Nematode

General characters:

1- Unsegmented worms. They are elongate and cylindrical or filiform in appearance, both ends are pointed.
2- The sizes show a great variation.
3- The body is covered with a cuticle.
4- There is body Caity (pseudocoelomate) in which the various organs are located such as digestive, genital, excretory and nerve systems.
5- The alimentary canal is complete consisting of an oral cavity, mouth, oesophagus, intestine and anus.the oral cavity may have teeth or cutting plate.
6- All the nematodes of man are separate sexes.

Reproductive systems:

The male genital system:consist of a long tube which can differentiated into testis, vas deferens, seminal vesicle and ejaculatory duct. The genital duct forms a common passage with intestine known as cloaca. Accessory copulatory organs such as spicule and gubernaculum, are also present.

The female genital system consist of a single or double tubes. Each part of the tube is differentiated into ovary, oviduct, seminal receptacle, uterus, vagina and vulva. The female genital pore opens either in the middle of the body or nearthe mouth.

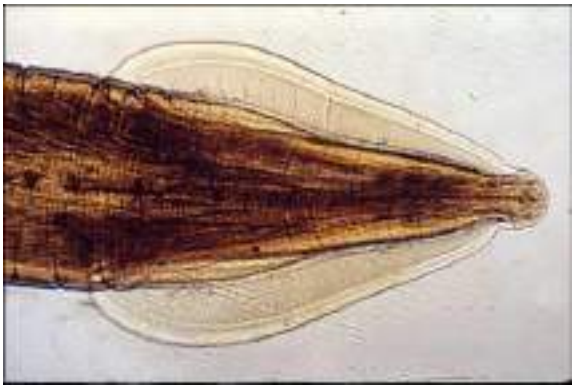
Mode of infection of nematode parasite:

1- By ingestion of egg (embryonated or non embryonated) contaminating food or drink(*Enterobius vermicularis)*
2- By penetraton of the skin *Ancylostoma deudenale*
3- By blooding sucking insects *Wuchereria bancrofti*
4- By unhalation of infected dust (*Enterobius vermicularis*)

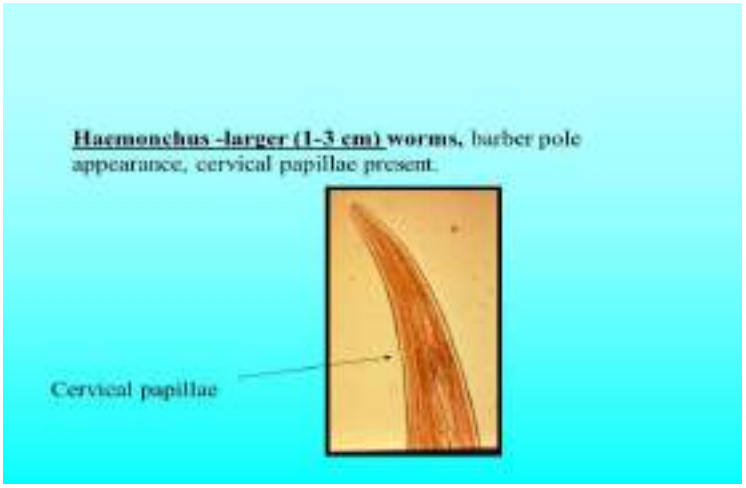
Terms used in the nematodes

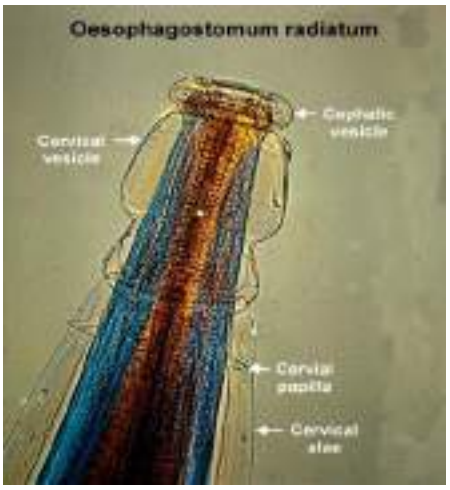
Cuticle: an outer hyaline, non-cellular layer forming the integument of nematodes

Cervical alea :wing like expansion of the cuticula near mouth

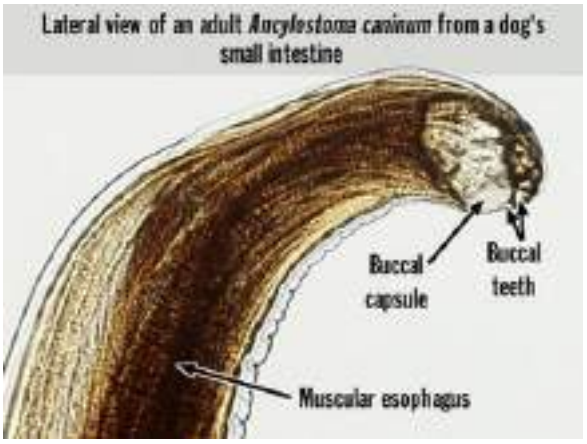


Cervical papillae: protuberances نتوءاتof cuticula near oesophagus

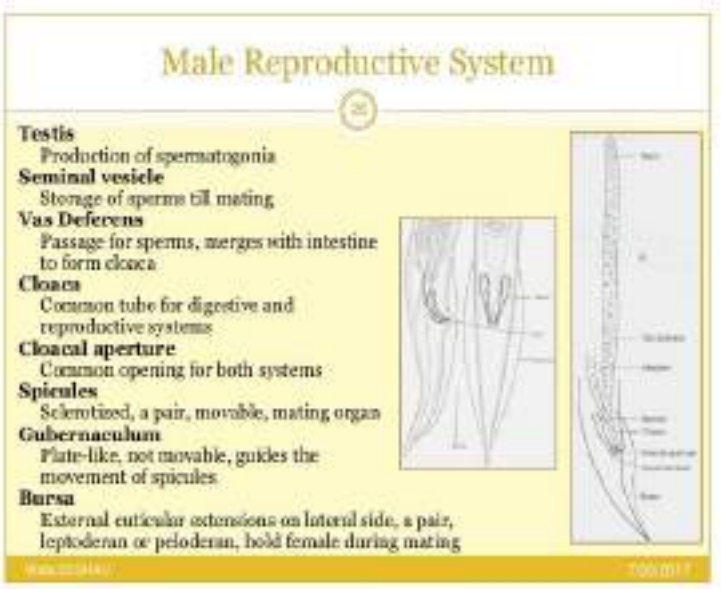


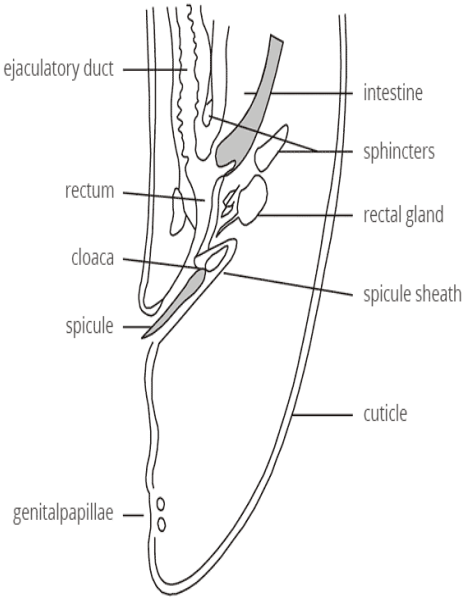


Buccal capsule when the oral فتحةaperture leads to a mouth cavity, it may contain teeth or cutting organs

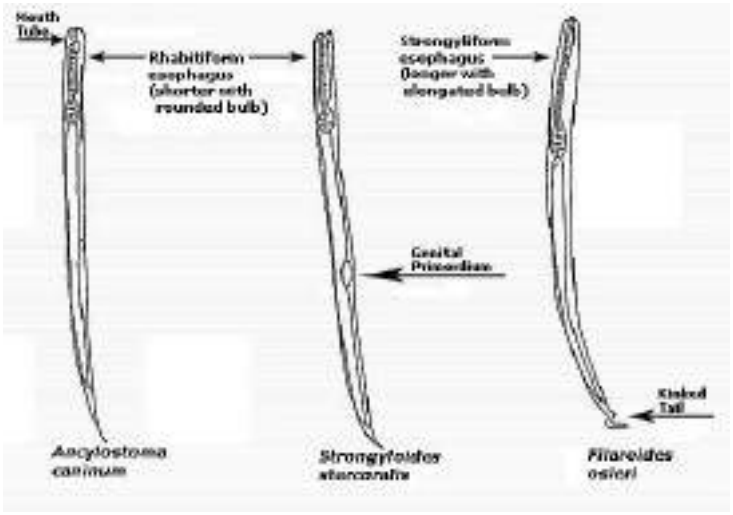


Cloaca: a coomon passage in male nematodes where the rectum and the genital duct open

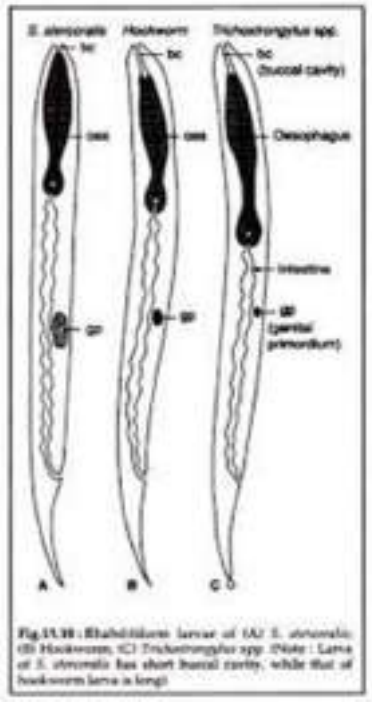




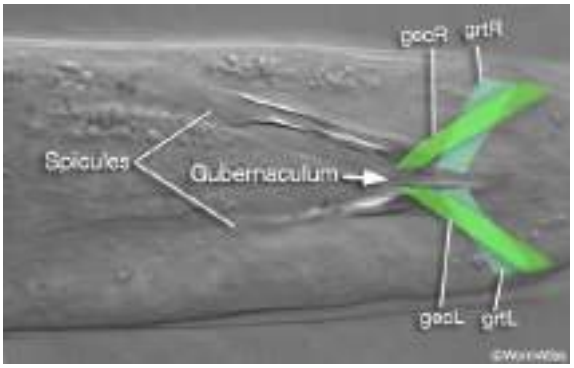
Filariform larva: the larva has long oesophagus and its posterior end not like انتفاخ او بصلةbulb



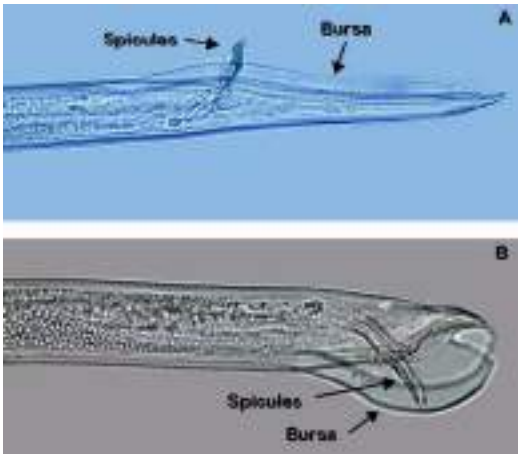
Rhabditiform larva: the larva has short oesophagus and its posterior end like bulb

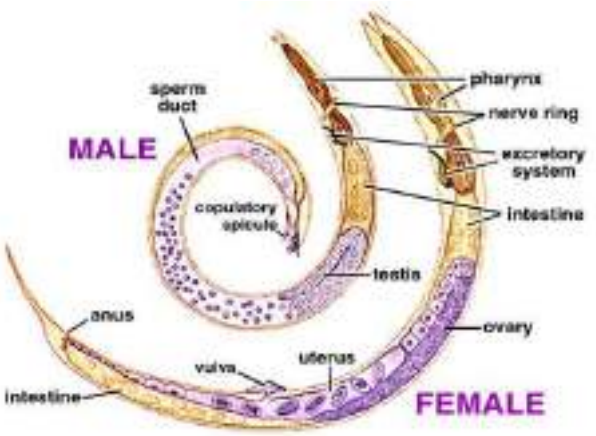


Gubernaculum: an elevation og the dorsal wall of coalca which guides the spicule during copulation

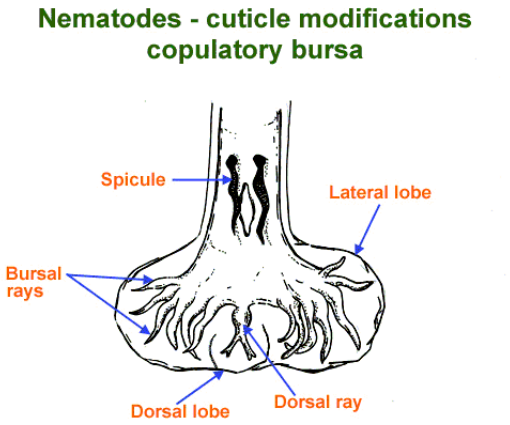


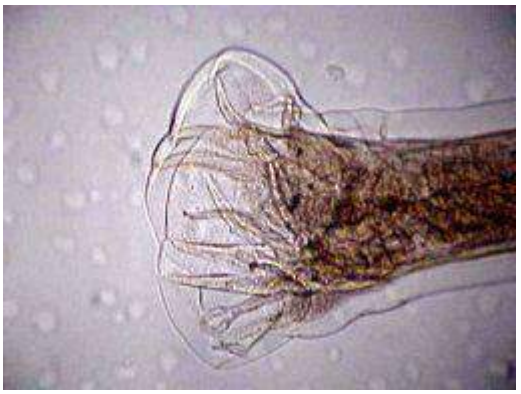
Spicule: represents the accessory copulatory organ it is rodعصا like



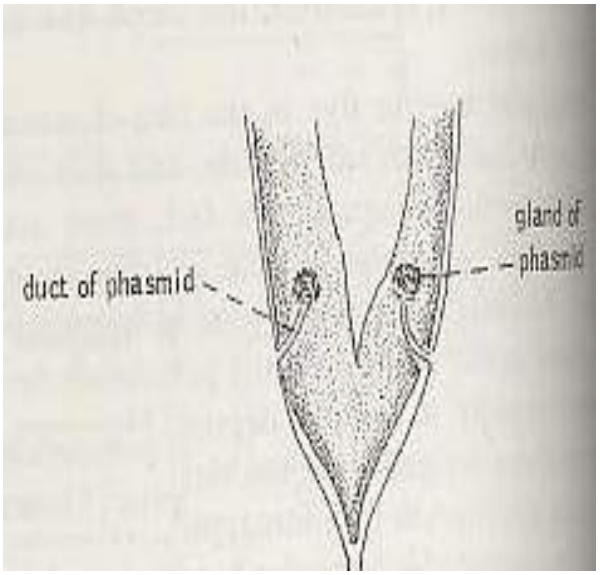


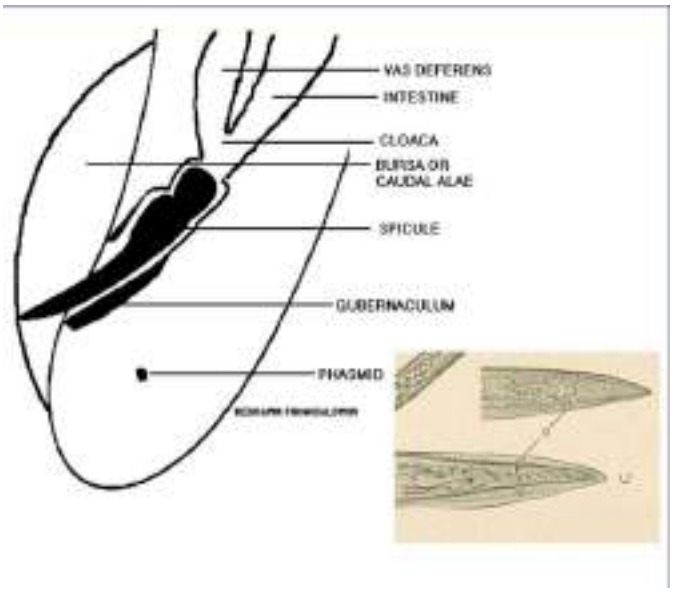
Copulatory bursa: an umbrella like expantion of cuticle surrounding the cloaca of the male nematode of some species. It is supported by fleshy rays comparable with the اشعة او اضلاعribs of umbrella.





Phasmid : pair of sensory structures located on the pair if papillae behnd the anus of nematode





Trichinella spiralis

Geographical distribution:

Common in Europe and united state, also reported from some parts of Africa, china and Syria. Natural human infection with this helminth has been reported in India.

Habitat:

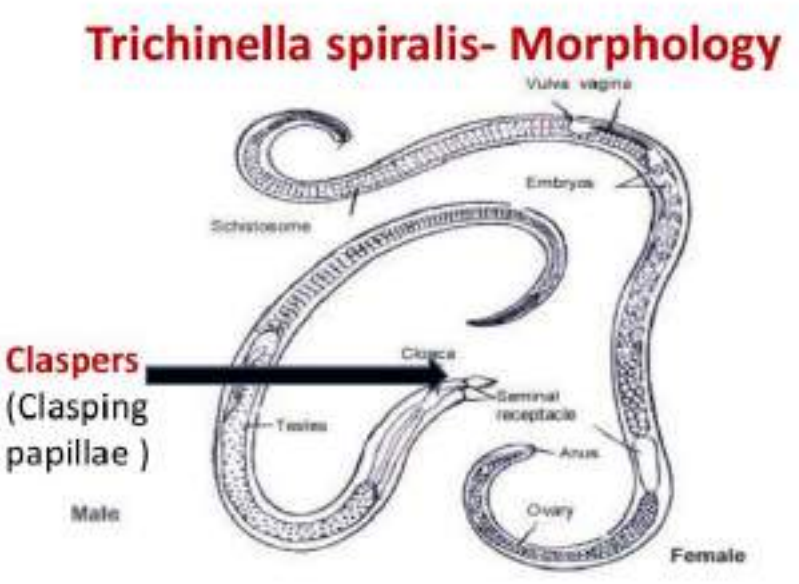
The adult lives in the small intestine of human, pig, rats, bear, fox, dog, cat and white whale. The larval stages live in striated muscles of the above host.

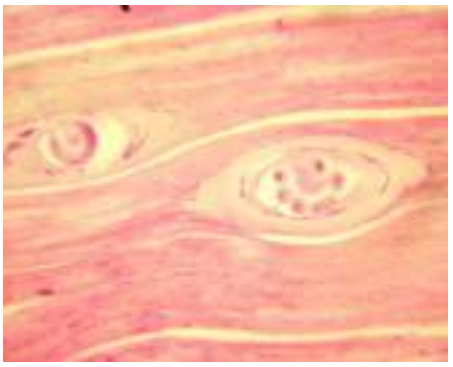
Morphology:

Adult worm it is one of the smallest nematodes infecting man. The male is shorter than female. The spicule and copulatory sheath are lacking, but at the tail end, there are two papillae on either side. The female is much longer than male . The female is discharge embryos instead of eggs.

Larvae; they remain encysted in striated muscle of the host. Inside the cyst the larva continues to devlop up to the stage of sexes differentiated anf when fully grown it becomes ten times its original size

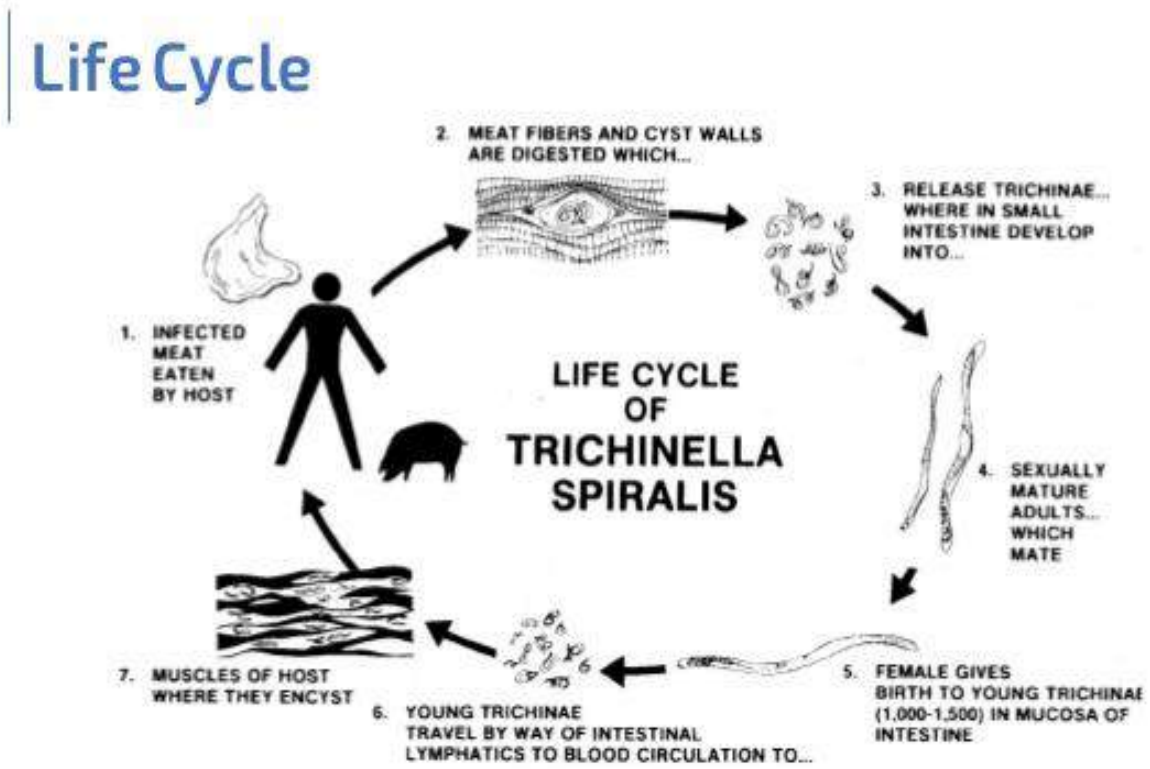
Encapsulation of the larva begins about the 21st day and completed within 3 months. A غمد بيضوي بشكل حاد ليموني الشكل blunt ellipsoidal lemon shaped sheath develops as a result of host tissue reaction around the larva.the calcification occurs after 6 to 18 month.

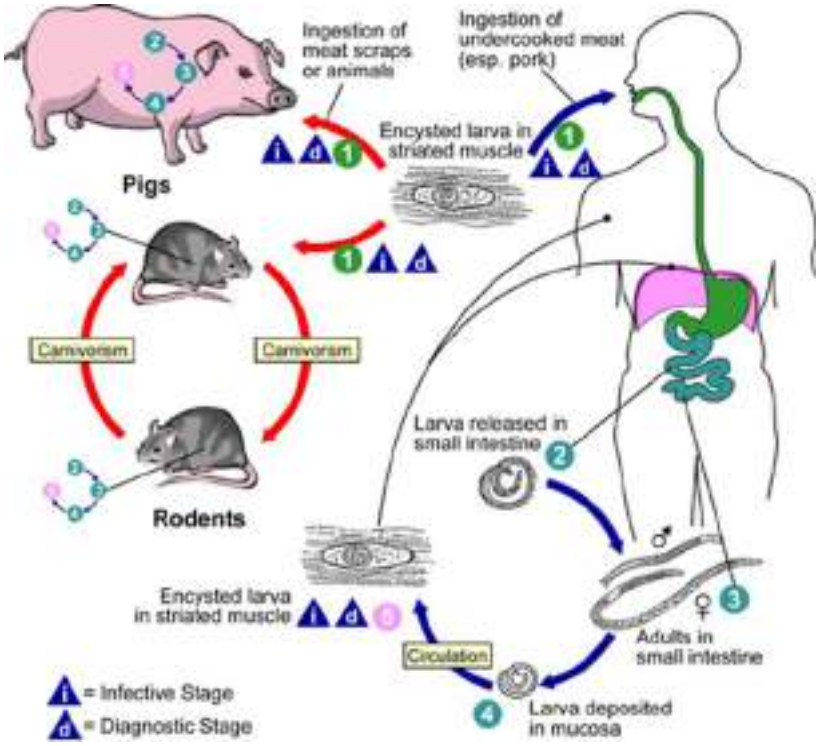




Life cycle:the life cycle is passed in one animal (pig, rat or man) but transference of the host is required for the preservation of the species from extinction. The animal (big) serves both as definitive and intermediate host, two hosts are required to complete the life cycle. The parasite entrance into man is unable to complete its life cycle.

The adult worm lives for not more than 2-3 month and the males die after copulation. The female enters to mucosa and submucosa of intestine and released larva which penetrates the intestine and migrate with lymph and blood to the most part of the body especially the striated muscle which supplied with an abundance of blood. The larva immediately develops and roll up to form spiral shape larva and become infective. The blunt ellipsoidal lemon shaped sheath develops as a result of host tissue reaction around the larva. The infection occurs when the host eats uncooked meat contain this larva. (when this infection occurs in man it is unable to complete, but when it occurs in pig or rat it able to complete by eat the scrapبقايا of animals or uncooked meat).





Pathogenicity:

The symptom are include:

1- Stage of intestine invasion (incubation): this is the period (5-7 days) during the larva grow to adult. The symptom include: abdominal pain, nausea, vomiting, fever)
2- Stage of larval migration: the invasion of muscle occurs from 7th to 10th day after infection.the symptoms include: oedema of the eyelids and face (تورم العين والوجه تيجه تجمع السوائل) and fever>
3- Stage of encystment: this occurs only in striated muscles swelling of the face and abdomen. The death may happen in the second or third stages according to myocarditis.

Diagnosis can only done by demonstrating the larva in the muscle by biopsy or autopsy, serological test and x- ray may used.

Trichuris trichiura: called whip worm

Geographical distribution:

Worldwide. Common in warm and moist regions.

Habitat : in large intestine of man particularly in caecumالاعور

Morphology:

Adult: the general apprearance the worm like whip, the anterior end three –fifth is very thin and hair like and the posterior end two fifth is thick like the handle of the whip.the whole anterior end consist long oesophagus and embedded in the mucous membrane of the large intestine. The thick posterior end consists the intestine and sex organs. Male its caudal extremity coiled ventrally. Female the caudal end shaped like a comma.

Eggs:

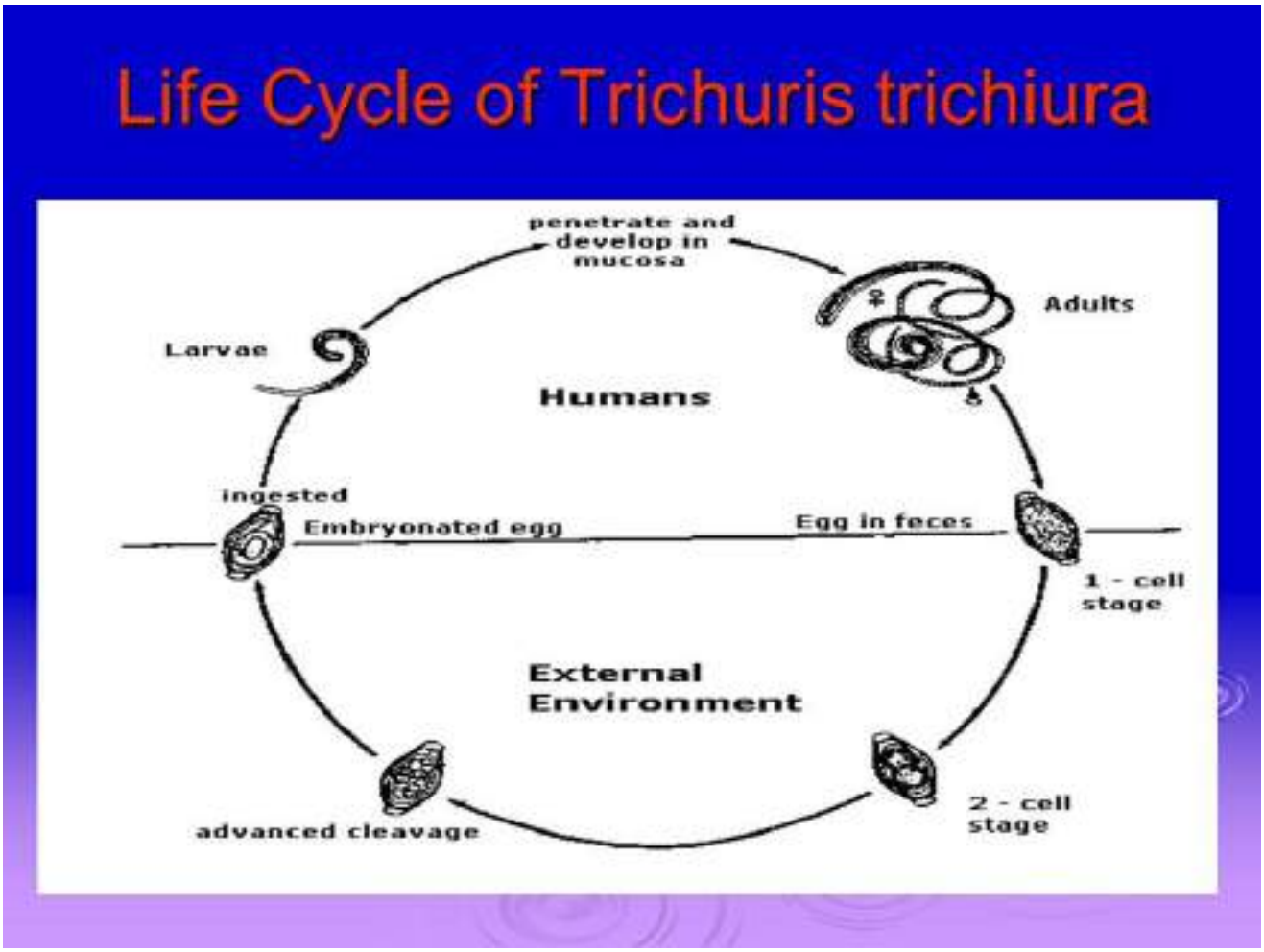
Brown (bile stained) has a double shell the outer layer is bile stained,barrel- shaped a mucous plug at each end, the notembryonated when exist.

Life cycle: no intermediate host is required. The eggs come out of human host with the faeces and the development proceeds slowly in water or damp earth depending on the environmental conditions. In tropical climate a rhabditiform larva develops within the egg in the course of 3 to 4 weeks. In temperate climate the larva takes a long time (6 to 12 months) to complete its development. The embryonated eggs are infective to man. The man is infected when the embryonated eggs are swallowed with food or water. The egg shell id dissolved by the digestive juices and the larva emerges through one of the poles of the eggs. The liberated larvae pass down into the caecum, their site of localization. They grow directly into adult worms and embed their anterior part in the mucosa of the intestine. The worms become sexualy mature within a month from the time of ingestion of the eggs and the gravid female begins to lay eggs. The cycle is then repeated.

Pathogenicity: infection with *T. trichiura* is known as tricuriasis. Usually the worms do not product y pathogenic effect. The worms inhabiting the vermiform appendix may give rise to symptoms of acute appendicitis. In heavy infections the patient often complains of abdominal pain, mucous diarrhoea often with blood streaked stool and loss of weight.

Laboratory diagnosis; this is done by finding eggs by direct microsopical examination in stool. The adult may be present in stool. Proctoscopy examination shows worm on the rectal mucosa.

Proper disposal of the night soil and prevention of eating the unwashed vegetables.


Life Cycle of Trichuris trichiura
penetrate and develop in mucosa
Adults
Larvae
Humans
ingested
Embryonated egg
Egg in feces
1 - cell stage
External Environment
advanced cleavage
2 - cell stage

Pa**thogenic Intestinal Amebae**

Genus: *Entamoeba histolytica* (the parasite causing diarrhea and liver abscess in man)

Lambl (18590nfirst discovered the parasite. Lösch (1875) proved its pathogenic nature. While Schaudinn (1903) differentiated pathogenic and non-pathogenic type of amoeba .

Geographical distribution: The parasite is world-wide. More common in tropical and sub-tropical than in temperate zone.

Habitat: Trophozoites and cyst of E. histolytica (the large race or the tissue invading forms) and cysts live in the mucous and sub mucous layers of the large intestine .

Morphology: the structural character of the parasite can be studied both in stained (iodine and iron haematoxylin) and unstained preparation there are two phase in the life cycle of the parasite:

(i)Fairly abundant in amoebic dysentery stools.

(ii) The size varies from 10-30µ

(iii) The outline shows finger like pseudopodia.

(iv) Actively motile in nature.

The cytoplasm is

(i)Ectoplasm—prominent;

(ii)Endoplasm—finely granular

(iii)Vacuoles—scanty, spherical and defined with ingested RBCs

Nuclear membrane uniformly stained with chromatin; karyosome is central. In saline preparation the nucleus is indistinct and small

The cyst is

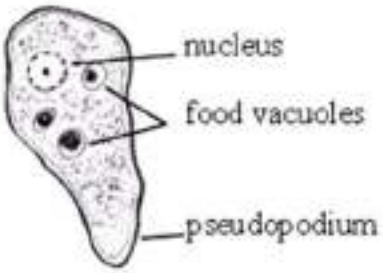
(i) Size: 6-18µ

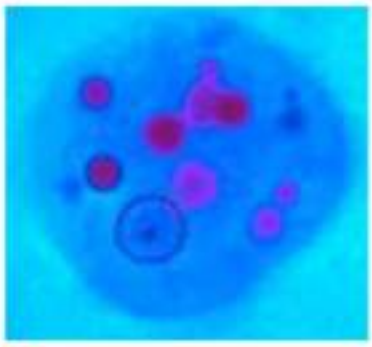
(ii) Chromatid body present with rounded ends and they are bar like.

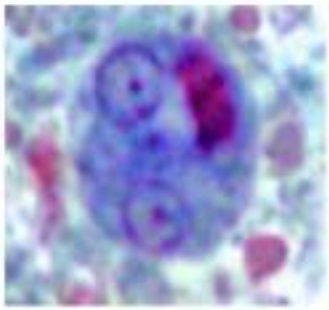
(iii) Usually one or four nuclei are seen.

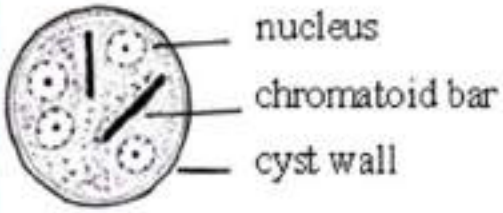
(iv) Nucleus not visible when unstained.

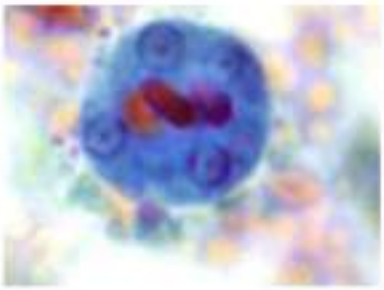
E. histolytica



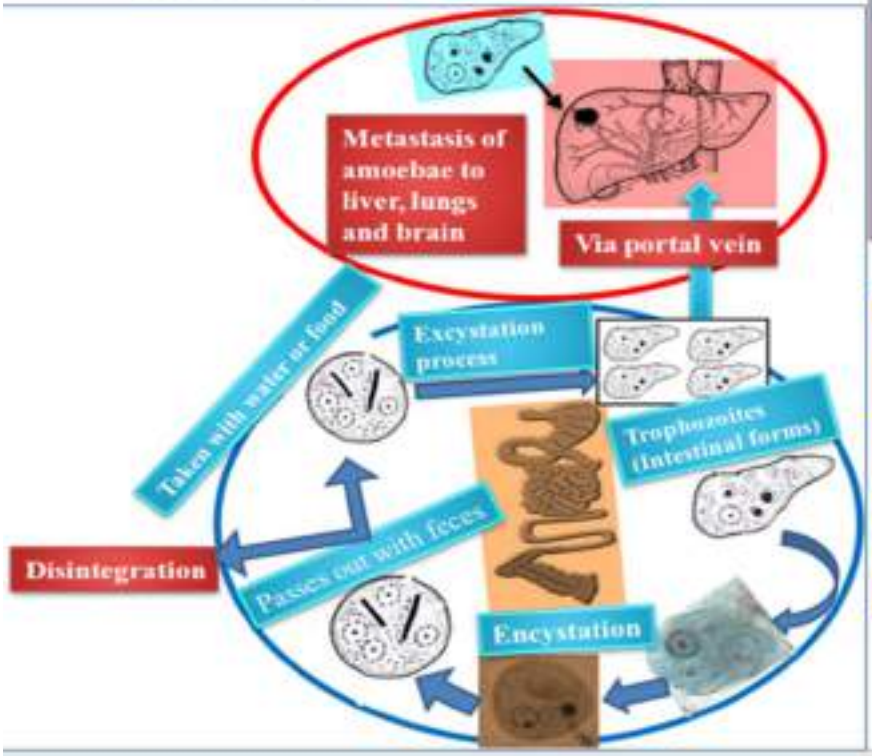








Life Cycle



Cycle: cyst—trophozite—cyst
Host: humans.
Site of infection: large intestine.
Infective stage: mature cyst (with 4 nuclei).
Infective route: mouth.

- Each mature cyst gives 4 trophozoites.
- The trophozoites adhere to the cecum, colon and rectum, and begin to divide.

Pathology:

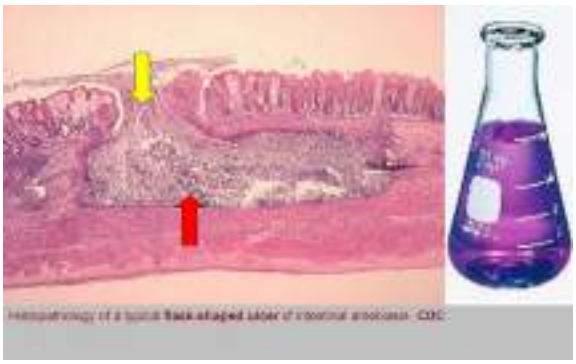
.1 Intestinal amebiasis (Primary lesion): Invasion of the wall of large intestine, – ulcer is flask shaped. Complications: Amoebic granuloma (ameboma), appendicitis, intestinal perforation.

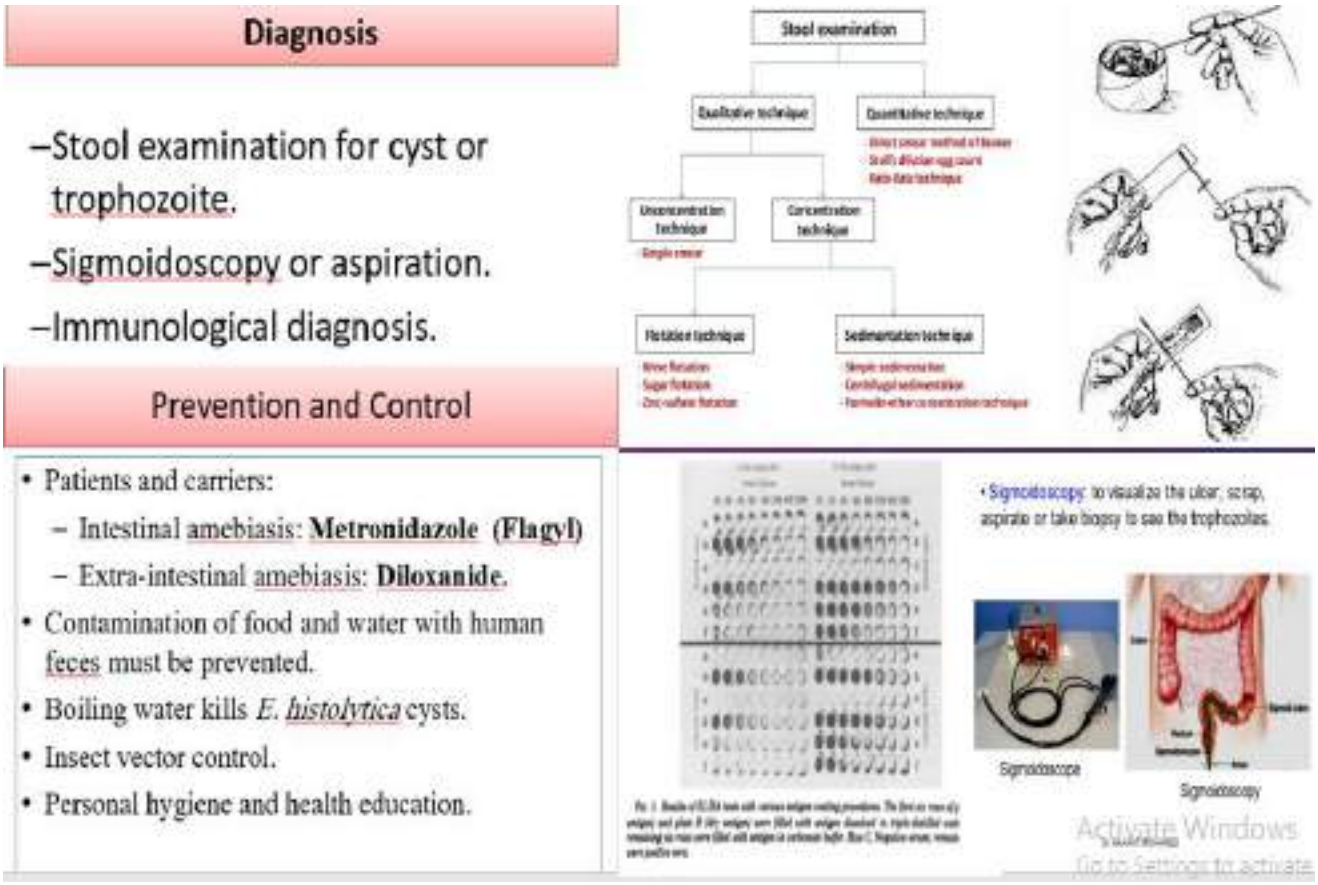
.2 Extra-intestinal amebiasis (Secondary lesions) occurs as a result of METASTASIS of trophozoites to extra-intestinal organs and cause hepatic amebiasis (abscess); pulmonary amebiasis; cerebral amebiasis; cutaneous amebiasis; spleenic abscess .

Symptoms :

Acute amebiasis: Diarrhea, dysentery (stool containing blood ,
mucous and shreds of necrotic mucosa), acute
abdominal pain, and fever .

Chronic amebiasis: Recurrent attacks of dysentery, hepatomegally and weight loss





Giardia lamblia

- **Disease:** Giardiasis or lambliasis.
- **Infective stage:** Mature cyst with 4 nuclei.
- **Site of infection:** Small intestine.
- **Life cycle**: Includes trophozoites and cysts. Trophozoites live in small intestine and attach themselves to intestinal mucosa but not invasive.
- **Treatment:** Flagyl

The trophozoites of G. lamblia are flattened pear shaped.

When stained, the trophozoite

1.has 2 nuclei, 2 slender median rods

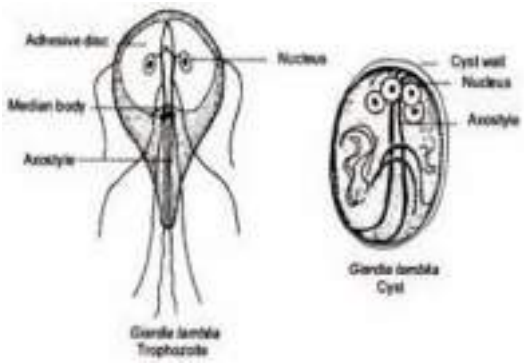
2 .(axostyles), and 8 flagella arising

from the anterior end.

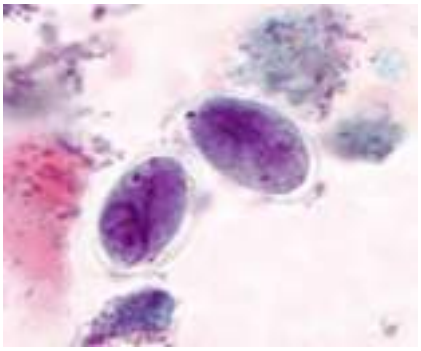
3 .The trophozoites have been described as

looking like tennis rackets without the

handle.



4. They are often have a comical face-like appearance when looking at the front view.



1.The cysts of G. lamblia are ellipsoid in shape .

2.They contain 4 nuclei which tend not to be obvious .

3.Longitudinal fibrils consisting of the remains of axonemes and parabasal bodies may also be seen .

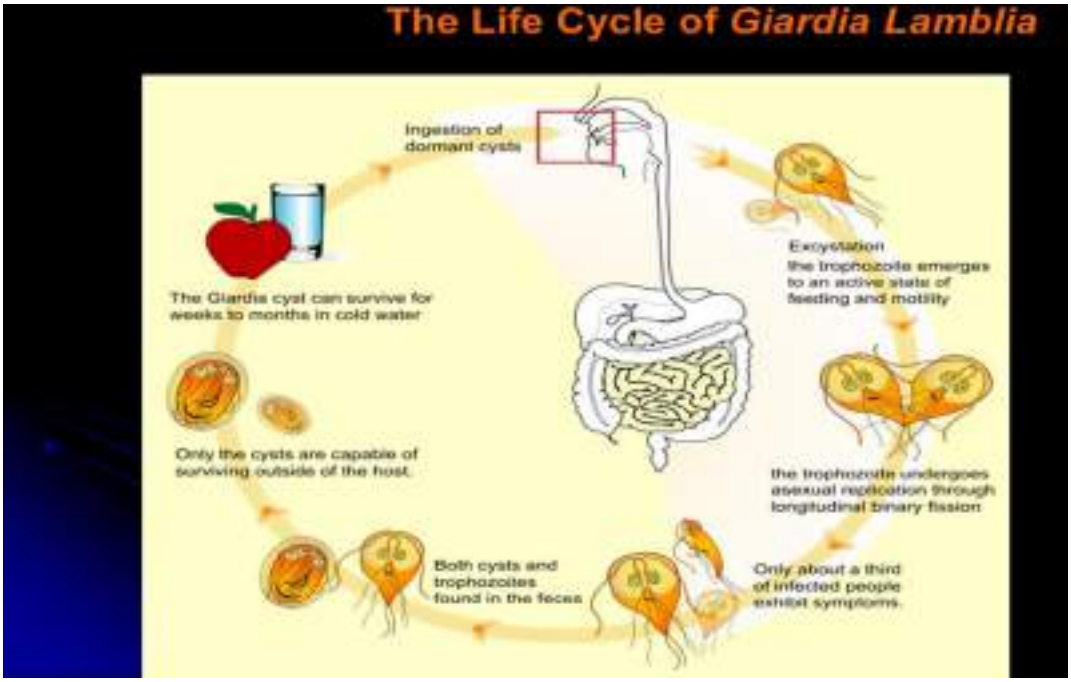
4.Cysts may appear to shrink from the cell wall .

5.The cysts are infective as soon as they are passed.

Host: Humans ;

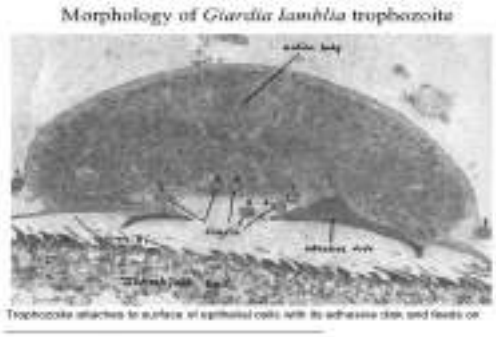
Residing site: small intestine

Infective stage: Cyst; Infective route: mouth



Pathology: Atrophy and shortening of the villi. Possibly, the mucosal abnormalities are due to mechanical, toxicity effect, and impaired absorption of vitamin B12. Uptake of bile salts by Giardia inhibits the digestion of fats by pancreatic lipase and this leads to Malabsorption syndrome (greasy stool called STEATORRHEA) .

OR MAY BE The parasite attach itself on surface of epithelial cell of intestine and in heavy infection may cause disturbance of intestinal function especially the absorbance of fat and vitamin B12



Clinical symptoms:

Diarrhea

Vomiting

Flatulence

Malabsorption syndrome

Weight loss.

Diagnosis:

1-Examination of stool for trophozoite or cyst.

2-Duodenal aspiration.

