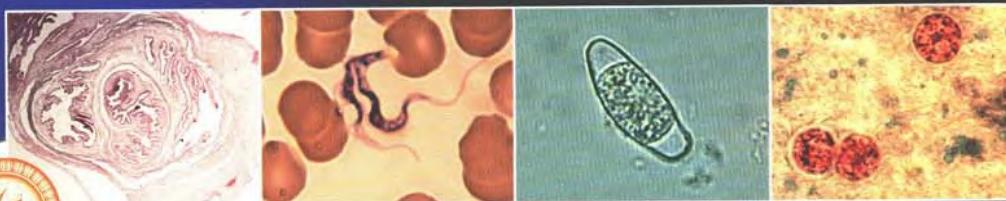
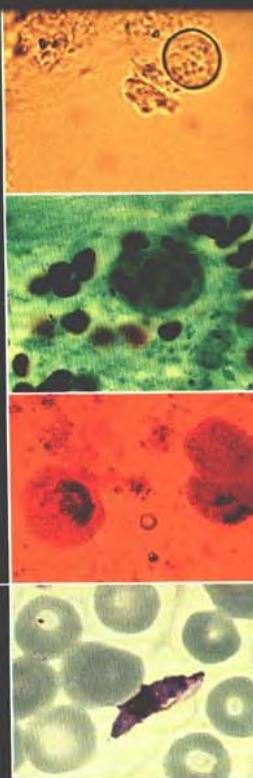
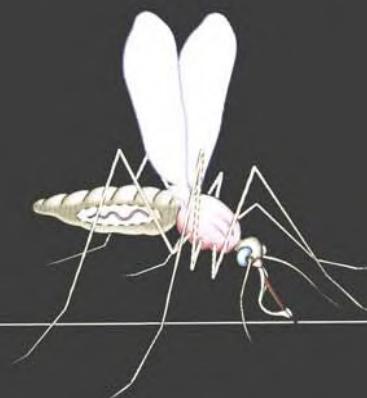


Fourth edition

Atlas of

Medical  
Helminthology  
and Protozoology



P.L. Chiodini  
A.H. Moody  
D.W. Manser

Atlas of

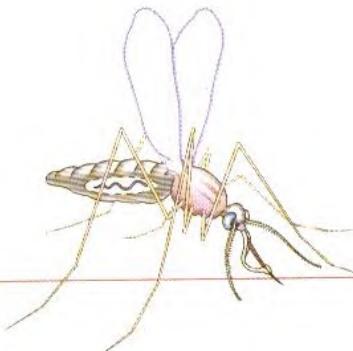
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and Protozoology



*Commissioning Editor:* Timothy Horne  
*Project Development Manager:* Jim Killgore  
*Designer:* Sarah Russell  
*Project Manager:* Nancy Arnott

Fourth Edition

Atlas of  
Medical  
Helminthology  
and Protozoology



Peter L. Chiodini BSc PhD FRCP FRCPath

Consultant Parasitologist, Department of Clinical Parasitology, The Hospital for Tropical Diseases,  
Honorary Professor, The London School of Hygiene and Tropical Medicine, London, UK

Anthony H. Moody MPhil MIBiol C.Biol FIBMS

Laboratory Manager, Department of Clinical Parasitology, The Hospital for Tropical Diseases, London, UK

David W. Manser BSc MSc FIBMS

Biomedical Scientist, Department of Clinical Parasitology, The Hospital for Tropical Diseases, London, UK

Illustrated by Robert Britton



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**Note**

Medical knowledge is constantly changing. As new information becomes available, changes in treatment, procedures, equipment and the use of drugs become necessary. The authors and the publishers have, as far as it is possible, taken care to ensure that the information given in this text is accurate and up to date. However, readers are strongly advised to confirm that the information, especially with regard to drug usage, complies with the latest legislation and standards of practice.

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# Preface

Since this atlas was first published, major advances in immunology and molecular biology have transformed our understanding of the parasitic diseases which affect humans. The programme to eradicate Guinea worm is well advanced and real progress is being made towards a malaria vaccine. However, none of the parasites described in the first edition have yet been consigned to history. Indeed, *Cyclospora* and the microsporidia are newly recognised as important human pathogens even since the third edition, and in some geographical areas the malaria situation is worse, with the spread of multi-drug resistant *Plasmodium falciparum* malaria. There is a great deal left to be done.

Effective action against parasitic disease requires a team approach, including epidemiologists, biologists, diagnostic

laboratory workers and clinicians. Common to all these disciplines is a need to understand the life cycles and morphology of the organisms they confront. It is hoped that this edition of the atlas will provide an appropriate introduction. The strong emphasis on diagnosis has been retained and since diagnostic parasitology still relies heavily on morphology, we have strengthened this area with the introduction of colour illustrations and photomicrographs.

We hope this book will help to kindle enthusiasm for the effort to control these parasites and the diseases they cause.

London

2001

P. L. C.

A. H. M.

D.W. M.

# Acknowledgement

This atlas first originated from the Royal Army Medical College, London. The late Major-General HC Jeffrey and the late RM Leach wrote the first two editions. Colonel, later

Major-General, GO Cowan undertook revision for the third edition and an abridged version of his introduction is included in this latest edition.

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# Introduction

*The protozoon and the helminth, as regards tropical pathology,  
are in the ascendant.*

Sir Patrick Manson (1899)

Parasites to the Ancient Greeks were those who sat at another's table and paid for their meal with flattery. In biology, a parasite is an animal or plant living in or on another (the host) and drawing nourishment from it. This definition could include viruses, bacteria and fungi as well as protozoa and helminths, but historically the first group has been studied in microbiology, the second in parasitology. In tropical diseases, Manson's dictum remains valid today.

**Protozoa** are small, unicellular organisms, which contain a nucleus and functional organelles. They reproduce quickly and asexually in the host, but may have a sexual phase of their life cycle in another host or vector.

**Helminths (worms)**, which are metazoa, are larger, multicellular organisms, normally visible to the naked eye in their adult form. They reproduce sexually, usually within the host, and have pre-adult stages (ova, larvae) which live externally or in other hosts.

**Transmission** of parasites requires:

- a source or reservoir which may be human or animal
- a route of infection, e.g. ingestion, penetration or an insect vector.

The **definitive** host is that in which sexual reproduction occurs (e.g. mosquitoes for malaria) or in which the mature form of the parasite occurs (e.g. humans for African trypanosomiasis). An **intermediate** host is another animal essential to the completion of the life cycle (e.g. snails for schistosomiasis).

Parasites cause disease in humans by:

- mechanical effects, e.g. hydatid cyst
- invasion and destruction of host cells, as in malaria
- allergic or inflammatory immune reaction by the host to the parasite, e.g. toxocariasis and trypanosomiasis
- competition for specific nutrients, e.g. *Diphyllobothrium latum* for vitamin B<sub>12</sub>
- or there may be no obvious disease, as in *Taenia saginata* in humans.

**Diagnosis in parasitic diseases** depends on:

- a history of exposure and the clinical pattern of illness in the patient
- identification of the parasite itself in excreta (stool, urine), blood, or specific tissues
- indirect evidence of the parasite by testing the patient's blood for antibodies
- detection of parasite antigens in clinical specimens
- detection of parasite DNA or RNA in clinical specimens.



# Helminthology

## Worms of medical importance

### Nematodes (round worms)

- Unsegmented
- Possess mouth, oesophagus and anus
  - Important in further diagnosis
- In general, sexes separate
- Reproduction
  - Oviparous
  - Larviparous
- Infection by
  - Ingestion of eggs, or
  - Penetration of larvae through surfaces, or
  - Arthropod vector, or
  - Ingestion of encysted larvae

### Cestodes (tape worms)

- Segmented
- Possess scolex, neck and proglottids
- Hermaphroditic
- Reproduction
  - Oviparous
  - Sometimes multiplication within larval forms
- Infection generally by encysted larvae

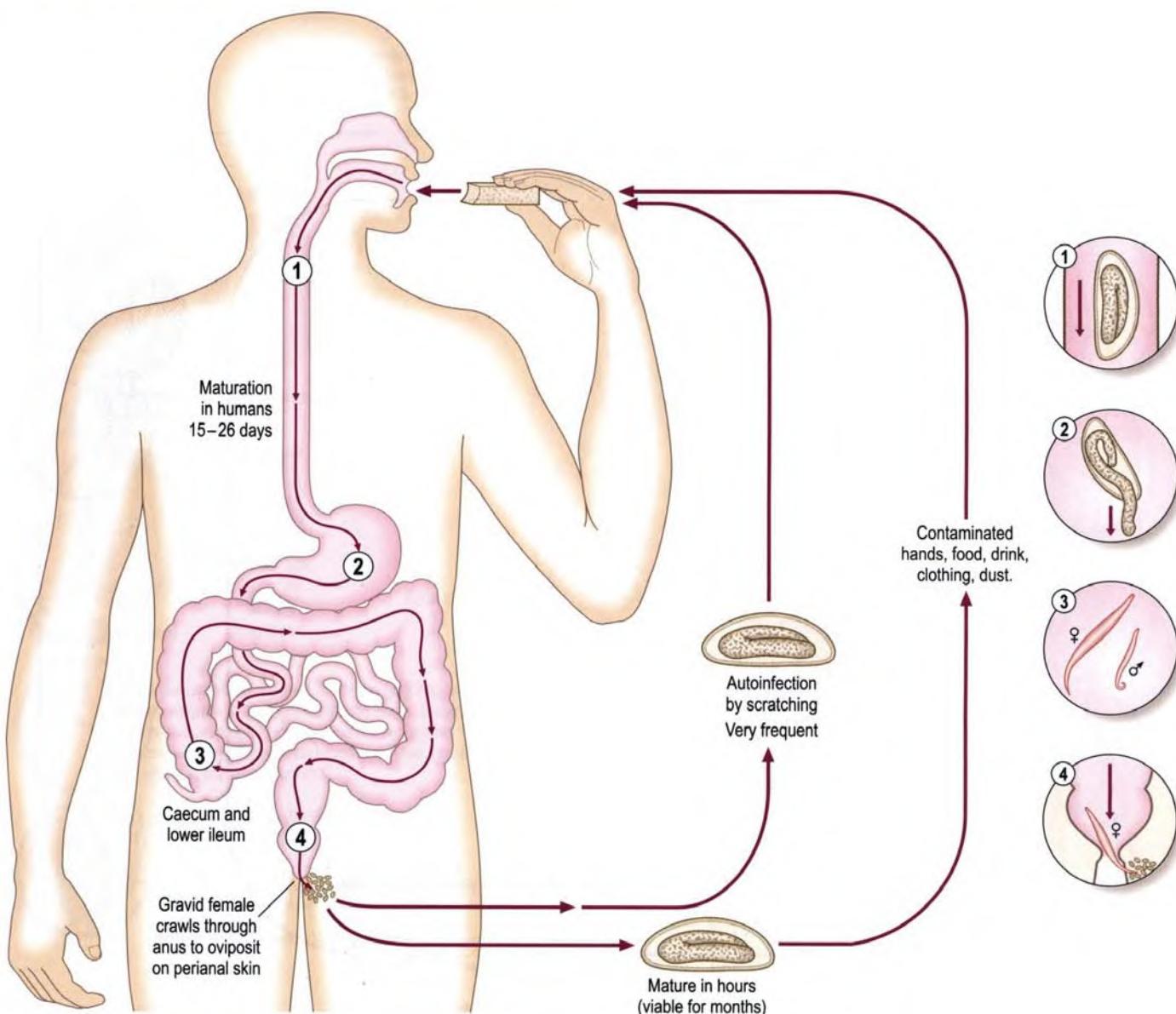
### Trematodes (flukes)

- Unsegmented
- Leaf-like or cylindrical
- Generally hermaphroditic
- Reproduction (digenic)
  - Oviparous
  - Multiplication within larval forms
- Infection mainly by larval stages entering intestinal tract, sometimes through skin

# Nematode (round) worms

## *Enterobius vermicularis* (thread or pin worm)

### Life cycle



### Distribution

350 million infected worldwide, often group or institutional infection.

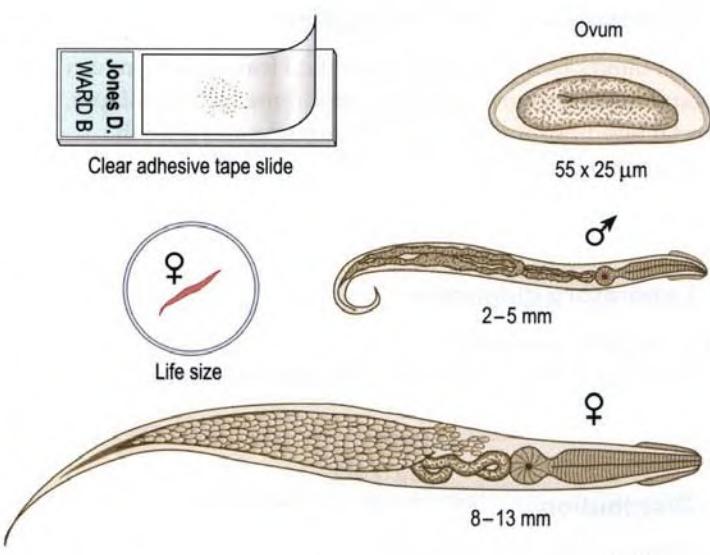
### Pathology and Clinical features

Most infections are asymptomatic. Perianal itching may be troublesome. In females, migrating worms may cause pruritis vulvae or vaginitis. Rarely, urinary tract infection or appendicitis can occur. Migration into the peritoneal cavity has been recorded.

### Laboratory diagnosis

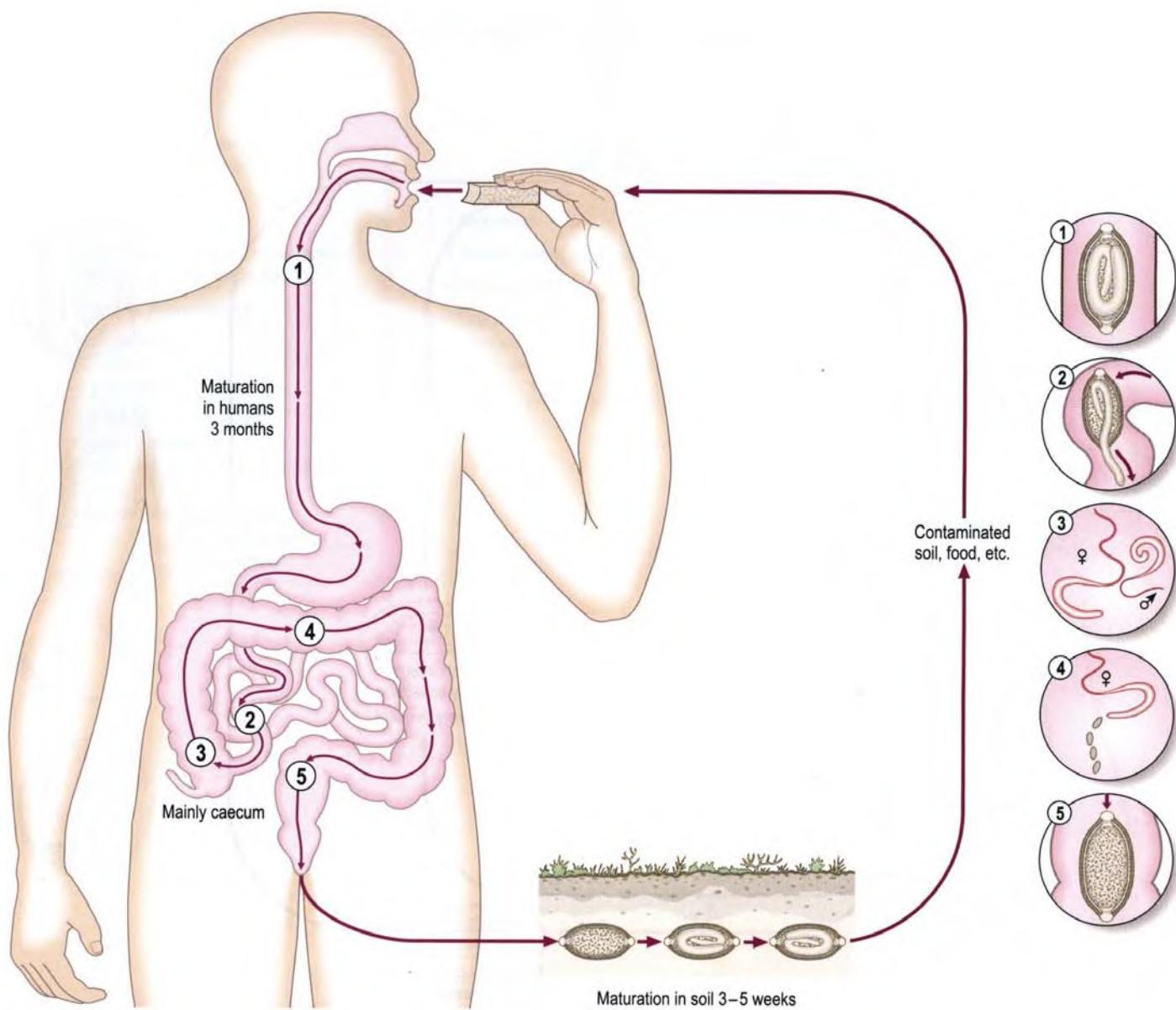
Mild eosinophilia.

Ova can be recovered from the perianal area using clear adhesive tape or a cotton swab moistened with saline. Early morning collection before washing gives best recovery. In females, ova may occasionally be recovered from urine.



# *Trichuris trichiura* (whip worm)

## Life cycle



## Pathology and Clinical features

Light infections may be asymptomatic. Heavy infections can result in the *trichuris* dysentery syndrome, rectal prolapse, rectal bleeding, anaemia, growth stunting and growth retardation in children.

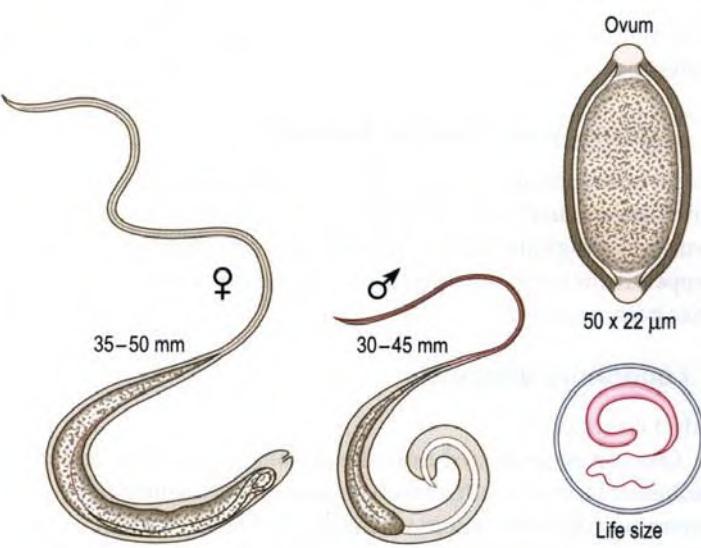
## Laboratory diagnosis

Eosinophilia may occur.

Ova may be recovered in faeces by concentration methods.

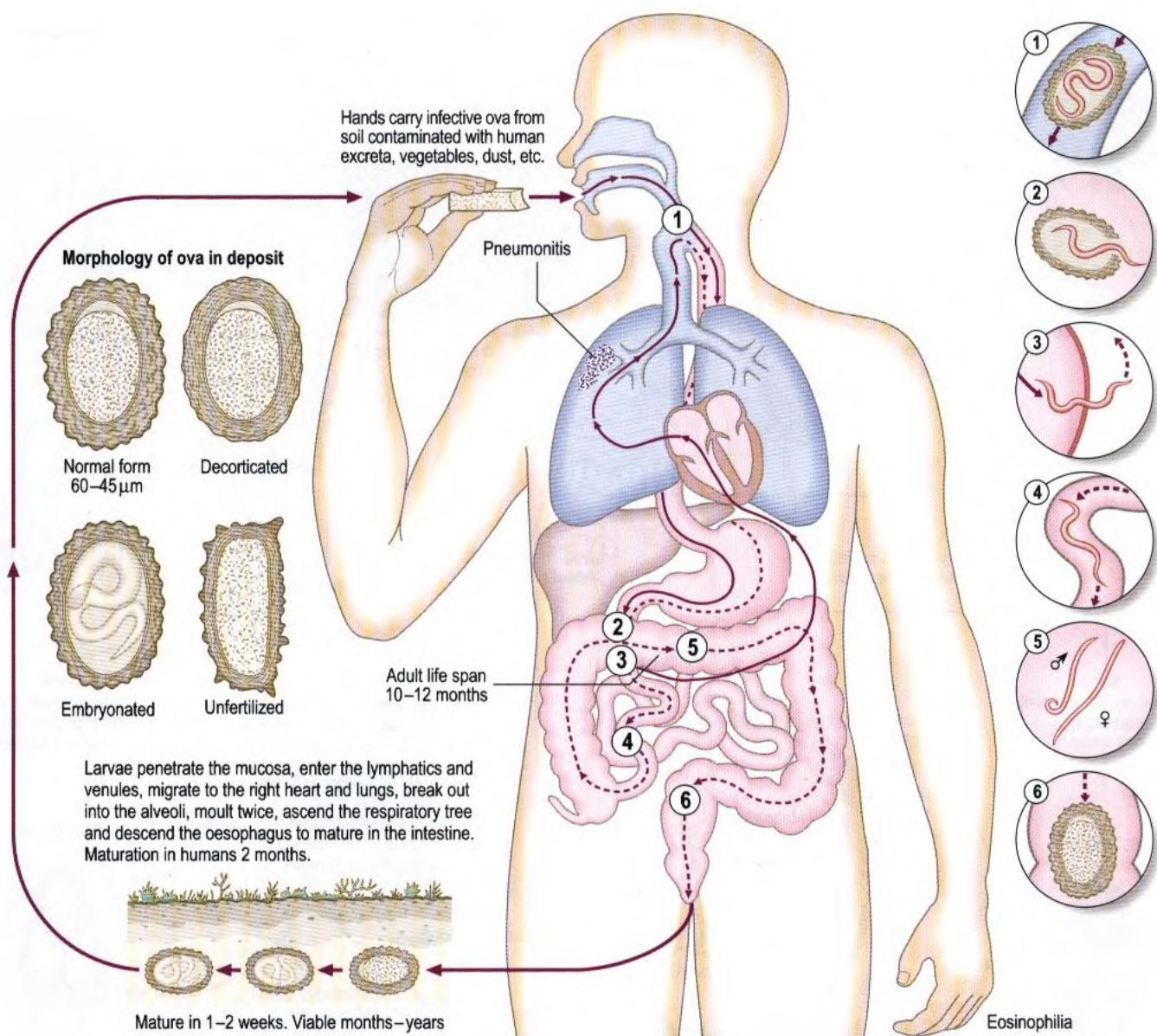
## Distribution

1.3 billion infected worldwide.



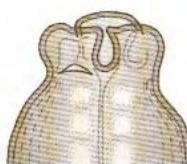
# Ascaris lumbricoides (round worm)

## Life cycle



## Pathology and Clinical features

Larvae can cause pneumonitis with eosinophilia. Adult worms can cause obstruction of the small intestine, bile ducts and trachea; also appendicitis, pancreatitis and peritonitis. Children may vomit up a bolus of adult worms, or cough up immature worms.



Head of adult to show arrangement of the three lips

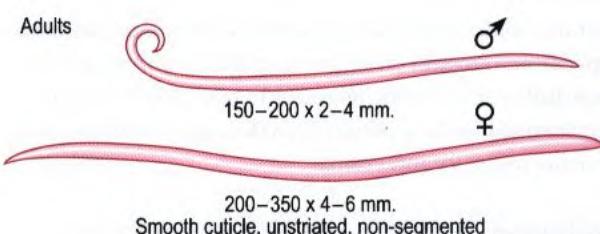
## Laboratory diagnosis

Ova may be recovered from faeces by concentration methods. Rarely larvae can be found in sputum, and must be distinguished from those of *Strongyloides*. Eosinophilia is present in the larval invasion stage.

No specific serology is currently available.

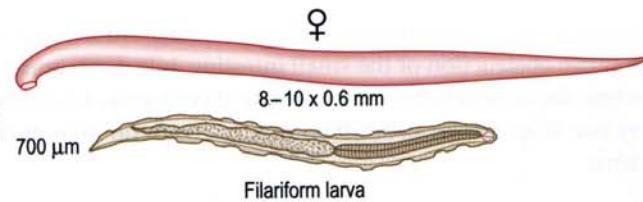
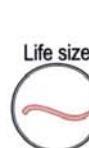
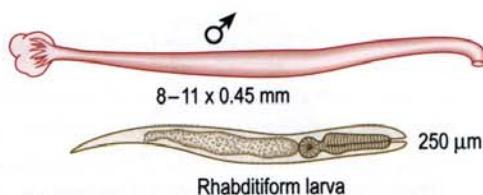
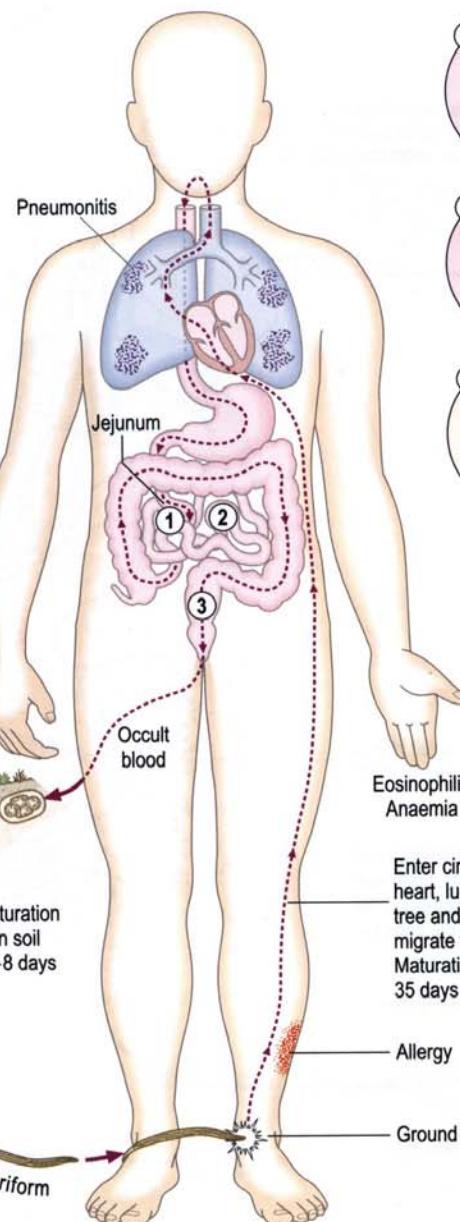
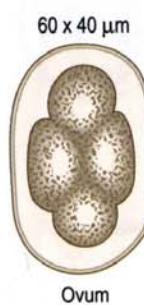
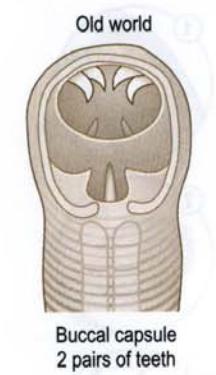
## Distribution

1.47 billion infected worldwide.



## Hookworms

### *Ancylostoma duodenale*



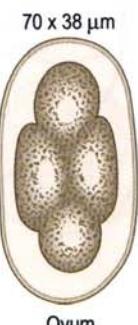
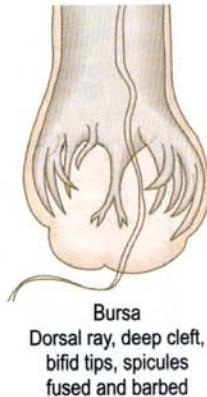
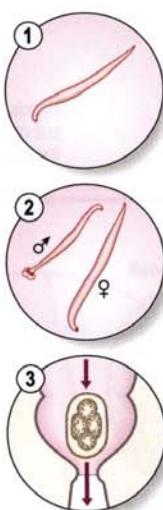
### Pathology and Clinical features

Ground itch may follow skin penetration by filariform larvae. Pneumonitis can result from larval migration through the lungs. Adult worms in the jejunum ingest blood. Occult gastrointestinal bleeding occurs. Iron deficiency anaemia and its sequelae in heavy infections.

### Distribution

900 million infected worldwide.

### *Necator americanus*



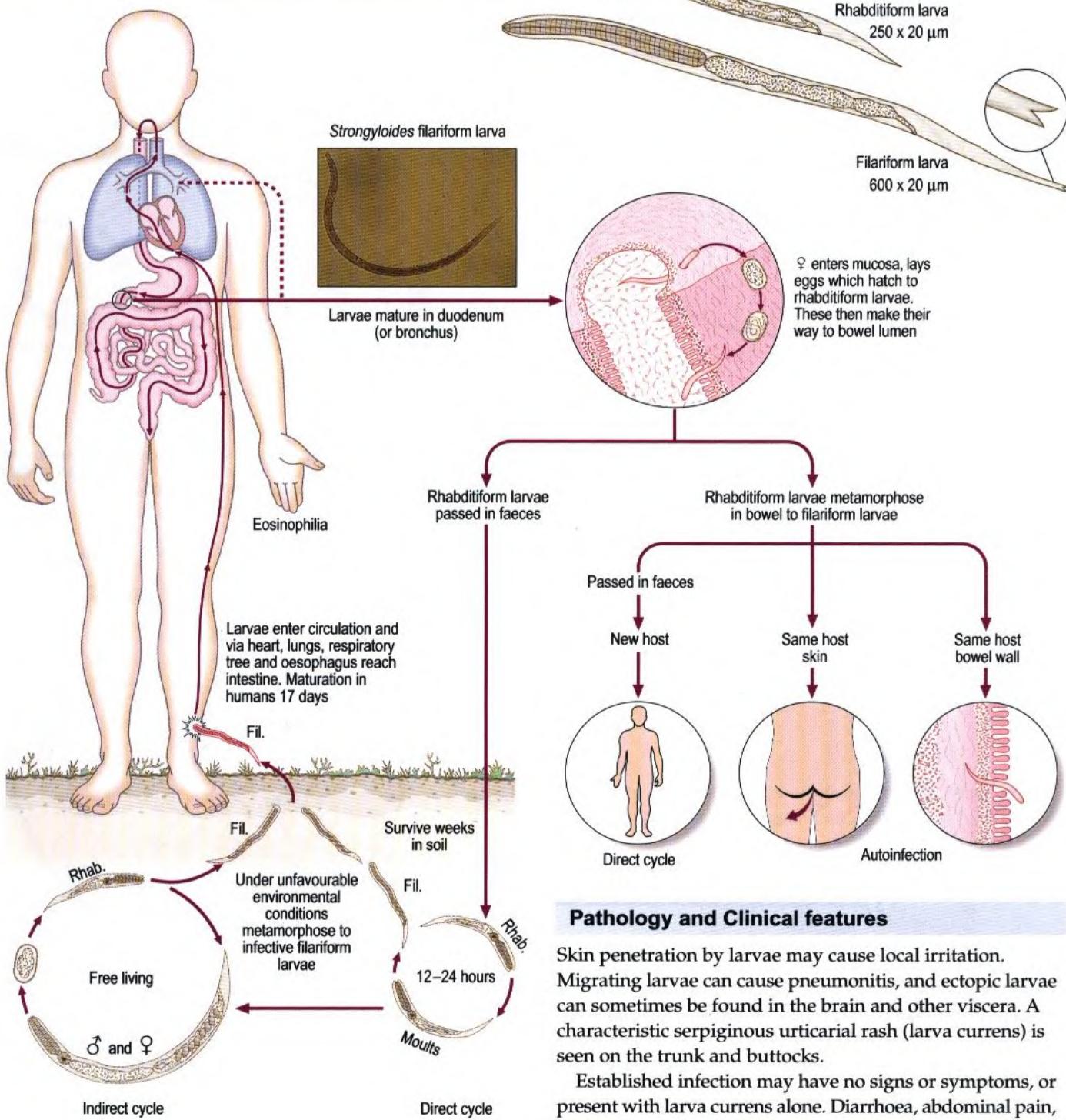
### Laboratory diagnosis

Eosinophilia.

Ova may be recovered from faeces by concentration methods. Rhabditiform larvae may be seen in old faecal specimens and must be distinguished from *Strongyloides* by the appearance of the buccal cavity.

# *Strongyloides stercoralis*

## Life cycle



## Pathology and Clinical features

Skin penetration by larvae may cause local irritation. Migrating larvae can cause pneumonitis, and ectopic larvae can sometimes be found in the brain and other viscera. A characteristic serpiginous urticarial rash (larva currens) is seen on the trunk and buttocks.

Established infection may have no signs or symptoms, or present with larva currens alone. Diarrhoea, abdominal pain, bloating and sometimes malabsorption can be found.

The *Strongyloides* hyperinfection syndrome results from massive autoinfection with filariform larvae in the presence of severe immunosuppression or cachexia. Risk factors include steroid and/or cytotoxic therapy, HTLV1 infections, HIV infections, malignancy, severe malnutrition and other severe systemic disorders. Clinical features include diarrhoea, gastrointestinal haemorrhage or perforation, pneumonitis, Gram-negative bacterial meningitis or septicaemia with high mortality.

## Distribution

70 million infected worldwide.

## Laboratory diagnosis

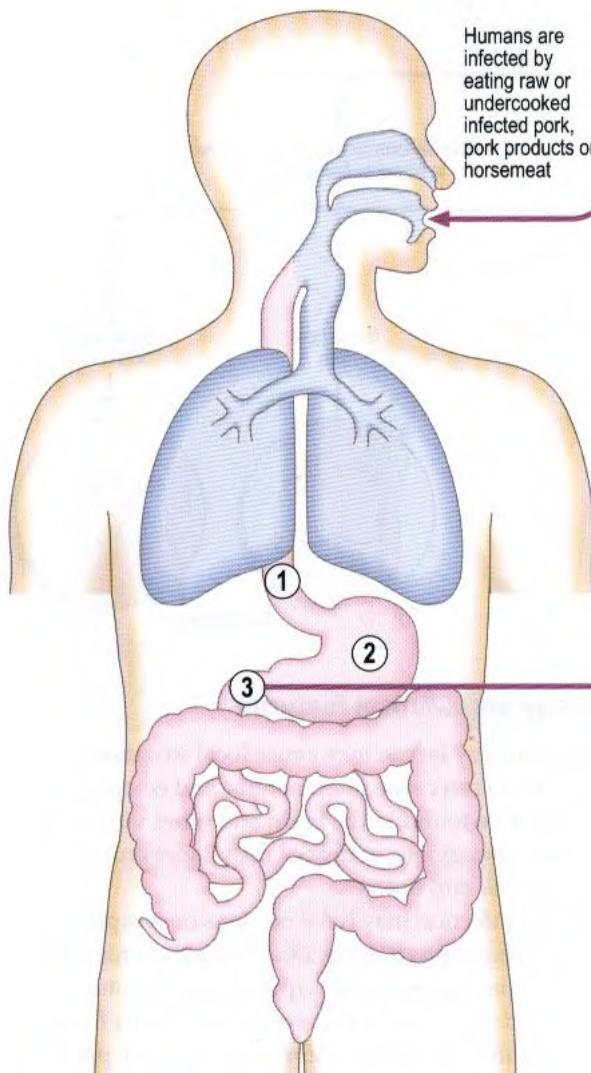
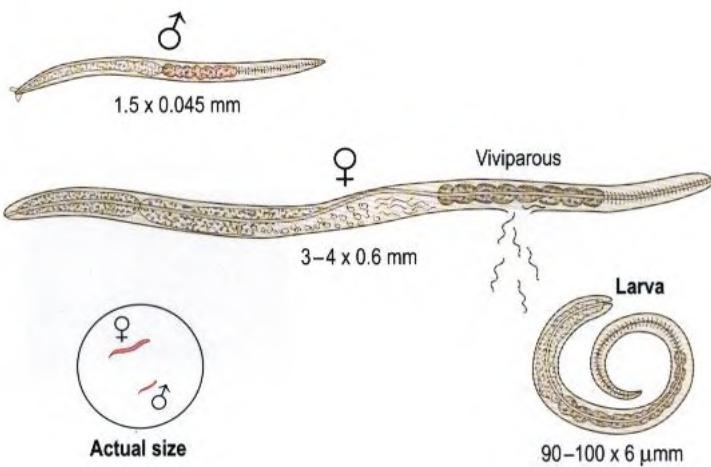
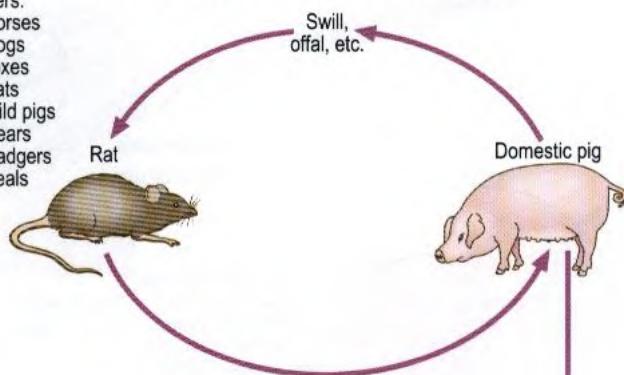
Eosinophilia may be present, but its absence does not exclude diagnosis. It is essential to examine fresh specimens. Rhabditiform larvae can be seen in faeces by direct microscopy or by concentration methods. Filariform larvae may also be seen in faeces, sputum and other body fluids, particularly in immunocompromised hosts. Faecal culture using charcoal is an important diagnostic method. Duodenal aspiration and the 'string test' are also recommended isolation methods. Serology by ELISA is useful in chronic infection.

# *Trichinella spiralis*

## Life cycle

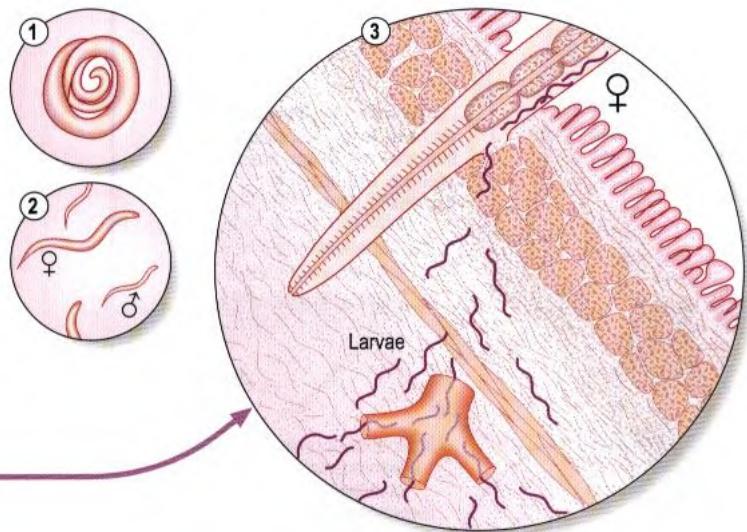
A given host can be definitive or intermediate, but two hosts, both carnivores, are required to complete the cycle

Others:  
 — horses  
 — dogs  
 — foxes  
 — cats  
 — wild pigs  
 — bears  
 — badgers  
 — seals

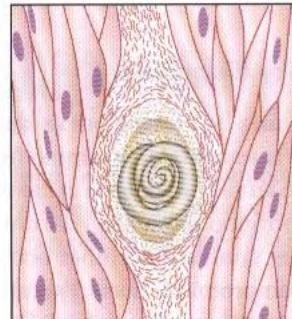


Humans are infected by eating raw or undercooked infected pork, pork products or horsemeat

Infected flesh is digested by gastric juices; the larvae are set free and develop into adults in duodenum. The gravid ♀ burrows into mucosa and releases larvae which enter circulation and are disseminated throughout the body



Dissemination through the blood and lymph



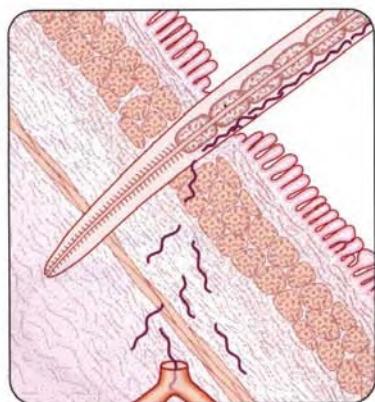
## Laboratory diagnosis

Eosinophilia and high serum CPK in the acute phase. At the encystment stage, use muscle biopsy, muscle crush preparation and serology (IFAT or ELISA).

## Distribution

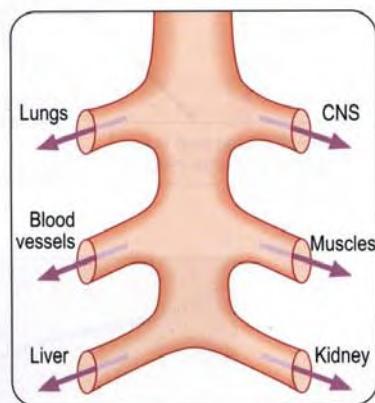
50 million infected worldwide.

Encysted larva in muscle



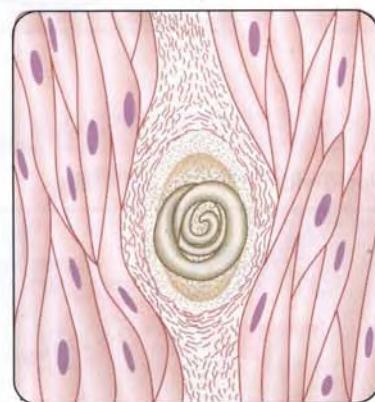
### Invasion

Intestinal inflammation leading to diarrhoea.  
Inflammatory response leading to periorbital oedema, haemorrhages under nails, muscle pains and myocarditis.



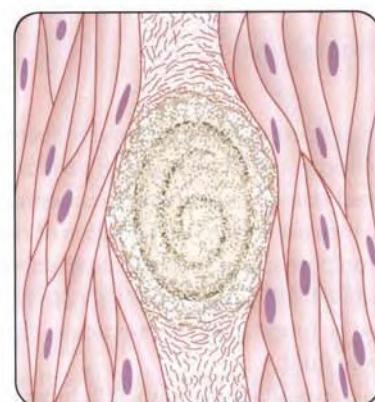
### Dissemination

Migration may occur through any tissue but larval encystment is only in striated muscle. A granulomatous response develops elsewhere.



### Localization

Especially muscles of respiration and tongue. Long term: eventual fibrosis and degeneration, resulting in calcification.



