**Entamoeba coli**

**MORPHOLOGY IN CONTRAST WITH E. histolytica**

<table>
<thead>
<tr>
<th></th>
<th>E. histolytica</th>
<th>Trophozoite</th>
<th>E. coli</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stained by iron haematoxylin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purpleish brown</td>
<td>Purpleish brown</td>
<td>Cytosol</td>
<td>Greyish blue</td>
</tr>
<tr>
<td>Faintly granular</td>
<td>Faintly granular</td>
<td>Inclusions</td>
<td>Coarsely granular</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>Red blood cells</td>
<td>Nucleus Membrane</td>
<td>Vacuoles black, as are bacteria etc.</td>
</tr>
<tr>
<td>Lined with minute black granules</td>
<td>Lined with minute black granules</td>
<td>Thick with plaques of black chromatin</td>
<td></td>
</tr>
<tr>
<td>Small black central dot</td>
<td>Small black central dot</td>
<td>Karyosome</td>
<td>Eccentric black dot or plaque</td>
</tr>
<tr>
<td>Trace only seen</td>
<td>Trace only seen</td>
<td>Fibre network</td>
<td>More conspicuous; may have chromatin plaques</td>
</tr>
</tbody>
</table>

**Precyst**

<table>
<thead>
<tr>
<th></th>
<th>E. histolytica</th>
<th>Trophozoite</th>
<th>E. coli</th>
</tr>
</thead>
<tbody>
<tr>
<td>Round</td>
<td>Round</td>
<td>As trophozoite</td>
<td>Round</td>
</tr>
<tr>
<td>Astrocyte</td>
<td>Astrocyte</td>
<td>Cytosol Nucleus</td>
<td>As trophozoite</td>
</tr>
<tr>
<td>Black chromidial bodies or bire</td>
<td>Black chromidial bodies or bire</td>
<td>Inclusions</td>
<td>May have slender black chromidial bire</td>
</tr>
<tr>
<td>Glycogen (dissolved) replaced by vacuoles</td>
<td>Glycogen (dissolved) replaced by vacuoles</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Cyst**

<table>
<thead>
<tr>
<th></th>
<th>E. histolytica</th>
<th>Trophozoite</th>
<th>E. coli</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grey-blue</td>
<td>Grey-blue</td>
<td>Cytosol</td>
<td>Greyish-blue, granular</td>
</tr>
<tr>
<td>As pre cyst less conspicuous or absent</td>
<td>As pre cyst less conspicuous or absent</td>
<td>Inclusions</td>
<td>As pre cyst less conspicuous or absent</td>
</tr>
<tr>
<td>Unstained, hyaline</td>
<td>Unstained, hyaline</td>
<td>Wall</td>
<td>Unstained, hyaline</td>
</tr>
<tr>
<td>As trophozoite 1-4</td>
<td>As trophozoite 1-4</td>
<td>Nuclei</td>
<td>As trophozoite 1-8</td>
</tr>
</tbody>
</table>
**Entamoeba coli** is a non-pathogenic species of *Entamoeba* that frequently exists as a commensal parasite in the human gastrointestinal tract. Clinically, *E. coli* (not to be confused with the bacterium *Escherichia coli*) is important in medicine because it can be confused during microscopic examination of stained stool specimens with the pathogenic *Entamoeba histolytica*. While this differentiation is typically done by visual examination of the parasitic cysts via light microscopy, new methods using molecular biology techniques have been developed.

The presence of *E. coli* is not cause in and of itself to seek treatment as it is considered harmless. However it should be noted that when a person becomes infected with this benign entamoeba, other pathogenic organisms may have been consumed at the same time.

**Entamoeba gingivalis**

*Entamoeba gingivalis* is a non-pathogenic protozoa and is known to be the first amoeba in humans to be described. It is found only in the mouth between the gingival pockets and near the base of the teeth. *Entamoeba gingivalis* is found in 95% of people with gum disease and in 50% of people with healthy gums. The cyst formation is not present, therefore transmission is direct from one person to another by kissing, or by sharing eating utensils. Only the trophozoites are formed and the size is usually 10 micrometer to 20 micrometer in diameter. *Entamoeba gingivalis* have pseudopodia that allow them to move quickly. Their spheroid nucleus is 2 micrometer to 4 micrometer in diameter and contains a small central endosome. There are numerous food vacuoles and contain cellular debris, blood cells and bacteria.
Entamoeba gingivalis is an Entamoeba histolytica-like amoebae that lives in/on the teeth, gums, and sometimes tonsils. It measures 10-35 micrometers in length. Endocytotic vacuoles are often numerous and the parasite will ingest bacteria, leukocytes, and erythrocytes (dark circles in trophozoites, above) although it is not itself invasive. No cysts are formed and transmission is entirely by oral-oral contact. Multiple samplings reveal the parasite to colonize the oral cavity of nearly all adult humans.

