# *Human intestinal parasites*

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# Human parasites

- Parasites are infective organisms belong to the animal kingdom
- It include protozoa , helminths , and arthropods
- Protozoa : unicellular organisms that are able to multiply within their host e.g. Amebiasis , Giardiasis.
- Worms or helminths: multicellular and usually do not divide within the human host e.g. Ascariasis & hookworms

# Protozoan diseases

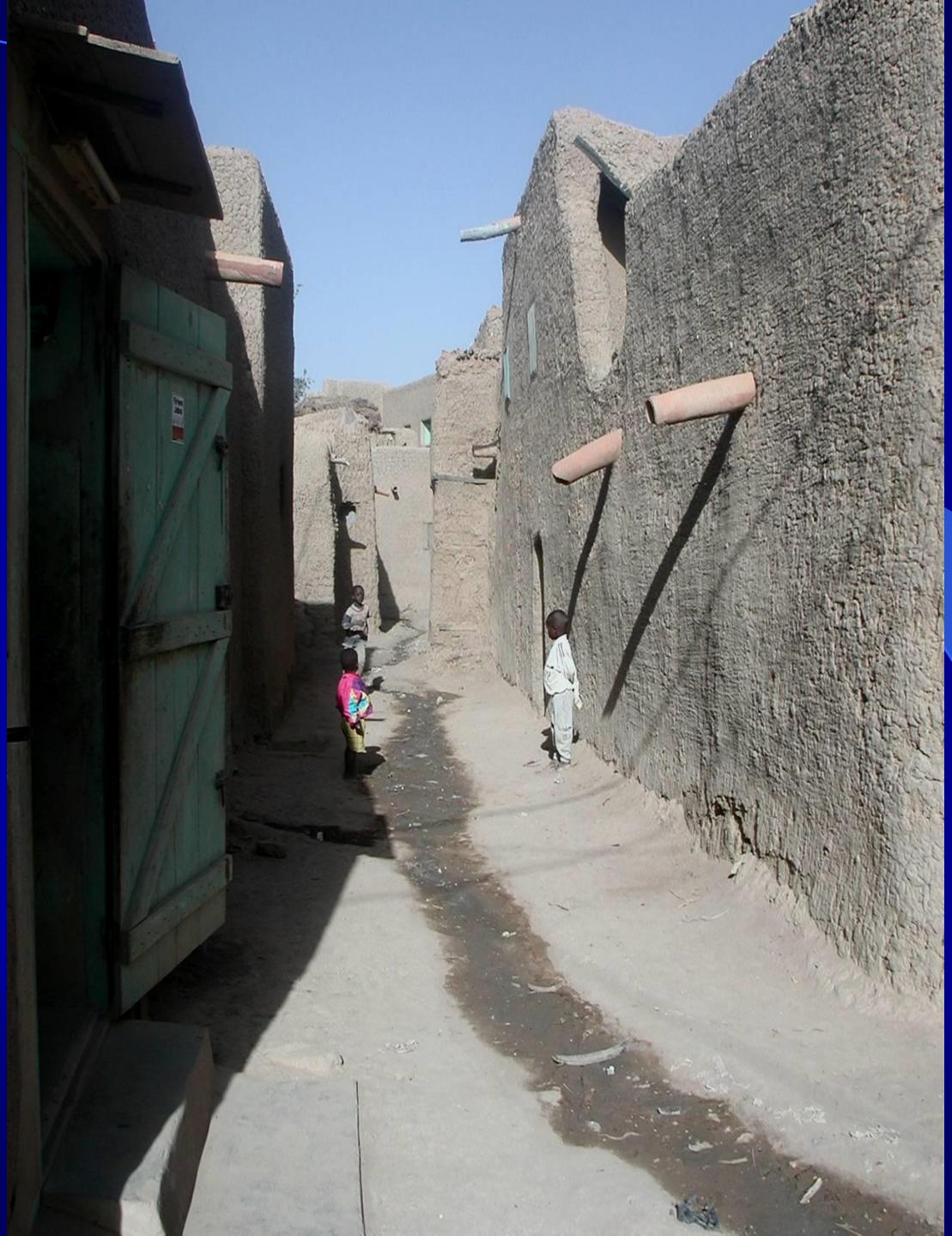
- Infection is usually acquired orally through fecal contamination of water or food.
- More endemic in countries with unsanitary water condition.
- Cryptosporidium & Isosporiasis have become major causes of diarrhea in patient with AIDS.

# Amebiasis

## A. Etiology & Epidemiology:

- ***Entamoeba histolytica*** is an intestinal protozoan causing amebiasis or amebic dysentery worldwide.
- it is more common in tropical countries or other areas with poor sanitary conditions.
- It is estimated that up to 10% of the world's population may be infected with *E. histolytica* and in many tropical countries the prevalence may approach 50%.
- There are an estimated 50 million cases of amebiasis per year and up to 100,000 deaths.
- *E. histolytica* is pathogen that exhibits a wide spectrum of virulence , ranging from asymptomatic or mild disease to a highly invasive and destructive organism.

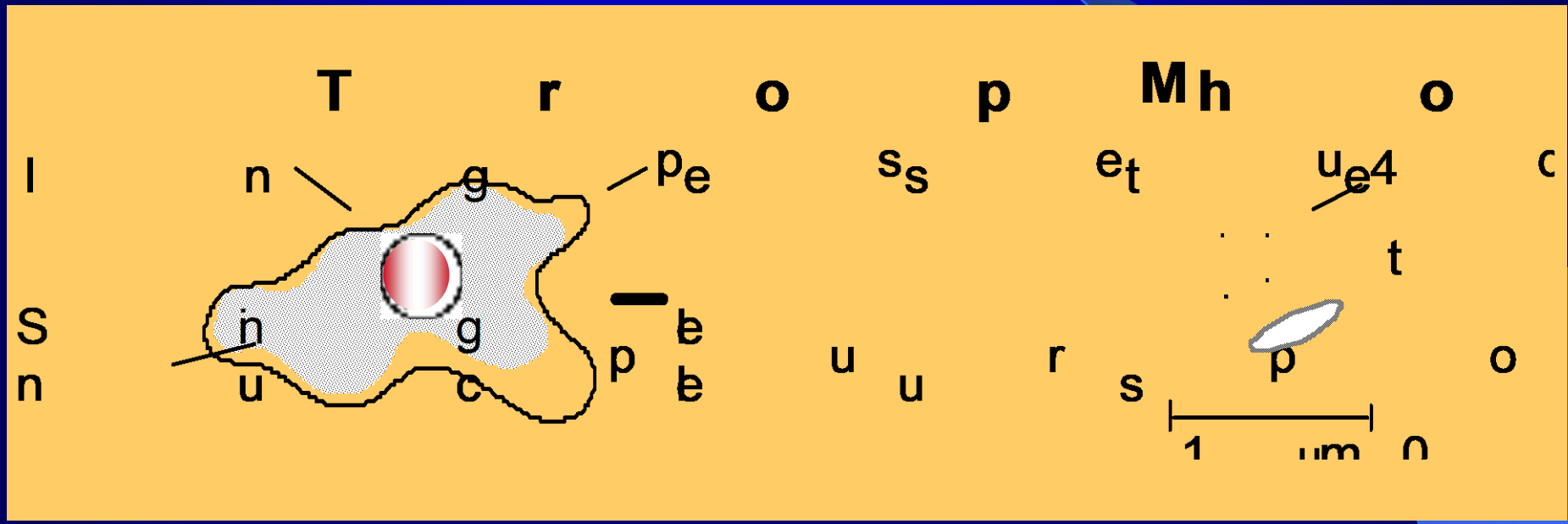
Poor sanitation is a factor in transmission of ***Entamoeba histolytica***



