We have protozoa and helminthes that are parasites in the GI tract

We're gonna start with the protozoa:

The first protozoa is going to be **amoeba**

Lots of amoebae genera & species are **free living**; they live in the water and soil, and do **not** cause disease at all in the humans.

As far as the humans are concerned, there are **6 species** which can inhabit the body of human beings, but again out of these six species, there is **only one** which is **pathogenic** & we're going to concentrate on, which is **Entamoeba histolytica**, the other five varieties, they can be in the body but they do not cause any disease

So we'll start talking about Entamoeba histolytica :

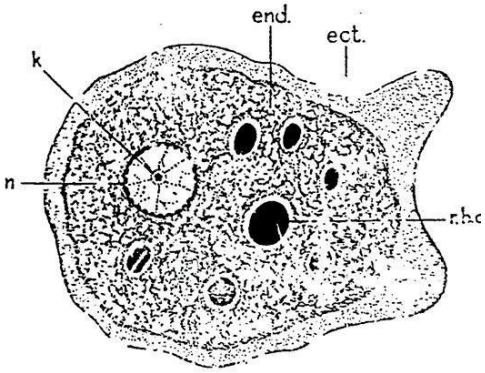
This is really a member of a family known as **Rhizopoda family.**

Rhizopoda...why? Because they move by means of **pseudopodia,** also, rhizo means crawling –their way of movement-.

Now if we look at the organism: this is a protozoa, which is directly spread **without an intermediate host**, so in that case we have what's known as **trophozoite and a cyst**

Trophozoite is the morphological entity of the organism which is live; it moves, it eats, excretes, metabolizes & causes diseases… it divides by binary fission

But because trophozoites are very delicate, they **die outside the body**, so they **can't transmit the disease**, so they have to **rely on** something else or another form of the organism, which is known as the cyst, and that's the one that transmits the disease




from one person to another.

Now if we look at trophozoite:

it measures about **20 to 40 microns** in diameter, and you can see the cytoplasm, which is actually divided into **2 areas**: one which is on the periphery and it's known as the **ectoplasm** which is clear as you can see, this is the bit of the cytoplasm which is **involved in movement**

you can notice the pseudopodia & the ectoplasm into them >>> pulls the organism towards it.

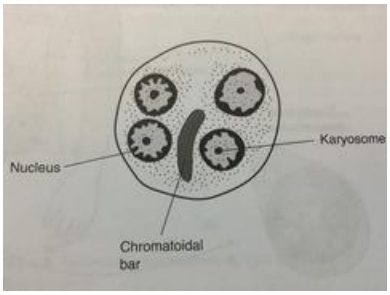
on the other aspect, in the center, we have the other bit of the cytoplasm which is known as **endoplasm**, and usually it's **more granular**, and there is **nucleus in the middle** .... .also, there are **vacuoles** which can contain digested bacteria, red blood cells *it depends on the species*

Of course we have **transformation of trophozoite into the cyst**...What happens?

There is **diminution** in the size, it becomes smaller and smaller, then we get the appearance of **chromatoid bodies**

chromatoid bodies are nucleic acids; **DNA** and **RNA** which actually appear during the formation of the cyst

Once the **cyst** is actually **completely formed (mature**), they are not present, they have **disappeared**, and as you can see there is a cyst, it has a **tough outer layer** and **4 nuclei**




The **number of nuclei** of course **depends on the species** that we are dealing with

Now before we move to discuss anything else, let's talk about the six varieties of amoeba that are encountered in the body

**1. Most important: entamoeba histolytica**

Because as we said it's the **pathological one** and it's believed to be present in **10% of all the people in the world**

**2. entamoeba coli**: this is not pathogenic but it happens to be present in **30% of people**

**3. entamoeba gingivalis**: its present in the mouth in case of **dental caries**

**4. endolimax nana**

**5. iodamoeba butschlii**

**6. dientamoeba fragilis**

These are again amoebae which can be present in GI tract, in the large intestine, but they are not pathogenic

