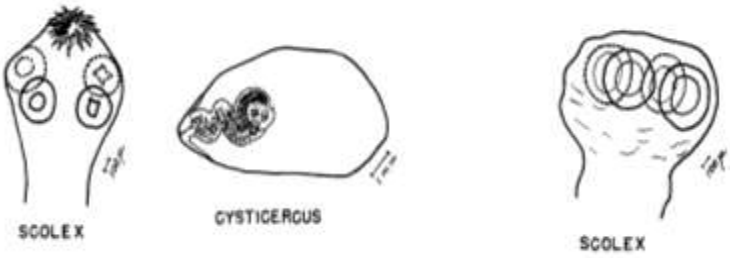
HELMINTHS – CESTODES (tapeworms)

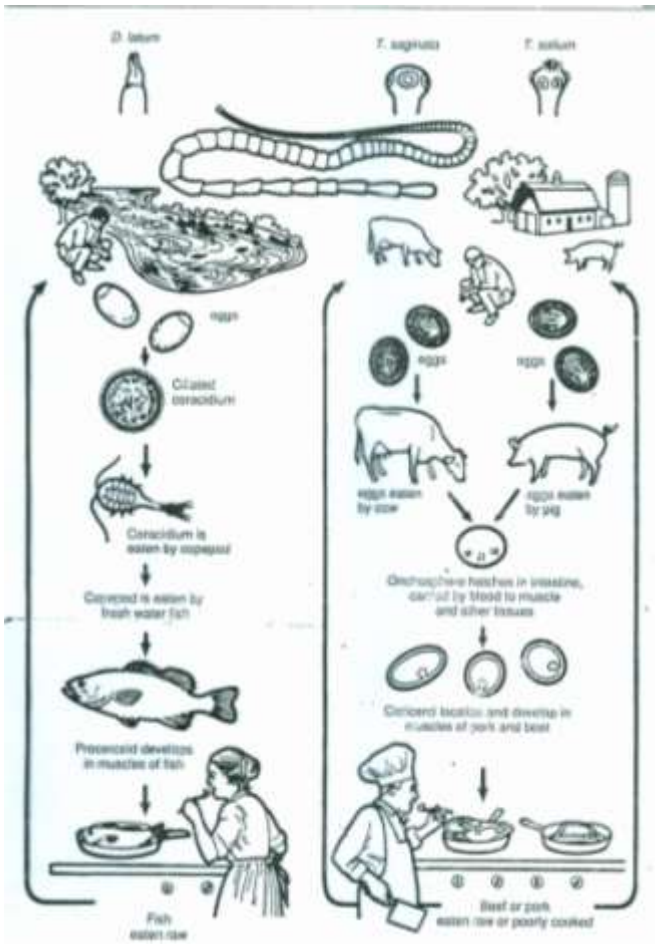
The eggs of cestodes as mentioned before have a striated outer covering. Inside the egg, there is a rounded embryo with six hooks. That's why it's called **hexacanth** (canth means spine). Those eggs are released by the primary host (human beings) in water or in soil during defecating or urination, the eggs are large and has a lid which called **operculum,** the operculum opens upon reaching water and the embryo is released, embryo must be adapted to be able to swim in water to find the intermediate host, that's why it's ciliated, this ciliated embyo is called **Ciliated Coracidium**. Once it reaches to the intermediate host (fish, cow or pig) it will be eaten by them. When the embryos (eggs) settle in the muscles (tissues) of the intermediate host, there they change their morphology (develop) and they become known as a **cysticercus** (bladder worm) a cystic form of parasite inside the tissues of the intermediate host it's also a fluid filled sac containing in one end invaginated structure which is **rudimentary scolex** .



In the figure here (in the middle) we have a cysticercus filled with yellowish fluid, it looks like a balloon invaginated at one end by a structure called rudiment scolex.

