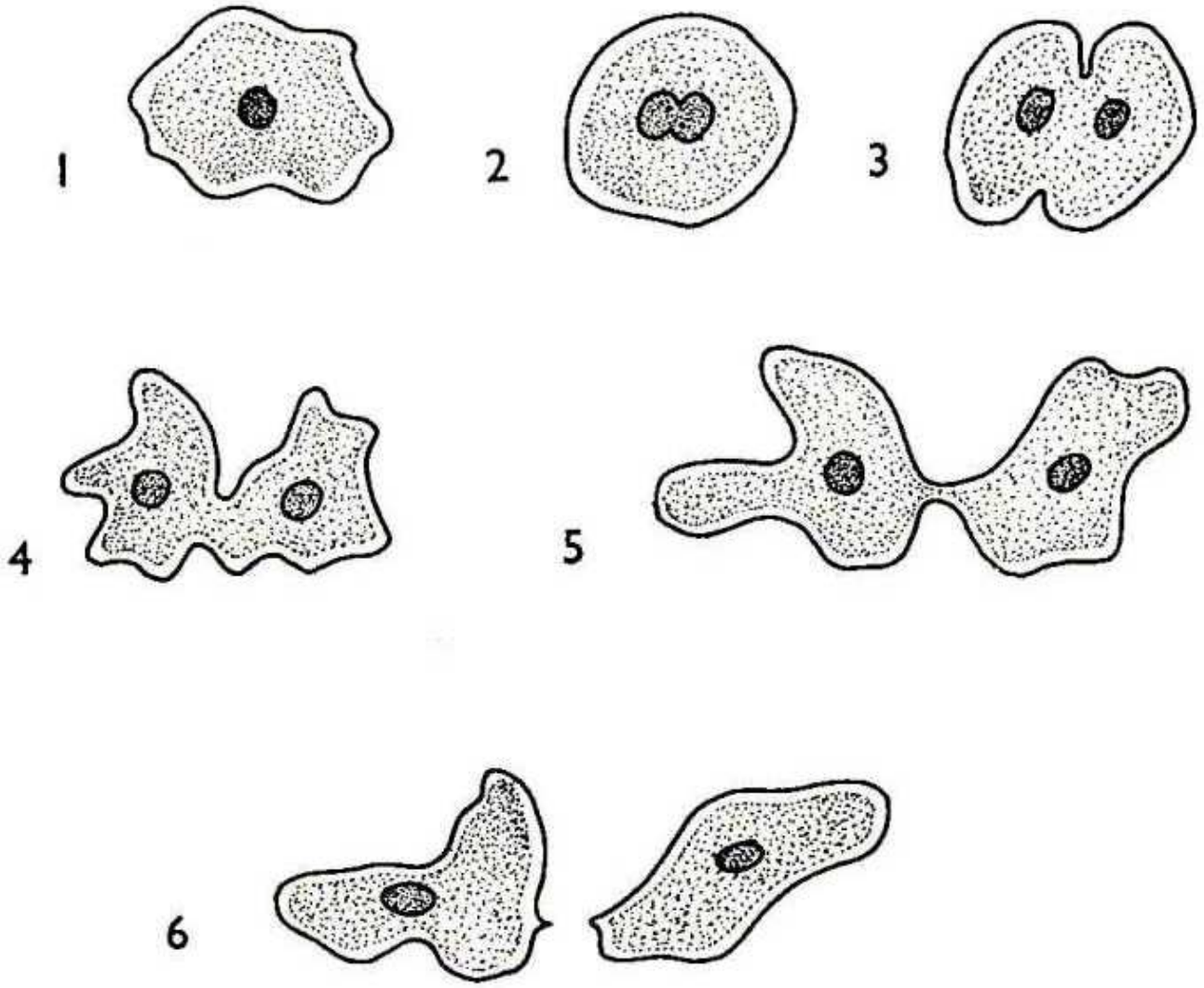