You have to know that there are some other types that can be there, but they are not really that important

From all of these we have to concentrate on 2 types:

**entamoeba histolytica** because it's **pathogenic**

**entamoeba coli** because it's **so common**

We don't want to be confused between them in the diagnosis

Some people with diarrhea (dysentery), we look for the feces, and we don't want to confuse the trophozoite of entamoeba coli and entamoeba histolytica & we don't want to confuse the cysts together; because unfortunately, many people who have diarrhea, they go to the lab and the result is entamoeba histolytica .. so really we have to be careful about the **differentiation between these 2 types**

1. If we look at trophozoite: look at the **ectoplasm**; it's very greater (its amount is very big) in entameba histolytica in comparison with entameba coli which has very small amount of ectoplasm

2. **Pseudopodia** are going to be larger in entameba histolytica than those in entameba coli (As a result of the difference in the ectoplasm amount between the 2 species)

So the **movement of entamoeba histolytica** is going to be **quicker** but **entamoeba coli** is as **slow** as a turtle because its pseudopodia are really very small due to the small amount of ectoplasm there

3. Endoplasm, the **vacuoles in the endoplasm** of **entamoeba histolytica usually have red blood cells** whereas **vacuoles in coli** have **bacteria** but never red blood cells

Which is related to the function of pathogenicity

4. **Cyst**, the cyst of **entamoeba histolytica is smaller and has only 4 nuclei**, **in coli it's larger and it has 8**

if you look at a very big cyst, what you see is chromatoid bodies

5. **chromatoid bodies** again are different, **in entamoeba histolytica**, they are like

**Sausage (cigar shape**) but in **entamoeba coli** they are **splinter shape**

splinter: such as the result of breaking glass شظايا

6. **Peripheral chromatin** here **in entamoeba histolytica** is actually **uniform and thin**, but chromatin in **entamoeba coli** is irregularly scattered (thick in regions & thin in others)

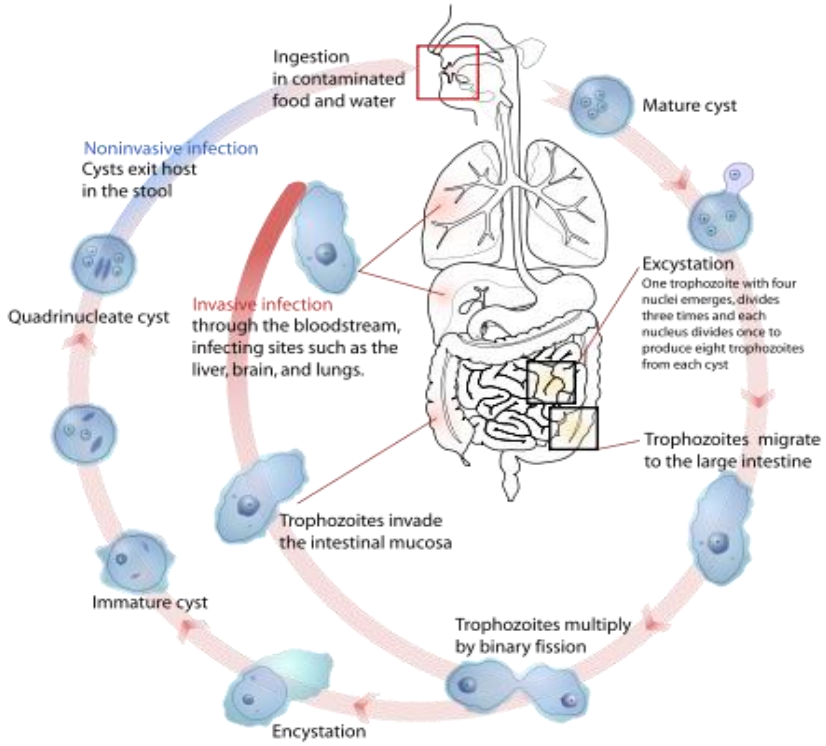
7**. Karyosome (equivalent to nucleolous)**, **in entamoeba histolytica,** is **central**, but **in coli** is **eccentric to one side of the nucleus**

