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TAPEWORMS (CESTODIASIS)

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Nearly all of the cestodes, or tapeworms (class Cestoda in the phylum Platyhelminthes, the flatworms), are parasitic as adults in the intestinal tract of vertebrates. Similar to other flatworms, they are bilaterally symmetric, usually flattened dorsoventrally, and lack a body cavity.[6, 46] Each tapeworm is a string of individuals having a complete set of reproductive organs in progressive degrees of maturity and budding off from a body attached to the host tissue by a head or scolex. Tapeworms consist of a head, or scolex; a neck, or germinal region; and a segmented body (strobila). The scolex may be equipped with a variety of holdfast organs (e.g., rounded suckers, hooks, or elongated grooves) used to maintain the worm's position in the gut. The undifferentiated neck gives rise to a linear sequence of sets of male and female reproductive organs (the proglottids, or segments) that become progressively more mature as they are moved distally by differentiation of younger proglottids in the neck. Subsequent enlargement of segments is chiefly due to the development and maturation of enormous numbers of eggs—up to 100,000 per proglottid in *Taenia* species. The result is a chain of segmentally arranged sexual units, each ultimately filled with eggs within a coiled or branched uterus. Also located within the tapeworm body are transverse, circular, and longitudinal muscles; a primitive nervous system consisting of central ganglia in the scolex and paired longitudinal nerves extending the length of the worms; and osmoregulatory, or excretory, canals connected by a series of transverse connecting tubules. Cestodes lack a digestive tract at any stage of the life cycle; thus, the body covering, or tegument, must serve not only as a protective coating, but also as a metabolically active layer through which nutritive material can be absorbed and secretions and waste materials transported. The tegument is a syncytial anuclear surface layer covered with minute projections, called microtriches, responsible for greatly increasing the absorptive surface, which usually lies in intimate contact with the host intestinal villar microtubules.

Tapeworms are hermaphroditic. A sexually mature proglottid can copulate

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with itself (self-fertilization) or with others in its strobila or can outcross with proglottids of other worms. In terminal proglottids, when development of eggs is completed, the egg-filled segments are called ripe or gravid. In some species (e.g., *Taenia*), these terminal proglottids detach (apolysis) and pass in the feces as segmented packets that may even retain independent motility. In other groups, the ripe segments degenerate, and the released eggs are mixed with the feces (e.g., *Hymenolepis nana*). In groups involving an aquatic life cycle (e.g., *Diphyllobothrium*), eggs are discharged continuously through a uterine pore. The life cycles of all but one cestode are indirect (i.e., they require development in one or more intermediate hosts before development can occur in the final host). The single exception is *H. nana*, a parasite of humans and rodents that is able to complete all stages of development in a single host, although it retains its ability to undergo indirect developmental cycles as well (Table 1).

Tapeworms of medical importance belong to two large orders: Pseudophyllidea and Cyclophyllidea. These differ in general morphologic and biologic characteristics.[6] Most infect human beings in the adult stage, causing intestinal cestodiasis; humans are the obligatory final hosts of two species (*Taenia saginata* and *Taenia solium*) and are incidentally involved in the life cycles of others (accidental zoonotic infections). Cestodes occurring in the intestinal tract of humans can be differentiated and speciated based on differences in morphologic characteristics of the scolex, proglottids, and, in most cases, the eggs (Figs. 1 and 2); these diagnostic considerations have been reviewed.[17] The larval or metacestode stages of a few species infect and invade human tissues (e.g., *Echinococcus granulosus* and certain species of *Taenia*); these diseases are discussed elsewhere in this issue.

TAENIASIS (BEEF AND PORK TAPEWORMS)

Taeniasis is intestinal infection by adult tapeworms of the genus *Taenia*, either *T. saginata*, the beef tapeworm, or *T. solium*, the pork tapeworm.

Morphology and Biology

Adult *T. solium* and *T. saginata* are 3- to 10-m, white or yellowish segmented cestodes that attach to the wall of the small intestine by a pinhead-sized scolex. In both species, the scolex has four circular muscular suckers; the *T. solium* scolex also has a double ring of 72 to 30 anterior movable hooks (see Fig. 1). Both species have a short neck (3 to 7 mm), where growth and segment differentiation occur, and a long strobila (4 to 10 m in *T. saginata* and 1 to 3 m in *T. solium*) that consists of a chain of several thousand sexually maturing, mature, and gravid proglottids. The gravid proglottids comprise the posterior portion of the worm; these terminal proglottids, which are broken off and shed in the feces, are rectangular, 20 × 5 to 7 mm in *T. saginata* and 12 × 5 in *T. solium* (see Fig. 1).

