

# Parasitology

## Protozoa

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*Dr. Usama Ali*

### Learning objective:

At the completion of this lecture, students will be able to:

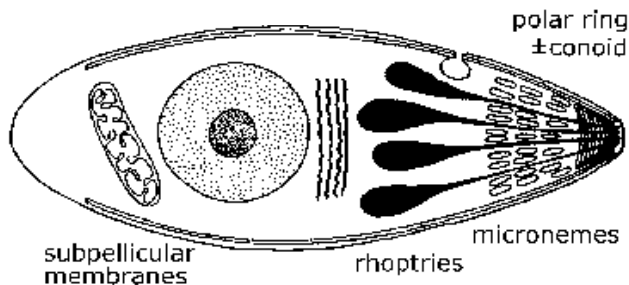
- ✓ Define apicomplexa phylum.
- ✓ Explain main differences among species.
- ✓ Make the diagnosis for each parasite.
- ✓ Explain their pathogenesis.
- ✓ Become familiar with treatment option of each parasite.

### Apicomplexa:

The phylum: **Apicomplexa** are a large group of protozoa, characterized by:

- ☒ The presence of an **apical complex (apicoplast)** that functions in the invasion of host cell.
- ☒ They are unicellular, spore-forming, and exclusively parasites of animals or human.
- ☒ Motile by **gliding motility**. Motile structures such as flagella or pseudopods are absent except in certain gamete stages.
- ☒ Is a diverse group including organisms such as coccidia (*Cryptosporidium* sp., *Isospora belli*, *Sarcocystis* and *Toxoplasma gondii*) gregarines, piroplasms and haemosporina (*Plasmodium* spp.).

### General Apicomplexan structures



# Coccidia

- ☒ Are microscopic, spore-forming, single-celled parasites.
- ☒ Are obligate, intracellular parasites (they must live and reproduce within host cells).
- ☒ **Coccidiosis is a parasitic disease of the intestinal tract of humans and animals**, caused by coccidian protozoa belong to four genera: *Cryptosporidium* sp., *Isospora belli*, *Sarcocystis* and *Toxoplasma gondii*.
- ☒ The disease spreads from one host to another by contact with infected feces, or ingestion of infected tissue.
- ☒ Diarrhea, which may become bloody in severe cases, is the primary symptom.
- ☒ Have a complex life cycle, involving both asexual (**schizogony** or **merogony**) and sexual (**gametogony** and **sporogony**) reproduction.
- ☒ The infective oocysts in species of *Isospora belli* and *Sarcocystis* produce two internal sporocysts, each with four sporozoites; in *Cryptosporidium* the sporocyst stage is omitted.
- ☒ Only two species of coccidian known to undergo schizogony and gametogony in man, viz., *Isospora belli* & *Cryptosporidium*.

## *Cryptosporidium* (cryptosporidiosis)

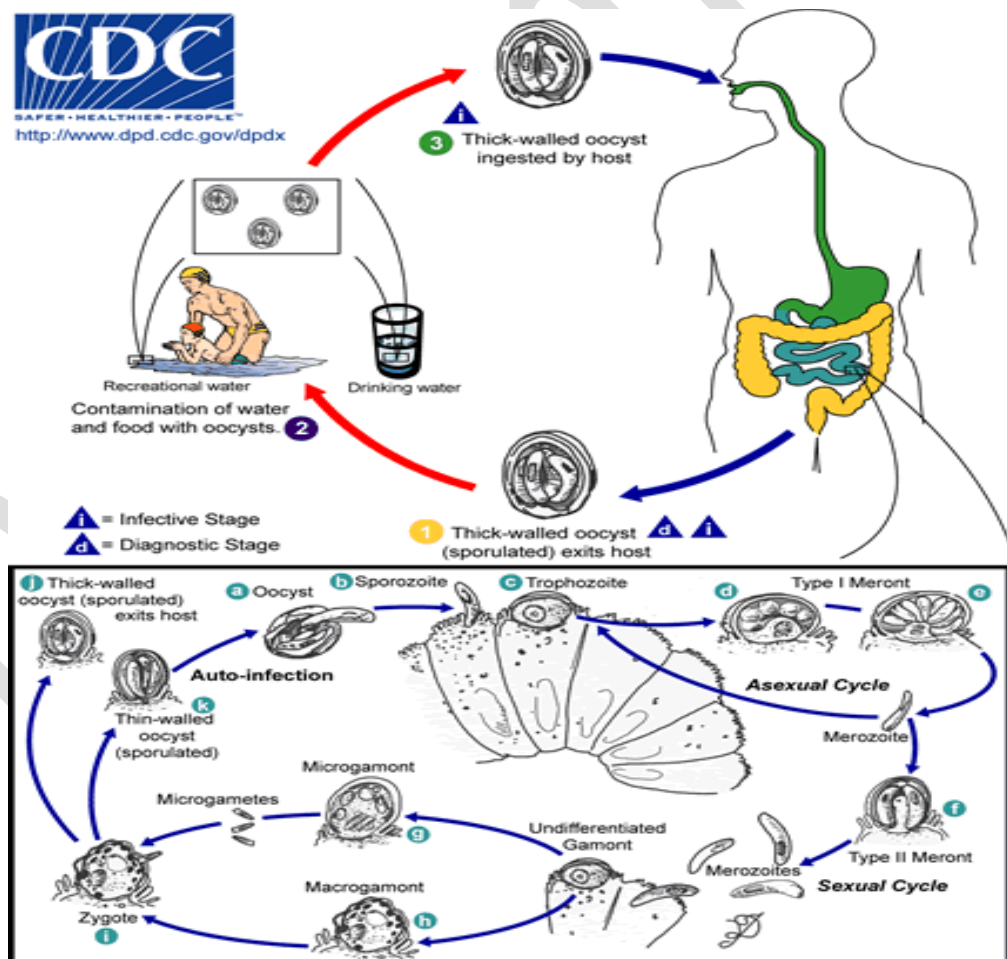
- ➞ The first human cases of cryptosporidiosis were reported in 1976.
- ➞ Initially it was believed to be a rare and exotic infection.
- ➞ Now recognized as a common human pathogen and a frequent cause of **diarrhea in humans**.
- ➞ In immunocompetent individuals this diarrhea is **self-limiting** and last about two weeks.
- ➞ The disease is quite serious and potentially life-threatening in immunodeficient patients (especially AIDS) and is characterized by a profuse watery diarrhea.

