What's Eating You? Trichuris trichiura (Human Whipworm)

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Trichuriasis is the third most common roundworm infestation in humans, with an estimated 800 million people infected worldwide.¹ The infestation is more prevalent in regions with a mild climate. In the United States, the infestation principally is seen in the southern states and is more prevalent among children. Poor sanitation conditions contribute to the spread of the infestation of *Trichuris trichiura*, or whipworm, because their eggs must contaminate soil where they will mature and thus complete their life cycle.

Eggs infected with T trichiura that contaminate the hands or food of humans and are ingested hatch in the human small intestine. The larvae migrate to the colon, where they mature and produce eggs. Sixty to 70 days after an individual becomes infected, the female worms begin to produce eggs (3000/d-20,000/d). The unembryonated eggs are passed in the stool and develop in soil, where they pass through a 2-cell stage and an advanced cleavage stage before becoming embryonated. This cycle takes approximately 15 to 30 days, after which time the eggs become infectious. If the eggs are ingested by a human host, the eggs' life cycle is complete.² Adult worms can live for approximately one year. These adult worms are found in the cecum and ascending colon of their host, where their anterior portion is threaded into the mucosa. Although the worms are thin, they are approximately 4 cm in length.

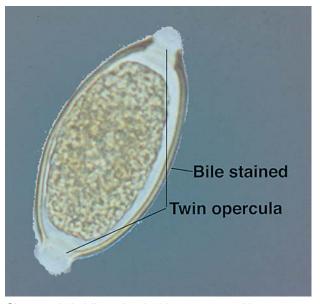
Some adult dipteran flies, such as *Chrysomya megacephala*, *Chrysomya rufifacies*, and *Sarcophaga* species, may act as carriers of helminthic parasites in humans living in tropical climates. Implicated flies tend to breed in areas of human refuse.³

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Characteristic bile-stained whipworm egg with twin operculae.

Most individuals infested with T trichiura are asymptomatic. In young children and individuals with heavy infestation, symptoms may include urticaria, pruritus, eczematous skin eruptions, abdominal cramping, diarrhea, rectal prolapse, and growth retardation.^{4,5} *T* trichiura also can produce a pruritic papular eruption that is associated with blood and tissue eosinophilia, as well as with elevated levels of serum immunoglobulin E. Therapy for these patients can be accompanied by a Jarisch-Herxheimer reaction.⁶

Heavy *T* trichiura infestation can result in severe colitis and significant blood loss, with impaired cognitive functions that are reversible with treatment.⁷ In some human immunodeficiency virus–positive populations, the prevalence of *T* trichiura infestation exceeds 44%.⁸ The degree of infestation may relate to the degree of immune suppression. Expulsion of gastrointestinal *Trichuris muris* has been shown to be mediated by a helper T cell type 2 response involving interleukins 4, 9, and 13 and dependent on

interleukins 1 α and 1 β .⁹ Some humans innately are more susceptible to gastrointestinal nematodes, and this susceptibility has been linked to a weak helper T cell type 2 response.¹⁰

The diagnosis of T trichiura infestation generally is established by finding ova in a stool sample. Rarely, ova may be found on a cellulose tape preparation for pinworm infestation. Whipworm ova measure 50 to 54 μ m long by 22 to 23 μ m wide and easily can be distinguished from pinworm eggs by the symmetrical barrel shape, lack of an embryo, twin opercula at both ends, and bile staining (Figure). The 2 polar plugs lack bile staining and contrast sharply with the brown, bile-stained wall. In contrast, pinworm eggs are asymmetrical (shaped like a rugby ball but flattened on one side) and embryonated, and they lack opercula and bile staining. In cases of heavy infestation, adult worms may be found by an endoscopy. The pointed mouthparts are at the thin anterior portion of the worm, which is deeply embedded in the host's mucosa. The posterior end is thick and resembles the handle of a whip. The bulk of the body is thin, like the remainder of the whip. The vulva is at the junction of the threadlike and thickened regions of the worm's body. The thicker posterior end lies free in the lumen of the host's large intestine.

Mebendazole generally is the drug of choice to treat whipworm infestation. Although albendazole is often used as an alternative therapy, a recent study of albendazole versus mebendazole demonstrated a low cure rate (17.1%) in the albendazole-treated group¹¹; however, longer treatment regimens with albendazole (at least 3 days for light infestations and 5–7 days for heavy infestations) still can produce acceptable cure rates.¹²

Some degree of parasitic infestation is part of the natural human condition. Inflammatory conditions such as atopic dermatitis, asthma, and inflammatory bowel disease are more common in industrialized nations that have a low incidence of infestation. Helminthic infestation causes down-regulation of some inflammatory responses, which may account for the lower incidence of these inflammatory diseases. Active infections with any geohelminth, as well as infections with *Ancylostoma lumbricoides* or *Ancylostoma duodenale*, are associated with protective effects against atopy.¹³

Trichuris suis recently has been used in clinical trials to down-regulate the inflammatory response in Crohn disease. In one study, 29 patients with active Crohn disease ingested 2500 viable T suis ova every 3 weeks for 24 weeks.¹⁴ By week 24, a total of 23 patients (79.3%) showed a beneficial response, and none experienced adverse events. Although most helminthic infestations will continue to be treated, therapeutic nematodes may enter the armamentarium for the treatment of inflammatory bowel disease and other inflammatory diseases.¹⁵

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