Here's a fun fact. Several reports have documented colonization of the uterus by Entamoeba gingivalis. One intriguing study (1980, Acta Cytol. 24: 413-420) revealed about 10% of all intrauterine devices (IUDs) to be colonized by the filamentous plaque causing bacterium, Actinomyces. This bacterium is one favorite food item of Entamoeba gingivalis. Of those women with IUDs colonized by Actinomyces, approximately 10% of those also harbored the amoeba (IUDs without the bacterium had no amoebae). Thus, about 1% of all females with IUDs are thought to harbor uterine E. gingivalis. Food for thought?
Three stages exist (1) the *amoeba*, (2) the bi-flagellate and (3) the *cyst*.

**WHAT IS *NAEGLERIA*?**

*Naegleria* is an ameba commonly found in warm freshwater and soil. Only one species of *Naegleria* infects people, *Naegleria fowleri*. It causes a very rare but severe brain infection. Most infections are fatal.

**HOW DOES INFECTION WITH *NAEGLERIA* OCCUR?**

*Naegleria* infects people by entering the body through the nose. Generally, this occurs when people use warm freshwater for activities like swimming or diving. The ameba travels up the nose to the brain and spinal cord where it destroys the brain tissue. Infections do not occur as a result of drinking contaminated water.

**WHERE IS *NAEGLERIA* FOUND?**

*Naegleria fowleri* is found around the world. The ameba grows best in warm or hot water. Most commonly, the ameba may be found in:

- Bodies of warm freshwater, such as lakes, rivers
- Geothermal (naturally hot) water such as hot springs
- Geothermal (naturally hot) drinking water sources
- Warm water discharge from industrial plants
- Poorly maintained and minimally-chlorinated or unchlorinated swimming pools
- Soil

*Naegleria* is not found in salt water locations like the ocean.

*Naegleria* infection cannot be spread from one person to another.
THE SYMPTOMS OF *NAEGLERIA* INFECTION:-

Infection with *Naegleria* causes the disease primary amebic meningoencephalitis (PAM), a brain infection that leads to the destruction of brain tissue. In its early stages, *Naegleria* infection may be similar to bacterial meningitis.

Initial symptoms of PAM start 1 to 14 days after infection. The initial symptoms include headache, fever, nausea, vomiting, and stiff neck. Later symptoms include confusion, lack of attention to people and surroundings, loss of balance, seizures, and hallucinations. After the start of symptoms, the disease progresses rapidly and usually causes death within 3 to 7 days.

LIFE CYCLE OF *NAEGLERIA AND ACANTHAMOEOBA*

This is an illustration of the life cycle of the parasitic agents responsible for causing “free-living” amebic infections.
Free-living amebae belonging to the genera *Acanthamoeba*, and *Naegleria* are important causes of disease in humans and animals. *Naegleria fowleri* produces an acute, and usually lethal, central nervous system (CNS) disease called **primary amebic meningoencephalitis** (PAM). *N. fowleri* has three stages, cysts (1), trophozoites (2) flagellated forms (3), in its life cycle. The trophozoites replicate by **promitosis** (nuclear membrane remains intact) (4). *Naegleria fowleri* is found in fresh water, soil, thermal discharges of power plants, heated swimming pools, hydrotherapy and medicinal pools, aquariums, and sewage. Trophozoites can turn into temporary flagellated forms which usually revert back to the trophozoite stage. Trophozoites infect humans or animals by entering the olfactory neuroepithelium (5) and reaching the brain. *N. fowleri* trophozoites are found in cerebrospinal fluid (CSF) and tissue, while flagellated forms are found in CSF.

*Acanthamoeba* spp. is opportunistic free-living amebae capable of causing **granulomatous amebic encephalitis** (GAE) in individuals with compromised immune systems. *Acanthamoeba* spp. have been found in soil; fresh, brackish, and sea water; sewage; swimming pools; contact lens equipment; medicinal pools; dental treatment units; dialysis machines; heating, ventilating, and air conditioning systems; mammalian cell cultures; vegetables; human nostrils and throats; and human and animal brain, skin, and lung tissues. Unlike *N. fowleri*, *Acanthamoeba* have only two stages, cysts (1) and trophozoites (2), in its life cycle. No flagellated stage exists as part of the life cycle. The trophozoites replicate by **mitosis** (nuclear membrane does not remain intact) (3). The trophozoites are the infective forms and are believed to gain entry into the body through the lower respiratory tract, ulcerated or broken skin and invade the central nervous system by hematogenous dissemination (4). *Acanthamoeba* spp cyst and trophozoites are found in tissue.
LAB DIAGNOSIS:

For practical purposes, *N. fowleri* meningoencephalitis must be rapidly diagnosed. Patients who present with a clinical picture of meningitis (ie, fever, headache, meningismus, nausea and vomiting) should undergo a spinal tap as soon as they present.