These criteria differentiate between morphology of entamoeba histolytica and entamoeba coli, so when you look at something, you're going to make sure that is this histolytica or coli in order to make a proper diagnosis

Now the infection is passed from person to another by means of fecal contamination, we call it fecal-direct, there is no intermediate host, it is **feco-oral**

You go and buy shawarma and falafel, Seller was in the bathroom and didn't wash his hands in a proper way, so he put in your sandwich some cysts, you eat it, and once you eat it, these cysts are going to go to your stomach (usually these **cysts are resistant to acid, so they are not destroyed in your stomach)** then go to your **small intestine**, and under the **hydrolytic** effect of enzymes, the **outer covering disintegrates**, releasing 4 nuclei, which divide immediately into 8, and these 8 develop into baby amoebae known as **(amoebulae**) and these will quickly go to the

**large intestine** and develop into **adult trophozoite**




so now you are really infected

By **infection**.... what do we mean?

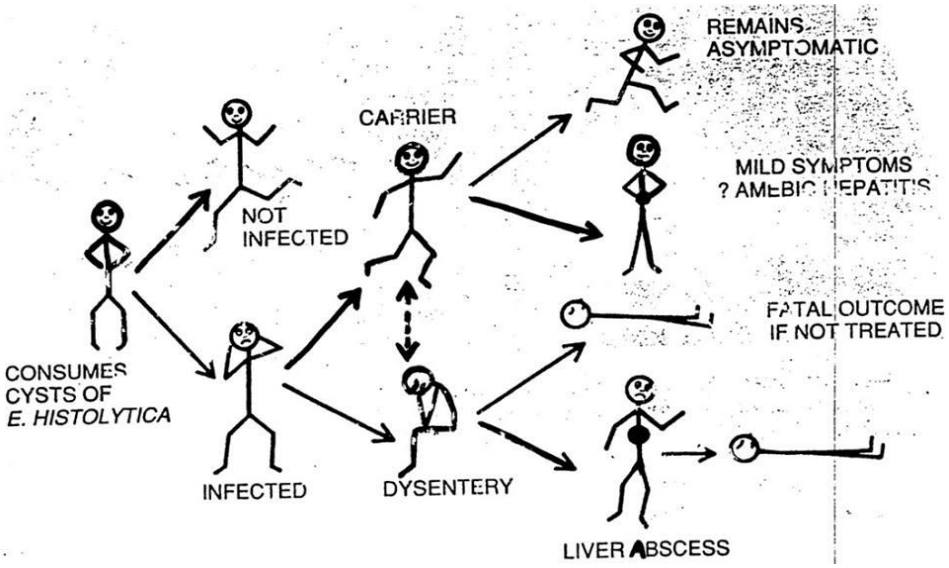
**having in your large intestine trophozoites of entamoeba histolytica**

But that doesn't mean that you are gonna have disease, infection does not necessarily mean that you are gonna be ill

So let's see what actually happen??

By doing experiments, people were infected on purpose by cysts voluntarily, some proportion of people although they ingested cyst, they never become infected at all, and no body knows why, may be the acidity of their stomach is so strong that it kills every thing مكيف ع حالو .. برقص وبغني

But the majority of people, they will become infected بلطم ع حالو .. وبنتف بشعرو



When you become infected what happens??

