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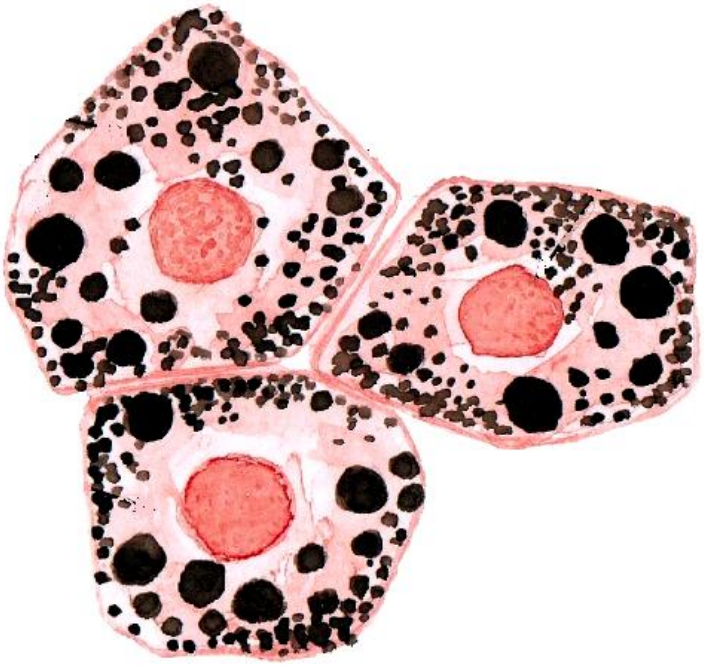
Ulyanovsk State University

Institute of Medicine, Ecology and Physical culture

N. A. KURNOSOVA, N. A. MICHEEVA

ATLAS OF BIOLOGY



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Kurnosova N.A.
К75 **ATLAS OF BIOLOGY**/ N.A. Kurnosova, N.A. Micheeva**. -** Ulyanovsk : Ulyanovsk State University,2018. - 120 p.

The Atlas includes all the illustrations (microphotographs and diagrams) on the subject "Biology with ecology". The Atlas is designed according to course of biology for students of Medical department. It will be a guide to action for students during their practical work.

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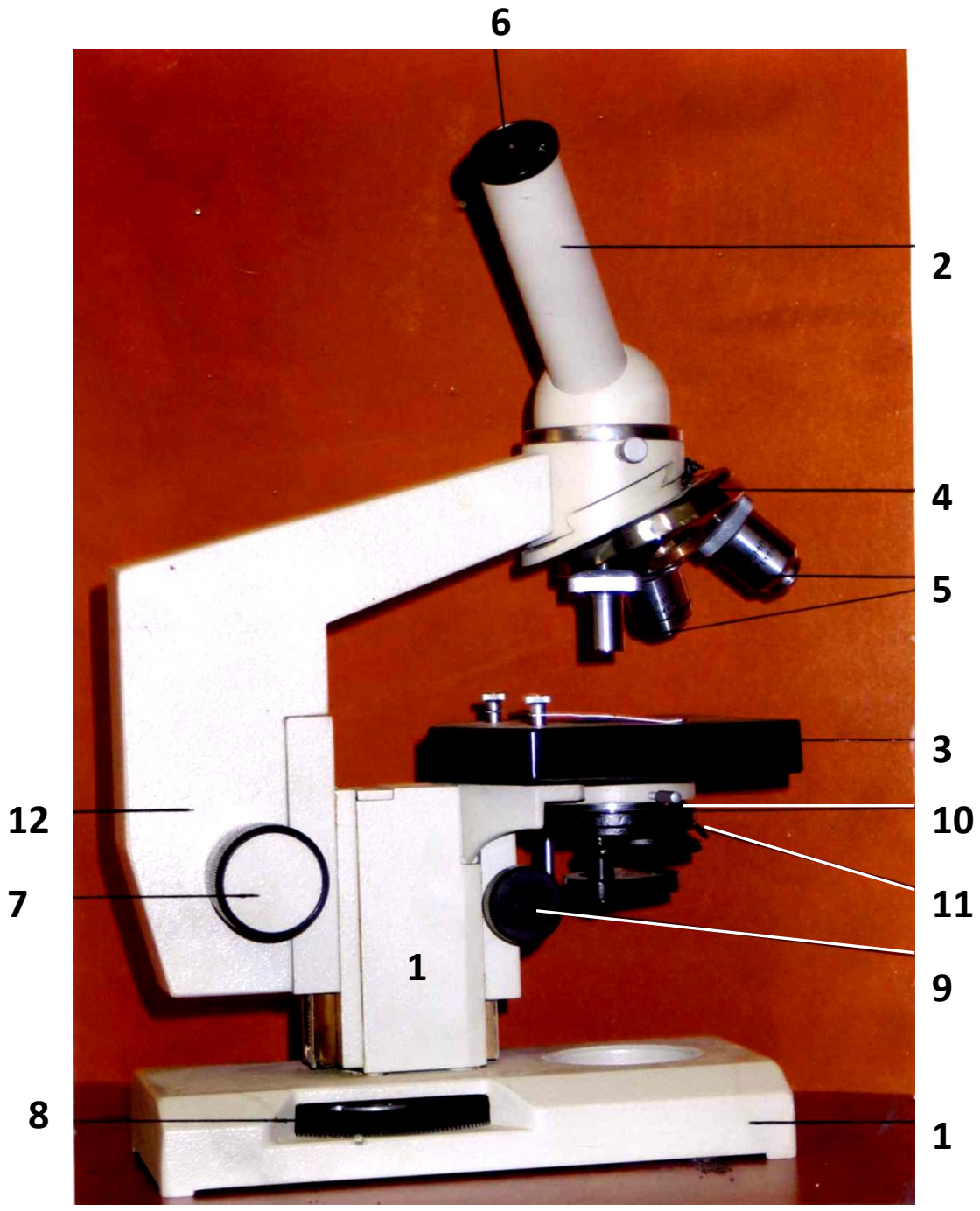
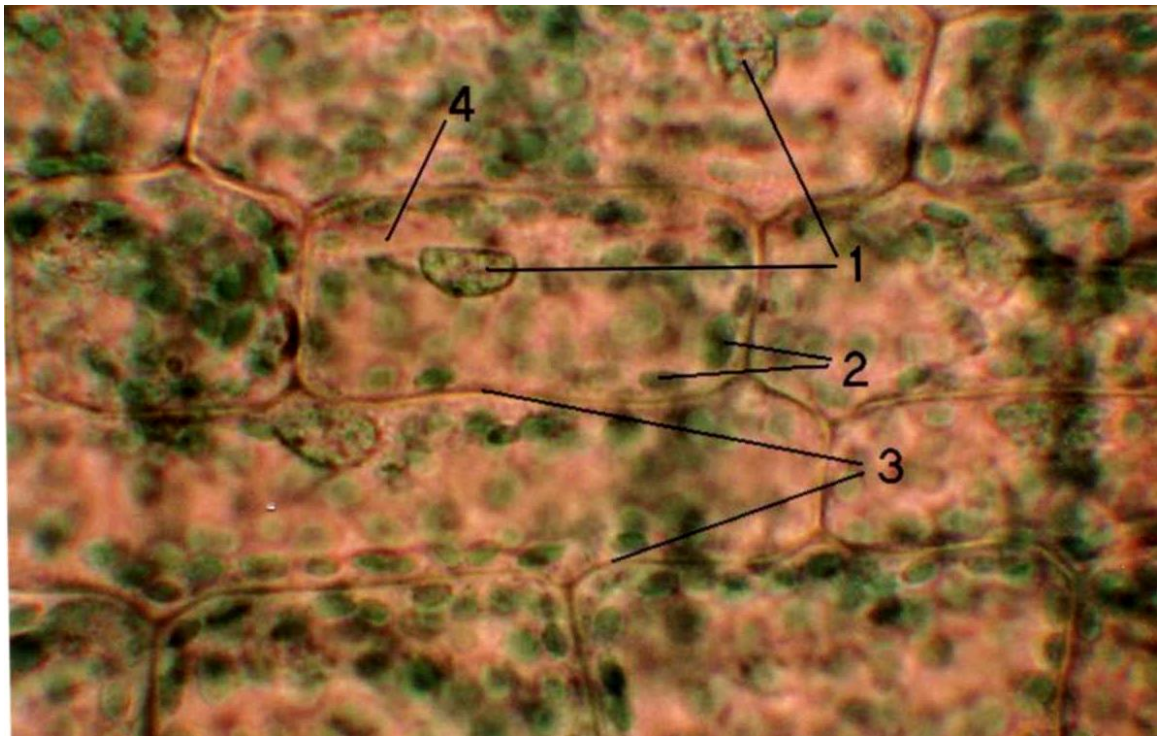
CONTENT OF COURSE:

Cytology



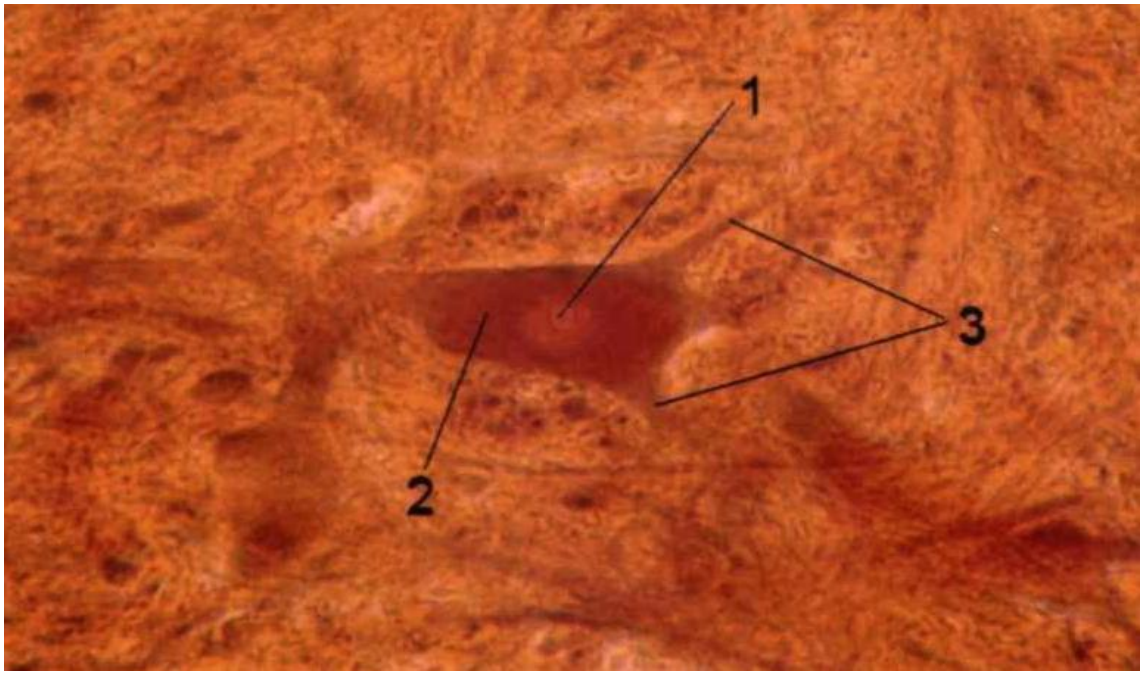
Microscope "Biolam P-11": 1-base; 2-tube; 3-mechanical stage; 4-revolver (revolving nosepiece); 5-objective lenses; 6-eyepiece; 7-coarse focus adjustment knob ; 8-fine focus adjustment knob; 9-condenser adjustment knob; 10-diaphragm; 11- lever on the right side of the condenser; 12-arm.

Plant cell



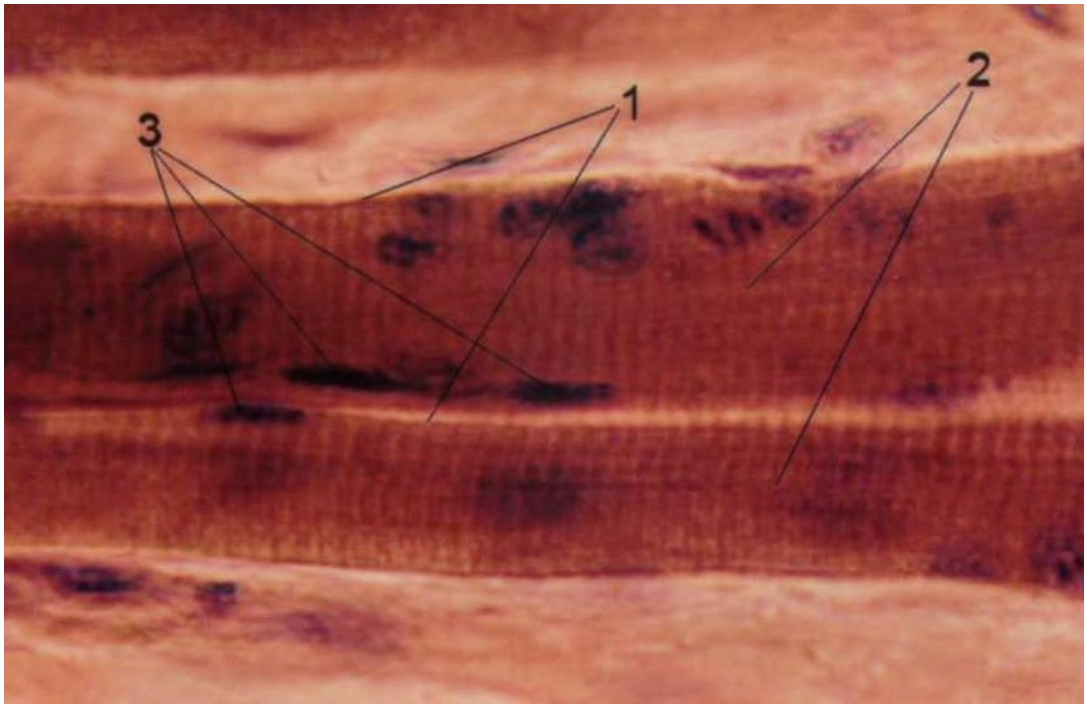
Indicate in the figure: 1) core; 2) chloroplasts; 3) cell boundaries; 4) vacuole.

The spinal cord of the dog



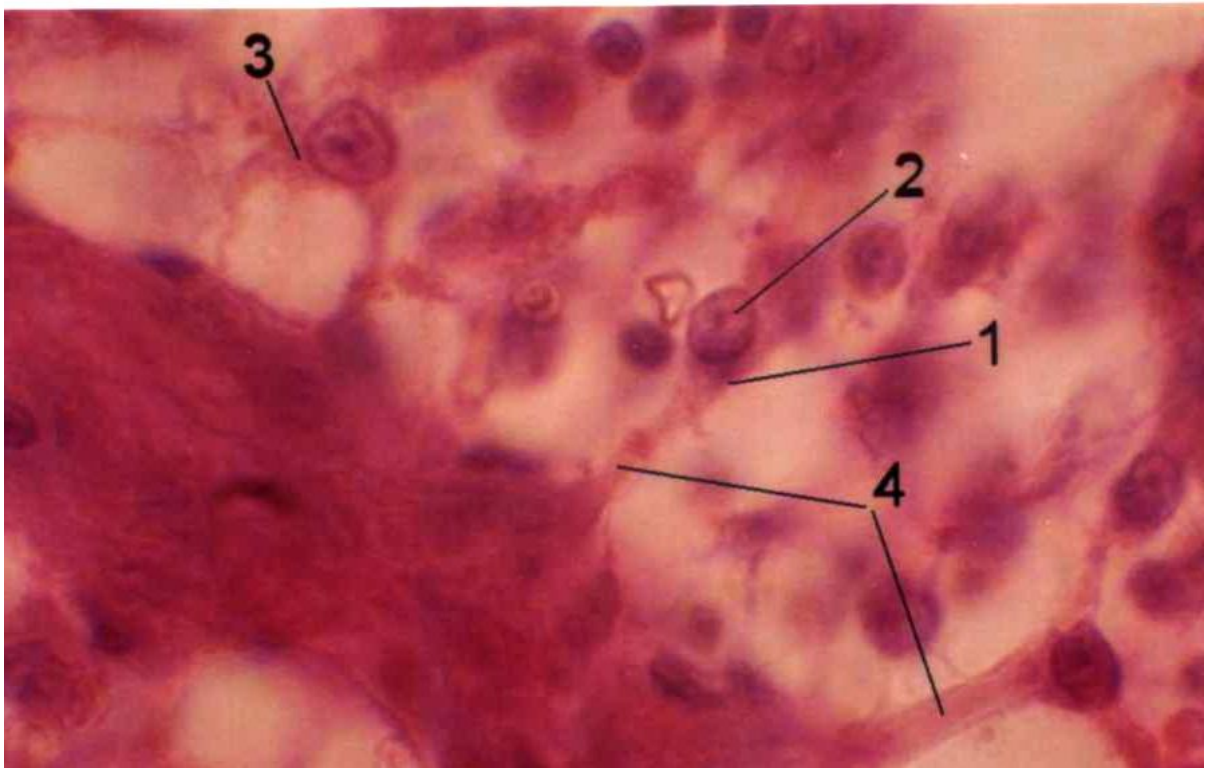
Indicate in the figure: 1) the core of the nerve cells; 2) cytoplasm; 3) the spikes of the neuron.

Simplast - cross-striated skeletal muscle fiber



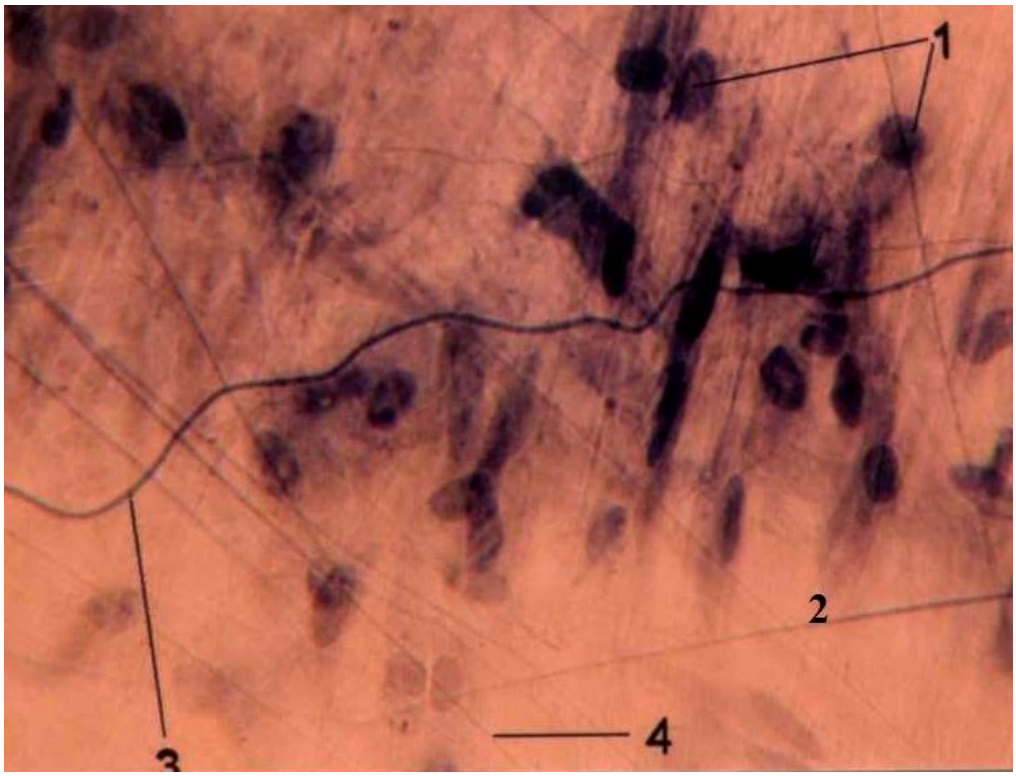
Indicate in the figure: 1) the external boundaries of the fiber; 2) cytoplasm (sarcoplasm); 3) nucleus

The syncytium - reticular tissue of bone marrow



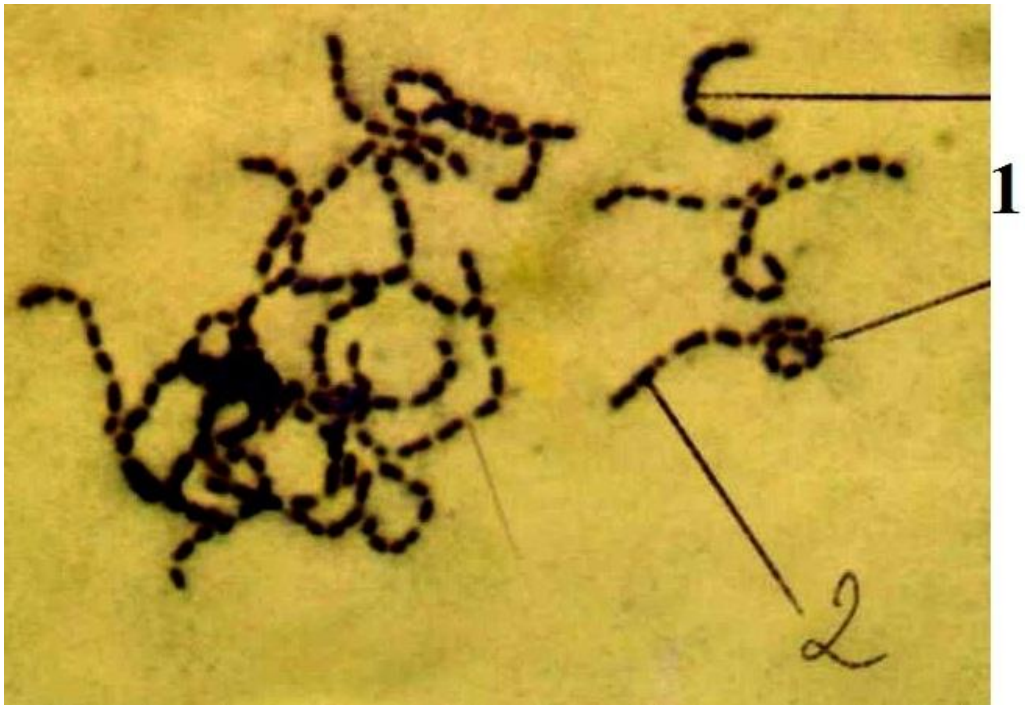
Indicate in the figure: 1) reticular cells of the syncytium; 2) the core of reticular cells; 3) the cytoplasm of reticular cells; 4) processes of reticular cells.

Intercellular substance of loose fibrous connective tissue



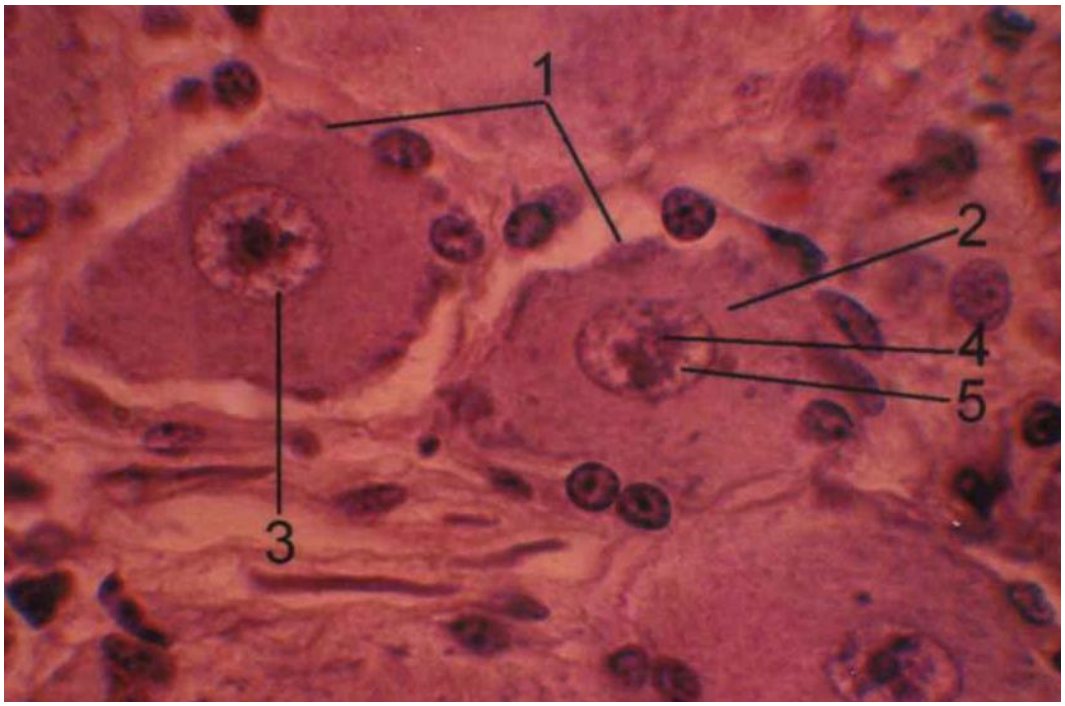
Indicate in the figure: 1) cell unformed loose fibrous connective tissue; 2) amorphous substance; 3) collagen fibers; 4) elastic fibers.

Bacterial cells



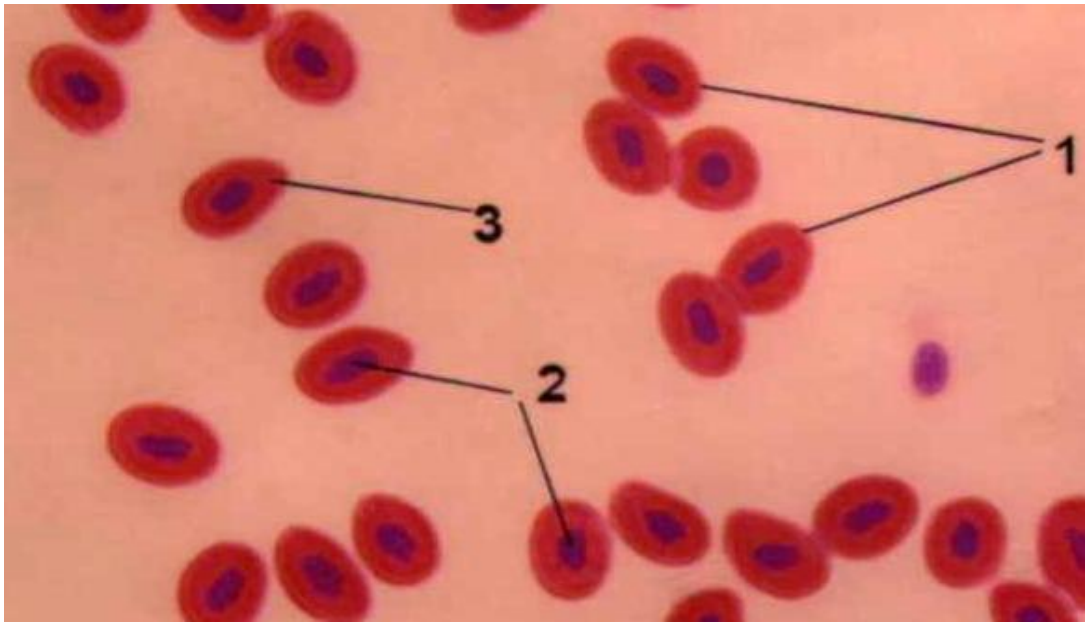
Indicate in the figure: 1) the chain of bacterial cells; 2) the bacterium is rod-shaped

Spinal ganglion of the dog



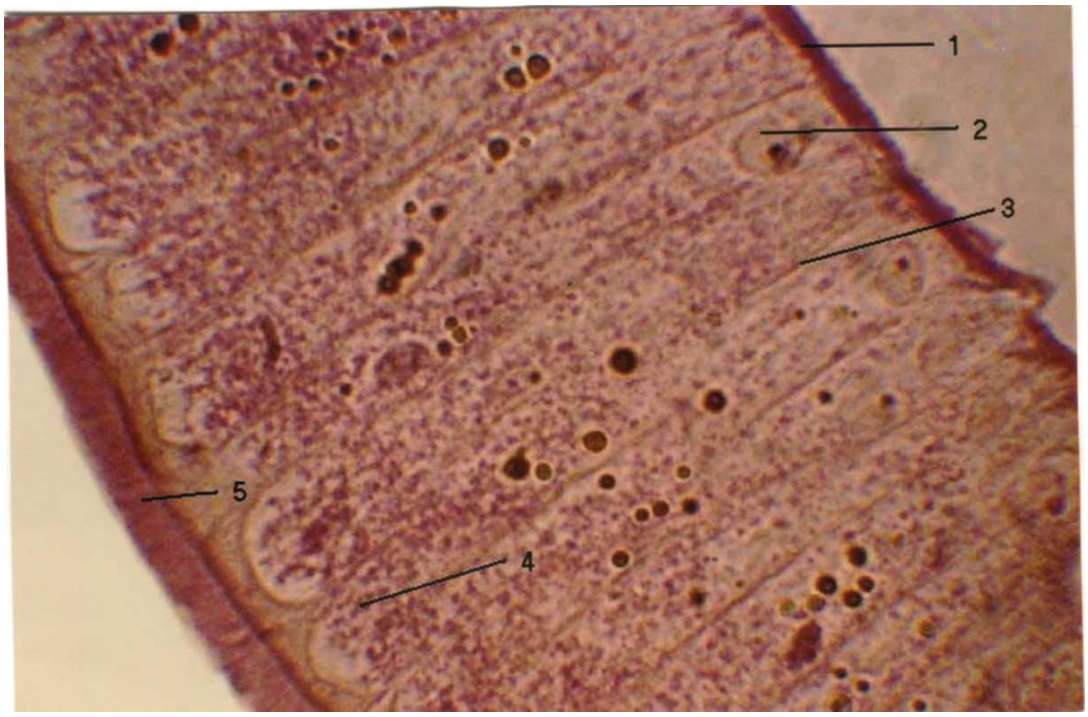
Indicate in the figure: 1) the cell boundaries; 2) the cytoplasm of nerve cells; 3) the core; 4) heterochromatin; 5) euchromatin.

Blood of frog



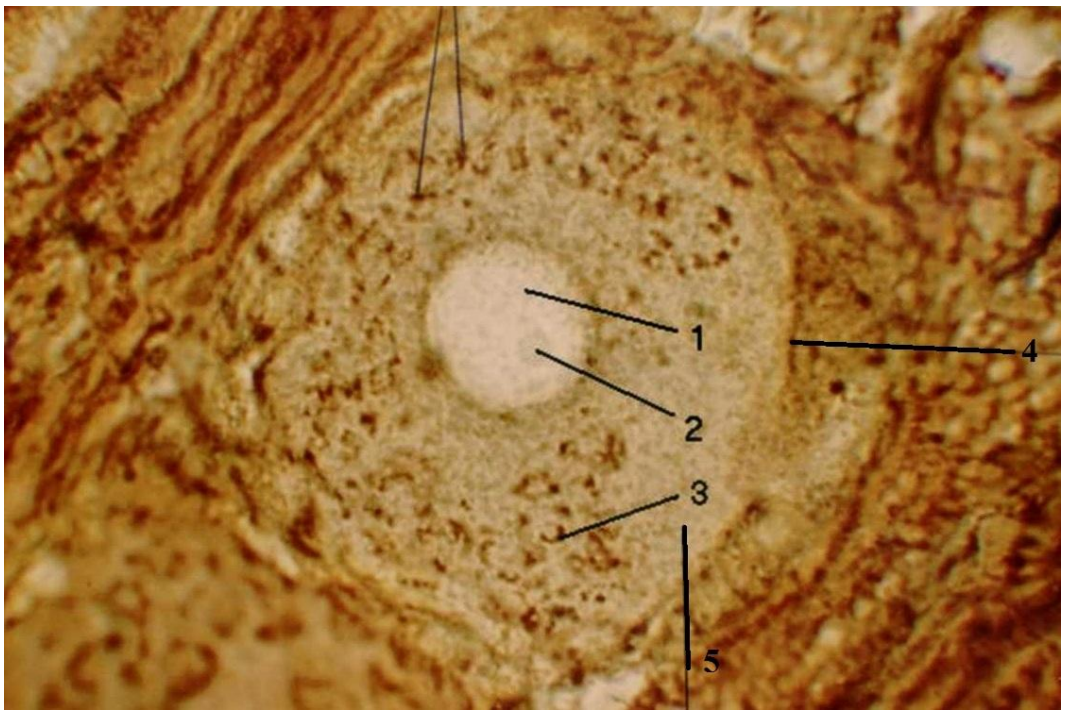
Indicate in the figure: 1) red blood cells; 2) the nucleus; 3) the cytoplasm

Chondriosomes (mitochondria) in the epithelial cells of the intestinal roundworm (dyed by iron haematoxylin)



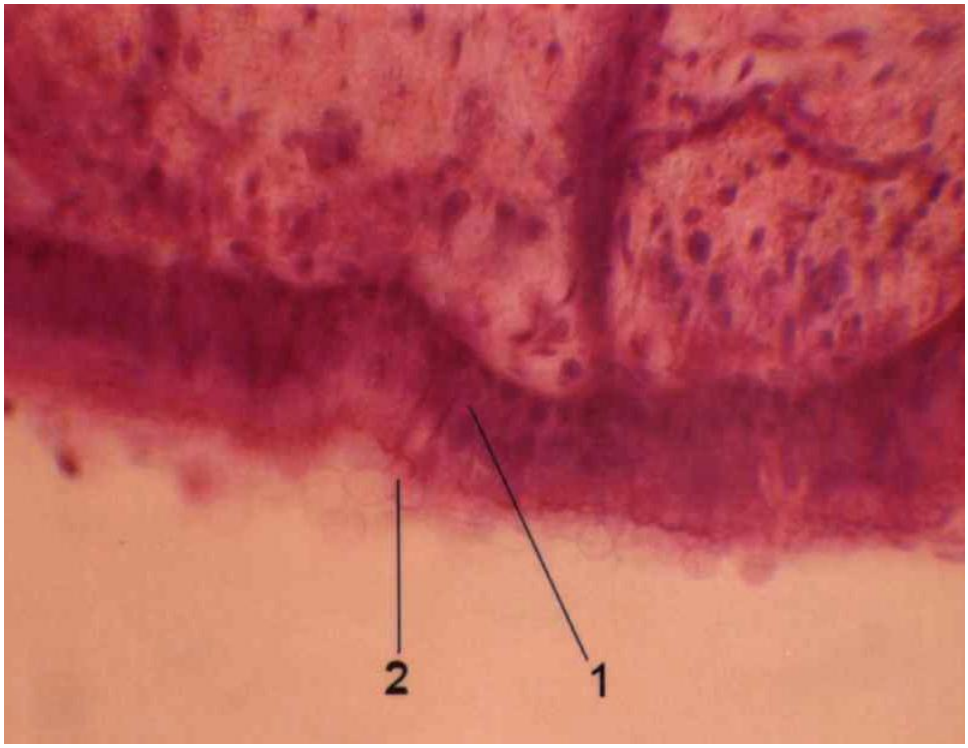
Indicate in the figure: 1) basal membrane; 2) the core; 3) plasmalemma; 4) mitochondria; 5) brush border (microvilli).

The Golgi complex (by impregnation with silver salts)



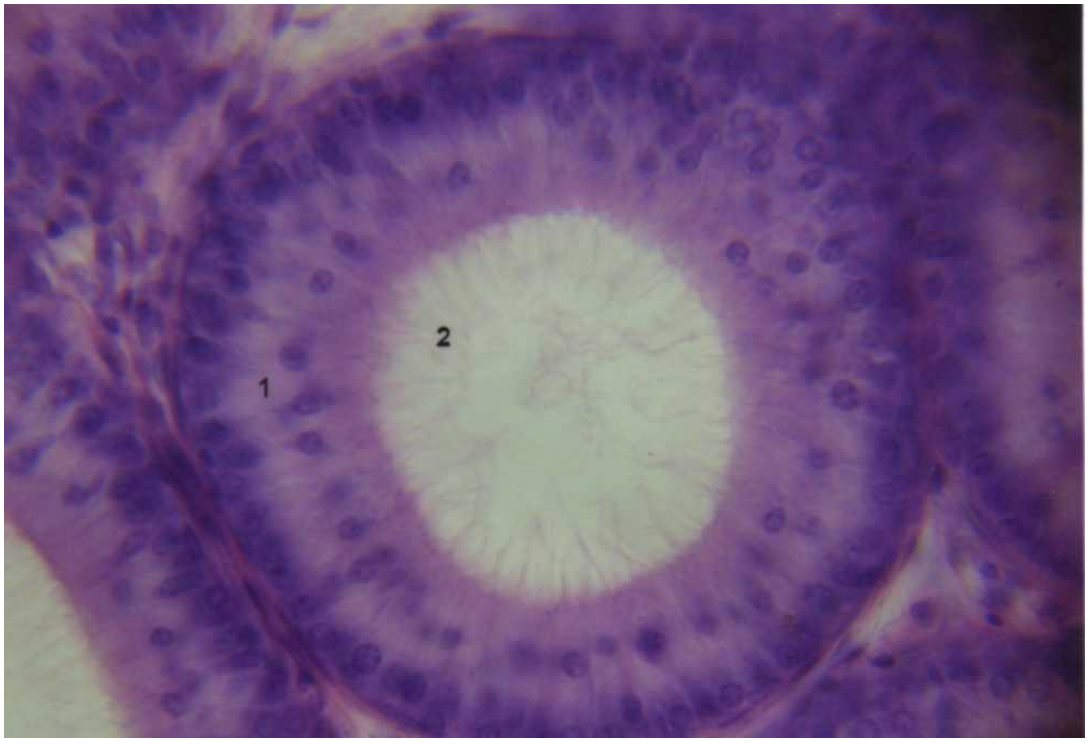
Indicate in the figure: 1) nucleus; 2) the nucleolus; 3) the Golgi complex; 4) the cell boundaries; 5) the cytoplasm

Ciliated cilia of epithelial cells of the trachea (coloring with hematoxylin-eosin) Note edging apical end of epithelial cells formed cilia.



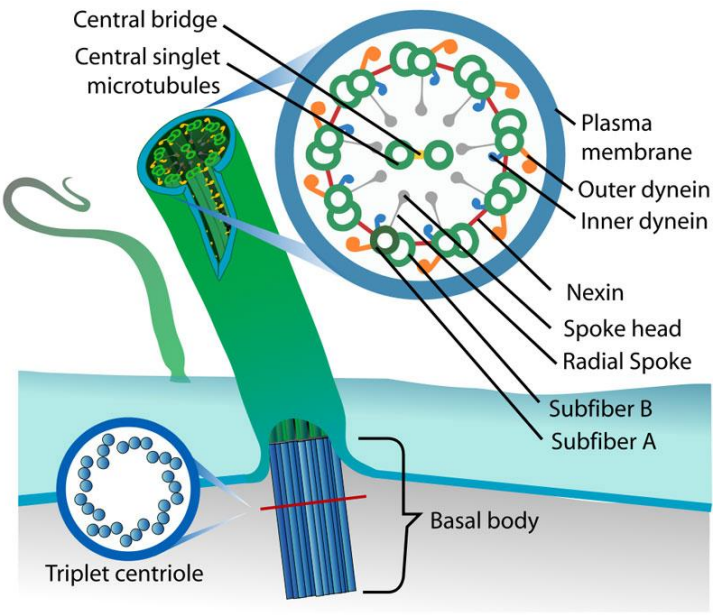
Indicate in the figure: 1) epithelial cell: 2) cilia

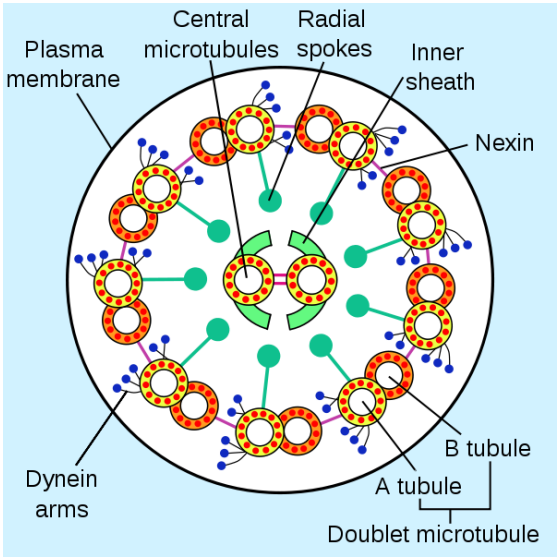
Ciliated cilia of epithelial cells of the canal of the epididymis testicles (dyed with hematoxylin-eosin).



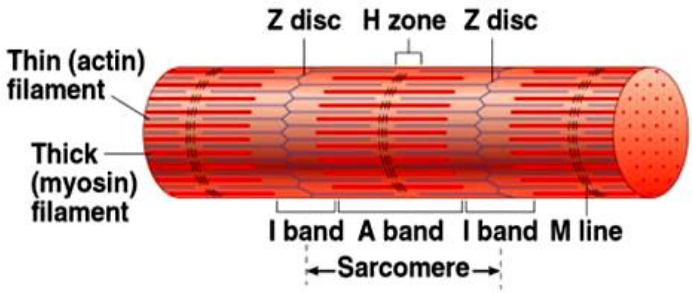
Indicate in the figure: 1) epithelial cell: 2) cilia

The scheme and draw the structure of cilia

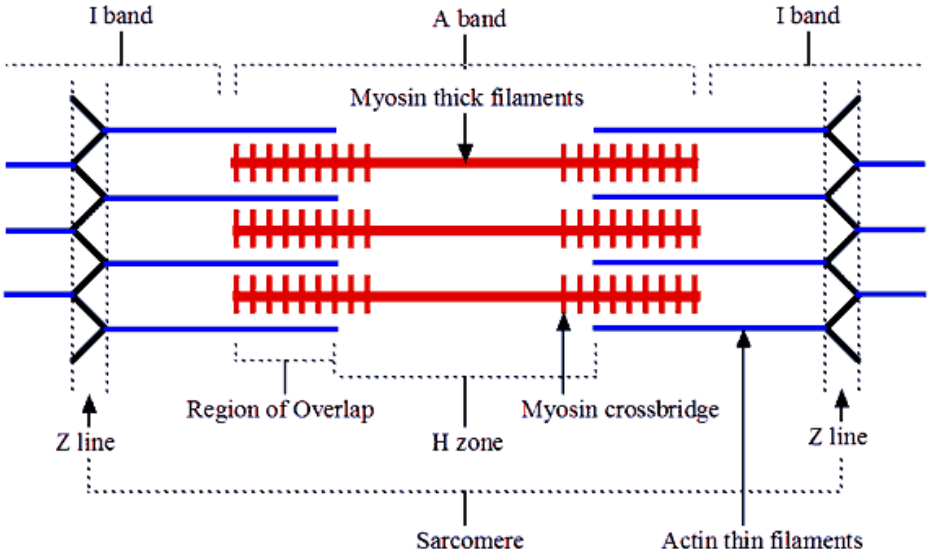




The scheme and draw the structure of myofibril



(b) Myofibril or fibril
(complex organelle composed of bundles of myofilaments)