In patients with PAM, the CSF pressure is often elevated, and the CSF is hemorrhagic. The WBC count can be within the reference range in early infections but rapidly increases to range from 400-26,000 cells/µL with a neutrophilic predominance. The CSF glucose level may be low or within the reference range, but the CSF protein is usually elevated. Results on a Gram stain of the CSF sediment are negative for bacteria. A wet mount must be made because the trophozoites of *N. fowleri* lyse during the heat fixation that precedes the Gram stain. On the wet preparation, motile trophozoites are evident. Care must be taken to avoid confusing *N. fowleri* trophozoites with WBCs and vice versa. In examining CSF for *N. fowleri*, a regular glass slide for a wet mount is preferred to a WBC counting chamber. The regular glass slide allows for better definition of internal structures.

The CSF is centrifuged at 150g for 5 minutes. The supernatant is carefully aspirated, and the sediment is gently suspended in the remaining fluid. A drop of this suspension is placed on a slide and covered with a No. 1 coverslip. The slide is observed under a compound microscope using 10 and 40 objectives. Phase contrast optics is preferable. The slide may be warmed to 35°C (to promote amebic movement). The amebae are detected based on their active directional movements. CSF indices in *N. fowleri* include the following:

- CSF protein levels are elevated.
- CSF glucose levels are within the reference range or reduced.
- CSF WBC count is elevated (400-26,000 cells/µL).
- CSF RBC count is high, and the CSF is often hemorrhagic.
- CSF Gram stain results are negative for bacteria.
- CSF wet mount is positive for motile trophozoites and is of paramount importance for the diagnosis.

Additional methods of diagnosing *N. fowleri* infection include polymerase chain reaction (PCR), monoclonal antibodies, DNA probes, and isoenzyme profile analysis. However, these methods are more time consuming and labor intensive than routine CSF studies. They are useful in postmortem diagnoses and for research purposes.
Other nonspecific laboratory findings in peripheral blood include the following:

- The WBC count is elevated with a neutrophilic predominance.
- Complete metabolic panel (CMP) may show abnormalities, including hyponatremia associated with acquired diabetes insipidus, hyperglycemia, or both.

Imaging Studies

- Limited data are available on imaging studies. One patient has been reported who had a CT scan of the head that demonstrated diffuse enhancement of the gray matter and obliteration of the interpeduncular and quadrigeminal cisterns.

Histologic Findings

- Amebic trophozoites in perivascular spaces and paraventricular areas
- Fibrinoid necrosis in some blood vessels
- Hemorrhage and necrosis
- Meningeal exudate composed of neutrophils, chronic inflammatory cells, and degenerating amebae
- Focal demyelination in the white matter of the brain and spinal cord
- Acute inflammatory reaction in nasal mucous membranes
- Trophozoites demonstrated on hematoxylin- and eosin-stained slides in involved tissues

**DRUG CATEGORY:** Polyene antibiotics

Amphotericin B desoxycholate (Fungizone) is the most effective drug against *N. fowleri*
Acanthamoeba Infection

*Acanthamoeba* are microscopic amoeba commonly found in the environment. Several species of *Acanthamoeba* have been found to infect humans, *A. culbertsoni*, *A. polyphaga*, *A. castellanii*, *A. healyi*, *A. astronyxis*, *A. hatchetti*, *A. rhysodes*, and possibly others.

**Trophozoite:**

*Acanthamoeba trophozoite*. Spiny surface structures called *acanthopodia*, distinguish *Acanthamoeba* from other free-living amebae that infect humans.

Scanning electron micrograph of an *Acanthamoeba trophozoite*. Spiny surface structures called *acanthopodia* (arrows) distinguish *Acanthamoeba* from other free-living amebae that infect humans, such as *B. mandrillaris*, *N. fowleri*, and *Sappinia diploidea*. Bar, 1 µm.
*Acanthamoeba* spp.: the trophozoite is irregular, 15-45 µm, having micropseudopodia called acanthopodia; in trichrome stain the cytoplasm of trophozoites appears greenish pink, the central located karyosome pink or red. (Trichrome stain).
Cyst:

The cysts are spherical, 15-20 µm in diameter, having a thick double wall. The outer wall may be spherical or wrinkled, the inner wall appear stellate or polyhedral.

