General Microbiology
Lecture 18
(Protozoa)
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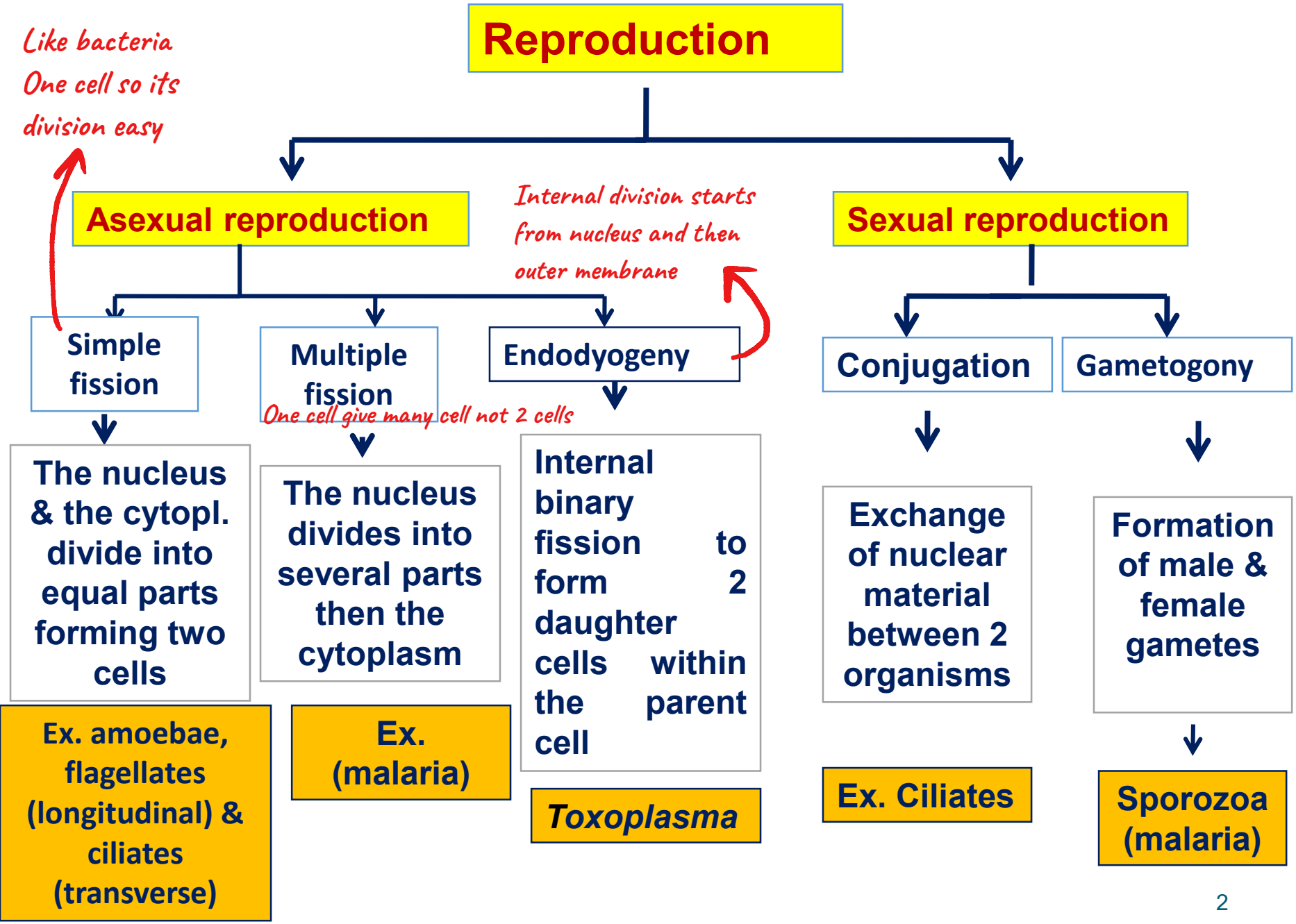