The structure of the nuclear pore

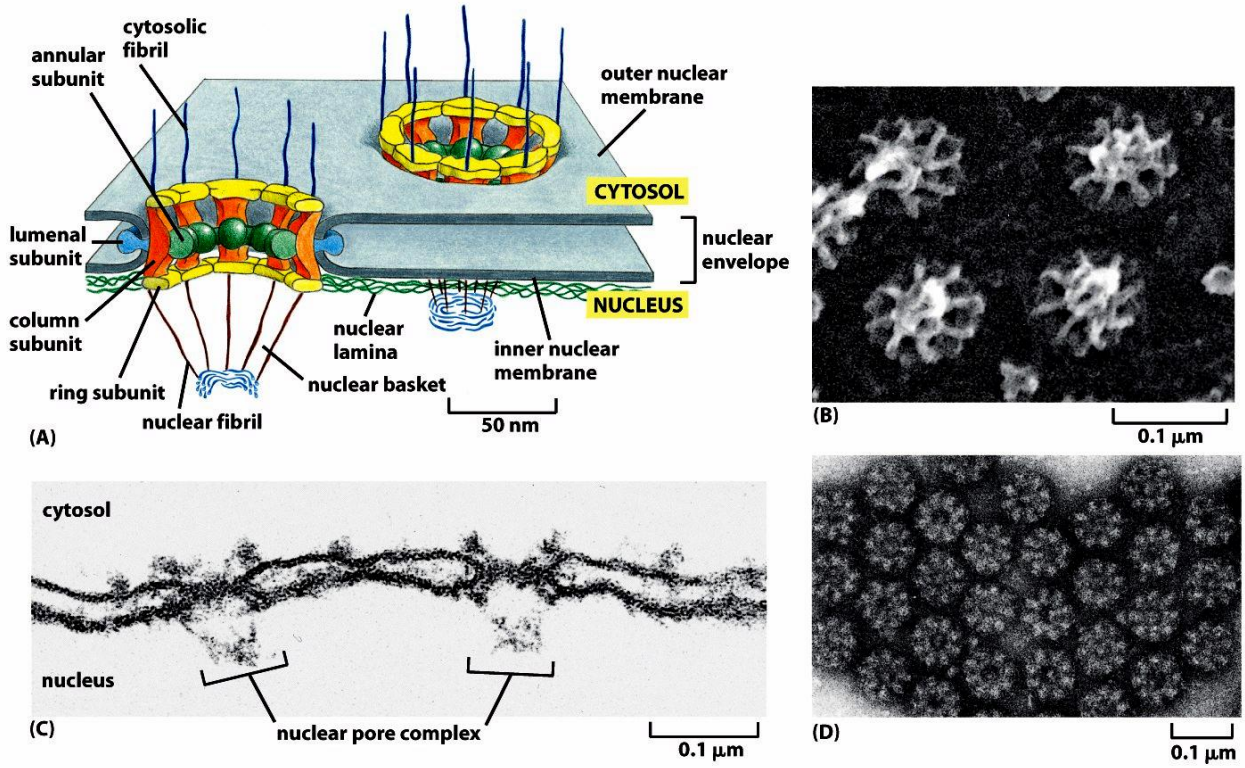
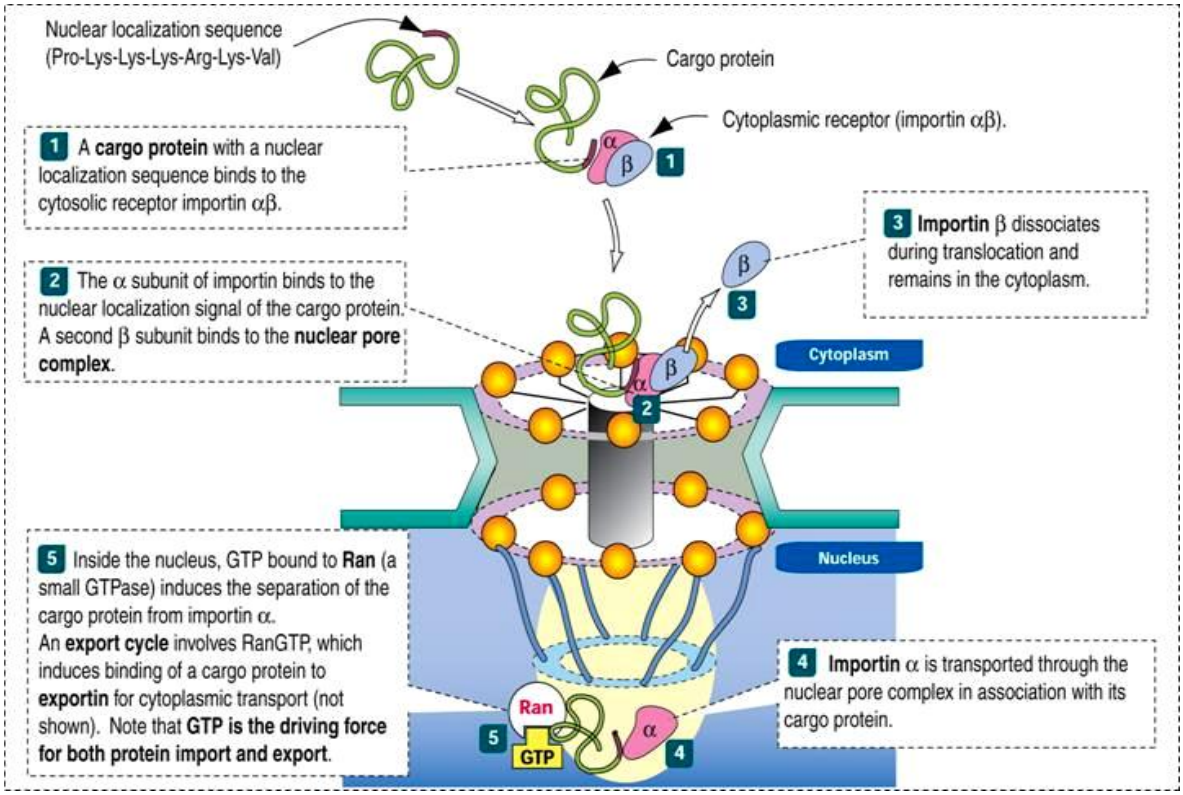
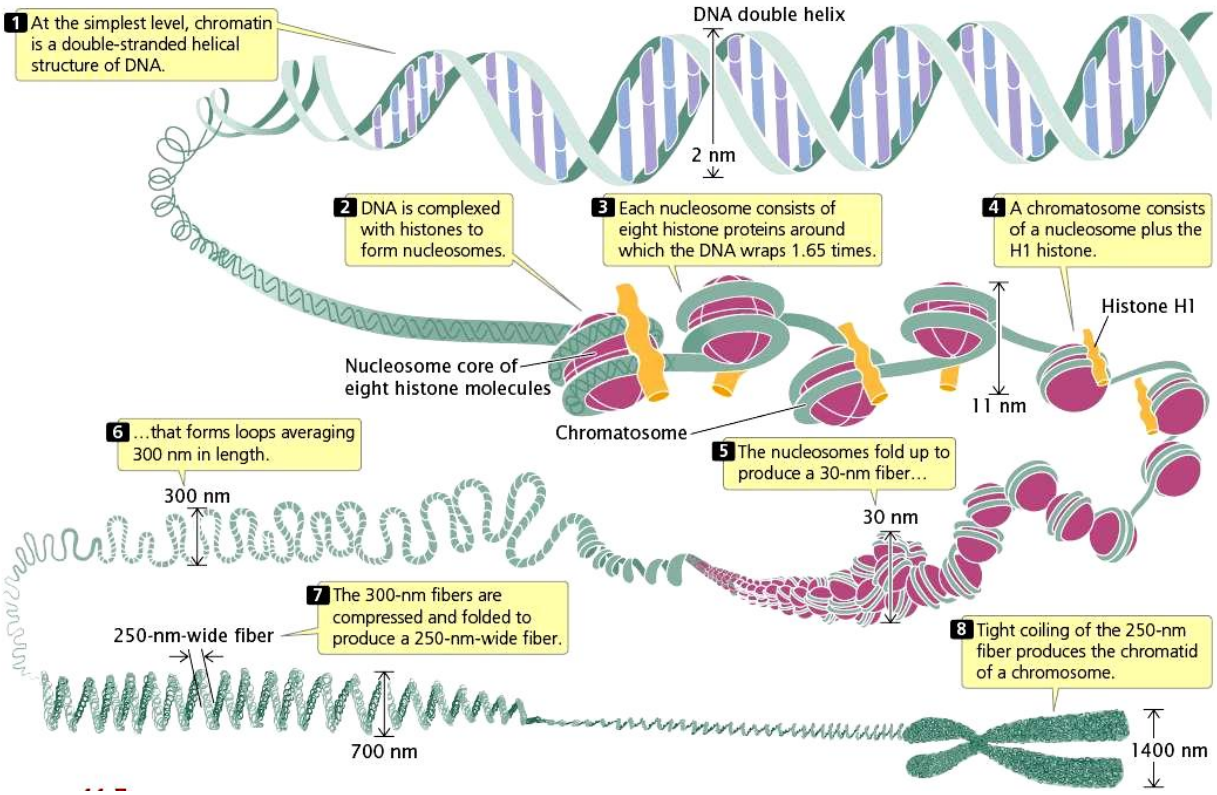
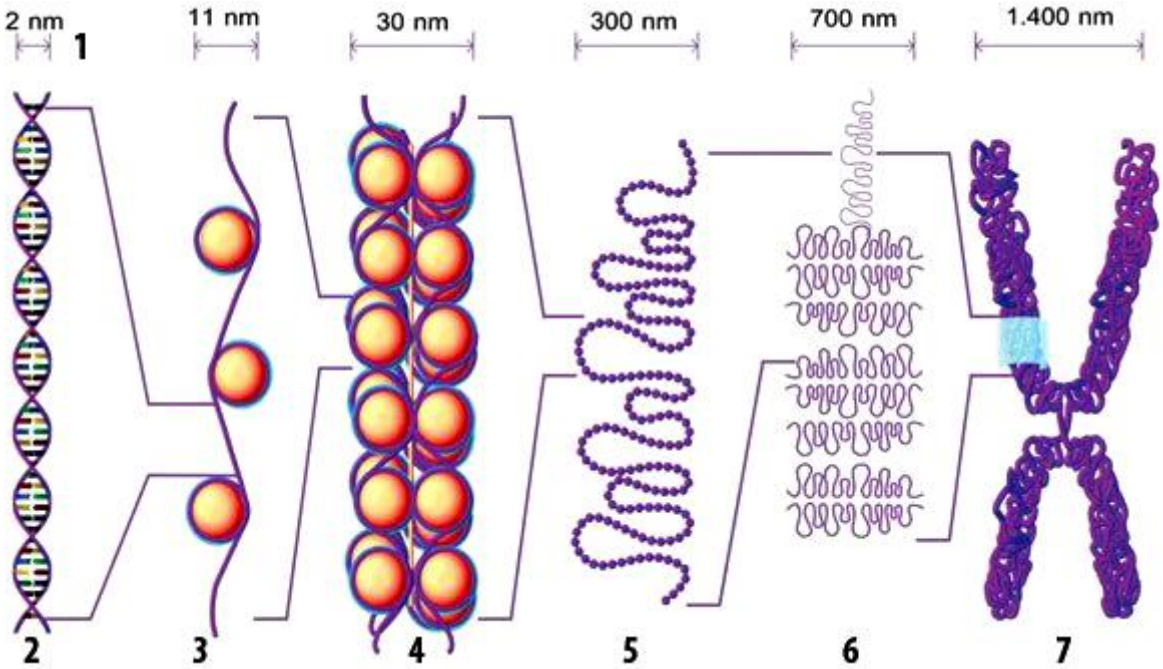


Figure 12-9 Molecular Biology of the Cell 5/e (© Garland Science 2008)

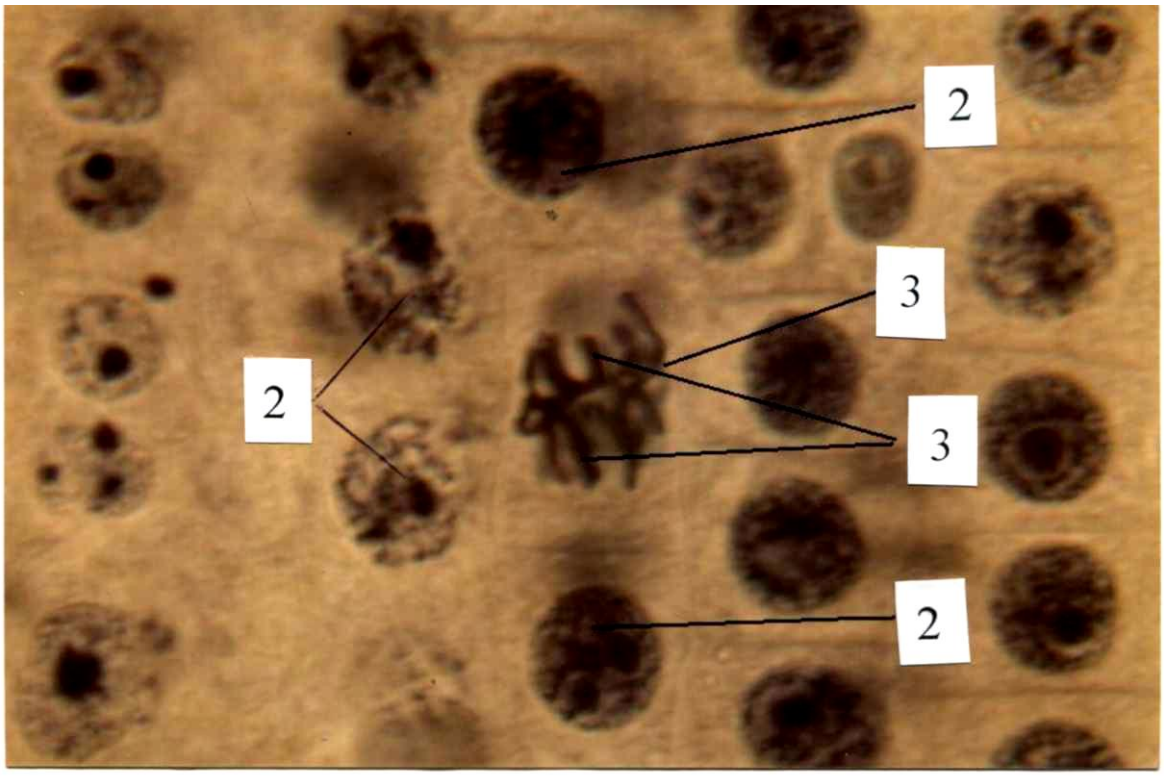


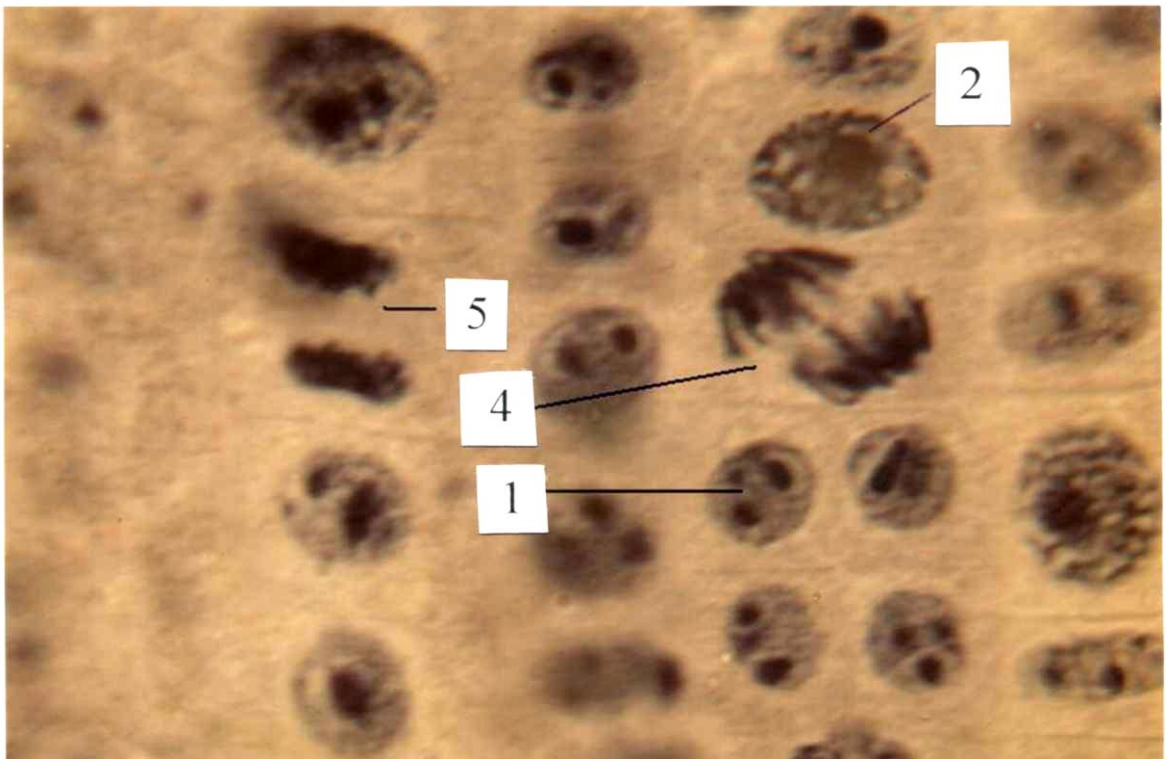
Levels of packaging of chromatin in the cell nucleus





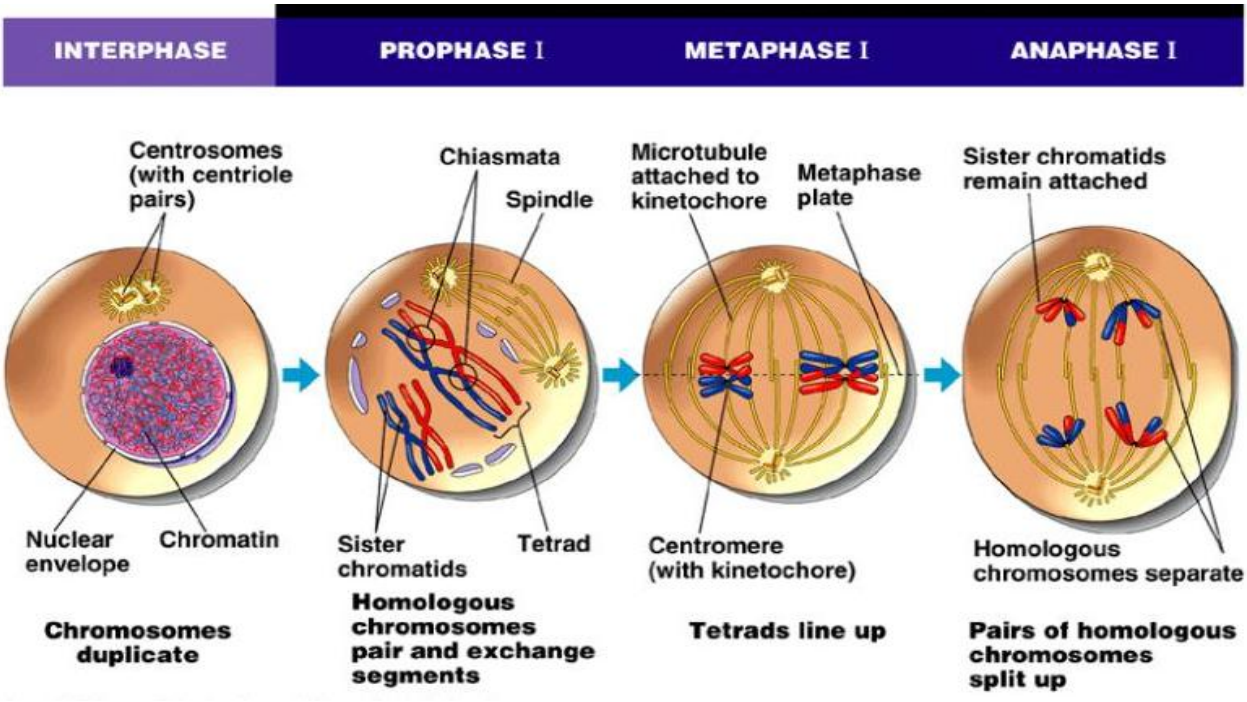
Main phases of mitosis

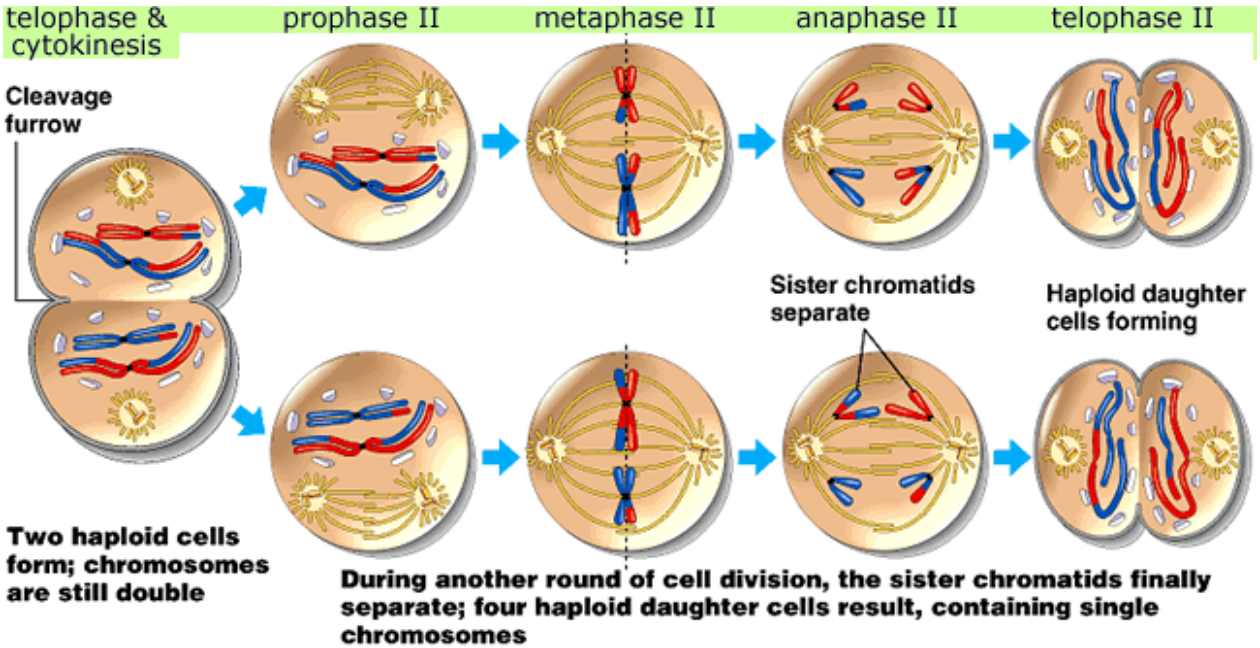




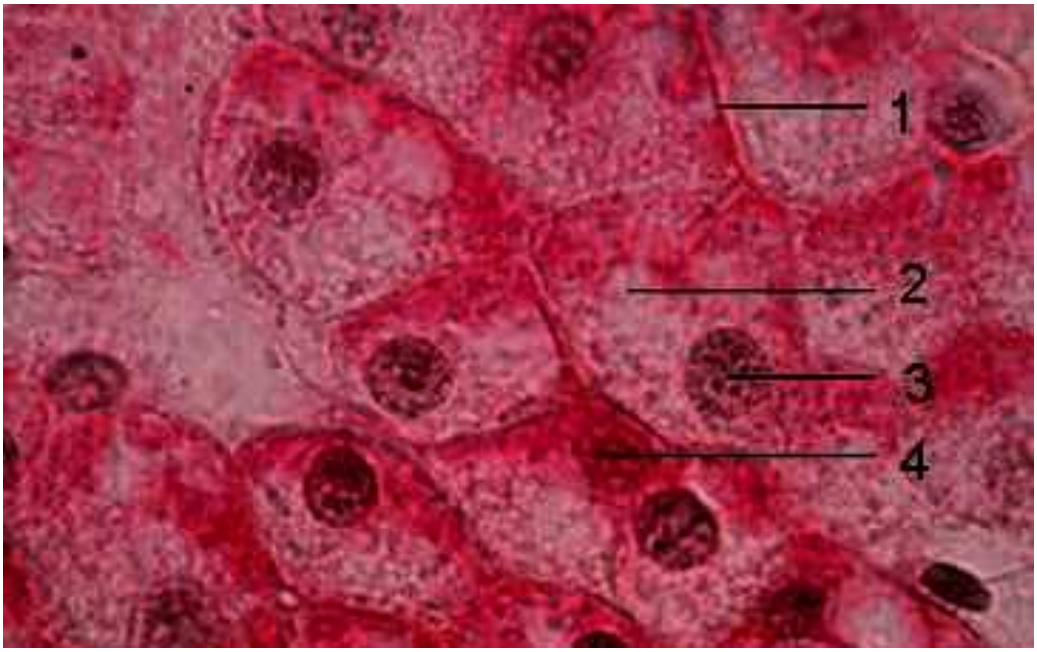
Indicate in the figure: 1) interphase; 2) prophase 3) metaphase; 4) anaphase; 5) telophase

Meiosis


INTERPHASE
PROPHASE I
METAPHASE I
ANAPHASE I
Centrosomes (with centriole pairs)
Nuclear envelope
Chromatin
Chromosomes duplicate
Chiasmata
Spindle
Sister chromatids
Tetrad
Homologous chromosomes pair and exchange segments
Microtubule attached to kinetochore
Metaphase plate
Centromere (with kinetochore)
Tetrads line up
Sister chromatids remain attached
Homologous chromosomes separate
Pairs of homologous chromosomes split up

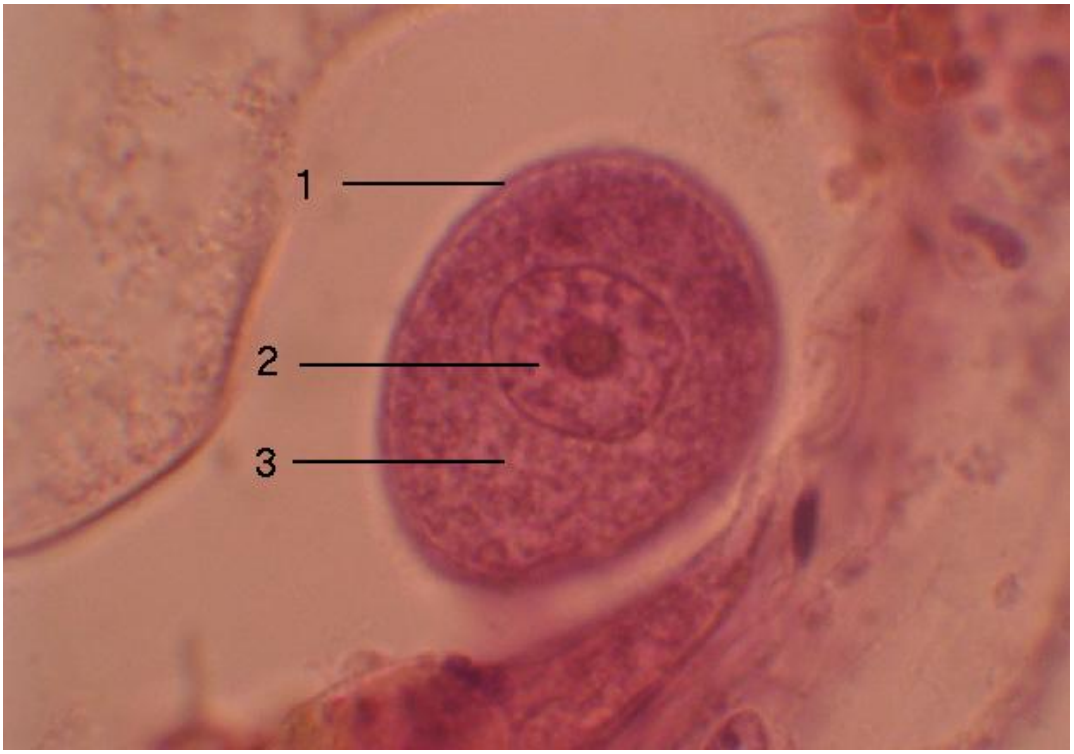

telophase & cytokinesis
prophase II
metaphase II
anaphase II
telophase II
Cleavage furrow
Sister chromatids separate
Haploid daughter cells forming
Two haploid cells form; chromosomes are still double
During another round of cell division, the sister chromatids finally separate; four haploid daughter cells result, containing single chromosomes

Inclusion of glycogen in liver cells



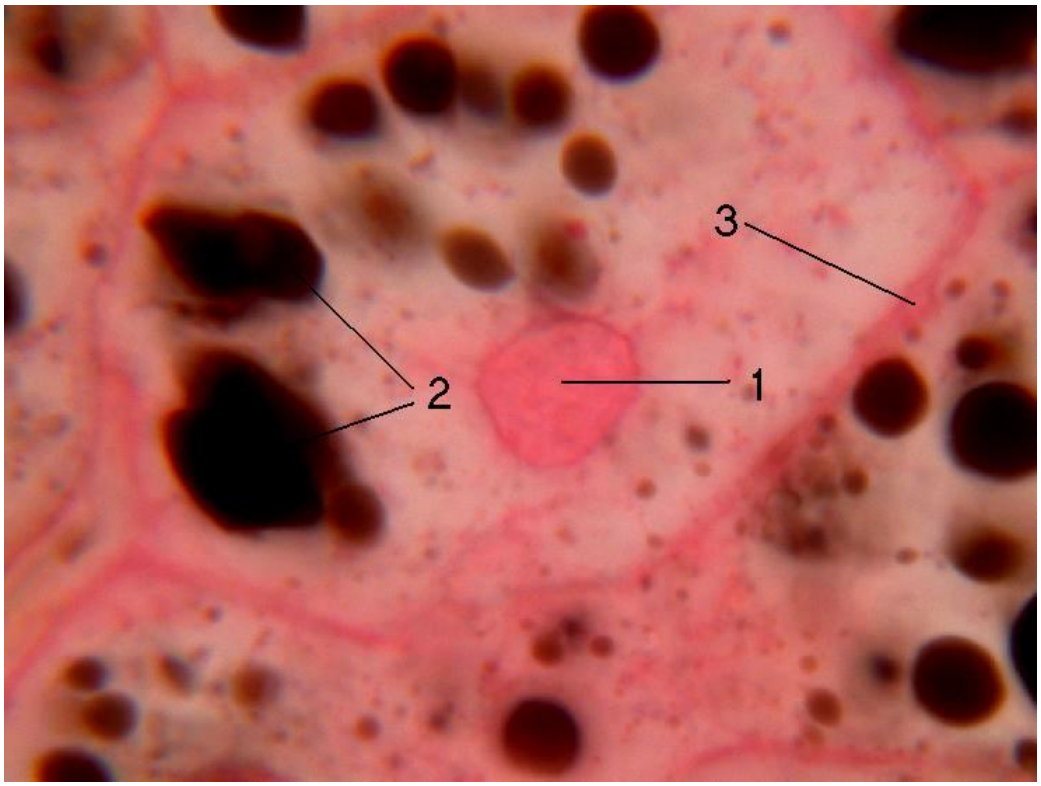
Indicate in the figure: 1) the cell boundaries; 2) the cytoplasm; 3) the nucleus; 4) inclusion of glycogen

The inclusion of protein in the egg of a shellfish



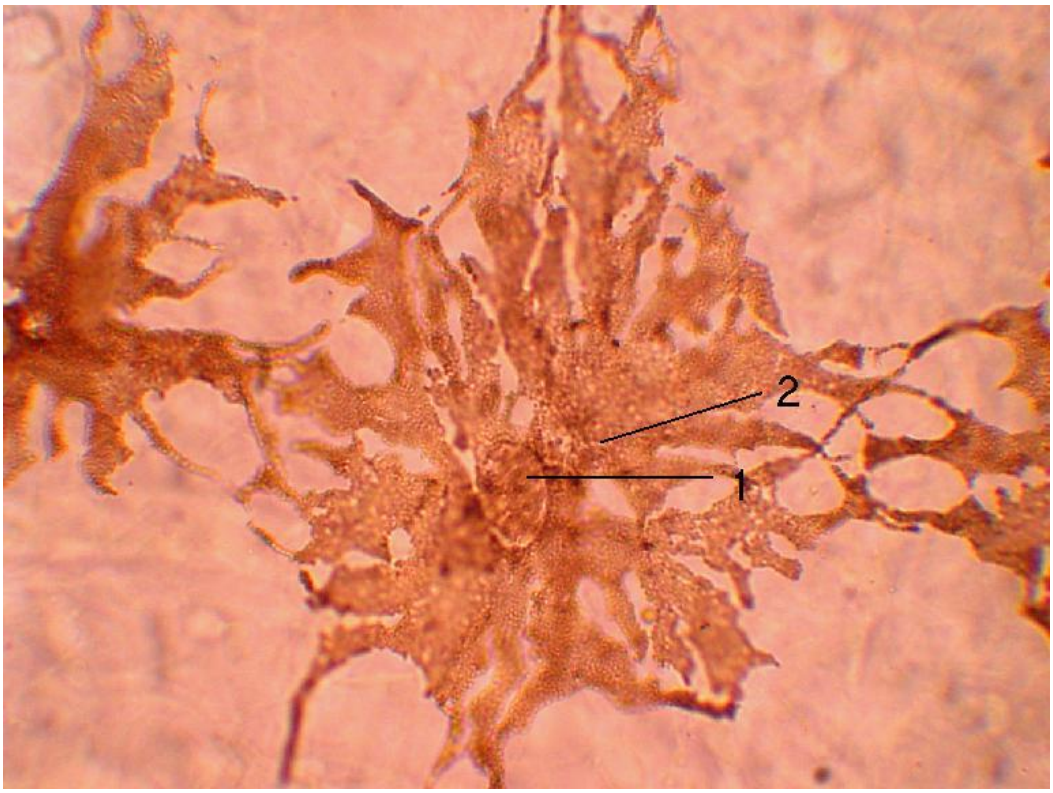
Indicate in the figure: 1) the cell boundaries; 2) the nucleus; 3) inclusion of protein

The inclusion of fat in liver cells



Indicate in the figure: 1) the cell boundaries; 2) the nucleus; 3) inclusion of fat

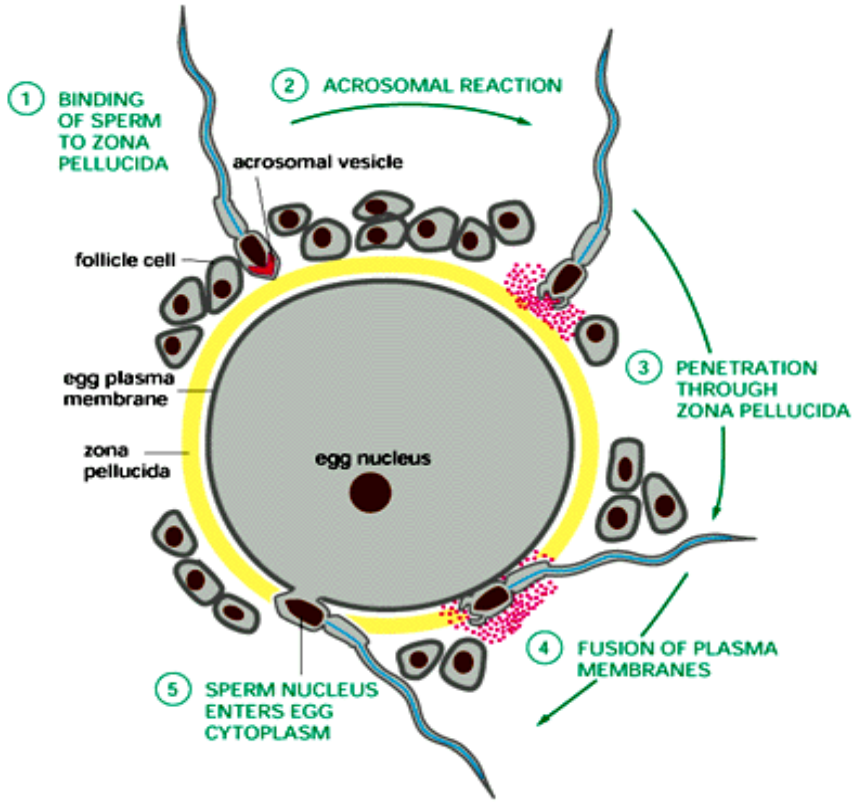
Inclusions of pigment in amphibian skin cells

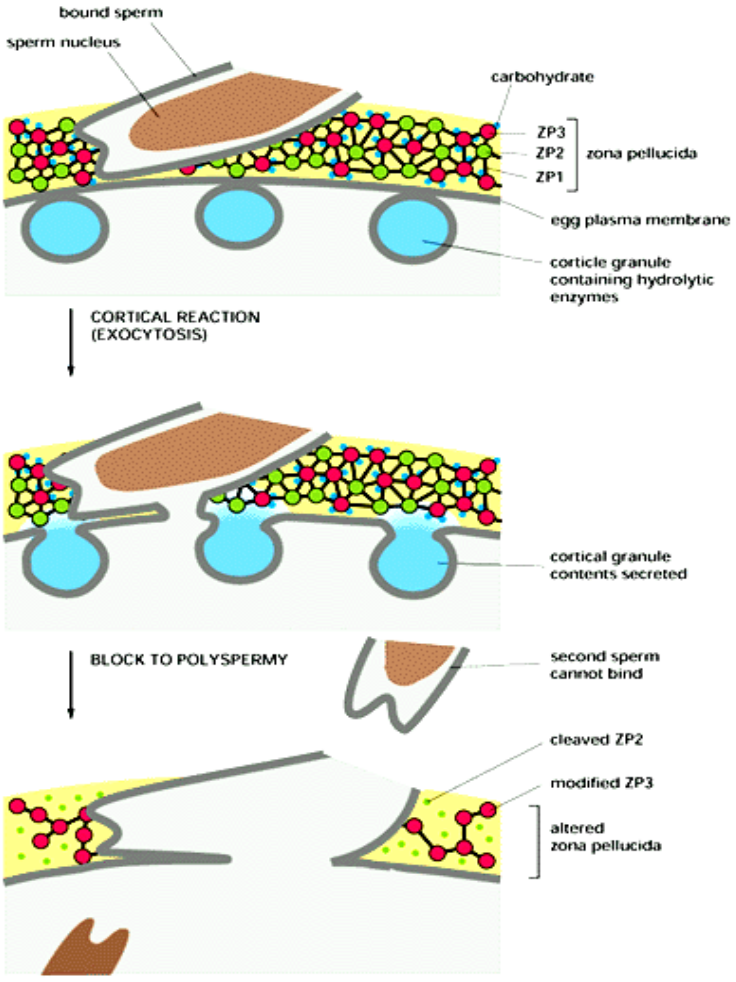


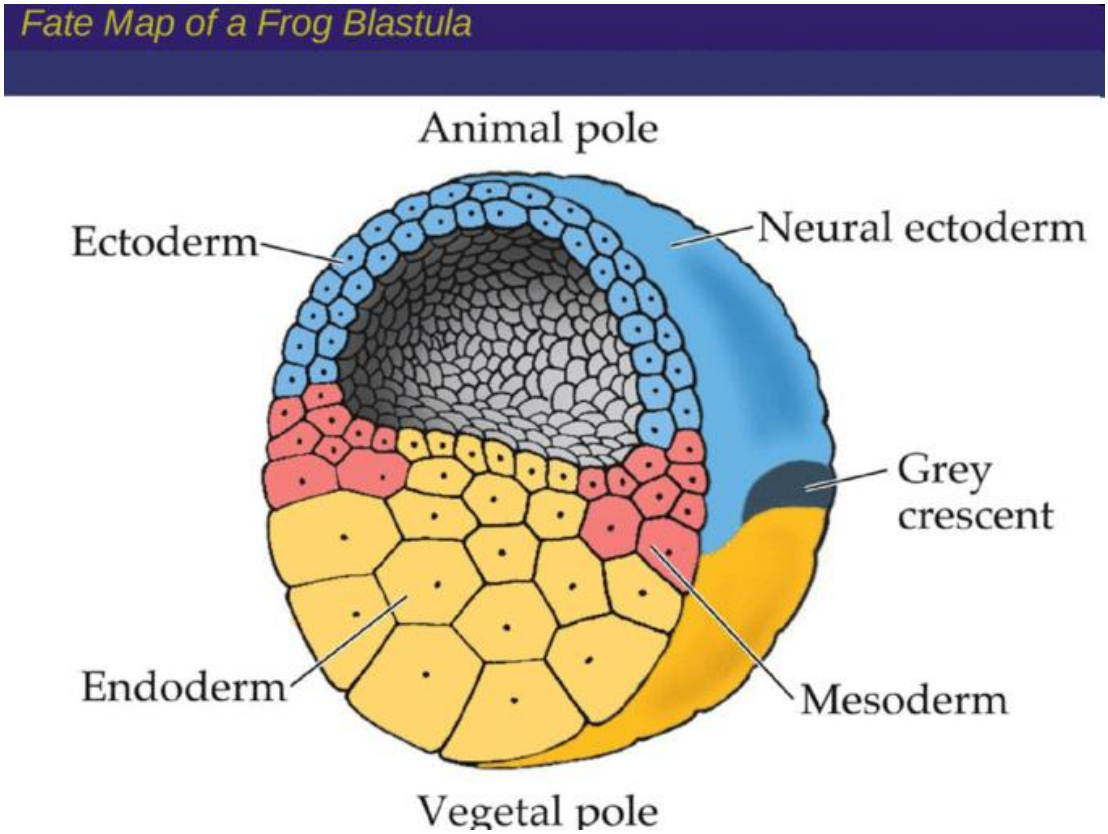
Indicate in the figure: 1) the nucleus; 2) inclusion of pigment

ONTOGENESIS

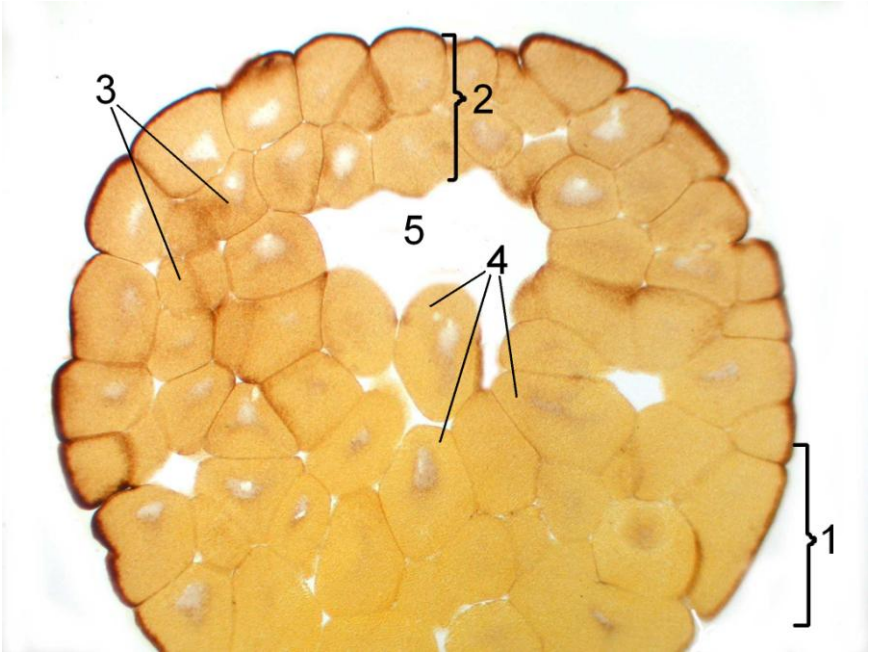
Fertiliz ation





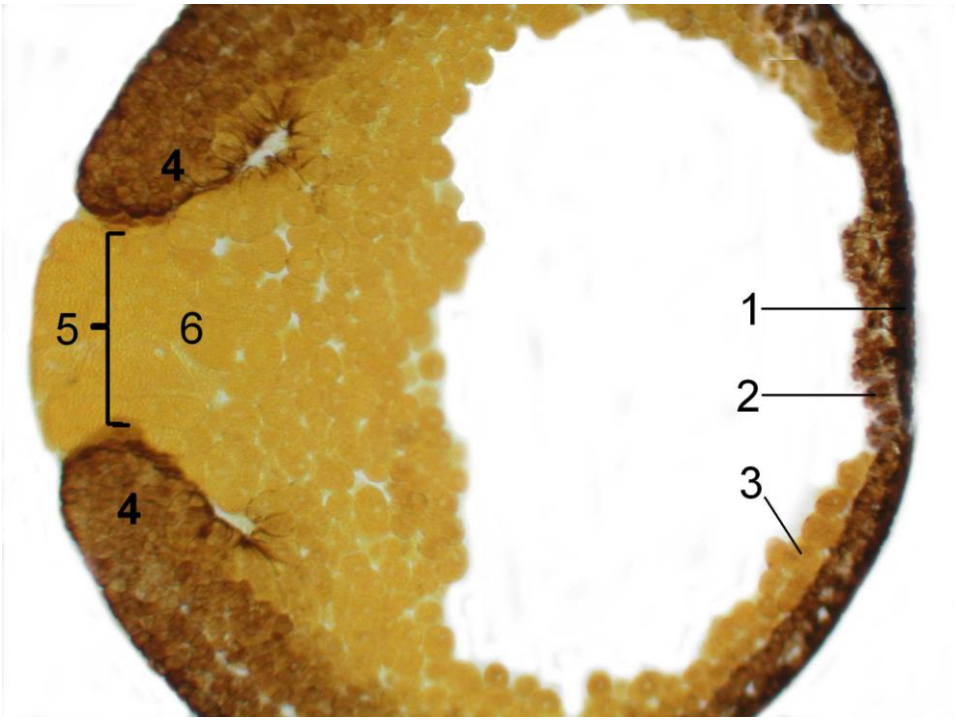


Blastula of the frog



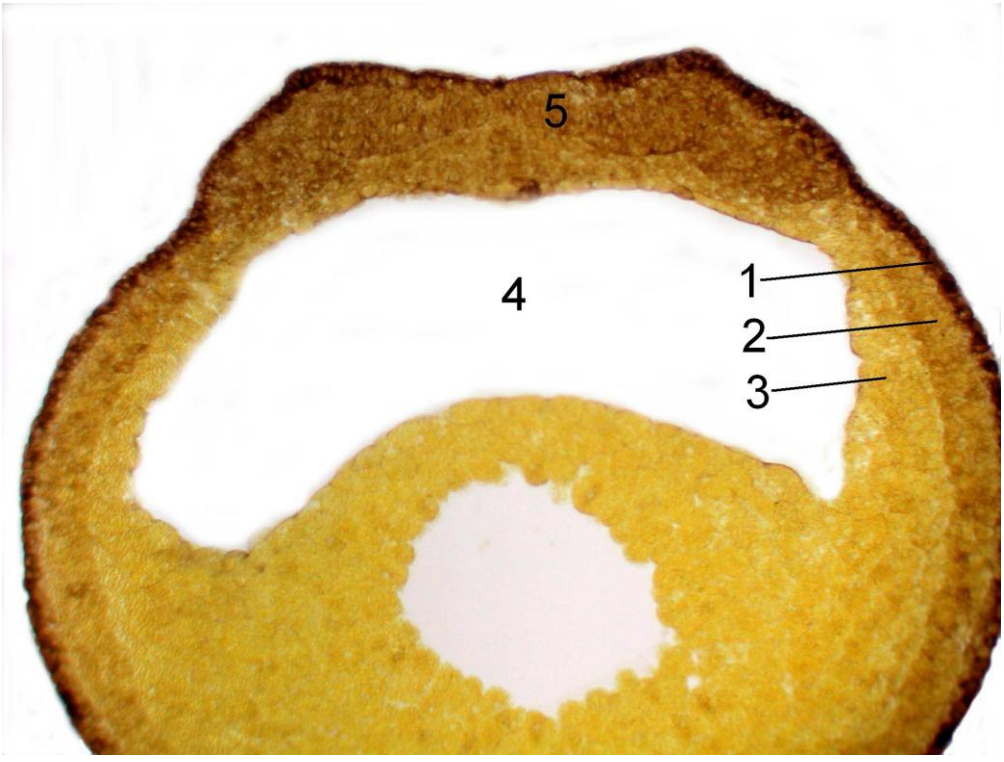
Indicate in the figure: 1) vegetative pole; 2) animal pole; 3) micrometers; 4)macromeres 5) blastocoel

Gastrula of frog



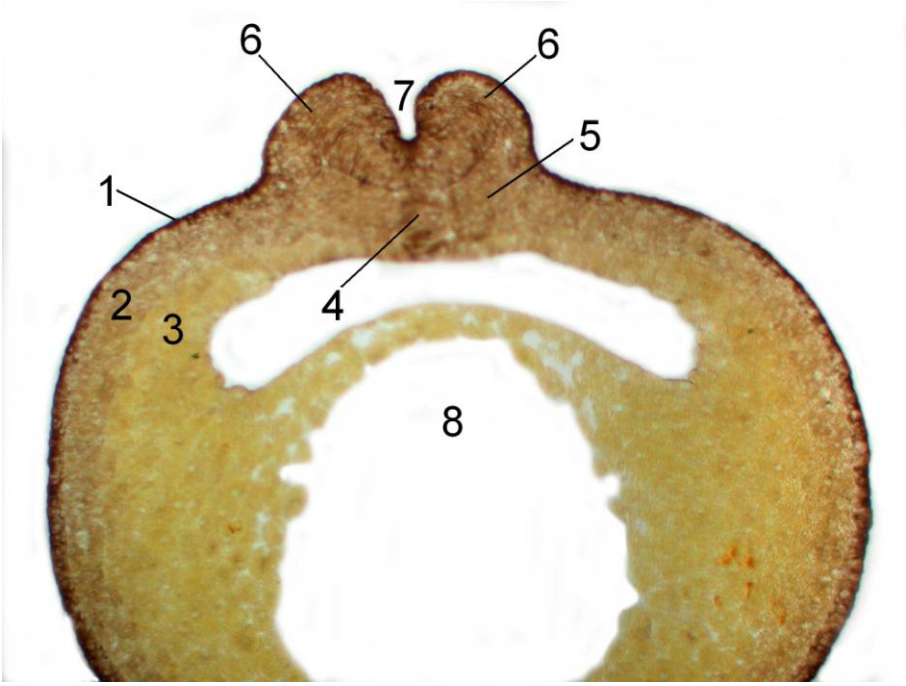
Indicate in the figure: 1) animal pole; 2) ectoderm; 3) endoderm; 4) lips of the blastopore; 5) blastopore; 6) yolk cork

Early neurula of frog



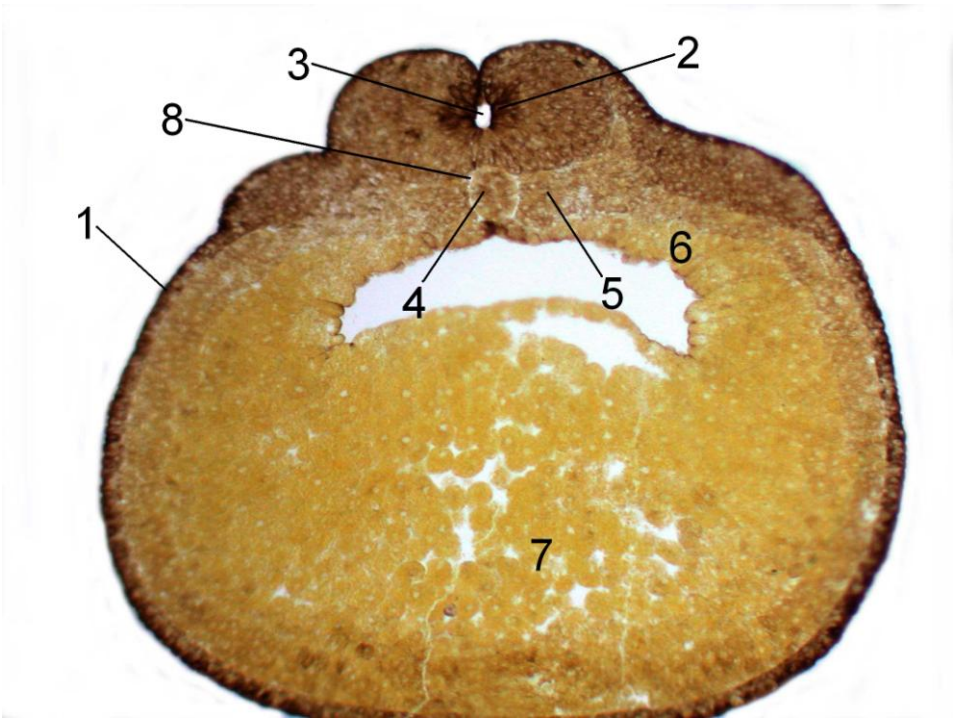
Indicate in the figure: 1) ectoderm; 2) mesoderm; 3) endoderm; 4) cavity ; 5) neural plate

Average neurula of frog



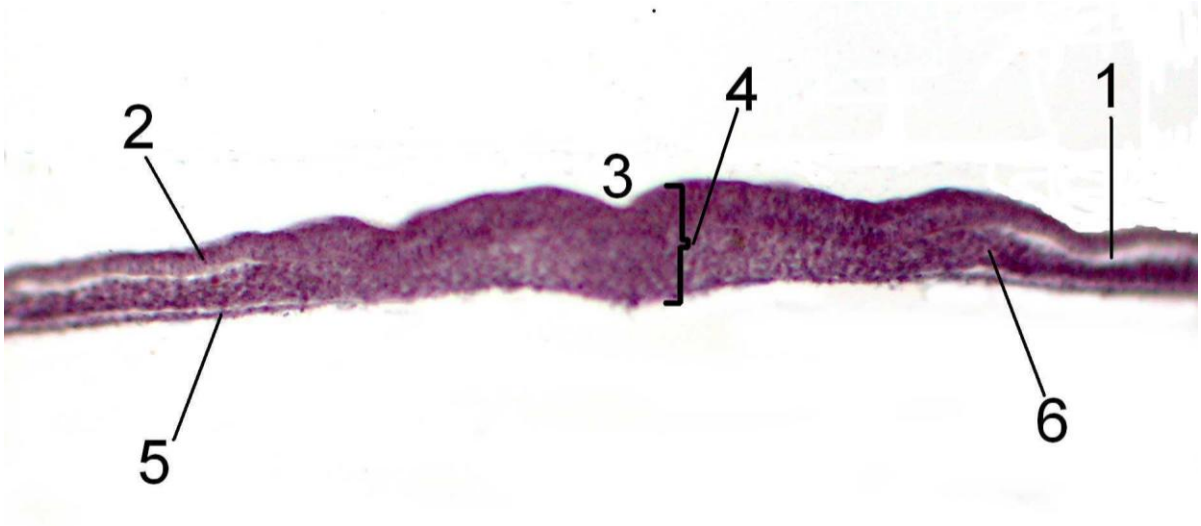
Indicate in the figure: 1) ectoderm; 2) mesoderm; 3) endoderm; 4) notochord; 5) somites; 6) neural fold; 7) neural groove; 8) primary gut