## Life cycle

All known stages of this parasite found in the **brush border** of the mucosal epithelium of stomach and intestine. *Cryptosporidium* is capable of completing its life cycle in one host (**monoxenous**). Sporulated oocysts, containing 4 sporozoites, are excreted by the infected host through feces and possibly other routes such as respiratory secretions ① Transmission of *Cryptosporidium parvum*, *C. muris* and *C. hominis* occurs mainly through contact with contaminated water (e.g., drinking or recreational water) or exposure to infected animals ② Following ingestion (and possibly inhalation) by a suitable host ③, excystation ④ occurs.

In contrast to other coccidia, *Cryptosporidium* sporozoites do not invade the enterocytes. Instead they induce an extension and fusion of the microvilli resulting in the parasite becoming surrounded by a double membrane of host origin. Even though the parasite is surrounded by host membranes it is not intracellular and is referred to as being 'extracytoplasmic'. A junction, called the '**feeder organelle**' or the '**adhesion zone**', forms between the parasite and the host enterocyte.

The parasite, now called a trophozoite, likely derives nutrients from the host cell via this junction. The trophozoites within the brush border of epithelial cells (b, c) of the gastrointestinal tract or other tissues such as the respiratory tract, undergo asexual multiplication (**schizogony** or **merogony**) (d, e, f) and then sexual multiplication (**gametogony**) producing **microgamonts** (male) (g) and **macrogamonts** (female) (h). Upon fertilization of the macrogametes by the microgametes (i), oocysts (j, k) develop that sporulate in the infected host. Two different types of oocysts are produced, the **thick-walled oocyst**, which is commonly excreted from the host (j), and the **thin-walled oocyst** (k), which is primarily involved in autoinfection. Oocysts are infective upon excretion, thus permitting direct and immediate **fecal-oral transmission**.



## Mode of Infection with *Cryptosporidium parvum*:

- Ingestion of thick-walled oocysts: In contaminated food or drink

(called heteroinfection).

By faeco-oral route (hand to mouth) in already infected patient (called external autoinfection).

- Thin-walled oocysts in intestinal lumen of already infected patient causes internal autoinfection.

## Pathogenesis:

The most common clinical manifestation of cryptosporidiosis is a mild to profuse watery diarrhea. This diarrhea is generally self-limiting and persists from several days up to one month. Recrudescences are common. Abdominal cramps, anorexia, nausea, weight loss and vomiting are additional manifestations which may occur during the acute stage. The disease can be much more severe for persons with AIDS which manifests as a chronic diarrhea lasting for months or even years.

Diarrhea can have **osmotic**, **inflammatory**, or **secretory** components.

- Osmotic diarrhea** : epithelia malfunction [epithelial cells damaged or killed:] which lead to:
  - Impaired absorption**: villus atrophy (blunting)  $\longrightarrow$  ( $\downarrow$   $\text{Na}^+$  absorption) and  $\uparrow$  intercellular permeability.
  - Enhanced secretion**: crypt cell hyperplasia  $\longrightarrow$   $\uparrow$   $\text{Cl}^-$  secretion.
- Inflammatory diarrhea** [inflammation in lamina propria]  $\longrightarrow$  mucosal invasion and leukocytes in stools. This phenomenon could also contribute to the secretory process via cytokines and neurohormones.
- Secretory diarrhea** (toxin associated & watery): The watery nature of the diarrhea has suggested the presence of an **enterotoxin**. However, there is **no evidence** for a **toxin-mediated secretory diarrhea** despite efforts to identify such a toxin.

