Introduction
Definition
Balantidiasis is infection by *Balantidium coli*, the largest protozoan parasite and the only ciliate parasite known to infect humans. Malmsten first described the organism in 1857 in 2 patients with severe diarrhea; one of these infections was fatal.1

Synonyms
Balantidiasis is also known as balantidiosis, balantidial dysentery, and ciliary dysentery.

Epidemiology
*Balantidium coli* is endemic worldwide. It is most prevalent in temperate and tropical regions, but has been reported in Norway, Sweden, Finland, and northern Russia.2 This ciliate parasite inhabits a variety of hosts, especially primates.3 Humans are most commonly infected by contact with infected pigs.4 In some pig-raising areas of New Guinea, human infection rates are as high as 28%.5,6 An outbreak of balantidiasis on the Pacific island of Truk in 1971 led to 110 human infections.7 Human-to-human transmission can occur when personal hygiene is poor, especially among institutionalized populations.8 Nonhuman primates are another source of infection. *Balantidium coli* has been reported in numerous primate species, including orangutans, chimpanzees, gorillas, and Old and New World monkeys.3,9,10 Other hosts for *B. coli* include rats, fowl, turtles, and cockroaches. Humans have been reported to be asymptomatic carriers of *B. coli*.4

Figure 15.1
*Balantidium coli* trophozoite in colon. Note cilia (ci), cell membrane (cm), large macronucleus (ma), and large contractile vacuole (cv). x570
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Morphologic Description

The trophozoite of *B. coli* is 50 µm to 200 µm by 40 µm to 70 µm; in tissue sections most are 40 µm to 80 µm by 25 µm to 45 µm. They are ovoid, with a cell membrane covered with uniform cilia (Figs 15.1 and 15.2). Near the anterior end is the funnel-shaped peristome, a ciliated area surrounding the cytostome (mouth) (Figs 15.3 and 15.4). Residual products are eliminated through the cytopyge, a small triangular opening at the posterior end. The granular cytoplasm contains food and usually 1 or 2 contractile vacuoles (Fig 15.1). The most obvious structure within the cytoplasm is the large, often kidney-shaped macronucleus (Fig 15.5). The tiny micronucleus lies within the concave depression of the macronucleus (Fig 15.6). The micronucleus is not observed in hematoxylin and eosin stained sections, but may be identified using special stains such as Brown-Hopps, Movat, and Masson. Unusual forms, possibly undergoing division, conjugation, or apoptosis, may rarely be seen (Figs 15.7 and 15.8). Precysts are round, ciliated, and have a peristome (Fig 15.9). Cysts, which are not observed in histologic sections of colon, are spherical and vary from 45 µm to 75 µm in diameter. They have a large macronu-
ucleus (Fig 15.10) and a tiny micronucleus which is rarely observed (Fig 15.6). Cysts may also contain cytoplasmic inclusions, cellular debris, mucus, and food vacuoles (Fig 15.11). The ultrastructural and molecular characterization of *B. coli* have been reported but the molecular nature needs more study to be a useful tool for identification.11

**Life Cycle and Transmission**

Trophozoites of *B. coli* usually inhabit the colon of the host. They divide asexually by transverse binary fission of the micronucleus, the macronucleus, and finally the cytoplasm. Rarely, they reproduce sexually by conjugation. Trophozoites encyst during transport through the intestine or externally in soft stool (Fig 15.12). In this process, a trophozoite becomes round, partially retracts its cilia, and secretes a cyst wall. Cysts can remain viable for several days in stool. The parasite excysts after it is ingested by another suitable host. Humans are infected by ingesting cysts, the infective stage of *B. coli*, in contaminated water or food. Pigs and rats are the most important reservoir hosts for *B. coli*.

**Clinical Features and Pathogenesis**

*Balantidium coli* may inhabit the bowel lumen without invading tissue or provoking clinical symptoms. Parasites that invade tissue do so by mechanical action of the cilia and by lytic action, particularly in patients weakened by underlying factors such as malnutrition or immunosuppression.12

Balantidiasis can mimic intestinal amebiasis. The acute form of the disease is marked by rapid onset of diarrhea or dysentery, with 20 bowel movements or more per day.13 Other frequent complaints are abdominal colic, tenesmus, nausea, and vomiting. Chronic balantidiasis produces intermittent diarrheal episodes alternating with normal bowel movements or constipation. Patients may occasionally have headache, insomnia, anorexia, weight loss, or muscular weakness. *Balantidium coli* may cause appendicitis and lung involvement, and has been attributed to urinary tract disease.14-17 Extra-abdominal balantidiasis occasionally develops in patients with cancer or post-organ transplantation.18-20 Peripheral eosinophilia is not a feature of balantidiasis.
Pathologic Features

Parasites usually invade the colon and appendix. Invasion of the ileum however, has been reported. Early lesions appear as flask-shaped ulcers a few millimeters in diameter, similar to those seen in intestinal amebiasis. Ulcer borders are frayed, swollen, and undermined (Fig 15.13). The surface is covered with a thick, friable, adherent mucous layer. Ulcers may be superficial or may perforate the intestinal wall (Fig 15.14), releasing parasites into the peritoneal cavity and causing peritonitis, and sometimes death. Extraintestinal infections involving the liver, vagina, ureter, bladder, and lung have been reported but are extremely rare.

Microscopically, coagulation necrosis containing trophozoites of *Balantidium coli* forms the base of the ulcer (Fig 15.15). Trophozoites stain well with hematoxylin and eosin and are readily seen within the ulcer (Figs 15.16 to 15.19). The macronucleus stains black and, although it may vary considerably in shape, a kidney-shaped configuration is diagnostic (Fig 15.5). Trophozoites are usually more numerous at the periphery of the ulcer than in the necrotic center. There is edema of adjacent tissues, with infiltrates of chronic inflammatory cells, primarily lymphocytes and plasma cells (Fig 15.20). Neutrophils are insignificant unless there is secondary bacterial infection (Fig 15.21). Eosinophils have been reported, but are not a common finding.

Diagnosis

Diagnosis is established by identifying *Balantidium coli* trophozoites or cysts in feces or scrapings from intestinal lesions. Only trophozoites are seen in biopsy specimens of infected tissues (Fig 15.1). Trophozoites usually appear in diarrheic stools; cysts appear in more solid stools. Trophozoites are actively motile in wet mounts of fresh feces. The macro-
nucleus stains well with iron-hematoxylin on smears from fresh stool (Fig 15.22). Diagnosis can also be made by identifying trophozoites in surgical, cytological, and autopsy specimens (Figs 15.16 and 15.17). Special care must be taken in differentiating *B. coli* from ciliocytophthoria in nasopharyngeal specimens.26
Treatment and Prevention

The drugs of choice for balantidiasis are tetracycline (for adults: 500 mg 4 times/day for 10 days), or iodoquinol (650 mg 3 times/day for 20 days), metronidazole (500 mg 2 times/day for 5 days); other drugs such as ampicillin, carbasone, diodoquin, nitrimidazine, and paromomycin have been used with varying results.22,21,28 Effective sanitation and use of water from protected sources are the most useful measures of protection. Limiting exposure to pigs is helpful but often impractical.29,32

References


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15.12 Drawing by Frank O. Raasch