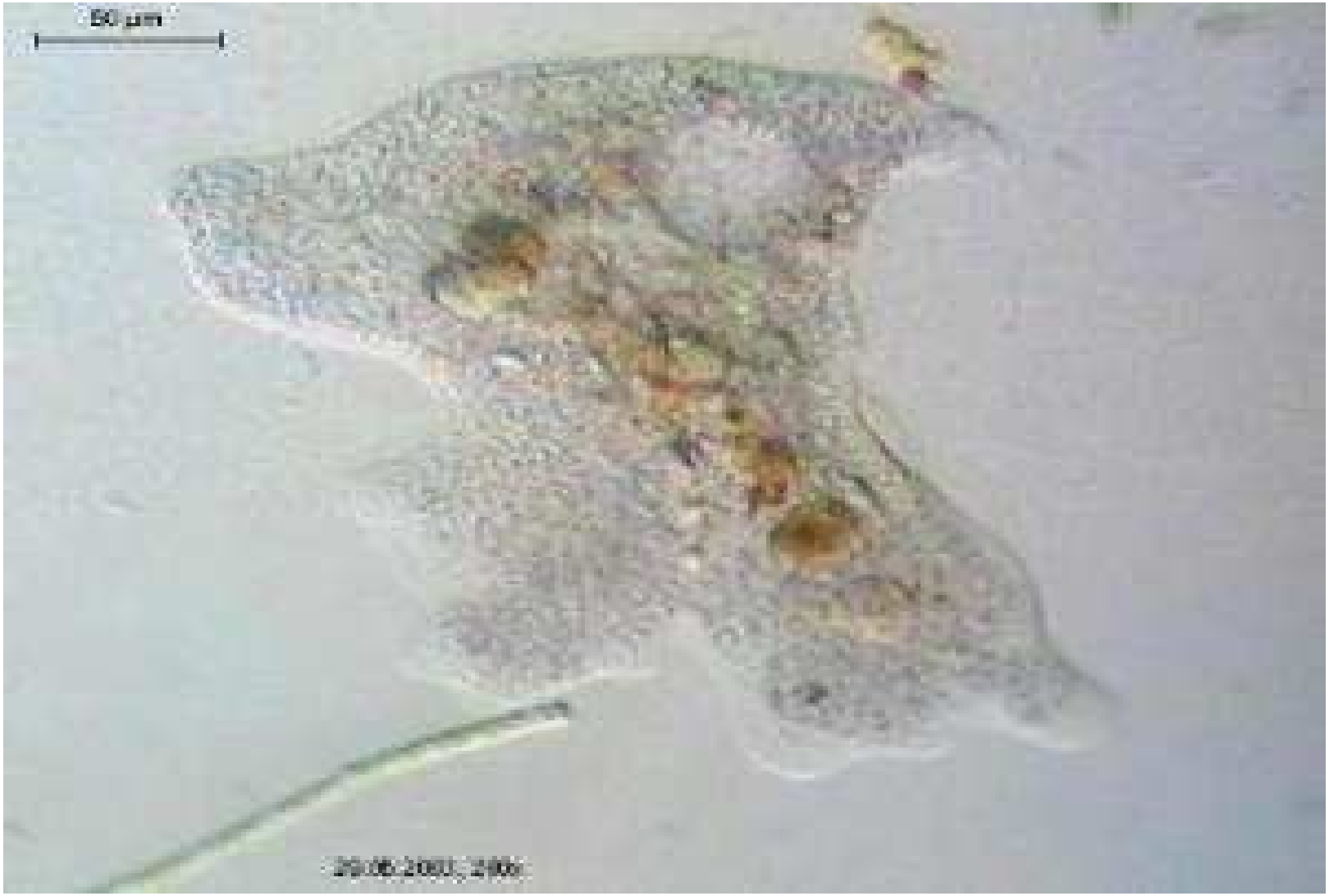
Intestinal Amoeba