**** Remember: primary host is a host in which the parasite grows mature: it's normally human, maybe an animal. Secondary or intermediate host is a host that harbors the parasite only for a short transition period it's normally animal or insect, humans (accidental)**

Now … If a human being eat the meat of (fish pig or cow) which has been undercooked or not cooked (raw) (the meat



of the intermediate host), then he will ingest cysticercus. cysticercus comes to the GI tract of the primary host then the scolex will evaginate and sticks to the wall of the small intestine and from the distal end (neck region) it will proliferate and produce a long complete worm.

If you cook food, the color of meat become grey and that's usually enough to kill cysticercus and prevent infection.

the life cycle of cestodes depends on the intermediate host, at the top of the figure (shown in the previous page) 3 different worms are illustrated, some body is defecating (human being is the primary host) and feces contain the eggs of worms, eggs are eaten by pigs or cows (intermediate host) then hexacanth becomes cystacercus - again – cystacercus are localized in the muscles (flesh) of those pigs or cows, raw meat is eaten by human being to repeate the same process.

Sometimes fish is the intermediate host and once you eat a raw fish you will be infected by that worm (this is how the life cycle occur passing from primary host to intermediate host and back to the primary host).

Trematodes (flukes)

Endoparasites and the third group of helminths

Shape

Flattened (leaf shaped) worms generally. (an exception is schistosomes which are the worms that cause bulharzia (schistosomasis) they are either flattened, elongated or rounded according to the sex. Males are usually flattened and elongated while females are rounded)

Size

2-8 cm

Lifespan

Can be many or few years.

- All trematodes are **hermaphrodites** (organism that has reproductive organs normally associated with both males and females sexes) except **schistosoma** which has separate sexes (there are males and females).

- All trematodes (including **schistosomes**) require an intermediate host which is usually a fresh water snail (mollusk) on the other hand all cestodes **with one exception** require an intermediate host.

Figure 1

- trematodes can be luminal or tissue parasites.

Surface (tegument)

It has ridges and spines (it's ridged and spiny) this serves as an anchorage of the worm to the wall of lumen or tissue.

- Trematodes have 2 suckers: **oral sucker** which is around the mouth opening and **ventral sucker** on the ventral aspect of the worm distal to oral sucker (**all trematodes without exceptions** have these true suckers).

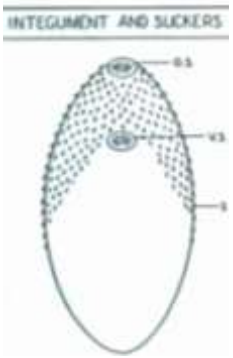


Figure 2

GI tract

They do have a GI tract which contains pharynx and esophagus then bifurcates into two parts, it's not complete, it has a blind ending (no anal opening) unlike nematodes. excretion of any undigested food (solid or semi solid waste is done by regurgitation (vomiting) - figure 1 -

- Note here the ridges or spines and the 2 suckers : oral sucker anteriorly and bellow it ventral sucker - figure 2-

Female and male reproductive system Usually superimposed in the same worm (**hemathodites**) except **schistosomes** that has separate sexes.

Nervous system

Ganglia with many nerve bundles extending forward and backward

Flame cells

Extending around the edges of the worm, functions like a kidney and secretion begins in these cells.

General morphology of their eggs

Large, operculated (have an operculum or lid) which is cap like structure, exception is schistosomes : their eggs are'nt operculated and have spines and depending on the species of schistosomes these spines are either terminal at the edge of the egg or it might be on the lateral side or it might be lateral but very small that's why we have 3 medically important types of schistosomes with different eggs and these eggs are differentiated by means of spines.

Life cycle

It's adapted to water

- the eggs are excreted out of human body (primary host) from various places depending on where the worms live inside the body : they can be excreted by feces (if they live in GI tract), urine (if they live in urinary tract) or sputum (if they live in lungs).

- the eggs are released and they have to reach water through feces, urine or sputum, once they go into water lid opens up out of the operculum, it will release the embryo inside.

- this embryo is ciliated (has cilia) so it can swim to find the intermediate host in water (which is the fresh water snail), this embryo is usually known as **miracidium,** it will penetrate the snail and once it goes into the snail it folds into a rounded ball of cells and undergoes morphological transformation producing **sporocyst** (a cyst that contains a nuclei and cells which divide), sporocyst divide again producing -inside it - daughter sporocysts which increase in number inside the large sporocyst.