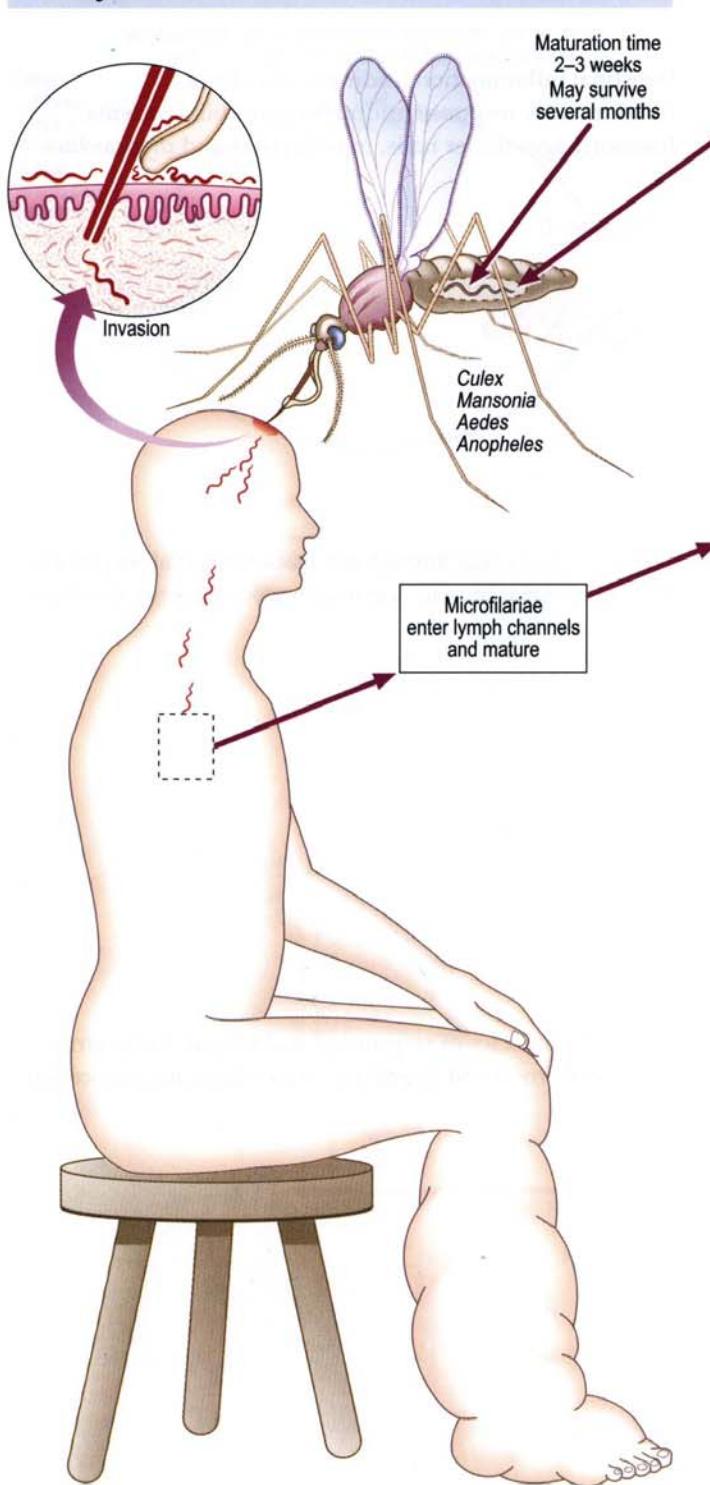
### Organization

### Laboratory diagnosis

At the diarrhoeal stage, adults and larvae may be found occasionally in faeces. Eosinophilia is high. At the encystment stage, use muscle biopsy, muscle crush preparation and serology (IFAT or ELISA).

# *Wuchereria bancrofti* (filariasis)

## Life cycle



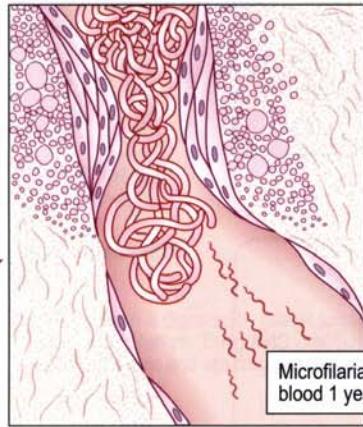
## Development in mosquito

The larvae penetrate stomach, migrate to thoracic muscles, develop, then migrate to head, mature and now infective



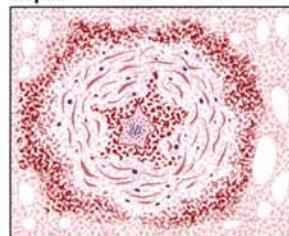
Nocturnal periodicity  
Head bluntly rounded  
♀ 80-100 x 0.25 mm  
♂ 40 x 0.1 mm

## Localization



Microfilariae appear in the blood 1 year after infection

## Repair



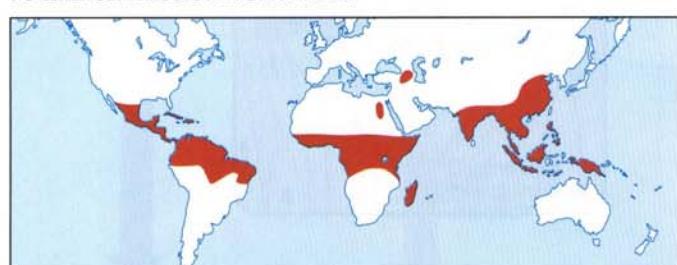
## Microfilaria

230-320 x 10 µm

Tail pointed, free from nuclei  
Sheathed

## Distribution

90 million infected worldwide.



## Laboratory diagnosis

Eosinophilia.

Microfilariae are found in peripheral blood collected between 10pm and 2am, or at midday for *W. bancrofti* var.*pacifica*. Thick blood films are examined stained or unstained, concentration by Knott's method will increase sensitivity. Filtration of citrated blood through a 5 micron pore size polycarbonate membrane is the method of choice.

Microfilariae can also be found in chylous exudate, chylous urine and in hydrocoele fluid.

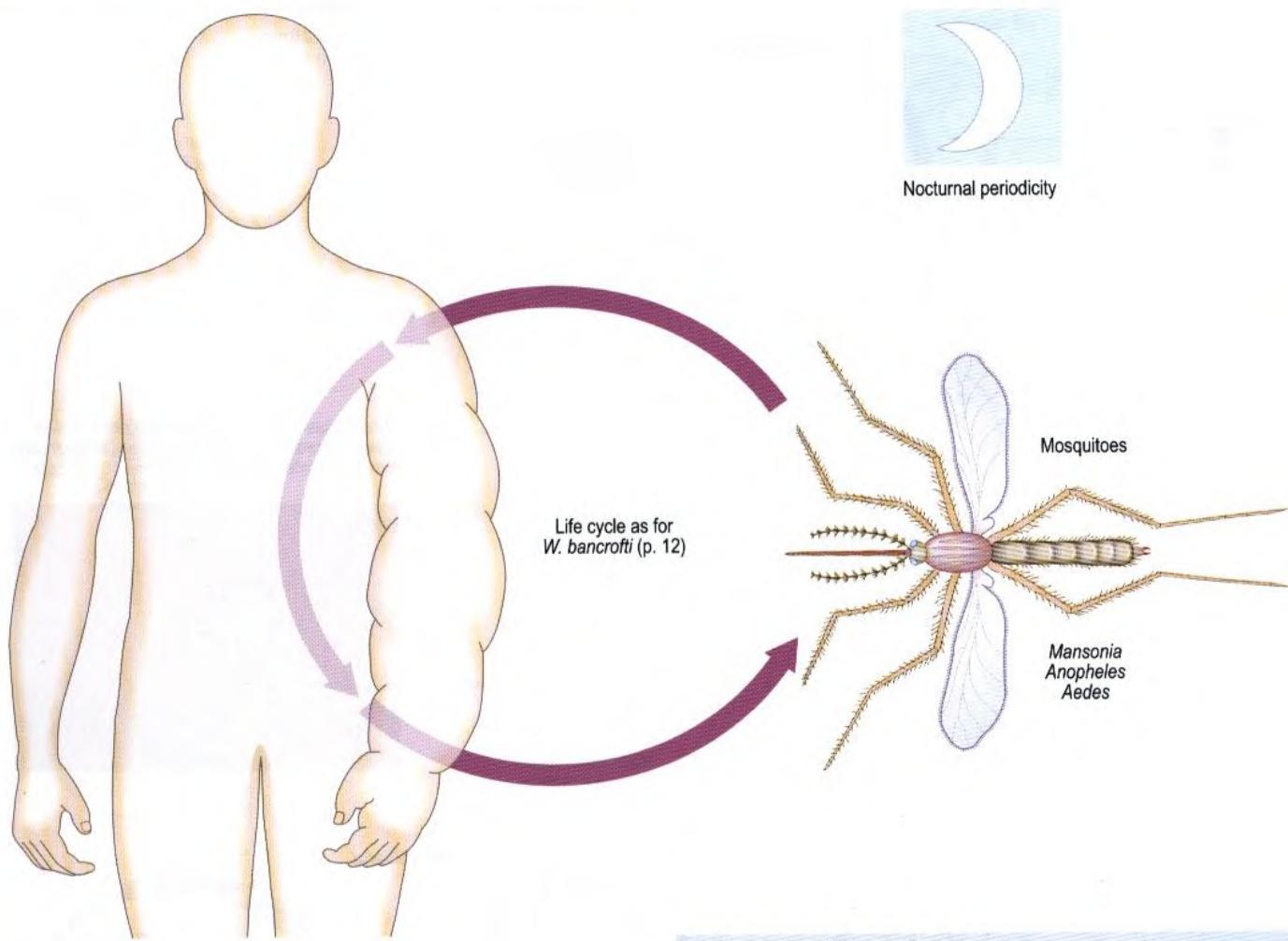
**Serology.** ELISA is of use. Patients with TPE have high filarial antibody levels. A specific *W. bancrofti* antigen immunochromatographic test is now commercially available.

## Pathology and Clinical features

Adult worms in the lymphatic channels cause proliferation of the lining of the endothelium. Surrounding infiltration of eosinophils, macrophages, lymphocytes and giant cells causes filarial granulation tissue leading to obstruction, secondary infection, fibrosis and calcification. The results of this are acute lymphangitis, filarial abscess, lymphadenopathy, elephantiasis, hydrocoele and chyluria. Tropical pulmonary eosinophilia (TPE) occurs in individuals who are hyper-responsive to filarial antigens, giving rise to nocturnal cough, wheeze and low-grade fever.

# *Brugia malayi*

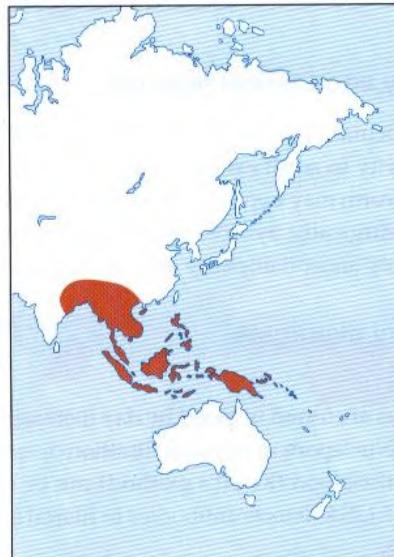
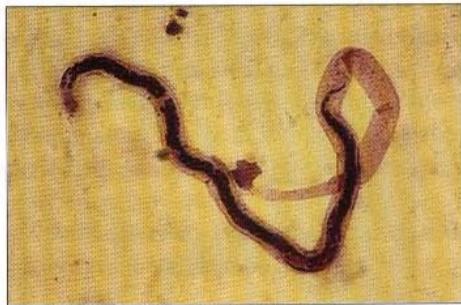
## Life cycle



## Laboratory diagnosis

As for *Wuchereria bancrofti* except for the specific antigen test.

## Distribution

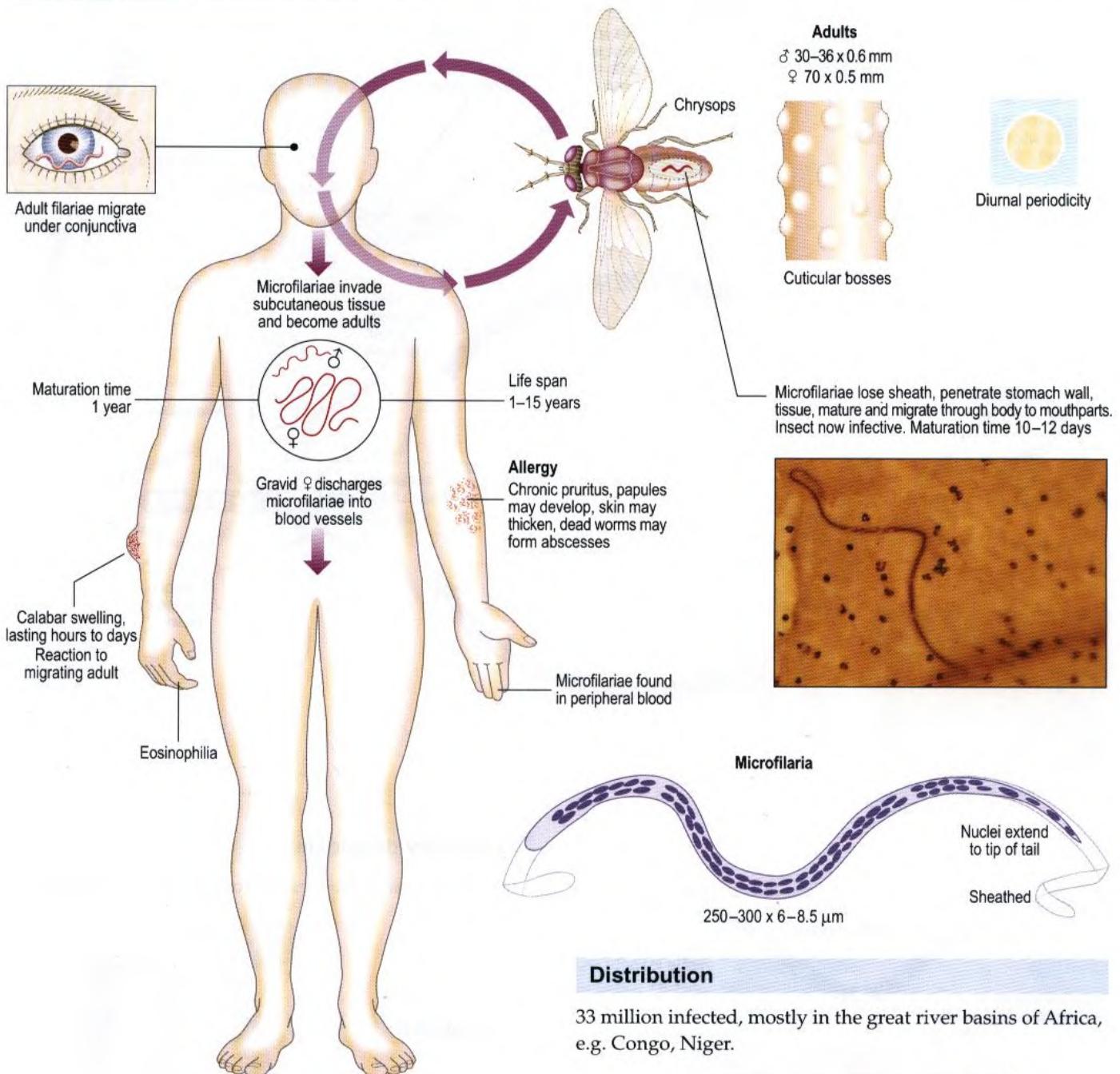


## Pathology and Clinical features

These are similar to those of *Wuchereria*, but *Brugia* more commonly affects the upper limbs. Hydrocoele, other genital lesions and chyluria are rare.

# *Loa loa* (eye worm)

## Life cycle



## Pathology and Clinical features

Transient subcutaneous (Calabar) swellings due to hypersensitivity to adult excretory products.

The adult worm may appear under the conjunctiva and can be removed surgically. Symptoms include fatigue, chronic pruritus, rarely encephalopathy or nephropathy.

## Laboratory diagnosis

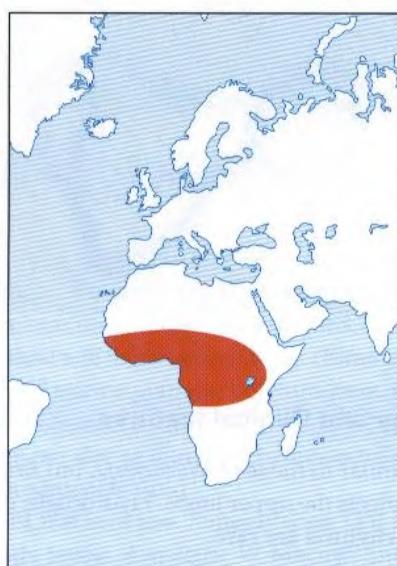
### Eosinophilia

Microfilariae are found in blood by day (between noon and 14:00 hours). Nuclepore membrane filtration or centrifugation after lysis of the blood (Knott's method) can be used.

**Serology.** ELISA detects antibodies to filarial antigens but is non-specific.

## Distribution

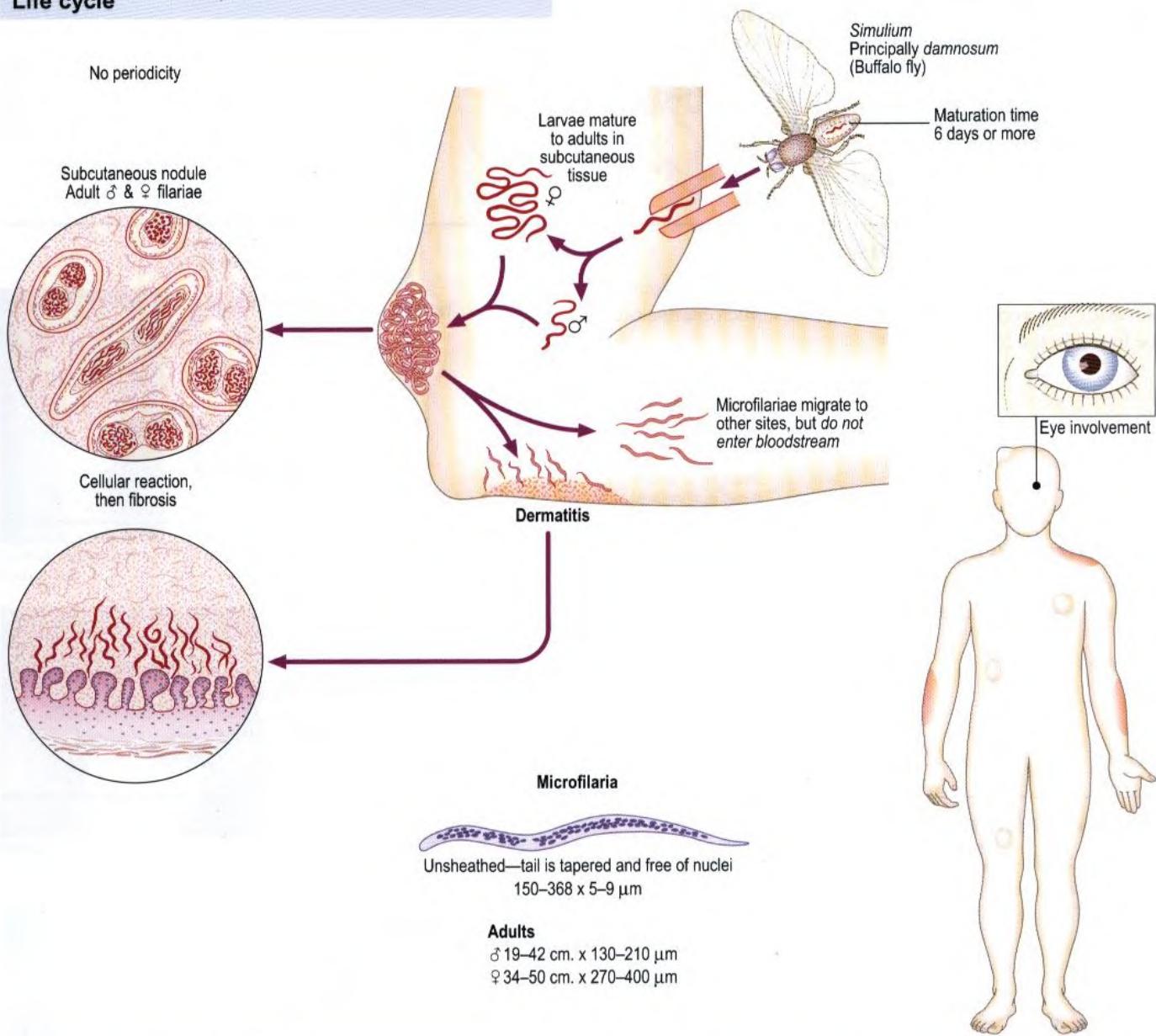
33 million infected, mostly in the great river basins of Africa, e.g. Congo, Niger.



## ***Onchocerca volvulus* (blinding worm)**

## Life cycle

No periodicity



## **Pathology and Clinical features**

Fibrous nodules develop round the adult worms, especially over the iliac crests. There may be some lymphatic obstruction; elephantiasis has been noted in Africa. The microfilariae cause itching, excoriation, urticaria, depigmentation, lichenification, 'sowda' and lymphadenopathy. When invading the eye, they can cause inflammatory lesions in any part of the eye such as sclerosing keratitis, choroidoretinitis and optic atrophy. Blindness may ensue.

Where microfilariae cannot be demonstrated, a Mazzotti test (DEC provocation test) can be useful.

### **Laboratory diagnosis**

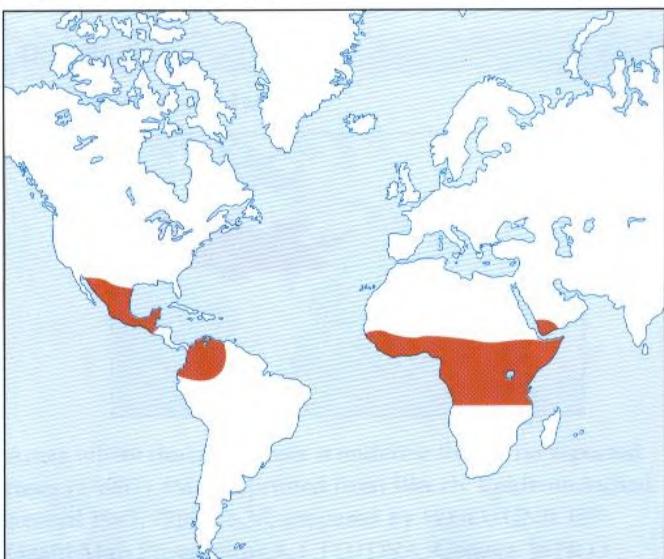
### Eosinophilia.

Adult worms can be detected in excised nodules, microfilariae in the anterior chamber of the eye (slit lamp), skin snips and rarely in blood and urine.

Specific serodiagnosis by ELISA and PCR for parasite DNA on skin samples is in use.

## Distribution

17 million infected worldwide.



## Other filarial worms

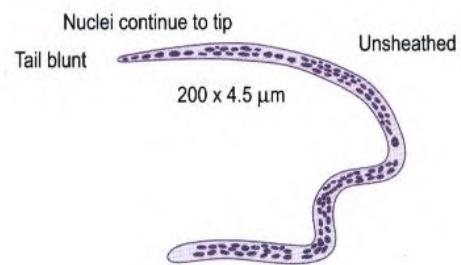
These worms are much less pathogenic. Microfilariae of other species are unsheathed, may be found in the blood and tissues and differentiation from *Wuchereria* and *Brugia* is necessary.

Filtration requires 3 micron pore size membrane, because of the smaller size of these microfilariae.

No periodicity.

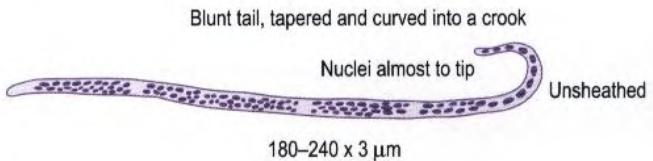
### *Mansonella perstans*

Found in Tropical Africa and the coasts of Central and South America. The vector is the midge *Culicoides*. Microfilariae can be found in the blood.



### *Mansonella streptocerca*

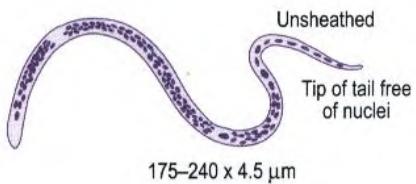
Found in Africa. The vector is the midge *Culicoides*. Microfilariae can be found in the skin.



### *Mansonella ozzardi*

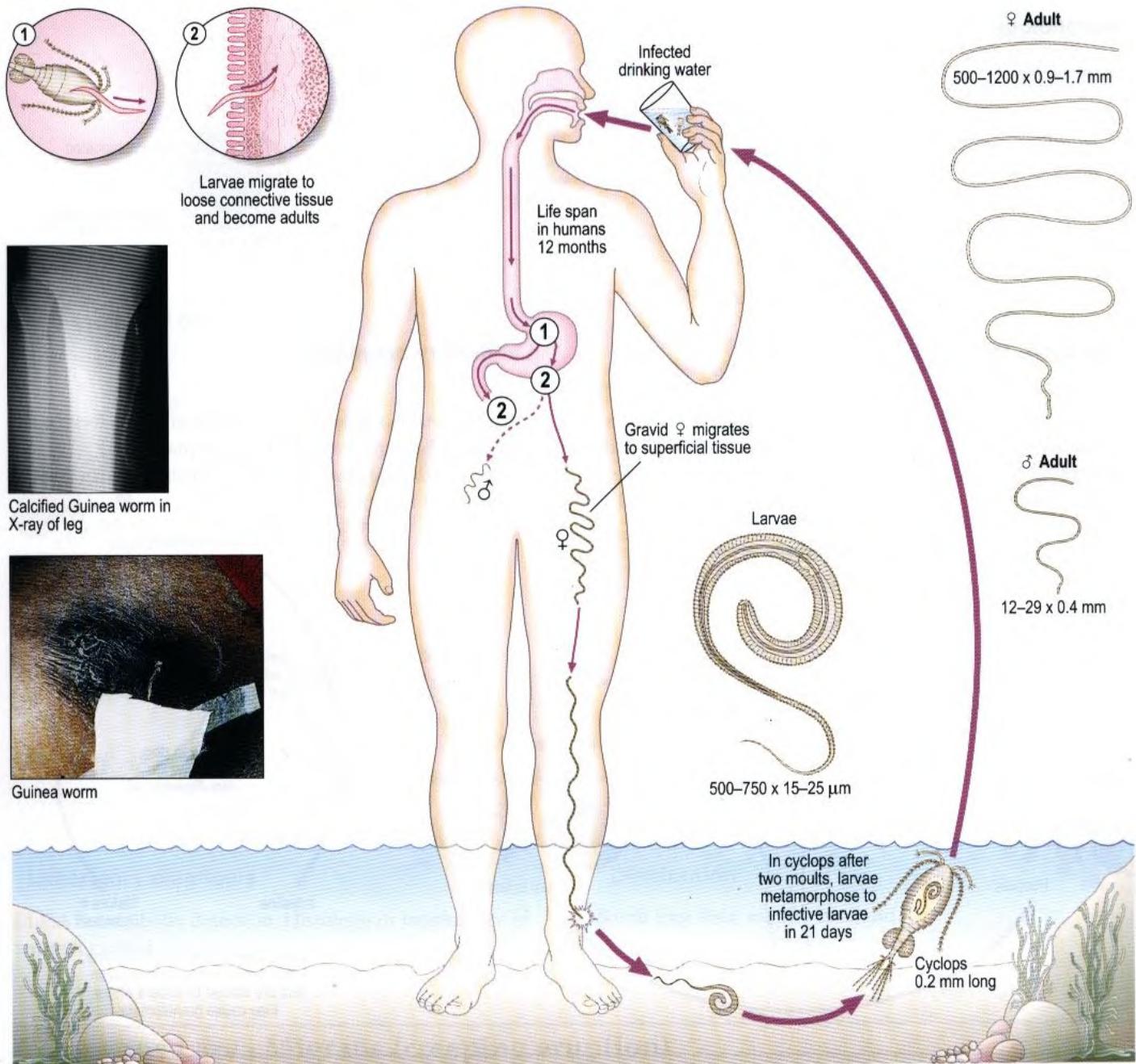
Found in South America and the Caribbean. The vector is the midge *Culicoides*.

Microfilariae can be found in the blood and skin.



# *Dracunculus medinensis* (Guinea worm)

## Life cycle



## Pathology and Clinical features

The gravid female causes itching, urticaria and a burning sensation. A blister appears which bursts to become an ulcer (usually leg) with discharge of embryos and some fibrosis. The adult female may be seen protruding from the ulcer. There is often secondary bacterial infection, and sometimes arthritis of the knee and ankle. Worms may fail to emerge, die and calcify.

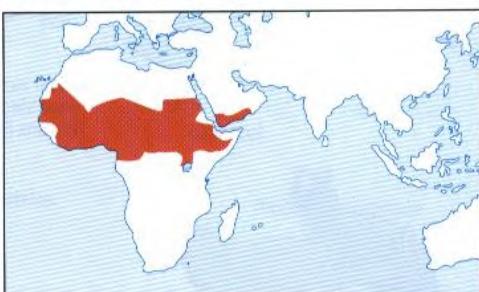
## Laboratory diagnosis

Eosinophilia.

Larvae may be found in fluid from the ulcer.

## Distribution

70 000 infected worldwide.



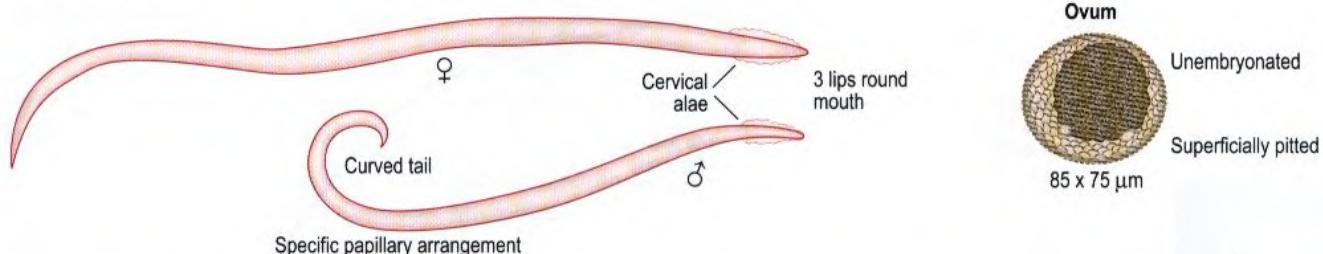
Areas where dracunculiasis is endemic (based on reported cases in 1997). (Map reprinted from Weekly Epidemiological Record 1997; 72(6):33-35; prepared by WHO/UNICEF HealthMap Programme & CTD/DRA, Geneva: WHO.)

# Phasmid Nematodes

## *Toxocara canis* (dog round worm)

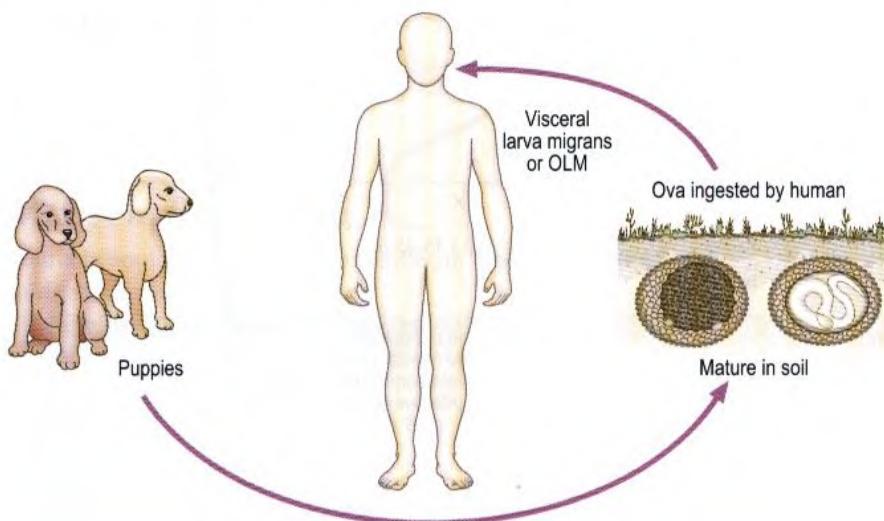
### Morphology

*Toxocara*: body is bent ventrally. *Toxascaris*: body is bent dorsally.



### Life cycle and occurrence

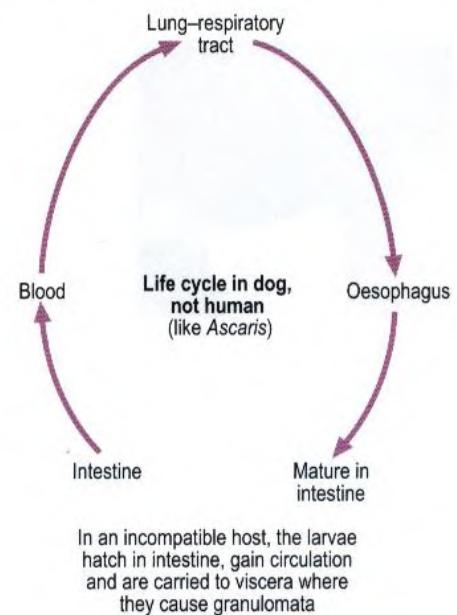
Ocular larva migrans (OLM) and visceral larva migrans (VLM) usually occur as distinct entities without overlap. VLM occurs in younger children and gives rise to fever, pneumonitis and hepatomegaly. Myocarditis, convulsions, psychiatric changes or encephalopathy may occur. OLM presents as unilateral visual loss, often with squint. Retinal detachment, endophthalmitis or papillitis may occur.



### Laboratory diagnosis

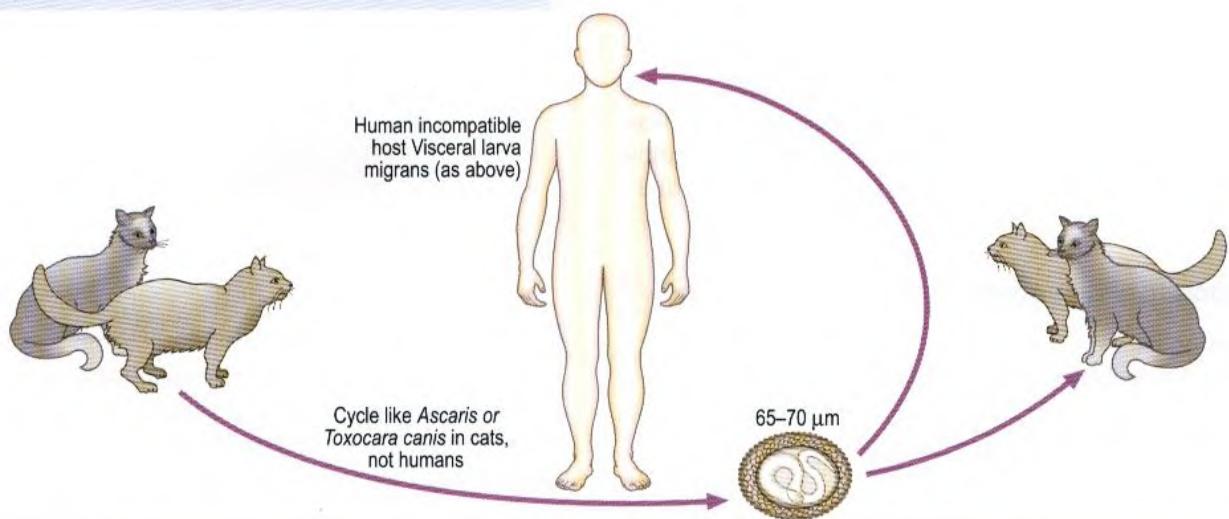
Eosinophilia.

**Serology.** Antibody detection by ELISA on serum. A vitreous sample may be required in OLM. Examination of environmental soil samples for ova by concentration techniques may be an aid to control.



## *Toxocara cati* (cat round worm)

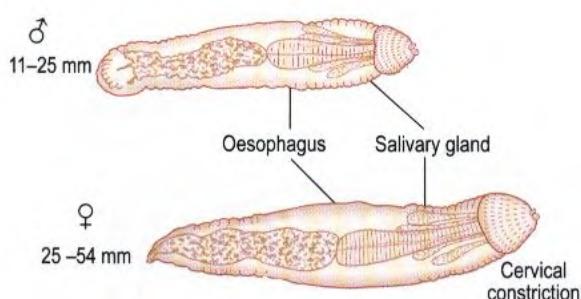
### Life cycle



# *Gnathostoma spinigerum*

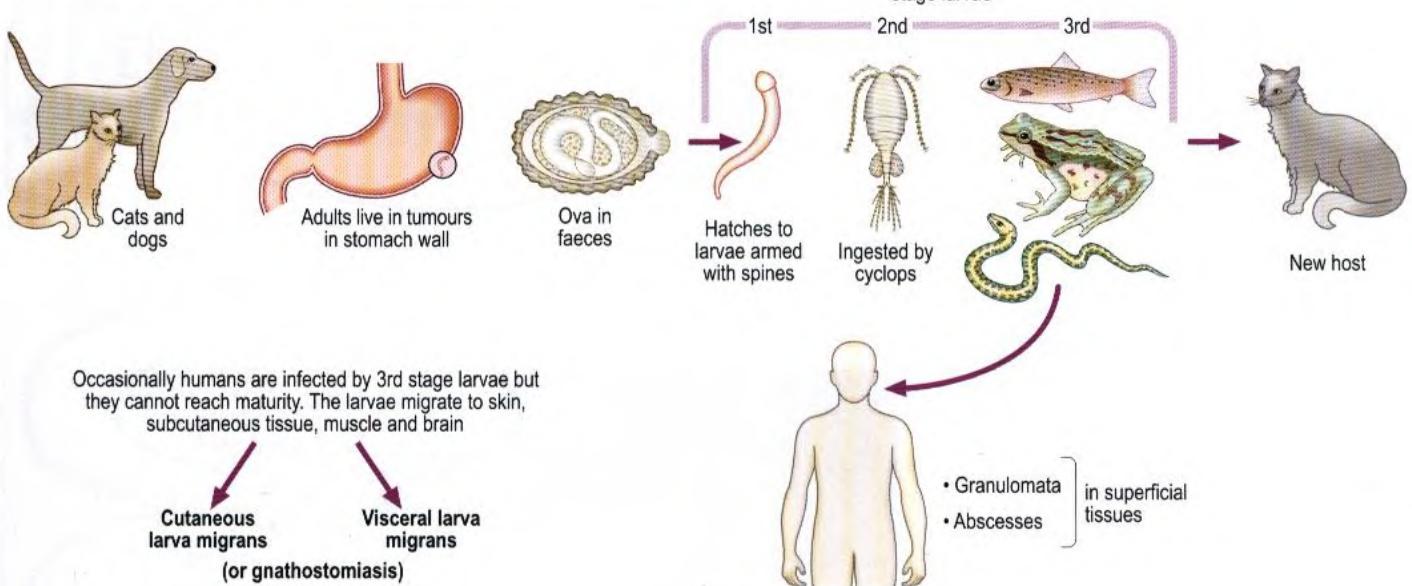
## Morphology

Stout, reddish-coloured worms



Ovum in definitive host  
Non-embryonated  
Plug at one end  
Superficially pitted  
67–70 x 38–40 µm

## Life cycle and occurrence



## Laboratory diagnosis

ELISA for antibody detection. Histology or morphology of worm if excised.

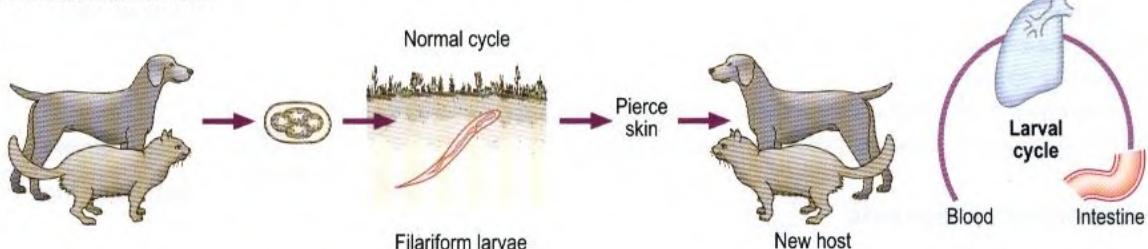
## Distribution

South East Asia, mainly Thailand.

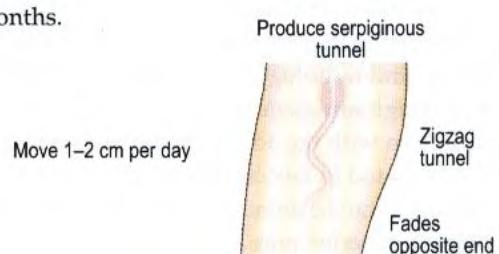
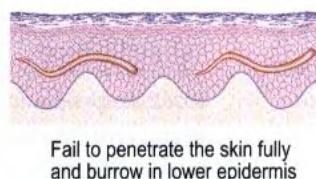
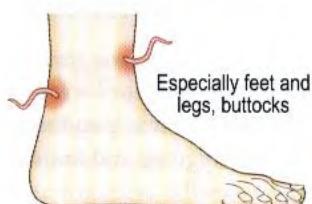
## Cutaneous Larva Migrans (creeping eruption)

Caused by non-human hookworm larvae.

*Ancylostoma brasiliense*  
*Ancylostoma caninum*  
*Uncinaria stenocephala*



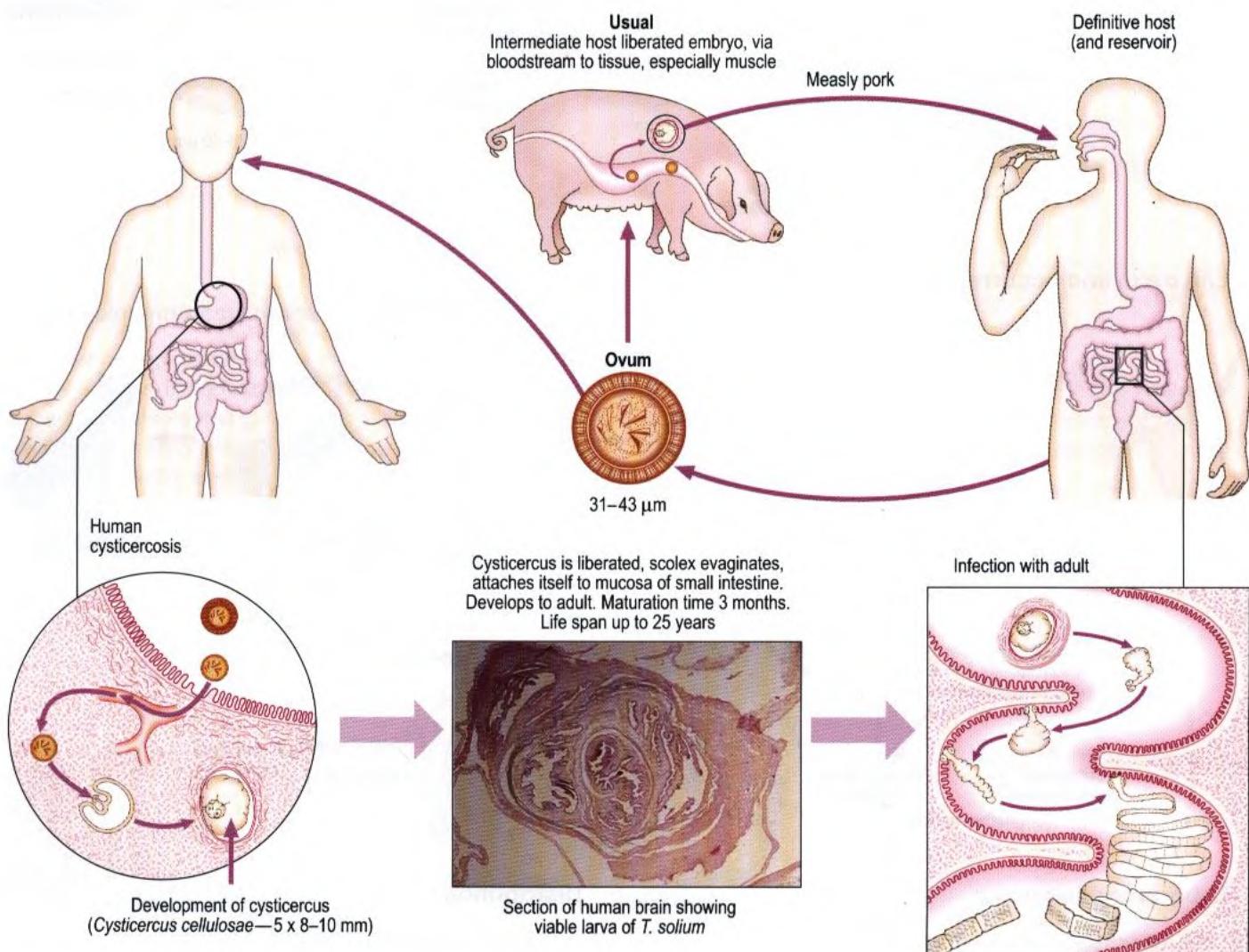
If they successfully invade humans, the intensely itchy infection lasts for months.