Reproduction
Like bacteria
One cell so its division easy
Asexual reproduction
Internal division starts from nucleus and then outer membrane
Sexual reproduction
Simple fission
Multiple fission
Endodyogeny
Conjugation
Gametogony
One cell give many cell not 2 cells
The nucleus & the cytopl. divide into equal parts forming two cells
The nucleus divides into several parts then the cytoplasm
Internal binary fission to form 2 daughter cells within the parent cell
Exchange of nuclear material between 2 organisms
Formation of male & female gametes
Ex. amoebae, flagellates (longitudinal) & ciliates (transverse)
Ex. (malaria)
Toxoplasma
Ex. Ciliates
Sporozoa (malaria)

Reproduction in amoebae

Parent cells

Binary fission

Karyokinesis *Nucleus division*

Cytokinesis *Cytoplasm division*

Two daughter cells

Multiple fission

مقاومة البيئة الخارجية

Cyst

بتنقسم بالداخل

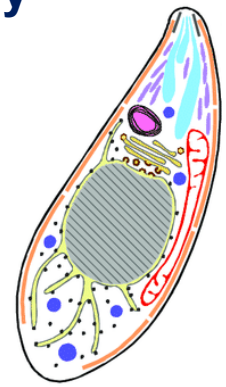
Divides many times

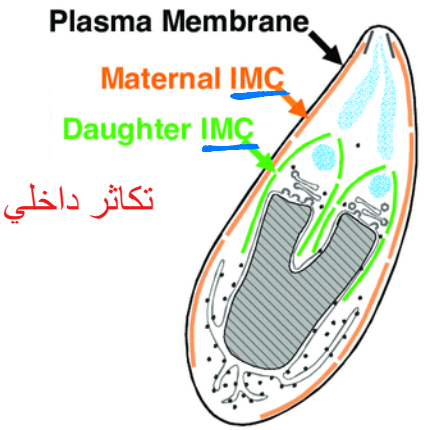
Rupture of cyst wall which produce mutiple no. Of protozoa

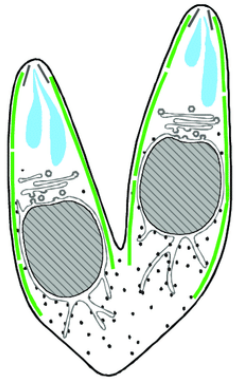
بتنقسم في بيئة محمية

Many daughter cells

Endodyogeny







inner membrane complex (IMC)

Class: Rhizopoda (Amoebae)

1- Amoebae of large intestine

- *Entamoeba histolytica* (The only pathogenic)
- *Entamoeba coli.*

2- Amoeba of buccal cavity

Entamoeba gingivalis

3- Free living amoebae

- Pathogenic free living amoebae → *Naegleria fowleri* & *Acanthamoeba*
- Non pathogenic free living amoebae → Coprozoic amoebae

✍**N.B.** Other species of amoebae are commensals in the large intestine such as ***E. hartmani*** & ***E. dispar*** & are morphologically similar to ***E. histolytica***. So **PCR** is required to differentiate bet. amoebae species.

NB. Someone we take from him stool sample we may see trophozoid (active form of amoebae)

But it is not ..... to be E.histolytica ==> PCR

Usually we give anti parasitic therapy (antiprotozoal therapy) بنعالج مباشرة

Histolytica = pathogenic = penetrate intestinal wall and to circulation and habitat in liver / brain ==> amoebic maninigoecephalitis

Entamoeba histolytica

- **Geographical distribution:** Worldwide especially in the temperate zone and more common in areas with poor sanitary conditions. *Malaria / Ameoba*
- **Habitat:** Large intestine (caecum, colonic flexures and sigmoidorectal region).
- **D.H:** Man
- **R.H:** Dogs, pigs, rats and monkeys.
- **Disease:** Amoebiasis or amoebic dysentery