- the sporocyst - depending on the species – will develop buds which called **rediae**, so the sporocysts may give rise to daughter sporocysts or undergo morphological transformation producing rediae and this depends on the species (some species develops rediae while others don't develop them and instead they only increase in number producing new sporocysts).

- within the sporocyst or rideae, a development of new morphology occur, this morphology looks like an adult worm, what do we mean with adult worm ??

This means that it's rounded, has a mouth, oral sucker, ventral sucker and a tail. these adult worms are known as **cercaria**.

- So **cercaria** develops either in **rediae** or **sporocysts**.

- circaria then released out of the snail into water (they have got tail to help them to swim in water looking for the primary host).

There are 2 methods by which these circariae can actually reach the primary host :

1) In some species (**tissue parasites like schistosomes**) **circaria** swims until it finds the body of the primary host (human being) and goes through the skin, these circareae are really adaptive for that because they look like an arrow (their head looks like an arrow) to help them penetrating the body of primary host, they will lose their tails after penetrating the skin because it's not required any longer, finally circareae go at certain tissues inside the body and become adult worms their.

2) In luminal or intestinal parasites, for circareae to get access to the primary host they first must become encysted متكيس)).

- How do they become encysted?
cicareae lose their tails, become rounded and covered by protective layer, they will look like an
adult worm (they have oral sucker, ventral sucker and GI tract) so now they are really covered by a protective layer in a cyst that's how they become encysted.

- Where do they become encysted?

They encyst in water either on aquatic plants which are eaten by people or on aquatic creatures (fish, shrimps or crabs) depending on species.

- When somebody comes and eat these plants or creatures while they are raw (un cooked or undercooked)(cooking again kills the worms and prevents infection) those encysted circareae which look like adult worms will reach to the GI tract and then the outercovering of the cyst disintegrates and then they become adult worms sticking to the wall of GI tract **and again this depends on species :**

- Luminal parasites or luminal species of termatodes get into the body of primary host by eating **but tissue parasites or tissue species of termatodes** (example : **schistosomes**) get into the inside of body by penetration through the skin.

Refer to figures :

A : there is an egg with operculum, the embryo inside it.

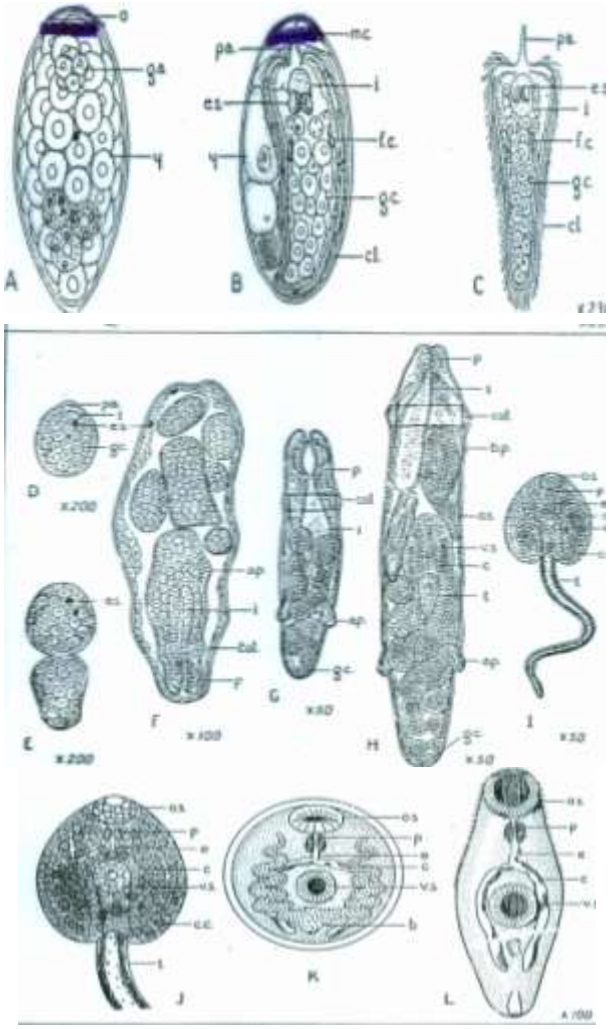
C: it shows **maracidium** which is ciliated, note the presence of pointed end which helps it penetrating the snail.

