Toxocariasis

Toxocarosis, Visceral Larva Migrans, Ocular Larva Migrans, Larval Granulomatosis, Toxocarial Retinitis

Last Updated: May 2005

Etiology

Zoonotic Toxocara species include Toxocara canis, T. cati, and possibly T. vitulorum and T. pteropodis. These nematode parasites all belong to the family Toxocaridae.

- T. canis is generally thought to be more important than T. cati in human disease. In Iceland, where dogs (the definitive hosts for T. canis) have been banned since the 1940s, visceral larva migrans is very rare and 0 of 300 human adults had antibodies to Toxocara spp.
- T. cati has been implicated particularly in ocular toxocariasis.
- T. vitulorum infection is thought to be a low level zoonosis mainly affecting children in the tropics. There is uncertainty about the zoonotic potential of this species: diagnostic tests for Toxocara are not always specific, and infections attributed to T. vitulorum could be due to T. canis or T. cati.
- T. pteropodis, a nematode of fruit bats, was implicated in an outbreak of hepatitis associated with feces-contaminated fruit in Palm Island, Australia. This association has been questioned by some authors.
- Two new species have recently been identified: T. malayasiensis in the domestic cat and T. lyncus in caracals. The zoonotic potential of these two organisms is unresolved.
- Toxocara species found in rodents and other wild animals, with no reported links to disease in humans or domestic animals, include T. tanuki, T. alienata and T. mackerrasae.

Geographic Distribution

T. canis and T. cati are found worldwide in the soil. The eggs of these species occur in 2-88% of soil samples collected in various countries and regions. T. vitulorum is found mainly in the tropics; cases have been reported from 50° north of the equator to 40° south. T. vitulorum is present in the U.S. but the prevalence of infection is low. The high ambient temperatures and humidity of the tropics favor the transmission of Toxocara species.

Transmission and Life Cycle

The life stages of Toxocara spp. include:

- Unembryonated eggs excreted in the feces.
- Infectious embryonated eggs containing third stage larvae. This stage is present after the eggs develop for at least 1 to 2 weeks in the environment.
- Immature larvae, which migrate through the tissues
- Dormant (‘hypobiotic’) immature larvae, found in various tissues
- Mature worms, found in the intestines

Toxocara canis in dogs

Dogs and other canids are the definitive hosts for T. canis. Mature worms, found in the intestines, shed large numbers of unembryonated eggs into the feces. The eggs become embryonated in the environment, in approximately 9-15 days at the optimum humidity and temperature (25-30°C), and 35 days at 16.5°C. Larvae do not develop at temperatures less than 10°C and die below -15°C. Cold temperatures can delay development for months or years. Only embryonated eggs are infectious.

When a dog ingests embryonated eggs, the larvae hatch in the intestines. In puppies less than 4 to 5 weeks old, the larvae penetrate the intestinal wall and are carried in the bloodstream to the lungs, where they enter the alveoli and migrate up the bronchioles, bronchi and trachea. Larvae in the pharynx are swallowed. When the parasites reach the intestines a second time, they develop into adults, mate and release eggs. Immature larvae can also be found occasionally in the feces. Adult T. canis have a lifespan of approximately 4 months in the intestines, and most of the parasites...
have been expelled within 6 months of infection. When older puppies and adult dogs ingest eggs, an increasing proportion of the larvae fails to complete the migration through the lungs. These larvae travel instead to the muscles, liver, kidneys and other viscera, where they become dormant.

Dogs of any age can develop patent infections if they eat tissues containing dormant (hypobiotic) larvae – for instance, larvae found in prey. These larvae can mature in the dog’s intestines without further migration.

Hypobiotic larvae serve as a reservoir of infection in pregnant dogs. They become reactivated during the last third of pregnancy and many of them enter the uterus or mammary gland, where they infect the fetus or puppy. Transmission can occur repeatedly to each subsequent litter, without reinfection of the dam. Parasites acquired in utero enter the fetal liver, migrate through the lungs, and develop into adults after approximately 3 weeks. Most of the larvae ingested in the milk do not migrate through the tissues, but complete their development in the intestines. Some bitches develop patent infections during lactation, either from the movement of hypobiotic larvae to the intestines or from the ingestion of larvae from the feces of their puppies; these infections disappear spontaneously 4 to 10 weeks after parturition.

Toxocara cati in cats

Cats are the definitive host for *T. cati*. The life cycle of *T. cati* is thought to resemble that of *T. canis*; however, *T. cati* is not transmitted in utero and kittens are infected only through the milk or colostrum. A recent study suggests that larvae are transmitted in the milk only if the queen is acutely infected in late pregnancy; hypobiotic larvae do not seem to be a source of lactogenic transmission.