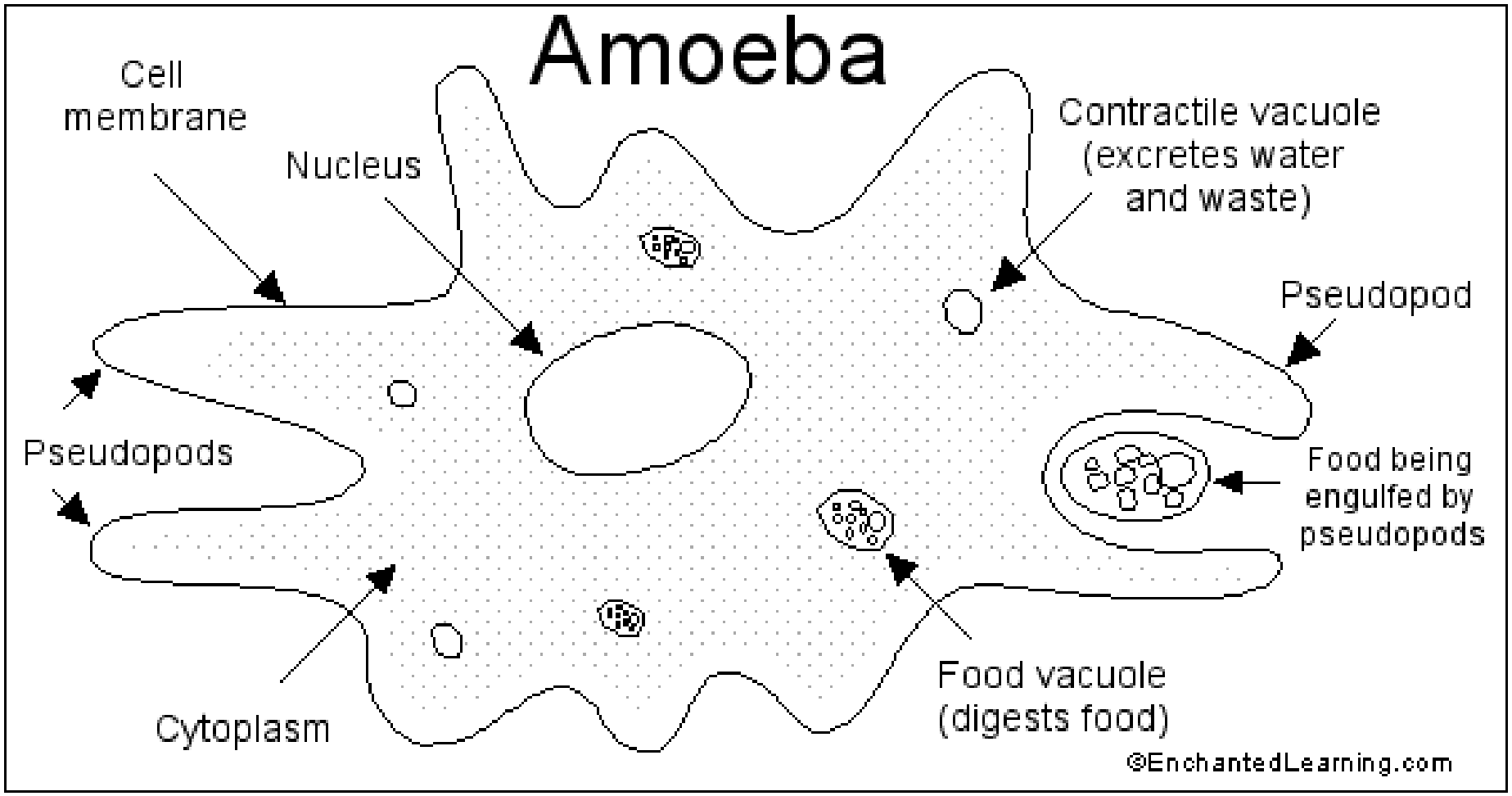
- ***Entamoeba histolytica***
- ***Entamoeba dispar***
- ***Entamoeba coli***
- ***Endolimax nana***
- ***Iodamoeba bütschlii***

