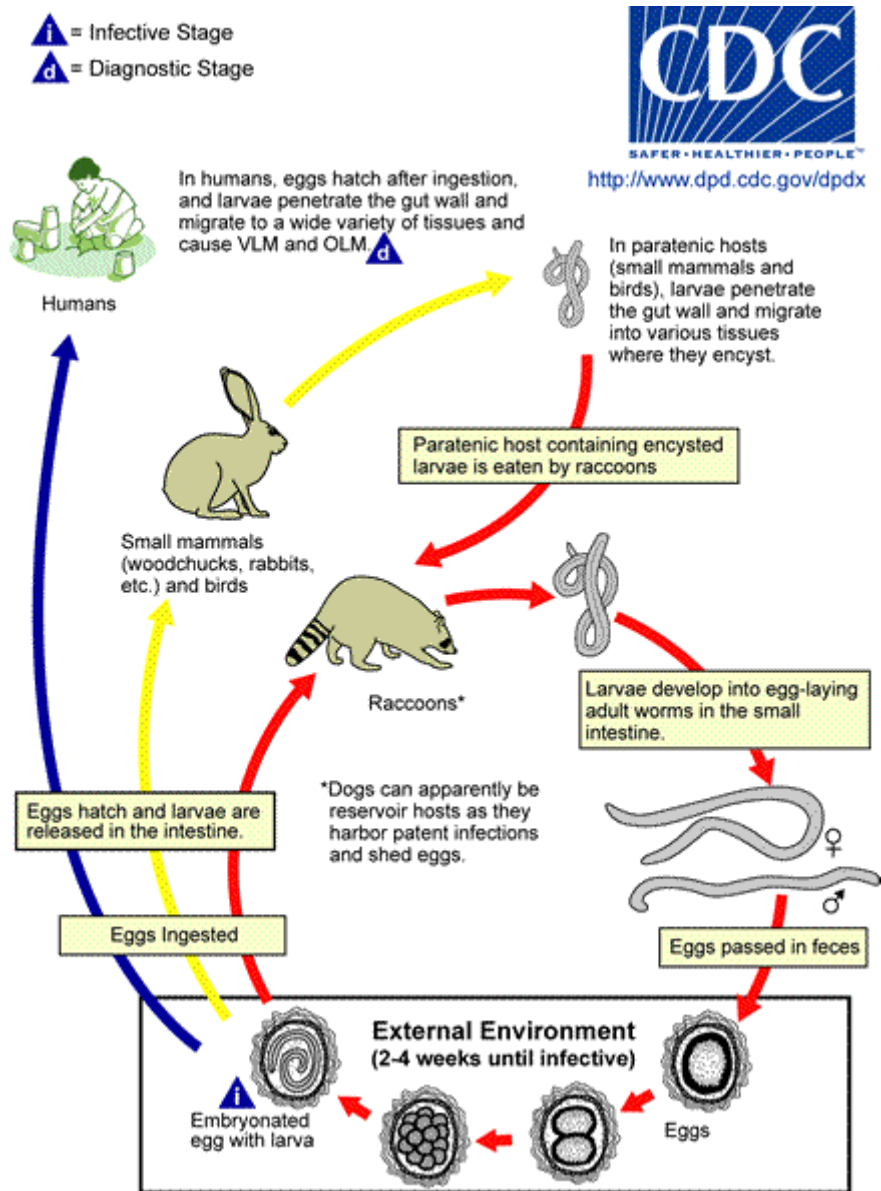


Baylisascariasis

Causal Agent:

Human baylisascariasis is caused by larvae of *Baylisascaris procyonis*, an intestinal nematode of raccoons.

Life Cycle:



Baylisascaris procyonis completes its life cycle in raccoons (*Procyon lotor*), with humans acquiring the infection as accidental hosts. Following ingestion by many different hosts (over 50 species of birds and mammals, especially rodents, have been identified as intermediate hosts) eggs hatch and

larvae penetrate the gut wall and migrate into various tissues, where they encyst. The life cycle is completed when raccoons eat these hosts. The larvae develop into egg-laying adult worms in the small intestine and eggs are eliminated in raccoon feces. People become accidentally infected when they ingest infective eggs from the environment; typically this occurs in young children playing in the dirt. After ingestion, the eggs hatch and larvae penetrate the gut wall and migrate to a wide variety of tissues (liver, heart, lungs, brain, eyes), and cause visceral (VLM) and ocular (OLM) larva migrans syndromes, similar to toxocariasis. In contrast to *Toxocara* larvae, *Baylisascaris* larvae continue to grow during their time in the human host. Tissue damage and the signs and symptoms of baylisascariasis are often severe because of the size of *Baylisascaris* larvae, their tendency to wander widely, and the fact that they do not readily die. Tissue damage and the signs and symptoms of baylisascariasis are often severe.

Geographic Distribution:

Raccoons infected with *Baylisascaris procyonis* appear to be common in the Middle Atlantic, Midwest, and Northeast regions of the United States and are well documented in California and Georgia. Proven human cases have been reported in California, Oregon, New York, Pennsylvania, Illinois, Michigan, and Minnesota, with a suspected case in Missouri.

Clinical Features:

Human infections can be asymptomatic. However, because these larvae continue to grow and wander in the human host, infections often result in severe disease manifestations. Much like toxocariasis, infection with *Baylisascaris* can result in visceral larva migrans (VLM) or ocular larva migrans (OLM) syndromes. The larvae of *B. procyonis* have a tendency to invade the spinal cord, brain, and eye of humans, resulting in permanent neurologic damage, blindness, or death. Human infection with *Baylisascaris* appears to be rare. To date, 13 well documented *Baylisascaris* encephalitis cases, and 1 suspected case in a young girl with CNS larva migrans, have been reported. The prevalence of subclinical cases is unknown. Because there is no widely available definitive diagnostic test for humans infected with this parasite, many cases are not diagnosed initially.

Laboratory Diagnosis:

Human infections are difficult to diagnose, and often the diagnosis is by exclusion of other causes. Results from complete blood count (CBC) and cerebrospinal fluid (CSF) examination would be consistent with parasitic infection, but tend to be nonspecific. Examination of tissue biopsies can be extremely helpful if a section of larva is contained, but removing an appropriate piece of tissue where the larva is actually present can be problematic. Ocular examinations revealing a migrating larva, larval tracks, or lesions consistent with a nematode larva are often the most significant clue to infection with *Baylisascaris*. Serologic testing can be extremely helpful in suspected cases; however, tests are not routinely in use nor widely available.

Diagnostic findings

- Microscopy

Treatment:

No drugs have been demonstrated to be totally effective for the treatment of baylisascariasis. Drugs such as albendazole have been recommended for specific cases.