# Cestode (tape) worms

## *Taenia solium* (pork tape worm)

### Life cycle



### Pathology and Clinical features

**Infection by larvae (cysticercosis).** Cysticerci, generally multiple, may occur in any site but are more frequent in the brain and muscle. They excite reaction in the area, especially when they die, which manifests as inflammation, fibrosis and later some calcification. This leads to focal CNS syndromes, especially epilepsy.

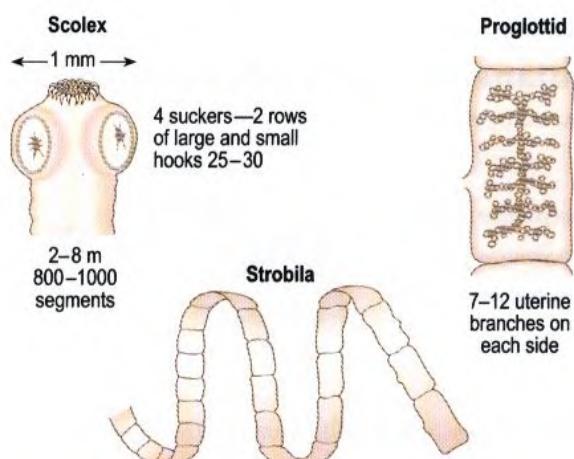
**Infection with adults.** Often there can be no pathology, but there might be mild irritation of intestinal mucosa.

### Laboratory diagnosis

Eosinophilia.

Larval infections. There are several methods, including histological examination of biopsy material, serology (IFAT, ELISA, EITB) and radiology (CT or MRI scan of the brain, X-ray of the thigh muscles).

Pure infection with the adult. Gravid segments, ova and scolex can be found in faeces. The uterine branches of the mature segments can be demonstrated by injection of Indian ink through the uterine pore.

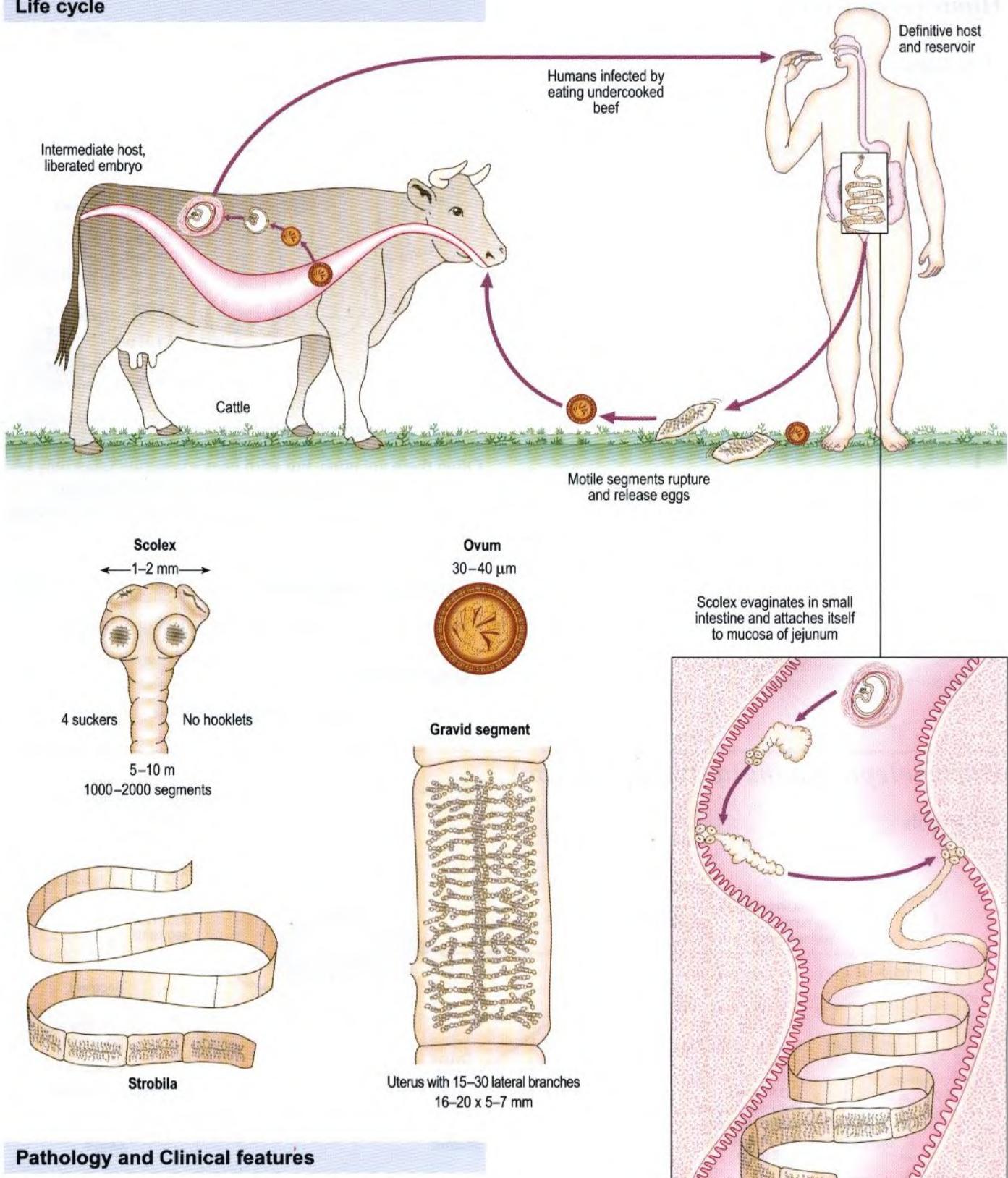


### Distribution

5 million people infected worldwide. *Taenia solium* is endemic in pig-rearing areas of the world where hygiene and animal husbandry are poor.

# *Taenia saginata* (beef tape worm)

## Life cycle



## Pathology and Clinical features

Usually there is no pathology as *Cysticercus bovis* is unknown in humans. Occasionally there is vague alimentary upset.

## Laboratory diagnosis

Gravid segments, ova and scolex can be found in faeces. Uterine branches of the mature segments may be seen in a crush preparation between two glass slides, or by Indian ink preparation, as in *T. solium*. Ova are also found on the perianal skin (on clear adhesive tape slides).

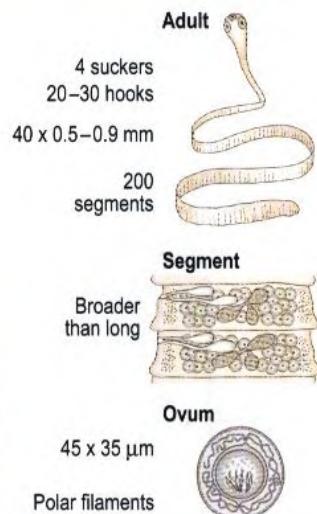
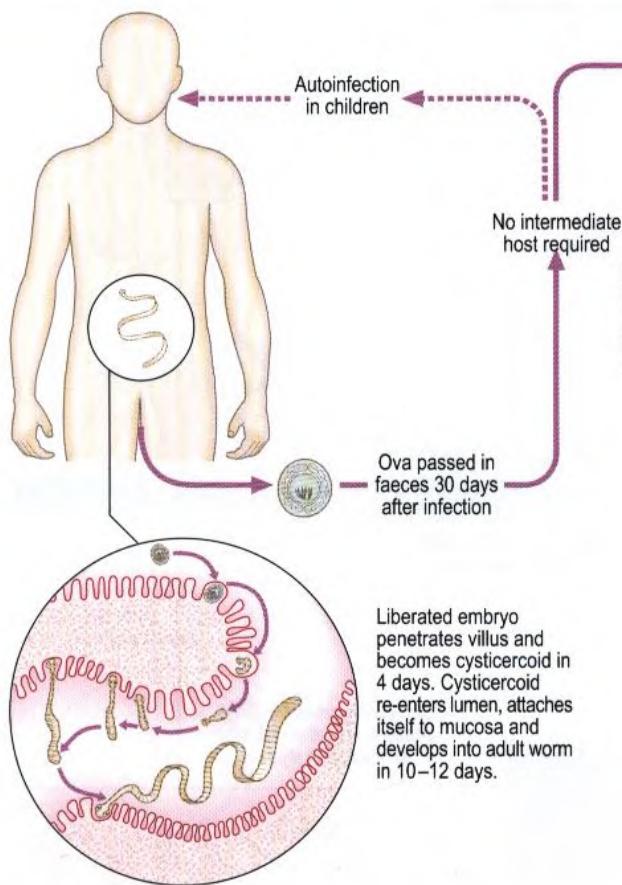
## Distribution

*Taenia saginata* is found in beef-eating areas, especially in the tropics.

# Dwarf tape worms

## *Hymenolepis nana*

### Life cycle



### Pathology and Clinical features

Often there are none, but with heavy infection there may be abdominal pain and diarrhoea. Anaemia and nervous symptoms, including dizziness and irritability, can occur in children.

### Laboratory diagnosis

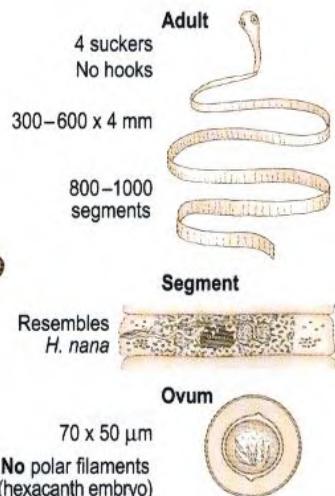
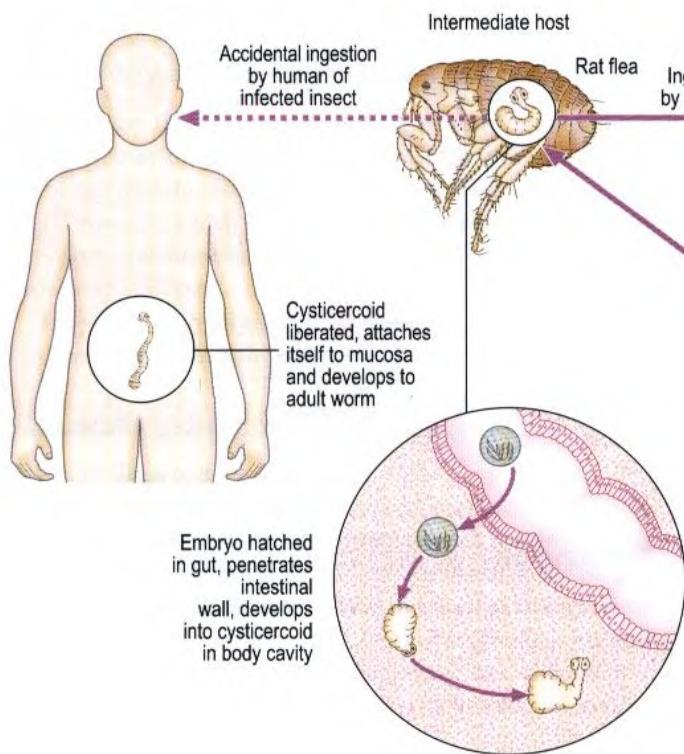
Eosinophilia may be present. Ova found in faeces.

### Distribution

36 million people are infected worldwide.

## *Hymenolepis diminuta* (rat tape worm)

### Life cycle



### Pathology and Clinical features

Generally there is no effect on the host.

### Laboratory diagnosis

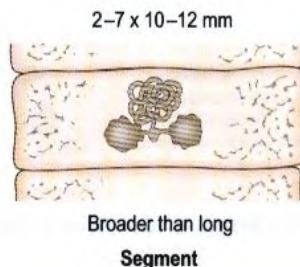
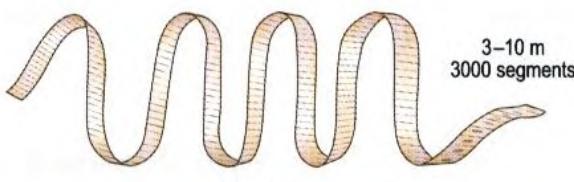
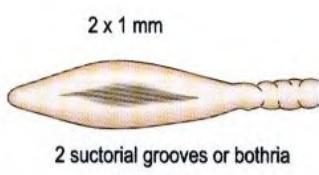
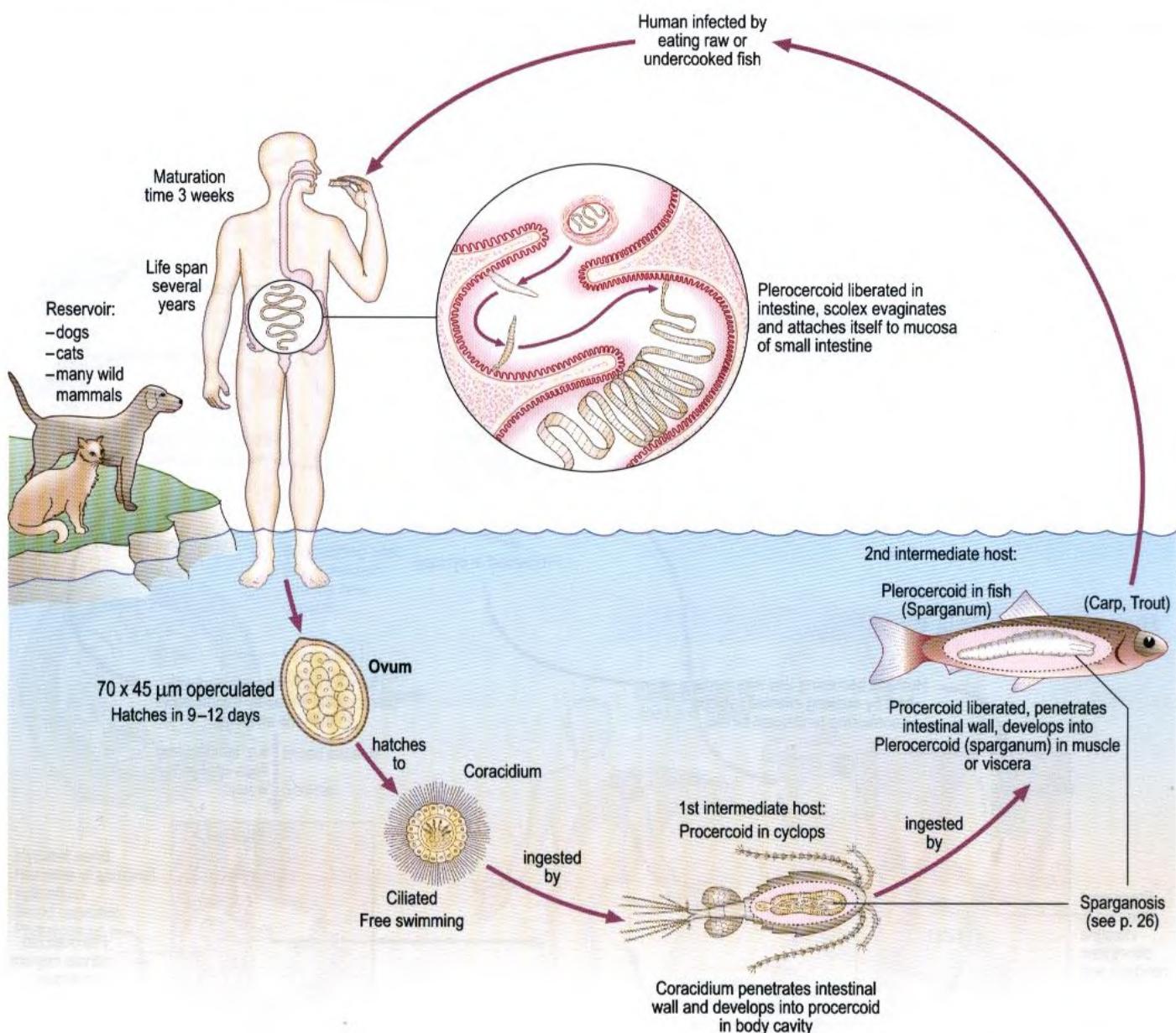
Ova in faeces.

### Distribution

Worldwide, but rare in humans.

# Diphyllobothrium latum (fish tape worm)

## Life cycle



## Pathology and Clinical features

Generally there is none, but occasionally there can be megaloblastic anaemia (through absorption of vitamin B<sub>12</sub> by the worm).

## Distribution

16 million infected worldwide in eastern seaboard of Canada and America, Brazil, Baltic States, parts of West Africa, North Siberia and South East Asia.

## Laboratory diagnosis

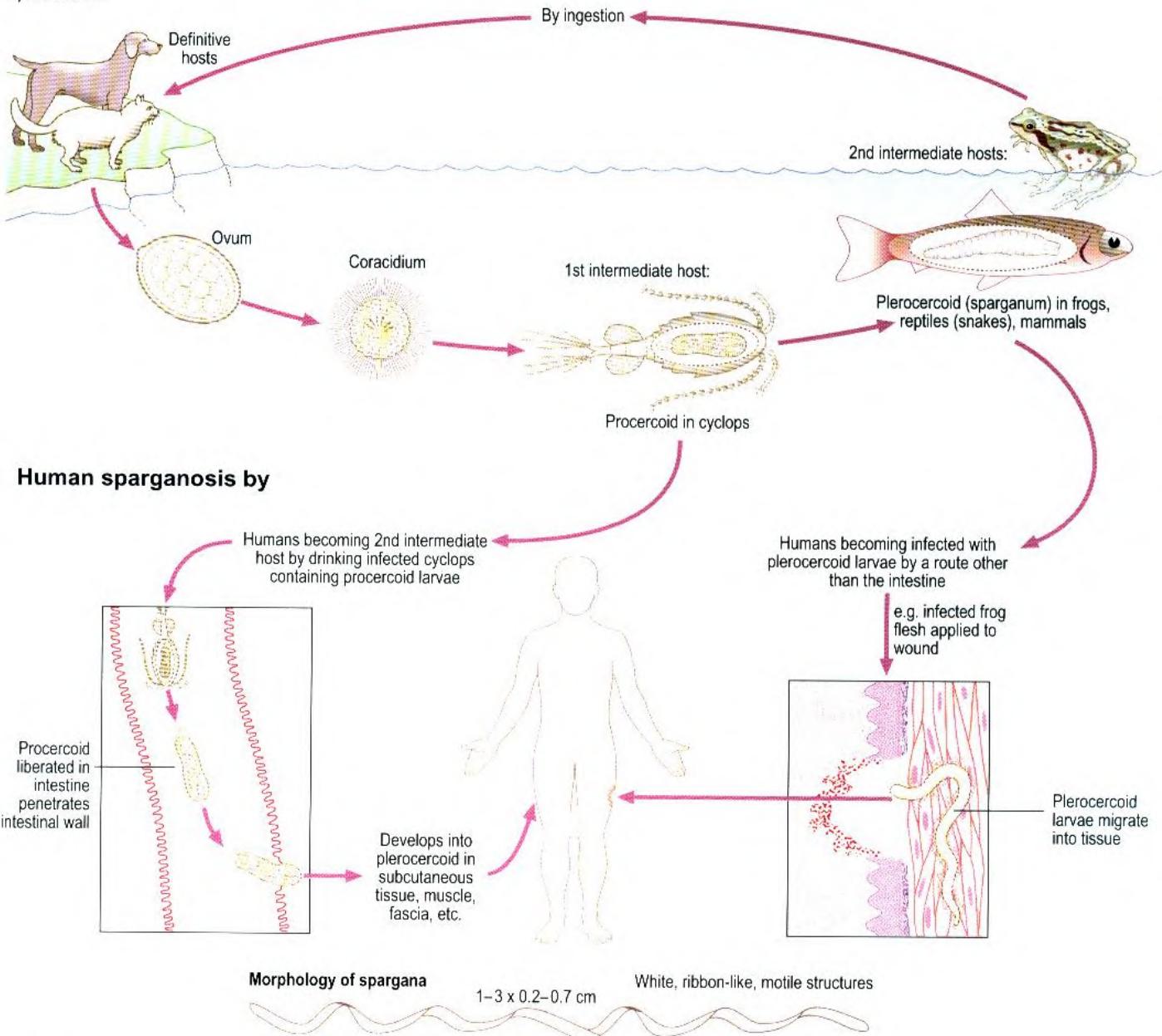
Eggs and gravid segments can appear in faeces. Megaloblastic anaemia (low serum B<sub>12</sub>).

# Larval forms of cestode infection in humans

## Sparganosis

### Life cycle of such tapeworms

Sparganosis is caused by the extra-intestinal presence in the human body of larvae of non-human tapeworms of the genus *Spirometra*.



### Pathology and Clinical features

Infestation with living larvae causes a painful oedematous reaction. Dead larvae cause intense local inflammatory reactions. There are numerous eosinophils and there can be abscess formation. There can be ocular sparganosis in the soft tissues near the eye, resulting in severe damage. Invasion of the CNS may occur.

### Types of spargana

Most Spargana do not proliferate in human tissues. *Sparganum proliferum* is a very rare parasite in which sparganum proliferates by lateral budding.

### Laboratory diagnosis

Diagnosis of the disease is by examination of biopsy material or excised larvae.

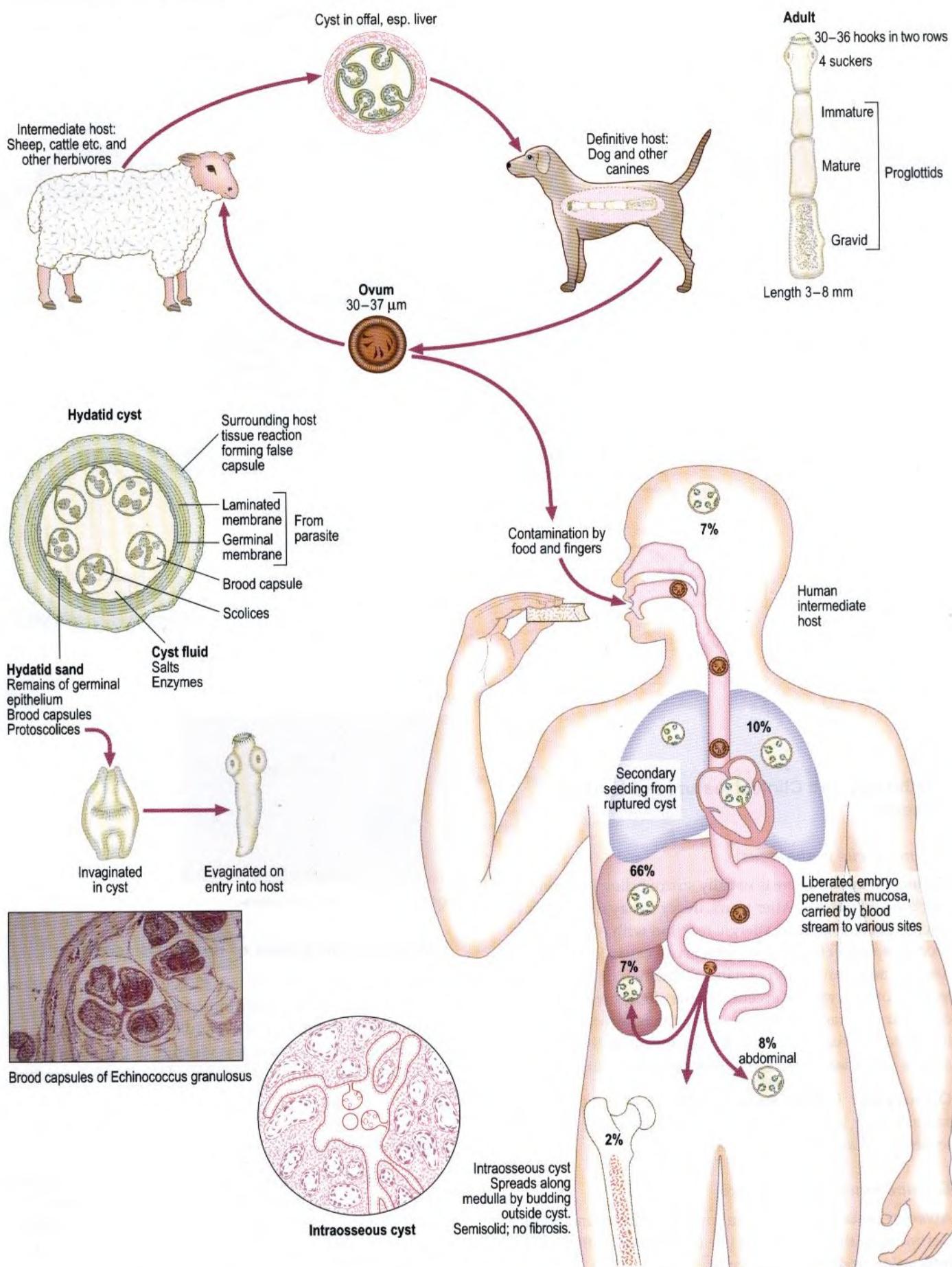
### Distribution

The Far East mainly but occasionally elsewhere.

## **Echinococcus granulosus (dog tape worm)**

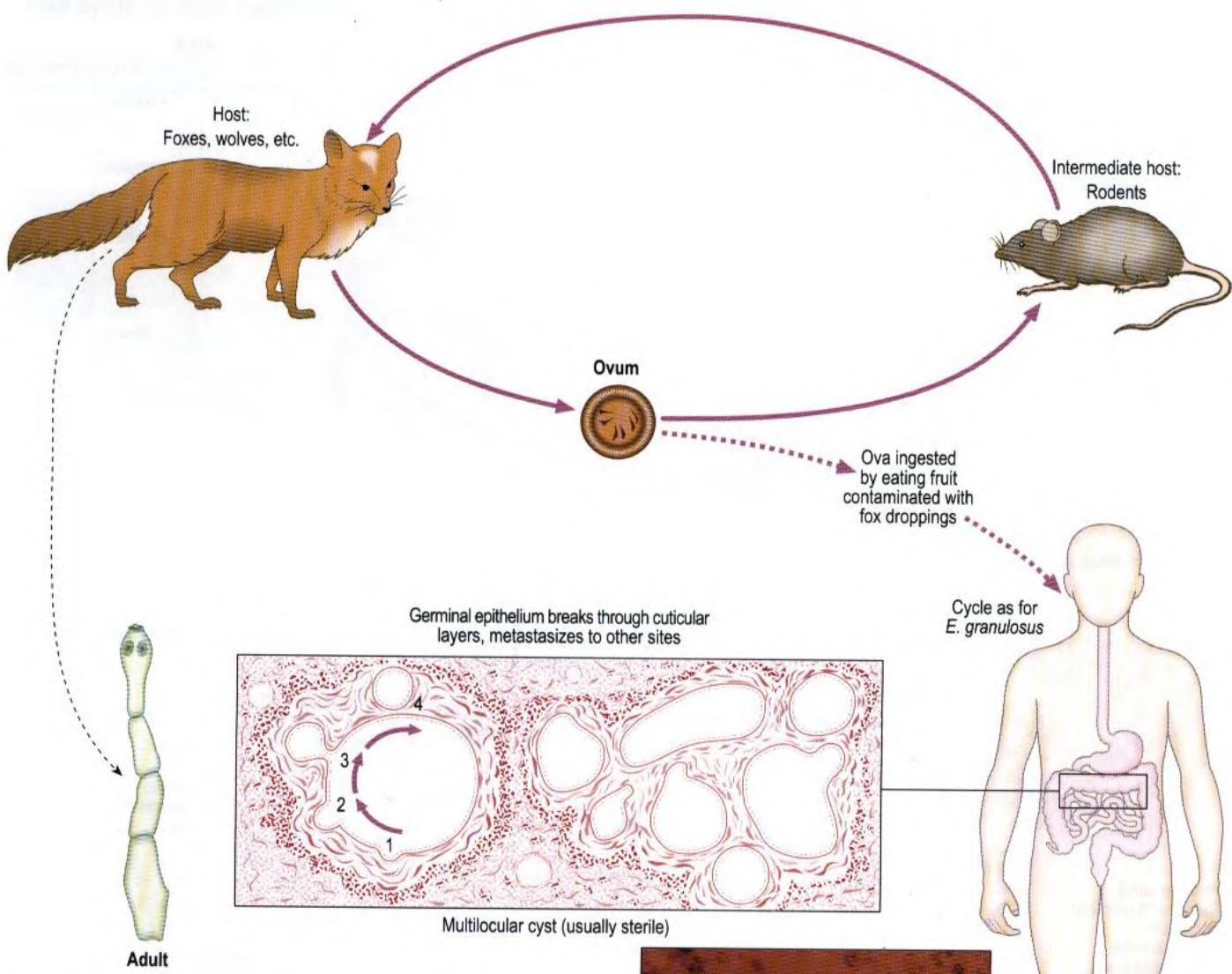
## Life cycle

*Echinococcus granulosus* causes hydatid disease.



# *Echinococcus multilocularis*

## Life cycle



## Pathology and Clinical features of hydatid disease

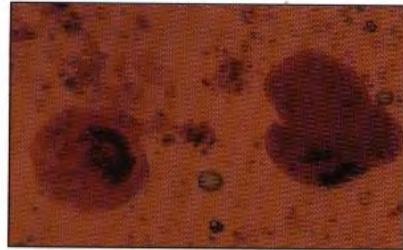
### *Echinococcus granulosus*

**Unilocular cysts.** There is usually surrounding inflammatory reaction and fibrosis. After years, the cyst may die, shrink and calcify. There is general allergic reaction with eosinophilia, bronchospasm, etc. Pressure effects can cause local tissue damage and obstruction of natural channels. Rupture or leakage of the cyst can accentuate the allergic reaction. There can be anaphylactic shock and sometimes secondary implantation, for example in the peritoneal region. There can also be secondary infection with formation of abscess.

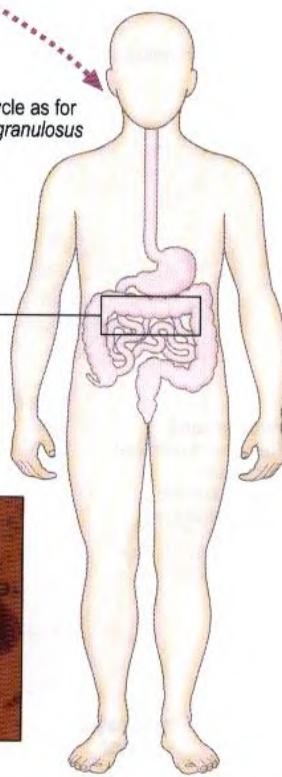
**Osseous cysts.** Usually there is no fibrosis although there is some cellular infiltration. Destruction of the bone can sometimes lead to spontaneous fracture.

### *Echinococcus multilocularis*

**Alveolar cysts.** There are local pressure effects and allergy. Germinal epithelium can act like a neoplasm with local infiltration or distant metastases.



Eosin penetration of dead protoscoleces



## Laboratory diagnosis of hydatid disease

Use serological tests on serum (e.g. ELISA, complement fixation, counter current immunoelectrophoresis for Arc 5 or immunoblot). Microscopy of cyst fluid from operative specimens can be used to assess viability of protoscoleces. Histological examination of a removed specimen is another possibility.

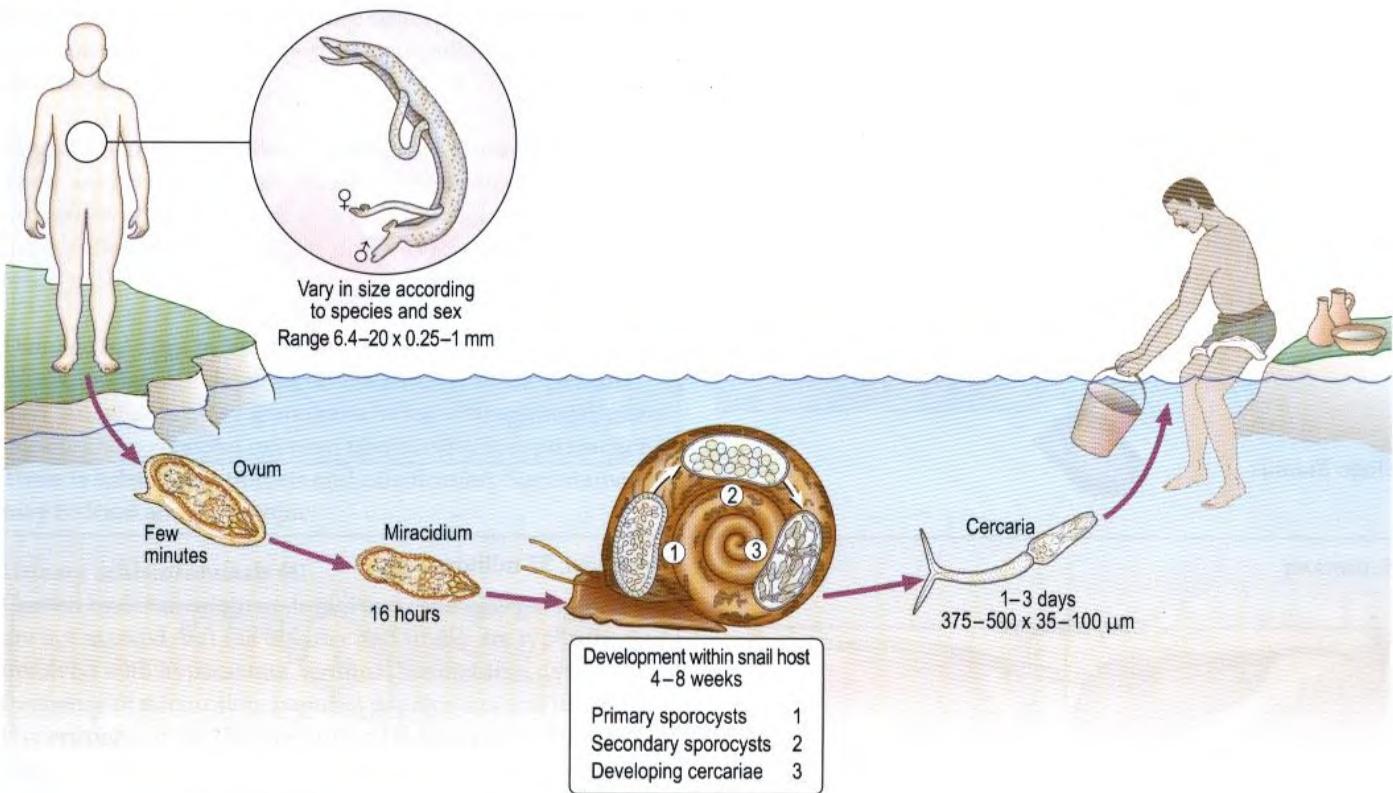
## Distribution

1 million infected worldwide. *E. multilocularis* is rare in humans, but occurs in Northern Europe, Asia, North America and Arctic regions. *E. granulosus* is widespread in sheep-rearing areas of the world. Eradication is well advanced in Australia and New Zealand.

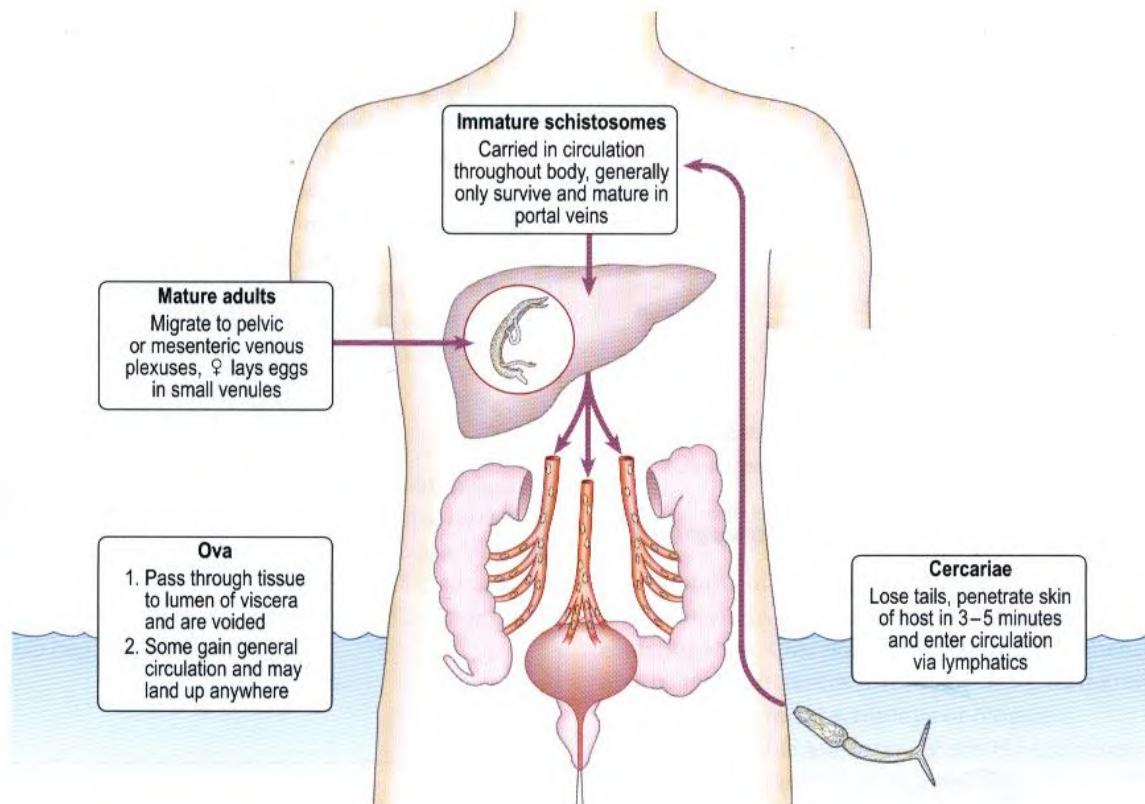
# Trematode (flat) worms

## *Schistosoma* species (blood flukes)

### Life cycle for all species



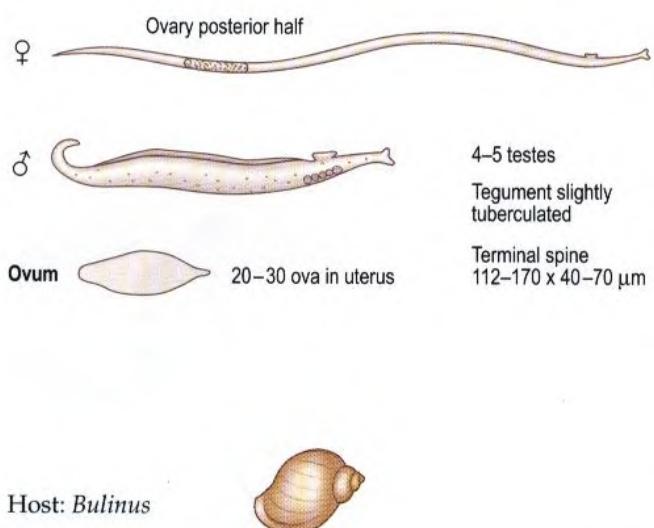
### Life cycle in humans



## *Schistosoma* species (blood flukes) (Continued)

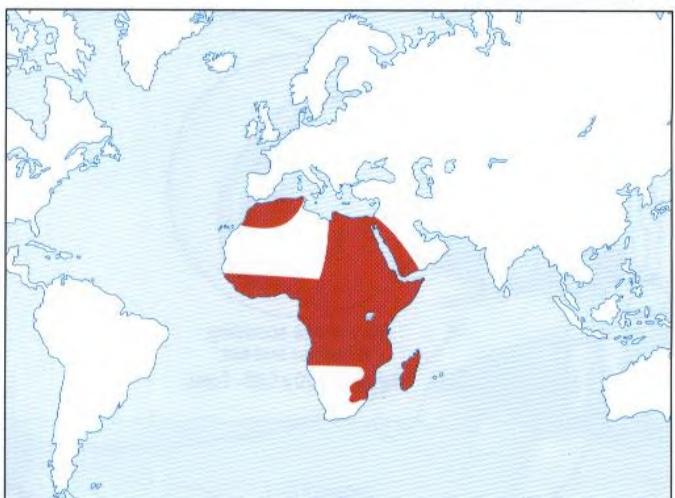
### Morphology

#### *S. haematobium*

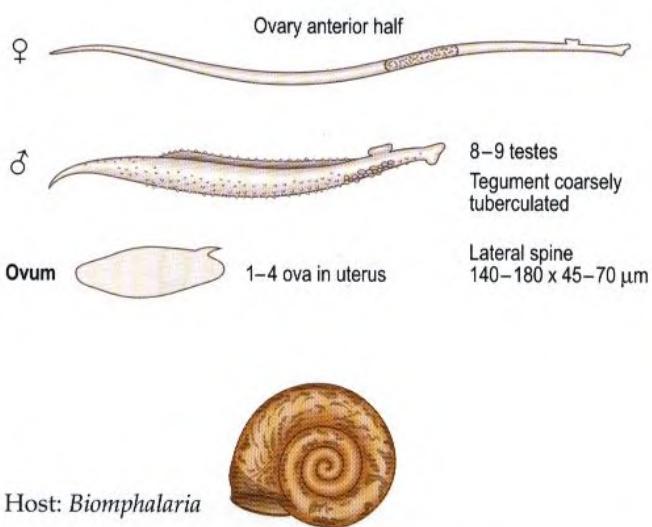


### Distribution

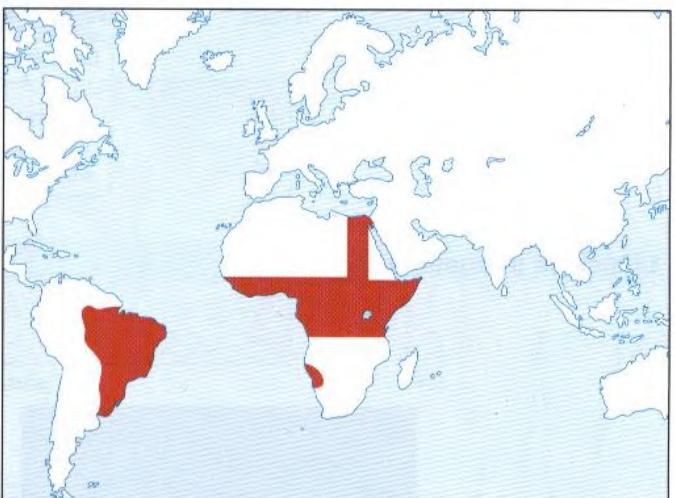
#### *S. haematobium*: 78 million



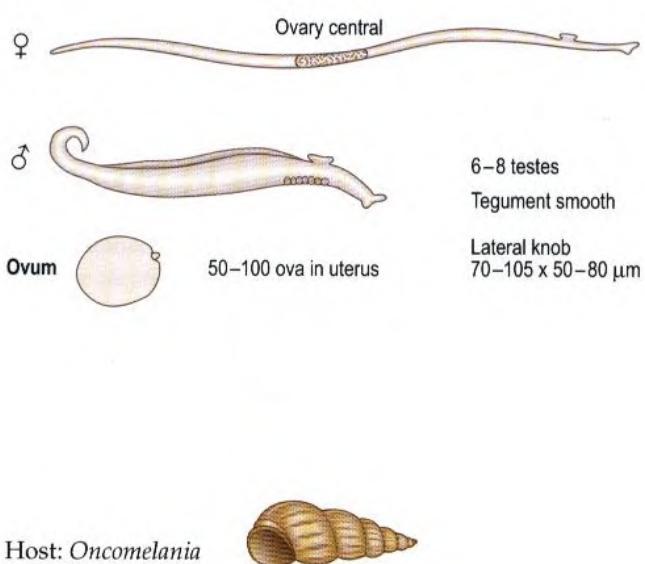
#### *S. mansoni*



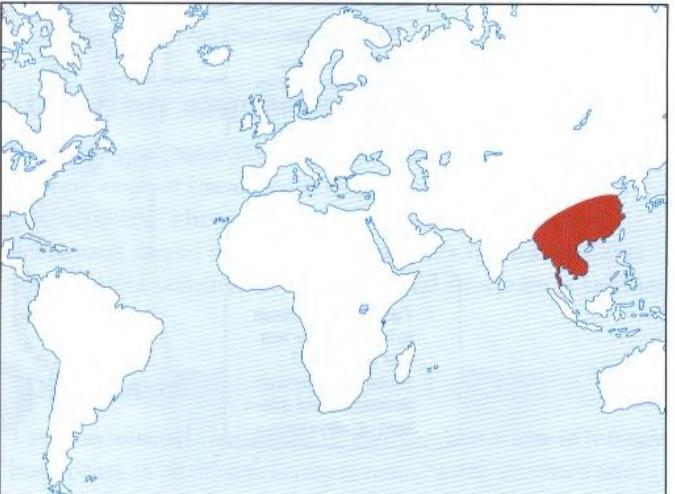
#### *S. mansoni*: 57 million



#### *S. japonicum*



#### *S. japonicum*: 69 million



# Schistosomiasis

## Pathology

### Penetration of the skin by cercariae (1)

Skin penetration may not be apparent. Human and some non-human *Schistosoma* species cause cercarial dermatitis (swimmer's itch). This manifests with papules, macules, vesicles and intense itching.