D: shows the **sporocyte** which may produces more daughter sporocytes or become rediae and within either the sporocyst or rediae (depending on species) circarea is developed.

I: shows circarea, note the presence of oral sucker ventral sucker and GI tract inside it, and it has a tail

J: circarea which has'nt encysted yet.

K, L : circarea is encysted (protected) and get inside aquatic plants or creatures.



** Schistosomes that penetrate the skin aren't illustrated here but its circarea is like an arrow with a head, body and tail (adaptation for penetrating the skin).

NOTE: the following figures (1, 2) shows the life cycle of tissue and luminal parasites respectively.

Life cycle of tissue parasites (schistosomes) *: refer to figure 1*

Their eggs are'nt operculated but have spines, these eggs get into water usually by urination or defecation.

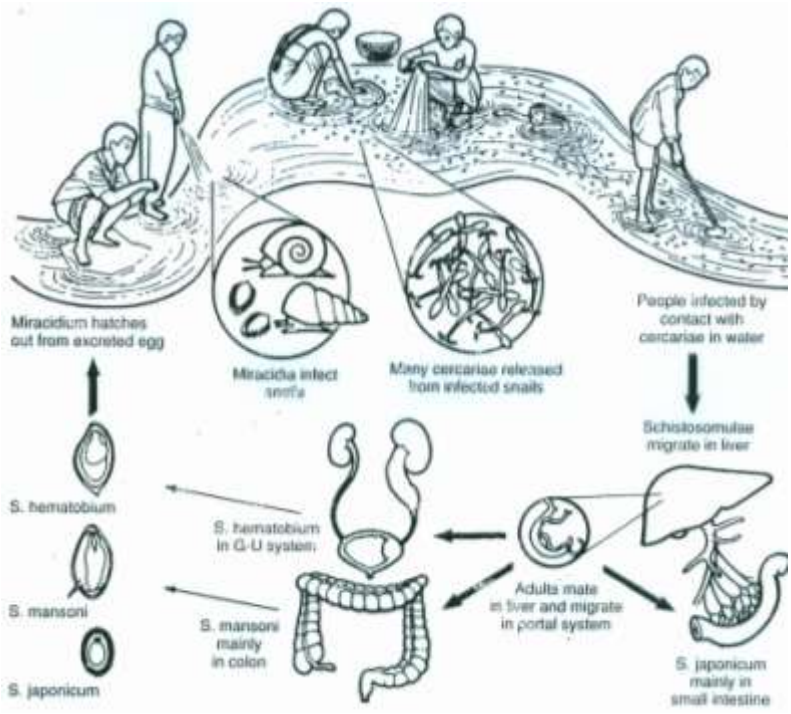


Figure 1

- In the right circle there are circareae, they look like arrows, if somebody comes in contact with water (by swimming, washing clothes or dishes) circareae go through the skin settled in tissues to repeat the cycle again.

