

Amoebiasis and Shigellosis

Transmission, Pathogenicity & Symptoms, Diagnosis, Treatment/Control and Prophylaxis

Amoebiasis and shigellosis are both faecal-orally transmitted infections of the large intestine that cause dysentery, but they are caused by very different organisms - a protozoan parasite versus a bacterium - with different mechanisms of tissue damage, diagnosis and treatment. This makes them a classic compare-and-contrast pair in microbiology and parasitology.

1. Amoebiasis

Amoebiasis is an infection of the large intestine caused by the protozoan parasite *Entamoeba histolytica*, which exists as an environmentally resistant **cyst** (the infective stage) and an actively feeding, potentially tissue-invasive **trophozoite**.

1.1 Mode of transmission

Transmission is faecal-oral: mature quadrinucleate cysts, passed in the stool of an infected person, contaminate food or water and are ingested by a new host. Less commonly, transmission occurs directly person-to-person through poor hand hygiene, or via sexual contact involving faecal exposure. The cyst survives gastric acid, and excystation in the caecum/lower ileum releases trophozoites that go on to colonise the colon.

Life Cycle of *Entamoeba histolytica*

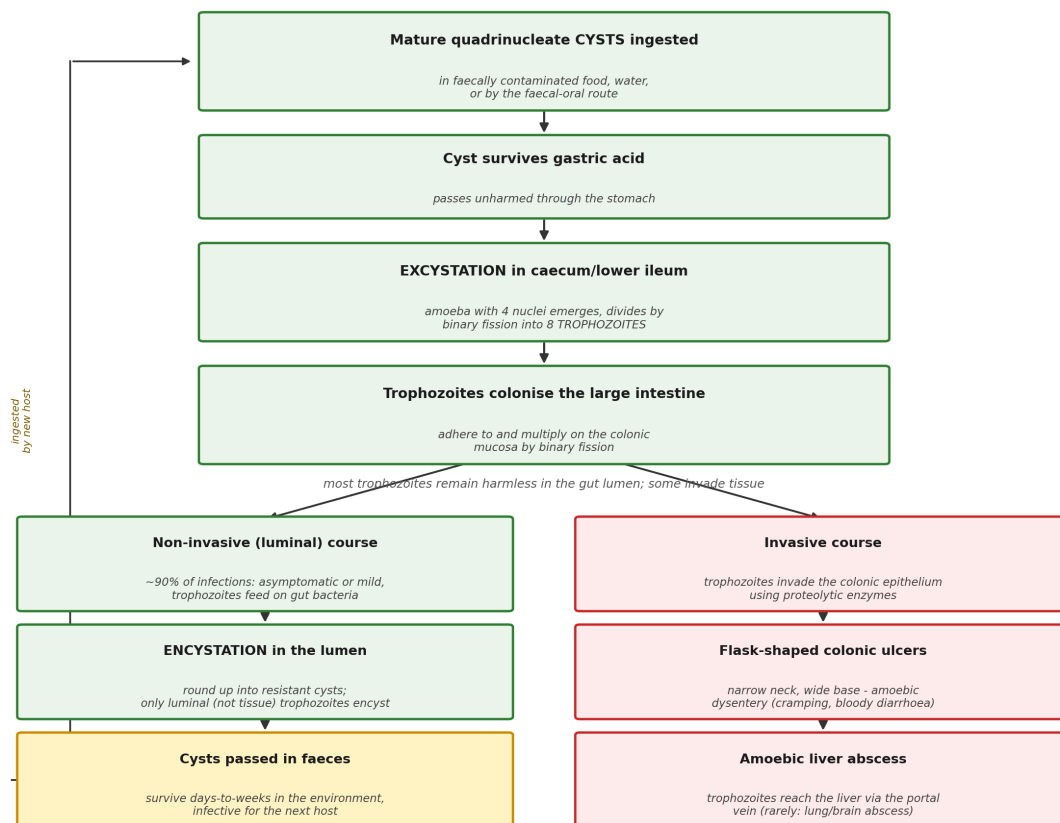


Fig. 1 - Life cycle of *Entamoeba histolytica*, showing the non-invasive (cyst-producing) and invasive (disease-causing) courses that trophozoites can take.

1.2 Pathogenicity and symptoms

About 90% of infections remain in the gut lumen and are asymptomatic or only mildly symptomatic, with trophozoites feeding harmlessly on colonic bacteria before encysting and being passed in the stool. In the remaining cases, trophozoites invade the colonic epithelium using proteolytic enzymes, producing characteristic **flask-shaped ulcers** (narrow at the mucosal surface, wider in the submucosa) and **amoebic dysentery** - cramping abdominal pain, weight loss, and diarrhoea that is often blood-streaked. Trophozoites can also enter the portal circulation and travel to the liver, producing an **amoebic liver abscess** (fever and right-upper-quadrant pain, typically 2-4 weeks after exposure) - the most common extraintestinal complication; rarely, abscesses form in the lung or brain.