Prevention and control

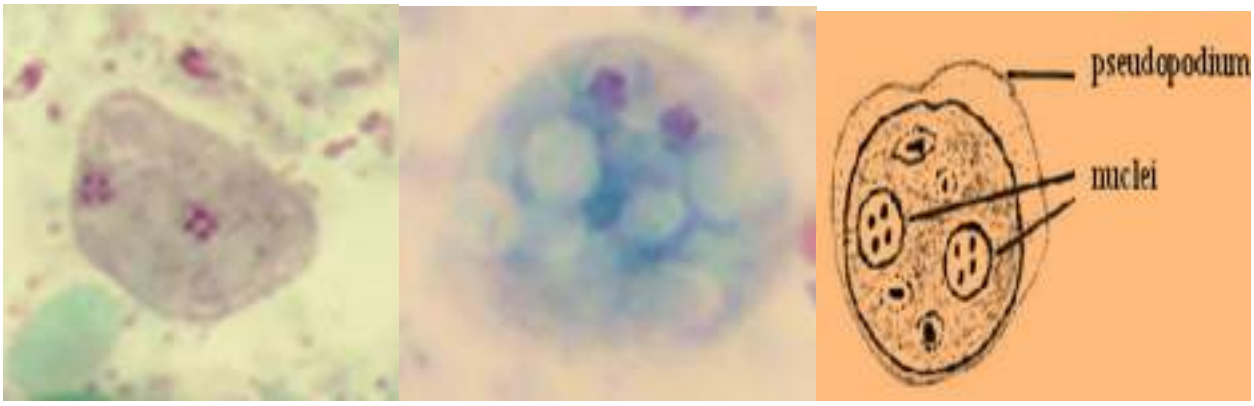
1-Patients and carriers: should treat with Metronidazole (Flagyl)

2-Insect vector control (flies, cockroaches)

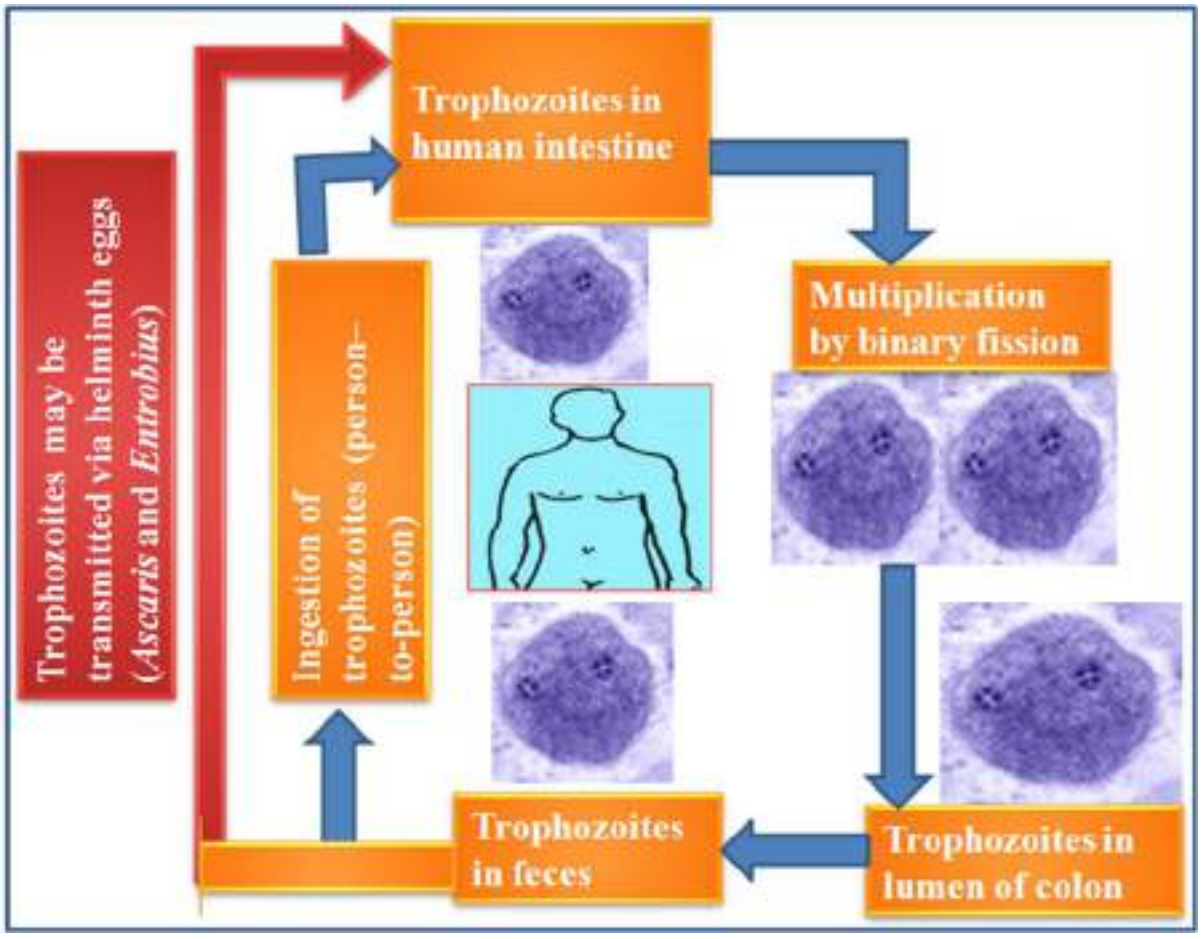
3-Personal hygiene and health education

Dientamoeba fragilis

Amoeboflagellate of the intestinal tract that is found only as trophozoite (no cyst). Has two nuclei; resembles trichomonads antigenically and ultrastructurally. Sometimes may ingest RBCs and produces a moderate, persistent diarrhea and other gastrointestinal symptoms.



The life cycle began when human ingest the troph with contaminated food and water . The multiplication occur in intestine by binary fission then the troph exist with feces . Thw troph may be transitted the eggs of some helminthes such *Ascaris* and *Enterobius*



Urogenital Flagellates

Trichomonas vaginalis has a worldwide distribution incidence is as low as 5%in normal female and high 70% among and prisoners. It is closely related to T hominis in shape it can be distinguished by having a short undulating membrane that extend only about half the distance to the posterior end of the body. It have four anterior flagella and lateral flagellum attached by undulating membrane it is pear shaped and has a single nucleus at the anterior end and two axostyle which run from the nucleus down the center of the body and extends from the end of the body and Disease: Trichomoniasis, Vaginitis, Urethritis, Prostatitis

Site of infection: Vagina, urethra, prostate

Flagellate of the lumen of the urogenital tract- vagina, urethra, and prostate .

Only trophozoite stage known to exist (absence of cyst stage) .

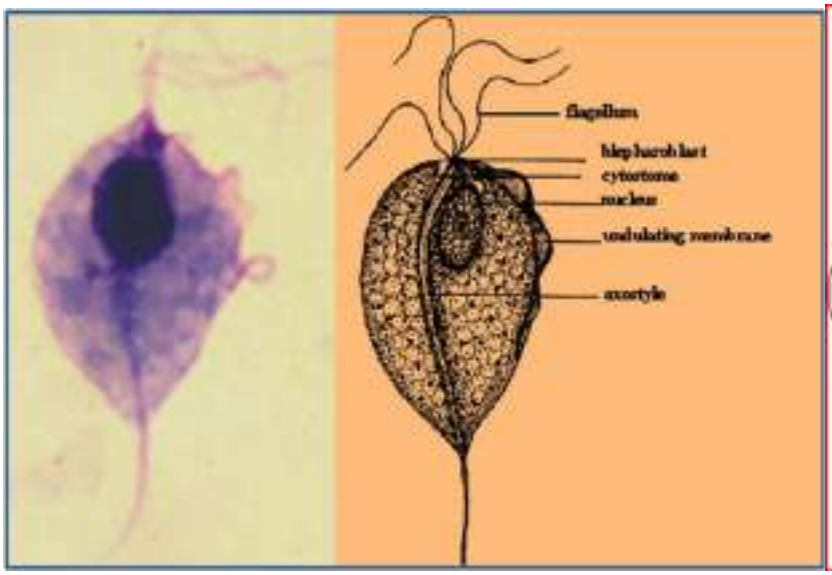
One seventh of female patients complaint of symptoms; but detection rate in their husbands are low.

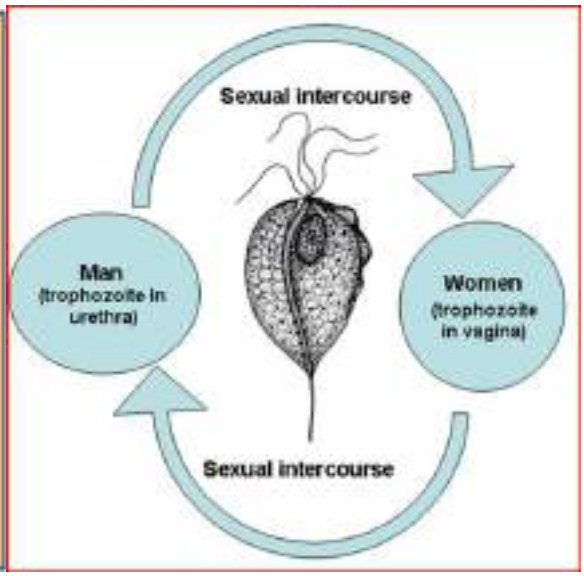
Mode of transmission: sexual contact with infected females or males, contaminated toilet articles e.g. towels, toilet seats and infection acquired in babies while passing the birth canal at birth.

Life cycle

The parasite lives in the vagina of women and the urethra (sometimes prostate) of men. Infection occurs mainly by sexual contact. The parasite does not encyst and divides by binary fission .

Pathology the infection is rarely symptomatic in men, although it may cause mild urethritis or occasionally prostatitisIn women, it is often asymptomatic, but heavy infections may cause mild to severe vaginitis (complain of itching and burning sensation) with foul-smelling yellowish, sometimes frothy discharge, and some areas become granular.





Diagnosis and Treatment

1 .Clinically, symptoms of burning sensation, frothing discharge ,

punctate lesions of the vagina .

2 .Parasitological-microscopic examination of motile parasites in fresh vaginal discharge and prostate secretion.

3. Laboratory culture: allowing the parasites to multiply in numbers and increase chance of detection.

Treatment:

1 .Metronidazole (Flagyl) is effective in both males and females.

2 .Vinegar douches: this parasite lives poorly at pH below 5.

Control :

Personal hygiene and the use of condoms are helpful.

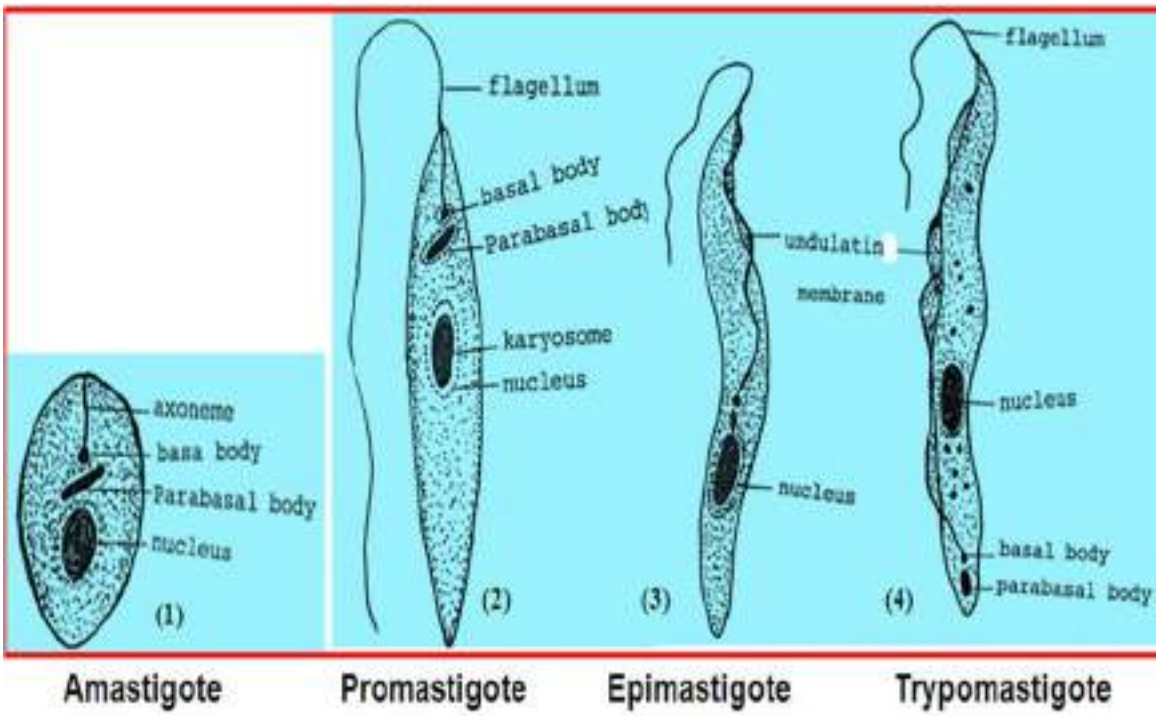
Tissue and Blood flagellates

Blood and tissue flagellate of major clinical significant include members of genera Trypanosoma and Leishmania. These two genera belong to the family Trypanosomatidae of the order Kintoplastida

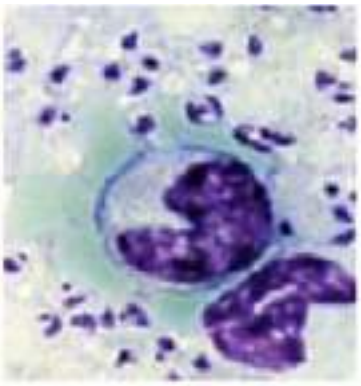
.The member of this family are characterized of being parasitic and possess a single flagellum and a DNA containing organelle called kintoplast which is located close to the flagellar basal body.

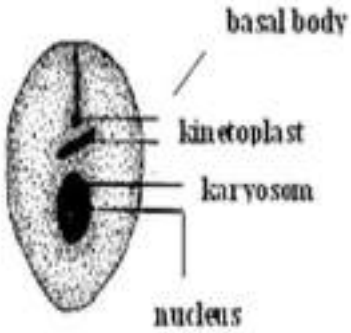
Hemoflagellates may be present in any of four different stages (amastigotes, promastigotes, epimastigotes and trypomastigote) which are named according to their flagellum

Morphological types seen in various hemoflagellates of humans

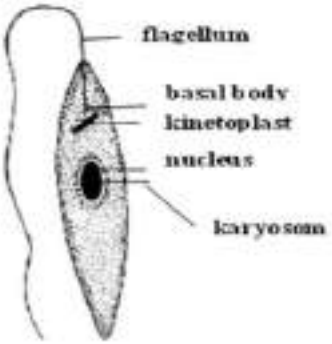


The amastigote forms (Leishman-Donovan body) are small, round or oval bodies and reside in macrophages. The large nucleus (located in one side) and small kinetoplast can be seen when stained with Giemsa stain. No free flagellum.

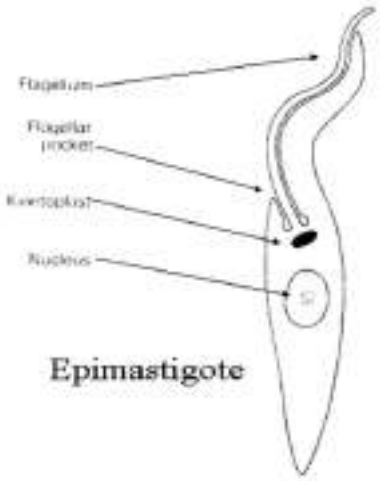




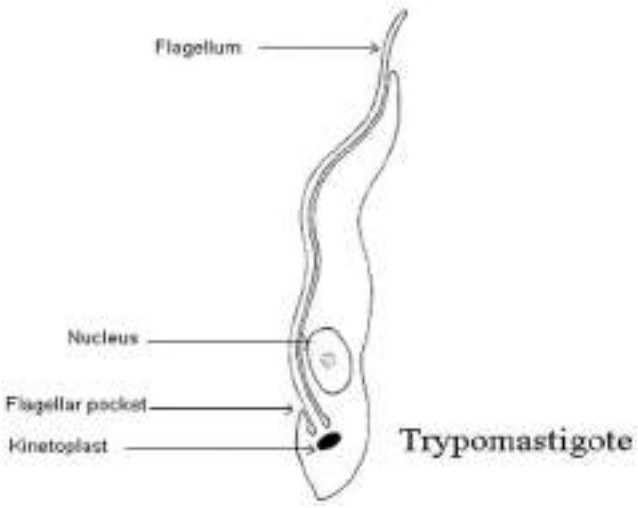
The promastigote stage (leptomonad) is a motile, slender organism with a single anterior flagellum without undulating membrane and multiplies by longitudinal fission in the gut of the insect. The kinetoplast located in the anterior end of the body



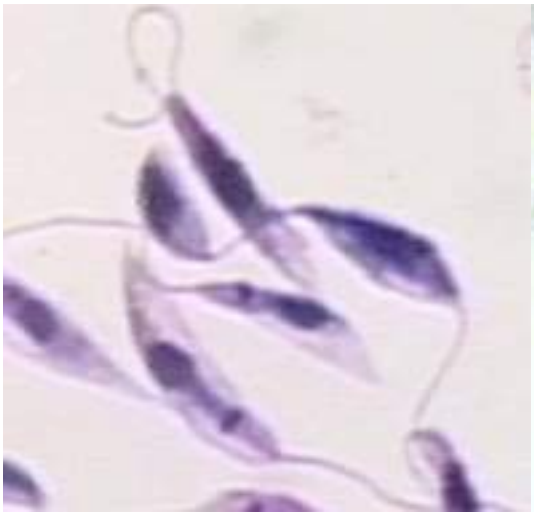
The epimastigote forms (crithidia body) has a free flagellum with undulating membrane. The kinetoplast located above the nucleus and basal body and extends to anterior end of the body



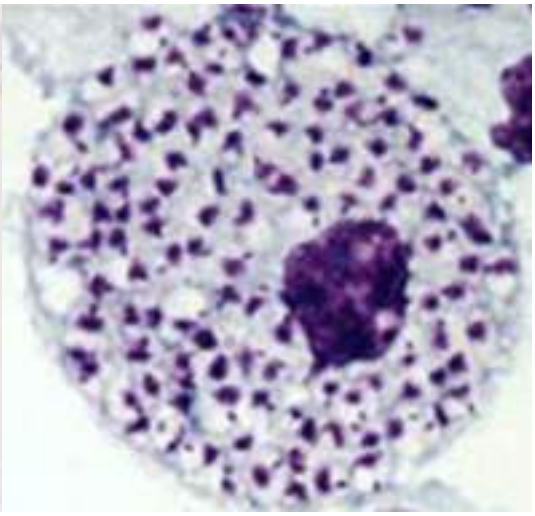
The trypomastigote forms (tryposome) has a free flagellum with an undulating membrane that extends along the body. The kinetoplast located behind the nucleus and basal body and extends to anterior end of the body



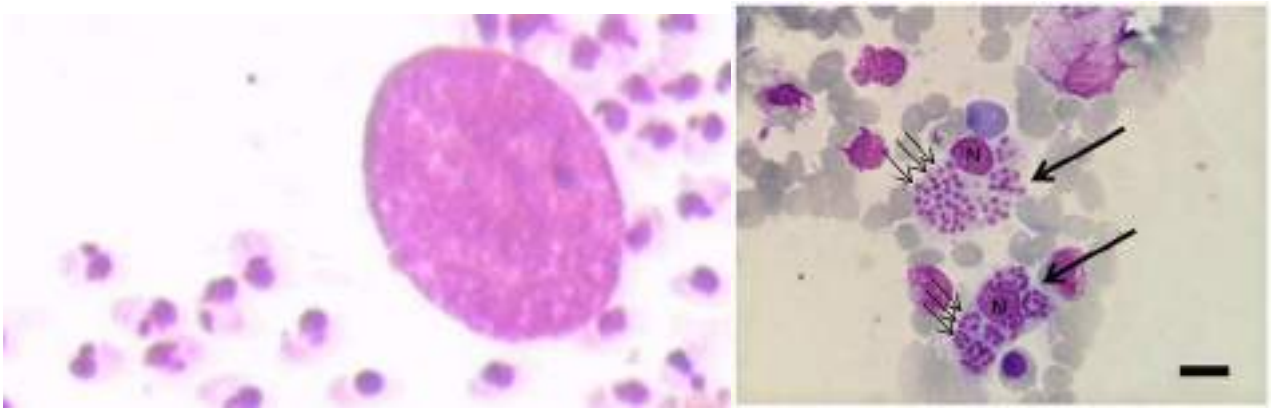
Leishmania spp.
Leishmania donovani:Causes a disease called Visceral leishmaniasis, Kala-azar, Black fever, or Dum-Dum fever.
Leishmania tropica: Causes a disease called Cutaneous leishmaniasis, Baghdad boil, or Delhi boil.
Leishmania brasiliensis: Causes a disease called Muco-cutaneous leishmaniasis.
Zoonotic parasites.
Visceral leishmaniasis is one of the 5 major parasitic diseases.
Both visceral and cutaneous leishmaniasis are endemic in south and middle of Iraq.



Leishmania **promastigotes (culture form)**



Leishmania **amastigotes (Giemsa stain)**



Leishmania **amastigotes, bone marrow aspirate (Giemsa stain)**

Human leishmaniasis is a group of diseases. These diseases are caused by infection with protozoan parasites from the Leishmania genus. Most species of Leishmania are zoonotic (affecting animals and human) many species of Leishmania are pathogenic for human. Leishmania donovani causes a disease called Visceral leishmaniasis, Kala-azar, Black fever, or Dum-Dum fever.

Leishmania tropica: Causes a disease called Cutaneous leishmaniasis, Baghdad boil, or Delhi boil.

Leishmania brasiliensis: Causes a disease called Muco-cutaneous leishmaniasis or uta or chiclero.

Visceral leishmaniasis is one of the 5 major parasitic diseases. The geographical distributin of leishmaniasis is limited by distribution of the sandfly, its susceptibility to cold climate, its tendency to take blood from humans or animals only and its capacity to support the internal development of specific species of Leishmania. Both visceral and cutaneous leishmaniasis are endemic in south and middle of Iraq.

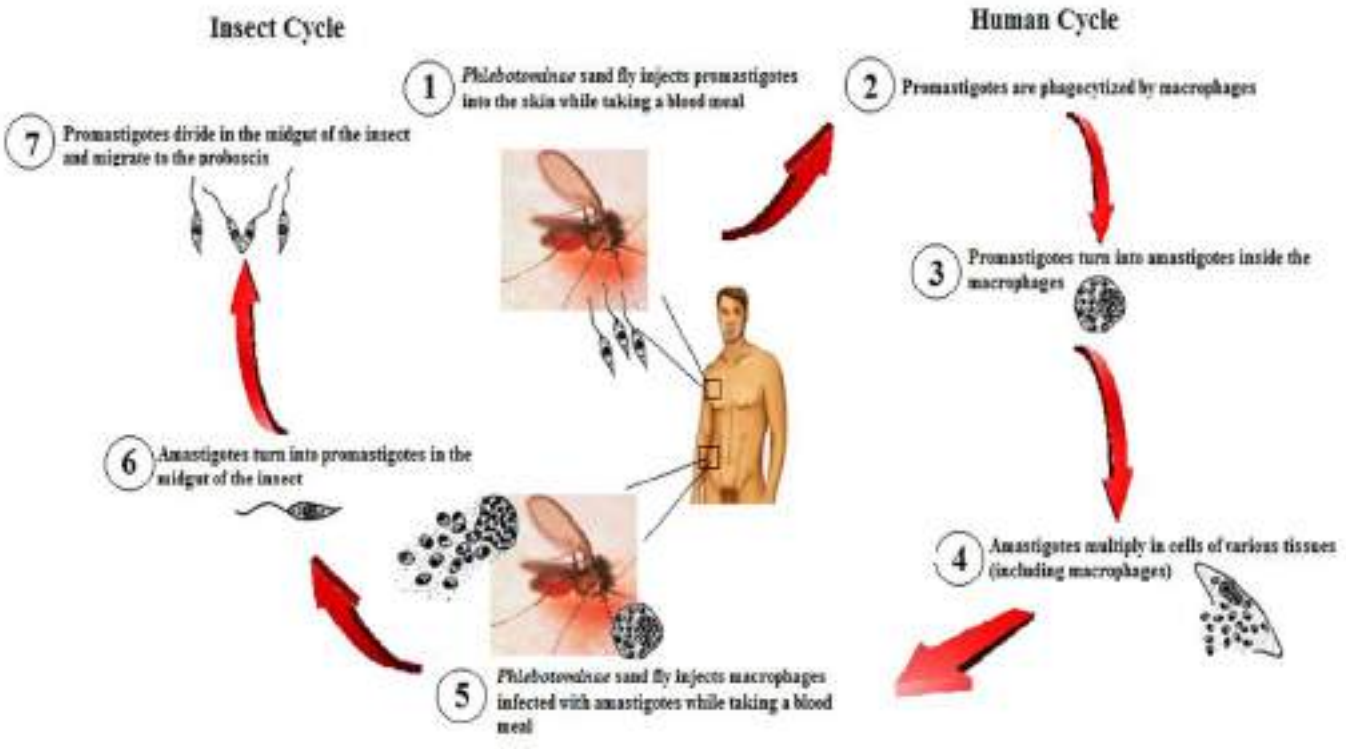
life cycle of Leishmania parasites has two distinct forms, amastigote and promastigote .

The parasites are transmitted by the bite of sandflies (Phlebotomus) which carry the promastigote in the anterior gut and pharynx .

During feeding, infected female sandflies inject the promastigotes into the skin of the host. When the promastigotes are engulfed by the macrophages, they transform into amastigotes which multiply by binary fission within the parasitophorous vacuole of the macrophage until the infected cell ruptures .

The librated amastigotes infect other cells and the infection spreads by movement of infected cells in the vascular system. The sandfly takes the amastigotes during the blood meal which transform back into promastigotes and multiply in the gut until the anterior gut and pharynx are packed .

The infected sandfly injects the promastigotes into a new host when it feeds. Dogs and rodents are common reservoir hosts.



Main characters of life cycle

Hosts: Humans and sandflies

No sexual development in the host

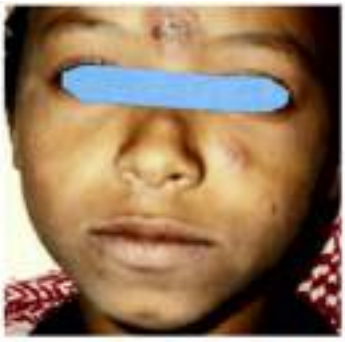
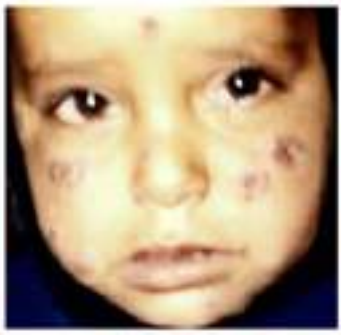
Residing site: Macrophages

Infective stage: Promastigote for humans and amastigotes for sandflies

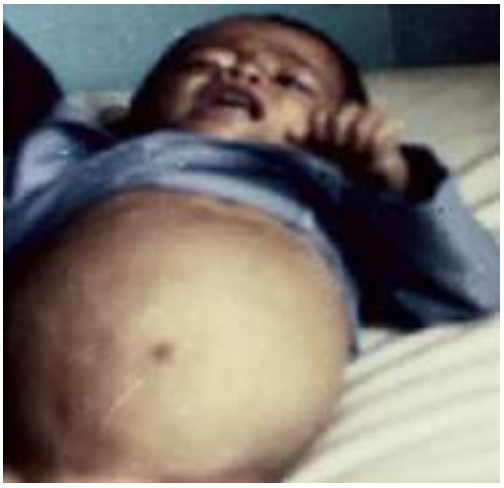
Infective route: Bite of sandfly

Reservoir host: Dogs and foxes

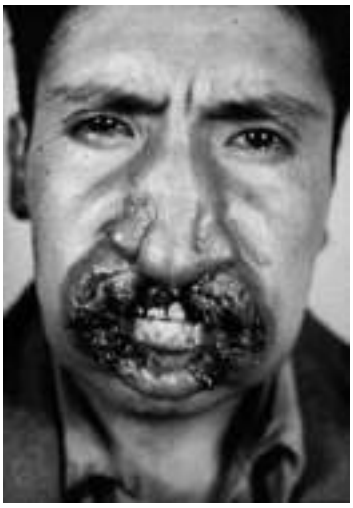
Other routes of infection: Blood transfusion

Cutaneous Leishmaniasis



Visceral Leishmaniasis



Mucocutaneous Leishmaniasis

Symptoms:

In visceral leishmaniasis amasitoge located and multiply in the mononuclear phagocytic cells of the liver , spleen ,lymph nodes, bone marrow, intestinal mucosa and other organs. The incubation period may vary between 2 weeks and 18 months. The following symptoms can be seen:

1 -abdominal enlargement due to the enlargement of liver and spleen

2-fever which may be continuous, intermittent, or remittent and happens again at irregular intervals

3-hepatomegaly and splenomegaly

4-with progression of the disease , skin develops hyper pigmented granulomatous (kala azar means black disease)

5-weight loss

6-anemia

7-untreat cases result in death

Cutaneous leishmaniasis

The incubation period is variable and may be a couple of months or as long as 3 years in L tropica and L athiopica infection in L major it is much shorter (about 3 weeks) .The following symptoms can be seen:

1-the first sign is the small red papule that gradually grows to form a relatively painless ulcer (some time itches). This ulcer heals in 2-10 months even if untreated but leaves a disfiguring scar.

2-in L major infection , the papule is covered with a serous exudate and ulcerates early whereas the papules are dry and ulcerate only after several months in L tropica and L athiopica infection

3-the disease may disseminate in people with a depressed immune function

4-host recovery in cutaneous leishmaniasis depends on the development of cell mediated

Mucocutaneous leishmaniasis

This disease is a chronic form of leishmaniasis cuased by L athiopica in athiopia and kenya and by many subspecies of L mexicana in central and south America it characterized

the destruction of the oropharynx and nose, resulting in extensive midfacial destruction. The skin lesion with mucocutaneous disease is often notable for its prolonged healing time and large size. In most cases, a healed scar can be identified based on careful examination

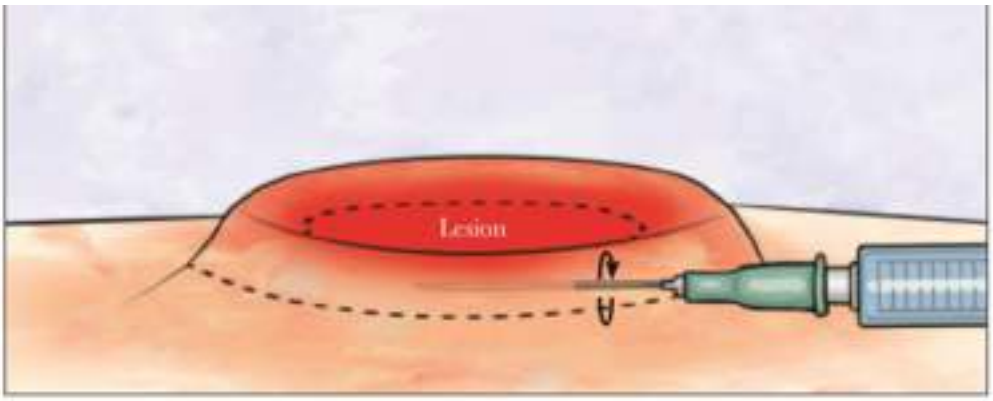
Diagnosis: Puncture smear: Bone marrow: safe, first choice

Skin biopsy

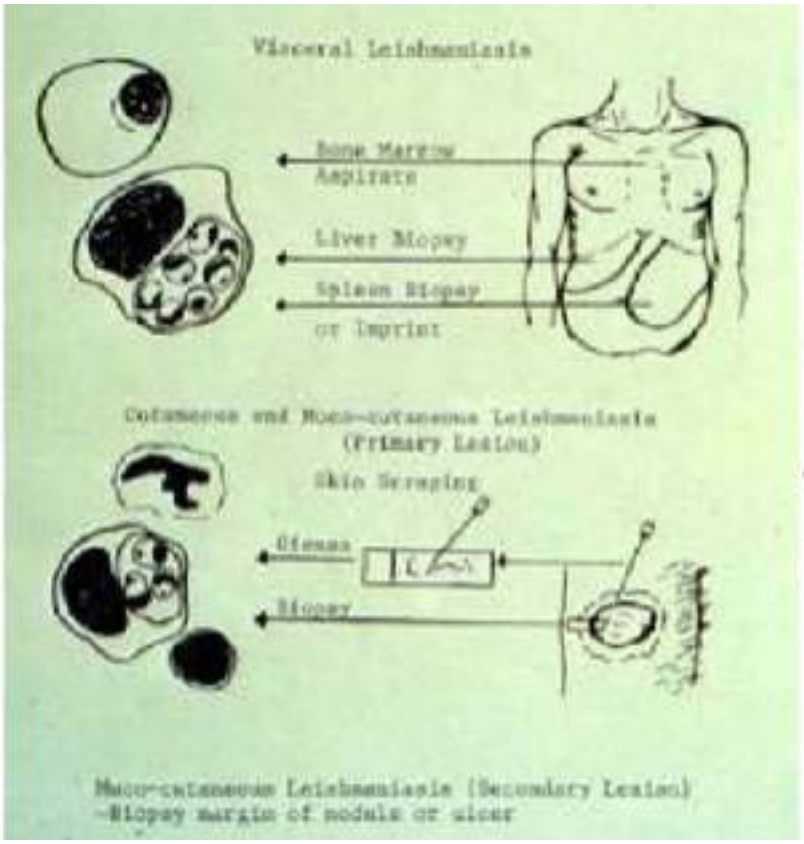
Tissue cultivation

Animal inoculation

Molecular (Probe) test: DNA



NOTE: If pertinent, consult CDC about obtaining/handling other types of fluids/aspirates (e.g., bone marrow).



Methods for Direct Tissue Examination for Leishmaniasis

Control: Treatment of patients: Sodium Stibogluconate (Pentostam)

Killing of infected dogs

Eradication of sandflies

Diphylobothrium latum

الدودة الشريطية السمكية العريضة

دودة السمك الشريطية (ديفيلوبوثريم لاتم)

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Subclass: Eucestoda

Order: Pseudophyllidea

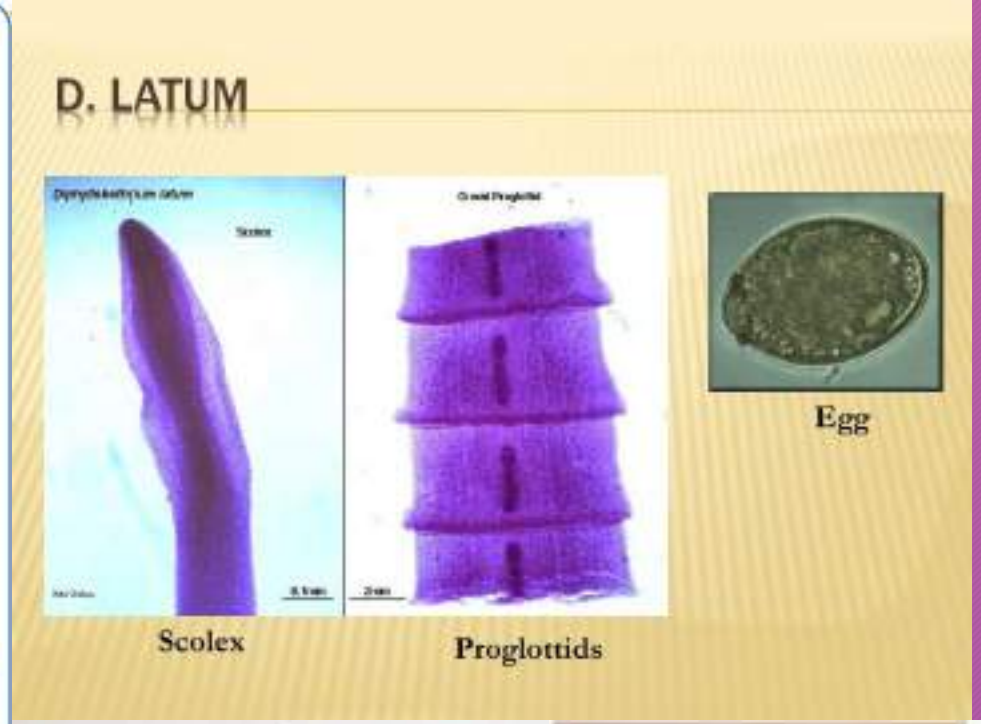
Family: Diphyllobothriidae

Genus: *Diphyllobothrium*

Diphyllobothrium latum

- يطلق على هذه الدودة الشريطية **دودة السمك الشريطية لأن الإنسان يصاب بالعدوى اذا ما تناول فى طعامة سمك المياه العذبة الذى يحتوى على الطور اليرقى المعدى**
- ويطلق على هذه الدودة فى بعض الاحيان **الدودة الشريطية العريضة** لان قطعها العرض اكبر من طولها
- وتعرف أيضاً **بالدودة الشريطية ذات الحفرتين الممصتين**
- وهى توجد فى **الأمعاء الدقيقة للانسان والكلاب والقطط والثعالب والخنازير والدببه وغيرها من الحيوانات اللاحمه التى تتغذى على الاسماك**
- وتنتشر الاصابه بهذا النوع من الديدان فى وسط اوروبا وخاصة فنلندا والاقطار المطله على شواطئ بحر البلطيق وايرلندا وسط افريقيا واليابان والفلبين ومنطقة البحيرات العظمى فى امريكا الشمالية
- وتعيش الدودة البالغة لهذا النوع من الديدان فى الإنسان لمدة قد تصل إلى **عشرين سنة** وهى **تعتبر اكبر دودة شريطية موجوده فى الإنسان**

تسمى بالدودة الشريطية العريضة
Broad tapeworm
او دودة السمك الشريطية العريضة
Broad fish tapeworm
تكثر الإصابة في المناطق التي يشيع فيها تناول الأسماك النيئة ةالمطبوخة بصورة غير جيدة في اوربا واسيا وامريكا وافريقيا



الدودة البالغة ذات لون اصفر او عاجي وهي أطول دودة شريطية تصيب الانسان تتكون من قطع جسمية (تسمى في بض المصادر الاسلات) (proglotitde)

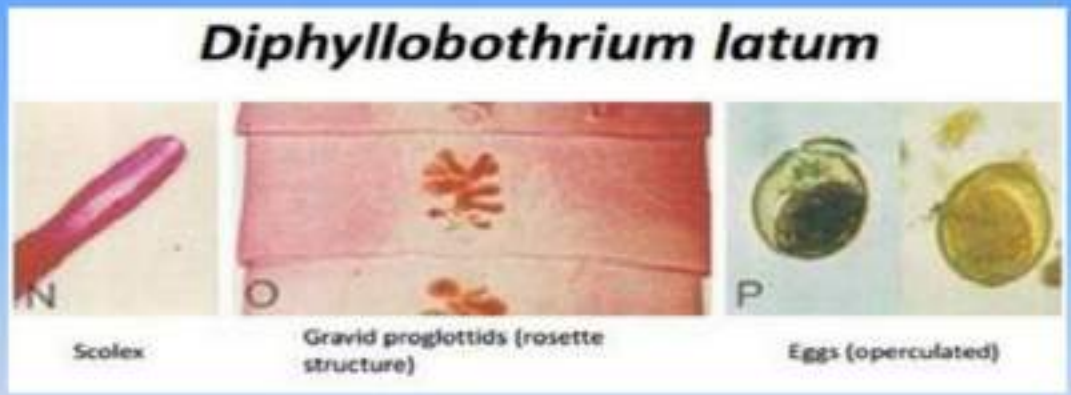
اذ يكون عدد القطع الجسمية او الاسلات حوالي 4000-3000 اسلة او قطعة

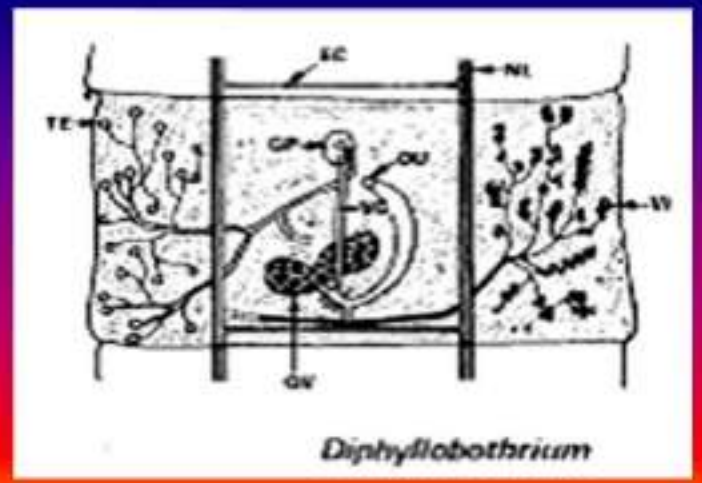
الراس متطاول او مستطيل او لوزي الشكل مزود باخدودين ماصين عميقين احدهما على الجهة البطية والأخر على الجهة الظهرية

القطع الناضجة يكون عرضها اكبر من طولها لهذا تسمى بالدودة الشريطية العريضة تقع الفتحة التناسلية المشتركة عند الناحية البطنية في الخط الوسطي في مقدمة القطعة والى الخلف منها قليلا هناك فتحة الرحم والذي يعد صفة تشخيصية نظرا لكونه قاتم اللون زهري الشكل واقع في وسط القطعة الناضجة البيوض ذات غطاء غير واضح في احدى النهايتين اما النهاية الأخرى فتحوي على تثخن يشبه العقدة ولون البيوض بني مصفر وتحوي على قشرة

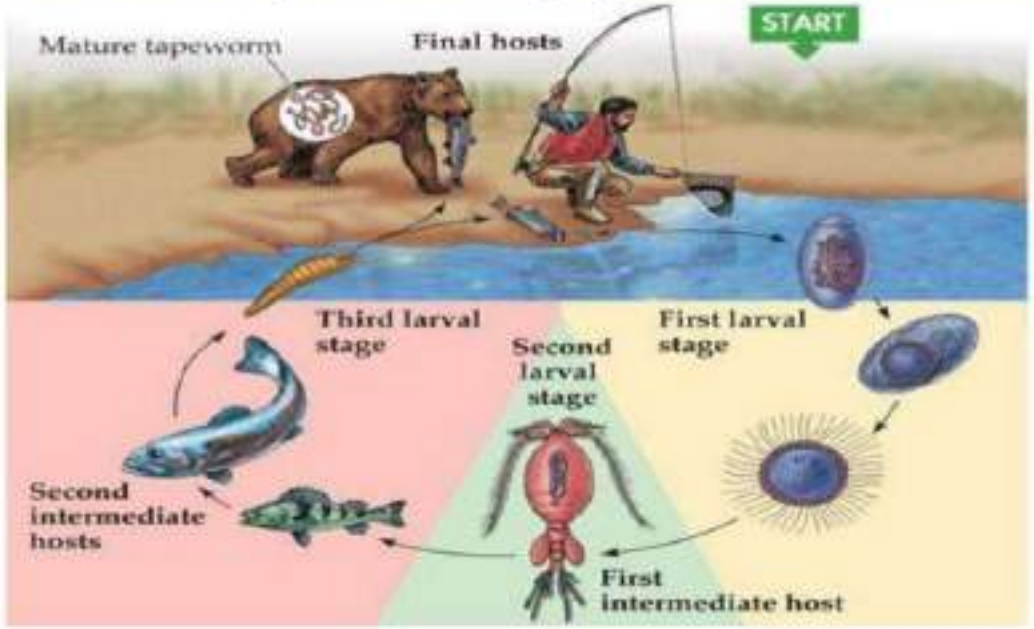
- والخصيات والغدد المحية منتشرة فى جوانب الأسلة
- **ويتكون المبيض من فصين يوجدان فى الجزء الخلفى من الأسلة** امام غدة مهليس ولا تنتج الدودة الشريطية العريضه البويضات المخصبة فى القطع النهائية فقط كما هو الحال فى ديدان البقر والخنزير الشريطية **ولكنها تنتج البويضات المخصبة بصفة دائمة ولذلك لا توجد بهذه الدودة اسلات مثقلة لان البيض يخرج من فتحة الرحم بمجرد تكونه ؟**
- **البيضه :-** بيضاوية الشكل ويبلغ طولها 55-75 ميكرون وعرضها 40-55 ميكرون **ولوهنا بنى مصفره وهى مزودة بغطاء فى احد طرفيهما وتحتوى على جنين غير تام النمو**

- وينمو فى كل قطعة كما هو الحال فى قطع الديدان الشريطية الاخرى كلا **الجهازين التناسلين الذكرى والانثوى فى وسط القطعه ولا تفتح فى الحافة الجانبية للقطعه كما يحدث فى الاجهزةالتناسلية لديدان البقر والخنزير الشريطية**
- ويقع **الرحم فى منتصف كل قطعه من القطع الناضجة** أو البالغة وهو يبدو على شكل انبوبة ملتوية ويظهر عند امتلائه بالبويضات على **شكل ورده سوداء** إلى حد ما فى منتصف كل قطعة
- وعلاوة على ذلك فإنه يختلف عن ديدان جنس (التينيا) بأنه **يفتح الرحم إلى الخارج وفتحة الرحم الخارجية تقع فى منتصف القطعة قريبا من الفتحات الانثوية والذكرية**





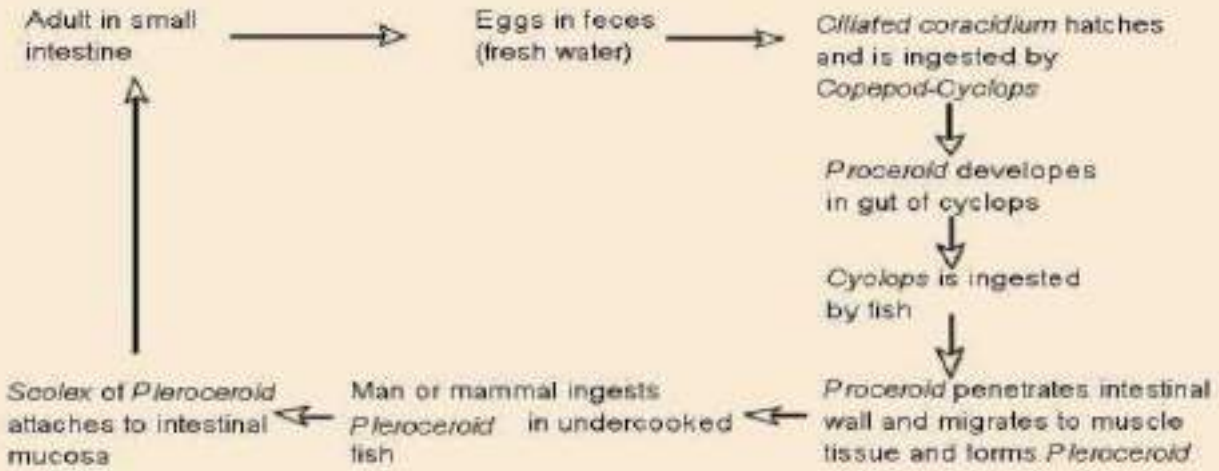
Broad fish Tapeworm *Diphyllobothrium latum*



دورة الحياة

- تخرج البويضات من فتحة الرحم وتختلط هذه بمحتويات القناة الهضمية للعائل النهائى وتخرج مع البراز إلى خارج الجسم ويصل عدد البيض الذى تضعه الدودة الناضجة حوالى مليون بيضة يوميا
- البويضات لا تكون ناضجة فور خروجها مع البراز ولكنها تحتاج لمدة أسبوعين أو ثلاثة اسابيع فى الماء حيث تنمو البيضه وتفقس ويخرج منها حيوان صغير يسمى كوراسيديوم corasidum وهو حيوان مستدير الشكل قطره حوالى 80-90 ميكرون ويحتوى على الجنين ذو الستة اشواك ومحاط بغطاء الجنين وهذا مزود بعديد من الاهداب
- يسبح الكوراسيديوم قريبا من سطح الماء ويعيش فقط لمدة 12-24 ساعة ويموت اذا لم تبتلعة العوائل الوسيطة والعوائل الوسيطة التى يستعملها هذا النوع من الديدان الشريطية هو أنواع من قشريات المياة العذبة التى تتبع مجدافية الارجل (الكوبيبودا Copepoda) وتنمتى إلى جنسين فقط هما : سيكلوبس وديابتومس .

Diphyllobothrium latum Life Cycle



- تبتلع القشريات الكوراسيديوم الذى يفقد غطاء الجنين بما عليه من أهداب فى أمعاء الحيوان القشرى ثم يخترق جدار الامعاء لهذا الحيوان يساعدة فى ذلك الستة خطاطيف حتى يصل إلى تجويف الجسم حيث ينمو فى خلال 3 أسابيع إلى الطور اليرقى الأول المسمى بروسيركويد procercoid وهذه اليرقة بيضاوية الشكل ويتراوح طولها بين 100 500 ميكرون ولها جزء أمامى منقبض وجزء خلفى مستدير يحتوى على الستة خطاطيف التى كانت موجودة أصلا فى الجنين
- عندما يبتلع الحيوان القشرى المحتوى على البروسيركويد التام النموبواسطة أنواع معينة من الاسماك مثل السلمون والثعابين واسماك أخرى قريبة منها (العوائل الوسيطة الثانية) فان الحيوان القشرى يهضم فى أمعاء السمك وينطلق البروسيركويد حيث يفقد الخلفية ويخترق جدار الامعاء ويستقر فى بعض الانسجة مثل العضلات وبعض أعضاء السمكة الداخلية وهناك تنمو فى مدة تتراوح بين سبعة – ثلاثين يوما إلى الطور اليرقى الثانى المعروف بيرقة بليروسيركويد plerocercoid .

وعندما ياكل الانسان الأسماك النيئة او غير المطبوخة بصورة جيدة تنشط هذه اليرقة داخل امعائه وتنمو الى ديدان بالغة خلال 3 اسابيع

- يرقة **البليروسيركويد** لها رأس مزود بحفرتين ممصيتين ولها جسم مستطيل عليه انكماشات جانبية عديدة ولكنة غير مقسم وهو طويل نسبيا ويبلغ طوله من 1-2 سم وعرضة من 2-3 ملليمتر
- وهذا هو الطور المعدى للانسان أو أى عائل نهائى أخر والسمك فى هذه الحالة يعتبر عائل وسيط ثانى
- **وقد تبتلع سمكة كبيرة السمكة الصغيرة المصابة وفى هذه الحالة لا تنمو يرقة البليروسيركويد فى السمكة الكبيرة ولكنها تستطيع أن تعيش فيها وتكون مهمة السمكة الكبيرة فى هذه الحالة هو نقل العدوى على نطاق أوسع والمقدرة على الانتقال من عائل وسيط ثانى إلى عائل وسيط اخر يزيد من فرص عدوى هذه الدودة الشريطية لعوائلها النهائية وتعويضها عن النقص الناجم من استعالمها لعدد قليل من العوائل الوسيطة الاولى .**

- يصاب الإنسان بالعدوى اذا ما أكل سمك غير مطهى جيدا ومصاب بالطور المعدى
- وفى هذه الحالة تثبت يرقة البليروسيركويد نفسها بجدار الامعاء ويساعدها فى ذلك الحفر الممصية حيث تنمو وتبدا فى تكوين الاسلات
- و تنتقل العدوى كذلك عندما يقوم الناس بتنظيف الاسماك المصابة وتنتقل البليروسيركويد إلى ايديهم ومنها إلى افواههم
- ويبدا ظهور البيض فى البراز 5-6 اسابيع بعد اكل وجبة من سمك مصاب بالطور المعدى
- **العلاقة بين العائل والطفيل** : قلما تصيب هذه الدودة الإنسان بضرر ظاهر وخاصة ان الإنسان لا يصاب باكثر من دودة واحدة ولكن قد يصاب الإنسان بفقر دم ورغبة متزايدة للاكل مع اسهال والام فى البطن وقد يصاحب ذلك نقص فى الوزن.