### Migration and maturation of immature worms (2)

There are general toxic and allergic symptoms including urticaria with eosinophilia, fever, abdominal pain and tender hepatosplenomegaly. This is known as Katayama or snail fever.

### Damage by eggs in tissue (3)

Resulting damage depends on the severity of the parasite load. An inflammatory granuloma forms with epithelial, giant, plasma and eosinophil cells and fibroblasts (Hoepli reaction). There is subsequent fibrosis and calcification. Such damage may be local and/or ectopic.

### Urinary schistosomiasis (4)

Caused by *S. haematobium*. Initial toxic and allergic symptoms are not marked, but the bladder and ureter are typically involved with hyperaemia, terminal haematuria, dysuria and frequency of micturition, papules, papillomata and ulceration. Hypertrophy of the bladder can lead to later contraction. There may be cystitis and calculus formation, with calcification and squamous cell carcinoma. Fistulae may develop. There can also be hydroureter and hydronephrosis. Ectopic lesions are less severe than in other species. Genital schistosomiasis may lead to lumpy semen, haematospermia or wart-like lesions on the vulva.

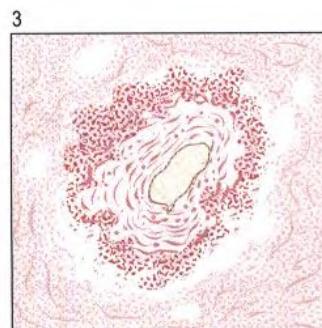
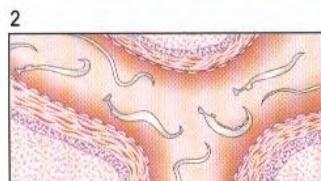
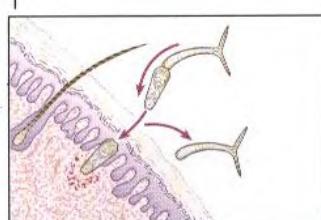
### Intestinal schistosomiasis (5)

Caused by *S. mansoni*. There are marked initial toxic and allergic symptoms. The large intestine and rectum are typically involved with polyposis, papules, abscesses, ulcers, papillomata, fistulae and ova in faeces. The bladder is sometimes involved, with pathology as for urinary schistosomiasis as above. There can be ectopic lesions; the liver is frequently involved (receiving eggs via the portal vein with inflammatory reaction and fibrosis leading to periportal ('pipe-stem') fibrosis with portal hypertension, oesophageal varices, splenomegaly and ascites; there can also be lesions in the brain, spinal cord and lungs).

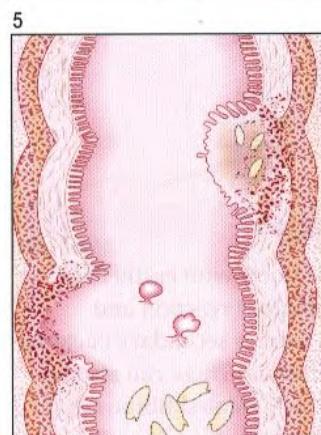
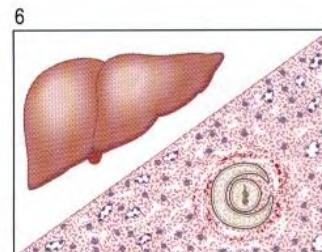
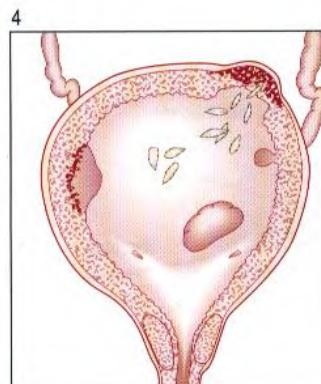
### Oriental schistosomiasis (6)

Caused by *S. japonicum*. Initial toxic and allergic symptoms are marked and can lead to myocarditis and death. Intestinal lesions are similar to those with *S. mansoni* infection, and the small intestine is often involved. The liver is infected as in *S. mansoni*. Hepatic involvement occurs as for *S. mansoni*. The brain may also become involved.

## General



## Particular



## Laboratory diagnosis

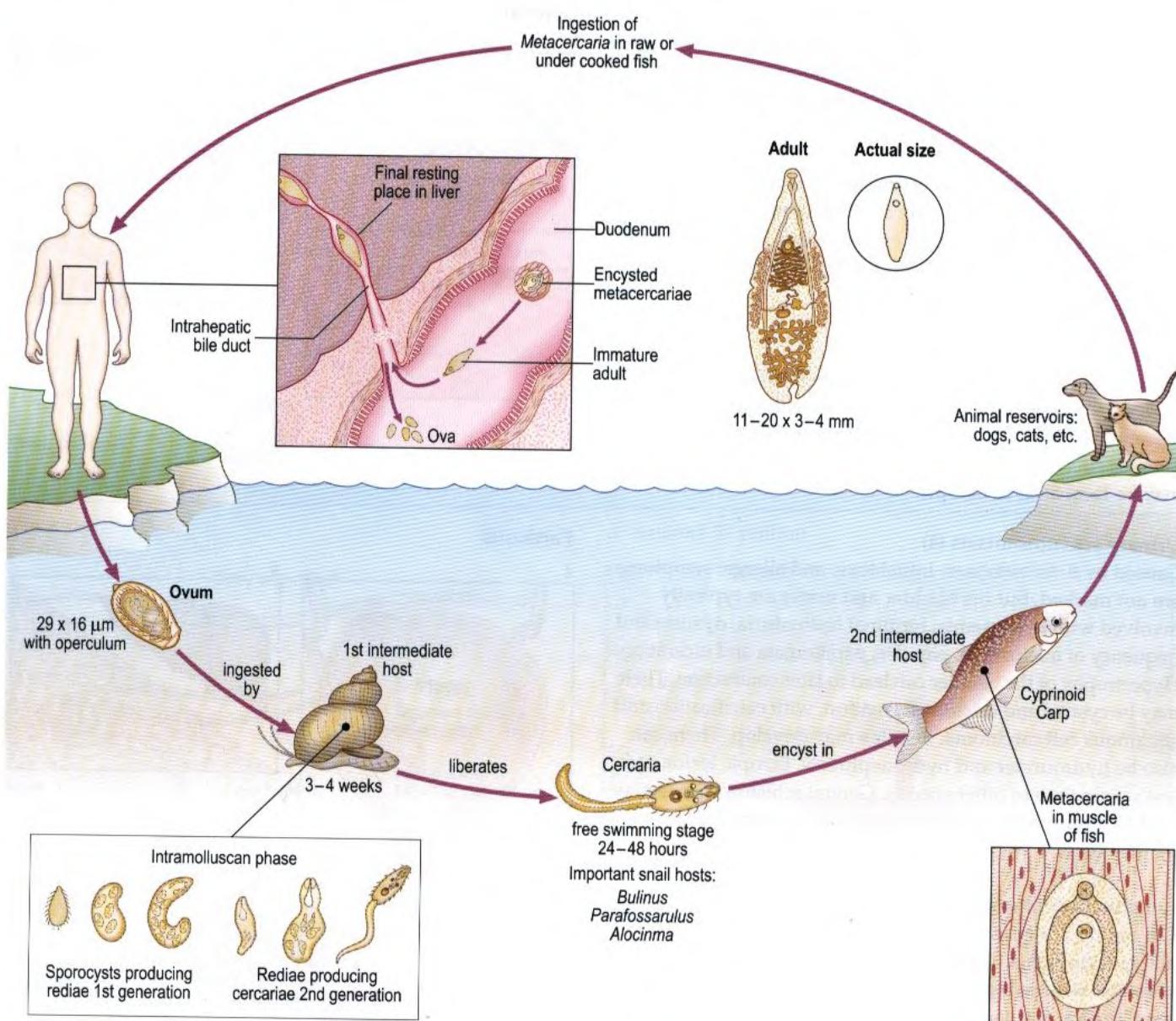
Eosinophilia may be present.

Ova found in terminal urine by Nuclepore filtration or after centrifugation. Ova may also be found in semen. Ova may also be found in faeces directly or using formalin-ether concentration, rectal scrapings or biopsies.

**Serology.** ELISA tests (using soluble egg antigen) are useful 6–12 weeks post-exposure. In many chronic cases, the diagnosis will be made by serology alone.

# *Clonorchis sinensis*, syn. *Opisthorchis sinensis* (Oriental liver fluke)

## Life cycle

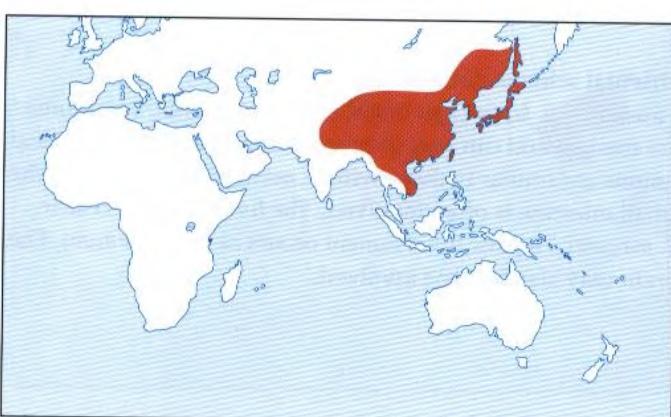


## Pathology and Clinical features

Adult flukes inhabit the distal bile ducts with epithelial proliferation, surrounding inflammatory reaction and ascending cholangitis. Sometimes there is secondary bacterial infection with jaundice and septicaemia. There can also be eosinophilia. All this can lead to thick, dilated fibrous ducts with adenomata of epithelium, bile duct stenosis and cholangiocarcinoma. Many cases are asymptomatic. Acute infection may lead to tender hepatomegaly. Chronic infection can result in anorexia, low-grade fever, epigastric pain and tender hepatomegaly.

## Distribution

28 million infected worldwide.

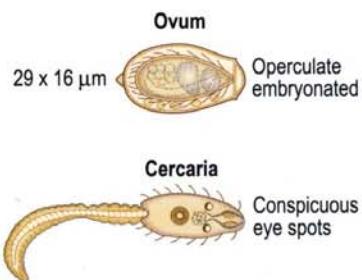
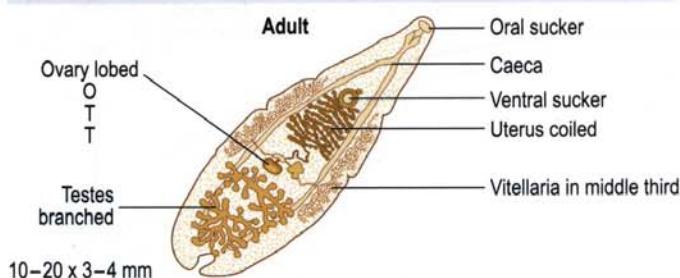


## Laboratory diagnosis

Ova are found in faeces and in bile (via duodenal aspiration or 'string test').

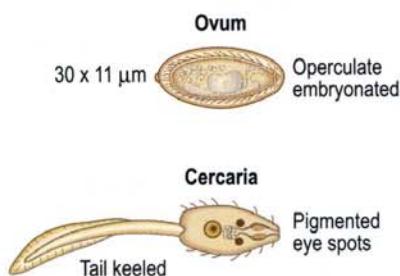
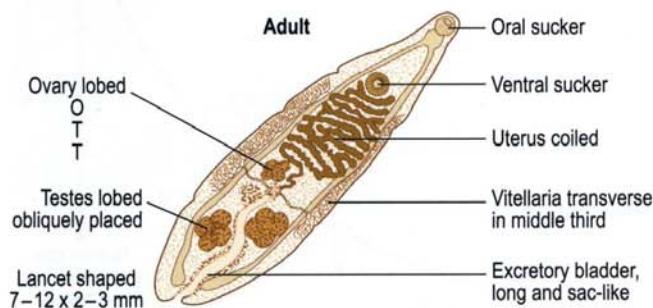
## *Clonorchis sinensis* (continued)

### Morphology

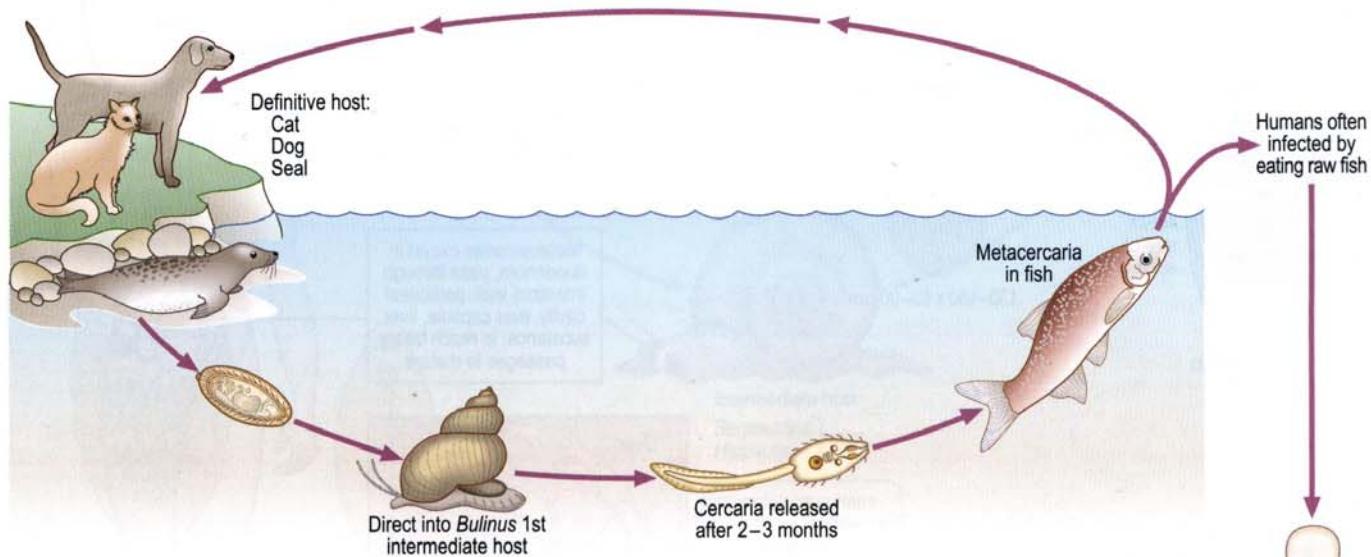


## *Opisthorchis felineus*, *Opisthorchis viverrini* (cat liver fluke)

### Morphology



### Life cycle



### Pathology and Clinical features

There are proliferative changes in the bile ducts. If the infection is massive or repeated then there may be chronic cholangitis. Clinical features are similar to those of clonorchiasis.

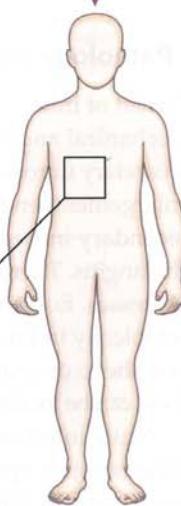
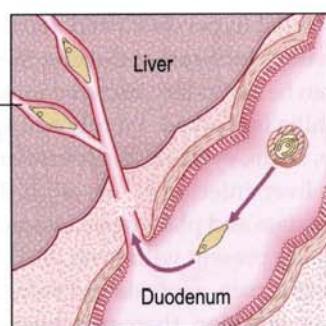
### Laboratory diagnosis

Ova can be found in faeces.

### Distribution

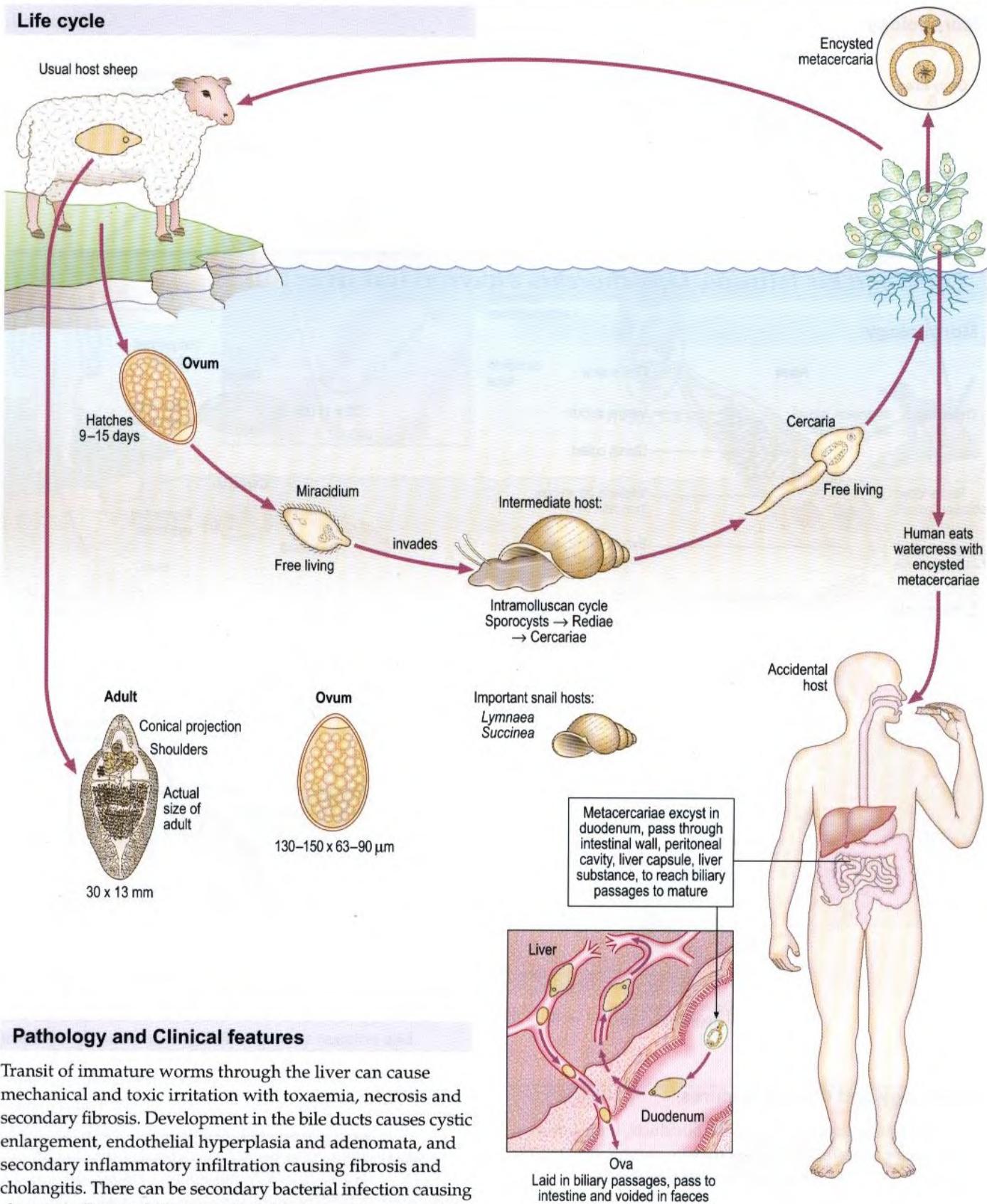
*O. felineus* is found mainly in Eastern Europe and Russia.  
*O. viverrini* occurs in Thailand.

Metacercariae excyst in duodenum  
Migrate through common bile duct to the smaller intrahepatic ducts  
Mature in 3–4 weeks



# Fasciola hepatica (sheep liver fluke)

## Life cycle



## Pathology and Clinical features

Transit of immature worms through the liver can cause mechanical and toxic irritation with toxæmia, necrosis and secondary fibrosis. Development in the bile ducts causes cystic enlargement, endothelial hyperplasia and adenomata, and secondary inflammatory infiltration causing fibrosis and cholangitis. There can be secondary bacterial infection causing abscesses. Eosinophilia is marked. Worms can appear ectopically in lungs, brain, eyes, etc. with similar reactions. If raw sheep or goat's liver, infected by the adult fluke, is eaten there can be local irritation and pharyngeal infection (Halzoun).

Acute infection may present with fever, tender hepatomegaly, epigastric pain, anorexia and vomiting. Jaundice may occur. In chronic infection, there may be no symptoms or epigastric/right upper quadrant pain, hepatomegaly and vomiting.

## Laboratory diagnosis

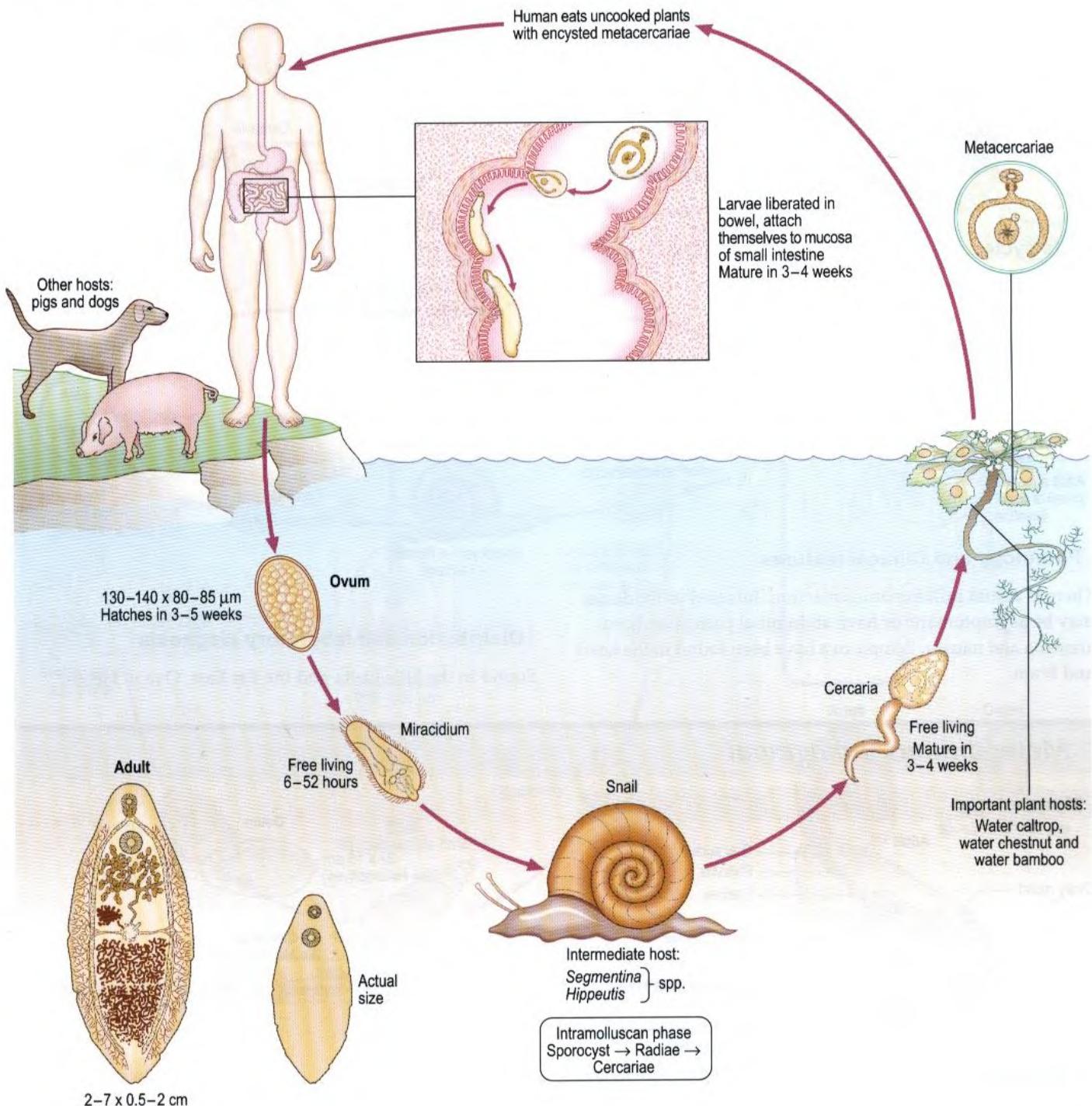
Ova are found in faeces. Serology (IFAT) is available.

## Distribution

The fluke is found in all sheep-rearing countries. About 1 million people are infected worldwide.

# Fasciolopsis buski

## Life cycle



## Pathology and Clinical features

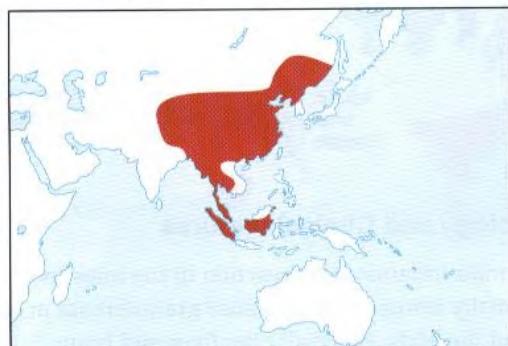
There is localized inflammation at the site of attachment with haemorrhages and occasional abscesses. There is also eosinophilia. Lightly infected individuals may be asymptomatic. Diarrhoea, abdominal pain, anorexia, nausea and vomiting may occur.

## Laboratory diagnosis

Ova, and sometimes adults, are found in faeces.

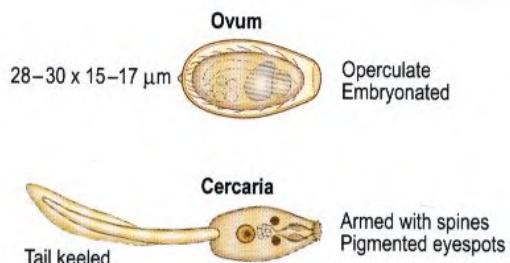
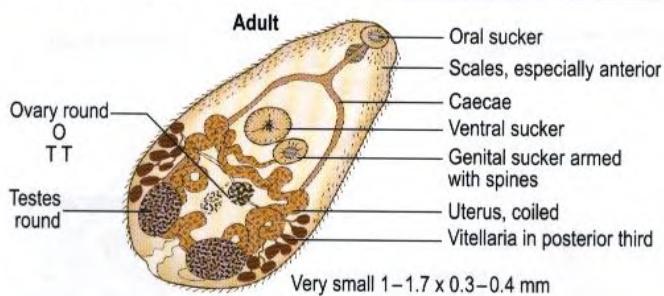
## Distribution

15 million infected worldwide.

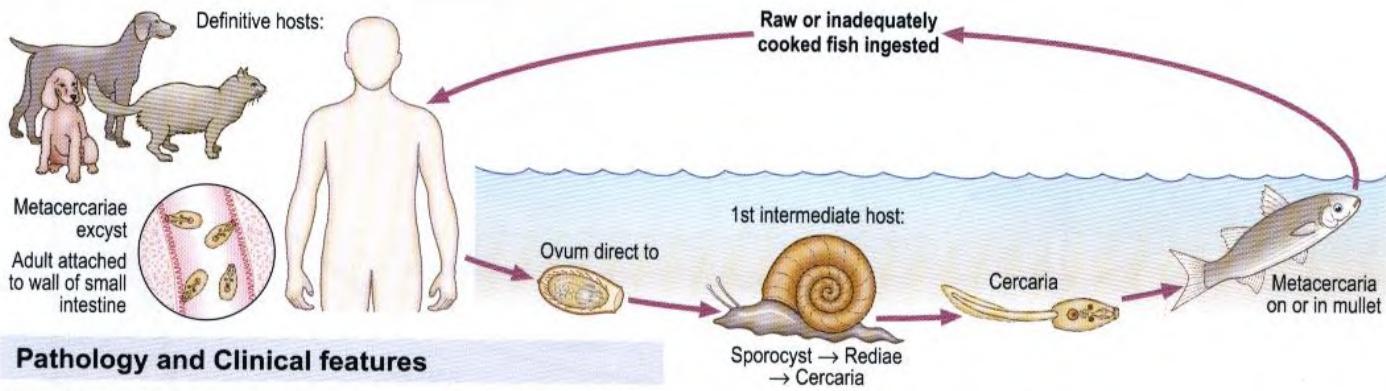


## *Heterophyes heterophyes*

### Morphology



### Life cycle



### Pathology and Clinical features

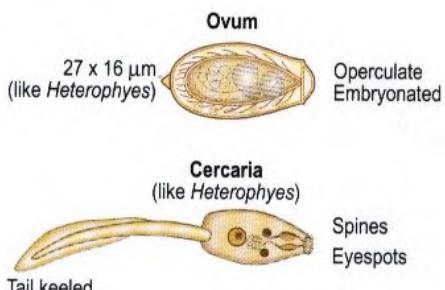
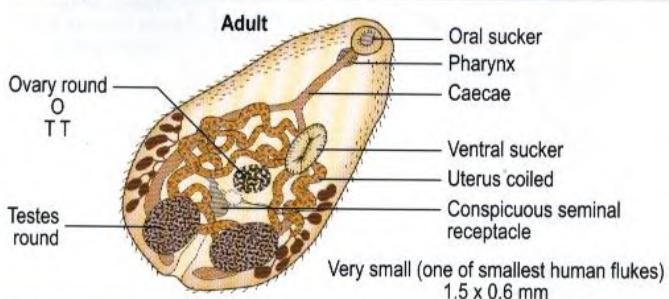
There is a mild inflammatory reaction. Infected individuals may be asymptomatic or have abdominal pain, diarrhoea, anorexia and nausea. Ectopic ova have been found in the heart and brain.

### Distribution and laboratory diagnosis

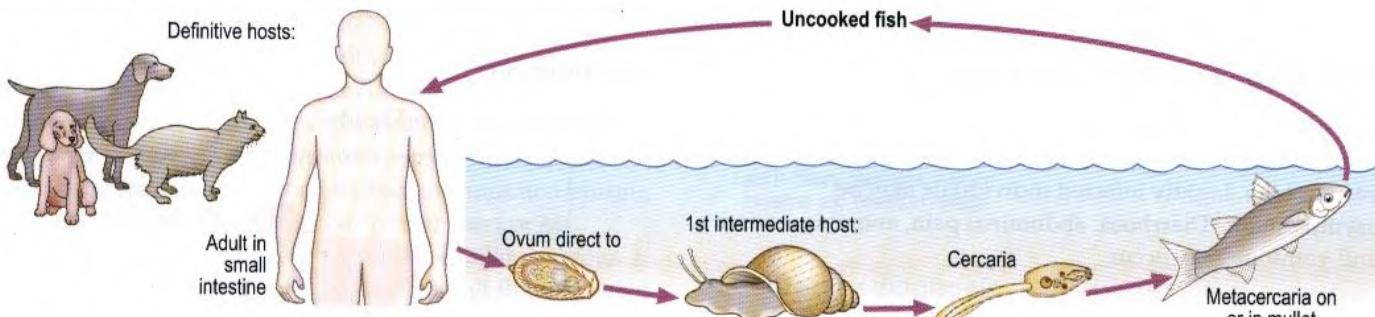
Found in the Nile Delta and the Far East. Ova in faeces.

## *Metagonimus yokogawai*

### Morphology



### Life cycle



### Pathology and Clinical features

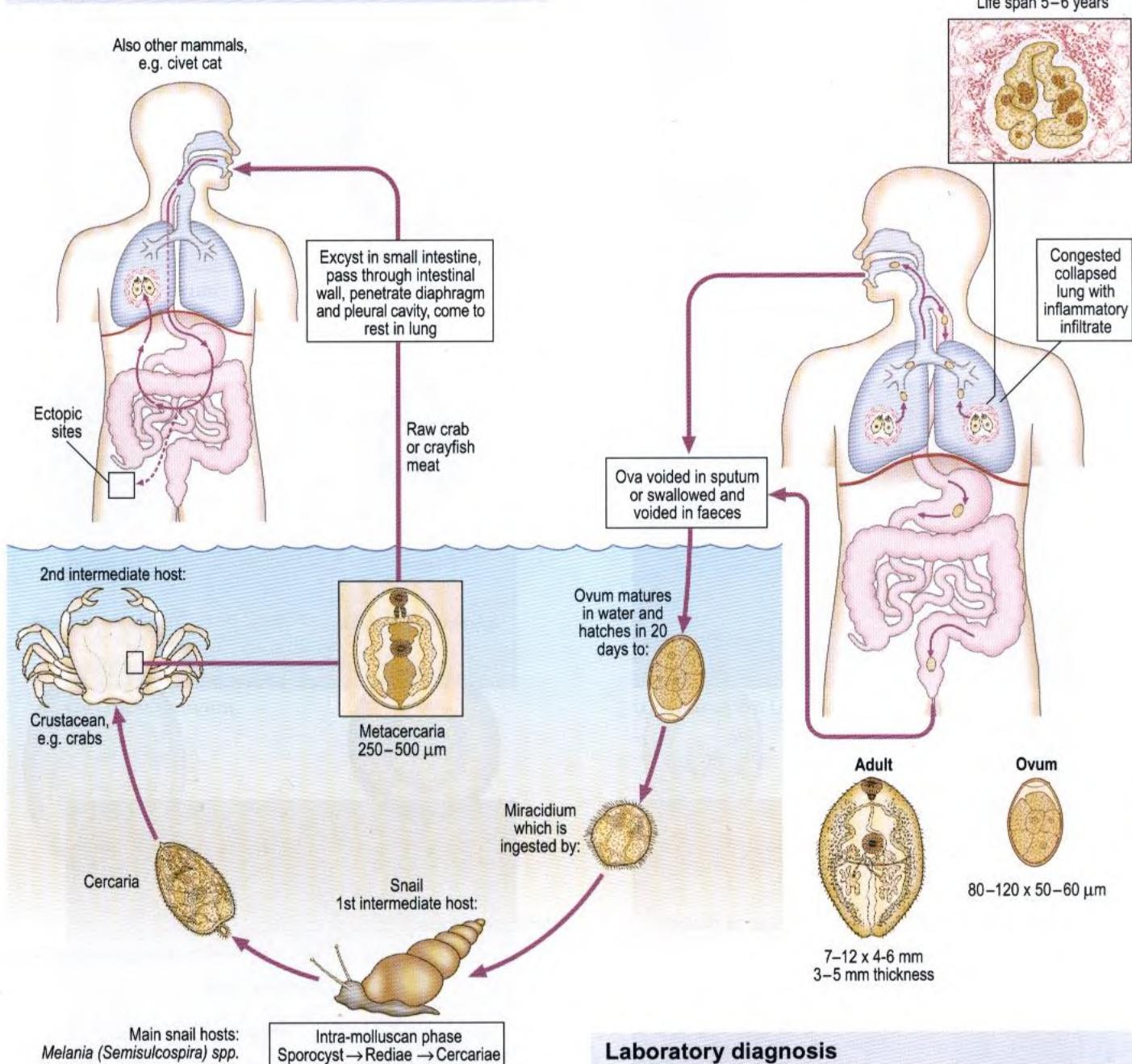
Causes mild inflammatory reaction in the intestine. Occasionally ectopic ova can cause granulomata in other organs of the body, especially the liver and brain.

### Distribution

Prevalent in the Far East.

# *Paragonimus westermani* (lung fluke)

## Life cycle

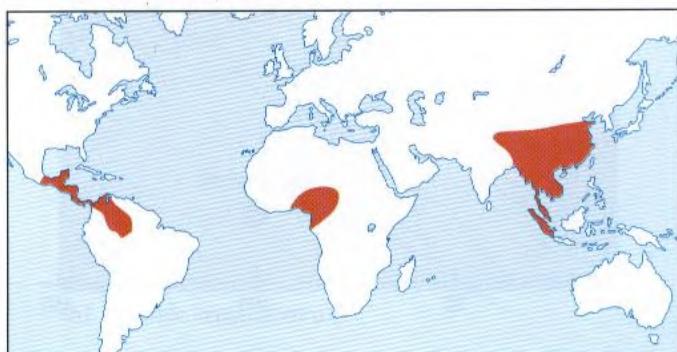


## Laboratory diagnosis

Ova are found in sputum after KOH digestion or faeces after formalin-ether concentration. Serological tests, when available, are CF or ELISA (using extract of adult flukes as antigen) or gel diffusion. Chest X-ray or CT can also be used.

## Distribution

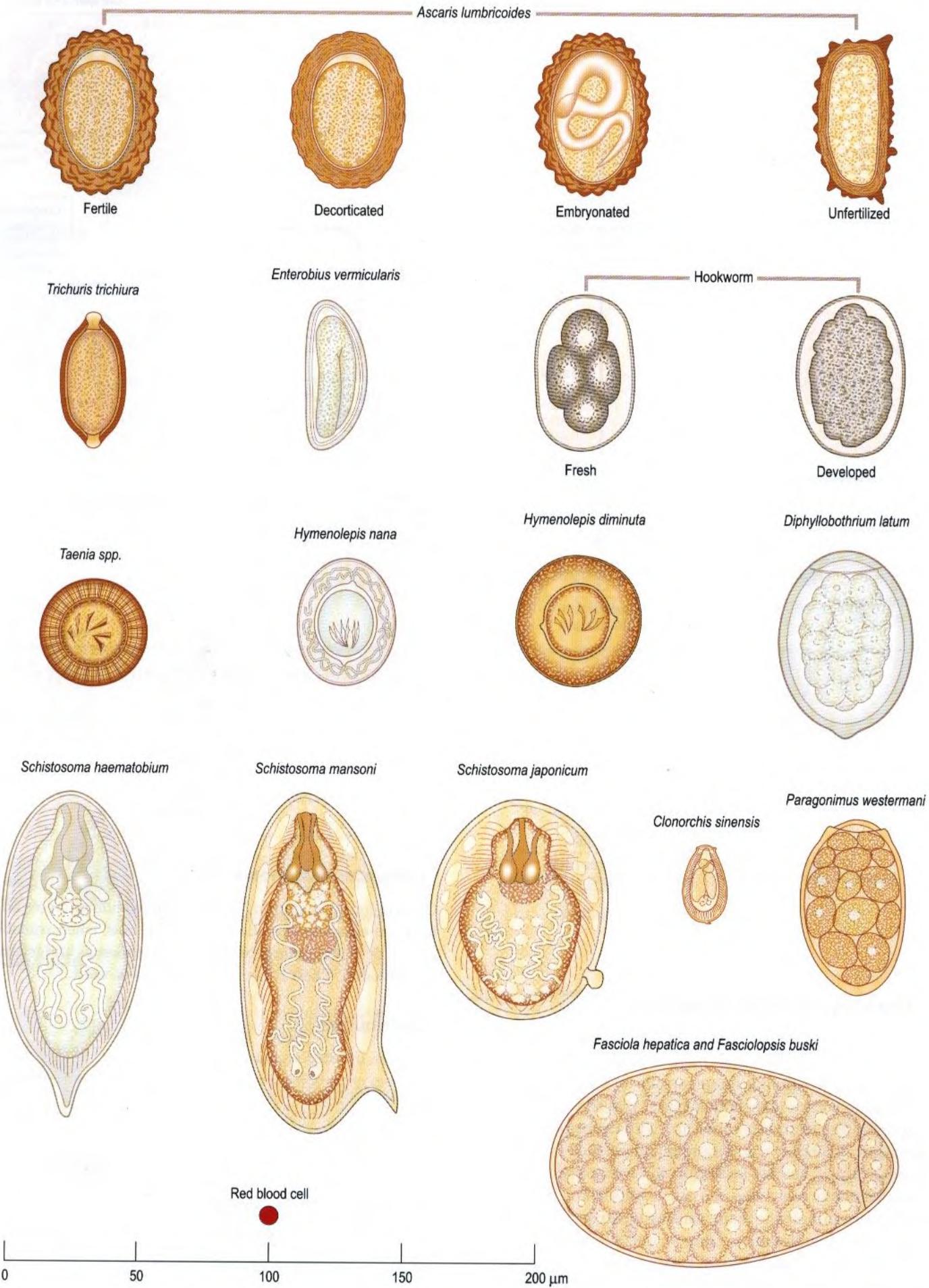
5 million infected worldwide.



## Pathology and Clinical features

The initial invasion has little pathological effect on the host. On localization in the lungs, there is tissue reaction leading to formation of a fibrous tissue capsule (of a slate blue colour) containing worms (generally in pairs), ova and inflammatory infiltrate. The capsule is connected with the respiratory passages. Secondary complications of these lung cysts include bronchiectasis, abscess formation and haemoptysis. Localization in other sites can cause cysts in any part of the body (for example the brain, causing epilepsy). Eosinophilia is a general manifestation. Chronic infection may be asymptomatic. Cough, brown gelatinous sputum, chest discomfort, shortness of breath and pleuritic chest pain may occur.

