 cm), and *Jejunascaris* (6–50 cm) (Dipylidiidae). Typically without clinical signs (Fig. 7.184; see also Figs. 4.59, 4.61, 4.63, 4.65, 8.41, and 8.42), infection can rarely result in impaction in young puppies.

*Mesocricoides* spp. (Mesocricoididae). Adults measure 30 to 250 cm. Typically, infection with this stage of the parasite is without clinical signs (see Figs. 4.64, 7.85, and 7.86). Segments crawling on the perianal region may cause pruritus and discomfort to animals and are unattractive.

*Dehnelocephalus latus*, *S. mansonioides* (Dipylidobothriidae). The former is the largest tapeworm of all (7–12 m), and the latter usually measures up to 1.5 m. These two species can be differentiated based on the morphology of the uterus. The uterus of *D. latus* is rosette shaped (Fig. 7.185), whereas in *Spermium mansonioides*, as the genus name suggests, it appears spiral. Typically, infection is without any signs, but the voiding of large strands of tapeworm tissue can be quite disconcerting to owners (see Figs. 4.30, 4.31, and 4.34 and for egg detail Fig. 7.87).

Trematodes. *Alaria americana* (5 mm), *Alaria artemisioides* (10 mm), *Alaria canis* (3.2 mm), *Alaria immodicatus* (1.9 mm), and *Alaria maritima* (1.2–1.6 mm) (Diplostomatidae) (Fig. 7.186; see also Figs. 4.22 to 4.24). Adult infections are typically asymptomatic, but immature flukes migrate through the peritoneal cavity and diaphragm en route

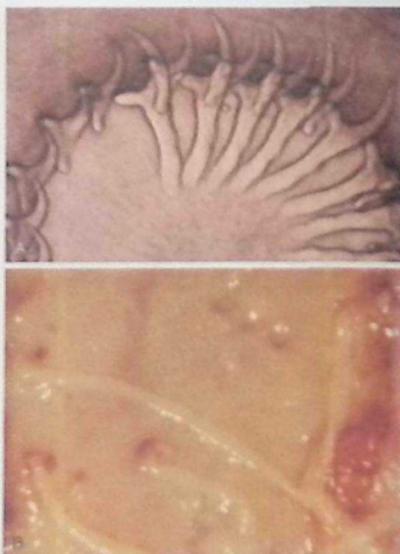


Fig. 7.183 Taenid tapeworm. (A) Rostellar hooks on the dissected border of a *Taenia* specimen recovered from a cystic; hook measurements correspond to those for *T. pisiformis* (large hooks ~240  $\mu$ m; small hooks ~147  $\mu$ m). (B) Attachment sites of three *Taenia* proglottid scolexes on the mucosa of the small intestine of a dog at necropsy.

TABLE 7.8 Number and Size of Rostellar Hooks Found on Common Species of *Taenia*

Species	Number of Hooks		Size (mm)	
	Large	Small	Large	Small
<i>Taenia saginata</i>	0	0		
<i>Taenia serialis</i>	23–34	110–117	85–140	
<i>Taenia multiceps</i>	23–34	130–180	75–140	
<i>Taenia axei</i> (adult)	22–26	131–195	85–141	
<i>Taenia axei</i> (larva)	20–36	139–200	50–130	
<i>Taenia hydatigena</i>	26–44	170–226	110–160	
<i>Taenia crassiceps</i>	28–34	171–200	121–150	
<i>Taenia marti</i>	29–40	175–220	130–160	
<i>Taenia nana</i>	36–46	201–258	159–196	
<i>Taenia pisiformis</i>	32–40	230–294	114–177	
<i>Taenia hydatigena</i>	32–46	275–280	120–180	
<i>Taenia canis</i>	36–44	233–267	160–210	
<i>Taenia taenioides</i>	26–52	200–450	167–263	
<i>Taenia latissima</i>	56–67	370–467	160–247	



Fig. 7.103 *Dipylidium caninum* in the intestine of a dog at necropsy.

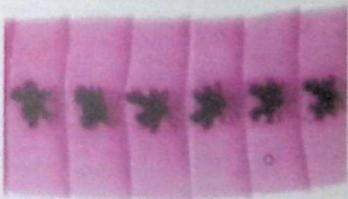


Fig. 7.104 Stained *Dipylidiosis* segments showing the rosette-shaped egg-filled uterus in mature segments. (Courtesy Dr. Stefano Catalano)



Fig. 7.105 *Alaria organiformis* in the small intestine of a dog (b10). *Alaria*, typical of the family Diplostomatidae, is divided into a forebody and hindbody.

to the lungs and may cause signs. Larval stages can be transmitted in milk vertically.

*Monostephanus appendiculatus* (1.8 mm) and *Monostephanus longicauda* (1 mm) (Cyathocystidae). These cyathocystids resemble *Alaria* in having a bulbous tribocytic organ but differ in not being divided into distinct forebody and hindbody regions. Infection is asymptomatic.

*Echinochasmus schwarzi* (2.1 mm) (Echinostomatidae) is a slender echinostomatid with a collar of spines surrounding the oral sucker. The parasite may cause severe enteritis in dogs that eat fish.

*Apophallus venustus* (1.4 mm), *Cryptocotyle lingua* (2.2 mm), and *Plagiorhynchus lingua* (1.2 mm) (Heterophyidae). Dogs ingesting fish and acquiring *C. lingua* can have severe enteritis.

*Plagiorhynchus* spp. These small (1.2 mm) plagiatorhynchids have spindle-shaped, spinous bodies with well-developed suckers the genital pore is anterior to the ventral sucker.

*Nanophyetus salmositica* (1.1 mm, see Fig. 7.22; see also Figs. 4.13 and 8.36) and *Solliacystis montana* (0.4 mm) (Troglorematidae) are ovoid and pear-shaped, respectively, and have spinous bodies and well-developed suckers. *Nanophyetus salmositica* is host to *Neorickettsia helmshteyni*, which causes salmon poisoning in dogs. Signs include hemorrhagic enteritis and lymphadenopathy.

*Acanthocephala*. *Oncicola canis* is small (14 mm) and spindle-shaped (see Fig. 4.196). *Macracanthorhynchus ingens* is very long but may contract with fixation (Fig. 7.106; see also Figs. 4.191 and 4.192). Dogs acquire infection by ingesting millipedes, with diarrhea as the main clinical sign. Identification can be facilitated by recovery of eggs and examination of the proboscis.

*Protista*. Flagellates, *Giardia* (Fig. 7.94) trophozoites (9–21  $\mu$ m long, 5–15  $\mu$ m wide, 2–4  $\mu$ m thick) on mucus of the small intestine can be visualized in scrapings examined by microscopy. Assemblages C and D are specific for dogs. Diarrhea and vomiting may occur, typically in younger animals. Other infected dogs may or may not have signs but may have periodically soft feces with a foul odor. Cysts are often excreted without clinical signs.

*Coccidia*. *Cystosporosus canis*, *C. ohioensis*, *C. burrowsi*, *H. heydorni*, and *N. caninum* (Apicomplexa) oocysts contain a single sporont when shed in the feces and resemble their feline counterparts (Fig. 7.97), and see Fig. 7.97). *Schistosoma* gamonts, and oocysts may also be found in histologic sections or smears of scrapings. These coccidia cause damage to host erythrocytes, but infection is often subclinical. Young animals and immunocompromised animals are most often affected. The main clinical sign



Fig. 7.106 *Macracanthorhynchus ingens* from a dog. (A) Hooks on the spiny proboscis. (B) Fixed, contracted, specimen. (C) Unfixed specimen showing its full length.

numbers of worms are present; animals may become emaciated; hyperinfections may occur, especially in association with prednisolone immunosuppression. Transmammary transmission establishes the infection in puppies and may be an important consideration in areas of the country where the parasite is endemic and anthelmintic treatments have not been used. Infective larvae may develop within the host and lead to autoinfection by penetration of the bowel wall or the skin around the anus; these larvae are carried to the lungs and can potentiate respiratory signs.

*Trichinella spiralis* (Trichinelloidea) (females: 3–4 mm by 60  $\mu$ m; males: 1.4–1.6 mm by 40  $\mu$ m). The small adults are found threaded through the mucosa of the duodenum and produce “prelarvae” that enter the intestinal mucosa (see Figs. 4.181 and 4.182). Vomiting or mild diarrhea may result from these initial stages of infection. Pre-larvae may sometimes be recovered in feces.

Cestodes. *Taenia pisiformis*, *T. hydatigena*, *T. evivi*, *T. multiceps*, and *T. serialis* (Taeniidae). Members of the genus *Taenia* are large tapeworms ranging in size from a few centimeters to several meters in length. These worms are comprised of hundreds to thousands of segments (proglottids), which individually measure 3–7 mm by 8–14 mm in size. A simple way to narrow down species identification of taeniids involves studying the morphology and morphometry of the large and small hooks through careful dissection (using a scalpel blade) of the rostellum from the scolex. Basic knowledge of the taenid species expected in a host, coupled with rostellar hook morphology and morphometry will help narrow down species identification. For example, Fig. 7.108 depicts the rostellar hooks of a *Taenia* specimen matching in size those expected for *T. pisiformis* (Table 7.8). PCR identification may also be used for taenid species confirmation. Adult tapeworms (Fig. 7.106; see also Figs. 4.38 to 4.40, and 4.42) typically cause no significant signs but may incite owner disgust.

*Echinococcus granulosus* (2–11 mm long with up to seven segments), *E. multilocularis* (1.2–4.5  $\mu$ m long with up to six segments), and other species (Taeniidae). Adult tapeworms (see Fig. 4.69) cause no significant signs. If *Echinococcus* is to be expected, the intestine collected during necropsy must be frozen at  $-80^{\circ}\text{C}$  for 3 days before examination and parasite collection. This is done to inactivate eggs that are otherwise zoonotic to humans and can lead to establishment of larval (hydatid) tapeworm infections.

*Dipylidium caninum* (20–70 cm), *Dipylidium* (4–12 cm), and *Jayusculis* (8–50 cm) (Dipylidiidae). Typically without clinical signs (Fig. 7.105; see also Figs. 4.59, 4.61, 7.83, 7.85, 8.41, and 8.42), infection can rarely result in impaction in young puppies.

*Monocestoides* spp. (Monecestoididae). Adults measure 30 to 250 cm. Typically, infection with this stage of the parasite is without clinical signs (see Figs. 4.64, 7.86, and 7.88). Segments crawling on the perianal region may cause pruritus and discomfort to animals and are unsightly.

*Dibothriocephalus latius*, *S. mansonioides* (Diphylobothriidae). The former is the largest tapeworm of all (2–12 m), and the latter usually measures up to 1.5 m. These two species can be differentiated based on the morphology of the uterus. The uterus of *D. latius* is rosette shaped (Fig. 7.106), whereas in *Spirurus mansonioides*, as the genus name suggests, it appears spiral. Typically, infection is without any signs, but the voiding of large strands of tapeworm tissue can be quite disconcerting to owners (see Figs. 4.30, 4.33, and 4.34 and for egg detail Fig. 7.109).

Trematodes. *Alaria americana* (5 mm), *Alaria urticae* (10 mm), *Alaria canis* (3.2 mm), *Alaria malayensis* (1.9 mm), and *Alaria macrura* (1.2–1.8 mm) (Diplostomidae) (Fig. 7.106; see also Figs. 4.22 to 4.26). Adult infections are typically asymptomatic, but immature flukes migrate through the peritoneal cavity and diaphragm en route



Fig. 7.108 Taenid tapeworm. (A) Rostellar hooks on the dissected scolex of a *Taenia* specimen recovered from a cystiter. Hook measurements correspond to those for *T. pisiformis*: large hooks = 240  $\mu$ m; small hooks = 147  $\mu$ m. (B) Attachment sites of three *Taenia pisiformis* scolexes on the mucosa of the small intestine of a dog at necropsy.

TABLE 7.8 Number and Size of Rostellar Hooks Found on Common Species of *Taenia*

Species	Number of Hooks		Small Hooks ( $\mu$ m)
	Large Hooks	Small	
<i>Taenia saginata</i>	0	0	0
<i>Taenia serialis</i>	23–34	110–117	95–100
<i>Taenia multiceps</i>	23–34	120–180	75–100
<i>Taenia ovis</i> (kattar)	23–36	121–195	85–101
<i>Taenia solium</i>	33–36	120–200	93–120
<i>Taenia hydatigena</i>	30–44	170–226	112–160
<i>Taenia brassicae</i>	28–34	170–200	121–150
<i>Taenia martii</i>	28–40	175–220	130–160
<i>Taenia nana</i>	36–40	207–250	159–186
<i>Taenia pisiformis</i>	32–42	220–294	114–177
<i>Taenia regis</i>	32–40	220–250	120–190
<i>Taenia citreus</i>	30–44	232–287	180–222
<i>Taenia taenioides</i>	30–52	300–400	167–200
<i>Taenia latifolia</i>	30–62	370–467	180–247



Fig. 7.103 *Dipylidium caninum* in the intestine of a dog at necropsy.



Fig. 7.104 Stained *Dibeothricostelae* segments showing the rosette-shaped egg-filled uteruses in multiple segments. (Courtesy Dr. Stefano Catalani)



Fig. 7.105 *Alaria* organisms in the small intestine of a dog (x10). *Alaria*, typical of the family Diplostomatidae, is divided into a forscbody and hindbody.

to the lungs and may cause signs. Larval stages can be transmitted in milk vertically.

*Monostephanus appendiculatus* (1.8 mm) and *Monostephanus longicauda* (1 mm) (Cystocotylidae). These cystocotylids resemble *Alaria* in having a bulbous triboytic organ but differ in not being divided into distinct forebody and hindbody regions. Infection is asymptomatic.

*Echinochaosmum schwartzi* (2.1 mm) (Echinostomatidae) is a slender echinostomatid with a collar of spines surrounding the oral sucker. The parasite may cause severe enteritis in dogs that eat fish.

*Apophyllus venustus* (1.4 mm), *Cryptocotyle lingua* (2.2 mm), and *Phagocota longa* (1.2 mm) (Heterophyidae). Dogs ingesting fish and acquiring *C. lingua* can have severe enteritis.

*Plagiorhynchus* spp. These small (1.2 mm) plagiorhynchids have spindle-shaped, spinous bodies with well-developed suckers; the genital pore is anterior to the ventral sucker.

*Nanophyetus salmositica* (1.1 mm, see Fig. 7.92; see also Figs. 4.13 and 8.36) and *Selacotyle mustela* (0.4 mm) (Troglosetimidae) are ovoid and pear-shaped, respectively, and have spinous bodies and well-developed suckers. *Nanophyetus salmositica* is host to *Neorickettsia helmsleyana*, which causes salmon poisoning in dogs. Signs include hemorrhagic enteritis and lymphadenopathy.

*Ascaridiphora*. *Oxyciola canis* is small (34 mm) and spindle-shaped (see Fig. 4.180). *Macracanthorhynchus ingens* is very long but may contract with fixation (Fig. 2.96; see also Figs. 4.191 and 4.192); dogs acquire infection by ingesting millipedes, with diarrhea as the main clinical sign. Identification can be facilitated by recovery of eggs and examination of the proboscis.

*Protista*. Flagellates. *Giardia* (Fig. 2.96) trophozoites (9–21 µm long, 3–15 µm wide, 2–4 µm thick) on mucosa of the small intestine can be visualized in scrapings examined by microscopy. Assemblages C and D are specific for dogs. Diarrhea and vomiting may occur, typically in younger animals. Other infected dogs may or may not have signs but may have periodically soft feces with a foul odor. Cysts are often excreted without clinical signs.

*Coccidia*. *Cystoisospora canis*, *C. plausens*, *C. burnetti*, *H. heydorni*, and *N. caninum* (*Apicomplexa*) oocysts contain a single sporozoite when shed in the feces and resemble their feline counterparts (Fig. 2.97, and see Fig. 2.98). Sclerozoites, gametes, and oocysts may also be found in histologic sections or mucosal scrapings. These coccidia cause damage to host enterocytes, but infection is often subclinical. Young animals and immunocompromised animals are most often affected. The main clinical sign



Fig. 7.106 *Macracanthorhynchus ingens* from a dog. (A) Hooks on the spiny proboscis. (B) Firm, contracted, specimen. (C) Uncoiled specimen showing its full length.

associated with *Cystispora* spp. is diarrhea, which is usually watery but may also contain mucus or blood. Infections are self-limiting.

*Sarcocystis cruzi*, *Sarcocystis evansi*, *Sarcocystis miescheriana*, *Sarcocystis lernaei*, *Sarcocystis foeyi*, and *Sarcocystis hominilarnaei* (Table 3.1 and Fig. 7.44) (Apicomplexa) have sexual stages in the mucosa, usually with no clinical signs.

*Cryptosporidium canis* (Apicomplexa) has minute stages on the apical margins of the enterocytes that would be difficult to see without histologic sections. Most infections are asymptomatic and occur in dogs less than 6 months old, or in dogs that are immunocompromised.

#### Cecum and colon

**Nematodes.** *Trichuris vulpis* (Trichinelloidea) (45–75 mm long) (Fig. 7.40); see also Figs. 4.186, 7.30, 8.76). In small numbers, worms are found in the cecum; in heavier infections, worms are also found with their anterior end embedded in the mucosa of the colon and rectum. Most dogs are without clinical signs. Dogs can have large bowel diarrhea characterized by hematochezia; mucus and straining are the main clinical signs when present. Diarrhea may lead to dehydration or pseudohypoadrenocorticism in middle-aged and older dogs as a result of isotonic fluid loss causing hyponatremia, metabolic acidosis, and hyperkalemia.

**Protozoa.** *Eimeria histolytica* and *E. coli* are cyst-forming amoebae. Trophozoites of *E. histolytica* may contain phagocytosed erythrocytes and may cause amoebic colitis. Infection with these organisms seems very rare in dogs in the United States.

**Trichomonas** spp. and **Prostatta-Acanthamoeba** **baumii** are coccidiforming microsporidians. They can be found by examination of mucus and will live in water, so saline solution preparations are required. These infections are asymptomatic.

**Nabalastium coli ciliatum** (see Figs. 3.13 and 7.31) has caused colitis in dogs on very rare occasions. Trophozoites are large (50–60  $\mu$ m in length, and sometimes up to 120  $\mu$ m) and found in the lumen of the large bowel but may invade the mucosa. This common ciliate of pigs is very rare in dogs.

#### Liver and pancreas

**Nematodes.** *Toxascaris canis* and *T. hominis* (Ascarioidea) sometimes erratically invade the common bile duct or pancreatic duct causing obstruction or rupture.



Fig. 7.187 *Trichuris vulpis* posterior ends of worms on the mucosa of the cecum; the anterior portions of the worms are embedded in the mucosa.

**Capillaria** (*Capillaria*) **hepatica** (Trichinelloidea) (Fig. 7.46) see also Fig. 8.78) is found in the liver of dogs usually as an incidental necropsy finding.

**Diactophyme** **renale** (Dioctophymatidae) adults (see Figs. 4.177 and 7.11) may situate themselves between the liver lobes and erode the surface of the organ. Eggs of the parasite may also be at times found encapsulated in the omentum or distributed in small nodules in the liver.

**Nematode** larvae, **Toxascaris canis** (Ascarioidea) can have encapsulated larvae widely distributed in adult animals, especially in skeletal muscle and the kidneys, but also the liver.

**Trematodes.** *Ophidiorhynchus tenuirostris*, *O. viverrini*, *Glacocercaria sinensis*, *Mesocercaria albicans*, and *M. zoonocerca* (Opisthorchiidae) in bile ducts (see Figs. 4.10 and 4.19); infections are usually asymptomatic unless a large worm burden is present, in which case severe hepatic dysfunction can result.

Eggs of *Heterobilharzia americana* (Schistosomatidae) in tissues are surrounded by granulomatous reaction; granulomatous lesions in the liver may be associated with an elevation of hepatic enzymes (see Figs. 4.28 and 4.29). Flushing the vascular system of the liver with saline solution may release large numbers of paired flukes. Clinical signs are nonspecific and may include anorexia, lethargy, weight loss, and diarrhea. Mineralization due to hypercalcemia and calcification of eggs may be evidenced radiographically.

**Cestode** larvae, **Echinococcus multilocularis alveolar** hydatid (see Figs. 4.50, 4.51, 8.85, and 8.86) have been reported in dogs in Europe and North America (Sappalao et al., 2007). Reports are still few in number and thought to be associated with dogs that have ingested very large numbers of eggs from an infected animal (likely a fox). These cysts can lead to organ failure owing to their ability to grow, involving the organ in a tumor-like fashion. Clinical signs include abdominal distention, anorexia, hepatomegaly, and others. Fatalities can occur. In areas where the parasite is endemic, or in dogs that have traveled to these areas, this presentation should be considered as a differential if hepatic tumors are suspected.

#### Peritoneum and peritoneal cavity

**Nematode.** **Mesocricaria tritrichocerca** (see Figs. 8.47) can be associated with massive infections owing to asexual multiplication that may be associated with diarrhea, abdominal distention, pain, and weakness.

**Nematode.** **Diactophyme renale**, the giant red worm (females: 200–1000 mm by 5–12 mm; males: 140–450 mm by 4–6 mm) (Dioctophymatidae) found in the peritoneal cavity or renal pelvis (see Figs. 4.177 and 7.11). Besides the occasional free adult in the peritoneal cavity, the third-stage larvae cross through the peritoneal cavity on their way to the liver, where they molt to the fourth stage. Fourth-stage larvae again traverse the peritoneal cavity before entering the renal capsule. A serofibrinous to chronic, fibrous peritonitis can result.

#### Respiratory System

##### Canal passages

**Nematodes.** **Eucoleus** (*Capillaria*) **baubeni** (Trichinelloidea) (females: 64.9 mm; males: 26.7 mm) may cause sneezing. Eggs in the sputum of patient females are typical of capillariids and contain a morulated embryo.

**Arthropods.** **Pneumonysses caninum** (Mesostigmata) (Fig. 7.146) see also Fig. 2.103). Clinical signs include reverse sneezing, chronic nasal discharge, nasal irritation, and epistaxis. Inflammation of the nasal cavity may result in the loss of the sense of smell.

**Linguatule serrata** (Pentastomida) (females: 80–120 mm; males: 18–20 mm long) (Fig. 2.139). These organisms are bloodsucking, wormlike parasites of the nasal cavity and paranasal sinuses. The body is flat



Fig. 7.108 *Pseudomonas aeruginosa* in the nasal sinuses of a dog at necropsy. (Courtesy Dr. John M. King.)

and elongated, somewhat tongue-shaped, and may appear segmented. Four hooks surround the mouth on the anterior end of the parasite. These can cause epistaxis, inflammation, and respiratory distress.

#### Trachea and bronchi

**Nematodes.** *Filaroides coleri* (Metastrongyloidea) (females 9–15 mm, males 5 mm long) (Fig. 7.49c; see also Figs. 4.124 and 4.125, and Fig. 7.60). *Filaroides coleri* occurs in nodules near the bifurcation of the trachea with clinical signs of respiratory distress.

**Cestrosoma vulpis** (Metastrongyloidea) (see Fig. 4.119 and Fig. 7.60) are small worms (females: 12–16 mm by 300–450  $\mu$ m; males: 3.5–8 mm by 280–320  $\mu$ m) found on bronchial and bronchiolar mucosa, causing most typical signs of chronic cough, dyspnea, and exercise intolerance.

**Baculina** (*Capillaria*) *serophilae* (Trichostrongyloidea) (females: 20–60 mm by 100–180  $\mu$ m; males: 15–25 mm by 60–100  $\mu$ m) is associated with signs of coughing.

#### Lung parenchyma

**Nematodes.** *Filaroides hirthi* (females: 8.8–13 mm; males: 2.3–3.2 mm) and *F. Andersonstrongylus* *milki* (females: 11 mm by 110–174  $\mu$ m; males: 3–4 mm by 58–101  $\mu$ m) (Metastrongyloidea) (Young, 1975; see Figs. 4.77, 1.60). Most dogs are asymptomatic,



Fig. 7.109 *Filaroides* (*Colerius*) *coleri* nodules at the tracheal bifurcation of a gray wolf, *Canis lupus*. The nodules containing adult nematodes varied in size from 3 to 11 mm. (Courtesy Dr. Padraig Duignan.)

but immunocompromised hosts may show signs of severe pneumonia that can be fatal.

**Dirofilaria immitis** (Filarioidea) (see Figs. 4.168 and 7.110) adults are large (30 cm) worms that occur in pulmonary arteries; allergic pneumonitis may be associated with the presence of these or other parasites. Adult worms found in ectopic locations such as these can elicit trauma and may impair organ function.

**Nematode larvae.** Petechial hemorrhages, areas of focal necrosis, and nodular inflammation of lung tissue may be caused by migrating nematode larvae. Such lesions should be investigated by preparing squashes and by the Baermann technique. Identification of nematode larvae in histologic preparations is considered in Chapter 8.

**Angiostrongylus vasorum** (Metastrongyloidea) (see Figs. 4.122, 4.123, 8.60, and 8.61) eggs and larvae cause respiratory lesions, and clinical signs are varied. Dogs may experience exercise intolerance, weight loss, subcutaneous edema due to congestive heart failure and lung damage, or coagulation abnormalities.

**Strongyloides stercoralis** (Rhabdioidea) migrating filariform larvae (Fig. 7.42) can also cause areas of acylotomic and petechial hemorrhage throughout the lung parenchyma.

**Ancylostoma caninum**, *A. braziliense*, and *U. stenocephala* (Ancylostomatoidea) larvae (Fig. 1.42) maybe present as a result of percutaneous transmission or penetration of the oral mucosa after ingestion.

**Thaanaos curvi** (Acarioides) migrating larvae can cause pneumonia (Fig. 7.107).

**Microfilariae of D. immitis** (Onchocercidae) may elicit allergic pneumonitis (see Fig. 8.74).

**Trematodes.** *Feruginosus kellicotti* (Diglostromatidae) (7–12 mm long, 4–8 mm wide, 4–6 mm thick) (see Figs. 4.14, 4.15, and 7.61B) live in fluid-filled cysts that are surrounded by large areas of granulation tissue around the escaping eggs. These organisms can cause severe loss of lung function.

**Alaria** spp. (1 to several mm in size) (Diglostromatidae) immature flukes migrate to the lungs and may cause migrating tracks with accompanying respiratory signs (see Fig. 4.25).

#### Vascular System

##### Pulmonary artery, right side of the heart, and vena cavae

**Protozoa.** *Trypanoplasma gondii*, cardiac muscle (Apicomplexa); may result in supraventricular and ventricular arrhythmias.

**Trypanosoma cruzi** (hemoflagellate) (see Fig. 8.11) amastigotes in heart muscle cause acute myocarditis by myocardial invasion and cycles of multiplication and cell rupture. Weakness, exercise intolerance, syncope, lymphadenopathy, pale mucous membranes, neurologic signs, and signs of right- or left-sided heart failure manifested on the electrocardiogram as decreased QRS complex and heart block can be seen clinically. Chronic infection can progress to dilated cardiomyopathy (see Fig. 3.5), and dogs can show signs of weakness, exercise intolerance, syncope, ventricular tachycardia, and sudden death. This infection is becoming more common in Texas and other southern states in the United States.

**Nematodes.** *Dirofilaria immitis* (Filarioidea) occurs in right ventricle, right atrium, pulmonary arteries, and rarely vena cavae (Fig. 3.10); see also Figs. 4.168, 4.171, and 8.71 to 8.73). Adult worms (females: 250–310 mm by 1–1.5 mm; males: 120–200 mm by 700–900  $\mu$ m) live in the pulmonary arteries and cause clinical signs indicative of cardiac, pulmonary, hepatic, and renal involvement. In heavy infections, worms can invade the right side of the heart and cause congestive heart failure and ascites. Clinical signs can include coughing, exercise intolerance, dyspnea, syncope, hepatomegaly, and abdominal fluid and lung sounds on auscultation. Vena cava syndrome, with associated hematuria, can also result owing to obstruction by adult worms.



Fig. 7.110 *Dirofilaria immitis* adults and induced villous proliferation of the intima of the pulmonary artery in a dog with heartworm disease. Photo courtesy of Dr. Joe Hostetler.

*Angiostrongylus vasorum* (Metastrongyloidea) is much smaller (females: 18–25 mm; males: 14–18 mm) than *D. immitis* and is found in the pulmonary arterial branches (see Figs. 4.122, 4.123, and 8.60 and 8.61). First-stage larvae resembling those of *Aelurostrongylus* (Fig. 3.69) are shed in the host's feces. Dogs may experience exercise intolerance, weight loss, subcutaneous edema due to congestive heart failure and lung damage, or coagulation abnormalities.

*Toxascaris canis* (Ascarioidea) larvae may be seen in cardiac muscle.

*Trichinella spiralis* (Trichinelloidea) may migrate through heart muscle and incite myositis.

#### Mesenteric and portal veins

Trematodes, *Heterobilharzia americana* (Schistosomatoidea) (see Figs. 4.28 and 4.29, and 8.40). Adults occur in pairs, and cause disease by the eggs that erode their way through the intestinal mucosa and cause granulomatous reactions in the liver or bowel wall. Mineralization associated with hypercalcemia and egg calcification may be evident on radiographs.

#### Blood

Nematode microfilariae, *Dirofilaria immitis* and *A. reconditum* (Filarioidea) (see roughly 240 to 350  $\mu\text{m}$  in length, see Fig. 7.103). *Dirofilaria immitis* microfilariae can remain in circulation for years even after the death of the adults and may be present in puppies after infection in utero. These cannot grow up to adulthood without obligate growth in the mosquito host. In other geographic areas other microfilariae may be present.

Protista, *Babesia canis* (Apicomplexa) (see Figs. 3.53 and 3.54 and Figs. 5.13–5.14) will be seen only at microscopy if blood films are made. Clinical signs of canine babesiosis include pale mucous membranes, icterus, hemoglobinuria and hemoglobinuria, depression, weakness, fever, anorexia, and splenomegaly.

*Trypanosoma cruzi* (hemoflagellate) trypomastigotes may be scarce in blood films (see Fig. 3.4 and 5.10). Examine heart muscle histologically for amastigotes (see Fig. 8.11).

Gamonts of *Hepatozoon* species may be found circulating within leukocytes within the blood stream (see Fig. 5.11)

#### Skeletal Muscles

Protista, *Nesoparia caninum* (see Fig. 3.37, Apicomplexa) causes disease mainly in dogs less than 6 months of age, which will show signs of paralysis. The pelvis, limbs are more severely affected than the thoracic, and signs of progressive muscle atrophy are present.

*Hepatozoon americanum* has cystic stages in the muscles associated with chronic pain that with biopsies assist in diagnosis (see Fig. 8.31).

Nematode larvae, *Trichinella spiralis* (Trichinelloidea) (see Figs. 4.182, 183 and 8.77) usually does not cause clinical signs in dogs.

*Ancylostomum caninum* (Ancylostomatoidea) larvae are present in vacuoles in muscle fibers with little or no evidence of host reaction (see Fig. 4.115).

#### Connective Tissues

Protista, *Hipponosema americanum* (Apicomplexa) (see Fig. 8.31) can cause myositis and peritoneal base proliferation with changes that might be evident on radiographs. The organisms can form large cysts in the muscles and muscle atrophy, hyposthenia, and reluctance to move can result.

Nematodes, *Asymphylotermes racematum* (Filarioidea) (females: 20–32 mm by 146–180  $\mu\text{m}$ ; males: 9–17 mm by 92–100  $\mu\text{m}$ ) without clinical signs.

*Dirofilaria immitis* (Filarioidea) (females: 250–310  $\mu\text{m}$  by 1–1.3 mm; males: 120–200  $\mu\text{m}$  by 700–900  $\mu\text{m}$ ) (see Fig. 4.168) migratory stages and ectopically migrating adults.

*Dracunculus insignis* (Spirurida) (females: 1.5–30 cm by up to 1 mm; males: 17–22 mm by 240  $\mu\text{m}$ ) (Figs. 7.111 and 7.112; see also Figs. 4.336, 4.357, and 7.120) causes subcutaneous nodules, local myositis, and ulcerations with or without associated pododermatitis. Female



Fig. 7.111 *Dracunculus insignis* discovered in canine dissection in part of an anatomy class in 1966.



Fig. 7.112 *Dracunculus insignis* (60 $\times$ ). Cross-section of a female nematode in the subcutaneous tissue of a raccoon. The two lateral coils on either side of the body and heavy dorsal and ventral muscle bands surrounding the stercoral tube packed with larvae are evident.

worms may be seen emerging from these sites. Larvae with very long tails may be expressed from the nodules or recovered from female worms.

#### Insect larvae

**Obligatory myiasis.** *Caterebra* (Caterellidae) (see Figs. 2.52, 2.53, 8.1, and 8.2) larvae (30 mm) migrate to the skin to create a warble. The skin can be sensitive in this area, and a draining tract may be present. The spiracles of the larvae may be seen protruding from the fistula, and the larvae can be extracted from the opening.

*Cochliomyia hominivorax* (larva 17 mm, Calliphoridae) (see Figs. 2.15 and 2.20). A reportable parasite in North America. Large numbers of maggots hatch from eggs deposited on wounds. Their feeding behavior causes extreme tissue damage, necrosis, and pain. Affected sites can be further affected by facultative myiasis (fly-strike) or other secondary infections. This parasite can be identified by visualization of their pigmented posterior tracheae.

**Facultative myiasis.** *Phormia sericata*, *Phormia regina*, and *Phormia irrorator* (larva 17 mm, Calliphoridae) (see Figs. 2.15 and 2.20).

*Wohlfahrtia vigil* and *W. exilis* (Sarcophagidae) (see Fig. 2.19).

Larvae of these free-living flies are associated with fly-strike on wounds or sores that have become heavily contaminated with organic matter. They elicit painful wounds and necrosis of surrounding tissue on which they feed. Note that these maggots are routinely associated with the carcasses of dead animals, thus consideration may need to be given to their antemortem or postmortem association with the host.

### Urogenital System

#### Kidney

**Nematodes.** *Dictyophyma renale* (Dictyophymidae). A giant red worm (females: 200–1000 mm by 5–12 mm, males 140–450 mm by 4–6 mm) found in the renal pelvis or peritoneal cavity (Fig. 7.113; see also Fig. 4.177). The right kidney is most often affected. Clinical signs include enlargement of the right kidney, hematuria, urinary tract infections, and rarely renal failure if both kidneys are affected. Eggs of *D. renale* are typically passed in the urine of the infected animal (Fig. 7.112).



Fig. 7.113 Adult *Dictyophyma renale* in the renal pelvis from a dog in Nevada. (Photo courtesy Dr. Miquela Marc-Cereval. Typically, the right kidney is parasitized, and worms lead to the destruction of the parenchyma of the organ. Worms may also be recovered from the peritoneal cavity.)