# Etiology & Epidemiology (cont.)

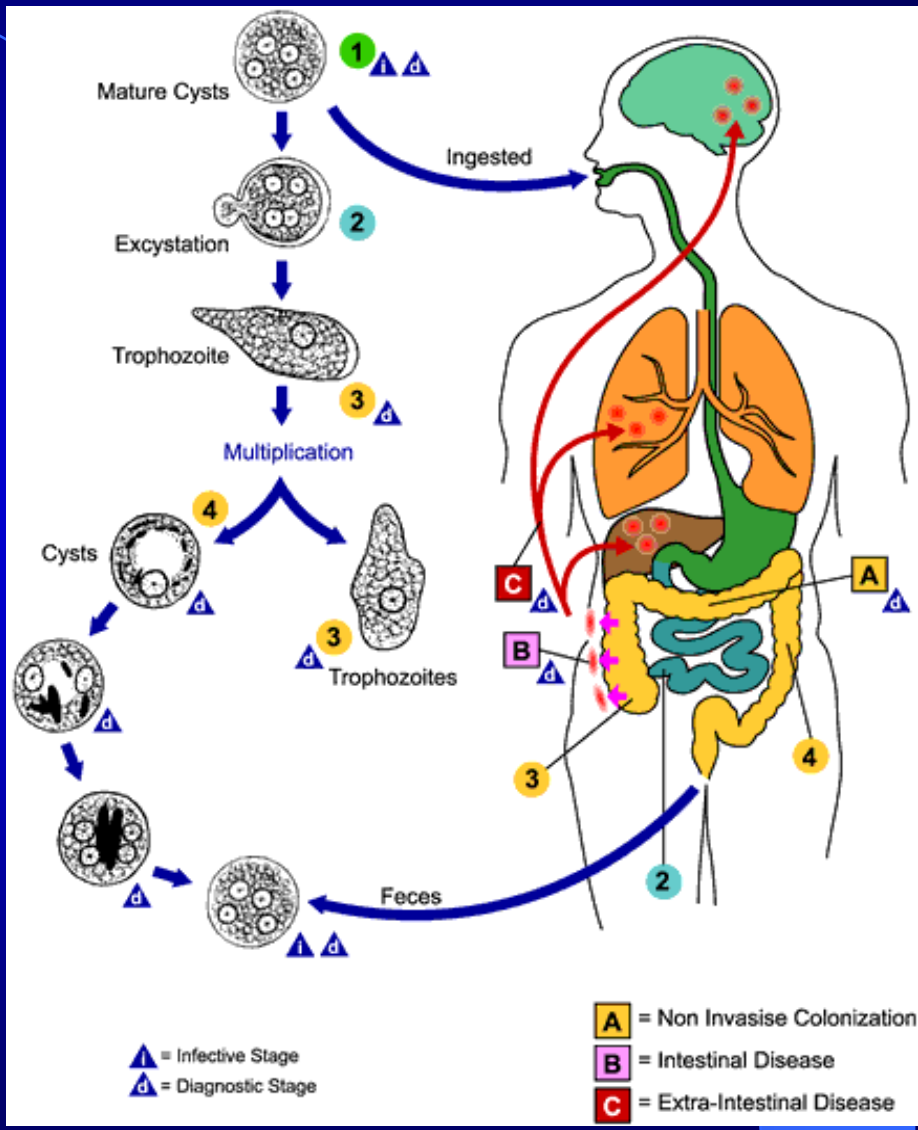
- Entamoeba dispar and Entamoeba moshkovskii are nonpathogenic parasites that are identical morphologically to *E histolytica*. but genetically distinct species ,E dispar  is more prevalent & associated only with asymptomatic carrier state.
- Other non-pathogenic species that infrequently colonize the G.I.T. :  E. coli , E. hartmanni , E.gingivalis & E.polecki .
- Infection is caused by cysts ingestion which resist low temperature , chlorine in usual concentration , gastric acidity and digestive enzymes , but can be killed by heating to 55 c.

# *E. histolytica* - parasitic forms



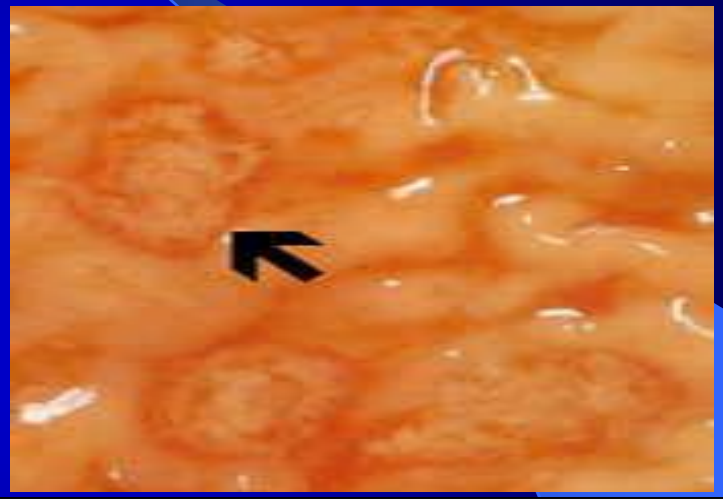
## B. Life Cycle:

- Mature cysts are ingested via contaminated water, food or hands (1)
- Excystation(2)occurs in the small intestine and trophoz (3) are released and migrate to the large intestine. They multiply by binary fission and produce cysts(4)which are passed in the feces.



# C. Pathology:

- When it invades the bowel wall (invasive phase) it causes a diarrheic syndrome & intestinal ulcers (Flask-shaped ulcer) ; intestinal disease and may spread to extra intestinal sites such as the liver, brain,and lungs, with resultant pathologic manifestations.
- Among various amebas,the *Entamoeba histolytica* is the one that invades tissues in man. in many cases, the trophozoites remain confined to the intestinal lumen (non-invasive infection), it causes no problems.



The lumenal side of the colon from fulminating amebiasis case showing several ulcers. Note raised edges (arrow).

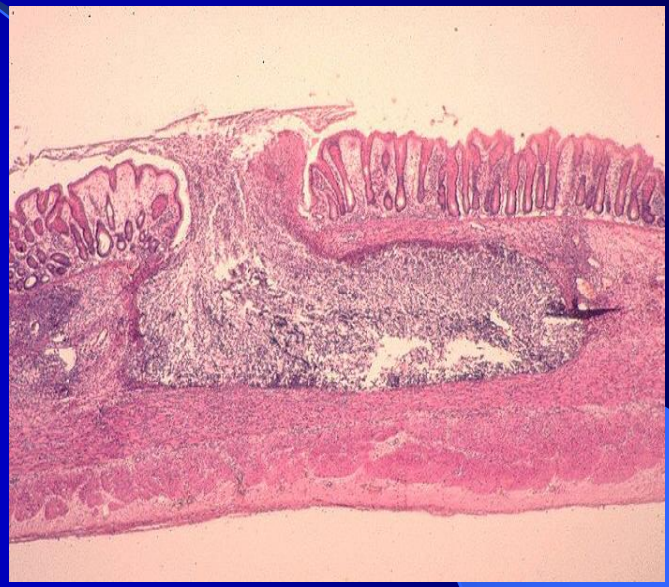
## Pathology (cont.)

- **non-invasive** Amoebiasis

  ameba colonize mucosal surface
  - asymptomatic cyst pass
  - non-dysenteric diarrhea
- **invasive**

  necrosis of mucosa ulcer
  - dysentery
  - hematophagous trophozoites
  - Flask shape ulcer
- **metastasis extraintestinal amoebiasis**

  ( less common in children than adult)
  - primarily liver amebic abscess
  - Rarly Brain , pleuropulmonary , skin , or genitourinary lesions.



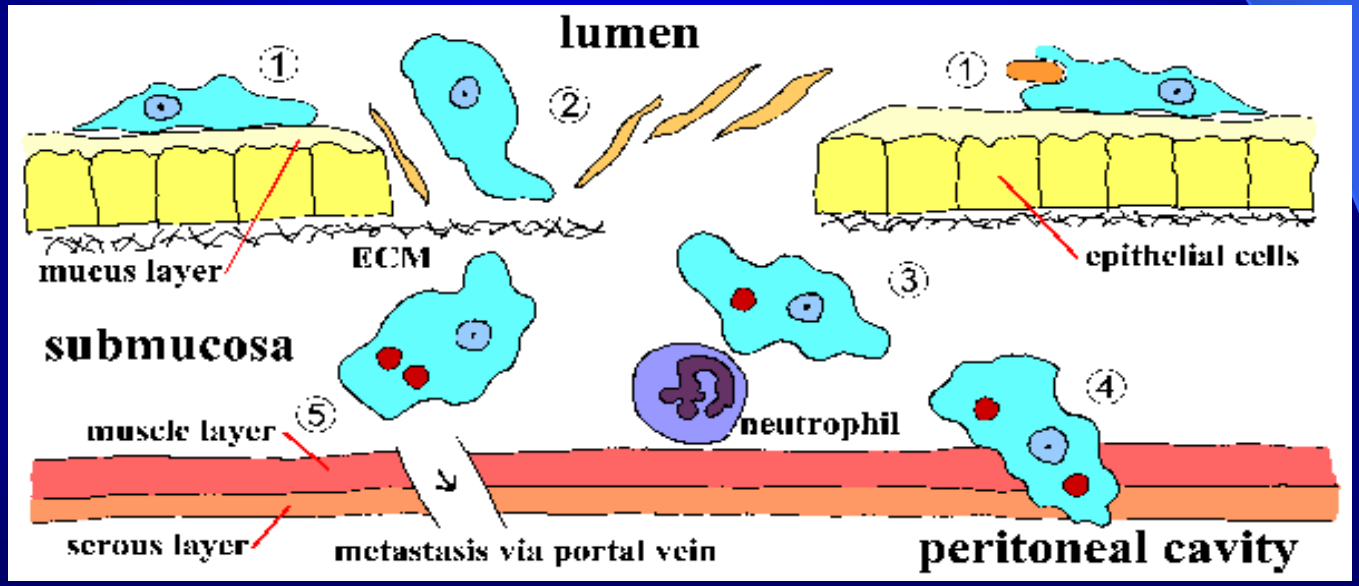
**Histopathology of a typical flask-shaped ulcer of intestinal amebiasis**

# Pathology (cont.)

## Invasion of intestinal mucosa occurs in 5 steps:

1: Trophozoites adhere to the mucus layer; Depletion of the mucus barrier allows for the trophozoite to come in contact with epithelial cells. 2: Epithelial cells are killed in a contact dependent manner leading to a disruption of the intestinal mucosa. 3: The trophozoites will continue to kill host cells in the submucosa and further disrupt the tissue as they advance. 4: Disruption of the intestinal wall. 5: metastasis via the circulatory system is also possible.