**You could develop disease or you could stay asymptomatic**

Once you become infected... what's the disease??

the disease is **dysentery**; diarrhea which is not copious غزير, the **feces are not very fluid**, you only **pass small amount of stools**, but you **go frequently** to the bathroom 10-15 times رايح جاي عالحمام .. بس الكمية اللي بتطلع بتكون قليلة جدا

There is **blood in it**, there is **mucus and pus**, and the smell of the stool is really very **very foul**

Dysentery; 2 types:

**1. Bacillary dysentery** caused by **shigella (bacillus bacteria)**

2**. Amoebic dysentery** caused by **amoeba**

Amoebic dysentery can cure itself sometimes by itself and it goes to become asymptomatic (carrier)

As we said infected people either become **ill** or become **carriers without symptoms (asymptomatic)**

If you don't eradicate the organism completely from the infected person > he either becomes a carrier, asymptomatic or **complications** may occur in which will lead actually to **death**

So from dysentery >> you can die

It’s not a very simple disease

The carrier either still carrier for the rest of his life or become ill or it can disappear (no longer hosts the organism), and he transmits the disease to other people all the time by passing the feces

For example, the **wall of the large intestine may burst**, and this rupture will lead to **peritonitis**, which lead to **death …** or the appendix may get involved, and you can get **appendicitis which also can kill.**

So what happens?? You can actually die; it's a very serious disease

In fact **amoeba ranks third in the frequency of fatality by protozoa**

1. the **most serious protozoa** and the **first one which can cause death** is **MALARIA**

2. Then **SCHISTOSOMA**

3. And the third is **AMOEBA**

Or sometimes you may get other complications like **swelling (chronic inflammation)** around the amoeba in the wall of the large intestine & you’ll get swelling in there, BE careful not to misdiagnose the swelling or tumor in such case with cancer

If you detect the swelling on the large intestine by **X-ray, ultrasound or CT scan**, usually the first thing that comes to your mind is cancer (you should always think of cancer as the first diagnosis) but in the differential diagnosis, you may have this tumor which is due to the amoeba (chronic inflammation), this is known as **meboma**

**ameboma: swelling due to inflammatory process caused by amoeba**

Or sometimes you can get like a chronic infection on the inside of the large intestine and this can actually mimic or resemble **inflammatory bowel disease (like ulcerative**

**colitis, crohn's disease)**: these are **chronic autoimmune diseases** which cause inflammation

But be careful here because ulcerative colitis sometimes can be **treated by immune suppressing drugs**, and **you do not want to use immuno suppressing drug in an infection**

If we misdiagnosed somebody, and reported him with ulcerative colitis and we gave him immune suppressing drugs, and the true diagnosis is amoeba chronic infection >> **we are going to make condition worse**

If you weren't 100% sure you do a colonoscopy, you examine the **inside of the large intestine; in the inflammatory bowel disease**, you'll find that the **whole mucosa** is involved, it's **universal**, the inflammation is **all uniform**, but in **amoeba**, the **lesions are scattered**; there are areas which are inflamed and others which are completely normal (because it's an infection)

Until now, **amoeba is** causing disease in the **lumen of the intestine**, so that is a **luminal parasite** BUT sometimes it can **penetrate the wall**, **gain access to the blood**, and from blood it will go **to portal circulation to the liver**, and there it will produce an **abscess**, now it becomes a **tissue parasite**

So **entamoeba histolytica is both luminal and tissue parasite** at the same time

So we have **liver abscess** which can **rapture into the peritoneum or the plural cavity** , and this can also **lead to death**

Again another cause of death is production of liver abscess

you may have other **abscess in other places of the body not just the liver**, can be in the **lungs**, or even in the **brain** and in some other areas but the **commonest** is the **liver** of course, and the second after the liver is actually lung abscess

So these are really the main pathological complications and entities (diseases) caused by amoeba

In the past, they always said that 10% of the people in the world are colonized by entamoeba histolytica, but really this is a bit un-true

Not 10% of people actually suffer from such diseases, so when you come & examine entamoeba histolytica itself, you find that there are strains that are pathogenic, and there are some strains that are non pathogenic

Indeed, **90% of entamoeba histolytica is non pathogenic**

When you look at **pathogenic and non pathogenic**; they are **morphologically exactly the same**, so morphologically we can't tell the difference

But this strain actually causes the disease and others which are the majority; they are really non pathogenic, indeed here we have **some sort of differences (biochemical & structural differences) that are not visible to the naked eye and differentiate between the 2 strains.**