Taeniasis is acquired by ingestion of raw or poorly cooked meat that contains living cysticerci (*measly* beef in *T. saginata* and *measly* pork in *T. solium* infections) (Fig. 3). Cysticerci are the larval or metacestode forms (formerly called *Cysticercus bovis* for *T. saginata* and *C. cellulosae* for *T. solium*) and are oval, translucent cysts with clear fluid, approximately 5 × 10 mm, with an opaque, invaginated protoscolex (the future scolex) visible within. Humans are the only naturally susceptible final host for both species of *Taenia*. After ingestion by the

Table 1. CLINICAL AND EPIDEMIOLOGIC CHARACTERISTICS OF ADULT CESTODES OF THE HUMAN GUT

Agent	Intermediate Hosts	Definitive Hosts	Prevalence in Humans	Geographic Distribution	Clinical Seriousness of Human Infection
Taenia saginata	Cattle	Only human beings	50 million worldwide; increasing in Europe since WWII	Widely distributed throughout world	Generally mild; complications rare
Taenia solium	Swine, human beings, dogs	Only human beings	5 million worldwide; decreasing in Europe	Latin America, Asia, Africa	Intestinal taeniasis mild; cysticercosis may present serious manifestations
Diphyllobothrium spp.	Copepods; fresh water and anadromous fish	Humans, dogs, and other fish-eating mammals	20 million worldwide; decreasing	Widely distributed throughout world	Generally mild; rarely causes serious B_{12} deficiency
Hymenolepis nana	Intermediate host optional; insects (fleas or beatles or none)	Humans, rodents	Most common cestode of humans	Widely distributed	Often asymptomatic or mild
Hymenolepis diminuta	Grain beetles and other insects	Rats	Sporadic; may be locally common	Widely distributed	Generally mild
Inermicapsifer spp.	Unknown; probably arthropods	Rodents	Rare	Africa, Caribbean, South America	Mild
Raillietina spp.	Insects	Rodents and birds	Rare except focally	Southeast Asia, Latin America	Mild
Dipylidium caninum	Flea	Dogs, cats, and humans	Rare	Widely distributed	Mild
Bertiella spp.	Oribatid mites	Monkeys	Rare	Kenya and Southern Asia	Mild
Mesocestoides spp.	First stage host unknown; rodents or reptiles for second larval stage	Mammals and birds	Rare	Africa, Asia, and elsewhere	Mild

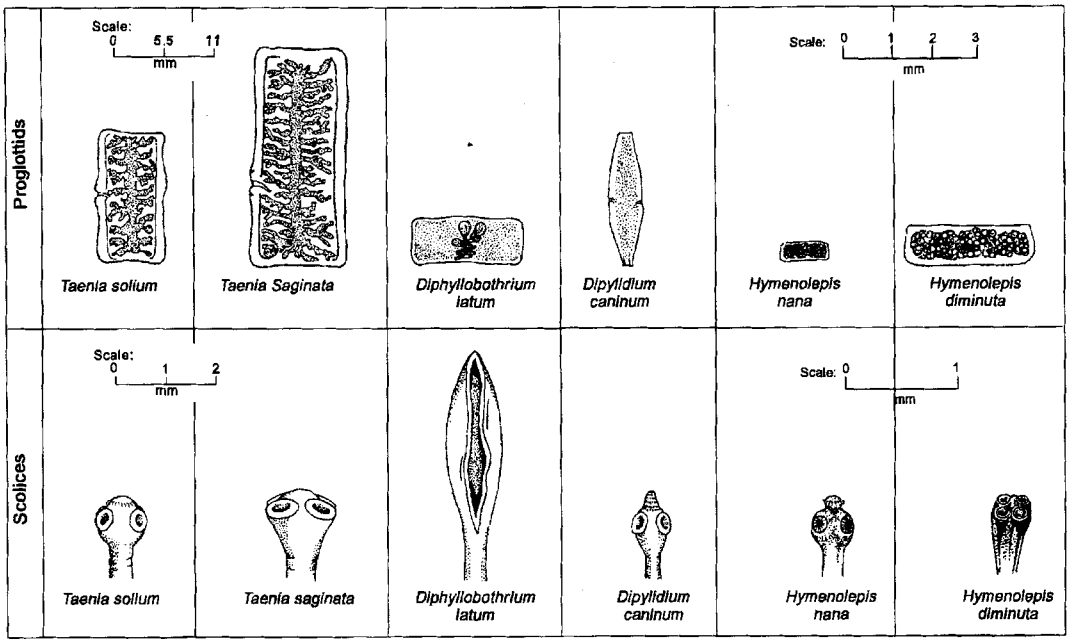

Figure 1. Cestode eggs found in human stool specimens.

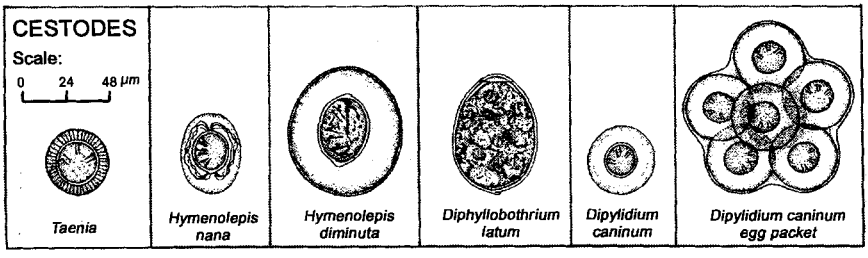

Figure 2. Gravid proglottids and scolices of cestode parasites of humans.

host, the cysticercus evaginates in the human small intestine and grows to become a fully differentiated adult tapeworm in about 2 to 4 months.