تقتصر الإصابة بهذه الدودة على المناطق التي يؤكل بها السمك نيا او مطبوخ بشكل غير جيد كما تحدث في الأشخاص الذين يقومون بتنظيف الأسماك في الأسواق او المصانع والنساء اليهوديات اثناء اعداد السمك للطبخ من خلال تذوق السمك نيا بعد إضافة الملح والتوال فتلتصق اليرقة باللسان وقد تصل نسبة الإصابة في بعض مجتمعات البلطيق الة 100% وذلك يعود الى تلوث البحيرات والجداول بنفايات المجاري القادمة من المدن الصغيرة كماتساعد الكلاب والقطط التي تاكل نفايات الأسماك من بقايا المعسكرات والبيوت والفنادق على ديمومة دورة الحياة

التاثيرات المرضية قد لاتظهر اعراض عند الإصابة بدودة واحدة ولكن في الإصابات الشديدة يظهر

انسداد الامعاء التهاب في الغشاء المخاطي زيادة خلايا الدم البيض الحامضية الم في لبطن غثيان توتر عصبي ضعف وقلة شهية نقص الوزن سزء تغذية فقر دم

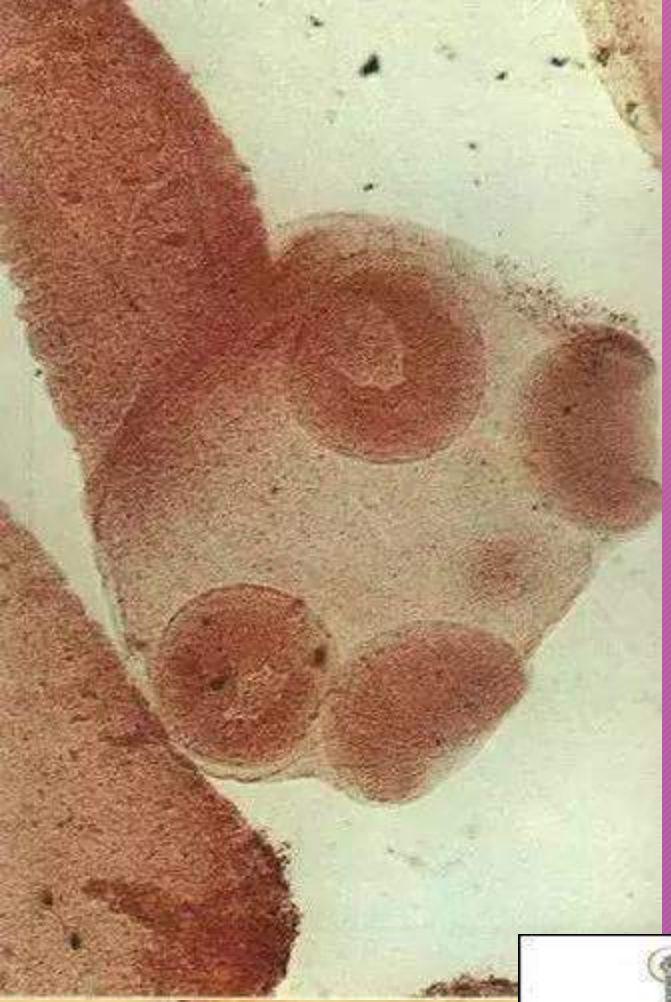
يعود فقر الدم المشابه لفقر الدم الخبيث الى قيام الدودة بامتصاص 80- 100% من فيتامين B12 و قد سجل ان ثلث حالات فقر الدم الخبيث في فنلندا يعود الى الإصابة بهذه الدودة

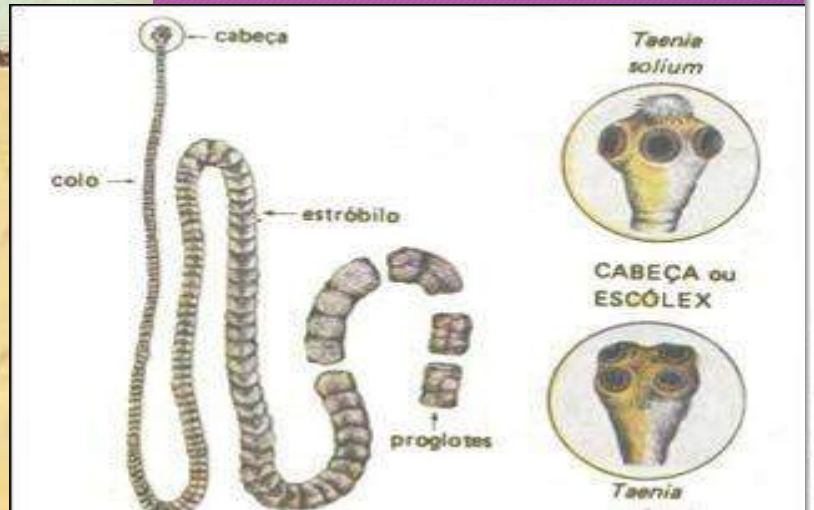
عدم رمي نفيات الأسماك (بقايا الاحشاء بعد الصيد)الموجود في الأمعاء
اخذ الحيطة عند تنظيف وطبخ الأسماك
تجنب اكل الأسماك نية او غير مطبوخة بشكل جيد
معاملة فضلات الانسان قبل طرحا في الماء او البرك

Taenia saginata

و

Taenia solium





رتبة Order Cyclophyllidea

تشمل هذه الرتبة بأجناس *Taenia* و *Echinococcus* و *Dipylidium* وفيما يلي توضيح لهذه الديدان .

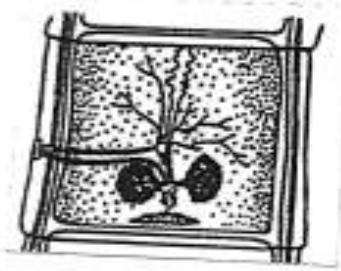
الدودة الشريطية البقرية *Taenia saginata*

تعرف الدودة الشريطية البقرية Beef tapeworm بالدودة العزلاء Unarmed .

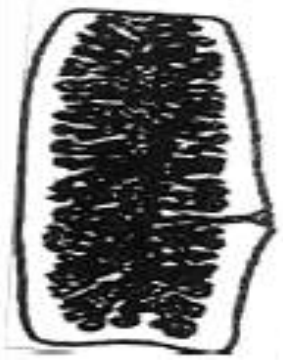
تعيش الدودة البالغة في أمعاء الإنسان أما الطور اليرقي فيصيب عضلات وقلب الأبقار وهي ذات انتشار عالمي و خاصة في المناطق الإسلامية التي يؤكل فيها اللحم نيئاً او مطهياً بصورة غير جيدة . وتعزى أصابة العديد من الأطفال بهذه الدودة الى العادات القديمة التي يُعطى فيها الأطفال قطعة من لحم البقر النيء أثناء فترة التسنين حيث يعتقد أن ذلك يساعد ظهور الأسنان .

المظهر الخارجي : يتراوح الدودة البالغة بين 4-8 متر و نادراً ما يصل إلى 15 متراً .

الرأس هرمي الشكل يحمل أربعة ممصات واضحة و لكنه لا يحوي على الخطم و لا الكلاليب . يشتمل الجسم على 1000-2000 قطعة .



القطع الناضجة ذات فتحات تناسلية جانبية غير منتظمة التبادل في الموقع . ويتراوح عدد الخصى فيها بين 300-400 خصية المبيض ثنائي الفص و الغدد المحية تقع خلف المبيض .

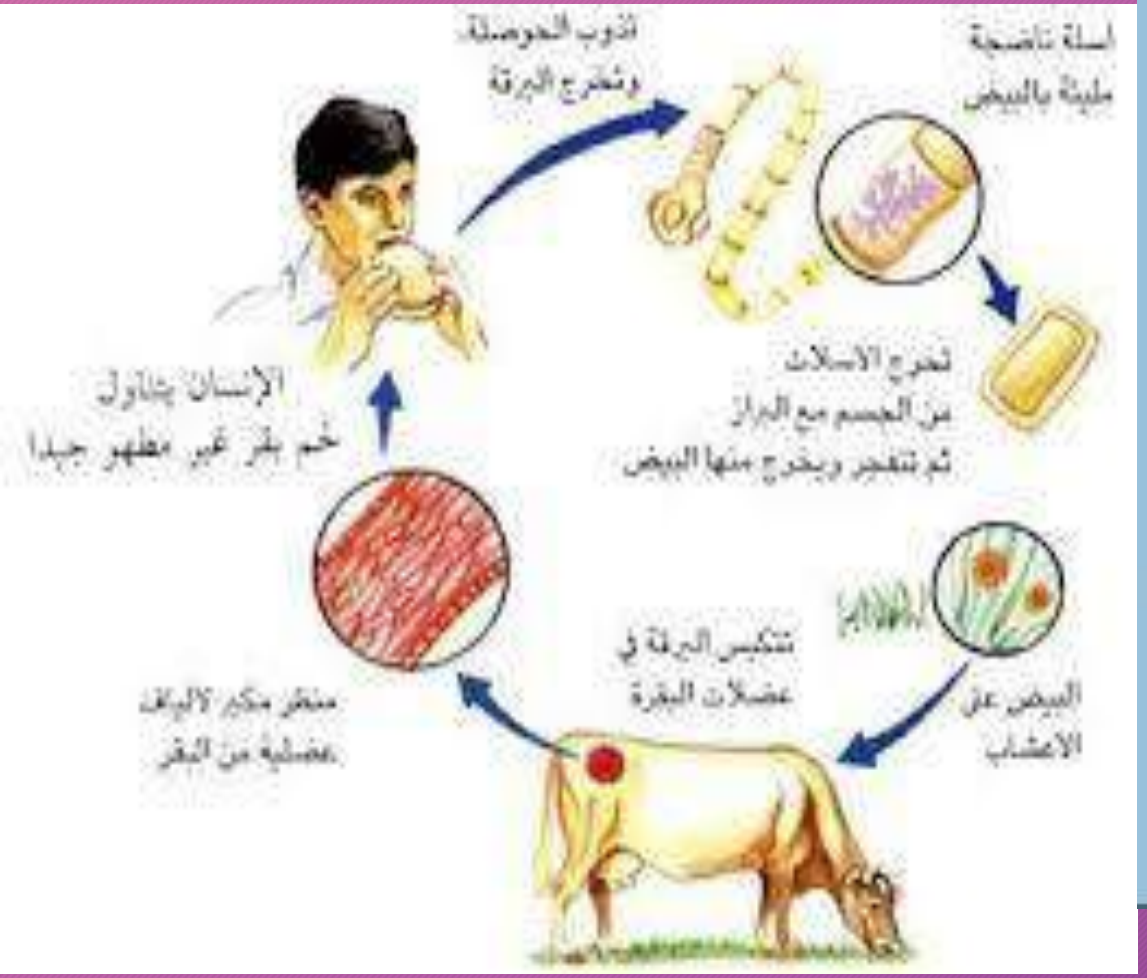


أما القطع الحبلى فتمتاز بكثرة التفرعات الجانبية في الرحم (15-35 فرعاً جانبياً) و التي قد تتفرع بدورها إلى فروع جانبية ثانوية و قد يحتوي الروحم حوالي 100000 بيضة. البيوض كروية وعندما تكون بالرحم تكون مغلفة بغشاء خارجي مزود بخيطين قطبيين رقيقين تفقدهما بعد خروجهما من القطعة الحبلى بتحلل القطع داخل أو خارج الأمعاء .

وتمتاز هذه اليرقات بشكلها الدائري إلى البيضوي ويتراوح طولها بين 7.5-9 ملم و عرضها 5.5 ملم وهي ذات لون ابيض حليبي وعنق معتم منغمد إلى الداخل و رأس مزود بأربعة ممصات. في حالة الاصابة الشديدة قد توجد هذه اليرقات في اعضاء أخرى غير العضلات و الأنسجة الرابطة الداخلية مثل الكبد و الرئتين و الكليتين . يصاب الإنسان بعد تناول هذه اليرقات مع اللحم النيئ او المطبوخ بصورة غير جيدة حيث ينقلب الرأس للخارج في الأمعاء الدقيقة و يلتصق بالطبقة المخاطية للأمعاء متحولاً الى دودة ناضجة في حدود 8-10 أسابيع . و غالبا ما توجد دودة واحدة ولكن مع ذلك فقد سجل وجود 28 دودة في احد المصابين في الاتحاد السوفيتي السابق. هذا وتعمر هذه الديدان حوالي 25 سنة .

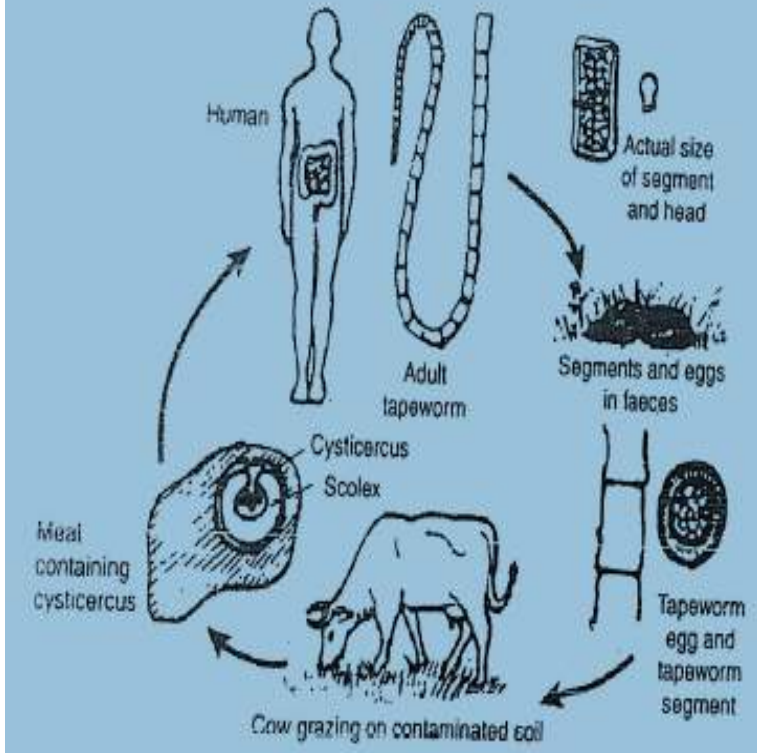
الوبائية : تحدث الاصابة في الانسان جراء تناوله لحم البقر النيئ او المطبوخ بصورة غير جيدة . أما المواشي فتصاب من خلال الرعي على التربة الملوثة ببيوض الدودة في المناطق التي تستعمل فيها مخلفات المجاري او فضلات الإنسان كأسمدة للنبات. و بإمكان البيوض البقاء حية تحت الظروف الطبيعية لفترة قد تصل إلى ستة اشهر و باستطاعة البيوض ان تمر من خلال امعاء بعض الطيور كالنوارس التي تتغذى على مياه المجاري مما يعمل على نشرها . كما ان العجول يمكن ان تصاب عن طريق المشيمة .

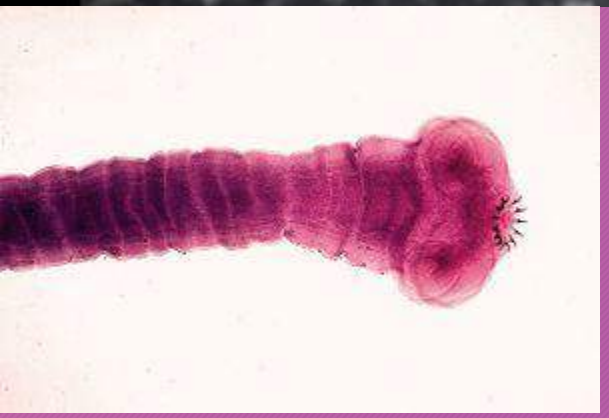
الأعرض المرضية : نادراً ما يسبب وجود الديدان البالغة اعراضاً ملحوظة ولكن احياناً ما تظهر حالات فقر الدم و زيادة عدد كريات الدم البيض الحامضية وحدوث الم وعدم ارتياح في البطن و حالات عصبية و دوار و تقيؤ واسهال . قد تستقر بعض القطع الحبلى بالزائدة الدودية فتؤدي الى التهابها . و قد يؤدي إمتصاص نواتج العمليات الايضية للدودة الى تسمم في الدم مشابه لما هو حاصل بسبب المخرم المعوي *Fasciolopsis buski*



دورة الحياة :

تنفصل القطع الحبلى عن جسم الدودة وتأخذ طريقها الى خارج جسم المضيف عبر فتحة المخرج أو ربما تخرج مع الغائط و تكون نشطة . وبعد خروجها مباشرة تطرح سائلاً حليبياً مليئاً بالبيوض . تصاب المواشي و العديد من المجترات الأخرى (بضمنها الأغنام و الماعز و الجمال) بعد تناولها للحشائش الملوثة ببيوض هذه الدودة و تعد العصارات المعدية مهمة لفقس البيوض، أما العصارات المعوية فتعمل على تحطيم الغشاء الجنيني وتنشيط الجنين الذي يخترق جدار الأمعاء ويحمله الدم أو الملف الى الانسجة العضلية او الرابطة مكوناً هناك الكيسة المذنبة البقرية Cysticercus bovis خلال 12-18 اسبوعياً .





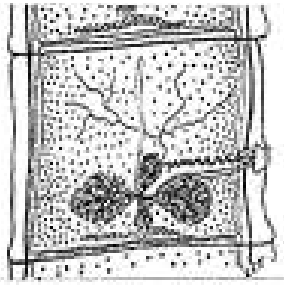
الدودة الشريطية الخنزيرية *Taenia solium*

تسمى دودة لحم الخنزير الشريطية Park tape worm أو الدودة الشريطية المسلحة Armed وتنتشر في ارجاء عديدة من العالم حيث يؤكل لحم الخنزير نيئاً او مطهياً بصورة غير جيدة و تكاد الإصابة ام تكون معدومة بين المسلمين و اليهود لعدم تناولهم لحوم الخنازير .

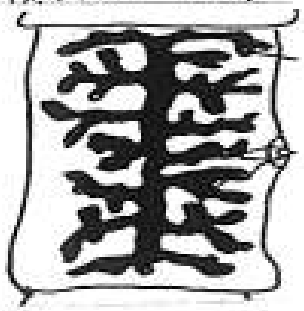
المظهر الخارجي : تشبه هذه الدودة الدودة البقرية في كثير من النواحي.

الدودة هذه اقصر طولاً حيث يتراوح طولها بين 1.8-3 متر وربما اطول ويتراوح عدد القطع الجسمية بين 800-1000 قطعة.

الرأس يحمل اربعة محاجم كروية الشكل و خطم مزود بصفين من الكلاليب .

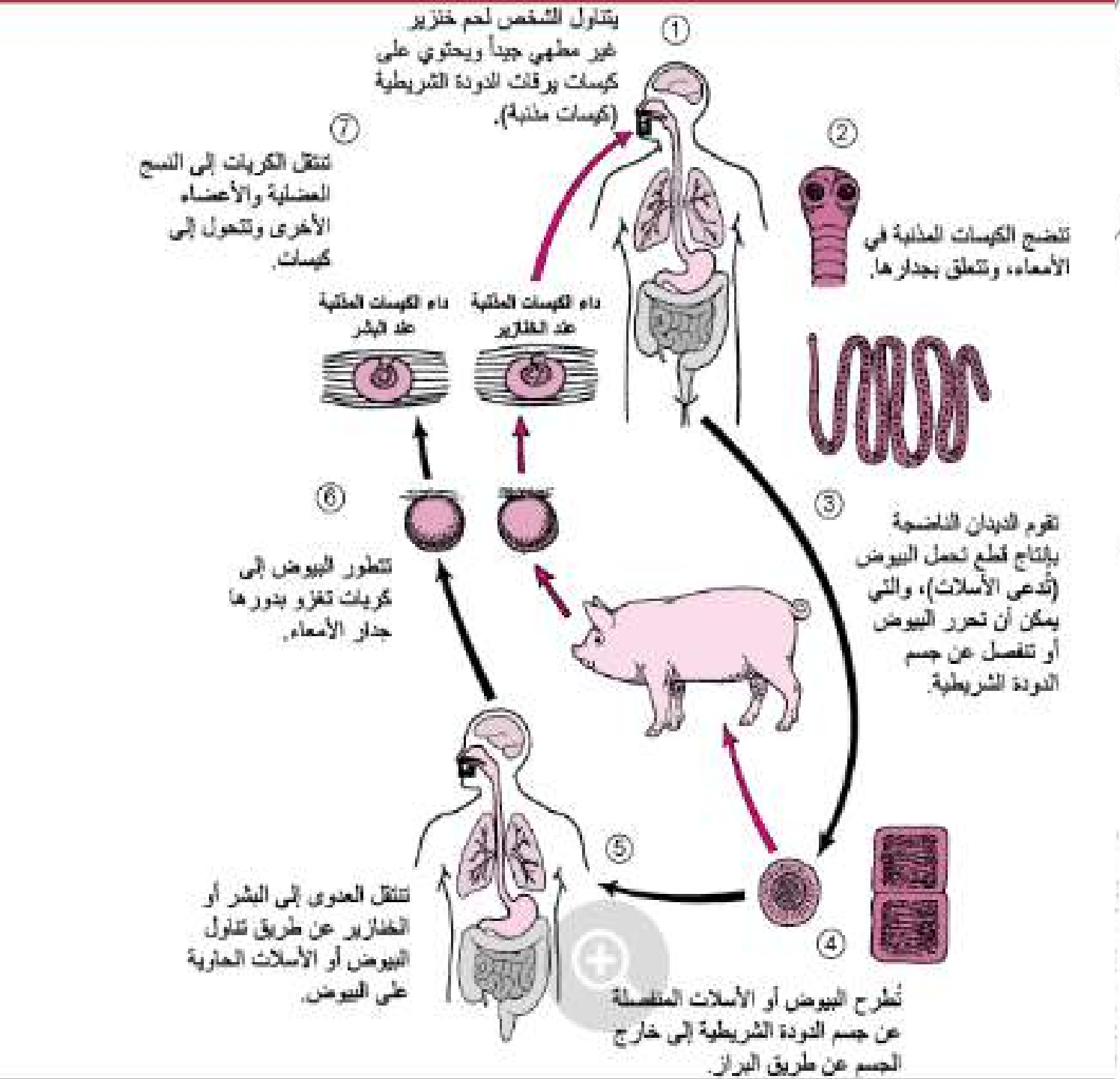


القطع الناضجة أقل استطالة مما في الدودة البقرية، والمبيض فيها ثلاثي التفصص.



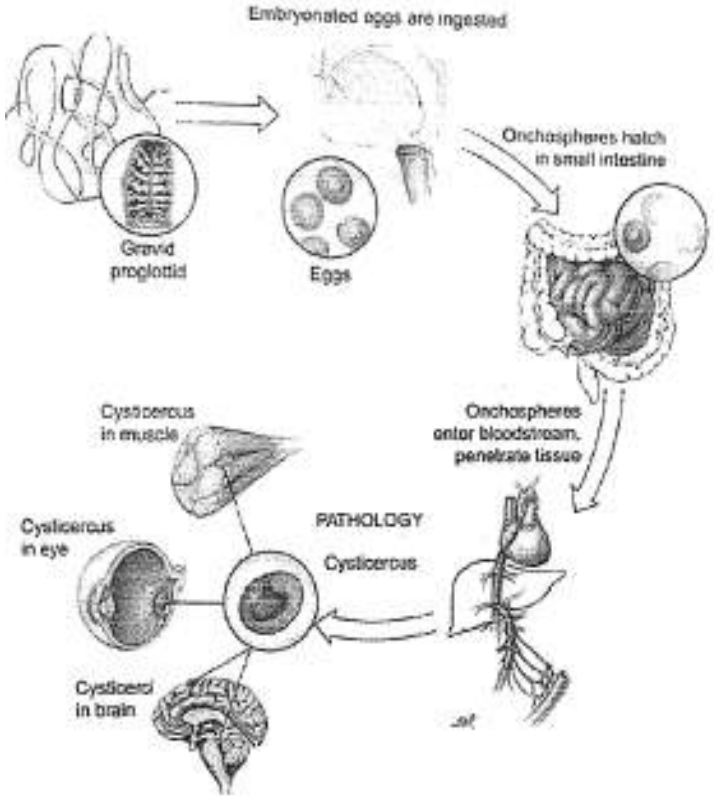
يشتمل الرحم في القطع الحبلى على 7-12 فرعاً جانبياً . القطع الحبلى اقل نشاطاً واكثر ترهلاً .

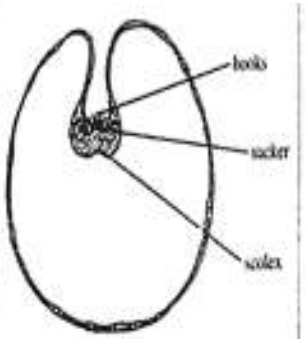
دورة الحياة : تنفصل القطع الحبلى من وقت لأخر على أشكال سلاسل (5-6قطع) ويتحرر من كل قطعة بين 30 ألف-50 ألف بيضة عند تمزقها داخل أو خارج جسم المضيف . ويصاب المضيف الوسطي (الخنازير ،



وتصاب احياناً الأغنام والغزلان والقردة والجرذان لكن بنسبة قليلة جداً) بأبتلاع البيوض حيث يتحرر الجنين سداسي الأشواك ويخترق الأمعاء و ينقله الدم أو الملف إلى العضلات الجسمية المختلفة فيتحول الى كيسة المسماة Cysticercus cellulosae.

تحصل إصابة الإنسان بتناول لحوم الخنازير نيئة او مطبوخة بصورة غير جيدة حيث يذوب جدار الكيس بفعل العصارات الهاضمة وينقلب الرأس للخارج ويثبت نفسه بجدار الأمعاء الدقيقة وينمو إلى دودة بالغة بغضون عدة اشهر . وتعمر الديدان البالغة 25 سنة أو أكثر. قد يصاب الأنسان بالأكياس المثانية أيضاً من خلال تلوث الأصابع ببيوض الدودة أو من خلال فقس البيوض بالأمعاء و إختراق الجنين لامعاء الانسان ووصوله الى العضلات و الانسجة تحت الجلدية



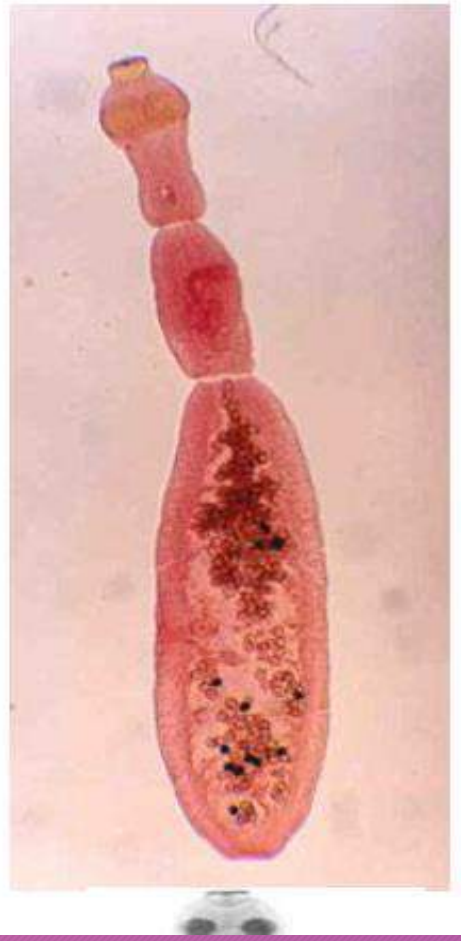


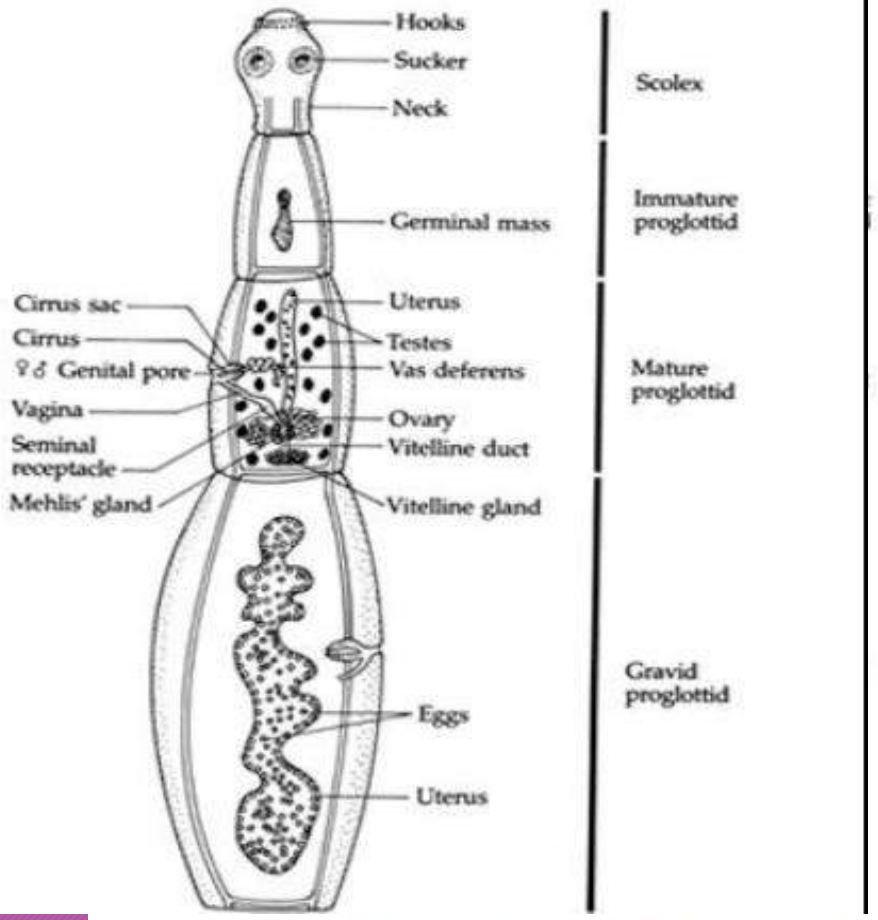
Cysticercus cellulosae ذات الشكل الأهليلجي و اللون الأبيض ويتراوح طولها بين 6-18 ملم و تحتوي على رأس داكن منغمد إلى الداخل ويحمل محصات و كلاليب

Echinococcus granulosus
Dipylidium caninum

الدودة الشريطية المشوكة الحبيبية *Echinococcus granulosus*

تعيش الديدان البالغة في الامعاء الدقيقة للكلاب والثعالب والذئاب وبنات أوى والقطط البرية أما الطور اليرقي فيعيش في عدة انواع من الحيوانات اكلة الاعشاب لاسيما الاغنام والماعز والابقار والخيول و الانسان حيث تصاب كل أعضاء الجسم خاصة الكبد و الرئتين . وهي ذات انتشار عالمي .



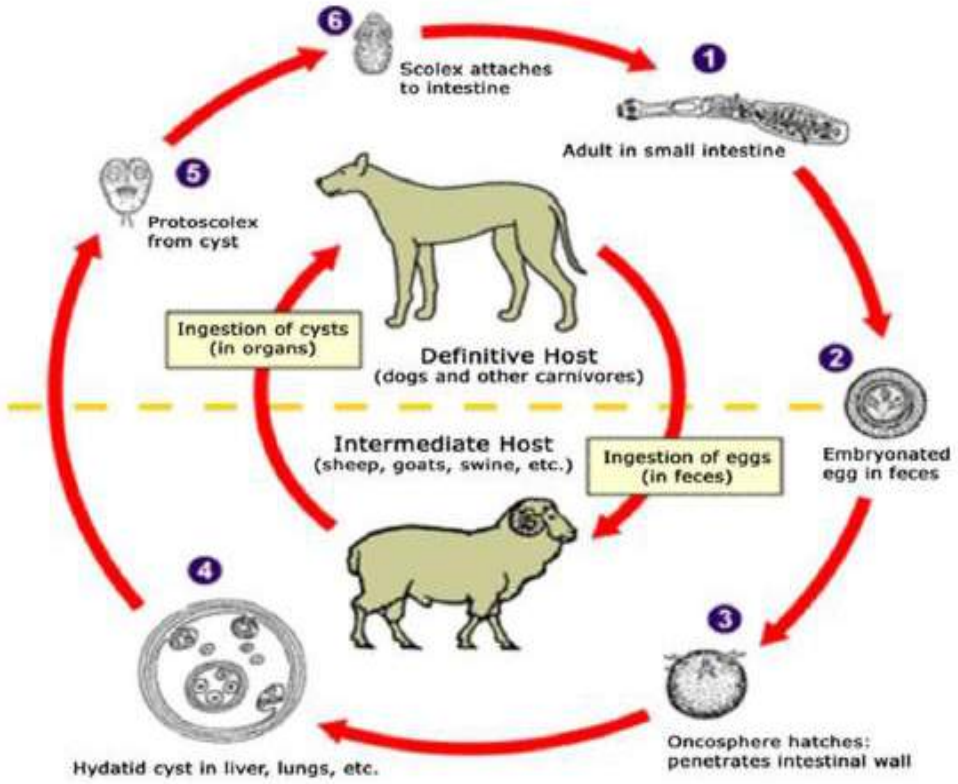


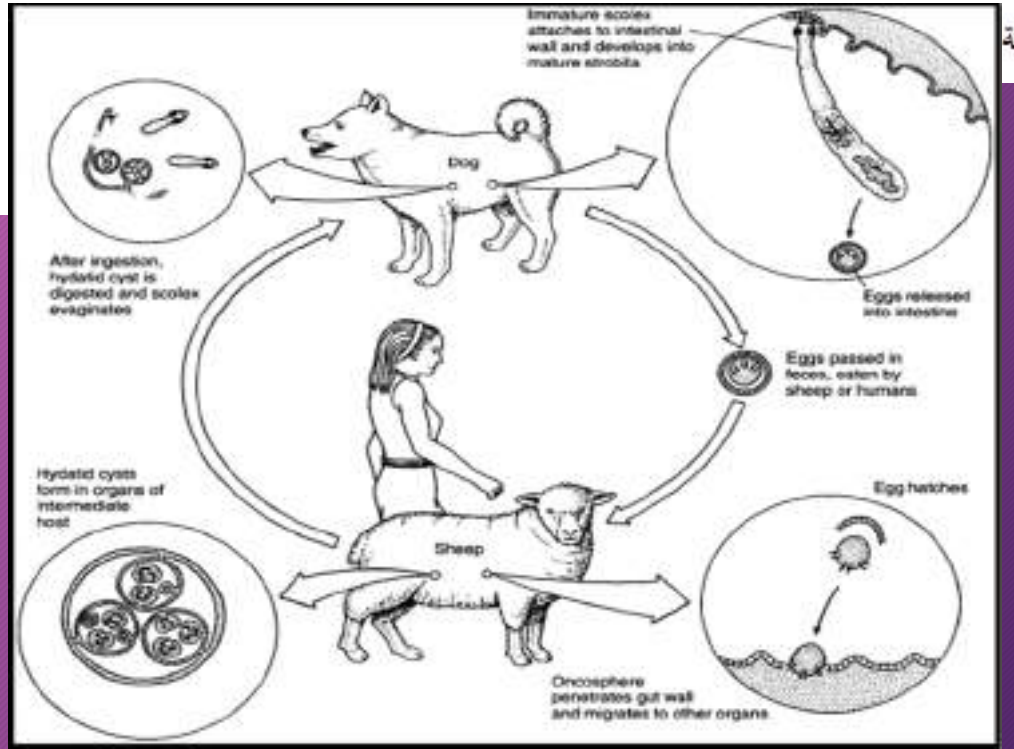
المظهر الخارجي : الديدان البالغة صغيرة الحجم حيث يتراوح طولها بين 2-9 ملم ولها رأس هرمي الشكل يحمل أربعة محاجم وخطماً واضحاً مزوداً بصفين من الأشواك يتراوح مجموعها بين 28-50 شوكة . يلي الرأس عنق وثلاث قطع هي غير ناضجة , وناضجة و حبلى . القطعة الناضجة مستطيلة الشكل وتحتوي على 45-65 خصية كمثرية الشكل وزوج من المبايض و عدة محية مفردة تقع خلف المبيضين . الفتحة التناسلية جانبية الموقع . القطعة الحبلى تشتمل على الرحم وسطي يتكون من 12-15 فراغ أو دهليز جانبي مملوء بالبيض وتشكل القطعة الحبلى لوحدها اكثر من نصف طول الدودة . البيوض شبه الدائرية المحاطة بغلاف مخطط دائرياً يتم طرحها مع القطعة الحبلى

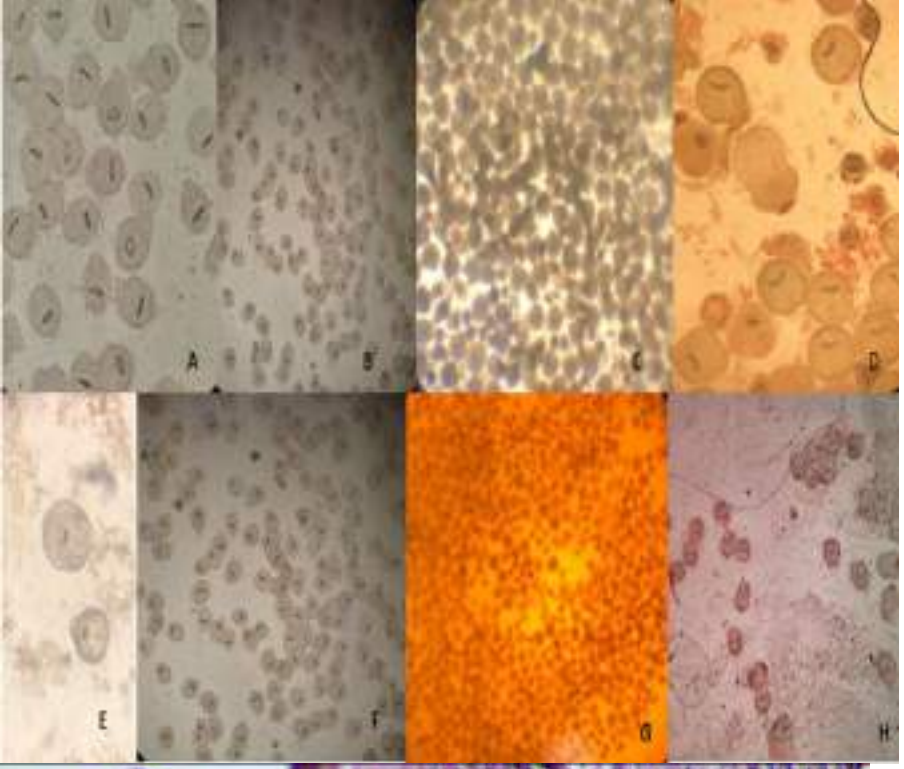
دورة الحياة : تمتاز البيوض بمقاومتها العالية للظروف البيئية. وعند ابتلاع أو استنشاق هذه البيوض من قبل الانسان او الحيوانات الداجنة كالأغنام والمواشي والماعز والجمال والخنازير والخيول فأن الجنين يتحرر منها في منطقة الاثني عشري و يلتصق بجدار الأمعاء بواسطة كلابية ومن ثم يخترق جدران الامعاء فينقله الدم الى اعضاء مختلفة في الجسم كالكبد او الرئتين وغيرها وهناك يتطور الى كيس مائي Hydatid cyst ينمو

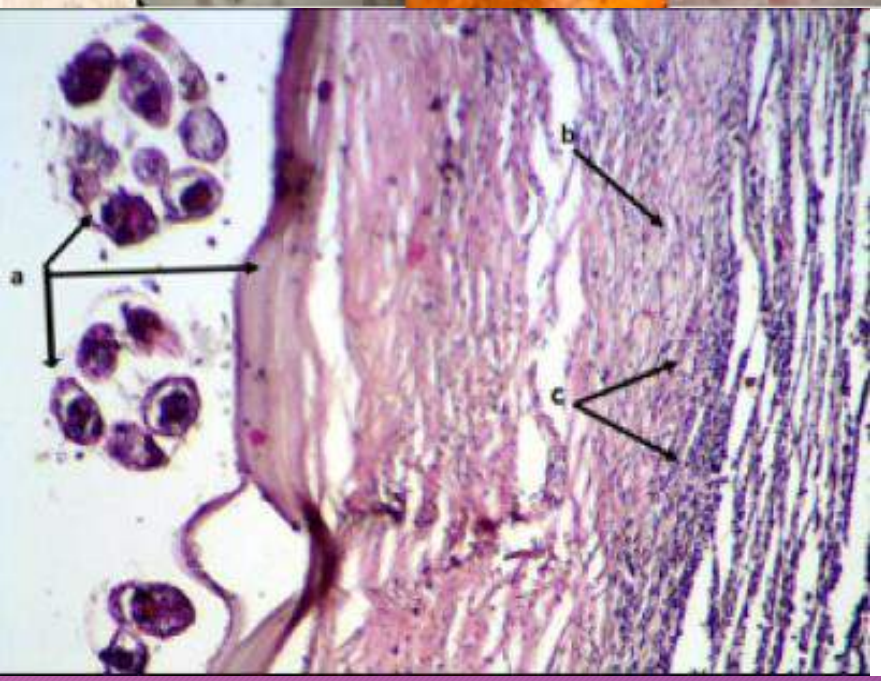
دورة الحياة

تعيش الديدان البالغة في أمعاء العائلة الكلبية حيث يتم طرح البيوض مع البراز وعند أبتلاع هذه البيوض من قبل العوائل الوسطية مثل الأبقار والأغنام والجمال وكذلك الأنسان الذي يصاب عن طريق تناول الغذاء والماء الملوث او عن طريق اللعب مع الكلاب المصابة ، يتحرر الجنين سداسي الأشواك في الأثني عشري بفعل العصارات الهاضمة وتخترق جدار الأمعاء وتصل الى الكبد والرئتين والأعضاء الأخرى لتنمو الى أكياس عدرية (مائية) . وتصاب الكلاب عن طريق تناولها للأعضاء المختلفة من الحيوانات آكلة الأعشاب الحاوية على الأكياس المائية التي تحتوي بدورها الرؤويسات البدائية Protoscoleces والتي تنمو الى ديدان بالغة في الأمعاء الدقيقة

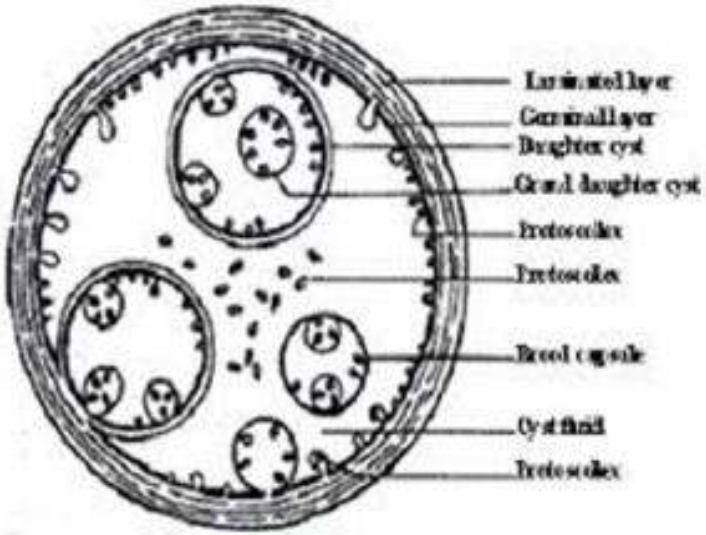








الكيس المائي Hydatid cyst



هذا الكيس كروي الشـــكل في الغالب مالم يتعرض للضـغط من قبل الأعضـاء الجسـمية الأخرى. يحاط الكيس بطبقة خارجية عديمة الأنوية ذات لون حليبي داكن تقع تحتها طبقة عشـــاء جرثومي داخلي نووي تحيط بسائل عديم اللون أو مصفر شاتب.

Figure 32.5. Protoscolex of *E. granulosus*.

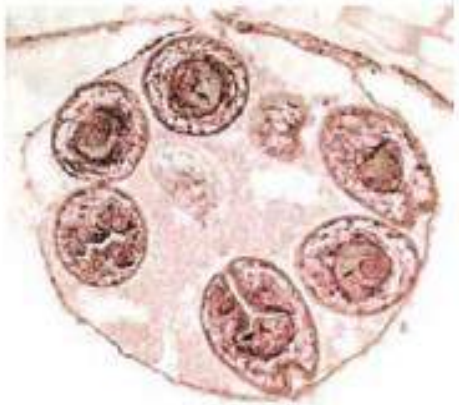


Figure 32.4. Brood capsule with protoscolices o
Echinococcus granulosus.

تبرز داخل الكيس محافظ الحضنة Brood capsules الحاوية على الرؤوس الأولية Protoscolices. تنشأ اكياس بنوية أما من الغشاء الجرثومي أو من محافظ الحضنة أو من الرؤوس الأولية. ينمو الكيس المائي ويحيطه المضيف بجدار متليف و تنشأ داخل الكيس المائي أكياس ثانوية جوفاء تسمى محافظ الحضنة والتي تنفصل عن جدار الكيس وتعرف عندئذ وهي داخل السائل المائي بأسم الرمل المائي أو الرمل العدري Hydatid sand وقد يحتوي الكيس الخصب المتوسط الحجم على حوالي مليوني رأس أولي ولكن بعض الأكياس تفشل في تكوين محافظ الحضنة فتسمى عندئذ بالأكياس العقيمة Sterile cysts .

الوبائية : يعتمد انتشار داء الأكياس المائية في الإنسان على الصلة الحميمة بينه وبين الكلاب المصابة . إن أعلى نسبة للإصابة بين الكلاب تحصل في الأقطار التي تكثر فيها المراعي وذلك لأستهلاك الكلاب لجثث الحيوانات المصابة. تحدث إصابة الإنسان في فترة الطفولة عادة وهي فترة العادات الغير صحيحة. و يتم انتقال المرض بابتلاع البيوض وخاصة من اليد إلى الفم. يحصل الإنسان على البيوض من التربة أو من التربة أو من فراء الكلاب المصابة أو من الكلاب غير المصابة التي يتلوث فراءها من الارض الملوثة بفضلات الكلاب المصابة . كما قد تحصل الاصابة بلعق الكلب لأيدي ووجوه الاطفال أو اواني الطعام حيث يحصل التلوث بالبيوض بعد لعق الكلاب لمخارجها.

التأثيرات المرضية و الأعراض : يعتمد المرض في الإنسان على موقع الأكياس المائية. وتبلغ أعلى نسبة للأكياس في الكبد (66%) فالرئتين (22%) فالكليتين(3%) فالعظام (2%) فالدماغ (1%) والانسجة والاعضاء الاخرى كلعضلات و الطحال و العيون والقلب والغدة الدرقية (0.06 %).

يتسبب الضغط و الخر الناجمين عن نمو الكيس في تدمير نسيج الكبد الطبيعي وتعطيل عمل الكبد الطبيعي وتنمو الأكياس في الكبد بصورة بطيئة حيث قد تصل إلى 30 سنة قبل أية اعراض ملحوظة وقد يؤدي ضغطها على القناة الصفراوية إلى مرض اليرقان الانسدادي Obstructive jaundice . اما في الحالبين فقد يؤدي الكيس إلى مشاكل و صعوبات بولية .

يؤدي انفجار الكيس المائي احياناً (نتيجة السعال أو ضغط العضلات أو النفخ أو الرشف أو اجراء العمليات الجراحية) إلى تحرر الرؤوس الاولية وقطع صغيرة من الغشاء الجرثومي وعلب الحضنة فتتكون أكياس السائل المائي احياناً إلى ظواهر الحساسية على شكل طفح جلدي وحكة وهذيان وقد يؤدي إلى حوادث صدمة مميتة .

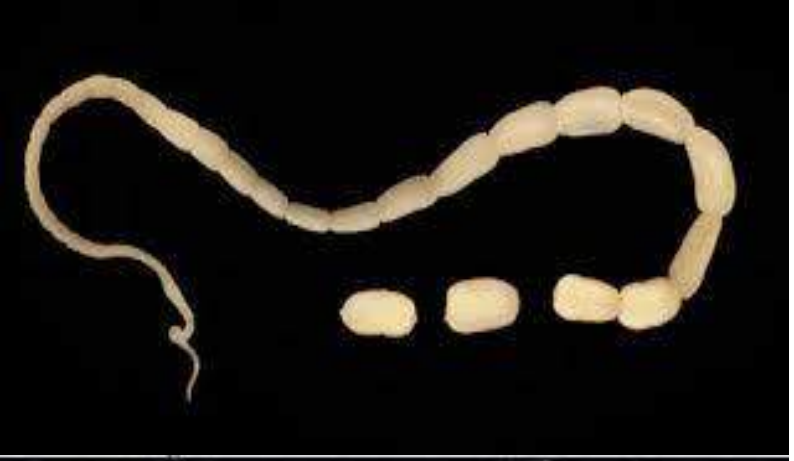
طرق انتقال العدوى:

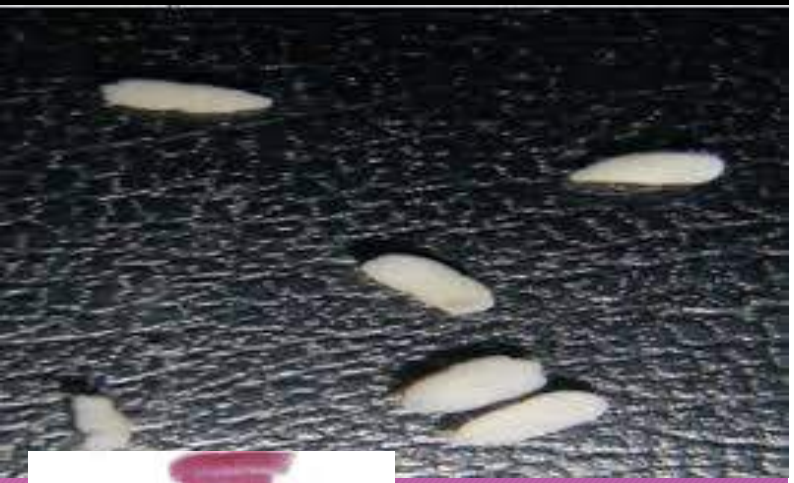
- بواسطة اليد إلى الفم من خلال التماس مع الأدوات الملوثة ببراز الكلاب المصابة بالمرض.
- تناول مواد غذائية مثل الخضروات الطازجة أو الماء الملوث ببويضات الدودة الشريطية الناتجة من براز الكلاب
- مباشرة من الكلاب المصابة إلى الإنسان من خلال ملامستها أو اللعب معها. للكلاب عادة لعق منطقة الشرج حيث يتلوث فمه بالبويضات أثناء اللعق وتنتشر البويضات على شعره وبالتالي تنتقل إلى الإنسان عندما يلامس الكلاب المصابة.
- تغذية الحيوانات آكلة الأعشاب على مراعي أو أعلاف ملوثة ببويضات الدودة الشريطية الناتجة من براز الكلاب.

التشخيص: يتم التشخيص بالفحص الشعاعي للأشخاص ذوي البطون والصدور المنتفخة. وقد يفيد أحياناً العثور على قطع من الأكياس في القشع أو البول نتيجة انفجار الأكياس داخل الجسم. كذلك يتم التأكد بإجراء بعض الاختبارات المصلية مثل اختبار كاسوني Casoni حيث يزرق 0.2 سم^3 من السائل المري المعقم تحت الجلد في أحد الذراعين وتزرق نفس الكمية من المحلول الملحي المعقم في الذراع الأخرى وفي حالة تكون حلقة حمراء حول المنطقة المزروقة بالسائل المائي فهذا يعني أن الشخص مصاب بداء الأكياس المائية .