الزحار الاميبي

بتأثر جدار الامعاء و بكون في دم مع البراز و حرارة و ألم

Entamoeba histolytica

Morphological characters

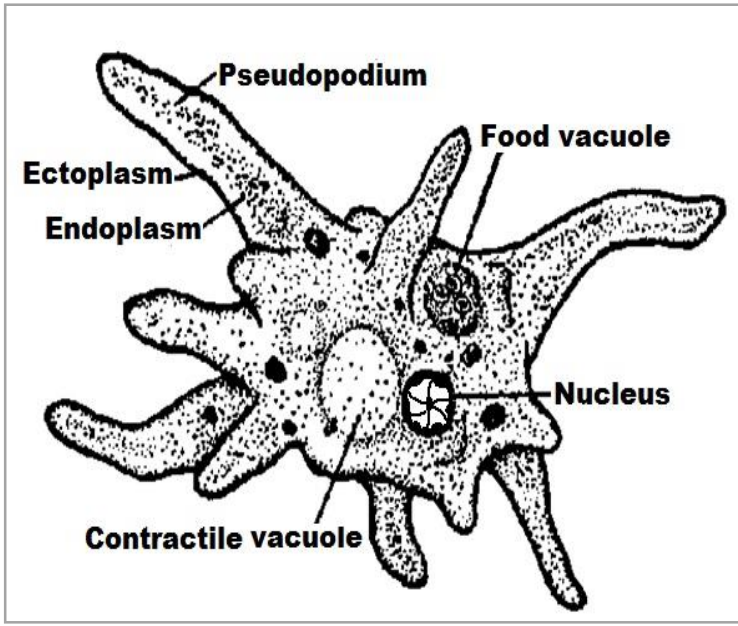
1- Trophozoite stage (Vegetative form or tissue form):

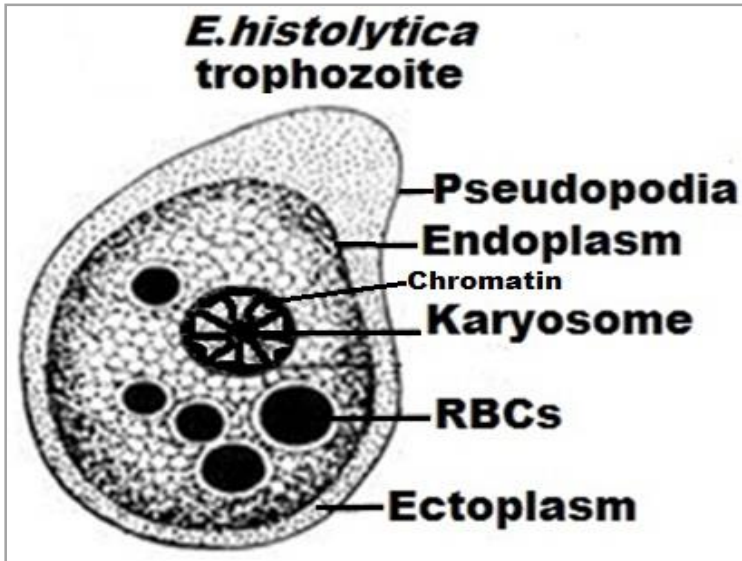
-Size: 20µm.

-Shape: Irregular. لانها اميبا بتطلع اقدام كاذبة

-Cytoplasm: Differentiated into ectoplasm and endoplasm.

- **Ectoplasm (Outer):** Clear with a single finger like pseudopodia





2- Cyst stage (Luminal form):

اذا كانت في بيئة غير مريحة خصوصا في نهاية الامعاء الغليظة ①
و يتحول الstool من soft الى hard
مباشرة بتحس انه البيئة غير مناسبة و مباشرة بتتحول إلى cyst

في الامعاء الدقيقة مبسوطة لانه stool is semiliquid لسا ما صار
absorption لكل water and nutrient

(a) Immature cyst (Uninucleate cyst and Binucleate cyst):

- Uninucleate cyst (one nucleus)
- Binucleate cyst (2 nucleus)