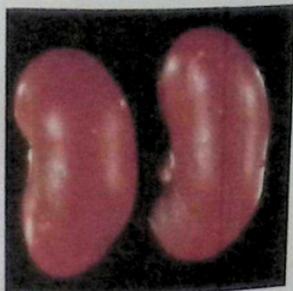


Fig. 7.114 *Toxocara canis* larvae in canine islets.

**Nematode larvae.** *Toxocara canis* (Ascaridoidea) (Fig. 7.114) larvae can cause nodular lesions to form in the kidneys but typically do not cause clinical signs.

#### Urinary bladder

**Nematodes.** *Parascaris* (*Capillaria*) *plica* (Trichostrongylidae) (females: 30–60 mm by 65  $\mu$ m; males: 13–30 mm by 48  $\mu$ m) can be found in the epithelium of the urinary bladder and usually do not cause signs. If large numbers are present, dogs can have polydipsia, dysuria, hematuria, and stranguria.

### Nervous System

#### Brain and spinal cord

**Protozoa.** *Neospora caninum* (Apicomplexa) (see Figs. 3.27 and 8.29) can appear in older dogs, causing signs of central nervous system involvement that include seizures and tremors, whereas cerebellar involvement will result in postural deficits.

**Nematodes.** *Baylisascaris* spp. (Ascaridoidea) larvae have on rare occasions caused neurologic disease in dogs (Fig. 7.115).

*Angiostrongylus cantonensis* causes severe neurologic disease in dogs in Australia. The presence of the worm in the southeastern United States and in Hawaii would suggest that sooner or later, unfortunately, cases will be reported in the United States.

#### Eye

**Nematodes.** *Toxocara canis* (Ascaridoidea) live on rare occasions been found in the retina (Figures 4.160, 4.161).

*Dirofilaria immitis* (Filarioidea) (see Fig. 4.166) can occur intracranially in the anterior chamber of the eye or the eyelid space.

**Oncophorus lupi** (Filarioidea) (female worms) have not been recovered without leakage; longest segments of females recovered from nodules are approximately 100–165 mm by 8.2–8.4 mm; males: 43–50 mm by 0.1–0.2 mm (Fig. 7.117). Cases are being reported with more frequency in the United States (64; [Sawyer et al., 2012; Henderson et al., 2013](#)). Worms are associated with subconjunctival and episcleral nodules that require surgical removal.

*Phlebotomus colchirensis* (Siphonuridae) (females: 12–18.8 mm; males: 7.7–13 mm) (see Fig. 4.166) can on occasion be found in the conjunctival sac and ducts of the lacrimal gland.

**Insects.** *Caterellus* spp. (Diptera) larvae range from a few millimeters to centimeters in size, depending on their age (Fig. 7.116). These larvae are often light in color with rows of thick black spines covering their bodies, becoming more dense as the host matures (see Fig. 2.53).

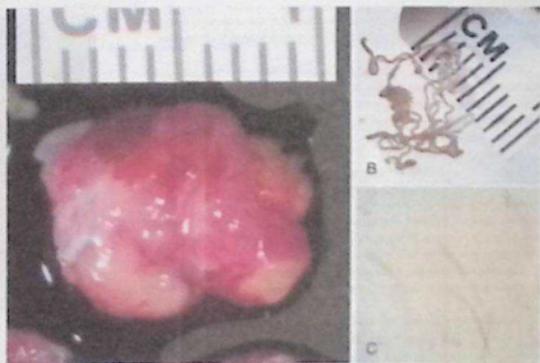


Fig. 7.115 *Othoecorea lupi* from a dog. (A) Resected subconjunctival nodule containing the nematode. (B) Adult worm teased out from the nodule. (C) Microfilariae recovered from captured adult females. (Sample and specimen provided by Dr. Eric Ledbetter).



Fig. 7.116 First-stage instar of a *Cuterebra* sp. recovered from a dog suffering from ophthalmomyiasis.

Aberrant migration of these larvae into the eye resulting in ophthalmomyiasis externa and ophthalmomyiasis interna has been reported (Edwards et al. 2014).

#### Skin and Hair

Infestations may lead to dermatitis and pruritus; anemia may be associated with severe infestations with blood-sucking types; tissue damage and necrosis may be associated with trypanis. Some of these organisms also act as vectors of disease agents.

**Insects.** Adult dipterans: Depending on type may lead to blood loss, injury, discomfort, and others.

Larval dipterans: primary or secondary myiasis (fly-strike).

*Cuterebra* spp. (Oestridae): localization of these bots to the subcutis generally does not result in much more than lesions through which the maggot's spiracular plates may protrude; these are generally not associated with disease. It should not be forgotten that these larvae have migrated through the tissues of the dog for several weeks before appearing in their final position in the skin.

*Emmagathus setosus* (Anophora) (see Figs. 2.55 and 2.57) all stages may be found on dogs.

*Trichodectes canis* (Mallophaga) (Fig. 7.117; see also Figs. 2.64 and 2.65).

*Heterodax spiniger* (Mallophaga) has club-shaped antennae that lie in cephalic grooves, and the anterior margin of the head is pointed (see Fig. 2.61); the organism is restricted to warm climates.



Fig. 7.117 *Trichodectes canis* in the hair of a dog.

*Ctenocephalides canis*, *C. felis*, *I. irritans*, and *E. gallinae* (Siphonaptera) (see Figs. 2.55, 2.36, 2.57, 2.39, 2.45, and 2.53).

**Acari.** *Rhipicephalus sanguineus*, *Dermacentor variabilis*, *I. Andersoni*, *Archipyzomus americanus*, *A. eruciculatus*, *Ixodes* spp., and others (Ixodidae) (see Figs. 2.72, 2.73, 2.76 to 2.86, and 2.88 to 2.91 and 2.93).

*Sarcoptes scabiei* (Sarcoptidae) (Fig. 7.118; see also Figs. 2.108, 7.56, 8.3, and 8.4) mites cause alopecia that usually spans the dorsum. Skin lesions are reddish and covered with yellowish crusty material. Severe self-trauma to the skin may result owing to intense pruritus.

*Otodectes cynotis* (Psocoptidae) (see Figs. 2.119 and 7.56) causes otitis with predisposition to secondary infections.

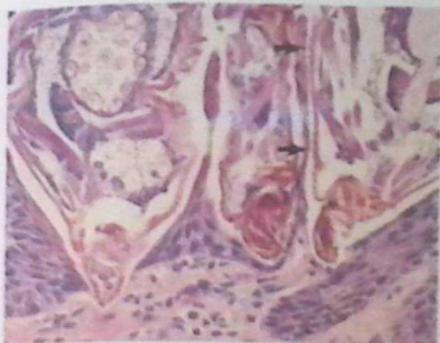


Fig. 7.110 *Sarcocystis mitis* in the skin of a dog (x200); arrows indicate apices on the cuticle.

*Demodex canis* (Demodicidae) (see Figs. 7.124, 7.125, 8.5, and 8.6) can normally be found on dogs in low numbers, and usually they do not cause disease. Demodicosis can be a localized problem, usually affecting the face and presenting as alopecia and scales surrounding the eyes and the mouth. Generalized demodicosis causes large reddened scaly alopecic coalescing patches on the head, legs, and trunk. Folliculitis and furunculosis can be present, and generalized lymphadenopathy is typical. Secondary bacterial infections cause inflammation and exudation.

*Cheyletiella vagans* (Cheyletidae) (see Figs. 2.126 and 7.16) is not generally associated with signs, may cause mild dermatitis.

**Nematodes.** *Diploscylus insignis* (Spirurida) is sometimes recovered from subcutaneous nodules or ulcerations in the skin of cats and dogs that have been hunting frogs (Figs. 7.229 and 7.27). Care must be taken not to rupture the female during resection so as not to induce a strong inflammatory reaction at the site. Wounds tend to heal rapidly and without complications after worms are no longer present. Larvae can often be readily recovered from female specimens (Figs. 7.228 and 7.230), and see Figs. 7.111 and 7.112.

**Nematode larvae.** *Rhabditi strongyloides* (Rhabditida) (see Figs. 4.128 and 8.72) larvae cause a pruritic hyperemic dermatitis. The larvae are usually free-living on decaying organic matter, and therefore the lesions are typically distributed on areas of the body that come in contact with the ground such as the feet and the ventral thorax and abdomen.

#### Annotated Host-Organ Listing of Parasites of Cats

*Toxoplasma gondii* may occur in any tissue of any host as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Figs. 3.35, 3.36, 8.27, and 8.28). Sexual reproduction (see Fig. 3.32) with formation of oocysts (Fig. 7.62) occurs only in the intestinal mucosa of members of the cat family (Felidae).

#### Alimentary System

##### Mouth

**Protista.** *Tetratrichomonas felistomae* (flagellate) is found around the gum margins and is mainly observed in cats infected with feline immunodeficiency virus, feline leukemia virus, or feline infectious peritonitis, or cats suffering from gingivitis; it is nonpathogenic.

##### Stomach and esophagus

**Nematodes.** *Gnathostoma spinigerum* (Spirurida) (female: 9–31 mm by 1–2.5 mm; male: 10–25 mm by 1–1.9 mm) (see Figs. 4.158, 4.159), with head attached to stomach mucosa, may cause gastric wall perforation.

*Physaloptera praecipua* and *P. rana* (Spirurida) (*P. praecipua* - female: 15–58 mm by 1–1.7 mm; male: 13–45 mm by 0.7–1.3 mm; and *P. rana* - female: 27–41 mm by 0.9–1.1 mm; male: 25–29 mm by 800  $\mu$ m) (Fig. 7.117; see also Figs. 4.160 and 4.161), with the anterior end attached to the gastric mucosa, may be diagnosed endoscopically and may cause vomiting.

*Ollulanis tricuspis* (Trichostrongyloidea) (female: 0.8–4 mm by 40  $\mu$ m; male: 0.7–0.8 mm by 35  $\mu$ m) (see Fig. 4.88) living in the stomach wall of infected cats causes chronic gastritis that results in vomiting, anorexia, weight loss, and possibly death.

*Acanthobasus (Capillaria) putori* (Trichostrongyloidea) (female: 9.4–15 mm by 68–88  $\mu$ m; male: 5.4–8 mm by 38–68  $\mu$ m) (Fig. 7.90) usually causes no clinical signs; it has been reported to cause perforation at the caudal aspect of the pylorus.

##### Small intestine

**Nematodes.** *Taeniae cati* (Ascariidae) (female: 6–12 cm long; male: 3–7 cm by 1–1.1 mm) (Fig. 7.115, and see Figs. 4.149, 7.60 and 7.61) infection is usually without clinical signs except in very heavy infections. In these cases, ill-thrift, vomiting, and other gastrointestinal signs may be present.



Fig. 7.115 *Physaloptera praecipua* in the stomach of a cat.



Fig. 7.120 *Taeniae cati* in the intestine of a cat at necropsy.

*Toxocara leonina* (Ascarioidae) (females 2-10 cm by 1.8-2.4 mm; males 2-7 cm by 1.5-2 mm) (Figs. 7.69 and 7.70) infection is typically without signs.

*Ancylostoma tubaeforme* (Ancylostomatidae) (females: 12-15 mm by 380-430  $\mu$ m; males: 9-5.11 mm by 300-350  $\mu$ m) (see Figs. 4.110, 7.37, and 7.68) usually causes no clinical signs, but cats may have weight loss, regenerative anemia, and loose, tarry stools, and infection has resulted in death owing to significant blood loss from the intestinal mucosa.

*Ancylostoma braziliense* (Ancylostomatidae) (females: 6.5-10.6 mm by 220-320  $\mu$ m; males: 5-7.5 mm by 190-270  $\mu$ m) (Fig. 7.121) causes less blood loss than *A. tubaeforme*, and experimentally infected kittens have maintained unaffected red blood cell parameters.

*Uncinaria stenocephala* (Ancylostomatidae) (females: 7-15 mm by 0.2-0.25 mm; males: 5-9 mm by ~0.2 mm) (see Figs. 4.109 and 7.37) infections in cats in the United States are very rare. No clinical signs are expected in natural infections.

*Strongyloides felis* (common in Australia; Speare and Timler, 1987) (parthenogenic females: 1.7-2.7 mm by 50-60  $\mu$ m, Rhabditidae) (see Fig. 4.131). Infections are generally asymptomatic.

*Trichostrongylus axei* (Trichostrongylidae) (females 3-4 mm by 60  $\mu$ m; males: 1.4-1.6 mm by 40  $\mu$ m) (see Fig. 4.118) causes signs referable to mild gastrointestinal upset, such as vomiting and diarrhea, maybe bloody diarrhea.

*Ancylostoma (Capillaria) pumilio* (Trichostrongylidae) (Fig. 7.68) is present in the small intestine and the stomach. May be associated with gastritis but is typically present without clinical signs.

**Cestodes.** *Taenia taeniiformis* (15-60 cm in length) (Taeniidae) (see Figs. 4.39 and 4.41) occurs typically without signs. In rare cases, impactions have been reported. Passage and movement of segments can be unobtrusive to owners.

*Echinococcus multilocularis* (Taeniidae) (1.2-4.5 mm long with up to six segments) (see Fig. 4.49) occurs typically without signs. Infections would be of concern to owners because of their zoonotic importance.

*Dipylidium caninum* (20-70 cm) (Dipylididae) (see Figs. 4.59 to 4.61, 7.65, 7.66, 8.41, and 8.42) occurs typically without signs.

*Mesocricoides lineatus* (30-250 cm) (Mesocricoididae) (see Figs. 4.64, 4.65, and 7.67) occurs typically without signs.

*Spirometra micronoides* (up to 1.5 m) (Dipylidobothriidae) (see Figs. 4.31 to 4.36, and 7.67) may be asymptomatic, or associated with diarrhea, emaciation, or vomiting.

**Trematodes.** *Alaria marcicuae* (5 mm, Diplostomatidae) (see Figs. 4.22-4.24) occurs typically without signs.



Fig. 7.121 Adults of *Ancylostoma braziliense* as viewed by endoscopy in the duodenum of a cat.

*Apophyllus venustus* (1.4 mm, Heterophyidae) occurs typically without signs.

*Phagocota longi* (1.2 mm, Heterophyidae) occurs typically without signs.

*Mesostephanos milis* (1.8 mm, Cyathocotylidae) occurs typically without signs.

**Acanthocephalids.** *Oncicola* spp. (see Fig. 4.195) occur typically without signs.

**Protists.** *Cystoisospora felis*, *C. viridis*, *Besnoitia* spp., *H. Hammondii*, and *T. gonali* (Coccidia) (Fig. 7.62; see also Fig. 8.129) stages occur in the intestinal epithelium, where they might cause enteritis and perhaps mild diarrhea.

*Sarcocystis boydii*, *S. tenella*, *S. parvifelis*, and *S. leptorum* (Coccidia) (see Table 3.1 and Fig. 7.66) occur with sexual stages in the intestinal epithelium and without clinical manifestations.

*Giardia* spp. (see Figs. 5.11 and 7.63) trophozoites (9-21  $\mu$ m long, 5-15  $\mu$ m wide, 2-4  $\mu$ m thick) present on intestinal epithelium may be detected in mucosal scrapings. *Giardia* infections in cats usually occur without signs, but diarrhea may occur. *Acanthamoeba* F species for cats.

*Cryptosporidium felis* (see Figs. 3.16 and 7.64, Apicomplexa) sexual and asexual stages occur in the apical portions of the epithelial cells; they are probably visible only via histologic sections. Infections are usually without signs, although occasionally it is accompanied by severe diarrhea.

#### Large Intestine

**Nematodes.** *Strongyloides stenocephalus* (parthenogenic females: 5 mm  $\times$  109  $\mu$ m, Rhabditidae) forms large tumor-like nodules in the large intestine, which are detected on abdominal palpation as a firm, fibrotic colitis.

*Trichostrongylus axei* (females: 1.7-3.15 mm by 60  $\mu$ m; males: 21.5 mm by 510-560  $\mu$ m) and *Trichostrongylus serratus* (females: 68 mm; males: 40 mm) (both exotic, South America; Trichostrongylidae) (see Figs. 4.183 and 7.68). Clinical signs have not been described for infections in cats.

#### Large bile ducts, and gallbladder; pancreatic duct

**Nematodes.** *Calodium (Capillaria) hepaticum* (Trichostrongylidae) (Fig. 7.120; see also Fig. 8.78). Reports of infections in cats are rare.

*Tanaisia canis* (Ascarioidae) larvae, granulomas (see Fig. 4.132) (Falciano et al., 1989).

**Trematodes.** *Opiothorchis tenuicollis* and *O. filiformis* (30 mm, Opiothorchidae) in gallbladder and bile ducts is likely to induce cirrhosis, cholecystitis, and the development of stoma and strictures owing to continuing periparturite fibrosis.

*Metorchis affinis* (4.8 mm) and *M. conjunctus* (6.8 mm) (Opiothorchidae) in bile ducts are associated with icterus and cholangiohepatitis, ascites, jaundice, and emaciation.

*Aspheteros pascualiformis* (22 mm, Opiothorchidae) occurs in gallbladder and bile ducts with ascites, weight loss, diarrhea, vomiting, and icterus.

*Parameisochis complexus* (10 mm, Opiothorchidae) occurs in bile ducts (see Fig. 4.139).

*Clonorchis sinensis* (Asia) (30-25 mm by 3-5 mm, Opiothorchidae) (see Fig. 4.10) occurs in gallbladder and bile ducts with occasional pancreatic duct involvement causing progressive liver cirrhosis.

*Platyosomum fastuosum* (*P. concinnum*) (8 mm, Dicrocoelidae) (see Figs. 4.21 and 7.67) occurs in tropical climates in gallbladder and bile ducts, causing anorexia, weight loss, vomiting, depression, mucoid diarrhea, jaundice, and hepatomegaly.

*Eurytoma praeconis* (3.5 mm) (Dicrocoelidae) occurs in pancreatic duct, bile ducts, and gallbladder, causing cirrhosis and pancreatic atrophy and fibrosis (Fig. 7.122).



Fig. 7.122 Long-axis ultrasonic image of the left lobe of the pancreas of a cat with *Eurytrema procyonis*. The pancreas is mildly enlarged, and the thickened walls of the mildly dilated pancreatic duct have a beaded appearance.

### Respiratory System

#### Nasal cavity, trachea, and bronchi

**Nematodes.** *Eucoleus (Capillaria) aerophilus* (Trichaelloidea) (females: 20–40 mm by 100–180  $\mu$ m; males: 15–25 mm by 60–100  $\mu$ m) (Fig. 7.104).

**Mammomonogamus spp.** (Syngnathidae) (females: 9–30 mm; males: 3.0–8.1 mm) (Fig. 7.123) occur in noses and nasopharynx; species seen in the middle ear. No clinical signs are reported for infections in cats.

#### Lung parenchyma

**Nematodes.** *Aelurostrongylus abstrusus* (Metastrongyloidea) (females: 9–10 mm by 80  $\mu$ m; males: 4–6 mm by 54–64  $\mu$ m) (Figs. 7.60 and 7.61; see also Figs. 8.58 and 8.59) occurs in terminal respiratory bronchioles and alveolar ducts, with the majority of signs being related to developing eggs lodged in the tissues; cats harboring large worm burdens may experience bronchopneumonia and show signs of open-mouthed abdominal breathing.

**Diofilaria immitis** (Filarioidea) allergic pneumonitis may be associated with immature adults. Death of these young stages can lead to bronchioalveolar disease, a manifestation known as Heartworm Associated Respiratory Disease, which presents with coughing, wheezing, and dyspnea. Also, adult worms found in ectopic locations such as these can elicit trauma and may impair organ function.

**Trematodes.** *Paragonimus kellicotti* (7–12 mm long, 4–8 mm wide, 4–6 mm thick) and other *Paragonimus* spp. outside the United States



Fig. 7.123 *Mammomonogamus auris* in the middle ear of a cat as viewed through an otoscope. (Courtesy Dr. Edgar Tudor, Paradise Animal Hospital, Seipon, United States.)

(Troglotremaidae) (see Figs. 4.14, 4.15, and 7.3 B) occur in nodules, typically in pairs or in greater numbers within the cysts; animals are generally without signs, but respiratory distress or even death may be associated with the infection.

### Vascular System

#### Heart and pulmonary arteries

**Protista.** *Trypanoplasma gondii* (Apicomplexa), cardiac muscle; may result in myocarditis and ventricular arrhythmias.

**Trypanosoma evansi** (kinetoplastid) (see Fig. 8.11) amastigotes in heart muscle cause acute myocarditis by myocardial invasion and cycles of multiplication and cell rupture.

**Nematodes.** *Diofilaria immitis* (Filarioidea) (see Figs. 4.167, 4.171, 8.71 to 8.74) occurs in arteries; cats very typically have few worms. Signs of infection are associated with cardiopulmonary disease and hypertension. Coughing, dyspnea, wheezing, and vomiting are reported. Dying worms can potentiate signs of Heartworm Associated Respiratory Disease (HARD).

**Tricostema cuniculi** (Ascaridoidea) larvae, granulomas (Fasson et al., 1990).

**Trichostrongylus axei** (Trichostrongyloidea) migrating prelarvae may cause myositis.

#### Microscopic veins

**Trematodes.** *Schistosoma japonicum* (females: 20 mm; males: 15 mm) (Schistostrongyloidea) in cats in southeast Asian countries. Infections appear to be asymptomatic.

#### Blood

**Protista.** *Cyanofoxon felis* (Theileriidae) (see Figs. 3.56 and 3.57 and 8.32) occurs with merozoites in erythrocytes and schizonts in macrophages in lumen of vessels in most organs. Cats may show signs of anemia, depression, anorexia, dehydration, fever, icterus, and enlargement of the liver and spleen.

**Nematode microfilariae.** *Diofilaria immitis* (Filarioidea) (see 7.71; see also Fig. 8.74) rarely produces microfilariae in cats; other filarid infect cats in other parts of the world.

### Skeletal Muscles

**Nematode larvae.** *Trichostrongylus axei* (Trichostrongyloidea) (see Figs. 4.182, 4.183, and 8.77).

### Connective Tissues

**Insect larvae.** *Catoptera* spp. (Diptera) (larva 30  $\mu$ m) (see Figs. 2.33, 7.105, 8.1, and 8.2) occur as migrating forms that vary in size. Larvae in these locations are typically asymptomatic. These organisms are covered in rows of dark spines and have two large black mandibles on the anterior end.

### Urogenital System

#### Bladder

**Nematodes.** *Tricostema cuniculi* (Ascaridoidea) larvae, granulomas (Fig. 4.153) (Fasson et al., 1990).

#### Urinary bladder

**Nematodes.** *Parascaris (Capillaria) pilosa* (females: 30–60 mm by 62  $\mu$ m; males: 13–30 mm by 68  $\mu$ m), and *P. feliscus* (females: 29–32 mm by 52–144  $\mu$ m; males: 25.5 mm by 52–64  $\mu$ m) (Trichostrongyloidea) (see 7.63). Obstruction of the ureters by worms can occur in cats with large numbers of *P. feliscus*. In these cases, signs associated with prostatic obstruction are reported.

### Nervous System

**Nematodes.** *Diofilaria immitis* (Filarioidea) adults migrate ectopically in meninges and ventricles (see Figs. 4.167 and 4.168) and may cause neurologic signs or death.

**Insect larvae.** *Caterella* spp. (Diptera) (larva 30 mm) (see Figs. 2.33, 7.116, 8.1, and 8.2) larvae can migrate through spinal cord and brain with clinical signs largely dependent on the path taken; seizures, vestibular signs, blindness, dermatitis, circling, disorientation, and death have all been noted.

#### Eye

**Protozoa.** *Toxoplasma gondii* (Coccidia) can cause iritis, uveitis, detached retina, iridocyclitis, keratic precipitates, mydriasis, anisocoria, and delayed pupillary reflex.

*Acanthamoeba* spp. (Amoebozoa) can cause facultative infections in the eye of cats leading to Acanthamoebic keratitis.

**Dipteran larvae.** *Caterella* spp. (Diptera) larvae range from a few millimeters to centimeters in size (30 mm), depending on their age (Fig. 7.116). These larvae are often light in color with rows of black black spines covering their bodies, becoming more dense as the host matures (see Fig. 2.33). Aberrant migration of these larvae into the eye resulting in ophthalmomyiasis externa and ophthalmomyiasis interna has been reported (Lidzanski et al. 2011).

**Skin and Hair.** Infestations may lead to dermatitis and pruritic anemia may be associated with severe infestations with blood-sucking types; tissue damage and necrosis may be associated with myiasis. Some of these organisms also act as vectors of disease agents.

#### Insects

**Adult insects.** Adult dipterans (biting flies).

*Felicola subrostratus* (Mallophaga) live clinging to the hairs of cats to which they attach their eggs (Fig. 7.124; see also Figs. 2.65 and 7.121).

*Ctenocephalides felis*, *C. canis*, and *E. gullinosa* (Siphonaptera) (see Figs. 2.36, 2.37, 2.45, and 7.30).

**Insect larvae.** Larval dipterans: primary or secondary myiasis (flystrike).

*Caterella* spp. (Diptera) ( $\approx 30$  mm) (see Figs. 2.33, 7.116, 8.1, and 8.2) larvae found in subcutaneous locations emerge through the skin of the cat after internal migrations and form a subcutaneous vesicle. The third-stage larvae and its spiracles can often be seen through the pore of the warble.

**Arachnida.** Dermacome spp., *Hemaphysalis leporispalustris*, and *Ixodes* spp. (Ixodidae) (see Figs. 2.73, 2.78–2.83, 2.90, 2.91).

*Notoedres cati* and *S. scabiei* (Sarcoptidae) (see Figs. 2.106, 2.108, 2.109, 2.113, and 7.36).

*Otiolaelus cynisii* (Psocoptidae) (see Figs. 2.107, 2.119, and 7.36).

*Erythraeus rasilvskyi* (Littorophoridae) clinging on to hairs may give the hair coat a salt-and-pepper appearance (see Figs. 2.122 and 7.36).

*Cheyletiella blakei* (Cheyletidae) all stages, including eggs attached to hair (Fig. 7.125; see also Fig. 2.126).



Fig. 7.124 Egg of *Felicola subrostratus* glued to a cat's hair.

*Demodex cati* (Demodicidae) (see Fig. 2.124) mites are found in the hair follicles of the cat, most often found on the eyelid, face, chin, and neck. Affected areas may have alopecia, crusts and scaling of varying degrees.

*Notoedres whartoni* and *Walchia americana* (Trombididae) (see Figs. 2.128 to 2.131). *Notoedres whartoni*, a bright red chigger, has been found in the external ear canal of cats. *Walchia americana*, normally a parasite of the gray squirrel *Sciurus carolinensis*, is capable of causing a severe and generalized dermatitis in cats (Lawson et al. 1976).

**Nematodes.** *Dracunculus imignis* (Spirurida) is sometimes recovered from subcutaneous nodules or ulcerations in the skin of cats and dogs that have been hunting frogs (Figs. 7.126 and 7.127). Care must be taken not to rupture the female during resection so as not to induce a strong inflammatory reaction at the site. Wounds tend to heal rapidly and without complications. Larvae can often be readily recovered from female specimens (Fig. 7.127).

#### Annotated Host-Organ Listing of Parasites of Ruminants

*Neogena certumina* may occur as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Fig. 8.29). *Toxoplasma gondii* may occur in any tissue of any host as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Fig. 8.28).



Fig. 7.125 Egg of *Cheyletiella blakei* attached to the hair of a cat.



Fig. 7.126 *Dracunculus imignis* adult worm extracted from the subcutaneous tissue of a cat.

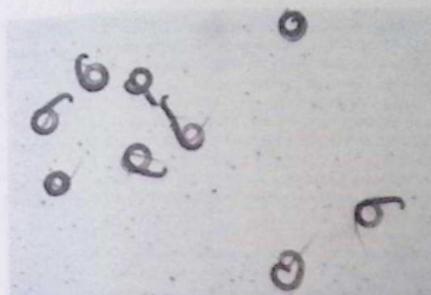


Fig. 7.127 *Diarcurulus insignis* first-stage larvae recovered from the fixation solution the adult worm was shipped in. Note the characteristic long tails.

### Alimentary System

#### Mouth, esophagus, and forestomachs

**Protozoa.** *Sarcocystis* (Apicomplexa) *sarcocystis* occur in muscles of the tongue and esophagus (see Figs. 8.26). Infection is asymptomatic. Calcified cysts can be seen on radiographs.

**Cestode larvae.** *Taenia* spp. (Taeniidae) cysticerci occur in muscle of the tongue (see Figs. 4.43 and 4.48), generally without ill effect.

**Insect larvae.** *Hypoderma lineatum* (Diptera: Hypodermatidae) in wall of esophagus. Treatments resulting in death of the larvae may incite inflammation, which may in turn result in difficulty swallowing and in bloat. These reactions, and indeed the bots themselves, are rare these days.

**Nematodes.** *Gyngyllemus pulchrum* (females: 80–145  $\mu\text{m}$  by 300–500  $\mu\text{m}$ ; males: 50–62  $\mu\text{m}$  by 150–300  $\mu\text{m}$ ), and *Gyngyllemus serruosum* (females: 70–85  $\mu\text{m}$  by 420–460  $\mu\text{m}$ ; males: 52–41  $\mu\text{m}$  by 250–300  $\mu\text{m}$ ) (Spirurida) (see Figs. 4.162, 4.163, 7.77, 7.78, 7.79, and 8.70) occur woven in a neat, sinusoidal pattern in the esophageal (G. pulchrum) or ruminal (G. serruosum) mucosa. Infections are asymptomatic.

**Trematodes.** *Cylophorus cylophorum*, *Paramphistomum cervi*, *P. bovis*, and *P. microbothroides* (Paramphistomatidae) (see Figs. 4.12 and 4.13). Adults in the rumen are not associated with clinical signs.

#### Abomasum

**Protozoan.** *Eimeria gibbata* (Coccidia) megacystis (see Fig. 3.28). Infections are without clinical signs.

**Cryptosporidium andersoni** (Apicomplexa) occurs usually without clinical signs (see Fig. 3.17).

**Nematodes.** Cattle: *Haemonchus contortus*, *H. placei*, *H. similis*, *Mecistocirrus digitatus*, *Ostertagia ostertagi*, O. (*Gyroscolopala*) *hyata*, *T. axei*.

Sheep and goats: *H. contortus*, O. (*Teladorsagia*) *circumcincta*, O. (*Gyroscolopala*) *occidentalis*, O. *trifurcata*, O. *orkelii*, O. (*Teladorsagia*) *daviana*, O. (*Pseudostertagia*) *hullea*, *Marshallagia marshalli*, *Trichostrongylus axei*.

Other related host species: *Haemonchus contortus*, *H. placei*, *M. digitatus*, O. *ostertagi*, O. *bicincta*, O. (*Teladorsagia*) *circumcincta*, O. *orkelii*, O. *trifurcata*, O. (*Gyroscolopala*) *hyata*, O. (*Gyroscolopala*) *occidentalis*, O. (*Teladorsagia*) *daviana*, O. (*Pseudostertagia*) *hullea*, *M. marshalli*, and *T. axei* (Strongylida: Trichostrongyloidea) (Figs. 7.74 and 7.75 and Table 7.9). Depending on the species of parasite, infection may cause anemia, diarrhea, abomasitis, and so on (Fig. 7.128; see also Fig. 8.54). Subclinical infections are also common.

TABLE 7.9 Nematodes in the Abomasum and Small Intestine

Genus	Length (mm)	Frequency
<b>Abomasum</b>		
<i>Haemonchus</i>	14–30	472/475
<i>Mecistocirrus</i>	43	477
<i>Ostertagia</i>	7–9	466/472
<i>Trichostrongylus axei</i>	7	470/472
<b>Small intestine</b>		
<i>Oxyuris</i>	6–16	472/478
<i>Trichostrongylus</i>	6–7	470/472
<i>Asmatidius</i>	20–25	472/476

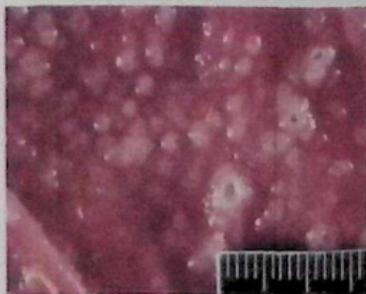


Fig. 7.128 Lesions caused by members of the Ostertagiinae. (A) Lesions in the abomasum of a cow caused by larvae of *Ostertagia ostertagi*. (B) Similar lesions in the C2 gastric compartment of an alpaca. (Photograph courtesy Dr. Mary C. Smith.)

#### Small intestine

**Nematodes.** Cattle: *Trichostrongylus axei* (Acuroidae) (females: 22–30 cm by 3–6 mm; males: 15–26 cm by 3–5 mm) is only rarely seen in the United States although it can be common in the developing world. It has an esophageal ventriculus and produces subperic egg with a pitted *T. axei*-like shell surface. *Ascaris suum*, a very rare,

aberrant parasite of ruminants, lacks a ventriculus and produces ellipsoidal eggs with a mammillated shell surface.

Cattle: *Cooperia oncophora*, *C. punctata*, *C. punctata*, *C. spatulata*, *Trichostrongylus colubriformis*, *T. longispicularis*, *Nematodirus helvetianus*, *N. spathiger*.

Sheep and goats: *Cooperia curvica*, *T. colubriformis*, *T. longispicularis*, *T. capricola*, *T. vitrimus*, *N. spathiger*, *N. filicollis*, *N. battus*.

Other related host species: *Cooperia curvica*, *C. bisona*, *C. onchophora*, *C. pectinata*, *C. punctata*, *C. occidentalis*, *T. colubriformis*, *T. longispicularis*, *T. capricola*, *T. vitrimus*, *N. helvetianus*, *N. spathiger*, *N. filicollis*, *N. abnormis*, *N. larcolatus*, and *N. battus* (Strongylidae: Trichostrongylidae) typical associated sign in heavy infections is diarrhea (Table 7.3; see also Fig. 8.50).

*Bunostomum phlebotomum* (cattle) (females: 18–19 mm by 500–600 µm; males: 10–12 mm by 470–475 µm) and *B. trigonocephalum* (sheep) (female: 19–26 mm; male: 12–17 mm) (Anchylostomidae) (see Fig. 4.107) are capable of causing anemia in younger animals with heavy infections.

*Strongylus vitalis* (cattle) and *S. papillosus* (most ruminants) (parthenogenic females: 3.5–6 mm, Rhabditidae) (see Fig. 4.131) can cause diarrhea and anemia when present in large numbers. Neurologic signs and sudden death syndromes have also been reported. Infection can be transmitted in milk or percutaneously.

*Ancylostoma (Capillaria) bovis* (mainly cattle, female: 18–25 mm by 80–116 µm; males: 11–13 mm by 50–75 µm) and *Ancylostoma (Capillaria) brevipos* (mainly sheep, female: 12 mm by 60 µm; males: 8–9 mm by 50 µm) (Trichinelloidea) (Fig. 7.30) occur without association with clinical signs.