Late neurala of frog



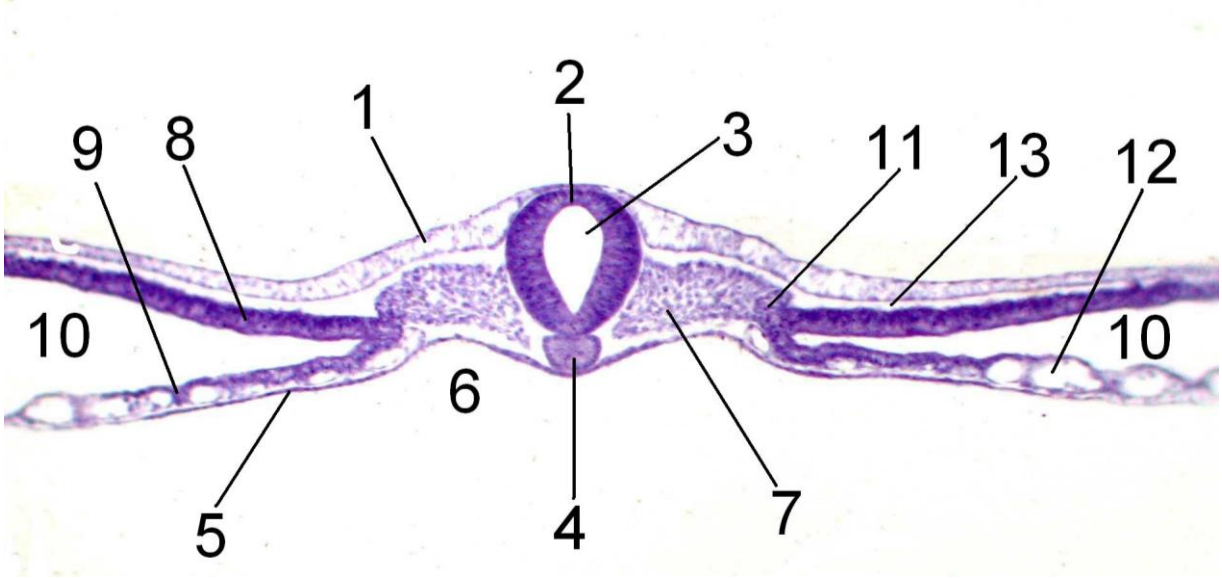
Indicate in the figure: 1) ectoderm; 2) neural tube; 3) neurocele; 4) notochord; 5) somites; 6) endoderm; 7) primary gut

Primary stripe of chicken embryo



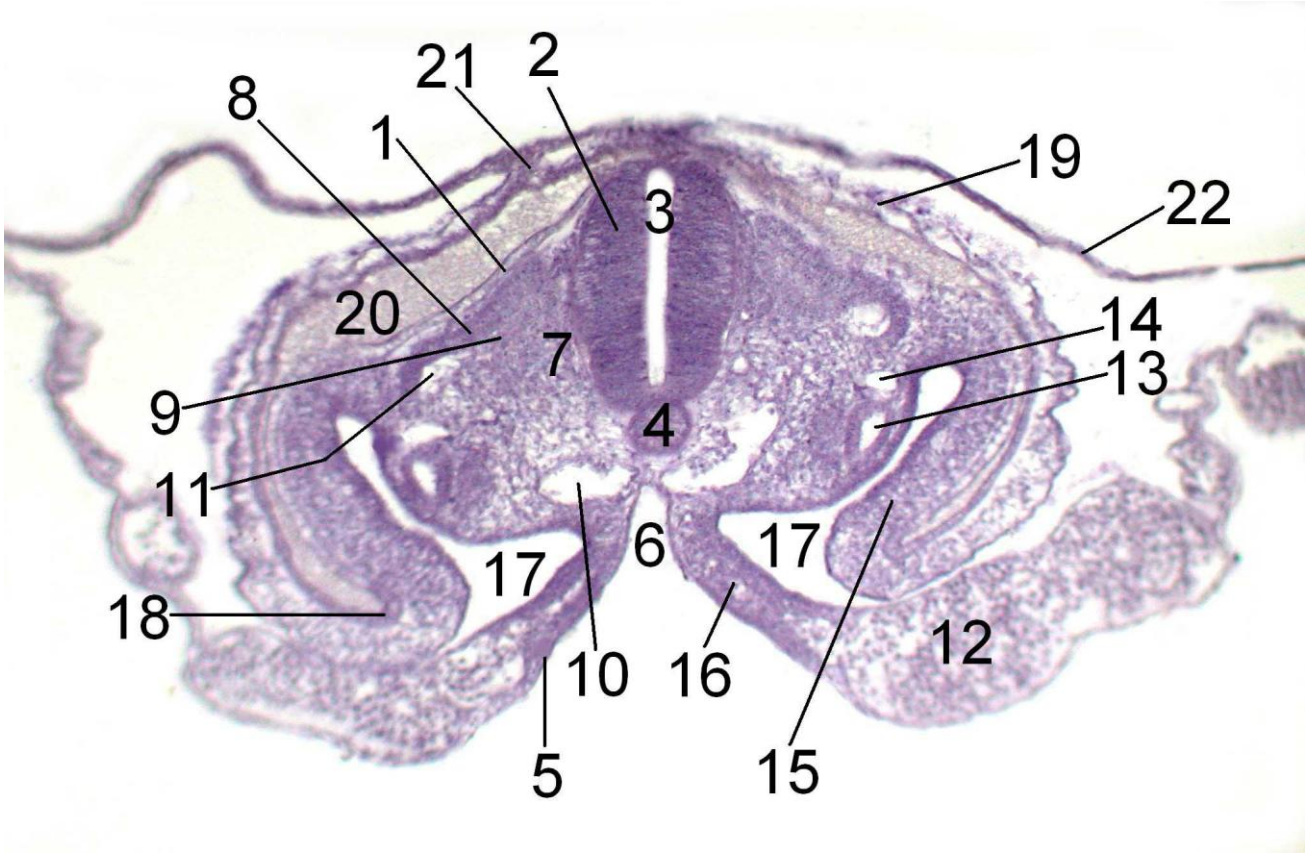
Indicate in the figure: 1) cavity; 2) ectoderm; 3) neural groove; 4) primary stripe; 5) endoderm; 6) mesoderm (somites)

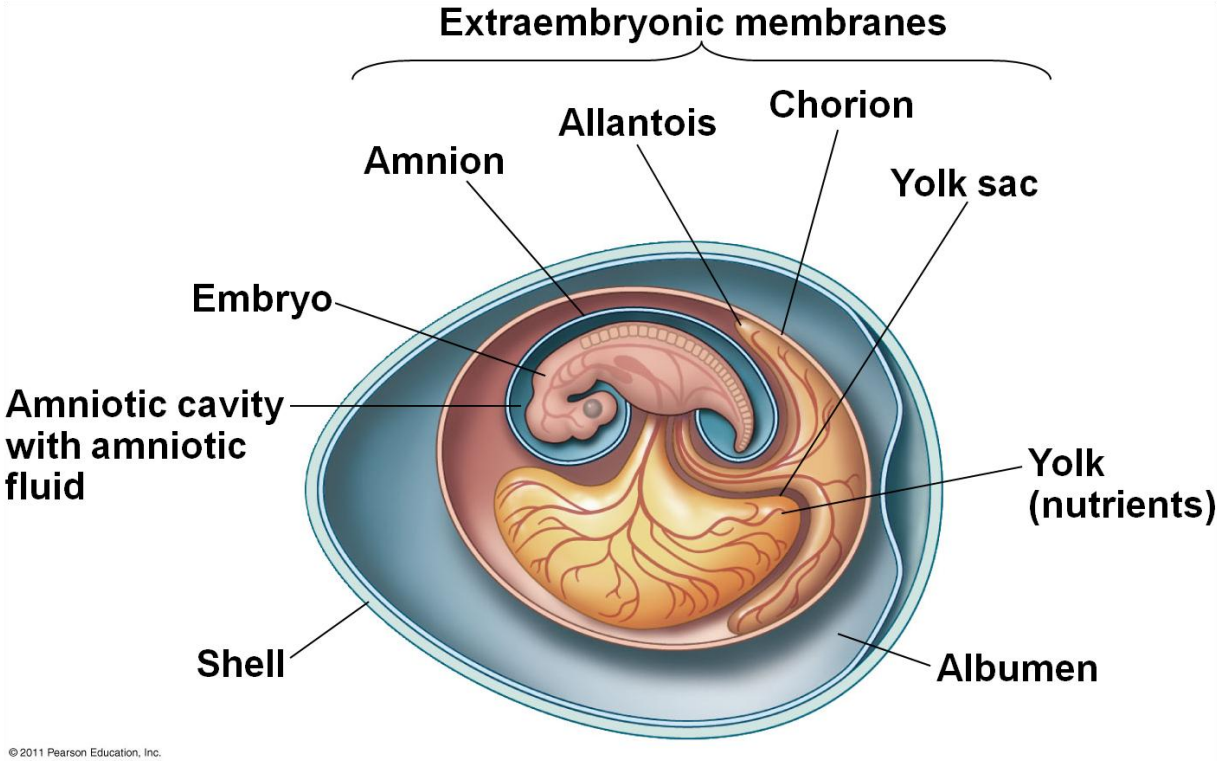
Chicken embryo at the stage of formation of primary organs



Indicate in the figure: 1) ectoderm; 2) neural tube; 3) neurocele; 4) notochord; 5) endoderm; 6) place of yolk; 7) somites; 8) somatopleurea - parietal layer of splanchnotome; 9) splanchnopleura - visceral layer of splanchnotom; 10) coelom; 11) intermediate mesoderm; 12) blood vessels

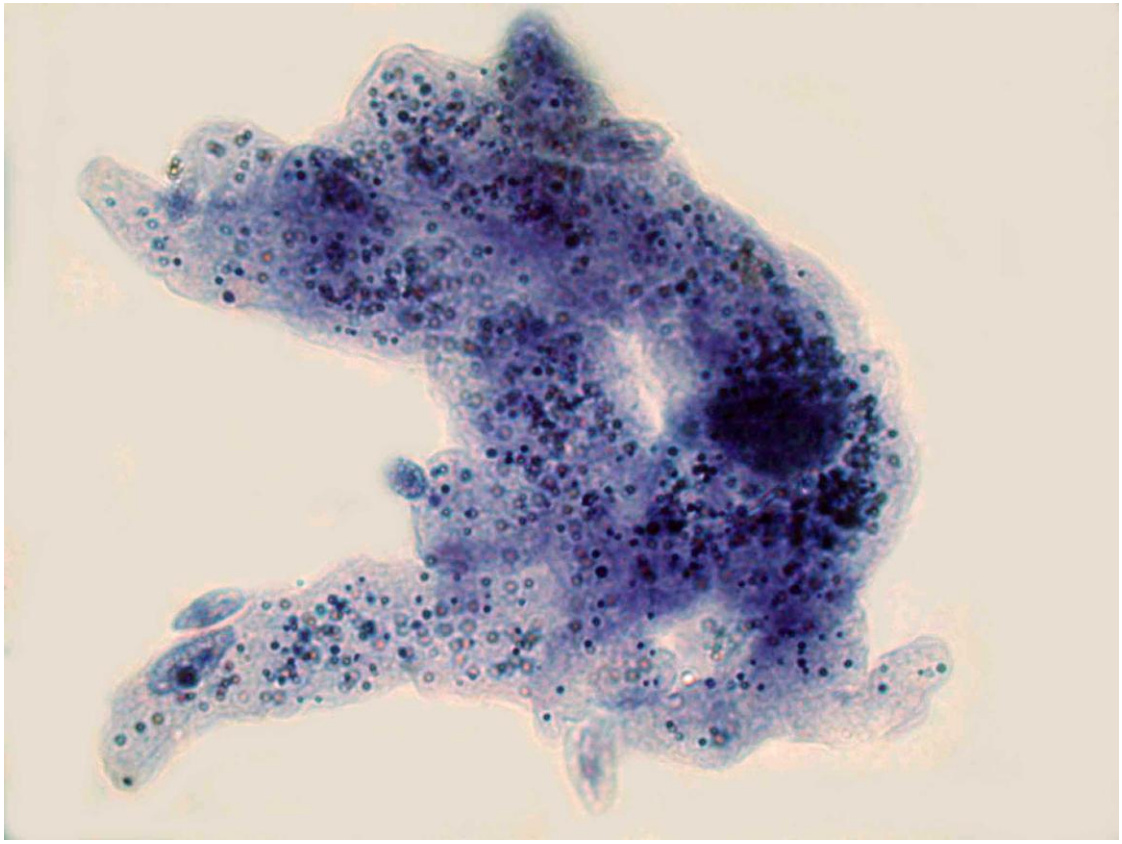
Chicken embryo at the stage of formation of extraembryonic membranes

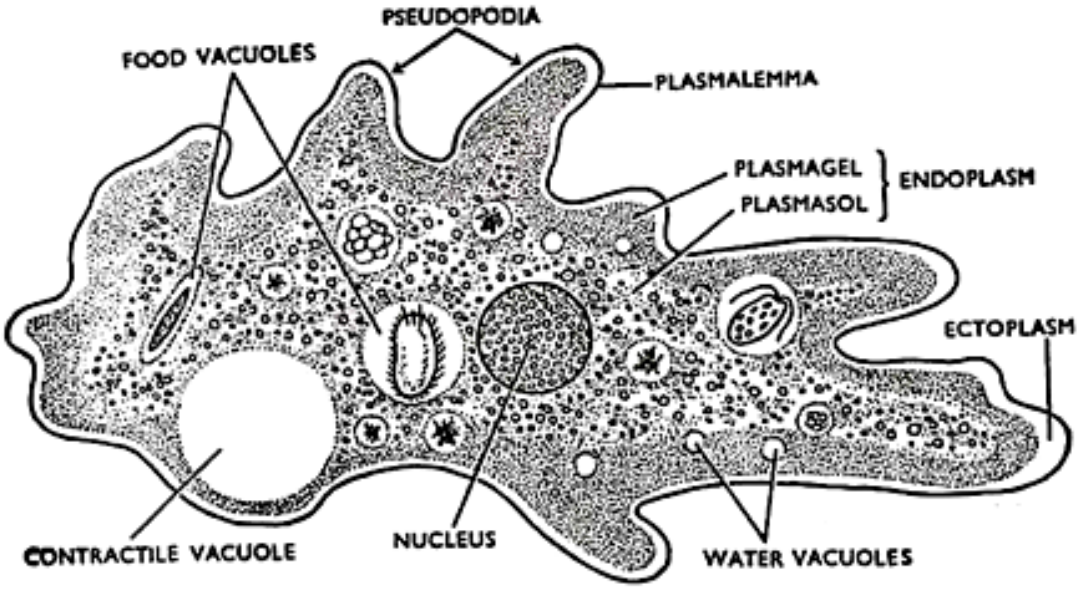




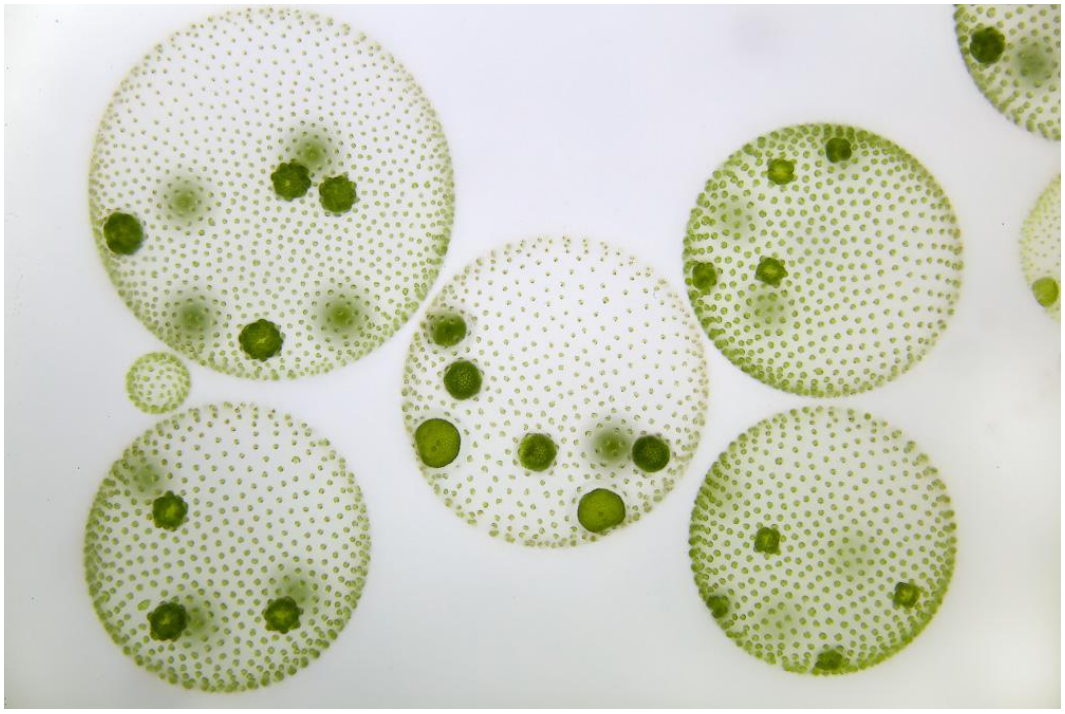
Parasitology

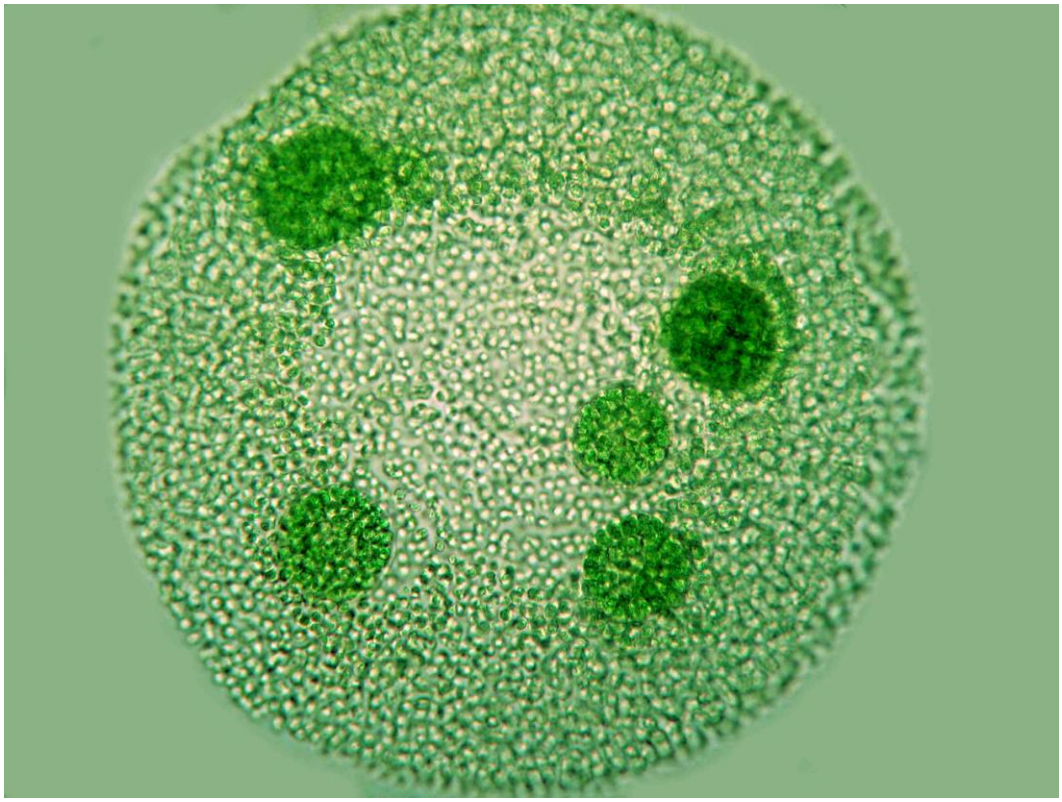
Amoeba proteus



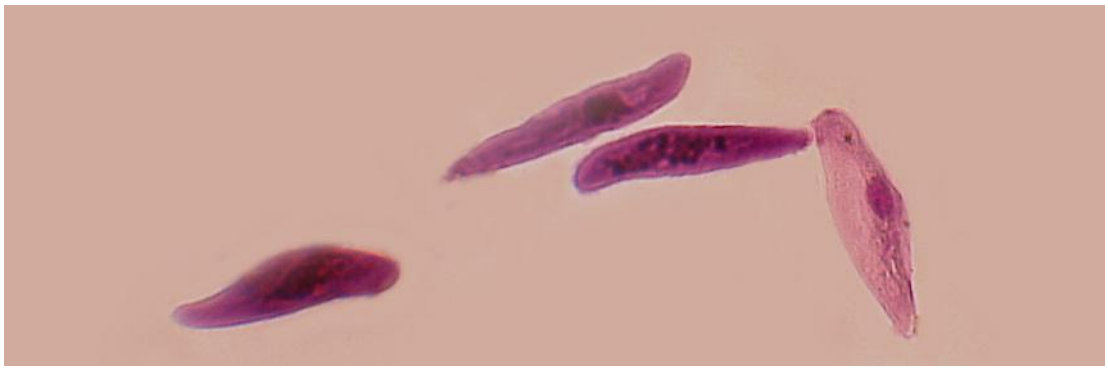


Volvox

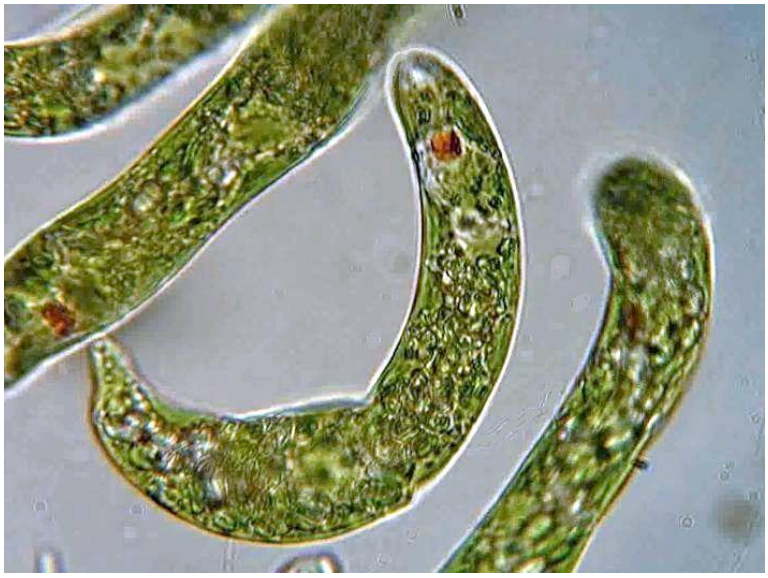


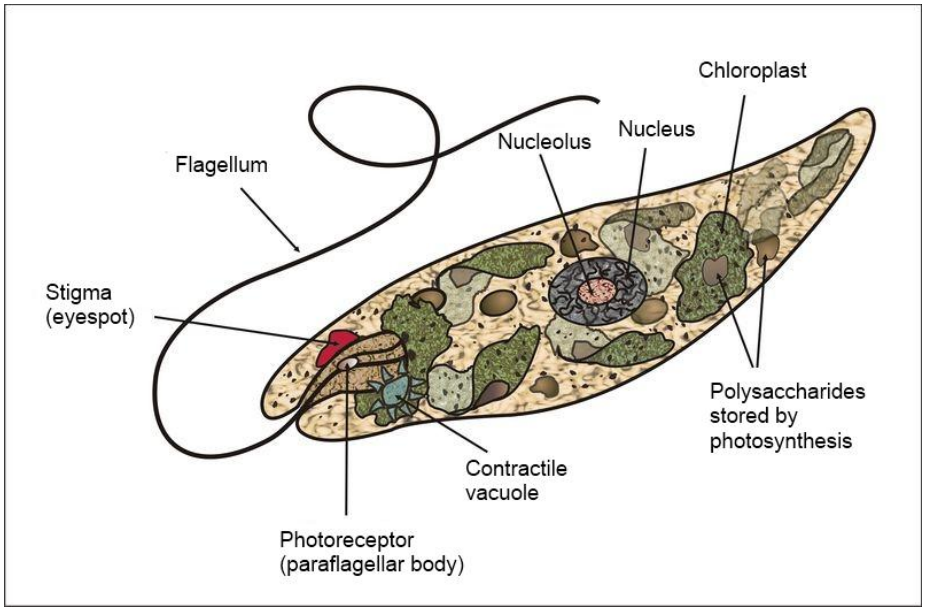


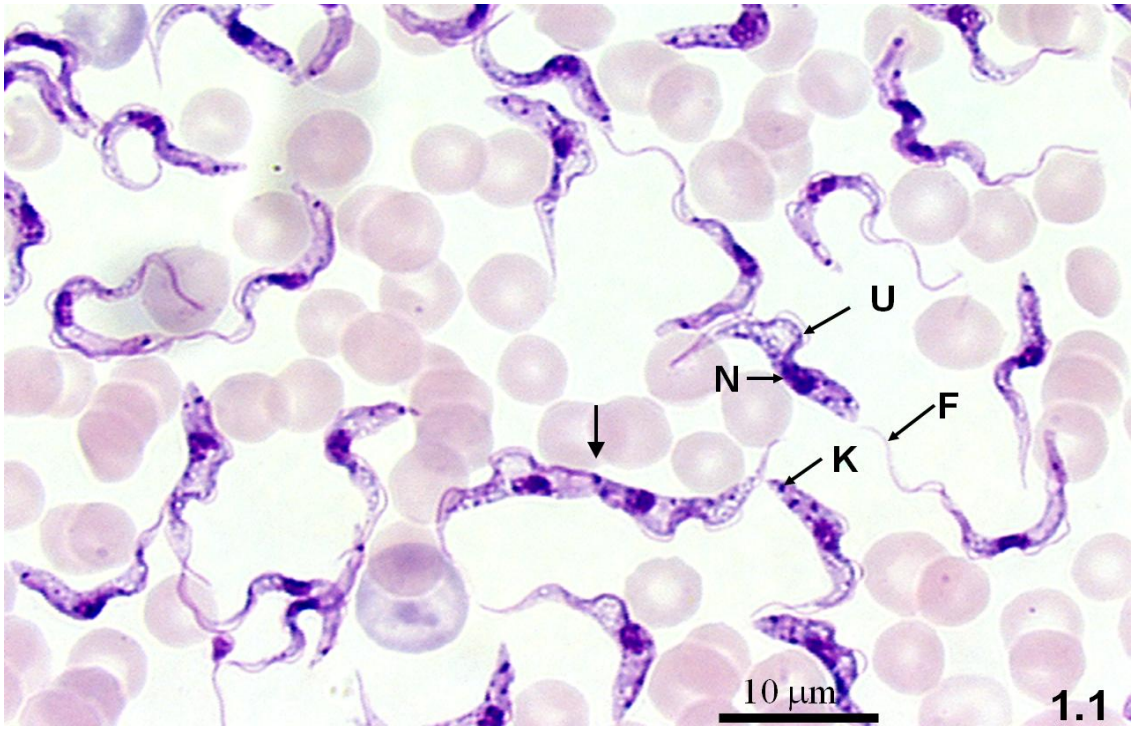
Paramecium Caudatum



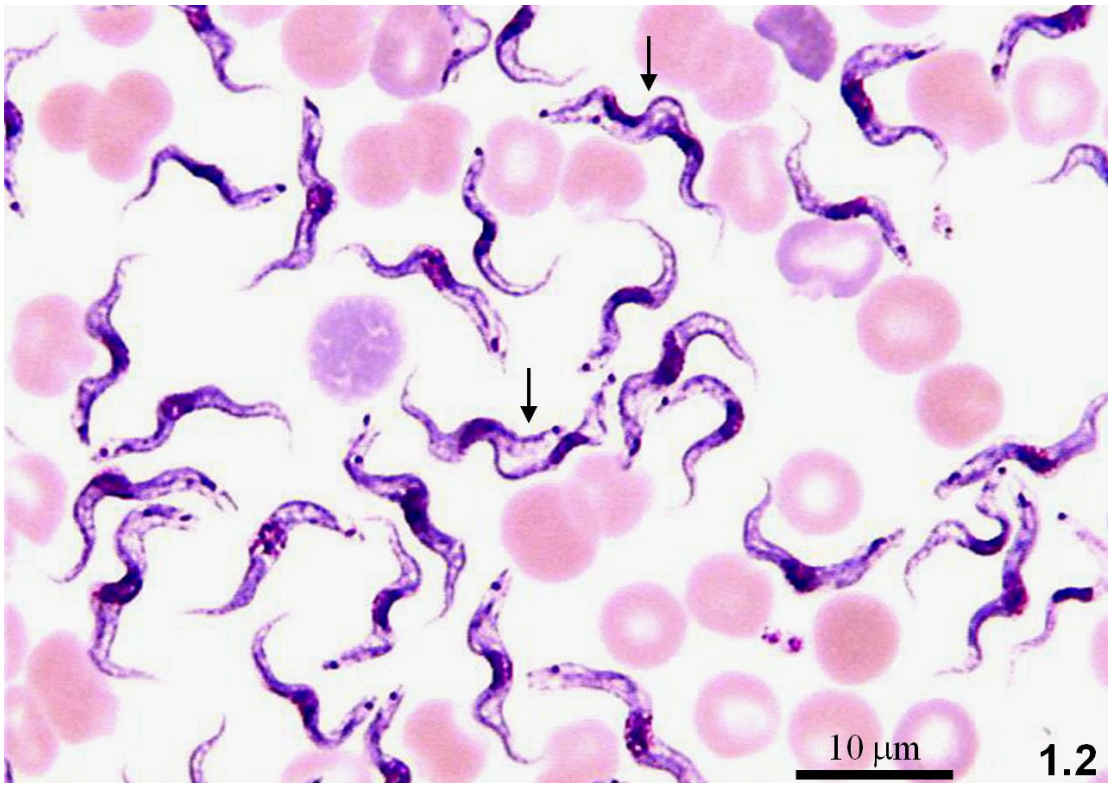
Euglena



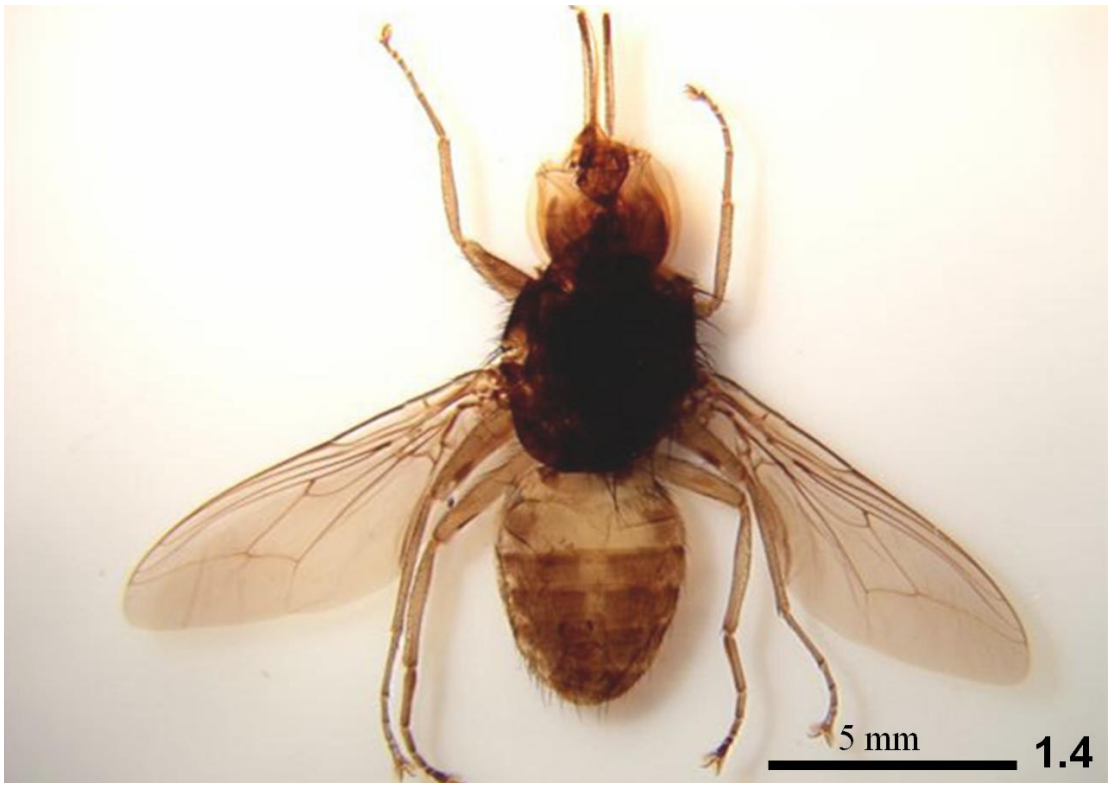




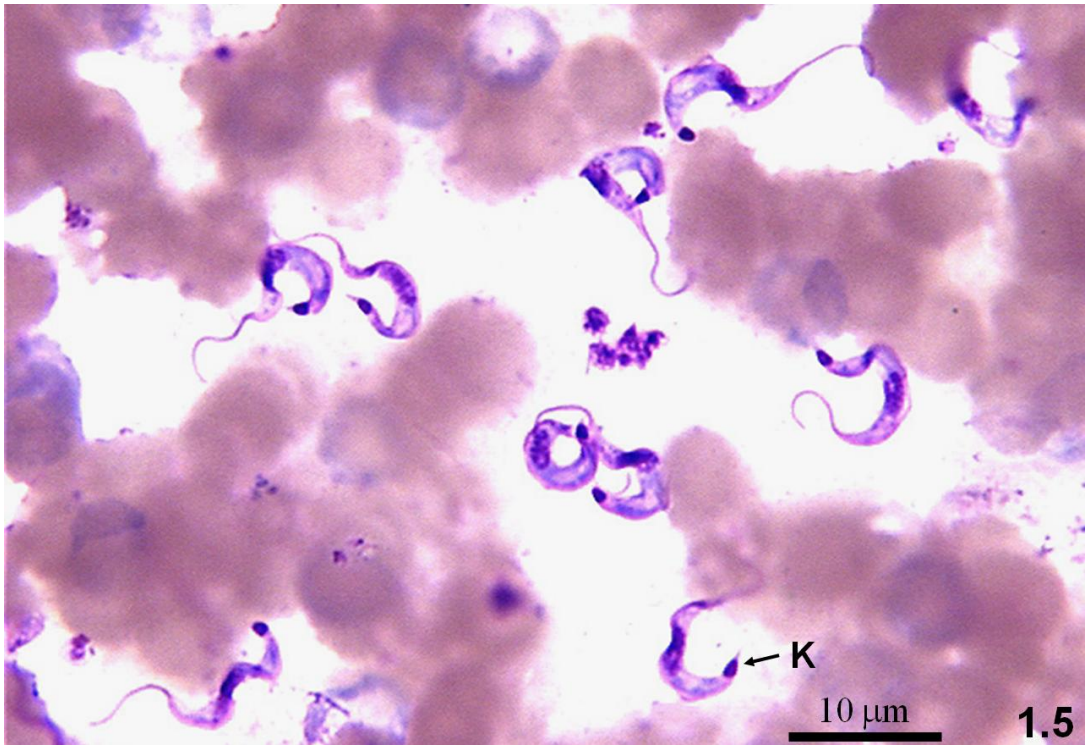
1.1 Trypomastigotes of *Trypanosoma brucei rhodesiense*



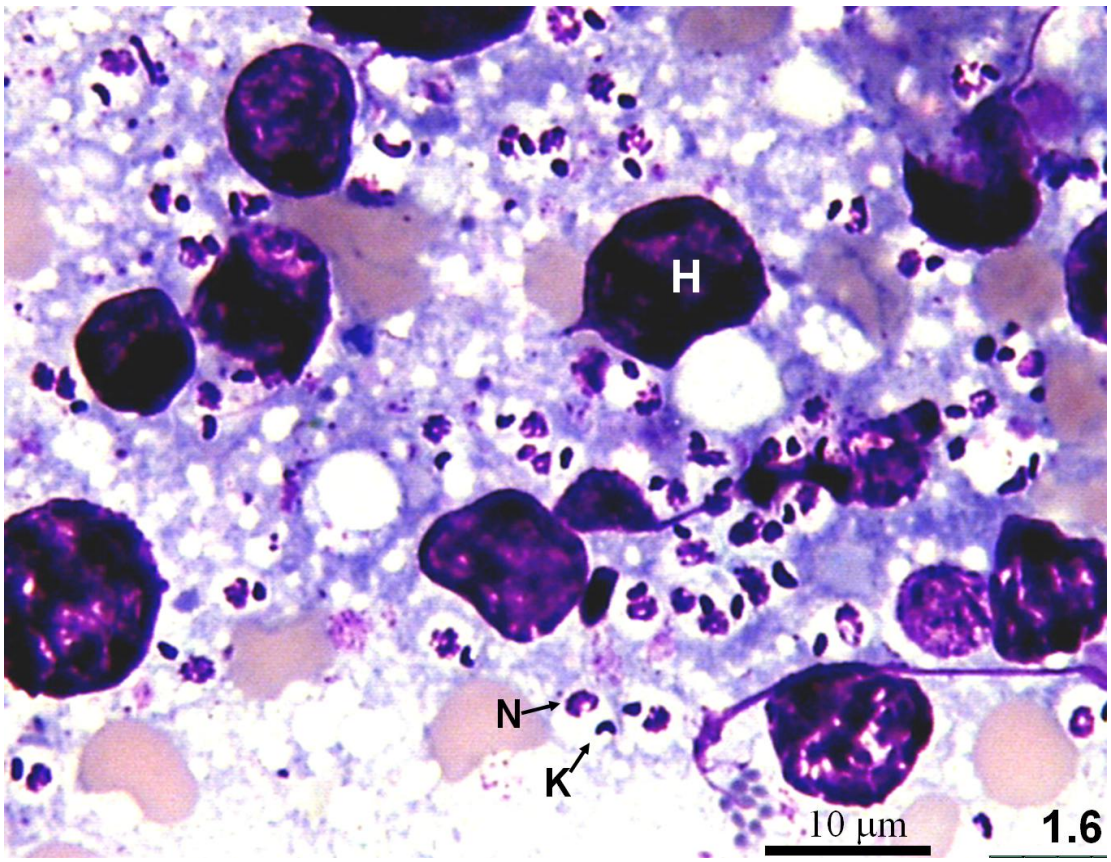
1.2 Trypomastigotes of *Trypanosoma brucei gambiense*,



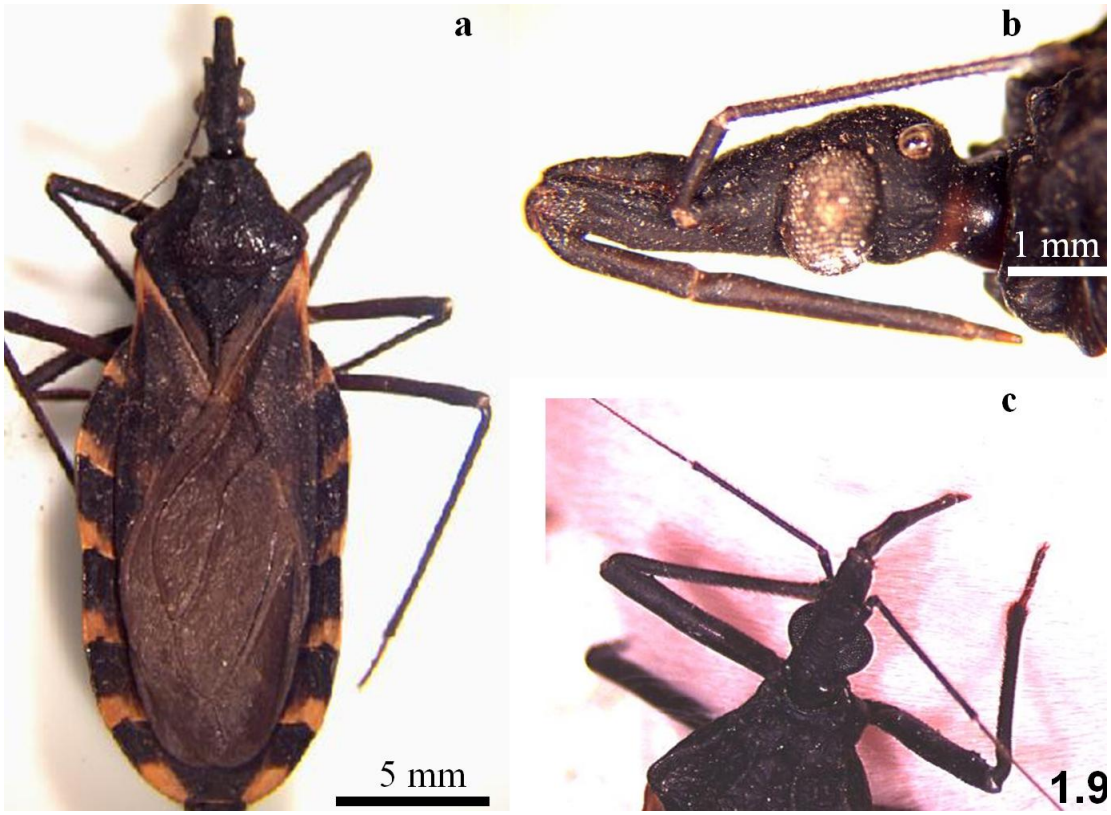
1.4 *Glossina* sp. (tsetse fly), the vector of African trypanosomiasis



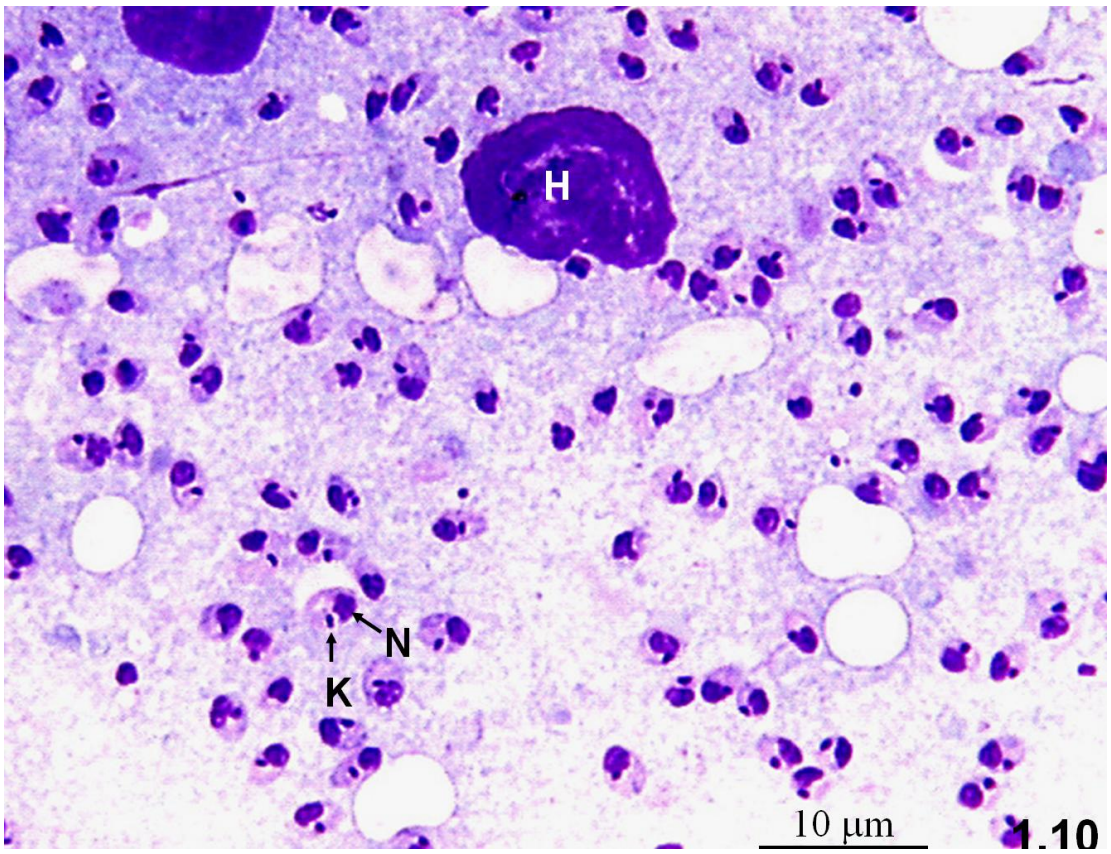
1.5 Trypomastigotes of *Trypanosoma cruzi*, peripheral blood smear.