**Adherence, cytotoxicity, and disruption of the tissues are important factors in the pathogenesis of *E. histolytica*.**



# Clinical classification

- Asymptomatic infection (carrier) in >90% of cases , so E. histolytica is symptomatic in ≤10% of cases .
- E dispar is 10-fold more common than E histolytica .
- 1% invasive amoebiasis from the total cases ( E dispar + E histolytica )
- Classified as Intestinal and Extra-intestinal amoebiasis

# A. Intestinal amoebiasis

- a. dysentery**:** dysenteric stools (pus and blood without feces). fever, dehydration, and electrolyte abnormalities. Tenesmus and abdominal tenderness.
- b. non-dysenteric colitis
- c. amoeboma:may become the leading point of an intussusception or may cause intestinal obstruction.

# B. Extra-intestinal amoebiasis

a. Hepatic (1-7% of children with invasive amebiasis)

(1) acute non-suppurative

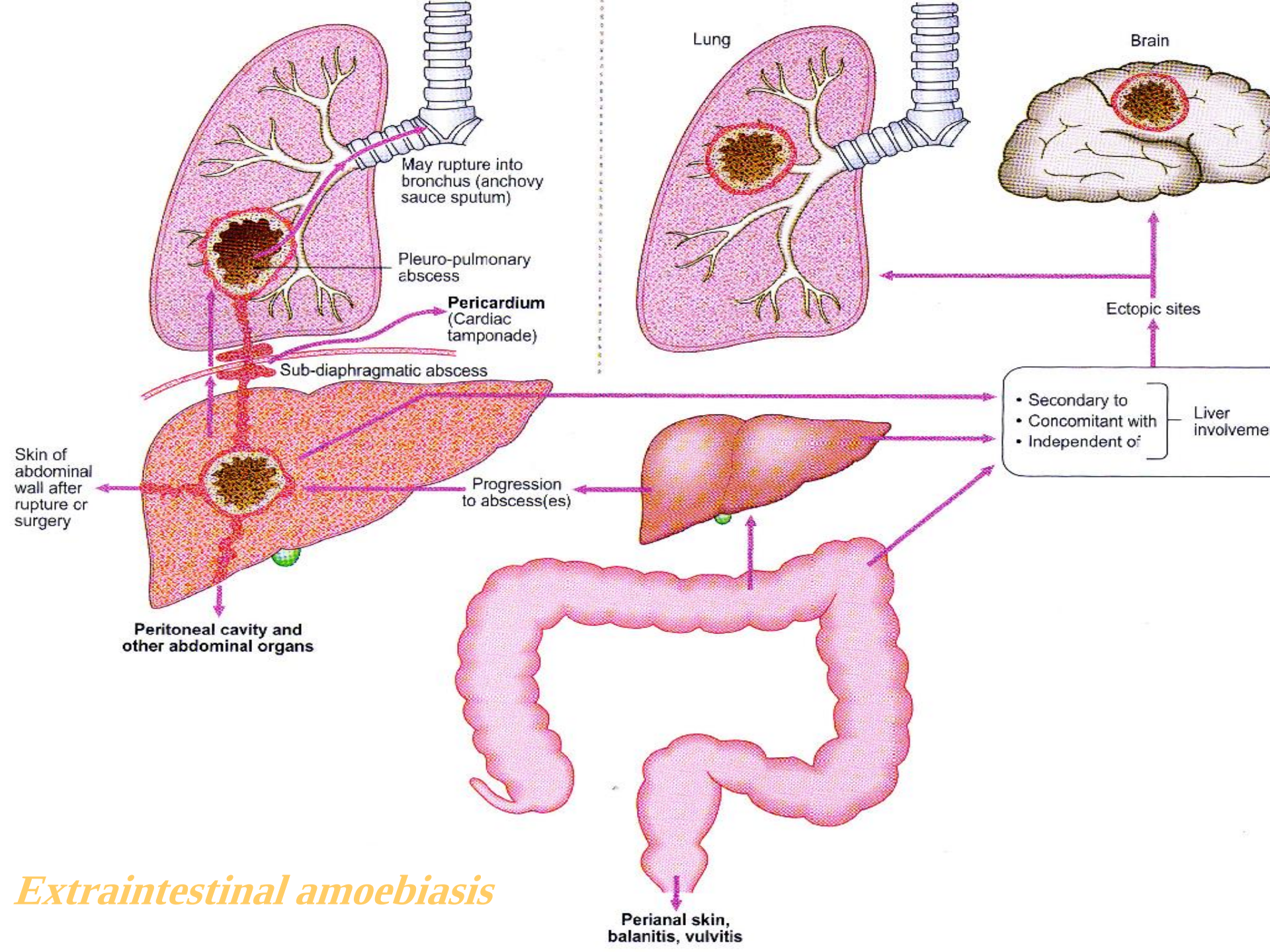
(2) liver abscess: right upper quadrant pain, referred to the right shoulder. Tender hepatpmegaly & elevation of diaphragm.

b. Pulmonary

c. Brain

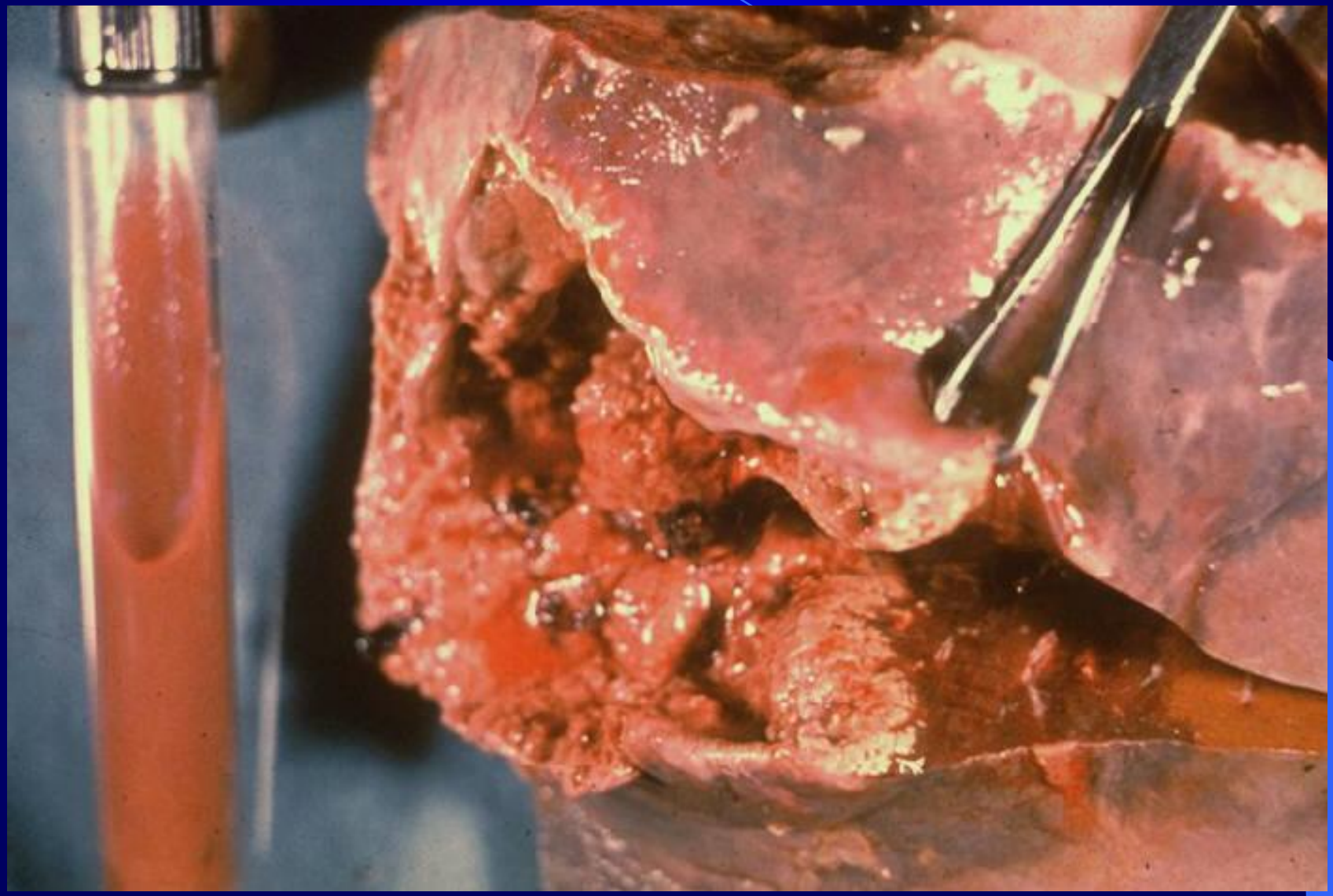
d. Skin , perianal infection

e. Other extra-intestinal amoebiasis




# *Extraintestinal amoebiasis*

# Gross pathology of amebic abscess of liver. Tube of "chocolate" pus from abscess.



# D. Diagnosis:

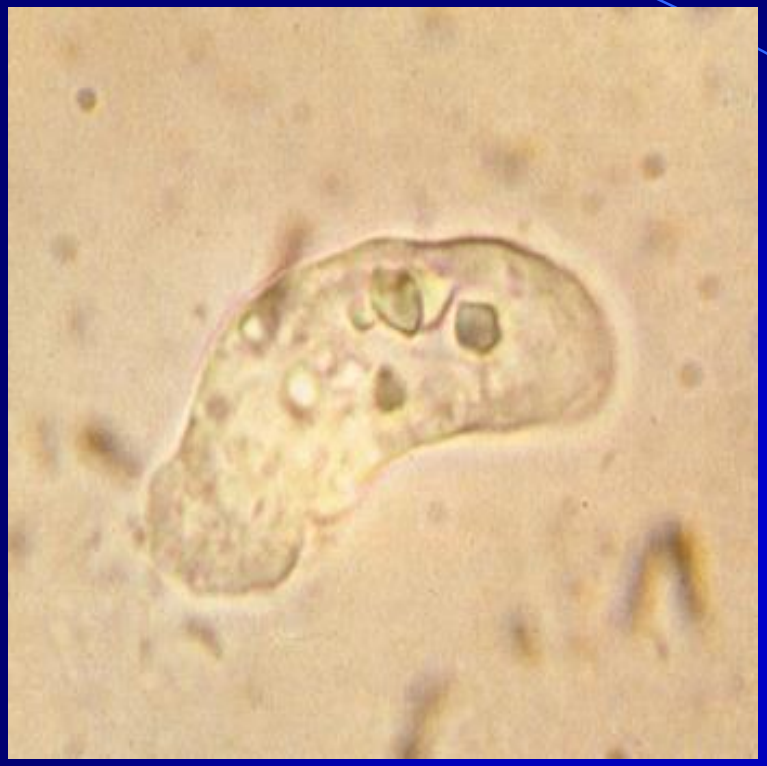
- **Microscopy (Stool examination):**

  Microscopic identification of cysts and trophozoites in the stool is the common method for diagnosis. To confirm the diagnosis motile trophozoites containing red blood cells must be identified: the presence of amoebic cysts alone does not imply disease.