## Common helminth ova





(a) *Ascaris* ovum



(e) *Toxocara canis* ova



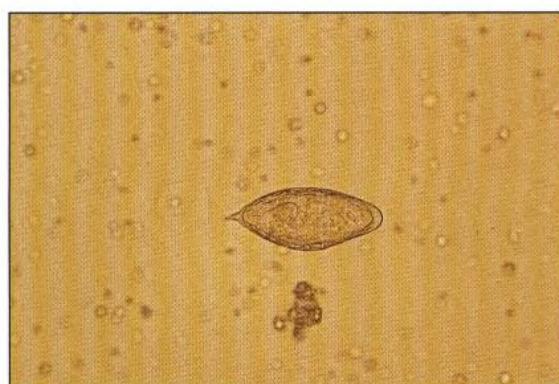
(b) *Trichuris* ovum



(f) Hookworm (*Ancylostoma*) ovum



(c) *Hymenolepis nana* ovum



(g) *Schistosoma haematobium* ovum



(d) *Schistosoma mansoni* ovum



(h) *Fasciola hepatica* ovum



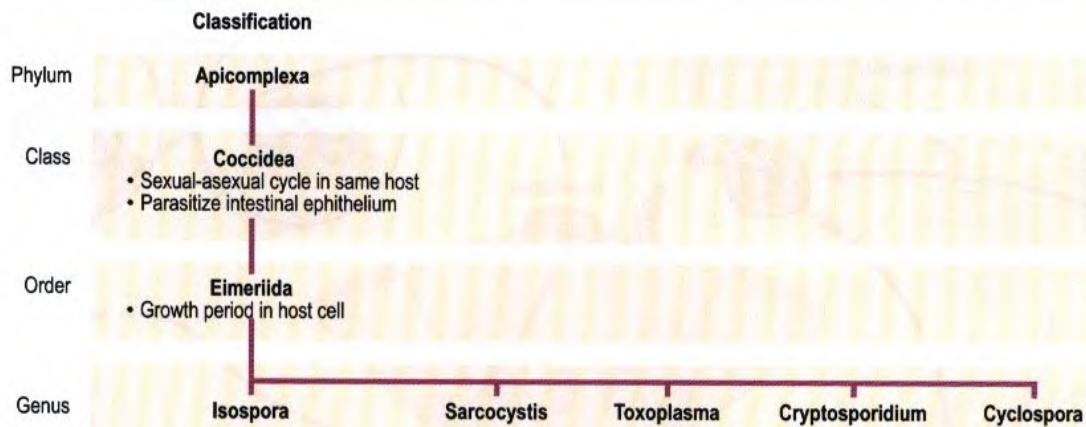
# Protozoology

# An outline classification of the parasitic protozoa of humans

| Empire    | Kingdom                | Phylum                 | Class                          | Order  | Genus   |
|-----------|------------------------|------------------------|--------------------------------|--|---|
| Eukaryota | Archezoa Haeckel 1894  | Metamonada             | Trepomonadea<br>Retortamonadea | Diplomonadida<br>Enteromonadida<br>Retortamonadida | <i>Giardia</i><br><i>Enteromonas</i><br><br><i>Chilomastix</i><br><i>Retortamonas</i>                           |
|           |                        | Microspora             | Microsporea                    | Microsporida                                       | <i>Encephalitozoon</i><br><i>Enterocytozoon</i><br><i>Nosema</i><br><i>Septata</i><br><i>Trachipleistophora</i> |
|           | Protozoa Goldfuss 1818 | Percolozoa             | Heterolobosea                  | Schizopyrenida                                     | <i>Naegleria</i>  |
|           |                        | Parabasala             | Trichomonadea                  | Trichomonadida                                     | <i>Dientamoeba</i><br><i>Trichomonas</i>  |
|           |                        | Euglenozoa             | Kinetoplastidea                | Trypanosomatida                                    | <i>Leishmania</i><br><i>Trypanosoma</i>   |
|           |                        | Ciliophora             | Litostomatea                   | Vestibuliferida                                    | <i>Balantidium</i>  |
|           |                        | Apicomplexa (Sporozoa) | Coccidea                       | Eimeriida  | <i>Cryptosporidium</i><br><i>Cyclospora</i><br><i>Isospora</i><br><i>Sarcocystis</i><br><i>Toxoplasma</i>       |
|           |                        |                        | Haematozoea                    | Haemosporida<br>Piroplasmida                       | <i>Plasmodium</i><br><i>Babesia</i>   |
|           |                        | Rhizopoda              | Lobosea                        | Acanthopodida                                      | <i>Acanthamoeba</i><br><i>Balamuthia</i>  |
|           |                        |                        | Entamoebidea                   | Euamoebida   | <i>Endolimax</i><br><i>Entamoeba</i><br><i>Iodamoeba</i>  |

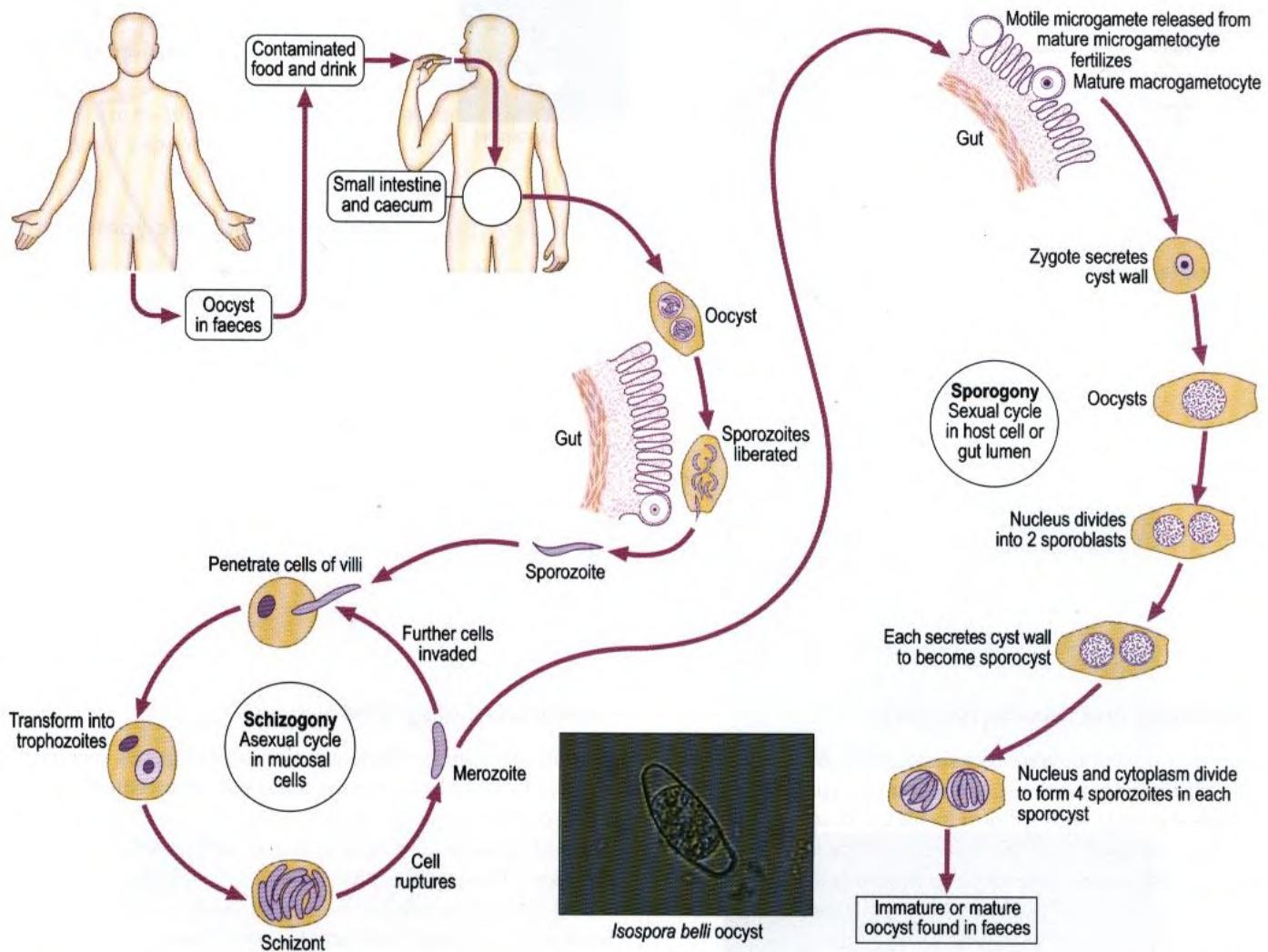
# Intestinal protozoa

## Coccidia



### *Isospora belli* (causing coccidiosis in humans)

#### Life cycle



#### Pathology and Clinical features

Small bowel mucosal atrophy. Watery diarrhoea or steatorrhoea, weight loss and sometimes cholecystitis occur in AIDS.

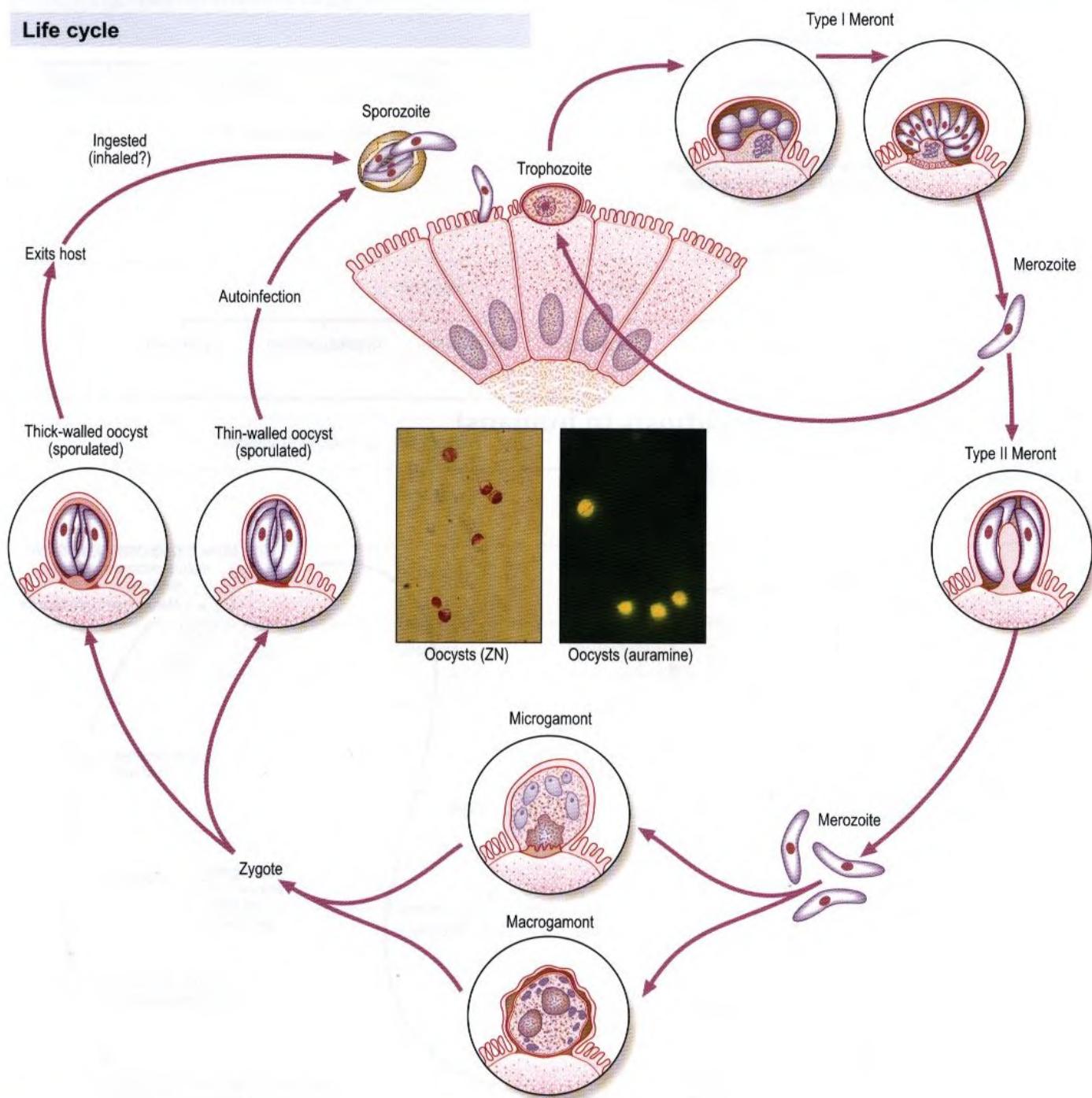
#### Laboratory diagnosis

Oocysts are seen in formalin-ether concentration of faeces or modified Ziehl-Neelsen or auramine-stained faecal smears. Intraepithelial parasites may be seen in small bowel biopsies.

## Coccidia (continued)

### *Cryptosporidium parvum*

#### Life cycle



#### Pathology and Clinical features

In the immunocompetent, there is short-term enteropathy with self-limiting diarrhoea. In the immunocompromised, for example a patient with AIDS or a child with severe combined immunodeficiency, there is chronic diarrhoea with malabsorption and weight loss. Extraintestinal infection of the respiratory tract, biliary tract and pancreas may occur.

#### Laboratory diagnosis

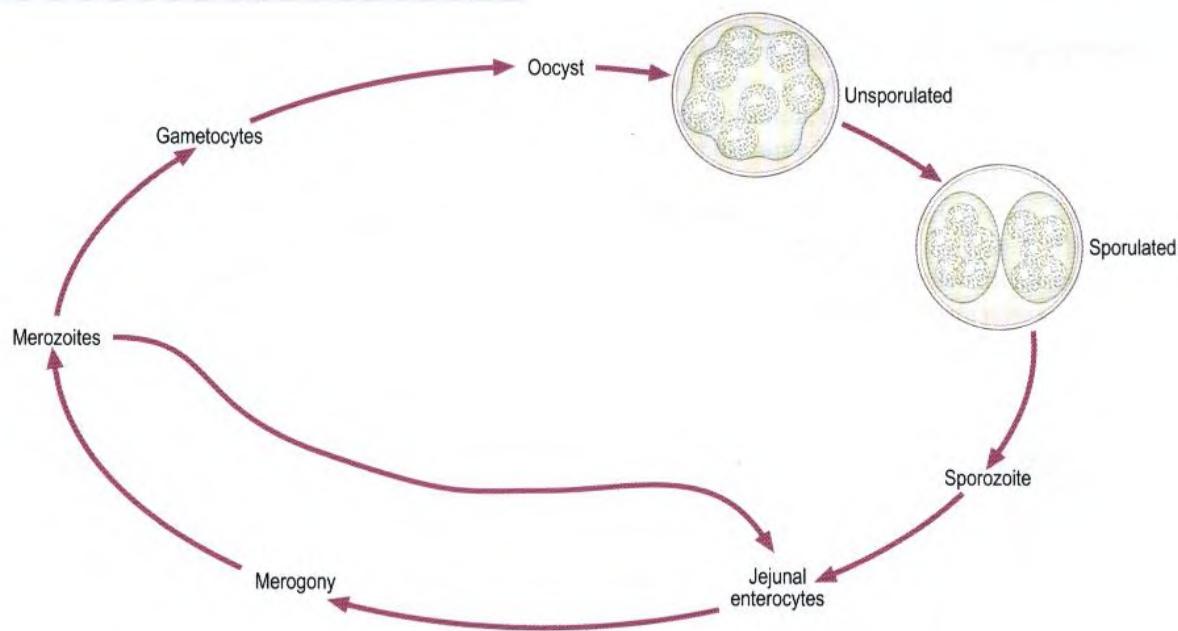
Oocysts (4–5 µm in diameter) are found in faeces, using modified Ziehl-Neelsen stain, auramine or specific FITC labelled monoclonal antibody staining. They can also be found in faecal concentrates, duodenal aspirates and duodenal biopsies. The oocysts are very small (5 µm in diameter) and round. Parasites may also be seen in small bowel biopsies. Sucrose floatation is an alternative to formalin–ether concentration.

#### Distribution

*Cryptosporidium* have a cosmopolitan distribution. Human and farm animal strains exist; both can cause human disease. Human infection is usually waterborne.

## *Cyclospora cayetanensis*

### Life cycle



## Cyclospora

Oocysts are 8–10 µm in diameter with a central morula of refractile spheres when unsporulated. These mature into a final division of 2 sporocysts.

### Pathology and Clinical features

Acute onset of diarrhoea, followed by steatorrhoea. Colicky abdominal pain and malaise. Partial villous atrophy may be seen.

### Distribution

Widespread, probably worldwide.

### Laboratory diagnosis

Oocysts are seen in faeces unsporulated when first passed. Diagnosis is either by formalin–ether concentration, modified Ziehl–Neelsen stain or by autofluorescence.



Unsporulated



Sporulated



Modified Ziehl–Neelsen stain

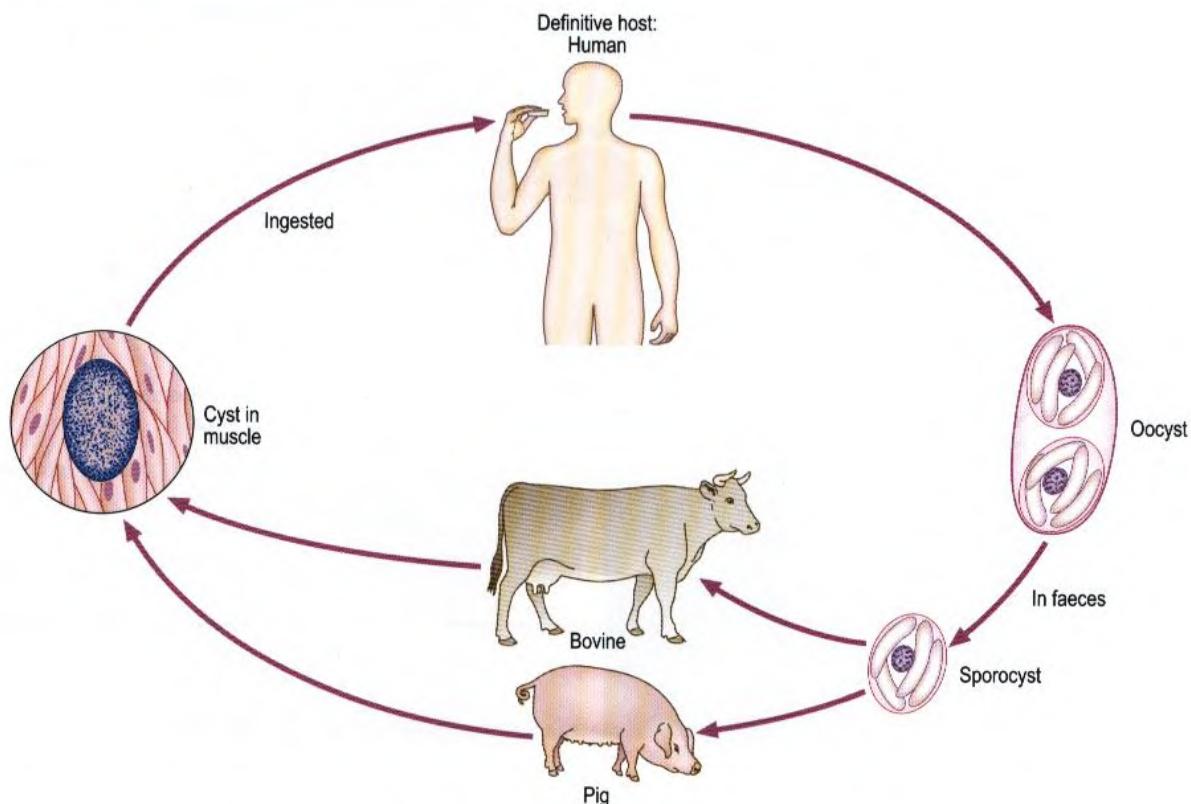


Autofluorescence

## Coccidia (continued)

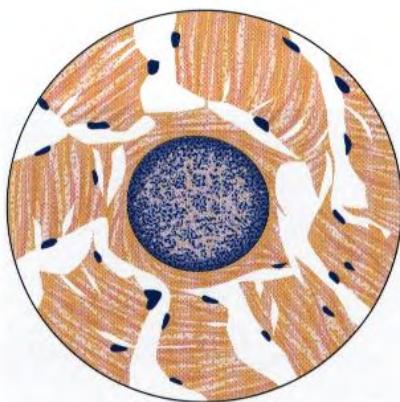
### *Sarcocystis hominis*

#### Probable life cycle

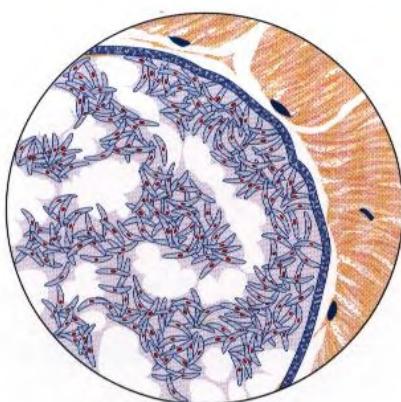


Occasionally humans can act as intermediate hosts for *Sarcocystis* of other animals.

#### Morphology



Cyst in human muscle  
Miescher's tube x 100



Enlarged portion of  
Miescher's tube showing  
Rainey's corpuscles (each  
12–16 x 4–9 µm): from  
a human case

#### Pathology and Clinical features

The intestinal stages produce diarrhoea and abdominal pain. The clinical significance of muscle cysts is unknown.

#### Laboratory diagnosis

Oocysts or free sporocysts are found in faeces. Histological examination of biopsy specimens may show the sexual stages in the intestinal epithelium.

Histology is the only way to diagnose the presence of sarcocysts, although these are almost invariably incidental findings.

## Microsporidia—general characteristics

All are obligate intracellular parasites. The vast majority of species are in invertebrates, especially insects, lower vertebrates and fish. Only a few have been reported from warm-blooded vertebrates.

They are considered to be primitive organisms. Their evolutionary history has been predicted from their prokaryote-like ribosomal characteristics — the absence of a separate 5.8S rRNA and the nucleotide sequence of the small subunit (16S) rRNA. They have no mitochondria. The infective stages are highly-resistant spores. These are very uniform in size for a given species.

When spores are ingested by a new host, the cells are penetrated by means of an apparatus known as the polar tube. When this is fully extended, the sporoplasm passes through the tube, to be inoculated into the cytoplasm of the host cell.

Following infection, there follows a phase of multiplication by binary or multiple fission (merogony). The transition to the spore-producing stage (sporogony) is heralded by the secretion of an electron dense surface coat — this will form the future exospore layer of the spore wall. The primary sporogonic cells are **sporonts**, which divide into **sporoblasts**, which mature into **spores**, which are released when the host cell ruptures.

Common species of microsporidia reported from humans. Most are AIDS associated.

| Species                                       | Localization             | Pathogenesis      |
|---|--------------------------|-------------------|
| <i>Encephalitozoon cuniculi</i>               | Generalized, brain, etc. | Convulsions, etc. |
| <i>Encephalitozoon hellem</i>                 | Corneal epithelia        | Keratopathy       |
| <i>Enterocytozoon bieneusi</i>                | Enterocytes—gut          | Diarrhoea         |
| <i>Encephalitozoon (Septata) intestinalis</i> | Enterocytes—gut          | Diarrhoea         |
| <i>Nosema connori</i>                         | Generalized              | Multi-organ       |
| <i>Nosema corneum</i>                         | Corneal stroma           | Keratitis         |
| <i>Microsporidium africanum</i>               | Corneal stroma           | Keratitis         |
| <i>Pleistophora</i> sp.                       | Muscle fibres            | Myositis          |

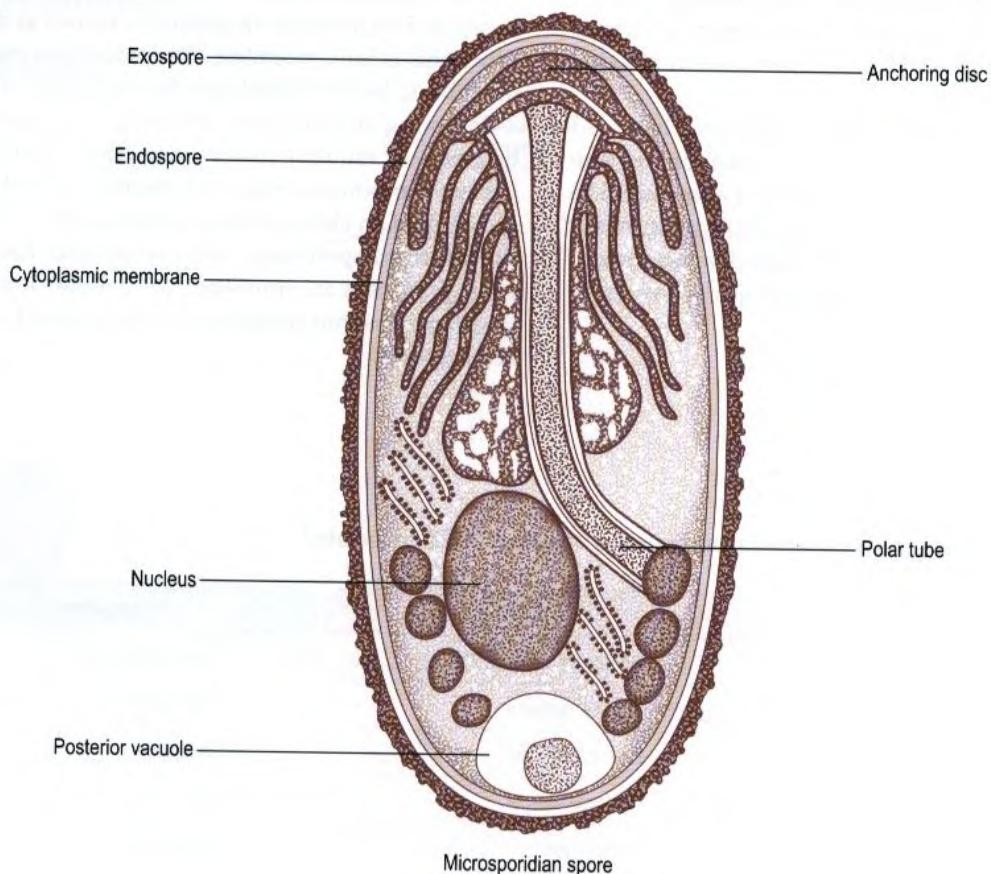
Infections of the gastrointestinal tract and urinary system can be detected by the presence of spores in faeces or urine. Spores from these sites can be visualized by staining them with the modified trichrome stain.

The spores of microsporidia are very small—1 x 0.5 µm (See below)

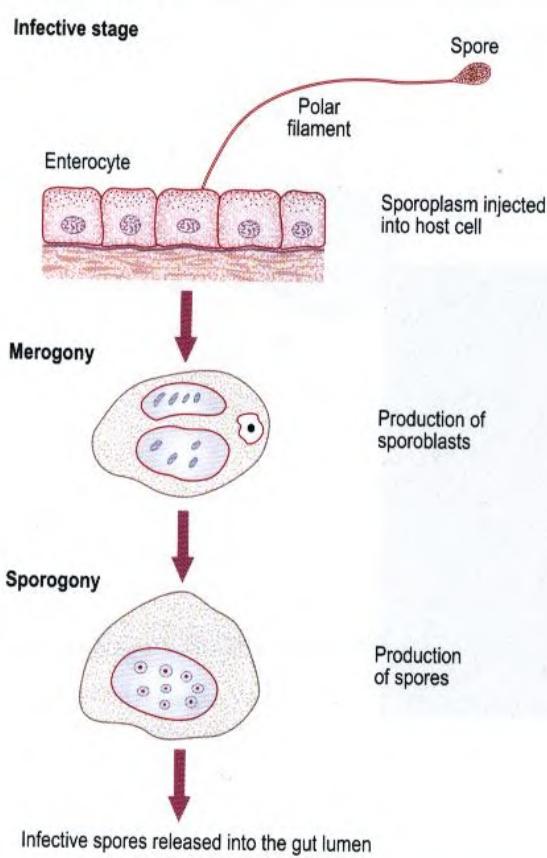


*Enterocytozoon bieneusi* spores

## Microsporidia (continued)



### Life cycle



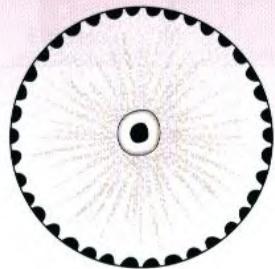
### Laboratory diagnosis

Alternative staining methods for microsporidial spores in stool samples are modified trichrome stain and uvitex 2B or calcofluor fluorescence.

# Amoebae

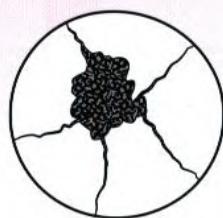
## *Entamoeba*

- Generally one nucleus in trophozoite
- Small karyosome at or near centre
- Nuclear membrane lined with chromatin granules
- Forms cysts



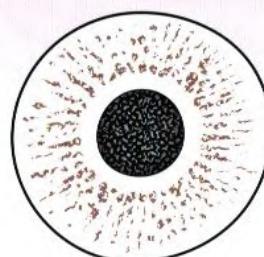
## *Endolimax*

- Generally one nucleus in trophozoite
- Large irregular karyosome attached to nuclear membrane
- No peripheral chromatin
- Forms cysts



## *Iodamoeba*

- Generally one nucleus in trophozoite
- Large karyosome surrounded by achromatic granules
- No peripheral chromatin
- Forms cysts



## *Dientamoeba*

- Minute
- Generally binucleate
- Central particulate karyosome
- No peripheral chromatin
- No cystic stage



## Species

*Entamoeba histolytica/dispar*  
*Entamoeba coli*  
*Entamoeba hartmanni*  
*Entamoeba polecki*

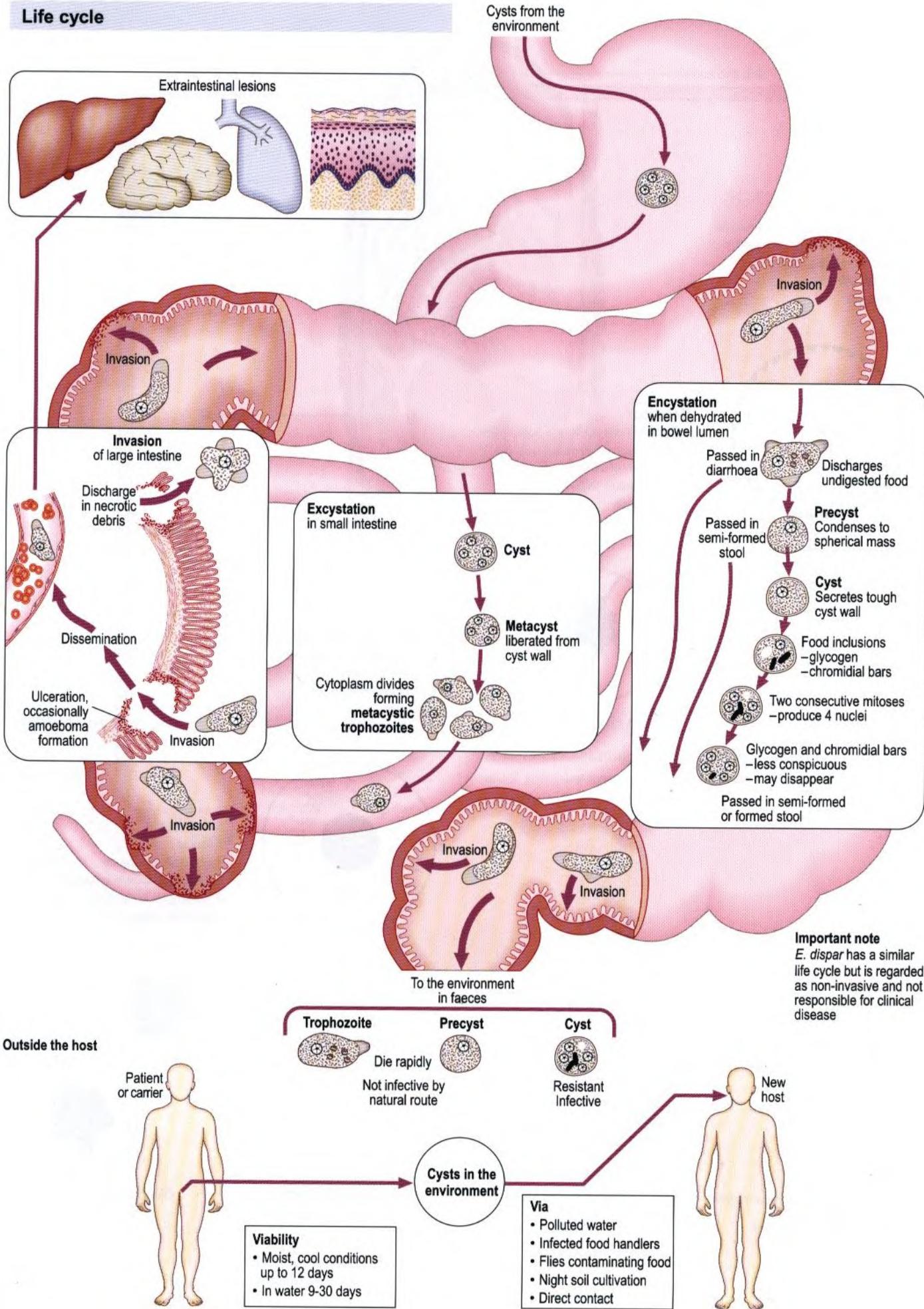
*Endolimax nana*

*Iodamoeba bütschlii*

*Dientamoeba fragilis*

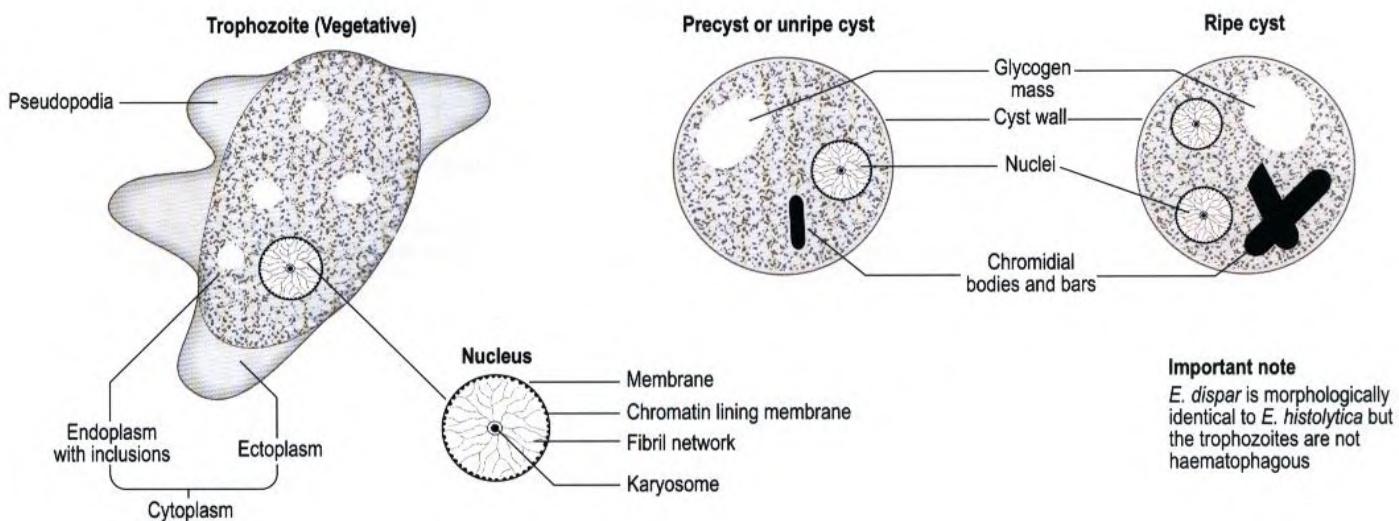
# *Entamoeba histolytica* (causing amoebiasis)

## Life cycle



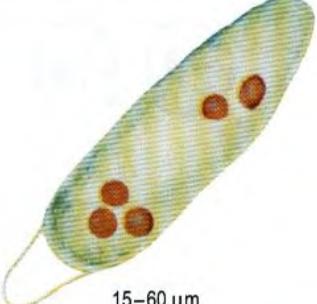
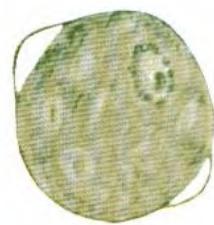
## Morphology

### General – nomenclature



**Particular - Includes differentiation from *Entamoeba coli*, an intestinal commensal.**

### Unstained preparations

| <i>E. histolytica</i>   | Trophozoite  |   |  | <i>E. coli</i>  |
|---|--|---|--|---|
| <br>15–60 µm | Granular<br>Clear finger-like<br><b>Active</b><br>Purposeful<br>Generally invisible<br><b>Red blood cells (RBCs)</b> | Cytoplasm<br>Pseudopodia<br>Movement<br>Nucleus<br>Inclusions | Conspicuously granular<br>Blunt<br><b>Sluggish</b><br>Not purposeful<br>Ring refractile granules with eccentric karyosome<br>Vacuoles, crystals, vegetable cells, bacteria, <b>no RBCs</b> | <br>15–50 µm |

### Precyst and unripe cyst

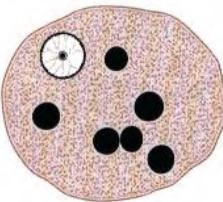
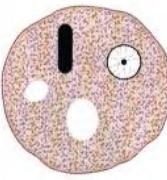
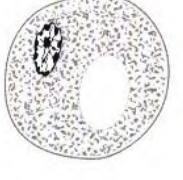
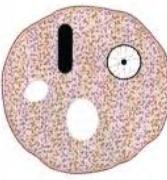
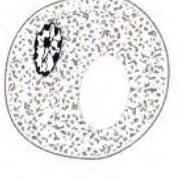
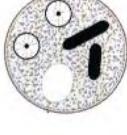
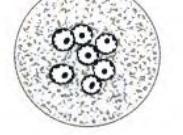
|   |  |                                    |  |   |
|---|--|------------------------------------|--|---|
|  | Granular<br>May be refractile ring<br>Rod-like refractile chromidial bars<br>Glycogen masses | Cytoplasm<br>Nucleus<br>Inclusions | Granular<br>Visible as refractile ring<br>May be slender refractile chromidial bars<br>Glycogen masses |  |
|---|--|------------------------------------|--|---|

### Ripe cyst

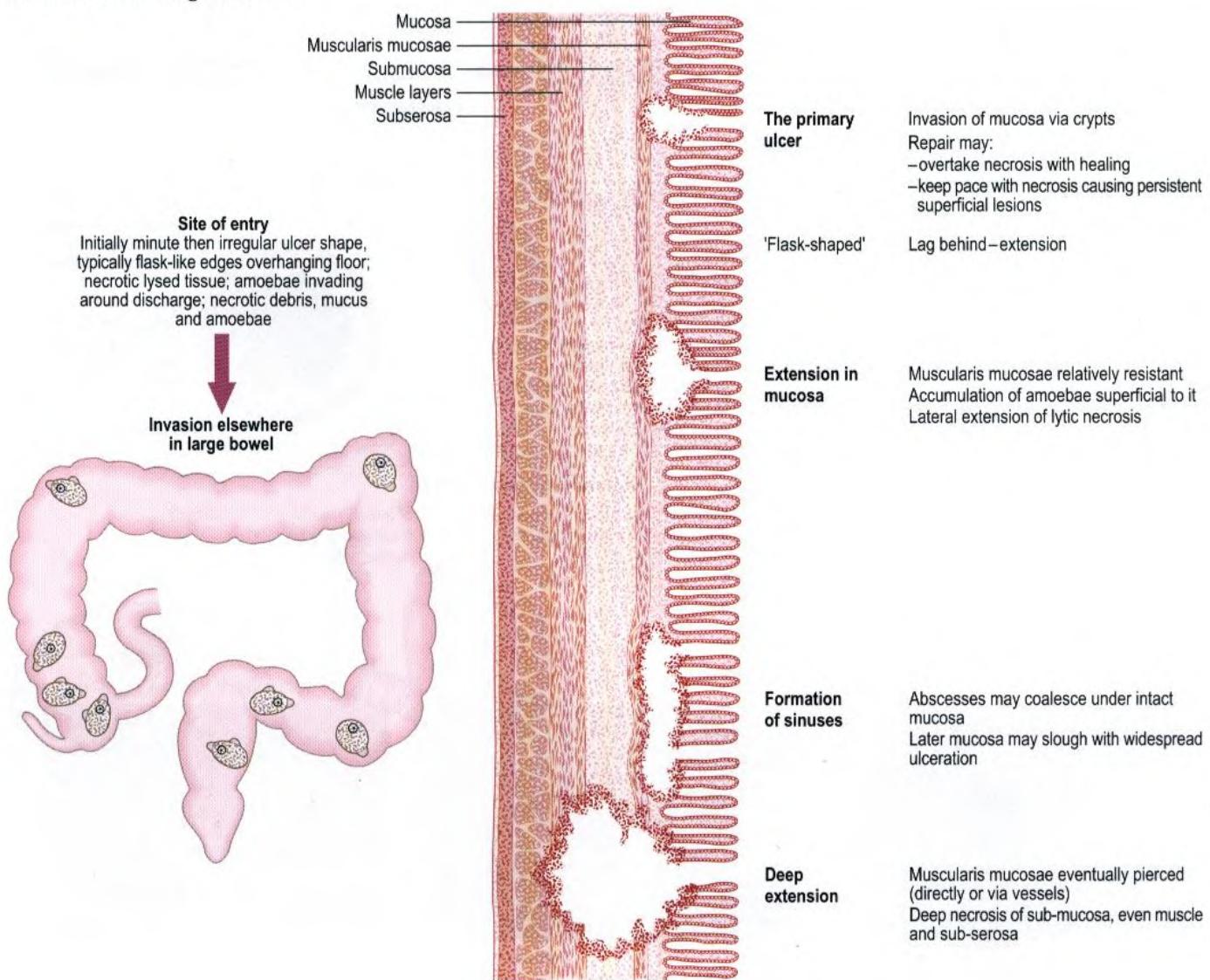
|   |   |                                       |   |   |
|---|---|---------------------------------------|---|---|
| <br>10–20 µm | Round<br>Refractile<br>1–4 refractile nuclei with central karyosome<br>Refractile chromidial bars often present | Shape<br>Wall<br>Nuclei<br>Inclusions | Round<br>Conspicuous refractile double outline<br>1–8 refractile nuclei with eccentric karyosomes<br>Only rudimentary slender chromidial bars | <br>10–33 µm |
|---|---|---------------------------------------|---|---|

## *Entamoeba histolytica* (causing amoebiasis) (continued)

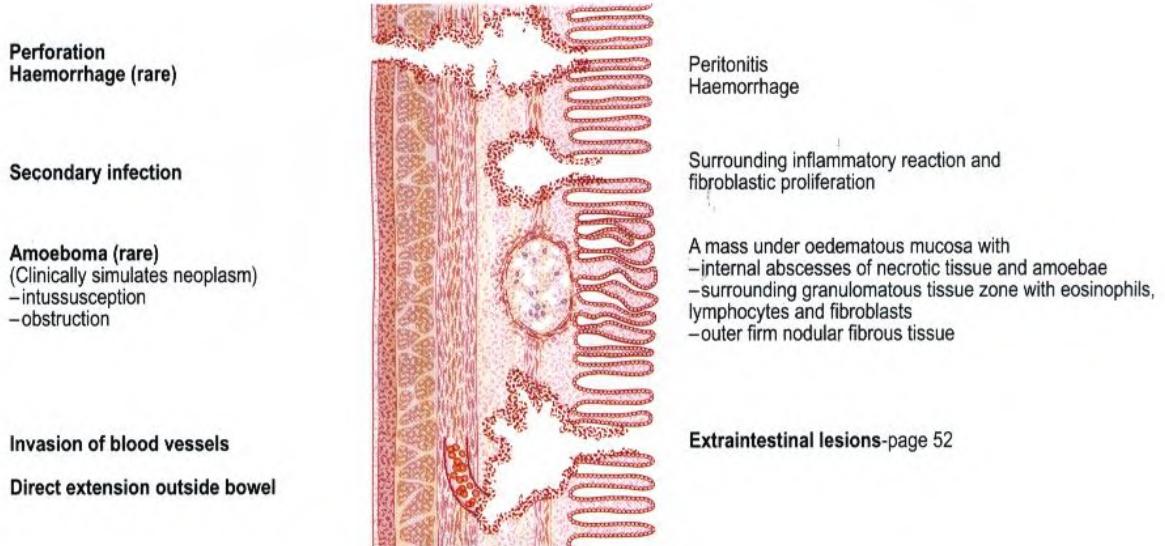
### Morphology (continued)

| Iodine preparations   |   |  |   |
|---|---|--|---|
| <i>E. histolytica</i>   | Precyst   | <i>E. coli</i>                               |   |
|      | Brown, diffuse<br>Finely granular yellow green<br>Yellow ring with central yellow dot (karyosome)       | Glycogen<br>Cytoplasm<br>Nucleus             | Brown, compact<br>Conspicuous granularity<br>Nuclear membrane with eccentric karyosome easily recognised  |
|    |   |  |   |
| Stained by iron haematoxylin  |   |  |   |
| Trophozoite   |   |  |   |
|     | Purplish brown<br>Faintly granular<br>RBC black<br>Lined with minute black granules                     | Cytoplasm<br>Inclusions<br>Nucleus: Membrane | Greyish blue<br>Coarsely granular<br>Vacuoles black, as are bacteria etc.<br>Thick with plaques of black chromatin  |
|    | Small black central dot<br>Trace only seen  | Karyosome<br>Fibril network                  | Eccentric black dot or plaque<br>More conspicuous; may have chromatin plaques   |
|    | Round<br>As trophozoite<br>Black chromidial bodies or bars<br>Glycogen (dissolved) replaced by vacuoles | Shape<br>Cytoplasm<br>Nucleus<br>Inclusions  | Round<br>As trophozoite<br>May have slender black chromidial bars<br>Glycogen (dissolved) replaced by vacuoles  |
|  |   |  |   |
| Precyst   |   |  |   |
|    | Round<br>As trophozoite<br>Black chromidial bodies or bars<br>Glycogen (dissolved) replaced by vacuoles | Shape<br>Cytoplasm<br>Nucleus<br>Inclusions  | Round<br>As trophozoite<br>May have slender black chromidial bars<br>Glycogen (dissolved) replaced by vacuoles  |
|  |   |  |   |
| Cyst  |   |  |   |
|    | Grey-blue<br>As precyst, less conspicuous or absent<br>Unstained, hyaline<br>As trophozoite 1–4         | Cytoplasm<br>Inclusions<br>Wall<br>Nuclei    | Greyish-blue, granular<br>As precyst, less conspicuous or absent<br>In 2 nuclei stage glycogen vacuoles may be dumb-bell-shaped<br>Unstained, hyaline<br>As trophozoite 1–8 |
|  |   |  |   |