(Acanthamoeba trophozoites and a cyst, trichrome stain).

Both forms have a single nucleus with a large centrally located nucleolus. With trichrome stain, the cysts stain red. Species identification is based on morphology of cysts (stellate, polyhedral).
LIFE CYCLE AS MENTIONED ABOVE WITH *NAEGLERIA*

LAB DIAGNOSIS IS THE SAME THAT IN *NAEGLERIA* ABOVE.

MOVEMENT TRACKING OF ACANTHAMOEBA

*Acanthamoeba* may be cultured on non nutrient agar flooded with Page's saline solution and overlaid with *Escherichiacoli*. "Feed" tracks on agar plate will be seen as the following:

(Feed_tracts_4x ; Feed_tracts_10x ).
Where are Acanthamoeba found?

Acanthamoeba spp. (spp. means several species) are found worldwide. Most commonly, Acanthamoeba are found in the soil and dust, in fresh water sources such as lakes, rivers, and hot springs and in hot tubs. Acanthamoeba may also be found in brackish water and in sea water. Amoeba can also be found in Heating, Venting, and Air Conditioner units (HVAC), humidifiers, and dialysis units.

Acanthamoeba have been found in the nose and throat of healthy people as well as those with compromised immune systems.

Infection with Acanthamoeba

Acanthamoeba can enter the skin through a cut, wound, or through the nostrils. Once inside the body, amoeba can travel to the lungs and through the bloodstream to other parts of the body, especially the central nervous system (brain and spinal cord).

Through improper storage, handling, and disinfection of contact lenses, Acanthamoeba can enter the eye and cause a serious infection.

The signs and symptoms of Acanthamoeba infection

There are several ways Acanthamoeba spp. can affect the body. Eye infections result from contact lens cases becoming contaminated after improper cleaning and handling. Risk of Acanthamoeba infection is higher for people who make their own contact lens cleaning solution. Acanthamoeba enter the eye via contact lenses or through a corneal cut or sore. Infection or a corneal ulcer results.

In addition, Acanthamoeba spp. can cause skin lesions and/or a systemic (whole body) infection.

Acanthamoeba spp. cause a serious, most often deadly infection called granulomatous amebic encephalitis (GAE). Once infected, a person may suffer with headaches, stiff neck, nausea and vomiting, tiredness, confusion, lack of attention to people and surroundings, loss of balance and bodily control, seizures, and hallucinations. Signs and symptoms progress over several weeks; death generally occurs.

The treatment for infection with Acanthamoeba:-

Amphotericin B desoxycholate (Fungizone) is the drug of choice. Eye and skin infections are generally treatable. However, infections of the brain (CNS) with Acanthamoeba are almost always fatal.
**Endolimax nana**

*Endolimax.nana*: The identification of intestinal amoebae depends on the size and shape of trophozoites and cysts and on morphology and number of nuclei. *Endolimax nana* has a world-wide distribution and is considered an harmless commensal of the intestine. *Endolimax* is a genus of amoebozoa that are found in the intestines of various animals, including the species *E. nana* found in humans. It causes no known disease and is most significant in medicine because it can provide false positives for other tests, notably the similar species *Entamoeba histolytica* responsible for amoebic dysentery, and because its presence indicates the host has consumed fecal material. It forms cysts with four nuclei which excyst in the body and become trophozoites. *Endolimax nana* nuclei have a large endosome somewhat off-center and small amounts of visible chromatin or none at all.

![Image of Endolimax nana trophozoite](image)

*E.nana* trophozoites are small (6-12 µm); the nucleus contain a large karyosome with a clear halo.
**Endolimax nana:** many cysts are visible, each with one to four nuclei. The cysts are smaller than 10 mm and contain four nuclei with a massive central karyosome.

*E.nana:* cysts are oval (6-10 µm) contain 4 small nuclei with a relatively large karyosome.

**LAB DIAGNOSIS AND TREATMENT:**

Examine the stool microscopically and see the *Endolimax nana* BUT Differentiation from *Enteromonas hominis* cysts is sometimes difficult. No treatment required, although it will probably vanish with some [metronidazole](https://www.mayoclinic.org/diseases-conditions/metronidazole/symptoms-causes/syc-20355568) (Flagyl).