**The strain which is non pathogenic** is known as **entamoeba dispar**

**The strain which is pathogenic** is known as **entamoeba histolytica**

What are these differences?? Sometimes they are very clear and sometimes they are very small variations

**Differences / properties make entamoeba histolytica pathogenic:**

**1. EH-lectin**: entamoeba histolytica lectin (lectin: is a kind of molecule which helps things **to adhere** مادة لاصقة)

So entamoeba histolytica has this material which **allows it to stick to mucous**, and we know that **adherence is a pathogenicity factor**

For any infection to establish itself, it needs to adhere properly to the surface in which it's present

EH-lectin is an important property in making entamoeba histolytica pathogenic

, helps it to stick to mucous & invade the mucosa

**2. Polypeptide and proteins** known as **amoeba pores (pore making peptide)**, they are **very effective in making holes**

**Amoeba release protein material** which **makes pores in cells**, the **cells are going to die**, then they are going to be **eaten by amoeba organism**

Pore making peptide is an important property in making entamoeba histolytica pathogenic

**3. Complement resistance**: complement is a **part of immune system** which is **responsible for lysing pathogenic organisms**, so if pathogens are sensitive to complement system, there is no chance to make an infection, they will die very quickly

So **entamoeba dispar** is **sensitive to complement system** of the body >> **die quickly**

**entamoeba histolytica** is **more resistant to complement system**, so it allows it to **survive and cause pathology**

**4. Growth**, you can actually grow them in culture, but **normally amoeba in general if you want to grow them you have to provide them with bacteria** (ie. You have to add a xenic culture)

**xenic culture**: it's a culture which contains bacteria

But **entamoeba histolytica** is somehow **strong / has got special properties** that it can **grow in a culture without bacteria (axenic culture)**

**5. Proteases, collagenases**; enzymes which **dissolve intercellular material between the cells**, so if that happens, the organism can invade, push itself in the tissues and so on ...

So **entamoeba histolytica** has strong variety of these enzymes which **destroy the tissues and allow histolytica to be pathogenic**, especially cysteine proteases, there are other proteases and collagenases, but cysteine protease is the **most abundant and the most important**

**6. Concanavalin**, which when added to amoeba histolytica can cause **agglutination**

Amoeba hysto. + concanavalin A = agglutination between them

But this doesn't happen with entamoeba dispar

There is something on the surface of these organisms that we can't see morphologically, which accounts for these differences in the properties

These are the properties that differentiate between the two groups, so even large number of people 10% have entamoeba histolytica, most of them have dispar strain and they do not have disease

**It could possibly switch between one another**

The **diet of the human** may **affect the pathogenicity of entamoeba** or even affect the bacteria which is present in GI tract (normal flora can affect the pathogenicity of entamoeba)

So, finally, if you get the disease you are having either dysentery or liver abscess or even the carrier people sometimes they can have **symptoms: abdominal pain, bloating نفاخ , or very slight amount of hepatitis (mild hepatitis)**

**Diagnosis** is usually by diagnosis of **feces**, when you examine feces, you may see trophozoite, but to see trophozoite, you have to examine the **fresh feces**

if you leave them, trophozoites will die, so you have to collect samples straight away from the patient, put it on slide, **keep it warm**, because **warmth** actually **allow the trophozoite to move (keep it alive and keep it moving), which helps you to detect trophozoite;** when you look at the stool under the microscope,حتلاقي بلاوي, so it's very confusing, but if you see something moving, you'll pick it up straight away, so you can see pseudopodia and trophozoites

This is not always possible, it's very difficult, because samples are taken to the lab and most probably trophozoites are going to die

So you want to look for the **cyst**, add **iodine** to help you (it kills the trophozoites), (**do not look for trophozoites because they have already died**), but the **cyst** will be **highlighted brownish-yellowish**, and with the **4 nuclei**, you can determine the diagnosis