1.6 Amastigotes of *Trypanosoma cruzi*, spleen smear.

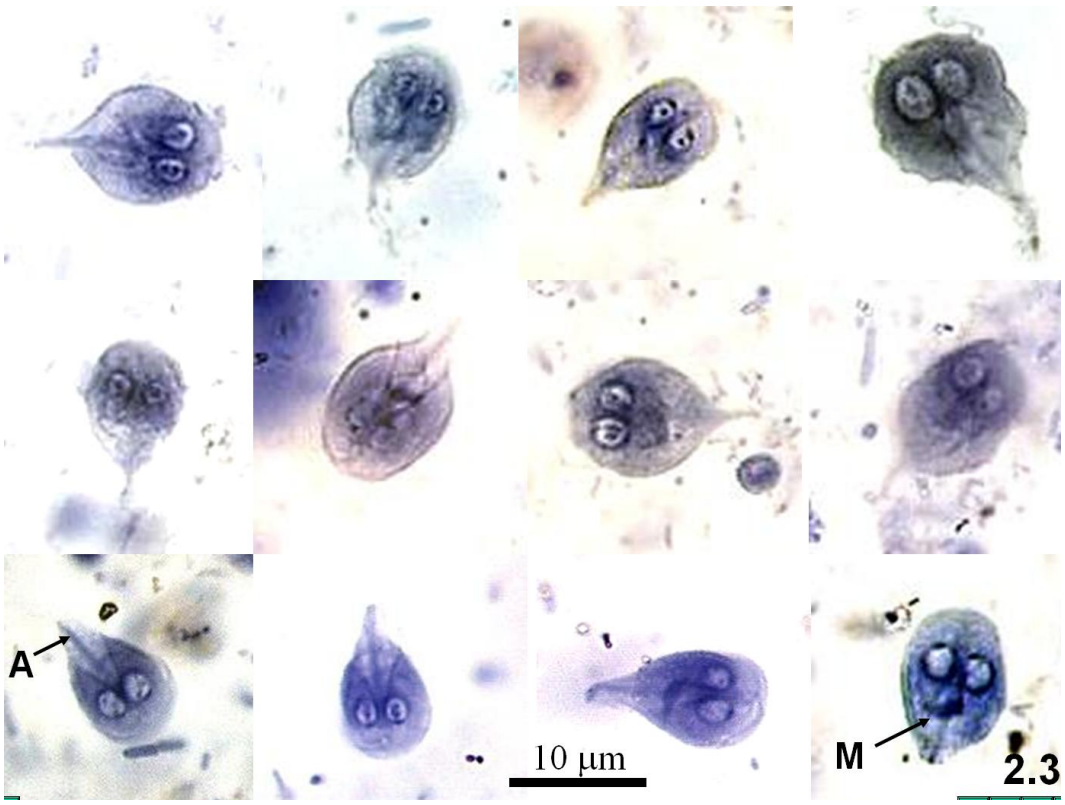


1.9 Kissing bug, *Triatoma gerstaeckeri*

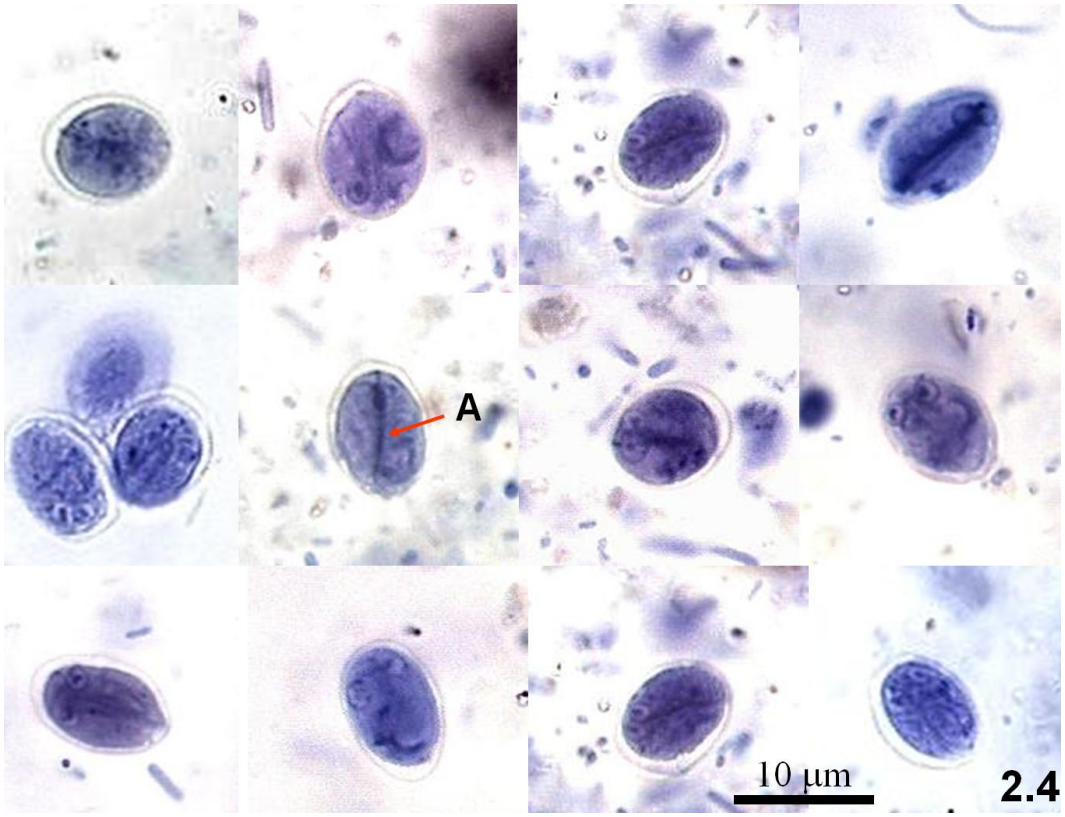


1.10 Amastigotes of *Leishmania donovani,* liver smear.

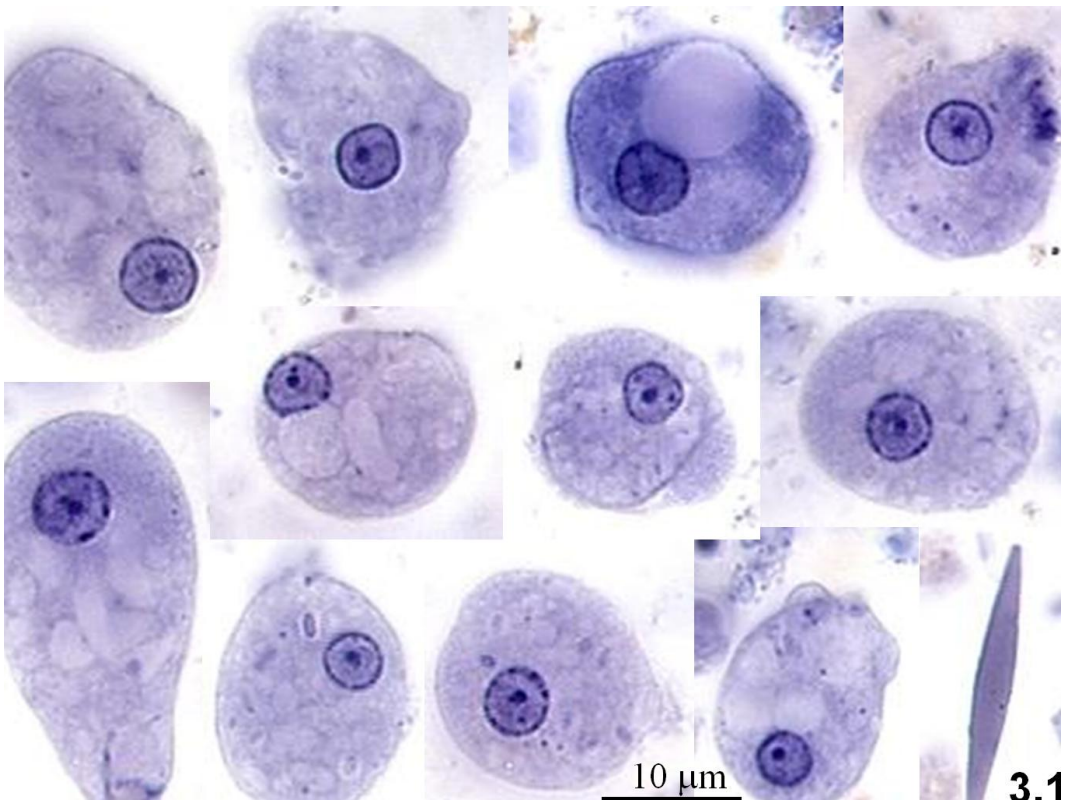
1.13 *Lutzomyia diabolica* (sand fly)



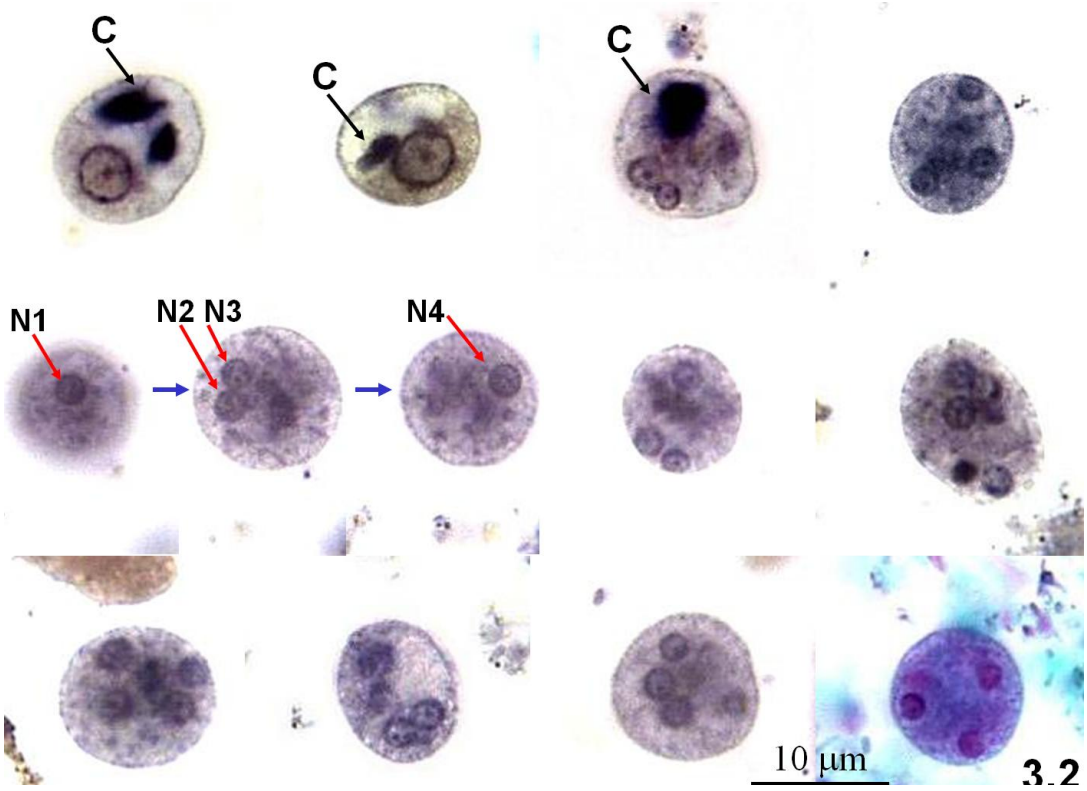
2.3 Trophozoites of *Giardia lamblia,* stool smear.



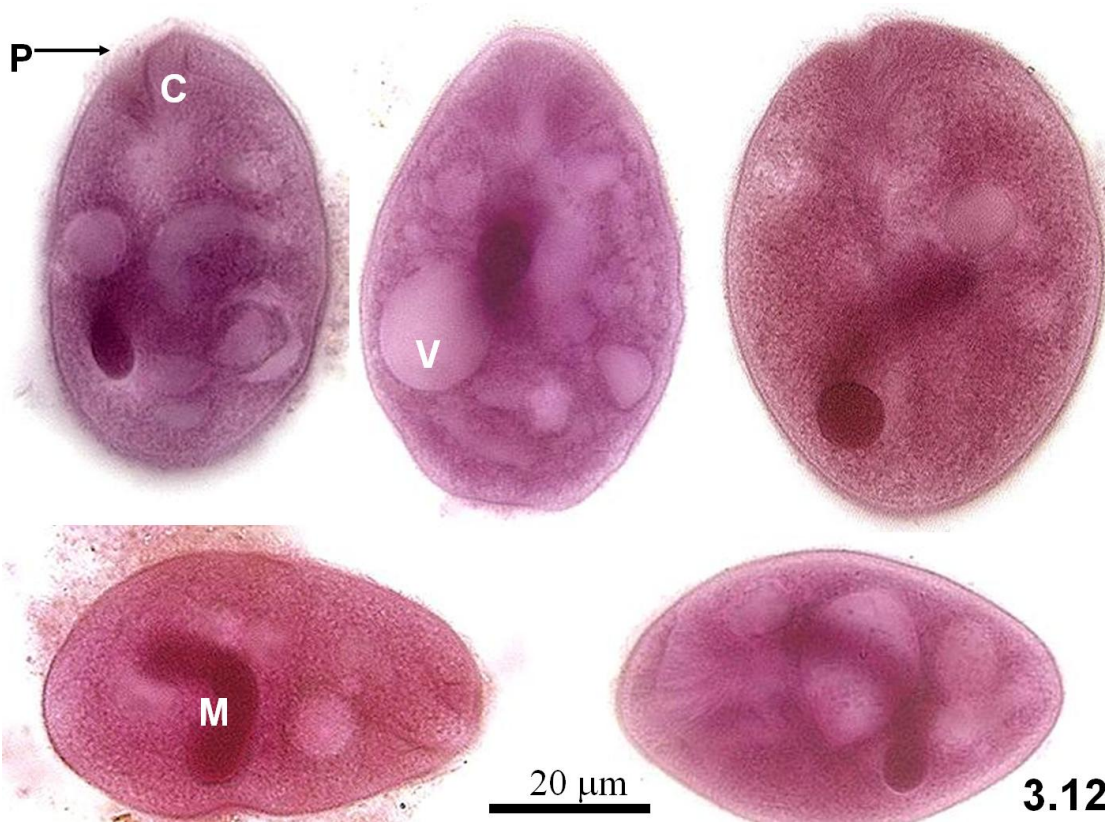
2.4 Cysts of *Giardia lamblia,* stool smear.



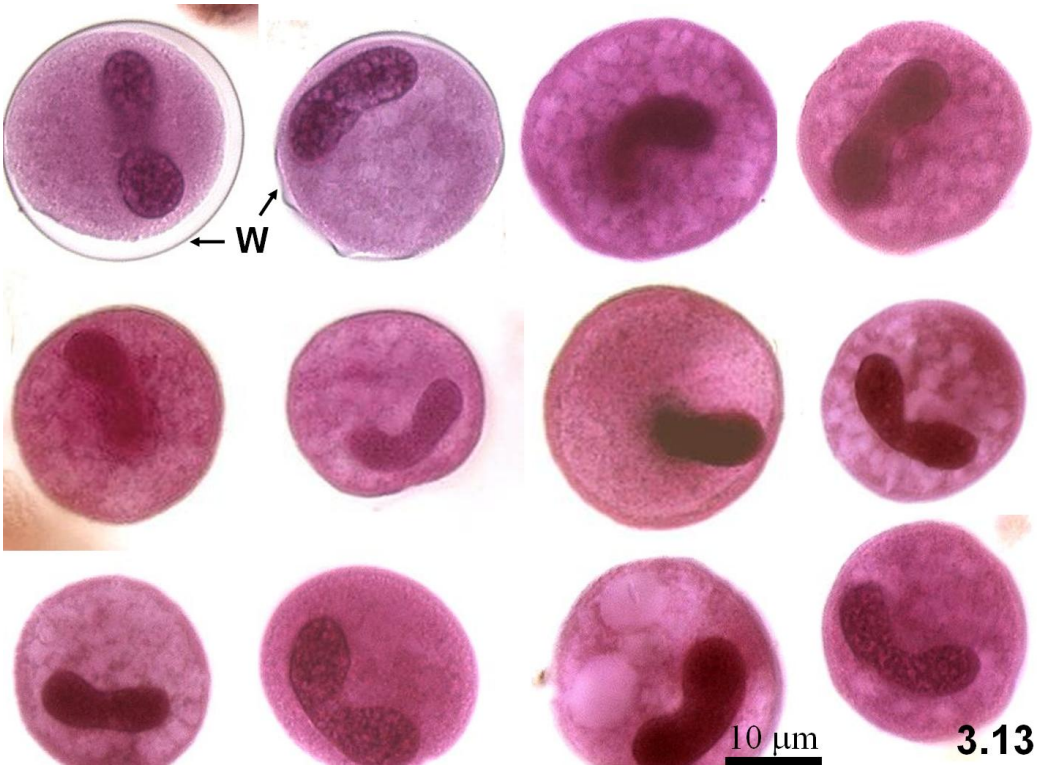
3.1 Trophozoites of *Entamoeba histolytica,* stool smear.



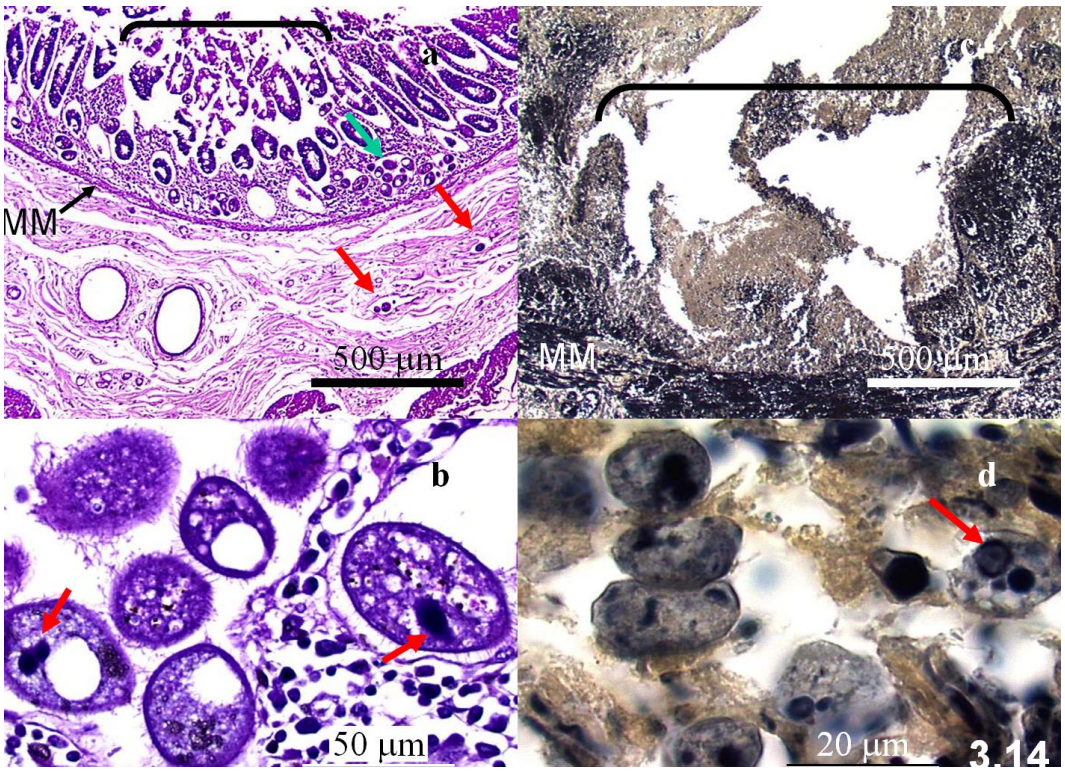
3.2 Cysts of *Entamoeba histolytica,* stool smear.



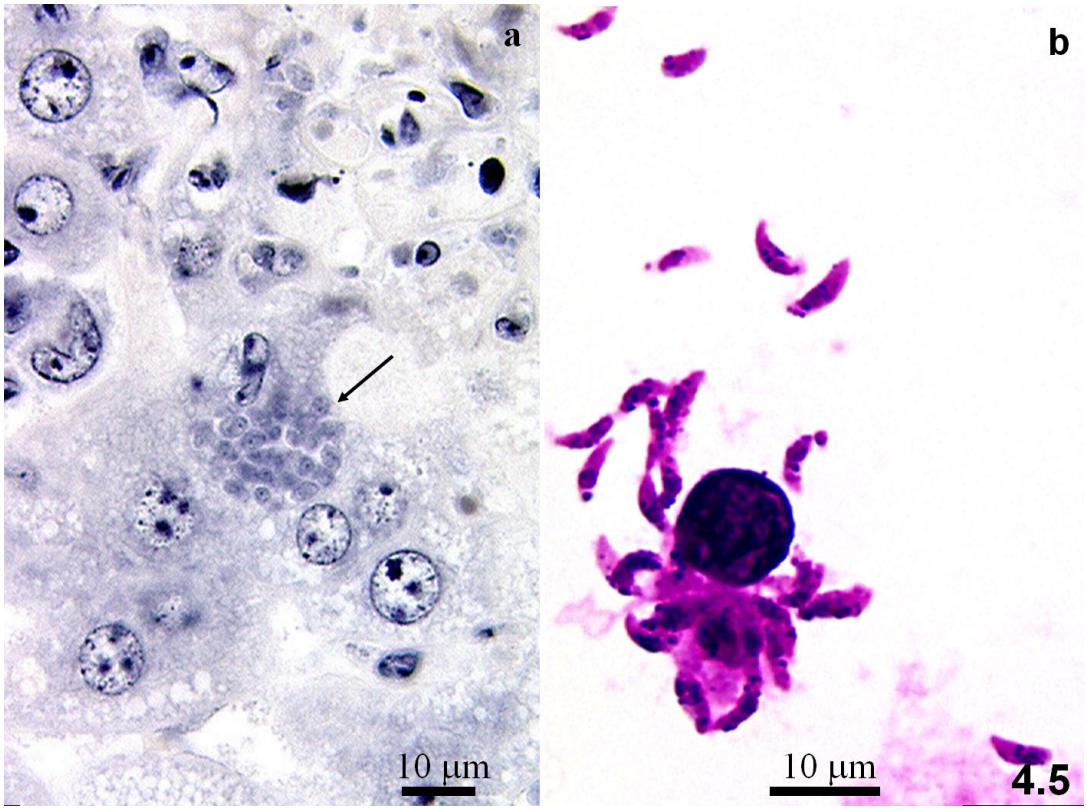
3.12 Trophozoites of *Balantidium coli,* stool smear.



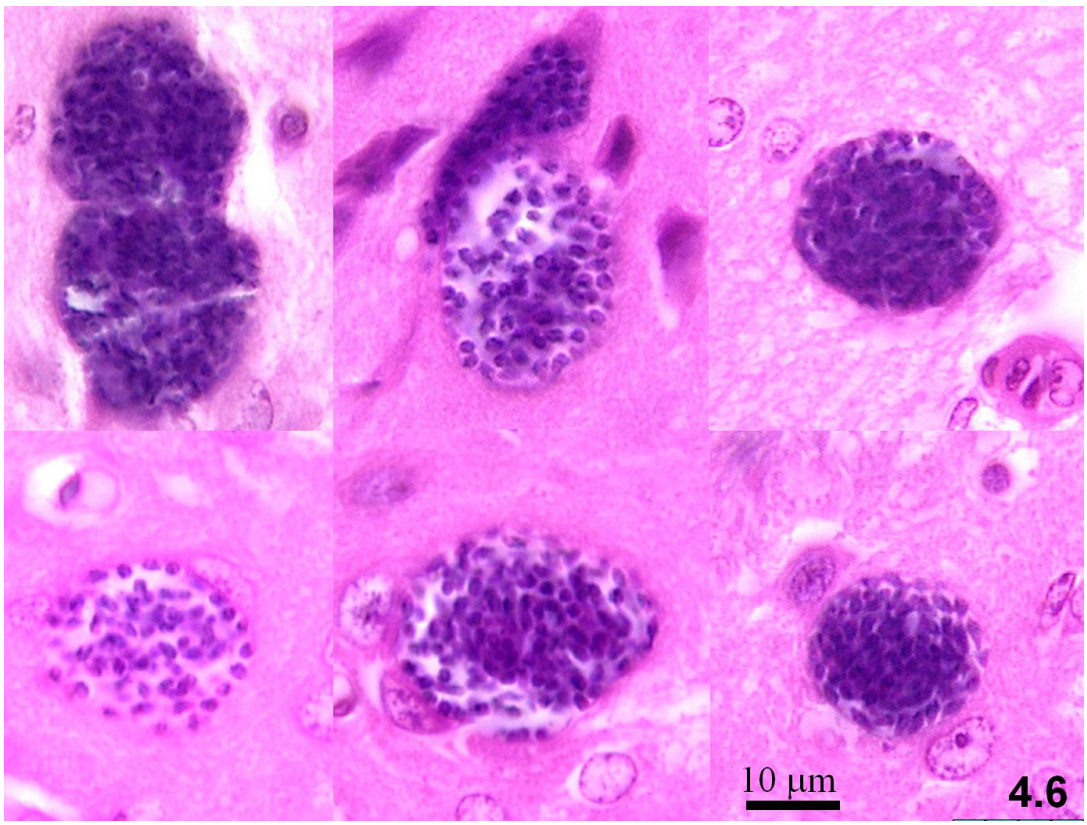
3.13 Cysts of *Balantidium coli,* stool smear.



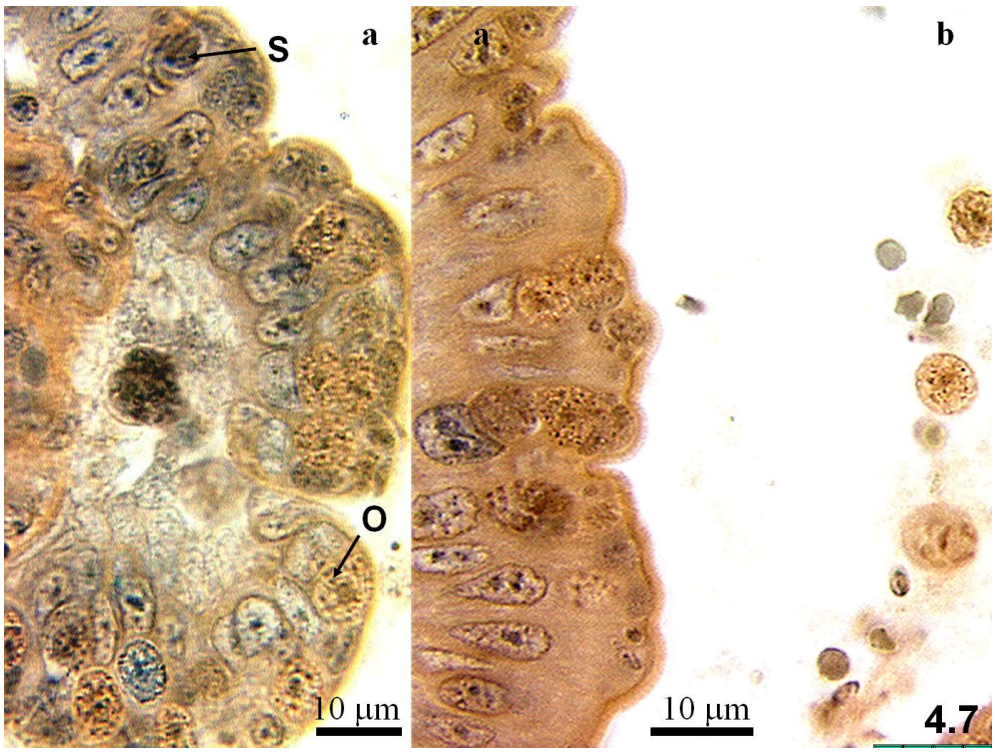
3.14 Intestinal balantidiasis and amebiasis, histological sections.



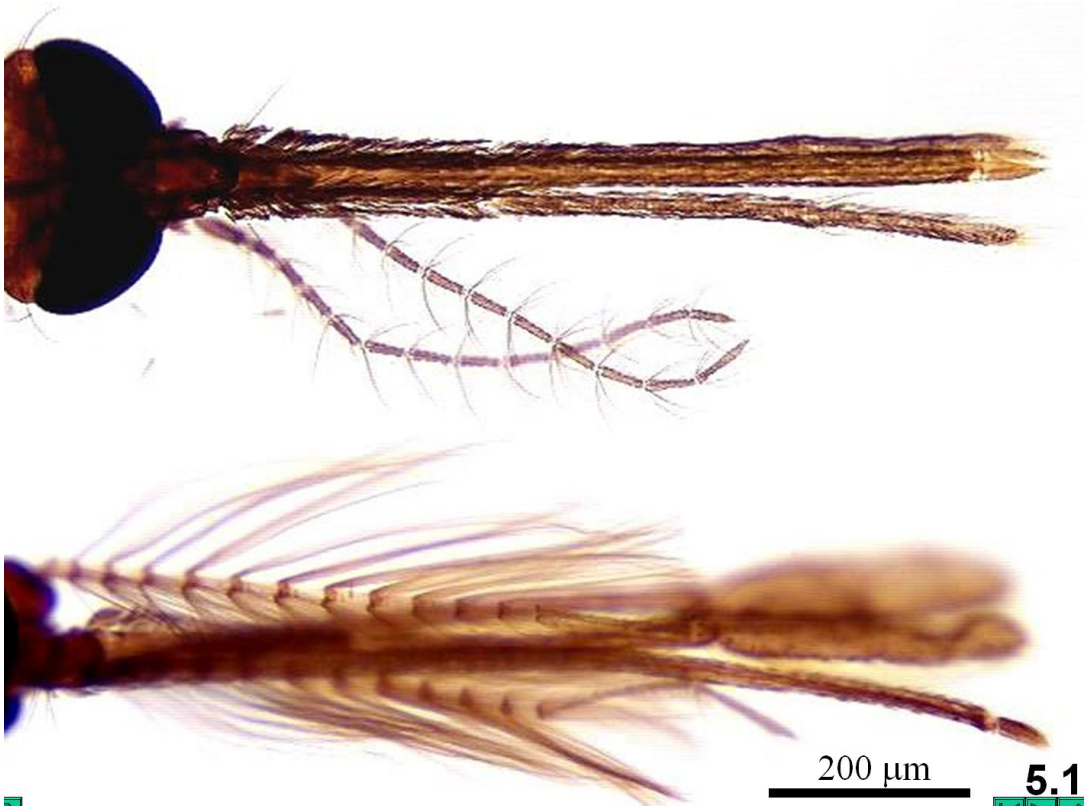
4.5 Extraintestinal cycle of *Toxoplasma gondii* in the mouse.



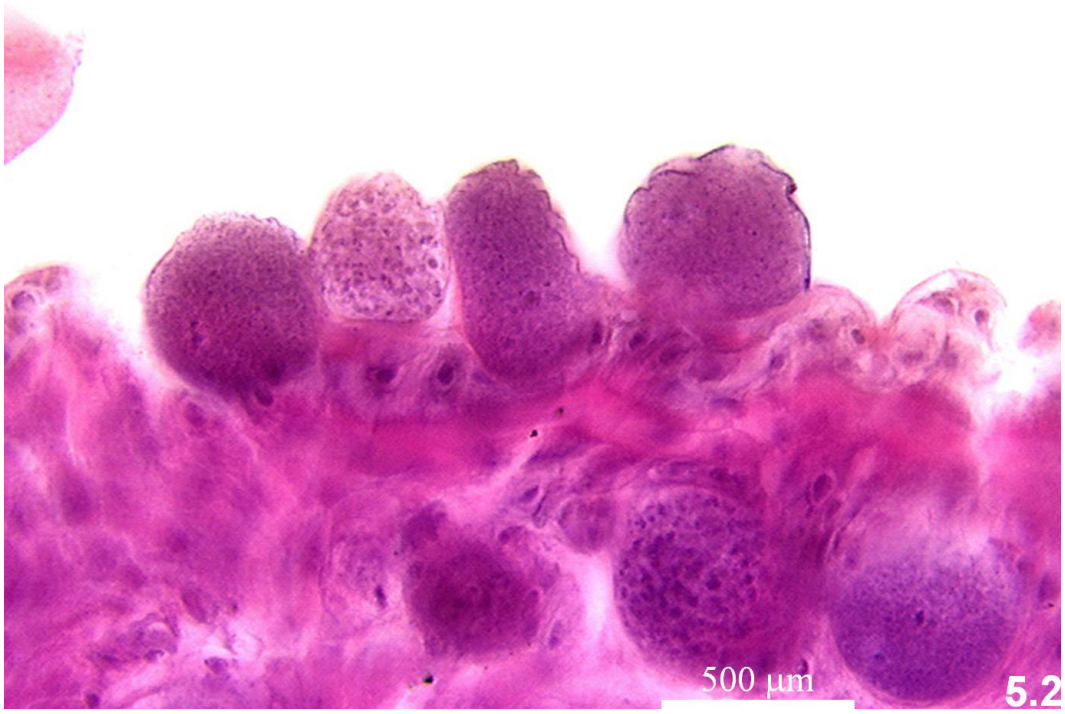
4.6 Histological sections of zoitocysts of *Toxoplasma gondii*



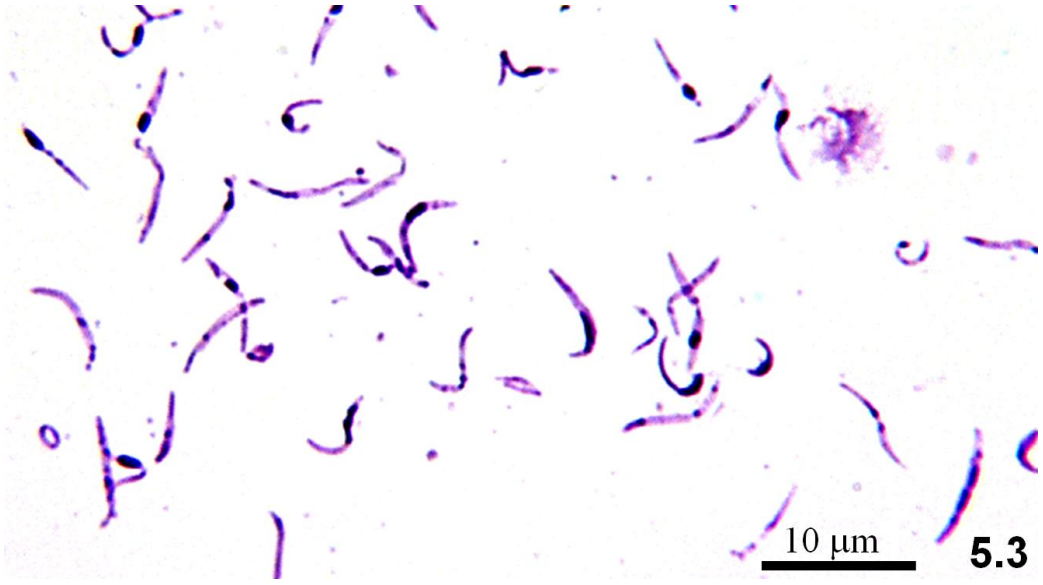
4.7 Enteroepithelial cycle of *Toxoplasma gondii*



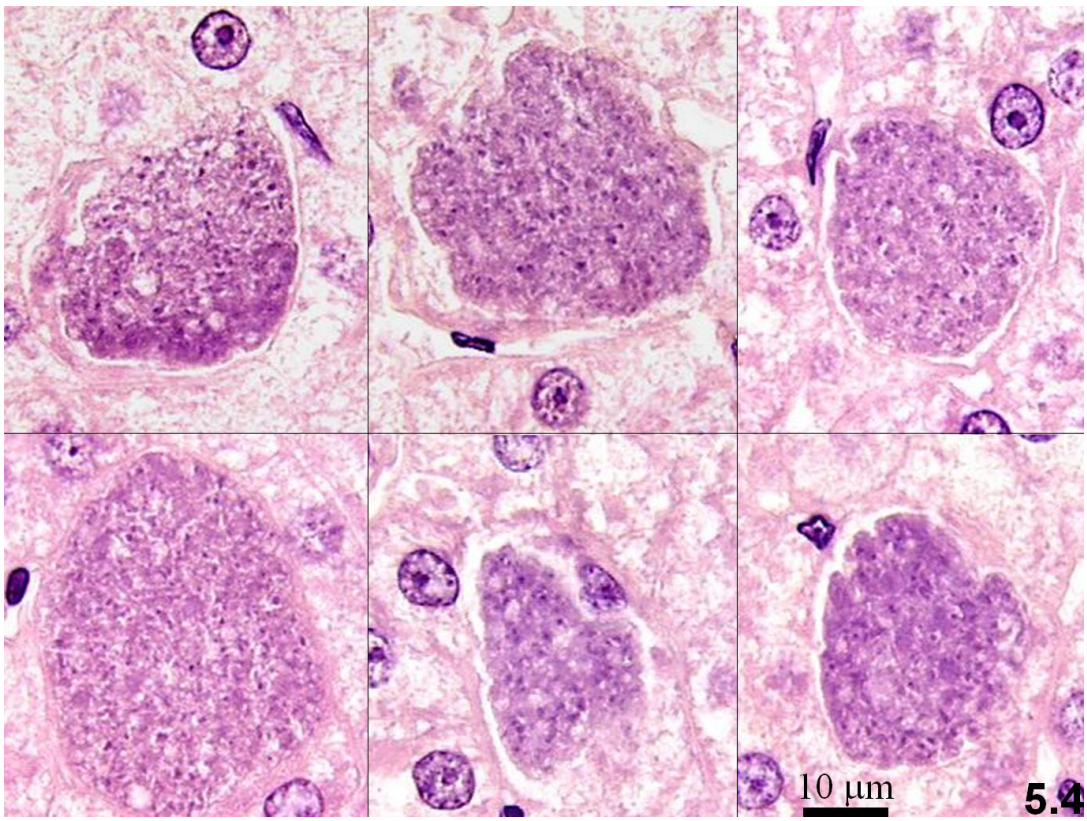
5.1 Mouthparts of *Anopheles* sp. mosquitos.



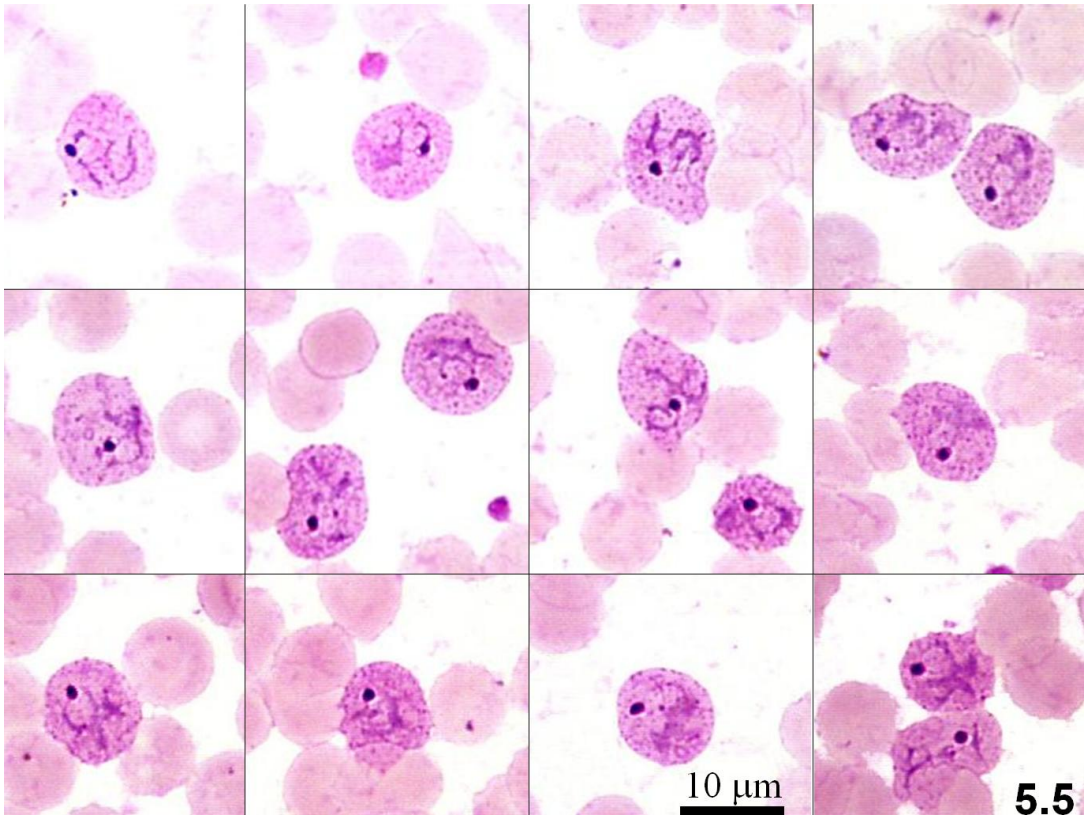
5.2 Oocysts of *Plasmodium* sp.



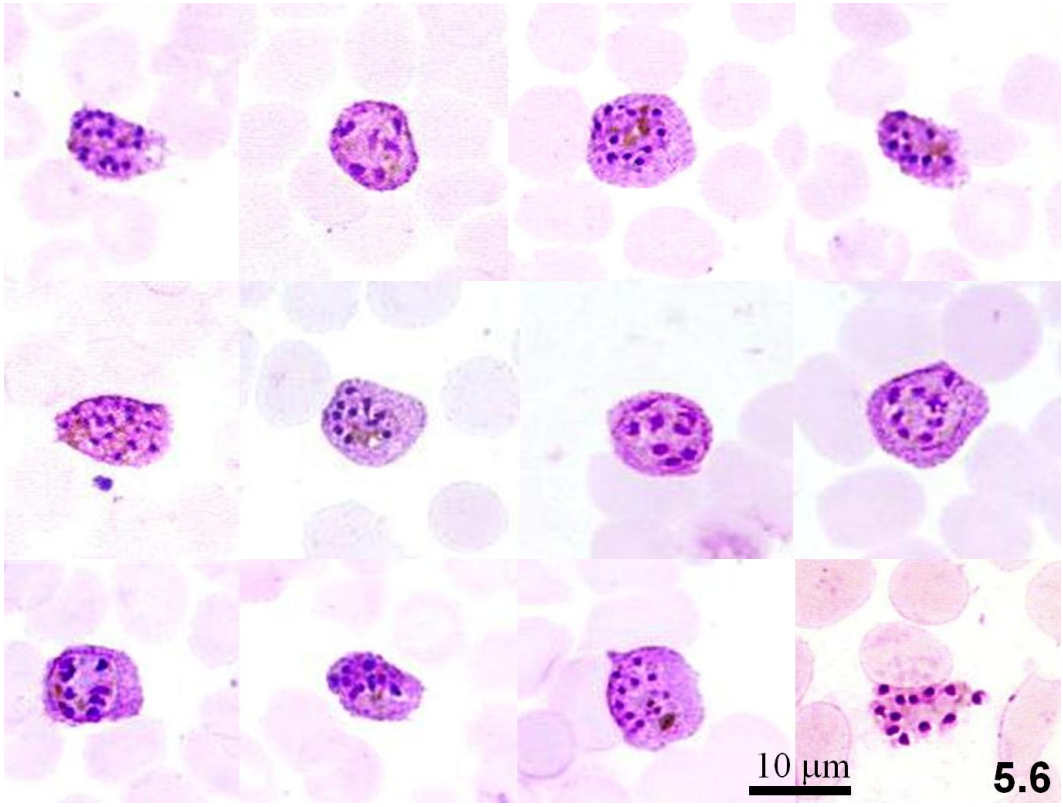
5.3 Sporozoites of *Plasmodium vivax*



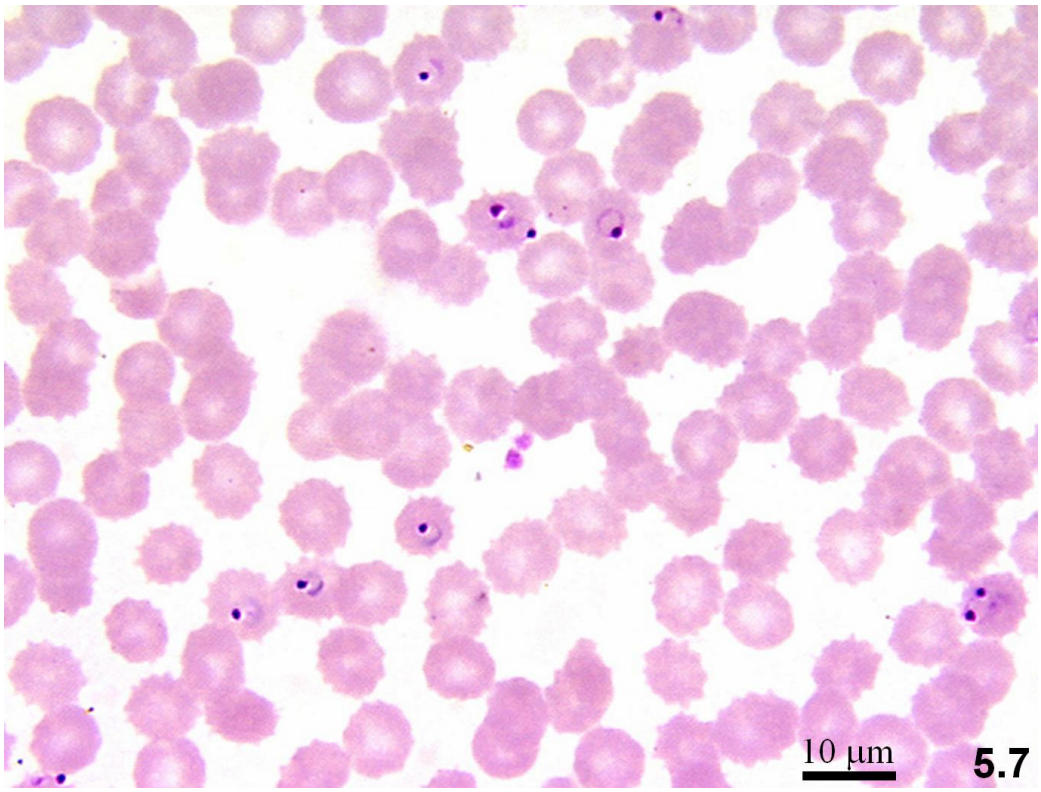
5.4 Exoerythrocytic schizonts of *Plasmodium* sp. in liver cells.



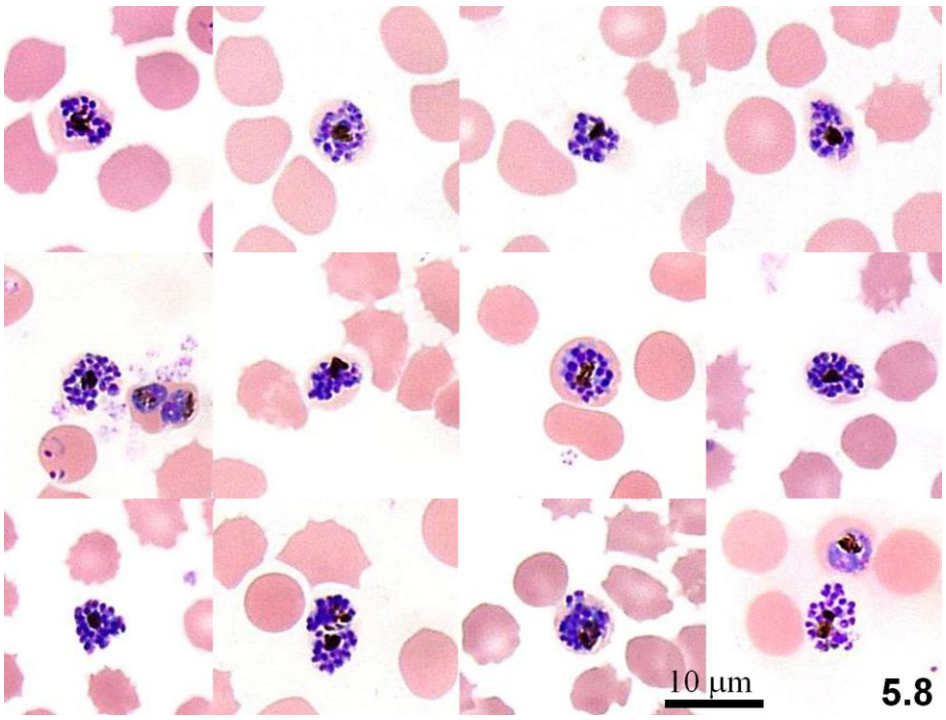
5.5 Trophozoites of *Plasmodium vivax*.