Infection is usually single in *T. saginata* but occasionally multiple with *T. solium*. The tapeworm attaches by the scolex to the intestinal mucosa of the jejunum, then lies along the mucosa and follows the small intestine loops for much of its length. Terminal segments and free eggs are shed periodically, in *T. saginata* perhaps 10 to 15 segments a day, but fewer and more irregularly in *T. solium*, and pass through the ileocecal valve into the large intestine and then are evacuated. *T. saginata* segments tend to crawl spontaneously out of the anus or away from the fecal bolus (adapted to the open grazing habit of cattle), whereas *T. solium* segments rarely crawl and remain in the feces (adapted to the coprophagic habit of pigs?). Each segment carries 50,000 to 100,000 eggs. The eggs of both *Taenia* species are indistinguishable: spherical, 30 to 40 μm, with a double-walled membrane with radiating striae (see Fig. 2). The six-hooked embryo, or oncosphere, is usually visible within the egg. The eggs are infective for the intermediate host as soon as they are released from the host, and when ingested by a susceptible intermediate host (cattle for *T. saginata*, pigs for *T. solium*), they "hatch." The hexacanth embryos emerge from the eggs, penetrate the intestinal wall aided by their six hooklets, and pass by blood or lymph channels to reach muscle, subcutaneous tissue, or other organs, where they grow and develop into cysticerci. These forms can live for several years, after which they degenerate and become fibrotic and calcified. In the human small bowel, the embryo of *T. solium* released from eggs can invade the bowel wall and disseminate hematogenously to muscle, subcutaneous tissues, central nervous system, and other tissues producing cysticercosis by autoinfection. How frequently this occurs is not known; from 5% to 40% of patients diagnosed with cysticercosis have concurrent taeniasis,[25, 32] and virtually all *T. solium* tapeworm carriers develop antibodies to larval antigens (Tsang VCW: unpublished data, 1995). Human cysticercosis by larval *T. saginata* does not occur.

Epidemiology

Occurrence and prevalence of *T. saginata* and *T. solium* infection is related to local habits of eating raw or undercooked beef or pork. *T. saginata* is a cosmopolitan parasite, with regions of high endemicity in Latin America, Africa, the Middle East, and Central Asia; in some populations sampled in East Africa, prevalence exceeded 50%.[34] Beef tapeworm prevalence is moderate in Europe, South Asia, Japan, and the Philippines and low in Australia and North America.

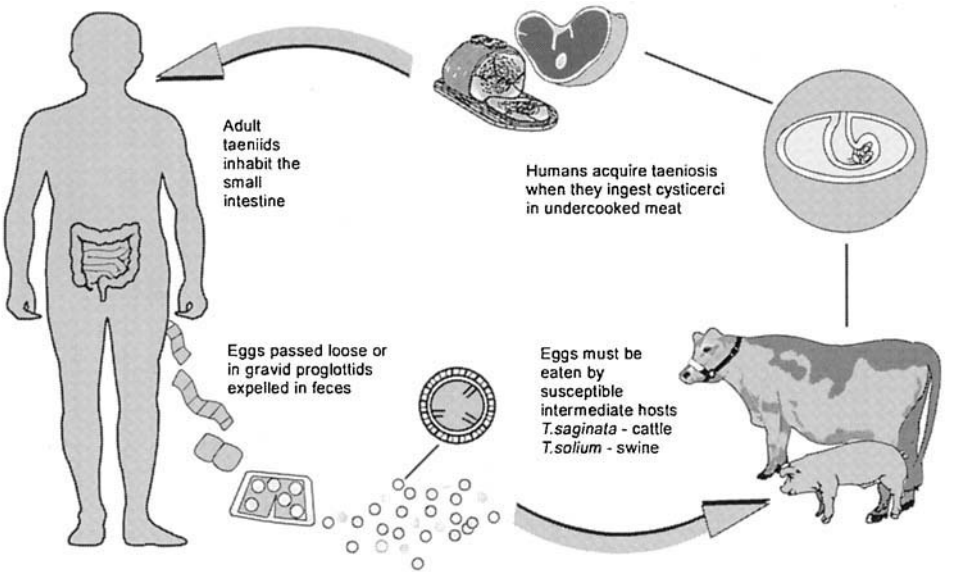

Figure 3. *Taenia* spp: life cycle.

In the United States, *T. saginata* cysticercosis is sporadic and at low prevalence in cattle,[43] *Taenia* eggs were found in less than 0.1% of 216,000 stool specimens examined at state health laboratories in 1988.[26]

T. solium also occurs in much of the world but is generally less common and more focal than *T. saginata*. The tapeworm is particularly prevalent in most of Latin America, the Slavic countries, Africa, Southeast Asia, India, and China. Prevalence is low or the parasite is declining in parts of northwestern Europe. In the United States and Canada, locally acquired *T. solium* is rare or absent, although increasing numbers of imported cases of both taeniasis and cysticercosis have been reported.[10, 44]

Persons of all ages and both sexes are susceptible, and age of exposure is mainly determined by the age at which raw meat consumption begins; in Mexican rural communities, *T. solium* taeniasis is reported with similar frequency in all age groups greater than 2 years of age.[39, 40]

Human fecal contamination of the environment is a crucial factor in sustaining the life cycles of *Taenia* species. Cattle become infected with *T. saginata* by grazing on ground or ingesting water or roughage contaminated with eggs from human feces. Indirect contamination of pasture by insufficiently processed human sewage may also be important. Pigs become infected with *T. solium*, often massively, because of their coprophagic habits.