الوقاية : للوقاية من الاصابة لابد من مراعاة مايلي :-

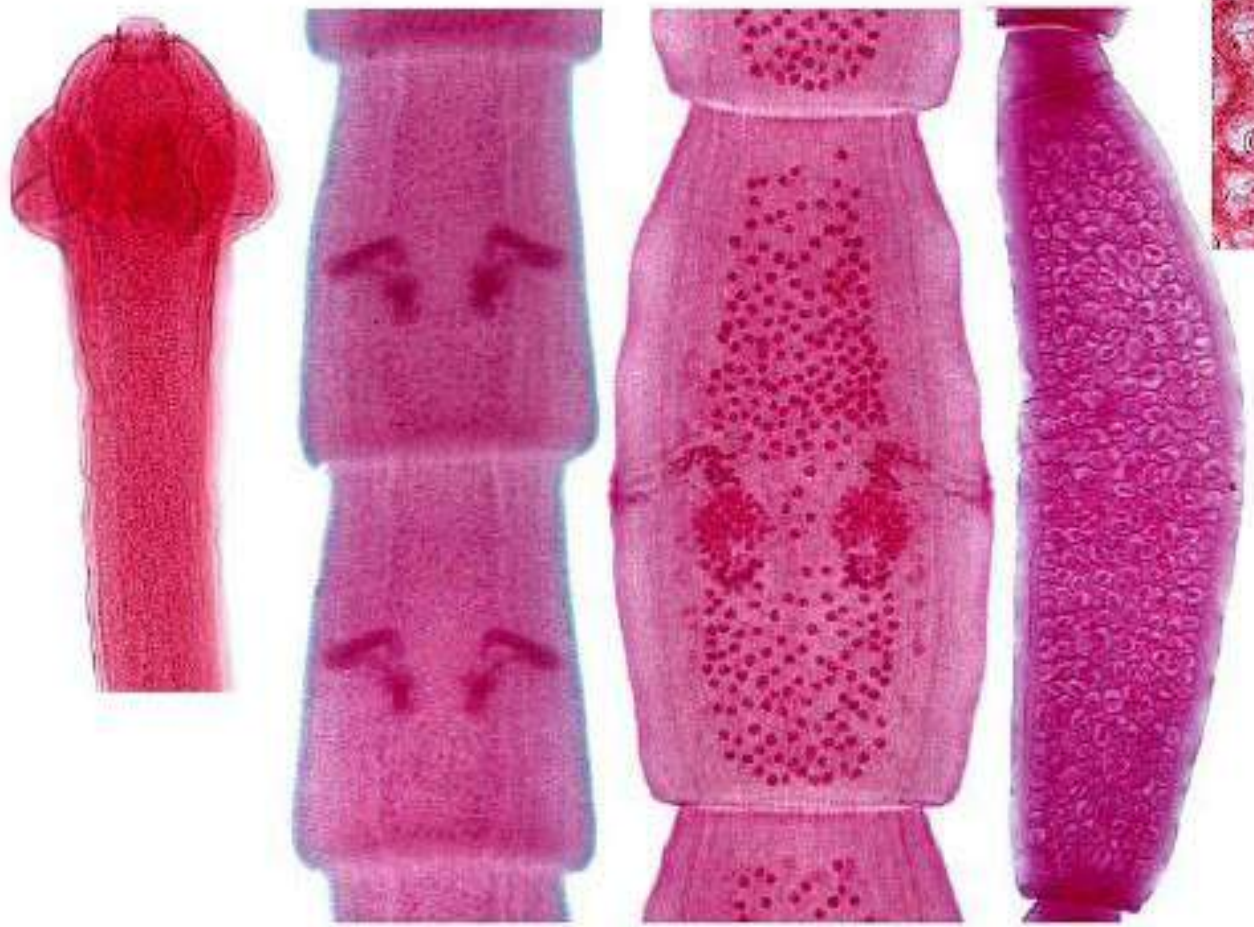
1- عدم اطعام الكلاب بقايا الحيوانات المذبوحة.
2- دفن الحيوانات الميتة و عدم السماح للكلاب بالتغذي عليها .
3- علاج الكلاب بطاردات الديدان Anthelmimtics مرة أو مرتين اسبوعياً .
4- القضاء على الكلاب السائبة Stray dogs .
5- تجنب الألفة مع الكلاب وخاصة لدى الأطفال.
6- على الماء وطهي الخضروات والتشديد على النظافة الشخصية في المناطق الموبوءة .



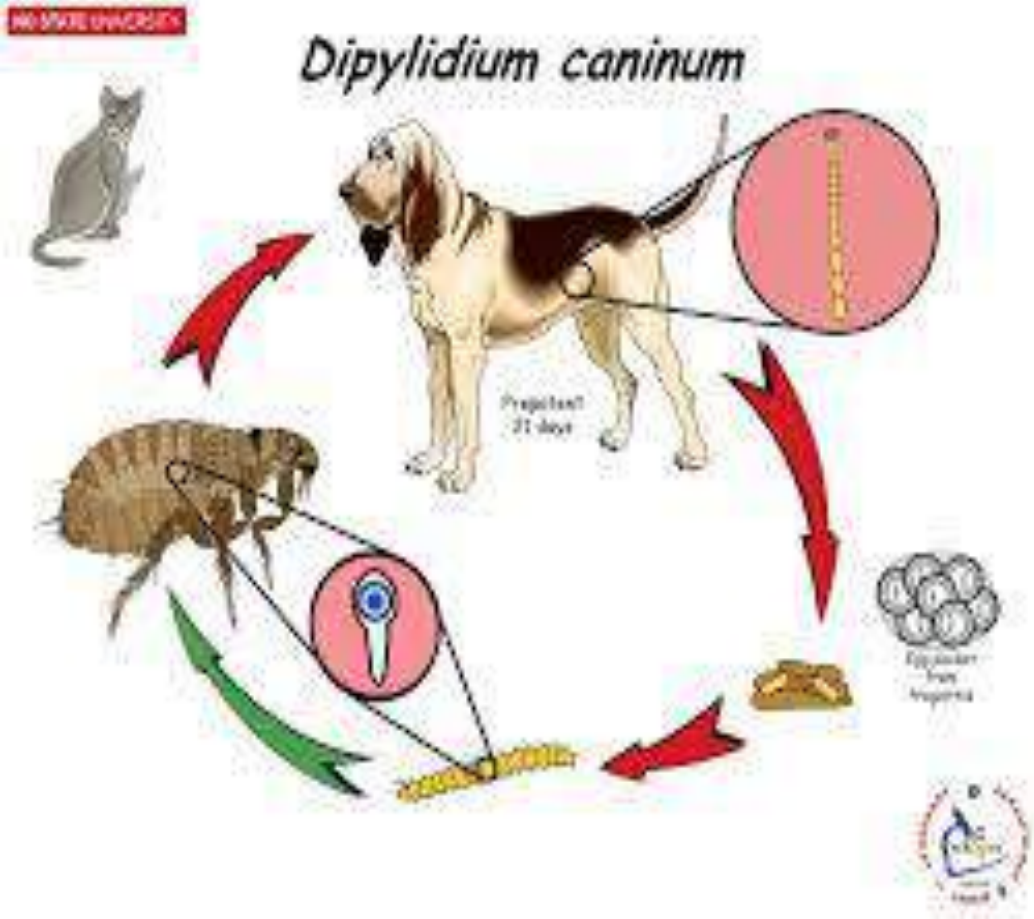


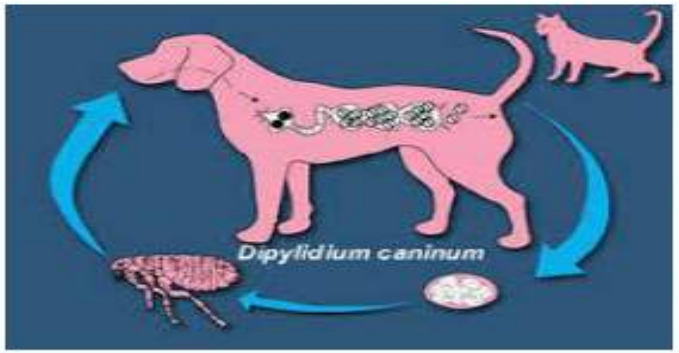
الدودة الشريطية الكلبية ذات الفتحتين *Dipylidium caninum*

تعرف بالدودة الشـريطية الكلبية ذات الفتحتين Double-pored dog tapeworm وهي من الديدان الشـائعة جداً في الأمعاء الدقيقة للكلاب و القطط والثعالب المصـابة بالبراغيث و القمل و أحياناً ما يصـاب بها الإنسان و بالأخص الأطفال. الإصابة بها عالمية الأنتشار .



يتراوح طول الدودة البالغة بين 10-70 سم (بالمعدل 30سم) ولها رأس صغير معيني الشكل مزود بخطم Rostellum قابل للأرتداد في كيس الخطم Rostellar sac ويحمل الخطم صفاً إلى 7 أو 8 صفوف من الكلاليب الشبيهة بشوكة نبات الجوري. كما يحمل الرأس أربعة ممصات. تحتوي القطع التناسلية الناضجة على زوج من الأعضاء التناسلية كل منهما له فتحة تناسلية جانبية الموقع و من هنا جاءت تسمية هذه الدودة بذات الفتحتين. يكون المبيض والغدة المحية عند كل جانب تركيباً يشبه عنقود العنب. أما القطع الحبلى فشبيهة بحبة القرع وينمو فيها الرحم مكوناً كرات من البيوض Egg balls حاوية ما بين 5-20 بيضة. وعالباً ما تشاهد القطع الحبلى وهي تتلوى بنشاط في الغائط أول خروجه من المصاب.





دورة الحياة

بعد طرح القطع الحبلى مع البراز تطرح منها البيوض. هذه البيوض يتم التهامها من قبل يرقات البراغيث و القمل (المضيف الوسطي) التي تصيب القطط و الكلاب و الإنسان. يفقس الجنين الموجود داخل البيضة في أمعاء يرقة الحشرة و يخترق أمعائها متجهاً إلى الجوف الجسمي حيث يتحول إلى طور الكيسانة المذنبة Cysticercoid وهذا الطور المتكيس يتحول إلى دودة بالغة بعد التهام أو تناول البراغيث البالغة أو القمل البالغ من قبل القطط أو الكلاب. ومن المحتمل أن تحصل إصابة الأطفال عن طريق لعق وجوههم من قبل الكلاب والقطط أثناء اللعب معها بعد تناولها البراغيث مباشرة , أو أحيانا ما تحصل الإصابة بسبب سقوط الحشرات المصابة على الغذاء أو في الماء . هذا و تحتاج يرقات الكيسانة المذنبة فترة 3-4 أسابيع حتى تتحول إلى دودة بالغة .

الوبائية: أكثر الناس تعرضاً للإصابة هم الذين في تماس مباشر مع الكلاب و القطط خاصة الأطفال حيث يحصل ابتلاع عرضي للبراغيث أو القمل أو تلوث الطعام أو الشراب أو الأيدي بها.

التشخيص: يعتمد على العثور على القطع الحبلى (ذات الشكل الشبيهة بحبة القرع) في الغائط أو العثور على كرات البيوض بعد تحطم القطع .

الوقاية: للوقاية من الإصابة لابد من تجنب ملامسة الكلاب والقطط. أما بالنسبة للكلاب والقطط المرباة في البيوت فلابد من معالجتها بصورة دورية فضلاً عن تعفيرها بالمبيدات الحشرية للتخلص من البراغيث والقمل والعمل على نظافة أماكن معيشتها.

الديدان الخيطية Nemathelminthes

الديدان الخيطية Nemathelminthes

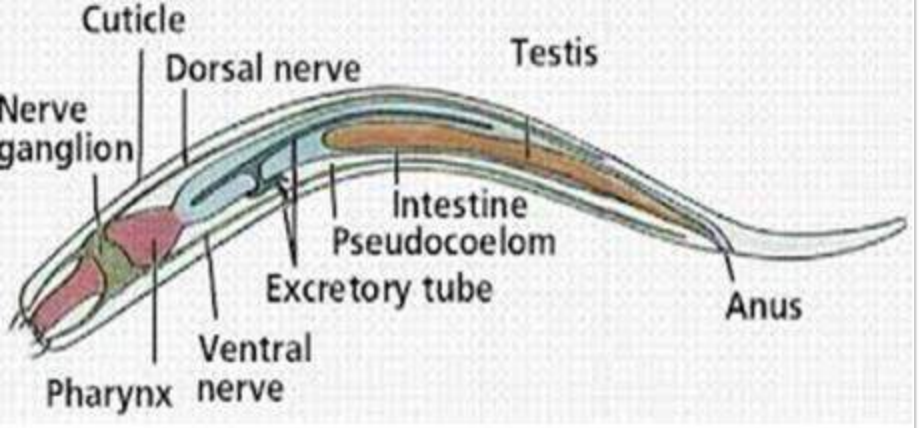
تعرف عامياً باسم Thread worms وهي الترجمة الحرفية لمصطلح Nemathelminthes ويسميها البعض بالديدان الاسطوانية أو المدورة إلا إن صفة الجسم الاسطوانية ليست مقتصرة فقط على هذه الديدان فحسب بل تلاحظ في ديدان أخرى غير الديدان الخيطية لذلك فان تسمية الديدان الخيطية بالديدان الاسطوانية عليها اعتراض. ومن ناحية أخرى يضع البعض الديدان الخيطية ضمن شعبة كبرى تسمى الديدان الكيسية Aschelminthes. إن ما يسمى بالديدان الكيسية عبارة عن خليط غير متجانس من أعداد كبيرة من أنواع الديدان التي لا تربطها علاقات تطورية واضحة جداً لذلك ستعامل الديدان الخيطية هنا كشعبة مستقلة بحد ذاتها.

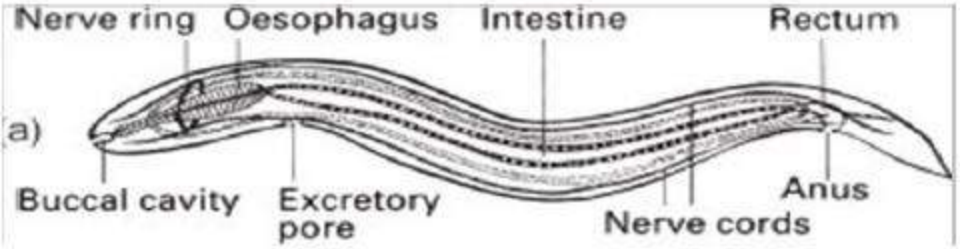
يعيش البعض من الديدان الخيطية حراً في التربة أو الماء ومنها ما يعيش متطفلاً على جذور وسيقان و أوراق وثمار النبات ومنها ما يتطفل على مختلف الحيوانات الفقرية و اللافقرية. عموماً تمتاز الديدان الخيطية المتطفلة بكونها اكبر حجماً من الديدان الخيطية الحرة المعيشة وأنها ذات دورات حياة اكثر تعقيداً.

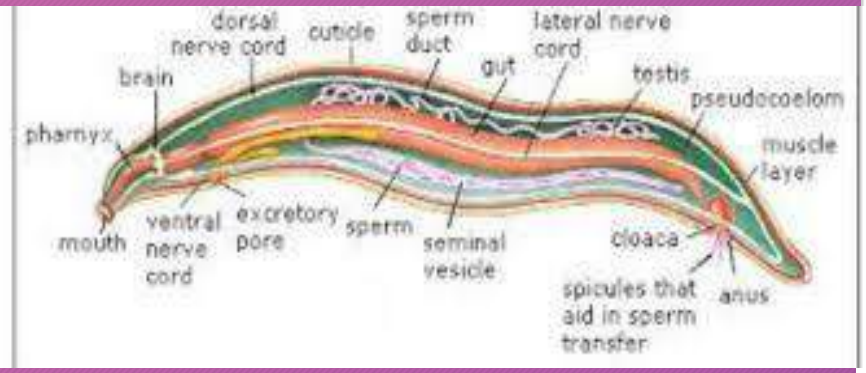
الديدان الخيطية النموذجية متطاولة اسطوانية و مدببة تقريبا عند طرفيها الامامي و الخلفي. الجسم محاط من الخارج بطبقة شفافة أو شبه شفافة من الكيوتكل. وهذا الكيوتكل قوي وغير ناضح ولا يتكون من مادة الكايتين الموجودة في مفصلية الأقدام. يفرز الكيوتكل من طبقة ما تحت الأدمة Hypodermis التي تقع تحتها طبقة عضلية .

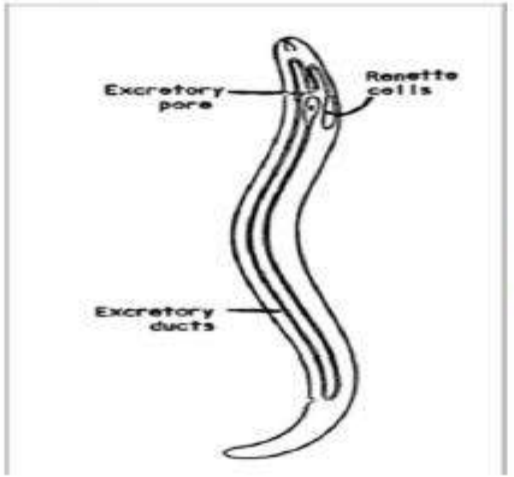
للديدان الخيطية جوف جسمي كاذب Pseudocoel غير مبطن بطبقة بريتون وتقع فيه اعضاء التناسل بصورة غير متصلة بمسارق ماعدا فتحتها الخارجية .

يتكون الجهاز العصبي من حلقة عصبية متميزة تقع حول المريء ومنها تمتد جذوع عصبية طولية أماما وخلفاً.



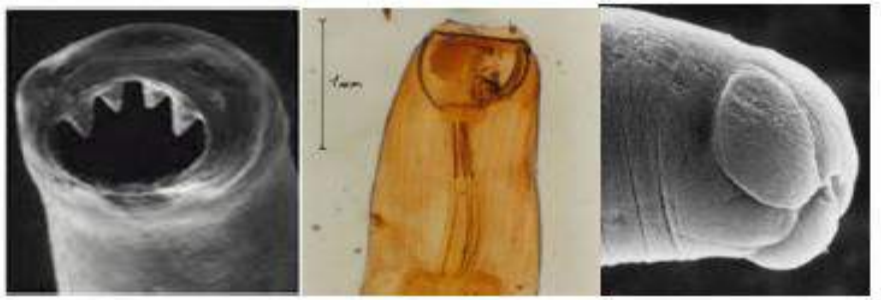






الجهاز البولي متباين في تركيبه إلا انه يشـتمل على فتحة بولية تقع في الناحية البطنية للمريء وهناك قنوات جانبية تتخذ شكل الحرف H وقد تختزل بعض هذه القنوات كما توجد خلية عدية واحدة أو خليتين Renette cells .

الجهاز الهضـمي يبدأ بالفم وينتهي بالمخرج الواقع عند أقصـى جزء من الطرف الخلفي للحيوان أو على مقربة منه. غالباً ما يحاط الفم بعدد من الشـفاه وقد يتحور الفم بشـكل محفظة فمية Buccal capsule مجهزة بتراكيب مختلفة كالأسنان أو الحافات القاطعة أو غيرها.

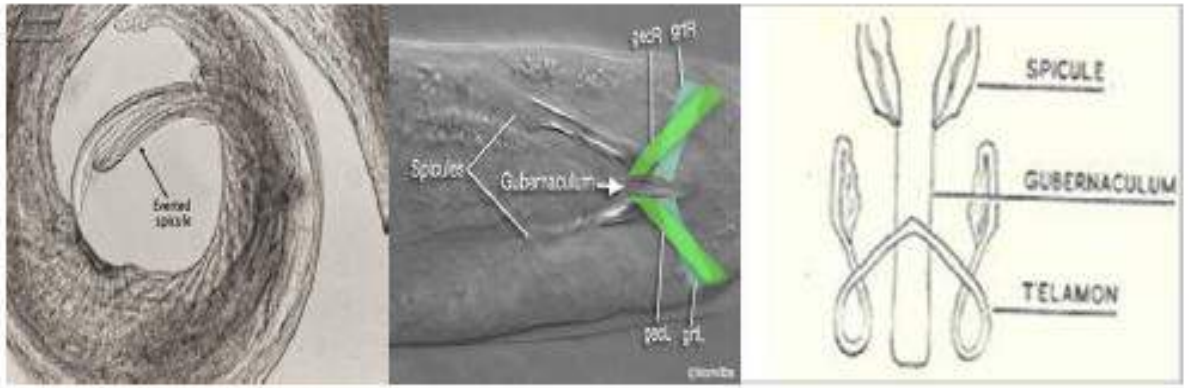


يؤدي الفم إلى مريء عضـلي أو مريء مكون من جزء عضـلي وآخر عدي. يؤدي المريء إلى الأمعاء التي تنتهي بالمخرج. وفي حالة الذكور ينتهي الجهاز الهضمي بنهاية مشتركة مع الجهاز التناسلي على شكل مجمع مشترك.

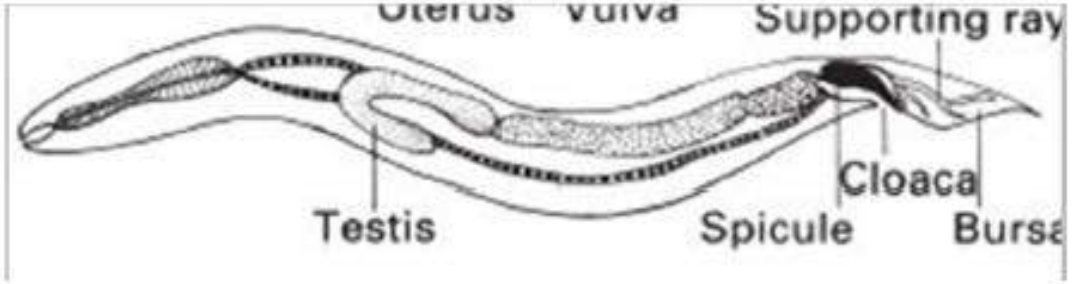
جهاز الدوران معدوم. التنفس يتم أساساً عن طريق الكيوتكل وربما عن طريق القناة الهضمية.

الأجناس منفصـلة والذكور عموماً أقصـر وأنحف من الأناث ونهاياتها الخلفية معقوفة نحو الناحية البطنية . وتمتلك ذكور الكثير من الأنواع أما شوكة واحدة أو شوكتي سفاد Copulatory spicules قابلة للنتوء تفرز في

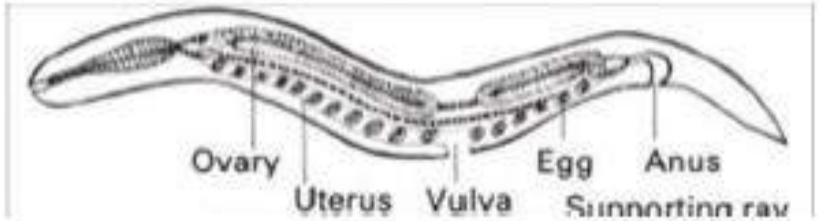
مهبل الأنثى أثناء السفاد لترشد الحيامن هناك. في العديد. من الديدان الخيطية هناك تثخن ظهري في جدار المجمع يسمى Gubernaculum يرشد شوكة أو شويكتي السفاد أثناء الاندفاع من المجمع عند التزاوج. وفي بعض الديدان الخيطية هناك تثخن أخر بطني الموقع يسمى Telamon يقوم بالوظيفة ذاتها.



في حالة الذكور يتكون الجهاز التناسلي عموماً من انابيب طويلة جزء منها يعمل كخصي وجزء اخر كقنوات ناقلة (وعاء منوي ناقل) وعادة ما تتوسع هذه القنوات أو حوصلة منوية تتبعها قناة قاذفة قبل إن تفتح للخارج بالمجمعCloaca. في غالبية الديدان الخيطية المتطفلة يختصر الجهاز التناسلي بالذكر على أنبوبة واحدة.



يتكون الجهاز التناسلي في حالة الإناث عموماً من انابيب طويلة جزء منها يعمل كمبيض أو وجزء أخر كقنوات ناقلة (قناة بيض) وعادة ما تتوسع هذه القنوات على شكل رحم قبل إن تفتح للخارج بفتحة الفرج Vulva التي تتخذ موقعاً امامياً عادة. هذا ويتحد الرحمان بمهبل قصير قبل افتتاحهما بالفرج. في غالبية الديدان الخيطية المتطفلة يختصر الجهاز التناسلي في الإناث فيمكن من أنبوبتين عدا بعض الشواذ.



تختلف دورة حياة الديدان الخيطية المتطفلة ما بين مباشرة وغير مباشرة تحتاج إلى مضيف وسطي واحد أو أكثر لإكمالها. قد يبدأ الجنين بالنمو و البيضة ما زالت غير قاقسة أو غير واصلة للبيئة الخارجية. يعاني الجنين

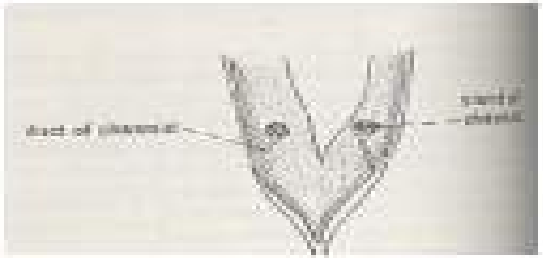
داخل أو خارج البيضة من عدد الانسلاخات التي تمكنه من النمو وزيادة حجمه حتى يصل إلى الطور المعدي (الطور اليرقي الثالث عادة) الذي يمكن ان يصيب المضيف النهائي أما باختراق جلده مباشرة أو بتحوله مع الطعام أو الماء الملوثين.

وفي جسم المضيف النهائي أما إن ينمو الطفيلي إلى مرحلة البلوغ مباشرة في الأمعاء أو إن عليه إن يقوم برحلة في عدد من أعضاء الجسم حتى يصل ثانية إلى الأمعاء فيصبح بالغاً. ويمكن التعبير عن مراحل النمو و الانسلاخات بالصورة الآتية :-

Egg → LI+m → L2+M → L3+M → L4+M → Adult

المرحلة الأولى والثانية تكون من النوع العصوي أو الرابتيدي Rhabditoid حيث يكون شكل المزيء من نوع Rhabditiform اي بشكل انتفاخ بالأمام يعقبه تضمر ثم إنتفاخ. أما الطور اليرقي الثالث فهو من النوع Strongyliform أو Filariform حيث يكون المزيء متجانس السمك لا يوجد فيه أي انتفاخ .

أما تصنيف الديدان الخيطية فيعتمد على وجود مايسمى بالفاسميدات Phasmids



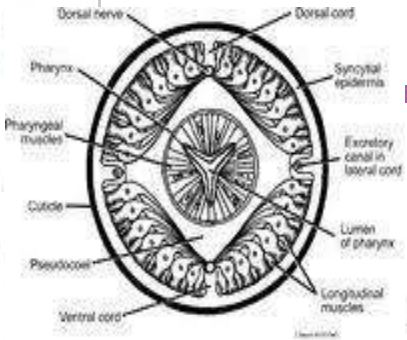
الفاسميدات Phasmids (وهي عبارة عن زوج من تراكيب حسية تقع على حلمتين دقيقتين خلف المخرج).

حيث تقسم الديدان الخيطية تبعاً لذلك إلى صنفين (أو صنفين ثانويين) هما :-

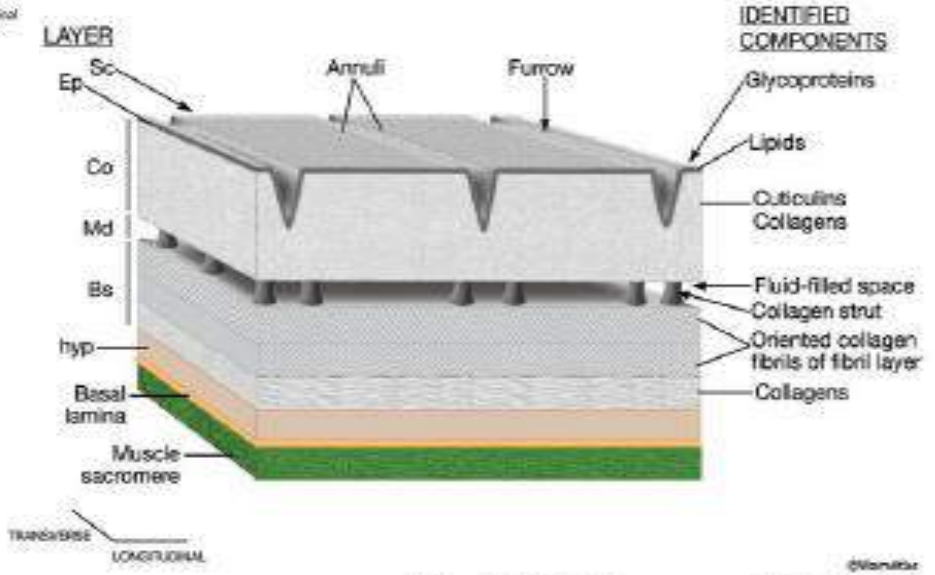
1- **الافاسميدات Aphasmidia** :- تمتاز حيوانات هذه المجموعة بانعدام الفاسميدات فيها. الجهاز الابرازي اثري أو معدوم. تضم هذه المجموعة انواعاً قليلة بالمقارنة مع المجموعة الثانية. ومن أمثلة الافاسميدات كل من الجنس *Trichinella* والجنس *Trichuris* .
2- **الفاسميدات Phasmidia** :- تمتاز حيوانات هذه المجموعة بامتلاكها للفاسميدات. الجهاز الابرازي موجد وليس اثرياً. تنتسب أغلب الديدان الخيطية لهذه المجموعة و التي تمثل بالجنس *Ascaris* و الجنس *Ancylostoma* والجنس *Enterobius* والجنس *Strongyloides* و الجنس *Wuchereria* والجنس *Dracunculus* . وفيما يلي إستعراض سريع لعدد من أنواع الديدان الخيطية التي تتطفل على الانسان وبعض حيواناته.

غطاء الجسم (الجلد) Integument

يتكون غطاءالجسم من:



اولا: الكيوتكل
ثانيا: الطبقة تحت الادمية
ثالثا: والطبقة العضلية



أولاً ــ الكيوتكل Cuticle (البشرة الشمعية)

يغلف الكيوتكل سطح الجسم الخارجي كما يبطن التجويف الفمي والمريء والفرج والمهبل والفتحة البولية والمجمع المستقيم . وهو يكون غطاء قوياً مرناً ومقاوماً للأنزيمات الهضمية كما انه يحوى عدداً من الانزيمات مما يدلل على كونه فعالاً ايضاً . يتكون الكيوتكل من ثلاث طبقات رئيسة هي:

A. القشرة Cortex
B. الطبقة الوسطى أو المادة البينية Matrix
C. الطبقة الليفية الثالثة Trifibrillar.

A: القشرة Cortex وتتألف من طبقتين :-

أ- قشرة خارجية Outer cortical layer تتكون من بروتينات تشبه الكيراتين keratin-like .
ب- طبقة قشرة داخلية Inner cortical layer تتكون بصورة أساسية من بروتين ليفي يسمى الكولاجين Collagen يشبه ما موجود في طبقات الكيوتكل الأخرى .

B: الطبقة الوسطى أو المادة البينية Middle layer *Matrix* وتتألف من طبقتين ليفيتين خارجية وداخلية هما :-

أ- طبقة ليفية خارجية Outer fibrillar layer وتحتوى على قنوات واضحة متقرعة تمتد تحت طبقتي القشرة الداخلية و الخارجية وتنتهي بتفرعات إلى الخارج إذ قد تساعد بنقل المواد إلى سطح الكيوتكل أو قد يكون لها دور هيكلي ساند
ب- طبقة ليفية داخلية Inner fibrillar layer وهذه سميكة متجانسة *Homogenous* وتتركب من مواد بروتينية تشبه الالبومين وبروتين ليفي كما تحوى بعض الكربوهيدرات والمواد الدهنية

C: الطبقة الليفية الثلاثية Trifibrillar layer وهذه تضم ثلاث طبقات ليفية (خارجية ووسطى وداخلية) مكونة من حزم متوازية من بروتين شبيه الكولاجين وتسمح هذه الالياف بتمدد أو تقلص طولي وذلك بتغير الزوايا بين الألياف هذه .

إلى الاسفل من الكيوتكل هناك طبقة الصفيحة القاعدية Basel lamella وهي طبقة من ليفات دقيقة تندمج مع الطبقة التي تحتها .

ثانياً ــ تحت الادمة Hypodermis

هذه الطبقة مسؤولة عن تكوين طبقات الكيوتكل وتتكون من مندمج خلوى Syncytium وتقع انويتها في اربعة تنخفات تبرز إلى الجوف الجسمي الكاذب Pseudocoel مكونة ما يسمى بالحبال تحت الادمية Hypodemal crods ممتدة طولياً وتقسم فضاءات الجسم من اربعة ارباع . يمتد في كل من الحبل الظهري والبطني حبل عصبي أما في كل من الحبلين الجانبين فتمتد قناة بولية .

ثالثاً ــ الطبقة العضلية Muscular layer

وهي طبقة عضلية سميكة لحد ما وتترتب فيها الخلايا العضلية المغزلية الشكل طولياً . وكل خلية عضلية مكونة من جزء خارجي قابل للتقلص Contractile وجزء داخلي غير قابل للتقلص . الطبقة العضلية مجهزة بتفرعات الاعصاب .

يحيط جدار الجسم بالجوف الجسمي الكاذب Pseudocoel لكونه غير مبطن بطبقة بريتون وهذ الجوف فيه سائل يسمى اللمف الدموي Haemolumph وهو محلول معقد خال من الخلايا ولونه صاف إلى ارجواني صاف وله اهمية في نقل المواد الذابة من نسيج لآخر

التراكيب المتخصصة الناشئة عن سطح الجسم

تنشأ من سطح الجسم في العديد من الديدان الخيطية تراكيب متخصصة *Specialized structures* على شكل فتحات , تنخفات , امتدادات , وانتفاخات تؤدى وظائف مختلفة منها :-

التراكيب المتخصصة الناشئة عن سطح الجسم

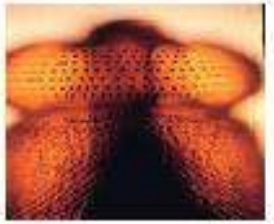
تنشأ من سطح الجسم في العديد من الديدان الخيطية تراكيب متخصصة *Specialized structures* على شكل فتحات , تجنات , امتدادات , وانتفاخات تؤدي وظائف مختلفة منها :-

1- **الشفاه Lips** (مفردها شفة) : تحاط افواه العديد من الديدان الخيطية المتطفلة بثلاث شفاه (واحد شهرية واثنين جانبيتين بطنيتين) وفي بعضها تنعدم الشفاه وفي الانواع الحرة المعيشة قد تحتوى 6 شفاه .

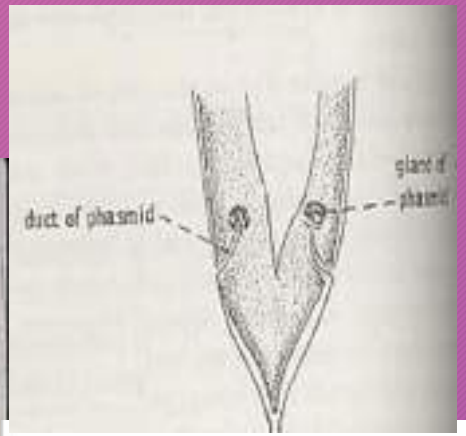
2- **دروع الراس Head Shields** : وهي تراكيب كيوتكلية خاصة تقع خلف الشفاه .

3- **الحبال Cordons** : لبعض الديدان الخيطية اربعة اخاديد أو حافات تسمى الحبال تبدأ من موقع الشفة الجانبية وتمتد خلف المسافات مختلفة حسب الانواع . وقد تكون هذه التركيبة مستقيمة أو منحنية أو جيبية أو حتى متشابهة ولها اهمية تصنيفية

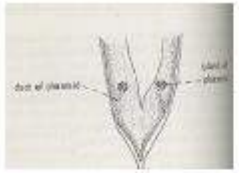
4- **انتفاخ الرأس Head Bulb**: لبعض الديدان الخيطية رأس منتفخ منفصل عن بقية الجسم بتخصر . الداخل ينقسم هذا الانتفاخ الراسي إلى اربع مساحات جوفاء تسمى *Ballonets* غير معروفة الوظيفة.



5- **الامفيدات Amphids**: زوج من أعضاء حسية (مستقبلات كيماوية) على جانبي الرأس في الديدان الخيطية .

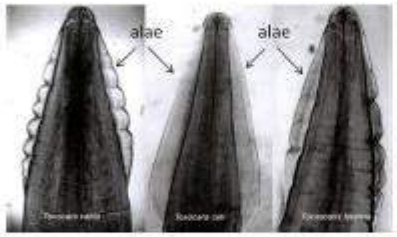


6- **الفاسميدات *Phasmids*** : تراكيب حسية (شمية) على شكل حفر موجودة على الجانبين قرب نهاية الذنب في الفاسميدات Phasmidia .

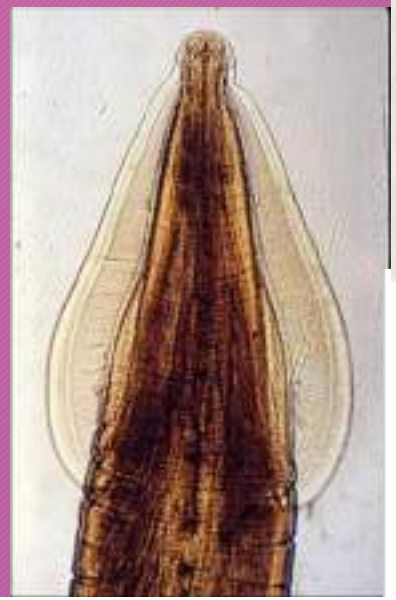


7-

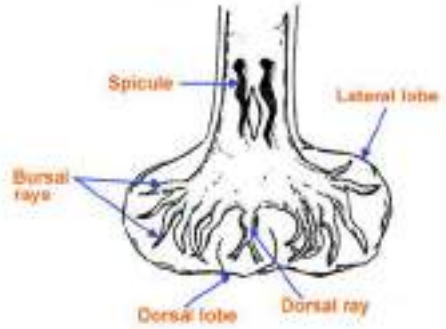
8- **الاجنحة *Alae*** : تراكيب ناشئة امتدادات الكيوتكل على جانبي الدودة بشكل اجنحة , بعضها امامية قتسمى عنقية Cervical وبعضها خلفية فتسمى ذنبية Caudal وبعضها تمتد طولياً جانبي الجسم عدا المقدمة و المؤخرة فتسمى طولية Longitudinal أو جانبية Lateral قد يكون لها دور بالموازنة والاستقرار



9- **كيس السفاد Copulatory bursa** : زوائد مسطحة من الكيوتكل تسندها عادة اشعة لحمية Rays توجد عند النهاية الخلفية لذكور بعض الديدان الخيطية كالديدان الشصية Hook worm تساعد الذكر مسك الأنثى اثناء السفاد .



Nematodes - cuticle modifications
copulatory bursa




شكرا لكم

TOXOPLASMOSIS

Toxoplasma gondii :

The parasite was discovered by Nicolle and Manceaux in 1908 in a small rodent of Africa (*Ctenodactylus gundi*). The medical importance of this organism for human was realised 30 years later. It is a small protozoan being widely distributed in man and animals all over the world. Toxoplasmosis is a disease of mammals and birds caused by Toxoplasma gondii. This obligate intracellular parasite has been found in many mammals, birds and even reptiles. This parasite can be transmitted to humans via uncooked meat, soil and food, by organ transplantation and in pregnant women it pass through placenta to the baby. It also transmits by contact with contaminated things (infected meat, culture, animals).This parasite exists in three forms in final host (schizont, gametocytes and oocyst) while in human and other mammals and bird and reptiles(the intermediate hosts) there are two forms tachyzoites (trophozoite), tissue cysts (which contain bradyzoites) . The tissue cysts and oocysts are the main forms of parasite involved in transmission (infective stages). The infection possesses a serious disease in immunosuppressed individuals and pregnant females. If immunity is impaired, dormant bradyzoites become reactivated. In some infected individuals this parasite may cause dementia due to brain damage caused by tachyzoites. Toxoplasmosis is prevalent in the Mediterranean countries, including Iraq.

Morphology

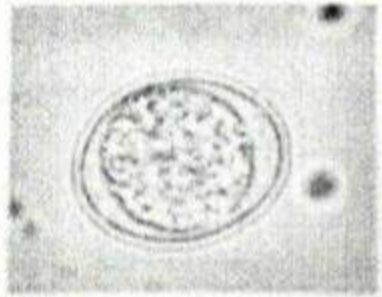
T. gondii is considered to have three stages of infection; the tachyzoite stage of rapid division, the bradyzoite stage of slow division within tissue cysts, and the oocyst environmental stage. When an oocyst or tissue cyst is ingested by a human or other warm-blooded animal, the resilient cyst wall is dissolved by proteolytic enzymes in the stomach and small intestine, freeing sporozoites form within the oocyst. The parasites first invade cells in and surrounding the intestinal epithelium, and inside these cells, the parasites differentiate into tachyzoites, the motile and quickly multiplying cellular stage of T. gondii. Tissue cysts in tissues such as brain and muscle tissue, form about 7–10 days after initial infection.The intracellular parasites (tachyzoite) are 3 x 6 µm, pear-shaped organisms that are enclosed in a parasite membrane to form a cyst measuring 10-100 µm in size.Cysts in cat feces (oocysts) are 10-13 µm in diameter

Tachyzoite (trophozoites) is often crescent or arc or pear shaped with a point anterior end and a rounded posterior end.rapidly multiplying trophozoite acute state found intracellular in parasitophourus vacuole

Oocyte oval in shape contain 2 sporocystes each contains 4 sporzoites

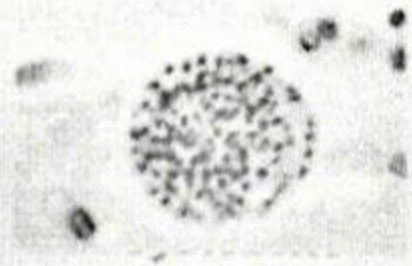
Bradyzoite (sporozoite): slowly multiplying within tissue cyst form, it is also crescent shaped

Tissue cysts grow and remain intracellular may contain hundreds of bradyzoites it is often spheroidal



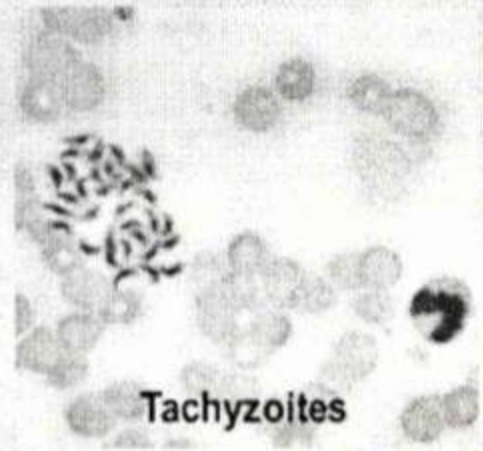
oocyst

Sporulated
oocyst

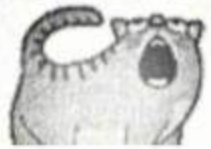


Bradyzoites
within
tissue cyst

Tachyzoites



Tachyzoites



Morphology

Life cycle

The life cycle of *T. gondii* can be broadly summarized into two components: a sexual component that occurs only within cats (felids, wild or domestic), and an asexual component that can occur within virtually all warm-blooded animals, including humans, cats, and birds.[26] Because T. gondii can sexually reproduce only within cats, cats are therefore the definitive host of T. gondii. All other hosts – in which only asexual reproduction can occur – are intermediate hosts

Sexual reproduction in the feline definitive host

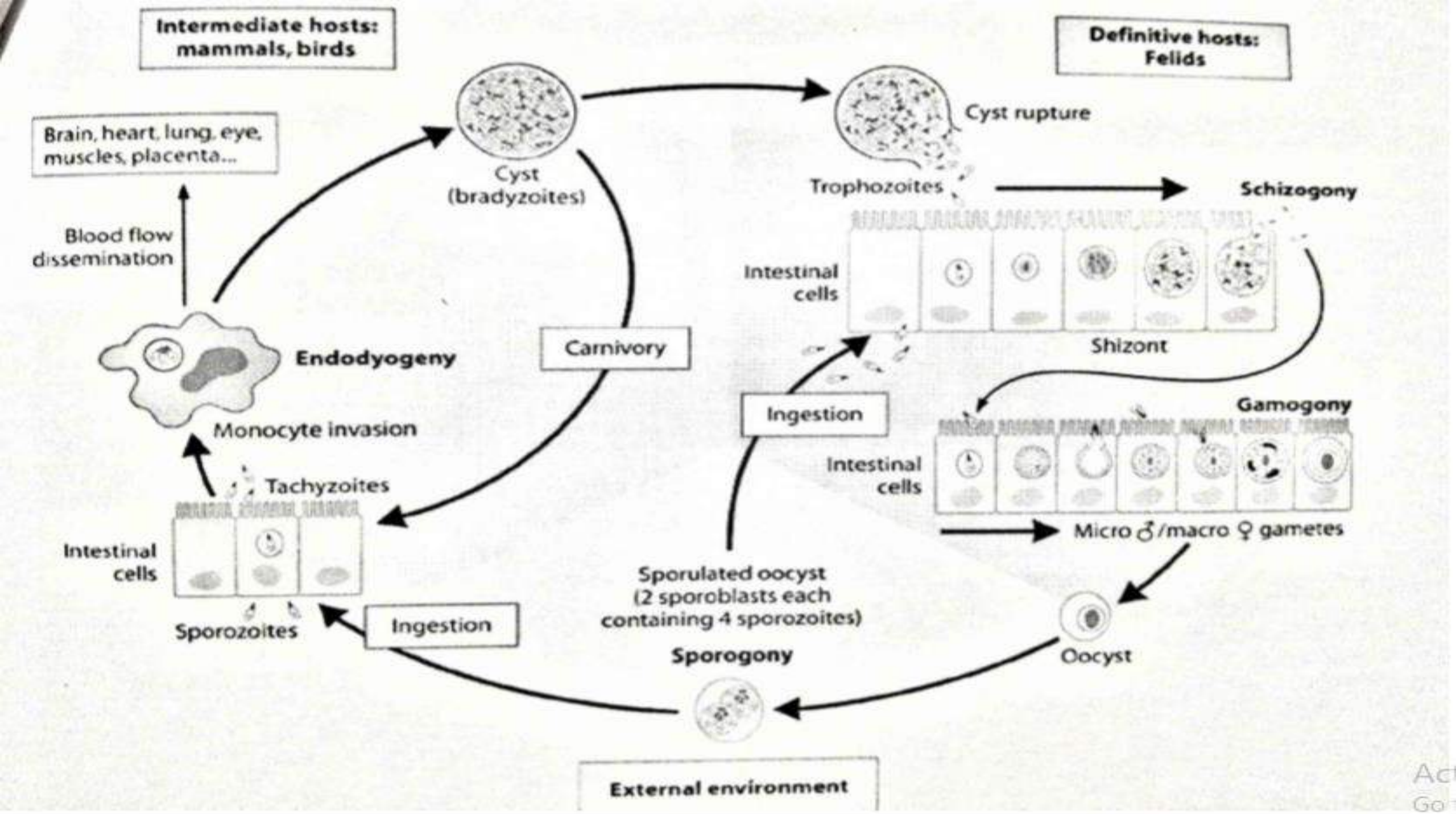
When a member of the cat family is infected with T. gondii (e.g. by consuming an infected mouse carrying the parasite's tissue cysts), the parasite survives passage through the stomach, eventually infecting epithelial cells of the cat's small intestine. Inside these intestinal cells, the parasites undergo sexual development and reproduction, producing millions of thick-walled, zygote-containing cysts known as oocysts.

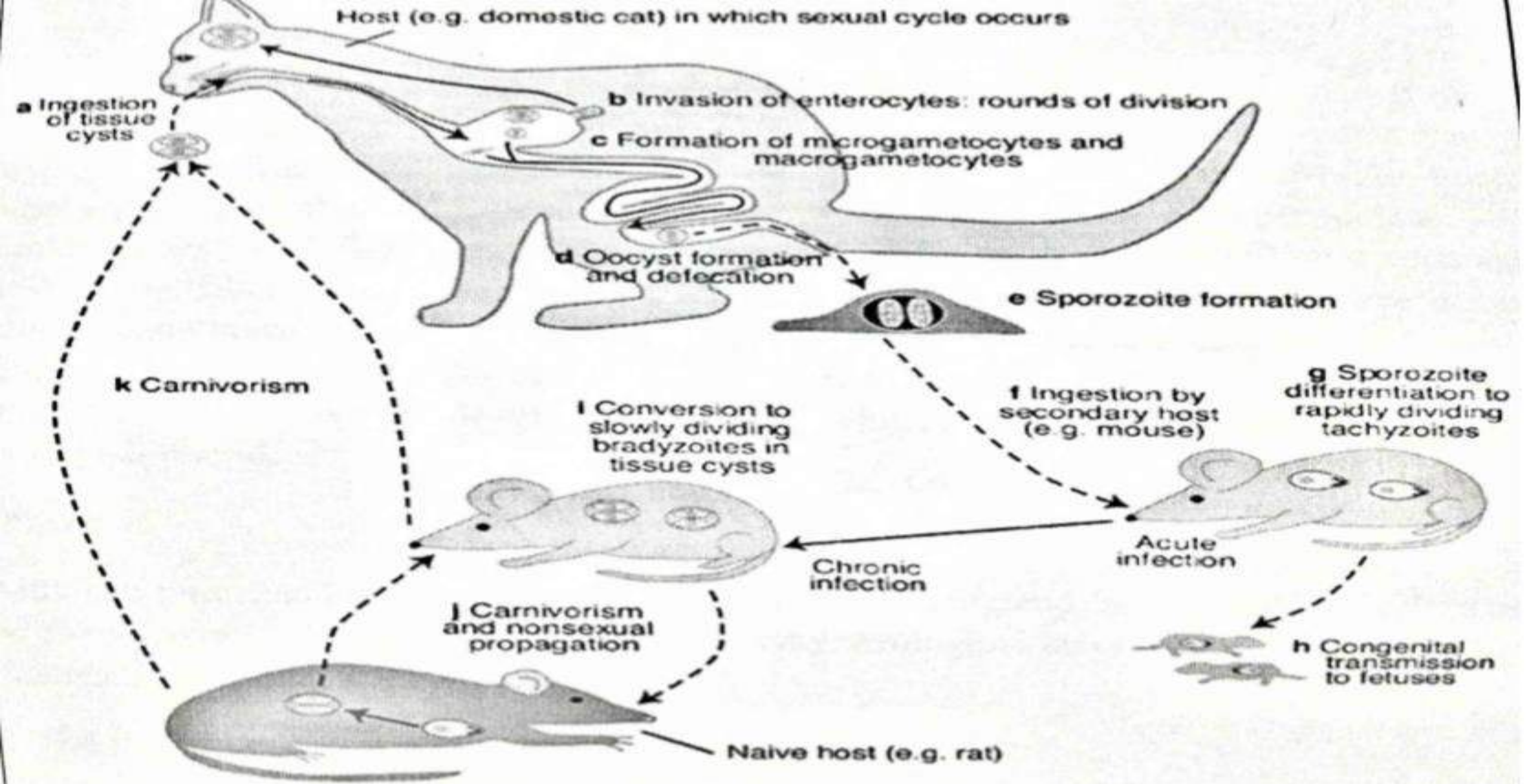
Infected epithelial cells eventually rupture and release oocysts into the intestinal lumen, and they spread to environment with feces . Oocysts can then spread to soil, water, food, or anything potentially contaminated with the feces. Highly resilient, oocysts can survive and remain infective for many months in cold and dry climates.