Infective stage

b) Mature cyst (Quadrinucleate cyst) (I.S):

The infective stage of amoeba is quadrinucleate cyst

هي الي بتقدر تدخل الجسم و تتحول الى *trophozoite*

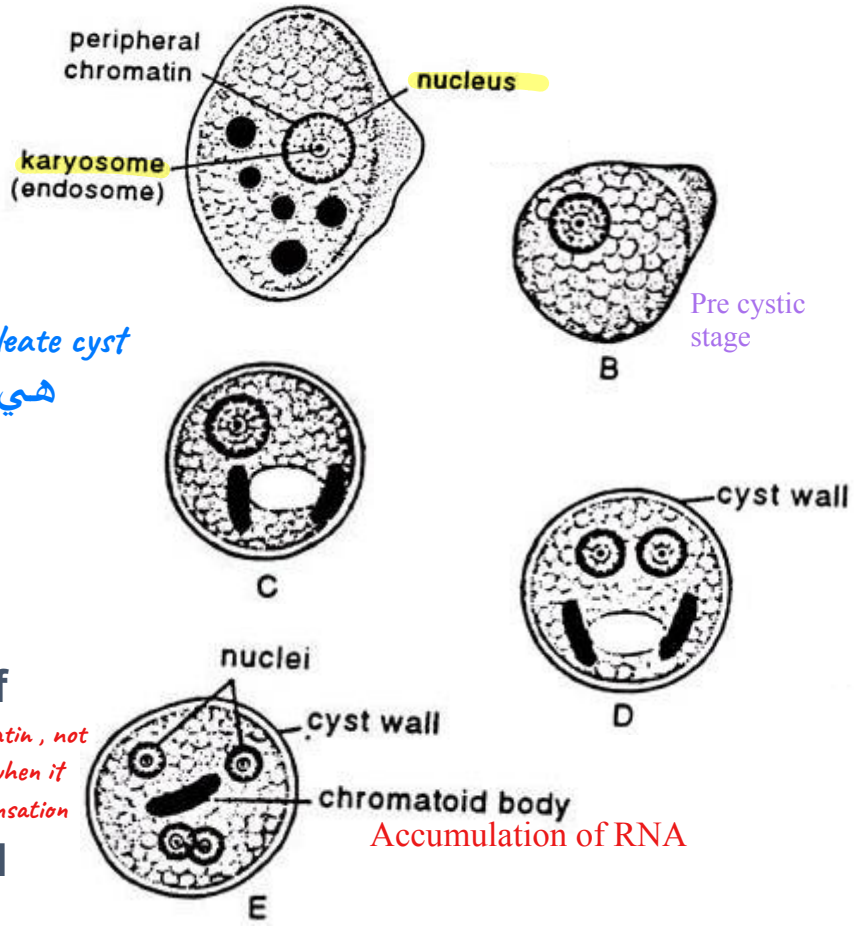
-Size: 15 µm.

-Shape: Rounded with thick cyst wall.

-Contents:

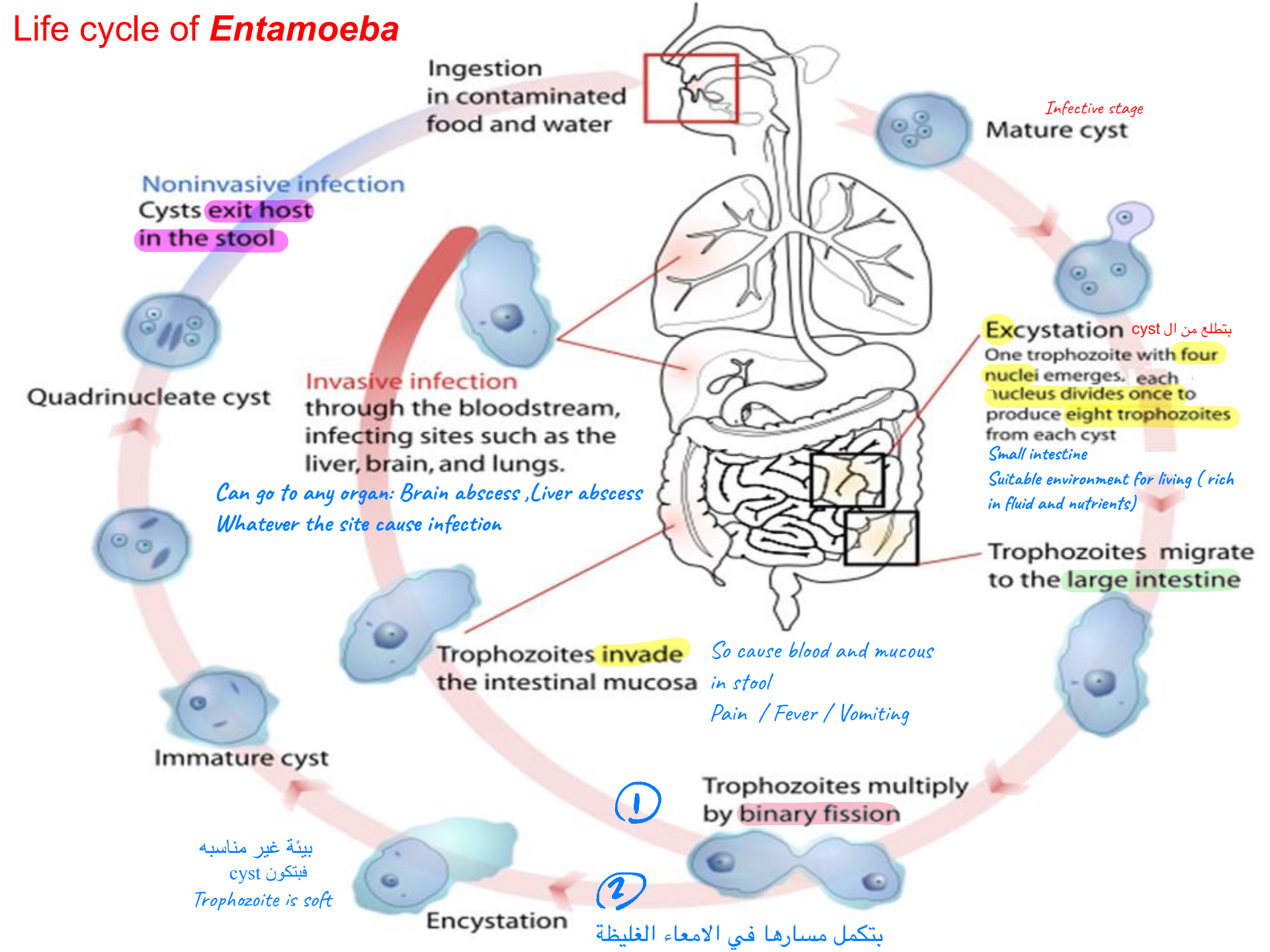
- 4 nuclei similar to the nucleus of trophozoite but smaller in size.
- Glycogen vacuoles and chromatoid bodies (stored food).

As it have DNA , chromatin , not condensed , active but when it became cyst DNA condensation



Entamoeba histolytica. A. Trophozoite stage. B. Precystic stage. C-E. Cysts, C. Uninucleate. D. Binucleate. E. Quadrinucleate stage

Life cycle of ***Entamoeba***



Mode of infection

parasite سهل تنقل اي

1- Contaminated foods (ex. green vegetables) or drinks or hands with human stool containing mature cyst.

2- Handling food by infected food handlers as cookers and waiters.

3- Flies and cockroaches that carry the cysts from faeces to exposed food.

Stool in external environment + flies... = spread through their foot to exposed food

4- Autoinfection (faeco-oral or hand to mouth infection).

From hands

5- Homosexual transmission.

Pathogenesis

Resistance of the host

Depends on

1-Host immunity.

2-Presence of debilitating diseases.

•Infection is severe in young children, pregnant women, elderly and immunodeficient patients.