*Oncophorus* spp. third- and fourth-stage larvae (Strongylidae) (see Figs. 4.101 and 4.102, 8.56, and 8.57) in the mucosa. These occur in nodules in sensitized animals, which can interfere with digestion in heavily infected animals leading to diarrhea and wasting.

Cestodes. *Moniezia expansa* and *M. benedeni* (Anoplocephalidae) (Fig. 7.123; see also Figs. 4.55, 4.56, and 7.70) typically occur with no clinical signs.

*Thysanotoma acrostoides*, *Wyominia itani* (Anoplocephalidae) typically occur with no clinical signs.

*Thysanotus*, *Selisia*, *Arivollina* (Anoplocephalidae) are exotic anoplocephalids of ruminants. Infections are asymptomatic.

Trematodes. Immature paramphistome flukes (*Paramphistomum* and *Capyllophorum*) can cause enteritis with associated diarrhea prior to their migration to the rumen.

Protozoa. *Eimeria* spp. (Coccidia) (see Figs. 7.76, 7.78, and 8.17, and Table 7.4.30–7.7), depending on the species involved, can cause severe enteritis with bloody diarrhea; stages may be visible in wet mount scrapings of the intestinal mucosa.

*Cryptosporidium parvum*, *C. bovis*, and *C. andersoni* (Apicomplexa) occur, with the *C. parvum* being an important cause of diarrhea in calves, and other hoofed-stock, less than 30 days of age (see Figs. 3.16 and 3.17).

*Giardia* spp. (flagellate) (Fig. 7.67) can cause diarrhea in young animals and sometimes adults. Assemblages A, B, and E affect ruminants.

#### Cecum and cecum

*Nematodes*. *Oesophagostomum radiatum* (cattle), *O. columbianum* (sheep and goats), *O. venulosum* (sheep and goats), and *Chabertia ovina* (sheep and goats) (for all four species, females: 13–24 mm by 300–600 µm; males: 11–17 mm by 230–400 µm) (Strongylidae) (see Figs. 4.87 to 4.102). The fourth-stage larvae of *O. radiatum* in cattle and *O. columbianum* in sheep may be found in abscesses in the gut wall (see Fig. 4.87).

*Traillius discolor* (52 mm, cattle) and *T. ovis* (70 mm, sheep and goats) (Trichinelloidea) (Fig. 7.29) can be associated with diarrhea in animals that are severely infected.

*Skipshinema ovis* and *S. caprai* (Oxyuridae) (both species, females: 5–10 mm by 300–520 µm; males: 2.3–5.7 mm by 180–180 µm) infections are usually without clinical signs (Fig. 7.13).

Protozoa. *Eimeria* spp. (Coccidia) (see Figs. 7.76 and 7.80, and Figs. 3.20 to 3.28 and 8.17, and Table 7.4 to 7.7).

*Haemonchus bovis* and other species of *Haemonchus* are considered non-pathogenic parasites or commensals of the large bowel of ruminants. *Haemonchus salicatus* (dillie) is a commensal of the large bowel of cattle (Fig. 7.3).

#### Liver

Nematodes. *Ascaris suum* (Ascarididae) of swine will on rare occasions appear in the bile ducts of sheep and cattle.

Nematode larvae. The larvae of *T. vitellorum* undergo a liver-lung migration on their way back to the intestine after the infective eggs are ingested.

*Ascaris suum* (Ascarididae) and *S. abnormis* (Strongylidae) of swine (see Figs. 4.105, 7.15, 7.16) have larvae that can migrate through the bovine liver and cause severe trauma.

Cestodes. *Thysanotoma acrostoides* and *W. itani* (Anoplocephalidae) can sometimes be found in the bile ducts of ruminants into which they have migrated soon after the animal's death, with rapid ligation of the duct, the worms will be found in the small intestine.

Cestode larvae. Hydatids of *E. granulosus* and *E. multilocularis* (Taeniidae) (see Figs. 4.49 to 4.53, 8.45, and 8.46) cause signs that may be severe depending on the location and size of the cysts produced.

Cysticercs of *T. hydatigena* (Taeniidae) (see Fig. 4.43) may be present without clinical manifestations. Migration of larvae through the liver can cause tracks and fibrosis, which can result in liver condemnation.

Trematodes. *Fasciola hepatica*, *F. gigantica*, and *Fascioloides magna* (Fasciolidae) (Fig. 7.109; see also Figs. 4.2 and 4.11, 7.70, 8.24, 8.35 and 8.37 to 8.39). *Fasciola hepatica* (30 mm) is endemic in western and Gulf States of the United States and in Hawaii, Puerto Rico, British Columbia, and eastern provinces of Canada. *Fasciola gigantica* (75 mm) is endemic in Hawaii and Africa. *Fascioloides magna* (100 mm) occurs in foci throughout North America; remember that the final host is typically the white-tailed deer. The migratory tracts and lesions produced by *F. magna* can be marked in ruminants with large deposits of black "flake pigment," often fatal in smaller



Fig. 7.123 *Moniezia benedeni* in the intestine of a cow at necropsy.



Fig. 7.130 Liver of a sheep that has been fatally infected with *Fasciolosis magna* showing the typical lesions and deposits of black "bile pigment."

ruminants. These infections may produce anorexia, painful abdominal distension, hepatic fibrosis, and hyperplastic cholangitis.

*Dicrocoelium dendriticum* (10 mm) (Europe, Asia, Africa, South America) has been introduced into North America, and it occurs in central New York State and the Pacific Northwest; it causes chronic hepatic fibrosis (see Fig. 4.20).

*Farysenia panaminticensis* (16 mm) (Asia and Brazil) (Dicrocoelidae) (see Fig. 3.69) may be associated with a failure to thrive.

#### **Peritonema and peritoneal cavity**

**Nematode.** *Setaria labatopapillifera* (Filarioidae) (see Fig. 4.174) are large white filarioids (females: 40–60 mm by 380–450  $\mu$ m; males: 10–12 mm by 470–475  $\mu$ m) that are sometimes found in incidental findings in the abdominal cavity of cattle.

**Cestode larvae.** Cysticerci of *T. hydatigena* (Taeniidae) (see Fig. 4.43) that often have a long "neck" anterior to the scolex. Often present without clinical signs. Migration of larvae through the liver can cause tracks and fibrosis.

**Pentastomid nymphs.** *Lingostola serrata* (Pentastomida) (see Figs. 2.137 and 2.139 and 8.9) larvae can be found in the abdominal cavity of viscera of ruminants, most commonly in Africa. Larvae may cause injury, inflammation, and space-occupying lesions depending on site and number of organisms present.

### **Respiratory System**

#### **Nasal cavity and paranasal sinuses**

**Insect larvae.** *Oestrus ovis* larvae in sheep and goats (Oestridae) (see Fig. 2.24) are found in the nasal sinuses; they may be rather small or quite large (10–20 mm) depending on their developmental age. May be associated with sinusitis as larvae can irritate the mucosa with their mandibular hooks and cuticular spines. Sneezing and nasal discharge may be present.

#### **Trachea and bronchi**

**Nematodes.** *Dictyocephalus viviparus* (cattle) (Trichostrongyloidea) (females: 23–80 mm long; males: 17–50 mm by 500  $\mu$ m) is the only lung-dwelling nematode found in cattle; these can cause severe respiratory distress when present in large numbers (Fig. 4.90).

*Dictyocephalus filaria* (sheep and goats) (Trichostrongyloidea) (females: 43–112 mm long; males: 25–80 mm long) (see Figs. 4.89 and 7.70) can cause respiratory distress in the infected host.

*Protostrongylus rufescens* (Metastrongyloidea) The red lung worm of sheep and goats (females: 25–65 mm by 150–200  $\mu$ m; males: 16–46 mm by 120–170  $\mu$ m) (see Figs. 4.76 and 7.71). Infection is generally asymptomatic but may predispose to bacterial infection.

*Mammomonogamus laryngus* (Syngamidae) (females: 8.7–9.8 mm by 350–370  $\mu$ m; males: 3.0–3.4 mm by 360–380  $\mu$ m) (Figs. 7.131 and 4.106). Blood red worms of cattle. Male and female worms are fused in copula; they are endemic in Puerto Rico and various Caribbean islands. These worms have a large stronglyd buccal capsule. If large numbers are present, coughing and loss of condition may be seen.

#### **Long paracystoma**

**Nematodes.** *Muelleria capillaris* (Metastrongyloidea) (sheep and goat) (females: 19–23 mm by 40–50  $\mu$ m; males: 11–14 mm by 32–35  $\mu$ m) (see Fig. 4.117). Infection is generally asymptomatic. Heavy infections may be associated with secondary bacterial infections. Interstitial pneumonia and coughing may occur.

**Larval nematodes.** *Oesophagostomum columbianum* larvae (erratic migration) (see Fig. 4.102).

Both *T. vividiorum* and *A. suum* are capable of causing and have caused respiratory signs in cattle during larval migration (see Fig. 8.66).

**Cestode larvae.** *Echinococcus granulosus* (Taeniidae) (see Figs. 4.50, 4.53, 8.45, and 8.46) cysts can become quite large when present in lung tissue. Severity of respiratory signs depends on the number and size of these cysts.

### **Vascular System**

#### **Heart**

**Cestode larvae.** *Taenia saginata* (Taeniidae) cysticerci are found in the muscles of cattle in the United States.

*Taenia ovis* (Taeniidae) cysticerci are found in the various muscles of sheep and have recently appeared among sheep in Canada (Fig. 7.132). Clinical signs are not present.

#### **Arteries**

**Nematodes.** *Elastophora schneideri* (sheep) (Filarioidae) (females: 60–120 mm by 500–800  $\mu$ m; males: 55–85 mm by 480–685  $\mu$ m) occurs in the western United States. Deer appear to be the normal host and are not clinically affected. Sheep may have facial dermatitis associated with the microfilariae.



Fig. 7.131 Muscle of a sheep from Canada with three evident cysticerci of *Taenia ovis*. (Photograph courtesy Dr. Andrew Peregrine, Ontario Veterinary College, University of Guelph, Ontario, Canada.)

*Elaeophora peeli* (cattle; Filarioidea) (females: 40–300  $\mu\text{m}$  by 350  $\mu\text{m}$ ; males: 45–70  $\mu\text{m}$  by 200–260  $\mu\text{m}$ ) is an exotic infection in Africa and Asia. May lead to arteriitis without any apparent clinical signs.

*Onchocerca armillata* (cattle; Filarioidea) (females: 78  $\mu\text{m}$  by 235–375  $\mu\text{m}$ ; males: 74–84  $\mu\text{m}$  by 200–230  $\mu\text{m}$ ) is an exotic infection in Africa and Asia. Clinical signs are not reported.

#### Veins

Trematodes. *Schistosoma* spp. (Schistosomatidae) (see Fig. 4.27) are all exotic. *Schistosoma japonicum* is found in Asia with a wide mammalian host range. Species in cattle, sheep, and goats include *S. bovis* (Africa, Asia, southern Europe) and *S. nasuti*, *S. matthei*, *S. indicum*, *S. spindale*, and *S. turkistanica* (Asia). Diarrhea or dysentery may result from heavy infection with most of these species. Anemia and emaciation are seen in chronically infected animals. *S. nasuti* causes snoring disease, in which severely affected animals present with rhinitis, profuse mucopurulent nasal discharge, sneezing, dyspnea, and snoring.

#### Lymph nodes

Feuistomatids. *Linguatula serrata* larvae.

#### Blood

Nematode microfilariae. *Setaria labracapillaria* (cattle; Filarioidea); *Elaeophora schenckii* (sheep; Filarioidea). May cause filarial dermatitis of the face.

Protozoa. *Babesia bigemina*, *B. bovis*, *B. divergens*, *B. argassiana*, *Theileria orientalis*, *T. parva*, *T. annulata*, and *T. mutans* (Piroplasmorida) (see Figs. 3.50, 3.51, and 3.55) are all basically exotic at this time in the United States. Infection leads to anemia.

*Trypanosoma theileri* (60–70  $\mu\text{m}$  and sometimes up to 120  $\mu\text{m}$  in length; cattle) (Fig. 3.3) and *T. encephalophagum* (50–60  $\mu\text{m}$ ; sheep) (hemoflagellates). Rarely seen in blood films, these organisms are readily demonstrable by blood culture, but are considered to be nonpathogenic. There are different pathogenic species, *T. brucei*, *T. congolense* and *T. evansi* that fortunately do not occur in North America (Figs. 3.1 and 3.2).

#### Skeletal Muscles and Connective Tissues

Cestode larvae. *Taenia saginata* (Taeniidae) cysticerci are found most frequently in the muscles of mastication, tongue, heart, and muscular portion of the diaphragm of cattle; scoxles with four suckers but no hooks. Clinical signs are not expected.

*Taenia hydatigena* (Taeniidae) (see Fig. 4.45) cysticerci are sometimes found in skeletal muscles but more commonly in liver or on peritoneal membranes. Clinical signs are not expected.

*Taenia ovis* (Taeniidae) cysticerci are pea-sized vesicles found in the heart and esophagus and beneath the epicardium and diaphragmatic pleura of sheep and goats (Fig. 2.55). Clinical signs are not expected unless cysts interfere with organ function.

Insect larvae. *Hypoderma bovis* and *H. lineatum* (Hypodermatidae) (see Fig. 2.26) larvae overwinter in the northern climates within cattle, with *H. bovis* being in the spinal canal and *H. lineatum* being in tissues around the esophagus. Treatments resulting in death of the larvae may incite inflammation, which may in turn result in neurologic signs, difficulty swallowing, and bloot. Prevalence of these bots has greatly decreased; thus such reactions are a rare occurrence.

Nematodes. *Onchocerca gutturosa*, *O. lienalis*, *O. bovis*, and *O. gibsoni* (Filarioidea). Adult *Onchocerca* worms (females: 140–520  $\mu\text{m}$  by 200–600  $\mu\text{m}$ ; males: 8–55  $\mu\text{m}$  by 72–190  $\mu\text{m}$ ) are found in deep connective tissues, whereas microfilariae are present in the dermis. In Australian cattle, *O. gibsoni* produces nodules in the brain that require extensive trimming. We have seen *O. gibsoni* in corned



Fig. 2.522 Computed tomographic image of the cranial portion of the head of a llama taken in the Cornell Hospital for Animals showing thickened lesions that were identified histologically as being sarcocysts.

beef purchased in a local supermarket. Adult parasites are of low pathogenicity.

Protozoa. *Sarcocystis* spp. (Apicomplexa) sarcocysts in muscle (Fig. 2.522); see also Table 3.1 and Figs. 8.24 to 8.26). Infections are subclinical.

#### Urugental System

Protozoa. *Trichostrongylus axei* (Flagellate) (see Figs. 3.7 and 3.8). These pear-shaped flagellates are 10 to 25  $\mu\text{m}$  by 5 to 15  $\mu\text{m}$ . They are found in the preputial cavity of bull and the vagina, uterus, and cervix of cows infected during calving. Vaginitis with or without mucopurulent discharge, placentitis, early-term abortion, and sterility are reported.

*Taeniaia gonidi* (Apicomplexa) placentas of aborting previously naive sheep or goats.

*Nematode caninum* (Apicomplexa) placentas of aborting cattle. Horizontal or vertical inflexion may lead to abortion even in subsequent pregnancies. Do not feed dogs aborted fetuses and placentas.

#### Nervous System

##### Brain, spinal cord, and meninges

Protozoa. Sarcocystis-like organism (Apicomplexa) in brain of cattle (Lambert et al., 1997). Infection was associated with multifocal granulomatous meningoencephalitis with focal malacia and resulted in ataxia and blindness.

Nematode. *Parabrostrongylus tenuis* (Pristionyxidae) (see Figs. 4.118, 8.62, and 8.63). Adults are typically found in white-tailed deer. Larvae and young adults that develop in sheep and goats (and other ungulates) migrate through the spinal cord and brain, causing paralysis. Infections in cattle are rare but are reported.

Cestode larvae. *Taenia multiceps* (Taeniidae) occurs in the brain of sheep and goats causing "gid" (see Fig. 4.47); the organism is exotic and supposedly no longer in North America. The cesturus can lead to neurologic signs due to increased pressure on the brain through its increasing size.

Insect larva. *Hypoderma bovis* (Hypodermatidae) larvae in the spinal canal of cattle. May be associated with neurologic signs.

##### Eye

Nematodes. *Thelazia californiensis* (sheep); *T. gulosa* (cattle), and *T. skrjabini* (cattle) (Spirurida) (females: 11–19 mm; males: 5–13 mm).

in conjunctival sac and lacrimal duct (see Fig. 4.166) may be associated with conjunctivitis and development of granulation tissue.

*Setaria cervi* (Filarioidea) has been reported in the eye of a goat.

### Skin and Hair

#### Insects

Dipteran adults. *Musca autumnalis*, *S. calcitrans*, and *H. irritans* (Muscidae) (see Figs. 2.14, 2.15, and 2.16) adults spend a good deal of time on cattle; *S. calcitrans* is more likely to rest off of cattle when not feeding. *Musca autumnalis* is mainly a nuisance and can distract animals from feed and water intake or from rest. The other two flies act in similar fashion, but also inflict painful bites and ingest blood. They can result in loss of condition and anemia.

*Glossina* spp. (Africa) (see Fig. 2.17). Flies feed on blood and can transmit trypanosomiasis.

*Melophagus ovinus* (Hippoboscidae) (see Fig. 2.18) pupae and adults of the kind are found on the fleece. Adults suck blood and elicit pruritus and wool breakage in sheep.

*Hypoderma bovis* and *H. lineatum* (Hypodermatidae), the gadflies, are rarely seen as they hover about cattle gluing their eggs to hairs on the animals. These flies are said to be capable of causing stampedes.

Tabanidae (see Figs. 2.10 and 2.11) land on cattle typically only long enough to feed on blood. These are painful bites that can also serve as vectors.

Dipteran larvae. *Hypoderma bovis* and *H. lineatum* (larvae 30 mm, Hypodermatidae) (see Figs. 2.23 and 2.28) bots mature in warbles in the skin of cattle typically along the back of the animal. These can damage the hide and result in economic loss.

Calliphoridae and Sarcophagidae (see Figs. 2.13, 2.20, 2.21) maggot can be serious pests of ruminant, newborn, and animals that are wounded or down and soiled for an extended period. They are either facultative or obligatory myiasis causing species. *Cochliomyia hominivorax* is identifiable by its pigmented posterior tracheae; it is reportable in North America.

Anoplurans. *Hemaphysalis rufipes*, *H. quadrifasciata*, *H. tuberculosa*, *Leontopithecus vitalis*, *Solenopotes capillaris* (cattle), *L. ovalis*, *L. padalis*, *L. ovisformis* (sheep), *L. equorum*, and *L. nematus* (goat) (see Figs. 2.50, 2.52, 2.53, 2.56, and 2.57). Sucking lice may be associated with pruritus, anemia, wool or fiber breakage, and loss of condition in heavy infestations.

Mallophagans. *Bovicola bovis* (cattle), *B. ovis* (sheep), *B. caprae*, *B. leontinae*, *Damaelina (Holoferites) crassipes* (goats) (see Figs. 2.62 and 2.67). Chewing lice may be associated with pruritus, anemia, wool or fiber breakage, and loss of condition in heavy infestations.

Siphonaptera. *Echinophaga gallinacea* (see Figs. 2.37, 2.45, and 2.58) heavy infestation may result in pruritus and anemia.

Ctenocephalides felis can cause severe distress in calves and has even been reported to cause the death of calves, lambs, and sheep, although mainly in tropical settings overseas (see Figs. 2.36 and 2.39). Pruritus and anemia are expected in heavy infestation.

#### Arachnida

Metastigmata: Ixodidae. Tick infestations may cause distress, anemia, and lesions that may predispose animals to fly-strike.

*Anoblyonema americanum*, *A. capense*, *A. maculatum*, *A. inornatum* (Mexico), *A. oblongoguttatum* (Central and South America), and *A. variegatum* (imported to Caribbean from Africa, eradication from area in process) (see Figs. 2.78 and 2.79).

*Rhipicephalus annulatus* and *R. microplus* (see Figs. 2.84 to 2.88, and 2.89); *R. annulatus* is considered exotic and should be reported if found on cattle.

*Dermacentor Andersoni*, *D. albipictus*, *D. occidentalis*, *D. variabilis*, and *Dermacentor (Oliventor) nitens* (see Figs. 2.90-2.92, and 2.93).

*Ixodes cookei*, *I. pacificus*, *I. scapularis* (see Figs. 2.79 to 2.82, and 2.83). *Hemaphysalis longicornis* is newly reported in the eastern United States, and its distribution is expected to increase.

Metastigmata: Argasidae. *Odobius nigrom* (spinose ear tick) (see Figs. 2.77 and 2.80), with larvae and nymphs in the ears. Infestations cause irritation and pain. Head shaking and signs of discomfort are the norm. Often large numbers of immature ticks are present in the ear canal.

*Oribothadros corvaceus* and *O. varians* (see Fig. 2.76) will get on the hosts only long enough to feed. Their bites elicit pruritus and anemia, when in large numbers. Dermatitis may be associated with these temporary infestations.

Argasidae. *Sarcoptes scabiei* (see Figs. 2.106, 2.108, 2.109, and 2.84) can cause severe dermatitis, especially in cattle.

*Chorioptes bovis* (see Figs. 2.107, 2.117, 2.118, and 2.85) is common and often without clinical signs, but pruritus and itchy dermatitis may be seen.

*Psoroptes ovis* (see Figs. 2.108, 2.115, 2.116, and 2.86) is considered eradicated for the most part from the United States, but this or other very similar mites turn up in the ears of llamas and other American camelids, various wild sheep, and cattle in the southern and western parts of the United States. Infestations can be associated with lichenification of the skin, alopecia, and erosive dermatitis.

Prostigmata. *Dermodes bovis*, *D. ovis*, and *D. caprae* (see Figs. 2.124, 2.125, 8.5, 8.6, and 2.87) can cause very large nodular lesions in the skin of goats and cattle, each containing thousands of mites. Follicular rupture may occur. Infestations may damage the hide.

*Phthirus bovis* (cattle) and *Phthirgaster ovis* (sheep and goats) (Phthirgasteridae) are the ruminant itch mites. Infestations are often subclinical; some animals may exhibit mild pruritus.

Trinobocidae (see Figs. 2.128 to 2.131). Chiggers are larvae of free-living adult mites and can cause severe pruritus often localizing within the ears. These stages can be characterized by having six legs, plumose setae, and a small scutum.

Meosostigmata. *Raillietia ovis* (1 mm cattle) and *R. caprae* (goats). Ear mites (see Fig. 2.100) living near the tympanic membrane. Head shaking, head tilt, and decreased food intake are sometimes seen.

*Psyllidia Resusina haemeri* (Coccidia), exotic. Cysts on skin can degrade the value of the hide. Fever and edema of the extremities may be present early in infection. Anorexia, tachypnea, diarrhea, and abortion are reported at the early stage. Disease may progress to hair loss, thickening (and sometimes cracking) of the skin; avascular necrosis may be produced from lesions. Severely affected animals may become emaciated.

*Monostolus*. *Stephanofilaria stilesi* (Filarioidea). Very small adult (females: 5.0-5.8 mm by 100-117  $\mu$ m; males: 3.0-3.5 mm by 40-50  $\mu$ m) filarids in skin of ventral abdomen of cattle causing erosive lesions during the grazing season, exotic.

*Bursellaria bovis* (Filarioidea). Adults (female: 4-5 cm by 400-500  $\mu$ m; male: 2-3 cm) in subcutaneous tissues cause "summer bleeding" in cattle, exotic.

*Nematode larvae*. *Oribovetres guttorum*, *O. linealis*, and *O. bovis* (Filarioidea). Microfilariae found in dermis of cattle.

*Elaphophora schrenkleri* (Filarioidea) microfilariae can be found in the skin, usually in the head region.

*Rhabdus strongyloides* (Rhabditiida) (see Figs. 4.128, 4.129) larvae will enter the hair follicles of animals on occasion if they are resting on damp hay or other bedding, can lead to dermatitis.

## Annotated Host-Organ Listing of Parasites of Horses

## Alimentary System

## Mouth

**Insect larvae.** *Gastrophilus intestinalis*, *G. nasalis*, and *G. haemorrhoidalis* (Diptera: Gastrophilidae) (Fig. 7.133; see also Figs. 2.23 and 2.27 to 2.31) larvae can be found in the tongue, in the interdental pockets, or at the base of the tongue. These larvae will be smaller and lighter in color than their more mature counterparts in the gastrointestinal tract. Infections are typically subclinical, but inflammation in the mouth and tongue could be present with associated pain and difficulties in eating or swallowing.

**Protozoan.** *Trichomonas equibuccalis* (mucosoflagellate) is found around gum margins of cheek teeth. Infection is asymptomatic.

## Stomach

**Nematodes.** *Dracchia megastoma*, *H. muscae*, and *H. microstoma* (Spirurida) (see Fig. 4.165) are found in the stomach, with *H. muscae* (females: 12–22 mm; males: 8–14 mm) and *H. microstoma* (females: 15–35 mm; males: 9–22 mm) being on the mucosa, and *D. megastoma* (females: 10–13 mm; males: 7–10 mm) in nodules at the *margo plicatus* (Fig. 7.133). Their presence at this site is not associated with clinical signs, although there may be increased mucus secretion, gastritis, and production of tumor-like fibrotic nodules that may contain caseous or necrotic material.

**Trichostrongylus axei** (Trichostrongyloidea) (females: 3.2–8 mm by 55–70  $\mu$ m; males: 2.3–6 mm by 30–60  $\mu$ m) (see Figs. 4.78 and 4.80) infections may cause hypertrophic gastritis with mucosal proliferations and are often associated with shared pasturage with cattle.

**Insect larvae.** *Gastrophilus intestinalis* (Diptera: Gastrophilidae) (Fig. 7.133; see also Figs. 2.23, 2.27 to 2.31), even with their specific designation, are found in the stomach. May cause ulcerations at their site of attachment, yet infections are typically subclinical. Large numbers could impact performance.

## Small intestine

**Nematodes.** *Parascaris equorum* and *P. univolvus* (Ascaridoidea) (females: 18–50 cm by up to 8 mm; males: 15–28 cm by 5–8 mm) (Figs. 7.134, and see Fig. 7.6) is seen in foals and in some adult horses. In young (3–9 months) horses without sufficient immunity to maintain only small numbers of adult worms, large numbers of mature worms compete for host nutrients and lead to ill-thrift, poor hair coat,



Fig. 7.134 Linear ulcer in a horse associated with a very heavy infection with *Parascaris equorum* seen at Cornell University in 1994. (Courtesy Dr. John M. King.)

pot-bellied appearance, and may cause diarrhea, obstruction, perforation or rupture of the intestine.

**Strongylidae worm** (Rhabditida) (parthenogenic females: 8–9 mm by 80–95  $\mu$ m) (see Figs. 4.127, 4.130 to 4.32, 7.99, and 8.53) is very small and threaded through the intestinal mucosa. This parasite may be associated with neonatal diarrhea in foals acquiring the parasite through nursing.

**Cestodes.** *Anoplocephala magna* (up to 800 mm by 25 mm), *Pentastrophale mammillata* (10–40 mm by 6–8 mm) (Anoplocephalidae) (see Figs. 4.57, 4.58, and 7.93). These large tapeworms are typically asymptomatic. Heavy infection with *A. magna* has been reported to cause hemorrhagic enteritis.

**Protista.** *Cyathostomum* spp. (Apsicomplexa) can cause serious watery diarrhea and dehydration in neonatal foals (see Figs. 3.16 and 7.93).

**Isomere leuckartii** (cestode) (Fig. 7.93; see also Fig. 8.18) has large scolices and oocytes that may be demonstrated in mucosal scrapings. Infections are asymptomatic, and typically seen only in foals.

**Giardia** spp. (flagellate) (Fig. 7.93, 7.94) can be found as trophozoites in light scrapings of the mucosal surface of the anterior small intestine. Clinical signs are not typical of infections in older animals, but young animals may have associated diarrhea.

**Insects.** *Gastrophilus nasalis* and *G. haemorrhoidalis* (Diptera: Gastrophilidae) larvae are found in the duodenum. Infections are typically subclinical. Inflammation and ulceration may be seen at the site of attachment.

## Large intestine

**Nematodes.** *Oxyuris equi* (females: 40–150 mm; males: 9–12 mm) and *P. vivipara* (females: 3 mm; males: 2 mm) (Oxyuridae) (see Figs. 4.134 to 4.136, and 7.96). *Oxyuris equi* (Fig. 7.97) is commonly seen because the females crawl out of the anus. The female's behavior at the time of egg laying, and the material with which eggs become adhered to the perianal region leads to tail rubbing. *Probstmayra vivipara* is almost never seen as the female is small and gives birth to live young within the intestine (Fig. 7.135), such infections are inapparent.

**Family Strongylidae:** The horse is host to about 60 species belonging to the family Strongylidae, and as many as 20 different species are often found in the same horse.

**Subfamily Strongylinae:** *Strongylus vulgaris* (up to 24 mm), *S. edentatus* (up to 45 mm), *S. equinus* (up to 47 mm), *Triodontophorus*



Fig. 7.133 Stomach of a horse showing the attachment of bots, *Gastrophilus intestinalis*, and a lesion produced at the *margo plicatus* by an infection with the spirid *Dracchia megastoma*.



Fig. 7.136 *Oxyuris equi* adults recovered from a horse at necropsy.

*sericus*, *T. berriensis*, *T. tenuicollis*, *T. nipponicus* (all *Trichostrongylus* medium-sized worms up to 25 mm long), *Oesophagostomum robustus* (up to 24 mm), and *Charavotiostomum acuminatum* (small worm up to 10 mm) (Fig. 7.439; see also 7.139 [bottom row], 8.49, and 8.55). Larvae of these "large strongyles" all mature in nodules in the bowel wall. These may become inflamed and have pinpoint ulcerations at the time of L4 emergence into the lumen. Very heavy larval infections may lead to colic, diarrhea, and failure to thrive. Adults in the human are plug feeders, which cause hemorrhagic ulcers at the sites of attachment. *Trichostrongylus* spp. feed in clusters, and thus may cause deep hemorrhagic ulcers where they are attached. Worms in the lumen ingest blood and large numbers could cause anemia.

**Subfamily Cyathostominae:** Genera: *Cyathostomum* (up to 12 mm), *Cylicocyclus* (10–25 mm), *Cylicostephanus* (up to 10 mm), *Cylicodontophorus* (up to 14 mm), *Poturiostomum* (up to 21 mm), *Pentapostomonum*, *Pentastoma*, *Ceratomyxus*, and *Gyaloclypeus* (up to 11 mm) (Figs. 7.437 to 7.443). The "small strongyles" are parasites of the intestine at all stages of their life, whose infections are typically inapparent. Very heavy infections cause damage to the intestinal mucosa as larvae emerge into the lumen after long periods of maturation. Inflammation of the mucosa may be apparent. When synchronous reactivation of very large numbers of hypobiotic larvae occurs, typically seen in late fall, winter, or early spring, severe and acute damage to the mucosa leads to profuse watery diarrhea, hypalbuminemia, and edema. Large numbers of immature red worms may be flushed out of the host at this time.

Each species can be identified by careful study of the stool region alone. With fresh specimens, detail sufficient for identification can be seen without recourse to clearing agents. Simply mount the specimen under a coverslip in a drop of water. With this simple preparation it is usually possible to roll the specimen so that both dorsal and lateral aspects may be studied. Even preserved specimens may be studied in this manner but tend to be considerably less transparent than fresh specimens. For comparisons to be made easily, illustrations of the species that bear the greatest resemblance to one another have been grouped together. The nomenclature used in the excellent body of work by Lichtenfels (1975) and by Lichtenfels et al (1998, 2008) is the system that has been applied in the following pictorial key (Figs. 7.436 to 7.487).

**Castode.** *Anoplocephala perfoliata* (*Anoplocephalidae*) (up to 80 mm long but usually 25–40 mm by 8–14 mm) (see Figs. 4.58 and 7.55)

is found mainly in the cecum; this tapeworm also tends to cluster in the ileum near the ileocecal valve, where it is associated with ulceration and chronic inflammation of the ileal wall, intussusception, and colic.

**Insect.** *Gasterophilus haemorrhoidalis* (Diptera: *Gasterophilidae*) larvae sometimes attach briefly as they make their way out of the intestinal tract into the environment. These temporary attachments may be associated with discomfort, pain, and colic.

#### Liver

**Nematode larvae.** *Parascaris equorum* (*Ascariidae*) passes through the liver on its way to the lung after the infective-stage eggs are ingested. May cause focal granulomas.

*Strongylus edentatus* and *S. equinus* (see Figs. 8.49, 8.55, and 4.71, 4.94) will wander through the liver for a period of time before patency, causing focal granulomas.

**Castode larvae.** *Echinostaco equinus* (*Trematode*) (see Figs. 4.50 and 4.51) hydatid cysts are very rare in horses of most of the world, and especially so in the United States. Clinical signs are associated with space-occupying lesions and subsequent compromise of organ function as cysts grow.

#### Pancreas

**Nematode.** *Strongylus equinus* (*Strongylidae*) larvae migrate sometimes into the pancreas before patency and may cause granulomas and fibrosis. McCraw and Slocombe (1985) reported depression and diarrhea in association with pancreatic invasion by larvae in experimentally infected horses.

#### Peritoneum and peritoneal cavity

**Nematodes.** *Setaria equina* (*Filaridea*) (see Figs. 4.174 to 4.176) adults (females, 70–130 mm by 0.75–1.20 mm; males, 50–80 mm by 0.4–0.6 mm) live in the peritoneal cavity. Infectious are non-pathogenic.

*Strongylus edentatus* (44 mm; *Strongylidae*) (see Fig. 4.94) larvae migrate through these regions.

#### Respiratory System

##### Paranasal sinuses

**Insect larva.** *Rhinocentrus pusporius* (*Oestridae*) is an exotic nasal bot causing irritation of the nasal cavity. Larvae can damage olfactory nerves and may invade deeper tissue leading to neurologic signs.

##### Bronchi and bronchioles

**Nematode.** *Diatypanulus arfieldi* (*Trichostonyxlophidae*) (females: 63–68 mm by 400–500  $\mu$ m; males: 25–43 mm by 133–259  $\mu$ m) (see Fig. 4.89) is found in horses; donkeys are thought to help maintain the infection among equines. Rapid breathing, coughing, and ill-thrift may be present.

##### Long paracystoma

**Nematode.** *Strongylus edentatus* (aberrant migration) migratory tracts may be seen.

*Parascaris equorum* (*Ascariidae*) larvae routinely make a liver-to-lung migration in the horse before returning to the intestinal tract. There is reason to believe that many of the larvae that do not develop to the adult stage in horses still make it to the lungs and cause eosinophil-associated pathology and possibly incite respiratory signs.

#### Vascular System

##### Arteries

**Nematodes.** *Strongylus vulgaris* (Figs. 7.546 and 7.549; see also Figs. 8.49 and 8.55) larvae migrating through the walls of the mesenteric arteries produce remarkably severe lesions in the walls of these vessels. Colic, gangrenous enteritis, and death can occur.