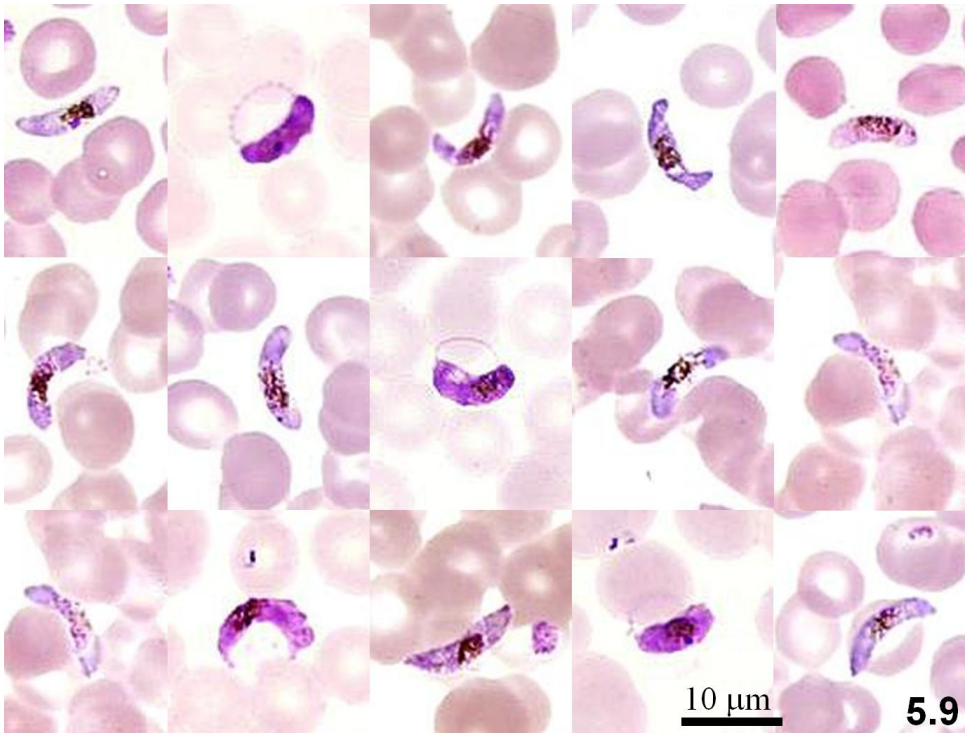
5.6 Erythrocytic schizonts of *Plasmodium vivax.*



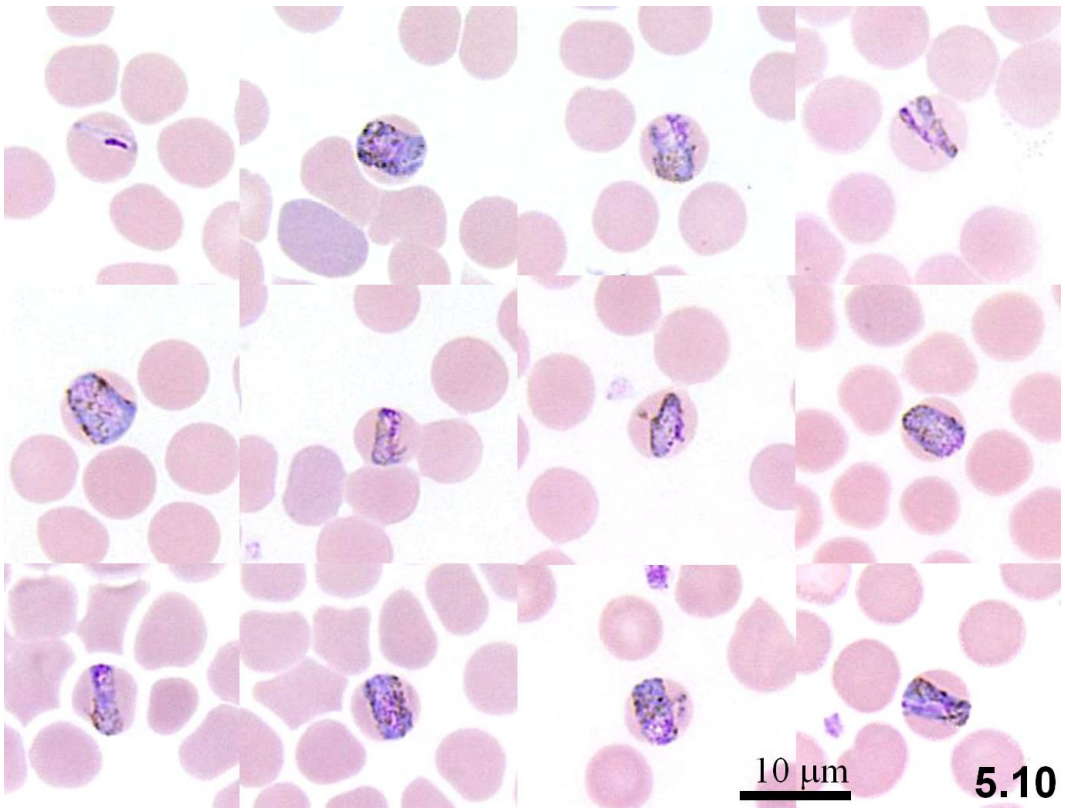
5.7 Young signet ring stage trophozoites of *Plasmodium falciparum.*



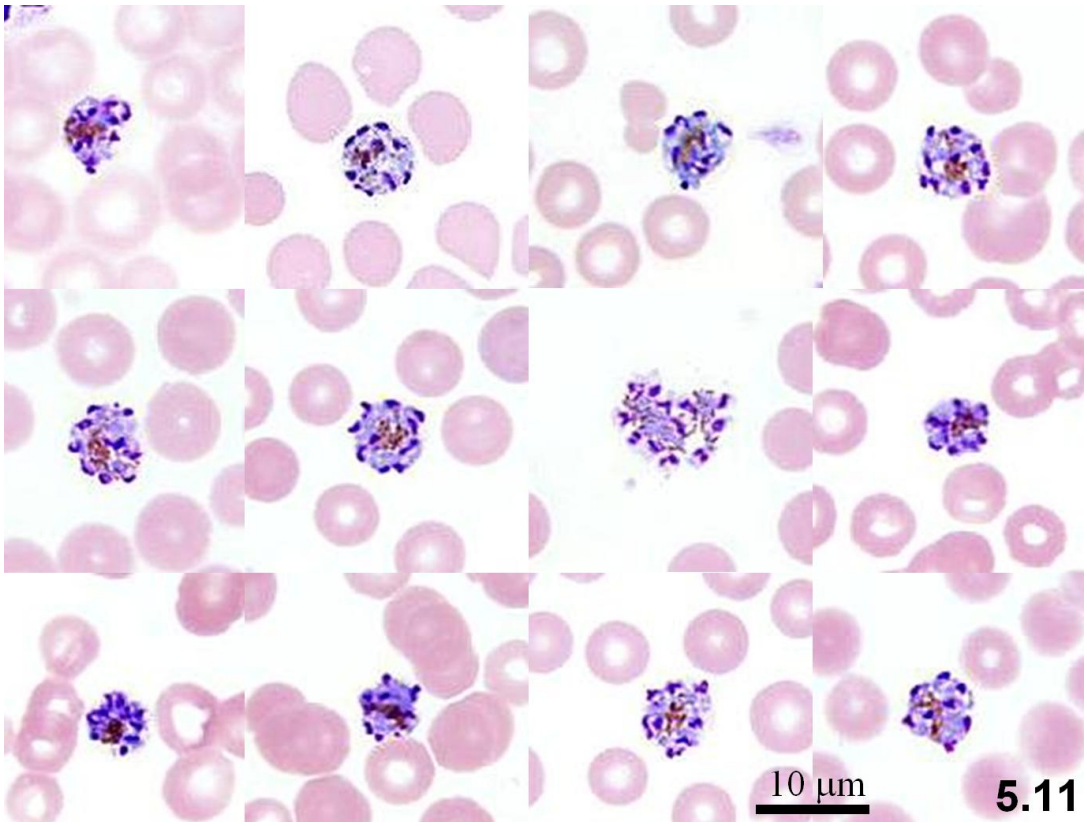
5.8 Erythrocytic schizonts of *Plasmodium falciparum*.



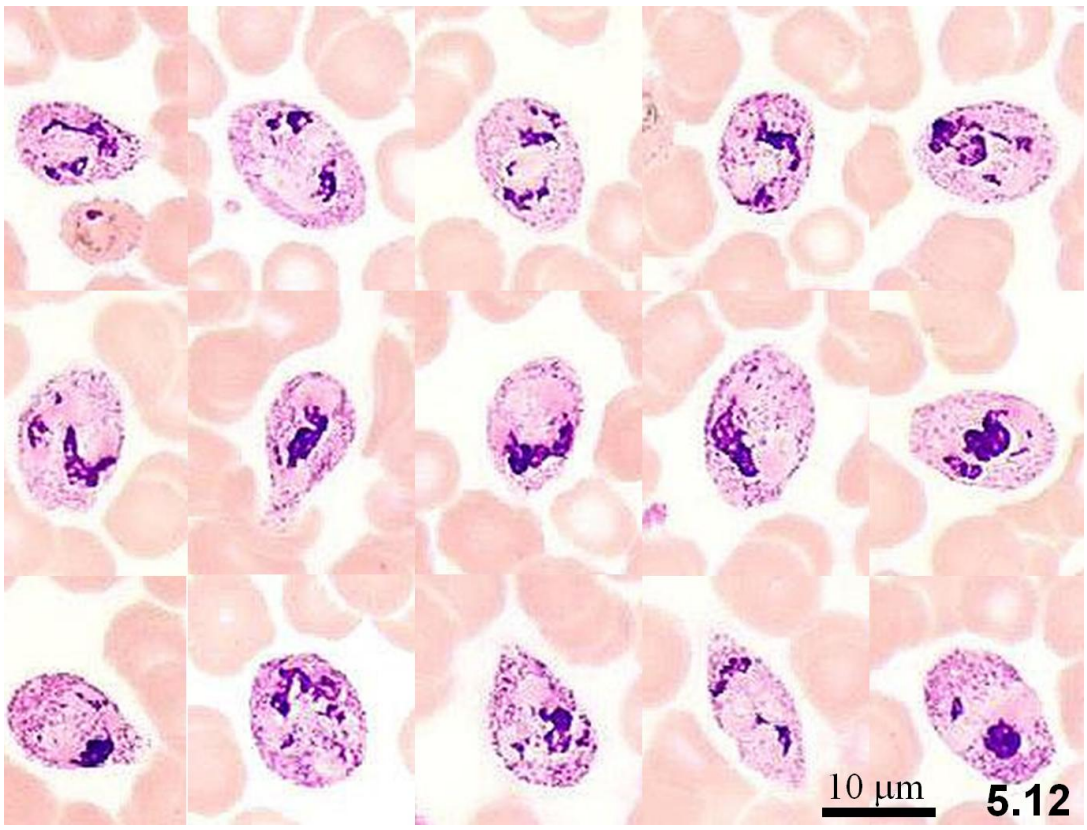
5.9 Gametocytes of *Plasmodium falciparum*.



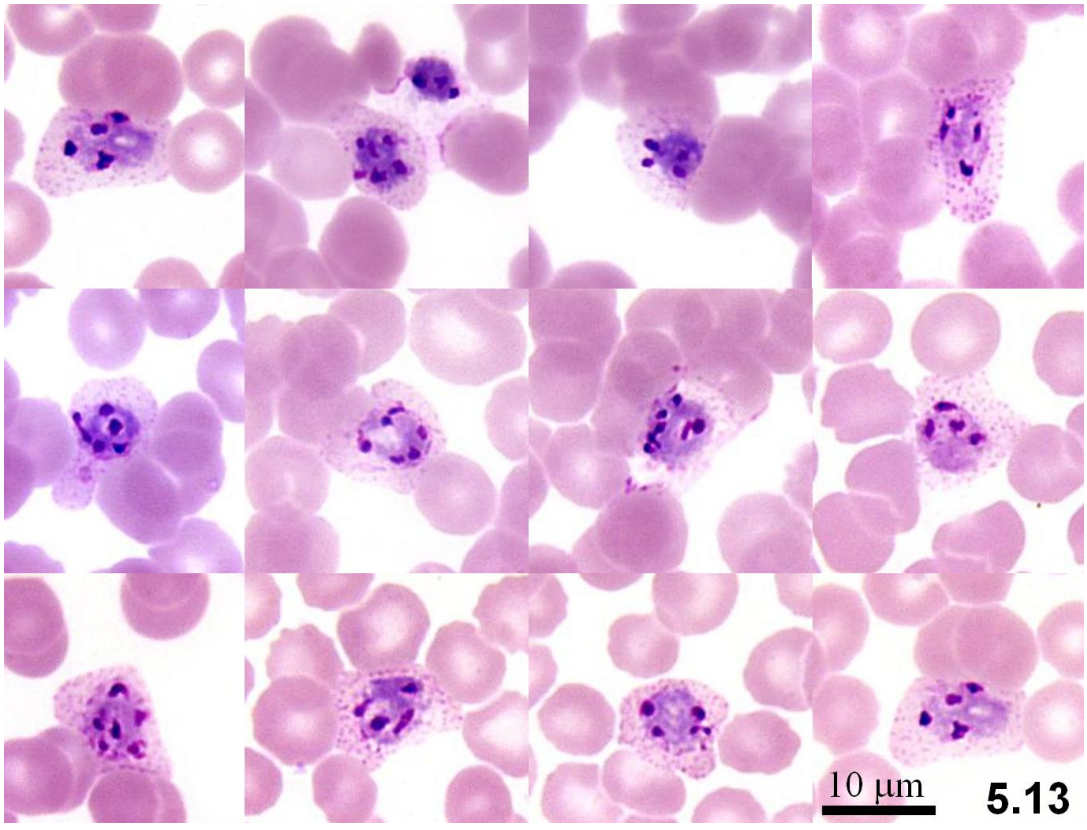
5.10 Trophozoites of *Plasmodium malariae*.



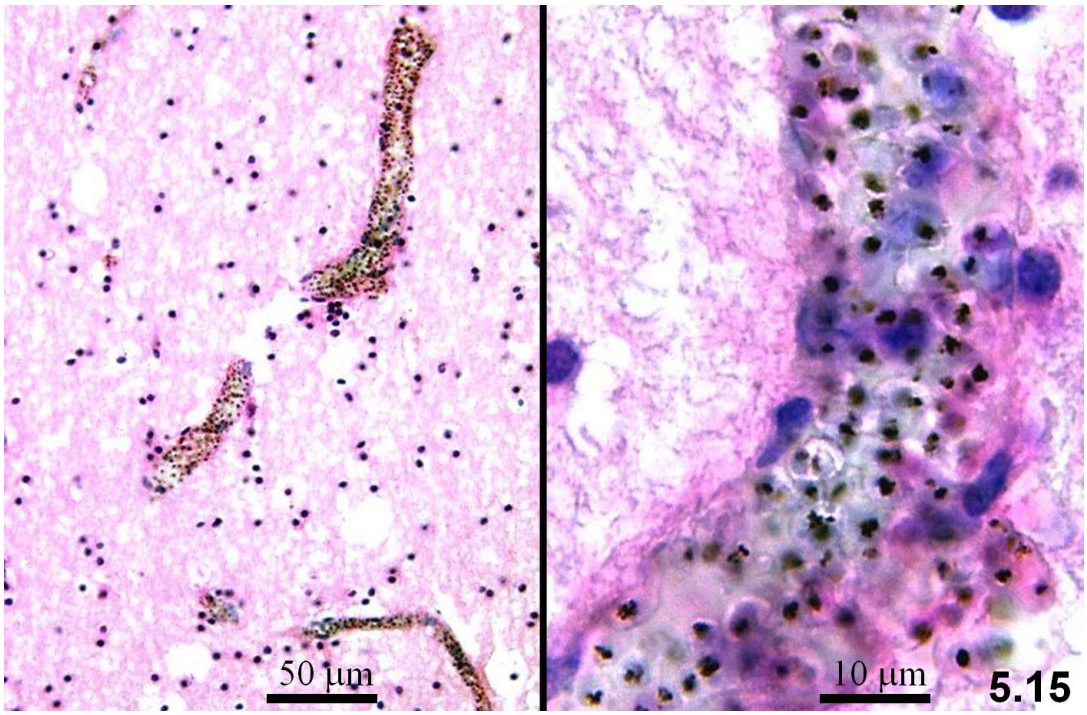
5.11 Erythrocytic schizonts of *Plasmodium malariae*.



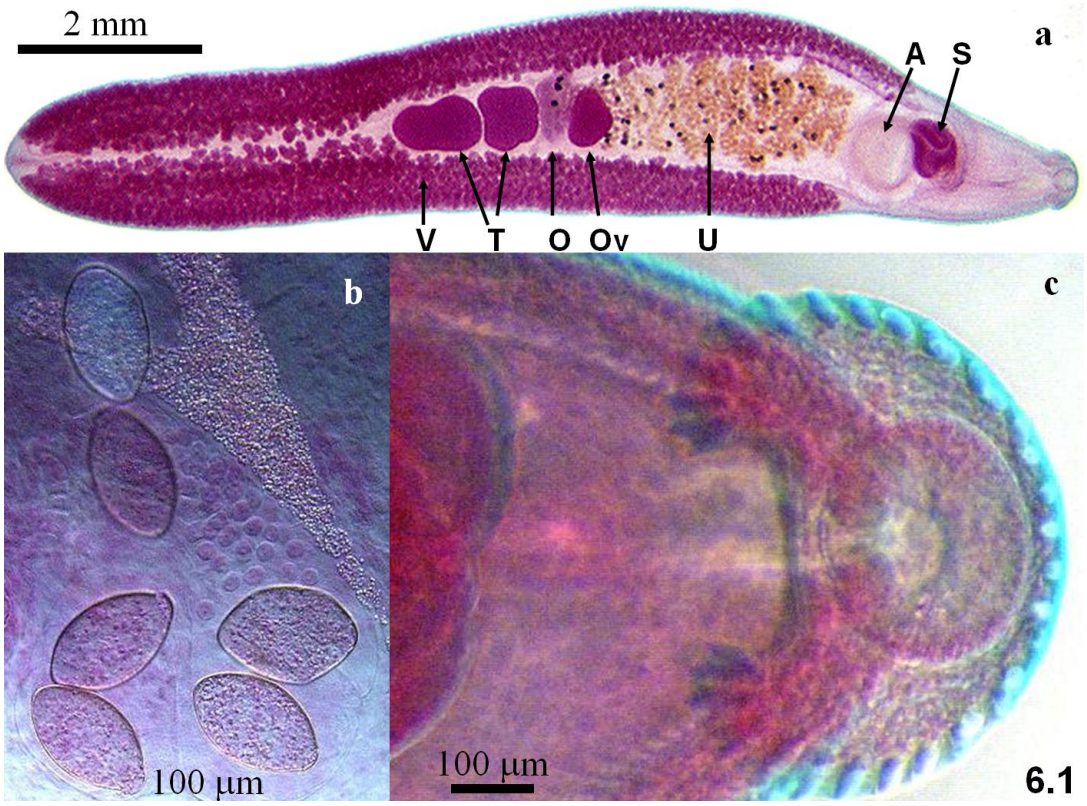
5.12 Growing trophozoites of *Plasmodium ovale*.



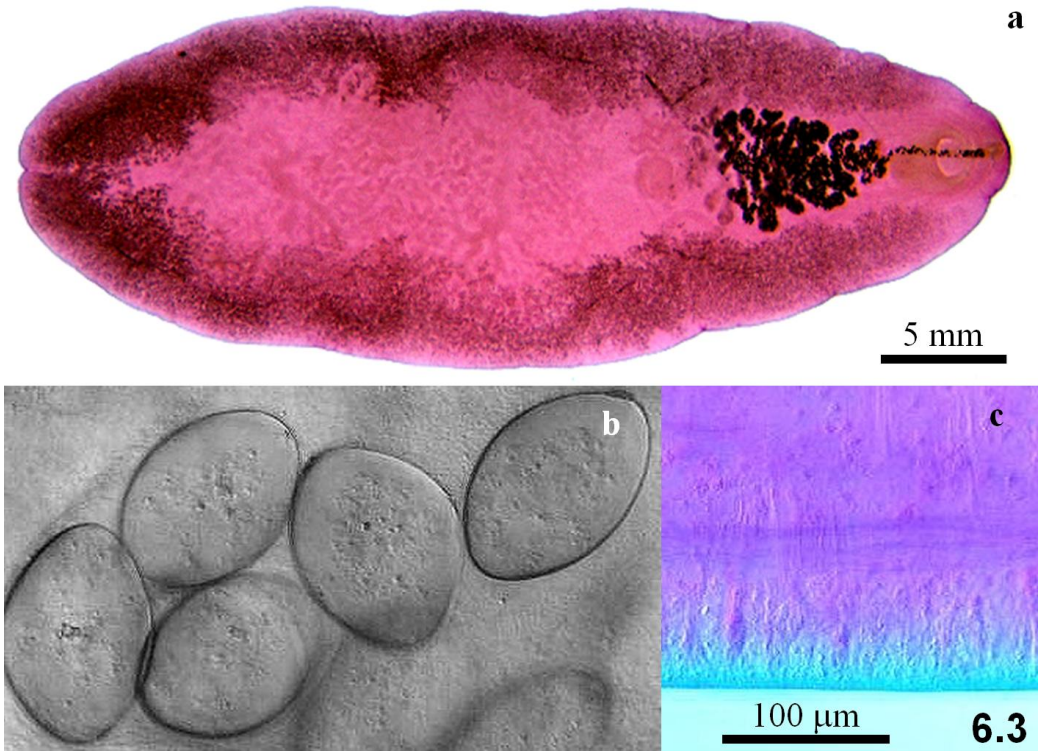
5.13 Erythrocytic schizonts of *Plasmodium ovale*.



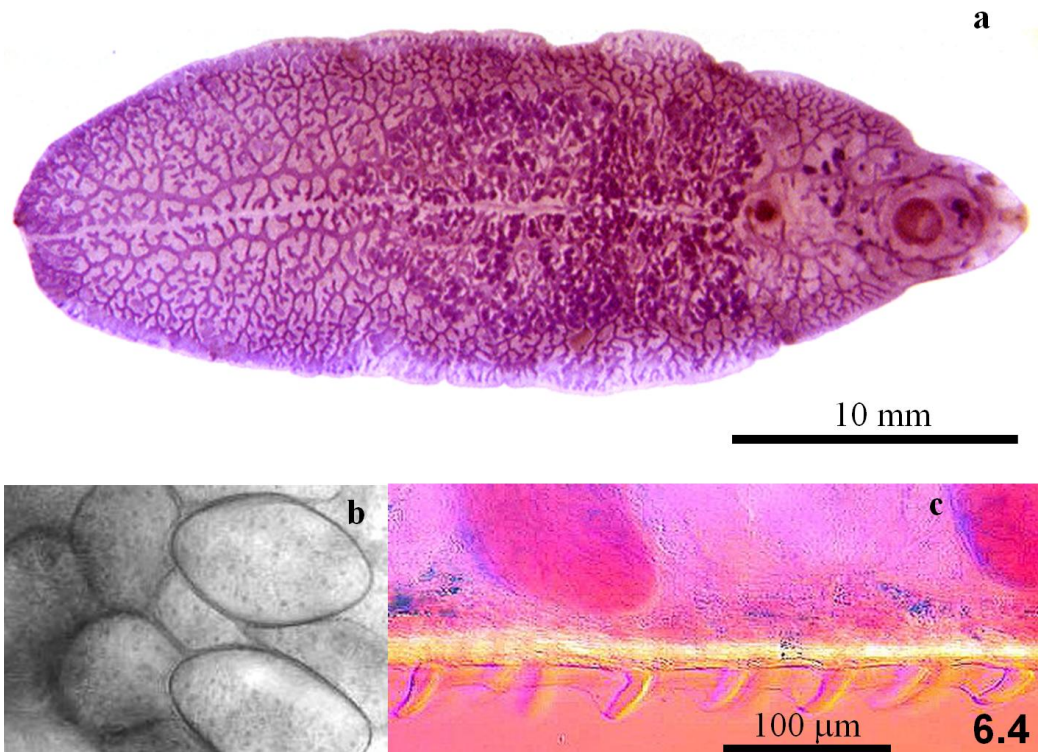
5.15 Histological section of brain from a case of cerebral malaria.



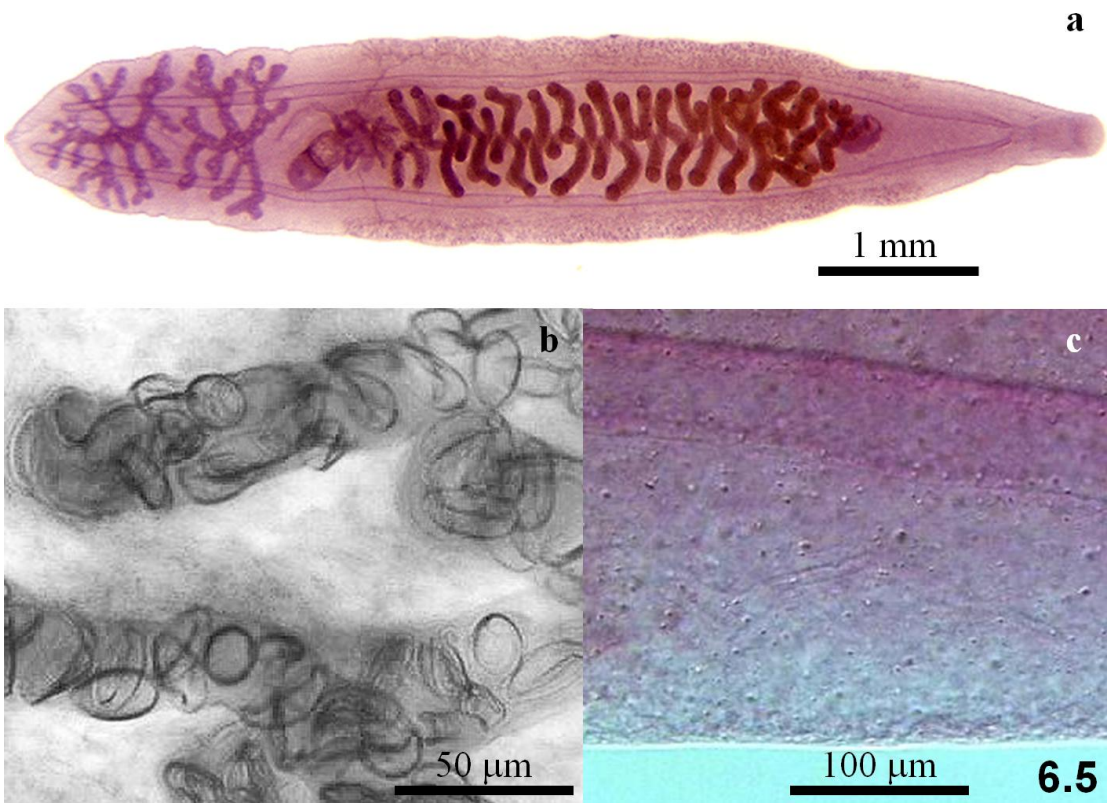
6.1 *Echinostoma revolutum.*



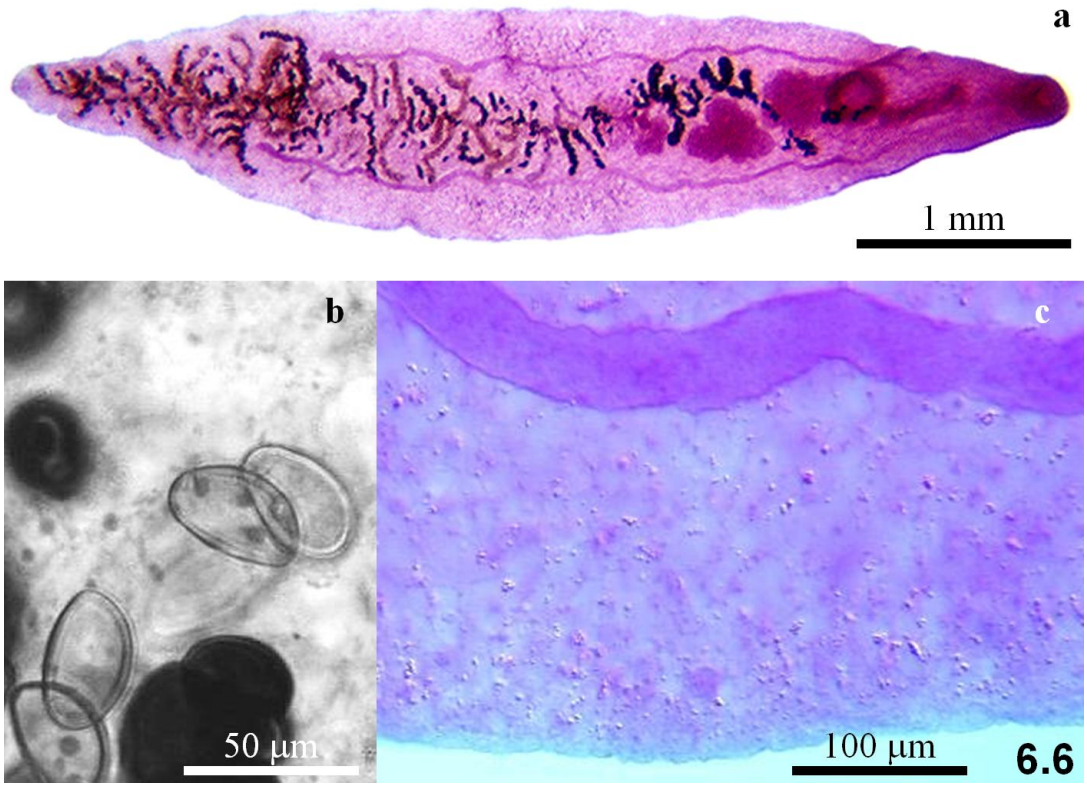
6.3 *Fasciolopsis buski.*



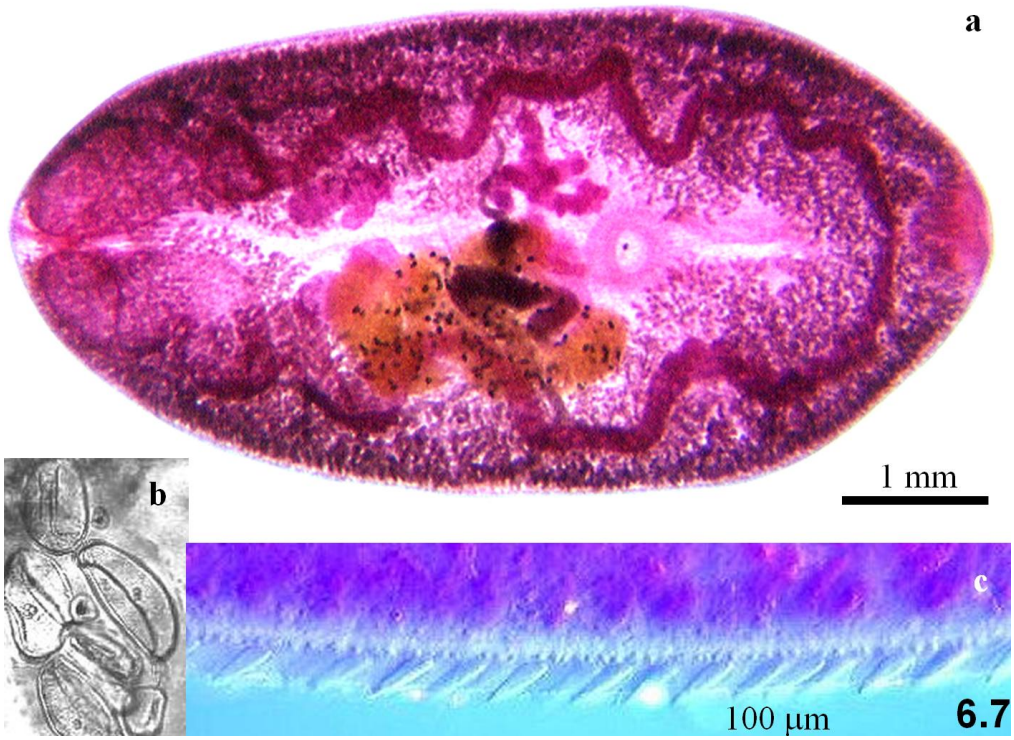
6.4 *Fasciola hepatica.*



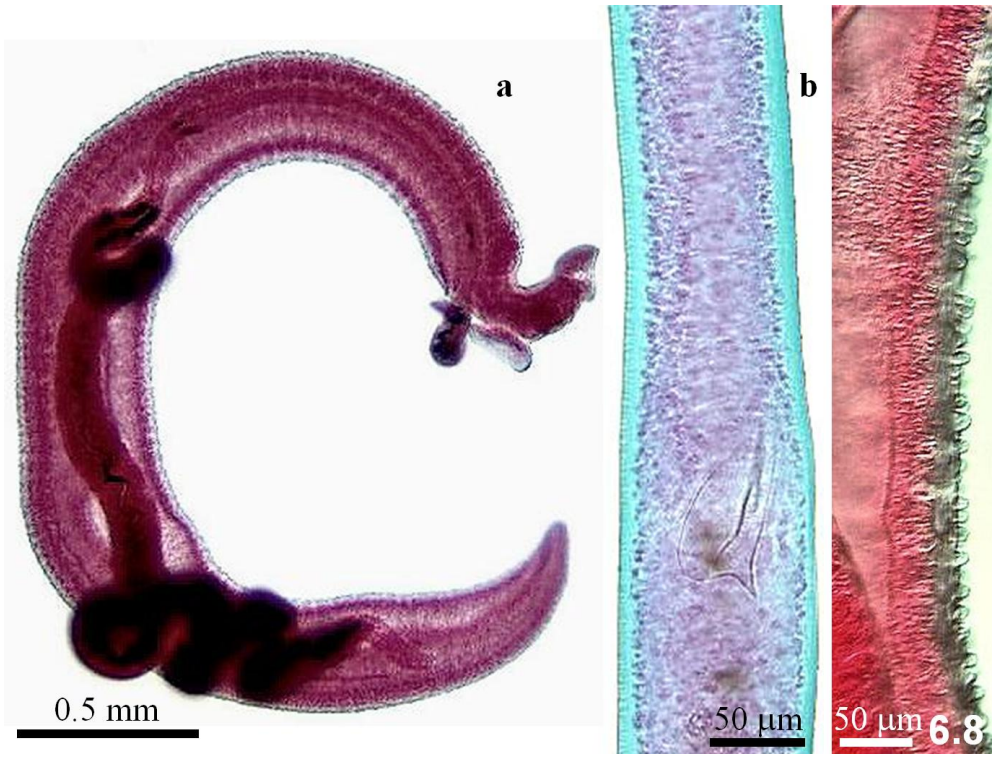
6.5 *Clonorchis sinensis.*



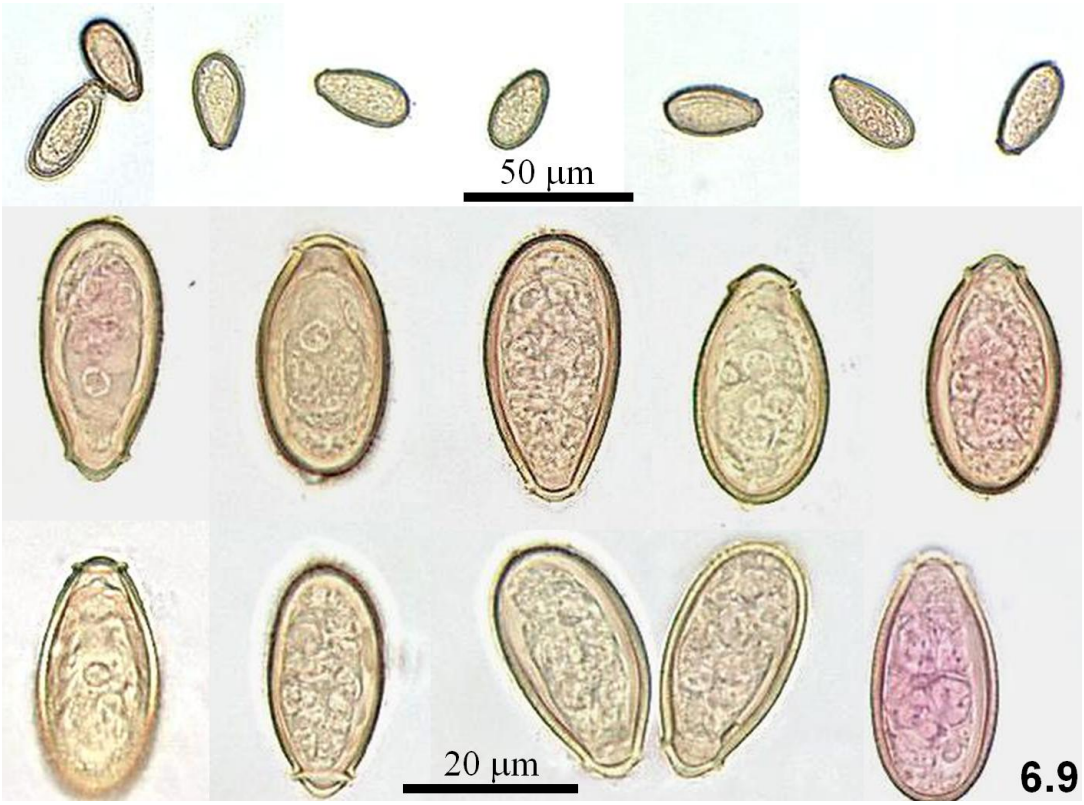
6.6 *Dicrocoelium dendriticum.*



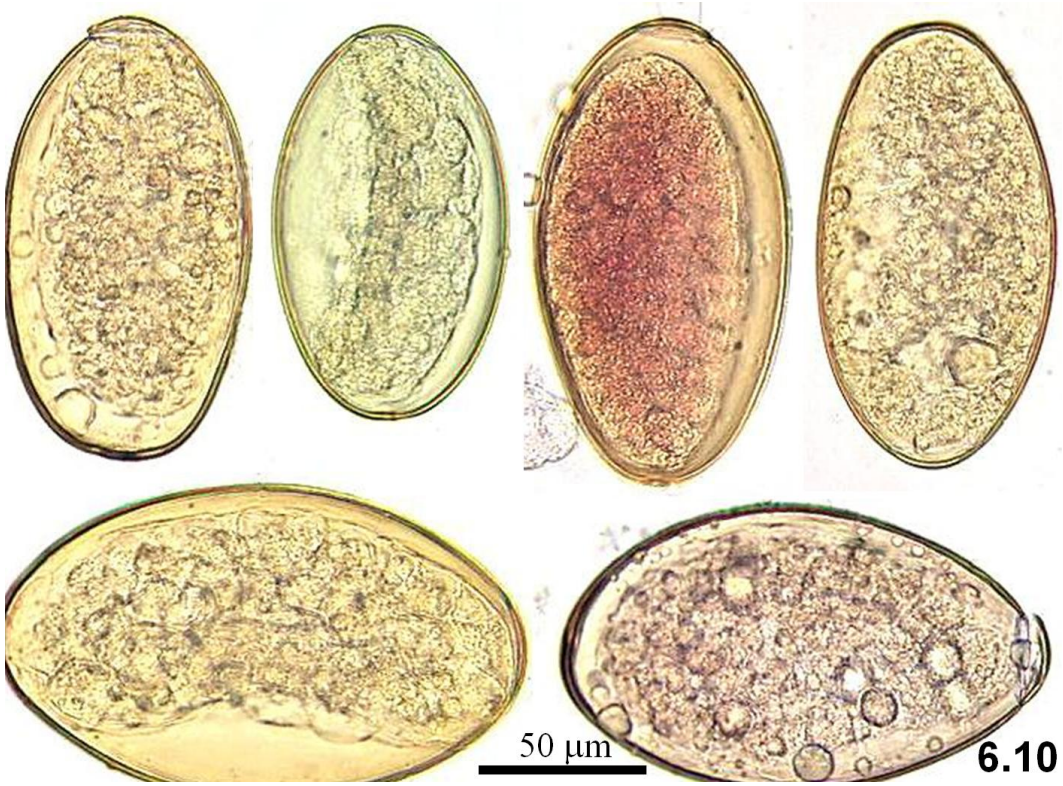
6.7 *Paragonimus westermani.*



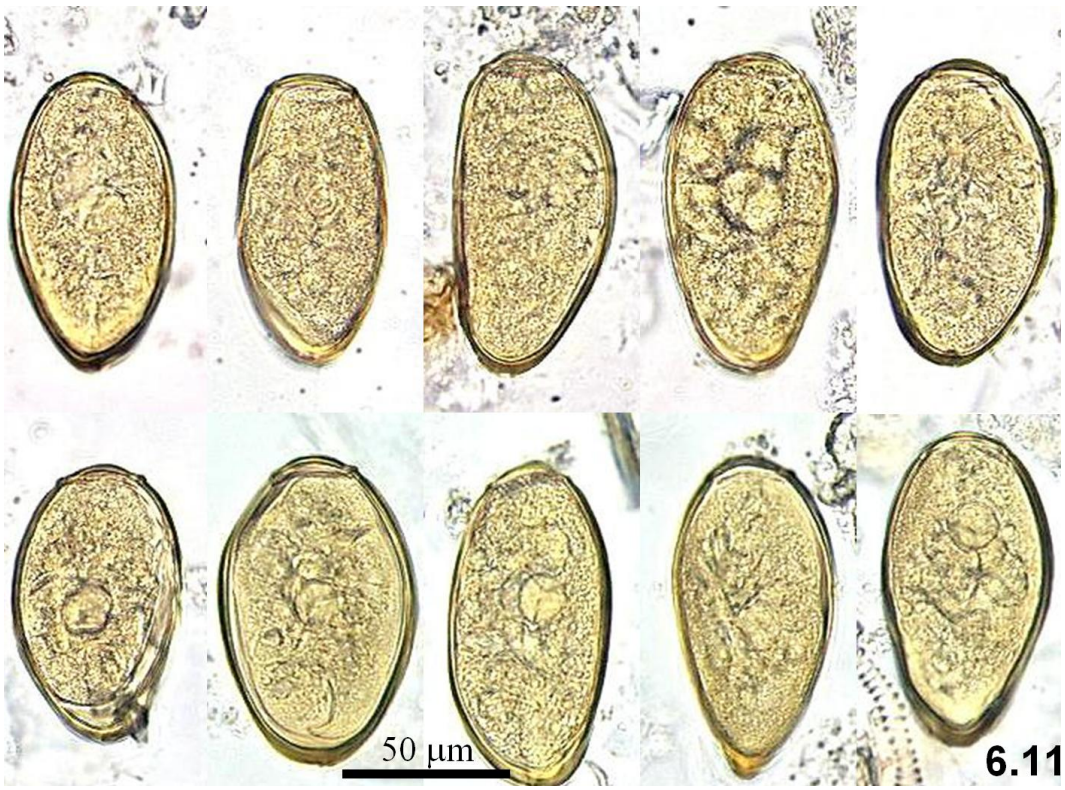
6.8 *Schistosoma mansoni.*



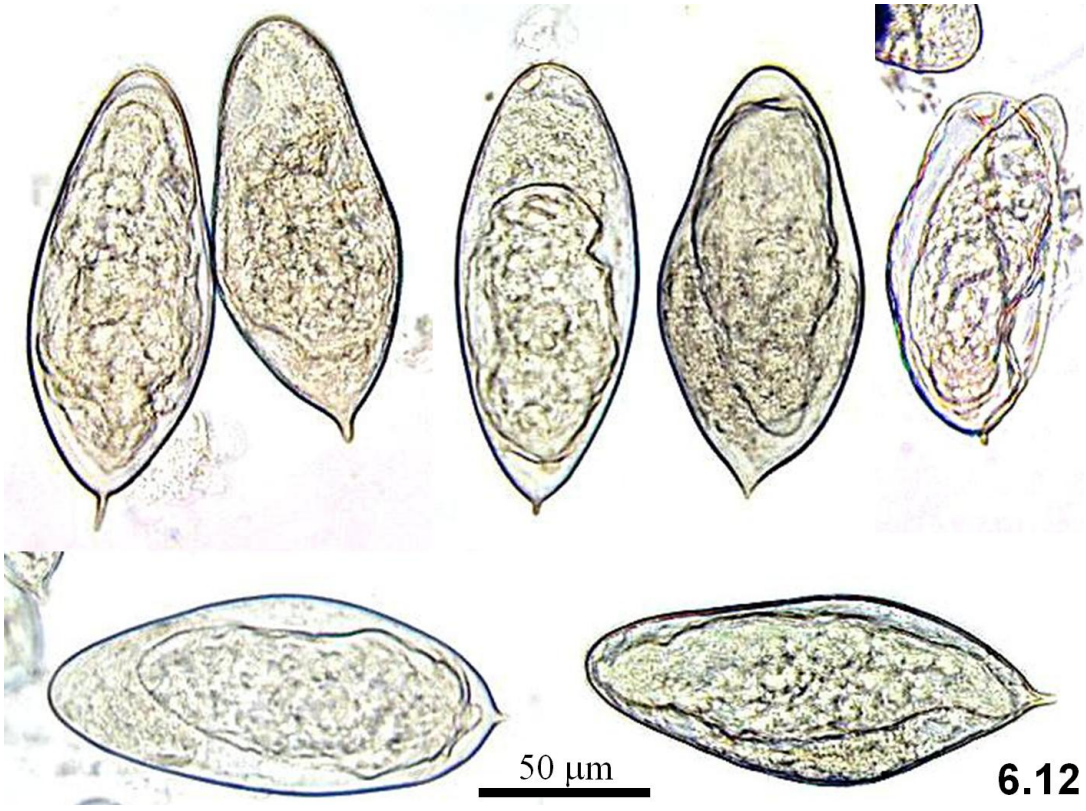
6.9 Eggs of *Clonorchis sinensis*.



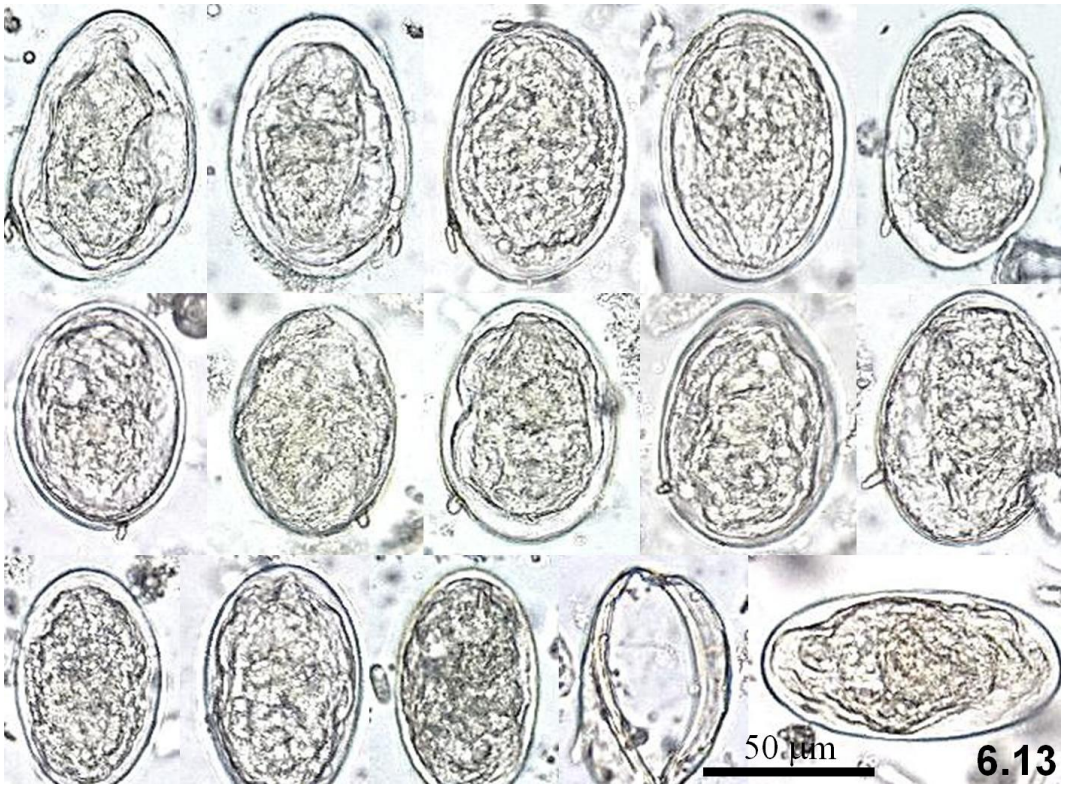
6.10 Eggs of *Fasciola hepatica*



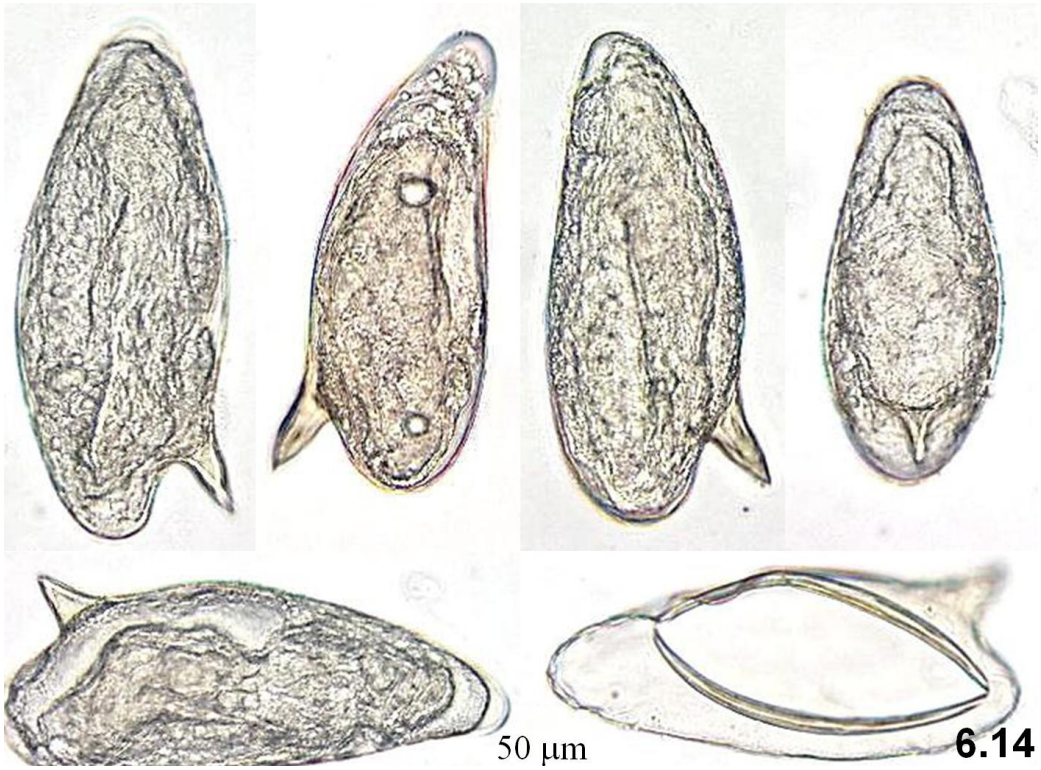
6.11 Eggs of *Paragonimus westermani*.