### Invasion of the large intestine

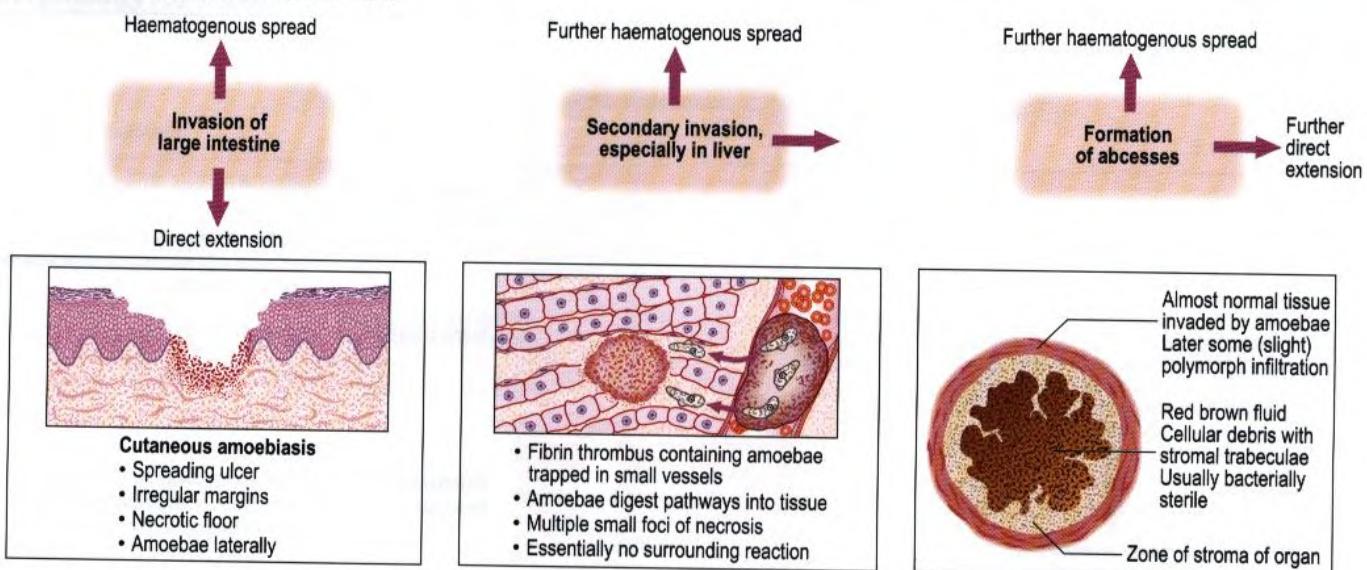


### Complications and sequelae

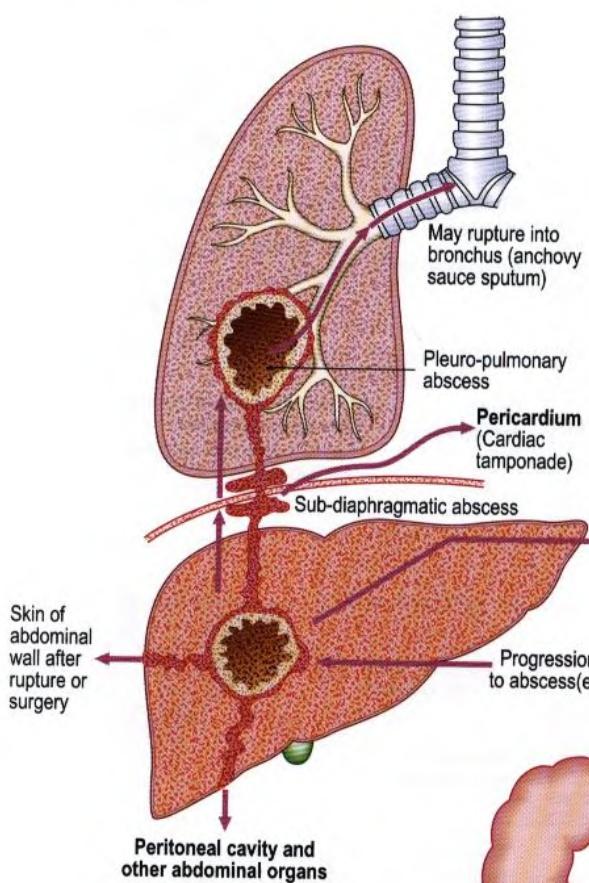


## *Entamoeba histolytica* (causing amoebiasis) (continued)

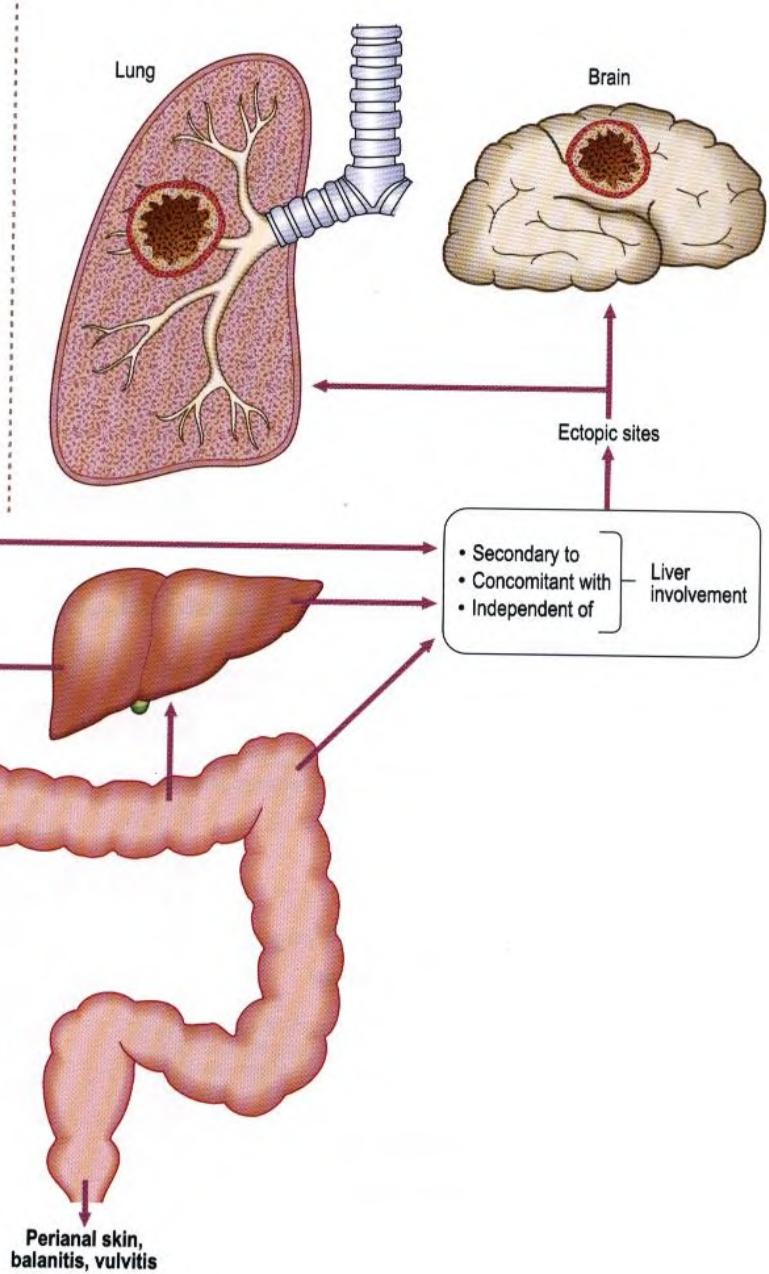
### Extraintestinal lesions in amoebiasis



### Direct extension



### Haematogenous spread



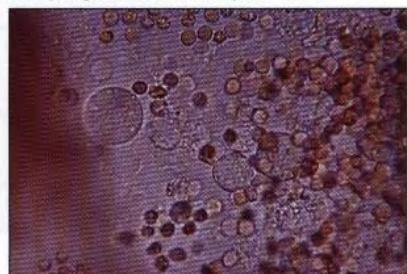
## Laboratory diagnosis

Diagnosis depends primarily on demonstration of haematophagous trophozoites of *E. histolytica* in stool samples, aspirates from intestinal and other organs, biopsy material (pinch biopsy at proctoscopy or sigmoidoscopy and surgical biopsy from elsewhere) and in mucus from rectal ulcers. ELISAs are available for the detection of *Entamoeba* antigen and specific *E. histolytica* lectin antigen in faecal samples. Serology is the method of choice for diagnosis of amoebic liver disease.

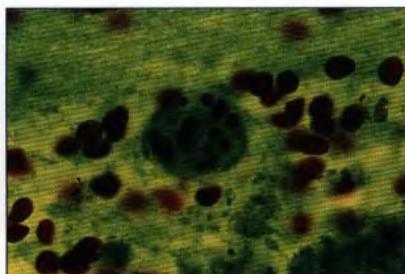
Faecal appearances in amoebic dysentery

|             |  | Character  |
|-------------|--|--|
| Naked eye   | Faecal matter<br>Mucus   | Always present<br>Not tenacious<br>Not abundant  |
| Microscopic | 1. Bacteria<br>2. Pus cells<br>3. Red blood cells<br>4. Large macrophages<br>5. Charcot-Leyden crystals<br>6. Haematophagous trophozoites of <i>E. histolytica</i>   | Numerous<br>Scanty, well preserved<br>Often in rouleaux<br>Not a feature<br>May be present but are non-specific<br>Present |
| Culture     | Usually limited to reference or research laboratories; lectin ELISA is used for specific identification of <i>E. histolytica</i> from cyst or trophozoite. Enzyme electrophoresis from cultured trophozoite is an alternative. |  |

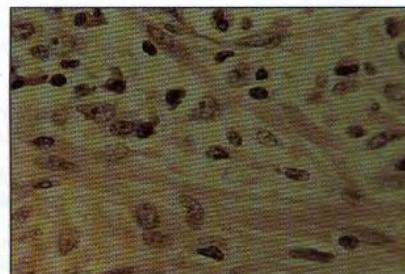
Haematophagous amoebic trophozoites.



(a) Direct microscopy



(b) Trichrome stain



(c) H&E stain

### Notes

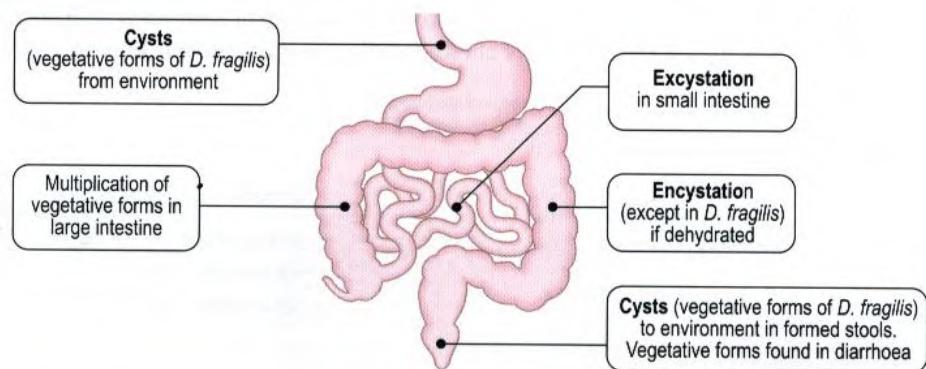
Vegetative *E. histolytica* when seen is actively motile and moves purposefully. There are finger-like, clear pseudopodia and ingested red cells. No nucleus can be seen. Precysts or cysts found in semi-formed or solid stool have typical nuclear characteristics (1–4 nuclei) and glycogen and chromidial bars can be demonstrated.

### Diagnostic tests

Polymorph leucocytosis. Examination of stool samples may show cysts and trophozoites of *E. histolytica*. Serological tests (IFAT, ELISA, cellulose acetate precipitin, latex agglutination) but serology is positive in no more than 75% of cases of amoebic colitis. Examine aspirated material for *E. histolytica*. Histology of rectal and colon biopsy material.

## Other intestinal amoebae

### Life cycle

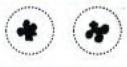


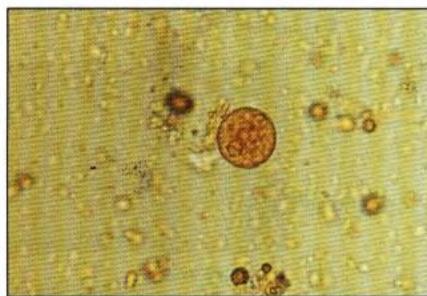
### Morphology

#### Unstained

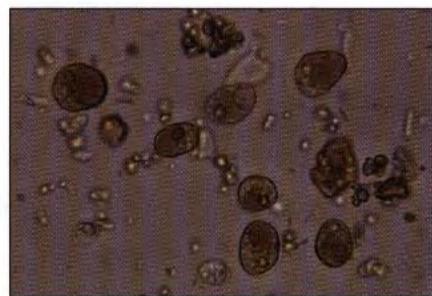
|                 | Vegetative forms (trophozoites)   |                        |                            |                             |                              |                          |                            |
|-----------------|---|------------------------|----------------------------|-----------------------------|------------------------------|--------------------------|----------------------------|
|                 | <i>Entamoeba coli</i>   | <i>Endolimax nana</i>  | <i>Iodamoeba bütschlii</i> | <i>Dientamoeba fragilis</i> | <i>Entamoeba histolytica</i> | <i>Entamoeba dispar</i>  | <i>Entamoeba hartmanni</i> |
| Size            | 15–50 µm  | 8–10 µm                | 8–20 µm                    | 5–12 µm                     | 15–60 µm                     | 15–60 µm                 | 15–60 µm                   |
| Motility        | Sluggish  | Sluggish               | Fairly active              | Very active                 | Very active                  | Active                   | Active                     |
| Ectoplasm       | Little  | Little                 | Little                     | Abundant                    | Abundant                     | Abundant                 | Abundant                   |
| Pseudopodia     | Blunt, mainly granular  | Blunt, mainly granular | Blunt, clear               | Leaf-like, clear            | Finger-like, clear           | Finger-like, clear       | Finger-like, clear         |
| Endoplasm       | All have granular cytoplasm with food particles, bacteria, crystals, vegetable cells, often in vacuoles. No ingested RBCs |                        |                            |                             | Ingested RBCs                | No ingested RBCs         | No ingested RBCs           |
| Nucleus         | Ring of refractive dots   | Generally invisible    | Generally invisible        | Two, collection of dots     | Generally invisible          | Generally invisible      | Generally invisible        |
|                 | <b>Precyst</b><br>(round up, discharge food particles, bacteria, etc.)  |                        |                            |                             |                              |                          |                            |
| Glycogen        | Often prominent vacuole   | Rare                   | Conspicuous                | None                        | Diffuse, soon disappears     | Diffuse, soon disappears | Diffuse, soon disappears   |
| Chromidial bars | Rarely seen   | Rare                   | None                       | None                        | Large refractile bars        | Large refractile bars    | Large refractile bars      |
|                 | <b>Cysts</b>  |                        |                            |                             |                              |                          |                            |
| Size            | 10–33 µm  | 5–14 µm                | 5–18 µm                    | None                        | 10–20 µm                     | 10–20 µm                 | 8–10 µm                    |
| Shape           | Spherical or oval   | Oval                   | Irregular                  | None                        | Spherical                    | Spherical                | Spherical                  |
| Wall            | Thick   | Thin                   | Thin                       | None                        | Thin                         | Thin                     | Thin                       |
| Glycogen        | Diffuse central   | None                   | Well-defined vacuoles      | None                        | Sometimes persists           | Sometimes persists       | Sometimes persists         |
| Chromidial bars | Not usual   | None                   | None                       | None                        | Sometimes present            | Sometimes present        | Sometimes present          |
| Nuclei numbers  | 1–8   | 4 (at one end)         | 1 only                     | None                        | 1–4                          | 1–4                      | 1–4                        |

## Stained

|                         | <i>Entamoeba coli</i>  | <i>Endolimax nana</i>   | <i>Iodamoeba bütschlii</i>  | <i>Dientamoeba fragilis</i>   | <i>Entamoeba histolytica</i>  | <i>Entamoeba dispar</i>   | <i>Entamoeba hartmanni</i>  |
|-------------------------|--|---|---|---|---|---|---|
| Cytoplasm inclusions    | With haematoxylin, stains bluish-grey<br>Stain black except glycogen as clear area |   |   |   | RBCs also stain black   |   |   |
| Nuclear characteristics |   |  |  |  |  |  |  |
| Membrane                | Thick  | Thin  | Thick   | Very delicate   |   | Delicate  |   |
| Chromatin on membrane   | Coarse   | None  | Sometimes granular  | None  |   | Fine granules   |   |
| Karyosome               | Coarse, generally eccentric  | Large irregular   | Large lateral   | Central granules  |   | Small central   |   |
| Fibril network          | May be chromatin particles   | No chromatin  | No chromatin  | Delicate fibrils  |   | Not often seen  |   |
| Pathogenicity           | Harmless commensal   | Harmless commensal  | Harmless commensal  | Disputed  | Invasive  | Harmless commensal<br>Non-invasive  | Harmless commensal<br>Non-invasive  |



*Entamoeba coli* cysts



*Iodamoeba bütschlii* cysts



*Entamoeba histolytica/dispar* cysts

# Intestinal flagellates

## Diagnosis

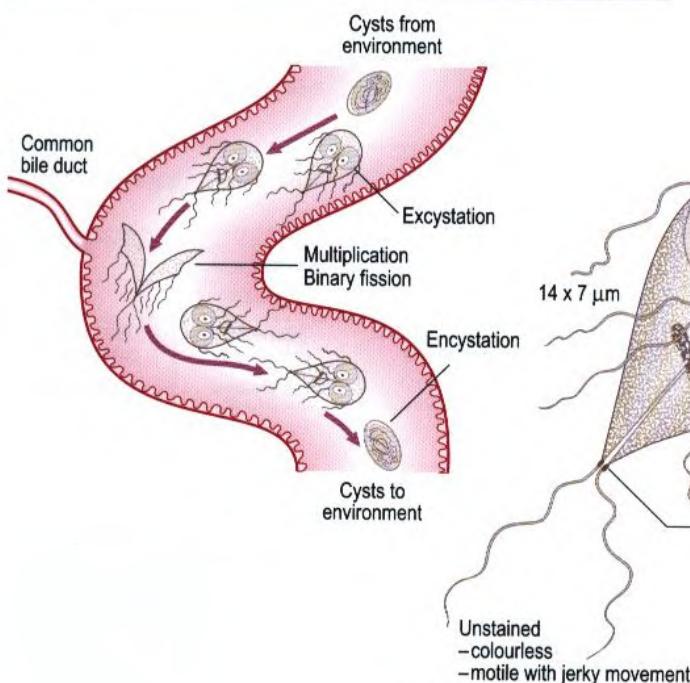
Trophozoites or cysts are found in stool samples or duodenal aspirates. Duodenal string test and stool antigen detection ELISA are also possible for the detection of *Giardia*.

## Distribution

These protozoa have a worldwide distribution.

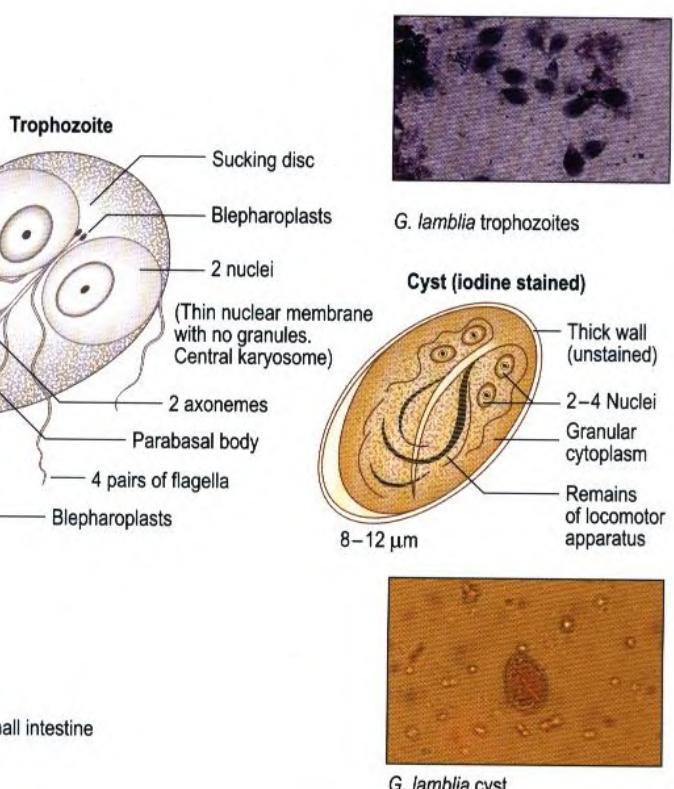
### *Giardia intestinalis* (*G. lamblia*)

#### Life cycle



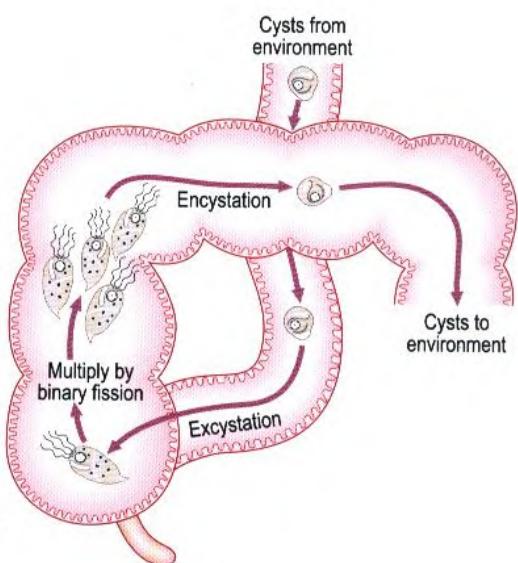
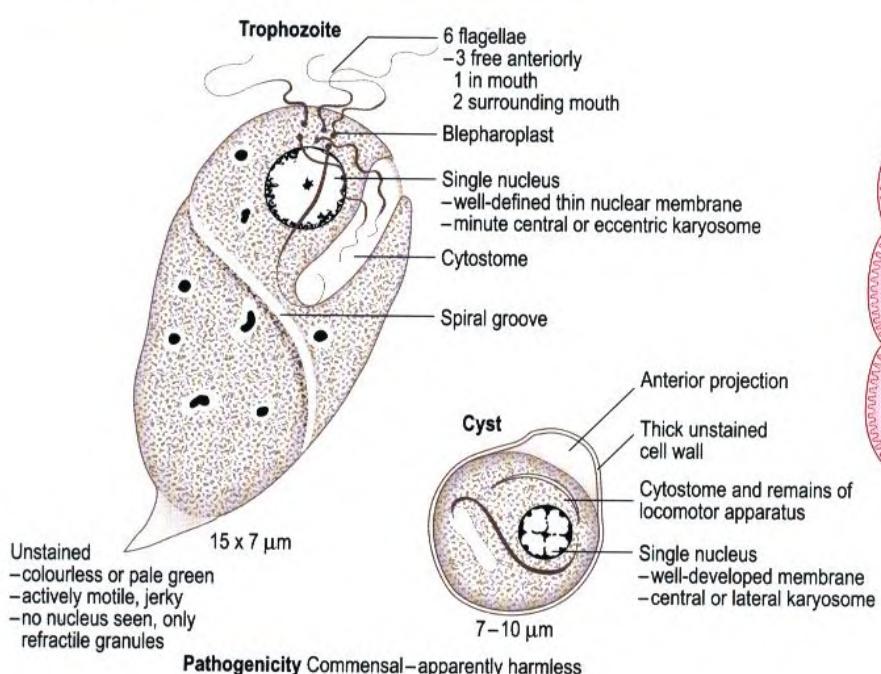
#### Pathogenicity

Common inhabitant of upper part of small intestine  
Enteropathy, diarrhoea, steatorrhoea

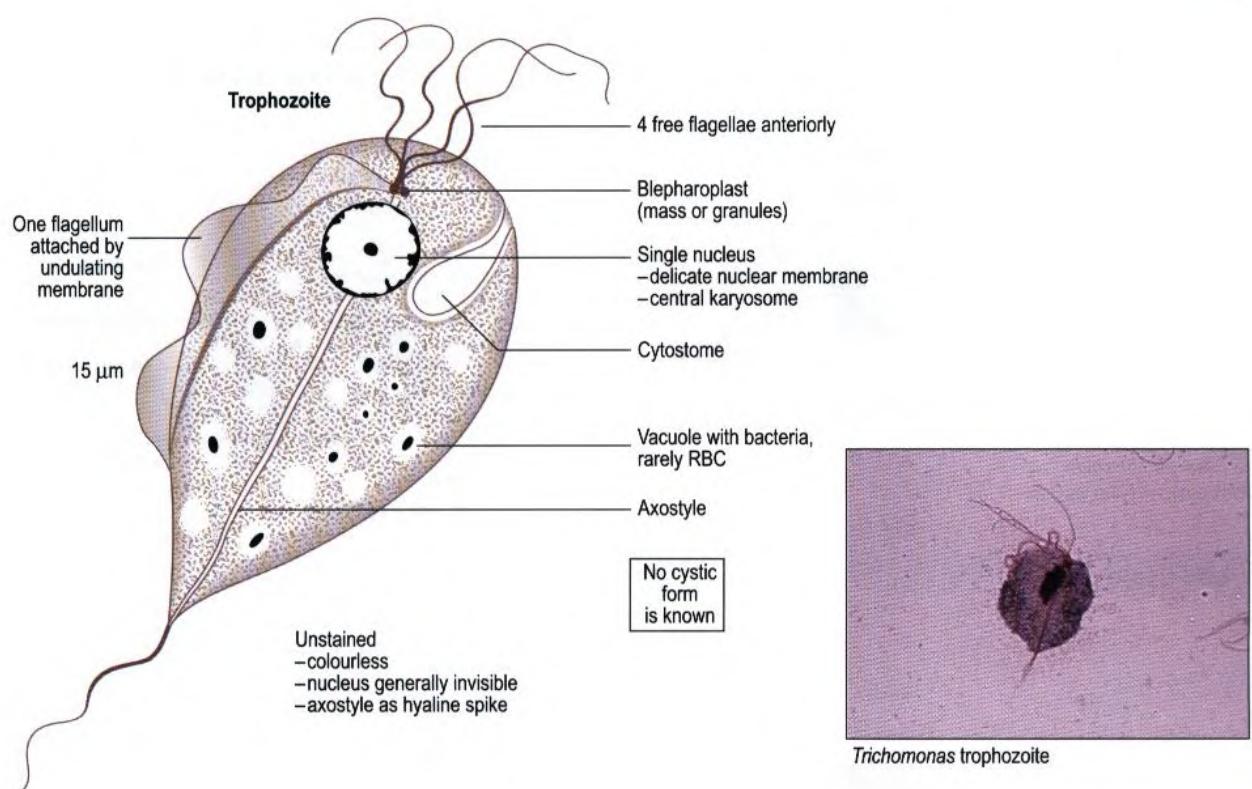


### *Chilomastix mesnili*

#### Life cycle



## *Trichomonas* species



### *T. hominis*

This is illustrated above. The trophozoite inhabits the small and large intestine. There is no proof as yet that it has any pathogenicity.

### *T. vaginalis*

Morphologically this is the same as *T. hominis* (above) but there is no free posterior flagellum beyond the undulating membrane. There is a marked parabasal body. It inhabits the urethra in the male and the vagina in the female, and is a cause of urethritis and vaginitis.

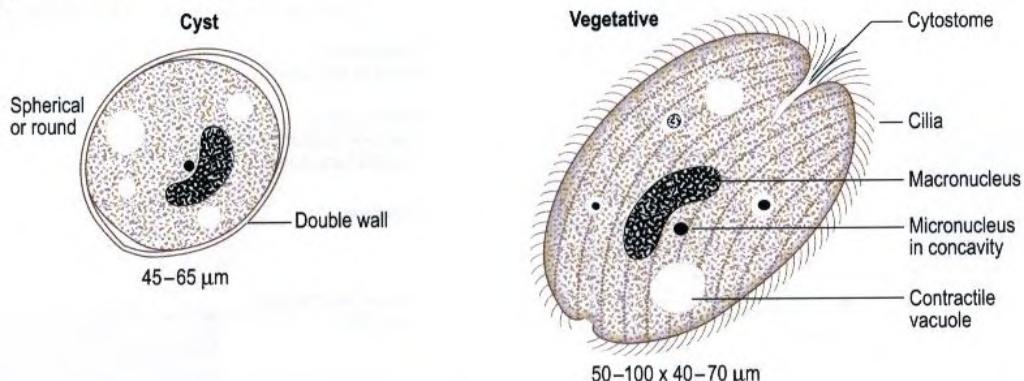
Demonstration of *T. vaginalis* is made by direct microscopy or after staining with acridine orange fluorescence stain. Cultures can be made using Feinberg-Whittington or Diamond's medium.

## Intestinal ciliates

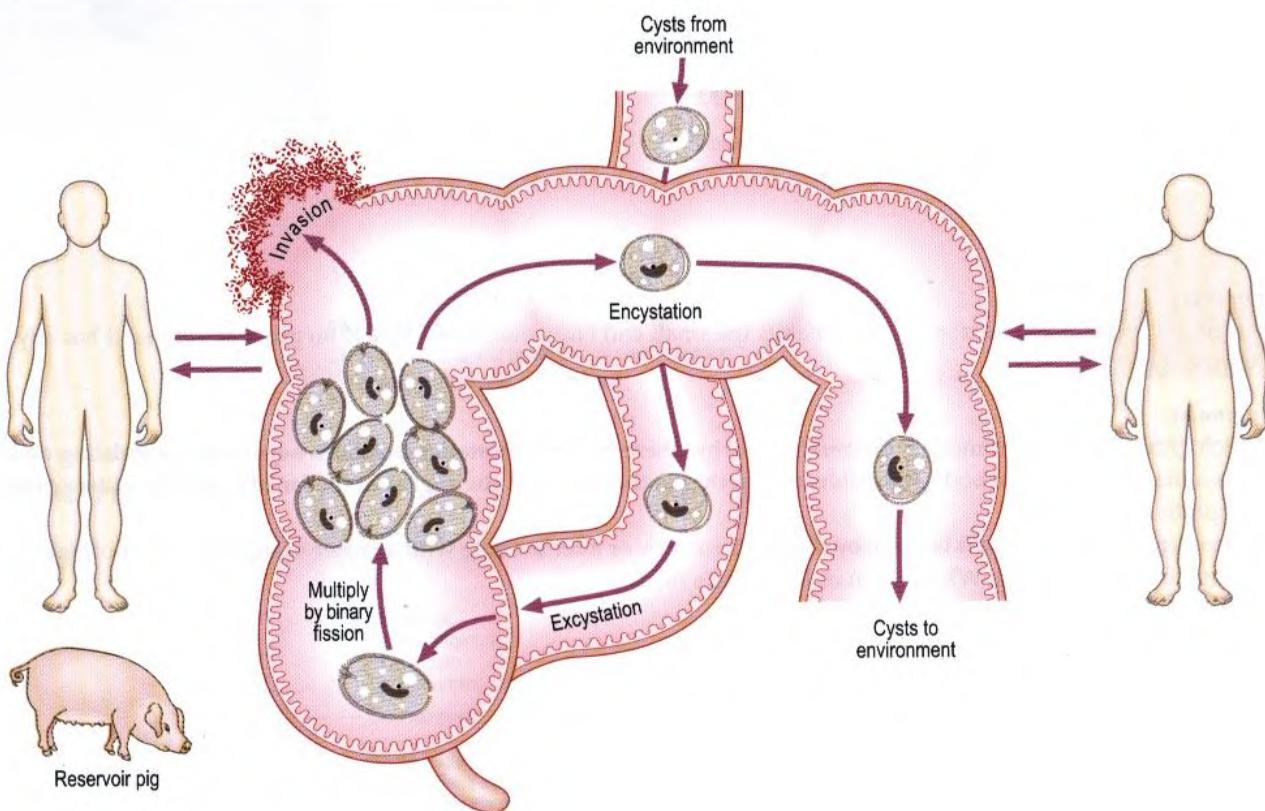
### *Balantidium coli*

Found in South and Central America, parts of Asia and some Pacific islands.

In its vegetative state, recognizable by the oval shape, coarse cilia, contractile vacuoles and the horseshoe- or kidney-shaped macronucleus. Reproduction is by binary fission.



### Life cycle

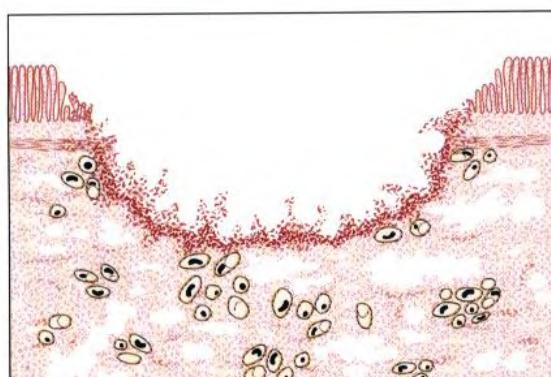


### Pathology and Clinical features

Problems occur in the ileum, colon and rectum but there is no extraintestinal spread. The parasite is a cause of dysentery, although the ulcers are wider mouthed than those of amoebic dysentery. Secondary infection is frequent. The main complication is perforation.

### Laboratory diagnosis

Trophozoites are found in diarrhoea and, in a fresh specimen, can be seen in active rotational movement. Cysts are found in semi-formed and formed stools.



# Tissue protozoa

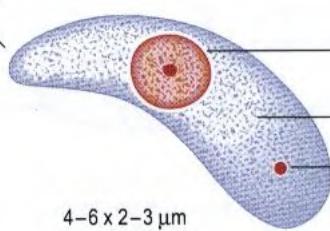
## *Toxoplasma gondii*

*Toxoplasma* has a very wide mammalian host range.

### Morphology

#### Tachyzoite

Pointed end



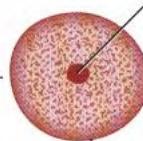
Red nucleus-ovoid, crescentic, or pyriform-nearer one end

Blue cytoplasm

Paranucleus stains as a small red dot

4–6 x 2–3 µm

Central karyosome



Nuclear membrane

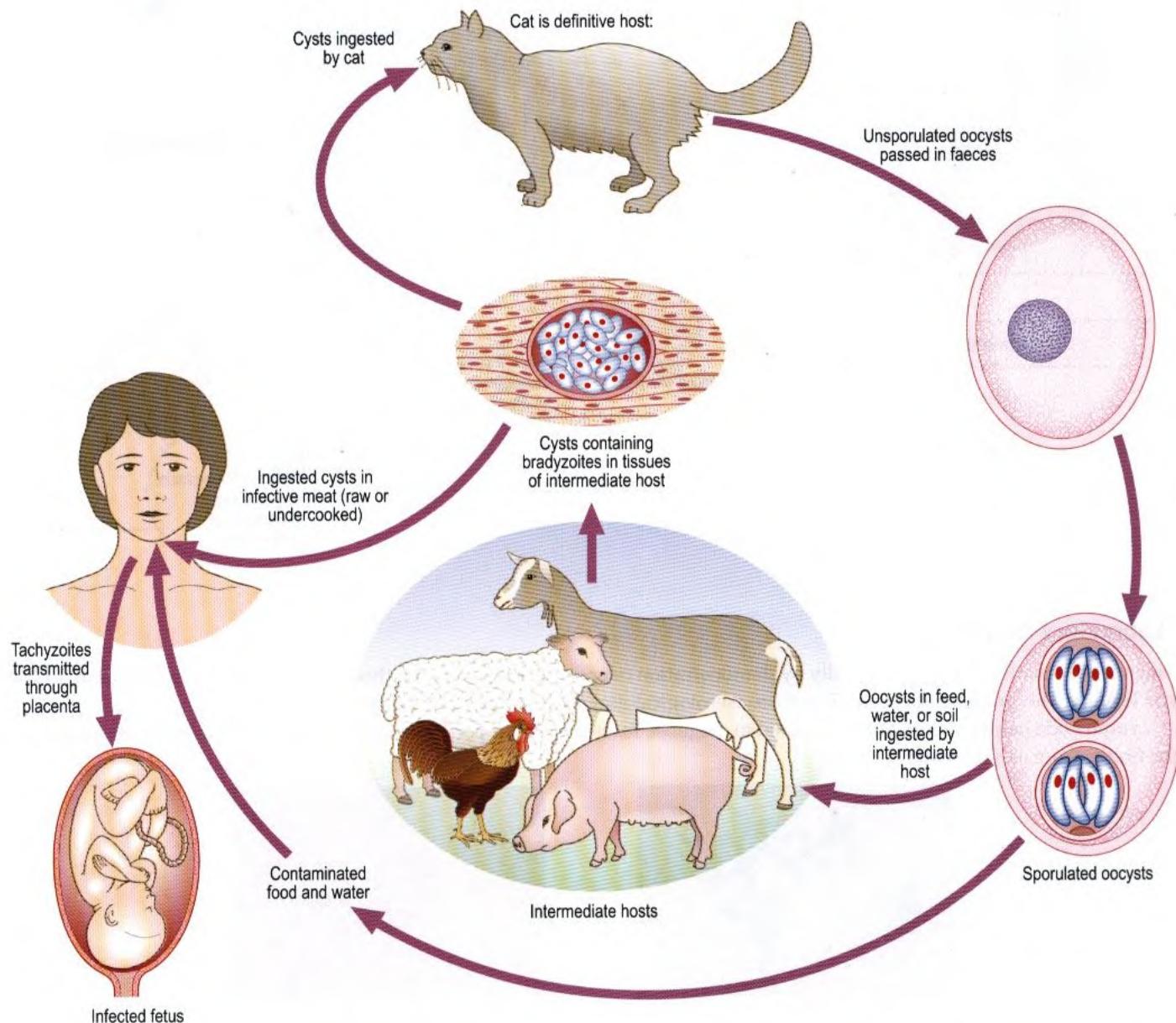
#### Habitat

Tachyzoites: single (free or intracellular) or in masses (pseudocysts)

In nucleated cells, especially macrophages

Bradyzoites (similar to tachyzoites but less active metabolically) in tissue cysts

### Life cycle

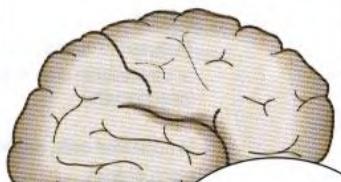


## Toxoplasma gondii (continued)

### Pathology and Clinical features

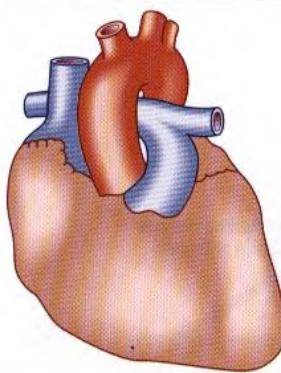
#### Congenital infection

**Marked calcification**  
Hydrocephalus or microcephaly

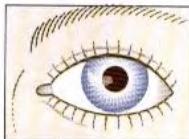


- Micro**
- Minute necrotic areas
  - Minute granulomata
  - Parasites in cells
  - Calcification

#### Myocarditis



#### Chorioretinitis

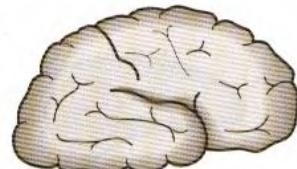


#### Other routes of infection

##### Inapparent effect

Woman may have affected child though herself shows no signs of disease

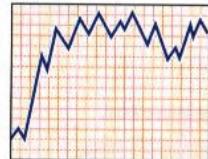
##### Acute encephalitis



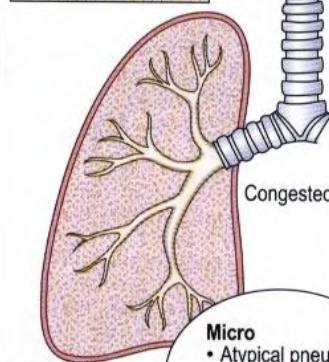
Cerebral abscess  
Frequently seen in patients with HIV infection

##### 'Glandular-fever-like' syndrome

###### Acute fever



Atypical pneumonia

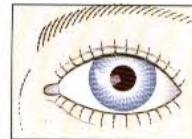


Serous effusions

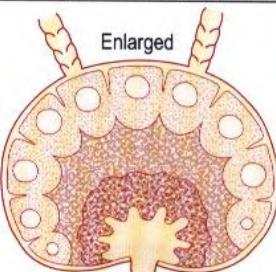
###### Micro

- Atypical pneumonia
- Parasitized mononuclears in bronchi

##### Chorioretinitis



##### Lymphadenopathy



###### Micro

- Reactive hyperplasia
- Conspicuous collections of histiocytes

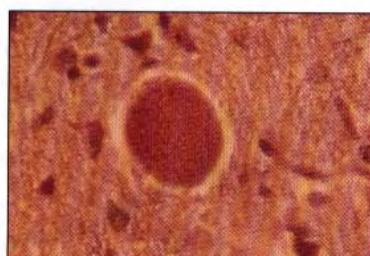
### Laboratory diagnosis

Diagnosis is usually made serologically by demonstration of specific antibodies. Methods include Latex agglutination, ELISA and ISAGA. The 'gold standard' for *Toxoplasma* serological diagnosis is the Sabin-Feldman dye test.

Lymph node biopsy should not be required to diagnose *Toxoplasma* but if performed because another diagnosis was suspected, the findings are as stated above.