Text continued on page 462

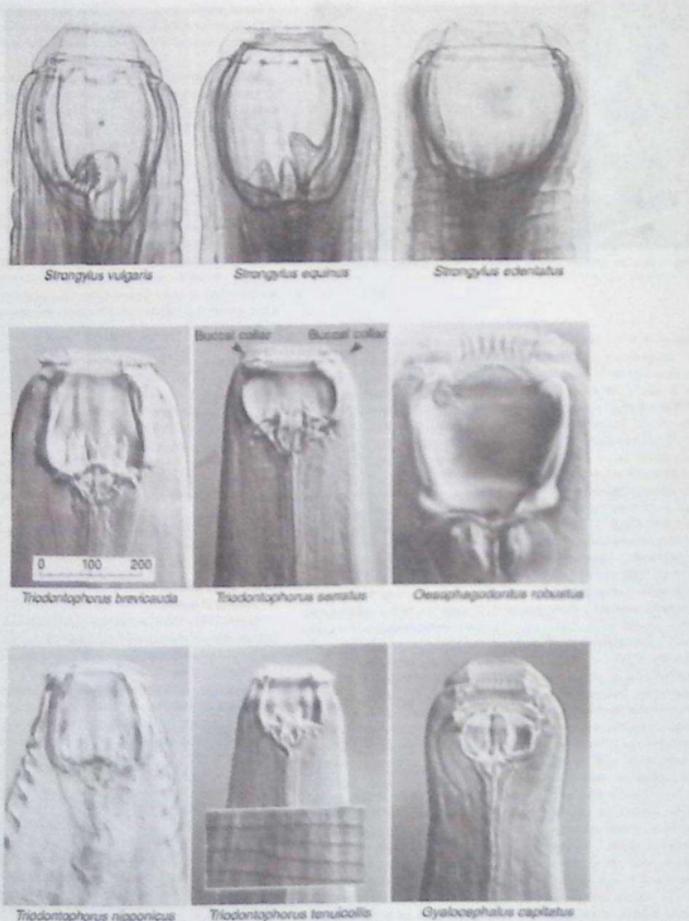


Fig. 7.136 Members of the subfamily Strongylinae (large strongyles) and Gyalocephalus capitatus (subfamily Cyathostominae). *Strongylus vulgaris* and *Oesophagodontus robustus* (x72); *Strongylus equinus* (x40); *Strongylus edentatus* (x33); *Triodontophorus* sp. and *Gyalocephalus capitatus* (x112). (*Strongylus* sp. cleared and mounted by the glycol methacrylate method of Pjanavski et al. A glycol methacrylate embedding technic for nematode whole mounts. *Cornell Vet* 62(2):333, 1973.)

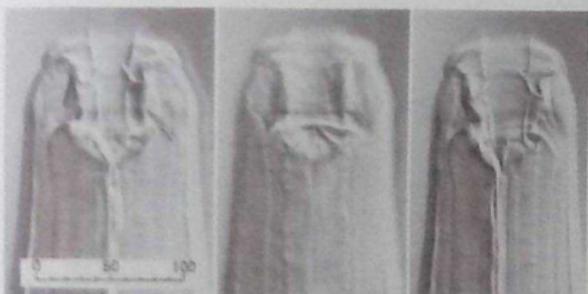
*Coronocylus coronatum**Cyathostomum catinatum**Cyathostomum tetraacanthum*

Fig. 7.137 Members of the subfamily Cyathostominae. Dorsoventral (left), dorsal surface (center), and lateral (right) views of the heads of *Coronocylus coronatus* (top row), *Coronocylus catinatum* (middle row), and *Cyathostomum tetraacanthum* (bottom row). All  $\times 282$ .

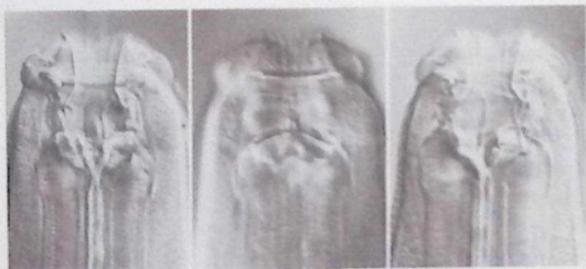
*Coronocylus labialis**Coronocylus labialis**Cyllostephanus goldi*

Fig. 7.128 Members of the subfamily Cyathostominae. Dorsoventral (left), dorsal surface (internal), and lateral (right) of the heads of *Coronocylus labialis* (top row), *Coronocylus labialis* (middle row), and *Cyllostephanus goldi* (bottom row). (A1 1283.)

*Cyclostephanus asymmetricus**Cyclostephanus bidentatus**Craterostomum acuticaudatum*

Fig. 7.138 Members of the subfamily Cyathostominae and *Craterostomum acuticaudatum* (subfamily Strongylinae). Dorsoventral (left), dorsal surface (top center), and lateral (right) views of the heads of *Cyclostephanus asymmetricus* (top row), *Cyclostephanus bidentatus* (middle row), and *Craterostomum acuticaudatum* (bottom row). (All  $\times 253$ .)

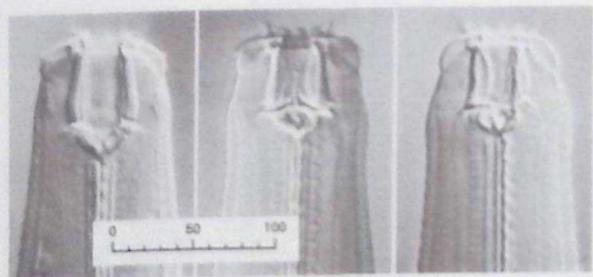
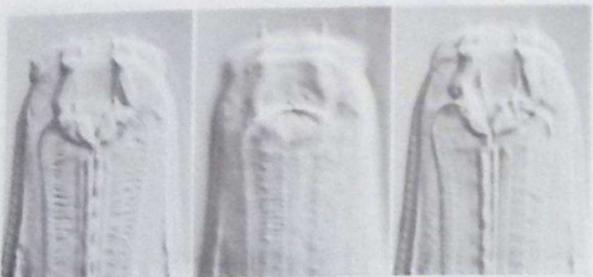
*Cyclostephanus calicatus**Cyclostephanus minutus**Cyclostephanus longibursatus*

Fig. 7.148 Members of the subfamily Cyathostominae. Dorsoverral (left), dorsal surface (vented), and lateral (right) views of the heads of *Cyclostephanus calicatus* (top row), *Cyclostephanus minutus* (middle row), and *C. longibursatus* (bottom row). (All  $\times 425$ .)

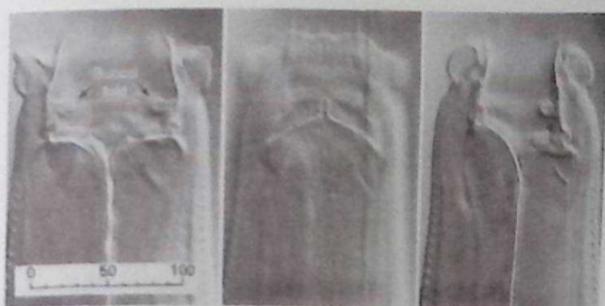
*Cylicocyclus nassatus**Cylicocyclus ashworthi**Cylicocyclus leptostomum*

Fig. 7.141 Members of the subfamily Cyathostominae. Dorsoventral (left), dorsal surface (center), and lateral (right) views of the heads of *Cylicocyclus nassatus* (top row), *Cylicocyclus ashworthi* (middle row), and *Cylicocyclus leptostomum* (bottom row). IC, *nassatus* and *C. leptostomum* (253); C, *ashworthi* (242).

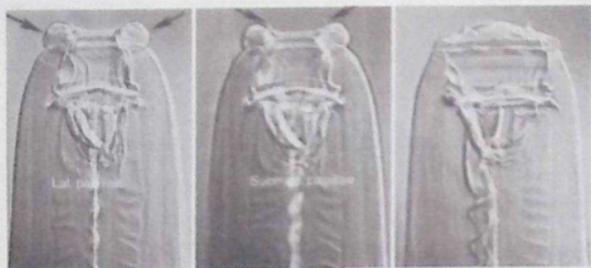
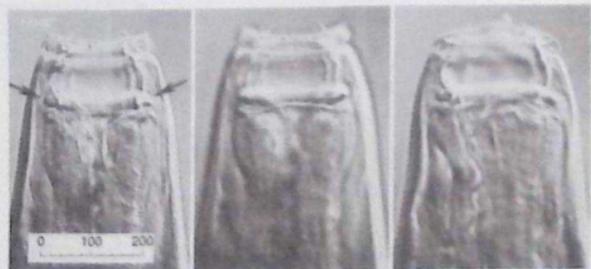
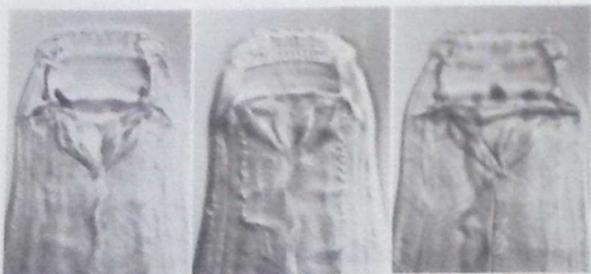
*Cyclocyclops elongatus**Cyclocyclops insignis**Cyclocyclops ultrajectus*

Fig. 7.142 Members of the subfamily Cysthoasominae. Dorsoventral (left, dorsal surface (center), and lateral (right) views of the heads of *Cyclocyclops elongatus* (top row), *Cyclocyclops insignis* (middle row), and *Cyclocyclops ultrajectus* (bottom row). (All x112.)

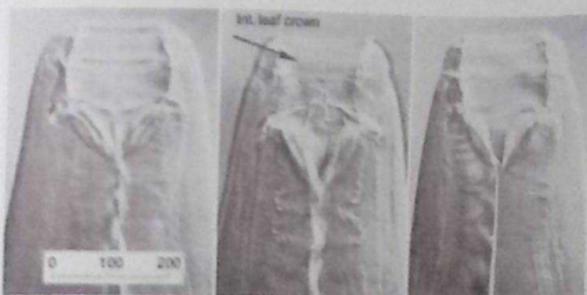
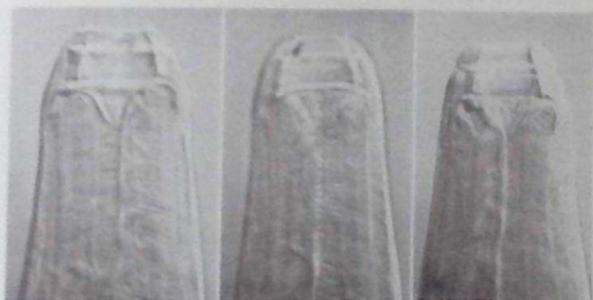
*Poteriosomum impudentum**Poteriosomum ratzi**Parapoteriosomum meliani*

Fig. 7.143 Members of the subfamily Cyathostominae. Dorsoventral (left), dorsal surface (center), and lateral (right) views of the heads of *Poteriosomum impudentum* (top row), *Poteriosomum ratzi* (middle row), and *Parapoteriosomum meliani* (bottom row). (A8 x112.)

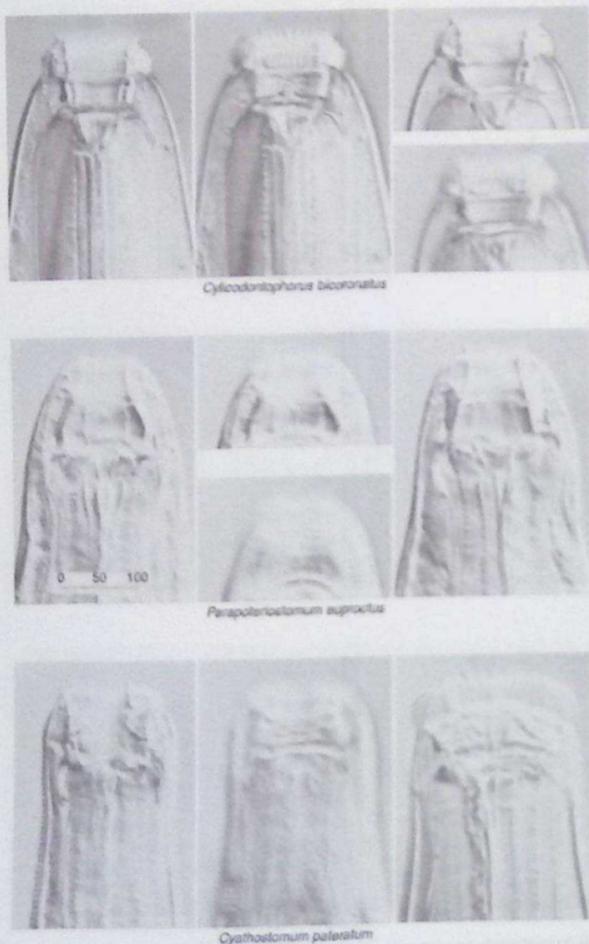


Fig. 7.144 Members of the subfamily Cyathostominae. Dorsoventral (left), dorsal surface (center), and lateral (right) views of the heads of *Cyclocodontophorus bicoronatus* (top row), *Parapoterostomum euproctus* (middle row), and *Cyathostomum patens* (bottom row). (48  $\times$ 170.)

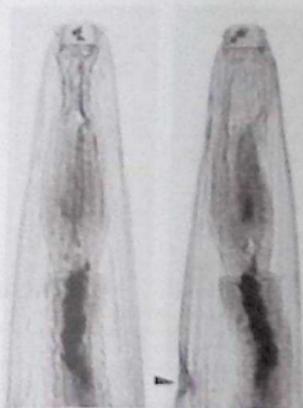


Fig. 7.145 *Cyclocoylus auricularis* (subfamily Cyathostominae) (65). Note prominent lateral head papillae. Arrow indicates position of excretory pore.



Fig. 7.146 Members of the subfamily Cyathostominae.



Fig. 7.147 *Cyclocoylus brevicaephalus*, the only harmful member of the subfamily Cyathostominae (x160).

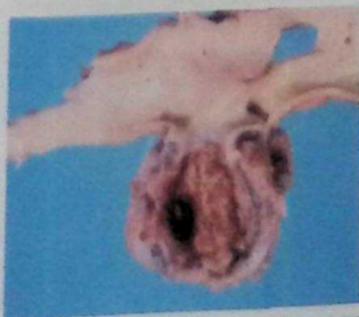


Fig. 7.148 *Strongylus vulgaris* vermiform enteritis and aneurysm in a pony aorta discovered during junior surgery.

*Elaeophora blumi* (Filarioidea) (Fig. 7.88) is found in intimal nodules of the wall of the aorta and other vessels. It is exotic and usually clinically inapparent.

#### Blood

Nematode microfilaria, *Setaria equina* (Filarioidea). Sheathed microfilaria (Fig. 7.88).

Protistan. *Babesia caballi* (piroplasm) (see Fig. 3.52) and *Theileria equi* could be seen in fixed red blood cells. Fever, anemia, icterus, and death have been reported associated with infection.

#### Skeletal Muscles and Connective Tissues

Nematodes. *Trichinella spiralis* (Trichinelloidea) first-stage larvae has been found in Europe in horses fattened for human consumption (Fig. 7.6). Infection is asymptomatic, but economic losses would occur due to meat condemnation.

Oncocercus cerviculus (Filarioidea) adults (females: up to 50 cm by 400  $\mu$ m; males: 6–7 cm long) are found in nuchal ligaments (see Fig. 4.7). *Oncocercus reticulatus* (Filarioidea) adults (females up to 70 cm long; males: up to 27 cm long) are found in flexor tendons and suspensory ligaments of the fetlock. Infections are typically asymptomatic, although heavy infection with *O. reticulatus* has been associated with lameness.

Protista. *Sarcocystis bertrami* and *S. fayeri* (coccidians) (see Table 3.1 and Figs. 8.25, 3.39 and 3.43) occur as sarcocysts within muscle fibers. Infection is inapparent.

Insect larvae. *Hypoderma bevis* and *H. lineatum* (Diptera: Hypodermatidae) (see Fig. 2.26) will on occasion migrate erratically into the subcutaneous dorsal tissues of horses. Neurologic signs may be seen if larvae invade the central nervous system.

Nematode microfilariae. *Oncocercus cerviculus* and *O. reticulatus* (Filarioidea) (see Fig. 8.75) microfilariae are found in the dermis. Some report microfilarial dermatitis can occur in some individuals.

#### Urogenital System

##### Kidneys

Nematode. *Halicephalebus gingivalis* (Rhabditiida) (see Fig. 8.52) can be found in various viscera of the horse as adult females and larvae, with one site of infection often being the kidney. Granulomas and nephritis have been reported associated with infections at this site.

Protistan. *Klebsiella equi* (coccidian) (see Fig. 3.48 and Fig. 8.23). Infection is typically inapparent.

##### Testes

Nematode. *Strongylus edentatus* (Strongylinae) sometimes immature adults are present in vaginal tunics.

#### Nervous System

##### Brain and spinal cord

Nematodes. *Strongylus vulgaris* (Strongylinae) (Fig. 7.44; see also Figs. 8.49, 8.55) may have fourth-stage larvae or young adults migrating erratically; even one worm can cause fatal neurologic disease.

*Setaria* spp. (Filarioidea) (see Figs. 4.175, and 7.88) can undergo erratic migration with neurologic disease; this seems to happen most often in Asia.

*Halicephalebus gingivalis* (Rhabditiida) causes neurologic disease that can be fatal.

*Draconchus megastoma* (Spirurida) (Dapton et al., 1980) adult aberrant migration leading to neurologic disease.

*Parataphrostrongylus tenuis* has also been reported to cause neurologic disease in horses (see Figs. 8.62 and 8.63).

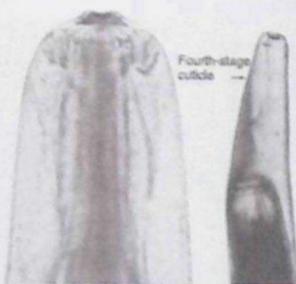


Fig. 2.148 *Strongylus vulgaris* fourth stage (left,  $\times 900$ ) and immature fifth stage (right,  $\times 300$ ) from a mural thrombus of the cranial mesenteric artery of a horse.

Insects. *Hypoderma bevis* and *H. lineatum* (Diptera: Hypodermatidae) may have larvae undergoing erratic migration in the atypical equine host; one larva can cause fatal neurologic disease.

Protista. Equine protozoan myelitis organisms (Apicomplexa) *Sarcocystis neurona* (see Figs. 3.43, 3.44, and 8.25). Causes severe neurologic disease in horses.

##### Eye

Nematodes. *Thelazia lacrymalis* (Spirurida) (females: 14–18 mm long; males: 8–12 mm long) (see Fig. 4.164) is found in the conjunctival sac and lacrimal ducts. Infections are typically asymptomatic.

*Setaria* spp. (Filarioidea) (see Figs. 4.174 and 4.176, and 7.88) can undergo erratic migration and lead to blindness.

Nematode larva and microfilariae. *Draconchus megastoma* and *Habronema* spp. (Spirurida) larvae may cause hemorrhagic conjunctivitis.

*Oncocercus* spp. microfilariae may sometimes be recovered from the eye (Fig. 7.88).

#### Skin and Hair

Insects. *Musca autumnalis* and *S. calcitrans* (Diptera: Muscidae) (see Figs. 2.14 and 2.15). These flies can be a nuisance and *S. calcitrans* feeds on blood and may serve as a vector.

*Hypobosca equina* and *Lipoptena cervi* (Diptera: Hippoboscidae) (see Fig. 2.18) are the keds of horses and may be associated with pruritus. *Hypobosca equina* tends to be rare in the United States; *Lipoptena cervi* does not seem to be common but fortunately only rarely gets on horses.

Gastrophilidae. *G. nasalis*, and *G. haemorrhoidalis* (Diptera: Gastrophilidae) females will hover around horses while they lay their eggs glued to hair.

*Tabanus* and *Chrysops* spp. (Diptera: Tabanidae) (see Figs. 2.10 and 2.11) will attack in bright sun long enough to inflict a painful bite.

*Harmatopinus asini* (Anoplura) (Fig. 3.37) sucking lice may cause pruritus.

*Bevisia equi* (Mallophaga: Ischnocera) (Fig. 7.77) chewing lice may cause pruritus.

*Echinophaga palliata* (Siphonaptera) (see Figs. 2.37 and 7.30). This tick-tight flea of chickens may cause dermatitis in horses.

*Triatoma sanguisuga* (Hemiptera: Triatominae) (see Fig. 2.69) kissing bugs may suck blood from horses.

Insect larvae. *Hypoderma bevis* and *H. lineatum* (Diptera) (see Fig. 2.26) larvae are found in the subcutis of the saddle area and may produce discomfort.

**Arachnida**

**Metastigmata:** Ixodidae, *Amblyomma*, *Annoyator*, *Rhipicephalus*, *Dermacentor*, *Haemaphysalis*, *Hyalomma*, and *Ixodes*, (see Figs. 2.76, 2.83, 2.85, 2.91, 2.93, 2.94 and 3.19) ticks feed on blood and can vector diseases.

**Metastigmata:** Argasidae, *Otobius megnini* (spinose ear tick) (Fig. 3.150; see also Fig. 2.77), with larvae and nymphs in the ears. Infestations of the ears can be quite painful and may elicit varying degrees of inflammation and discomfort.

**Astigmata:** Sarcoptes scabiei (Sarcoptidae) (see Figs. 2.106, 2.108, 2.109, and 7.81). Papules and pruritus in early infestation progress to thickened, alopecic skin that can crack and weep. Secondary infections can occur and with progressive disease emaciation, cachexia, and death are reported.

*Psoroptes ovis* and *C. bovis* (Psoroptidae) (see Figs. 2.106, 2.115-2.118, and 7.84). *Psoroptes ovis* can lead to crusting and scabbing in areas where mites are feeding. *Chorioptes bovis* is found on the fetlocks and can be associated with pruritus and scabbing, especially in horses with "feathers." Kicking, stomping, and biting at the area are common signs of infestation.

**Prostigmata:** Trombiculidae (see Figs. 2.128 to 2.131). Larvae of these mites can lead to extreme pruritus.

*Demodex equi* (see Figs. 2.124, 2.125, and 7.83) has been reported to cause pruritus, poor hair coat, and alopecia.

**Nematode microfilariae and larvae:** *Parafilaria multipapulosa* (Filarioidea) (Fig. 7.85) has microfilariae in serosanguineous discharge from ulcerated nodules.

*Onchocerca cervicalis* and *O. reticulata* (Filarioidea) (see Fig. 8.75) have microfilariae almost universally present in the dermis of horses, especially the dermis of the ventrion, if they have not been on routine ivermectin therapy.

*Rhabditis strongyloides* (Rhabditida) (see Figs. 4.128 and 4.129) can cause dermatitis in horses if they are drossed, as for example on straw for a day or two after surgery.

*Dirofilaria immitis*, *H. muscae*, and *H. microstoma* (Spirurida) have larvae that elicit exuberant granulomatous reactions in skin wounds, areas of skin subject to frequent wetting, and ocular conjunctiva.



Fig. 7.150 *Otobius megnini* nympha collected from the ear canal of a 4-month-old foal imported from Tennessee.



Fig. 7.151 *Besnoitia* sp. Note the white papillike skin cysts caused by this parasite on the muzzle of a donkey. (Photo courtesy Dr. Salyere L. DeBatta.)

(Fig. 7.85). Lesions are nonhealing, scabbed (often known as "summer sores"), and contain yellow granular tissue.

**Protozoa:** *Besnoitia* spp. (Coccidia) may affect horses and donkeys. Clinical manifestations are characterized by small nodules on the face, including in the muzzle and ears, and may be distributed throughout the body (Fig. 7.151; see also Figs. 5.46, 3.47, and 8.30). "Scleral pearls" may also be apparent in the eyes of affected animals.

**Annotated Host-Organ List of Parasites of Swine**

*Trichostrongylus axei* may occur in any tissue of any host as extracellular or intracellular trichostrongyles or as headstomps in cysts (see Fig. 8.28).

**Alimentary System****Mouth and esophagus**

**Nematodes:** *Gongylonema pulchrum* (Spirurida) (females: 80-145 mm by 300-500  $\mu$ m; males: 30-62 mm by 150-300  $\mu$ m) (see Figs. 4.162, 4.163, 7.77, 7.78, 7.79, 7.80, and 7.82) worms in unimolal tracks are not associated with clinical manifestations.

*Trichostrongylus axei* (Trichostrongylidae) is found in the epithelia of the tongue of wild pigs. No clinical signs are reported.

**Stomach**

**Nematodes:** *Physophorus esulatus* (females: 10-22.5 mm by 330-450  $\mu$ m; males: 6-13 mm by 300  $\mu$ m) (see Fig. 4.164). *Acarops strongylina* (females: 15-22 mm by 380  $\mu$ m; males: 10-15 mm by 300  $\mu$ m). *Gnathostoma hispidum* (females: 21-45 mm by 1.8-2.5 mm; males: 15-25 mm by 1.2-2 mm) (see Figs. 4.158 and 4.159), and *Strongylus parvulus* (females: 15 mm long; males: 12-15 mm long) (Spirurida). Infections are often subclinical. Heavy infections may present with gastritis and anorexia.

*Hyostrogylus rubidus* (females: 5-9 mm; males: 4-7 mm) and *Ollulanis tricuspis* (females: 0.8-1.0 mm by 40  $\mu$ m; males: 0.7-0.8 mm by 35  $\mu$ m) (Trichostrongylidae) (see Figs. 4.88 and 4.84). Both parasites have been associated with severe gastritis and ulceration in heavy infectious diarrhea and loss of condition are expected in such cases.

*Anatolostoma (Capillaria) gemmosa* (Trichostrongylidae). Infection is expected to be subclinical.

**Small intestine**

**Nematodes:** *Ascaris suum* (Ascaridoidea) (females: 20-40 cm by 5-6 mm; males: 15-25 cm by 3-4 mm) (Fig. 7.132; see also Figs. 4.137



Fig. 7.182 Lesions induced in the liver of a pig exposed to the infectious eggs of *Ascaris suum* (right); normal liver on left.



Fig. 7.183 Rectum of pig with attached *Trichuris suis* (Spaceman courtesy Dr. Mary C. Smith.)

to 4.139, 7.93, and 8.66). Unthriftness, failure to gain weight, rough hair coat, and distended abdomen may be seen in growing pigs. Young animals with large numbers of worms may have impaction, perforation, or rupture of the intestine.

*Globocephalus strutholatus* (Ancylostomatidae) (females: 3.6 mm by 330-385  $\mu$ m; males: 4.5-5.5 mm by 300-313  $\mu$ m) (see Fig. 4.108). These hookworms feed on blood, so heavy infections could be expected to cause anemia.

*Strongyloides ransomi* (parthenogenetic females: 3.3-4.5 mm by 54-62  $\mu$ m; Rhabditida) (see Figs. 4.131 and 4.132). Heavy infections in neonates can cause anemia, diarrhea, dehydration, emaciation, and death. Infections can be transmitted in milk.

*Trichinella spiralis* (Trichinelloidea) (females: 3-4 mm by 60  $\mu$ m; males: 1.4-1.6 mm by 40  $\mu$ m) (see Figs. 4.179-4.181, and 7.91). Adults in the intestine show little pathogenicity.

**Acanthocephala.** *Macracanthocephalus hirudinaceus* (470 mm) (see Fig. 4.189) are present with their robust proboscis embedded deep in the bowel wall where they elicit inflammation and granulomas. Light infections may be subclinical, but in larger numbers failure to thrive and emaciation can be seen. Perforation of the bowel wall may result in peritonitis.

**Prentista.** *Eimeria debilis* and about 10 other species of *Eimeria* (coccidians), usually infection is without clinical signs.

*Cystoisospora suis* (coccidians) causes enteritis and diarrhea in young animals as little as 5 days old (see Fig. 8.21).

*Cryptosporidium parvum* (Apicomplexa) is of little clinical relevance.

*Giardia* spp. (tricostophyllates) (see Figs. 3.11, 7.87, and 7.90); infection is usually without signs.

#### Cecum and cecum

**Nematodes.** *Oesophagostomum dentatum*, *O. breviscolum*, *O. georgianum*, and *O. quadrispinulosum* (Strongyloidea) (females: 10-14 mm by 310-500  $\mu$ m; males: 8-10 mm by 200-300  $\mu$ m) (see Figs. 4.97 and 4.98). Immature stages of these worms cause nodule formation in the bowel wall (see Figs. 4.101, 4.102, 8.56 and 8.57), enteritis, anorexia, and hematochezia; heavy infections are fatal. Additional economic losses also occur due to cecum condemnation.

*Trichuris suis* (Trichinelloidea) (females: 35-50 mm; males: 30-40 mm) (Fig. 7.83); see also Figs. 4.183 and 7.83). Anorexia, ill-thrift, mucoid or mucobloody diarrhea, dehydration, and death are possible in heavy infections.

**Prentista.** *Zenarhcha polidici*, *Z. nana*, *Isidomoda burtschli*, and others (astrochei) are considered for the most part to be commensals. *Chilomonas monili*, *Etmacanthomonas butryi*, *Trichomonas rufus*, and *Trichomonas suis* (trichomonads) are considered for the most part to be commensals.

*Neofilaria celi* (celites) (see Figs. 3.13, 7.92, and 8.13) is a commensal organism that can on occasion cause colitis.

#### Liver, pancreas, and peritoneal cavity

**Nematode larvae.** *Ascaris suum* (Ascarioidea) (see Figs. 4.138 and 7.83) has migrating larvae that cause "villous spine" lesions on the liver surface. May lead to organ condemnation.

*Stephanurus dentatus* (Strongyloidea) migrating larvae in liver and pancreas (see Fig. 4.103). Lesions may lead to organ condemnation.

**Tricostophyllates.** *Fasciola hepatica* (30 mm by 13 mm) and *F. gigantica* (25-75 mm by 12 mm) (Fasciolidae) (see Figs. 4.2 and 4.11) produce migratory tracks and hepatic fibrosis, which can result in liver condemnation.

**Cestode larvae.** *Echinococcus* spp. (Taeniidae) (see Figs. 4.50 to 4.52, 4.65, and 8.66) hydatids are very rare in the United States. Clinical signs would be expected if the cyst grows large enough to compromise the function of the organ.

*Taenia hydatigena* (Taeniidae) (see Fig. 4.43) cysticerci can be found on rare occasions, mainly in wild pigs. Migration of larvae through the liver can cause tracks and fibrosis, which can result in liver condemnation.

#### Respiratory System

##### Branchi and bronchioles

**Nematodes.** *Metastrongylus apri*, *M. salmi*, and *M. pudendiculus* (Metastrongyloidea) (females: 28-60 mm by 320-450  $\mu$ m; males: 11-26 mm by 120-324  $\mu$ m) (see Figs. 4.116 and 7.82) can cause signs of respiratory distress in pigs.

##### Lang parenchyma

**Nematode larva.** *Ascaris suum* (Ascarioidea) organisms migrate through the lungs after passage through the liver and cause disease (paroxysmal coughing, "chumps") in reaction to their passage (see Fig. 8.66).

**Cestode larva.** *Echinococcus* spp. (Taeniidae) (see Figs. 4.50 to 4.52, 8.45 and 8.46) hydatids in pigs in the United States seem to be very rare. Clinical signs expected when cysts compromise with organ function.

**Trematode.** *Fargasinus lillivieri* (Troglorematidae) (7.5-16 mm by 4-8 mm) (see Figs. 4.14 to 4.16, and 7.83) would be an excellent

parasite of wild pigs and is liable to do very well in pigs fed crayfish. Respiratory signs could result.

#### Skeletal Muscles and Connective Tissues

**Nematode larva.** *Trichinella spiralis* and *T. murrelli* (Trichinellidae) (Fig. 7-64; see also Figs. 8-77 and 4-182) larvae can be present in very large numbers per gram of pig muscle without the pig showing signs of disease. Economic loss would occur due to condemnation of meat.

**Cystode larva.** *Taenia solium* (Taeniidae) (see Fig. 4-48) cysticerci (elliptical bladder, 5–20 mm long by 5–10 mm wide) in muscle are a potential problem in areas where humans who might be infected with adults, especially those from certain developing countries, are working around pigs as animal handlers; cysts cause carcass condemnations but are not associated with clinical signs in pigs.

**Spinozoite massonoides** (Dipyllobothridae) (see Figs. 4-36 and 8-48) spargana can occur in pigs, which serve as paratenic hosts. Clinical signs are not reported.

**Nematode larva.** *Aleria* spp. (Mecynocercariae, Diplostomatidae). Infections are typically without signs (see Fig. 4-25).

**Protozoa.** *Sarcocystis mansuetaria*, *S. porcilis*, and *S. mithamni* (coccidians) (see Table 3-1 and Figs. 3-39, 3-41 and 8-26) sarcocysts occur in the muscles of pigs. Infections are subclinical.

#### Urogenital System

**Nematode.** *Stephanurus dentatus* (Strongylidae) (see Fig. 4-103). Stout, white worms (females: 30–65 mm by 1.5–2.2 mm; males: 20–30 mm by 1.1–1.9 mm) occur in the kidneys, ureters, urinary bladder, perirenal fat, pork chops, spinal canal, and elsewhere as a result of erratic migrations.

#### Skin and Hair

**Insects.** *Musca* and *Stomoxys* (Diptera) (see Figs. 2-14 and 2-15). These flies affect pigs in much the same way they affect other hosts.

**Haematopinus suis** (Anoplura) (Fig. 7-154; see also Figs. 2-53 (left) and 7-55) is the sucking louse of pigs. It can be associated with pruritus, alopecia, anemia, restlessness, and failure to thrive. Additionally, it may transmit swine pox.

**Pulex irritans**, *E. gullinsoni*, and *T. penetrans* (Siphonaptera) (see Figs. 2-58, 2-57, 2-58, 2-45, 2-46, and 7-56) may all cause infestations on pigs with associated pruritus and blood loss.

**Acarids.** *Metastigmata* (ticks) (see Figs. 2-78, 2-85, 2-94, and 2-96). Ticks of many types may infest pigs, cause injury, disturb and deplete their blood.

*Sarcoptes scabiei* (Arachnida) (see Figs. 2-106, 2-108, 2-109, and 2-111) continues to be a problem in pigs. Infestations can be extremely

pruritic, cause severe thickening and crusting of the skin, alopecia, and pyoderma. Infested animals fail to thrive, can become cachectic, and die.

*Demodex phylloides* (Prostigmata) (see Figs. 2-124, 2-125 and 7-57) causes dermatitis in the form of pimples on pigs. Nodules are full of huge numbers of mites; those may coalesce and rupture.

#### Annotated Host-Organ Listing of Common Parasites of Rabbits

*Toxoplasma gondii* may occur in any tissue of any host as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Figs. 3-35, 3-36, and 8-28). *Neospora caninum* may occur in similar locations (see Figs. 3-37 and 8-29).