T. saginata larval infection may also occur in other domesticated bovines, including the water buffalo and yak, but it rarely occurs in other ungulates. *T. solium* larval infection can occur also in domestic dogs, cats, and sheep.

Clinical and Pathologic Manifestations

Taenia species reside loosely within the intestinal lumen; other than minor local mucosal inflammation at the site of attachment of the scolex, there is little physiopathologic alteration of the gut. Adult *Taenia* are weakly immunogenic and may induce a moderate eosinophilia and increased levels of serum gamma immunoglobulin E (IgE).[5] Rare acute complications may occur, more commonly with *T. saginata* than *T. solium*, following migration of proglottids to unusual sites, such as the appendix or pancreatic and bile ducts.

Many, perhaps most, tapeworm carriers are asymptomatic and become aware of the infection only as they notice proglottids passed with feces or the disconcerting sensation caused by the spontaneous movement of *T. saginata* segments through the anus. Mild gastrointestinal symptoms may occur in some patients, including nausea and vague epigastric or periumbilical pain. Other clinical findings occasionally attributed to the worms include anorexia (or increased appetite), weight loss, headaches, convulsions, and allergic symptoms (urticaria, pruritus, other skin disorders), but these symptoms are sometimes associated with concomitant infection with other intestinal parasites or other potential causes. Cysticercosis is a potentially serious complication of *T. solium* infection.

Diagnosis

In many diagnosed cases, especially of *T. solium* infection, the patient recognizes infection incidental to elimination in the feces of individual or short chains of motile tapeworm segments. The patient's description is usually sufficient for a tentative diagnosis of taeniasis, but confirmation of species is advis-

able for evaluation of the possible risk of cysticercosis. Proglottids eliminated spontaneously or after treatment should be collected in water or saline solution, using strict precautions to avoid contamination. The standard method for differentiating the species is to count the number of primary uterine branches in gravid proglottids: *T. saginata* has 12 or more, and *T. solium* has 10 or less (see Fig. 1). The uterine structure is usually well visualized by pressing the gravid proglottid between two microscope slides; however, India ink injected into the lateral genital opening further distinguishes the uterine branches. In the absence of gravid proglottids, stained mature proglottids show two differences: a three-lobed ovary in *T. solium* and a vaginal sphincter in *T. saginata*.[17]

The lack of sensitive methods for diagnosis of intestinal *Taenia* infections has been a major factor in limiting clinical and epidemiologic studies. Microscopy is known to be a relatively insensitive technique because eggs are periodically absent from feces during infection. Egg concentration techniques, such as that of Ritchie,[37] are generally considered to be the most sensitive coproparasitologic test, although few definitive comparative studies of egg detection methods have been carried out. Visual demonstration of *Taenia* eggs in feces is not specific because *T. solium* and *T. saginata* eggs appear identical under the light microscope. Microscopic diagnosis can be supplemented with questioning of individuals to determine if they are aware of passing proglottids[21]; however, false-positive and false-negative reports of infection are sometimes made. Mass treatment and identification of tapeworm fragments in subsequent bowel evacuations has also been employed as a means of determining infection,[11, 21] although significant numbers of false-negative reports occur. A variety of immunologic and molecular techniques have been employed in an attempt to improve diagnosis. Intradermal tests have shown problems with both sensitivity and specificity.[29, 36] Molecular techniques such as protein analysis of proglottids and DNA probes for eggs or other worm material have been developed to differentiate the species of *Taenia* present in an infection,[8, 16, 23] but the periodic absence of eggs or proglottids from feces limits sensitivity. Detection of *Taenia*-specific antigens in host feces may be the most sensitive diagnostic technique. Coproantigen (CoAg) assays are based on capture-type enzyme-linked immunosorbent assays (ELISAs) with polyclonal antisera raised against either worm somatic[3, 4, 28] or excretory-secretory products.[12, 13] Diagnosis by CoAg assay of individuals from egg-negative fecal samples has been demonstrated, as have high levels of specificity, at least to genus level. Extensive field studies in Mexico, Guatemala, and China have shown that a CoAg test detects up to 2.5 times as many cases of taeniasis as microscopy[4] (and unpublished). Unfortunately, CoAg assays are still only research tools and not widely available for clinical use.

DIPHYLLOBOTHRIASIS (FISH TAPEWORM INFECTION)

Diphyllobothriasis is infection of the intestinal tract by the fish tapeworms, *Diphyllobothrium* species. Most patients are asymptomatic or mildly symptomatic, but a small proportion may develop megaloblastic anemia.