Asexual reproduction in the intermediate host

Ingestion of oocysts by humans or other warm-blooded animals is one of the common routes of infection. Humans can be exposed to oocysts by, for example, consuming unwashed vegetables or contaminated water, or by handling the feces (litter) of an infected cat. Although cats can also be infected by ingesting oocysts, they are much less sensitive to oocyst infection than are intermediate hosts. Excystation occurs in the small intestine and the librated tachyzoites penetrate the epithelial cells, Inside host cells, the tachyzoites replicate inside specialized vacuoles (called the parasitophorous vacuoles) created during parasitic entry into the cell. Tachyzoites multiply inside this vacuole until the host cell dies and ruptures, releasing and spreading the tachyzoites via the bloodstream to all organs and tissues of the body, including the brain.

It is important to mention that humans are dead-end intermediate host for this parasite unless they are eaten by a cat.


Intermediate hosts: mammals, birds
Definitive hosts: Felids
Brain, heart, lung, eye, muscles, placenta...
Cyst (bradyzoites)
Cyst rupture
Trophozoites
Schizogony
Blood flow dissemination
Intestinal cells
Shizont
Endodyogeny
Carnivory
Ingestion
Gamogony
Monocyte invasion
Tachyzoites
Intestinal cells
Micro ♂/macro ♀ gametes
Sporulated oocyst (2 sporoblasts each containing 4 sporozoites)
Sporozoites
Ingestion
Oocyst
Sporogony
External environment



The *Toxoplasma gondii* life cycle

TRANSMISSION

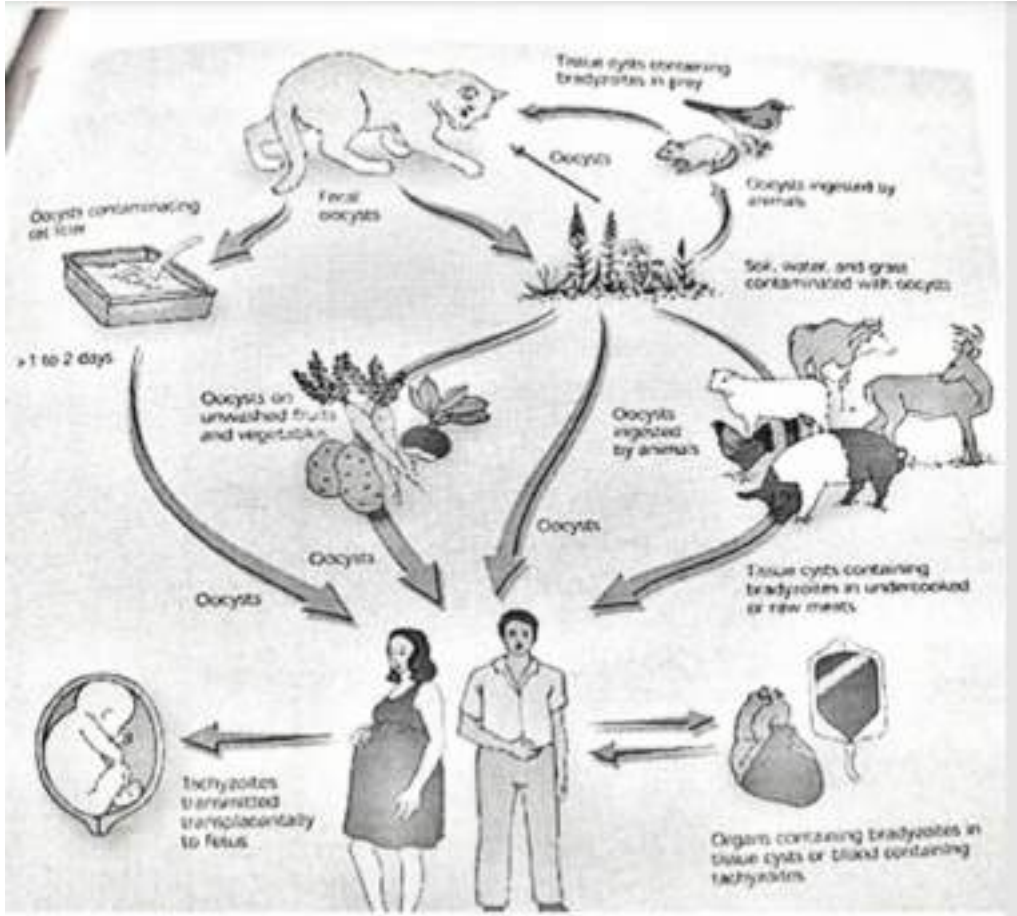
T. gondii is transmitted to humans by 3 principal routes:

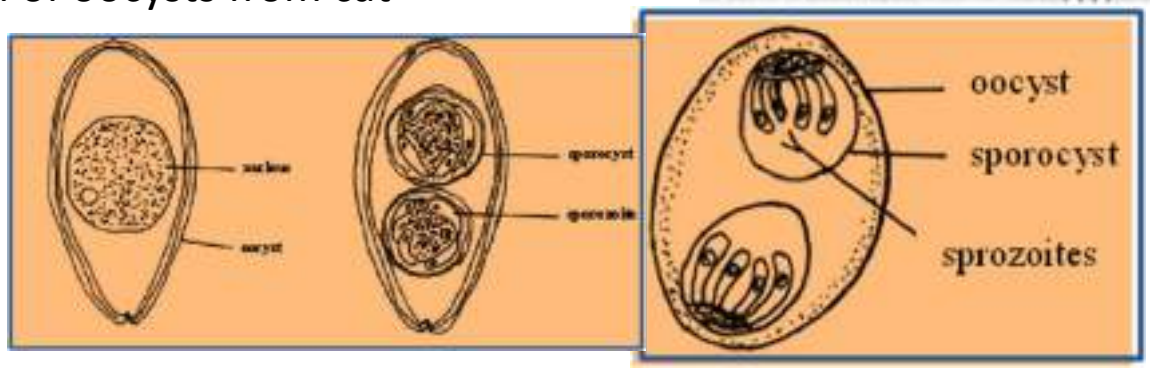
By eating raw or poorly cooked infected meat, especially pork, mutton, and wild animals or uncooked foods that have come in contact with infected meat.

Humans can accidentally (by mistake) ingest oocysts that cats have passed in their feces, either from a litter box or from soil (soil from gardening, on unwashed fruits or vegetables, or in unfiltered water).

Women can transmit the infection via placenta to their unborn fetus.

In adults, the incubation period for T. gondii infection ranges from 10 to 23 days after the ingestion of undercooked meat and from 5 to 20 days after the ingestion of oocysts from cat feces.





Immature oocyst (non-infective) **Developing oocyst** **Mature oocyst (infective sage)**

RISK FACTORS

Recent epidemiologic studies have identified the following risk factors for T. gondii infection:

Owning a cat.

Cleaning a cat litter box.

Eating raw or undercooked pork, mutton, lamb, beef, or minced-meat products.

Gardening.

Eating raw or unwashed vegetables or fruits.

Eating raw vegetables outside the home.

Having contact with contaminated soil.

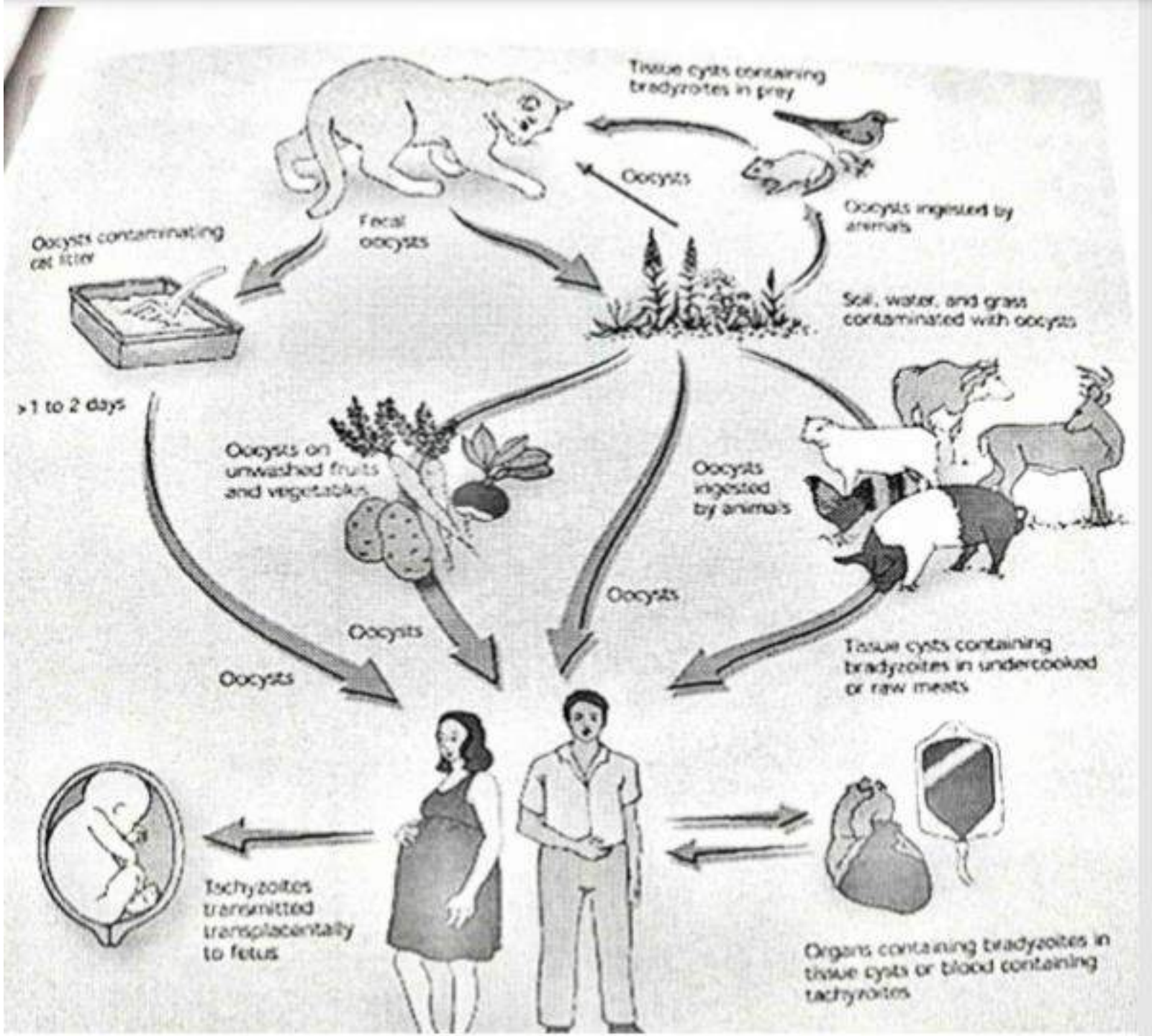
Washing kitchen knives.

Having poor hand hygiene.

Drinking water from a contaminated reservoir.

Women infected with T. gondii before conception rarely transmit the parasite to their fetus, but those who become acutely infected or have reactivation of T. gondii during pregnancy (i.e., because of immunosuppression) can transmit the organism transplacentally.

The risk of congenital disease is lowest (10 to 25 percent) when maternal infection occurs during the first trimester and highest (60 to 90 percent) when maternal infection occurs during the third trimester. However, congenital disease is more severe when infection is acquired in the first trimester.



Symptoms

•Some people (In immunocompetent adults) who have toxoplasmosis may feel as if they have the "flu" with swollen lymph glands (lymphadenopathy) or muscle aches and pains that last for a month or more, , while in immunocompromised individuals, involvement of brain, liver,lung and other organs, and often death . The majority of human infections are asymptomatic and the serious consequences are limited to pregnant women (congenital transmission) and immunodeficient hosts. Congenital infections may result in miscarriage and serious brain and eye damage to the fetus or may born with hydrocephalus, a buildup of fluid in the skull and with enlarged liver and spleen. Although up to 70% of infected women can give birth to a normal offspring, a small proportion of babies will develop active retino-chorditis or mental retardation in childhood or young adulthood.

Diagnosis

Although parasites can be isolated from tonsil or lymph gland biopsy, diagnosis does not depend on the recovery of the parasites. There are many serological methods that have been employed for diagnostic purposes such as:

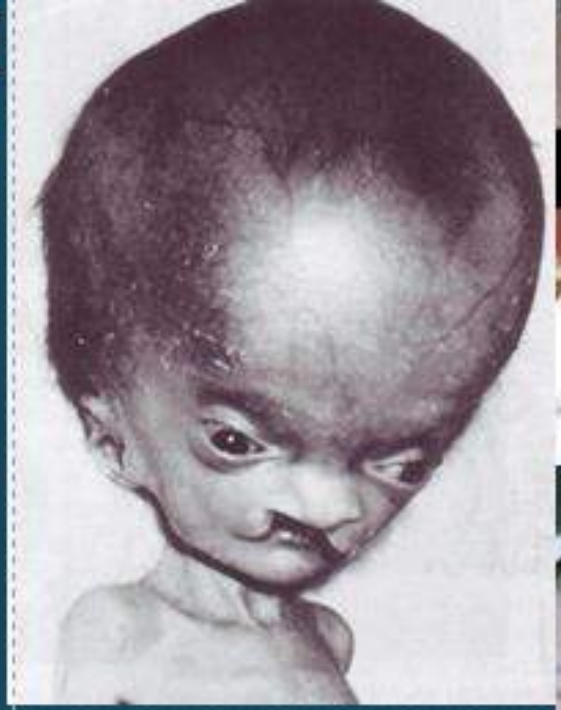
1-The toxoplasma dye test.
2-Fluorescent antibody test (FAT)
3-complement fixation test (CFT.)
4-Latex agglutination test.

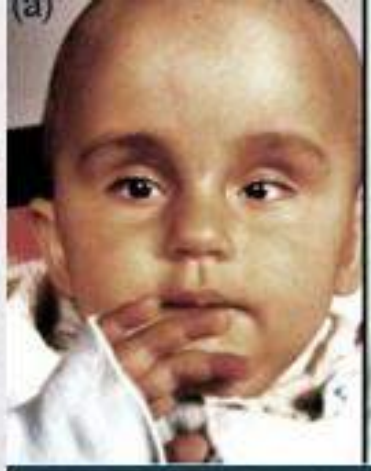
Symptoms

The majority of human infections are asymptomatic and the serious consequences are limited to pregnant women (congenital transmission) and immunocompromised hosts.

Congenital infections may result in miscarriage and serious brain and eye damage to the fetus. Although up to 70% of infected women can give birth to a normal offspring, a small percentage of babies will develop mental retardation in childhood or young adulthood.

In immunocompromised individuals, involvement of brain, liver, lung and other organs, and often death.





Pathogenesis

Eye lesion; lymphadenopathy
Congenital toxoplamosis:
Abortion;
Still birth (abnormities): hydrocephalus, mental retardation

Photograph of an infant with hydrocephalus and bilateral cleft palate

5-Enzyme-linked immunosorbent agglutination assay (*ELISA*), *IgG* for chronic infection whil
IgM for acute infection.

Control

To prevent human infection, the hands of people handling meat, meat cutting boards, sink tops, knives and other materials coming in contact with uncooked meat should be washed with water and soap. Pregnant women are advised to avoid cat litter and to handle uncooked and undercooked meat carefully

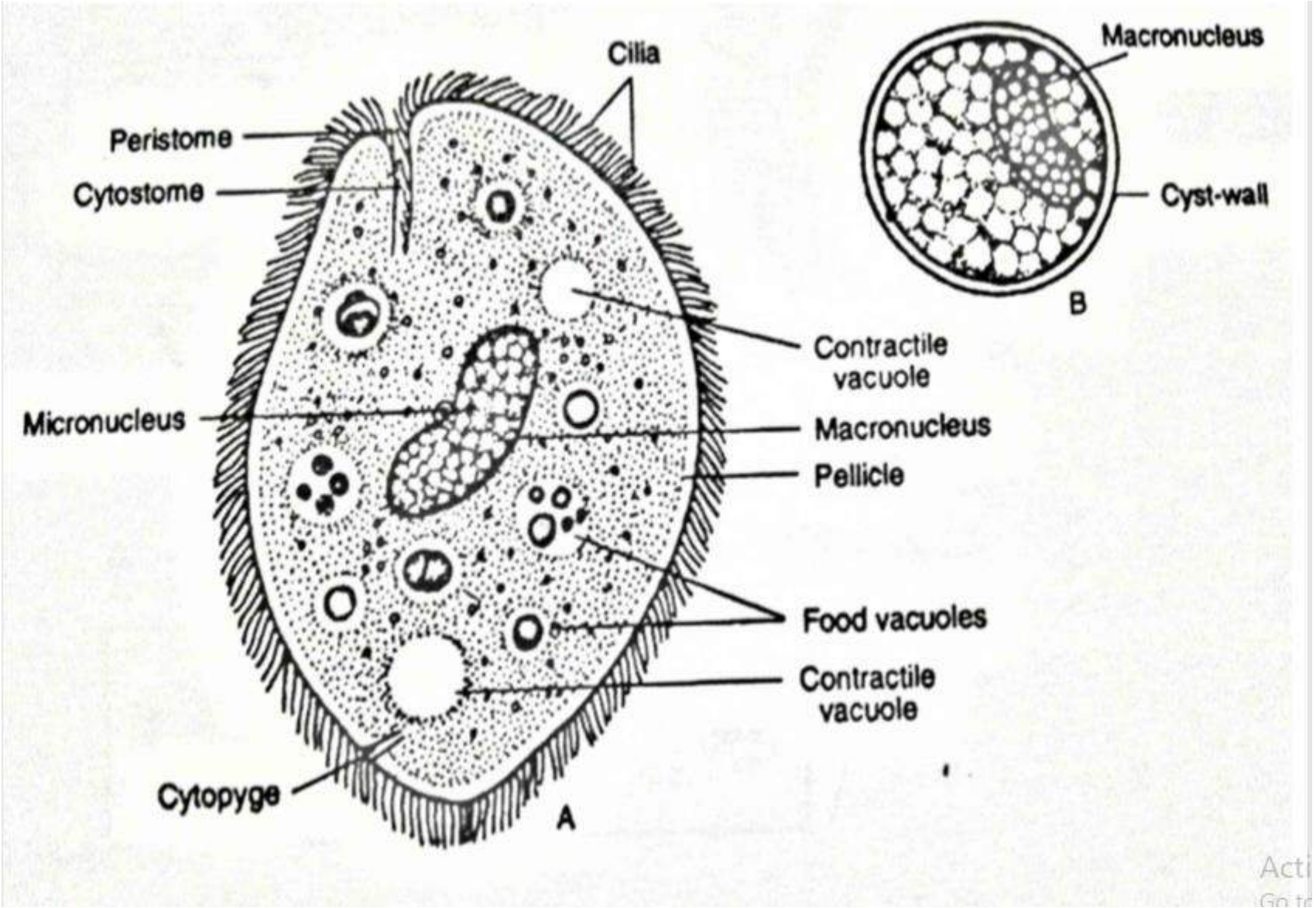
Congenital toxoplasmosis

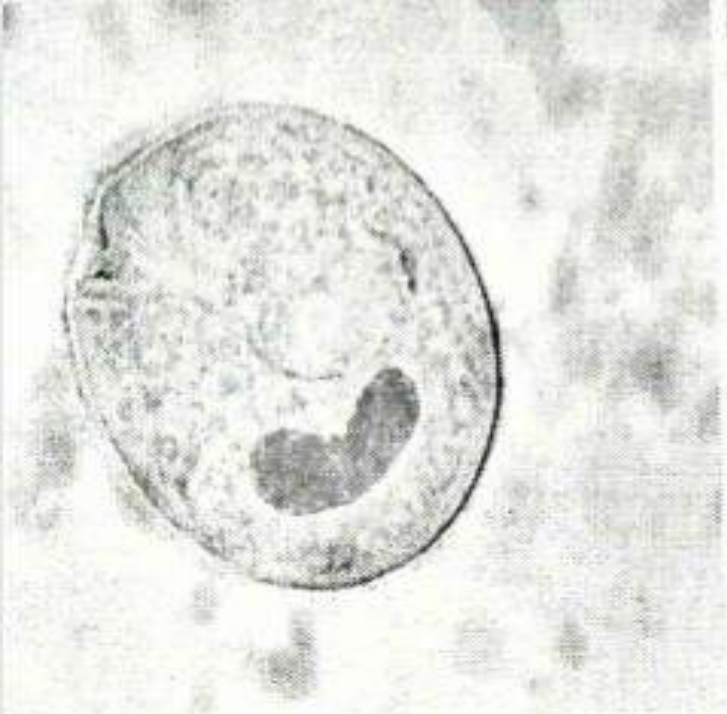
Balantidium coli is largest protozoan of the human in-testine inhabits the caecum. IT is the only member of the phylum Ciliophora which parasitizes humans and worldwide in its distribution. It moves by means of cilia and is acquired through faecal-oral transmission. A morphologically identical organism occurs in cows, pigs and horses. The infection occurs mostly in farm workers and other rural dwellers by ingestion of cysts in fecal material of farm animals. Human-to-human transmission is rare but possible.

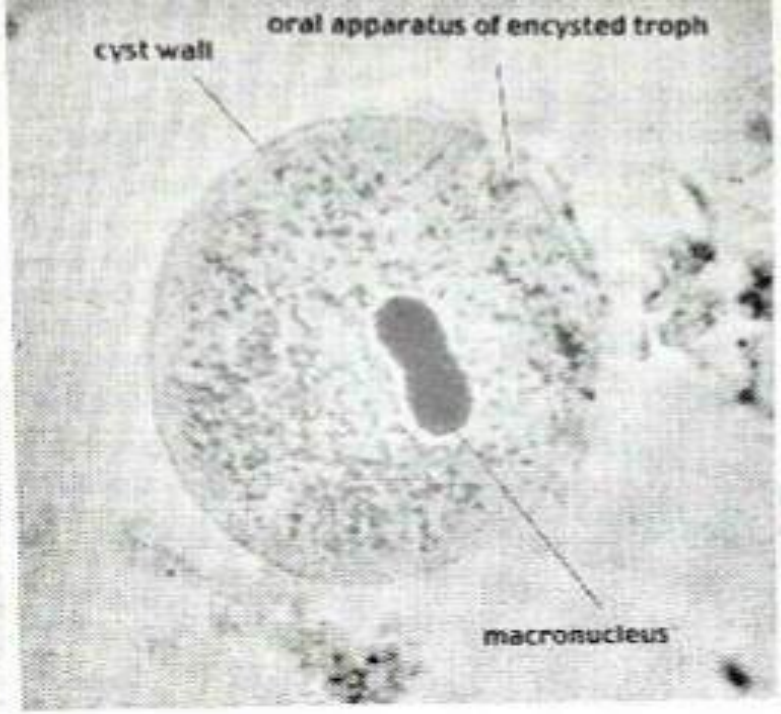
Morphology of the trophozoite:The outer surface, pellicle , is solid. Cilia are embedded in longitudinal rows. Near the middle of the frontal extremity the pellicle bears a funnel-shaped depression, called peristome. The peristome is provided with an opening, the cytostome at its The cilia, lining the peristome are larger than the rest of the body cilia. There are two contractile vacuoles and a number of food vacuoles. The cytopyge is an unclear anal opening situated at the end. Embedded in the cytoplasm there are kidney-shaped macronucleus(and may be spherical, ellipsoid, elongate, curved) and a very small, rounded micronucleus hidden within the concavity of the macronucleus .

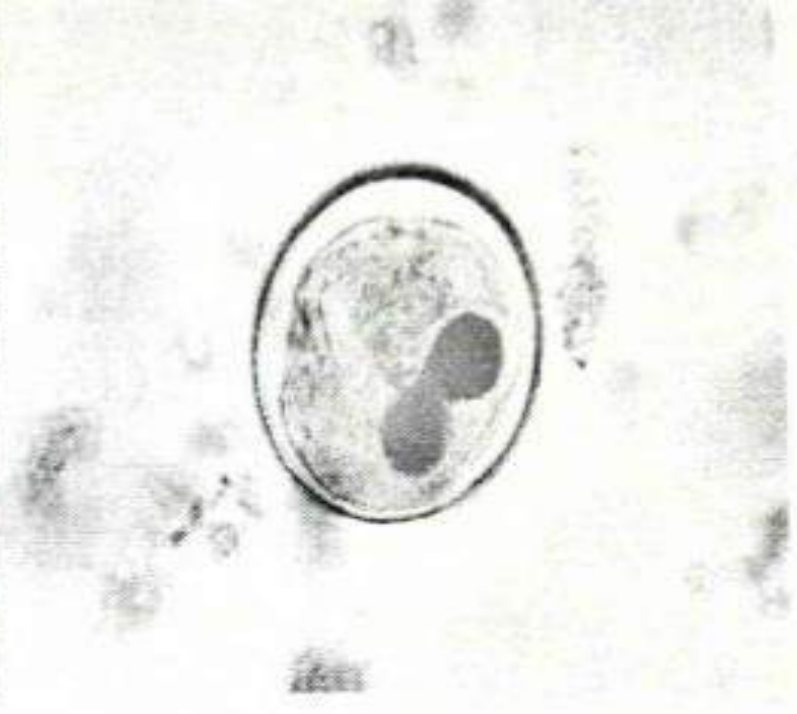
Morphology of the cyst

The cyst The cysts are nearly spherical, the walls are thick and refractile. In stained specimens the macronucleus can usually be seen within the cyst wall, but other structures usually are not observed.


Cilia
Peristome
Cytostome
Micronucleus
Contractile vacuole
Macronucleus
Pellicle
Food vacuoles
Contractile vacuole
Cytopyge
A
Macronucleus
Cyst-wall
B

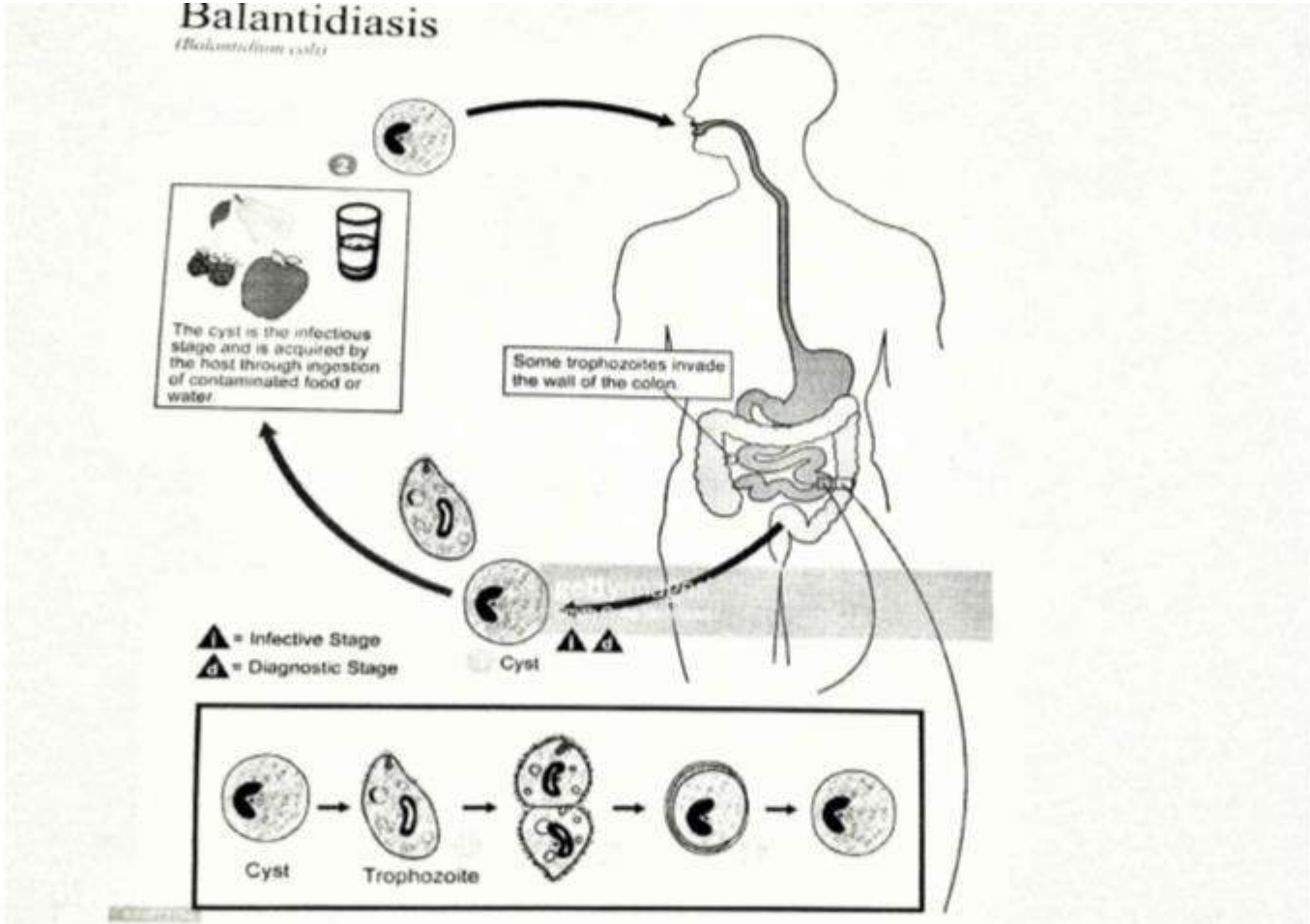






Life cycle: Balantidiasis trasmits from one host to another by cysts . When the host ingests the cyst with the contaminated food or water, excystation occurs in the small intestine, and the trophozoites colonize the large intestine. The trophozoites live in the lumen of the large intestine of humans and animals, where they replicate by binary fission. Under certain conditions, trophozoites encyst and the cysts are passed with the feces.

Sexual reproduction occurs by conjugation. The process resembles that of paramecium in minute details

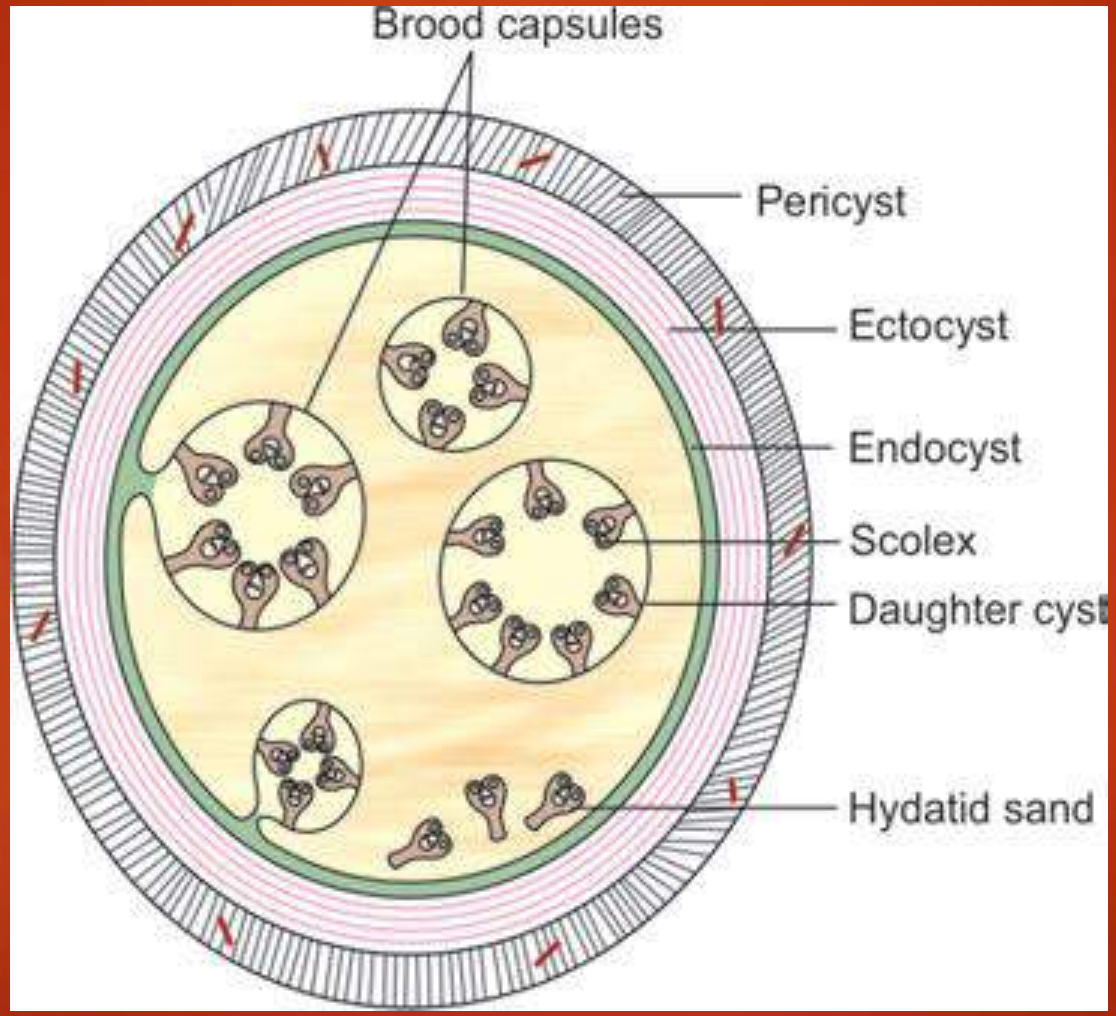


Transmission of Disease from Balantidium Coli :

Transmission from one host to another occurs in encysted condition through drink-ing water and contaminated food .

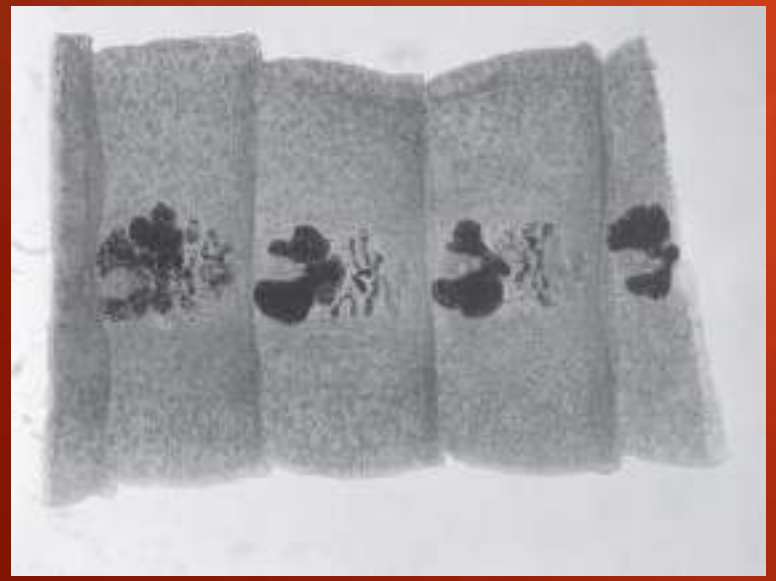
The specific damages done by Balantidium are not known and no more than one- fifth of the infected subjects experiences symptoms. So some observers prefer to consider it as a commensal .But there is Symptoms Generally occure in some cases, symptoms of balantidiasis are similar to those seen in amoebic dysentry, including intestinal epithelial erosion. Although liver, lung and brain abscesses have been reported, they are uncommon. The trophozoites produce an enzyme called hyaluronidase, which possibly Diagnosis facilitates the invading of the mucosa. a few fatal cases have been reported

Diagnosis Difinitive diagnosis depends on the detection of either trophozoites or cysts in feces of infected persons.



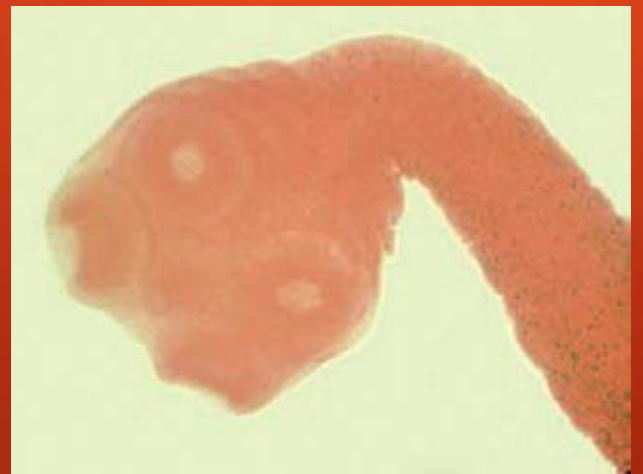
Microscopy hydatid cyst
For previous lecture

The adult tapeworm has three regions. Following the scolex, there is a short "neck" region followed by any number of segments, called proglottids (Figure 1). Another characteristic feature of an adult tapeworm is the absence of a digestive tract, because the adult worms inhabit the small intestine, a principal digestive system of the human host. This lack of an alimentary tract requires nutritive substances to enter the tapeworm across the tegument (membranous structure resembling skin that covers the segments). This structural arrangement is well adapted for transport functions because it is covered with numerous microvilli (hair-like projections) resembling those lining the lumen (open area of a tube-like structure) of the mammalian intestine. These villi serve to increase the surface area of the lumen and provide for better absorption. The excretory system is a specialized excretory cell type called a flame cell that functions in a similar manner to both kidneys and anus in mammals.



Of the two species in humans, Taenia saginata, or the beef tapeworm, is the larger of the two and T. solium, the pork tapeworm, use cattle and pigs as their respective intermediate hosts. The scientific study of the taeniid tapeworms of humans can be traced to the late seventeenth century and the observations of Edward Tyson, a British physician and scientist of anatomy who studied the tapeworms of humans, dogs, and other animals. Tyson was apparently the first person to recognize the "head" (scolex) of a tapeworm, and descriptions of the anatomy and physiology of the adult tapeworms led to the current body of knowledge regarding the biological life cycle of the taeniid tapeworms of humans. By the time of Tyson's contributions, it was quite evident that there were basic differences between the broad fish tapeworm and other tapeworms now categorized as taeniids, but the differentiation between T. solium and T. saginata was not fully discovered until later.

It was not until some years later that observations led to the belief that there were actually two species of Taenia responsible for infections by what was originally thought to be a single organism. The life cycles of the two species, T. solium and T. saginata, are practically the same. However, humans are capable of acting as the intermediate host for T. solium but not as an intermediate host for T. saginata. In the mid-nineteenth century, a some scientist fed the pork tapeworm to others who became infected. They been credited with recognizing the differences between T. solium and T. saginata based on the morphology of the scolex of the two tapeworms, which are now named after their primary hosts (pig and the cow). The morphology of a Taenia solium tapeworm scolex reveals four suckers and two rows of hooks, making it almost impossible to dislodge the scolex from the tissue where it is attached . In the human intestine the cysticercus, or larval stage, for the organism develops over approximately a 2-month period into an adult tapeworm. These adults can survive for years by attaching to and residing in the small intestine by using the suckers and hooks located on its scolex.



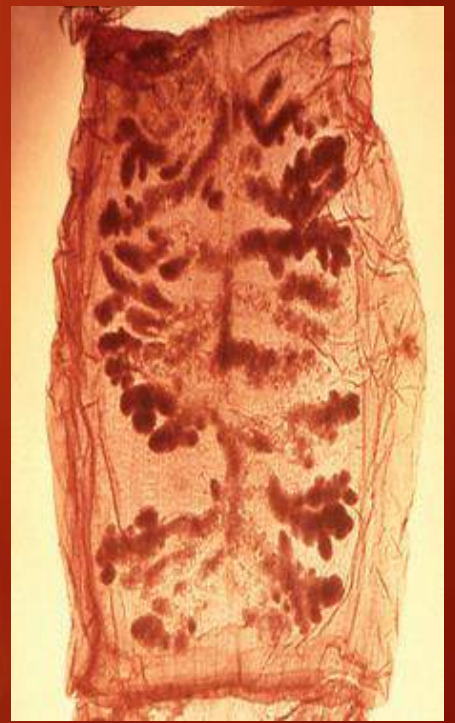


Table 10.4: Differences between *Taenia saginata* and *Taenia solium*

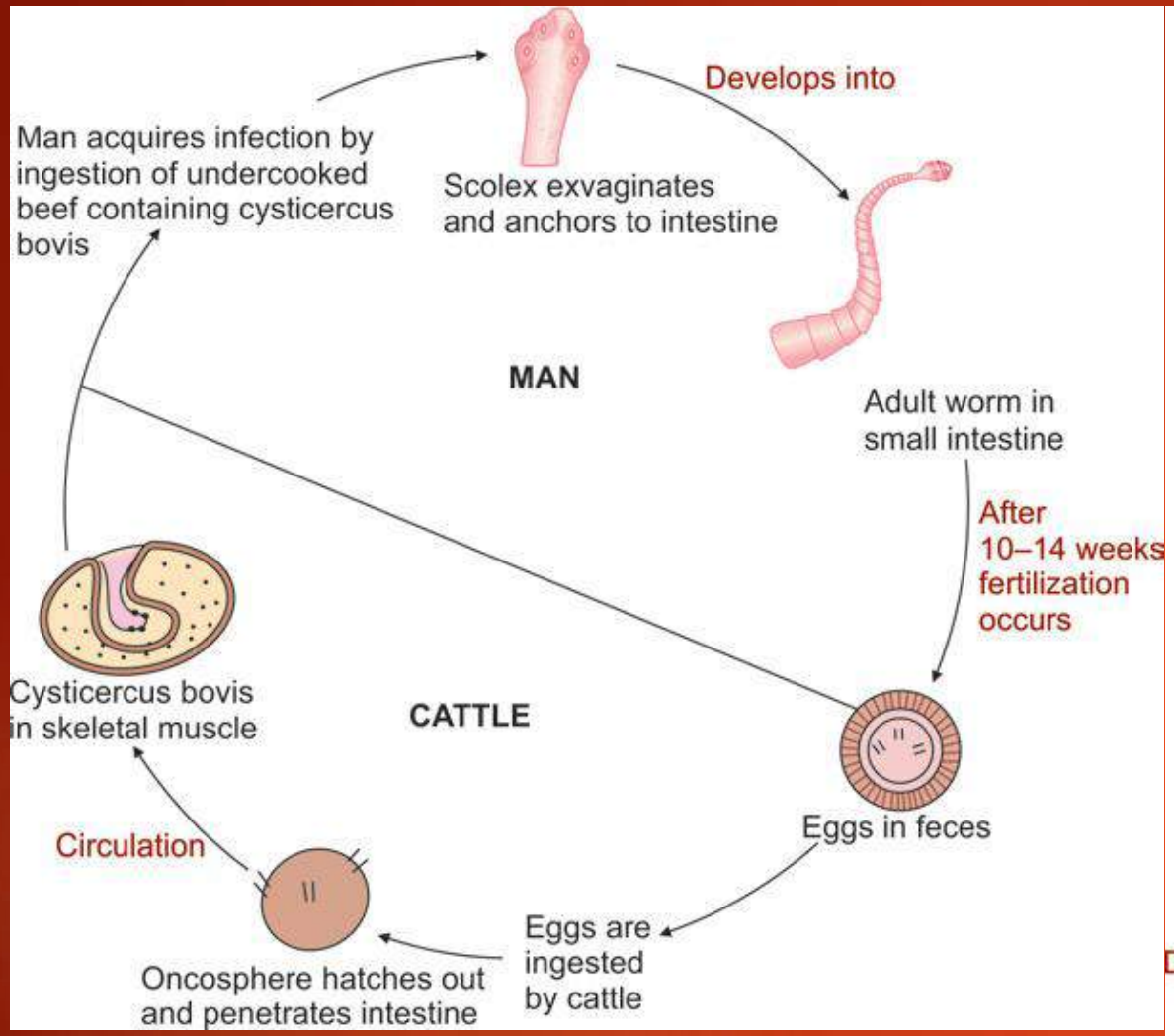
Features	*Taenia saginata*	*Taenia solium*
Adult worm		
Length	4–6 meters or more	2–4 meters
Head/scolex	• Large and quadrangular • Four suckers present which may be pigmented • No rostellum, No hooklets	• Small and globular • Four suckers present—not pigmented • Bears rostellum with two rows of hooklets • Hence called as armed tapeworm
Neck	Longer	Shorter
Proglottids		
No. of Proglottids	1,000–2,000	800–1,000
Uterus	Bears in 15–20 lateral branches	Bears in 7–13 lateral branches
Lobes of Ovary	Two, No accessory lobe	Three–two lobes with an accessory lobe
Testes	300–400 follicles	150–200 follicles
Vaginal sphincter	Present	Absent
Measurement	Gravid segment—20mm× 5mm	Gravid segment—12mm × 6mm
Expulsion of segments	Expelled singly in the feces	Expelled in chain of 5–6 segments
Eggs per segment	80,000 eggs per gravid segment	40,000 eggs per gravid segment
Larva		
	Cysticercus bovis present in cattle's muscle, but not in man	Cysticercus cellulosae present in pig's muscle and also in man (muscle, eye and brain)
Egg		
	Acid fast	Non-acid fast
Life cycle		
Disease	Causes intestinal taeniasis	Causes intestinal taeniasis & cysticercosis
Host	Definitive host: Man Intermediate host: Cattle	For intestinal taeniasis- • Definitive host: Man • Intermediate host: Pig For Cysticercosis- • Both definitive and intermediate host: Man
Infective form	Larva (cysticercus bovis)	For intestinal taeniasis—Larva (cysticercus cellulosae) For cysticercosis—egg
Diagnostic form	Egg	For intestinal taeniasis—egg For cysticercosis—larva (cysticercus cellulosae deposited in tissue)
Mode of transmission	Ingestion of contaminated beef	For intestinal taeniasis—ingestion of contaminated pork For cysticercosis • Contaminated food and water • Autoinfection

Cysticercus is the larval stage of Taenia. It contains a muscular organ with bladder like sac. It is called as: Cysticercus cellulosae in T. solium Cysticercus bovis in T. saginata Larval stage of T. saginata and T. solium is infective to man (to cause intestinal taeniasis). Life cycle of T. solium depends on the disease it causes. When it causes intestinal taeniasis, the life cycle is exactly similar to that of T. saginata except: The intermediate host is pig (hence called as pork tapeworm) Men harboring the adult worm excrete the eggs in feces which can infect the same individual by autoinfection . In pigs, the development time is shorter (7–9 weeks). But when it causes cysticercosis, the life cycle is different and given as below:

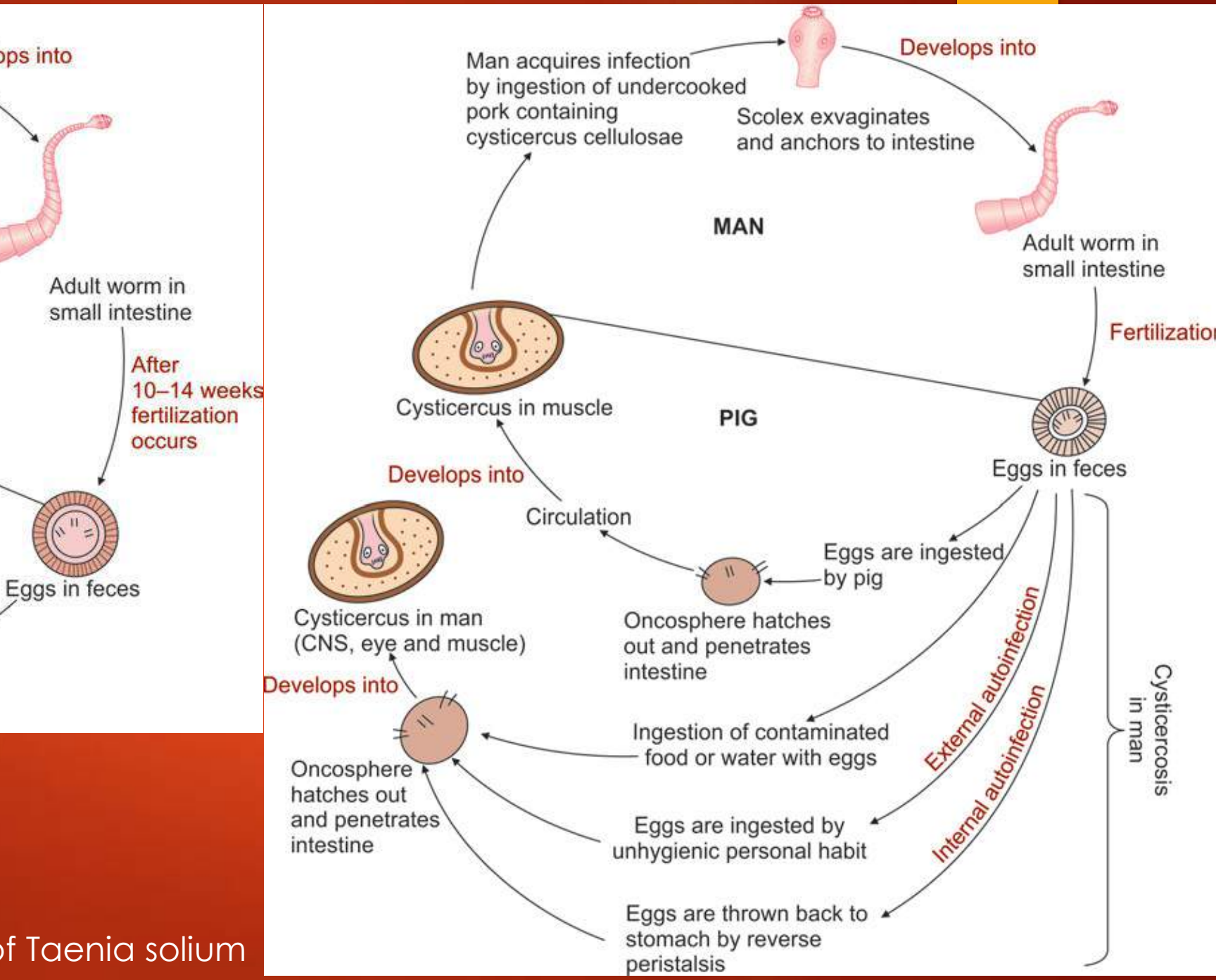
Host: Man acts as both defi nitive and intermediate host.