#### Alimentary System

##### Stomach

**Nematodes.** *Oblolucoides cucullis* (females: 13–18.5 mm long; males: 10–14 mm long) and *Graphidium strigosum* (females: 11–20 mm long; males: 8–16 mm long) (Trichostrongylidae) (Fig. 7-153). Spicules of *O. cucullis* are 0.40 to 0.54 mm; of *G. strigosum*, 1.1 to 2.4 mm.

##### Small Intestine

**Nematodes.** *Trichostrongylus retortaeformis* (in Europe, females: 6–9 mm long; males: 5–7 mm long), *T. affinis* (in continental rabbits, United States, size range similar to *T. retortaeformis*), *T. calcaratus* (in United States, females: 5.8–7 mm long; males: 4.7–6.6 mm long), and *Nematodirus leporis* (females: 16–20 mm long; males: 9–13 mm long) (Trichostrongylidae) (see Figs. 4-78 and 4-80). Spicules of *T. retortaeformis* and *T. affinis* are 100 to 155  $\mu$ m; of *T. calcaratus* is 170 to 190  $\mu$ m; whereas *N. leporis* are of 650 to 1000  $\mu$ m.

*Strongylides papillaris* (parthenogenic females: 3.5–6 mm; Rhabditida).

**Cystodea.** *Cinetaria cinerea* (Anoplocephalidae) (Fig. 2-126), and see Fig. 7-93.

**Protozoa.** *Eimeria* spp. (coccidia) (Fig. 7-59) parasite the intestinal epithelium and cause diarrhea and emaciation. *Eimeria cucullata*, *E. rajpuri*, *E. flavescens*, *E. intestinalis*, *E. irradialis*, *E. magna*, *E. media*, *E. perforans*, and *E. vesicularis*.

*Cryptosporidium cucullata* is a potential zoonotic agent.

*Gardia* (microspogonites) is a potential zoonotic agent (Fig. 7-60).



Fig. 7-154 *Haematopinus suis* from a pig.



Fig. 7-155 *Oblolucoides cucullis*: sternal end (left) and bursa and spicules of male (right) ( $\times 120$ ).



Fig. 7.158 *Cittotaenia* from a rabbit. (Specimen courtesy Dr. Lenora Schultz-Powell.)

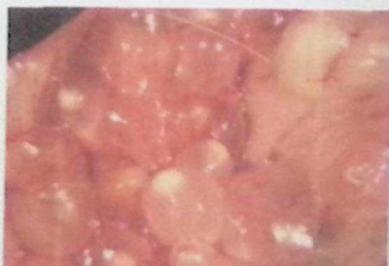


Fig. 7.159 Cysticercus of *Taenia proformis* in the abdominal cavity of an experimentally infected domestic rabbit.

#### Cecum and colon

Nematodes. *Passalurus ambiguus* (females: 8–12 mm by 515–590 µm; males: 3–5 mm by 250–275 µm), *Dermatostax redigera* (females: 16–17 mm by 600 µm; males: 8–11.5 mm by 435 µm) (Oxyurida) (see Fig. 4.133).

*Trichouris leporis* (Trichinelloidea) (females: 17.4–20.9 mm long; males: 19–21 mm long).

Protista. *Eimeria flavescens*, *E. poriformis* (coccidian).

*Entamoeba caniculi* (amoeba). Nonpathogenic.

#### Uterus and peritoneal cavity

Protista. *Eimeria stiedae* (Coccidia) causes biliary coccidiosis (see Fig. 8.20).

Larval nematodes. *Toxocara canis* migrates through the livers of rabbits on the way to the lungs and musculature (Fig. 7.157).



Fig. 7.157 *Toxocara* larva from a rabbit's liver (x250).

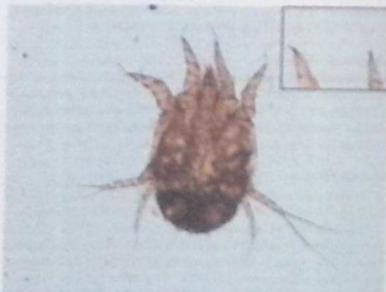


Fig. 7.159 *Psoroptes curvicuti* from a rabbit; inset shows the segmented pretarsi.



Fig. 7.160 Ear of a rabbit infested with *Psoroptes curvicuti*.



Fig. 7.161 *Leporacarus gibbus*, a hair-clasping mite of rabbits (x100). (Courtesy Dr. Stephen Westwood.)

**Cestode larvae.** *Taenia pisiformis* (Taeniidae) (Fig. 7.160) cysticerci initially migrate through the liver but ultimately settle down to mature in the peritoneal cavity.

#### Skin and Hair

**Arachnida.** *Pteroporus cuniculi* (Astigmata) (Figs 7.153 and 7.160; see also Figs. 2.106, 2.115, 2.116, and 7.81) can cause severe ear canker in rabbits.

*Saroptes* and *Oxyuris* (Astigmata) (see Figs. 2.106, 2.108, 2.109, and 7.81), *Leporacarus gibbus* (Trichostromatidae) (Fig. 7.161), *Cheyletiella parasitovorax* (Protostigmata) (see Fig. 2.126).

#### Annotated Host-Organ Listing of Common Parasites of Rats

*Toxoplasma gondii* may occur in any tissue of any host as intracellular or intracellular tachyzoites or as bradyzoites in cysts (see Figs. 3.35, 3.36, and 8.26). *Neospora caninum* may occur in similar locations (see Figs. 3.37 and 8.29).

#### Alimentary System

##### Esophagus, stomach, and intestines

**Nematodes.** *Esocolor bacillatus* (Trichostrongyloidea) (females: 52–69 mm by 150–212  $\mu$ m; males: 15–29 mm by 94–115  $\mu$ m). Seen embedded in the mucosa of esophagus.

*Nippostrongylus brasiliensis* (Trichostrongyloidea) (females: 2.5–6.2 mm long; males: 2.1–4.5 mm) (Fig. 7.162).

*Strongyloides ratti* (paratubercular) (females: 1.8–3.1 mm by 30–38  $\mu$ m, Rhabditida) (see Figs. 4.131 and 4.132).

*Gongylonema neophisicum* (Spirocarida) (females: 35–80 mm by 170–360  $\mu$ m; males: 7–4–20 mm by 85–200  $\mu$ m) (Figs. 7.159 and 7.177).

*Syphacia muris* (females: 2.8–3.4 mm by 180–250  $\mu$ m; males: 1.2–1.5 mm by 100  $\mu$ m) and *Aspicularis ratti* (Oxyurida) (females: 3.1–3.25 mm by 100–100  $\mu$ m; males for this species were not described by Johnston, S. 1970).

*Heterakis spumosa* (Ascariidida) (females: 6.8–8.3 mm long; males: 3.4–8.1 mm long).

*Trichinella spiralis* (females: 3–4 mm by 60  $\mu$ m; males: 1.4–1.6 mm by 40  $\mu$ m) and *T. muris* (Trichinelloidea) (see Figs. 4.180 to 4.182 and 7.177). Eggs of *H. diminuta* lack the polar filaments (See Fig. 4.62), *Trichuris muris* (Trichinelloidea).

**Cestode.** *Hymenolepis dimorpha* (Hymenolepididae) (26–60 mm long by 3–4 mm wide) (Fig. 7.163). Scolex without hooks.

**Protistans.** *Eimeria stuebeli* and other species (coccidians) (Fig. 7.89).

*Giardia* (mucocollagellate) (Fig. 7.93).



Fig. 7.162 *Alloglyphingylus brasiliensis*. (A) Bursa and spicules of male (x120). (B) Caudal end of female (x150). (C) Esophageal region (x150).

#### Liver

**Nematode.** *Calodium (Capillaria) hepaticum* (Trichostrongyloidea) (females: 52–104 mm by 78–184  $\mu$ m; males: 22 mm by 26–78  $\mu$ m) (Fig. 7.163; see also Fig. 8.78). Male and female worms in tunnels sometimes filled with eggs are seen embedded in the parenchyma.

**Cestode larvae.** *Strobilicercus* of *T. taeniiformis* (Taeniidae) (see Fig. 4.46).

**Hydatid cysts of E. multilocularis** (Fig. 7.160; see also Figs. 4.55, 8.45 and 8.46). Appears as a solid granuloma, mostly. Protoscolices may be appreciated in a squash mount preparation of a cut open granuloma.

**Protozoa.** *Hepatozoon muris* (plasmodium) has schizonts occurring in the hepatic cells; gamonts are found in the monocytes of the circulating blood (see Fig. 3.49). The vector is a mesostigmatid mite, *Echinolaelaps echinatus*.

#### Urogenital System

**Nematodes.** *Trichostrongylus crassicauda* (Trichostrongyloidea) (females: 10–19 mm by 200–221  $\mu$ m; males: 1.3–3.5 mm by 29–37  $\mu$ m) (see Figs. 4.188 and 8.120) lives threaded through the Muller epithelium; the male lives in the reproductive system of the female worm.

#### Skin and Hair

**Insect.** *Polyphax spinulosus* (Anoptera) (Fig. 7.160) large numbers may lead to anemia and debilitation; hypersensitivity reactions result in pruritus and self-trauma.

*Xenopsylla cheopis* (Siphonaptera) (see Fig. 2.158) a vector of plague and typhus.

**Arachnida.** *Ornithonyssus bacoti* (Mesostigmata) may lead to anemia and debilitation. These mites are likely to cause dermatitis in people.

*Basilofolia esuifera* (Protostigmata) hypersensitivity induced pruritus and self-trauma.

*Notonius muris* (Astigmata) (see Figs. 2.110, 2.111, and 2.112) leads to crusty dermatitis, typically on the pinnas, but may affect other areas of the face and body.



Fig. 7.163 *Calodium* (Capitellid) hepatocin. (A) Extensive egg-filled nematode tracks observed in the liver parenchyma of an urban Norway rat, *Rattus norvegicus*. (B) Liver parenchyma on a higher magnification demonstrating egg-filled tracts. (C) Histological section of a rat liver (300 $\times$ ) showing cross-sections of a female worm and eggs with bipolar plugs surrounding the worm.

#### Annotated Host-Organ Listing of Common Parasites of Mice

*Toxoplasma gondii* may occur in any tissue of any host as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Figs. 3.35, 3.36, and 8.28). *Nasipora cetransum* may occur in similar locations (see Figs. 3.37 and 5.29).



Fig. 7.164 Liver of a cotton rat experimentally infected with *Ectoparasitiscus multifasciatus* producing the alveolar hydatid cysts that have replaced most of the liver tissue.

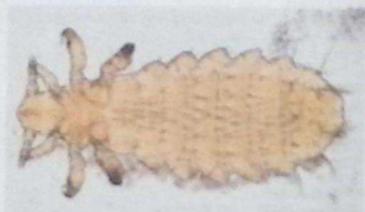


Fig. 7.165 *Polypilex spinulosus* male (x100).

#### Alimentary System

##### Stomach and Intestine

*Protista*. *Cryptosporidium muris* (stomach) and *C. parvum* (small intestine) (see Figs. 3.16 and 3.17).

**Nematodes**. *Heligmosomoides polygyrus* (eye), *Himantospiridae* (duodenum), *Trichostrongylidae* organisms are reddish and tightly coiled (females: 13 mm long; males: 6 mm long).

*Nippostrongylus brasiliensis* (*Trichostrongylidae*) (females: 2.5–6.2 mm long; males: 2.1–4.5 mm) (Fig. 7.162).

*Syphacia obvelata* (females: 3.4–5.8 mm by 240–400  $\mu$ m; males: 1.1–1.5 mm by 68–90  $\mu$ m) and *Aspicularis tetrapeta* (females: 3–4 mm by 215–275  $\mu$ m; males: 2–4 mm by 120–190  $\mu$ m) (*Oxyuroidea*) (Fig. 7.166).

*Heterakis spumens* (*Acaridida*) (females: 6.8–8.3 mm long; males: 3.4–8.1 mm long).

*Trichuris muris* (*Trichinelloidea*).

*Cestodes*. *Rolessiopsis* (*Hymenolepis*) *nana* (7–100 mm long by 500–600  $\mu$ m wide) and *Hymenolepis diminuta* (20–60 mm long by 3–4 mm wide) (*Hymenolepidae*) (see Fig. 5.29 for egg morphology). The sucker of *R. nana* is armed with hooks; that of *H. diminuta* is unarmored.

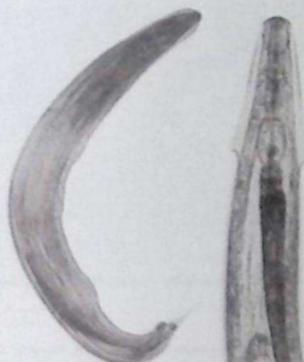


Fig. 7.168 Parasitoids of mice: *Syphacia obvelata* male (left) and *Aspicular tetractera* anterior and (right) ( $\times 60$ ).

**Larval nematodes.** Migrating *Toxascaris canis* can cause disease in small mammals as it migrates (Fig. 7.167).

#### Urogenital System

##### Kidneys

**Protozoa.** *Klebsiella muris* (coccidian) usually seen on histosections (see Fig. 8.23).

#### Skin and Hair

**Insects.** *Polypilus serratus* (Anoplura) (see Figs. 2.57, 2.58, and 7.165) large numbers may lead to anemia and debilitation; hypersensitivity reactions result in pruritus and self-trauma.

**Archebids.** *Myobia musculi* and *Radfordia affinis* (Prostigmata) (see Fig. 2.127). Infestations may be inapparent or result in varying degree of dermatitis, which may include pyoderma. Myobids do not migrate away from a dead host; the carcass must be scanned carefully with a stereoscopic microscope to find them.

*Myocoptes musculorum* (Anigmata) (see Fig. 2.121); generally infestations are asymptomatic.

*Ornithonyssus harti* and *Allodermanyssus sanguis* (Mesostigmata) (see Fig. 2.96, *Oreohelnyssus sylvianus*) infestations may be inapparent or lead to anemia and debilitation. These mites are likely to bite and cause dermatitis in people.

#### Annotated Host-Organ Listing of Common Parasites of Guinea Pigs

*Toxoplasma gondii* may occur in any tissue of any host as extracellular or intracellular tachyzoites or as bradyzoites in cysts (see Fig. 3.33, 3.36, and 8.28). *Neospora caninum* may occur in similar locations (see Figs. 3.37 and 8.29).

#### Alimentary System

**Nematode.** *Paraspidodera wicmatia* (Oxyurida) (females, 16–27.5 mm by 400  $\mu$ m; males: 11–22 mm by 300  $\mu$ m); typically asymptomatic.

**Cestode.** *Rodentolepis* (*Hymenolepis*) *nana* (7–100 mm long by 500–800  $\mu$ m wide) (see Fig. 5.94 for egg morphology); infections are asymptomatic but may be directly transmitted to people.

**Protozoa.** *Eimeria canis* (coccidian).

*Nyctalendrium* spp. (ciliate) (Fig. 7.166; see also Fig. 3.13).

*Cryptosporidium whartii* (see Fig. 3.16, *C. parvum* and Fig. 7.66, *C. felis*).

#### Skin and Hair

**Insects.** *Gliccola porcellis*, *Cyrtopus oralis*, and *Timonemys hioplani* (Mallophaga) (Fig. 7.166; see also Fig. 2.67) may be asymptomatic, or associated with unthriftness, pruritus, alopecia, and a matted hair coat.

**Archebids.** *Chirodiscoides canis* (Anigmata) (see Fig. 2.120) may be asymptomatic or associated with unthriftness, pruritus, overgrooming, and alopecia.

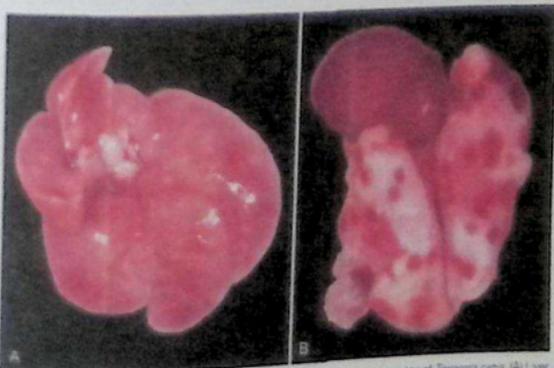


Fig. 7.167 Liver and lungs of mice experimentally infected with the infective eggs of *Toxascaris canis*. (A) Liver removed from a mouse 2 weeks after a challenge infection imposed on a month old infection. (B) Lungs of a mouse 3 days after being infected with 125 eggs of *Toxascaris canis*.



Fig. 7.168 *Neobalantidium coli* cyst in the feces of a guinea pig.



Fig. 7.169 Guinea pig infested with the louse *Glincola porcella*.

*Trichostrongylus axei* (Asiigmata) can cause severe pruritic mange in guinea pigs that can be fatal.

#### Annotated Host-Organ Listing of Common Parasites of Monkeys and Apes

##### Alimentary System

**Nematodes.** *Cephalobus parviticus* (Rhabditiida) (females: 0.9–1.45 mm by 35–48  $\mu$ m; males: 780–1120 by 35–40  $\mu$ m). These hairless parasites of the stomach and intestines of *Macaca irio* mandrill (and probably others) resemble the free-living generation of Strongyloidea. Their rhabditiform larvae may be confused with those of Strongyloidea on fecal examination. They do not, however, develop into filariform larvae, so the oömma may be resolved by culturing the fecal specimen.

*Strongyloides fuelleborni* (parthenogenetic; females: 2.9–4.6 mm by 43–68  $\mu$ m) and *S. stercoralis* (parthenogenetic; females: 1.7–2.7 mm by 30–46  $\mu$ m) (Rhabditiida) (see Figs. 4.131 and 8.53). Simian strongyloidosis may be associated with disseminated infection in debilitated animals and is a human health hazard.

*Nochta nachti* (Trichostrongyloidea). Bright red worms (female: 7.6–10 mm; males: 5.7–6.5 mm) lie within or protrude from gastric papillomata in the prepyloric region of the stomach. Cross-sections of *N. nachti* in histologic preparations display 16 distinct longitudinal cuticular ridges and channelled lateral alae.

*Trichostrongylus, Melinus,* and *Nematostium* (Trichostrongyloidea) (Fig. 7.176; see also Fig. 4.78).

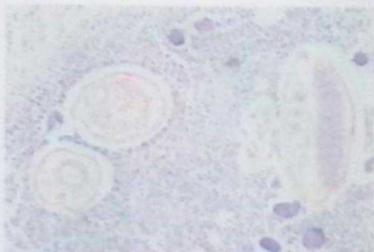


Fig. 7.174 *Melinus barbatus* in the small intestine of a *Cebus* monkey (x200).

*Oesophagostomum* (Gonasteriea) *apicostomum* (females: 8.5–10.5 mm by 200–300  $\mu$ m; males: 8–10 mm by 300–350  $\mu$ m), *O. nephelostomum* (size in the same range as previous), and *Terradus deimostomus* (Strongyloidea) (females: 12–14 mm by 650–750  $\mu$ m; males: 9.5 mm by 560  $\mu$ m) (Fig. 7.175; see also Figs. 4.97, 8.56, and 8.57). Stout-bodied "nodular worms" with leaf crowns and transverse ventral cervical groove.

*Nisus,* *Ancylostoma,* and *Globocephalus* (Ancylostomatidae) (see Figs. 4.108 to 4.110).

*Ancyrocephalus* (Ascariidae) (females: up to 41 cm by 5 mm; males: 15–25 cm by 5 mm) (see Figs. 4.137 and 4.138).

*Trichouris* spp. (Trichostrongyloidea) (Fig. 7.177; see also Fig. 4.183). *Entamoeba* spp. (Ciliata) (Fig. 7.178). Pinworms are quite host-specific. Generally speaking, a species of pinworm infests a genus of monkeys. *Pentostemon vermiformis* (female: 6–13 mm; males: 2–5 mm) and *Entamoeba anthrophagathus* (female: 6.5–5.6 mm by 450–540  $\mu$ m; males: 1.5–1.9 mm by 120–160  $\mu$ m) occur in chimpanzees. *Entamoeba* spp. are usually considered nonpathogenic, but there are very rare reports of invasion of the wall of the intestine resulting in enteritis or even fatal disease.

*Prabonysus* spp. occur in various primates and sometimes are found in fecal specimens (Fig. 7.179).

*Strophophagus, Gongylomonas, Prostopyrus, Physocaphus,* and *Rictularis, Physaloptera* (Spiridae) (Figs. 7.174 and 7.175; see also Figs. 4.160 to 4.164, and 7.171). *Protospirurus maculosa* (female: 33–42 mm by 612–1290  $\mu$ m; males: 19–26 mm by 400–800  $\mu$ m), a parasite of rodents that uses the cockroach *Leopoldus modestus* as intermediate host, has been observed to cause perforation of the stomach in captive monkeys (Cox and Johnson, 1959).

**Cestodes.** *Bertiella zaidii* (25–30 cm by 10–15 mm) (Ancylocephalidae) is large and has four suckers and no hooks (egg shown in Fig. 7.173). *Roulestele* (*Hymenolepis*) *nana* (Hymenolepididae) (7–100 mm long by 500–600  $\mu$ m wide) (Fig. 7.180) is very small, has four suckers, and has hooks.

**Acanthocephalus.** *Prosthenorchis* and *Momodjirini* (see Figs. 8.79 and 8.80, and see egg depicted in Fig. 7.170).

**Flukes.** *Gastrodiscoides hominis* (Paramphistomidae) (5–10 mm by 4–6 mm).

**Protists.** *Neobalantidium coli* (ciliate) (see Fig. 7.168, and 3.13) causes acute enteritis (Cox and Johnson, 1962).

*Entamoeba coli* or *Entamoeba coli*-like amoebae are common in primates (Fig. 7.178), but they can also occasionally be infected with the pathogenic *Entamoeba histolytica*.

*Gardia lamblia* (flagellate) (Fig. 7.194).

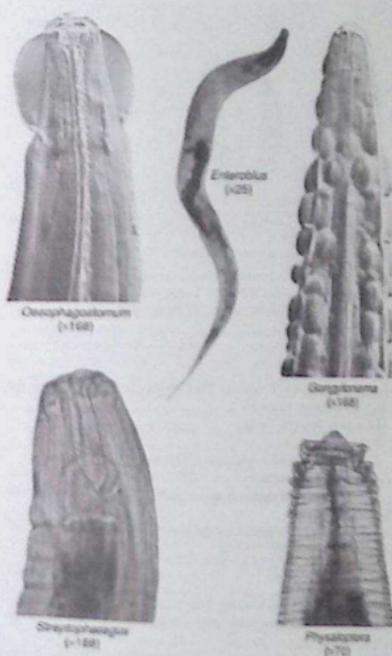


Fig. 7.131 Some nematode parasites of monkeys and apes. (Courtesy Dr. M.M. Rubenstein.)



Fig. 7.172 *Trichostrongylus* sp. from the feces of a patas monkey (*Erythrocebus patas*). Also, there are two unstained amoebic cysts present.

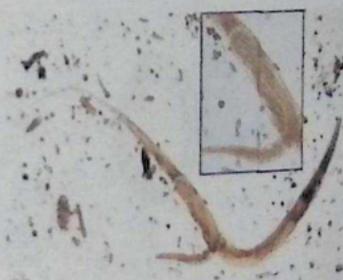


Fig. 7.173 *Probstmayria* sp. from the feces of a gorilla (*Gorilla beringei beringei*) identified in an ethyl acetate formalin sediment examination (Specimen provided by Dr. Jessica M. Rubman, Hunter College, NY)



Fig. 7.174 *Gongylonema* (x22), cross-section through gravid female embedded in the esophagus of stump-tail macaque monkey.



Fig. 7.175 *Gongylonema* (x125) at higher magnification showing the presence of uterine lateral cords and embryonated eggs in uterus, many containing larvae.

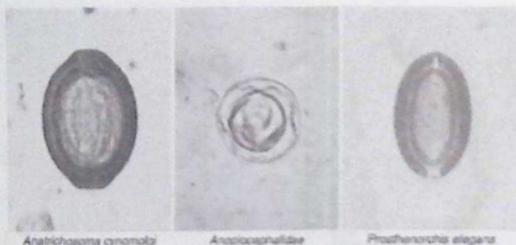


Fig. 7.176 Three parasites of primates. For a more complete listing of simian parasites by host and organ, see text. *Anatrichoeca cynomagi* adult worms burrow in the nasal mucosa. *Anoplocephalidae* eggs have a pear-shaped entricyphore surrounding the oncosphere. *Prosthenocheila elegans* (Acanthocephala) eggs have a thick outer shell and thin inner shells enclosing the embryo (acanthid).



Fig. 7.177 *Entamoeba coli*-like cyst in the feces of a patas monkey (*Erythrocebus patas*): the one cyst has eight visible nuclei.

### Liver and Pancreas

**Protozoans.** *Hepatozoyti kochi schizonts*.

*Entamoeba histolytica* (amoeba) can cause hepatic abscess.

**Nematodes.** *Calodium* (*Capillaria*) *hepaticum* (*Trichinelloidea*) (female: 52–104 mm by 79–184  $\mu$ m; male: 22 mm by 26–78  $\mu$ m) (Fig. 7.163; see also Figs. 6.78) occurs with worms and eggs in hepatic parenchyma.

*Trichostrongylus leptostomus* (*Spiruridae*) is a 10- to 20-mm worm with a long capillary pharynx; associated with varying degrees of fibrosing pancreatitis. Found in the pancreatic duct of American primates.

### Respiratory System

**Nose and throat**

**Nematode.** *Anatrichoeca* (*Trichinelloidea*) (Fig. 7.176).

**Annelids.** The leeches that attack the pharyngeal mucosa of monkeys are large, black annelids with a large cup-shaped caudal sucker. The presence of this bloodsucking parasite is suggested by chronic opisthus in a recently captured monkey. When the host drinks infested water, the young leeches enter the mouth, nose, pharynx, or larynx and

attach to the mucous membrane. They remain in these locations for several weeks unless removed.

**Acariid.** *Rhinophaga* spp.

**Lepid**

**Nematodes.** *Filaroides* (*Metastrongyloidea*).

*Alatachlamys* (*Spiruridae*).

**Cestode larvae.** *Echinococcus* spp. (*Taeniidae*) (see Figs. 4.50 to 4.53, 8.45, and 8.65).

**Acariid.** *Pseudozonurus simicola* (*Mesostigmata*) (less than 1 mm in length) (see Fig. 8.7).

### Serous Cavities

**Nematode.** *Dipetalonema* spp. (*Filarioidea*) (see Fig. 4.173).

**Cestode larvae.** *Theria hydatigena* (*Cysticercidae*) (up to 6 cm long) (see Fig. 4.43).

*Mesocercaria* (*tetrathyridium*) (1 cm or more long) (see Fig. 8.47).

*Sporontia mammosoma* (*plerocercoid*) (32 by 2.5 mm) (see Figs. 4.36 and 4.43).

**Podostomid acyphes.** *Paracaphalus*, *Arctidifur*, and *Leptostole* (see Figs. 2.137, 2.138, 2.139 and 8.9).

**Acanthocephalans.** *Prosthenocheila* spp. (see Figs. 8.79 and 8.80, and see Fig. 7.176 for egg depiction).

### Blood

**Nematode microfilariae.** *Dirofilaria*, *Dipetalonema*, *Tetraepetalonema*, *Loa*, and *Brugia* (*Filarioidea*). Differentiation of the many kinds of microfilariae found in monkeys from all parts of the tropics is a task for the specialist. Many species remain to be described.

**Protozoans.** Simian malarial *espansions*, *Plasmodium* and *Hepatocystis*.

### Muscle and Connective Tissues

**Nematodes.** *Oncocerca*, *Dirofilaria*, *Dipetalonema*, *Tetraepetalonema*, *Loa*, and *Brugia* (*Filarioidea*) (see Figs. 4.173). *Oncocerca* microfilariae are found in the dermis.

**Cestode larvae.** *Theria* (*Cysticercidae*).

*Mesocercaria* (*tetrathyridium*) (see Fig. 8.47).

*Sporontia* (*plerocercoid*) (see Figs. 4.36 and 8.48).

### Skin and Hair

**Insects.** *Podococcus* and *Pthirus* (*Anoplura*) (see Fig. 2.39).

**Nematodes.** *Anatrichosoma cutaricum* (Tracheoeloides). Very slender (25 mm by 0.2 mm) worms give rise to subcutaneous nodules, edema about the joints, and elongated, elongated blisters of the palm and sole. Adult females burrow in the epidermis of the palms and soles (Fig. 7.279).  
*Onchocerca microfilariae*.  
*Dracunculus* (Spirurida) (see Figs. 4.156, 4.157, 7.443, 7.445, 7.446 and 7.447).

## REFERENCES

- Agreiros, J., Claerebout, E., & Vercruyssen, J. (2003). Development of a copro-antigen capture ELISA for detecting *Ostertagia circumcincta* infections in cattle. *Veterinary Parasitology*, 97, 227.
- Ash, I. R., & Orford, T. C. (1993). *Atlas of Human Parasitology*. Chicago: ASCP Press.
- Balboobeh, L. R., Beugnot, F., Marchiondi, A. A., et al. (2014). American Association of Veterinary Parasitologists review of veterinary fecal flotation methods and factors influencing their accuracy and use—there really are best techniques! *Veterinary Parasitology*, 204, 73–80.
- Bowman, D. D., Hendrix, C. M., Lindsay, D. S., et al. (2002). *Feline Clinical Parasitology*. Ames, Iowa: Iowa State University Press.
- Cringoli, G., Rinaldi, L., Venturini, V., et al. (2004). The influence of flotation solutions, sample dilution and the choice of McIlwain slide area (volume) on the reliability of the McMaster technique in estimating the faecal egg counts of gastrointestinal strongyles and *Trichostrongylus axei* in sheep. *Veterinary Parasitology*, 123, 121–131.
- Dassil, E. D., & Lindquist, W. D. (1993). Determination of the specific gravity of certain helminth eggs using sucrose density gradient centrifugation. *Journal of Parasitology*, 83, 918.
- Deplains, F., Allhet, P., Yarnet, J., et al. (1999). *Echinococcus multilocularis* coproantigen detection by enzyme-linked immunosorbent assay in fox, dog, and cat populations. *Journal of Parasitology*, 85, 118.
- Deplains, F., Rinaldi, L., Alvarez Rojas, C. A., et al. (2007). Global distribution of alveolar and cystic echinococcosis. *Advances in Parasitology*, 55, 313.
- Dikmans, G., & Anderson, J. S. (2002). A comparative morphological study of the infective larvae of the cestode *Monostomum parvum* in the alimentary tract of sheep. *Transactions of the American Microscopical Society*, 32, 1.
- Dubey, J. P. (1979). A review of *Sarcocystis* of domestic animals and of other *Sarcocystis* of cats and dogs. *Journal of the American Veterinary Medical Association*, 169, 1081.
- Dubey, J. P., Perry, A., & Kennedy, M. J. (1987). Encephalitis caused by a *Sarcocystis*-like organism in a steer. *Journal of the American Veterinary Medical Association*, 181, 231.
- Eddleman, M. L., Lucero-Farfan, A., Kern, T. J., et al. (2014). Ophthalmomyiasis interna anterior in a dog: heretofore and extraction of a *Cuterebra* sp. larva. *Veterinary Ophthalmology*, 37, 448.
- Elliott, D. A., Gong, J., Cote, J., et al. (2017). Enzyme-linked immunosorbent assays for coproantigen detection of *Ancylostoma caninum* and *Trichostrongylus axei* in dogs and *Trichostrongylus axei* in cats. *Journal of Veterinary Diagnostic Investigation*, 29, 645.
- Elliott, D. A., Gong, J., Flynn, L., et al. (2014). Enzyme-linked immunosorbent assays for coproantigen detection of *Trichostrongylus axei* in dogs. *Journal of Veterinary Diagnostic Investigation*, 26, 604.
- Fales, K., & Fales, K. (1984). Improved detection of intestinal parasites. *Modern Veterinary Practice*, 65, 273.
- Fumes, A. O., & Johnson, C. M. (1993). A preliminary note on the identity, life cycle, and pathogenicity of an important nematode parasite of captive monkeys. *American Journal of Tropical Medicine and Hygiene*, 19, 285.
- Georgi, I. R. (1975). Differential characters of *Filaroides milkeri* Whitlock, 1958 and *Filaroides hirthi* Geopel and Anderson. *Proceedings of the Helminthological Society of Washington*, 45, 142.
- Georgi, I. R., & Anderson, R. C. (1973). *Filaroides hirthi* sp. n. (Nematode: Metastrongyloidea) from the lung of the dog. *Journal of Parasitology*, 63, 337.
- Gordon, H. M., & Whitlock, H. V. (1939). A new technique for counting nematode eggs in sheep faeces. *Journal of the Council for Scientific and Industrial Research*, 12, 52.
- Greve, J. H. (1903). Identifying nematode larvae in feces of dogs and cats. *Iowa State University Veterinarian*, 67, 98.
- Hobert, P. D. N., Oviwinda, A., Ball, S. L., et al. (2003). Biological identifications through DNA barcodes. *Proceedings of the Royal Society of London B Biological Sciences*, 270, 313.
- Hogins, P. L., Dabbling, B. R., & Karacas, K. R. (1987). Multilocus retinitis in New Zealand sheep dogs. *Veterinary Pathology*, 24, 22.
- Jenkins, D. J., Frazer, A., Bradshaw, H., & Crup, P. S. (2008). Detection of *Echinococcus granulosus sensu lato* in Australian rams with natural or experimental infection. *Journal of Parasitology*, 86, 148.
- Johanson, S. (1976). On a new enyural nematode of the genus *Aspicularia* from the common house rat, *Rattus norvegicus*. *Indian Journal of Helminthology*, 21, 147–148.
- Johanson, D. A., Belske, J. M., & Cole, G. C. (2004). Copro-antigen capture ELISA for the detection of *Taenia* (*Ostertagia circumcincta*) in sheep: improvement of specificity by heat treatment. *Parasitology*, 129, 115.
- Kaenzl, G. P., & Gordon, H. M. (1941). A useful mixing apparatus for the preparation of suspensions of faeces for helminthological examinations. *Journal of the Council for Scientific and Industrial Research (Australia)*, 14, 304.
- Kath, R. K. (1953). Infective larvae of cattle nematodes. *Australian Journal of Zoology*, 1, 223.
- Knafl, I. (1939). A method for making microfilarial curves on dry blood. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 33, 191.
- Lichtenfels, J. R. (1975). Helminths of domestic equids. *Parasitology of Helminthological Society of Washington*, 42, 1.
- Lichtenfels, J. R., Kharochuk, V. A., & Drojnos, G. M. (2004). Illustrated identification keys to strongylid parasites (STRONGYLIDAE: Nematoda) of horses, reeves and asses (Equidae). *Veterinary Parasitology*, 56(1–2 special issue), 4.
- Lichtenfels, J. R., Kharochuk, V. A., Krecel, K. C., et al. (1998). An annotated checklist by genus and species of 90 species level names for 34 recognized species of small strongyles (*Nematoda: Strongylidae: Cyathostominae*) of horses, asses, and reeves of the world. *Veterinary Parasitology*, 79, 65.
- Lindsay, D. S., Upton, S. J., & Dubey, J. P. (1999). A structural study of the *Spirocerca caninum* cyst. *International Journal for Parasitology*, 29, 1521.
- Lorenzetti, L. I., Caporaso, J. L., & O'Connell, B. M. (1979). Trichostrongylosis in a cat. *Journal of the American Veterinary Medical Association*, 175, 209.
- Lucero-Farfan, A., Liria, I. L., Yarnet, J. P., et al. (2011). Morphological differentiation of eggs of *Ancylostoma caninum*, *Ancylostoma tubaeforme*, and *Ancylostoma braziliense* from dogs and cats in the United States. *Journal of Parasitology*, 98, 1941.
- McIntosh, I. G., Lichtenfels, J. R., Griner, E. C., et al. (1983). Migration of a spicular nematode through the brain of a horse. *Journal of the American Veterinary Medical Association*, 286, 1386.
- McGraw, B. M., & Slocum, I. O. (1983). *Strongylus equorum*: development and pathological effects in the equine host. *Canadian Journal of Comparative Medicine*, 45, 372.
- McGarry, J. W., & Morgan, E. R. (2009). Identification of first-stage larvae of metastrongyles from dogs. *Veterinary Record*, 165, 236.
- McLeary, N. J., Newkirk, K., & Adams, C. M. (2017). Canine ocular onchocercosis: a retrospective review of the diagnosis, treatment, and outcome of 19 cases in New Mexico (2011–2015). *Veterinary Ophthalmology*, 20, 349.
- Mederet, K. L., Lucero-Farfan, A., & Bowman, D. D. (2018). Evaluation of parasite egg and cyst recovery using devices designed for centrifugal or stationary flotation. *J Am Anim Hosp Assoc*, 54, 38.
- Newton, W. L., & Wright, W. H. (1950). The occurrence of a dog filarial child. *Journal of Parasitology*, 42, 208.
- Newton, W. L., & Wright, W. H. (1957). A reevaluation of the canine filarosis problem in the United States. *Veterinary Medicine*, 52, 75.
- O'Hanley, B. M. O., Olores, M. F., Frazer, D., et al. (2003). Prevalence and genotypic characteristics of *Gleisnia* in dairy calves from Western Australia and Western Canada. *Veterinary Parasitology*, 100, 193.
- Quaranta, D., Giannelli, A., Latella, M. S., et al. (2015). Canine infections with *Oncocercus lupi* nematodes, United States, 2011–2014. *Emerging Infectious Diseases*, 21, 668.
- Prentiss, J. C., Bowman, D. D., Gillette, D. M., & Greve, R. B. (1988). Documented granulomatous disease in a cat caused by larvae of *Taenia canis*. *Journal of Comparative Pathology*, 99, 343.