Morphology and Biology

Diphyllobothrium species are the only pseudophyllidean cestodes that commonly use humans as definitive hosts. *D. latum* is the most common and widely studied species; however, numerous other species may also infect humans.[7]

Adult *D. latum* ranges from less than 1 m long to 12 m or more; the largest worms have thousands of proglottids. Other *Diphyllobothrium* species are smaller, rarely more than 1 m long. The scolex is finger-shaped and has dorsal and ventral sucking grooves (see Fig. 1). Proglottids are usually wider than they are long. There are numerous vitelline follicles scattered in the outer layer, whereas the testes lie in a central layer in the proglottid (see Fig. 1). Male and female genital pores open midventrally. The bilobed ovary is near the rear of the segment. The uterus consists of short loops and extends from the ovary to a midventral uterine pore from which eggs are continuously discharged. The ovoid eggs measure about 60 × 40 μm and have a lidlike operculum at one end and a small knob on the other (see Fig. 2).

Maintenance of the life cycle of *Diphyllobothrium* species requires that feces of infected hosts be discharged into freshwater that contains susceptible crustaceans and fish and that the infected fish be eaten raw by definitive hosts (Fig. 4). The three required hosts can be supplemented by additional species of fish serving as paratenic hosts that infect humans and other fish-eating hosts. After the egg hatches, the motile embryo (coracidium) is ingested by minute crustacea, *water fleas* (*Cyclops* and *Diaptomus*), in which the first-stage larva (procercoid) develops. When the procercoid is ingested by the second intermediate host—a fish—further development leads to the plerocercoid or *sparganum* larva, which is infective for the final host. The site of localization of the plerocercoid in fish differs with species of *Diphyllobothrium* and, to some extent, with species of fish. When the final host ingests the *Diphyllobothrium* plerocercoid, however, the worm remains in the gut, the larval portion of the body is shed, and the adult worm develops to maturity in the small intestine. Within 3 to 5 weeks, egg

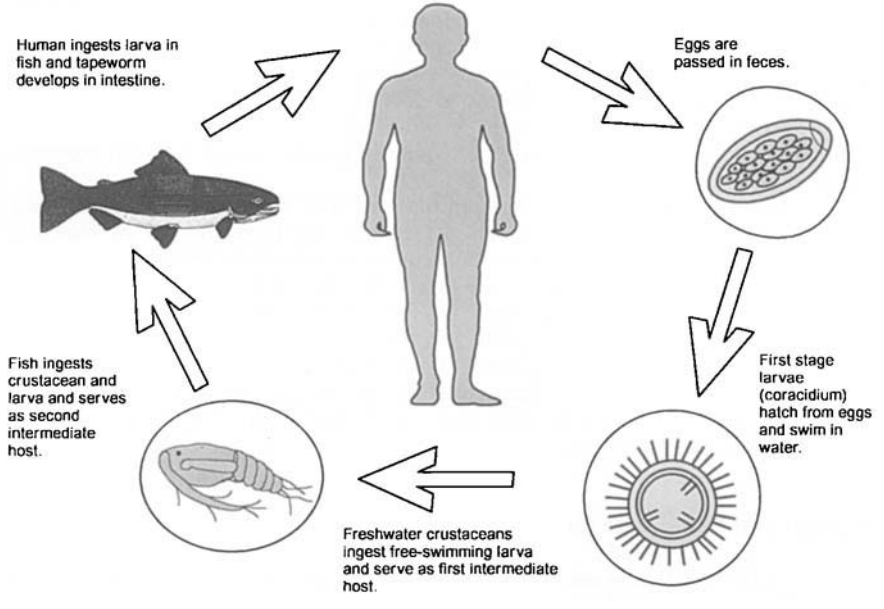

Figure 4. *Diphyllobothrium* spp: life cycle.

production is initiated; a million or more eggs may be passed daily. Adult *Diphyllobothrium* may survive 10 years or more.

Epidemiology

D. latum is a common human infection in northern Europe (Finland, East Prussia, Russian Karelia).[1] In North America, *D. latum* infections have been reported in Eskimos in western Alaska, where the infection was apparently introduced by Russians during their occupation of that region. *D. latum* may have been introduced also to the Great Lakes region by settlers from Scandinavia; however, the absence of recent reports suggests that it may no longer occur there. Tapeworms identified as *D. latum* have also been reported from Canada, Africa, Japan, Taiwan, Manchuria, Siberia, Papua New Guinea, Australia, South America, and elsewhere; however, some of these reports may represent erroneous identifications of other *Diphyllobothrium* species. In Alaska, at least six species of *Diphyllobothrium* are known to exist, and all of them occasionally infect humans.[1,42]

Humans become infected by ingesting *Diphyllobothrium* plerocercoid larvae in fish or fish roe or liver that is raw or incompletely cooked. A variety of freshwater as well as some marine fishes are sources of infection. Among the most common freshwater fish are pike, perch, and burbot; anadromous fish (e.g., salmon and their predators) may also be infected. Salmon has been reported to be responsible for transmission in Japan and the West Coast of the United States.[38]

Diphyllobothrium species are not host specific. Although human beings may be the most important final hosts for *D. latum*, bears, dogs, cats, and other carnivores often permit normal development of adult worms. Other *Diphyllobothrium* species are maintained primarily in wild animal hosts and only incidentally infect humans. For example, *D. dendriticum* occurs widely in fish-eating birds and mammals in the Northern Hemisphere and is not uncommon in certain human populations in Alaska. *D. ursi* is a common parasite of bears but has also been reported in humans in Alaska and Canada. *D. pacificum*, a parasite of pinnipeds off the coast of Peru, occasionally infects people who eat contaminated marine fish.