Binary fission





Entamoeba histolytica - trophozoite


Amoeba
Cell membrane
Nucleus
Contractile vacuole (excretes water and waste)
Pseudopod
Pseudopods
Food being engulfed by pseudopods
Cytoplasm
Food vacuole (digests food)
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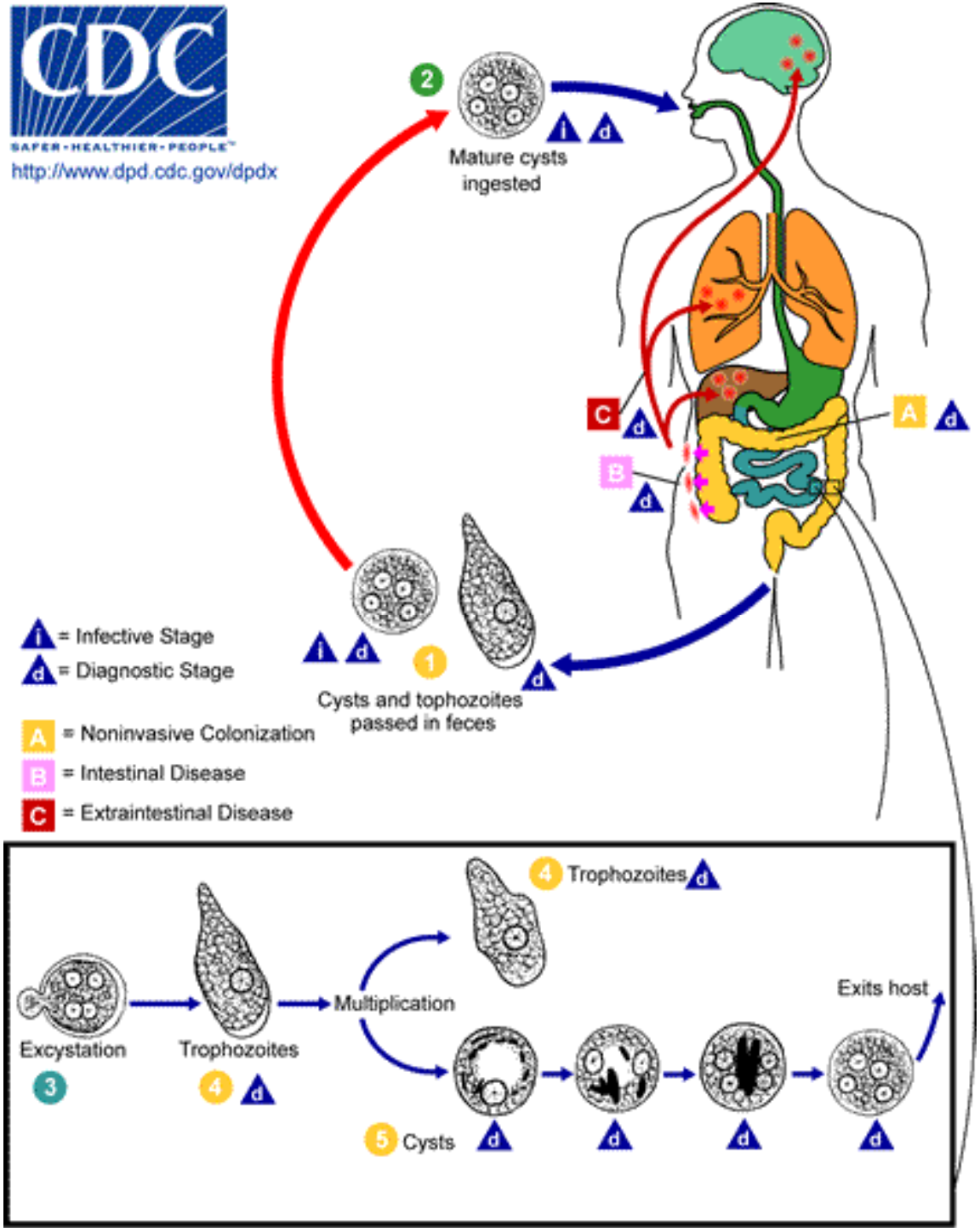
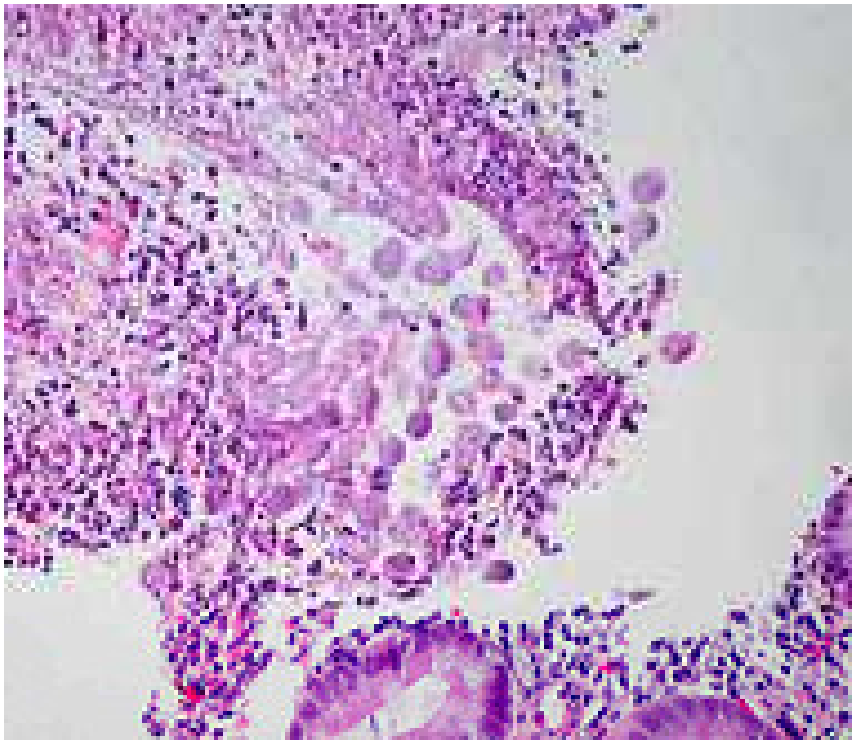
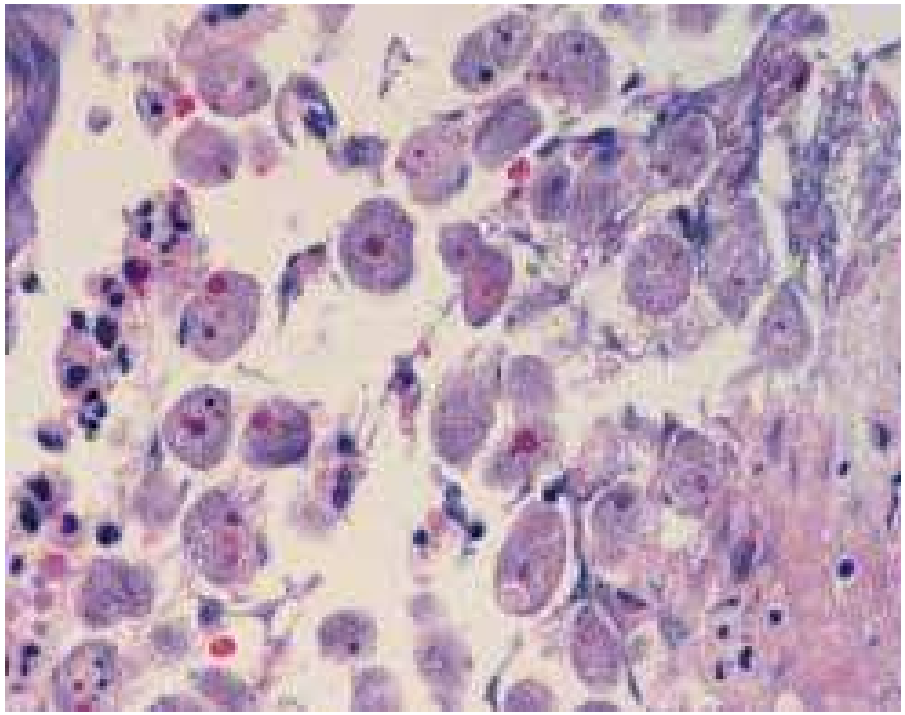