Infective stage: Eggs of T. solium.

Mode of transmission: Firstly man acquire the infection by—(1) ingestion of contaminated food or water with eggs of T. solium and (2) autoinfection



Life cycle of Taenia saginata



Life cycle of Taenia solium

Autoinfection: Eggs excreted from men reinfect the same individual. Autoinfection can be of two types:
external autoinfection: Due to unhygienic personal habit, e.g., contaminated finger
Internal autoinfection: Due to reverse peristaltic movements by which the gravid segments throw the eggs back into the stomach (equivalent to swallowing of the eggs) Further life cycle in men is similar to that in pigs: Oncosphere is released from the eggs, penetrates the intestine and enters into the portal circulation or mesenteric lymphatics and reaches to various organs like subcutaneous tissue, muscle, eye and brain where it is transformed to the larval stage cysticercus cellulosae in 7–9 weeks and deposited as cyst. Full development to mature cysts takes 2–3 months of time

pathogenesis and Clinical features: Intestinal Taeniasis
Both T. saginata and T. solium can cause intestinal taeniasis. Often, it is asymptomatic; patients become aware of the infection most commonly by noting the passage of proglottids in their feces . The proglottids are often motile, and patients may experience perianal discomfort (or pruritus) when proglottids are discharged . Mild abdominal pain or discomfort, nausea, loss of appetite, weakness, weight loss, headache and change in bowel habit (constipation or diarrhea) can occur . Occasionally obstruction by the migrating proglottids can result in appendicitis or cholangitis.

Neurocysticercosis
z NCC is the most common form and accounts for 60–90% cases of cysticercosis

Neurocysticercosis

NCC is the most common form and accounts for 60–90% cases of cysticercosis. NCC is considered as the most common parasitic CNS infection of man and the most common cause of adult onset epilepsy throughout the world . Based on the site of involvement, NCC is of two types:

1. Parenchymal: Involves brain parenchyma
2. Extraparenchymal sites are meninges and spinal cord .

Asymptomatic neurocysticerosis (NCC):Sometimes NCC remains in the brain without causing any symptoms

NCC exists in four morphological stages: It starts as vesicular form, gradually develops into necrotic, followed by nodular and finally into calcified stage

The clinical presentation is variable and depends on number, location & size of the cyst, the morphological stage of the cyst and the host immune response.

laboratory diagnosis Intestinal taeniasis

- Stool examination—Detects eggs, proglottids
- Taenia specifi c coproantigen detection in stool—ELISA
- Antibody detection in serum—ELISA, CFT, Immunoblot
- Molecular method—PCR

laboratory diagnosis Cysticercosis

- Radiodiagnosis— CT scan and MRI
- Antibody detection in serum or CSF—ELISA, Western blot (EITB)
- Antigen detection in serum or CSF- ELISA
- Lymphocyte transformation test
- Histopathology of muscles, eyes, subcutaneous tissues or brain biopsies

Helminth parasites have developed complex and versatile mechanisms to evade the immune responses of their hosts, utilizing immunoregulatory strategies to avoid immune effector mechanisms. In general, these processes are necessary for the parasites to complete their long life cycles and/or to favor host survival [2]. Despite their great evolutionary divergence and variety of stages, life cycles, and pathogenic and invasive mechanisms, helminths have developed similar strategies and induce strikingly similar immune responses, which have been called "stereotypical Th2-type immune responses." However, there are differences in the immune responses evoked by distinct helminths, mainly with regard to leukocyte involvement and the roles of these cells.

The Th2 response induced by helminth parasites is characterized by the secretion of high levels of anti-inflammatory cytokines such as interleukin-6 (IL-6), IL-9, IL-10, IL-25, IL-33, and transforming growth factor-β (TGF-β), but the main cytokines are IL-4 and IL-13. As a result and/or of this cytokine secretion, there are alterations in leukocyte activation, such as high levels of CD4+ T lymphocytes that differentiated into Th2 and T regulatory (Treg) subsets, the activation of immunoglobulin G1 (IgG1)- and IgE-producing by B cells, eosinophilia, basophilia, Interestingly, there are another type of cell can be found in this response such as : an immature dendritic cell (iDC) and huge populations of alternatively activated macrophages (AAMs) with the ability to suppress lymphocyte proliferation. Furthermore, another characteristic of Th2 responses is the suppression of the immune response to by antigens, which may compromise the effectiveness of vaccination and alter the immune response to several other antigens, even autoantigens.

It is commonly accepted that most of these changes in leukocyte phenotype and activation, as well as in the induction of the inflammatory milieu, are dependent upon the ability of the parasite to excrete/secrete antigens with immunoregulatory properties .

Taenia solium is responsible for two diseases in humans, i.e. taeniosis and neurocysticercosis, which are caused by the adult tapeworm and the larval stage (cysticercus), respectively. Tapeworms lodge in the small intestine of human beings after ingestion of live cysticerci in contaminated, undercooked pork meat; develop into adult tapeworms and expel gravid proglottids full of eggs in feces. Accidental intake of eggs by humans results in the development of neurocysticercosis, due to the establishment of cysticerci in the central nervous system. Neurocysticercosis is a public health problem in many developing countries. Taeniosis is usually asymptomatic but epidemiological studies have shown that tapeworm carriers are the main risk factor for developing neurocysticercosis . Human beings are the only definitive hosts for T. solium. Thus, the use of experimental models, such as the Syrian golden hamster (Mesocricetus auratus), is necessary to study the mechanisms involved in the immune response elicited by the tapeworm.
Helminth infections generally induce Th2 responses. Molecules derived from helminths that stimulate Th2 responses have been subject of current research for their potential regulatory immune functions. However, only few molecules involved in triggering such responses have been recognized.

Hymenolepis nana

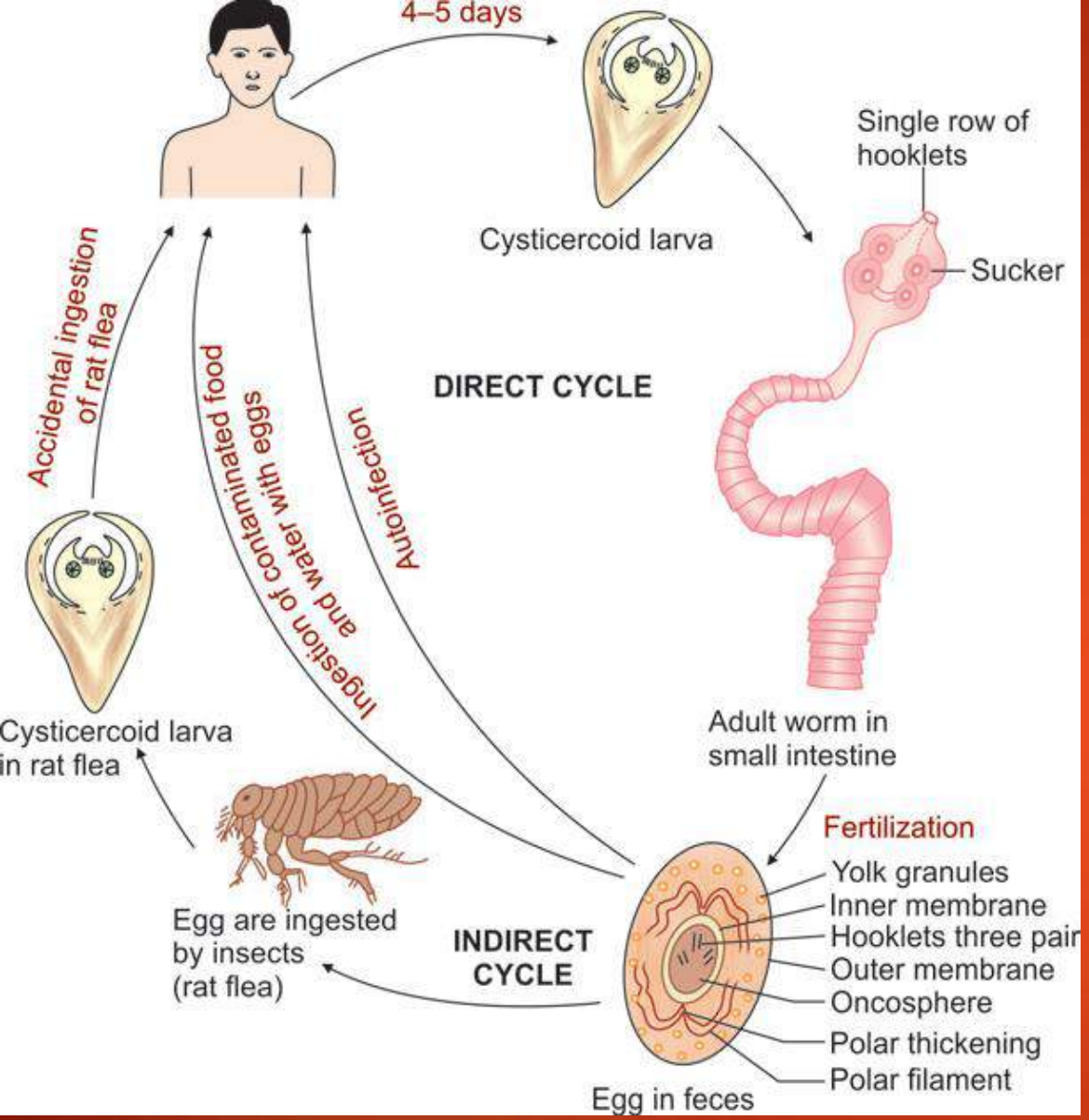
It is the smallest cestode infecting man, hence called as dwarf tapeworm. Name Hymenolepis refers to a thin membrane covering the eggs (Hymen membrane, lepis covering, and nana small size). It was first detected by Bilharz in 1857 in small intestine. H. nana is considered as the most common tapeworm infection throughout the world infecting 50–75 million of people. The overall prevalence ranges from 0–4% with higher prevalence in children (16%).

Morphology

Adult Worm is small, 1–4 cm in length and con sists of head, neck and strobila. It resides in the small intestine (upper two third of ileum). Head/scolex: It is globular with four suckers and a rostellum bearing single row of 20–30 hooklets . Neck: It is long and gives rise to proglottids . Strobila: Consists of 200 segments (proglottids). Mature proglottids contain both male and female reproductive organs. Genital pore opens laterally on the same side. Uterus has lobulated wall and there are only three testicular follicles (Rest description is similar to any other cyclophyllidean cestodes described earlier).

Egg

Eggs are the infective form as well as the diagnostic form of the parasite. Egg is round to slightly oval, 30–47 µm size. It has two membranes (outer egg shell and an inner embryophore) and an oncosphere .with six hooklets. Space between the two membranes is filled with yolk granules . Polar filaments: Both the poles of embryophore are thickened from which four to eight polar filaments emerge . Non bile stained (colorless in saline mount): It is the only cestode egg that is not stained by bile when passed through intestine. The larval form is called cysticercoids. It is solid except the proximal part which is vesicular and contains the scolex



Life cycle of Hymenolepis nana

Two life cycles are noted, i.e. direct and indirect cycle
Direct Cycle
Host: Man is the only host. There is no intermediate host. Rodents (rat and mice) are the other hosts. Infective form: Eggs Mode of transmission: Men acquire the infection by: 1-Ingestion of food and water contaminated with eggs. 2- Autoinfection with their own eggs released in the small intestine.
In the small intestine, eggs hatch out, penetrate the intestinal wall and develops into cysticercoid larvae in 4–5 days.Thereafter, the intestinal villi rupture and cysticercoids larvae become free in the gut lumen and transform into the adult worms in 10–12 weeks. Adult worm, when fully mature undergoes fertilization to produce eggs. Eggs are passed in the feces which are infective to man. Though the adult worm lives only about 4–10 weeks, the infection persists due to autoinfection.

Indirect Cycle

Host: Man is the definitive host. Insects act as intermediate host such as rat flea Xenopsylla cheopis and beetles like Tribolium castaneum or T. confusum . Mode of transmission: Men acquire the infection rarely, by accidental ingestion of insects containing the cysticercoid larva. In human intestine: Th e larva develops into adult worm in human smalll intestine which then produces eggs that are passed in the feces. In rat fleas: Eggs are ingested by the insects, embryo hatches out, penetrate the intestine and develop into the larval stage cysticercoid

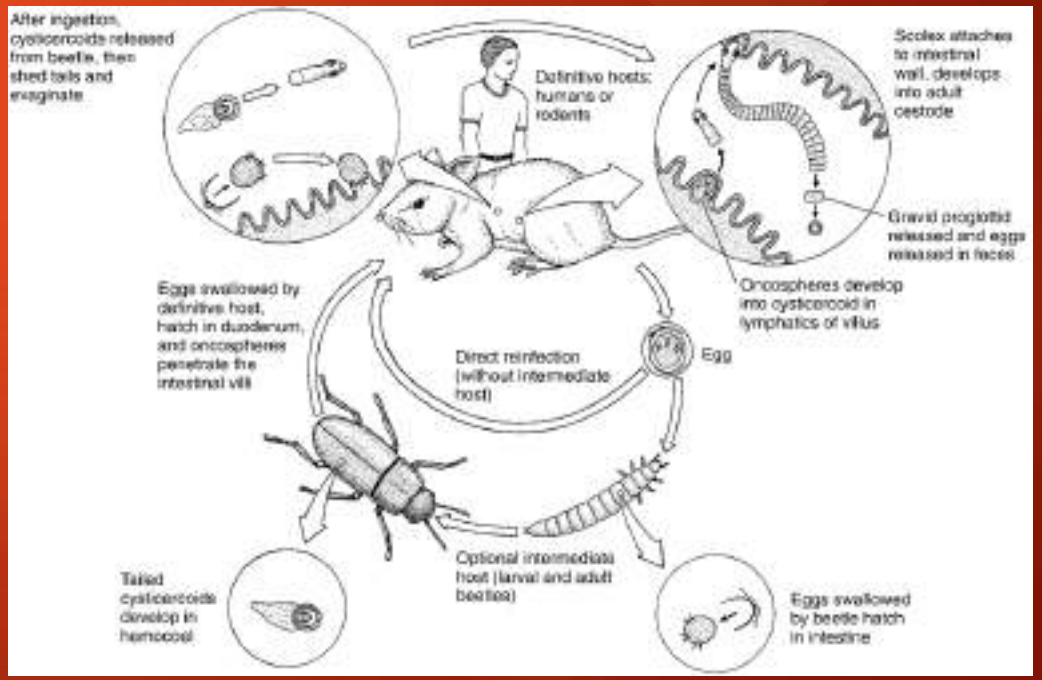
larva in the insect's body cavity. Th is stage is infective to men.

Clinical features and pathogenicity

H. nana infection is usually asymptomatic. When infection is intense and the worm burden exceeds 1000-2000 worms, patients develop symptoms like anorexia, abdominal pain, headache, dizziness and diarrhea

Stool examination (detects non bile stained eggs with polar fi laments between the shell membranes)

Eosinophilia are the diagnosis methods



has two sets of male and female reproductive systems and a genital pore on each side .The scolex has a retractable, rather pointed rostellum with several circles of rose thorn-shaped hooks. Its uterus disappears early in its development and is replaced by hyaline, noncellular egg capsules, each containing 8 to 15 eggs. Gravid proglottids detach and either wander out of the anus or are passed with feces. They are very active at this stage and are the approximate size and shape of cucumber seeds. As detached segments begin to desiccate, egg capsules are released. Fleas are the usual intermediate hosts, although chewing lice have also been implicated. Unlike adults, larval fleas have simple, chewing mouthparts and feed on organic matter, which may include D. caninum egg capsules. The resulting cysticercoids survive their host's metamorphosis into the parasitic adult stage, when fleas may be nipped or licked out of the fur of a dog or cat, thereby completing the life cycle. This, by the way, is an example of hyperparasitism, since the flea is itself a parasite. Nearly every reported case of infection of humans has involved a child. Adult humans may be more resistant, or else children may have increased chances of accidentally swallowing a flea. The symptoms and treatment are the same as for Hymenolepis nana. The only feature separating this family from Hymenolepididae is a larger number of testes, usually more than 12. This family, too, consists of hundreds of species that parasitize birds and mammals.

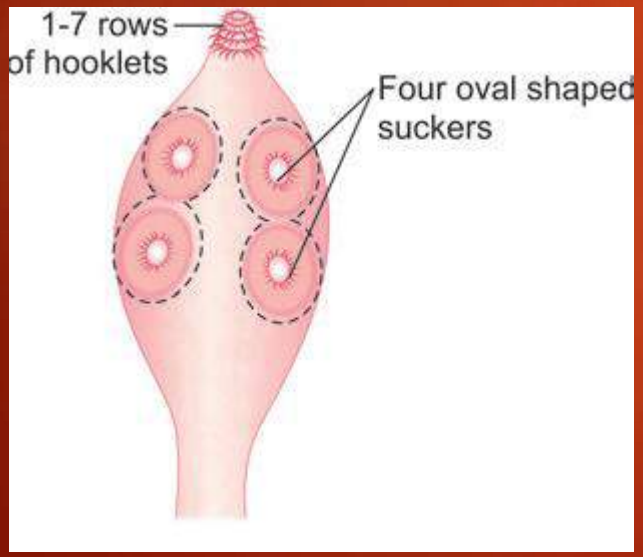
THE INFECTION Mostly, asymptomatic, but rarely symptoms like indigestion, loss of appetite, diarrhea, pruritus ani, abdominal pain may be reported. Children are aff ected commonly

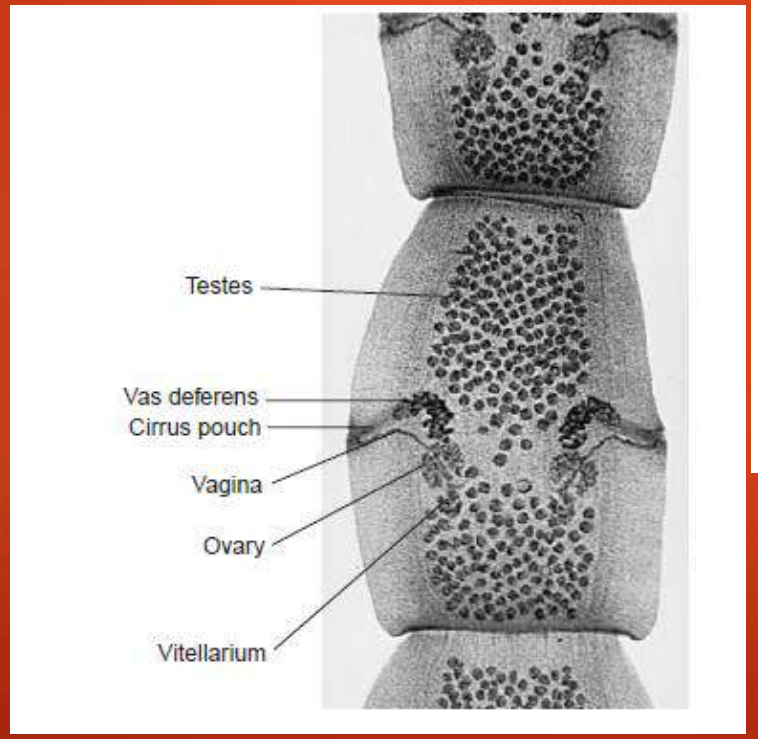
Dipylidiun caninum (double pored tapeworm)

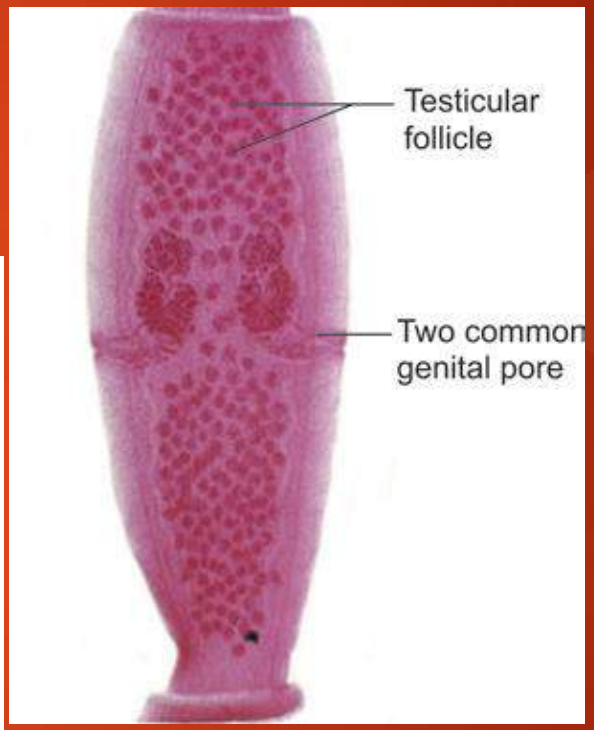
Th is is a common tapeworm of dogs and cats.Morphology:Adult worm is 10–70 cm long, scolex contains four oval suckers and is armed with rostellum and 1–7 rows of hooklets .life Cycle: It resembles with the indirect cycle of H. nana.Host: There are two types of hosts:1. Defi nitive host: Dogs and cats (rarely m

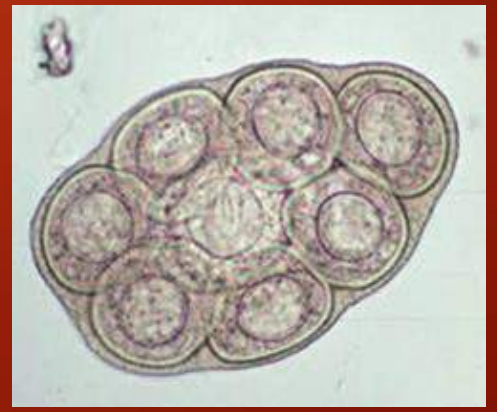
Intermediate host: Insects (fleas)Man acquires infection by ingestion of flea containing cysticercoid larva

Dipylidium caninum A cosmopolitan, common parasite of domestic dogs and cats, *Dipylidium caninum* often occurs in children.[66] It is easily recognized because each segment





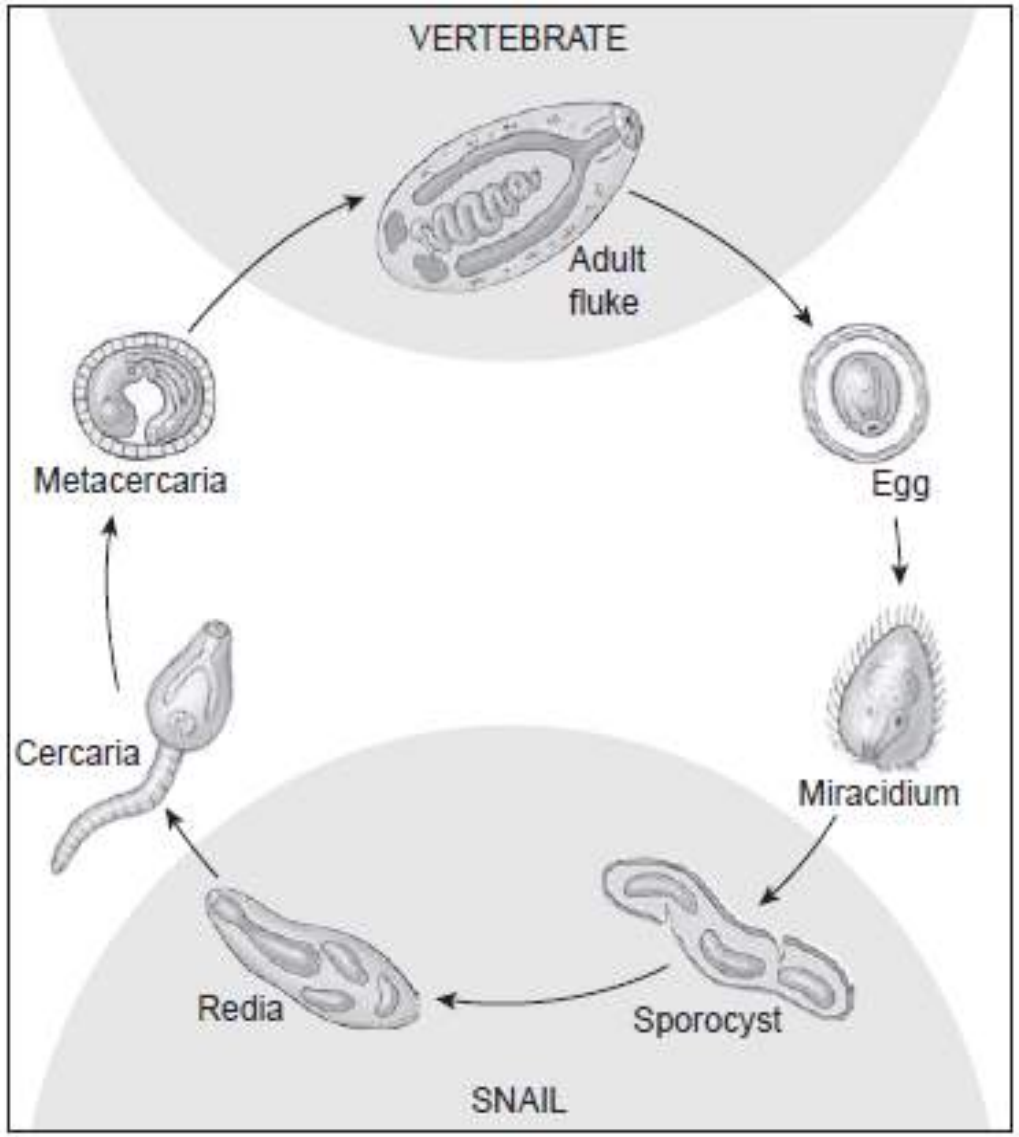




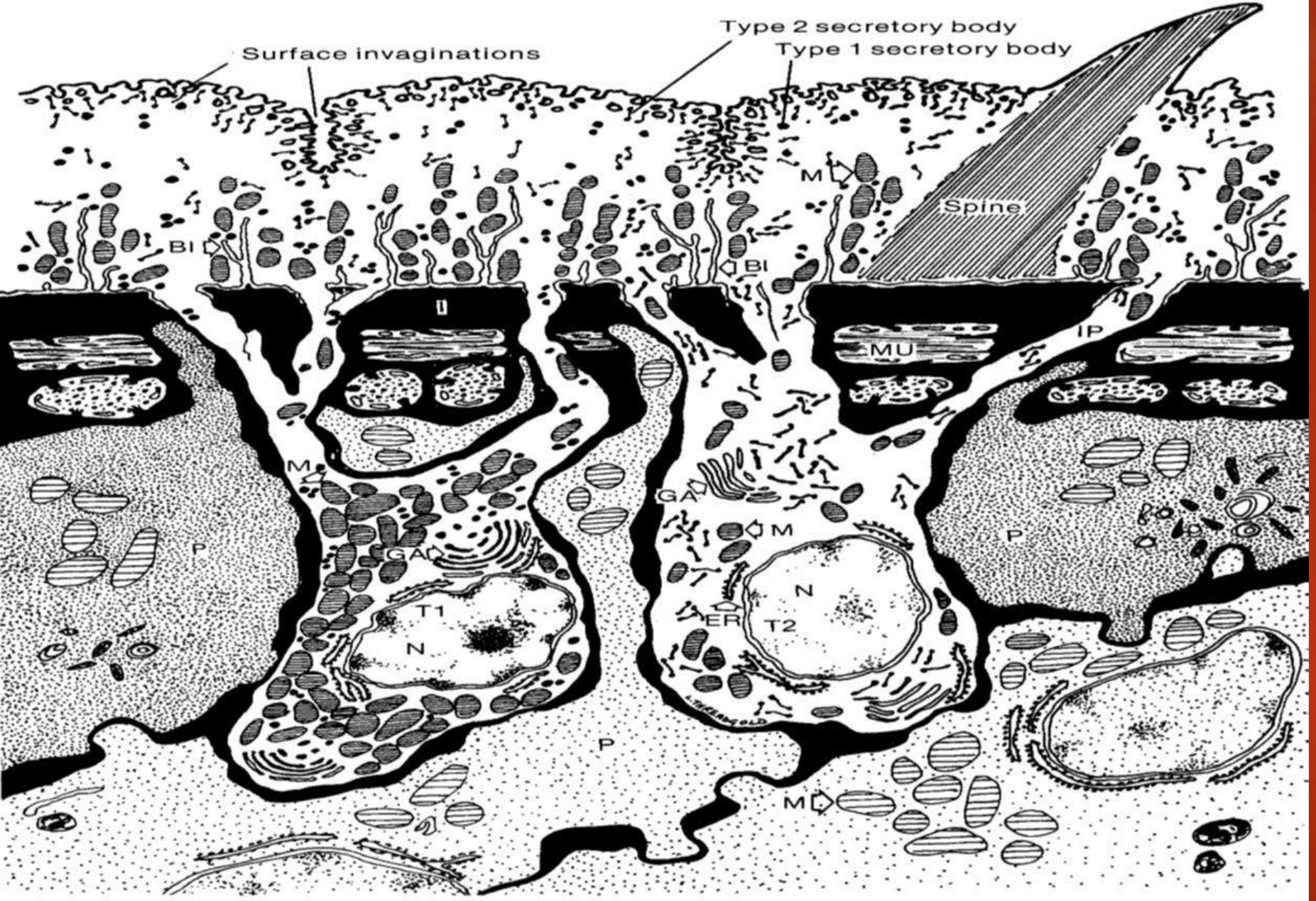
Digenea

Digenetic trematodes, or flukes, are among the most common and abundant of parasitic worms, second only to nematodes in their distribution. They are parasites of all classes of vertebrates, especially marine fishes, and nearly every organ of the vertebrate body can be parasitized by some kind of trematode, as adult or juvenile. Digenean development occurs in at least two hosts. The first is a mollusc or, very rarely, an annelid. Many species include a second and even a third intermediate host in their life cycles. Several species cause economic losses to society through infections of domestic animals, and others are medically important parasites of humans. Because of their importance, Digenea have stimulated vast amounts of research, and literature on the group is immense. This chapter will summarize digenean morphology and biology, illustrating them with some of the more important species.

Trematode "typical" life cycle is as follows: A ciliated, free-swimming larva, a miracidium, hatches from its shell and penetrates a first intermediate host, usually a snail. At the time of penetration or soon after, the larva discards its ciliated epithelium and metamorphoses into a rather simple, saclike form, a sporocyst. Within the sporocyst a number of embryos develop asexually to become rediae. Rediae are somewhat more differentiated than sporocysts, possessing, for example, a pharynx and a gut, neither of which are present in a miracidium or sporocyst. Additional embryos develop within the redia, and these become cercariae. Cercariae emerge from the snail. They usually have a tail to aid in swimming. Although many species require further development as metacercariae before they are infective to a definitive host, cercariae are properly considered juveniles; they have organs that will develop into an adult digestive tract and suckers, and genital primordia are often present. A fully developed, encysted metacercaria is infective to a definitive host and develops there into an adult trematode. Many trematodes have a second intermediate host which bears their encysted metacercariae. Their vertebrate definitive hosts are then infected when they consume the second intermediate host.



Flukes exhibit a great variety of shapes and sizes as well as variations in internal anatomy. They range from the tiny Levinseniella minuta, only 0.16 mm long, to the giant Fascioloides magna, which reaches 5.7 cm in length and 2.5 cm in width. Most flukes are dorsoventrally flattened and oval in shape, but some are as thick as they are wide. Some species are filiform, round, or even wider than they are long. Flukes usually possess a powerful oral sucker that surrounds the mouth, and most also have a midventral acetabulum or ventral sucker. The tegument of Trematodes, like that of cestodes, formerly was considered a nonliving, secreted cuticle; but, as in cestodes, electron microscopy reveals that the body covering of trematodes is a living, complex tissue. In common with Monogenoidea and Cestoidea, digenetic trematodes have a "sunken" epidermis; there is a distal, anucleate layer. IN NUCLEATED LAYER there is a cell containing the nuclei (cytons) lie beneath a superficial layer of muscles, connected to the distal cytoplasm by way of channels (cytoplasmic bridges). Because the distal cytoplasm is continuous, with no intervening cell membranes, tegument is syncytial. Although this is the same general organization found in cestodes, trematode tegument differs in many details, and striking differences in structure may occur in the same individual from one region of the body to another

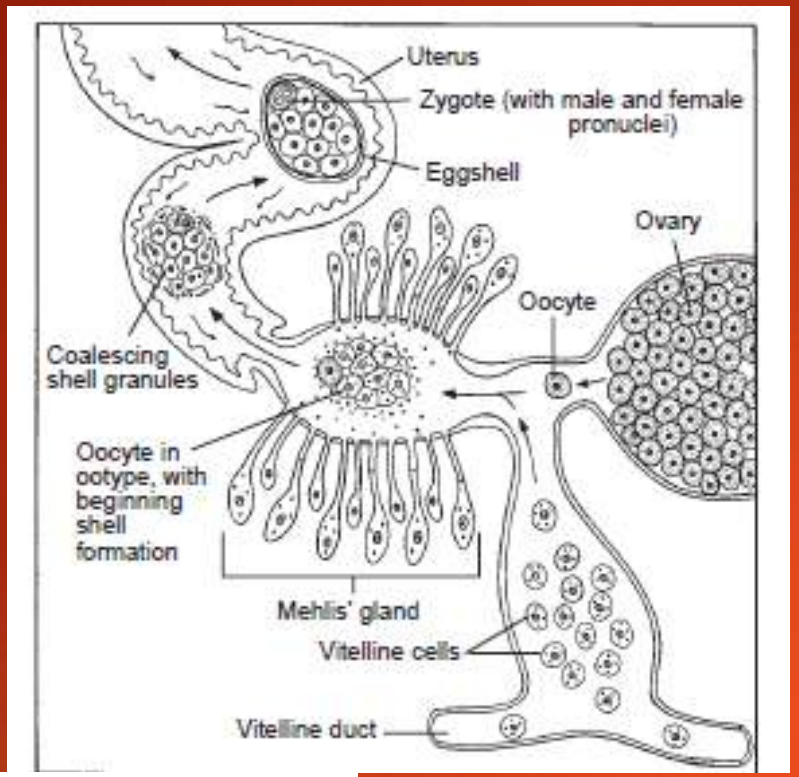


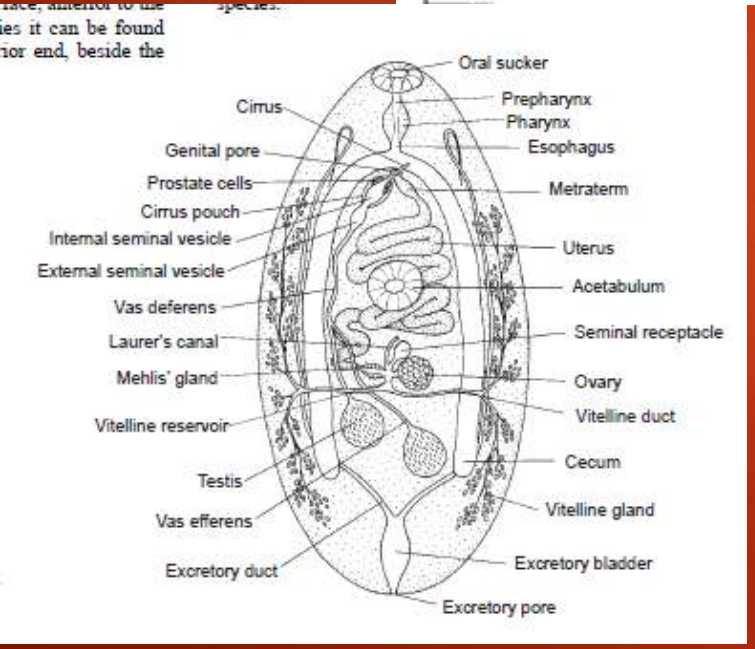
Muscles that occur most consistently throughout Digenea are circular muscles lying just beneath the basal lamina of the tegument, with longitudinal and diagonal layers underlying the circular muscles. These muscle layers envelop the rest of the body like a sheath

cerebral ganglion, and three main pairs of trunks—dorsal, lateral, and ventral supply posterior parts of the body. Ventral nerves are usually the most developed. Branches provide motor and sensory endings to muscles and tegument. The anterior end, especially the oral sucker, is well supplied with sensory endings

The excretory system of Digenea is based on flame bulb protonephridia (units of an excretory system closed at the proximal end and opening to the exterior at the distal end by way of a pore). Most trematodes are hermaphroditic (important exceptions are schistosomes), and some are capable of self-fertilization. Others, , require cross-fertilization to produce viable progeny. Some species inseminate themselves readily; others however will do so if there is only one worm present, but they always seem to cross-inseminate when there are two or more in the host. Some species will not inseminate themselves or even mature unless there is another adult worm present. Worms find each other by means of chemoattractants, and, except in schistosomes, the active compound appears to be cholesterol. A few instances are known in which adult trematodes can reproduce parthenogenetically

Male reproductive systems usually include two testes, although the number varies with species from one testis to several dozen. Shape of the testes varies from round to highly branched, according to species. Each testis has a vas efferens that connects with others to form a vas deferens; this duct then courses toward the genital pore, which is usually found within a shallow genital atrium. Before reaching the genital pore, the vas deferens usually enters a muscular cirrus pouch where it may expand into an internal seminal vesicle (within the pouch) for sperm storage. Female Reproductive System The single ovary in the female reproductive tract is usually round or oval, but it may be lobated or even branched. A short oviduct is provided with a proximal sphincter, or ovicapt, that controls passage of ova. The oviduct and most of the rest of the female ducts are ciliated. A seminal receptacle forms as an outpocketing of the wall of the oviduct. It may be large or small, but it is almost always present. At the base of the seminal receptacle there often arises a slender tube, Laurer's canal, which ends blindly in the parenchyma or opens through the tegument. Laurer's canal is probably a vestigial vagina that no longer functions as such (with a few possible exceptions), but it may serve to store sperm in some species





Female Reproductive System The single ovary in the female reproductive tract is usually round or oval, but it may be lobated or even branched. A short oviduct is provided with a proximal sphincter, or ovicapt, that controls passage of ova. The oviduct and most of the rest of the female ducts are ciliated. A seminal receptacle forms as an outpocketing of the wall of the oviduct. It may be large or small, but it is almost always present. At the base of the seminal receptacle there often arises a slender tube, Laurer's canal, which ends blindly in the parenchyma or opens through the tegument. Laurer's canal is probably a vestigial vagina that no longer functions as such (with a few possible exceptions), but it may serve to store sperm in some species

Schistosomatidae

1- include individuals live in blood vessels
2-life cycle lack metacercaria and redia
3- the individuals are dioecious
4- fork-tail cercariae
5-require one intermediate host (snail)

people in 74 countries at risk.12 Some 200 million people are infected with schistosomes, of whom 120 million are symptomatic and 20 million suffer from severe disease. Three species of schistosomes are of vast medical significance: Schistosoma haematobium, S. mansoni, and S. japonicum. All parasites of humans since antiquity. Bloody urine was a well recognized disease symptom in northern Africa in ancient times. At least 50 references to this condition have been found in surviving Egyptian papyri, and calcified eggs of S. haematobium have been found in Egyptian mummies dating from about 1200 B.C. Hulse presented a well-reasoned hypothesis that the curse that Joshua placed on Jericho can be explained by an introduction of S. haematobium into the communal well by the invaders. Removal of the curse occurred after abandonment of Jericho and eliminated the snail host, Bulinus .Today Jericho (Ariha, Jordan) is well-known for its fertile lands and healthy, well-nourished people The first Europeans to record contact with S. haematobium were surgeons with Napoleon's army in Egypt (1799–1801). They reported that hematuria (bloody urine) was prevalent among the troops, although the cause, of course, was unknown. Nothing further was learned about schistosomiasis haematobia for more than 50 years, until a young German parasitologist, Theodor Bilharz, discovered the worm that caused it. He announced his discovery in letters to his former teacher, Von Siebold, naming the parasite Distomum haematobium

While information was accumulating on biology of S. haematobium, some investigators began to doubt whether it was a single species or whether two or more species were being confused. The problem was confounded by the observation in some patients of eggs with terminal spines in both urine and feces. Whenever eggs with lateral spines were noticed they were ignored as "abnormal." In 1905 Sir Patrick Manson decided that intestinal and vesicular (urinary bladder) schistosomiasis usually were distinct diseases, caused by distinct species of worms. He reached this conclusion when he examined a man from the West Indies who had never been to Africa and who passed laterally spined eggs in his feces but none at all in his urine.38 Sambon argued in favor of the two-species concept in 1907, and he named the parasites producing laterally spined eggs Schistosoma mansoni. (Japanese zoologists had already detected still another species by this time, but their reports were generally unknown to Europeans.)

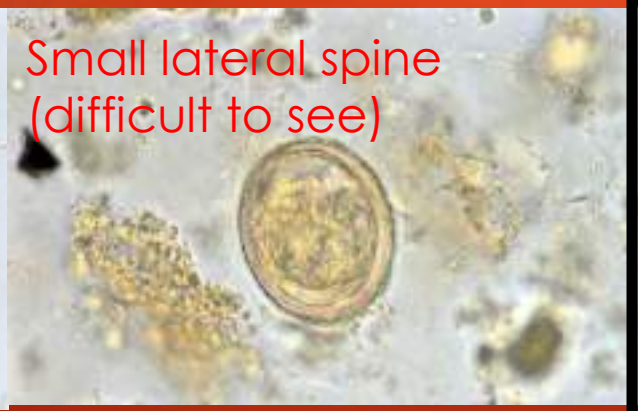
Although Schistosoma spp. are generally similar structurally, several differences in detail are listed in Table bellow .Considerable sexual dimorphism exists in the genus, males being shorter and stouter than females The males have a ventral, longitudinal groove, the gynecophoral canal, where the female normally resides. The mouth is surrounded by a strong oral sucker, and the acetabulum is near the anterior end. There is no pharynx. The paired intestinal ceca converge and fuse at about the midpoint of the worm and then continue as a single gut to the posterior end. Males possess five to nine testes, according to species, each of which has a delicate vas efferens, and these combine to form a vas deferens. The latter dilates to become a seminal vesicle, which opens ventrally through the genital pore immediately behind the ventral sucker. Cirrus pouch, cirrus, and prostate cells are absent. The suckers of females are smaller and not so muscular as those of males, and tegumental tubercles, if any, are confined to the ends of females. The ovary is anterior or posterior to or at the middle of the body, and the uterus is correspondingly short or long, depending on species.

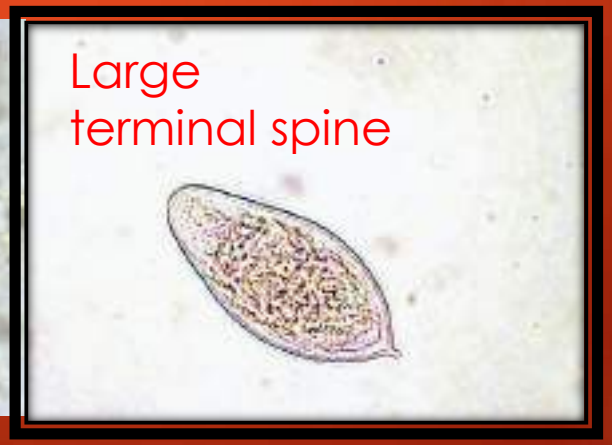
Table 16.1 Comparative Morphology of the Three Primary Species of Human Schistosomes

Characteristic	*S. haematobium*	*S. mansoni*	*S. japonicum*
Tegumental papillae	Small tubercles	Large papillae with spines	Smooth
Size			
Male			
Length	10–15 mm	10–15 mm	12–20 mm
Width	0.8–1.0 mm	0.8–1.0 mm	0.50–0.55 mm
Female			
Length	ca. 20 mm	ca. 20 mm	ca. 26 mm
Width	ca. 0.25 mm	ca. 0.25 mm	ca. 0.3 mm
Number of testes	4–5	6–9	7
Position of ovary	Near midbody	In anterior half	Posterior to midbody
Uterus	With 20–100 eggs at one time; average 50	Short; few eggs at one time	Long; may contain up to 300 eggs; average 50
Vitellaria	Few follicles, posterior to ovary	Few follicles, posterior to ovary	In lateral fields, posterior quarter of body
Egg	Elliptical, with sharp terminal spine; 112–170 μm × 40–70 μm	Elliptical, with sharp lateral spine; 114–175 μm × 45–70 μm	Oval to almost spherical; rudimentary lateral spine; 70–100 μm × 50–70 μm

Parasite species	Definitive host	Site of infection	Egg excretion	Snail vector	Geographic location
S.haematobium	humans, primates	veins of urogenital system	urine	*Bulinus*	Africa
S. mansoni	humans, rodents	intestinal mesenteric veins	faeces	*Biomphalaria*	Africa, America
S. japonicum	humans, ruminants, carnivores	intestinal mesenteric veins	faeces	*Oncomelania*	SE Asia

Schistosoma haematobium Common name: Bladder fluke.
Common Schistosoma spp. disease and condition names: Schistosomiasis, bilharziasis, swamp fever, Katayama fever.
There are many species of schistosomes but three important species infect humans. Although the differences among the Schistosoma are numerous, the three species of human significance have many similarities.. Schistosomiasis has recently been recognized as a major parasite cause of morbidity and occasional mortality, especially in sub-Saharan Africa. Along with HIV and malaria, disability from schistosomiasis caused by anemia, chronic pain, diarrhea, exercise intolerance, and undernutrition makes it a major problem in many parts of Africa and other areas of the world. Eggs. The average Schistosoma egg is comprised of a developed miracidium. The presence of lateral or terminal spines, as well as the organism's shape and size, aid in species identification. Schistosoma mansoni is relatively large, measuring 112 to 182 µm by 40 to 75 µm. The organism is somewhat oblong and possesses a prominent large lateral spine.
The somewhat roundish Schistosoma japonicum is the smallest of the Schistosoma spp., measuring 50 to 85 µm by 38 to 60 µm. The egg is characterized by the presence of a small lateral spine, which is often difficult to detect on microscopic examination. Schistosoma haematobium resembles S. mansoni in size and shape. The somewhat oblong egg measures 110 to 170 by 38 to 70 µm



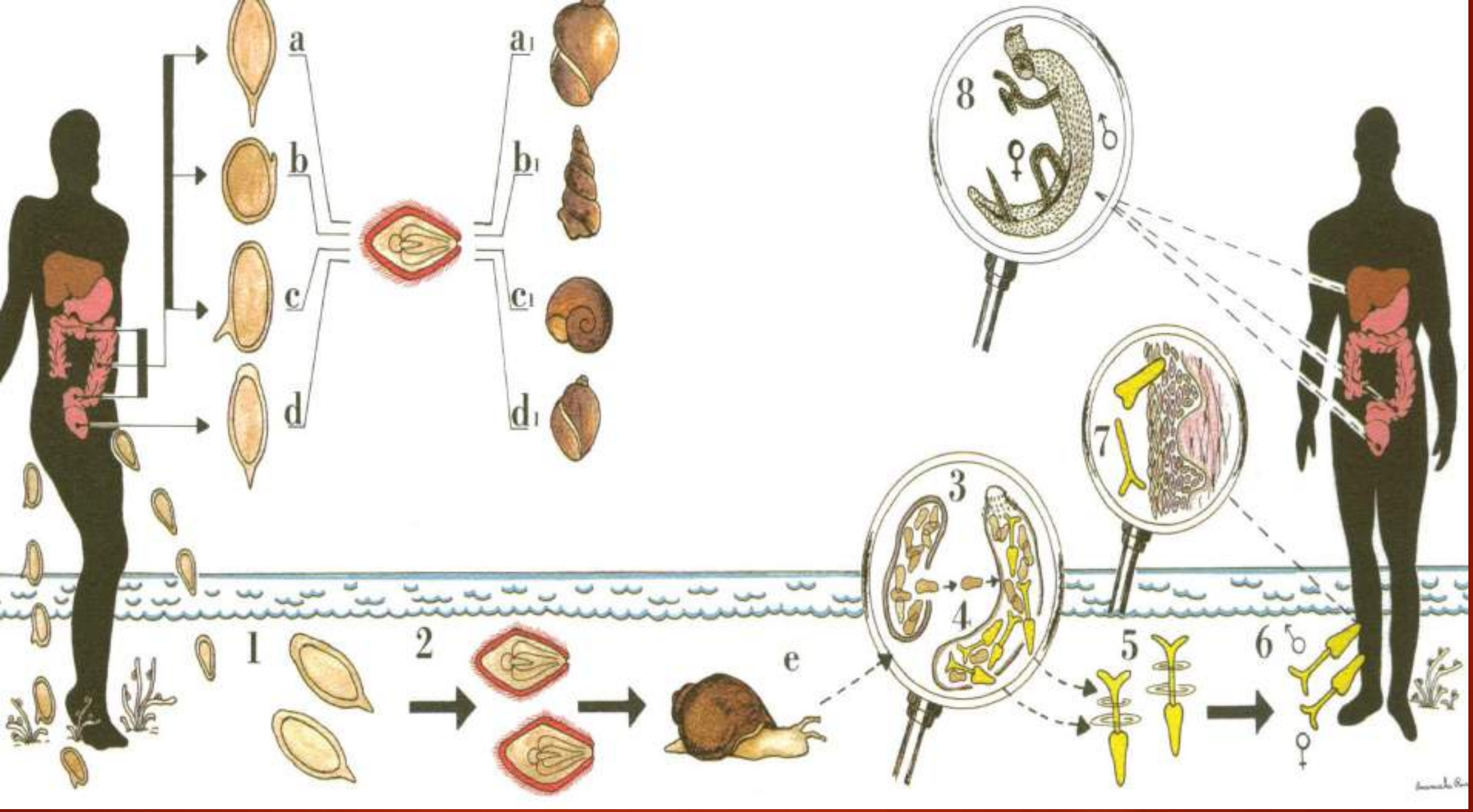


The presence of a large, prominent, terminal spine distinguishes the egg from that of other Schistosoma spp.