1.3 Diagnosis

- **Stool microscopy:** demonstration of cysts or haematophagous trophozoites in fresh stool, though sensitivity is limited and cannot reliably distinguish *E. histolytica* from the non-pathogenic *E. dispar*.
- **Stool antigen testing / PCR:** antigen ELISA (~88% sensitivity) and stool PCR (the current gold standard, 92-100% sensitivity and specificity) both distinguish pathogenic *E. histolytica* specifically.
- **Serology:** highly sensitive and specific for invasive disease, but cannot distinguish a current infection from a past one.
- **Ultrasound/CT:** the preferred imaging for detecting and characterising an amoebic liver abscess.

1.4 Treatment/control

Effective treatment needs two drug classes in sequence: a **tissue amoebicide** such as **metronidazole** or **tinidazole** to kill invasive trophozoites, followed by a **luminal amoebicide** such as **paromomycin** or **diloxanide furoate** to eradicate any remaining cysts/trophozoites in the gut and prevent relapse or further transmission. A large liver abscess may also need image-guided percutaneous drainage in addition to drug therapy.

1.5 Prophylaxis

- Safe drinking water and proper sanitation/sewage disposal to prevent faecal contamination of food and water supplies.
- Personal and food-handler hygiene - handwashing with soap after defecation and before handling food.
- For travellers to endemic areas: avoiding tap water and uncooked/unpeeled produce washed in untreated water.
- Prompt identification and treatment of both symptomatic cases and asymptomatic cyst passers to reduce onward transmission (no vaccine is currently available).

Key points

- Only the cyst is infective (resistant to gastric acid and the environment); the trophozoite is the actively multiplying and tissue-invading form but cannot survive outside the host or re-establish infection if ingested.
- Amoebic liver abscess is the classic "extraintestinal" complication to remember - it can occur even without current diarrhoea, sometimes years after the original infection.

2. Shigellosis

Shigellosis (bacillary dysentery) is an acute intestinal infection caused by *Shigella* species - Gram-negative, non-motile bacilli comprising four species/serogroups: *S. dysenteriae*, *S. flexneri*, *S. boydii* and *S. sonnei*.

2.1 Mode of transmission

Shigella spreads by the faecal-oral route: contaminated food, water, surfaces and objects, and direct person-to-person contact (including sexual contact involving faecal exposure) all transmit infection. A distinguishing feature is its very low infective dose - as few as 10-100 organisms can cause disease - which makes person-to-person spread in households, childcare settings and crowded or low-sanitation conditions particularly efficient.

2.2 Pathogenicity and symptoms

After surviving passage through the acidic stomach, *Shigella* crosses the colonic epithelium by transcytosis through specialised **M cells**, is taken up by macrophages, escapes the phagolysosome and triggers macrophage apoptosis - releasing IL-1-beta and provoking a strong inflammatory response. The bacteria then invade neighbouring enterocytes from their basolateral surface and spread directly from cell to cell using actin-based motility, without needing to re-enter the gut lumen. The resulting massive neutrophil infiltration damages the mucosa, causing ulceration and microabscesses. Some strains (mainly *S. dysenteriae* type 1) also produce **Shiga toxin**, which is not required for basic symptoms but increases severity and can trigger **haemolytic uraemic syndrome (HUS)** in severe cases. Clinically this produces **bacillary dysentery**: fever, abdominal cramps, tenesmus, and frequent small-volume stools containing blood, mucus and pus, usually starting 1-2 days after exposure and lasting about a week.