- Rainey, T., Occi, I. L., Robbins, R. G., et al. (2018). Discovery of *Haemaphysalis longicornis* (Ixodida: Ixodidae) parasitizing a sheep in New Jersey, United States. *Journal of Medical Entomology*, 55, 757.
- Schneider, T., Heise, M., & Epe, C. (1999). Genus-specific PCR for the differentiation of eggs or larvae from gastrointestinal nematodes of ruminants. *Parasitology Research*, 85, 895.
- Speare, R., & Tinsley, D. J. (1987). Survey of cats for *Strongylidae felis*. *Australian Veterinary Journal*, 64, 191.
- Stoll, N. R. (1925). Investigations on the control of hookworm disease. XV: an effective method of counting hookworm eggs in human feces. *American Journal of Hygiene*, 3, 59.
- Stoll, N. R. (1930). On methods of counting nematode ova in sheep dung. *Parasitology*, 22, 116.
- Supperer, B. (1953). Filariosen der Pferde in Österreich. *Wiener Tierärztliche Monatsschrift*, 40, 214.
- Tauxe, J. A., & Loomis, M. R. (1982). Epirostitis in *Ixodes* gerrilli. *Journal of the American Veterinary Medical Association*, 181, 1545.
- Thomas, J. S. (1988). Encephalomyelitis in a dog caused by *Baylisascaris* infection. *Veterinary Pathology*, 25, 94.
- Traversa, D., Iorio, R., & Klein, T. R., et al. (2007). New method for simultaneous species-specific identification of equine strongylids (Nematoda, Strongylidae) by reverse line blot hybridization. *Journal of Clinical Microbiology*, 45, 2917.
- Tropen, C. V., & Todd, K. S. (1979). Life cycle of *Isospora bussoni* n. sp. (Protozoa: Eimeriidae) from the dog *Canis familiaris*. *American Journal of Veterinary Research*, 38, 95.
- Whitlock, J. E. (1941). A practical dilution egg count procedure. *Journal of the American Veterinary Medical Association*, 88, 466.
- Whitlock, J. E. (1980). *The Diagnosis of Veterinary Parasitosis*. Philadelphia: Lea & Febiger.
- Xiao, L., Berra, C., Kimon, J., et al. (2003). Identification of 5 types of *Cryptosporidium* parasites in children in Lima, Peru. *Journal of Infectious Diseases*, 287, 492.
- Zajac, A. M., & Conboy, G. A. (2012). *Veterinary Clinical Parasitology*. Hoboken: Wiley-Blackwell.
- Zarlenga, D. S., Chait, M. R., Guthrie, L. C., & Boyd, P. C. (2001). A multiplex PCR assay for differentiating economically important gastrointestinal nematodes of cattle. *Veterinary Parasitology*, 97, 199.

## Histopathologic Diagnosis

Andrew D. Miller

The microscopic identification of parasites in tissue sections is an interesting challenge. Often a diagnostician is provided with a single slide that shows only pieces of the parasite. In an attempt to identify an object believed to be a parasite, one should gather as much information about the patient as possible, including life history and clinical signs. It is also important to be familiar with the kinds of parasites most likely to be found in the particular host and tissue under study, as well as in the specific geographic area. The host-organ listing of parasites in the preceding chapter should be considered as a checklist of possibilities. The main objective of this section is to emphasize some of the major microscopic anatomic features of parasites that can be helpful in their identification in histologic sections. For arthropods and mite-like parasites, several defining characteristics can be listed for each group of parasites, but the presence or absence of a body cavity and digestive tract and the type and distribution of muscle fibers are important criteria to be considered in making an initial placement into a major group.

For further reading and assistance with diagnosis of parasites in tissues, the following sources are helpful. A report dealing with the present subject is *Identifications of Parasitic Metazoa in Tissue Sections*, by Maybelle Charwood and J. Ralph Lichtenthal, first published in *Experimental Parasitology*, volume 52, pages 607 to 519, 1972, and later reprinted as a monograph by the US Department of Agriculture. Texts dealing with the subject include *Parasitology of Tropical and Extraordinary Diseases*, volumes 1 and 2, edited by C.H. Binford and D.H. Connor, Washington, DC, 1976, Armed Forces Institute of Pathology (AFIP); *Parasitology of Infectious Diseases*, volumes 1 and 2, by D.H. Connor, F.W. Chandler, D.A. Schwartz, H.J. Mann, and E.E. Lack, Stamford, Connecticut, 1967, Appleton & Lange; *An Atlas of Protozoan Parasites in Animal Tissues*, by C.H. Gardner, R. Fayer, and J.P. Dubey, USDA Agriculture Handbook No. 651, US Government Printing Office, Washington, DC, 1988, and edition 2, published by AFIP, American Registry of Pathology, Washington, DC; *Diagnostic Pathology of Parasitic Infections with Clinical Correlations*, edition 2, by Y. Gustave, Philadelphia, 1990, Lea & Febiger; *Parasites in Human Tissues* by E.C. Ortel and L.B. Adu, Chicago, 1995, American Society of Clinical Pathology (ASCP) Press; *Parasitic Diseases in Nonhuman Primates*, volume 2, by K. Stritt, J.G. Else, and M.L. Eberhard, in *Nonhuman Primates in Biomedical Research: Diseases*, edited by C.R. Abbe, K. Mansfield, S.D. Tarzitt, and T. Morris, San Diego, 2012, Academic Press; and *Parasitology of Infectious Diseases*, volume 1, *Helminthiasis*, by W.M. Meyers, R.C. Neafie, A.M. Martey, and D.J. Weir, AFIP, 2000, American Registry of Pathology, Washington, DC.

### ARTHROPODS

Arthropods, composed of hundreds of thousands of species, have such diverse features that describing them succinctly is nearly impossible in

a single histologic section. Arthropods do have some shared features—for example, they have a segmented body, a chitinous exoskeleton, 4 exosom, and jointed appendages. The chitinous exoskeleton, the cuticle, in histologic sections usually appears thick and dark, but usually the exoskeleton itself does not take up stain. Over some parts of the body, especially in areas between segments or joints in an appendage, the cuticle can be very thin. The striated muscle of arthropods is diagnostic for this group of pathogens if they can be found in the section. The larger arthropods also have a respiratory system that is composed of a tracheal system, which in sections appears as variously sized tubes coursing throughout the body. The larger of the tracheal branches have chitinous reinforcing rings. Arthropods also can contain fat bodies that often appear darkly stained in sections. Smaller parasitic arthropods often have rounded to elongated bodies that are apparent in tissue sections, and sometimes one is fortunate enough to observe sections through paired, jointed legs. All together, these features are fairly complete in defining an arthropod in section.

Three major groups of arthropods are likely to appear in histologic sections. The insects (subphylum Mandibulata, class Insecta) contain the maggots of various sepsis-producing flies, and these commonly appear in histologic sections. The mites are in the class Arachnida of the subphylum Chelicerata, and these creatures because of their small size and ability to colonize various mainly superficial body surfaces, such as skin and respiratory mucosae, also appear in sections of lesions. Ticks tend to remain superficial to the host, attaching only long enough to feed, so typically, unless there is a strange clinical presentation or an interested researcher, they do not appear in histologic sections. The Pentastomida are a group of parasitic crustaceans that have larval stages that parasitize vertebrates.

### Maggots

Maggots in tissue are the larvae of dipteran flies and may represent species that require a living host or species, causing secondary myiasis such as that caused by various *Calliphora* and *Sarcophaga*. Both types of maggots display similar features, and the difficulty lies in making a generic diagnosis based strictly on morphology. The spiracular plate is important in identification of fly larvae and may need to be retrieved from the wet tissues or paraffin block (see Fig. 212).

Sections of maggots will display the typical features of an arthropod (e.g., body cavity), segmentation, striated muscles attached at various points to the chitinous exoskeleton, and tracheae, often with cuticular rings (Figs. 211 and 212). Some species have prominent spines (Fig. 212). *Catolpa* larvae are obligate endoparasites of rodents and lagomorphs; these larvae may invade dogs, cats, and occasionally humans. Typically, they are found in cervical subcutaneous tissues, but in dogs and cats they migrate into the central nervous system with disastrous results (Fig. 212). First-stage *Hypoderma* larvae migrate

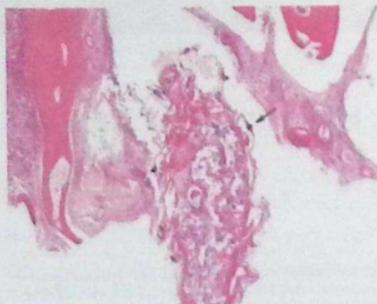


Fig. 8.1. *Cuterebra* larvae in the nasal cavity of a rabbit. Note the prominent spines (arrow) (20x).

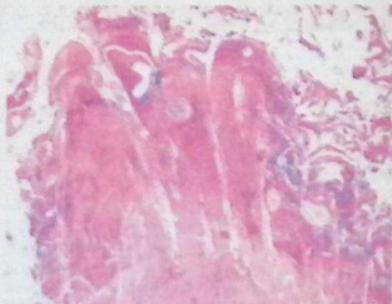


Fig. 8.3. *Sarcopista* mites embedded in dense parakeratotic hyperkeratosis in a fox (20x).

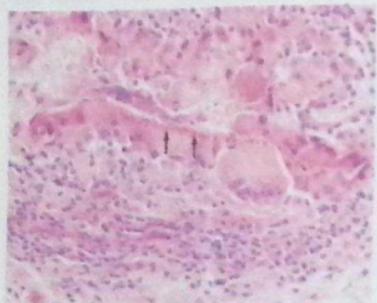


Fig. 8.2. Degenerate *Cuterebra* larva surrounded by granular inflammation in the brain of a cat (400x). The arrows highlight the dark brown spines.

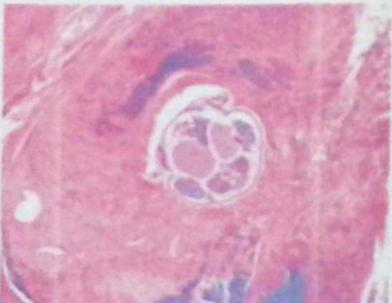


Fig. 8.4. *Sarcopista scabiei* in a flea. The chitinous appendages and ectotillar spines are evident (200x).

extensively in cattle, and erratic migration through the brain of horses has been reported.

### Mites

Mites tend to be rather small—millimeters or smaller in size. With many species, eggs, larvae (six legs), nymphs (eight legs), and adults (eight legs) are all found in section, and in a section of an adult can be found all the component parts of a typical arthropod—segmented legs, spines, and hairs externally, with striated muscles, reproductive organs, intestine, yolk glands, and developing eggs internally, may be seen in section. Mites that live in the skin—*Sarcoptes*, *Notoedres*, *Knemidocoptes*, and *Trixacarus*—are very small and round, feed at the stratum basale and dermis, and are typically found embedded in dense hyperkeratosis (Figs. 8.1 and 8.3) and have spines on their dorsum. In some hosts, such as the red fox, and pigs, sarcoptic mange is characterized by extraordinary hyperkeratosis (Fig. 8.3) and

similar hyperkeratosis is seen in cats with *Notoedres* infection. Hyperkeratosis is typical of mange caused by *Chorioptes* and *Chrysiella* in certain hosts, but the mites lie more superficially in the stratum corneum.

Demodex organisms are cigar-shaped mites found in hair follicles or associated sebaceous glands (Figs. 5.2 and 5.3), although some such as *Demodex cecis*, and *Demodex tejanii* tend to be superficial. In dogs with severe demodectic mange, *Demodex canis* may be found in the lymph nodes. Very large nodular lesions can be found in the skin in goats and can be seen occasionally with demodectic mange in cattle and swine.

Mites of the respiratory tract (e.g., *Parasitomyia*, *Sternostoma*) have more delicate endoskeletons than their ectoparasitic relatives. *Parasitomyia simicola* and *Parasitomyia stans* of the primate lung and canine nasal passages look like any other mesostigmatid mite (Fig. 8.7).



Fig. 8.5. Myriax *Dermodes canis* filling multiple hair follicles in the skin of a dog (20x).

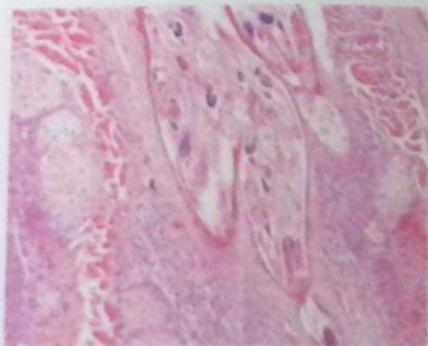


Fig. 8.5. Higher magnification of Fig. 8.5 illustrating the elongate body and large number of organisms filling the hair follicle (400x).

Trombiculid larvae (chiggers) feed through a styletome or feeding tube extending into the dermis; very typically, the stylet becomes dislodged, and all that remains is the very pruritic lesion.

#### Pentastomids

Pentastomids are so called because of the early belief that they had five mouths; in reality, they possess one mouth surrounded by four hooks (see Fig. 2.100). The adults of these bizarre crustaceans are worm-like parasites in the respiratory passages of predaceous reptiles, birds, and mammals that for the most part become infected when they ingest nymphs encysted in the tissue of their prey (Fig. 8.8). It is in the vertebrate prey that the nymphs appear in tissue sections after the host has ingested an egg (Fig. 8.9) containing a larva with four or six appendages. The pseudosegmented body of the nymph has a spheric to oval shape and is covered by a thick cuticle with sclerotized openings—stomata (Fig. 8.10). Pentastomids have a complete digestive system with a mouth and an anus, and in section the intestine is often



Fig. 8.7. *Pneumovax zinnicola* in the lung of a rhesus monkey. The mite has prominent yolk material (arrow). These parasites live in the distal airways and lead to significant bronchiectasis (40x).



Fig. 8.8. Numerous pentastome larvae encysted in the diaphragm of a dog. (Photo courtesy the Indiana Animal Diagnostic Disease Laboratory, Purdue University.)



Fig. 8.9. Pentastomid nymph from near the bladder of a cynomolgus monkey (100x). The cuticle is marked by stoma, aedepta, and the nymph contains large ectosymbiotic glands (asterisk).

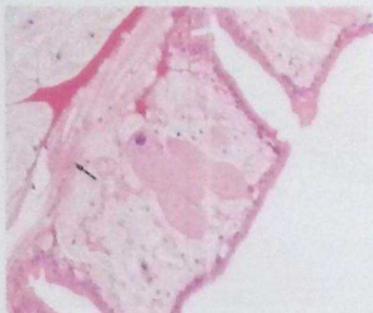


Fig. 8.10. Cross section through the body wall of a pentastome nymph illustrating the pseudosegmentation and thin bands of skeletal muscle (arrow) (400x).

surrounded by large acidophilic glands (Fig. 8.9). These acidophilic glands are a good distinguishing characteristic for this group of organisms; they stain bright pink with prominent blue nuclei in hematoxylin and eosin-stained sections. The musculature is striated and is located within the subcuticular region.

## PROTOZOA

Protozoa that are found in sections tend to be highly specialized individual cells with distinctive nuclei and other structures that may occur singly or in "nests" either within or external to the cells of the host. At the light microscope level, it is often difficult to ascertain many details of the individual cells, and often electron microscopy of material will provide the added detail required for a diagnosis. Also, immunohistochemical or *in situ* hybridization methods can often be used for some infections (e.g., for *Toxoplasma gondii*, *Neospora caninum*, and *Sarcocystis neurona*) to make a definitive diagnosis as to a genus or species of parasite in a particular case.

With a great many of the protozoa, it is often difficult to distinguish even distantly related organisms purely on the basis of the structures seen in sections because of preparation, because of the way they are fixed and stained, and because one is near the working resolution of the light microscope. Therefore a group of amastigotes of *Trypanosoma cruzi* may look very similar to a pseudocyst of rounded nuclei of *T. gondii*. This should be fairly straightforward because the amastigotes should be seen to contain the identifying kinetoplast, but they may be visible in only a portion of the organisms, and the typically elongate nuclei of *T. gondii* may appear simply as round small nucleated cells in some sections. Often it is very helpful to also consider the history, clinical signs, and overall pathologic changes seen in a case when making a diagnosis.

## Flagellates

Typical flagellates that occur in the tissues of vertebrates include two that are known as amastigote stages living within host cells. Amastigotes are small, round to oval bodies measuring 1.5 to 4  $\mu\text{m}$  in diameter (often smaller after tissue processing); they contain a nucleus and a rod-shaped kinetoplast. They do not store periodic acid-Schiff (PAS)-positive material. The two groups of organisms that have these

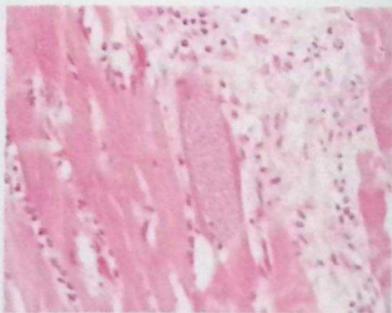


Fig. 8.11. *Trypanosoma cruzi* amastigotes in cardiac muscle of a sheep (400x).

stages are *Trypanosoma cruzi* and various species within the genus *Leishmania*.

Both trypomastigote and amastigote stages of *T. cruzi* occur in the vertebrate host, but generally only the amastigotes are seen in tissue sections; the trypomastigote stage is found almost exclusively in the blood. *T. cruzi* amastigotes are generally found in muscle cells of the esophagus, colon, and heart, where they may be responsible for megacosophagus, megacolon, and myocarditis (Fig. 8.11), respectively.

The amastigotes of *Leishmania parasites* only one cell type in the vertebrate host, the macrophage. Therefore they can be found in skin, bone marrow, and visceral organs such as the spleen and Kupffer cells of the liver. Again, the diagnostic organism within the parasite is the kinetoplast, but diagnosing the infection in tissue sections can be difficult because of shrinkage of cells during fixation, which can make visualization of the nucleus and kinetoplast challenging. One of the major differentiations to be considered is whether one is dealing with leishmaniasis or an infection with *Histoplasma capsulatum*. Needle biopsy or impression touch prep from cutaneous lesions or lymph node and bone marrow aspirates may be prepared and stained with Wright-Giemsa solutions, and in these preparations, the full structure of the organism, including both the nucleus and the kinetoplast, is generally more clearly visible (Fig. 8.12).

## Ciliates

*Neobalamandru coli* trophozoites live within the contents of the cecum and colon of pigs but may secondarily invade the wall of the large intestine of swine that have various forms of enteritis. Trophozoites are characterized by their large size and the presence of a macronucleus and a micronucleus and cilia (Fig. 8.13). Ruminant ciliates may be found in the lung as a result of terminal inhibition of ruminal contents, in which case no evidence of an inflammatory reaction is found. They can also become invasive postmortem and should not be overinterpreted as a causative agent of enterocolitis in large animals (Fig. 8.14). Rumen ciliates may also be found in hepatic vessels in cases of very severe enteritis. In horses with severe enteritis, the extravagantly shaped ciliates normally present in the large intestine may secondarily penetrate the submucosa. These ciliates have large, often polymorphic macronuclei, and some have tufts of long cilia.

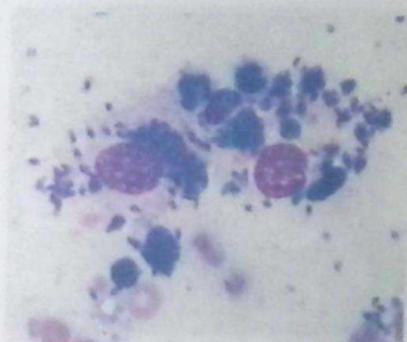


Fig. 8.12. *Leishmania amastigotes* in a lymph node of a dog. Both intracytoplasmic and extracytoplasmic organisms are evident. The nucleus and kinetoplast are obvious in the organisms.

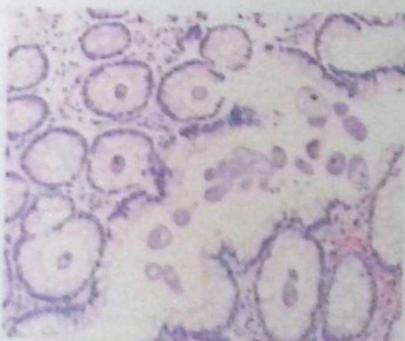


Fig. 8.13. *Neobabesium coli* in the lumen of the large intestine of a pig (DOG).

### Apicomplexa

**Coccidia.** The coccidia are members of the phylum Apicomplexa. Included in this discussion are members of the genera *Eimeria*, *Kleinstella*, *Cystoisospora*, *Homonadus*, *Besnoitia*, *Sarcocystis*, *Neospora*, and *Toxoplasma*. The life history and development of the major genera of coccidians are described in Chapter 3. There seems to be a good deal of consensus around placement of the genus *Cryptosporidium* within the coccidians rather than with the coccidia, but for convenience, these species are still included in this section. The genera *Eimeria* and *Cryptosporidium* seem to be completely monoxenous (i.e., with transmission always between members of one type of host with no paratenic or intermediate hosts), and almost all of the stages seen in section occur within the epithelium of the gastrointestinal tract, or rarely the gallbladder. *Kleinstella* is also apparently monoxenous, with direct transmission between hosts and almost all stages found in the epithelium of the renal system. The other coccidia are facultatively



Fig. 8.14. Postmortem invasion of ciliates in the small intestine of a horse (DOG).

(*Cystoisospora* and *Toxoplasma*) or obligatorily (*Sarcocystis*, *Homonadus*, *Neospora*, and *Besnoitia*) heteroxenous, that is, they have a paratenic or intermediate host. For heteroxenous species of coccidia, the stages often seen in tissue are the stages causing disease in the prey animal that is serving as the paratenic or intermediate host. A description of the histologic appearance of the various stages follows, but host specificity, site specificity, life cycle, and details of development characteristic of the genera and species of coccidia must be considered in arriving at a diagnosis.

### *Eimeria* and *Cystoisospora*

**Asexual stages.** The infective stage contained in the oocyst is the sporozoite, which is a product of a reduction division that occurs in the oocyst (Apicomplexa are haploid except immediately after fusion of the gametes). When a sporozoite enters a cell, it rounds up as a trophozoite in a membrane-lined parasitophorous vacuole. Not every species of coccidian stays within a parasitophorous vacuole, and this fact can be a useful adjunct in generic and specific diagnoses.

Trophozoites multiply asexually within cells by several processes. In the case of *Eimeria*, they typically undergo a spiral type of cellular division called *schizogony* (other terms that describe this form of division with various nuances are *merogony* and *endopolygony*). In this type of division, the apical complex divides into numerous copies around the periphery of the cell, the nucleus lobulates with portions associated with each apical complex, and finally the cell membrane contracts and divides to form a few to thousands of individual organisms. Depending on the species, schizonts may be found in enterocytes, biliary epithelial cells, endothelial cells, renal epithelial cells, or even uterine epithelial cells. Ordinary meronts contain from less than 10 to hundreds of merozoites; some meronts (megaschizonts) may contain more than 100,000 merozoites.

**Sexual stages.** A merozoite produced by the final schizontic generation enters a fresh host cell and develops into a male or a female gametocyte. The female gametocyte enlarges, stores food materials, and induces a hypertrophy of both the cytoplasm and the nucleus of its host cell. When mature the female gametocyte is called a *macrogamete* (Fig. 8.15). The male gametocyte also induces hypertrophy of the cytoplasm and the nucleus of its host cells, it undergoes repeated nuclear division and becomes multinucleated. Each nucleus is finally

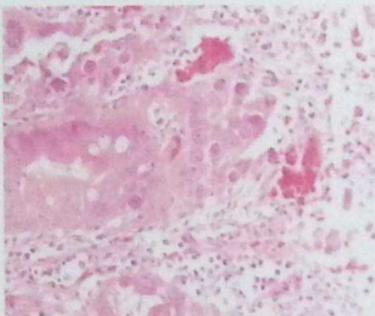


Fig. 8.15. Numerous intraepithelial macrogametes of *Eimeria necatrix* in a chicken. Note the peripheral ring of eosinophilic granular material.



Fig. 8.16. Numerous stages of *Eimeria macdanielis* in compartment three of an alpaca. Solvents, macrogametes, microgametes, and oocysts are present.

incorporated into a flagellated microgamete (Fig. 8.38) (the microgametes of *Cryptosporidium* spp. are without flagella). When a microgamete is penetrated and fertilized by a macrogamete, it becomes a zygote. Wall-forming bodies already present in the macrogamete, then become clearly visible as large, spheric, eosinophilic granules in the cytoplasm of the zygote (Fig. 8.16). These later coalesce to form the oocyst wall.

**Examples.** In small ruminants, small intestinal coccidiosis produces a characteristic gross lesion with multiple, raised, white nodules throughout the small intestine (Fig. 8.17). In the horse, *Eimeria leuckarti* forms large schizonts and very thick-walled and obvious oocysts (Fig. 8.18). The oocyst of *Cystoisospora* spp. develop within the lamina propria rather than in the epithelial cells (Fig. 8.19). *Eimeria gibbata* is atypical in that it forms megaloschizonts in the abomasum that are visible to the naked eye. *Eimeria stiedae* of the rabbit lives in and causes proliferation of the biliary epithelium and can produce a lethal



Fig. 8.17. Proliferative enteritis in a goat associated with small intestinal coccidiosis.

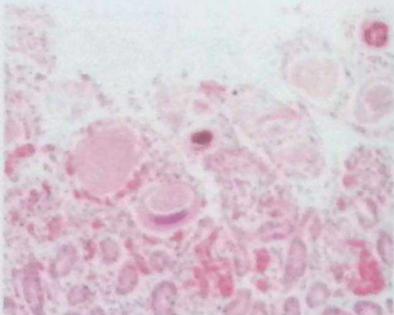


Fig. 8.18. *Eimeria leuckarti* schizont and developing oocyst in the intestinal mucosa of a horse (G906).

hepatitis (Fig. 8.20). For *Cystoisospora* spp., the released sporozoites colonize intestinal epithelium whose sporozoites are typically observed as they undergo endodyogeny (Fig. 8.21).

**Cryptosporidium.** The minute (5 to 7  $\mu$ m) stages appear as basophilic spheres on the luminal surface of epithelial cells of the gastrointestinal tract of vertebrates (Fig. 8.22). On rare occasions, typically in the immunocompromised host, infection of respiratory, pancreatic, or gallbladder epithelium may also occur. The infection is very superficial and appears to protrude from the surface of the cell, but these are intracellular parasites, and all stages—schizonts, gametes, oocysts, and so on—form underneath the host cell membrane.

**Klebsiella.** A parasite of the equine kidney; *Klebsiella equi* is usually an accidental histopathologic finding. Schizogony occurs in the glomerular endothelium and in the proximal convoluted tubules of the kidneys. The distinctive sporonts (Fig. 8.23) in the renal tubular

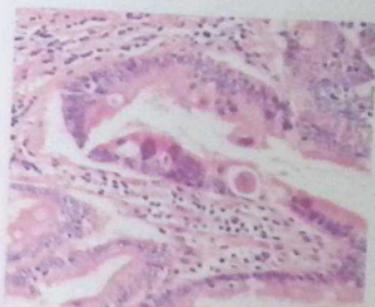


Fig. 8.18. Meronts of a *Cystoisospora* sp. developing in the small intestine of a cat (400x).

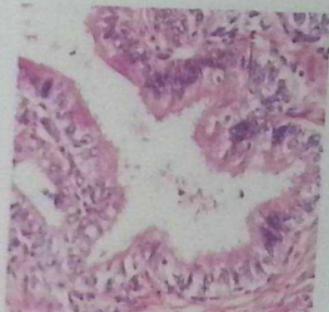


Fig. 8.22. *Cryptosporidium parvum* infection in the bile ducts of an immunosuppressed mouse. Note the numerous round protozoa intimately associated with the surface of the epithelial cells (400x).

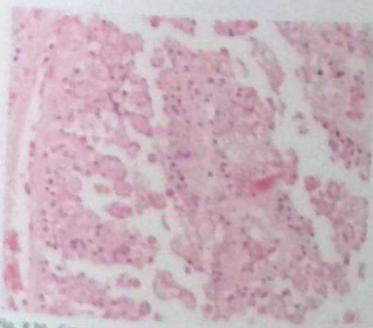


Fig. 8.26. *Eimeria acidae* developing in the bile duct epithelium of a rabbit (200x).

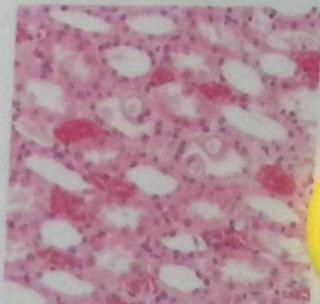


Fig. 8.23. Sporozoites of *Klebsiella equi* in the renal tubular epithelium of a horse (400x).



Fig. 8.21. *Cystoisospora suis* meronts (arrows) in the small intestine of a pig (400x).

epithelium produce as many as 40 sporoblasts, which develop into sporocysts, each of which may contain eight to 15 sporozoites. A similar species, *Klebsiella muris*, will show up in histologic sections of murine kidneys.

**Sarcocystis.** The early schizonts of *Sarcocystis* occur in endothelial cells of different organs and can be especially pathogenic in several organ systems, including the central nervous system. In the central nervous system merizonts induce necroinflammatory disease (Figs. 8.24 and 8.25). Sarcocysts, the stages found in the intermediate host, are found in skeletal and cardiac muscle fibers (Fig. 8.26); they vary in size from a few micrometers in diameter to macroscopically visible objects, stain intensely with hematoxylin, and are packed full of bradyzoites that are larger than those of *Toxoplasma*. Septa subdivide the interior of the sarcocyst but may escape notice because they stain poorly or not at all with hematoxylin and eosin. Often the cyst wall is

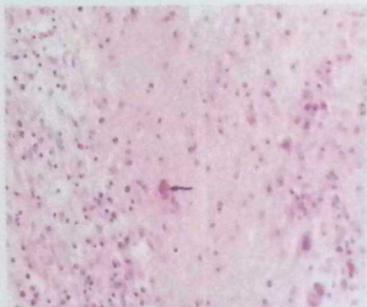


Fig. 8.24. *Sarcocystis falcatula*-associated meningionephritis in a falcon. Merozoites are commonly found associated with necrosis and inflammation (arrow) (400x).



Fig. 8.25. Sarcocyst of *Sarcocystis* spp. in skeletal muscle of a cat (400x).

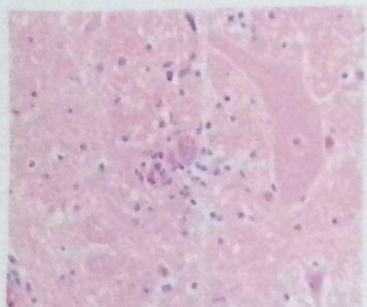


Fig. 8.26. Degenerating cyst of *Sarcocystis neurona* in the brainstem of a horse with encephalitis (400x).

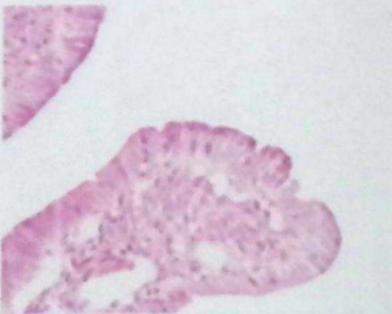


Fig. 8.27. *Toxoplasma gondii* development stages in the intestinal epithelium of an experimentally infected cat (400x).

described as hairy because of the many prolongations that give the cyst its apparent striated border. The hairy wall and the septa dividing the zites within the sarcocysts are often diagnostic.

**Hammondia.** *Hammondia* appears very similar morphologically to *Toxoplasma gondii*; the distinctions are biologic and molecular more than structural. The life cycle is obligatorily heteroxenous, but stages very similar to those described for *Toxoplasma* in the next paragraph are found in the tissues of many warm-blooded vertebrate animals that serve as prey to dogs and cats. These parasites have not been found to cause disseminated disease in immunosuppressed or immunocompromised hosts.