The eggs of the worm are eliminated via the urine of those infected. When an egg reaches freshwater, it hatches rapidly. The ensuing larva (an immature form of life called miracidium) is an oval shaped organism clothed with minute cilia swimming in search of the Bulinus snail. The miracidium] is attracted by the snail's mucous secretion and burrows into the soft tissue of the mollusc. Once inside, the miracidium transforms itself into an elongated, thin walled sack called mother sporocyst , which gives rise to about four hundred daughter sporocysts. Each one forms within its cavity numerous fork-tailed cercariae . A single miracidium may produce thousands of cercariae in only six weeks and this production may carry on in the snail for months the cercariae, stimulated by the bright light and the high temperature of the day, abandon the snails. Seen under the microscope they look like miniature tadpoles, with a pear shaped buoy and a long tail ending in a y-shaped fork which acts as a propeller to move the organism through the water . They are now swimming in a desparate race against time, searching for a human host to ensure their survival. They will die within 48 hours if the search fails. Attracted by the oily secretion lubricating the human body, they attach themselves to the skin with their oral suckers. They do not need to find a wound or break in the skin since they secrete an enzyme which splits the 'cement' holding the cells of the skin together. As soon as the cercariae start to penetrate the outer layers, they shed their tails and burrow a tunnel through the epidermis. Attracted by the oily secretion lubricatingThe tail-less cercariae, now called schistosomulae, then reach the lymph vessels which drain into the two major veins, allowing the schistosomulae of both sexes to reach first the lungs and then the liver, where they mature and mate. When they mate, the edges of the larger male worm fold over to form a groove to hold the slender and longer female close to him . Then, tightly embraced, they leave the liver, swimming against the flow of the venous blood, to settle in the blood vessels surrounding the bladder and the intestine . Since the only way to ensure the survival of the species is to get the eggs into freshwater, the worms live close to the natural openings of the human body – the bladder or the openings of anus so that the eggs can easily escape to the outside

The human body doesn't suspect their presence because the worms disguise themselves with a coating of protein similar to that of the host. This coating fools the human's defense system, which otherwise would fight off foreign particles. Undisturbed, the worms may live for years producing an incredible number of eggs.

The eggs, which are oval-shaped with a spike protruding from one end, are deposited in large numbers in the tissue of the bladder. Some reach the innermost part through the tiniest of blood vessels. They are now only separated from the urine by a thin layer of tissue, which they pierce with the spike to allow them to fall into the urine. When the eggs are eliminated from the bladder and reach fresh water, they will hatch into miracidia, search out the snails, and start the cycle anew.. However, a great number of eggs remain trapped in the tissue. Unlike their shrewd parents, they are not able to cheat the defense system of the host. Immediately, they are surrounded by scores of white blood cells, the body's main defense system. These 'immune fighters', known as phagocytes , they recognize as foreign by grinding the invaders to bits and swallowing them up with enzymes. However, the phagocytes are powerless in their fight against so many giants, each one 10 times their size. Not even with the help of larger cells, the macrophages , which fuse to form giant cells, are able to turn the battle in favour of the host. They are only able to lay siege by completely surrounding the eggs forming granulomas bladder and intestine tissue. Within about a month the eggs die and their shells calcify into rigid structures forming polyps, nodules, and ulcers. Slowly these are replaced by fibrous scar tissue,


a
b
c
d
a1
b1
c1
d1
8
7
3
4
5
6
1
2
e

Schistosomiasis is often divided into three phases: migratory, acute, and chronic. The migratory phase encompasses the time from penetration until maturity and egg production; it is often symptomless. Penetration of cercariae may produce a dermatitis if a patient's immune system has been sensitized by earlier experiences of cercarial penetration. Schistosome dermatitis is usually most severe when caused by bird schistosomes probably because cercariae are) killed The acute phase is sometimes called Katayama fever (for the Katayama region of Japan, a former endemic area and occurs when the schistosomes begin producing eggs about 4 to 10 weeks after initial infection. By this time a host has had exposure to various schistosome antigens, sufficient to mount a humoral response, but the advent of egg production
And increases the amount of antigen release. The change in antigen-antibody ratio leads to formation of large immune complexes that must be cleared by cells production . The syndrome is marked by chills and fever, fatigue, headache, malaise, muscle aches, lymphadenopathy, and gastrointestinal discomfort. There is a high eosinophilia, and granulomas around eggs contain large numbers of eosinophils, as well as neutrophils and macrophages. Macrophages in early acute granulomas secrete predominantly IL-1, and then TNF increases after one to two weeks. Chronic granulomas are dominated by macrophages, lymphocytes, fibroblasts, and multinucleated giant cells These become small fibrous granulomas, or pseudotubercles, so called because of their similarity to the localized nodules of tissue reaction (tubercles) in tuberculosis. Many eggs are carried by the hepatic portal circulation back up into the liver, where they stimulate granuloma formation

Chronic phase patients indigenous to endemic areas are commonly asymptomatic, or, with intestinal schistosomiasis, they may show mild, chronic, bloody diarrhea with mild abdominal pain and lethargy. With schistosomiasis haematobia, there may be pain on urination and blood in the urine. Affected people usually accept these conditions as normal and only seek medical assistance with heavy infections or when more serious complications develop. In most cases the patient's immune responses are modulated so that granulomatous reactions do not become too severe. For example, although macrophages secrete fibroblast growth factors, which mediate fibrosis, they also secrete collagenases that digest collagen fibers. A balance between collagen synthesis and degradation in most cases prevents progression to serious hepatic fibrosis. In about 8% of cases of infection with S. japonicum and S. mansoni, development of egg granulomas and fibrosis in the liver . As the eggs accumulate and the fibrotic reactions in the liver continue, a periportal cirrhosis and portal hypertension appeared . A marked enlargement of the spleen (splenomegaly) occurs, partly because of eggs lodged in it and partly because of the chronic passive congestion of the liver. accumulation of fluid in the abdominal cavity) is common at this stage . Some eggs pass the liver, lodging in the lungs, nervous system, or other organs and produce pseudotubercles there.

Several species of Schistosoma cause a severe rash when their cercariae penetrate skin of an unsuitable host; S. spindale and S. bovis are agents of dermatitis in humans throughout the range of these schistosomes. More importantly, several species of bird schistosomes are distributed throughout the world and cause "swimmer's itch" when their cercariae attack anyone on whose skin the organisms land

Major disease manifestations in chronic S. haematobium infections are urinary tract blockages, chronic urinary bacterial infections, bladder cancer, and bladder calcification

Clearly, economic and education level of a population will influence transmission of the disease, and age and sex are important factors as well. Males usually show the highest rates of infection and the most intense infections, and the most hazardous age is the second decade of life. This distribution of disease appears to reflect occupational and recreational differences, rather than sex or age resistance to infection. In places where both sexes work in the fields,the highest prevalence occurs in adults of both sexes. Certain other factors, such as immunity and egg release in chronic infections, must be considered when a population is surveyed and transmission is studied . Extending snail habitats, agricultural projects intended to increase food production in underdeveloped countries plyed an important role in transmission of the diseae

A simple, cheap, sensitive, and specific technique for routine diagnosis of schistosomiasis is demonstration of eggs in urine or feces therefore, direct smears and concentration techniques and other diagnostic methods, such as biopsy and immunodiagnosis were also used

Serological tests based on detection of antibodies in the patient's blood have several inherent problems: (1) they become positive some time after infection, (2) they become negative some time after cure, and (3) they may cross react with other helminth infections
diagnostic technique of the polymerase chain reaction to detect parasite DNA may used especially in negative cases in endemic area (why)

Adult and larval worms migrate through the host's blood circulation avoiding the host's immune system. The worms have many tools that help in this evasion, including the tegument, antioxidant proteins, and defenses against host membrane attack complex (MAC).

-Tegument

The tegument coats the worm and acts as a physical barrier to host antibodies and complement.

-Antioxidant proteins

Host immune defenses are capable of producing superoxide, which has a tremendous detrimental effect on the worm. However, they are able to produce a number of antioxidant proteins that block the effect of superoxide. Schistosomes have four superoxide dismutases, and levels of these proteins increase as the schistosome develops and matures.

Antioxidant pathways were first recognised as a chokepoints for schistosomes and later extended to other trematodes and cestodes. Targeting of this pathway with different inhibitors of the central antioxidant enzyme thioredoxin glutathione reductase (TGR) results in reduced viability of worms.

-Defense against host MAC

Schistosomes have ways to block host complement proteins. Immunocytochemistry techniques have found decay accelerating factor (DAF) protein on the tegument. DAF is found on host cells and protects host cells by blocking formation of MAC. It has been found that the schistosome genome consists of human CD59 homologs. CD59 inhibits MAC

Parasitology: a study of parasites and their relationships with their hosts.it is a one of the most wonderful area of biology. The study of parasitism is overlaps with other sciences branches (ecology, morphology, embryology, histology, biochemistry, immunology, pharmacology, molecular, cytology and nutrition, else). The term of parasite has Greek origin consists from two words , the first is (para) which mean besides and (sitos) which mean place, so it is mean(living beside food place).

Many organisms may have classified as parasites (bactecria, viruses and fungi, as well as parasite. But the term of parasitology has traditionally been limited to parasitic protozoa and helminthes as well as arthropods including arthropods that serve as vectors.

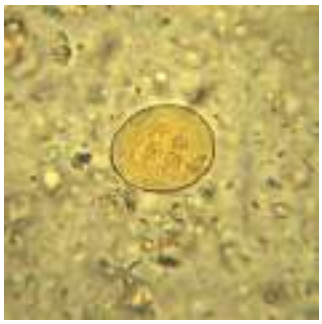
The importance of studying parasitology:

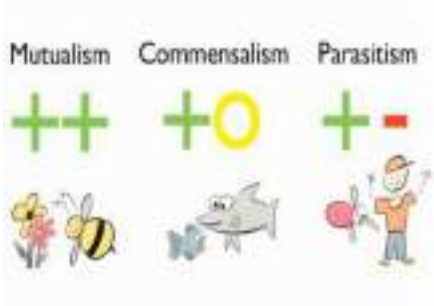
1- For their medical importance: parasites play importance role in the medicine and public health as six major tropical diseases to which WHO pay great attention to them including malaria, schistosomiasis, filariasis, leishmaniasis, trypanosomiasis, entamoebiasis and scabies for their importance.
2- For their economic importance: many of parasitic diseases are zoonotic and many of them are may infect the domestic animals and cause in economic loss.
3- For their scientific importance: it is very important to study the parasites to know their effects on health and to know how we can control of these diseases.
4- Some of them are used as biological control.

The relationships of animals:

There are diverse organism that are present in the community, and because of that different relationships are developed between them. These including two major types of relationship: interspecific relationship (is the relationship that show the interaction between organisms belong to the same kind of species like cooperation among bee and ants). And intraspecific relationship (is the relationship that show the interaction between the organisms belong to different species and these including:

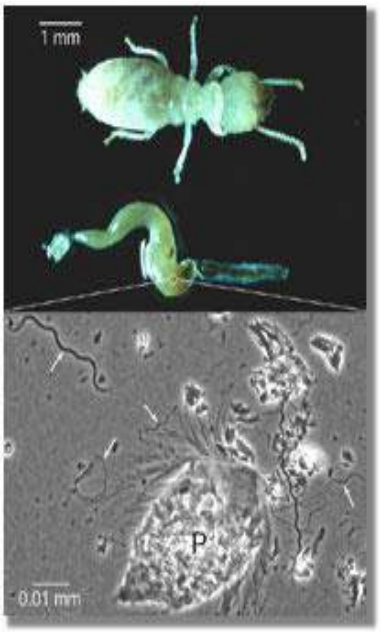
a- **Commensalism:** is a relationship between two species in which one of the organism benefits from this relationship while the other is neither benefits nor harmed. It does not involve physiologic interaction or dependency between the two partner of this relationship. Literally, it means ((eating on the same table)). An example of commensalism: hermit crabs and the sea anemones on its borrowed shells (ecto commensalism) and *Entamoeba coli* that live in human intestine (endo commensalism)

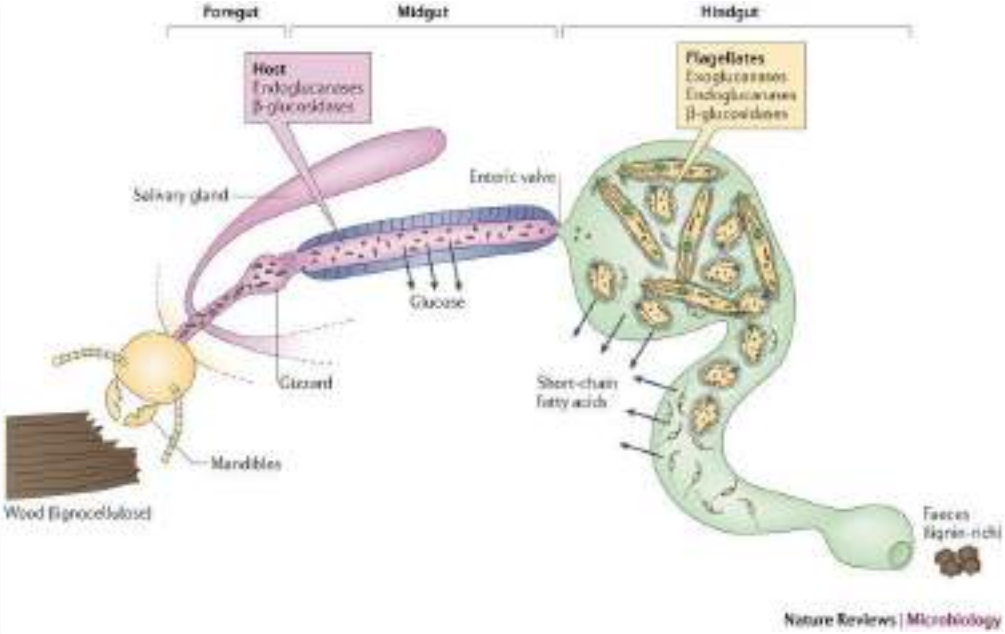




b- **Mutualism:** is a relationship between two organisms in which both of the organisms benefit from this relationship. It involves physiologic dependency between the two partner of this relationship. A classic example of this type of relationship is flagellated protozoan

(*Trichonympha*) that live in the gut of the termites. The flagellated protozoan depends entirely on a carbohydrate (as nutrition) from wood ingested by the termite. In return, the flagellated protozoan synthesizes and secretes cellulases (cellulose- digesting enzyme) , as the termite incapable to synthesizing its own cellulases.





c- **Parasitism:** is a relationship between two organisms in which one of the organisms benefit from this relationship while the other is harmed. The parasite may partially or entirely physiologic depended on its host.

The parasite gain shelter and nutrition from its host, the host suffer from wide range of damage. The parasites are usually smaller of the two (smaller than the hosts)

d-Predation: is a kind of relationship between to species in which one of the organism serve as food to other species. The first one called prey and the second called predator.

The difference between the parasitism and predation has been well expressed by Elton (1936) who has said that ((the difference between a carnivore and parasites is simply the difference between living on the capital and income)). As the parasites do not destroy the animals on which they parasitized so they keeping their food sources feed while the predator kill the prey.

The benefits gained by parasites from their host:

1-the host consider as a continues source of nutrition for parasites

2-the parasites use the host for transmitting and spreading

3-the host obtains a stable environment for parasites

4-the host obtain the protection for parasites

Damages done to host by parasites:

1-the parasites loot the food , blood or causing in blood losing for the host

2-mechanical injuries perforation of penetrate the tissue or organs or blockage cavities.

3-chemical injuries: resulting from secretion of parasites

4-histological changes:

a-hypertrophy:the increase in tissue or organs volume resulting from enlargement of cells as in *Plasmodium* infection

b-hyperplasia: increase in tissue or organs volume resulting from increase in the number of cells as in *Fasciola hepatica* infection

c-neoplasia: is an abnormal growth of tissue which may forms a mass develop to cancer as in *Schistosoma* infection

d-metaplasia: is a transformation or differentiated one tissue or cell to another type as in *Paragonimus westermani* (change the lung tissue to fibrous tissue)

5-the parasite transmit pathogenic agents (*Dientamoeba fragilis* on the egg of *Enterobius vermicularis, Plasmodium* in mosquitoes

6-growth activation as in some species of snails

7-Sex reversal as in crabs when infected with crustacean (the male become female)

8-abnormal behavior

9- death

Parasite: an animal or organism live on or in another organism belong to anther species (known as host) and it obtains nutrition and protection from this host. A parasite is completely or partly depended on its host and it may spend all or part of its life in or on the host

Type of parasites:

1-depending on the place:

a- Ecto-parasites: Parasite which lives on the body surface of its host. Example: Lice.

b- Endo-parasite: Parasite which lives inside the body of its host. Example: *Entamoeba histolytica*

2- depending on nature of life

a-Facultative parasite: Parasite which may exist as a commensal and if opportunity exists, may become pathogenic. Example: *Naegleria fowleri*

b-Obligate parasite: Parasite which always requires a host. Example *Plasmodium falciparum.*

3- depending on the period that parasite spend it in or on the host:

a-Periodic parasite: A parasite that lives on the host for short periods of time. And it visits its host for get a meal of food as mosquitoes

b-Stationary parasites: A parasite that lives in or on the host for a period of time. and it may include:

Temporary parasite: parasites are organisms whose parasitic mode of life is limited to a few or even one stage of development (An organism which is obligatory parasite at one or more stages of its life cycle but free living at other stages). Example: *Strongyloides stercoralis*.

Permanent parasite: A parasite that all stages of its life cycle permanently parasitize a host. Have no free living stages. Example: *Entamoeba histolytica*

4-Accidental parasite :A parasite that parasitizes an organism other than the usual host

Type of hosts:

Host: The organism in or on which the parasite lives.

1-Intermediate host: The organism in which the larval stages of the parasite live. or it is host where asexual reproduction take place. Example: Snails are intermediate hosts for *Fasciola hepatica.*

2-Definitive (final) host: The organism in which the adult stage of the parasite lives. or it is host where sexual reproduction take place.Example: Humans are the final hosts for *Taenia solium*.

3-Reservoir host: The host which harbours the parasite and act as additional source of human infection.(the reproduction of parasites is absent Example: Dogs are reservoir hosts for *Leishmania donovani* .

4-Accidental host: A host in which the parasite cannot successfully develop. Example: *Toxocara canis* in humans.

5-Vector: A living carrier (usually an insect) that transports a pathogenic parasite from one host to another. It includes two type

a-biological vector : when parasite multiplies and develops inside this host :A classical example is the mosquito which transmits malaria to humans.

b-mechanical vector: when host carry the parasite on its body without any development stages happen for the parasite: the fly carries the cyst of *Entamoeba histolytica*

Mode of parasitic infections:

1-by ingestion of the contaminated food or drinking contaminated water or uncooked meat

2-direct contact through the skin or sexually

3-pentration the skin due to contact with contaminated soil or water stream

4-Inhalation of the dust that carry the infective stages

5-by vectors

6-congenital: from mother to fetus

7-autoinfection: occure when infective stages are carried from host to the same host.

8-blood transfusion

Some Key definitions

Medical Parasitology: Is the study of parasites that infect humans.

Veterinary Parasitology: The study of parasites which infect animals .

Parasite: Is an organism which lives in or on another organism (host), from which it obtains food and shelter.

Pathogenic parasite: Parasite which causes harm to its host. Example: *Entamoeba histolytica*.

Non-pathogenic parasite (commensal): Parasite which benefits from the host without causing any harm. Example: *Entamoeba coli*.

Zoonosis: Simply means parasitic disease infects both animals and humans. An example of zoonosis in Iraq is the human infection with the larval stages (hydatid cysts) of the dog tapeworm, Echinococcus granulosus, causing a condition known as hydatid disease .

Incubation Period: The period between infection and the appearance of symptoms.

Intensity of infection: is the mean number of parasitic individuals found in infected hosts

Percentage of infection (occurrence, prevalence of incidence): is the proportion of infected host among all the host examined

Percentage of infection= $\frac{number\ of\ infected\ host}{total\ number\ of\ examined\ host}$x100

Infective stages:it is a period of the life cycle that causes infection when it becomes contact with the host in somehow.

symptoms

The parasites cause a wide range of symptoms (signs of the parasitic infection) and the intensity of these symptoms and their appearance depend on:

1-Number of parasites in the host (intensity of infection).

2-Location of the parasite in the host.

3-Size of the parasite.

4-Period of the infection.

5-Response of the host to the metabolic products of the parasites (toxins and secretions) (hypersensitivity).

Laboratory diagnosis:

The samples that used for diagnosis the parasitic infection are;

a-blood: blood smear is used for diagnosis of *Plasmodium, Trypanosoma* and microfilaria of *Wuchereria*

b -stool: the cyst and trophozoite of some protozoa found in stool and the ova of some worms

c-urine: for diagnosis of the ova of *Schistosoma heamatobium*

d-sputum: for diagnosis of the ova of *Paragonimus westermani*

e-biopsy from skin and liver for diagnosis the *Leishmania tropica* and *Leishmania donovani,* respectively

General features of protozoa

1-They are known as acellular or non-cellular organism. A protozoan body consists of only mass of protoplasm, so they are called acellular or non-cellular animals.

2-Habitat: mostly aquatic, either free living or parasitic or commensal

The majority are free living and less than 20% are parasitic.

3-Grade of organization: protoplasmic grade of organization. Single cell performs all the vital activities thus the single cell acts like a whole body.

4-Body of protozoa is either naked or covered by a pellicle.

5-Locomotion: Locomotory organ are pseudopodia (false foot) or cilia or absent.

6-Nutrition: Nutrition are holophytic (like plant) or holozoic (like animal) or saprophytic or parasitic.

7-Digestion: digestion is intracellular, occurs in food vacuoles.

8-Respiration: through the body surface.

9-Osmoregulation: Contractile vacuoles helps in osmoregulation.

10-Reproduction:

a-Asexually reproduction is through binary fission or budding.

b-Sexual reproduction is by syngamy conjugation.

11 -Excretion of protozoa by one of:

a-diffusion

b-contractile vacuoles

c-food vacuoles

d-cytopyge: it is an opening (cell "anus") found in some protozoa, such as the rumen-dwelling ciliates, through which waste matter is ejected

According to degree of pathogenicity, protozoa can be divided into:

(I) Pathogenic.

(2) Non-pathogenic (commensals).

(3) Opportunistic (whose pathogenicity are debatable).

According to types of locomotive (movement) organs, protozoa can be divided into 4 groups:

1 - Amebae – pseudopodia.

2 - Flagellates – flagella.

3 - Ciliates – cilia .

4 - Sporozoans – absence of locomotive organs.

Pathogenic Intestinal Amebae

Genus: *Entamoeba histolytica* (the parasite causing diarrhea and liver abscess in man)

Lambl (18590nfirst discovered the parasite. Lösch (1875) proved its pathogenic nature. While Schaudinn (1903) differentiated pathogenic and non-pathogenic type of amoeba .

Geographical distribution: The parasite is world-wide. More common in tropical and sub-tropical than in temperate zone.

Habitat: Trophozoites and cyst of E. histolytica (the large race or the tissue invading forms) and cysts live in the mucous and sub mucous layers of the large intestine .

Morphology: the structural character of the parasite can be studied both in stained (iodine and iron haematoxylin) and unstained preparation there are two phase in the life cycle of the parasite:

(i)Fairly abundant in amoebic dysentery stools.

(ii) The size varies from 10-30µ

(iii) The outline shows finger like pseudopodia.

(iv) Actively motile in nature.

The cytoplasm is

(i)Ectoplasm—prominent;

(ii)Endoplasm—finely granular

(iii)Vacuoles—scanty, spherical and defined with ingested RBCs

Nuclear membrane uniformly stained with chromatin; karyosome is central. In saline preparation the nucleus is indistinct and small

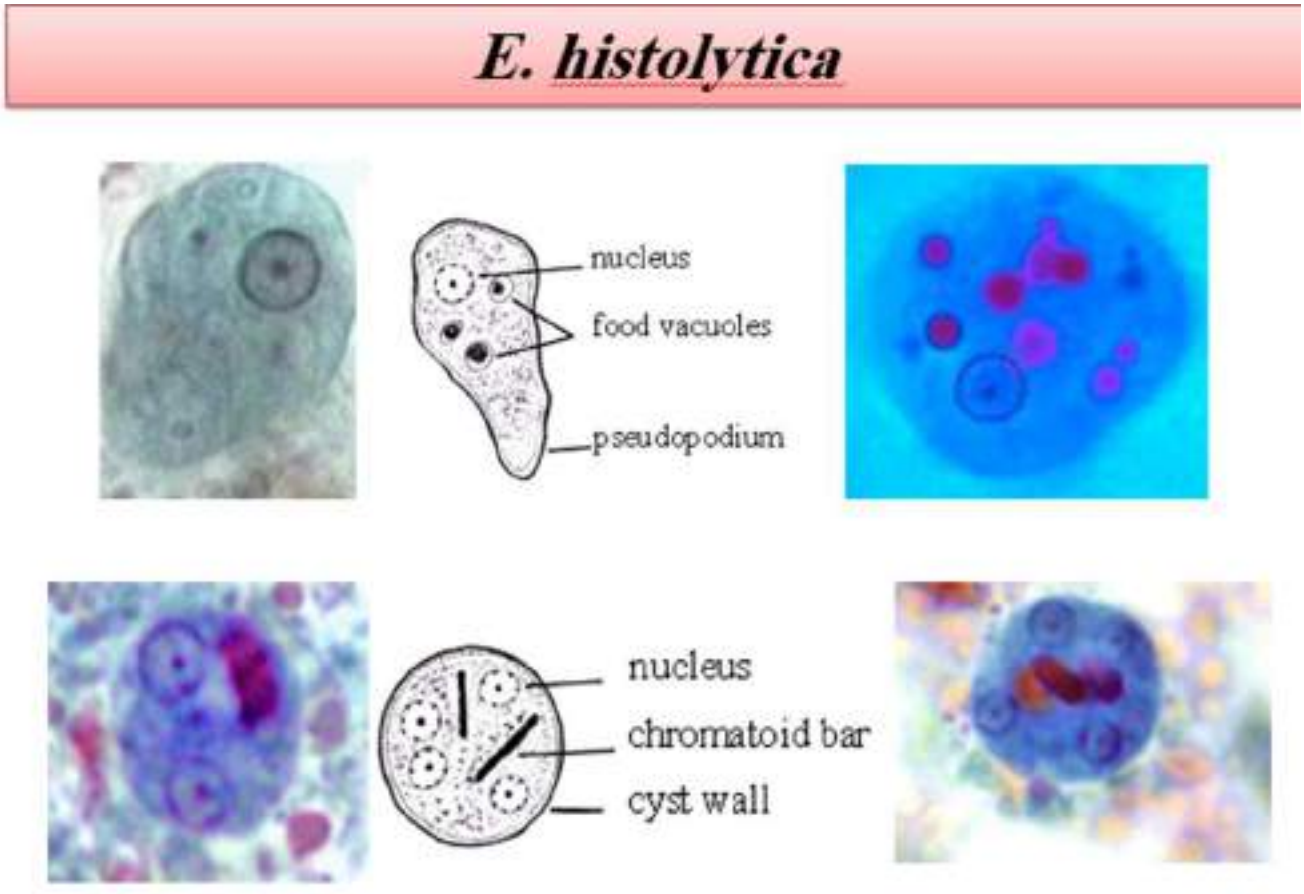
The cyst is

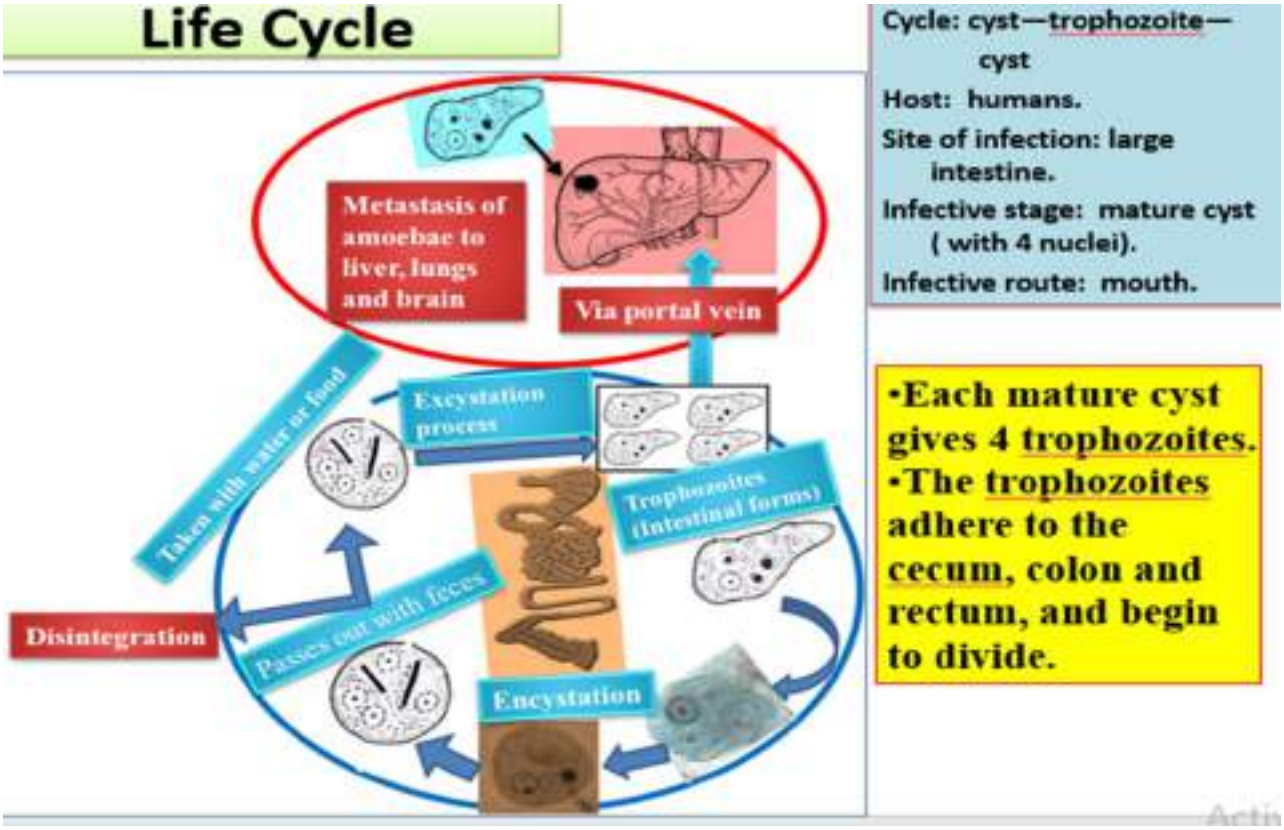
(i) Size: 6-18µ

(ii) Chromatid body present with rounded ends and they are bar like.

(iii) Usually one or four nuclei are seen.

(iv) Nucleus not visible when unstained.





Pathology:

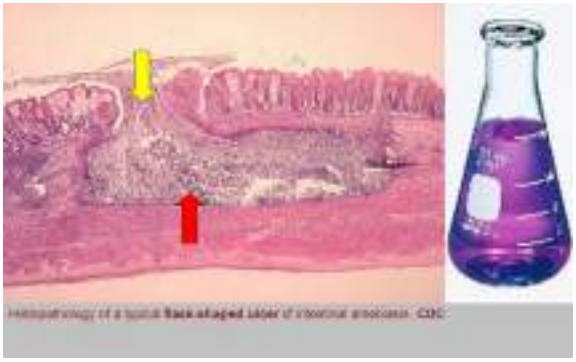
.1 Intestinal amebiasis (Primary lesion): Invasion of the wall of large intestine, – ulcer is flask shaped. Complications: Amoebic granuloma (ameboma), appendicitis, intestinal perforation.

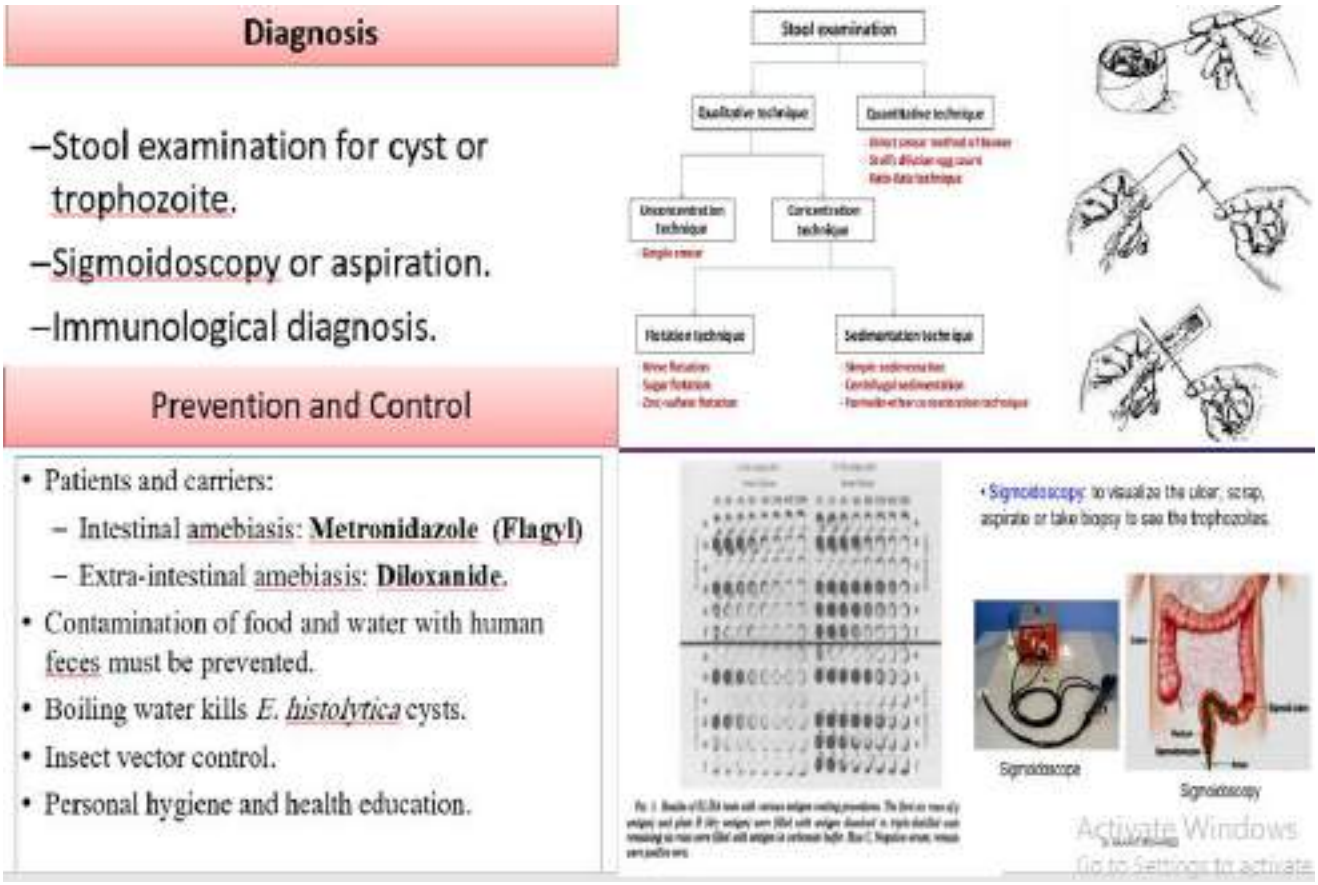
.2 Extra-intestinal amebiasis (Secondary lesions) occurs as a result of METASTASIS of trophozoites to extra-intestinal organs and cause hepatic amebiasis (abscess); pulmonary amebiasis; cerebral amebiasis; cutaneous amebiasis; spleenic abscess .

Symptoms :

Acute amebiasis: Diarrhea, dysentery (stool containing blood ,
mucous and shreds of necrotic mucosa), acute
abdominal pain, and fever .

Chronic amebiasis: Recurrent attacks of dysentery, hepatomegally and weight loss





Giardia lamblia

- **Disease:** Giardiasis or lambliasis.
- **Infective stage:** Mature cyst with 4 nuclei.
- **Site of infection:** Small intestine.
- **Life cycle**: Includes trophozoites and cysts. Trophozoites live in small intestine and attach themselves to intestinal mucosa but not invasive.
- **Treatment:** Flagyl

The trophozoites of G. lamblia are flattened pear shaped.

When stained, the trophozoite

1.has 2 nuclei, 2 slender median rods

2 .(axostyles), and 8 flagella arising

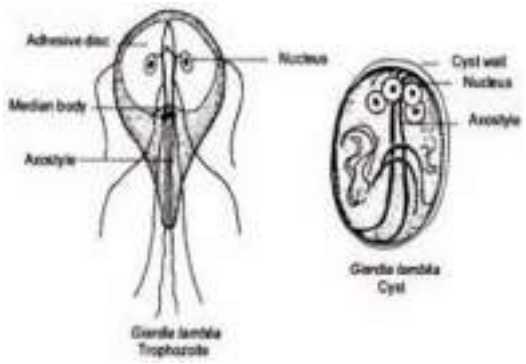
from the anterior end.

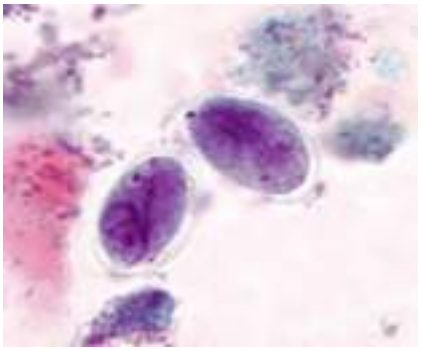
3 .The trophozoites have been described as

looking like tennis rackets without the

handle.

4. They are often have a comical face-like appearance when looking at the front view.





1.The cysts of G. lamblia are ellipsoid in shape .

2.They contain 4 nuclei which tend not to be obvious .

3.Longitudinal fibrils consisting of the remains of axonemes and parabasal bodies may also be seen .

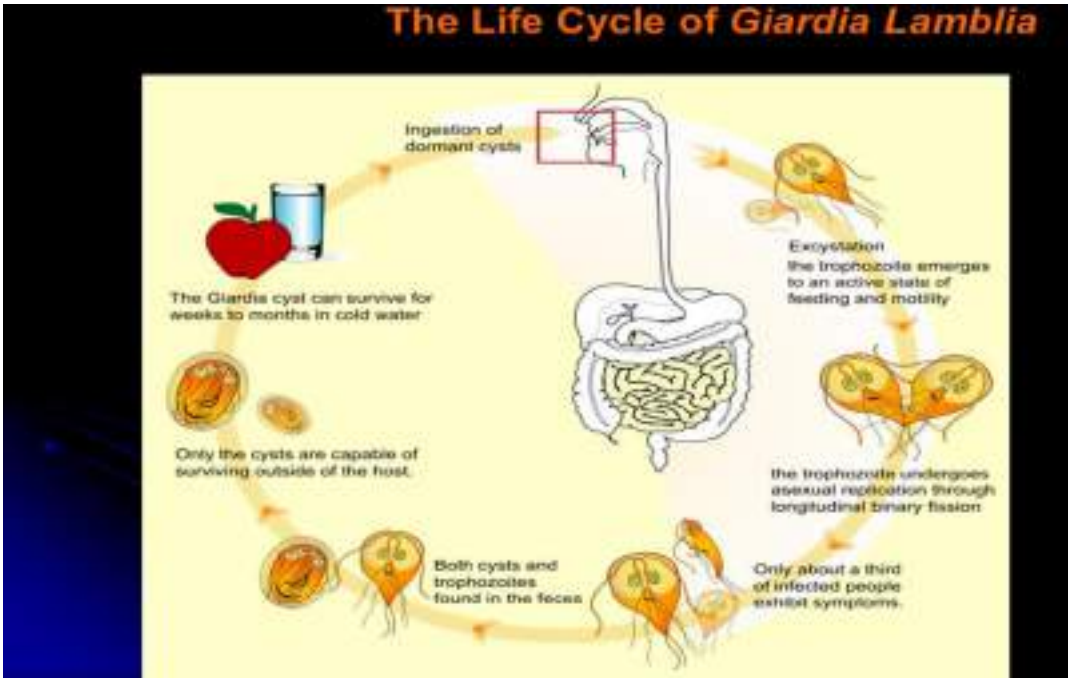
4.Cysts may appear to shrink from the cell wall .

5.The cysts are infective as soon as they are passed.

Host: Humans ;

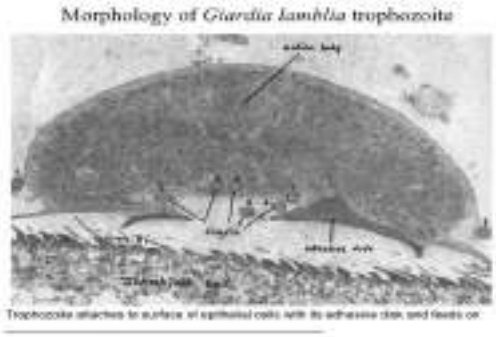
Residing site: small intestine

Infective stage: Cyst; Infective route: mouth



Pathology: Atrophy and shortening of the villi. Possibly, the mucosal abnormalities are due to mechanical, toxicity effect, and impaired absorption of vitamin B12. Uptake of bile salts by Giardia inhibits the digestion of fats by pancreatic lipase and this leads to Malabsorption syndrome (greasy stool called STEATORRHEA) .

OR MAY BE The parasite attach itself on surface of epithelial cell of intestine and in heavy infection may cause disturbance of intestinal function especially the absorbance of fat and vitamin B12



Clinical symptoms:

Diarrhea

Vomiting

Flatulence

Malabsorption syndrome

Weight loss.

Diagnosis:

1-Examination of stool for trophozoite or cyst.

2-Duodenal aspiration.

Prevention and control

1-Patients and carriers: should treat with Metronidazole (Flagyl)

2-Insect vector control (flies, cockroaches)

3-Personal hygiene and health education

Success is not the key to happiness. Happiness is the key to success. If you love what you are doing, you will be successful.

Learn English 100

"النجاح ليس مفتاح السعادة
وإنما السعادة هي مفتاح
النجاح. فإن أحببت عملك
فسوف تكون ناجحاً"

ALBERT SCHWEITZER

Apicomplexa:

this phylum including a large group of protozoa all of them are parasites without any exception. They do not possess any special organs of locomotion such as cilia or flagella (they may have flagella in some stages of their life cycle or may show amoeboid stage in some stages). They reproduce asexually by schizogony followed by sexual syngamy; this is known as alternation of generation. Most of them need two hosts in their life cycle.

Classification:

Phylum: Apicomplexa
Class : Sporozoa
Subclass: Coccidia
Order:Eucocciida
Suborder:

Eimeriina | Haemosporina

Genus:*Toxoplasma* Genus:*Plasmodium*

Order Euccociida has suborders of medical importance to human which are Eimeriina Haemosporina.

Suborder: Haemosporina.
Genus: Plasmodium
Plasmodium was first identified by Laveran when he described parasites in the blood of malaria patients in 1880. The fact that several species may be involved in causing different forms of malaria was first recognized by Camillo Golgi in 1886. The contribution of insect hosts to the Plasmodium life cycle was described by Ronald Ross in 1898. This parasite cause disease called malaria. Malaria comes from “mal” and “aria,” which means “bad air.” Before the parasite that caused malaria was discovered, people thought the disease was caused by foul air, and associated it with marshes and low-lying swamps. They were not 100% wrong—those areas are perfect breeding grounds for the mosquitoes which transmit malaria, and so infection often occurs in and around these areas. There are four species responsible for human malaria and these are P. falciparum, P. vivax, P. ovale and P. malariae.

The WHO estimates that in 2015 there were 214 million new cases of malaria resulting in 438,000 deaths. Others have estimated the number of cases at between 350 and 550 million for falciparum malaria. The majority of cases (65%) occur in children under 15 years old. About 125 million pregnant women are at risk of infection each year; in Sub-Saharan Africa, maternal malaria is associated with up to 200,000 estimated infant deaths yearly. P. falciparum (malignant tertian malaria) and P. malariae (quartan malaria) are the most common species of malarial parasite and are found in Asia and Africa. P. vivax (benign tertian malaria) predominates in Latin America, India and Pakistan, whereas, P. ovale (ovale tertian malaria) is almost exclusively found in Africa. Plasmodium falciparum is the most dangerous malarial parasite and the infected red blood cells develop surface knobs which cause them to stick to endothelial cells, this causes blockages and brain damage, often resulting in death, which can occur within a few days of infection.

Morphology: there are different stage of malarial parasite (sporozoite which inter to the human by insect bite they do not appear in blood because they immediately enter to liver cell) hepatic schizont (or sometime called cryptozoite when they remain inside hepatic cell), merozoite, trophozoite (ring stage and amoeboid stage which represent immature trophozoit and mature trophozoite which contain the schizonts),gametocyte all these stages are found in human and here are the definition of some morphological forms

Hepatic schizont: the actively dividing, multinucleated, parasite form in hepatocytes; produces no inflammatory response.

Trophozoite: a metabolically active form of the malaria parasite living within the RBC; sometimes called the ring form or amoeboid stage.

Erythrocytic schizont: multinucleated stage in a red blood cell (RBC) resulting from asexual multiplication of trophozoite. Each schizont contains a species determined number of meroziotes.

Merozoite: the name given to infective schizont components that break out of RBC or hepatocyte and then adhere to and penetrate a new RBC.

Gametocyte: morphologically distinctive sexual (male or female) form of the parasite which develops from some trophozoites in RBCs. It is infective to mosquito.

Sporozoite: the morphological form which develops in the mosquito salivary gland and is injected when the mosquito feeds and consequently infecting humans.