With the **liver abscess** you can **aspirate from the wall of the abscess not from the middle**, because at the middle, it will be fluid and you won't see anything, but if you examine the wall, you will see trophozoites

Or you can use **serology**, **studying the blood**, look for the **serum** of the patient**, if he has antibodies against antigens of the amoeba or not**, so wherever there is infection, there are antibodies against antigen of the amoeba in the blood (+ve serology)

Then you will start the **treatment**

There are many amoebae that **live free (free living amoebae),** and these **very rarely cause disease**, but there are **a couple that actually can cause disease in human beings**, they are **very serious**, but fortunately they are **very rare**, (very unlikely that you ever see any case of these)

**One is: naegleria fowleri**

Actually sometimes exist as **trophozoite, rhizopoda, flagellate or cyst**, so it **can change**

They **live in water**, they produce **encephalitis and meningitis**, which are usually **invariably fatal,** usually **no case of such encephalitis has ever survived**, **and they always die**

It's believed that swimming in a swimming pool which has naegleria fowleri in the water, it's believed that it goes up your nose, through cribriform plate to meninges and cause encephalitis

**Diagnosis** is by examination of **CSF**, and **usually the result is death**

**The second is: Acanthamoeba**

More likely here, the **infection is acquired through blood through injury**, **goes to the brain** through blood and cause **encephalitis**, again it's **invariably fatal**

Also, it can produce **keratitis**, which is **inflammation of cornea**, again it's very difficult to treat, and it actually produces **blindness, the only treatment is corneal graft (transplantation),** otherwise, he will be blind

*****that's the finish of amoeba story*****

Another parasite that causes disease in GI tract**: giardia lamblia**

This is a trophozoite that is flagellated because it has high flagellae, and this one lives **in the small intestine**

**(All amoeba live in the large intestine)**

**Pear shaped**

**20-30 microns**

It has **four pairs of flagella (4 in each side)**

It has **2 nuclei**; each of them has **very prominent nucleolus**, **owl-like appearance or kite-like appearance**

Looking at it from the side, you will see that it looks like lady bird أم علي

 (it is **convex on dorsal aspect**, and it's **flat on the ventral aspect)**

The ventral aspect which is flat has **a sucker on it (sucking pad/disk) which** is the one by which it **attaches itself to the inner surface of the small intestine**

Now as they **attach themselves to the wall of small intestine**, they actually cause effacement **of the microvilli,** we know that **on the villi of small intestine there are microvilli to increase the surface area for absorption,** so when you have **extensive infection** by these organisms, you find that the **microvilli are effaced (NO more microvilli)**

It's not known, some people say it's mechanical; these **organism sit on the inner surface** so they **destroy the microvilli**, or some people may think that there is some sort of **toxin produced by the organism** which causes actual effacement **of these microvilli**

**No microvilli >> malabsorption >> diarrhea**

This diarrhea is really malapsorptive & **very distinct, no fat is absorbed**, you find that the **stools are grayish-whitish**, **very sticky**, and its **smell is really very foul**, and this is known as **malabsorption of fat – presence of excess fat in feces (steatorrhea)**

The person doesn't feel very well, he is **underweight,** may be **anemic, has gases**

This **Giardiasis disease** is **mainly a disease of children** because maybe they haven't got very good hygienic practices

**Giardiasis disease can sometimes be water born,** contamination of water can give rise to it… In Ganges river/india, millions of people dive in there together, as a sort of sacraments, if anyone drinks from that water, he will end up with giardiasis, because the water is contaminated, so very often the disease can be water born

Transmission: by **feco-oral route** by eating contaminated food

**Diagnosis**: you look for the **cysts**, because trophozoites are very unlikely to be found in the feces, but the cysts will be present in large numbers

The cyst -that you are going to see- have **tough outer covering**, and they have **4 nuclei**

You may see some **flagellae** as well **inside**

You make the **diagnosis**, and then give the **treatment**

**ولا بد من قيادة للبشرية جديدة!**