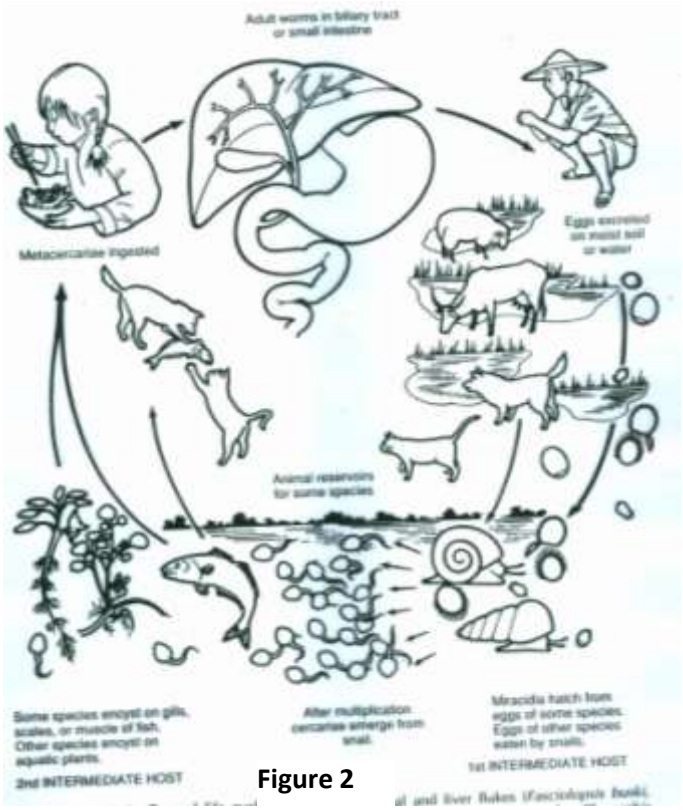


Figure 2

Life cycle of luminal parasites: *refer to figure 2*

Again this figure shows the life cycle of luminal parasites, someone (the primary host that has got the worms inside GI tract) is defecating in water, the feces contain eggs which are operculated , eggs hatch releasing the embryo which is ciliated and swim to find the intermediate host (fresh snails) it became cercaria after penetrarting the snails then those circareae are cysted on plants or creatures that will be eaten by human to reach the GI tract and form adult worms.. Then the cycle is repeated.

- Once schistosoma has many exceptions in many things consedering trematodes, then why did we consider schistosoma as trematodes ??

Because schistosoma has 2 scuckers, mouth, GI tract with blind ending, genetically we consider that schistosomes belong to trematodes.

Pathogenesis

What about the diseases that caused by parasites?

We have said before that ectoparasites are insects that live on skin and feed on blood, here we are concerned about pathogenesis of endoparasites:

- luminal parasites : sometimes you may have worms inside your GI tract for a long period of time and you don't feel something wrong which means that symptoms are absent or minimal and you have no pathology (no damage to the person).

-luminal parasites don't usually evoke an immune response (technically speaking as if they are outside the body), and that's why you don't have usual changes that occur when you are infected with tissue parasites.

- tissue parasites : in case of having tissue parasites inside the body you may have changes, the body will react with those parasites by an immune response resulting in the production of special cells and immunoglobulins in this case all symptoms will occur depending on where tissue parasite lives (liver, lung, urinary tract, eye, brain) so the actual presence of those worms cause production of inflammation which is chronic and tend to produce tissue damage and clinical consequences .
- in some cases it's believed that damages actually is like **hypersensitivity**, the patient becomes allergic to the constituents of the organ (parasite), this will result in tissue damage, this response is known as **delayed type hypersensitivity (DTH).**

- Pathology may be induced by the physical presence of the parasite, like in the following cases :

1) Luminal parasite that stuck to the wall of GI tract will suck blood causing **bleeding (blood loss)** resulting in **iron deficiency anemia.**

2) Some of these worms are very long (their length may reach 10 meters) and they extend into the terminal ileum, they can **compete for the absorption** of certain nutrients such as vitamin B12 (which is absorbed in the terminal ileum), this will result in **vitamin B12 deficiency** which causes **megaloblastic anemia.**

3) Sometimes worms may concentrate together inside the abdomen and cause **intestinal obstruction**, they cause blocking of the lumen.

4) Disturbances of absorption may result causing the patient to eat too much.

5) Ascaris worms release certain chemicals that cause **suppression of appetite** specially in children who become very thin with face turning yellow.

6) Giardia protozoa can damage the microvilli of small intestines causing **malabsorption** (mechanical action).