Dietary preferences of certain populations and the practice of discarding human excreta into ponds and rivers facilitate the transmission of *Diphyllobothrium* species. In Japan and the Scandinavian countries, the strong cultural preferences for eating raw fish dishes have favored high rates of transmission in the past. Earlier in this century, infection was relatively common among Jewish women who prepared gefilte fish (spiced, minced fish meat) and tasted it for flavor before cooking. In general, the incidence of fish tapeworm in humans seems to be declining, but in the United States, the growing popularity of raw fish dishes, such as Japanese *sushi* and *sashimi* and Latin American ceviche, is placing consumers at continued risk of infection.[9,41]

Clinical and Pathologic Manifestations

A single *D. latum* is usually present, but infection with multiple worms can occur. The worm is not invasive, and there is little pathologic damage associated with the infection other than that related to competition between host and parasite for absorption of vitamin B_{12}.[7] *D. latum* has a marked affinity for vitamin

B_{12}, and, in some individuals, depletion of vitamin B_{12} produces a megaloblastic anemia resembling pernicious anemia both clinically and hematologically. This requires that the tapeworm be in the proximal part of the intestine and probably that the host be defective in intrinsic factor secretion with diminished capacity to absorb vitamin B_{12}.[7] Only *D. latum* is known to be associated with macrocytic anemia. Among persons harboring the worm, approximately 40% have reduced vitamin B_{12} levels, but less than 2% develop anemia.

Most infections are asymptomatic. Although there is no regular discharge of individual proglottids, such as occurs with *Taenia* species; a length of the tapeworm is sometimes evacuated or vomited. In one large clinic study that compared the frequency of complaints between nonanemic carriers and noninfected persons, only fatigue, diarrhea, dizziness, weakness, numbness of extremities, and a sensation of hunger were reported more frequently in infected persons. No difference was found in the incidence of abdominal pains. Tapeworm-induced anemia is rare, but when fully manifest, it resembles pernicious anemia (i.e., a hyperchromic, macrocytic, megaloblastic anemia with thrombocytopenia and mild leukopenia). For unknown reasons, the anemia is generally limited in its occurrence to Scandinavian countries. The anemia is usually moderate but can become severe, with findings of pallor, glossitis, dyspnea, and tachycardia. Neurologic findings, including weakness, numbness, paresthesia, disturbance of movement and coordination, and impairment of deep sensibilities, can occur in the absence of hematologic abnormalities. The anemia and the neurologic manifestations respond to vitamin B_{12} and do not return after the worm has been expelled. Severe anemia associated with diphyllobothriasis is rarely observed nowadays, possibly related to improved levels of general nutrition.[1]

Diagnosis

Diagnosis requires demonstration of the characteristic eggs (see Fig. 2). Several stool specimens should be examined because eggs are not present in all specimens. Segments of proglottids are occasionally passed with feces or vomited, and, if they are intact, their internal morphology is diagnostic. If differential diagnosis of the eggs is difficult, a saline purge may provide proglottids for examination. Discrimination of *Diphyllobothrium* species requires expert assistance and is still a matter of considerable uncertainty.[1] No satisfactory serologic or coproantigen test for *Diphyllobothrium* infection is available.

In tapeworm-induced anemia, free hydrochloric acid may be present in the gastric juice, whereas true pernicious anemia is invariably associated with achlorhydria. Vitamin B_{12} levels may be diminished.

HYMENOLEPIASIS (DWARF TAPEWORM)

Hymenolepiasis nana is an intestinal infection by both adult and larval stages of *H. nana*, the dwarf tapeworm. *H. nana* differs from all other human tapeworms by being able to complete its entire cycle in a single host.

Morphology and Biology

H. nana seldom exceeds 40 mm long and 1 mm wide. The scolex bears a retractable rostellum, armed with a single circle of 20 to 30 hooks (see Fig. 1).

The neck is long and slender, and the proglottids are wider than they are long. Genital pores are unilateral; each mature segment contains three testes. Gravid segments break off from the strobila and disintegrate, releasing eggs 30 to 47 μm in diameter. The oncosphere is covered with a thin hyaline outer membrane and an inner thick membrane, with polar thickenings that bear several hairlike filaments embedded in the inner membrane (see Fig. 2).

The life cycle of *H. nana* is unique among tapeworms in that it does not require an intermediate host. Both adults and larval forms are found in the intestines. Autoinfection occurs commonly. When eggs are ingested by humans or a rodent, they hatch in the duodenum, releasing oncospheres that penetrate the mucosa and enter the lymph channels of the villi, where each develops into a cysticercoid larva. In 5 to 6 days, the cysticercoid emerges into the lumen of the small intestine, evaginates and attaches by means of its scolex, and matures, mainly in the ileum. The prepatent period from egg ingestion until eggs appear in the feces is 20 to 30 days. The life span of the adult tapeworm is usually limited to a few weeks.