المقام ضعيف حسب المعادلة لذلك التأثير كبير

Virulence of the parasite

Depend on

-Type of the strain.

-Invasiveness .

-Number of the amoebas.

Infection dose

GIT condition

Invasion ↑

-By carbohydrate diet.

-Injury of the mucosa (chemical).

-Stasis.

Blockage

Cancer

تضيق او تليف ← بقلل حركة الstool

مهم في الاكل الالياف لانه بتسرع من عملية التخلص من Stool

لانه مش صحي يضل لفترة طويلة

indirect contact with mucosa as there is a lot of antigens

With heavy infection and lowering of host immunity

Loos of surface continuity

The trophozoites of *E. histolytica* invade the mucosa and submucosa of the large intestine by secreting lytic enzymes ➲ amoebic ulcers

The ulcer is flask- shaped with deeply damaged edges containing cytolyzed cells, mucus and trophozoites.

Inter intestinal wall
4 mm diameter
When reach submucosa
It moves right and left
By lyric enzyme

The most common sites of amoebic ulcers are caecum, colonic flexures and sigmoidorectal regions due to decrease peristalsis & slow colonic flow at these sites that help invasion.

If it is fast It can't bind and cause infection

Clinical pictures

I) Intestinal amoebiasis

1-Asymptomatic infection

Most common and trophozoites remain in the intestinal lumen feeding on nutrients as a commensal without tissue invasion (Asymptomatic patient known as a healthy carrier and cyst passers)

2-Symptomatic infection

Damage of intestines
Bleeding

a) Acute amoebic dysentery

Presented with fever, abdominal pain, tenderness, tenesmus (difficult defecation) and frequent motions of loose stool containing mucus, blood and trophozoites.

b) Chronic infection

-Occurs if acute dysentery is not properly treated.
-With low grade fever, recurrent episodes of diarrhea alternates with constipation.
- Only cysts are found in stool.

3-Complications

- Haemorrhage due to erosion of large blood vessels.
- Intestinal perforation ➲ peritonitis.
- Appendicitis.
- Amoeboma (Amoebic granuloma) around the ulcer ➲ stricture of affected area.

تضيقات

Damage and stimulation of nerves

بسبب الالم و التقلصات في جدار الامعاء بسبب

Parasite present But the patient doesn't follow the treatment course / duration of treatment Some is treated but some goes to chronic (still in intestine)

فكل فترة بصير انفجار للاعراض و بتهدأ

ثقب في جدار الامعاء

Due to ulcer

Fibrosis (stricture) if it was large (stenosis of intestinal wall) and surgical treatment is needed

Or Strictures after perforation

II) Extra-intestinal amoebiasis

Due to invasion of the blood vessels by the trophozoites in the intestinal ulcer ➲ reach the blood ➲to spread to different organs as:

→ **Liver** →

-Amoebic liver abscess or diffuse amoebic hepatitis.
-Affect commonly right lobe either due to spread via portal vein or extension from perforating ulcer in right colonic flexure.
-CP: include fever, hepatomegaly and pain in right hypochondrium.

الاعراض معتمدة على العضو الي راحت إله

→ **Lung** →

•Lung abscess ➲ pneumonitis with chest pain, cough, fever.
•Amoebic lung abscess usually occur in the lower part of the right lung due to direct spread from the liver lesions through the diaphragm or very rarely trophozoites may reach the lung via blood.

→ **Brain** → Brain abscess ➲ encephalitis (fatal).

→ **Skin** → **Cutaneous amoebiasis due to either extension of acute amoebic colitis to the perianal region or through rupture on the abdominal wall from hepatic, colonic or appendicular lesions.**

Laboratory diagnosis

I)Intestinal amoebiasis

a) Direct

بشوف الطفيلي نفسه

- **Macroscopic:** Offensive loose stool mixed with mucus and blood.
- **Microscopic:**

1-Stool examination: Reveals either trophozoites (in loose stool) or cysts (in formed stool) by direct smear, iodine stained & culture.

