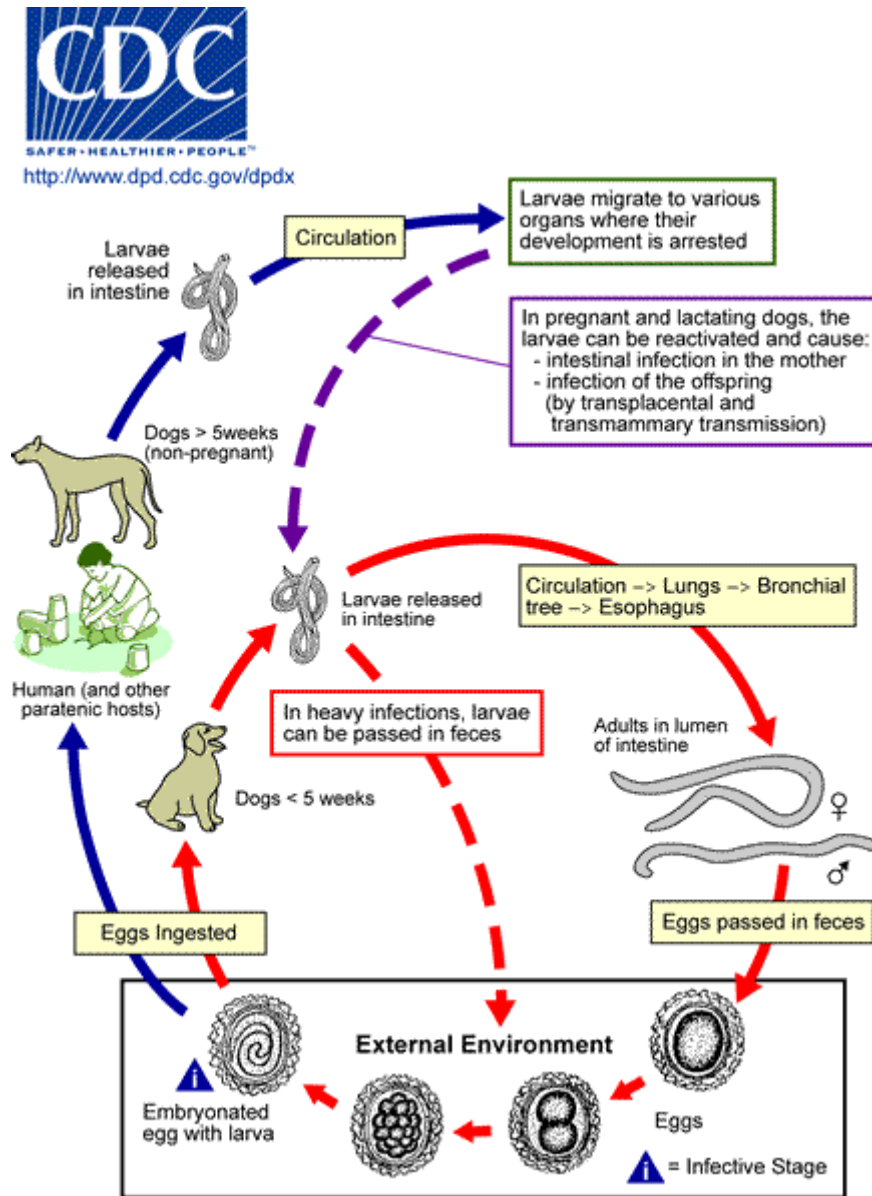


Toxocariasis

Causal Agents:

Toxocariasis is caused by larvae of *Toxocara canis* (dog roundworm) and less frequently of *T. cati* (cat roundworm), two nematode parasites of animals.

Life Cycle:



Toxocara canis accomplishes its life cycle in dogs, with humans acquiring the infection as accidental hosts. Following ingestion by dogs, the infective eggs hatch and larvae penetrate the gut wall and migrate into various tissues, where they encyst if the dog is older than 5 weeks. In younger dogs,

the larvae migrate through the lungs, bronchial tree, and esophagus; adult worms develop and oviposit in the small intestine. In the older dogs, the encysted stages are reactivated during pregnancy, and infect by the transplacental and transmammary routes the puppies, in whose small intestine adult worms become established. Thus, infective eggs are excreted both by lactating bitches and puppies. Humans are accidental hosts who become infected by ingesting infective eggs in contaminated soil. After ingestion, the eggs hatch and larvae penetrate the intestinal wall and are carried by the circulation to a wide variety of tissues (liver, heart, lungs, brain, muscle, eyes). While the larvae do not undergo any further development in these sites, they can cause severe local reactions that are the basis of toxocariasis. The two main clinical presentations of toxocariasis are visceral larva migrans (VLM) and ocular larva migrans (OLM)*.

* *Baylisascaris procyonis*, a roundworm of raccoons, has been reported to cause similar VLM and OLM syndromes in humans.

Geographic Distribution:

Worldwide.

Clinical Features:

Many human infections are asymptomatic, with only eosinophilia and positive serology. The two main clinical presentations of toxocariasis are visceral larva migrans (VLM) and ocular larva migrans (OLM). In VLM, which occurs mostly in preschool children, the larvae invade multiple tissues (liver, heart, lungs, brain, muscle) and cause various symptoms including fever, anorexia, weight loss, cough, wheezing, rashes, hepatosplenomegaly, and hypereosinophilia. Death can occur rarely, by severe cardiac, pulmonary or neurologic involvement. In OLM, the larvae produce various ophthalmologic lesions, which in some cases have been misdiagnosed as retinoblastoma, resulting in surgical enucleation. OLM often occurs in older children or young adults, with only rare eosinophilia or visceral manifestations.

Laboratory Diagnosis:

In this parasitic disease the diagnosis does not rest on identification of the parasite. Since the larvae do not develop into adults in humans, a stool examination would not detect any *Toxocara* eggs. However, the presence of *Ascaris* and *Trichuris* eggs in feces, indicating fecal exposure, increases the probability of *Toxocara* in the tissues.

For both VLM and OLM, a presumptive diagnosis rests on clinical signs, history of exposure to puppies, laboratory findings (including eosinophilia), and the detection of antibodies to *Toxocara*.

Diagnostic findings

- Microscopy
- Antibody detection

Treatment:

VLM is treated with antiparasitic drugs, usually in combination with antiinflammatory medications. The antiparasitic drug recommended in The Medical Letter is albendazole*, with mebendazole* as an alternative.

* This drug is approved by the FDA, but considered investigational for this purpose.