The direct life cycle is a modification of the ancestral or typical two-host life cycle, found in all other members of the family Hymenolepididae. The cysticercoid of *H. nana* can still develop normally within fleas and beetles, then infect humans by accidental ingestion of an infected grain beetle or flea. Domestic mice and rats also serve as hosts for *H. nana*, but their strain may be ill adapted to humans, so some authors consider this form a distinct subspecies, *H. nana fraterna.*

Epidemiology

The dwarf tapeworm is the most common cestode of humans, occurring most commonly where personal hygienic practices and the quality of sanitary facilities are poor. Infection rates are highest among children. Prevalence is especially high in warm and arid countries (e.g., in the Mediterranean region, the Near East, the Indian subcontinent, and South America). In these regions, prevalence in children reaches 5% to 20%. It is the most frequently diagnosed cestode infection in the United States[43]; hymenolepiasis nana was diagnosed in 0.4% of 216,000 stool specimens submitted to state diagnostic laboratories during 1987.[26] Epidemics may occur in orphanages, institutions for the mentally retarded, and other closed communities where fecal-oral transmission is likely.[52]

Humans are the natural reservoir of infection with the human strain; rodent-adapted strains are also found. Eggs passed in the feces are immediately infective. Person-to-person transmission of eggs by the fecal-oral route is probably the most common route of infection.[32] Transmission by fomites, water, and food may rarely occur.

Clinical and Pathologic Manifestations

The extent of pathologic changes depends on the number of parasites present. Patients whose egg density in stool is greater than 15,000 per gram invariably have abdominal cramps, diarrhea, and irritability. The factors that regulate worm numbers in humans include host immunity and nutrition. Infections are usually *self-cleared* by adolescence and are infrequent in healthy adults. Developing cysticercoids destroy the villi that they occupy; heavy infections, therefore, may result in extensive enteritis. Most infections are asymptomatic

and are probably caused by light or moderate worm numbers. Heavy infections frequently cause nonspecific intestinal symptoms, such as abdominal cramps, diarrhea, and anorexia. Other complaints such as dizziness, irritability, and seizures have been attributed to these infections but may be related to concomitant pathologic conditions.

Diagnosis

Diagnosis is made by identification of the characteristic eggs in feces (see Fig. 2); the tiny *H. nana* proglottids are broken up in the intestine and not seen. Because egg output may be irregular and in low numbers, two or more stool specimens should be examined by concentration techniques, preferably on alternate days.

HYMENOLEPIS DIMINUTA (RODENT TAPEWORM)

H. diminuta occurs worldwide, primarily as a parasite of domestic rats, but it can infect humans.[6] Infections are common in poor communities infested by rodents. Most infections are in young children. *H. diminuta* is a much larger worm than *H. nana* (up to 90 cm × 4 mm) and differs from it by lacking an armed rostellum on the scolex. It has unilateral genital pores; three testes per proglottid (see Fig. 1); and eggs without polar filaments, bile-stained, and twice as large as those of *H. nana* (see Fig. 2).

Rats, mice, and other rodents are the usual hosts of the adult parasite: Humans are a rare and accidental host, infected by swallowing insects that contain cysticercoid larvae. Numerous insects or their larvae are possible intermediate hosts infected by ingesting *H. diminuta* eggs passed in rodent feces. The minute grain beetle (*Tribolium* species) is probably most commonly involved in synanthropic cycles. Human infection probably occurs most often by accidental ingestion of mealworms or grain beetles that infest dried grains, cereals, and flour or other foods such as dried fruit. Infections with multiple worms are common. The worms attach to the duodenal or jejunal mucosa; gravid proglottids break off and disintegrate, releasing eggs into the stool.

Most human infections are apparently asymptomatic, but headache and mild gastrointestinal symptoms, including anorexia, nausea, abdominal cramps, and diarrhea have been described. Eosinophilia has been noted in experimental infections in humans. The diagnosis is made by finding characteristic eggs in the feces; proglottids may also be found but usually disintegrate.

DIPYLIDIASIS (DOG TAPEWORM)

Dipylidium caninum is one of the most common parasites of domestic dogs and cats throughout the world; the infection may also rarely and accidentally occur in humans, more commonly in children than in adults.[19, 22] The adult tapeworm measures 10 to 70 cm × 2 to 3 mm and attaches to the wall of the small intestine. Each proglottid (3 × 12 mm) has two sets of male and female reproductive systems and a genital pore on each side (see Fig. 1). The scolex has a retractable, somewhat pointed rostellum with several circular rows of hooklets. Eggs are encapsulated in clusters, each capsule containing 8 to 15 eggs (see Fig. 2). Actively motile, gravid proglottids the size and shape of cucumber seeds

become detached and either migrate out of the anus or are passed in feces. As the proglottids dry up, the egg capsules are released; are later ingested by flea larvae, the usual intermediate hosts; and develop in the flea body cavity into cysticercoids that survive the flea metamorphosis from larva to pupa and adult stages. The life cycle is completed when a dog, cat, or child ingests the cysticercoid-infected flea.