In mosquite there are the flollowing stages:

Microgamete: the first phase of development inside the gut of insect it is thread like filamentous it is form by process called ex-flagellation

Macrogamete: it is one gamete which form by process of nuclear reduction

Ookinte: it is the zygote lengthen and mature zygote form by fertilization of macro and microgametes

Oocyst:itb is a spherical mass surrounded by a structureless capsule

Early Stage Trophozoites

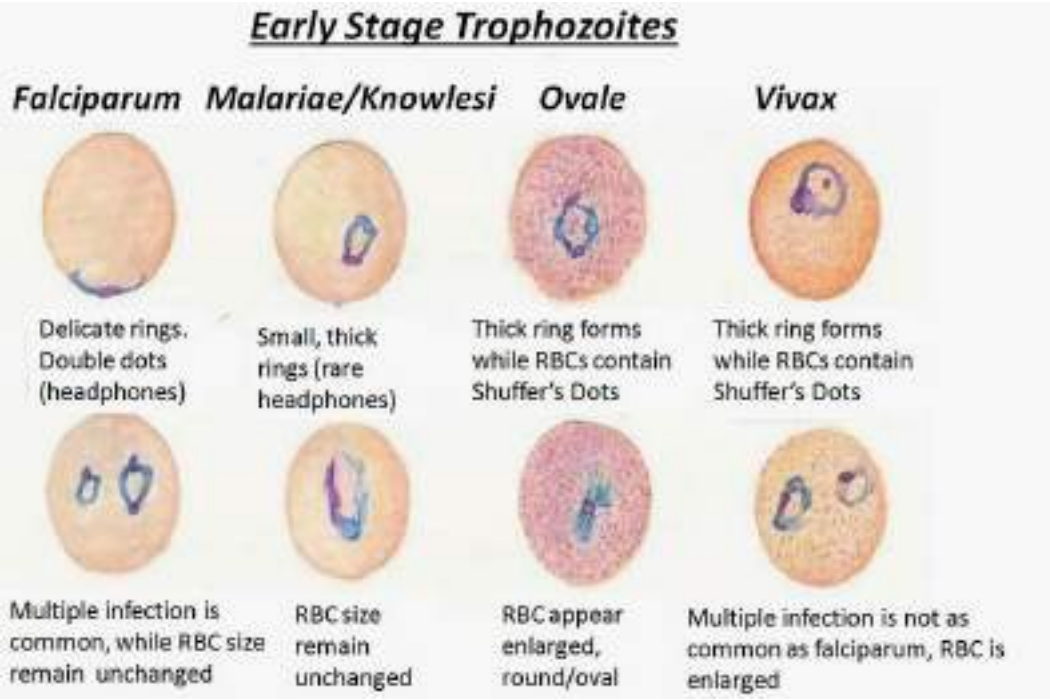
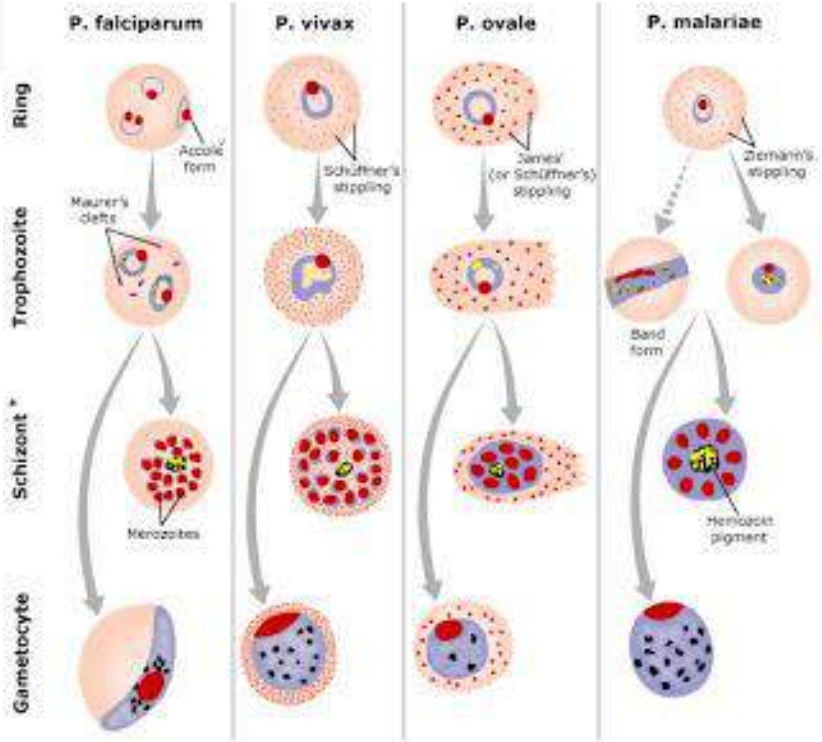
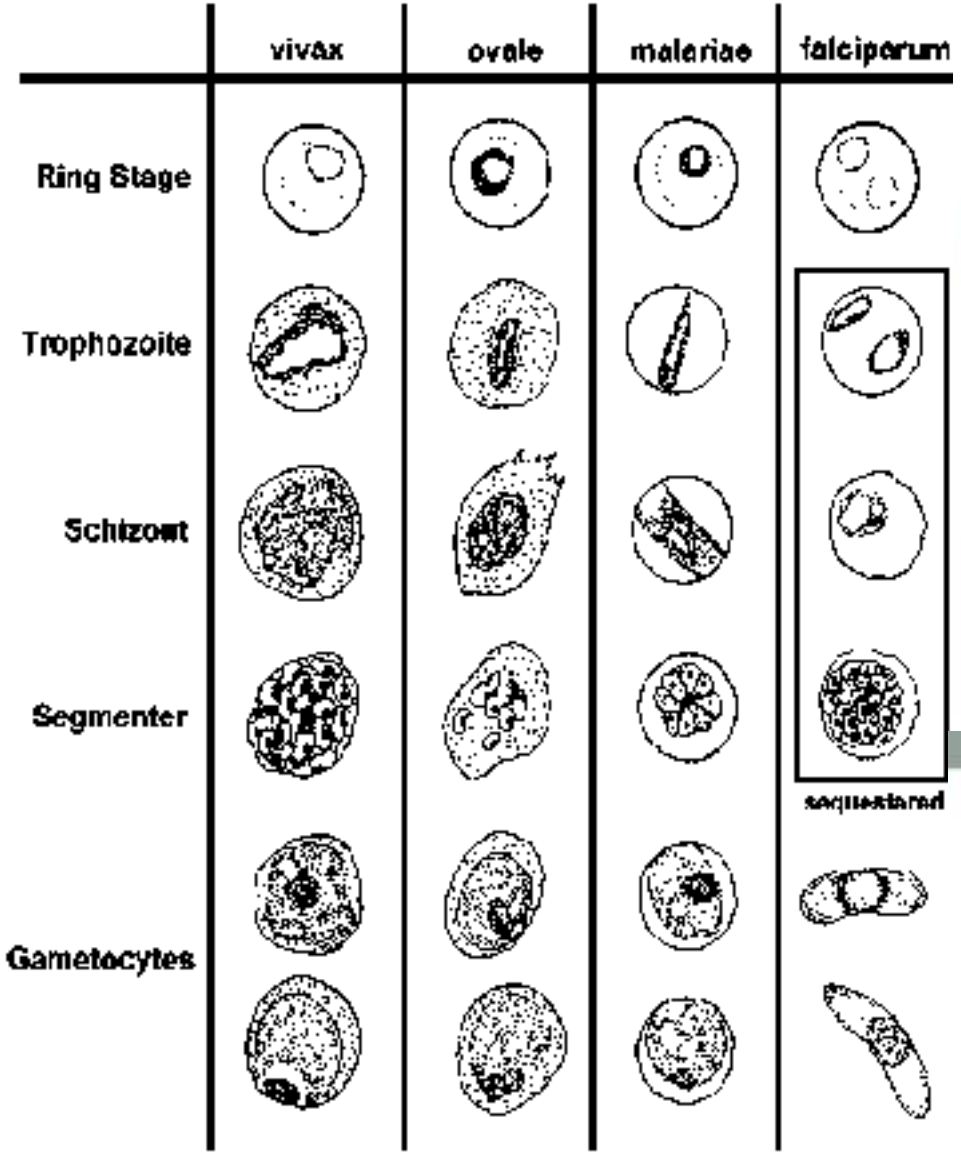


Table 2. Characteristics of infection with the five species of Plasmodia infecting humans

Characteristics	*Plasmodium falciparum*	*P. knowlesi*	*P. malariae*	*P. ovale*	*P. vivax*
Pre-erythrocytic stage (days)	5-7	8-9	14-16	9	6-8
Pre-patent period (days)	9-10	9-12	15-16	10-14	11-13
Erythrocytic cycle (days)	48	24	72	50	48
Red cells affected	All	All	Mature erythrocytes	Reticulocytes	Reticulocytes
Parasitaemia per µL • Average • Maximum	20,000-500,000 2,000,000	600-10,000 236,000	6000 20,000	9000 30,000	20,000 100,000
Febrile paroxysm (hours)	16-36 or longer	8-12	8-10	8-12	8-12
Severe malaria	Yes	Yes	No	No	Yes
Relapses from liver forms	No	No	No	Yes	Yes
Recurrences	Yes (treatment failure)	Yes	Yes (as long as 30-50 years after primary attack)	No	Yes (treatment failure)





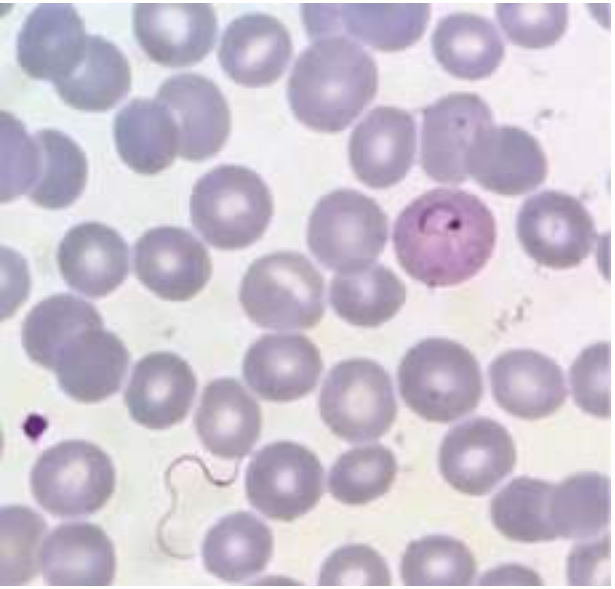
Key Morphological Differences Between Human *Plasmodium* Species in Blood Smears

P. vivax	*P. ovale*	*P. malariae*	*P. falcipar.*
1- enlarged erythrocyte. 2- Schüffner's dots. 3- ameboid trophozoite.	1-elongated oval erythrocyte. 2- Schüffner's dots. 3- compact trophozoite. 4- usually fewer merozoites in schizont.	1- compact trophozoite. 2- merozoites in rosette. 3- Band shape schizont.	1- numerous rings. 2- smaller rings. 3- no trophozoites or schizonts. 4- crescent-shaped gametocytes.

Species Differentiation on Thin Films

Feature	*P. falciparum*	*P. vivax*	*P. ovale*	*P. malariae*
Enlarged infected RBC		+	+	
Infected RBC shape	round	round, distorted	oval, fimbriated	round
Stippling infected RBC	Maurer clefts	Schuffner spots	Schuffner spots	none
Trophozoite shape	small ring, applique	large ring, amoeboid	large ring, compact	small ring, compact
Chromatin dot	often double	single	large	single
Mature schizont	rare, 12-30 merozoites	12-24 merozoites	4-12 merozoites	6-12 merozoites
Gametocyte	crescent shape	large, round	large, round	compact, round

CDC



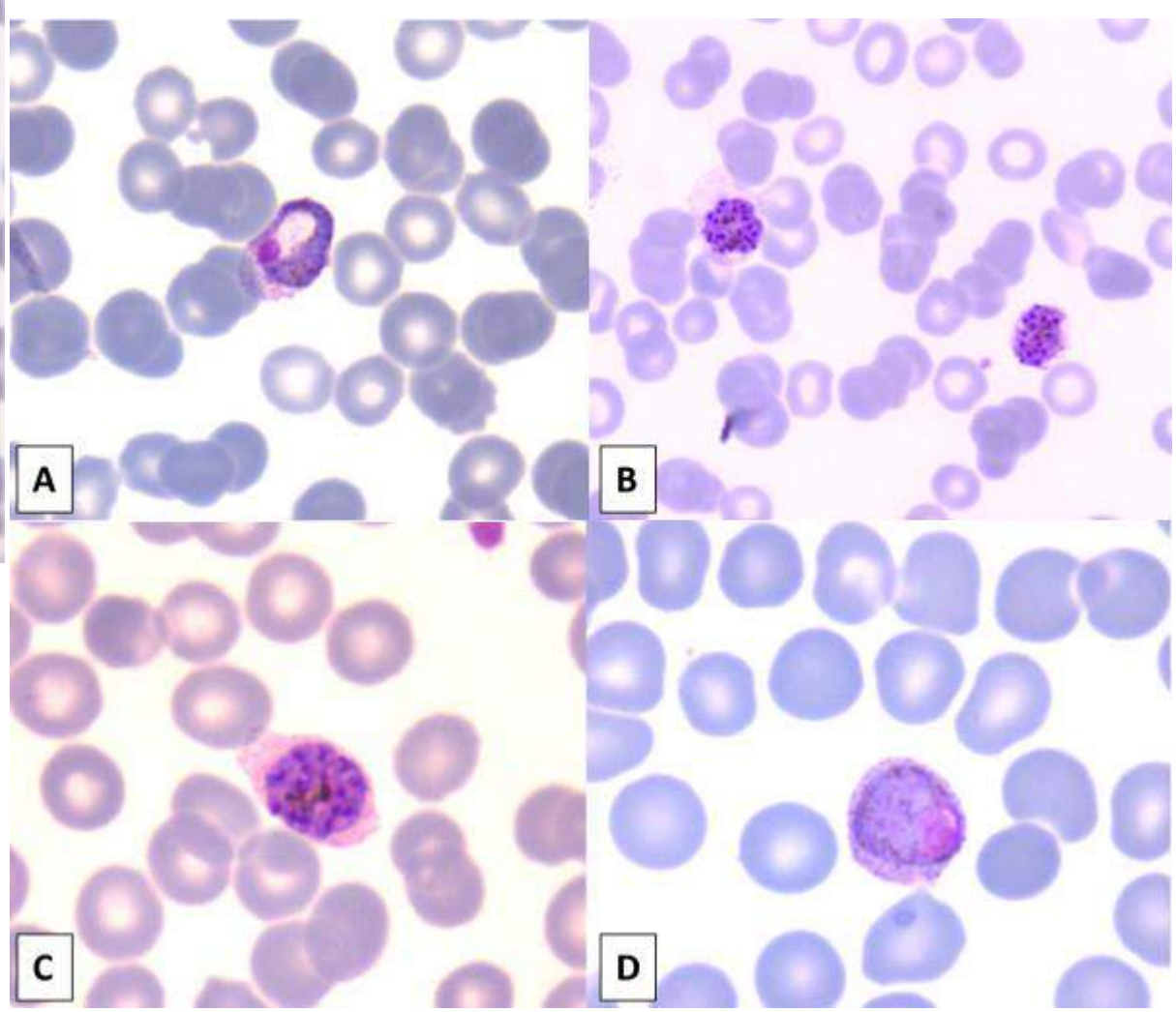


Figure 1: Plasmodium vivax in Giemsa-stained thin blood smear with all developmental stages present in peripheral blood. (A) Growing amoeboid trophozoite in enlarged red blood cell (RBC) with eosinophilic stippling (schuffner's dots). (B) Immature schizonts with clumps of brown pigment almost fill the enlarged RBCs. (C) Mature schizont with merozoites (about 14) and clumped pigment. (D) Macrogametocyte with diffuse brown pigment and eccentric...

Life cycle:

The There are mainly two phases of development in **the human asexual life cycle;**

1-Inside the liver (tissue phase)

I)pre- erythrocytic (or primary or Exo-erythrocytic) Schizogony- no clinical symptom and no pathological damage.

ii)Hypnozoite stage: cause of malarial of relapse (it is the phase persists in the liver cell as dormant form known as hypnozoite which is capable of developing into merozoite and it is responsible for relapses of vivax and ovale

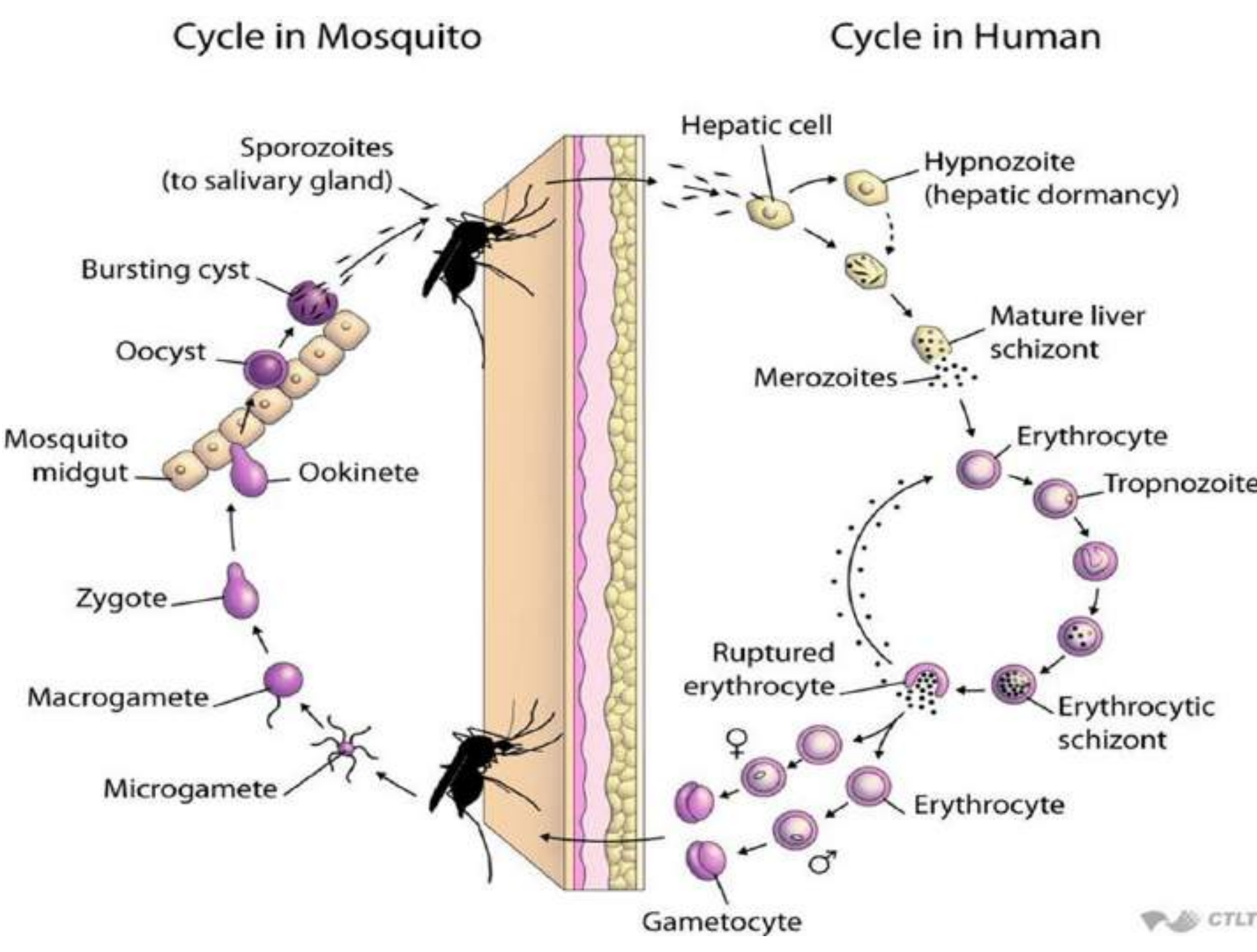
2-Inside the red blood cell (Erythrocytic phase)

i) Erythrocytic schizogony; cause of malarial paroxysmذروة

ii)Gametogony- infects mosquito

while in the mosquite the sexual cycle is occurred

The life cycle of Plasmodium involves several distinct stages in the insect and vertebrate hosts. In infected mosquitoes, parasites in the salivary gland are called sporozoites. When the mosquito bites a vertebrate host, sporozoites are injected into the host with the saliva. From there, the sporozoites enter the bloodstream and are transported to the liver(asexual cycles:1-in liver:exerythrocytic cycle will begin), where they invade and replicate within hepatocytes. At this point, some species of Plasmodium can form a long-lived dormant stage called a hypnozoite which can remain in the liver for many yearsThe parasites that emerge from infected hepatocytes are called merozoites, and these return to the blood to infect red blood cells.


Cycle in Mosquito
Cycle in Human
Sporozoites
(to salivary gland)
Bursting cyst
Oocyst
Mosquito
midgut
Ookinete
Zygote
Macrogamete
Microgamete
Hepatic cell
Hypnozoite
(hepatic dormancy)
Mature liver
schizont
Merozoites
Erythrocyte
Tropnozoite
Ruptured
erythrocyte
Erythrocytic
schizont
Erythrocyte
Gametocyte

Within the red blood cells(In blood: erythrocytic cycle will begin), the merozoites grow first to a ring-shaped form, amebiod stage and then to a larger form called a trophozoite. Trophozoites then mature to schizonts which divide several times to produce new merozoites. The infected red blood cell eventually bursts, allowing the new merozoites to travel within the bloodstream to infect new red blood cells. Most merozoites continue this replicative cycle, however some merozoites upon infecting red blood cells differentiate into male or female sexual forms called gametocytes. These gametocytes circulate in the blood until they are taken up when a mosquito feeds on the infected vertebrate host, taking up blood which includes the gametocytes.

In the mosquito(In insect: sexual cycle), the gametocytes move along with the blood meal to the mosquito's midgut. Here the gametocytes develop into male and female gametes(microgamete and macrogamete) which fertilize each other, forming a zygote. Zygotes then develop into a motile form called an ookinete, which penetrates the wall of the midgut. Upon traversing the midgut wall, the ookinete embeds into the gut's exterior membrane and develops into an oocyst. Oocysts divide many times to produce large numbers of small elongated sporozoites. These sporozoites migrate to the salivary glands of the mosquito where they can be injected into the blood of the next host the mosquito bites, repeating the cycle

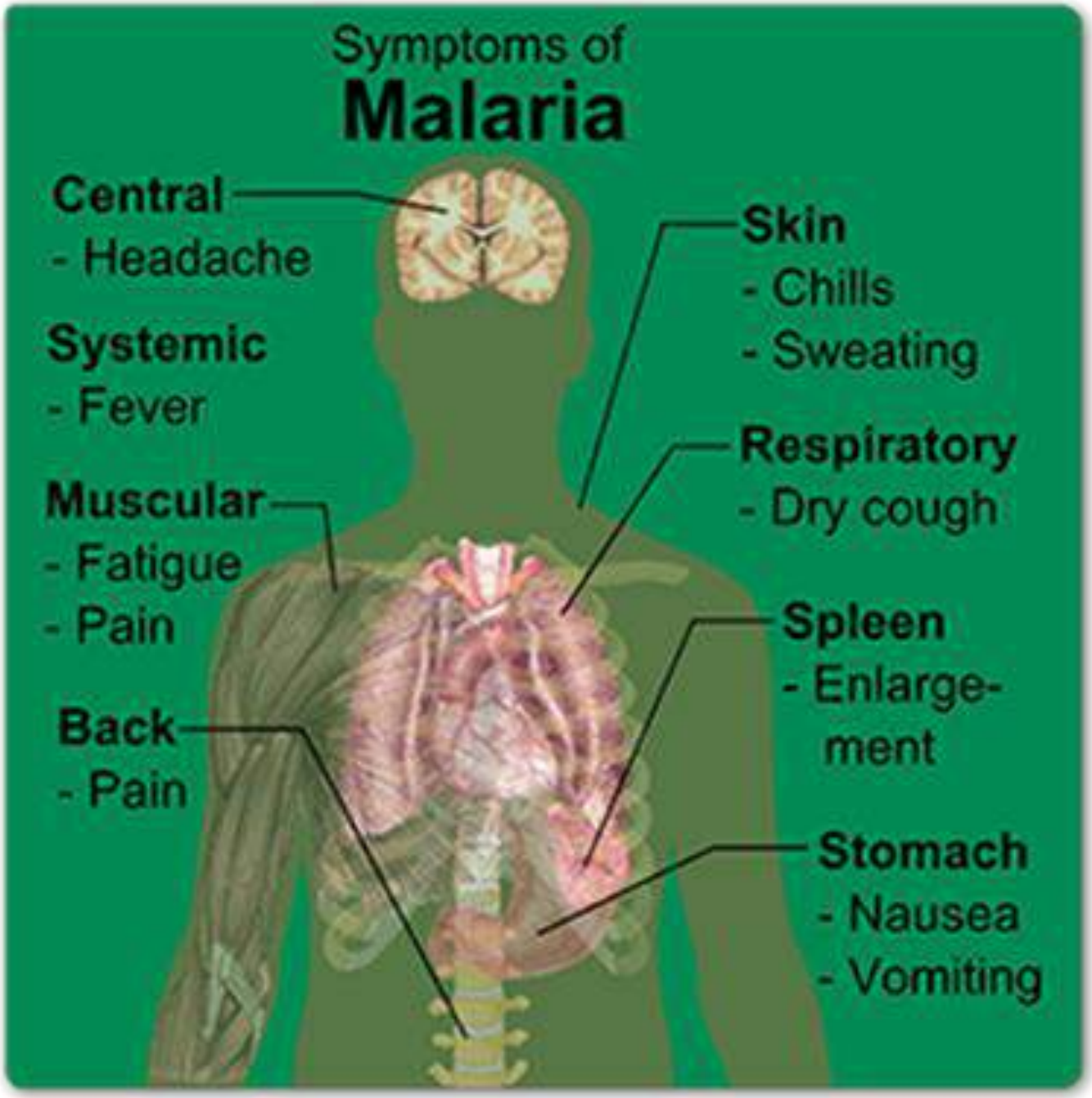
symptoms

The symptoms of malarial infection depend on the species of *Plasmodium* that causes the infection and also on the location and numbers of the parasites. *Plasmodium falciparum* normally takes 7-14 days to show symptoms while *P. Vivax* and P. ovale normally take 8-14 days and *P. Malariae* 7-30 days. In general, malaria causes the following symptoms: headache, vague pains in the bones and joints, chilly sensations and fever. As the disease progresses, the chills and fever become more prominent. The chill and fever follow a cyclic pattern (paroxysm) with the symptomatic period lasting 8-12 hours depending on the species of *Plasmodium*. It is the synchronous (متزامنة) rupture of red blood cells which gives the periodic fever. In between the symptomatic periods, there is asymptomatic period, the duration of which depends upon the species of the infecting parasite. This interval is about 34-36 hours in the case of *P. vivax* and *P.ovale* (tertian malaria), and 58-60 hours in the case of *P. malariae* (quartan malaria). In contrast،Classical tertian paroxysm is rarely seen in *P. falciparum*.Without treatment, all species of human malaria may ultimately result in spontaneous cure except with *P. falciparum* which becomes more severe progressively and results in death. This organism causes blockage of capillary vasculature in the brain, gastrointestinal and renal tissues. Chronic malaria results in splenomegaly, hepatomegaly and nephritic syndromes.

Plasmodium falciparum malaria is an acute **life-threatening disease and rapid diagnosis is required. Diagnosis is based on: 1. -Clinical picture: fever, chills, travel history and fever pattern. In Plasmodium falciparum daily fever and rarley every 2 day fever while in other species, most of the time intermittent fever every 2 or 3 days. 2. -Examination of blood: [thin, thick smear, buffy coat and antigen capture (ELISA and PCR). The examination of Giemsa stained blood smears for characteristics of species depends mainly on:**

- **The shape and size of trophozoite, schizont and gametocyte.**
- **Percentage of red blood cells with parasites (very rarley more than 1% parasitemia) in P. vivax, P. ovale or P. malariae.**
- **The presence of metabolic debris in red blood cells around parasites (called Schuffner's dots) in P. vivax infection.**
- **Size of red blood cells which contain parasites (P. ovale and P. vivax infect younger red blood cells.**

Control:Control measures include removing the breeding sites (larviciding, larva eating fish, and larva-killing bacteria), chemoprophylaxis and avoidance (using nets, screens, repellents, and insecticide treated bed nets). Chemoprophylaxis is a medication taken at regular intervals to kill one or more of the morphological forms (stages) of the malaria parasite and therefore prevent the onset of clinical illness.

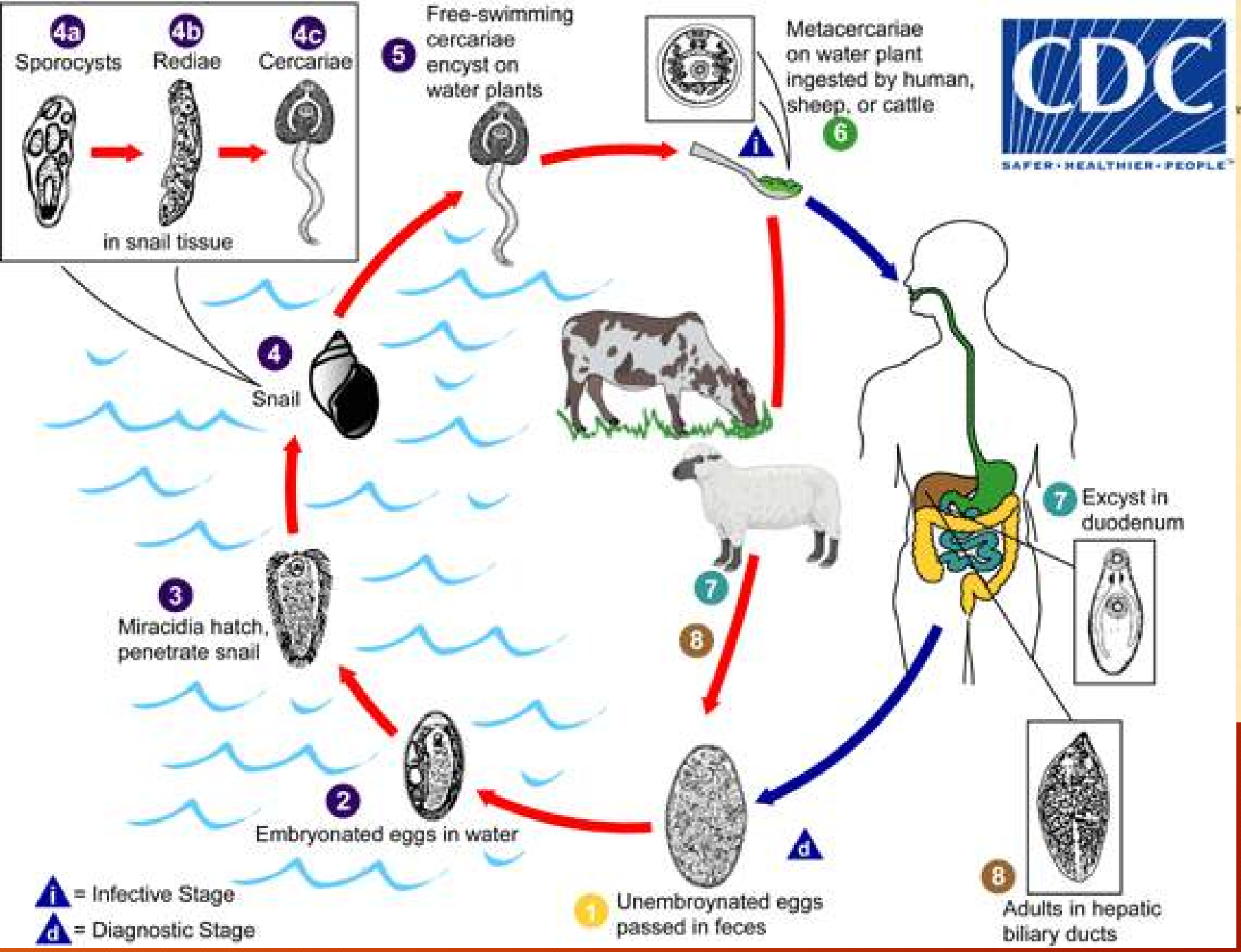

Symptoms of
Malaria
Central
- Headache
Systemic
- Fever
Muscular
- Fatigue
- Pain
Back
- Pain
Skin
- Chills
- Sweating
Respiratory
- Dry cough
Spleen
- Enlarge-
ment
Stomach
- Nausea
- Vomiting

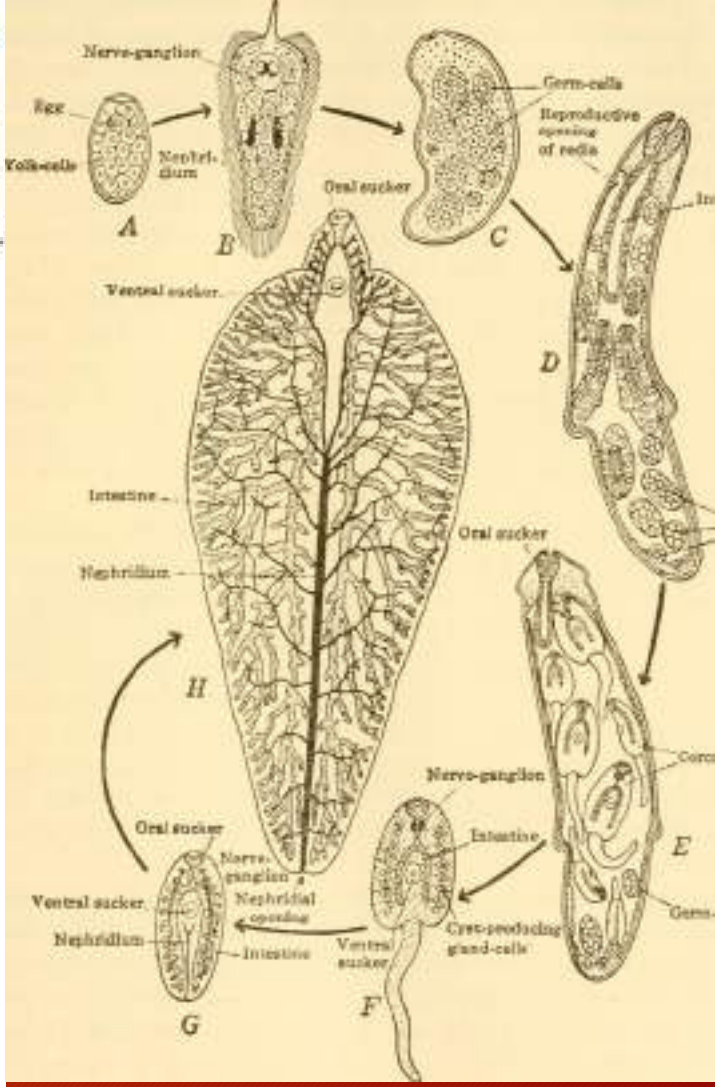
Trematoda

Liver fluke : *Fasciola hepatica*

- Liver fluke is a collective name of a polyphyletic group of parasitic flat worms under the phylum Platyhelminthes. They are principally parasites of the liver of various mammals, including humans. They parasitize in bile ducts, gallbladder, and liver parenchyma. In these organs, they produce pathological lesions leading to parasitic diseases. They have complex life cycles requiring two or three different hosts, with free-living larval stages in water.
- Fasciola hepatica, also known as the common liver fluke or sheep liver fluke, is a parasitic trematode (fluke or flatworm, a type of helminth) of the class Trematoda, phylum Platyhelminthes. It infects the livers of various mammals, including humans. The disease caused by the fluke is called fasciolosis or fascioliasis, which is a type of helminthiasis and has been classified as a neglected tropical disease. Fasciolosis is currently classified as a plant/food-borne trematode infection, often acquired through eating the parasite metacercariae encysted on plants
- The body of liver flukes is leaf-like, and flattened. The body is covered with a tegument. They are hermaphrodites having complete sets of both male and female reproductive systems. They have simple digestive system, and primarily feed on blood. The anterior end is the oral sucker opening into the mouth. Inside, mouth lead to a small pharynx which is followed by an extended intestine that runs through the entire length of the body. The intestine is heavily branched and anus is absent. Instead the intestine runs along an excretory canal that opens at the posterior end. Adult flukes produce eggs which are passed out through the excretory pore. The eggs infect different species of snails (as intermediate hosts) in which they grow into larvae. The larvae are released into the environment from where the definitive hosts (humans and other mammals) get the infection. In some species, another intermediate host is required, generally a cyprinid fish. In this case, the definitive hosts are infected from eating infected fish. Hence, they are food-borne parasites
- Fasciola hepatica is one of the largest flukes of the world, reaching a length of 30 mm and a width of 13 mm (Fasciola gigantica, on the other hand, is even bigger and can reach up to 75 mm.It is leaf-shaped, pointed at the back (posteriorly) and wide in the front (anteriorly). The oral sucker is small but powerful and is located at the end of a cone-shape projection at the anterior end. The acetabulum is a larger sucker than the oral sucker and is located at the anterior end

Fasciola hepatica occurs in liver of definitive host and its life cycle is indirect. Definitive hosts of the fluke are cattle, sheep and buffaloes. Wild ruminants and other mammals, including humans, can act as definitive hosts as well. The life cycle of F. hepatica goes through the intermediate host and several environmental larval stages.Intermediate hosts of F. hepatica are air-breathing freshwater snails from the family Lymnaeidae. Although several lymnaeid species susceptible to F. hepatica have been described, the parasite develops only in one or two major species on each continent. Lymnaea viator, L. neotropica, are most common intermediate hosts. Several other lymnaeid snails may be naturally or experimentally infected with F. hepatica but their role in transmission of the fluke is low. The metacercariae are released from the freshwater snail as cercariae, and form cysts on various surfaces including aquatic vegetation. The mammalian host then eats this vegetation and can become infected. Humans can often acquire these infections through eating freshwater plants such as watercress. Inside the duodenum of the mammalian host, the metacercariae are released from within their cysts. From the duodenum, they burrow through the lining of the intestine and into the peritoneal cavity. They then migrate through the intestines and liver, and into the bile ducts. Inside the bile ducts, they develop into an adult fluke.In humans, the time taken for F. hepatica to mature from metacercariae into an adult fluke is roughly 3 to 4 months. The adult flukes can then produce up to 25,000 eggs per fluke per day. These eggs are passed out via stools and into freshwater. Once in freshwater, the eggs become embryonated, allowing them to hatch as miracidia, which then find a suitable intermediate snail host of the Lymnaeidae family. Inside this snail, the miracidia develop into sporocysts, then to rediae, then to cercariae. The cercariae are released from the snail to form metacercariae and the life cycle begins again.


4a Sporocysts
4b Rediae
4c Cercariae
in snail tissue
5 Free-swimming cercariae encyst on water plants
Metacercariae on water plant ingested by human, sheep, or cattle
6
1
4
Snail
3 Miracidia hatch, penetrate snail
2 Embryonated eggs in water
7 Excyst in duodenum
8 Adults in hepatic biliary ducts
1 Unembroynated eggs passed in feces
i = Infective Stage
d = Diagnostic Stage


A
B
C
D
E
F
G
H
Nerve-ganglion
Germ-cells
Oral sucker
Ventral sucker
Intestine
Nephridium
Nephridial opening
Cyst-producing gland-cells

The alimentary canal of F. hepatica has a single mouth which leads into the blind gut; it has no anus. The mouth is located within the anterior sucker on the ventral side of the fluke. This mouth leads to the pharynx, which is then followed by a narrow oesophagus. The oesophagus, which is lined with a thin layer of epithelial cells, then opens up into the large intestine. As there is no anus, the intestine branches, with each branch ending blindly near the posterior end of the body.It has been shown that flukes migrate into smaller capillaries and bile ducts when feeding within the host. They use their mouth suckers to pull off and suck up food, bile, lymph and tissue pieces from the walls of the bile ducts.F. hepatica relies on extracellular digestion which occurs within the intestine of the host. The waste materials are egested through the mouth. The non-waste matter is adsorbed back in through the tegument and the general surface of the fluke. The tegument facilitates this adsorption by containing many small folds to increase the surface area.

Respiratory systemF. hepatica has no respiratory organs: the adult flukes respire anaerobically (without oxygen). Glycogen, taken from within the host is broken down via glycolysis to produce carbon dioxide and fatty acids. This process provides the fluke with energy. In contrast, the free-living miracidia stages of the parasite generally develop within oxygen rich environments. It is therefore believed that the free-living stages of the parasite respire aerobically, to gain the most energy from their environment.

Excretory system

F. hepatica's excretory system contains a network of tubules surrounding one main excretory canal. This canal leads to the excretory pore at the posterior end of the fluke. This main canal branches into four sections within the dorsal and ventral regions of the body. The role of F. hepatica's excretory system is excretion and osmoregulation.Each tubule within the excretory system is connected to a flame cell, otherwise known as protonephridia. These cells are modified parenchyme cells. In F. hepatica their role is to perform excretory, but more importantly, osmoregulatory functions. Flame cells are therefore primarily used to remove excess water.

Nervous system and sensory organs[edit]

The nerve system of Fasciola hepatica consists of a pair of nerve ganglia, each one is located on either side of the oesophagus. Around the oesophagus is a nerve ring. This nerve ring connects the two nerve ganglia together. The nerves stem off from this ring, reaching all the way down to the posterior end of the body. At the posterior end, one pair of nerves become thicker than the others these are known as the lateral nerve cords. From these lateral nerve cords, the other nerves branch. Sensory organs are absent from F. hepatica.

Reproductive system

F. hepatica adult flukes are hermaphrodite, this means each fluke contains both male and female reproductive organs. The male and female reproductive organs open up into the same chamber within the body, which is called the genital atrium. The genital atrium is an ectodermal sac which opens up to the outside of the fluke via a genital pore.The testes are formed of two branched tubules, these are located in the middle and posterior regions of the body. From the epithelium lining of the tubules sperm is produced. The sperm then passes into the vas deferens and then into the seminal vesicle. From the seminal vesicle projects the ejaculatory duct and this is what opens up into the genital atrium, many prostate glands surround this opening.On the right hand side of the anterior testis there is a branched, tubular ovary. From here, a short oviduct passes to the vitelline duct. This duct connects, via a junction, the ovaries, the uterus and the yolk reservoir. From this junction, the uterus opens into the genital atrium, this opening is surrounded by Mehlis glands. In some flukes, the terminal end of the uterus is strengthened with muscles and spines

F. hepatica reproduces both sexually, via the hermaphrodite adult flukes, and also asexually. The miracidia can reproduce asexually within the intermediate snail host.

Fasciola hepatica’s tegument protects it from the enzymes of the host's stomach, whilst still allowing water to pass through.Free-swimming larvae have cilia and the cercariae have a flagella-like tail to help them swim through the aquatic environment and also allow them to reach the plants on which they form a cyst.To attach within the host, F. hepatica has oral suckers and body spines. Their pharynx also helps it to suck onto the tissues within the body, particularly within the bile ducts.The adult fluke's respiration is anaerobic, this is ideal as there is no oxygen available in the liver.F. hepatica is adapted to produce a large number of eggs, this increases its chances of survival, as many eggs are destroyed on release into the environment. Also, F. hepatica is hermaphrodite, thus all flukes can produce eggs, increasing the number of offspring produced by the population.

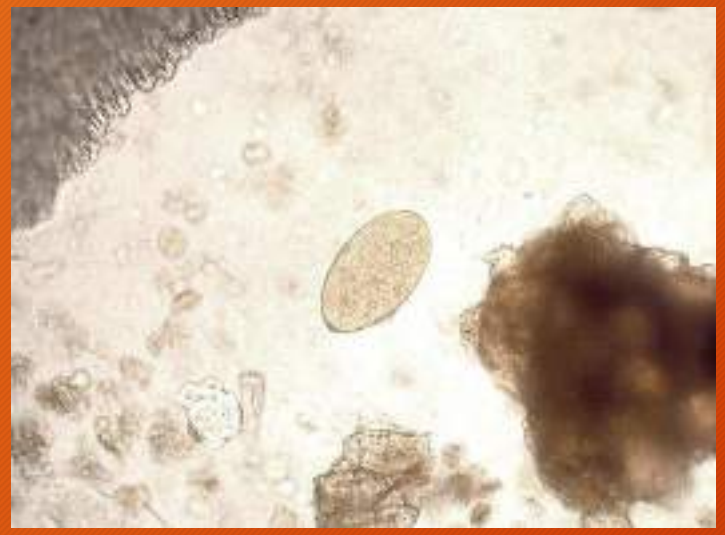
The genome for Fasciola hepatica was published in 2015. F. hepatica's genome, is one of the largest known pathogen genomes. The genome contains many polymorphisms, and this represents the potential for the fluke to evolve and rapidly adapt to changes in the environment, such as host availability and drug or vaccine interventions.

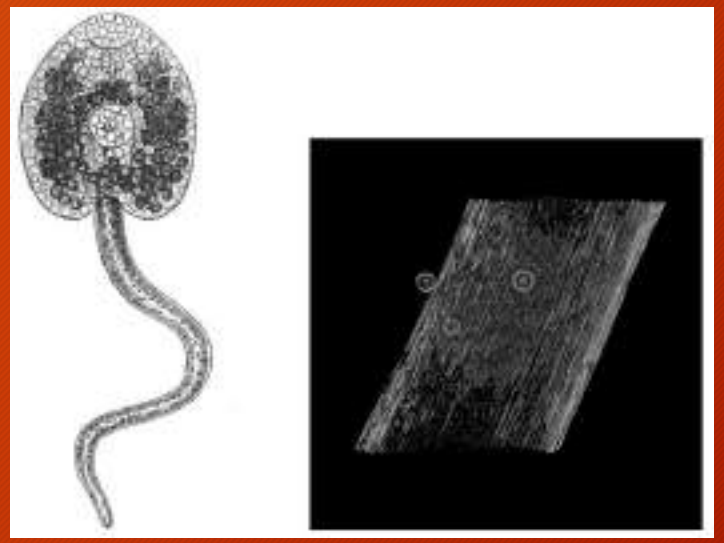
Both F. hepatica and F. gigantica can cause fasciolosis. Human symptoms vary depending on if the disease is chronic or acute. During the acute phase, the immature worms begin penetrating the gut, causing symptoms of fever, nausea, swollen liver, skin rashes and extreme abdominal pain.The chronic phase occurs when the worms mature in the bile duct, and can cause symptoms of intermittent pain, jaundice and anemia.In cattle and sheep, classic signs of fasciolosis include persistent diarrhea, chronic weight loss, anemia and reduced milk production. Some remain asymptomatic. F. hepatica can cause sudden death in both sheep and cattle, due to internal hemorrhaging and liver damage.
Fasciolosis is an important cause of both production and economic losses in the dairy and meat industry. Over the years, the prevalence has increased and it is likely to continue increasing in the future. Livestock are often treated with Flukicides, which are chemicals toxic to flukes. The two chemicals used are triclabendazole and bithionol. Ivermectin, which is widely used for many helminthic parasites, has low effectivity against F. hepatica, as does praziquantel.For humans, the type of control depends on the setting. One important method is through the strict control over the growth and sales of edible water plants such as watercress. This is particularly important in highly endemic areas. Some farms are irrigated with polluted water, hence, vegetables farmed from such land should be thoroughly washed and cooked before being eaten.

The best way to prevent Fasciolosis is by reducing the lymnaeid snail population or separating livestock from areas with these snails.These two methods are not always the most practical, so control by treating the herd before they are potentially infected is commonly practiced.

A diagnosis may be made by finding yellow-brown eggs in the stool. They are indistinguishable from the eggs of Fascioloides magna, although the eggs of F. magna are very rarely passed in sheep, goats, or cattle. If a patient has eaten infected liver, and the eggs pass through the body and out via the faeces, a false positive result to the test can occur. Daily examination during a liver-free diet will unmask this false diagnosis.

An enzyme-linked immunosorbent assay (ELISA) test is the diagnostic test of choice. ELISA is available commercially and can detect anti-hepatica antibodies in serum and milk; new tests intended for use on faecal samples are being developed. Using ELISA is more specific than using a Western blot or Arc2 immunodiffusion.Proteases secreted by F. hepatica have been used experimentally in immunizing antigens





Clonorchis sinensis,
the Chinese liver fluke, is a human liver fluke belonging to the class Trematoda, phylum Platyhelminthes. This parasite lives in the liver of humans, and is found mainly in the common bile duct and gall bladder, feeding on bile. These animals, which are believed to be the third most prevalent worm parasite in the world, are endemic to Japan, China, Taiwan, and Southeast Asia, currently infecting an estimated 30,000,000 humans. 85% of cases are found in China. The infection called clonorchiasis generally appears as jaundice, indigestion, biliary inflammation, bile duct obstruction, even liver cirrhosis, cholangiocarcinoma (CCA), and hepatic carcinoma.
It is the most prevalent human trematode in Asia, and still actively transmitted in Korea, China, Vietnam and also Russia, with 200 million people at constant risk. Recent studies have proved that it is definite cancer-causing agent in the liver (carcinoma) and bile duct (CCA). For this reason the International Agency for Research on Cancer has classed it as a group 1 biological carcinogen in 2009.

An adult C. sinensis is a flattened (dorso-ventrally flat) and leaf-shaped fluke. The body is slightly elongated and slender, measuring 15–20 mm in length and 3–4 mm in width. It narrows down at the anterior region into a small opening called oral sucker, which act as the mouth. From the mouth run two tubes called caeca throughout the length of body. They are the digestive and excretory tracts. The posterior end is broad and blunt. A poorly developed ventral sucker lies behind the oral sucker, at about one-fourth of the body length form the anterior end. A common genital pore opens just in front of it. As a hermaphrodite, it has both male and female reproductive organs. A single rounded ovary is at the centre of the body, and two testes are towards the posterior end. The uterus from the ovary, and seminal ducts from the testes meet and opens at the genital pore. The testes are highly branched. Another highly branched organs called vitellaria (or vitelline glands) are distributed on either side of the body. The eggs are similar to those of other related flukes such as Opisthorchis viverrini and O. felineus, and are often confused during diagnosis. They small and oval in shape, measuring about 30x15 µm in diameter. They are sharply curved and with a clear convex operculum towards the narrower end. At the broader end is a stem-shaped knob. The larva called miracidium can be seen inside the fertilised egg.

The eggs of a C. sinensis are released through the biliary tract, and excreted out along with the faeces. The eggs are embryonated and contain the larvae called miracidia. Unlike most other flukes in which the miracidia undergo development and swim in water to infect suitable host, the eggs of C. sinensis are simply deposited in water. The eggs are then eaten up by snails.

First intermediate host

Freshwater snail Parafossarulus manchouricus often serves as a first intermediate host for C. sinensis in China, Japan, Korea and Russia.Other snail hosts include Alocinma longicornis Bithynia longicornis, Bithynia fuchsiana, Bithynia misella, Parafossarulus anomalospiralis, Melanoides tuberculata, Semisulcospira cancellata, Semisulcospira amurensis, Semisulcospira libertina, and Assiminea lutea in China.and Melanoides tuberculata in Vietnam.

Once inside of the snail body, the embryonic membrane is dissolved by the snail's digestive enzymes so that the miracidium hatches from the egg. The ciliated miracidium can move about, penetrating the intestine, enters the haemocoel and digestive gland. Here it undergoes metamorphosis into a sporocyst. The sporocyst gives rise to small larvae called rediae. The rediae burst out from the sporocyst to become the next-stage larvae called cercaria. This system of asexual reproduction allows for an exponential multiplication of cercaria individuals from one miracidium. This aids the Clonorchis in reproduction, because it enables the miracidium to capitalize on one chance occasion of passively being eaten by a snail before the egg dies. The mature cercariae actively bore out of the snail body into the freshwater environment. But they are non-feeding and must find a fish host within 2–3 days, otherwise they die.

The ceraciae of C. sinensis are different from those of other flukes in that they do not actively swim. In stead, they initially hang upside-down in the water, and then sink to the bottom. They swim to the water surface to resume their initial position, and the movement is repeated again. They simple attack fish when they feel any disturbance in their life-style.

Second intermediate host

When they detect fish, they attached themselves on the scales using their suckers. Boring their way into the fish's body, they penetrate into the fish muscle within 6 to 13 minutes. Within an hour of penetration they develop hard coverings called cysts, and become metacercariae. This protective cyst proves useful when the fish muscle is consumed by a host. The metacercariae gradually develop and become infective to the next hosts after 3–4 weeks.

The common second intermediate hosts are freshwater fish: common carp (Cyprinus carpio), grass carp (Ctenopharyngodon idellus), crucian carp (Carassius carassius), goldfish (Carassius auratus), Pseudorasbora parva, Abbottina rivularis, Hemiculter spp., Opsariichthys spp., Rhodeus spp., Sarcocheilichthys spp., Zacco platypus, Nipponocypris temminckii , and pond smelt (Hypomesus olidus). In China (including Taiwan), 102 species of fish and four species of shrimp are known to be the intermediate hosts. In Korea, 40 species of freshwater fish are recorded as hosts. Seven species of fish are known as hosts in Russia.

Definitive host

The metacercariae are eaten along with raw or undercooked fish. The cysts of the metacercariae are gradually digested by the human gastric acids, and upon reaching the small intestines, the entire cyst is lost. The free metacercariae penetrate the intestinal mucosa and enters the bile ducts. It takes 1-2 day for migration into the bile ducts. They start feeding on the bile secreted from the liver, and gradually grow. They become adult in about a month, and start laying eggs. The average lifespan of an adult fluke is 30 years. An individual fluke can produce 4,000 eggs in a day.

The definitive hosts are fish-eating mammals such as dogs, cats, rats, pigs, badgers, weasels, camels, and buffaloes.

Dwelling in the bile ducts, Clonorchis induces an inflammatory reaction, epithelial hyperplasia and sometimes even cholangiocarcinoma, the incidence of which is raised in fluke-infested areas.
One adverse effect of Clonorchis is the possibility for the adult metacercaria to consume all bile created in the liver, which would inhibit the host human from digesting, especially fats. Another possibility is obstruction of the bile duct by the parasite or its eggs, leading to biliary obstruction and cholangitis (specifically oriental cholangitis).
Unusual cases of liver abscesses due to clonorchiasis have been reported. Liver abscesses may be seen even without dilatation of intrahepatic bile ducts.
Symptoms
While normally asymptomatic most pathological manifestations result from inflammation and intermittent obstruction of the biliary ducts. The acute phase consists of abdominal pain with associated nausea and diarrhea. Long-standing infections consist of fatigue, abdominal discomfort, anorexia, weight loss, diarrhea, and jaundice. The pathology of long-standing infections consist of bile stasis, obstruction, bacterial infections, inflammation, periductal fibrosis, and hyperplasia. Development of cholangiocarcinoma is progressive.

Diagnosis and treatment

Infection is detected mainly on identification of eggs by microscopic demonstration in faeces or in duodenal aspirate. But other sophisticated methods have been developed such as ELISA, which has become the most important clinical technique. Diagnosis by detecting DNAs from eggs in faeces are also developed using PCR, real-time PCR, and LAMP, which are highly sensitive and specific. Imaging diagnosis has been studied in depth and is now widely used. Drugs used to treat infestation include triclabendazole, praziquantel, bithionol, albendazole, levamisole and mebendazole. However, benzimidazoles are very weak as vermicide. As with other trematodes, praziquantel is the drug of choice. Lately, tribendimidine has been acknowledged as an effective and safe drug.
Genetics
The draft genome sequence of C. sinensis was published in 2011. The total genome size is estimated to be 580 Mb, and the GC content was calculated as approximately 43.85%. About 16,000 genes are predicted, out of which 13,634 genes have been identified.