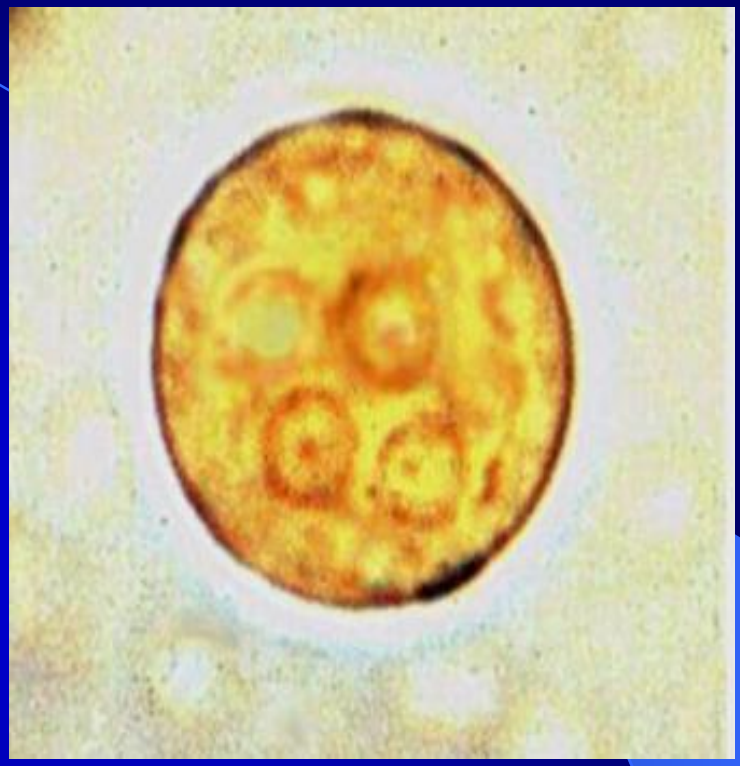
- Examination of a fresh stool smear for trophozoites that contain ingested RBCs is rather insensitive. Routine microscopy cannot distinguish the *E dispar* and *E moshkovskii* from *E histolytica.*

- **Morphologic comparison with other intestinal parasites**

  *E. histolytica* must be differentiated from other intestinal protozoa including :*E. coli* ,E. hartmanni ,Differentiation is possible, but not always easy, based on morphologic characteristics of the cysts and trophozoites.



**Trophozoite in stool**



**Cyst in stool**

- An enzyme immunoassay kit to specifically detect *E histolytica* in fresh stool specimens is commercially available.
- PCR-based diagnostic tests have been developed but are not widely available
- Serum tests for Antibody : Serum antibodies against amebae are present in 70-90% of individuals with symptomatic intestinal *E histolytica* infection. Antiamebic antibodies are present in as many as 99% of individuals with liver abscess who have been symptomatic for longer than a week, Seropositivity is low in asymptomatic cyst passers
  - Sigmoidoscopy and barium enema examination may show colonic ulceration but are rarely diagnostic
  - samples are always heme positive.
  - Fecal leukocytes may be absent.

## Diagnosis (cont.)

In hepatic amebiasis investigation may include :

-chest x ray – raised diaphragm.

-U/S.

-CT scan,MRI ,Isotope scan.

-CBC , LFT (leukocytosis, anemia & raised liver enzymes)

# Treatment

Treat all symptomatic, and also asymptomatic if not in endemic area

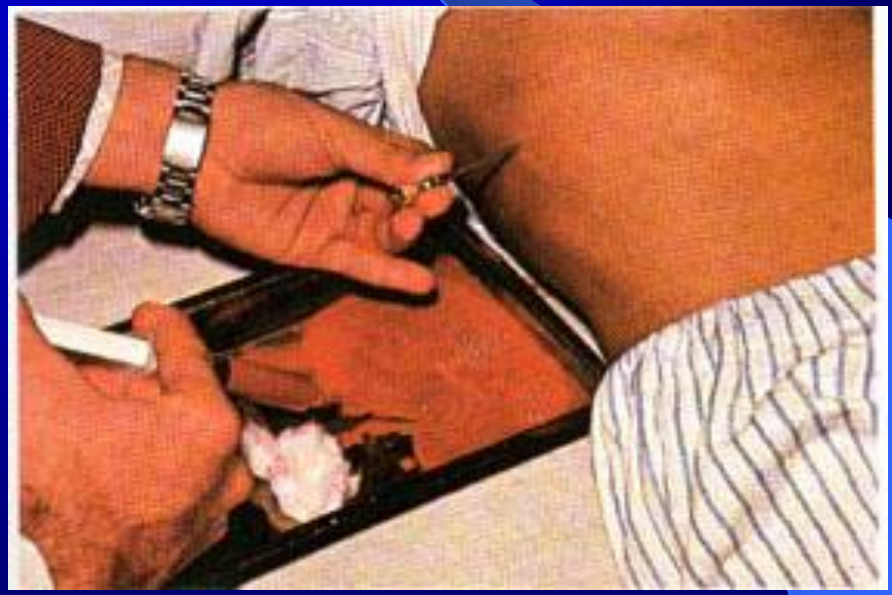
- Metronidazole is the mainstay of therapy for invasive amebiasis
- Tinidazole is also effective: dehydroemetine and chloroquine are alternative drugs, but are rarely used.
- Luminal amebicides: Iodoquinol, Paromomycin & Diloxamide furoate ,used in asymptomatic individual.
- After treatment of the invasive disease, the bowel should be cleared of parasites with a luminal amoebicide .

# Treatment

- Broad-spectrum antibiotics may be added to treat bacterial superinfection in a case of fulminant amebic colitis and suspected perforation.
- Surgical aspiration of liver abscess may be needed.
- Stool examination should be repeated 2 weeks after stopping therapy.

# An Amoebic Liver Abscess Being Aspirated.

- Note the reddish brown color of the pus This color is due to the breakdown of liver cells.



# Prevention

- Human feces should not be used as fertilizer
- Food and drinks must be protected from flies
- Personal hygiene:  wash hands after defecation and before meals.
- Regular examination of food handlers .
- Investigation of diarrhea episodes.
- There is no prophylactic drug or vaccine.

# prognosis

- Most infection end with eradication or asymptomatic carrier state, death in about 5% of extraintestinal infection.

# *Giardiasis*

## A. Epidemiology:

- ***Giardia intestinalis*** *{Giardia lamblia}* is the causative organism of an infection to small intestine called Giardiasis.
- It has worldwide distribution. The incidence is estimated at 200 million cases per year, prevalence varies from 0.5 to 50%.
- Typically *Giardia* is non-invasive and quite often results in asymptomatic infections. Symptomatic giardiasis is characterized by acute or chronic diarrhea and/or other gastro-intestinal manisfestations.
- Giardiasis usually represents a zoonosis with cross-infectivity between animals and humans.
- The organism is known to have multiple strains with varying abilities to cause disease, and several different strains may be found in one host during infection.