Most human infections are probably asymptomatic, but clinical findings attributed to the parasite include abdominal pain, diarrhea, anal pruritus, and psychological irritability. Urticaria and eosinophilia have also been reported. Multiple infections are not uncommon.

Parents often observe proglottids in children's diapers or feces. The characteristic gravid proglottids are diagnostic. Fecal examinations for eggs are unreliable because the proglottids usually do not release eggs within the intestines.

OTHER TAPEWORM INFECTIONS

Several other tapeworms that normally infect lower animals may on occasion infect humans. These infections are usually asymptomatic, and diagnosis depends on identification of characteristic eggs or gravid proglottids in the feces. Most appear to be readily eliminated by treatment with drugs effective against other intestinal cestodes.

Somewhat more than a dozen cases of human infection with *Mesocestoides* infections have been reported from Asia, Africa, and North America. Infections were usually recognized by identification of proglottids passed in the stool; *Mesocestoides* species have a distinctive parauterine organ that contains eggs. The normal definitive hosts of these tapeworms are birds or mammals. The first intermediate hosts are believed to be coprophagous arthropods. The second larval stage, called a tetrathyridium, is parasitic in a variety of vertebrates, including the dog, cat, birds, reptiles, and amphibians. Humans probably acquire the infection by ingesting tetrathyridia in flesh or blood of intermediate hosts. Most cases from Asia were associated with the use of snake's blood, liver, or gallbladder. One patient had drunk blood from a turtle. An African patient had eaten raw partridge liver. The source for several cases was unknown.

Several species of *Raillietina*, cestodes infecting rats and birds, have been described in humans in East Asia and in South America. *Inermicapsifer madagascariensis* is common in African rodents and has been reported from humans in Africa and South America. *Bertiella studeri* is a tapeworm parasitizing subhuman primates. Human infections are reported not infrequently in parts of Asia. Related *Bertiella* species are found in the Western Hemisphere and have also been reported in human infections.

TREATMENT

General Considerations

Historically, treatment of tapeworm infections involved medicines of variable modes of action and efficacy and was often quite an ordeal. Traditional treatments often include locally available seeds and barks, which are mainly purgative in nature. Most early medical compounds used to treat tapeworms (aspidium oleoresin, quinacrine, carbon tetrachloride, and tin compounds) were relatively toxic and often involved purging or extensive preparation of the

patient before and after treatment.[18] Other, nonmedical treatments that have been employed on a limited scale with reported success include manipulation of the spine[47] and administration of radiologic contrast medium.[50] Treatment of intestinal cestodes has greatly improved and simplified in recent decades; the development of efficient taeniacidal drugs, first niclosamide and later praziquantel, have made most older anthelmintics obsolete.[18]

Niclosamide, a chlorinated salicylanilide, was introduced into human and veterinary medicine about 1960[51] and shown to be effective against a wide variety of tapeworms and well tolerated, a "worm cure without tears."[14] At a dose of 2 g for adults and about half this amount for children, it was widely concluded that the drug gave a cure rate of about 90% for taeniasis and somewhat lesser efficacy for diphyllobothriasis.[18] Subsequent experience in many countries, however, led to reports of variable efficacy, and some batches of the drug were less effective than expected, possibly because of variations in particle size.[18, 49] The strong cestocidal activity of praziquantel, a new type of acylated isoquinoline-pyrazine, was first reported in 1975.[48] Clinical trials and long experience have shown that, at taeniacidal doses, there are no contraindications to the use of praziquantel in healthy persons.[51] Precautions must be observed, however, in populations in which *T. solium* is endemic; a report from Mexico indicated that mass treatment of a population at a dose rate of 10 mg/kg may have activated a case of latent cerebral cysticercosis.[15] Praziquantel is highly efficacious (>95%) against taeniasis, diphyllobothriasis, and most other intestinal cestodes when given as a single dose of 5 to 10 mg/kg.[20, 24, 51] Efficacy of 100% was reported against *T. saginata* infections at doses as low as 2.5 mg/kg.[33] For *H. nana* infection, a single dose of 25 mg/kg body weight achieves similar high cure rates (>95%) as five daily doses of niclosamide.[45]

Follow-up

After treatment with niclosamide and praziquantel, the proximal part of the large tapeworms, *Taenia* and *Diphyllobothrium* species, usually disintegrate. Therefore, although most of the strobila is often evacuated within a few hours, it is difficult to find and confirm removal of the scolex. Passage of intact or disintegrating segments and of eggs continues for several days after treatment. Treatment can be considered successful if no eggs reappear in several stool specimens examined at sufficient intervals posttreatment to allow regrowth of worms: 3 months for *Taenia* species and 1 month for *Hymenolepis, Diphyllobothrium*, and other species. Because *H. nana* has the ability to renew its population through internal autoinfection and because rates of reinfection are high among some populations of children, certain patients may appear to remain infected despite repeated treatments. Nevertheless, drug therapy nearly always reduces worm burdens to tolerable levels, and infections in children are usually lost spontaneously in adolescence.

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