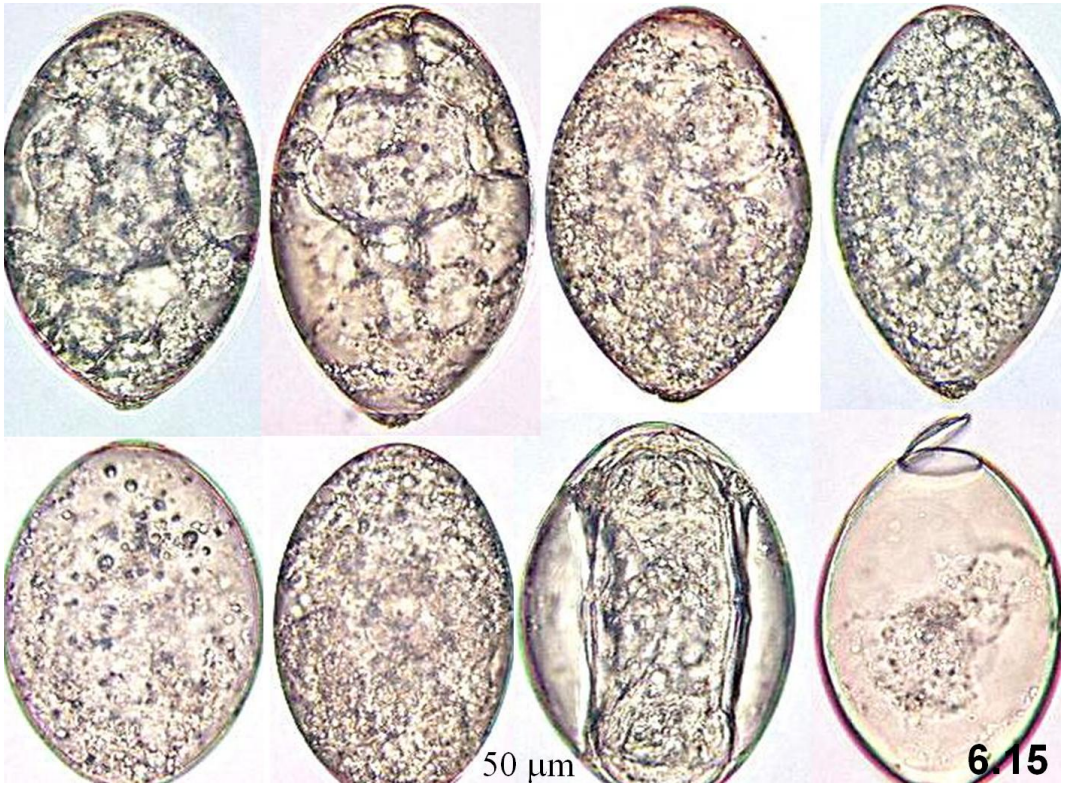
6.12 Eggs of *Schistosoma haematobium*.



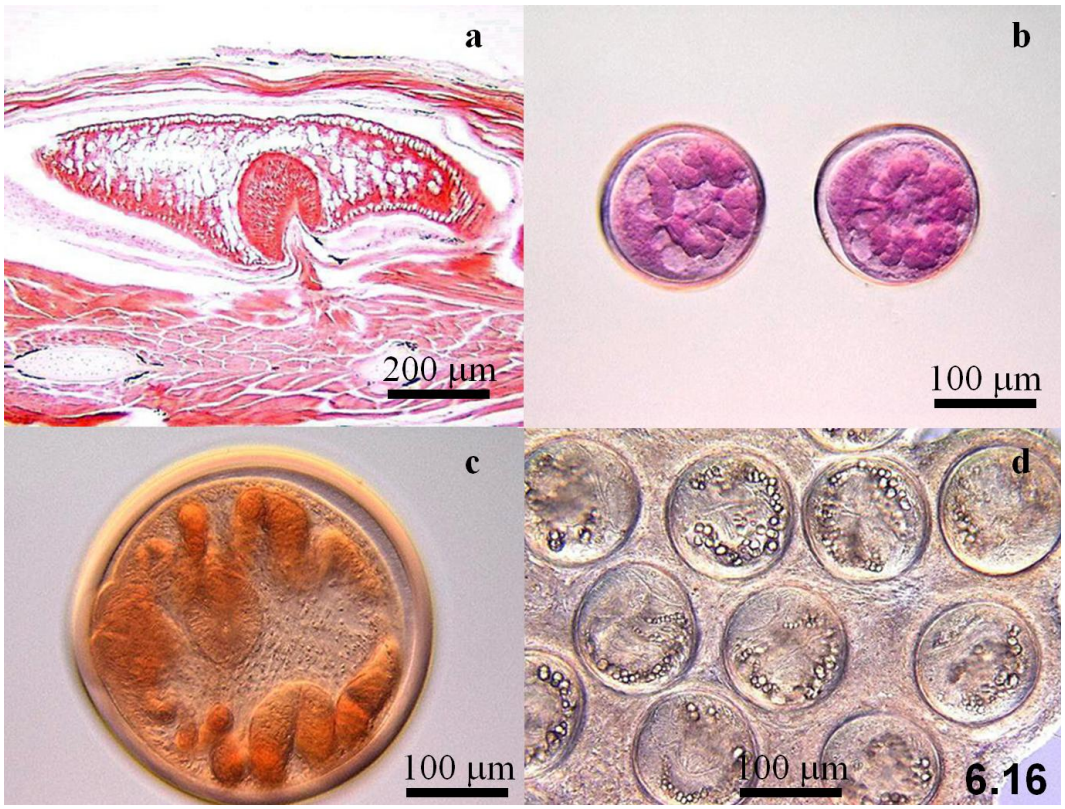
6.13 Eggs of *Schistosoma japonicum*.



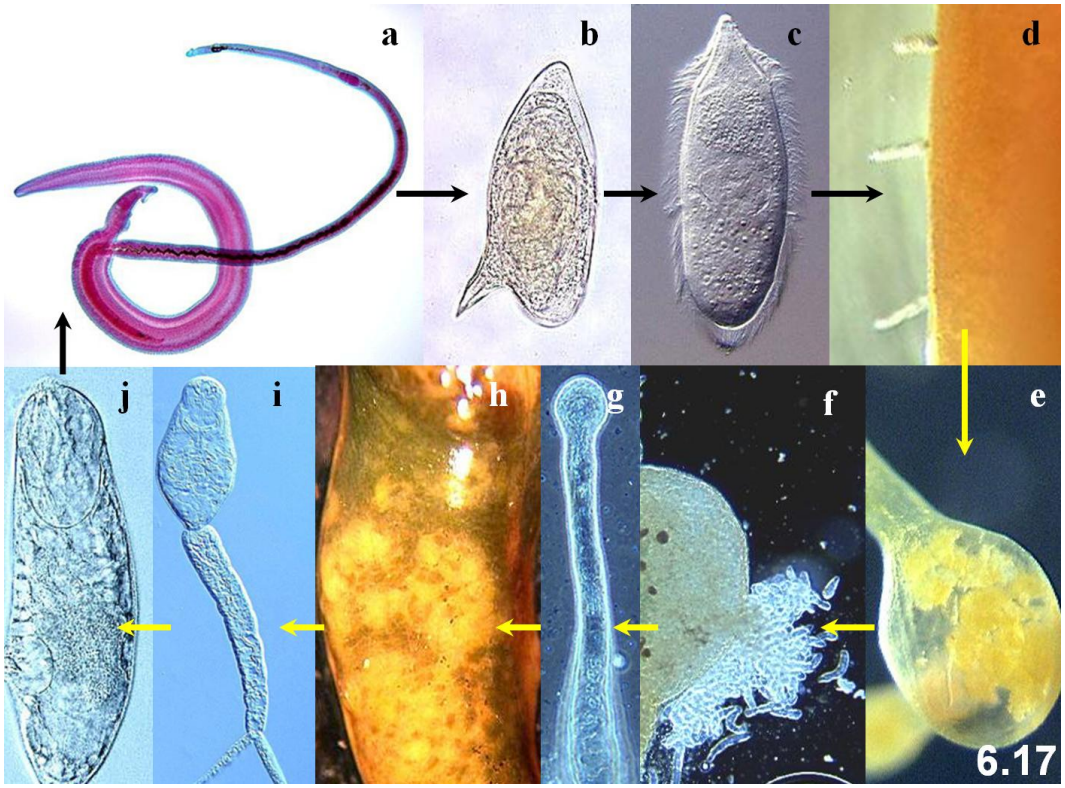
6.14 Eggs of *Schistosoma mansoni*



6.15 Eggs of *Echinostoma paraensei*.

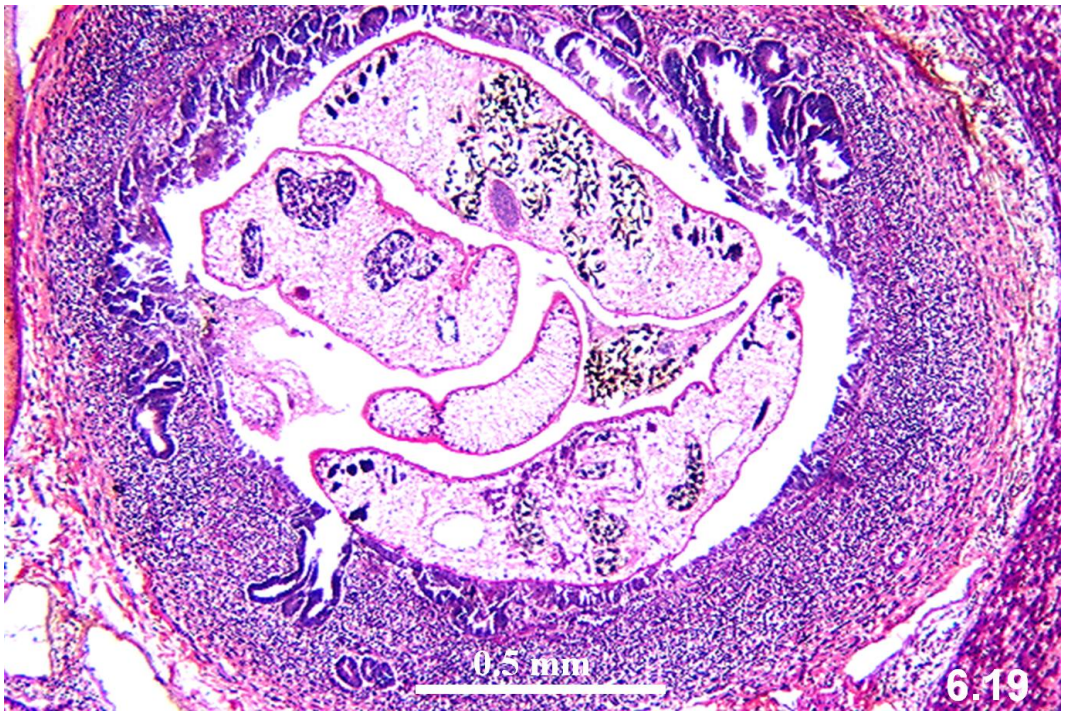


6.16 Metacercariae.

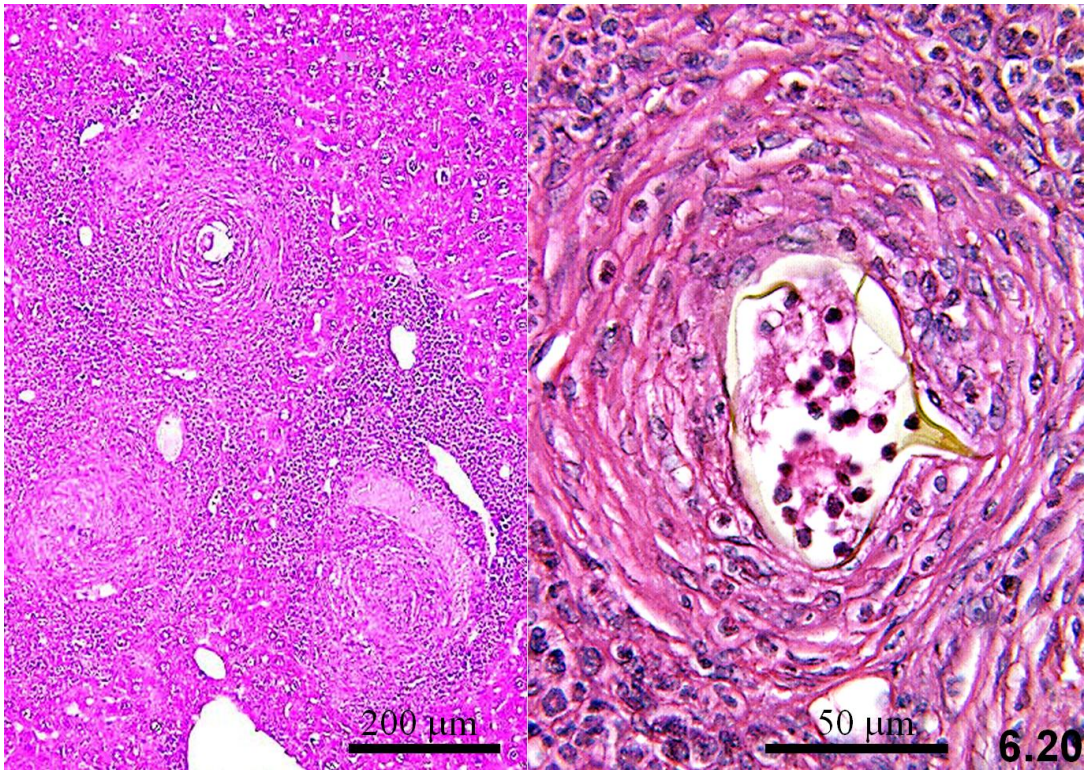


6.17 Life cycle of *Schistosoma mansoni*.

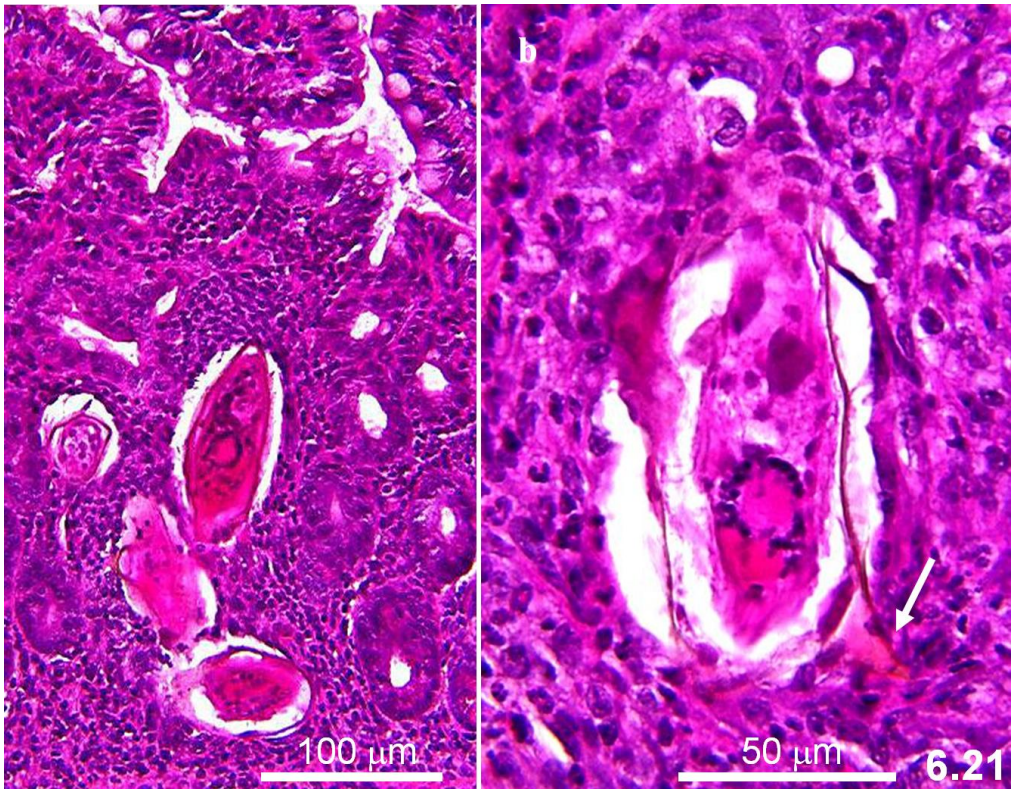
6.18 Snail vectors of schistosomiasis.



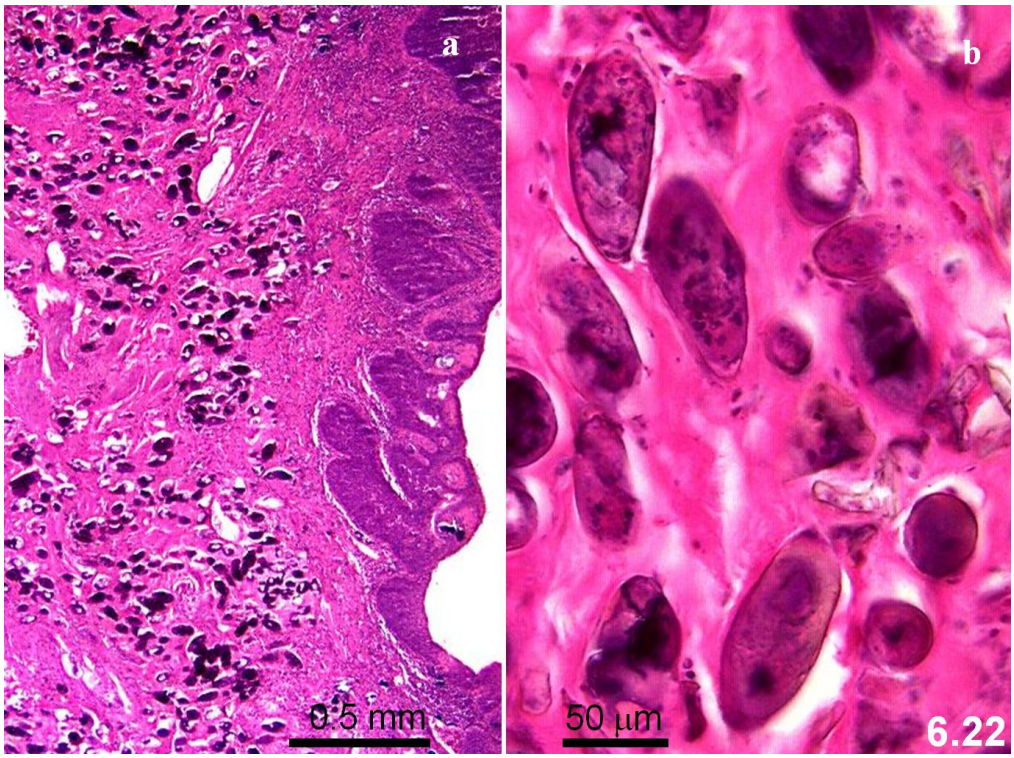
6.19 Histological section of bile duct infected with adults of *Clonorchis sinensis.*



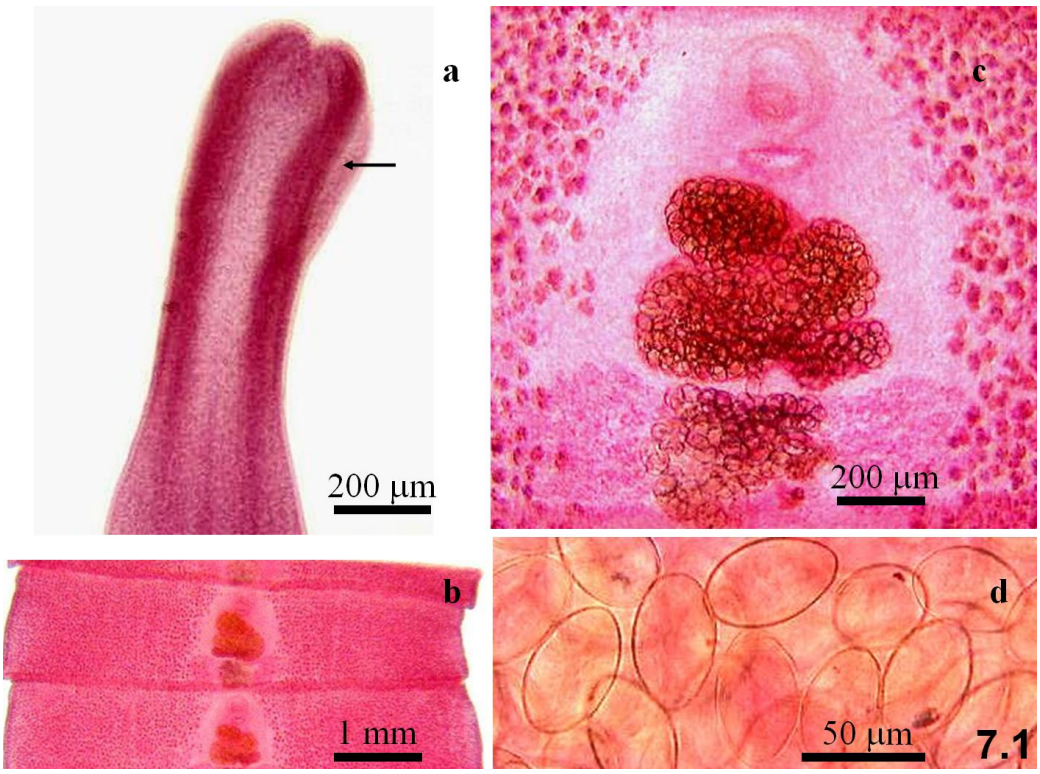
6.20 Histological section of liver from mouse infected with *Schistosoma mansoni.*



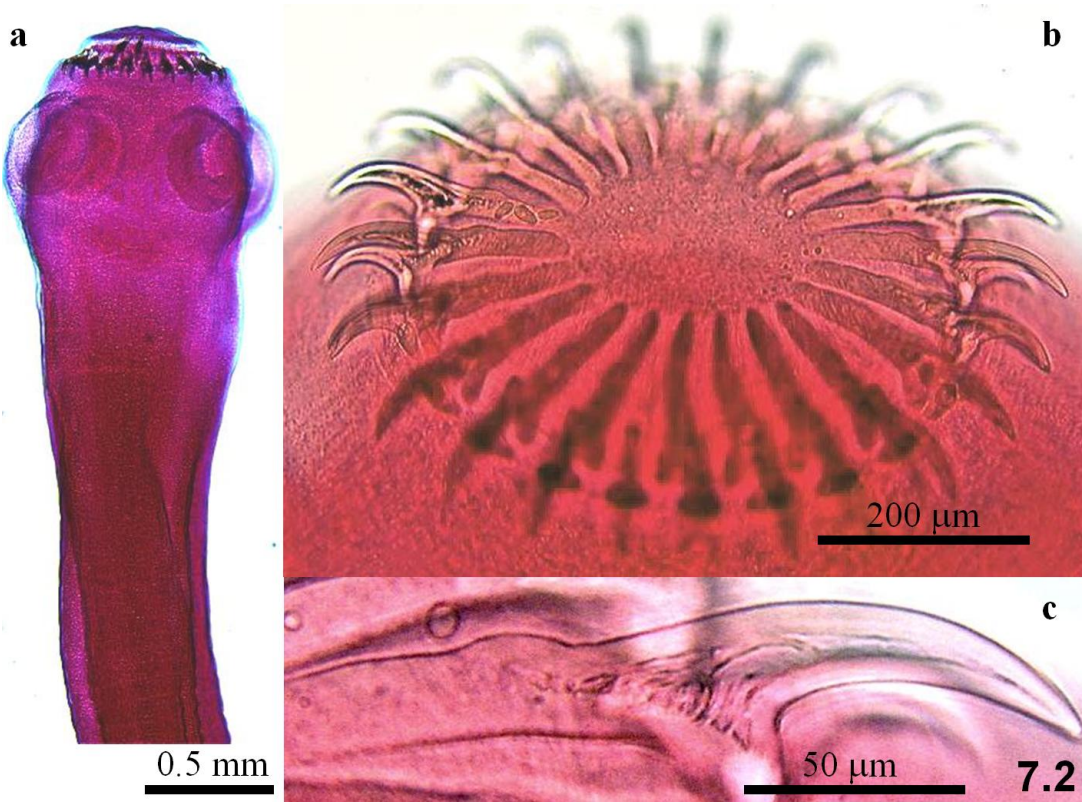
6.21 Histological section of small intestine from mouse infected with *Schistosoma mansoni.*



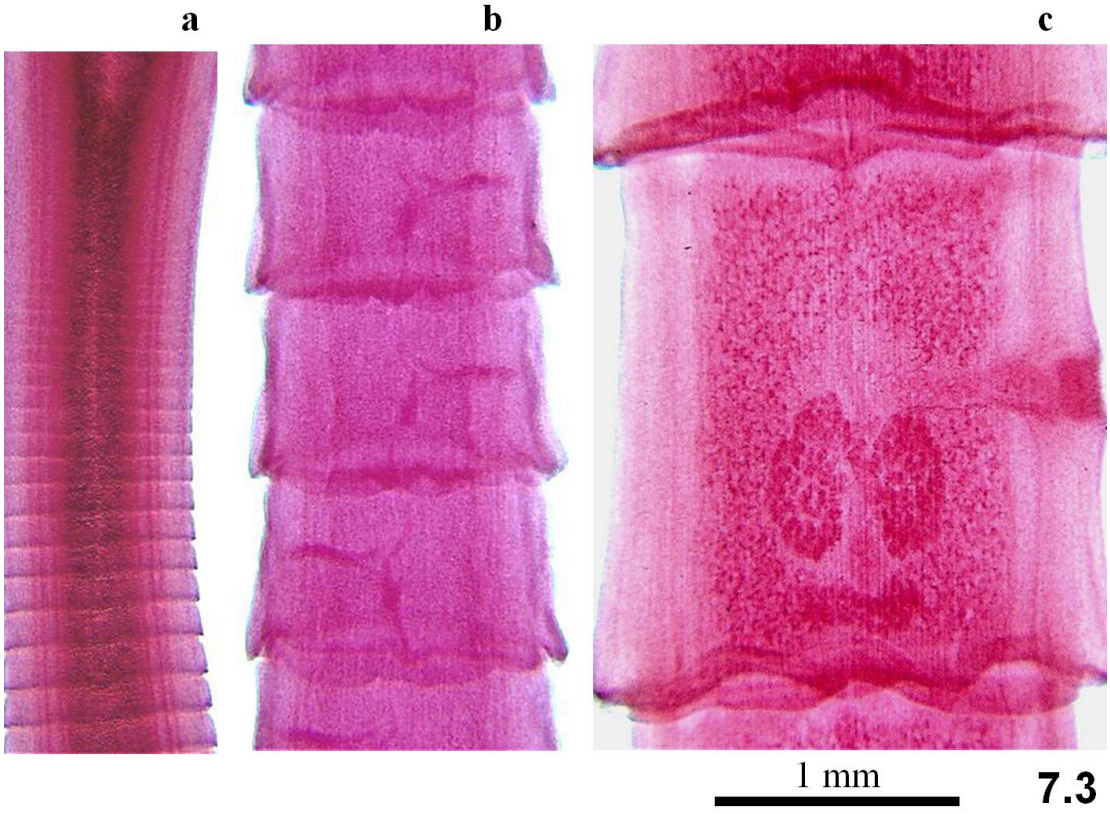
6.22 Eggs of *Schistosoma haematobium*



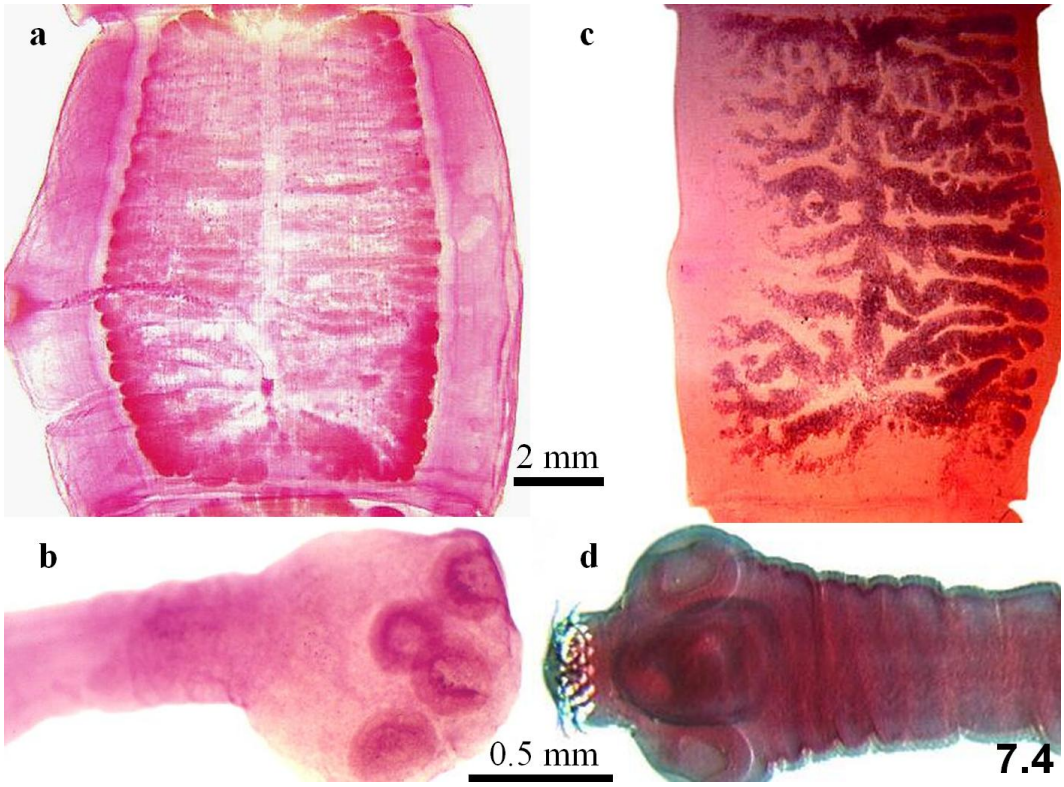
7.1 Adult of *Diphyllobothrium latum*.



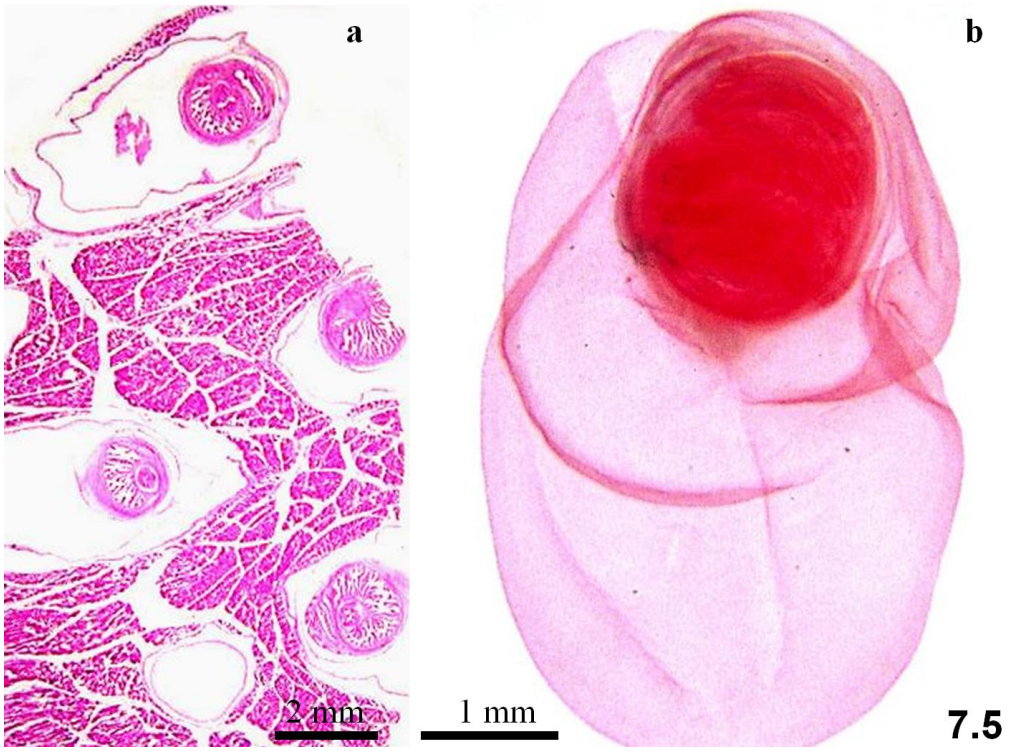
7.2 Scolex of *Taenia pisiformis*, a tapeworm of dogs



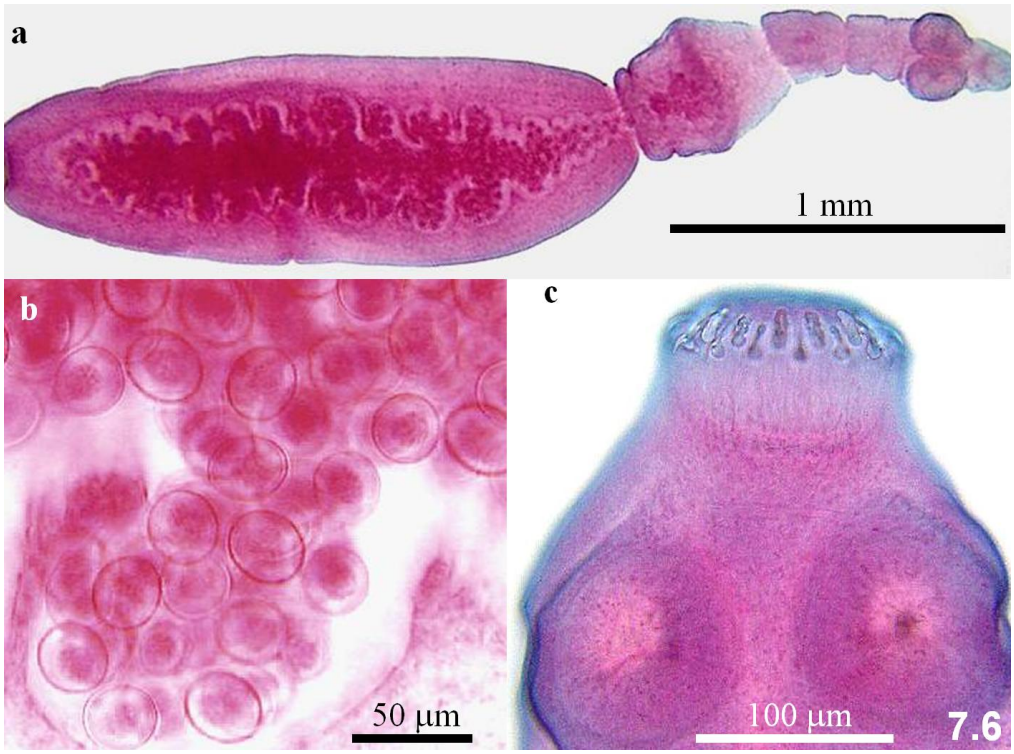
7.3 Strobila (body) of *Taenia pisiformis*.



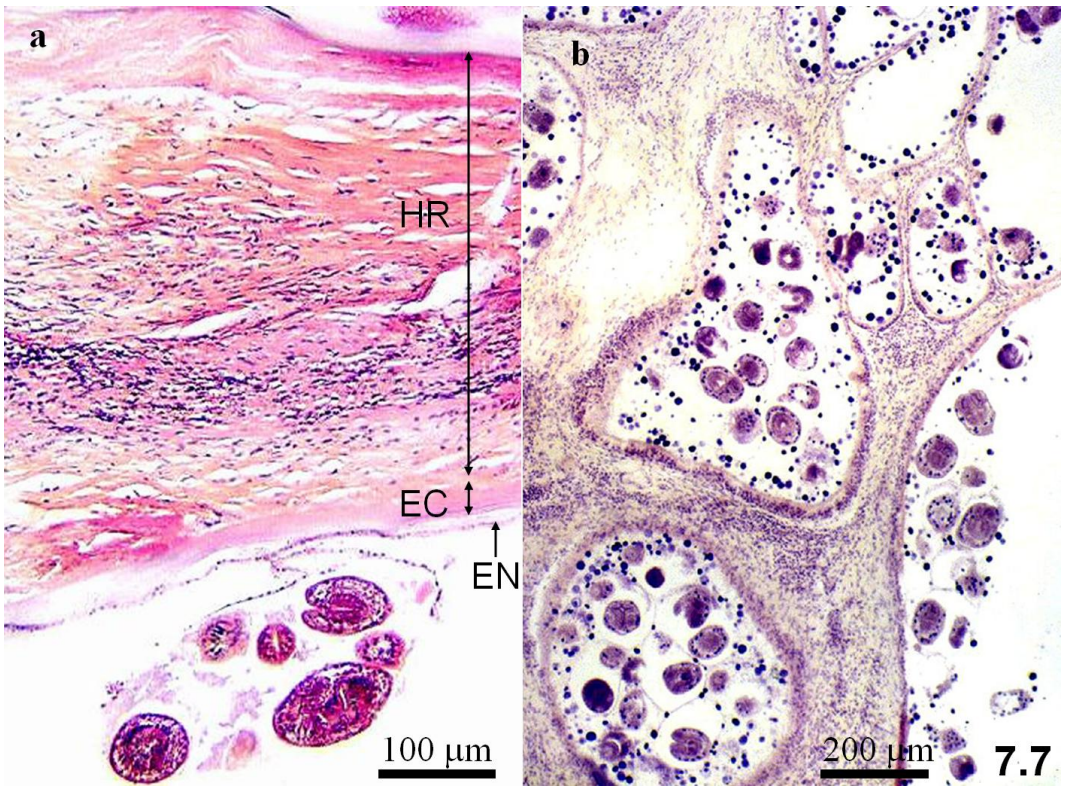
7.4 *Taenia saginata* vs. *T. solium*.



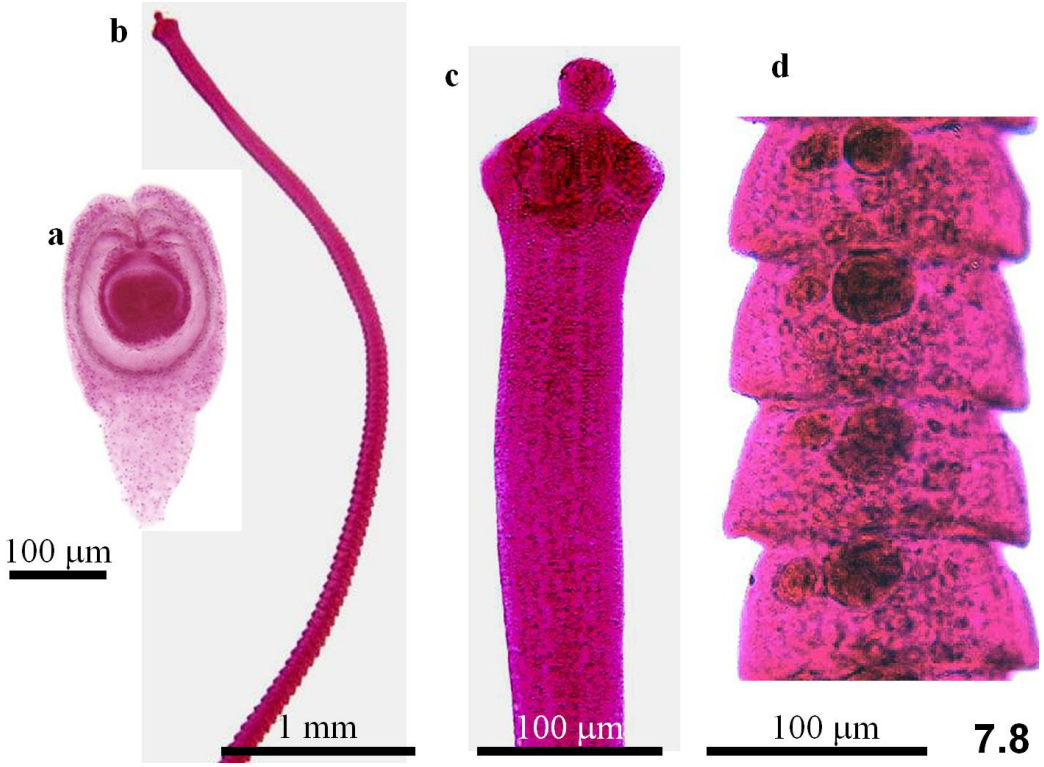
7.5 Cysticercus of *Taenia solium*.



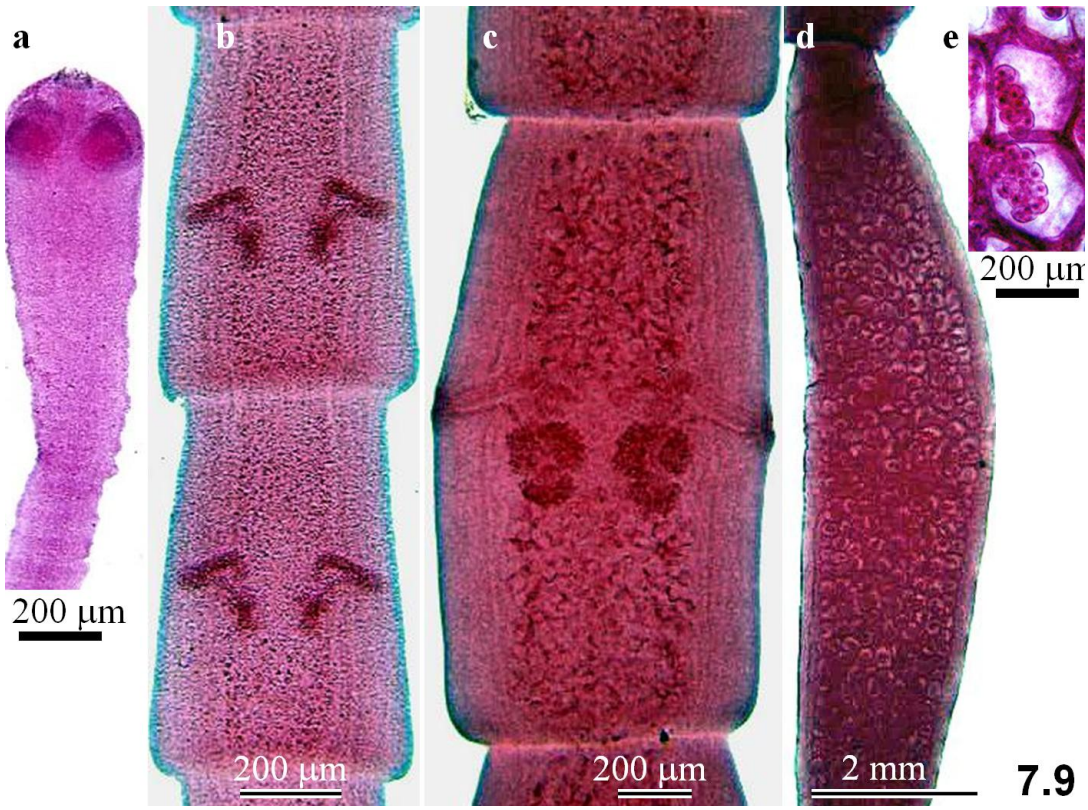
7.6 Adult of *Echinococcus granulosus*.



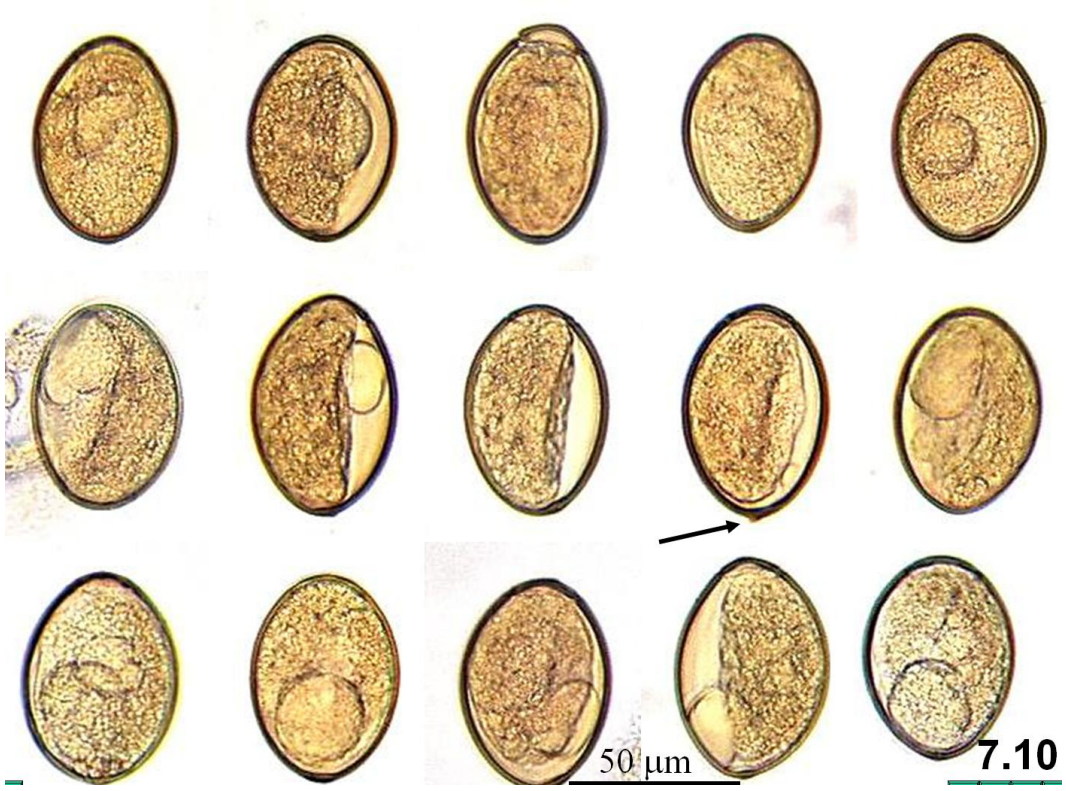
7.7 Hydatid cysts.



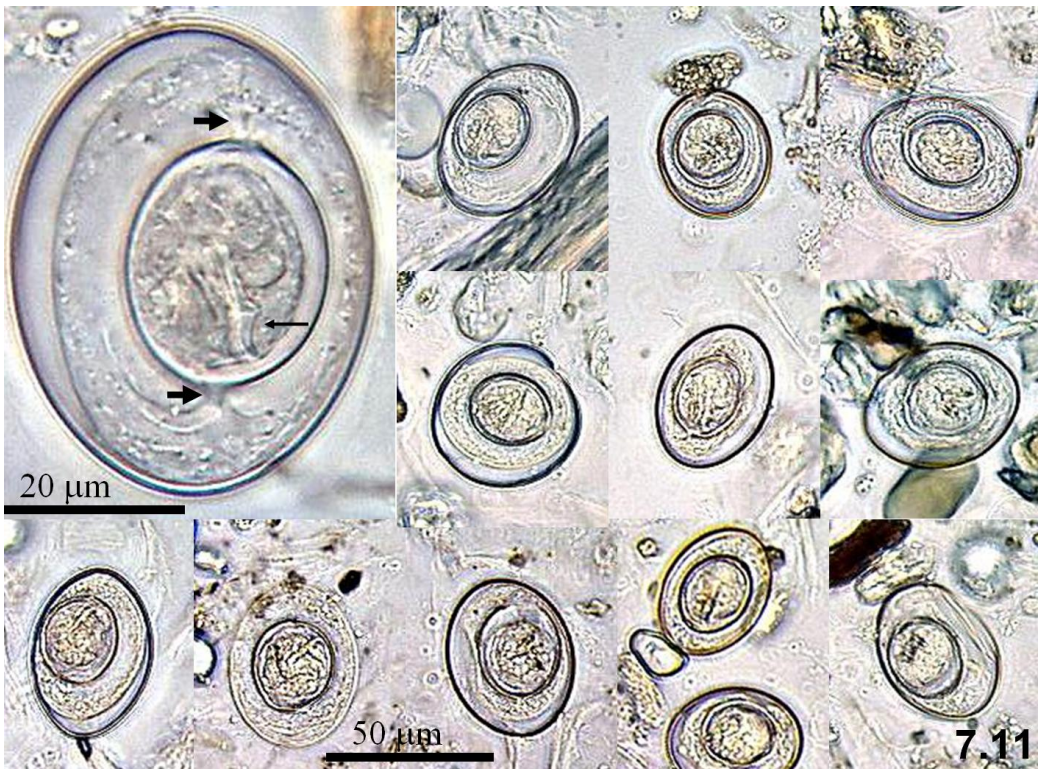
7.8 *Hymenolepis nana*.