- Megaloblastic anemia : an anemia (blood with insufficient concentration of hemoglobin) that result from inhibition of DNA synthesis during red blood cells production, the defect in red blood cell DNA synthesis is due to vitamin B12 deficiency . --- --
Megaloblastic anemia may caused also by **folic acid deficiency .** -
Pernicious anemia : is a type of megaloblastic anemia result when there is lack of vitamin B12 but it can't be caused by the presence of luminal parasites in GI tract because it's an auto immune disease .

- So the presence of these luminal parasites in GI tract can lead to problems with the health of patient.

Diagnosis of parasitic diseases

1) General parasitic diseases give rise to eosinophils :

- Eosinophils are one of the white blood cells. White blood cells make up approximately 1% of the total blood volume in a healthy adult and eosinophils make up about 3% of white blood cells.

- Why do they called eosinophils ??

Because their granules take up eosin upon staining and appear red so they are "acid loving" granules which means these granules must contain something basic, the major content of these granules is actually **basic proteins**.

- Why do parasitic diseases give rise to eosinophils ?

The basic proteins (contents of eosinophil's granules) are toxic to parasites. when eosinophils come in contact with parasites it pours it's basic proteins on it to kill it.

- so in parasitic infection especially that's caused by tissue parasites you will have a high degree of eosinophils (eosinophilia), so during diagnosis if you have eosinophilia then the patient might suffer from a parasitic infection.

NOTE: eosinophilia is present in parasitic diseases but not in all of them and if you have eosinophilia this doesn't always mean that you have a parasitic disease because there are other causes of eosinophilia (other than parasitic disease) such as asthma, fever, lymphoma ….etc.

2) Serological test; looking for specific antibodies in patient's serum, the antibodies produced against parasites are usually of IgE class, but you can also find IgM, IgG.

Serology: looks for antibodies in the serum against certain antigens (parasites, bacteria, viruses...)

- During prarsitic disese there will be a production of certain class of immunoglobulins which is IgE (the patient will have an immune response and the body will produce IgM initially then switching to IgE specially against tissue infections (tissue parasites).

- IgE has receptors on eosinophils and when IgE recognize the worms it sticks to it, this process is kown as **opsonization,** eosinophils come and IgE binds to it's receptors on eosinophils causing them to pour the basic proteins and kill the worms.

- So IgE is the major antibody that is produced in parasitic diseases, however other immunoglobulins might be produced like IgG which is specific against antigens produced by particular parasites (worms).

- **in case of tissue parasites** you examine a blood sample in parasitic disease, and look for to see whether there are eosinophils and mostly you look for IgEs to see whether they are raised or not also you can look for specific IgGs (when you suspect a certain worms to be present in the body, you look for whether there is IgG present against that particular specific worms or not), **in case of luminal parasites** all those changes may not occur (they don't elicit an immune response) that's why luminal parasites are considered as if they were outside the body.

3) **You can diagnose parasitic disease Directly from the parasite itself.** Ex: in cases of Ascaris, the patient might come to the doctor holding a worm that just came out of him!! (But this occurs rarely).

4) We can look for forms of the parasites in body excretion : 1) feces (because many parasites are present in the GI tract / 2) blood (in cases of malaria) / 3) urine or any body fluid or by skin or tissue biopsy (in cases of tissue parasites)
- we mainly look for :cyst → in case of protozoa / eggs → in case of worms / in case of nematodes we look for microfilaria which has characteristics like (sheath, nuclei..)
- in this method we mainly look for the eggs rather than other forms because each worm has got characteristic morphology of eggs which help to distinguish the species of the worm.

Treatment

1) All parasites have drugs that are useful in treatment; some of these drugs can be toxic or have side effects.
2) Prevention of transmission is a way to treat parasitic disease this is done by maintaining hygiene.
3) Elimination of intermediate host by cleaning water to reduce the number of schistosomes is one of the ways used to reduce infections by parasites.

Vaccination

There is no vaccination against parasites (on the other hand there are vaccines against viruses or bacteria) why??

Because parasites have many morphologies and keep changing always from one to another this means their antigens are also changing a lot, so it's difficult to find a suitable antibodies to use as vaccines against those antigens.