*Toxoplasma* tachyzoites



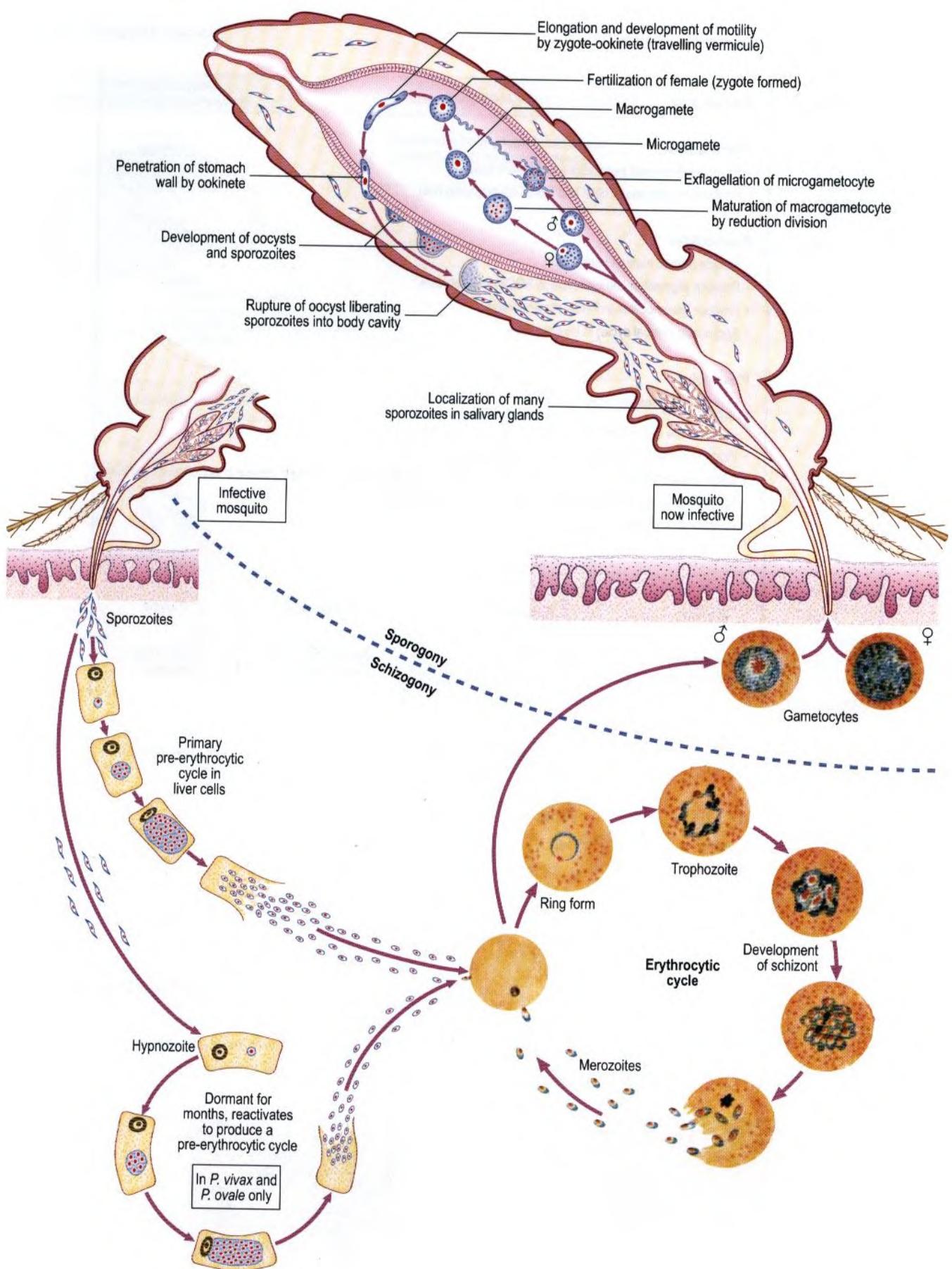
*Toxoplasma* pseudocyst (brain)

# Malaria parasites

## Classification

|                               |  |                                  |                      |                      |                 |                               |                        |                                  |                      |
|-------------------------------|--|----------------------------------|----------------------|----------------------|-----------------|-------------------------------|------------------------|----------------------------------|----------------------|
| Class                         | <b>Haematozoa</b>  |                                  |                      |                      |                 |                               |                        |                                  |                      |
| Order                         | <b>Haemosporida</b> <ul style="list-style-type: none"><li>• Sexual and asexual generations in different hosts</li><li>• Parasitic in fixed tissue cells and RBCs of vertebrate host</li></ul>  |                                  |                      |                      |                 |                               |                        |                                  |                      |
| Family                        | <b>Plasmodiidae</b> <ul style="list-style-type: none"><li>• Include human malaria parasites</li><li>• Produce pigment in asexual cycle in RBCs of vertebrates</li><li>• Produce gametocytes in RBCs of vertebrates</li><li>• Sporogony (sexual cycle) in invertebrates</li></ul>   |                                  |                      |                      |                 |                               |                        |                                  |                      |
| Genus                         | <b>Plasmodium</b> <ul style="list-style-type: none"><li>• Schizogony (asexual cycle) in:<ul style="list-style-type: none"><li>-RBCs</li><li>-other tissue cells of vertebrate host</li></ul></li><li>• Pigment derived from haemoglobin of infected RBC</li><li>• Gametocytes develop in some RBCs. These undergo sporogony (sexual cycle) in female anopheline mosquitoes</li><li>• Sporozoites produced in mosquito, infective to vertebrate host</li><li>• All malaria parasites included in this genus</li></ul> |                                  |                      |                      |                 |                               |                        |                                  |                      |
| Class                         | <table><tr><td><i>P. vivax</i></td><td><i>P. malariae</i></td><td><i>P. falciparum</i></td><td><i>P. ovale</i></td></tr><tr><td>Causes benign tertian malaria</td><td>Causes quartan malaria</td><td>Causes malignant tertian malaria</td><td>Causes ovale malaria</td></tr></table>   | <i>P. vivax</i>                  | <i>P. malariae</i>   | <i>P. falciparum</i> | <i>P. ovale</i> | Causes benign tertian malaria | Causes quartan malaria | Causes malignant tertian malaria | Causes ovale malaria |
| <i>P. vivax</i>               | <i>P. malariae</i>   | <i>P. falciparum</i>             | <i>P. ovale</i>      |                      |                 |                               |                        |                                  |                      |
| Causes benign tertian malaria | Causes quartan malaria   | Causes malignant tertian malaria | Causes ovale malaria |                      |                 |                               |                        |                                  |                      |

## Life cycle

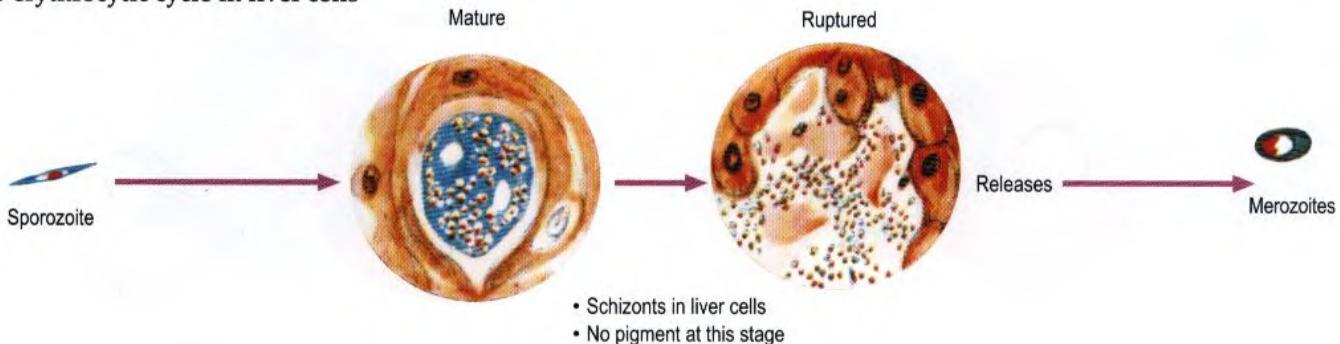


# Morphology

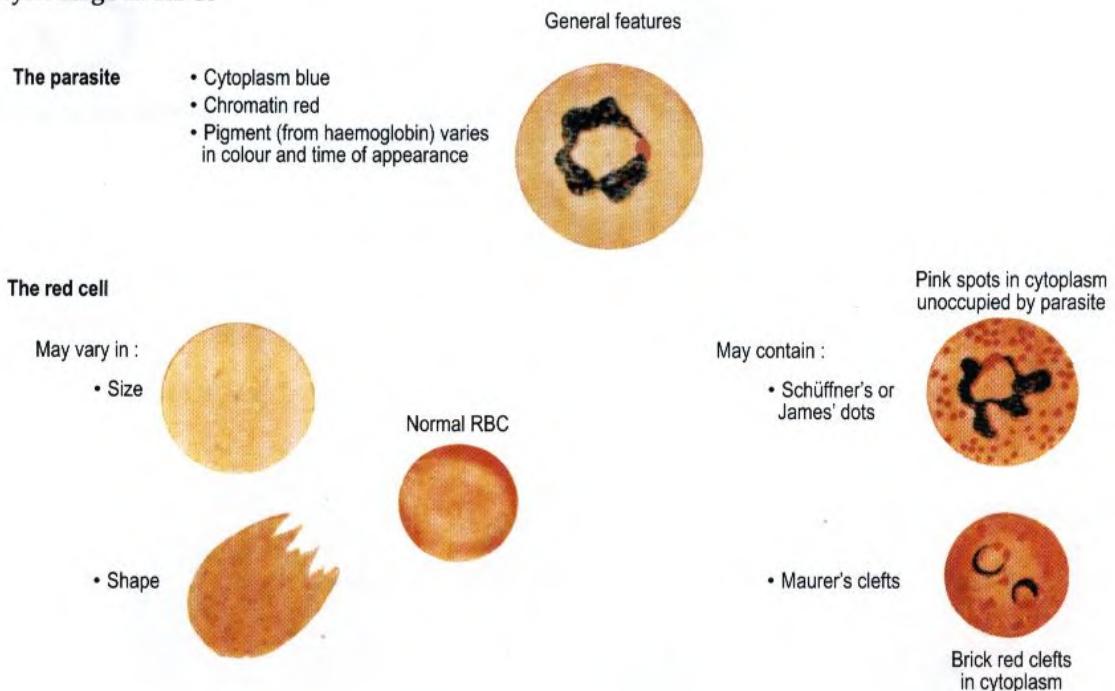
## Stained by Leishman or Giemsa

### Schizogony (asexual cycle)

#### Pre-erythrocytic cycle in liver cells



#### Erythrocytic stage in RBCs



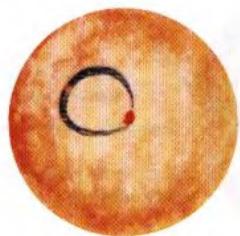
| RBC characteristics           | <i>P. vivax</i>          | <i>P. malariae</i> | <i>P. falciparum</i>                                | <i>P. ovale</i>           |
|-------------------------------|--------------------------|--------------------|---|---------------------------|
| <b>Size</b>                   | Larger than mature RBC   | Smaller, older RBC | Mature RBC  | Larger than mature RBC    |
| <b>Colour</b>                 | Pale                     | Normal             | Normal  | Pale                      |
| <b>Shape</b>                  | Round                    | Round              | Round<br>May be crenated                            | Oval<br>May be fimbriated |
| <b>Cytoplasmic inclusions</b> | Schüffner's dots present | None               | Maurer's clefts may be present in late trophozoites | James' dots conspicuous   |

## Morphology (continued)

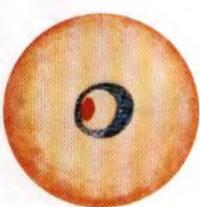
### Stages in thin films

#### Ring forms (early trophozoites)

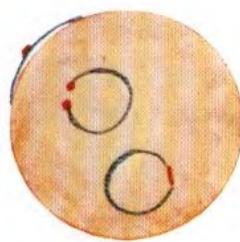
*P. vivax*



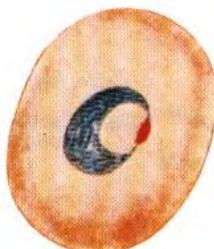
*P. malariae*



*P. falciparum*



*P. ovale*



|       |               |               |                    |            |
|-------|---------------|---------------|--------------------|------------|
| Size  | 1/3 RBC       | Up to 1/3 RBC | 1/3 RBC            | 1/3 RBC    |
| Shape | Delicate ring | Compact ring  | Very delicate ring | Dense ring |



|                            |                    |                            |                                   |                          |
|----------------------------|--------------------|----------------------------|-----------------------------------|--------------------------|
| Chromatin                  | Fine dot           | One mass often inside ring | Fine dots<br>Frequently two       | Dense, well-defined mass |
| Accolé forms*              | Sometimes          | None                       | Frequent                          | None                     |
| Pigment                    | None at this stage | May be present             | None at this stage                | None at this stage       |
| Multiple parasitized cells | Sometimes          | Rare                       | Frequently with high parasitaemia | Rare                     |

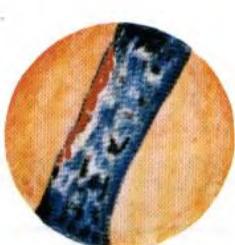
\* Forms situated on margin of RBC

#### Developing trophozoites

*P. vivax*



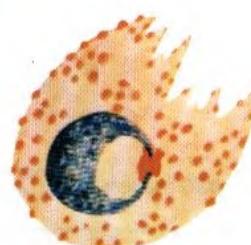
*P. malariae*



*P. falciparum*



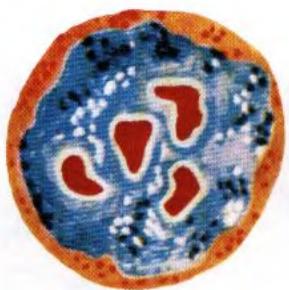
*P. ovale*



|               |                          |  |                                       |                            |
|---------------|--------------------------|--|---------------------------------------|----------------------------|
| Size          | Large                    | Small, but appears large relative to size of RBC | Small                                 | Small                      |
| Shape         | Very irregular, amoeboid | Compact, often band forms                        | Compact, with cytoplasmic vacuolation | Compact                    |
| Chromatin     | Dots or threads          | Prominent, often as a band                       | Dots or threads                       | Large irregular clumps     |
| Pigment       |                          |  |                                       |                            |
| —texture      | Fine                     | Coarse   | Coarse                                | Coarse                     |
| —colour       | Yellow brown             | Dark brown                                       | Black                                 | Dark yellow brown          |
| —quantity     | Medium                   | Abundant   | Medium                                | Medium                     |
| —distribution | Scattered fine particles | Scattered clumps and rods                        | Aggregated in one or two clumps       | Scattered coarse particles |

## Immature schizonts

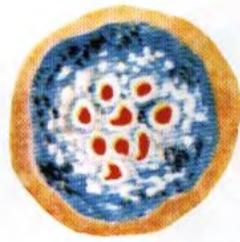
*P. vivax*



*P. malariae*



*P. falciparum*



*P. ovale*

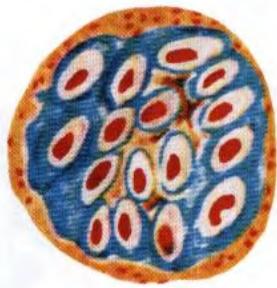


(rarely seen in peripheral blood)

|                  |                           |                      |                  |                      |
|------------------|---------------------------|----------------------|------------------|----------------------|
| <b>Size</b>      | Almost fills RBC          | Almost fills RBC     | Almost fills RBC | Almost fills RBC     |
| <b>Shape</b>     | Somewhat amoeboid         | Compact              | Compact          | Compact              |
| <b>Chromatin</b> | Numerous irregular masses | Few irregular masses | Irregular masses | Few irregular masses |
| <b>Pigment</b>   | Scattered                 | Scattered            | Single clump     | Scattered            |

## Mature schizonts

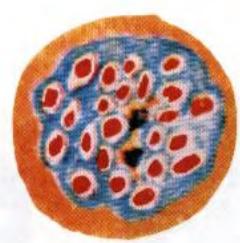
*P. vivax*



*P. malariae*



*P. falciparum*



*P. ovale*



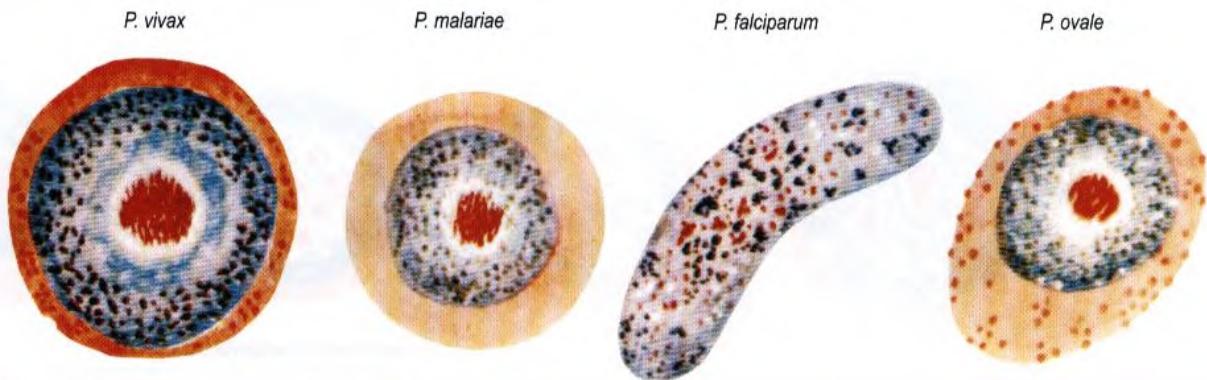
(rarely seen in peripheral blood)

|                   |                                     |                                   |                              |  |
|-------------------|-------------------------------------|-----------------------------------|------------------------------|--|
| <b>Size</b>       | Fills RBC                           | Nearly fills RBC                  | Nearly fills RBC             | Fills 3/4 RBC                            |
| <b>Shape</b>      | Segmented                           | Segmented daisy head              | Segmented                    | Segmented                                |
| <b>Merozoites</b> |                                     |                                   |                              |  |
| —range            | 14–24                               | 6–12                              | 8–32                         | 6–12                                     |
| —mean             | 16                                  | 8                                 | 24                           | 8  |
| —size             | Medium                              | Large                             | Small                        | Large                                    |
| <b>Pigment</b>    | Aggregated in centre (yellow brown) | Aggregated in centre (dark brown) | Aggregated in centre (black) | Aggregated in centre (dark yellow brown) |

## Morphology (continued)

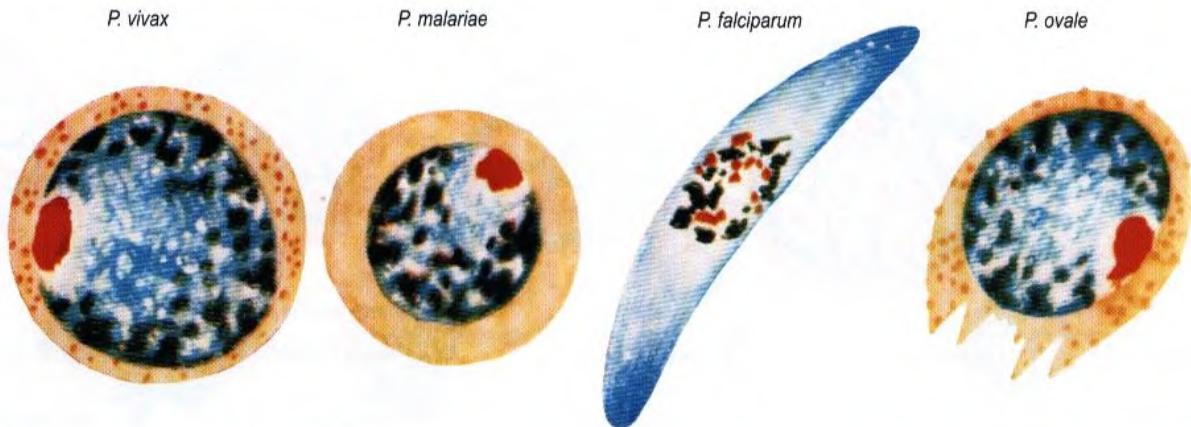
### Stages in thin films (continued)

#### Microgametocytes (male)



| Time of appearance    | 3–5 days                           | 7–14 days              | 7–12 days                           | 12–14 days             |
|-----------------------|------------------------------------|------------------------|-------------------------------------|------------------------|
| Number in bloodstream | Many                               | Scanty                 | Many                                | Scanty                 |
| Size                  | 3/4 fills RBC                      | 1/2 to 2/3 fills RBC   | Larger than RBC                     | 1/2 to 2/3 fills RBC   |
| Shape                 | Round or oval compact              | Round compact          | Kidney-shaped<br>Bluntly round ends | Round compact          |
| Cytoplasm             | Pale blue                          | Pale blue              | Reddish blue                        | Pale blue              |
| Chromatin             | Single chromatin mass              | As for <i>P. vivax</i> | Fine granules scattered throughout  | As for <i>P. vivax</i> |
| Pigment               | Abundant brown granules throughout | As for <i>P. vivax</i> | Dark granules throughout            | As for <i>P. vivax</i> |

#### Macrogametocytes (female)

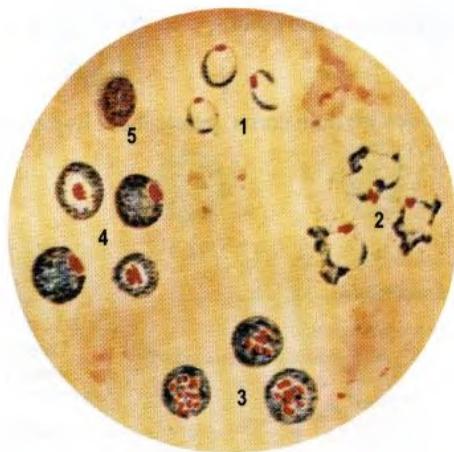


| Time of appearance    | 3–5 days                     | 7–14 days              | 7–12 days                                  | 12–14 days             |
|-----------------------|------------------------------|------------------------|--|------------------------|
| Number in bloodstream | Many                         | Scanty                 | Many                                       | Scanty                 |
| Size                  | 3/4 fills RBC                | 1/2 to 2/3 fills RBC   | Larger than RBC                            | 1/2 to 2/3 fills RBC   |
| Shape                 | Round or oval compact        | Round compact          | Crescentic-sharply rounded or pointed ends | Round compact          |
| Cytoplasm             | Dark blue                    | Dark blue              | Dark blue                                  | Dark blue              |
| Chromatin             | Compact peripheral mass      | As for <i>P. vivax</i> | Compact masses near centre                 | As for <i>P. vivax</i> |
| Pigment               | Small masses round periphery | As for <i>P. vivax</i> | Black, rod-like granules round nucleus     | As for <i>P. vivax</i> |

## Morphology in stained thick films

Note that the parasites are not flattened in the film and so appear smaller than in thin film. The red cells are haemolyzed in processing so there is no guide to the size, shape or colour of the RBCs. Schüffner's dots are indefinite and there are no Maurer's clefts.

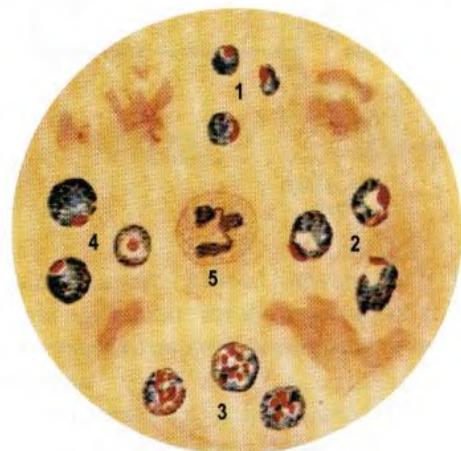
### *P. vivax*



1. Ring forms, small fine rings often broken
2. Trophozoites, markedly irregular cytoplasm
3. Schizonts, many (average 16) small merozoites
4. Gametocytes, compact parasites with features of ♂ and ♀ as described
5. White blood cell

### *P. malariae* and *P. ovale*

Almost identical but James' dots may be visible in the latter



1. Ring forms, compact rings
2. Trophozoites, solid regular cytoplasm
3. Schizonts, few (average 8) large merozoites
4. Gametocytes, very difficult to distinguish from *P. vivax*
5. White blood cell

### *P. falciparum*



1. Ring forms, very small, fine rings usually unbroken trophozoites (with vacuolated cytoplasm) and schizonts are rarely seen in peripheral blood
2. Gametocytes, characteristic crescentic ♂ and ♀ forms
3. White blood cell

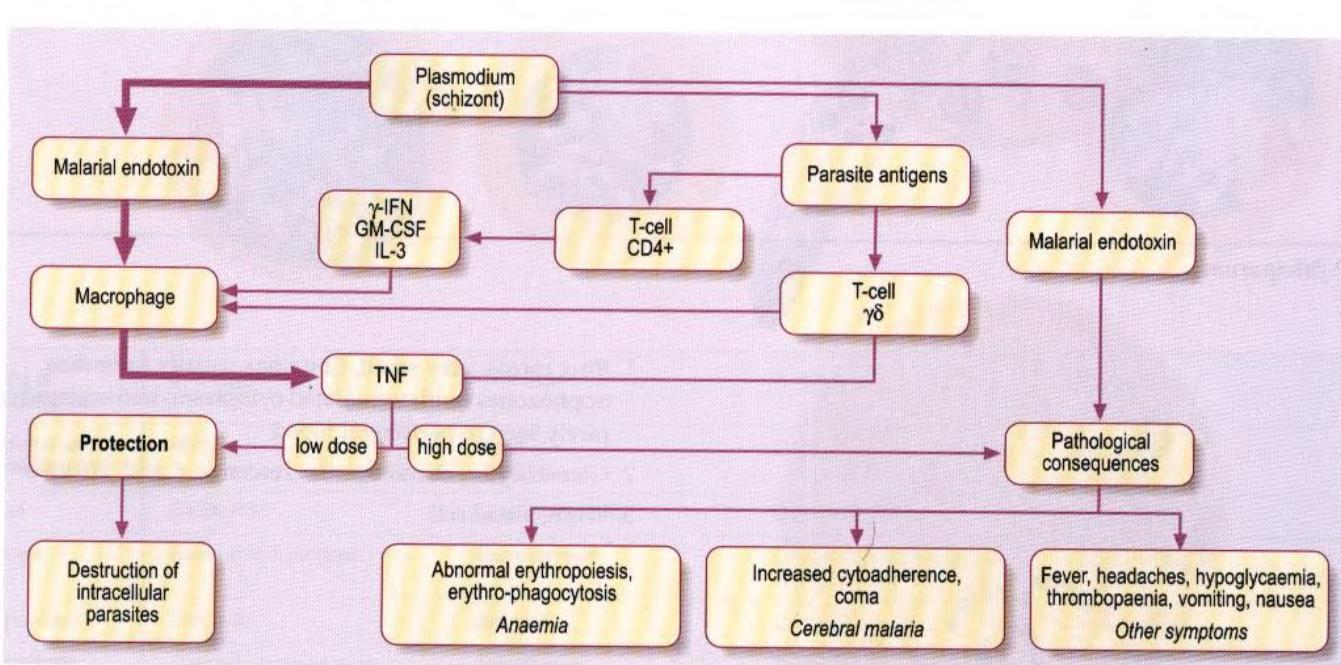
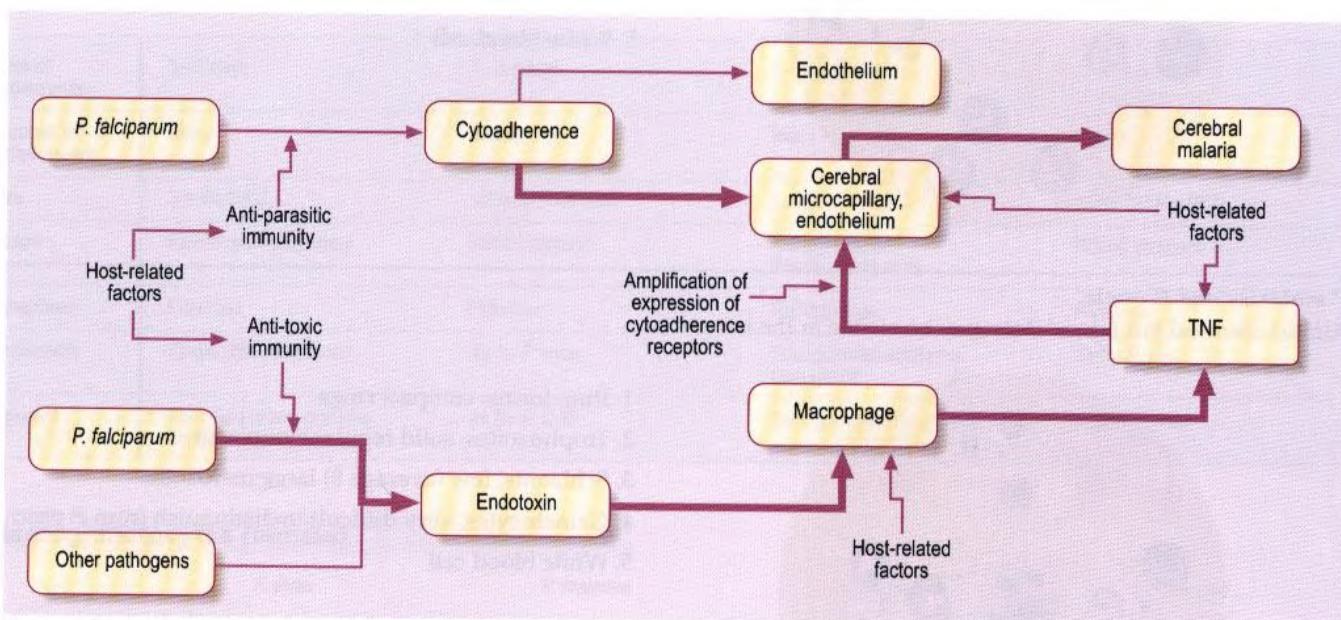
## **Pathology and Clinical features**

*Plasmodium vivax*, *P. ovale*, *P. malariae* and uncomplicated *P. falciparum* malaria have similar features with fever, rigors, headache, muscle aches, malaise and anorexia. Anaemia may develop and the liver and spleen may become enlarged. Because the clinical appearances are non-specific, malaria may be misdiagnosed, e.g. as a viral infection, with severe consequences.

*Plasmodium falciparum* infection can readily progress to severe malaria, the clinical criteria of which have been defined by a World Health Organisation working group. One or more of the following features in the presence of asexual parasitaemia indicate severe falciparum malaria:

- cerebral malaria
- severe anaemia
- renal failure
- pulmonary oedema or adult respiratory distress syndrome
- hypoglycaemia
- circulatory collapse or shock
- spontaneous bleeding from the gums, nose, gastrointestinal tract and/or laboratory evidence of disseminated intravascular coagulation
- repeated generalised convulsions (more than two in 24 hours despite cooling)
- acidaemia (arterial pH < 7.25) or acidosis (plasma bicarbonate < 15 mmol/L)
- macroscopic haemoglobinuria.

Other features of severe falciparum malaria include impaired consciousness less severe than coma, prostration, hyperparasitaemia, jaundice and hyperpyrexia.

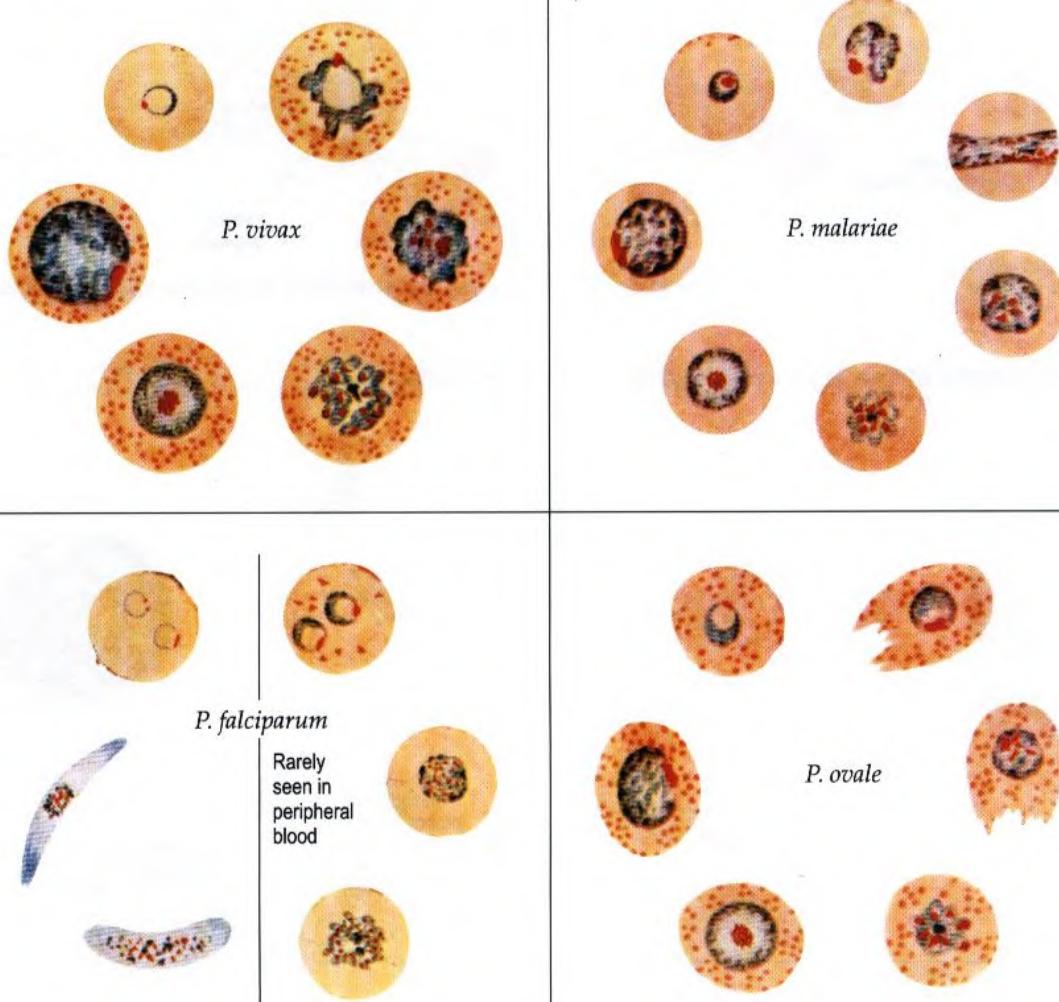


## Role of TNF

(Both of the above figures after Figs 20.17 and 20.18 in Hommel M, Gilles H M, *Malaria* (Chapter 20). In Cox E G, Kreier J P, Wakelin D, eds. Topley and Wilson's *Microbiology and Microbial Infections*, Vol 5. Parasitology. London: Arnold; 1998)

## Laboratory diagnosis

Malaria parasites in thin blood film. Stained by Leishman or Giemsa at pH 7.2



It is also possible to use thick blood films stained by Field or Giemsa. Bone marrow films may also be examined. Serology (IFAT or ELISA) is not appropriate for the detection of acute malaria but is deployed as a retrospective test for epidemiological use to establish the cause of nephrotic syndrome or hyperreactive malarial splenomegaly (HMS).

### Antigen Detection

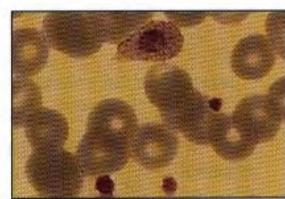
*P. falciparum* expresses a specific antigen HRP2 on the surface of the parasitized RBC. This can be detected by using immunochromatographic antigen capture techniques (AMRAD ICT, Becton Dickinson ParaSight F). Parasite lactate dehydrogenase (pLDH) is biochemically and antigenically distinct from human LDH and is produced by all *Plasmodium* species. Gold-labelled monoclonal and polyclonal antibodies can be used in an immunochromatographic technique to detect pLDH in whole blood (OptiMAL, Flow Inc., Portland OR).



*P. vivax* trophozoites



*P. malariae*: late trophozoite



*P. ovale* trophozoite



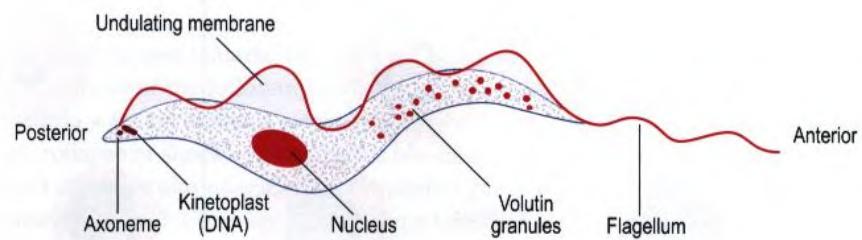
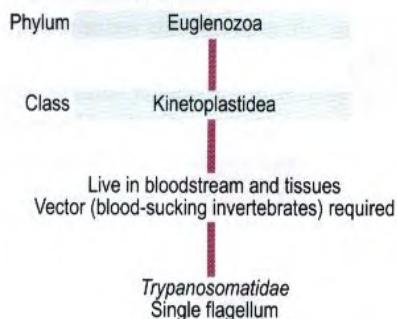
*P. falciparum* trophozoites



*P. falciparum* gametocyte

# Body-fluid and tissue flagellates

## Classification



## Morphological stages of the Trypanosomatidae affecting humans

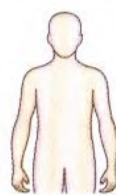
### *Leishmania* spp.



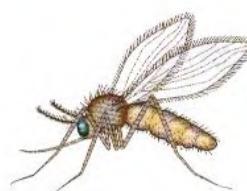
*Leishmania* amastigotes



Intracellular in macrophages in humans



In midgut, then proboscis of sandfly (transfer stage to human, also in culture)

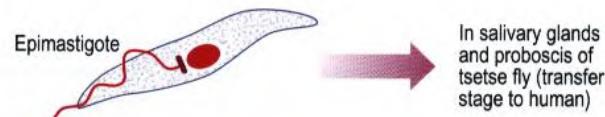


### *Trypanosoma* spp.

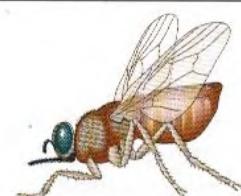
*Trypanosoma brucei rhodesiense*  
*Trypanosoma brucei gambiense*



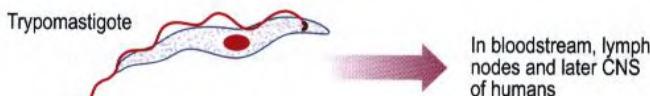
Trypomastigote



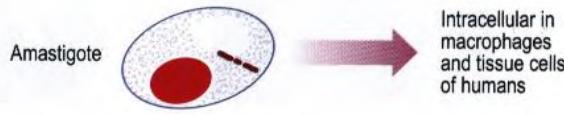
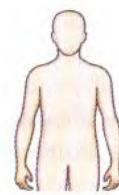
In salivary glands and proboscis of tsetse fly (transfer stage to human)



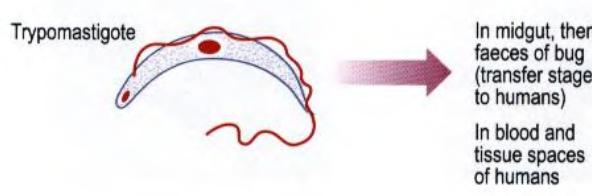
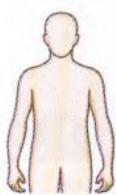
### *Trypanosoma cruzi*



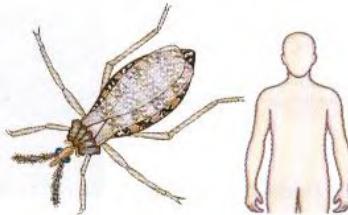
In bloodstream, lymph nodes and later CNS of humans



Intracellular in macrophages and tissue cells of humans



In midgut, then faeces of bug (transfer stage to humans)  
 In blood and tissue spaces of humans



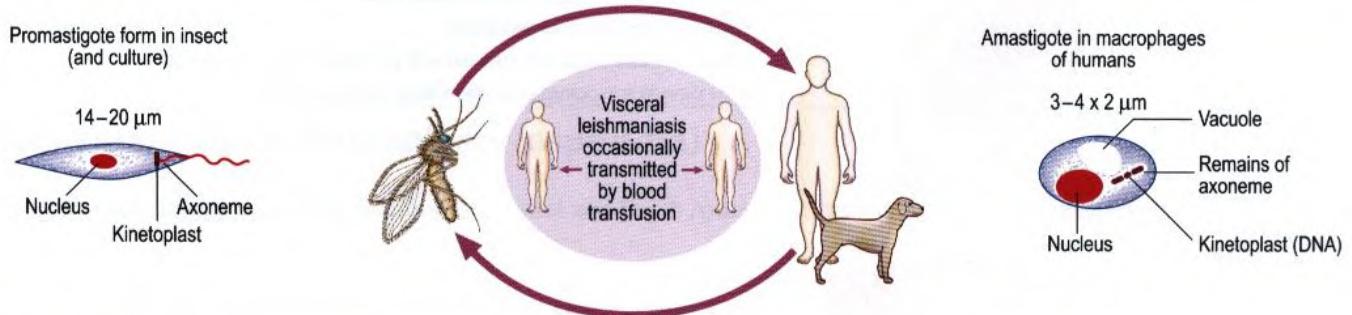
# Leishmaniasis

|                |   |  |   |
|----------------|---|--|---|
| <b>Species</b> | <i>L. donovani</i> complex<br><i>L. infantum</i><br><i>L. donovani</i><br><i>L. chagasi</i> | <i>L. tropica</i><br><i>L. major</i><br><i>L. aethiopica</i><br><i>L. infantum</i> | <i>L. braziliensis</i> complex<br><i>L. amazonensis</i><br><i>L. mexicana</i> |
| <b>Disease</b> | Visceral (kala azar)  | Cutaneous  | Muco-cutaneous (Espundia)   |

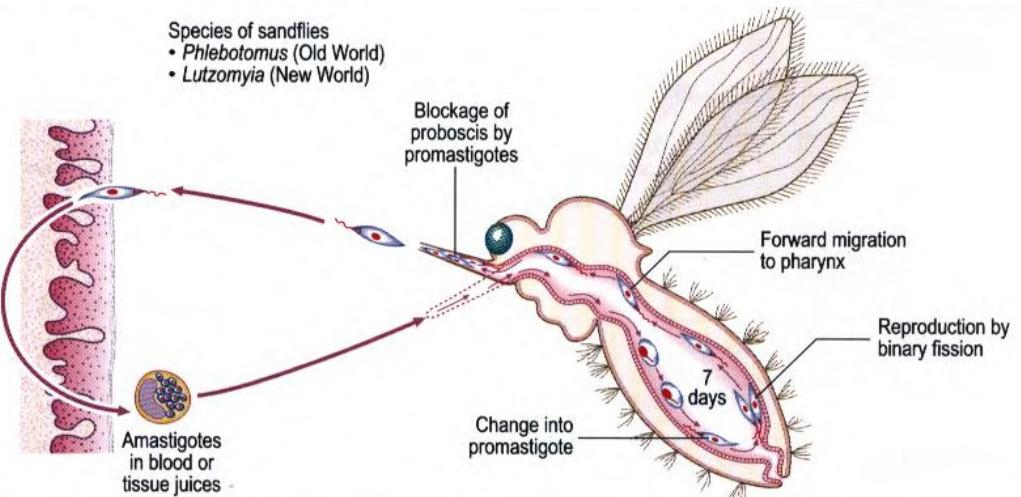
## Distribution



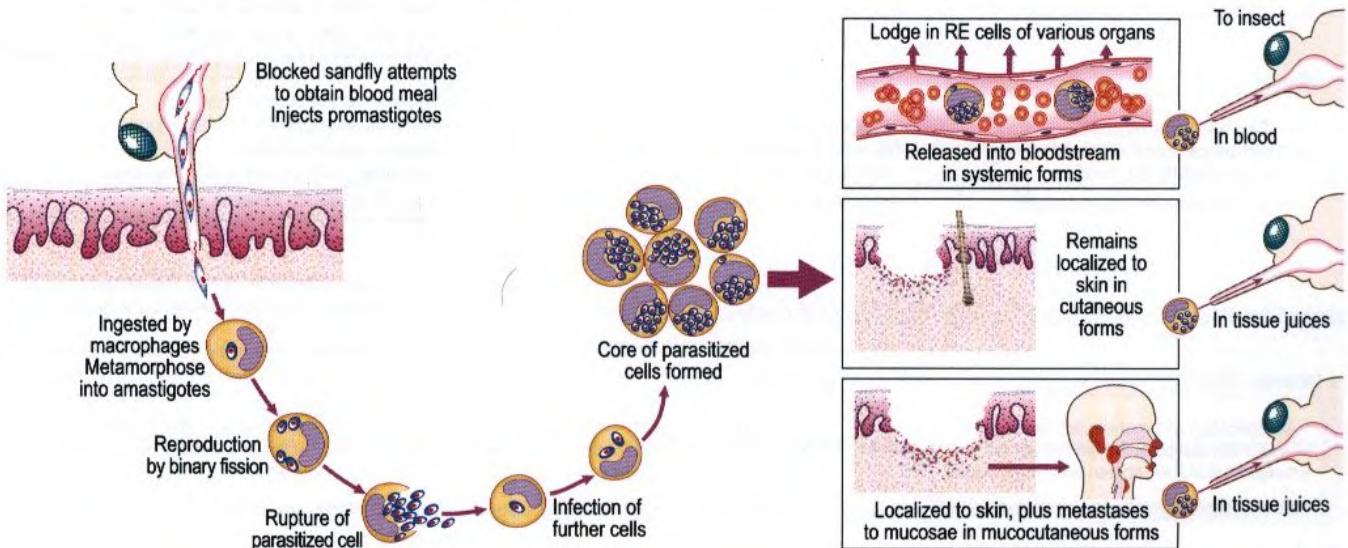
## Life cycle and morphology of *Leishmania* (similar in all three species)



