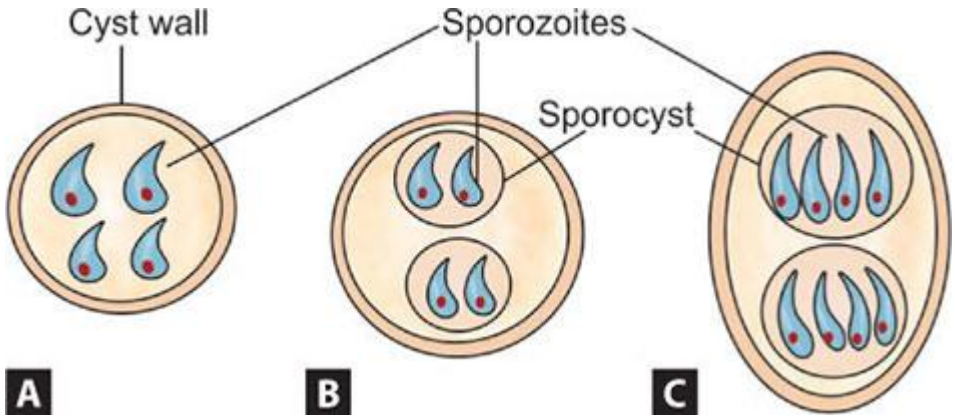
Sporozoa—II(Eimeriida)

2. *cryptosporidium parvum*

- *Cryptosporidium* is an intestinal coccidian parasite affecting various animals and men. It causes self limiting acute diarrhea in immunocompetent healthy individuals; where as it is an opportunistic pathogen in immunocompromised patients (including HIV infected patients), causing chronic persistent life threatening diarrhea
- Tyzzer (1907) was the first to describe it in gastric crypts of laboratory mice. Subsequently it was found to affect many animals like rats, guinea pigs, pigs, horses, etc. The first human case was reported in 1976.
- It belongs to the family Cryptosporidiidae. It is different from other coccidian parasites in such a way that it doesn't go deep into the host cells, but is confined to an **intracellular extra cytoplasmic location**.
- All the sexual and asexual stages of development take place within a **parasitophorous vacuole** that lies just below the cell membrane of the brush border epithelium of the small intestine.



Figs 7.5A to C: Sporulated oocysts (schematic diagram) of (A) *Cryptosporidium*; (B) *Cyclospora*; (C) *Isospora*.

Life Cycle (Fig. 7.6)

C. parvum completes its life cycle (both sexual and asexual stages) in single host (man or other animals).

Infective stage: Sporulated oocyst is the infective form of the parasite. Thick walled oocyst is infectious to other persons, where as the thin walled oocysts can cause autoinfection (through contaminated fingers).

Mode of Transmission: Man acquires infection by:

- Ingestion of food and water contaminated with feces containing thick walled oocysts
- **By autoinfection:** Thin walled oocyst can infect the same host.

Development in Man

Excystation: In the small intestine, the suture present in the inner wall of the oocyst gets dissolved and four slender crescent shaped sporozoites are released from each oocyst. Various factors like pancreatic enzymes and bile salts help in excystation

Invasion: Sporozoites invade the brush border epithelium of the small intestine and lie inside a parasitophorous vacuole near the microvilli surface, within which all the stages of development take place

Schizogony:

- The sporozoites subsequently differentiate into trophozoites which then undergo asexual multiplication (schizogony) to produce type I meronts
- Each type I meront undergoes schizogony to release eight merozoites, which then again invade the adjacent enterocytes and undergo repeated schizogony to produce type II meronts
- Four merozoites are released by the schizogony of each type II meront

Gametogony:

- The merozoites undergo gametogony and transform into sexual forms (microgamont and macrogamont) Each microgamont releases 16 microgametes while only one macrogamete is produced from each macrogamont

Sporogony:

- Fertilization takes place between microgamete and macrogamete to produce the zygote
- ✓ Subsequently, about 80% of zygote transform into highly resistant double layered thick walled oocyst and remaining 20% transform into single layered thin walled oocyst
- ✓ Sporulated oocysts are excreted in the feces. Thick walled oocyst infects the new hosts where as the thin walled oocysts infect the same host (autoinfection).

Epidemiology

Cryptosporidiosis is a zoonotic disease.

Pathogenesis and Clinical features

- **Attachment:**

Sporozoites attach to the brush border epithelium of the small intestine with the help of a unique protein called as **CP47** (47 kDa *C.parvum* protein)

- **Penetration:**

1. Discharges from the apicomplex (rhoptries, micronemes and dense granules) present in the anterior end of the sporozoites help in invasion
2. the parasite activates the host cell kinase signaling pathway that liberates proinflammatory cytokines like tumor necrosis factor (TNF)-α, interleukine (IL)-8, prostaglandins, etc

3. Cytokines released from the inflammatory site can activate the phagocytes; attract fresh leukocytes which in turn liberate soluble factors
4. These factors increase intestinal secretion of chloride and water and decrease the sodium absorption coupled to glucose transport.

Symptoms:

- Usually the infection is asymptomatic
- Sometimes, patient develops self-limiting watery nonbloody diarrhea
- Other features like abdominal pain, nausea, anorexia, fever, and/or weight loss may be present
- Symptoms develop after an incubation period of 1 week and subside within 1–2 weeks
- *C. parvum* accounts for 2–6% of cases of traveler's diarrhea.