E. histolytica

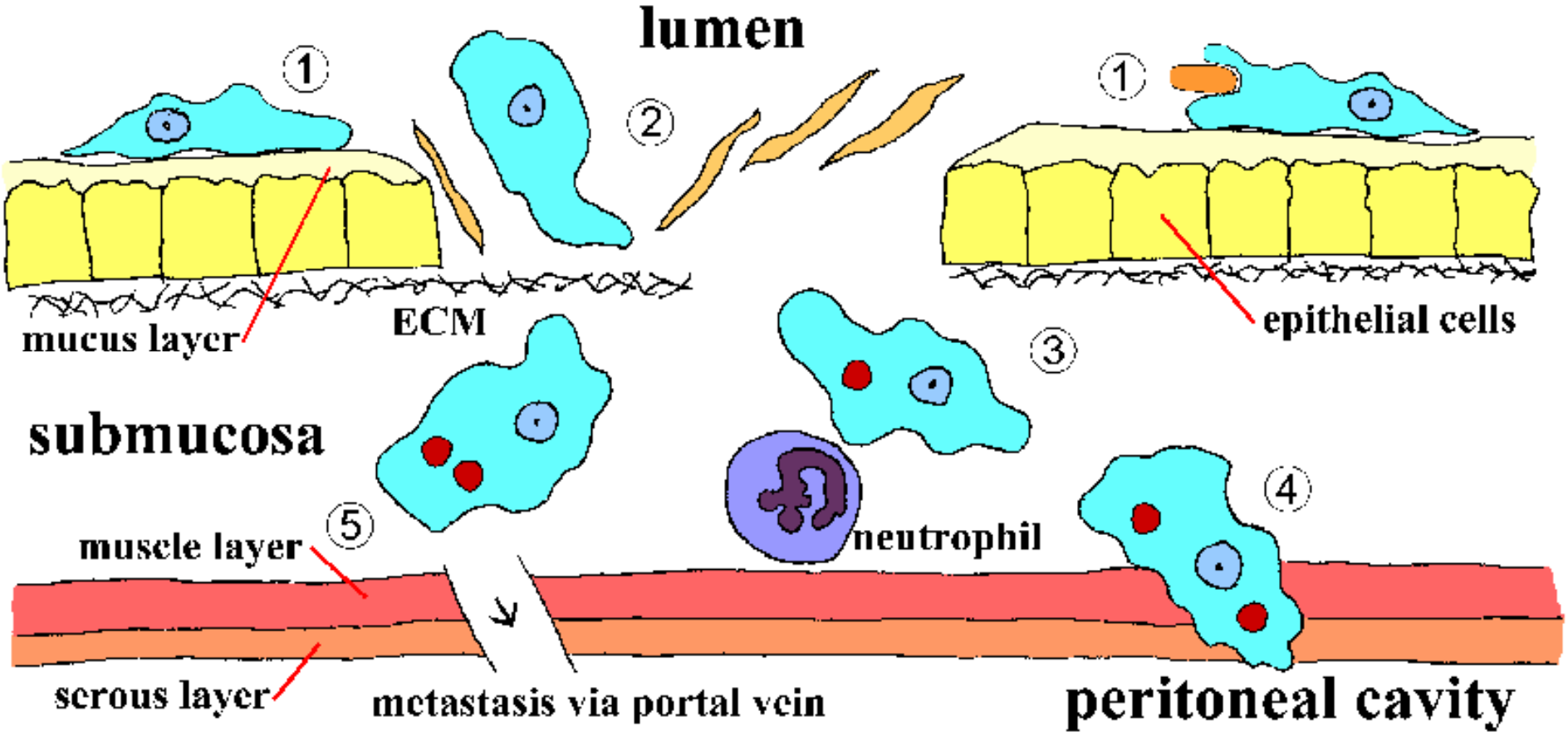
cycle





Entomoeba histolytica – Amoebic colitis

Amebiasis Pathogenesis



- **Step 1. Trophozoites adhere to the mucus layer. This adherence per se probably does not contribute to pathogenesis and is simply a mechanism for the ameba to crawl along the substratum. Depletion of the mucus barrier allows the trophozoite to come in contact with epithelial cells.**
- **Step 2. Epithelial cells are killed in a contact dependent manner leading to a disruption of the intestinal mucosa**
- **Step 3. The trophozoites will continue to kill host cells in the submucosa and further disrupt the tissue as they go.**
- **Step 4. Disruption of the intestinal wall or (Step 5) metastasis via the circulatory system are also possible.**