2-Sigmoidoscopy: To see the ulcer or the trophozoites in aspirate or biopsy of the ulcer.

3-X-ray after barium enema: to see the ulcer, deformities or stricture.

By naked eye

Solid
Liquid
Blood
Mucus

Damage

b) Indirect

AB
Antigen
Effect

-Serological tests: CFT, IHAT, IFAT, ELISA and GDPT (gel-diffusion precipitin test).

Contact

These serological tests are positive only in invasive intestinal amoebiasis but negative in asymptomatic carriers.

damage and invasive the intestine
There will be contact with immune system and AB will present in circulation so we take blood sample

Ag in stool

II)Extra- intestinal amoebiasis

According to the organ affected

a) Direct

1- X- ray:

In liver ➲ space occupying lesion.

In lung ➲pleuritis with elevation of the diaphragm

2- Ultrasonography, CT scan& MIR:

For liver abscess.

3- Aspiration of abscess content:

For liver abscess to detect trophozoites.

بدخل needle
ويسحب من الabscess و مباشرة على المختبر

b) Indirect

1- Serological tests: As intestinal amoebiasis. They are positive and can persist for years.

2- Molecular by PCR.

3- Blood examination: Leucocytosis.

4- Liver function tests: Increased in amoebic liver abscess.

Treatment

1) Asymptomatic intestinal carrier

↓

Luminal amoebicides

↓

Paromomycin or

Diloxanide furoate

2) Intestinal amoebiasis

↓

Tissue & luminal amoebicides

↓

Metronidazol (Flagyl) is the drug of choice +

Paromomycin or

Diloxanide furoate

In poising we give usually drugs that cover bacteria and parasite

3)Extra-intestinal amoebiasis

↓

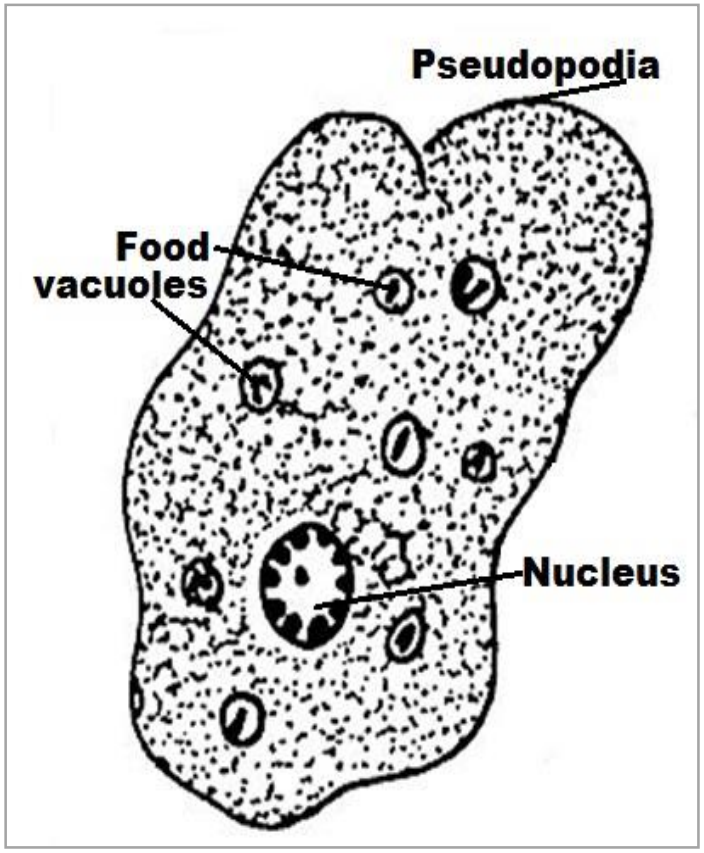
Tissue & luminal amoebicides

↓

Metronidazol (Flagyl) +

Paromomycin or

Diloxanide furoate



***E. Coli* trophozoite**

E. Coli cyst