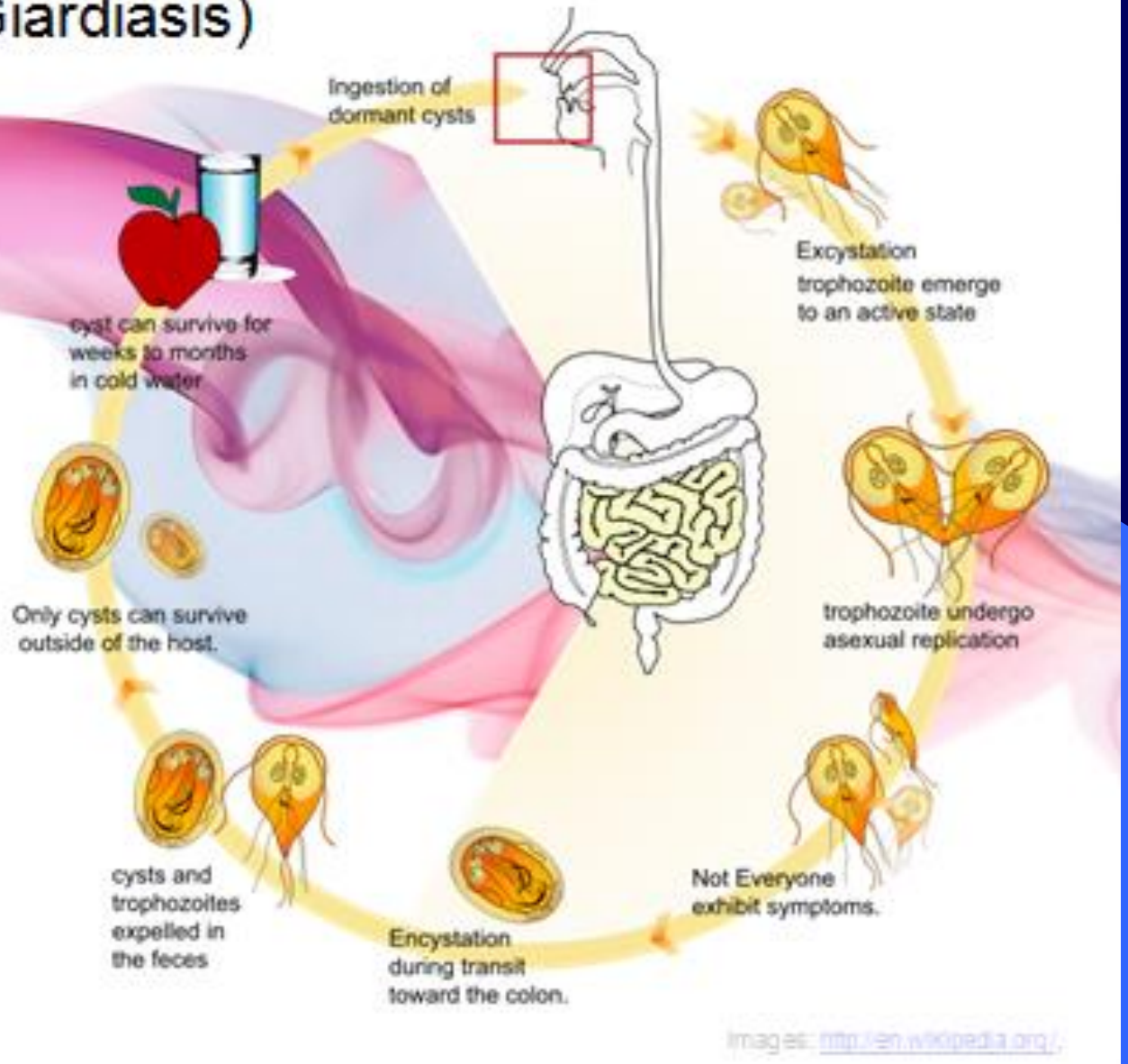
## . Epidemiology:

- The infective dose is low in humans; 10-25 cysts are capable of causing clinical disease
- It is the most common identified cause of water-borne disease associated with breakdown of water purification systems.
- Children are affected more than adult.
- Important cause of morbidity in developing world.
- Significant pathogen in people with malnutrition,humoral immune deficiency & cystic fibrosis.
- Cyst viability often is not affected by the usual concentrations of chlorine used to purify water for drinking.

# *Giardia lamblia* (Giardiasis)

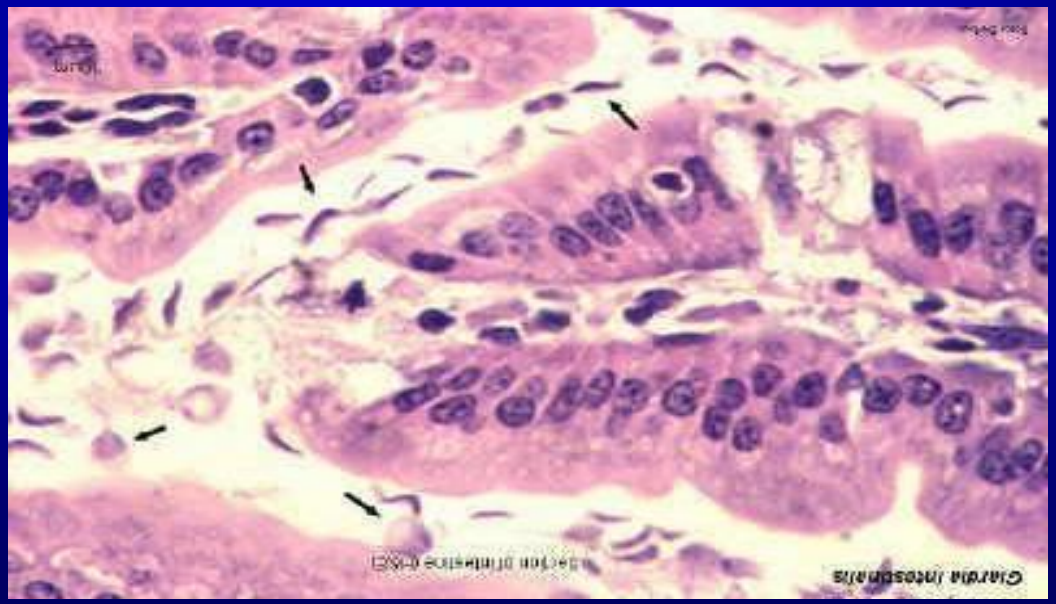
## B. Life Cycle:

After ingestion of mature cysts via contaminated water or food, the trophozoite emerges in the small intestine, rapidly multiplies, and attaches to the small intestinal villi. Mature infective cysts pass in feces and complete the cycle.



# C . Pathology

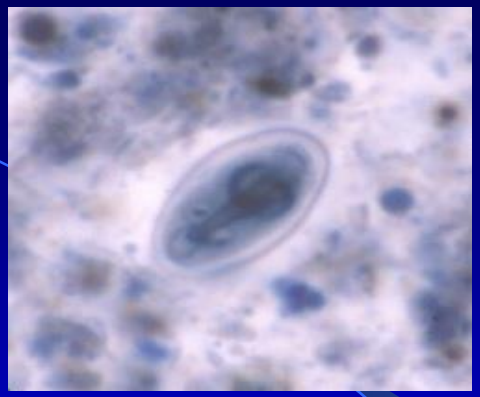
- The mechanisms of action are probably multifactorial and not yet fully elucidated Postulated mechanisms include damage to the endothelial brush border, enterotoxins, immunologic reactions, and altered gut motility and fluid hypersecretion via increased adenylate cyclase activity.
- Giardia infection can also lead to lactase deficiency as well as other enzyme deficiencies in the microvilli. This reduced digestion and absorption of solutes may lead to an osmotic diarrhea and could also explain the malabsorption syndromes.

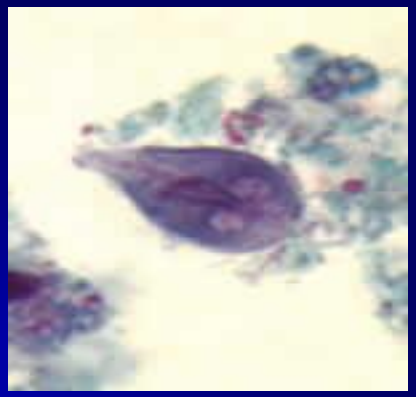


# D. Clinical manifestation

- Majority are asymptomatic.
- Children are more symptomatic than adult .
- The first signs of acute giardiasis include nausea, loss of appetite and an upper gastro-intestinal uneasiness.
- These signs are often followed or accompanied by a sudden onset of explosive, watery, foul-smelling diarrhea.
- Stools associated with Giardia infection are generally described as loose, bulky, frothy and/or greasy with the absence of blood or mucus, which may help distinguish giardiasis from other acute diarrheas.
- Other gastro-intestinal disturbances associated with giardiasis include: flatulence, bloating, anorexia, cramps & vomiting.
- May be self- limiting or produce severe protracted diarrhea & malabsorption .

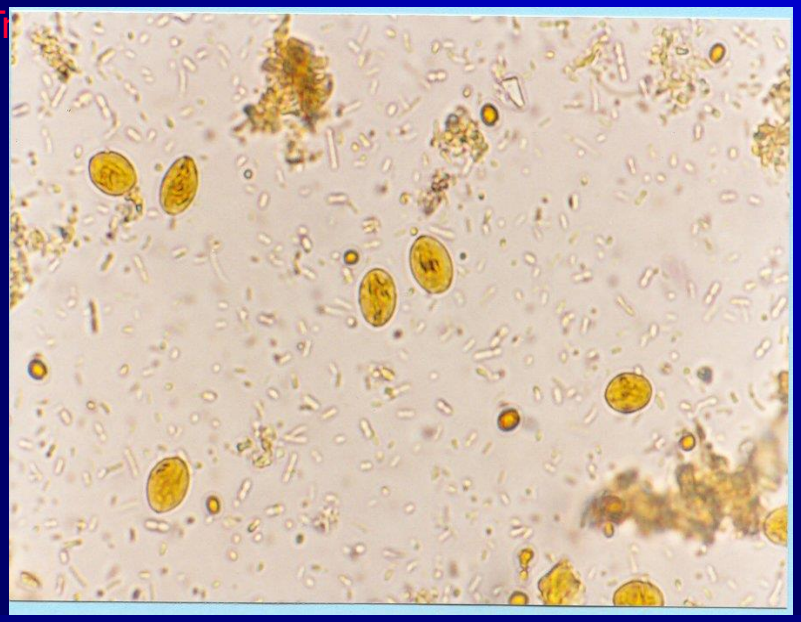
# E. Diagnosis:

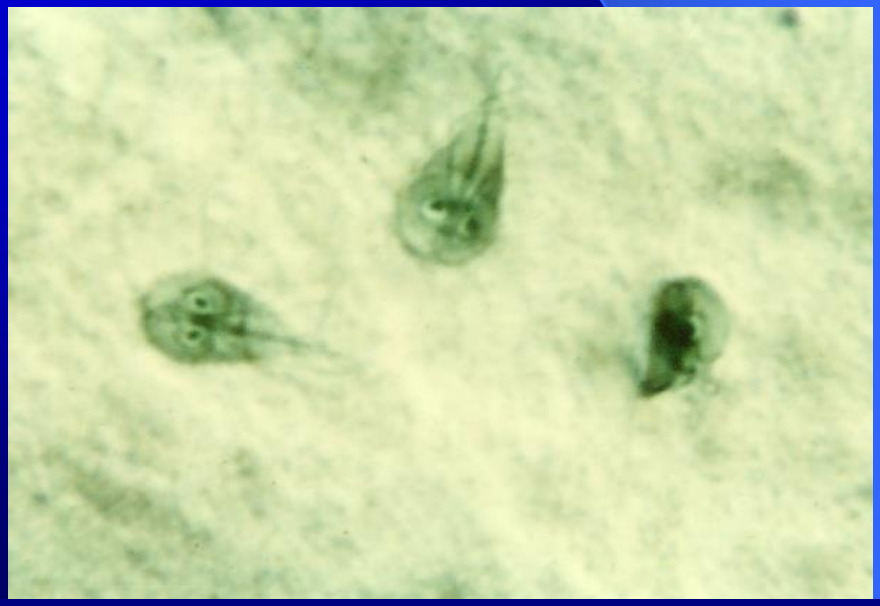




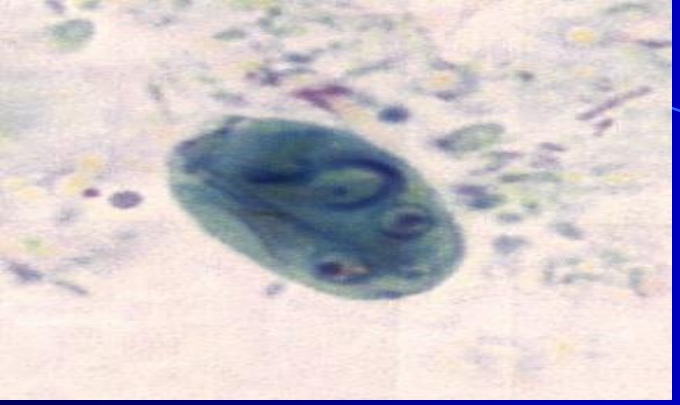
- **Stool examination**

  Trophozoites may be found in fresh, watery stools but disintegrate rapidly. Cysts are passed in soft and formed stools. 3 samples have sensitivity of up to 95% .

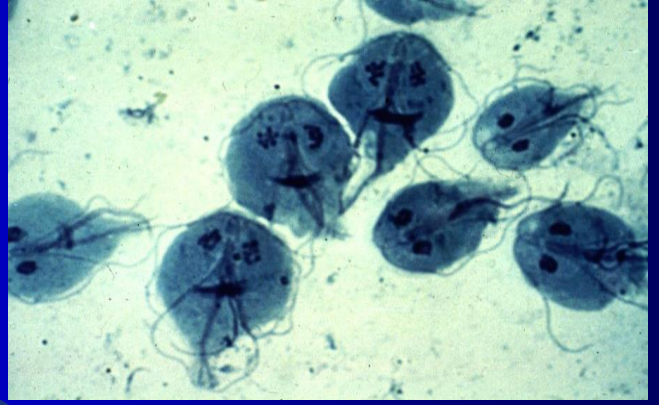




# Diagnosis cont.



**Cyst in stool**



**Trophozoites in duodenum**

- Stool antigen detection

  Several commercially available tests to detect Giardia antigen in the stool exist.. These tests have a sensitivity of 85-98% and a specificity of 90-100%.

- Examination of duodenal fluid or duodenal biopsy:-

  - To identify *Giardia lamblia* and other enteric pathogens.

  -To visualize changes in histologic features.

# F. Treatment

- Self limited.
- Asymptomatic - generally no treatment .
- Symptomatic – it should be treated .
- Drugs :

- Metronidazole , Tinidazole ,nitazoxanide.

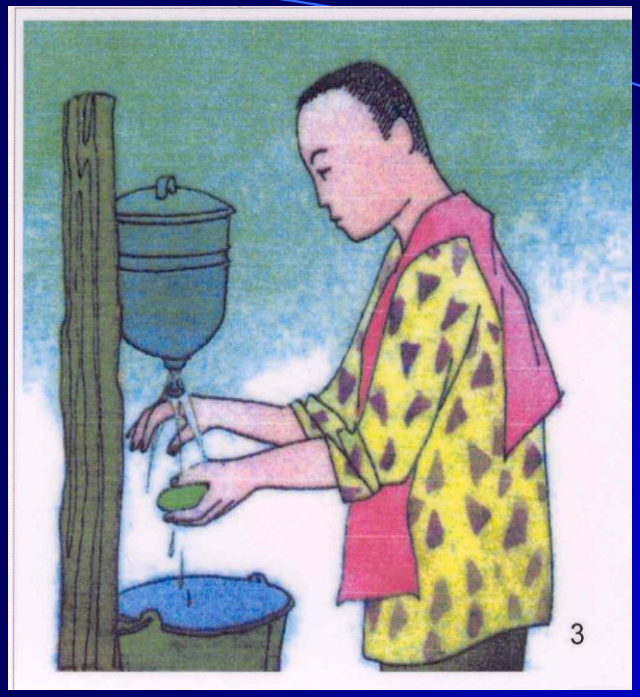
-Second line alternatives for the treatment of patients with giardiasis include furazolidone, albendazole, paromomycin, and quinacrine .

# Treatment

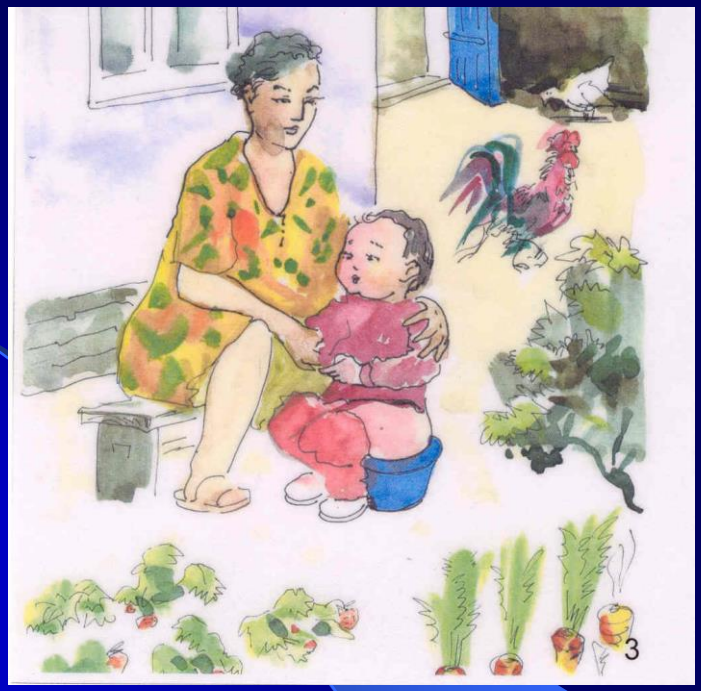
- Refractory cases : mepacrine,nitazoxanide,prolonged courses of tinidazole, or a combination of metronidazole and quinacrine.
- Failure to response – look for risk factors e. g. hypogammaglobulinemia .

# G . prevention

- Strict hand washing.
- Purification of drinking water.
- Avoid contaminated food .




Hand washing after defecation




Safe collection of child feaces


Boiling water for drinking and safe storage


Safe disposal of child feaces

# H. Prognosis

- The prognosis for patients with giardiasis is generally excellent. Most patients are asymptomatic, and most infections are self-limited. Giardiasis is not associated with mortality except in rare cases of extreme dehydration, primarily in infants or malnourished children.
- If the parasite persists in the stool, Reinfection is possible.
- Weight loss, disaccharidase deficiency, malabsorption, and growth retardation are possible complications
- *G intestinalis* has been implicated as the chief cause of growth retardation in infected children.

# *Cryptosporidium*

## *Epidemiology*

- **A worldwide cause of diarrhea**
- **Worldwide prevalence about 10% , children more than adult.**
- **Zoonosis, human and animal genotypes**
- **Transmission from animal to human , human to human , or contaminated water.**
- **Resistant to usual chlorination.**
- **Up to 20% of general population may be considered at higher risk**

# Etiology

Two species{1&2} mainly infect humans:

1. *Cryptosporidium hominis* which infects only humans.
2. *C parvum* which infects humans and animals.
3. *Cryptosporidium canis* infects dogs and humans.