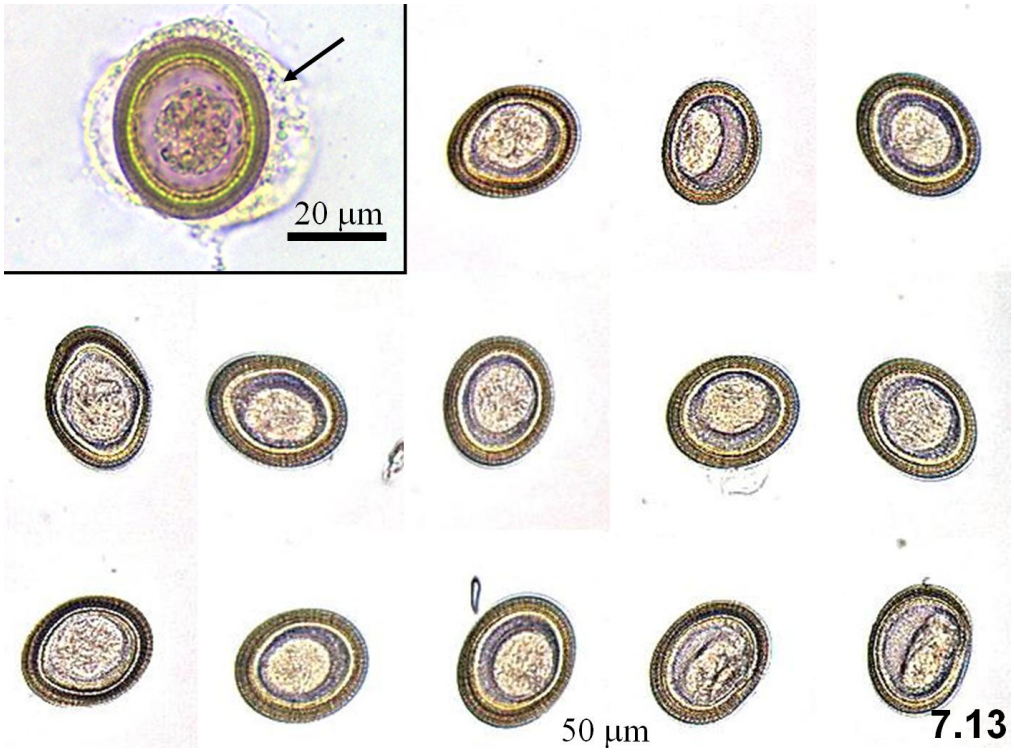
7.9 *Dipylidium caninum*.



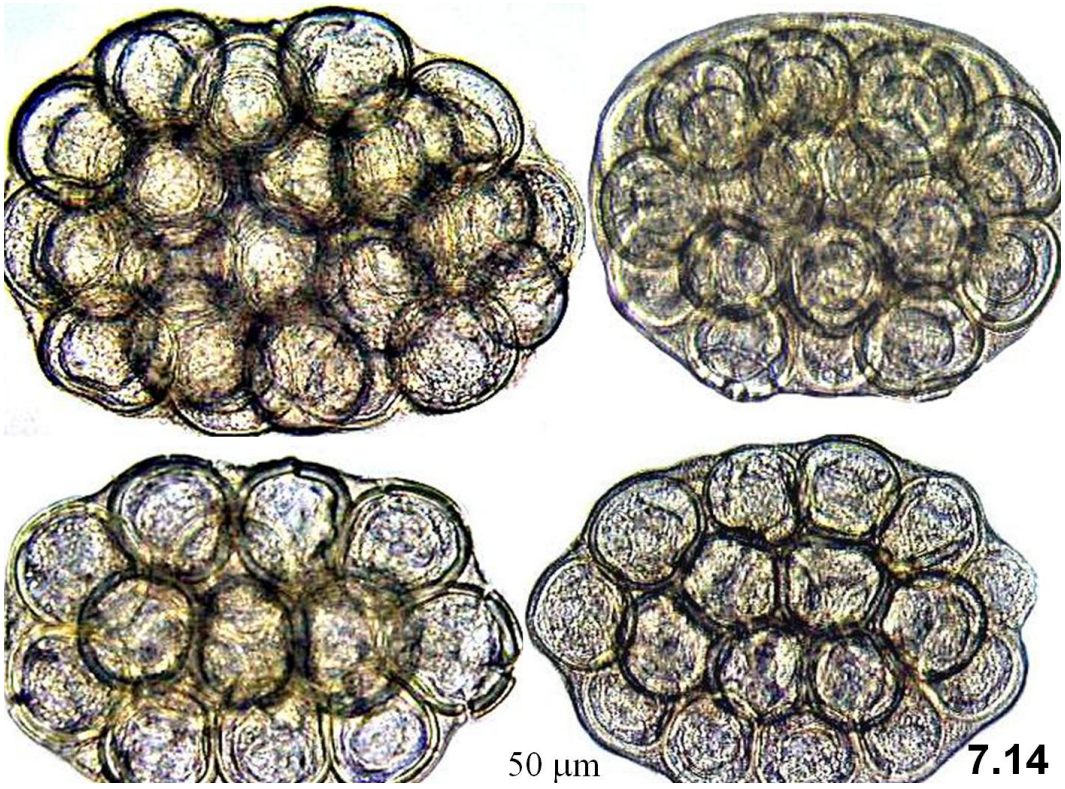
7.10 Eggs of *Diphyllobothrium latum*.



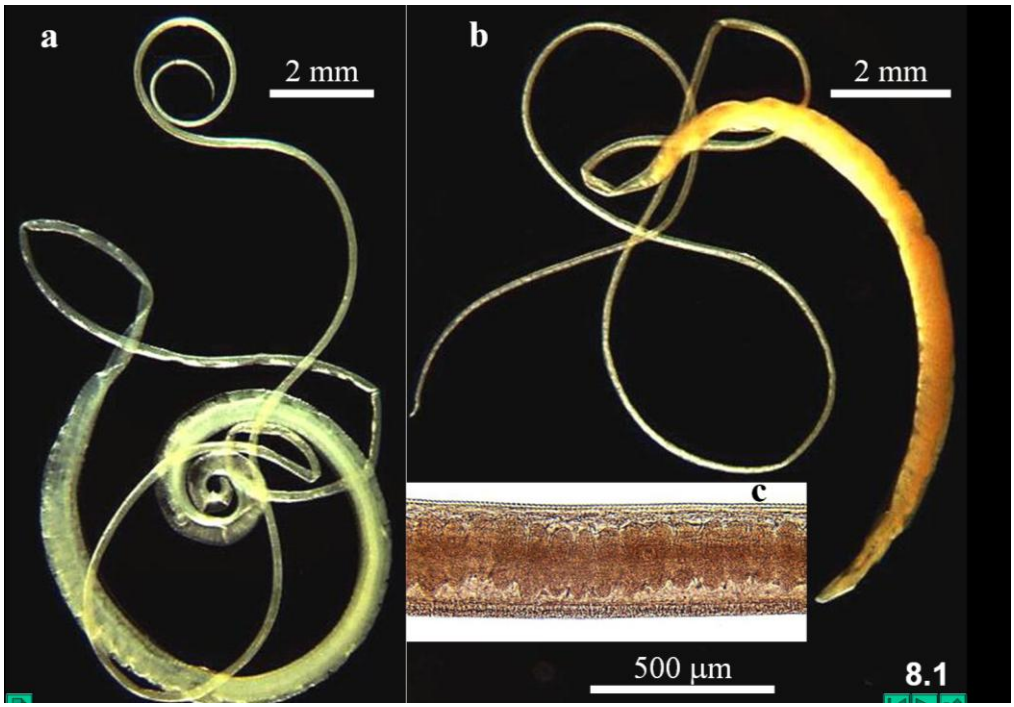
7.11 Eggs of *Hymenolepis nana*.



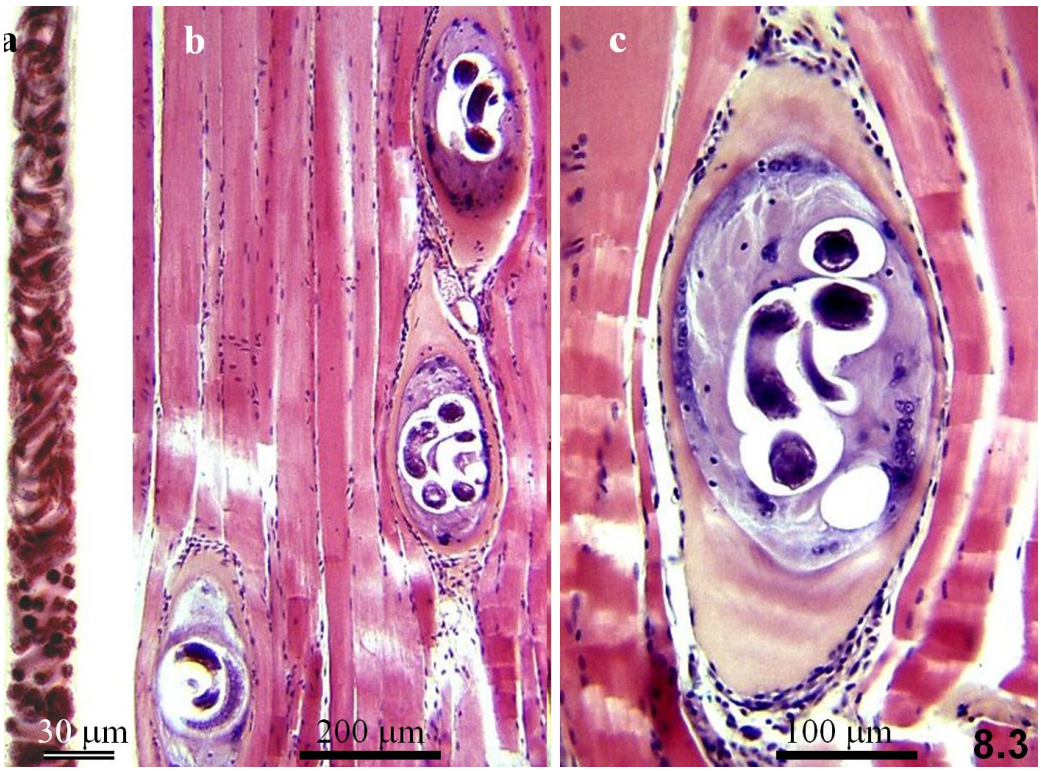
7.13 Taeniid eggs.



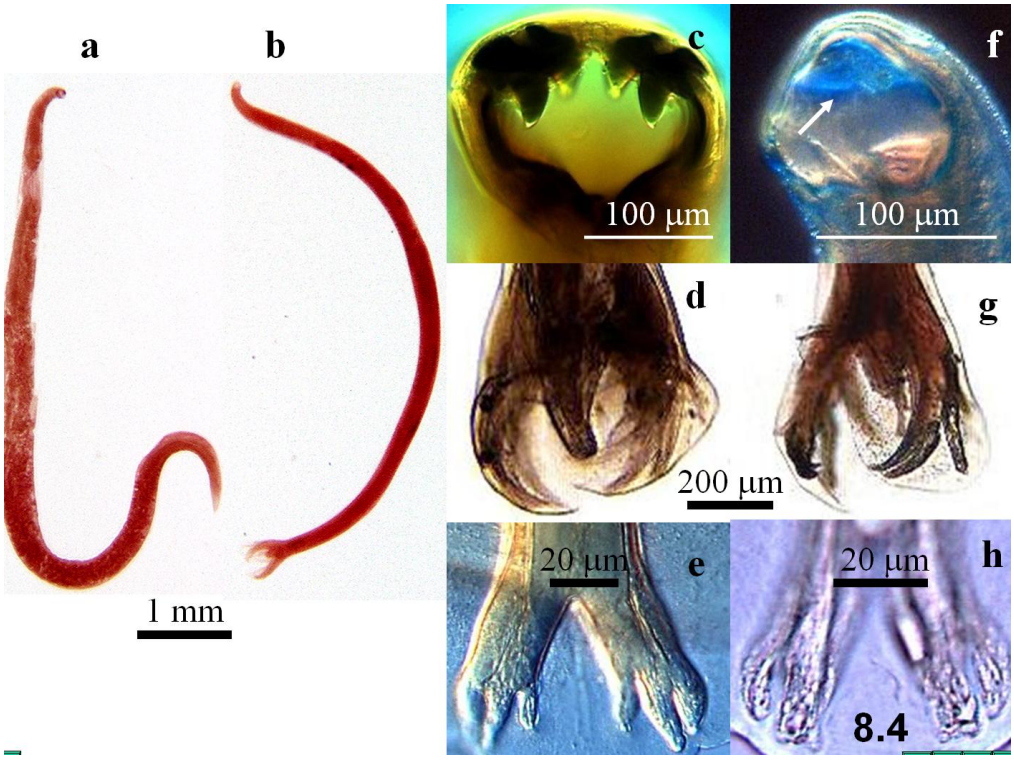
7.14 Egg capsules of *Dipylidium caninum*.



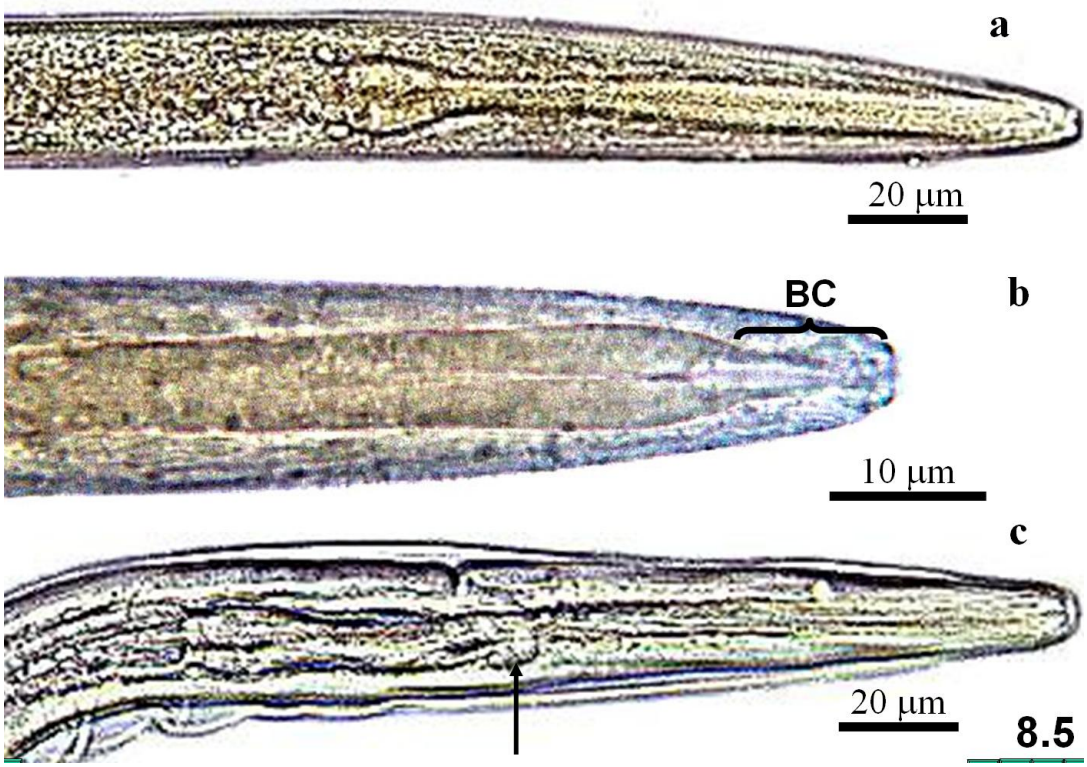
8.1 *Trichuris trichiura*.



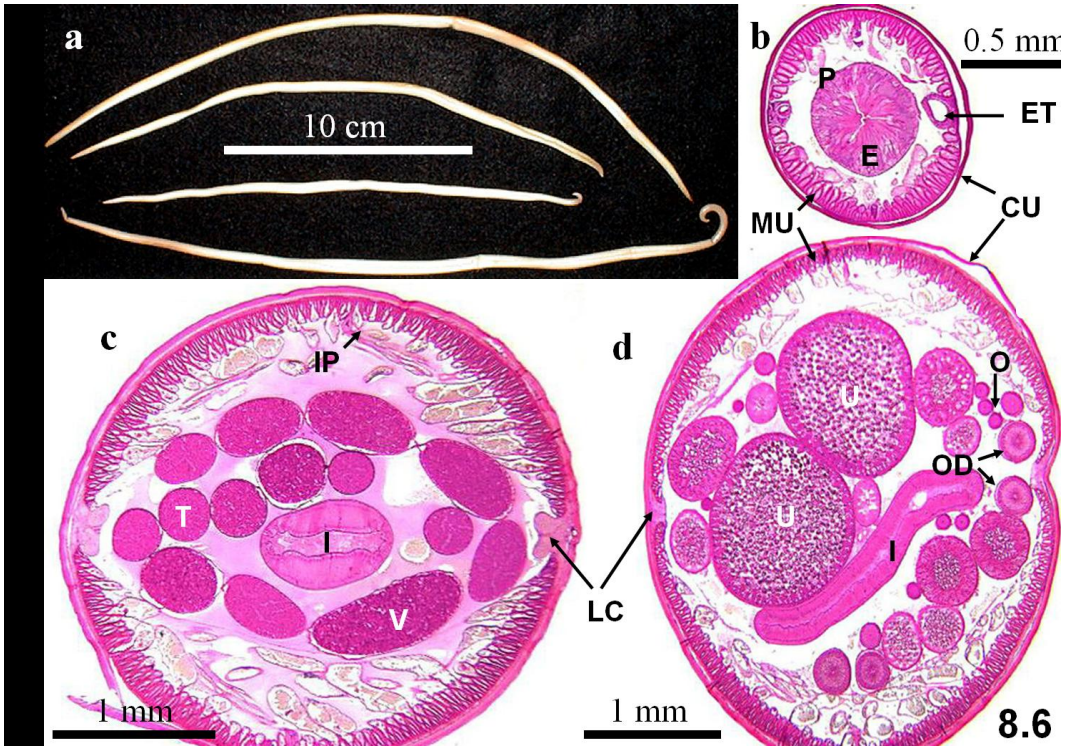
8.3 *Trichinella spiralis*.



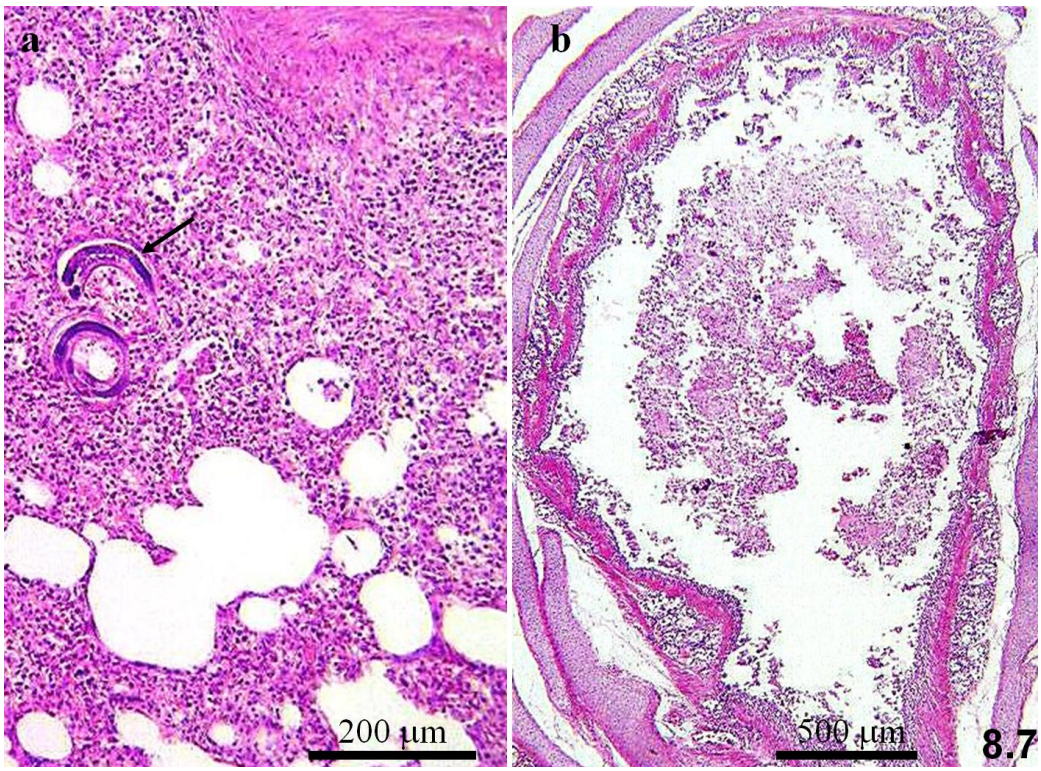
8.4 Hookworms.



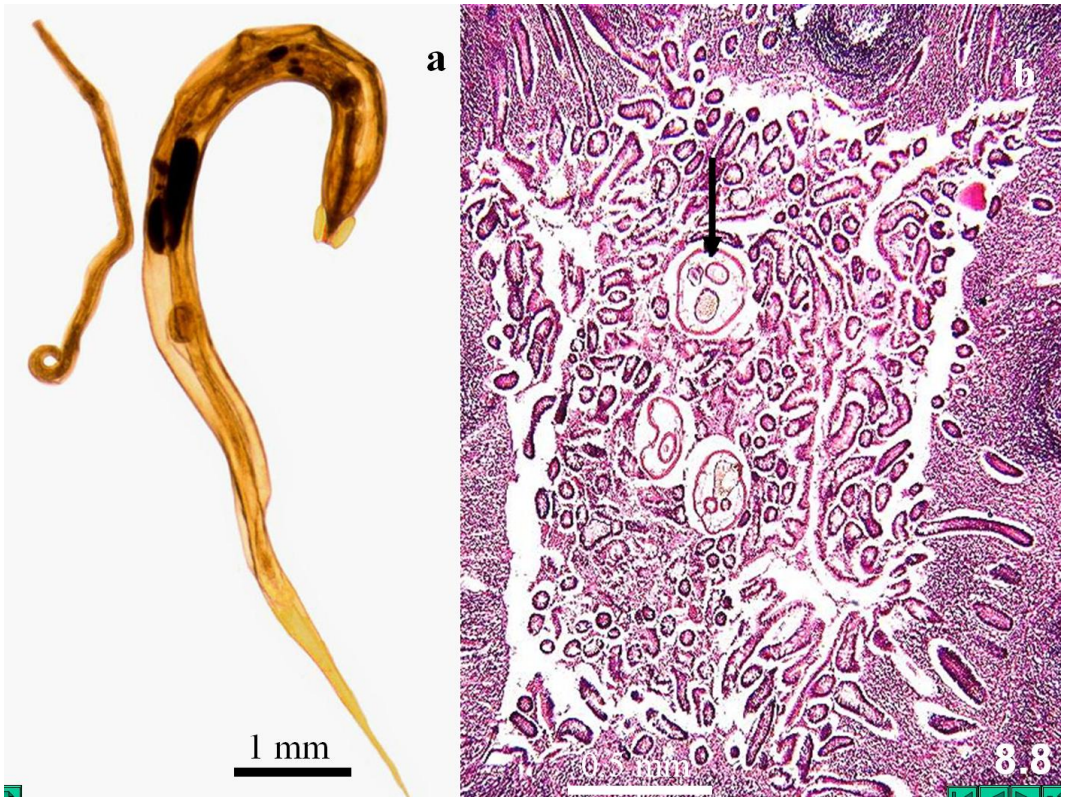
8.5 Rhabditiform vs. filariform larvae.



8.6 *Ascaris lumbricoides* adults.

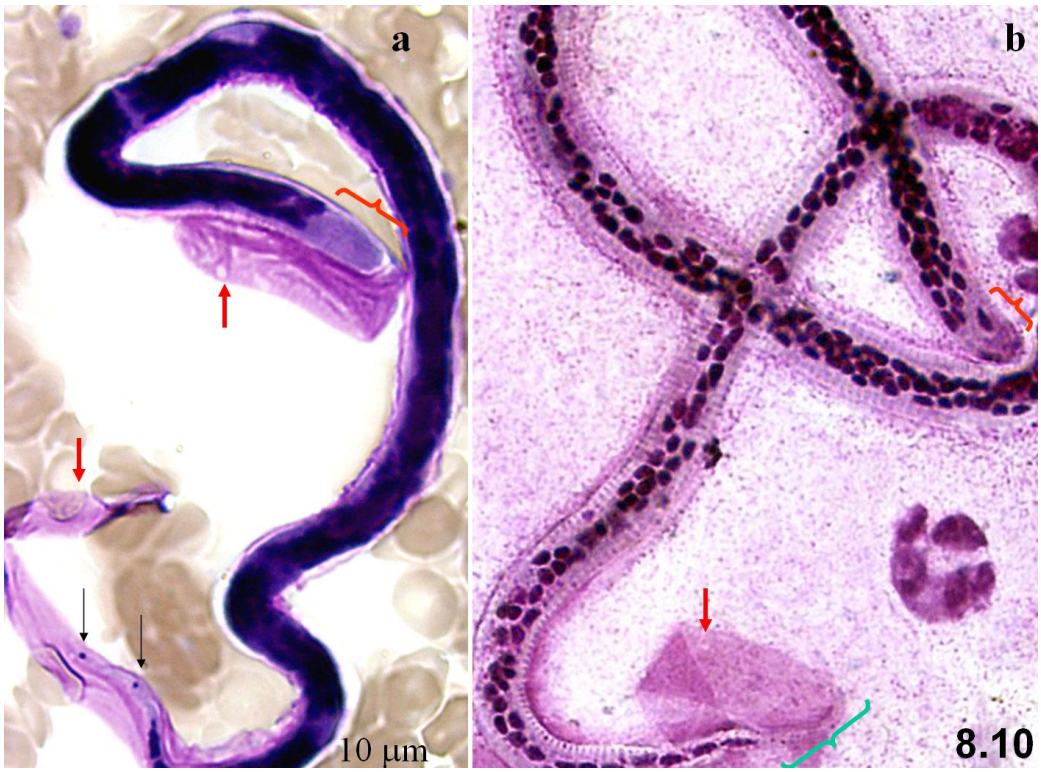


8.7 J_2s of *Ascaris lumbricoides* in lungs.

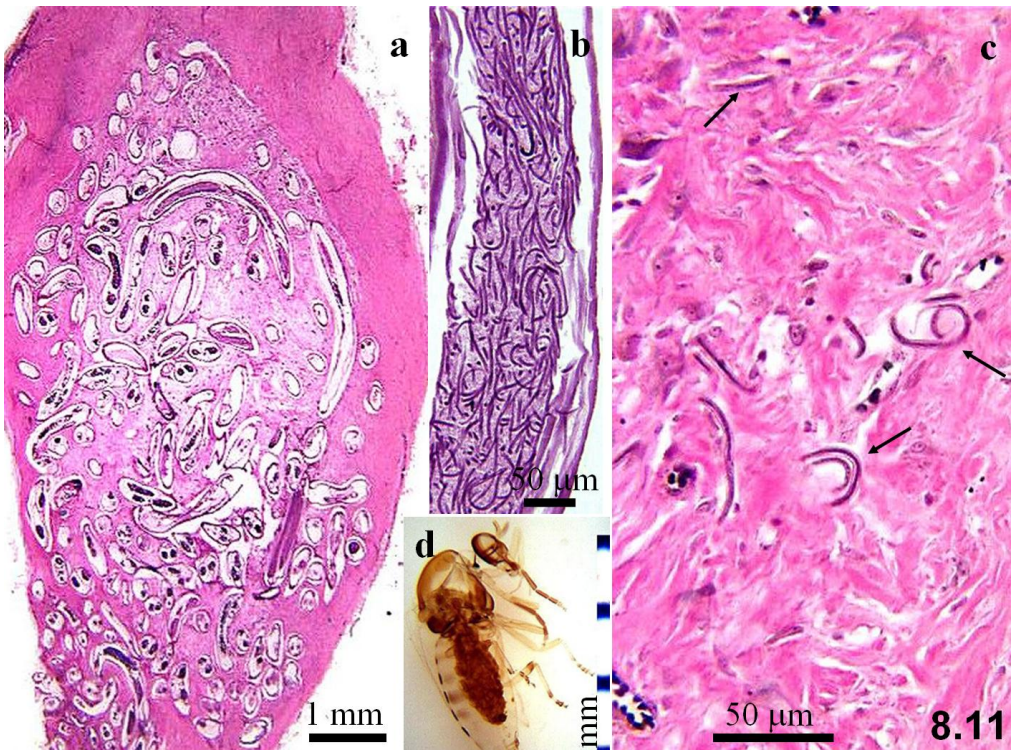


8.8 *Enterobius vermicularis.*

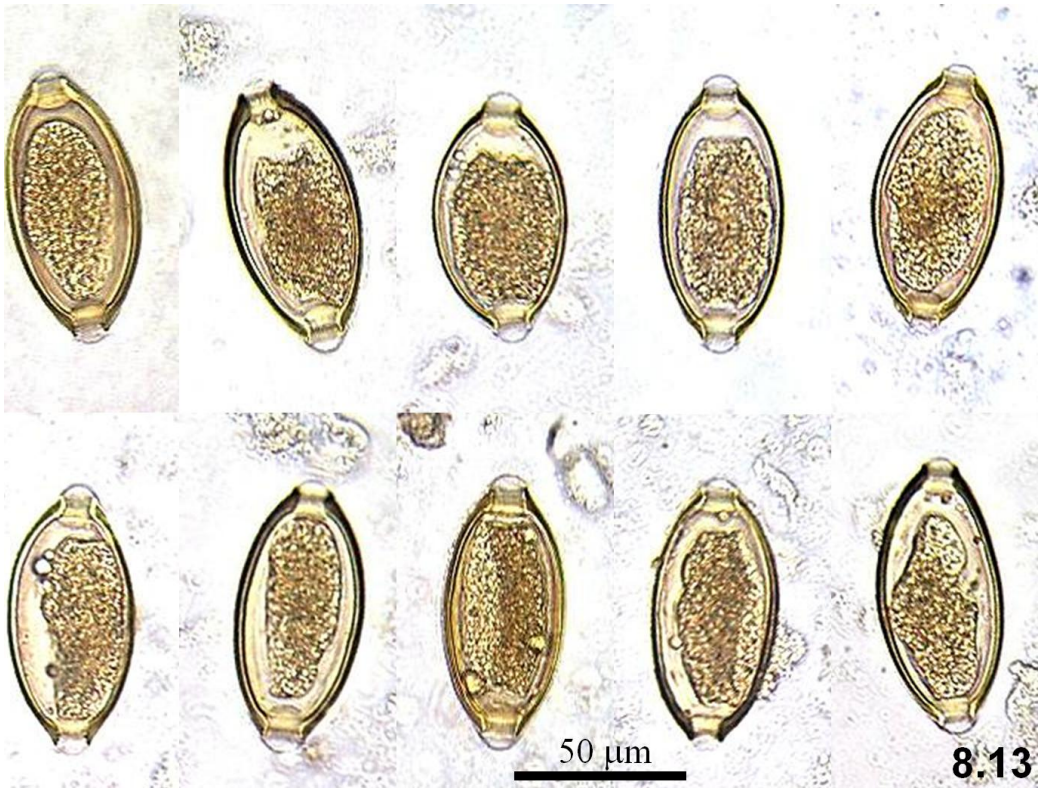
8.9 *Dirofilaria immitis.*



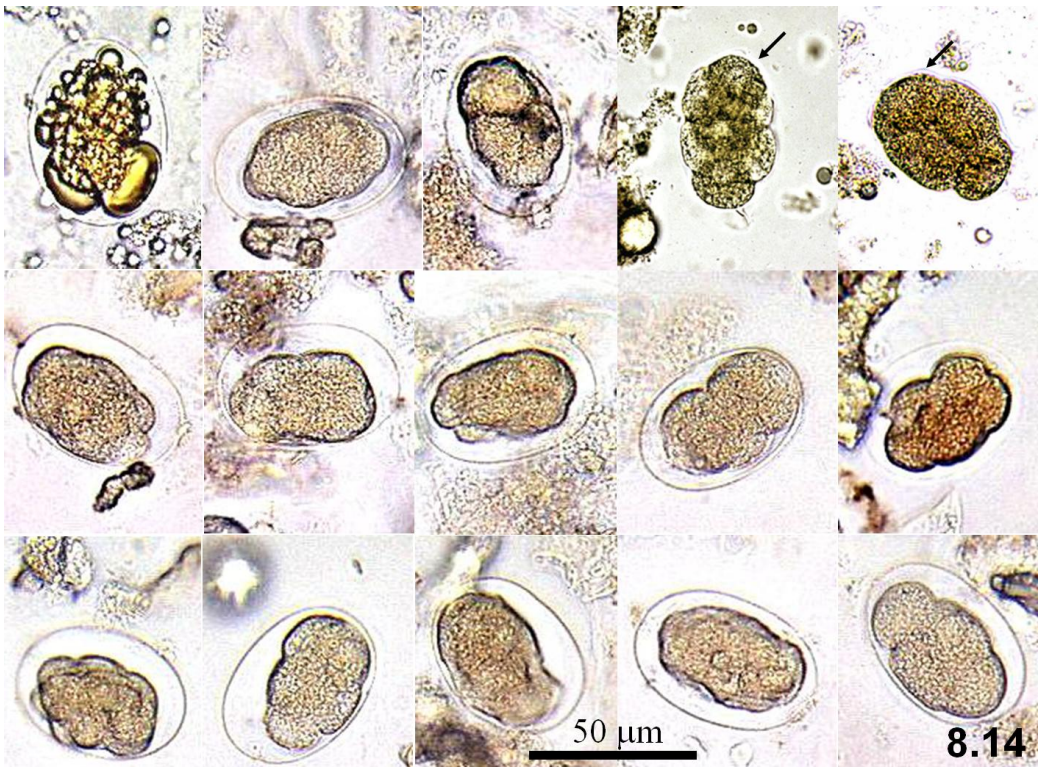
8.10 Microfilariae in peripheral blood smears, Geimsa stain.



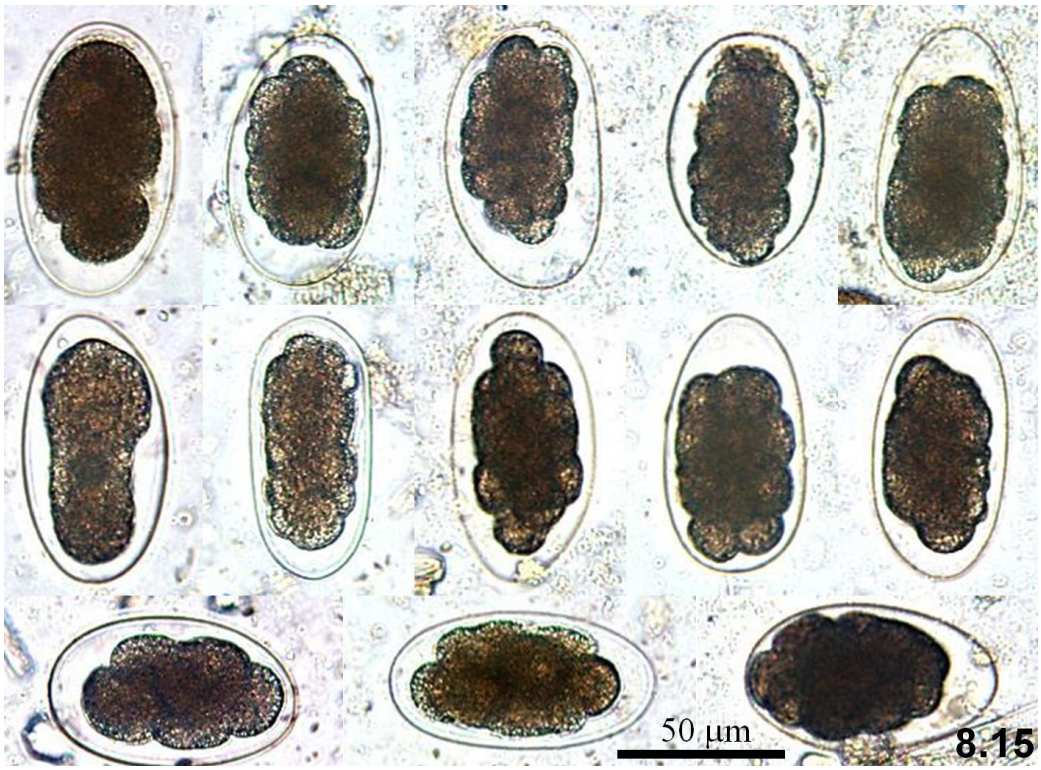
8.11 *Onchocerca volvulis.*



8.13 Eggs of *Trichuris trichiura.*

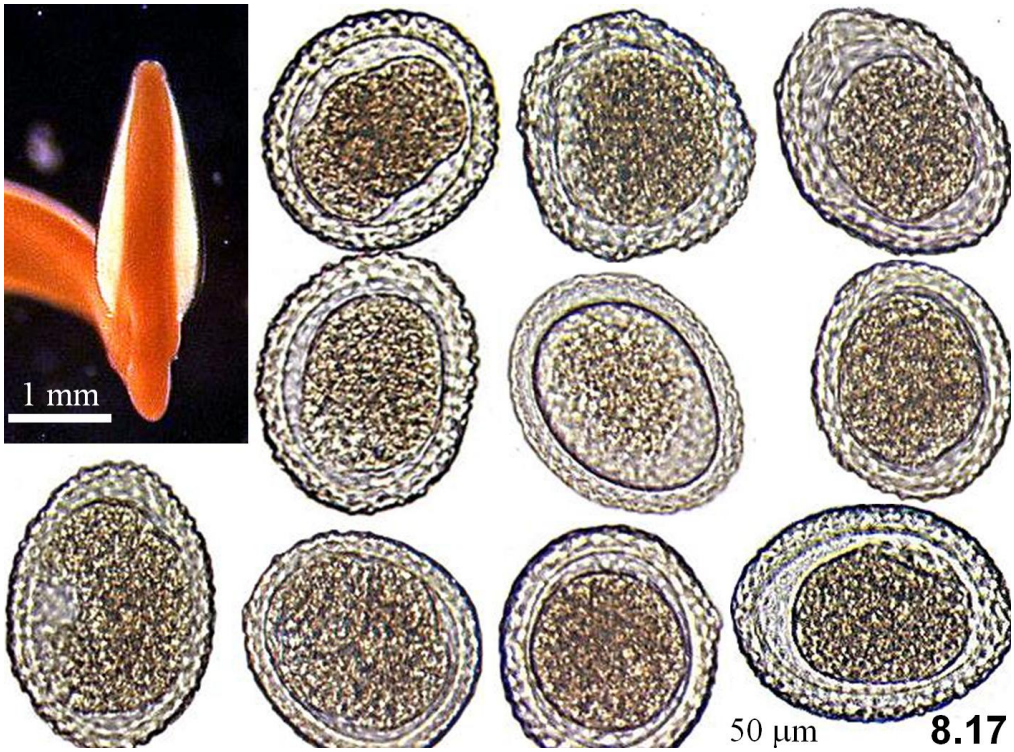


8.14 Hookworm eggs.



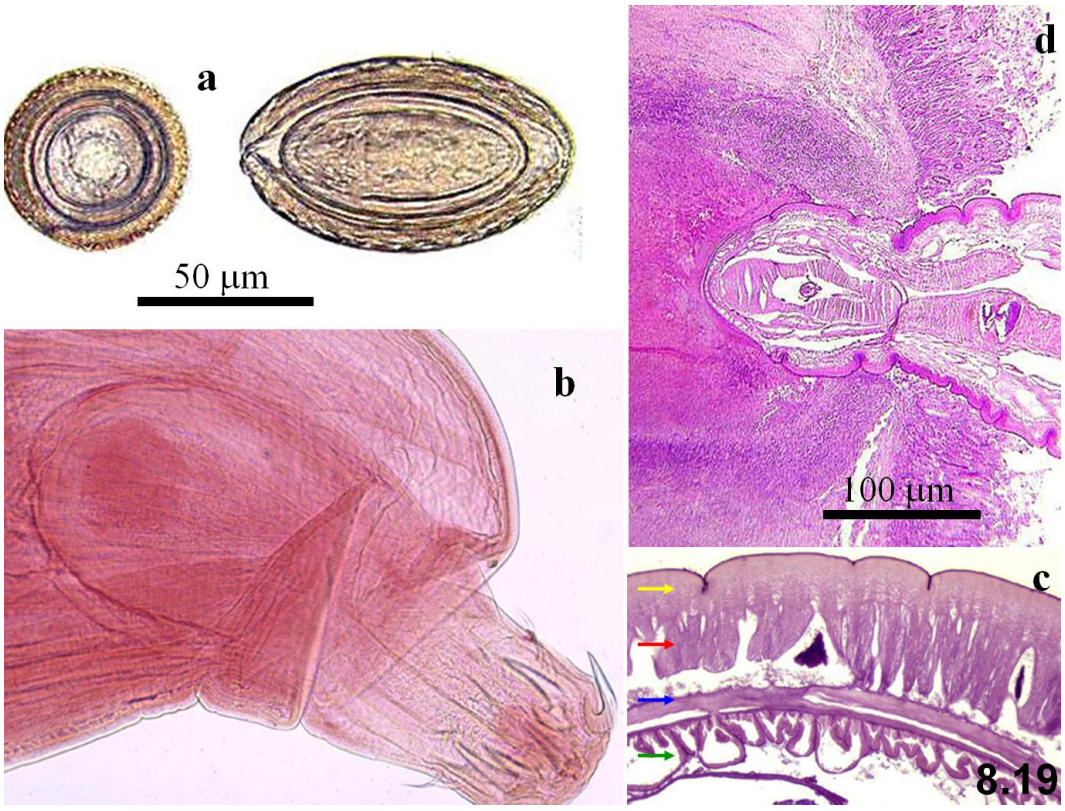
8.15 Eggs of *Trichostrongylus* spp.

8.16 Eggs of *Ascaris lumbricoides*.



8.17 Eggs of *Toxocara canis*.

8.18 Eggs of *Enterobius vermicularis*.



8.19 Acanthocephalans.

Attachment

1.1 Trypomastigotes of *Trypanosoma brucei rhodesiense*. Note the undulating membrane (U), anterior flagellum (F), and posterior location of the kinetoplast (K) relative to the nucleus (N). These would be indistinguishable from trypomastigotes of *T. b. gambiense*. The long slender form of trypomastigote is the dividing form (arrow), whereas the short, stumpy form (not shown) is infective for the intermediate host. *T. b. rhodesiense* is transmitted by the tsetse fly. *Glossina morsitans*, *G. pallidipes* and *G. tachinoides* in the savannah regions of east Africa. Numerous wild game animals serve as reservoir hosts. This parasite is highly pathogenic for humans, causing 100% mortality unless treated. *T. b. rhodesiense* typically does not cause the symptoms of African sleeping sickness seen with infections of *T. b. gambiense* simply because death occurs first.

Life cycle:

trypomastigotes in blood -- ingested by tsetse fly -- dividing epimastigotes in fly gut -- metacyclic trypomastigotes in fly saliva -- fly blood meal --trypomastigotes in blood

1.2 Trypomastigotes of *Trypanosoma brucei gambiense*, peripheral blood smear. Note the undulating membrane and posterior location of the kinetoplast relative to the nucleus. These would be indistinguishable from trypomastigotes of *T. b. rhodesiense*. The long slender form of trypomastigote is the dividing form (arrow), whereas the short, stumpy form (not shown) is infective for the intermediate host. *T. b. gambiense* is transmitted by the tsetse fly. *Glossina palpalis* and G. *tachinoides* in riverine regions of west and central Africa. There are no reservoir hosts. This species causes immune-mediated damage to capillaries in the brain (perivascular cuffing), resulting in African sleeping sickness and, if untreated, death.

Life cycle:

trypomastigotes in blood --ingested by tsetse fly-- dividing epimastigotes in fly gut -- metacyclic trypomastigotes in fly saliva -- fly blood meal --trypomastigotes in blood.

1.4. *Glossina sp.* (tsetse fly), the vector of African trypanosomiasis due to *Trypanosoma brucei brucei* in animals, and *T. b. gamhiense* and *T. b. rhodesiense* in humans. The wings, which arc extended outward on this mounted specimen, normally are folded on top of each other when resting. Although most flies are not susceptible to infection, both males and females can scrve as vectors.

1.5. Trypomastigotes of *Trypanosoma cruzi*, peripheral blood smear. Note the posterior location and large size of the kinetoplast (K), and the characteristic "C “- shape of several cells. The trypomastigote of this species is nondividing, and serves instead to disseminate the infection to tissue cells and to the vector. *T. cruzi* is transmitted in South and Central, and rarely North, America by several species of **kissing bugs**. Numerous wild and pcridomestic animals (e.g., dogs and cats) serve as reservoir hosts. This parasite causes Chagas' disease, which has both an acute stage, sometimes fatal in young children, and a chronic stage, which includes a gastrointestinal form (megaesophagus and megacolon. due to destruction of autonomic ganglia) and a **cardiac form** (cardiomegaly. ventricular aneurism, and arrhythmia, due to destruction of heart muscle and conducting cells).

Life cycle:

intracellular amastigotes (dividing) -- burst cell-- trypomastigotes in blood (nondividing) --re-invade cell or ingested by kissing bug – dividing epimastigotes in bug gut -- metacyclic trypomastigotes in bug feces -- rubbed into bite wound or eye -- intracellular amastigotes.

1.6. Amastigotes of *Trypanosama cruzi,* spleen smear. Note the absence of an undulating membrane or emergent flagellum The kinetoplast (K) is more darkly stained than the nucleus (N), and the parasite's cytoplasm is unstained. Large purple objects are host spleen cell nuclei (H). Amastigotes of *T. cruzi* would be indistinguishable from those of *Leishmania donovani*.

1.9. Kissing bug, *Triatoma gerstaeckeri* (Family Reduviidae, Subfamily Triatominae), a potential vector of *Trypanosoma cruzi.*
a. Entire bug viewed from above. **b.** Side view of bead, showing proboscis folded underneath. **c.** Bug feeding on a mouse. Note the extended proboscis. In addition to its large size, other features of this vector include a cone-shaped head with prominent eyes, dorsoventrally flattened body with wings folded in a concavity on top of the abdomen, margin of the abdomen with orange stripes, antennae in 4 segments, and a 3-segmented labial tube. Infectious metacyclic trypomastigotes of *T. cruzi* pass out in the bug's feces, and are rubbed into the bite wound or the eye, usually while the victim is sleeping. This specimen was collected in San Antonio.

1.10 Amastigotes of *Leishmania donovani,* liver smear. These are indistinguishable from amastigotes of *Trypanosoma cruzi*. Note the minute size, and absence of an emergent flagellum or undulating membrane The kinetoplast (K) is the dark rod-shaped inclusion near the round nucleus (N). Large purple structures are host liver cell nuclei (H). *L. donovani* is transmitted by *Phlebotomus* spp. sand flies in the old world (Africa, Asia, Middle East) and by *Lutzomyia* spp. in the new world (C and S America). Dogs and rodents are important reservoir hosts. Amastigotes of *L. tropica* or *L. mexicana* from skin lesions and *L. braziliensis* from mucocutaneous lesions would appear identical. Amastigotes of *L. donovani* infect macrophages of internal organs and cause **visceral leishmaniasis** (kala azar), characterized by hepatosplenomegaly. immunosuppression, anemia and death in 2-3 years if untreated.
Life cycle:
dividing amastigotes in macrophage -- burst macrophage -- free amastigotes -- re-phagocytosed or ingested by fly – dividing promastigotes in fly gut – injected into bite wound, phagocytosed -- dividing amastigotes in macrophage.

1.13 *Lutzomyia diabolica* (sand fly), a potential vector of *Leishmania mexicana* in the New World. In addition to its small size, the sand fly can be recognized by its hairy wings and body, and the 60-degree angle at which the resting wings are held from the body. This insect possesses cutting rather than piercing mouthparts. The specimen pictured here is a nonbiting male.

2.3 Trophozoites of *Giardia lamblia,* stool smear. Note the pyriform shape, two nuclei, and median bodies (M). The trophozoite stage normally would be found in loose stools. Flagella (which number 8) are not visible in these photographs, although axonemes (A) can be seen in the cytoplasm of some cells as dark lines. While many infected people are asymptomatic, this flagellate can cause protracted diarrhea.
Life cycle:
trophozoite in small intestine -- cyst in stools – ingested -- trophozoite in small intestine

2.4 Cysts of *Giardia lamblia*, stool smear. Note the oval shape, smooth cyst wall, axonemes (A), and four nuclei, usually not all of which are visible in one focal plane. The cytoplasm often is retracted from the cyst wall in fixed specimens, leaving a clear space. The cyst normally would be found in formed stools, and is the infective stage. Although ingestion of cysts from human feces accounts for most cases of giardiasis, there are numerous reservoir hosts, e g., beavers, and infections have been acquired by persons who drank what appeared to be pristine stream water or melted snow contaminated with cysts of animal origin.