## Life cycle in insect

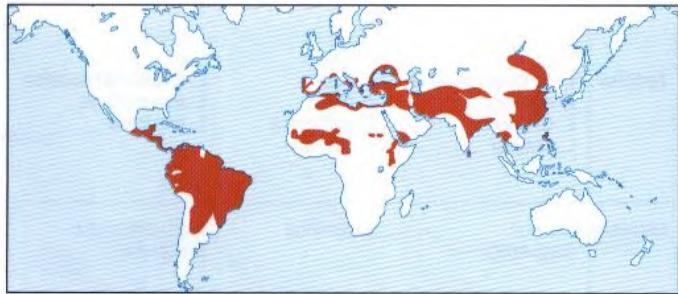


## Life cycle in humans and reservoir animals

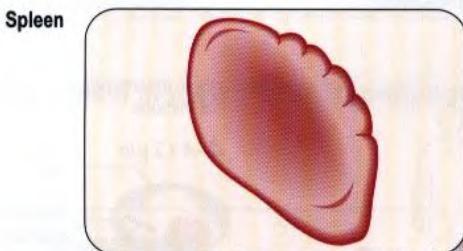


### **Visceral leishmaniasis (kala azar)**

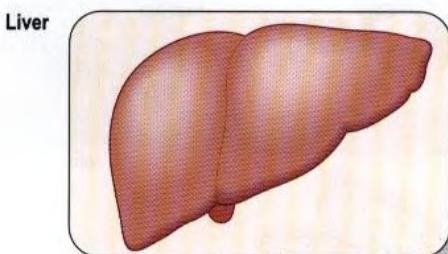
## Distribution



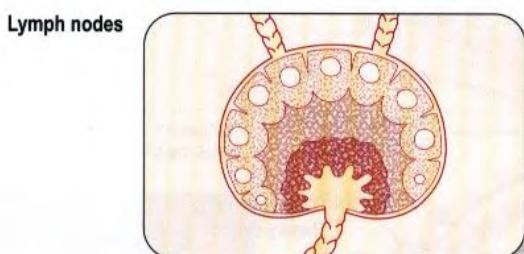
## Clinico-pathological correlation



- Parasitized macrophages and endothelial cells
  - Splenomegaly, pain from perisplenitis
  - Spleen appears congested, dark red, soft and friable. Markedly enlarged
  - The capsule is thickened and, later, infarcts and fibrosis occur

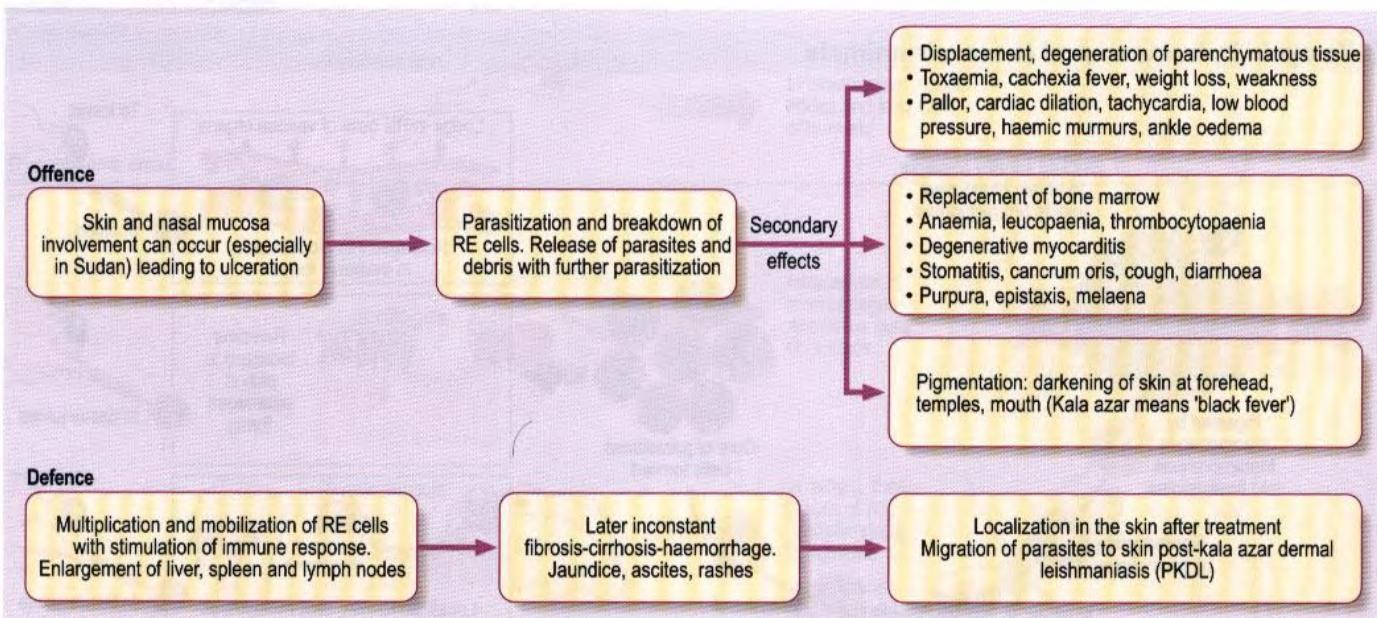


- Hepatomegaly
  - Liver appears enlarged, fatty congested and later may become cirrhotic
  - Parasitized proliferated Kupffer cells with atrophy of the liver cells and later fibrosis



- Lymphadenopathy
  - Reactive hyperplasia with parasitized macrophages

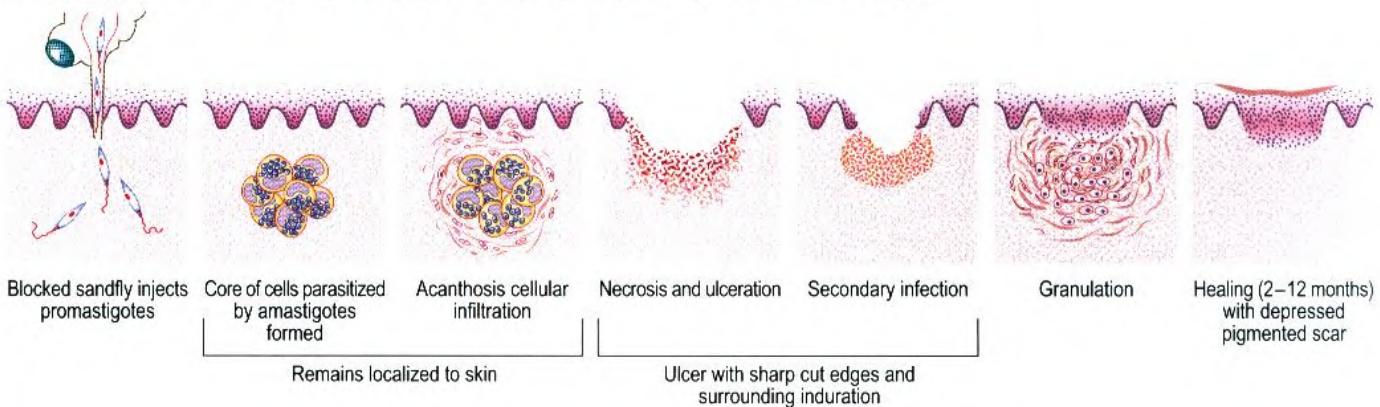
## Mechanism of pathology



Both offensive and defensive processes give rise to increased serum globulin and reversal of a/g ratio.

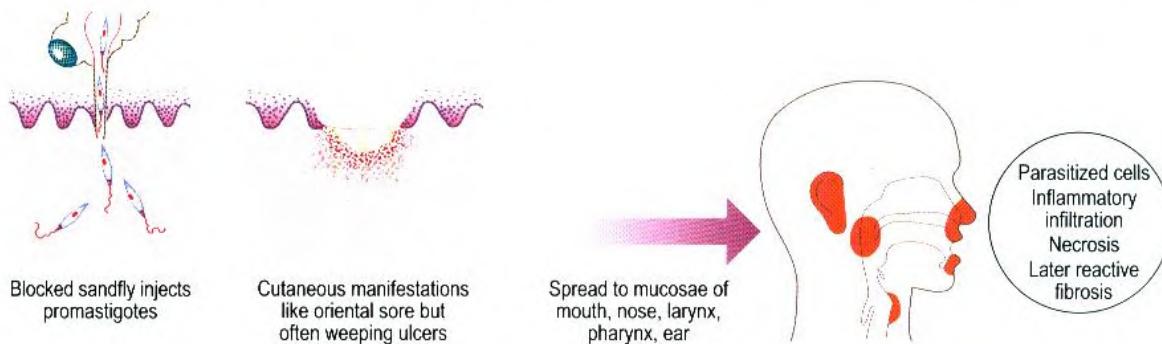
## Cutaneous leishmaniasis

Caused by *Leishmania tropica*, *L. major*, *L. aethiopica*, *L. infantum*, *L. braziliensis* complex



## Mucocutaneous leishmaniasis (espundia)

Caused by some infections with *L. braziliensis* (Central and South America), *L. aethiopica* (Ethiopia), *L. mexicana*



### Secondary effects in loose mucosal tissues

- Oedema and capillary involvement
- Interference with local blood supply
- Necrosis - extensive destruction
- Secondary infection
  - Deep erosion locally
  - Spread of infection to lungs or elsewhere
- Healing with fibrosis

#### Leading to:

- Extensive disfiguring lesions
- General constitutional upset (fever, pain, anaemia)
- Bronchopneumonia and septicaemia

## Diagnosis of Leishmaniasis

### Visceral

Amastigotes can be demonstrated by staining bone marrow, lymph node fluid, nasal scrapings (in the Sudan), liver biopsy or splenic aspiration specimens (although this can be a dangerous procedure). Rarely, amastigotes can be demonstrated in buffy coat preparations from peripheral blood.

### Cutaneous and mucocutaneous

Demonstration of the parasite is possible in stained films from slit-skin smears taken from the indurated edge of an ulcer, biopsy of the margin of the ulcer and from mucosal scrapings in mucocutaneous type.

Culture (NNN or a liquid medium such as Schneider's Drosophila medium or 199 medium with added fetal calf serum) is used for all types of material for diagnosis. Animal inoculation is rarely used now.

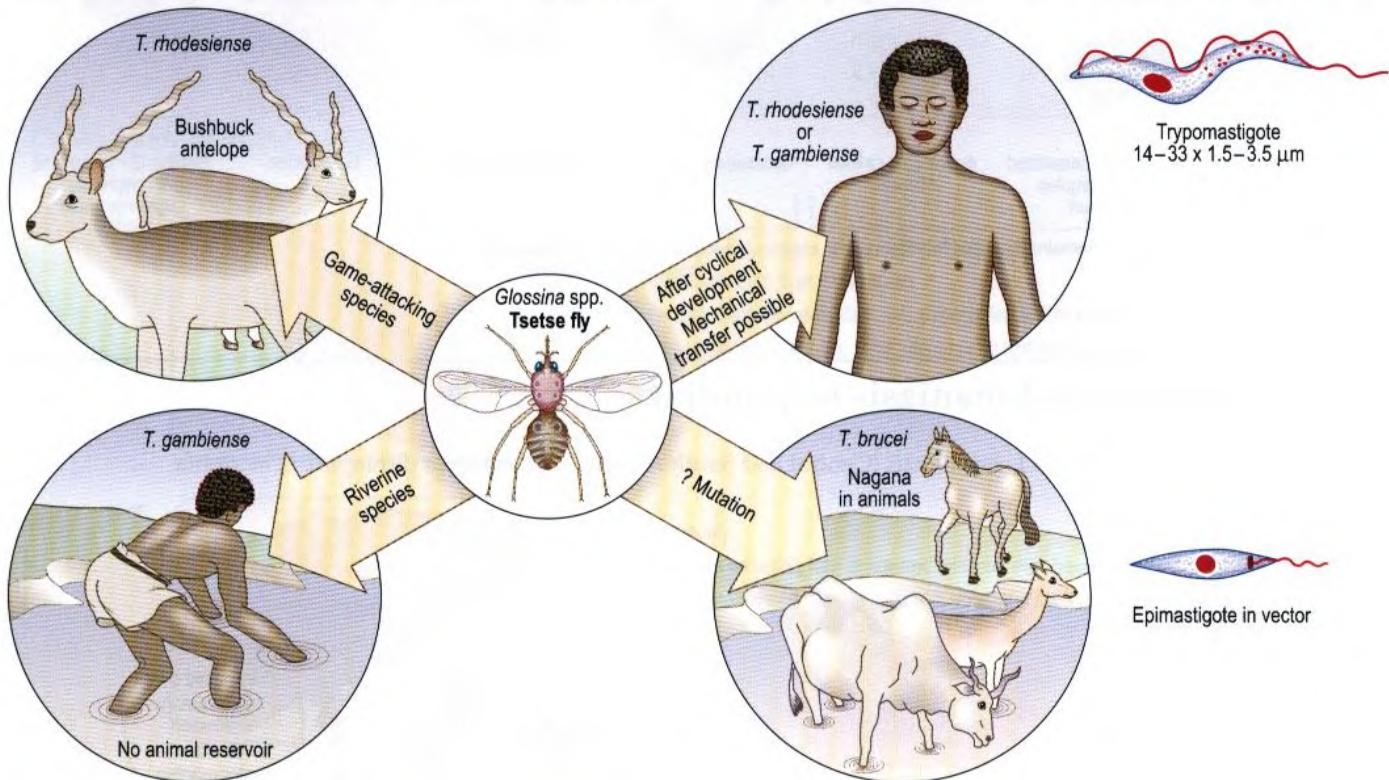
Polymerase chain reaction (PCR) can be used to diagnose and type the species of *Leishmania* present in biopsy or culture material.

Specific serological tests are IFAT, ELISA, direct agglutination test (DAT), or latex agglutination for IgG antibodies. An immunochromatographic test for rK39 antibody detection is also available.

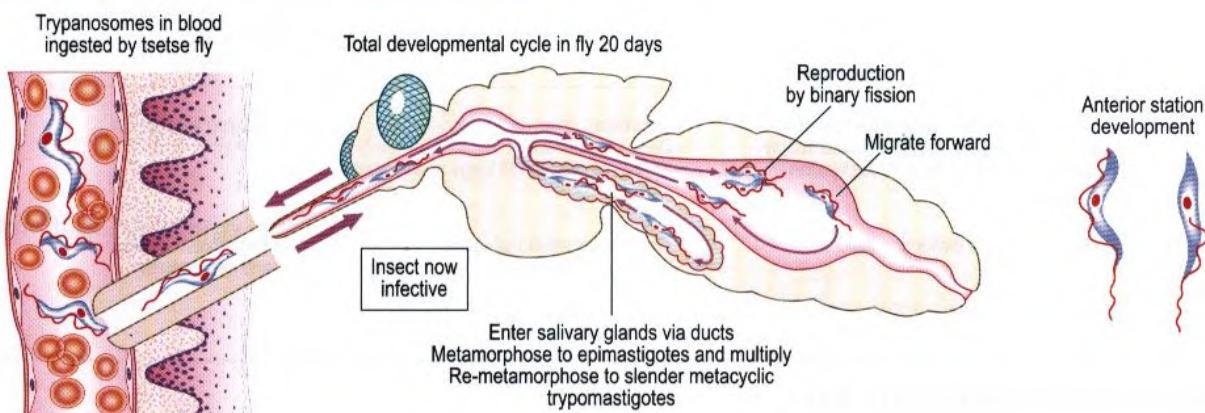
# Trypanosomiasis

## African type: sleeping sickness

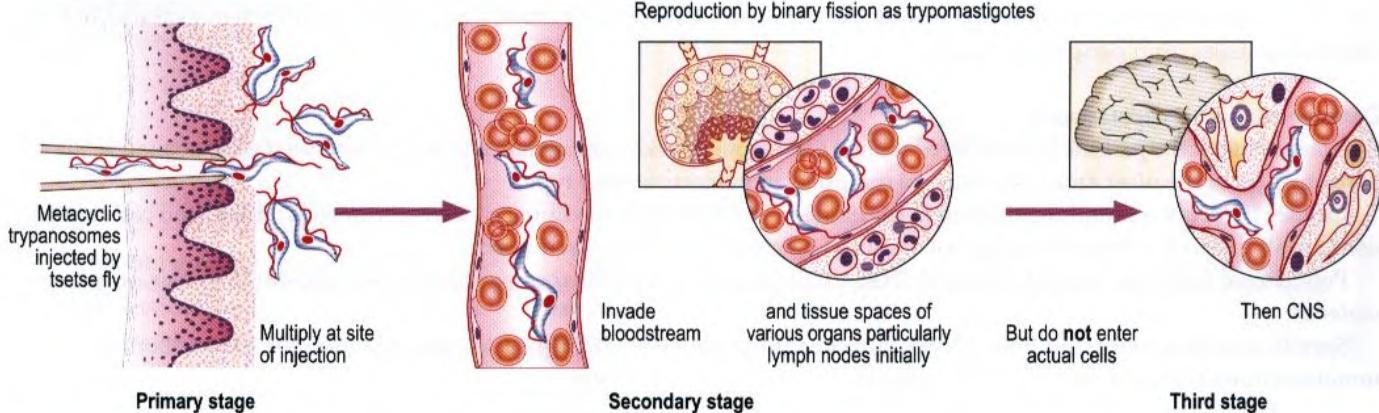
Caused by either *Trypanosoma gambiense* (chronic sleeping sickness, found in West Africa, the Congo, Zaire) or by *T. rhodesiense* (acute sleeping sickness, found in Zimbabwe, Tanzania, Zambia, Angola). Both have similar life cycle and morphology.



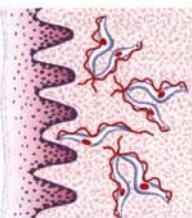
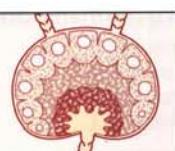
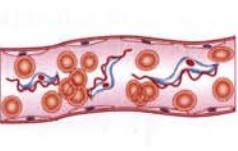
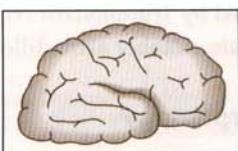
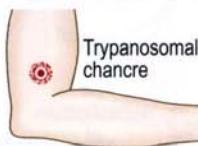
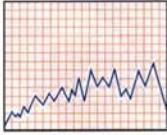
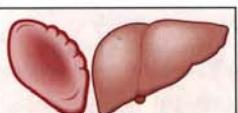
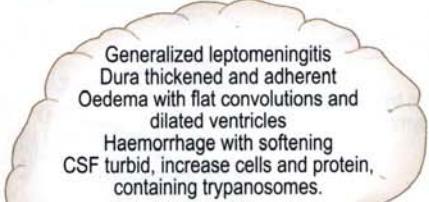
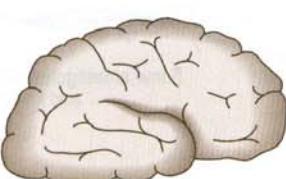
### Life cycle in insect



### Life cycle in humans



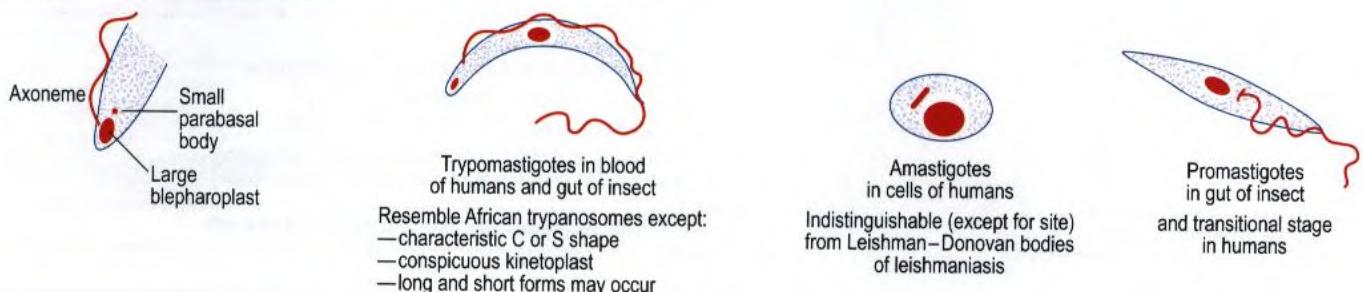
## Pathogenesis and pathology

| Primary stage   | Secondary stage  | Third stage  |
|---|--|--|
|  <ul style="list-style-type: none"> <li>Multiplication at site of injection</li> <li>Surrounding inflammatory reaction</li> </ul> <p>Local inflammatory lesion</p>   |   <ul style="list-style-type: none"> <li>Parasitaemia and toxæmia</li> <li>Invasion of tissue spaces (<b>not cells</b>) of various organs</li> </ul> <p>Predominantly</p> <ul style="list-style-type: none"> <li>Lymph nodes</li> <li>CNS</li> </ul> <ul style="list-style-type: none"> <li>Damage to endothelial cells of blood vessels, surrounding (perivascular) granulomatous reactions and haemorrhages</li> </ul>  |  <p>Toxic degeneration and pressure atrophy of tissue cells</p>   |
|   |  |  |
|   | <p><b>Chronic sleeping sickness</b><br/>(Due to <i>T. gambiense</i>)</p> <p>Differ only in degree</p> <p><b>Primary stage</b></p>  <p>Trypanosomal chancre</p>  | <p><b>Acute sleeping sickness</b><br/>(Due to <i>T. rhodesiense</i>)</p> <p>Clinico-pathological correlation</p> <p>As in chronic</p>  |
| <p><b>Fever</b></p> <ul style="list-style-type: none"> <li>Low</li> <li>Irregular</li> <li>Recurrent</li> </ul> <p><b>General toxic symptoms</b></p> <ul style="list-style-type: none"> <li>Backache</li> <li>Headache</li> <li>Tachycardia</li> <li>Irregular skin rashes (circinate)</li> <li>Transient oedema face</li> </ul> <p><b>Lymphadenopathy</b></p> <ul style="list-style-type: none"> <li>Typically post-cervical</li> </ul> <p><b>Later anaemia</b><br/>monocytosis</p> <p><b>Slight enlargement liver,</b><br/>spleen</p> | <p><b>Secondary stage</b> – predominantly blood and lymph node involvement</p>   <p>Congestion<br/>Sinus catarrh<br/>Perivascular cuffing</p> <p>Enlarged, soft, red.<br/>Later regressive, fibrotic</p>  <p>Congested, slightly enlarged</p>  <p>Toxic depression<br/>Bone marrow</p>   | <p><b>Fever</b></p> <ul style="list-style-type: none"> <li>High</li> <li>Persistent</li> </ul> <p><b>Severe toxic symptoms</b></p> <ul style="list-style-type: none"> <li>Headache</li> <li>Vomiting</li> <li>Shivering</li> <li>Oedema face</li> <li>Serous effusion</li> <li>Bone pain</li> </ul> <p><b>Lymphadenopathy</b><br/><b>Myocarditis</b><br/><b>Anaemia</b><br/><b>Purpura</b><br/><b>Hepatitis</b></p> <p>Often death at this stage</p> |
| <p><b>Progressive involvement of the CNS</b></p> <p>General symptoms of progressive encephalitis</p> <p>Focal signs uncommon</p> <p>Dementia</p>  | <p><b>Third stage</b> – CNS involvement</p>  <p>Generalized leptomeningitis<br/>Dura thickened and adherent<br/>Oedema with flat convolutions and dilated ventricles<br/>Haemorrhage with softening<br/>CSF turbid, increase cells and protein, containing trypanosomes.</p> <p>Perivascular cuffing with round and plasma cells, macrophages and endothelial cells<br/>Neuroglial proliferation<br/>Pressure atrophy neurones</p>  <p>Death before CNS involvement<br/>or<br/>Similar changes but more acute</p> | <p>May have early onset of encephalitis with rapid development of <b>coma</b></p>  |
| <p><b>Note on epidemiology</b> – Vectors of <i>T. gambiense</i> are riverine species, hence disease often epidemic:</p> <ul style="list-style-type: none"> <li><i>G. palpalis</i></li> <li><i>G. tachinoides</i></li> </ul>   | <p>Vectors of <i>T. rhodesiense</i> are game-attacking species, hence disease more often sporadic:</p> <ul style="list-style-type: none"> <li><i>G. morsitans</i></li> <li><i>G. pallidipes</i></li> <li><i>G. swynnertoni</i></li> </ul>  |  |

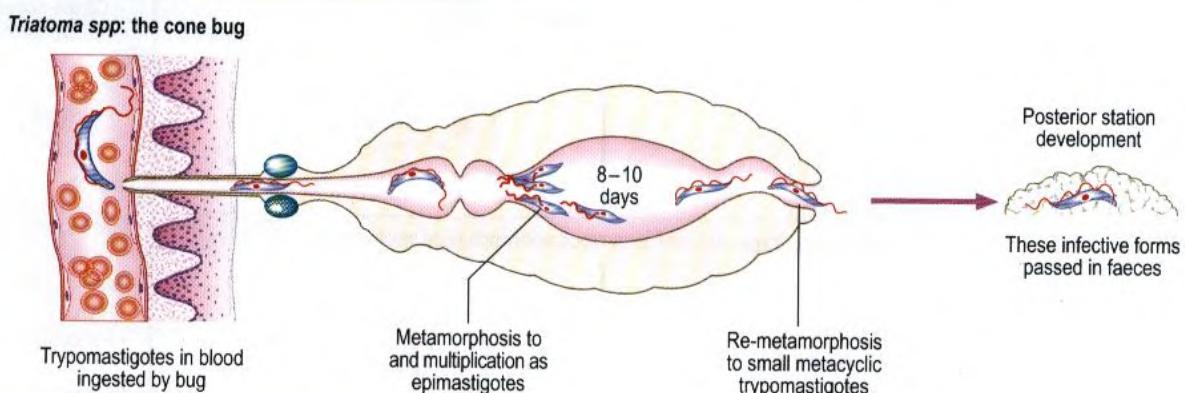
### South American type: Chagas' disease

Caused by *Trypanosoma cruzi*. The parasite is harboured in humans, domestic animals such as cats and dogs, and some wild animals, notably armadillos and opossums.

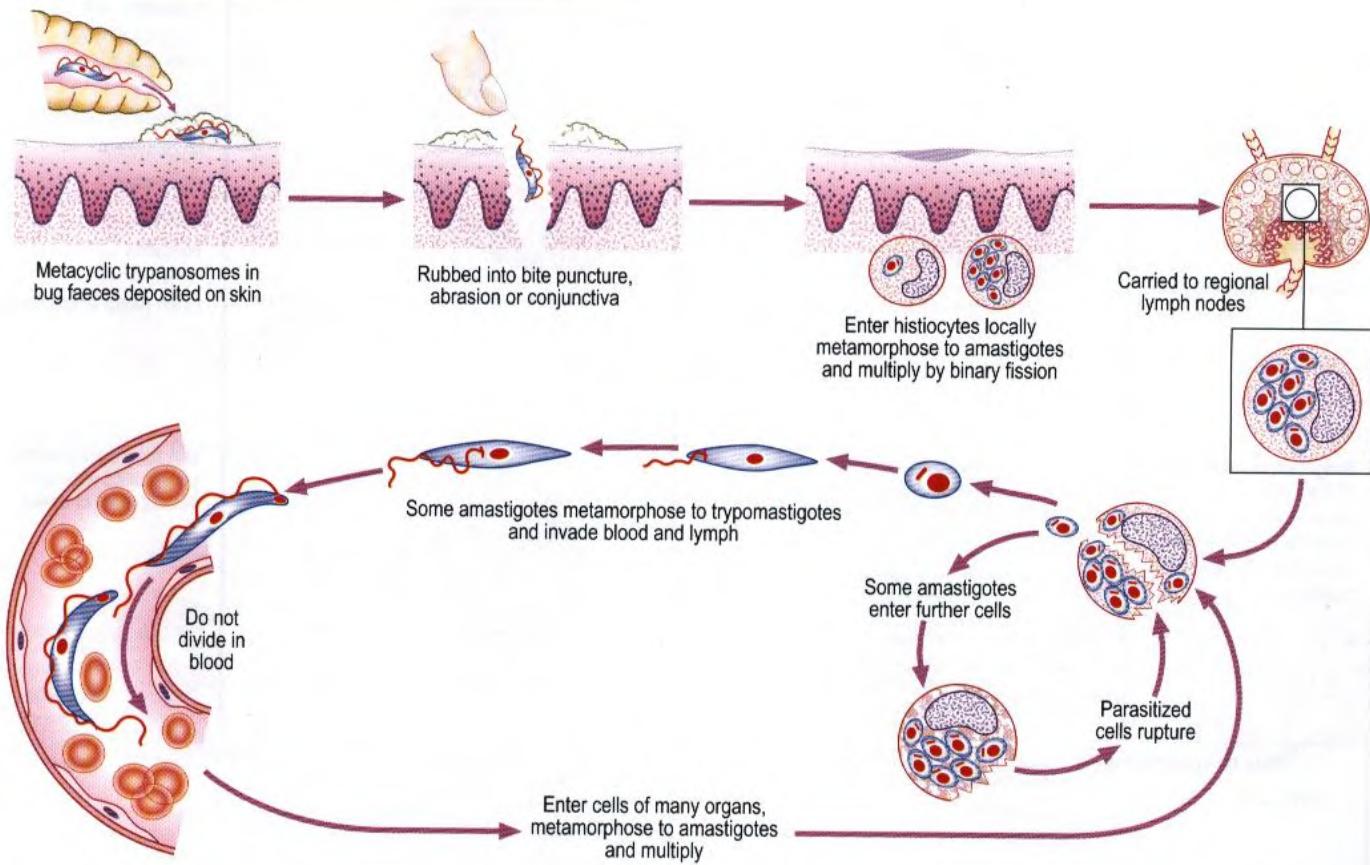
#### Morphology



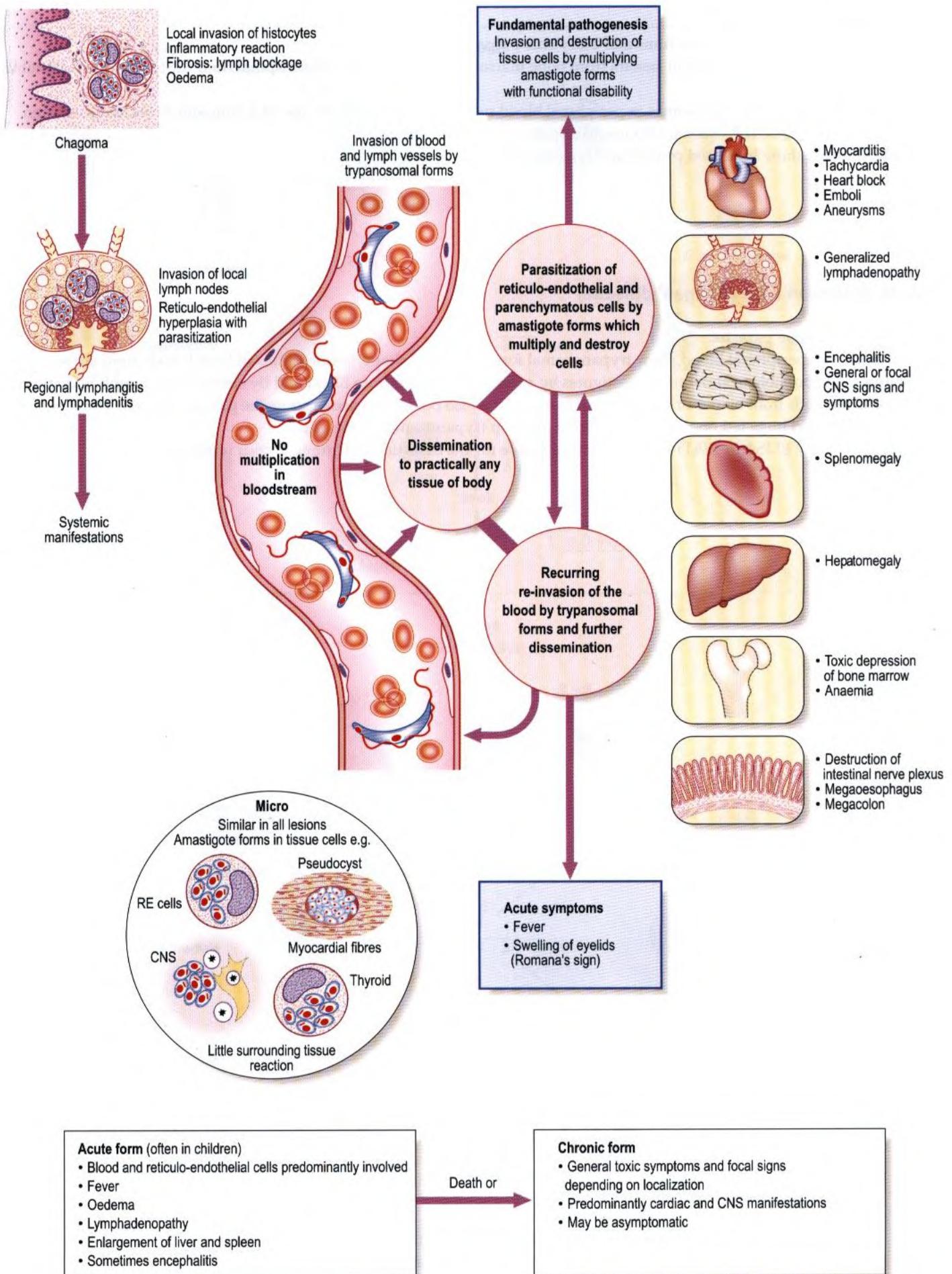
#### Life cycle in insect



#### Life cycle in humans



## Pathogenesis and pathology



## Laboratory diagnosis of trypanosomiasis

### African type (sleeping sickness)

#### Demonstration of the parasite.

Microscopy of thin and thick blood films and buffy coat preparations.

Trypanosomes can also be seen in smears from bone marrow and centrifuged cerebrospinal fluid (CSF). Culture is possible but difficult.

Microscopic detection of trypanosomes in peripheral blood may be improved by the use of a mini-anion- exchange column or by the use of the QBC11® (Becton Dickinson) to concentrate the parasite.

The CSF might show increased protein and lymphocytes.

### South American type (Chagas' disease)

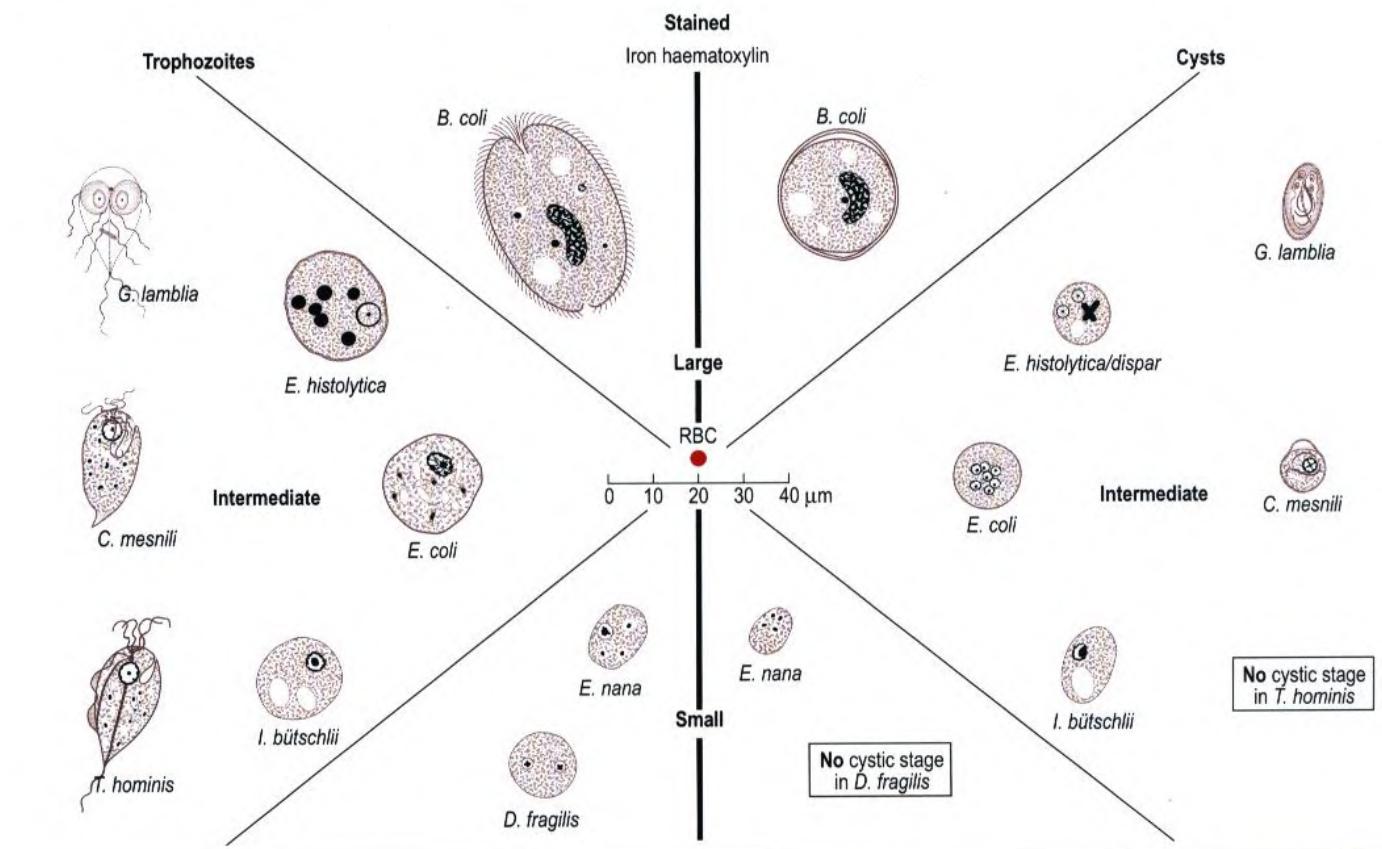
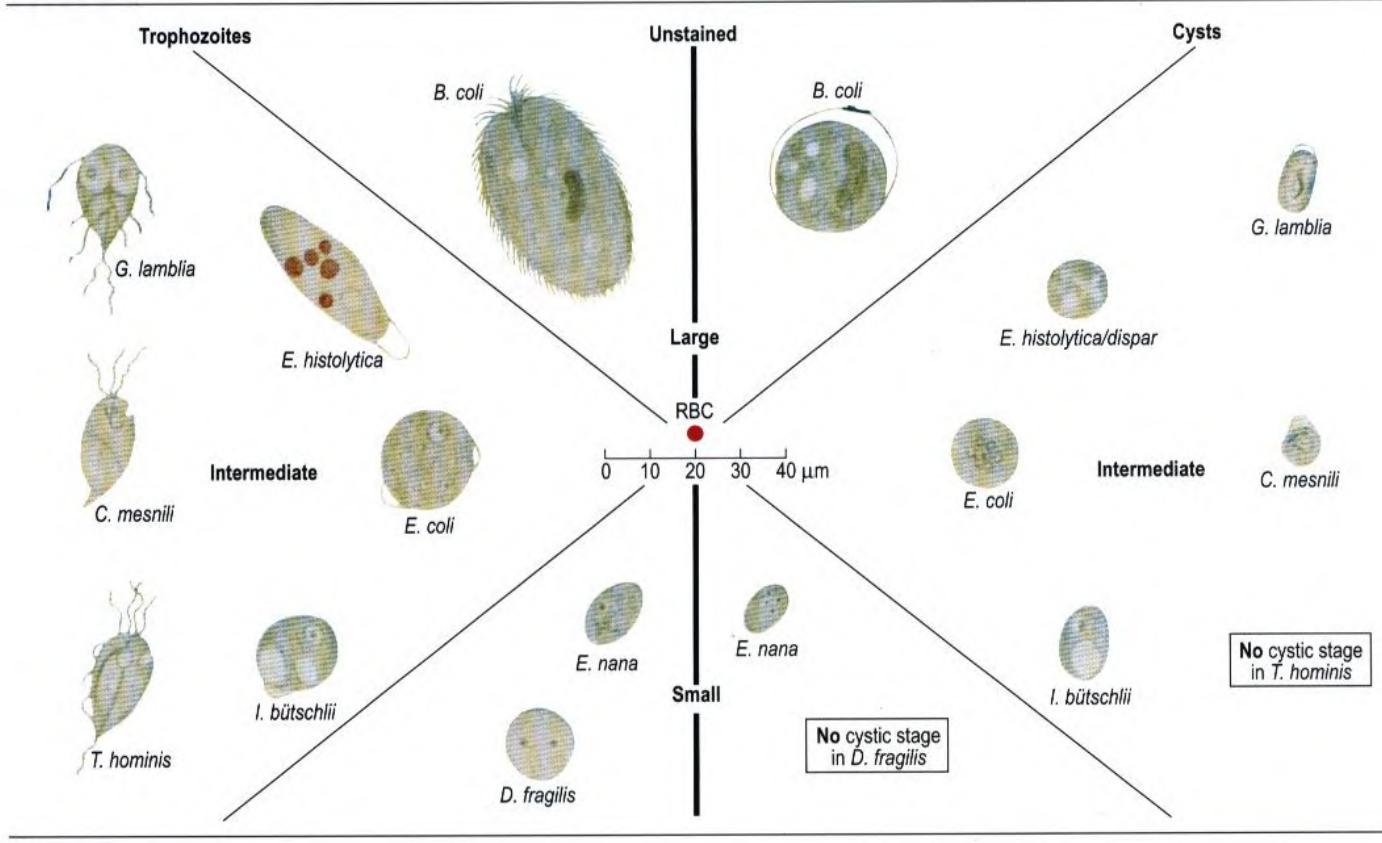
#### Demonstration of the parasite.

Stained smears of peripheral blood show trypanosomal forms in C or S shape. Stained films of lymph node fluid show amastigotes. It is possible to show trypomastigotes by animal inoculation from blood and by culture from lymph node fluid. Histological methods from biopsy or post-mortem material. It is also possible to demonstrate the parasite through xenodiagnosis: clean bred triatomid bugs fed on the patient's blood develop trypanosomes in the gut.

Serology (e.g. by ELISA or IFAT) is the method of choice for the detection of chronic *T. cruzi* infection.

# Recapitulation

## Luminal intestinal protozoa



Refer to text for the following additional stained appearances:

- trichrome stain for coccidia and microsporidia spp.
- modified Ziehl-Neelsen for *Cyclospora*, *Isospora* and *Cryptosporidium* spp.



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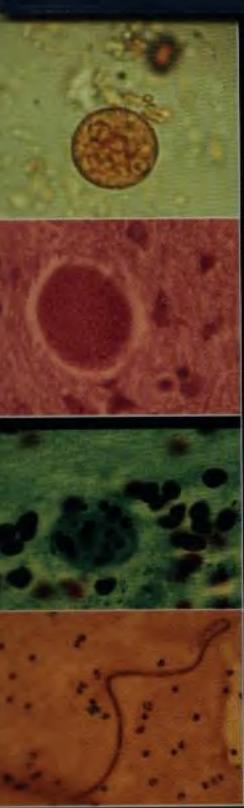
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