# *Etiology - cont.*

*Cryptosporidium cause the infection by ingestion of oocyst→ 4 sporozoites→ invade enterocytes of small intestine , infection progress through 2 stages :*

1. *asexual → reinfection at luminal surface of epithelium .*
2. *sexual →oocyst →infect others or the same host .*

# Cryptosporidiosis: The Disease

- Serious disease in immunocompromised host and can also affect immunocompetent person .
- watery diarrhea , cramps , nausea , vomiting , anorexia , myalgia , weakness , headach , fever .
- Malabsorption , lactose intolerance & wt. loss may occure.
- In immunocompetent it is self-limiting lasting 2-30 days (usually<2weeks) .
- in immunocompromised may lead to chronic diarrhea malnutrition anorexia & wasting .
- Infectious dose in healthy humans is low: about 130 oocysts

# Diagnosis

- Cryptosporidium can be difficult. Most often, stool specimens are examined microscopically using different techniques:

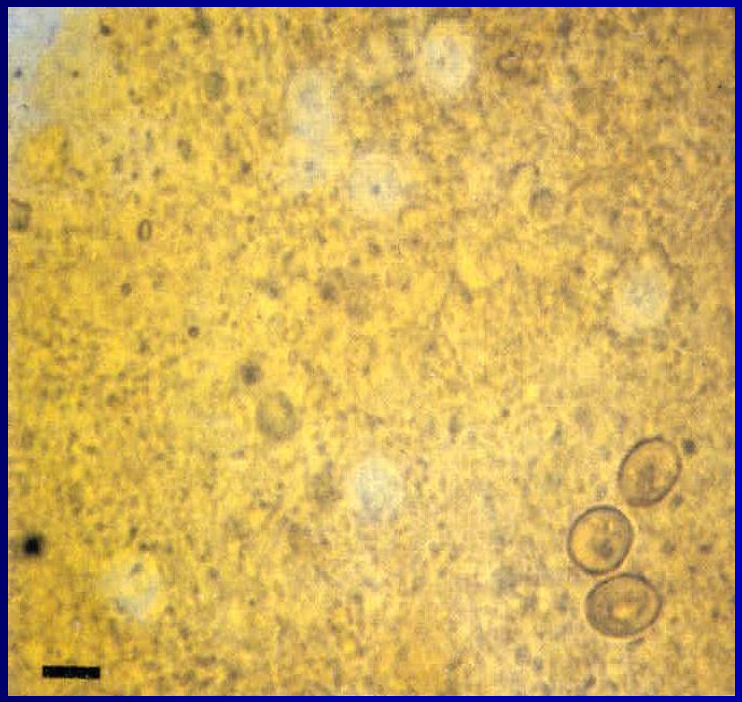
  - acid-fast staining for Oocys – 3 samples.

  - direct fluorescent antibody [DFA].

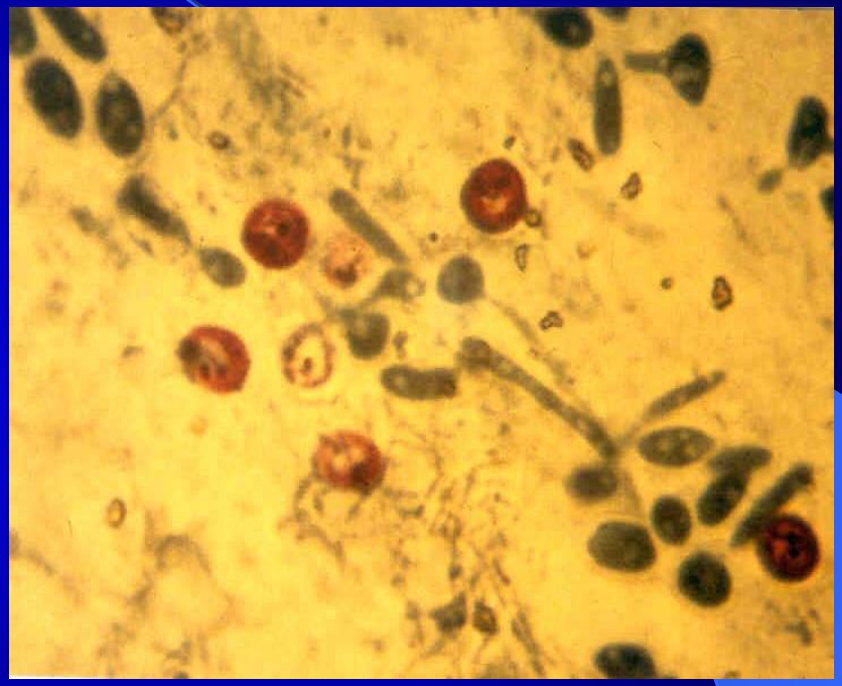
  -enzyme immunoassays for detection of *Cryptosporidium* species antigens.

- PCR .

# Cryptosporidium



Iodine stain of stool



Acid-fast stain of stool

***oocysts are rounded. Sporozoites are sometimes visible inside the oocysts.***

# treatment

- In immunocompetent it is self limiting , so no specific therapy .
- In immmunocomprised , No effective chemotherapy available
- Nitazoxamide shortens duration of diarrhea
- Combination therapy with paromomycin and azithromycin for 4 weeks followed by paromomycin monotherapy for 8 wk has been successfully used in adult patients with AIDS

# Helminthic disease

## Nematode (Roundworms)

- The most common helminthasis of humans .
- Common in tropical and subtropical climates , children > adult .
- Infection acquired by ingestion of mature eggs or larval penetration of skin .
- Adult warm live in intestinal tract and do not multiply in the lumen (except strongyloides stercoralis) .
- Eosinophilia often develop during larvae migration through host tissue .

# Enterobiasis (pin worm)

## etiology & epidemiology

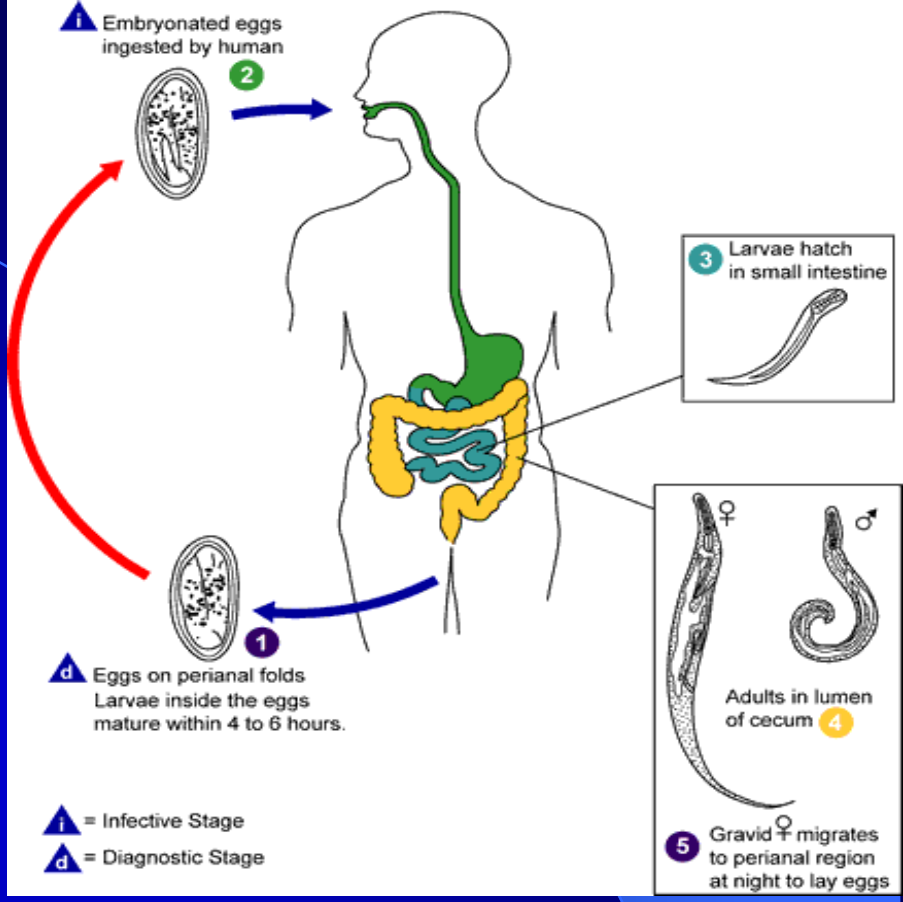
- Caused by Enterobias vermicularis .
- Worldwide, 30% of children are infected ,affect all ages (> children) , all socioeconomic levels
- Generally Harmless , ? More social than medical proplems .
- Humans are the only host .
- More in the ages 5-14 years , ↓ in adulthood

  (? Reduced exposure , ? Acquistion of immunity).

