

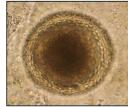
ESCCAP V MISSION OF THE DOG ROUNDWORM TOXOCARA CANIS

Toxocara canis are large fecund nematodes inhabiting the small intestine of dogs and foxes. The life cycle is complex and can involve several transmission routes

1. Eggs passed by adult *Toxocara canis* in dog or fox faeces embryonate on the ground and can accumulate extensively in the environment. Development to the infective larva inside the egg proceeds at a rate determined by environmental conditions and can take as little as 2 weeks in the UK summer. Being protected by a thick shell, larvated eggs can remain viable for four weeks or more; undeveloped eggs can survive for several years. Ingestion of infective eggs results in new infections in dogs. In younger dogs, larvae from ingested eggs undergo a hepatic-trachael migration (see 2. below). In older animals, however, larvae from eggs usually do not migrate in this way but instead travel to various tissues where they accumulate. These are the so-called 'somatic larvae' the arrested stage of this parasite which locates in the liver, lung, brain, heart and skeletal muscle, and the wall of the alimentary tract. The pre patent period (i.e. from infection to eggs seen in faeces) is 4-5 weeks











4. When infection takes

place in dogs older than

about 2-3 months of age

very few larvae undergo

PARATENIC HOSTS

5. As well as infective eggs, dogs are exposed to another source of infection: somatic larvae in the tissues (often neurological) of a variety of scavenged/prey animals such as rodents and birds which themselves have become infected following ingestion of T. canis eggs. Larvae acquired by dogs through this route do not the undertake hepatictracheal migration: development to adult worms is confined to the small intestine. Patency occurs faster when definitive hosts consume infected paratenic hosts compared to eggs



2. Larvae of *Toxocara canis* undergo an extensive journey before establishing and growing in the small intestine: The larvae enter the hepatic portal blood system to be transported to the liver, then on to the venous blood to the heart and from there to the lung capillary bed. They then break out into the alveoli and ascend via the bronchioles, bronchi and trachea. The larvae have by now moulted to the L4 stage. When swallowed, these immature develop to adults in the small intestine

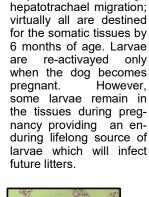


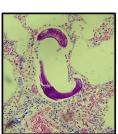
3. In the pregnant bitch, pups become infected following trans-placental migration of re-activated somatic larvae or while suckling (transmammary) and larvae undertake hepatic-tracheal migration. Eggs can actually start to appear in the faeces of puppies after only the second week of life, since activation of somatic larvae in the bitch happens around the last three weeks of gestation. Pups may be affected by large burdens of adult worms ('pot belly') and huge numbers of eggs may be excrted during the suckling period. Infection can also establish in the intestines of the lactating bitch so that both mother and pups can heavily contaminate the environment, if left untreated.





Larvae migrating through lungs of puppies can cause severe pathology







L3 in the liver

of a mouse

Infective egg

Eggs in the environment are an enduring source for potential human infection, in soil in parks and in suburban gardens, for example. Children are particularly susceptible and clinical syndromes include: Visceral larval migrans where many larvae cause a swollen liver and allergic signs; Ocular larva migrans caused by a single larva affecting unilateral vision impairment: Covert toxocariasis associated with high antibody titres and non-specific signs. Seroconversion in people has been identified as a risk factor for a range of conditions including epilepsy, cognitive dysfunction and asthma.