**Diagnosis**: stool examination for detection of Oocysts.

**Treatment**: In immunocompetent persons is self-limited diarrhea. Rapid loss of fluid managed by fluid and electrolyte replacement.

## **Epidemiology:**

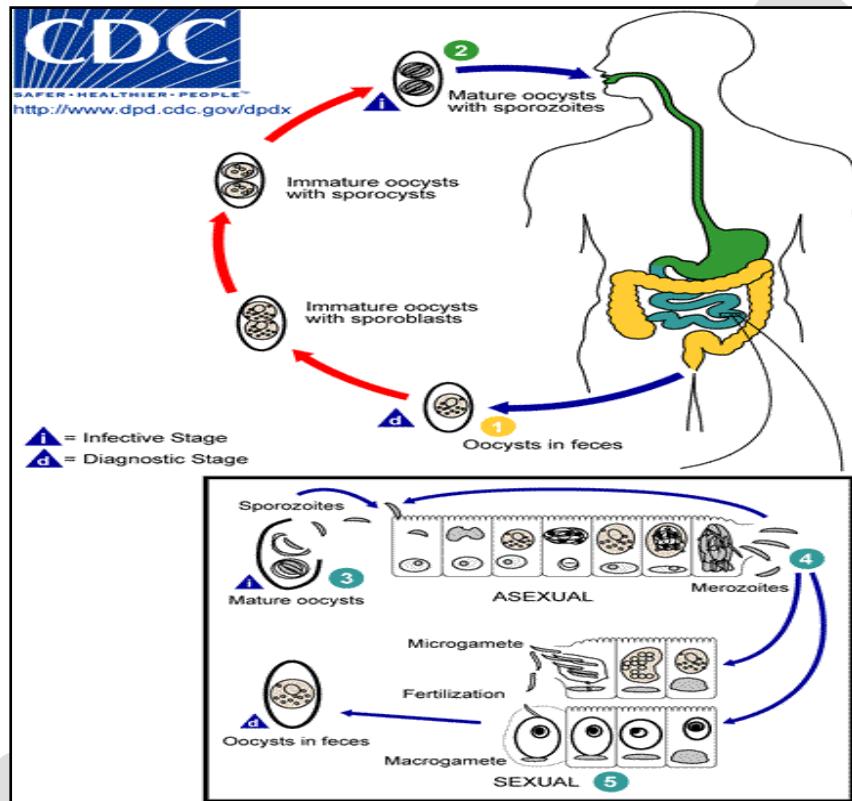
- Fecal- oral disease.
- two distinct transmission cycles in humans involving two different populations of *Cryptosporidium*:
  - 1) An exclusively anthroponotic (i.e., human-to human) cycle caused by genotype 1 (or *C. hominis*) and
  - 2) A zoonotic cycle caused by genotype 2 (or *C. parvum*).
- **Factors Favoring Waterborne Cryptosporidiosis:**
  1. small size of oocysts (4-5 mm)
  2. reduced host specificity and monoxenous development
  3. close associations between human and animal hosts
  4. large number of oocysts excreted (up to 100 billion per calf per day)
  5. low infective dose (<30)
  6. robust oocysts; resistant to chlorine

**Control:** Improvement in sanitary and hygienic conditions is indicated.

## *Isospora belli* (coccidiosis belli)

- ☒ *I. belli* is a rare infection of normal humans.
- ☒ The infection occurs via the oral-fecal route.
- ☒ Infects the epithelial cells of S.I. and produces self limiting diarrhea in normal individuals.
- ☒ It has worldwide distribution but more common in tropical regions and areas with poor sanitation.

### Life cycle:



At time of excretion, the immature oocyst contains usually one sporoblast (more rarely two) <sup>1</sup>. In further maturation after excretion, the sporoblast divides in two sporoblasts. The sporoblasts secrete a cyst wall, thus becoming sporocysts; and the sporocysts divide twice to produce four sporozoites each <sup>2</sup>. Infection occurs by ingestion of sporocysts-containing oocysts: the sporocysts excyst in the small intestine and release their sporozoites, which invade the epithelial cells and initiate schizogony <sup>3</sup>. Upon rupture of the schizonts, the merozoites are released, invade new epithelial cells, and continue the cycle of asexual multiplication <sup>4</sup>. Trophozoites develop into schizonts which contain multiple merozoites. After a minimum of one week, the sexual stage begins with the development of male and female gametocytes <sup>5</sup>. Fertilization results in the development of oocysts that are excreted in the stool <sup>1</sup>

**Pathogenesis:**

Infection causes acute, non bloody diarrhea with crampy abdominal pain, which can last for weeks and result in malabsorption and weight loss. In immunodepressed patients, and in infants and children, the diarrhea can be severe. Eosinophilia may be present (differently from other protozoan infections).

**Diagnosis:** Microscopic demonstration of oocysts in stool examination.

**Treatment:** Trimethoprim is the drug of choice.

**Control:** Improvement in sanitary and hygienic conditions is indicated.

*End of lecture*