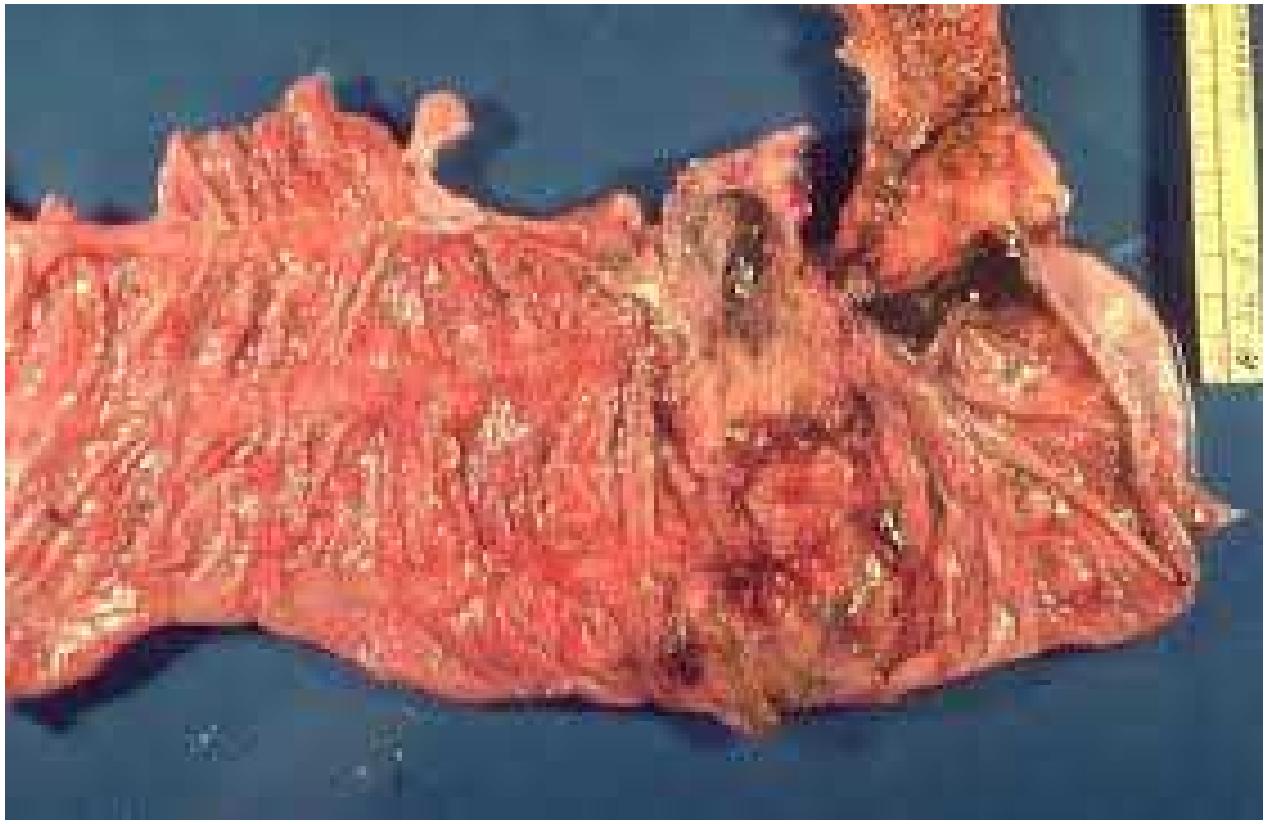
Adherence, cytotoxicity, and disruption of the tissues are important factors in the pathogenesis of *E. histolytica*.