**Toxoplasma.** The stages that occur within the epithelial cells of the cat are to a great extent comparable with what occurs with *Eimeria* and *Cyrtospora* (Fig. 8.27). It is within the genus that the names tachyzoite and bradyzoite were first used to describe the different life stages

that occur in the parasitic hosts. Within these hosts the only form of division that occurs is endodyogeny, which is similar to schizogony, but only two daughter cells are formed in each dividing organism. The only asexuals seen with *T. gondii* occur in the intestinal epithelial cells of felids (Fig. 8.27). Tachyzoites divide rapidly and for the most part cannot withstand pepsin digestion for any length of time. Bradyzoites divide slowly, are resistant to pepsin digestion, and form cysts in tissue that are most easily observed in histologic sections of brain stained with PAS because the slowly dividing forms store PAS-positive material. Cats can have cysts of bradyzoites throughout their bodies just as other hosts (Fig. 8.28). Tachyzoites accumulate as "groups" intracellularly; bradyzoites become tightly packed in "intracellular cysts." The latter, when found in striated muscle fibers, might be confused with sarcocysts or accumulations of *T. cruzi* amastigotes.

**Neospora.** Under the light microscope, the cysts of *N. caninum* are almost indistinguishable from those of *T. gondii*. The major distinction

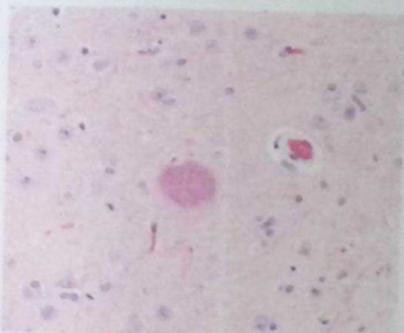


Fig. 8.28. *Toxoplasma gondii* bradyzoites in a cyst in the brain of a cat (400 $\times$ ).



Fig. 8.29. *Neospora caninum* bradyzoites in a cyst in a skeletal myofiber of a dog (400 $\times$ ).

that was recognized in the early description of this species was the thicker "cyst wall" that occurred around bradyzoites. In skeletal muscle infections in the dog, degeneration will ensue in the skeletal muscle and free zoster will cause significant necrosis and inflammation (Fig. 8.29).

**Besnoitia.** *Besnoitia* is mainly considered a pathogen of wildlife, specifically the opossum. It has also been described extensively as a cause of dermal and conjunctival infection in cattle and horses (Gubaj et al., 2005). The typical presentation are very large cysts without septa that are often found in the skin, although viscera may also be affected (Fig. 8.30).

**Hemosporidians.** A number of the Apicomplexa genera have heterocoenous life cycles with the sexual stages occurring in invertebrates and the asexual stages occurring in vertebrates (e.g., *Plasmodium*, *Theileria*, *Hepatozoon*, *Leucocytozoon*). With most of these parasites there is a good deal of description relative to the stages found in the blood of



Fig. 8.30. Skin of a donkey with multiple *Besnoitia* cysts characterized by a thick cyst wall (200 $\times$ ).

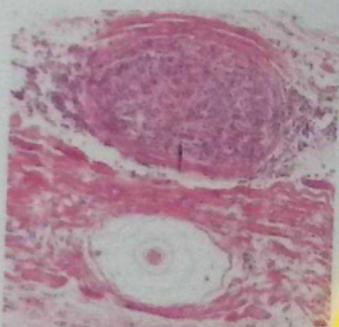


Fig. 8.31. *Hepatozoon americanum* infection in the skeletal muscle of a dog (200 $\times$ ). Both a paritized canine cell forming an uncoiled cyst and a localized granulomatous myositis associated with free merozoites (arrows) are present in this image.

the host, whereas very little time is spent describing the various stages, typically schizontous stages, that may occur in the viscera of hosts that can be seen in sections. *Babesia* infects only red blood cells, whereas *Theileria* infects erythrocytes and lymphocytes, because these are two of the most important hemoparasitans of domestic animals, there is little need to focus much attention on the schizont stage that occur in tissues. However, some other species do cause pathology and have stages in the tissues—schizonts that can be quite large and damaging.

**Leucocytozoon.** These species in chickens—*Leucocytozoon caulleryi* and *Leucocytozoon simondi*—produce megalo-schizonts in chickens and geese, respectively, that can be highly pathogenic. These schizonts can be very large and detrimental to the host.

**Hepatozoon.** *Hepatozoon* in the United States is associated with *Hepatozoon americanum*, which has cystic stages in the muscle of the dog host that are associated with chronic muscle pain in these animals. These stages are often used to assist diagnosis, which is often made as a result of muscle biopsy (Fig. 8.31).

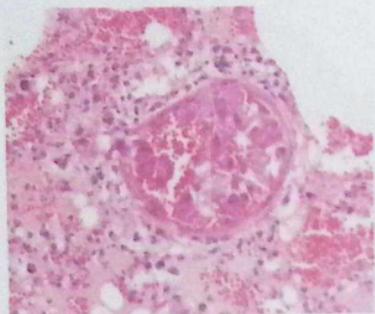


Fig. 8.32. Cytosuxion felis. A pteryneal vein of a cat filled with multiple, enlarged, mononuclear cells containing schizonts (400x).

**Cytosuxion.** Cytosuxion is a parasite that kills cats, often very acutely. Large schizonts can occur within the macrophages and cause them to become enormous. This is the reason for the name of the genus, and it is what makes the infection so deadly for cats. Sections throughout the body will have vessels plugged with these macrophages (Fig. 8.32).

### Amoebae

Amoebae are extracellular parasites that feed through the process of engulfing bacteria, cell debris, or other cells as food material. A vast majority of these organisms are nonpathogenic parasites or commensals living typically in the large intestine of animals. However, two forms do cause disease. Primates are host to *Entamoeba histolytica*, which can colonize the bowel wall and move to ectopic sites where the organisms establish cysts that most often include the liver, but they can also be found in lungs or brain tissue; reptiles have a similar pathogen, *Entamoeba invadens*, which can cause serious disease in these hosts with extraintestinal lesions. These amoebae tend to have nuclei typical of the genus with a central dot of chromatin, the karyosome, endosome, or nucleolus, and chromatin is also clumped around the inner surface of the nuclear membrane. These parasites can be found to contain erythrocytes, sometimes several, in various states of digestion. The other main group of disease-causing amoebae are facultative parasites that include the genera *Naegleria*, *Acanthamoeba*, and *Balamuthia*, which have infected dogs, sheep, cattle, primates, and humans. These forms live in the environment but can invade the tissue if they gain access through the nose or through wounds with lesions typically occurring in the brain or skin, but they can be found elsewhere. In sections, these amoebae tend to appear in clear spaces from artificial contraction of surrounding tissues during fixation and specimen preparation, to have foamy cytoplasm, and to have characteristic nuclei that contain a very dense endosome surrounded by a clear halo internal to the nuclear wall (Fig. 8.33).

### HELMINTHS

In examining helminths in section, basically two types can be seen: solid-bodied (the acolomates), and bodies in which tubes are suspended within a tube (the pseudocolomates). Trematodes and cestodes are of the solid body type; nematodes and acanthocephalans

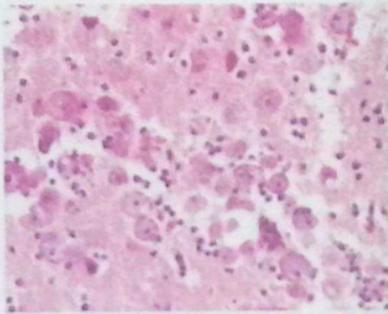


Fig. 8.33. Balantidium coli in the brain of a nonhuman primate causing extensive necrosis and granulomatous inflammation (400x). Note the gliotic nature of the amoebae and the prominent, typically central, magenta nucleolus.

represent the "hollow" body types. The problem can be that a trematode or a cestode may have all sorts of cavities in various organs that give them the appearance of having a pseudocoel, and nematodes may be so packed with organs and eggs or larvae that one starts to doubt whether one is looking at a nematode or not. The trematodes and cestodes are covered with a syncytial tegument, whereas the nematodes and acanthocephalans are covered with a secreted cuticle.

### Trematodes

Most trematodes are parasites of the digestive tract, but they only rarely show up in tissue sections. Trematodes in tissues are typically those in which the adults live in other tissues. Trematodes can be found throughout the bodies of vertebrates, including in the bile ducts, pancreatic ducts, body cavities, lungs, ureters, and blood vessels. In a few cases, larval stages can be found in domestic animals in which they may or may not cause disease.

The characteristics of trematodes in sections from a composite group of useful features, but because often the goal is to differentiate trematodes from cestodes, part of the characterization includes how they differ from cestodes. Of course, for almost every characteristic, there is one group that composes an exception. Trematodes have a solid but spongy body that usually contains no large cavities and is not divided into cortical and medullary layers as the body of cestodes is. Trematodes have an intestine that is usually bifurcate ending in a blind caecum (an example of an exception is the Cyclocoelidae, in which fusion of the posterior gut forms a continuous loop). Unlike tapeworms, trematodes do not contain calcareous corpuscles. The body is covered with the syncytial integument that often has spines (Fig. 8.34). Muscles are present below the integument, usually in an outer circular layer, a middle longitudinal layer, and an inner diagonal layer (which may also be external to the longitudinal layer or absent). Sex organs in the adult flukes are monocoelous hermaphrodites, except for Schistosomatidae, which have separate males and females. The eggs have typical shapes, and the shells are often brown to golden in sections. There are typically two suckers—one around the mouth and one ventral (often anterior to midbody)—and an excretory system that is difficult to see and empties through a pore at the posterior end of the body.



Fig. 8.34. *Fascioloides hepatica* in the liver of an ox. Note the lack of body cavity, body parenchyma, and surface tegument (20x).

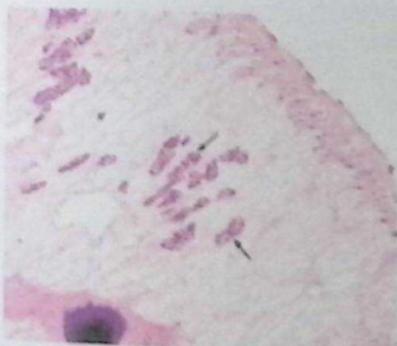


Fig. 8.35. Prominent vitelline glands removed in *Fascioloides magna* in the liver of an ox (200x).

Once a trematode is identified as such, the next step is to try to determine the family or genus. This involves calculating or guessing at the overall size and looking at the arrangement of the sex organs, the types of suckers if they are sectioned, and the extent and branching of the mesenteric and excretory systems. If there are eggs, they can be very helpful once the size, shape, type of operculum, and state of development (with or without a miracidium) have been noted. The spines on the surface of the body can also be very helpful in diagnosis; they will have to be examined to determine their number, size, and location on the body of the fluke.

Although both trematodes and cestodes have suckers, the oral sucker of trematodes is connected to a gut, whereas a gut is lacking in cestodes. The ventral sucker of trematodes is not connected to a gut. Sections through the uterus may contain eggs, which by their size, shape, and state of embryonic development may provide clues to the identity of the specimen. The arrangement of the sex organs and its distribution of vitelline glands in the trematode body are a much-used taxonomic characteristic (Fig. 8.37). For example, these glands lie both

dorsal and ventral to the gut in *Fasciola*, but all lie ventral to the gut in *Fascioloides*. The body form of some trematodes is quite distinctive. For example, heterophyids have small bodies with distinct spines and tend to be inserted in intestinal crypts, whereas diplostomatids are divided into a flattened forebody and a cylindrical hindbody. In the dioecious schistosomatids, the slender female is enclosed in the gynephoral groove of her stouter male partner. Mature, elongate sperm are often able to be visualized in multiple trematode species (Fig. 8.38), as opposed to the case in nematodes in which sperm are small and amoeboid. Adult trematodes lay eggs (Fig. 8.37) that can persist in the tissues for a long time, causing granulomatous inflammatory reactions in tissues (Figs. 8.39 and 8.39).

Larval trematodes, specifically monocercariae and metacercariae, are not uncommonly seen in tissue sections. They are often rather small, sometimes they are encountered singly, and other times numerous organisms are present. They are, like adult trematodes, composed of a solid parenchymal body with an outer tegument, but often little

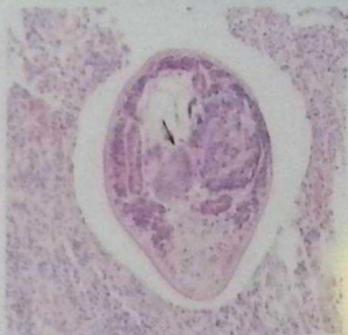


Fig. 8.36. *Nanophyetus salmicosolus* in the small intestine of a dog with prominent testes filled with sperm larvae (200x).



Fig. 8.37. Prominent, thick-shelled, yellow-brown eggs of *Fascioloides magna* in an ox (400x).



Fig. 8.38. Irregular, serpiginous tracts of fibrosis and pigmentation in the liver of an alpaca infected with *Fascioloides magna*.

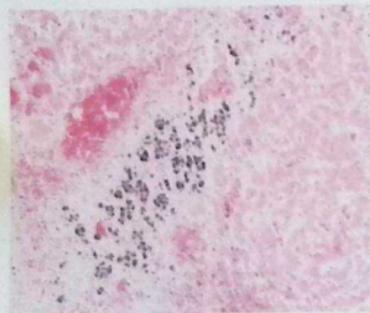


Fig. 8.39. Tracts of fibrosis and fluke pigment in the liver of an alpaca infected with *Fascioloides magna*. Even though no parasites are seen in this image, the pattern is typical for aberrant fluke migration (100x).

other internal structure is seen (Fig. 8.40). Because they represent immature stages, no reproductive structures are evident. No calcareous corpuscles are present, and this helps to distinguish them from larval tapeworms.

### Cestodes

Tapeworms seen in sections are most likely to be larval forms, although there is always a chance that a section of a proglottid may be seen in an unusual location. Tapeworms, unlike trematodes, have no intestine in any stage of the larvae or adults. Like trematodes, the internal organs of cestodes are embedded in a parenchymatous matrix; there is no body cavity. There are two principal zones of nonstriated muscle fibers—subtegumental and parenchymal (Fig. 8.41). It is through the tegument that tapeworms absorb their nutrients from the host, and the syncytial surface, especially in adult forms, is thrown into numerous villus-like projections for this purpose. Within the tapeworm, the parenchymal zone divides the parenchyma into a cortex lying outside

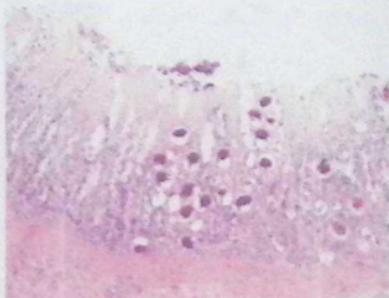


Fig. 8.40. *Heterobothrium americanum* eggs in the intestine of a naturally infected dog (700x).

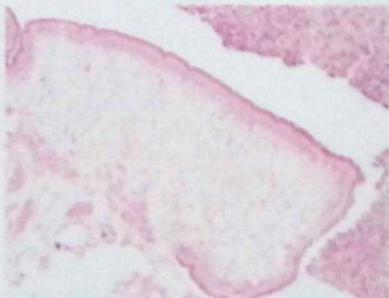


Fig. 8.41. *Diphylidium caninum* in the intestine of a dog (40x). Note the lack of body cavity and the surface tegument. Cestodes lack an intestinal tract.

a longitudinal layer of fibers, and a medulla lying within a transverse layer of muscle fibers; the medulla contains the osmoregulatory ducts and reproductive organs if these are present. Cestodes produce larvae in a vertebrate host (described later), and in some cestode species (i.e., *Diphylidium caninum*) egg sacks are often abundant in tissue sections (Fig. 8.42). Calcareous corpuscles are typical of cestode tissues and, especially in larvae, may provide the only evidence that the specimen is a tapeworm (Fig. 8.43). Tapeworms are covered by a tegument formed by the cytoplasmic projections of epidermal cells, which appears in histologic sections as a thick, homogeneous noncellular external layer supported by a basal membrane.

The larva of a tapeworm that is found in a vertebrate host represents the precursor to the adult form, and typically bears the holdfast or scolex of the adult in some rudimentary or embryonic form. After the host is ingested, much of the larva will be digested away and the small holdfast will attach to the intestinal mucosa and grow the adult



Fig. 8.42. Egg sacs of *Dipylidium caninum* in the perianal tissue of a dog (200x).



Fig. 8.44. Paired suckers of an adult Taenid tapeworm (40x).



Fig. 8.43. Prominent calcareous corpuscles (arrows) embedded in the parenchyma of a cestode (100x).

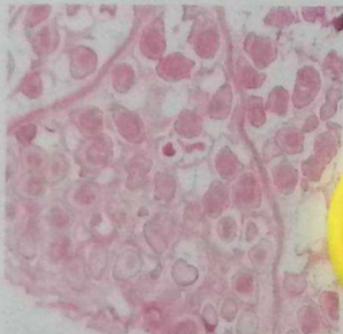


Fig. 8.45. *Ethnioncosus multilocularis* asexual hyalids in an experimentally infected vole showing a number of protozoa (120x).

strobili, which contains all the varied sexual organs and associated structures. In veterinary medicine, although it often seems as though we are dealing with a huge number of types and forms of bothria, suckers, and hook shapes (Figs. 8.44 to 8.46), the reality is that compared with the large numbers of tapeworm families with different forms of bothria occurring in a wide range of vertebrates (e.g., the

Trypanorhyncha, the Tetraphyllidea), we are really dealing only with the few forms that occur in terrestrial mammals. If one is lucky, the sections are through the head of the larva, which aids greatly in identifying the parasite beyond the simple designation of larval tapeworm based on body structure and the presence of calcareous corpuscles, but unfortunately, much of the time one has only sections through the body of the larva, and then the diagnosis, based simply on morphology, almost always remains somewhat obscure.

Because veterinary medicine until very recently focused almost exclusively on the common domestic mammals used as food and human companions, the most common tapeworm larvae seen are those of the taenid tapeworms that have a mammalian final host and a mammalian intermediate host. The typical taenid metacestode larvae are the cysticercus, the coenurus, the strobilocera, the unilocular hydatid, and the alveolar hydatid (Figs. 8.47 and 8.48). For information on hosts and site specificity, refer to the appropriate host-organ list and details in the previous chapters. If the histologic section includes only the bladder wall of the larvae, there will be little more

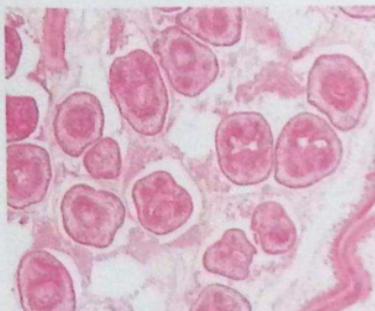


Fig. 8.46. A higher magnification of Fig. 8.45 of *Echinococcus multilocularis* with hooks evident on the scolexes (400x).

than calcareous corpuscles to identify it as cestode tissue. A section through the scolex of the larva that includes the typical claw-hammer-shaped hooks of this group identifies the specimen as a taeniid. *Taenia saginata*, the "beef tapeworm" of man, forms an exception in not having hooks in the larval nor adult stages. Often the scolex of the tapeworm is inverted in the body and will not evert until the larva is ingested by the final host.

Tentative identification of species of taeniid larvae may be based on their host and site specificity. For example, a cysticercus attached to the peritoneal membranes of a cottontail rabbit is very probably the larva of *Taenia pisiformis*, whereas a cysticercus on the peritoneal membranes of a ruminant of pig is most likely the larva of *Taenia hydatigena*. Further evidence is provided by hook length measurements if both long and short hooks happen to lie in the plane of section, or if they can be isolated from the wet tissues. *Vispaw* (1969) may be consulted for hook-length data. In the coenurus more than one scolex is connected to the same bladder wall. *Taenia crassiceps* presents a source of confusion in this regard by forming many cysticerci by budding. These all lie within the same host cysts but are not attached to a common bladder wall. Strobilocerci of *Taenia taeniiformis* are cysticerci that have precociously begun to elongate and segment as larvae and are found in the liver of rodents.

Hydatid cysts manifest expansive growth and have thick, laminated membranes separating the germinative layer, which bears sessile small scolices (termed protoscolices) or brood capsules, from the surrounding host connective tissue capsule. In "sterile hydatid cysts" (cysts without protoscolices), the laminated membrane is the only diagnostic characteristic available. Abolear hydatids have much thinner laminated membranes, and their manner of growth is invasive instead of expansive.

The remaining tapeworm larvae that are typically found in the tissues in sections are solid-bodied worm-like threads or ribbons (that can be very long) that course through the tissues or peritoneal cavities of the host. These two larvae are the tetrahyridium of the Meosentoididae (Fig. 8.47) and the plerocercoid (or sparganum) of the dipyllobothrid tapeworms (Fig. 8.48). Plerocercoids of *Sparganum* organisms (Fig. 8.49) are ribbon-like larvae that are unsegmented and undifferentiated. They have no bladder, and the scolex is not always developed, so no sections through bothria may be evident no matter how many serial sections are prepared. Calcareous corpuscles observed in a parenchymatous matrix without evidence of other structures may



Fig. 8.47. Mesocotyle tetrahyridium from the peritoneal cavity of a baboon (*Papio* sp.) (200x).



Fig. 8.48. *Sparganum maricostoides* plerocercoid from the subcutaneous tissues of a dog (100x).

be the only feature by which to identify a plerocercoid. Tetrahyridids of Meosentoidae organisms differ from plerocercoids in that they possess four suckers with no hooks, and their calcareous corpuscles tend to be large but not as dense as those of other larvae. Tetrahyrids can undergo massive asexual proliferation in the intermediate host (often seen in dogs), forming thousands of organisms that are atypical perhaps owing to the rapid multiplication, and are very difficult to identify as anything other than tapeworm tissue.

### Nematodes

Living nematodes are pseudocoelomic animals that have a fluid-filled body. The body externally is covered with a cuticle composed of collagen, and movement occurs by muscle cells in quadrants along the body wall working in opposition to the cuticle, which allows the worms to move sinusoidally. Nematodes typically have a mouth and an anus connected by a digestive tract. In sections the worms typically appear round and do have the internal organs floating within the

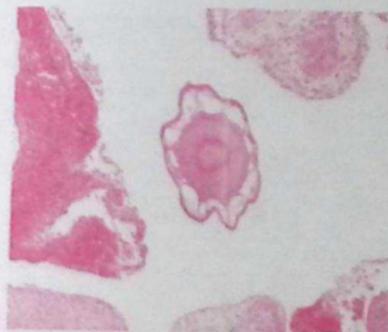


Fig. 8.48. *Strongylus vulgaris*. Cross section through *Strongylus vulgaris* showing the division of somatic musculature into dorsal and ventral fields by the lateral cords (200X).

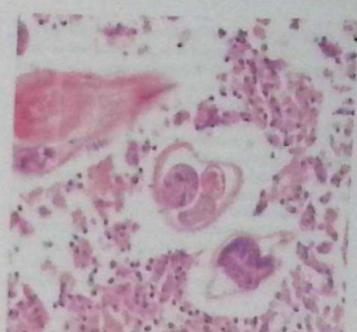


Fig. 8.50. *Trichostrongylus* sp. in the intestine of a goat. Illustrating prominent cuticular ridges (400X).

pseudocoelomic cavity. The genital primordium appears in larvae, but it tends not to grow to any extent until the worms reach the fourth larval stage. One feature of worms becoming adults after the fourth and final molt of their development is that the vulva of the female finally opens through the cuticle (becomes patent).

In sections (Fig. 8.49) the nematode is most cases is divided into two dorsolateral and two ventrolateral quadrants by the hypodermis. A syncytial layer below the cuticle secretes the cuticle. The hypodermis tends to extend into the body of the worm in dorsal, ventral, and lateral cords, which is what divides the body into its four apparent sections. The nervous system of a nematode consists of a major nervous ganglion that typically encircles the esophagus and that has major sets of fibers running through the ventral and dorsal hypodermal cords. Nematodes also have an excretory-secretory system that usually empties through an excretory pore, which is located on the ventral side of the worms near the level of the nerve ring or more anteriorly. The excretory system may have columns that extend posteriorly in the form of arms that extend into each of the lateral cords. In some adenophorean nematodes, cords may be found in addition to the typical four. Also, in the Trichinelloidea the hypodermis tends to be organized into bacillary bands rather than lateral cords, with one in *Trichuris*, two in *Trichinella*, and three or four typical in the various capillaria. From the bacillary bands, pores can be seen extending from the band through the cuticle.

The cuticle covers the external surface of the worms and, to varying degrees in different groups, the lining of the esophagus, the posterior portion of the digestive tract, the vagina, and the opening of the excretory system. The cuticle may appear layered in histologic sections, especially in forms with thicker cuticles. The cuticle may have major modifications above the lateral hypodermal cord where it forms large wings of cuticle called *ophelic alae* (when only on the head), which may or may not be continuous with lateral alae running the length of the worms. Some forms, mainly the Trichostrongyloidea, have numerous additional longitudinal ridges running the length of the body (Fig. 8.50). Adult worms are liable to have all sorts of modifications on the anterior end that are usually less apparent in the larvae; these consist of lips, large buccal capsules, teeth, and so on. The cuticle may also have spines, atriations, bosses (thickened bumps), pores (in some Adenophorea), and so on. In adult male nematodes with spicules, the



Fig. 8.51. *Esostongyloides* sp. from a great blue heron (40X). Each somatic muscle cell is composed of a basement membrane adjacent to the hypodermis, contractile muscle fibers, and a delicate sarcoplasmic portion containing the nucleus. The collagenous muscle cells have a dark-staining contractile portion extending up the lateral sides of the muscle cell, which gives the cell a cylindrical appearance, and an abundant, noncontractile cytoplasmic portion that appears to be empty with most stains.

spicules are composed of sclerotized cuticle and have a myriad of forms and shapes with some being spined.

The muscle cells of the body that provide locomotion lie along the body underneath the hypodermal layer (Fig. 8.51). These muscle cells have their long axis oriented along the length of the worm and vary in number and shape; the muscle cells of nematodes are unlike those of many other organisms because the muscles send processes to the dorsal and ventral nerve cords rather than having the nerves extending to the muscle cells. When a section has only a few muscle cells (about three to five) per quadrant, this is termed *macroxyarian*, and when there are more cells, the worms are described as *polyxyarian*. Muscle

cells are also described as to their appearance. If the cells have their contractile elements all appressed to the hypodermis with an empty cell body above them, they are termed **platymyarian**. If the cells have contractile portions that extend up the side of the cell body, they are called **coelomyarian**. Typically, cells that are platymyarian are few in number per quadrant, hence **meromyarian**, whereas coelomyarian muscle cells tend to be numerous per quadrant and **polymyarian**. The *Ascaridida* and *Spirurida* tend to have polymyarian, coelomyarian muscles; the *Rhabditida*, *Oxyurida*, and *Strongylida* tend to have meromyarian, platymyarian muscles. The *Adenophorea* are varied.

The digestive tract of the nematodes consists of an esophagus, an intestine, and a rectum (in male nematodes this is actually a cloaca, but the distinction is almost never used). Many of the characteristics of the digestive tract of adult nematodes are also present in their respective larvae stages. This can be a very useful asset in diagnosis using histologic sections.

The **esophagus** tends to be divided into a dorsal and two subventral portions by a triradial lumen that is typically lined with cuticle. There are muscles within the esophagus that pull on the lumen to open the esophagus for feeding. Within the different sections there may also be various glandular elements. The esophagus may be muscular throughout its length or may have an anterior portion that is muscular and a posterior portion that is glandular. The *Rhabditida* have a muscular esophagus typically divided into a distinct corpus, isthmus, and bulb. The *Oxyurida* have a muscular esophagus with a large valved bulb before the junction with the intestine. The *Strongylida* for the most part have a simple muscular esophagus. The *Ascaridida* have an esophagus that may have a large glandular area, the *ventriculus*, at its base, and they may also have ventricular caeca. The *Spirurida* typically have an esophagus that is muscular anteriorly and glandular posteriorly. The *Trichinelloidea* tend to have a stichosome esophagus (described later), whereas the *Dioctophymatoidea* have a muscular esophagus with many large branching glands.

The **intestine** of nematodes is fairly simple in all nematodes; it is composed of a single layer of columnar cells that have a microvillous border. In the *Strongylida*, the intestine is lined with a very few cells (**oligocytous**) that are syncytial and polynucleate, and it will often appear that only two such cells line the lumen at any given section. In the *Rhabditida*, the intestine appears lined by only two cells at each level. The *Oxyurida*, *Ascaridida*, and *Spirurida* have many cells (**polycytous**) to myriad cells (**myriocytous**) lining the intestinal lumen; these cells tend to be uninnucleate for the most part but can vary markedly in height around the lumen, especially within the *Spirurida*. In the *Adenophorea*, those we are concerned with typically have a polycytous intestine with uninucleate cells. In most of the nematodes that we will see in section, the anus is subterminal (i.e., there is a tail beyond the anus). The only group for which this is not the case is the *Adenophorea*, in which the anus is terminal.

**Rhabditida.** *Pinotaxia strongylodes* larvae are found in the hair follicles of dogs, swine, and cattle. They have double lateral alae.

*Helicophobus gingivalis* is a saprozoitic nematode that has been reported to invade mammalian tissue and disseminate to various sites, most notably the brain (Fig. 8-52), with fatal outcome. The infection has been reported widely in horses. These worms are small—adult females are 250 to 450  $\mu\text{m}$  in length by no more than about 25  $\mu\text{m}$  in diameter—and only females and larvae have been reported in tissues, suggesting that they are parthenogenetic. Distinctive features in section, in addition to the small size and location, include the presence of a rhabditoid esophagus, a single genital tube, and a thin body wall in which the cuticle, hypodermis, and muscle layers cannot be distinctly separated.

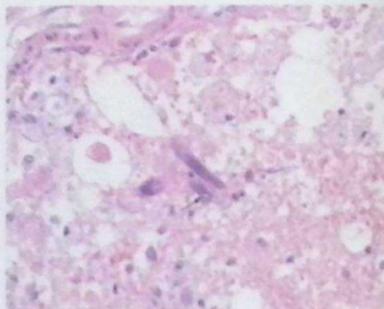


Fig. 8-52. *Helicophobus gingivalis* in brain of horse with the typical rhabditoid esophagus (arrow) (400 $\times$ ).



Fig. 8-53. *Strongyloides stercoralis* in the mucosa of the small intestine of a puppy (200 $\times$ ).

*Strongyloides* is a group of parthenogenetic species, and only female worms and larvae are found in the tissues. The adult parasitic female worms of this species are found deep in the mucosa membrane of the small intestine (Fig. 8-53) and are characterized by numerous and platymyarian muscles, a simple intestine composed of only two cells, and the eggs in utero, which are few in number, lined up in single

rows, and often with developing larvae. *Strongyloides* larvae (see Fig. 7.27) have double lateral alae.

**Strongyloidea.** There are four superfamilies: Trichostrongyloidea, Strongyloidea, Ancylostomatoidea, and Metastrongyloidea.

**Trichostrongyloidea.** The adults of this group tend to be small worms that typically inhabit the stomach or small intestine. In cross section, they are characterized by a small number of platymerian muscle cells and an intestine composed of few cells, often with prominent nuclei and a microvillous border. Most trichostrongyles, with the exception of *Trichostrongylus axei*, have marked longitudinal ridges on the surface of the cuticle (Fig. 8.56). Fourth-stage larvae are found throughout the mucosa of the stomach and intestine of ruminants and a wide range of other hosts. *Trichostrongylus axei* fourth-stage larvae and juvenile adults are found between the basement membrane and epithelial cells of the stomasal mucosa. *Ostertagia* fourth-stage larvae and juvenile adults are found in dilated gastric glands of the abomasum and produce a characteristic Moroccan leather appearance to the mucosa (Fig. 8.54).

**Strongyloidea.** Most adult strongyles inhabit the intestinal tract and are larger than the trichostrongyles. In section they exhibit characteristic features, including platymerian muscles and the typical strongyle intestine. The cuticle is not adorned with ridges. In the strongyles the presence of a large buccal capsule and specialized mouthparts is of great taxonomic value, but these features are often not seen in tissue sections.

Some of the larval stages of strongyles are spent in tissues other than the gut, whereas some form nodules in the intestinal wall. *Strongylus vulgaris*, *Strongylus edentatus*, and *Strongylus equinus* migrate extensively and sometimes erratically in the horse (Fig. 8.55) and see Fig. 9.55). *S. edentatus* tends to migrate retroperitoneally and it is characterized by a thick, multilayered cuticle. *S. equinus* immature adults are frequently found in the pancreas; sections through the buccal capsule reveal the presence of teeth at their base.

Cesophagostomum and related worms are common parasites of livestock and monkeys and have worldwide distribution. They are often referred to as nodular worms because developing larvae produce remarkable nodular abscesses in the intestinal wall of the vertebrate host during development, leading to the adult stage. Most often seen in

section as developing worms inside these nodules (Fig. 8.56 and 8.57), the larvae have a relatively thick but smooth cuticle, prominent lateral cords, and muscle cells that are platymerian and meromyarian, typically with only a small number of muscle cells per quadrant. The gut is composed of a small number of multinucleated cells with a conspicuous macrovillous (brush) border.

**Ancylostomatoidea.** The Ancylostomatoidea, typically referred to as hookworms, inhabit the gut as adults and have typical strongyle features in section. The larvae of hookworms are relatively small, usually only 14 to 16  $\mu\text{m}$  in diameter, and have double lateral alae.

**Metastrongyloidea.** Adult metastrongyles, often referred to as lungworms, typically parasitize the lungs or airways, but some may invade blood vessels or the central nervous system. In section, the body wall tends to be thin, the musculature is often polymyarian and ecdomyarian in nature, and the gut is the typical strongyle type, although



Fig. 8.55. *Strongylus vulgaris*. Cranial mesenteric artery in a horse associated with *Strongylus vulgaris* (40 $\times$ ).



Fig. 8.54. Markedly proliferative and colobated abomasum of an American bison infected with *Ostertagia ostertagi*.



Fig. 8.56. *Cesophagostomum* sp. Section through a nodule in the wall of the large intestine of a cynomolgus monkey containing multiple sections of *Cesophagostomum* larva (40 $\times$ ).



Fig. 8.57. *Cestopogonostomum* sp. Higher magnification of Fig. 8.56 showing a section through *Cestopogonostomum* larva (200x). Note the small number of platymerian muscle cells and a prominent brush border on the epithelial cells of the gut.

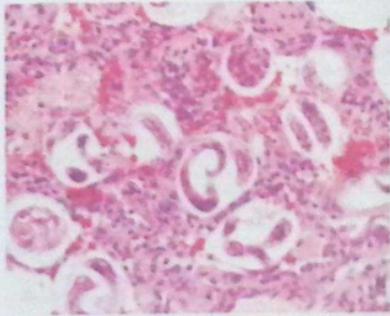


Fig. 8.58. A higher magnification of Fig. 8.57 with *Aelurostrongylus abstrusus* eggs and larvae in the lung of an infected cat (400x).