Pathogenesis of Shigellosis

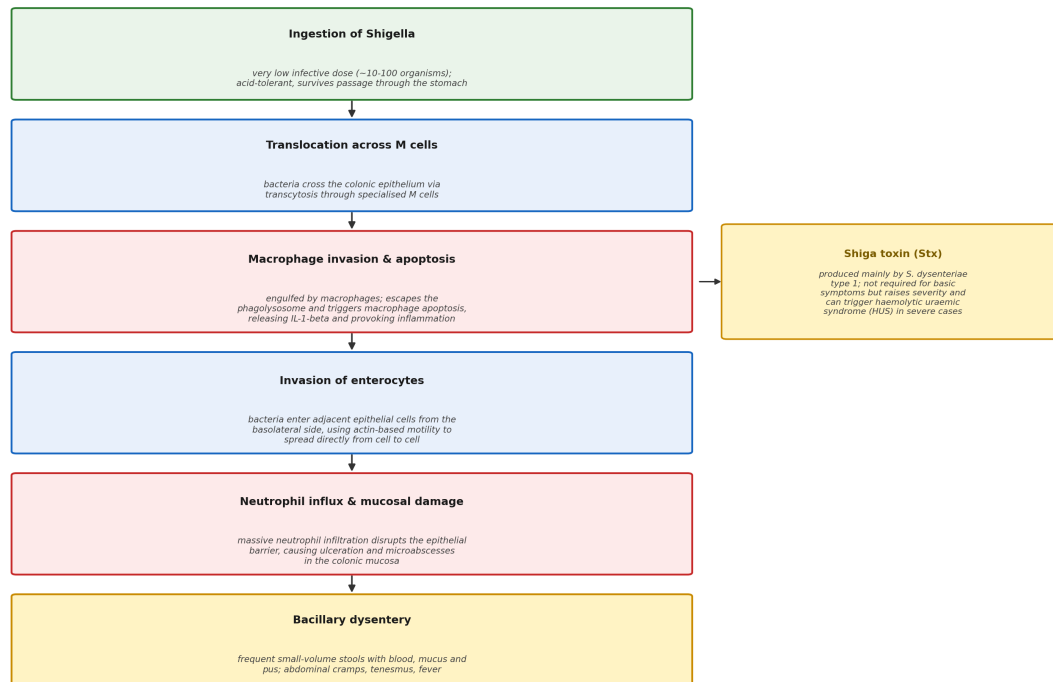


Fig. 2 - Pathogenesis of shigellosis, from ingestion to bacillary dysentery, with the role of Shiga toxin.

2.3 Diagnosis

- **Stool culture with antimicrobial susceptibility testing** remains the definitive diagnostic method, and is essential for guiding antibiotic choice given rising resistance.
- **Culture-independent diagnostic tests (CIDTs)**, such as nucleic acid amplification tests (NAATs), are increasingly used for faster results, though they cannot distinguish *Shigella* from enteroinvasive *E. coli* or provide resistance data.
- Stool microscopy typically shows numerous faecal leukocytes and red cells, supporting (but not confirming) the diagnosis.

2.4 Treatment/control

Mild shigellosis is usually self-limiting within 5-7 days and managed with supportive care - oral rehydration to prevent dehydration. Antibiotics are reserved for more severe or prolonged cases, young children, the elderly, or immunocompromised patients, since **antimicrobial resistance in *Shigella* is increasing worldwide**, including extensively drug-resistant strains; antibiotic choice should therefore be guided by local resistance patterns and, where possible, culture susceptibility results rather than empiric treatment alone. Anti-motility (anti-diarrhoeal) drugs are generally avoided, as they can worsen the illness by delaying pathogen clearance.

2.5 Prophylaxis

- Careful handwashing with soap, especially after using the toilet, changing nappies/diapers, and before preparing or eating food.
- Safe food and water practices, particularly for travellers to areas with poor sanitation.
- Avoiding swallowing water from pools, lakes and other recreational water bodies.
- Isolating symptomatic individuals from food handling/childcare duties, and abstaining from sexual activity that risks faecal exposure until diarrhoea has resolved.
- No vaccine is currently licensed for routine use, though several candidates are in development.

Worth noting

- "Shigella" and "dysentery" are not synonyms - amoebiasis (Section 1) also causes dysentery, but by a completely different mechanism (a protozoan directly digesting tissue vs a bacterium triggering an inflammatory/immune-mediated mucosal injury). Distinguishing bacillary from amoebic dysentery by mechanism, not just by symptoms, is a common exam point.

3. Quick Comparison

	Amoebiasis	Shigellosis
Causative organism	<i>Entamoeba histolytica</i> (protozoan parasite)	<i>Shigella</i> spp. (Gram-negative bacillus)
Infective stage/dose	Mature quadrinucleate cyst	Very low infective dose (~10-100 organisms)
Core mechanism	Trophozoites invade colonic epithelium using proteolytic enzymes	Bacteria invade via M cells, spread cell-to-cell, trigger neutrophil-driven mucosal damage
Hallmark lesion/complication	Flask-shaped colonic ulcers; amoebic liver abscess	Colonic ulceration/microabscesses; Shiga toxin-associated HUS in severe cases
Stool findings	Trophozoites/cysts on microscopy; antigen/ PCR for confirmation	Numerous faecal leukocytes; culture for definitive diagnosis and susceptibility
First-line treatment	Tissue amoebicide (metronidazole/tinidazole) + luminal amoebicide (paromomycin)	Supportive care; antibiotics only for severe/high-risk cases, per local resistance
Vaccine available	No	No (candidates in development)

Key point

- Both diseases share the same core prophylaxis - safe water, sanitation and hand hygiene - because both are faecal-orally transmitted; the difference lies almost entirely in the organism, its mechanism of tissue damage, and the drugs needed to treat it.

Notes prepared for study purposes. Consult current primary literature and clinical guidelines for details required in formal citation or clinical practice.