## *Enterobius vermicularis (pinworm)*

# *Life cycle & Pathogenesis*

*Infection occurs by ingestion of eggs which carried on fingernails , clothing , bedding or house dust , Eggs hatch in the stomach & larvae migrate to cecal region & mature to Adult warm (1cm-white in color) . The gravid female migrate by night to perianal region to deposit masses of Eggs , after 6 hours maturation period a single larva can be seen within each ovum , the larvae remain viable for 20 days. Oviposition induce perianal irritation → scratching → eggs carried under fingernails → mouth or environment .*



## *Symptoms*

- *Enterobiasis is relatively harmless and rarely produces serious lesions. The most common symptom is perianal pruritus, nocturnal perineal and vaginal irritation caused by the female migration. The itching results in insomnia and restlessness.*
- May present as enuresis or abdominal pain which may sometimes be severe and can mimic acute appendicitis.
- *Diagnosis: Diagnosis is made by finding the adult worm or eggs in the perianal area, particularly at night . adhesive cellophane tape (Scotch tape) is used to obtain eggs – pressed against the perianal region early in the morning .*

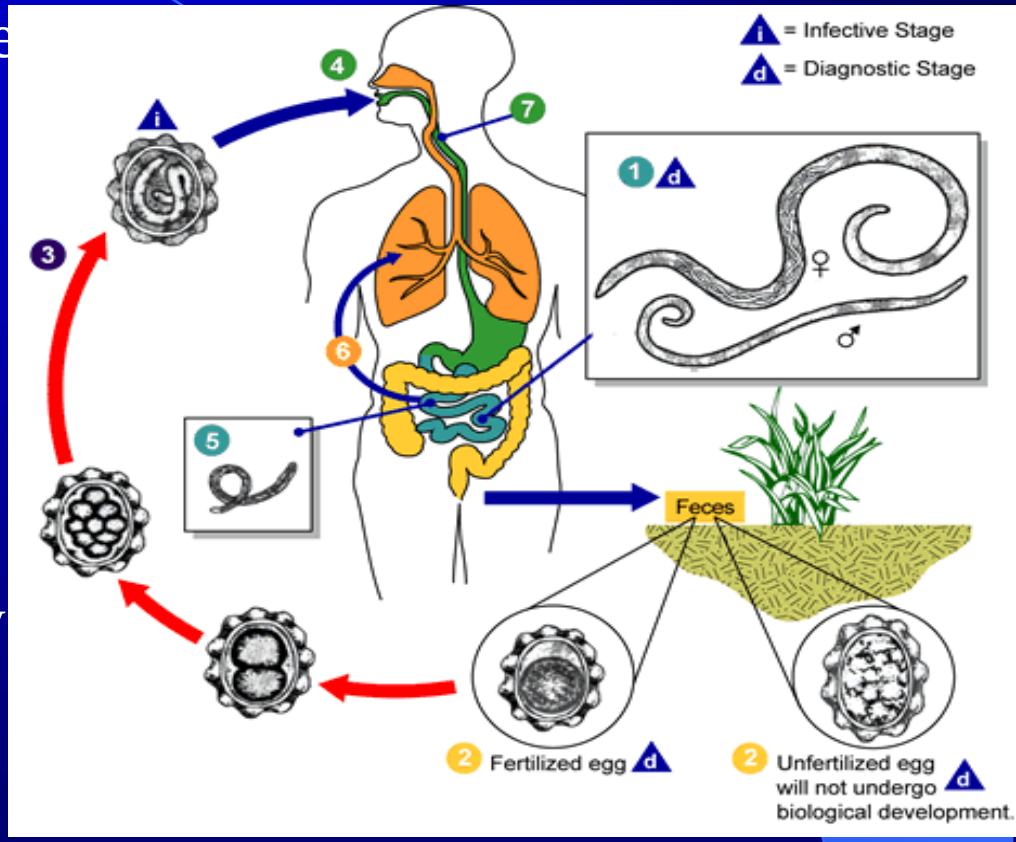
  *Eosinophilia is usually absent .*

# Treatment & Control

- All infected individual and their family members should be treated .
- Albendazole 400mg once orally , repeat the dose in 2 weeks .
- Mebendazole 100mg single dose orally repeat the dose in 2 weeks .
- Pyrantel pamoate 11mg/kg once repeat the dose in 2 weeks .
- In constant exposure you can repeat treatment every 3-4 months .

# Ascariasis

Ascaris lumbricoides is the most prevalent human helminthiasis , produce billion cases worldwide




## Etiology :

Eggs in the stool mature in 5-10 days → ingestion → Larvae → penetrate the intestinal wall → venous circulation → Lungs →break through pulmonary vessels into alveolar space → bronchial tree → trachea → reswallowed →small intestine →maturation , ♂ 15-25cm , ♀25-35cm , life for 1-2 years , producing 200,000 eggs/day .

# Clinical manifestations

- Disease occur in few infected people .
- Symptoms are relative to the worm burden.
- Most symptoms are due to physical presence of the worm..
- During larvae migration through the lungs Loffler-like syndrome may occur –seasonal pneumonitis- cough , blood stained sputum , eosinophilia & transient pulmonary infiltration .
- Warm in the bowel is associated with vague complaint such as abdominal Pain , distention & rarely obstruction .
- Migration to billiary tract result in pain , nausea , vomiting , fever & rarely jaundice .
- Steatorrhea &↓ vit. A absorption may occur .

# Diagnosis

Diagnosis is based on identification of eggs in the stool. (fertile or infertile) .

**Treatment and Prevention**:

-Albendazole 400mg orally , single dose .

-Mebendazole, 100 mg BD, for 3 days or 500mg once is effective.

-Pyrantel pamoate 11mg/kg once .

-Piperazine salts if there is obstruction by warms .

-Good hygiene is the best preventive measure.

# *Hookwarms*

## A. Epidemiology:

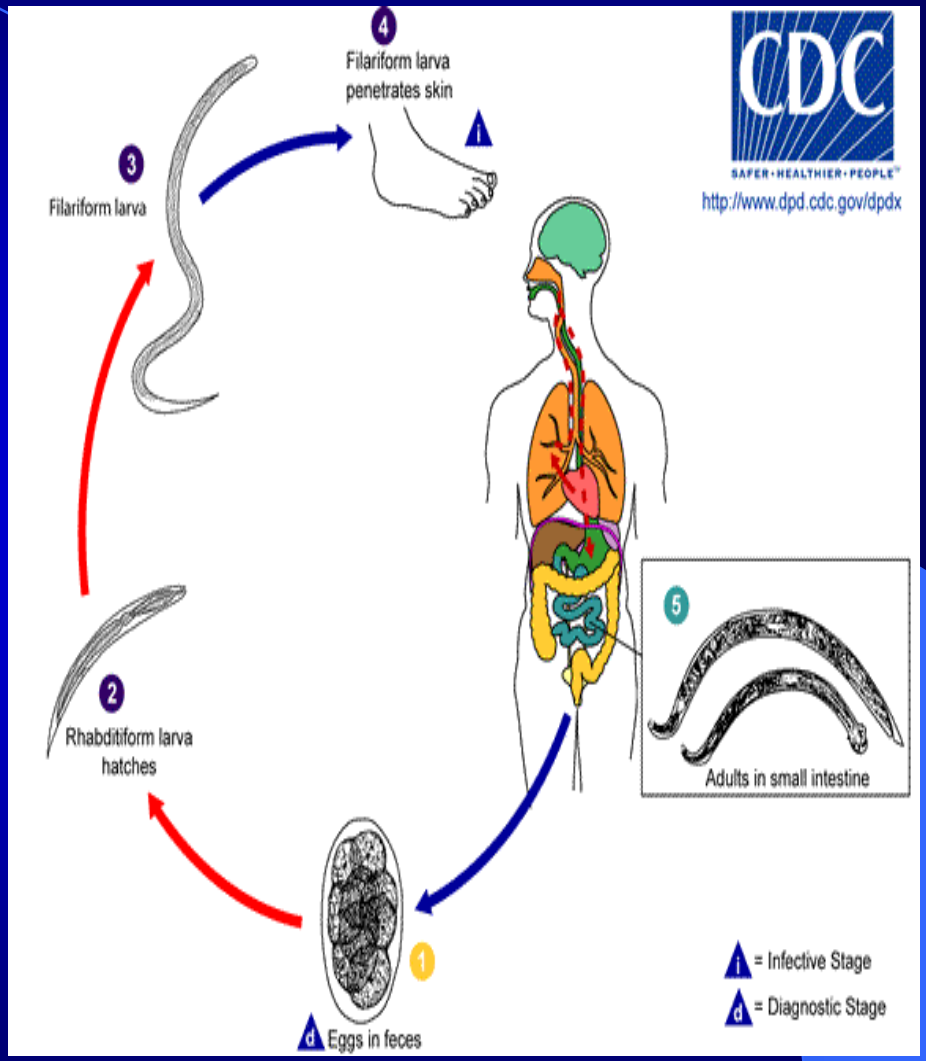
- Hookworms affect the intestine of more than 900 million people worldwide and cause daily blood loss of 7 million liters.
- 3 main species : ancylostoma duodenale , Necator americans & ancylostoma cerylenicum .
- *A.* duodenale (old world hookworm) is the dominant species in the Mediterranean region, North Africa, southern Europe and northern Asia .
- *N. americanes* (new world hookworm) is most common in the Americas, central and southern Africa, southern Asia, Indonesia, Australia and Pacific Islands .

# B. Life Cycle:

Eggs are passed in the stool , larvae hatch in 1 to 2 days. The released larvae grow in the feces and/or the soil , and they become filariform (third-stage) larvae that are infective .

On contact with the human host, the larvae penetrate the skin and are carried through the veins to the heart and then to the lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed

The larvae reach the small intestine, where they reside and mature into adults.



# C. Pathology:

- Hook worms bite and suck on the intestinal wall, which can cause bleeding and necrosis with diarrhea & bloody stool
- In severe infections lead to iron deficiency anemia.
- Hook worms Toxins may lead to physical and mental retardation.

# D. Diagnosis:

- **Microscopic identification of eggs in the stool** is the most common method for diagnosing hookworm infection.

# Treatment

- Iron therapy or blood transfusion .
  ( if needed )
- Albendazole , or
- Mebendazole , or
- Pyrantel pamoate .
  ( doses as mentioned before )


Thank you
7 4 2006