Pathology/Clinical features

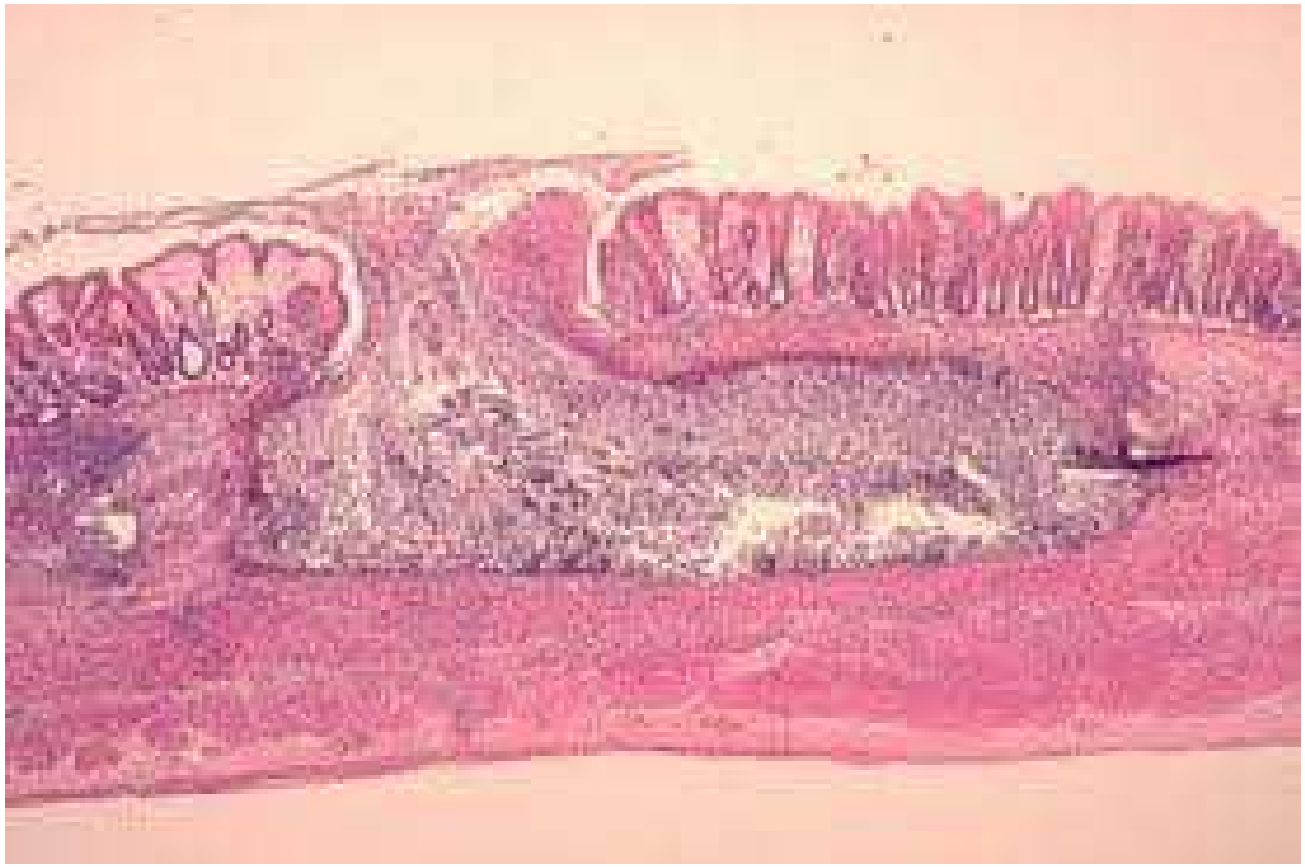
The clinical spectrum of *E. histolytica* infection ranges from asymptomatic carrier state or acute colitis, to fulminant colitis with perforation.

- **Asymptomatic infection**
- **Intestinal amoebiasis**
- **Amoebic liver abscess**
- **Peritoneal amoebiasis**
- **Pericardial amoebiasis**
- **Cerebral amoebiasis**
- **Genitourinary amoebiasis**
- **Cutaneous amoebiasis**

Gross pathology of intestinal amebiasis showing extensive ulceration



Histopathology of a typical flask-shaped ulcer of intestinal amebiasis (from CDC)



Immunology (a)

- **First contact of trophozoite with intestinal epithelial cells stimulates them to produce interleukin (IL)8. Neutrophils are rapidly recruited. Cell infiltration around invading amoeba leads to tissue necrosis**
- **Invasive infection with *E. histolytica* produces a marked immune response which results in the development of protective immunity though incomplete**

Immunology (b)

- **Recurrence of amoebic colitis & abscess is unusual**
- **Patient with AIDS do not appear, surprisingly, to be more susceptible to severe infection though asymptomatic carriage is common**
- **Intestinal invasion by *E. histolytica* results in antibody response. Circulation of antibodies as early as 1 week after onset**
- **All immunoglobulin classes are involved (IgG2 are predominant) but those can be degraded by proteinases of the amoeba and limit the effectiveness of humoral response**

Management/treatment (a)

Asymptomatic intestinal carrier:

- **1st choice DILOXANIDE FUROATE**
- **2nd choice PAROMOMYCIN**

Intestinal infection:

- **1st choice METRONIDAZOLE followed by DILOXANIDE FUROATE**
- **Or TINIDAZOLE followed by DILOXANIDE FUROATE**
- **2nd choice PAROMOMYCIN**

Management/treatment (b)

Amoebic liver abscess:

1st choice METRONIDAZOLE followed by DILOXANIDE FUROATE

Or TINIDAZOLE followed by DILOXANIDE FUROATE

2nd choice DEHYDROEMETINE followed by DILOXANIDE FUROATE