3.1 Trophozoites of *Entamoeba hisiolytica,* stool smear. A nucleus with a central endosome and a fine peripheral ring of chromatin distinguishes this parasite from *E. coli*. Trophozoites would be found mainly in loose stools. Charcot-Leyden crystals, representing the crystallized contents of granules from eosinophil leukocytes (bottom right frame), may also be found in a fecal smear. Infection occurs when the cyst is ingested in fecally contaminated food or water. This parasite is

capable of causing **ulcerative colitis**, resulting in severe dysentery, as well as extraintestinal amebiasis, including fatal brain infections.
Life cycle:
trophozoite in large intestine -- cyst in feces -- ingested -- trophozoite in large intestine

3.2 Cysts of *Entamoeba histolytica*, stool smear. Note the presence of four or fewer nuclei. Although all four nuclei may not be visible in the same plane, they can be counted by carefully adjusting the fine focus control (NI-N4. second row). Chromatoidal bars (C), when present in immature cysts, usually have blunt ends, versus splintered ends in cysts of *E. coli*. Cysts would be found mainly in formed stools, and are the infective stage.

3.12. Trophozoites of *Balantidium coli*, stool smear. Note the oval shape, large curved macronucleus (M), cytostome (C), peristomal cilia (P), and food vacuoles (V). The only pathogenic ciliate parasite of humans, *B. coli* can cause **intestinal lesions** that result in a disease similar to amoebic dysentery, although this is rare. Trophozoites would be found mainly in loose stools. Infections are established when the infective cyst is ingested with fecally-contaminated food or water. Because this parasite also infects pigs, human infections with *B. coli* are especially common in pig-rearing areas.
Life cycle:
trophozoite in large intestine -- cyst in feces -- ingested -- trophozoite in large intestine

3.13. Cysts of *Balantidium coli*. stool smear. Note the smooth round shape, large curved macronucleus, and cyst wall (W), which has separated from the cell in some specimens. Cysts would be found mainly in formed stools, and are the infective stage.

3.14. Intestinal balantidiasis and amebiasis, histological sections. **a.** Lesion (area under bracket) in colon caused by *Balantidium coli*. MM, muscularis mucosae. In addition to trophozoites in the lesion itself (green arrow), there are also trophozoites in lymphatic vessels in the submucosa (red arrows), demonstrating how the infection may spread to other sites. *B. coli* only rarely causes this pathology in humans. **b.** Higher magnification of trophozoites in lesion. Note the macronucleus in some specimens (arrow). **c.** Lesion caused by *Entamoeba histolytica* (area under bracket). MM, muscularis mucosae. **d.** Trophozoites at base of the lesion, some of which contain darkly stained, phagocytosed red blood cells. Arrow points to the nucleus of a trophozoite.

4.5 Extraintestinal cycle of *Toxoplasma gondii* in the mouse. When an intermediate host, which could be almost any mammal or bird, ingests *oocysts* (containing sporozoites) from cat feces or *zoitocysts* (containing bradyzoites) in the tissues of another intermediate host, repeated, rapid cycles of schizogony occur, releasing merozoites (tachyzoites). which infect cells in a wide variety of tissues. **a.** Histological section of liver, showing an infected cell in the center (arrow) and a necrotic area above. **B**. Smear of peritoneal fluid, showing tachyzoites. This stage of the infection usually produces mild, if any, symptoms in healthy persons, but can be lethal in immunocompromised individuals, (acquired toxoplasmosis), and can cause severe damage to the fetus if transmitted transplacentally (congenital toxoplasmosis).

4.6 Histological sections of zoitocysts of *Toxoplasma gondii* in the brain of a mouse. The proliferation of tachyzoites elicits an immune response that slows down schizogony, resulting in accumulations of slowly-dividing merozoites, called bradyzoites, within an infected cell. These intracellular accumulations, known as zoitocysts, may persist for years in nervous tissue. Ingestion of zoitocysts will initiate the **enteroepithelial cycle** in cats, and the **extraintestinal cycle** in birds and mammals.
Extraintestinal life cycle:

Infective oocyst or zoitocyst -- ingested -- sporozoites or merozoites in intestinal lumen -- invade cells -- intracellular trophozoite – mitosis – schizont – cytokinesis, release from cell -- tachyzoites – reinvade cell – intracellular trophozoite – mitosis –schizont – host immune response -- zoitocyst containing bradyzoites

4.7 Enteroepithelial cycle of *Toxoplasma gondii* in the epithelial lining of the cat intestine, histological section. **a.** Intestinal epithelial cells infected with oocysts (O) and schizonts (S) containing merozoites. **b.** Oocysts in infected epithelial cells and free in lumen. Ingestion of oocysts (containing sporozoites) or intermediate hosts infects with tachyzoites or zoitocysts (containing bradyzoites) results in schizogony and sexual reproduction, leading to the production of oocysts. This sexual cycle occurs only in the cat, whereas in intermediate hosts, only asexual schizogony occurs.

Enteroepithelial life cycle:

Infective oocyst or zoitocyst -- ingested -- sporozoites or merozoites in intestinal lumen -- invade cells -- intracellular trophozoite – mitosis – schizont – cytokinesis, release from cell -- merozoite in lumen -- reinvade cell -- gametogenesis – differentiation – gametes – fertilization, cyst wall formation, release from cell – oocyst in feces – sporulation (2 to 3 days) -- infective oocyst

5.1 Mouthparts of *Anopheles* sp. mosquitos. *Plasmodium* spp. infections are transmitted to humans by the bite of the female *Anopheles* mosquito (top). Males (bottom) are physiologically capable of supporting parasite development, but since they do not feed on blood, they cannot transmit infection. Males are easily recognized by the plumose hairs on their antennae. This genus of mosquito can be identified by its palps, which are almost as long as the proboscis, and the 45-degree angle at which it sits when resting or feeding.

5.2 Oocysts of *Plasmodium* sp. on the surface of an *Anopheles* sp. mosquito midgut. **Gametocytes** taken up in a blood meal develop into gametes in the midgut, fertilization occurs, and the zygote (ookinete) penetrates the midgut wall to develop into an oocyst, which produces sporozoites. The sporozoites then migrate to the salivary gland.

Life Cycle (mosquito stages in orange):

Sporosoite in salivary gland – injected during feeding sporozoite in blood – invades hepatocyte -- trophozoite in hepatocyte – mitotic division – schizont in hepatocyte -- hepatocyte bursts -- merozoites in blood – invade RBC -- trophozoite in RBC -- mitotic division -- schizont in RBC – RBC bursts -- merozoites in blood – reinvade RBC -- schizont or gametocyte in RBC – gametocytes ingested by mosquito – gametes in midgut – fertilization – zygote – elongation – ookinete -- penetrates midgut epithelium, meiotic and mitotic division – oocyst containing sporozoites – sporozoite – migration in hemolymph – sporozoites in salivary gland.

5.3. Sporozoites of *Plasmodium vivax* in a squash of an oocyst from an infected *Anopheles* mosquito. Sporozoites develop in oocysts on the wall of the stomach, and then migrate in the hemolymph to the salivary glands. Several thousand may be injected into the host by one mosquito during feeding. Upon reaching the liver, each will penetrate into an hepatocyte and develop into an *exoerythrocytic schizont*.

5.4. Exoerythrocytic schizonts of *Plasmodium* sp. in liver cells. Once introduced into the bloodstream, *sporozoites* of *Plasmodium* penetrate hepatocytes within 30 minutes. Each undergoes schizogony, to produce an exoerythrocytic (EE) schizont, which is a single multinucleate cell. Cytokinesis occurs, and thousands of merozoites burst from the hepatocyte within 1-2 weeks post infection, depending upon the species (e.g. 40,000 merozoites in 5-7 days in the case of *P. falciparum*). These merozoites then infect erythrocytes. During infections with *P. vivax* and *P. ovale*, some EE schizonts develop into dormant hypnozoites, and upon becoming active may cause a relapse of the disease years after a supposed cure.

5.5. Trophozoites of *Plasmodium vivax*. These can be identified as *P. vivax* by the following features: enlarged, decolorized infected erythrocytes; prominent Schuffner's dots; and the ameboid shape of the trophozoite. Decolorization is not apparent here. Hemozoin granules may be relatively difficult to identify in this species. Malaria caused by *P. vivax* usually is not life-threatening.

5.6. Erythrocytic schizonts of *Plasmodium vivax*. When merozoites invade host erythrocytes, most undergo schizogony to produce 12 to 24 merozoites (average of 16) within approximately 48 hours. These burst out of the cell and immediately infect new cells. Because the infection becomes synchronous in the host, large numbers of infected erythrocytes burst more or less simultaneously, causing a rapid rise in body temperature at 48-hour intervals.

5.7. Young signet ring stage trophozoites of *Plasmodium falciparum*. This organism kills more people annually than all other parasites combined. Because of its pathogenicity, a failure to recognize *P. falciparum* infection in a smear like the one shown here may have fatal consequences. Diagnostic features are: high parasitemia; presence of only signet ring trophozoites; applique forms, double chromatin dots, and multiple infections in some cells; and absence of Schuffner's dots. Soon after this stage, infected cells disappear from the circulation by adhering to endothelial cells of blood vessels in the tissues, making diagnosis difficult and potentially leading to **cerebral malaria**. Schizogony results in new infected erythrocytes, which reappear in the peripheral blood at 48-hour intervals. Some individuals who are apparently "cured" of infection may develop symptoms years later due to resurgence of previously low, nondetectable levels of parasitemia (a phenomenon called recrudescence, not to be confused with relapse).

5.8. Erythrocytic schizonts of *Plasmodium falciparum.* This stage usually is not observed in peripheral blood, except in very heavy infections. Each schuzont produces from 6 to 32 merozoites, with an average of 20 to 24, every 48 hours. Hemozoin pigment is clumped in the center of the infected RBC. Note that the merozoites are very small, and that the schizont usually does not fill up the RBC.

5.9. Gametocytes of *Plasmodium falciparum*. Some merozoites penetrate erythrocytes and differentiate into gametocytes instead of undergoing schizogony. Although the gametocytes of all four human-infecting species can be distinguished, those of *P. falciparum* have a unique appearance, and therefore are valuable in diagnosis. Macrogametocytes of this species are elongate, and have a nucleus less than one-half the length of the cell. Microgametocytes may be shorter and more blunt-ended, have a lighter blue cytoplasm, and have a nucleus that is greater than one-half the length of the cell. Gametocytes do not produce gametes until they reach the midgut of a mosquito. The gametes fuse to produce a zygote that elongates into an ookinete. The ookinete penetrates the midgut wall and develops into an *oocyst*, in which *sporozoites* are produced.

5.10 Trophozoites of *Plasmodium malariae*. Occasionally trophozoites form a band shape, stretching across the red blood cell, as shown in these photographs. The cytoplasm of the parasite stains more darkly than in *P. vivax* and there are no Schuffner's dots. Hemozoin granules are much more conspicuous in this species than in the other three. Unlike the case with *P. vivax*, infected erythrocytes are not enlarged.

5.11. Erythrocytic schizonts of *Plasmodium malariae*. From 6 to 12 merozoites (average of 8) form in each infected cell at 72-hour intervals. The merozoites are often, but not always, arranged in a rosette around the periphery, with the hemozoin granules at the center.

5.12 Growing trophozoites of *Plasmodium ovale*. This species has the lowest prevalence among malarial parasites, occurring mainly in tropical Africa. Although sometimes difficult to distinguish from *P. vivax*, up to 20-60% of infected cells show oval distortion (versus around 5% in *P. virax*), as depicted in this plate. Schuffner's dots and hemozoin granules are prominent, as is the large chromatin mass. Also, infected cells are enlarged (although usually not as enlarged as with *P. vivax*), and some of the infected cells have fimbriated (ragged) edges.

5.13 Erythrocytic schizonts of *Plasmodium ovale*. This stage shares many diagnostic features with the trophozoite stage: enlarged, ovally-distorted host cells, and prominent Schuffner's dots. From 6 to 14 merozoites (average = 8) are produced by each schizont in 48 hr. The schizonts shown in these photographs are at a relatively early stage of development.

5.15. Histological section of brain from a case of cerebral malaria. In *Plasmodium falciparum* infections, the surfaces of infected red blood cells express parasite proteins that cause them to adhere to the endothelial lining of blood vessels in the organs. Note the brown (hemozoin)-tinted blood vessels in the low magnification view on the left, which at higher magnification on right are seen to be clogged with infected red blood cells, visible by their black deposits of hemozoin pigment in the parasite cytoplasm. Cerebral malaria can be rapidly lethal, especially in small children and nonimmune adults, and is a major reason that *P. falciparum* is one of the greatest killers of humans.

6.1. *Echinostoma revolutum*. **a.** Adult worm. Echinostomes are intestinal parasites with a worldwide distribution. Infection results from ingesting metacercariae, usually in uncooked molluscs. Mammals and birds serve as reservoir hosts. Heavy infections may cause diarrhea. Structures visible are two tandem testes (T), faintly-stained ootype (O), ovary (OV), uterus (U) filled with eggs, acetabulum (A), and seminal vesicle (S). Extensive vitelline glands (V) occupy lateral margins. **b.** Newly formed eggs in the uterus, which are passed out unembryonated in the feces of the definitive host. **c.** Collar of spines surrounding mouth, the feature that gives echinostomes their name.

Life cycle:

adult in small intestine of definitive host – egg in feces – 2 wk -- miracidium – penetrates snail (1^{st} intermediate host) -- sporocyst -- produces many -- mother rediae – each produces many -- daughter rediae -- each produces many -- cercariae -- penetrate 2^{nd} intermediate host (mollusc) -- metacercaria – ingested by definitive host -- adult

6.2. Fasciolopsis buski **a.** Adult worm. This giant (up to 75 mm) fasciolid intestinal fluke is contracted in Asia by eating uncooked aquatic plants on which metacercariae have encysted. Dogs and pigs serve as reservoir hosts. Heavy infections can cause diarrhea, intestinal obstruction, and systemic toxicity, which may be fatal. Tandem, dendritic testes are faintly visible posteriorly. Unlike the related *Fasciola hepatica*, *F. buski* possesses unbranched intestinal ceca (not visible here), and does not possess "shoulders" at the anterior end. Extensive vitelline glands occupy the lateral margins, and the gravid uterus is visible at the anterior end. **b.** Eggs in uterus. **c.** Tegument, showing absence of spines.

Life cycle:

adult in small intestine of definitive host – egg in feces – 7 wk -- miracidium – penetrates *Segmentina* snail (1^{st} intermediate host) -- sporocyst -- produces many -- mother rediae – each produces many -- daughter rediae -- each produces many -- cercariae – encyst on vegetation -- metacercaria – ingested by definitive host -- adult

6.4. *Fasciola hepatica.* a. Adult worm. This large (up to 30 mm) liver fluke has a worldwide distribution. Infections are contracted by eating uncooked aquatic plants on which metacercariae have encysted. Sheep and cattle are reservoir hosts. Juvenile worms cause anemia, damage to the

liver parenchyma, and may lodge in ectopic locations, e.g., the brain or eye, whereas adults damage the bile ducts. This species can be recognized easily by its cephalic cone and "shoulders," as well as the highly branched intestinal ceca, clearly visible at the posterior end. Two darkly stained, tandem, dendritic testes occur in the middle of the body. **b.** Eggs in uterus, similar to those of *Fasciolopsis buski*. **c.** Tegument, showing spines.

Life cycle:

adult in bile ducts of definitive host – egg in feces – 9-10 days -- miracidium – penetrates lymnaeid snail (1st intermediate host) -- sporocyst -- produces many -- mother rediae – each produces many -- daughter rediae -- each produces many -- cercariae – encyst on vegetation -- metacercaria – ingested by definitive host – juvenile -- penetrate through intestine into coelom, then info liver -- 2 months in parenchyma -- adult in bile ducts

6.5. *Clonorchis sinensis.* **a.** Adult worm. Infections with this liver fluke are contracted in Asia by eating uncooked freshwater fish in which metacercariae have encysted. Piscivorous mammals can serve as reservoir hosts. Although the liver parenchyma is not invaded, heavy infections may cause such *damage to the bile ducts* that jaundice, hepatomegaly, ascites, and death occur. Note the posterior, tandem, dendritic testes, the relatively large seminal receptacle anterior to the testes, the gravid uterus, and vitelline glands, confined to the middle of the body. **b.** Eggs in uterus. **c.** Tegument, showing absence of spines.

•back to *adult* of *Dicrocoelium dendriticum*

Life cycle:

adult in bile duct of definitive host -- eggs in feces – eaten by *Parafossarulus* snail (1st intermediate host) -- miracidium – penetrates gut – sporocyst -- produces many -- rediae – each produces many -- cercariae – penetrate 2nd intermediate host (freshwater) -- metacercaria – ingested by definitive host – juvenile – migrates up bile duct -- adult

6.6. *Dicrocoelium dendriticum* **a.** Adult worm. This liver fluke occurs in Europe. Asia, and N. America. It has an unusual terrestrial life cycle, and infections are contracted by eating ants infected with metacercariae. Infected ants display negative geotaxis during cool times of the day, causing them to crawl up blades of grass where they may be eaten by sheep and cattle. Thus, human infections are rare, but can mimic biliary disorders seen with other liver flukes. Note pointed ends of body, and extensive posterior uterus (unlike that of *Clonorchis sinensis*, with which it may be confused). **b.** Eggs in uterus. **c.** Tegument, showing absence of spines.

Life cycle:

adult in bile ducts of definitive host – egg in feces – eaten by *Cionella* land snail (1st intermediate host) -- miracidium – penetrates gut – mother sporocyst -- produces many -- daughter sporocysts – each produces many – cercariae in snail mucus – ingested by ant (2nd intermediate host) – metacercaria in ant hemocoel -- ingested by definitive host -- juvenile -- migrates up bile duct -- adult in bile ducts

6.7. *Paragonimus westermani*. **a.** Adult worm. Infections with this lung fluke are contracted in Asia, the S. Pacific, and S. America by eating uncooked freshwater crabs that are infected with metacercariae. Numerous mammals, especially cats, serve as reservoir hosts. The lung lesions containing worm pairs result in hemoptysis, chronic cough, and frequent bacterial superinfection. Also, the juvenile worms may travel to ectopic locations, e.g., the brain, causing seizures. Note the coffee-bean shape, long, wavy ceca, adjacent, branched testes, and off-centered, lobate ovary. **b.** Eggs in uterus, collapsed during specimen processing. **c.** Tegument, showing spines.

Life cycle:

adult in lungs of definitive host -- eggs in sputum or feces – 2 wks -- miracidium – penetrates thiarid snail (1st intermediate host) -- sporocyst -- produces many -- mother rediae – each produces many -- daughter rediae -- each produces many -- cercariae -- penetrate freshwater crab

(2nd intermediate host) -- metacercaria – ingested by definitive host – juvenile – penetrates through intestine into coelom, then through diaphragm into lung -- adult

6.8. *Schistosoma mansoni*. **a.** Adult worms. Infections with this blood fluke are acquired in Africa, S. America, and parts of the Caribbean when cercariae from *Biomphalaria* spp. snails penetrate through the skin. Thus, no metacercarial stage is involved. Eggs deposited by the female in mesenteric veins are swept into the liver and elicit an inflammatory response, resulting in fibrosis, which may cause portal hypertension and hemorrhage of collateral vessels. **Intestinal lesions** may also occur. Note that the worms have separate sexes, and that the female is wrapped within the gynecophoral canal of the male. **b.** Single egg within uterus. **c.** Male tegument, showing papillae. The tegument of *S. haematobium* (which occurs in Africa) has fewer papillae, and that of *S. japonicum* (in Asia) is smooth.

Life cycle:

adult in mesenteric veins of definitive host -- egg in feces – miracidium – penetrates Biomphalaria snail (1st intermediate host) -- mother sporocyst -- produces many -- daughter sporocysts – each produces many – cercariae -- penetrates skin of definitive host -- schistosomulum -- migrates to liver, mates, migrates to mesenteric veins -- adult

6.9. Eggs of *Clonorchis sinensis*. Top row photographed at the same magnification as eggs of other species in following figures. The small size of this egg is a key diagnostic feature. Second and third rows photographed at a higher magnification, to show the operculum sitting in a rim, and a fully formed miracidium, which does not hatch from the egg until ingested by a snail. A small abopercular knob is visible on some eggs.

6.10. Eggs of *Fasciola hepatica*. Note the unembryonated condition, smooth oval shape, and relatively small (compared to eggs of *Paragonimus westermani*) operculum, which is most easily seen at the bottom right, where it has partially opened due to the pressure of the coverslip. Usually, yolk granules completely fill the immature egg.

6.11. Eggs of *Paragoninms westermani*. This egg would be found in human sputum, but also occurs in feces when swallowed. Note the thick abopercular wall, unembryonated condition, and relatively wide (compared to eggs of *Fasciola hepatica*) operculum, which sits in a rim.

6.12. Eggs of *Schistosoma haematobium.* These would be found mainly in urine, because the adult worms inhabit veins surrounding the bladder, but eggs also are found in feces. They are easily identified by the terminal spine and fully formed miracidium, which hatches immediately in fresh water through a tear in the egg shell (upper right photo). *S. haematobium* occurs in Africa and the Middle East, and is transmitted by *Bulinus* snails. In addition to hematuria and fibrosis of the bladder and ureters, infections are associated with bladder cancer.

6.13. Eggs of *Schistosoma japonicum*. Note the fully formed miracidium, which hatches immediately in fresh water through a tear in the shell (bottom row), and the minute lateral spine, which is not apparent in several photographs because it is not viewed in profile. This species, which occurs in Asia, is the most pathogenic of the human-infecting schistosomes due to the high egg production by females and the tendency of the small eggs to pass through the liver and enter the systemic circulation, causing pathology in other organs, especially the central nervous system. *S. japonicum* is transmitted by operculate snails in the genus *Oncomelania*.

6.14. Eggs of *Schistosoma mansoni*. Note the fully formed miracidium, which hatches immediately in fresh water through a tear in the shell (lower right photo), and the large lateral spine, which may be inconspicuous if pointing straight up (top right photo). This species is transmitted in Africa, the Caribbean, and Brazil by snails in the genus *Biomphalaria*.

6.15. Eggs of *Echinostoma paraensei*. Unembryonated when first deposited in the feces, a miracidium develops within two weeks and hatches through a small operculum (bottom right two photos). Compare with eggs of *Fasciofa hepatica*.

6.16. Metacercariae. These encysted stages occur in the life cycle of nearly all trematodes except for the schistosomes. **a.** Histological section of *Clinostomum*, a parasite of bird definitive hosts, in fish muscle. **b.** *Fasciola hepatica*, metacercariae of which would be found on the surface of aquatic plants, e.g., watercress. **c.** *Paragonimus westermani*, from the muscle of infected crab. **d.** *Echinostoma paraensei*, from the pericardial cavity of a snail. Note the thick tunic of snail hemocytes surrounding each echinostome metacercaria.

6.17. **Life cycle** of *Schistosoma mansoni*. **a.** Adult worms *in copula*. **b.** Egg in feces. Eggs hatch immediately upon reaching fresh water, releasing a miracidium. **c.** Miracidium, which must penetrate into a susceptible snail within several hours. **d.** Miracidia penetrating head foot of *Biomphalaria glabrata.* Each miracidium transforms into a single mother sporocyst at the site of penetration. **e.** Mother sporocyst in tentacle of *B. glabrata* several weeks after penetration of the miracidium. **f.** Squash of tentacle infected with mother sporocyst, showing released daughter sporocysts. **g.** Higher magnification of daughter sporocyst, showing typical elongation and enlargement of anterior end. These migrate posteriorly to the digestive gland. **h.** Digestive gland of infected snail (green), largely replaced by daughter sporocysts (yellow). These give rise to cercariae. **i.** Cercaria, with characteristic forked tail. These attach to human skin, drop their tail, penetrate, and are then called schistosomula. **j.** Schistosomulum, which pairs with a worm of the opposite sex in the liver, develops to adulthood, and migrates to the mesenteric veins.

6.18. Snail vectors of schistosomiasis. **a.** *Biomphalaria glabrata*, intermediate host of *Schistosoma mansoni* in the New World. Note the planospiral shell and the red tissue coloration, due to free hemoglobin m the hemolymph, a characteristic of planorbid snails (i.e., in the family Planorbidae). These snails can grow to well over 20 mm in diameter. Since they are hermaphroditic and can self fertilize, and because they can survive desiccation by entering a state of dormancy, they are difficult to control with molluscicides. **b.** *Bulinus truncatus* from Egypt, the intermediate host of S. *haematobium*. Note the more conical, globose shell. This snail is also a planorbid. **c.** *Oncomelania hupensis*, the intermediate host of *S. japonicum*. This snail is much smaller than the other two (only several mm long), and unlike them lacks hemoglobin, has separate sexes, possesses a gill, and has an operculum on its head foot that it uses to seal the aperture of its shell. Due to its operculum and because it is amphibious rather than aquatic, it too is difficult to control with molluscicides.

6.19. Histological section of bile duct infected with adults of *Clonorchis sinensis*. These worms feed upon, and damage, the epithelium of the duct, which is eroded in some areas of this section. Note the inflammatory response in the lamina propria of the duct, possibly caused by bacterial infection, which results in further tissue damage. Routine ingestion of uncooked freshwater fish harboring metacercariae can lead to infections with thousands of adults, resulting in biliary dysfunction, jaundice, ascites, and sometimes death.

6.20. Histological section of liver from mouse infected with *Schistosoma mansoni*. **a.** Low magnification view, showing three granulomas in the parenchyma. **b.** Higher magnification view of single granuloma surrounding an egg, which has a clearly visible lateral spine. These granulomas protect the surrounding hepatocytes from lytic enzymes released by the egg, but unfortunately occlude the sinusoids and presinusoidal capillaries through which blood must flow. As a result, pressure is elevated in the hepatic portal vein carrying blood from the intestine to the liver (portal hypertension), causing seepage of fluid into the abdominal cavity (ascites fluid). More importantly, thin-walled collateral vessels form to carry the blood around the liver

blockage and back to the heart. If a large vessel bursts, especially in the wall of the esophagus (rupture of esophageal varices), fatal hemorrhage may result. Liver disease occurs in about 8% of cases of infection with *S. mansoni* and *S. japonicum*.

6.21. Histological section of small intestine from mouse infected with *Schistosoma mansoni* **a.** Several eggs in mucosa, surrounded by a diffuse cellular infiltrate. **b.** Higher magnification of an egg, showing lateral spine (arrow). In humans, the most severe lesions in the case of *S. mansoni* infections occur in the colon. Symptomatic intestinal schistosomiasis is most common with S. *japonicum*.

6.22 Eggs of *Schistosoma haematobium* in the wall of the urinary bladder. **a.** Large numbers of calcified eggs in the muscularis. Note the thickened epithelium. **b.** Higher magnification of eggs. The terminal spines of the eggs are not visible in this section. Damage to the bladder wall can lead to hematuria, bacterial infections, as well as metaplasia and possibly cancer of the bladder epithelium.

7.1 Adult of *Diphyllobothrium latum*. **a.** Scolex, showing groove-like bothrium (arrow) used to grasp the host mucosa. **b.** Mature proglottid. **c.** Higher magnification of mature proglottid, showing bilobed ovary, uterus with eggs, and follicular vitelline glands. The pink background is due to numerous follicular testes, situated between the dorsal and ventral layers of vitelline follicles. In this species, eggs are released through the uterine pore, located anterior to the uterus. **d.** Higher magnification of eggs in uterus. Infection results from eating second intermediate or paratenic hosts infected with plerocercoids. Symptoms range from none to fatigue, to diarrhea; however, approximately 2% of infected individuals develop megaloblastic anemia, due to the worm's unique affinity for vitamin B12. Ingestion of first intermediate hosts (or of intermediate or paratenic hosts of non human-infecting pseudophyllideans, e.g., *Spirometra mansonoides*) leads to development of plerocercoids in the tissues (sparganosis).
Life cycle:
adult in intestine of definitive host (piscivorous mammals) -- eggs in feces – l-several weeks -- coracidium hatches from egg --eaten by freshwater crustacean (1^{st} intermediate host) -- procercoid in hemocoel -- eaten by fish/frog (2^{nd} intermediate host) -- plerocercoid in muscles -- eaten by fish/snake/swine (paratenic host) -- plerocercoid in muscles -- eaten by definitive host -- adult

7.2 Scolex of *Taenia pisiformis*, a tapeworm of dogs, that shows typical cyclophyllidean anatomy. **a.** Scolex and neck (where immature proglottids are formed), showing armed rostellum and 4 acetabula (suckers). The scolex of *T. solium* would appear similar, whereas that of *T. saginata* lacks hooks. **b.** Higher magnification of rostellum, showing circle of hooks. **c.** Higher magnification of individual hook.

7.3 Strobila (body) of *Taenia pisiformis*. **a.** Immature proglottids being formed from neck region. **b.** Immature proglottids containing developing reproductive organs. **c.** Mature proglottid, showing a bilobed ovary, follicular testes, lateral genital pore, and posterior vitelline mass. Ventral canals of the excretory system are visible laterally.

7.4 *Taenia saginaia* vs. *T. solium*. **a.** Proglottid of *T. saginata*. **b.** Scolex of *T. saginata*. Note the absence of hooks. **c.** Proglottid of *T. solium*. **d.** Scolex of *T. solium*. Note the rostellum, armed with hooks. The two species can be distinguished both on the basis of their scolex, and by counting the number of side branches of the gravid uterus, numbering >14 in *T. saginaia* and <14 in *T. solium*. Human infections with the adult worm are acquired by eating undercooked beef (*T. saginaia*) or pork (*T. solium*) infected with cysticerci. Infections with adults usually cause mild or no symptoms. However, *T. solium* is quite hazardous, because its eggs (unlike

those of *T. saginata*) if ingested by humans release an oncosphere that develops into a bladder-like cysticercus larva, which can cause severe disease in the brain or eye. Also, gravid proglottids may rupture in the intestinal lumen, resulting in overwhelming cysticercosis.
•back to scolex of *Taenia pisiformis*

Life cycle:

adult in small intestine -- gravid proglottid in feces – ruptures – embryonated eggs – ingested by cow (*T. saginata*), pig (*T. solium*), human (*T. solium*) – oncosphere hatces from egg – penetrates gut, enters blood – cysticercus in internal organs – ingested by human -- adult

7.5 Cysticercus of *Taenia solium*. **a.** Histological section of infected pork, showing 4 cysticerci, each consisting of a fluid-filled bladder containing an invaginated, introverted (inside-out) scolex. Cysticercosis results from ingestion of *T. solium* eggs from human feces, and occurs in both pigs and humans. Also, humans harboring the adult tapeworm may develop heavy burdens of cysticerci when gravid proglottids are retained in the intestine and then rupture, releasing thousands of infective eggs. Cysticerci can lodge in any organ, and may be visible as surface swellings if occurring subcutaneously. Depending on numbers and location, cysticerci in the brain may cause symptoms of epilepsy. Note the absence of inflammation around viable cysticerci. However, dead cysticerci elicit a strong inflammatory response, which can be fatal if occurring in the brain. **b.** Whole mount of cysticercus, showing bladder and introverted, invaginated scolex (hooks not visible at this magnification).

7.6 Adult of Echinococcus granulosus. **a.** Adult worm, consisting of a scolex and 3 proglottids. **b.** Eggs in uterus. These are indistinguishable from eggs of other taeniids that are found in dog feces. **c.** Higher magnification of the scolex, which bears 4 suckers and an armed rostellum. When eggs of this parasite are ingested by intermediate hosts, including humans, the oncosphere develops in internal organs into a large fluid-filled hydatid cyst, in which infectious protoscolices are produced asexually. These cysts grow slowly, sometimes filling with up to 2 liters of fluid, eventually compressing surrounding tissues. Disease may result from pressure effects or from rupture of large cysts, causing anaphylactic shock. This parasite is more common in sheep-rearing areas, where sheep, rabbits, and other mammals eaten by dogs become infected by ingesting eggs from dog feces. Dogs develop heavy infections, because a single cyst contains many protoscolices, each of which develops into an adult.

Life cycle:

adult in dog intestine -- eggs in feces – ingested by intermediate host -- oncosphere hatces from egg – penetrates gut, enters blood vessels – hydatid cyst in internal organs -- protoscolices (hydatid sand) -- ingested by dog -- adult

7.7 Hydatid cysts. These develop in the internal organs of intermediate hosts, including humans, that ingest eggs from feces of infected canids. **a.** Histological section of the wall of a unilocular cyst of *Echinococcus granulosus*. Hanging into the fluid filled cavity is a brood chamber, composed of germinal membrane and containing protoscolices. The inner germinal layer of the cyst (endocyst, EN) is surrounded by an acellular, laminated ectocyst (EC). A thick fibrous tissue host response (HR) surrounds the ectocyst. **b.** Histological section of a multilocular cyst of *E. multilocularis*. Because the cyst of this species lacks a restraining laminated ectocyst, the germinal membrane can grow outward into surrounding tissues or break off and metastasize to other sites. Therefore, prognosis is grave unless the infection is diagnosed early and the cyst surgically removed. *E. multilocularis* is mainly a parasite of wild canids, especially foxes, and consequently fur trappers are at risk of infection.

7.8 *Hymenolepis nana.* **a.** Cysticercoid (possibly of either *H. nana* or *H. diminuta*). **b.** Adult. **c.** Scolex and neck. The rostellum is retractable into a sac and is armed with hooks, not visible at this magnification. **d.** Mature proglottids. This species is unique among tapeworms in not

requiring an intermediate host to complete its life cycle (a direct or homoxenous life cycle). However, an indirect life cycle, utilizing an insect intermediate host, also can occur. Humans become infected by ingesting eggs from rodent or human feces, or by ingesting intermediate hosts infected with cysticerci. Light infections are asymptomatic. However, because eggs may hatch in the intestine of the definitive host, autoinfection can produce heavy parasite burdens (thousands of worms) leading in humans to diarrhea and symptoms of toxicity.
Life cycle (direct):
adult in intestine – proglottid detsches, disintegrates – eggs in feces -- ingested by of definitive host -- oncosphere hatces from egg – penetrates villus -- cysticercoid in lamina propria – leaves villus (5-6 days) -- adult in intestine

7.9 *Dipylidium canina*. **a.** Scolex, consisting of 4 suckers and an armed rostellum, retracted into a sac in this photograph. **b.** Immature proglottid. **c.** Mature proglottid. Note the two lateral genital pores, diagnostic for this species. **d.** Gravid proglottid, in which the uterus has broken up into egg capsules, each containing 5-20 eggs. Gravid proglottids are quite motile for a short time after being passed out in the feces. **e.** Two egg capsules in the uterus. This relatively harmless tapeworm is a cosmopolitan parasite of dogs and cats, and rarely humans, most often children. Infections are acquired by ingesting a flea intermediate host infected with the cysticercoid stage.
Life cycle:
adult in small intestine -- gravid proglottid in feces – disintegrates -- egg capsule -- ingested by flea – oncosphere hatces from egg – penetrates gut – cysticercoid in hemocoel – ingested by definitive host -- adult

7.10. Eggs of *Diphyllobothrium latum*. Because of the large size of the adult, a million eggs/day may be shed in the feces from each worm. The eggs are operculate, symmetrically oval, and sometimes have an abopercular knob (arrow). Structurally they are similar to a trematode egg. The embryo is undeveloped when the egg is released from the uterus, and must complete its development to the coracidium stage (a ciliated oncosphere) in fresh water.

7.11 Eggs of *Hymenolepis nana*. Top left photograph shows a higher magnification. Note the oncosphere with hooks (thin arrow), surrounded by an embryophore that has polar filaments at either end (thick arrows). The outermost capsule is separated from the embryophore by a gelatinous granular layer. These eggs are infective not only for an insect intermediate host, but also for the rodent or human definitive host.

7.13 Taeniid eggs. The oncosphere is surrounded by a 2-layered embryophore, and the thicker outer layer has prominent radial striations, a diagnostic feature. The surrounding gelatinous layer and capsule (arrow, top left photograph) are flimsy and often detach from the striated embryophore, which then becomes the outermost layer. Eggs of various taeniids (e.g., *Echinococcus. Multiceps*, and *Taenia* spp.) are indistinguishable from one another. Taeniid eggs in human feces would indicate infection with either the relatively harmless *Taenia saginata* or the quite pathogenic *T. solium*, and therefore further diagnostic tests would be advisable, e.g., examination of scolices and gravid proglottids recovered from feces. Taeniid eggs on the ground may remain infective for over 5 months.

7.14 Egg capsules of *Dipylidium caninum*. These are released from disintegrating gravid proglottids, which are quite active after being passed in the feces until they desiccate. The capsules are formed from compartments of the uterus.

8.1 *Trichuris trichiura.* **a.** Adult male. Note curved posterior end. **b.** Adult female. **c.** Portion of esophagus, surrounded by unicellular glands called stichocytes. These cosmopolitan intestinal parasites have an easily recognizable whip-like shape, hence the common name whipworm. The

long, thin anterior end lies buried in the mucosa of the ileocecal region. Because of the mechanical and possibly immune-mediated damage to the mucosa, heavy infections can result in dysentery, anemia, and rectal prolapse. Heavy infections in children, common in some tropical countries, can retard physical development and cognition.

Life cycle:

adult in ileocecal region – unembryonated eggs in feces – 2 wk -- J1 in egg – ingested, hatches -- J1 in intestinal crypts – 4 molts -- adult in ileocecal region

8.3 *Trichinella spiralis*. **a.** Portion of adult female, showing uterus packed with J_1 larvae. Each female releases approximately 1,500 J_1s in the intestine of the definitive host. **b.** Several juveniles encysted in pork skeletal muscle. **c.** Higher magnification of juvenile in nurse cell. This nematode is unusual in that it is an intracellular parasite, and the definitive host also serves as intermediate host. Infection occurs when infective encysted larvae are eaten in uncooked meat, usually pork (although an outbreak occurred in France due to ingestion of infected horse meat). Humans are compatible hosts, but they are a "dead end" for the parasite in the sense that their muscles usually are not eaten by another animal. This parasite is quite pathogenic, damaging not only skeletal muscles but also intestinal mucosa, lungs, heart myofibers, and the brain. A single bite of heavily infected, undercooked pork, often in the form of sausage, can be fatal.

Life cycle:

adult in intestinal mucosa -- J1 in lymphatics, blood (some pass out in feces) – penetrate skeletal myofibers -- J1 in nurse cell – ingested by definitive host, molts 4x -- adult in intestinal mucosa

8.4 Hookworms. **a.** Female *Necator americanus*. **b.** Male *N. americanus*. Note posterior copulatory bursa. Both specimens show, dorsal curvature of the anterior end, hence the name "hookworm." **c.** Buccal capsule of *Ancylostoma duodenale*, showing the large ventral teeth. **d.** Copulatory bursa of *male A. duodenale*. **e.** Dorsal ray of the bursa of *A. duodenale*, showing 3 branches at the end of each fork. **f.** Buccal capsule of *N. americanus*, showing cutting plate (arrow). **g.** Copulatory bursa of male *N. americanus*. **h.** Dorsal ray of the bursa of N. americanus, showing 2 branches at the end of each fork. Besides structural differences, *N. americanus* is more prevalent everywhere except southern Europe, and its life cycle differs slightly from that of *A. duodenale* (i.e., no developmental arrest in immune hosts; a requirement for migration through the lung). Heavy infection with these blood-feeding worms causes anemia and symptoms of protein malnourishment, which can be fatal.

Life cycle:

adult in small intestine – unembryonated eggs in feces – 24 hr -- rhabditiform J1 in soil – 2 molts – filariform J3 in soil – penetrates skin, travels in blood to lungs, breaks through alveolus, coughed up, swallowed, molts 2x -- adult in small intestine

8.5. Rhabditiform vs. filariform larvae. **a.** Anterior end of rhabditiform (J1) hookworm larva. A corpus, isthmus, and end bulb are all present in the esophagus. **b.** Higher magnification of anterior end of rhabditiform hookworm larva showing large buccal capsule (BC). This feature allows it to be distinguished from the rhabditiform larva of *Strongyloides stercoralis*. **c.** Anterior end of filariform (J3) hookworm larva. No isthmus is present in the esophagus, although a nerve ring is visible (arrow). This is the infective larval stage.

8.6 *Ascaris lumbricoides* adults. **a.** Adult females (top) and males (bottom). Note curved posterior end of male. **b.** Cross section at the level of the esophagus. Note the triradiate lumen of the esophagus (E), a feature of all nematodes. **c.** Cross section of male. **d.** Cross section of female. CU, cuticle; ET, excretory tubules; I, intestine; IP, innervation processes; LC, lateral cords; MU, longitudinal muscles; O, ovary; OD, oviduct; P, pseudocoel; T, testis; V, vas deferens. In heavy infections pneumonitis results from **lung damage** during larval migration.

Large numbers of adults can cause malnutrition, allergic symptoms, and fatal intestinal blockage, and wandering worms can invade visceral organs or cause asphyxiation if aspirated.
•back to eggs of *Toxocara canis*

Life cycle:
adult in intestine – unembryonated eggs in feces – 2 wk – J2 in egg – ingested, hatches – J2 in intestine – penetrates mucosa, travels to lungs in blood, penetrates alveolus, moves up to pharynx, swallowed, molts 2x -- adult in small intestine

8.7 J2s of *Ascaris lumbricoides* in lungs. Although larvae hatch from eggs in the duodenum, which is the location for the adult worm, they undergo a complex migration by penetrating into the mucosa and travelling via the blood to the lungs, where they molt twice to the J4 stage. They then break into an alveolus, pass up to the pharynx, and are swallowed. Upon reaching the duodenum for the second time, they develop into adults. **a.** Juvenile (arrow) breaking into alveolus. Note severe inflammation (pneumonitis) in interstitial tissue. **b.** Bronchus containing inflammatory cells and larvae, which are travelling upward to the pharynx prior to being swallowed. Damage from heavy infections, especially if complicated by bacterial superinfection, can be fatal.

8.8 *Enterobius vermicularis*. **a.** Adult male (on left) and female. Note long pointed tail of female, hence the name "pinworm". **b.** Histological section of appendix, showing three cross sections of pinworms in the lumen (arrow). Although often residing in the appendix, the worms are not believed to cause appendicitis. Infection occurs when the eggs containing J3 are inhaled or swallowed. The major symptom of infection is perianal itching, although more serious problems can result if tissue invasion occurs, and adults sometimes wander into the female reproductive tract.
Life cycle:
adult in ileocecal region -- female migrates to perianal skin – unembryonated eggs on perianal skin – 6 hr – J3 in egg – swallowed, hathes -- J3 in duodenum – molts 2x, migrates posteriorly -- adult in ileocecal region

8.9 *Dirofilaria immitis.* **a.** Microfilaria in dog peripheral blood smear. In this species the microfilaria, which is an incompletely developed J1, does not possess a sheath (i.e., the egg capsule), and nuclei do not extend to the tip of the pointed posterior end. **b.** Adults removed from the heart of an infected dog. Note the threadlike appearance, which is typical of filarial worms. Cardiac and pulmonary pathology occurs in heavily infected dogs. In humans, worms die before attaining adulthood, and are washed into the lung microcirculation, where they form granulomas.

Life cycle:

adult in right heart and pulmonary artery of dog -- microfilariae in peripheral blood – ingested by mosquito, molt twice – J3 in Malpighian tubules – migrates to mouthparts, invades bite wound, migrates to heart, molt twice – adult in right heart and pulmonary artery

8.10 Microfilariae in peripheral blood smears, Geimsa stain.

a. Microfilaria of *Brugia malayi*. **b.** Microfilaria of *Wucheraria bancrofti*. These species are distinguished by the following criteria: presence of a terminal and subterminal nucleus in the tail of B. *malayi* (black arrows), separated by an indentation, vs. the absence of nuclei in the tail of *W. bancrofti* (region enclosed by green bracket); large cephalic space in *B. malayi* vs. small cephalic space in *W. bancrofti* (region enclosed by red bracket); compact column of nuclei in *B. malayi* vs. more dispersed nuclei in *W. bancrofti*. Note the prominent sheath in both specimens (red arrows).

8.11 *Onchocerca volvulis*. **a.** Histological section of adults in a fibrous capsule below the skin. **b.** Histological section of uterus of female, showing microfilariae. **c.** Histological section of dermis, showing wandering microfilariae (arrows). **d.** *Simulium* sp. (blackfly), the vector of *O. volvulis*. Note humped appearance of thorax. Due to the inflammatory response against the microfilariae in the skin and eyes, severe dermatitis and blindness ("river blindness") result from longstanding heavy infections. Human infections occur in Africa and South America.

Life cycle:

adult in subcutaneous nodules – microfalariae in skin –ingested by blackfly, molt twice – J3 in flight muscles – migrates to mouth parts, enters bite wound, molt twice -- adult in subcutaneous nodules

8.13 Eggs of *Trichuris trichiura*. Note the symmetrical barrel shape and plugs on each end. Embryonation to the infective J1 requires approximately two weeks.

8.14 Hookworm eggs. The embryo is surrounded by an extremely thin shell, which sometimes collapses during specimen preparation (arrow). Eggs of the two major human-infecting hookworms, *Necator americanus* and *Ancylostoma duodenale*, are indistinguishable. Embryos are usually at the 4 or 8-cell stage when eggs are passed out in the feces.

8.15 Eggs of *Trichostrongylus* spp. Eggs of these bloodsucking intestinal nematodes appear similar to those of hookworms, with a thin shell and morula embryo. However, they are larger, one end is more tapered, and the embryo contains a greater number of cells.

8.16 Eggs of *Ascaris lumbricoides*. The outermost layer of the shell is proteinaceous and mammillated (bumpy). The eggs typically contain a zygote when passed in the feces (top two rows), and the embryo develops to the J2 (bottom row) in 9-13 days. The eggs are extraordinarily resistant to adverse environmental conditions, and can survive for 10 years in shaded soil. Eggs sometimes are decorticated, i.e., without the mammillated coat, and consequently appear smooth surfaced (last photo, middle row). These can be distinguished from hookworm eggs by their thick shell. Unfertilized eggs also are found; these are more elongate than the fertilized eggs, and do not contain a spherical embryo (last photo, top row).

8.17 Eggs of *Toxocara canis*. This ascarid parasite infects dogs and has a life cycle similar to that of *Ascaris lumbricoides*. Infection occurs when an egg containing a J2 is ingested. Although not capable of developing to the adult stage in humans, its larvae wander in the visceral tissues of persons accidentally ingesting the infective egg, causing the condition known as visceral larva migrans. Most larvae become encapsulated in the liver, causing minimal symptoms (fever, eosinophilia), but those wandering into the brain or eye can cause disease. Because the parasite is capable of transplacental infection, most puppies are born infected and therefore are quite hazardous until dewormed. A related species, *T. cati*, infects cats, but is not capable of transplacental infection. Both species have cervical alae, shown in the upper row for *T. cati*. As with *Ascaris lumbricoides*, eggs of *Toxocara* spp. survive for years in the soil, and many backyards and playgrounds in the U.S. are contaminated with infective eggs.

8.18 Eggs of *Enterobius vermicularis*. These eggs are deposited on the perianal skin by the female at night, and embryonate to the infective stage within 6 hours. Eggs soon contaminate bed sheets, clothing, and, because they are easily lifted aloft in air currents, the surrounding room. When viewed on edge, the eggs appear flat on one side.

8.19 Acanthocephalans. These worms bear some similarity to the nematodes in that they are pseudocoelomates, yet also show affinities with tapeworms in having no digestive tract and possessing a tegument rather than a cuticle. The defining feature is the spiny proboscis. **a.** Egg of

Macracanthorhynchus hirudinaceous viewed on end and lengthwise. The embryo (acanthor) is surrounded by several membranes. **b.** Anterior end of acanthocephalan from turtle intestine, showing proboscis and proboscis receptacle. **c.** Histological cross section of the body wall of a male acanthocephalan. The tegument has a relatively homogeneous outer layer (yellow arrow) and an underlying radial fiber zone (red arrow), in which are found the longitudinal spaces called lacunae. A layer of circular (blue arrow) and longitudinal (green arrow) muscle lies below. Finally, the ligament sac (black arrow), containing the testis, is found in the pseudocoelom. **d.** Histological section of *Macracanthorhynchus hirudinaceous* nearly perforating the intestinal wall. Infections occur in humans who ingest insects harboring the infective larval stage (cystacanth).

List of recommended literature:

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4. Roberts, L.S. and Janovy, J.J. Jr. 2000. Gerald D. Schmidt & Larry S. Roberts. Foundations of Parasitology. 6th ed. McGraw-Hill Publishers.