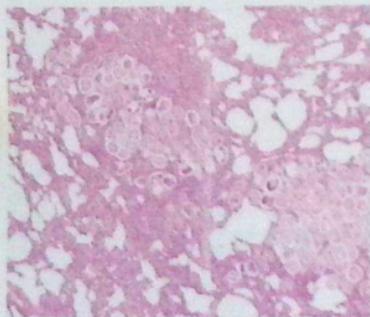


Fig. 8.58. *Aelurostrongylus abstrusus* larvae and eggs in section of a nodule in the lung of a cat (100x).



Fig. 8.60. *Angiostrongylus vasorum* in the pulmonary artery of a dog (100x).

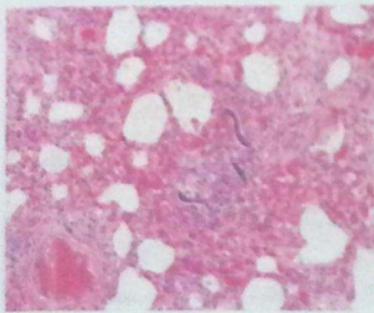


Fig. 8.61. Larvae of *Angiostrongylus vasorum* in the lung of an infected dog (400x).

the microvilli are less prominent than in other strongyles. Many metastrongyles contain embryonated eggs or larvae in utero and shed these stages into the surrounding tissues.

Cats are typically host to only a single lungworm, *Aelurostrongylus abstrusus*. Adults, eggs in varying stages of development, and larvae are found in nests in the lung parenchyma (Fig. 8.57 and 8.58). Diagnosis is usually fairly easy because domestic cats have few other worms causing similar lesions; however, wild felids may be host to related forms.

Dogs can be infected with several lungworms, but they tend to live in markedly different locations, making diagnosis easier than it would be otherwise. *Angiostrongylus vasorum* adults may be found in the right side of the heart and in pulmonary vessels of dogs, whereas the eggs and larvae are found in the lung parenchyma. This infection was exotic to North America but has now appeared in the far east of Canada and in some regions of the United States (Fig. 8.60 and 8.61). *Filaroides hirthi* adults are found in the lung parenchyma of the dog.

Eggs contain first-stage larvae when laid, and the eggs do not accumulate in lung tissue. Autoinfection by *F. hepatis* may lead to a state of hyperinfection in which lung tissue is almost completely replaced by adult worms, and larvae may be found widely scattered in lymph nodes, pancreas, intestinal tract, liver, and brain. *Ostertagia circumcincta* adults are found in fibrous nodules projecting into the lumen of the trachea and principal bronchi.

Sheep and goats can be host to several species of lungworms. *Mastomys capillaris* is found in nodules in the lung parenchyma. These nodules contain adult worms, eggs in varying stages of development, and larvae. If the tails of larvae can be located in the tissue section, *Mastomys* organisms can be distinguished from *Protostrongylus* organisms (see Fig. 7.65). *Protostrongylus* spp. adults may be found in either parenchymal nodules or airways. *Dicrocoelium* spp. (*Trichostrongylidae*) adults are found in airways. *Paraphosphocorynus tenuis* adults are found in the meninges and nervous tissue of the spinal cord and brain of sheep and goats (Figs. 8.62 and 8.63) but their eggs and larvae, which are indistinguishable from those of *Mastomys* organisms, are found

widely scattered in the lung parenchyma rather than concentrated in nests.

**Oxyurids.** The oxyurids are generally smallish worms that as adults typically inhabit the large intestine or cecum. In section most species have prominent lateral alae. The esophagus has characteristic sections consisting of corpus, isthmus, and terminal bulb, which can occasionally be seen in sections. The musculature is platymerarian and mesenteric, and generally only two or three muscle cells are located in each quadrant. The intestine is variable but is cuboidal to columnar with a single nucleus per cell. The presence of typical eggs in other sites assists with identification.

**Ascaridida.** The ascarids comprise a diverse group of worms, and as adults, some, such as *Ascaris* and *Purpura* spp., are the largest of the internal nematodes. In tissue section, in addition to their large size, the ascarids characteristically have a thick, multilayered cuticle, polymyarian-coelomyarian muscles (often with cytoplasmic processes that extend into the body cavity), an intestine with numerous columnar epithelial cells and short macrovilli, and large lateral cords (Figs. 8.64 to 8.66). The Ascaridida are often divided into two large groups or superfamilies. One, the Ascaridoidea, parasitize land-dwelling vertebrates, whereas the second group, the Heterocheiloidae, parasitize birds, fish, and marine mammals. Members of the Ascaridoidea, including the genera *Ascaris*, *Purpura*, *Trichostrongylus*, and *Baylisascaris*, have three simple lips on the anterior end, a thick, multi-layered cuticle, a club-shaped esophagus, columnar epithelial gut cells with a single nucleus near the base of each cell, prominent coelomyarian-polymyarian muscle, and typical eggs in the uterus that have a thick shell often wrinkled or sculptured on the surface. Genera in Heterocheiloidae, such as *Aspicaris*, *Terranova*, *Comptosia*, and *Pseudascaris*, have much the same features in section, except that all in the group also have a cecum (anteriorly directed), a ventricular appendix (posteriorly directed), or both. Those may be obvious if sections are cut through the level of the esophageal-intestinal junction.

Those ascarids that parasitize mammals often have larvae that are capable of tissue migration, and larvae of genera such as *Trichostrongylus*, *Baylisascaris* (Fig. 8.68), and *Lagebilia* (see "larval



Fig. 8.62. *Paraphosphocorynus tenuis* in the spinal cord of an alpaca (200x).

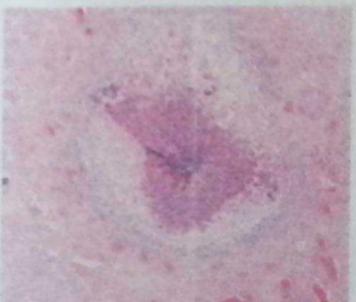


Fig. 8.63. Degenerating *Paraphosphocorynus tenuis* parasite with remnant cuticle (arrow) surrounded by an intense granuloma in the spinal cord of an infected sheep (100x).

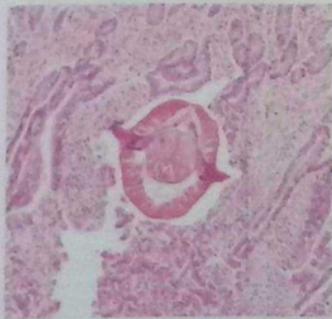


Fig. 8.64. *Trichostrongylus axei* adult nematode in the small intestine of a dog revealing the prominent lateral alae and coelomyarian musculature (200x).

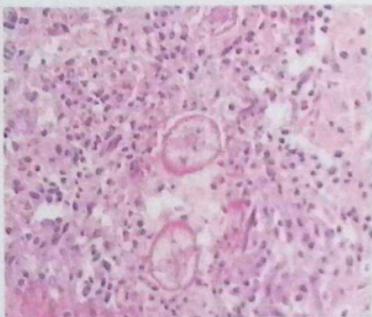


Fig. 8.65. *Baylisascaris procyonis* in the brain of a woodchuck showing the large excretory columns, the intestina with a patent lumen, and lateral alveolus (400x).



Fig. 8.67. *Spirivora* eggs embedded in a subcutaneous histiocytoma in a dog (H&E). The lateral cords projecting into the postcuticle and the intestine with a prominent brush border are noted.

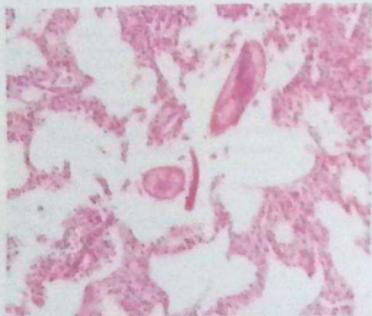


Fig. 8.66. Larval *Ascaris suum* in the lung of an infected piglet (100x). Lateral alveoli are evident.

migrans" syndrome. Ascarid larvae have single lateral cuticular alveoli. They also have a single excretory cell with H-shaped anterior and posterior projections called excretory columns. The presence of single lateral alveoli and paired excretory columns makes ascarid larvae relatively easy to distinguish in tissue sections (Fig. 8.65). *Toxocara* larvae migrating or arrested in somatic tissues tend not to exceed 21  $\mu$ m in diameter, but *Baylisascaris* larvae continue to grow as they migrate and may reach 55 to 70  $\mu$ m in diameter.

**Spirivora.** The order Spirivora consists of the superfamilies Gnathostomatoidae, Physalopteroidae, Rictularioidae, Theziaioidae, Spirivoridae, Dracunculoidae, and Filarioidae. The spirivora represent an extremely diverse group of nematodes that parasitize a wide range of hosts and anatomic locations in those hosts. As adults, spirivora range in size from thin and thread-like in the case of *Gongylonema*, to stout, robust worms such as *Gnathostoma*, to incredibly long worms in the

case of *Dracunculus*. Some species localize in the lumen of the gut, others are associated with the wall of the gut, and others have moved away from the gut entirely. Despite this variability, a number of similarities in both biologic and morphologic aspects have been noted. As a group, the spirivora use insects as intermediate hosts. In many species, small, thick-shelled eggs containing a well-developed larva are passed in the feces and are ingested by an insect intermediate host. In the *Dracunculoidae*, female worms migrate to the surface and release first-stage larvae into water, where they are ingested by copepods. In the *Filarioidae*, not only have the adult worms moved away from the gut, but the female worms release minute larvae called microfilariae that may circulate in the blood or reside in the skin and are picked up by bloodsucking insects that serve as intermediate hosts. Features of spirivora in tissues include a cuticle that often has some ornamentation, including spines, bosses, transverse striations, or longitudinal ridges. The esophagus tends to be long and divided into anterior muscular and posterior glandular portions; the glandular portion is very cellular and stains much more intensely. The general spirivora intestine is often large and folded on itself and is composed of many cells, often with the nuclei arranged in a row, a prominent brush border, but a rather weak basement membrane. The lateral cords are prominent, and the musculature is polymyarian-coelomyarian in nature. In most spirivora female worms contain small, thick-shelled eggs containing a larva. In the case of the *Dracunculoidae* and *Filarioidae*, large numbers of larvae or microfilariae, respectively, are contained in utero. This combination of features makes the spirivora relatively distinctive in sections.

Spirivora larvae are, on occasion, seen in tissue sections and have some of the same morphologic features seen in adult worms, including polymyarian-coelomyarian muscle cells, prominent lateral cords, and a distinctive intestine composed of many tall columnar cells.

*Spirivora lapi* (Fig. 8.67) provides an example of the superfamily Spirivoridae. The adults typically are found in nodules in the wall of the esophagus and stomach, and sometimes in the wall of the aorta or rectum. In cross section they are characterized by large lateral cords that project into the body cavity; an intensely stained glandular esophagus; an intestine with a prominent brush border and many cells with the nuclei lined up in a row, which gives the appearance of three layers; a uterus filled with small eggs containing intensely stained larvae; and coelomyarian-polymyarian muscle cells (Fig. 8.67). The larvae have

books and combs associated with the stoma, although these structures require oil immersion microscopy to be seen properly. *Tetrameres* sp. are commonly found in the proventricular glands of various bird species. These parasites have a characteristic, deeply eosinophilic fluid that fills the pseudocoelom (Figs. 8.80 and 8.81).

The genus *Gorgysinema*, another member of the Spiruroidea group, is encountered in the tissues of animals with some frequency and has several distinctive morphologic features. Typically found threaded in the mucosa of the mouth, tongue, esophagus (Fig. 8.79), or stomach, the members of *Gorgysinema* have characteristic spiraled features in section, including a divided esophagus, a polymyarian-coelomyarian musculature, and the presence of small, thick-shelled, embryonated eggs. *Gorgysinema* organisms are distinctive, however, in that the anterior end has large cervical alae and is covered with cuticular plaques or bosses on the anterior end, and the lateral cords are asymmetric.

*Dicranocaulis insignis*, of the superfamily *Dicranocaulidae*, is characterized by flat lateral cords separating semilunar dorsal and ventral



Fig. 8.79. *Gorgysinema pulchrum* in the tongue of a cow (100x).



Fig. 8.80. *Tetrameres* sp. in proventricular glands of a pigeon (200x). Note the numerous spiraled eggs and the eosinophilic fluid in the pseudocoelom.

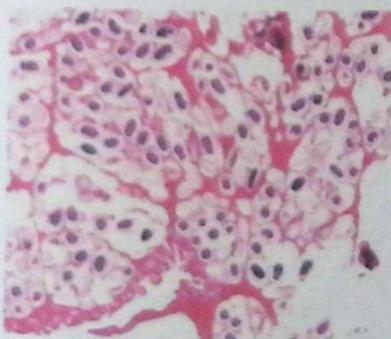


Fig. 8.81. Higher magnification of Fig. 8.80 highlighting the typical spiraled eggs (400x).

muscle fields composed of coelomyarian-polimyarian muscle, a very retracted intestine, and a large uterus filled with larvae.

Members of the superfamily *Filarioidea*, although they have many typical spiraled features in section, are relatively distinct. Most distinctive is their location, as adults, in virtually all tissues except the gut. *Filaria* range greatly in size, from some that are only 1 or 2 cm in length to others, such as *Dirofilaria immitis*, in which the female worm may reach 30 cm in length by 1 mm in diameter; however, all tend to be slender. The cuticle may be thin or thick and, in some groups, contains distinctive ridges or striations. The musculature is coelomyarian-polimyarian, the esophagus may be divided but is generally not as prominent as in other spirurids, and the intestine is typically a simple tube. One of the most characteristic features of filariae is the presence of microfilariae filling the uterus. Many species of filaria infect animals, and several examples will serve to illustrate the group.

*D. immitis*, the dog heartworm, is well recognized for the disease it produces in canines, felines, and humans (Fig. 8.77). The adult worms live in the circulatory system, typically in the chambers and great vessels of the heart. The worms, as just stated, are large; have a thick, multilayered but smooth cuticle; have prominent coelomyarian-polimyarian muscles; have broad lateral cords; have a weak intestine; and, in the female, have paired uteri filled with microfilariae (Fig. 8.77).

Many other *Dirofilaria*, such as *Dirofilaria repens* of the dog and *Dirofilaria tenuis* of the raccoon, live in subcutaneous locations and are distinctive in that the cuticle has prominent longitudinal ridges marked with transverse striations, giving the external surface a beaded or crenate appearance.

The genus *Onchocerca*, another common filarial infection of domestic animals, provides a good example of species' filarial anatomy in section. Adult female *Onchocerca* organisms are thin and extremely long and have distinctive cuticular structures. These worms possess external circular ridges and striae in the inner layer of the cuticle (Fig. 8.72). Not only are these ridges and striae specific to the genus *Onchocerca*, but the number of striae per ridge has been shown to have great value in distinguishing various species within the genus. Also distinctive of adult female *Onchocerca* organisms are the muscle cells, which often appear to be weak and poorly developed, and a prominent amount of hypodermal tissue, even underlying the muscle cells. As far as it is known, adult *Onchocerca* organisms inhabit dense connective tissues, are tightly coiled, and, in some species, form distinct fibrous nodules.



Fig. 8.71. *Dirofilaria immitis* in the pulmonary artery of a dog.

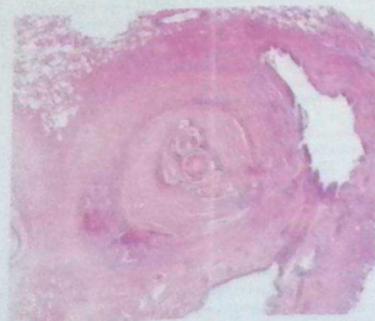


Fig. 8.72. *Dirofilaria immitis* in the pulmonary artery of a dog (20x). The thick, smooth cuticle and large coelomycytoplasmic muscles are evident at this magnification.

#### Enoplida

**Trichinelloidea.** This group contains the trichinelloids, the trichuroids, the capillarids, and the trichosomoides. In this group the most characteristic feature, both grossly and in section, is the **stichosome** esophagus, a small cylindrical tube surrounded by individual **stichocytes** that compose the stichosome. The other distinctive feature of



Fig. 8.73. *Dirofilaria immitis* in the pulmonary artery of a dog (200x). The small intestine and paired uteri containing microfilariae are evident.

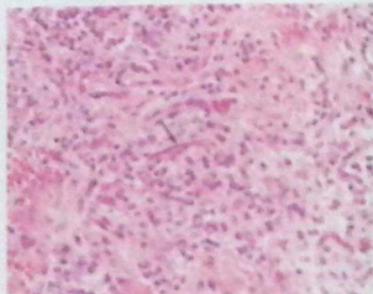


Fig. 8.74. *Dirofilaria immitis* microfilariae lodged in interstitial capillaries in the lung of an infected cat (400x).

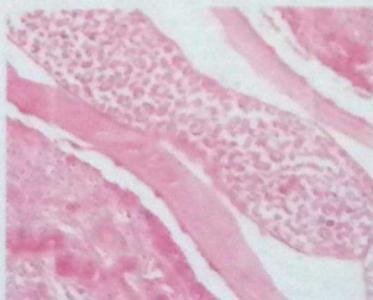


Fig. 8.75. *Onchocerca cervicalis* female in the nuchal ligament of an ox (200x). The outer circular cuticular ridges and striae in the inner layer of the cuticle are evident.

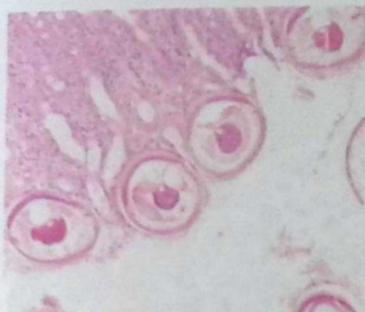


Fig. 8.76. *Trichouris vulpis* in the esophagus of a dog showing a cross section of the esophageal region with the prominent basophilic stichosome and the bacillary band (200x).



Fig. 8.77. *Trichinella spiralis* first-stage larva in a skeletal muscle fiber of a cat (200x).

these worms in section is the presence of a bacillary band(s). The bacillary band is a specialized section of the cuticle and hypodermis that includes specialized hypodermal gland cells. In *Trichouris* there is a single bacillary band in the esophageal region (Fig. 8.76) whereas in *Trichinella* and capillarids, two bacillary bands run the length of the esophagus. In addition, the female reproductive tract is a single tube, the anus is usually terminal, the muscles are colorless/anisopolymyarian, and the eggs typically have bipolar prominences (plugs) and are frequently in an unembryonated state when passed or seen in tissues. Occasionally, eggs may develop and hatch in vitro, as in the case of *Trichinella*. The first-stage larva is typically the infective stage for the definitive host. Most worms in this group display a high order of site specificity and, except for *Trichinella*, a high order of host specificity as well. The host-organ listings should prove helpful in dealing with this group of parasites.

Adult *Trichouris*, as their common name whipworm suggests, have a whip-shaped body. The thin "whiplash" anterior portion is threaded through the epithelium of the large intestine, whereas the stout "handle" portion normally lies free in the lumen. Immature *Trichouris* lie entirely within the mucosa and are of uniform diameter.

Adult *Trichinella* are found threaded in the mucosa of the small intestine, and in tissue section the adults resemble Strongyloides, except that they have a tubular esophagus embedded in the stichosome, male worms exist, and in female worms the uterus contains prelarvae instead of segmenting eggs. *Trichinella* larvae are found characteristically coiled in a "nurse cell" (Fig. 8.77) in striated muscle, and they are characterized by stichocytes surrounding the esophagus. Capillarids infecting the intestinal mucosa are somewhat larger than *Trichinella* and have eggs with bipolar plugs in their ova.

The presence of single-cell eggs with bipolar plugs in the uterus is the best criterion for identifying capillarids in tissue sections (Fig. 8.78). *Trichouris* spp. have larger eggs and are found only in the large intestine of mammals, practically the only epithelium in which capillarids will not be found.

Other common but less frequently seen members of this group include *Anatrichosoma* in the nasal mucosa or palate of primates and marsupials and *Trichonosis* in the bladder of rats. Both have larvated eggs with bipolar plugs and two or one bacillary band(s), respectively. *Anatrichosoma*, although occurring in the same general

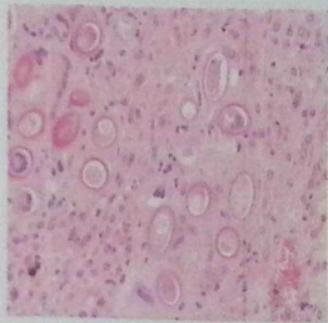


Fig. 8.78. *Capillidum hepaticum* in the liver of a nonhuman primate (200x). Eggs with bipolar plugs are visible in the tissue.

location (i.e., mouth and throat) as *Gongylosoma*, can easily be distinguished on morphologic features, including smaller diameter, presence of stichosomes and bacillary bands in the anterior end, and polar plugs in the eggs.

#### Acanthocephalans

Adult male and female Acanthocephala are pseudocoelomates that live in the intestine of vertebrates, where they gain nutrients through their external covering (i.e., they have no intestinal tract). Hosts include all vertebrate classes, fish, amphibians, reptiles, birds, and mammals. Eggs passed in feces are ingested by the intermediate host, an arthropod, and infection is acquired through ingestion of the intermediate host. The worm-like adults possess a spiny proboscis that is used for attachment to the intestinal mucosa and can be retracted inside the body; this is why they are often called *thorax*- or *spiny-headed* worms (Fig. 8.79). The fluid-filled pseudocoelom contains cells of the reproductive system, testes, and cement glands in males. Females have a reproductive system wherein balls of ovaries (ovary flut about and sperm migrate into the pseudocoelom to fertilize the eggs.



Fig. 8.78. Adult *Prosthenechis* sp. in the caecum of a common marmoset.



Fig. 8.80. Cross section through *Prosthenechis* sp. in a common marmoset revealing the two, thick layers of muscle (M2).

A "uterine bell" sorts the eggs according to their developmental stage, and mature eggs, containing a larva called an *acanthur*, pass into the uterus, out of the body, and into the feces. The intermediate host is typically an arthropod in which a stage called a cystacanth develops; the cystacanth can sometimes use vertebrate paratenic hosts, and this is typically the stage that will be seen in histologic sections.

The body wall is thick and multilayered and is very distinctive in histologic sections. There is an outer tegument (outer plasma membrane and three fibrous layers that contain lacunae [channels] that may serve as a means of moving nutrients around the body), a thin "dermis" layer, and a layer of circular and longitudinal muscle tubules that are highly distinctive (Fig. 8.80). In the cystacanth there are no reproductive organs, but there are two lemnisci—muscular and glandular structures that serve to evert and retract the thorny proboscis. The thick hypodermis lying external to the muscle layer provides the major clue as to the identity of a cystacanth.

## REFERENCES

- Benford, C. H., & Connor, D. H., (Eds.). (1976). *Pathology of tropical and extraordinary diseases*, vol. 1 and 2. Washington, DC: Armed Forces Institute of Pathology (AFIP).
- Chatwood, M. B., & Lathamelis, J. R. (1972). Identification of parasite reservoirs in tissue sections. *Experimental Parasitology* 31, 407-619.
- Connor, D. H., Charlton, E. W., Schwartz, D. A., et al. (1987). *Pathology of infectious diseases*, vol. 1 and 2. Stamford, Conn: Appleton & Lange.
- Dubey, J. P., Sankaranar, C., Duncan, T., et al. (2005). *Bioherpetology of Ruminant Lemnisci (Protozoa: Apicomplexa) from the donkey (Equus asinus)*. *International Journal for Parasitology* 35, 678.
- Goodman, C. H., Fern, R., & Daley, J. P. (1983). *An atlas of protozoan parasites in animal tissues*. Washington, DC: AFIP American Registry of Pathology.
- Gutierrez, V. (1995). *Diagnostic pathology of parasite infections with clinical correlation*. Philadelphia, Pa: Lea & Febiger.
- Meyer, W. N., Steele, B. C., Marty, A. M., & Witt, D. J. (2000). *Pathology of infectious diseases*, vol. 1. Philadelphia, Washington, DC: AFIP American Registry of Pathology.
- Oxley, T. C., & Adl, L. R. (1995). *Parasites in human tissue*. Chicago: American Society of Clinical Pathology (ASCP).
- Strick, F., Thee, J. G., & Herbrand, M. L. (2012). Parasitic diseases of nonhuman primates. In C. R. Allen, K. Mansfield, S. D. Yowell, & T. Morris (Eds.), *Nonhuman primates in biomedical research: diseases*, vol. 2. San Diego: Academic Press.
- Viviers, A. (1965). A taxonomic revision of the genus *Thelazia* Linnæus, 1758. *Onderstepoort Journal of Veterinary Research*, 36, 3.

**Abstract:** The microscopic identification of parasites in tissue sections is an interesting challenge. Often a diagnosis is provided with a single slide that shows only pieces of the parasite. In an attempt to identify an object believed to be a parasite, one should gather as much information about the patient as possible, including life history and clinical signs. It is also important to be familiar with the kinds of parasites most likely to be found in the particular host and tissue under study, as well as in the specific geographic area. The host-organ listing of parasites in the preceding chapter should be considered as a

checklist of possibilities. The main objective of this section is to emphasize some of the major microscopic anatomic features of parasites that can be helpful in their identification in histologic sections. For arthropods and metazoan parasites, several defining characteristics can be listed for each group of parasites, but the presence or absence of a body cavity and digestive tract and the type and distribution of muscle fibers are important criteria to be considered in making an initial placement into a major group.

## Antiparasitic Vaccines

Marshall W. Lightowers

Vaccines are biological preparations that improve immunity to disease. The terms vaccination and immunization are used interchangeably. Most vaccines incorporate a source of antigen that is specific to a particular pathogen. Before the discoveries of Edward Jenner and Louis Pasteur, it had been recognized that for some diseases, those people or animals that recovered were immune to reinfection. This led to practices whereby individuals were deliberately exposed to virulent pathogens to induce subsequent immunity—at least in those that survived the vaccination! Examples include variolation for smallpox and leishmanization for cutaneous leishmaniasis in humans. Later the practice was used to prevent tick fever in cattle and is used even now, either as a carefully controlled exposure to virulent parasites for prevention of coccidiosis in chickens, or together with chemotherapy for poultry coccidiosis and also for East Coast fever in cattle in parts of Africa. The term “vaccine” was coined by Louis Pasteur in honor of Edward Jenner’s discovery of the protective efficacy of cowpox infections against smallpox (Lewin and Logan, 1997). The potential for vaccines to transform man’s ability to overcome some diseases was recognized from the earliest times and was embodied in Thomas Jefferson’s 1806 letter to Edward Jenner in which he wrote “Future nations will know by history only that the loathsome smallpox has existed” (Jefferson, 1987). Vaccination has also transformed disease control for many animal diseases, playing a pivotal role in eradication of the first veterinary disease, rinderpest, declared in June 2011.

An advance on the use of virulent organisms as vaccines has been the use of attenuated pathogens; notable examples include the Sabin vaccine for polio in humans, and several vaccines of this type are in current commercial use for diseases of veterinary importance, including some parasitic diseases. Live vaccines have advantages in that the immunity they stimulate is generally strong, however, they have disadvantages associated with aspects such as the vaccine strain reverting to virulence, possibility for inadvertent transmission of contaminant microorganisms, particularly viruses, and the need to maintain a cold chain to maintain the vaccine’s infectivity. For safety and quality control reasons, there is a preference for vaccines to contain nonliving antigens, preferably defined antigens.

As a general rule, eukaryotic parasites do not stimulate a high level of immunity, and persistent infections and reinfections are the norm. In comparison with bacterial and viral pathogens, vaccination has proved to be ineffective against the majority of parasitic diseases. The past decade has been characterized by an “omics” revolution. Whether this will represent a “new dawn” in development of practical outcomes, as some have claimed, remains to be seen; as yet there are no antiparasitic vaccines that could be construed as having had their origins in “omics.” Although the development of vaccines against eukaryotic parasites has lagged well behind progress with bacterial and viral pathogens, there are notable exceptions among the parasites,

particularly in which the existence of naturally acquired immunity has favored successful vaccine development.

The vaccines dealt with here are those that are currently registered commercial vaccines. A number of commercial antiparasitic vaccines were produced in the past but are no longer marketed, such as Imvax-Sabrey Laboratories’ Canine Hookworm Vaccine, marketed during the 1970s. Several antiparasitic vaccines have entered, then left the marketplace, over the past decade. These vaccines are not considered here. A summary of commercial antiparasitic vaccines is provided in Table 8.1. There has never been a commercial antiparasitic vaccine for use in humans.

### PROTOZOAL INFECTIONS

#### Coccidiosis Vaccines

Some of the earliest commercial antiparasitic vaccines to be developed were against coccidiosis in chickens. Development of the vaccines was made possible following the realization that an initial exposure of chickens to *Eimeria* spp. parasites stimulated a high level of resistance to challenge infections (Beale and Clark, 1943; Torres, 2002), and this could be achieved without causing serious pathology or death through the initial administration of low numbers of oocysts (Oshonowo, 1997; Johnson, 1997). In practice, one of the most challenging aspects of applying this knowledge to the development of commercial vaccines was in establishing procedures to reliably deliver oocysts in the birds en masse without a high risk of inducing clinical disease (see reviews by Shirley et al. 2007b; Williams, 2002).

The earliest commercial use of vaccines based on live wild-type parasites incorporated the concomitant application of chemotherapeutic agents to prevent clinical disease in some birds, and the practice continues in some circumstances to the present day. Safe live vaccines incorporating attenuated parasites followed. Effective vaccines have been developed for coccidiosis in both chickens and turkeys. Many of the commercially available vaccines for coccidiosis are summarized in Table 8.1. Anticoccidial vaccination is a complex topic, in which different vaccine formulations are available comprising different combinations of *Eimeria* spp. for application in particular target birds (broilers, layers, broilers), and for different target species (chickens and turkeys). More extensive discussions concerning the various vaccine formulations can be found in Williams (2002a, 2002b), Shirley et al. (2005), and Ahmad et al. (2016). A single, commercial vaccine against coccidiosis in chickens incorporating nonliving antigens was developed and marketed by Abic in Israel from 2002, however, it has since been discontinued.

A variety of technologies have been developed for delivering live coccidiosis vaccines, including administration in drinking water, spray on feed or within an edible gel, intraocular, intrayolk sac, and in ovo. Consideration of these various methodologies can be found in Williams (2002a).

TABLE 9.1 Commercial Antiparasite Vaccines

Parasite	Vaccine Recipient	Registered Name	Company <sup>a</sup>	Antigen Type
<b>Antiprotozoal</b>				
<i>Eimeria</i> spp.	Chicken	Numerous	Numerous	Live, nonattenuated
<i>Eimeria</i> spp.	Chicken	Numerous	Numerous	Live attenuated
<i>Eimeria</i> spp.	Turkey	CocciVac T, Immunovac T	MSD, Vetech Laboratories	Live, nonattenuated
<i>Toxoplasma gondii</i>	Sheep	Toxovac	MSD	Live attenuated
<i>Besnoitia besnoiti</i>	Cattle	Not indicated	Local	Live, nonpathogenic
<i>Babesia bovis</i>	Cattle	Several	Local	Live attenuated
<i>B. biguttata</i>	Dog	Prostag	Boehringer Ingelheim	Subunit
<i>Theileria parva</i>	Cattle	Not indicated	Local	Live, nonattenuated
<i>Theileria annulata</i>	Cattle	Not indicated	Local	Live attenuated
<i>Leishmania donovani</i>	Dog	Leish-Tec	Herpax Calix	Recombinant subunit
<i>Leishmania infantum</i>	Dog	Can-Leish	Vetvac	Subunit
<i>Leishmania infantum</i>	Dog	Leifland	Labortonus (ETI) and MSD	Recombinant subunit
<i>Bertrichomonas testis</i>	Cattle	Trichivac	Boehringer Ingelheim	Inactivated parasites
<i>Trichostrongylus axei</i>	Cattle	Trivox	Laboratoire Biologique de Tardif	Inactivated parasites
<b>Antihelminth</b>				
<i>Dicrocoelium viverrinum</i>	Cattle	Bovilis Hyliver, Dicotar	MSD	Live attenuated
<i>Haemonchus contortus</i>	Sheep	Berberox	Wormax Australia	Subunit
<i>Fasciola circum granulosa</i>	Sheep/cattle	EDS	Changsheng Aukin Biological Technology	Recombinant subunit
<i>Taenia mulleri</i>	Pig	Genvac	Indian Immunologicals Ltd.	Recombinant subunit
<b>Antitick</b>				
<i>Rhipicephalus microplus</i>	Cattle	Genvac	IBBC Cuba	Recombinant subunit
<i>R. microplus</i>	Cattle	Tek-Vac MR Go Tick	Tecogenics SA/ Linar de Colombia	Subunit
<b>Antibiosis</b>				
<i>Caligus rogerscraffi</i>	Fish	Poissonian Aquario-Sas Licé	Tecovox	Synthetic peptides

<sup>a</sup>Marketing and availability of these vaccines are subject to commercial decisions at any time, and hence the list cannot be considered to be accurate or comprehensive on an ongoing basis. These vaccines were being commercially marketed to some extent around the time of writing or in the recent past.

<sup>b</sup>Marketing arrangements may lead to vaccines being sold under license in particular regions, hence the company specified may not be responsible for marketing the indicated vaccine in some areas.

<sup>c</sup>Up to eight different species of *Eimeria* may be included in a combined vaccine. Vaccines may be marketed in a number of different variants containing different combinations of different species' oocysts.

<sup>d</sup>A number of different companies or institutes involved with manufacture and marketing.

<sup>e</sup>Vaccines prepared using similar methodologies in a number of countries.

TABLE 9.2 Coccidiosis Vaccines for Chickens

Vaccine (Manufacturer)	Parasite Species	Vaccine Type	Bird Type	Administration	First Registered
ADVIN <sup>a</sup> (Pharapharm)	<i>Eac, Ema, Eto</i>	WT	Broilers	Hatchery spray, water, or feed	1992 (USA)
CocciVac-B (MSD Animal Health)	<i>Eac, Ema, Ema, Eto</i>	WT	Broilers	Hatchery spray or feed	1982 (USA)
CocciVac-C (MSD Animal Health)	<i>Eac, Eto, Eto, Ema, Ema, Ema, Eto, Eto</i>	WT	Broilers/layers	Oral, hatchery spray, or feed	1989 (USA)
CocciVac-BB2 (MSD Animal Health)	<i>Eac, EmaX2, EmaX, Eto</i>	WT, A (P)	Broilers	Hatchery spray or feed	2011 (USA)
CocciVac-DD (MSD Animal Health)	<i>Eac, Eto, Ema, Ema, Ema, Eto</i>	WT	Broilers/layers	Spray or feed spray	2012 (USA)