Adult cats can develop patent infections after ingesting either eggs or larvae. Although fewer larvae complete the tracheal migration in adult cats than in kittens, the decrease is not as significant as in the dog. In cats, hypobiotic *T. cati* larvae are found mainly in the muscles.

Toxocara vitulorum in ruminants

Buffalo and bovine calves are the definitive hosts for *T. vitulorum*. Mature *T. vitulorum* are found almost exclusively in the duodenum of 3 to 10 week old calves, although there are rare reports of patent infections in older animals. Unembryonated eggs are shed in the feces. *T. vitulorum* eggs develop to the infective stage in 7 to 12 days at 28-30°C, the optimal temperature. No development occurs below 12°C, but the eggs survive and development can be completed when the temperature is raised. Embryonated *T. vitulorum* eggs can survive in the environment for several months and possibly up to two years.

Pregnant cows become infected by ingesting embryonated eggs from the environment. *T. vitulorum* larvae migrate through the liver, lung, muscle, brain, kidney, lymph nodes, mammary gland and other organs; however, mature worms are not found in the intestines of adult animals. The number of hypobiotic larvae in adult cows halves within a year, but a few dormant larvae can survive through two pregnancies.

Calves mainly become infected by vertical transmission in milk. The larvae are most abundant in the milk during the first week after calving but have been found for up to 18 days. Few larvae are found in colostrum. In utero transmission is either much less important than lactogenic transmission or does not occur. Calves may be infected by embryonated eggs under some conditions but this has not been conclusively demonstrated.

Toxocara infections in paratenic hosts including humans

Embryonated *Toxocara* eggs will release their larvae in the intestines of most mammals, as well as birds and some invertebrates such as earthworms and flies. In species other than the definitive host, the larvae do not complete the migration through the lungs and into the intestines. They migrate only through the tissues, where they eventually become encapsulated as hypobiotic larvae. Some of these larvae can remain viable indefinitely; larvae have been found for at least 9 years in experimentally infected macaques, and small rodents can be infected for life. Larvae can be transmitted between paratenic hosts, with no growth or development, by carnivorism or cannibalism. If a paratenic host is eaten by a dog or cat, the larvae usually continue their development to adult worms directly in the intestinal tract, without migration through the lungs. *T. canis* larvae can remain viable for several weeks or longer in frozen carcasses.

Transmission to the fetus is possible in some paratenic hosts. In mice, *T. canis* and *T. cati* can infect the fetus if the mother is infected during pregnancy, but hypobiotic larvae from the tissues do not infect the fetus. Transmission is also possible in the milk in rodents.

Most human infections occur in small children who eat dirt that contains embryonated eggs. Humans can also be infected if they ingest eggs on unwashed hands or in contaminated food or water, or larvae in raw or undercooked tissues (particularly the liver). Some authors suggest that humans could be infected by drinking *T. vitulorum* larvae in unpasteurized milk; other authors consider this to be unlikely. Direct contact with pets is unlikely to be a source of infection with *T. canis* or *T. cati*, as the eggs must develop for at least 9 to 15 days before they become embryonated. Vertical transmission is not known to occur in humans.

Disinfection

*Toxocara* eggs are very resistant to chemical disinfectants but can be destroyed by aqueous iodine, ultraviolet light (direct sunlight), high temperatures and prolonged drying. Isolated *T. vitulorum* eggs can be
destroyed by exposure to sunlight for 1.5 hours, boiling water, or immersion in 3% Lysol® for 15 minutes, but eggs in feces are more difficult to destroy and disinfection is unreliable. T. canis eggs can be removed from kennels by removal of the feces, followed by thorough cleaning. A solution of 1% sodium hydroxide is recommended as an aide to cleaning; the sodium hydroxide removes the sticky outer protein coat and makes the eggs easier to remove but does not kill the developing larvae. A disadvantage to the use of sodium hydroxide is that the decorticated eggs are more infective than eggs with an intact protein coat. Toxocara eggs can survive both composting and sewage treatment.

**Infections in Humans**

**Incubation Period**

The incubation period in humans is weeks to months.

**Clinical Signs**

Three syndromes have been described in humans: visceral larva migrans, ocular larva migrans and covert toxocariasis. Some authors categorize neurological disease as a fourth syndrome; others include neurological disease in the visceral form. Small numbers of larvae do not usually cause disease.

Most cases of visceral larva migrans are asymptomatic, and are recognized mainly by persistent eosinophilia. Typical signs in more severely affected children include chronic eosinophilia, malaise, fever, hepatomegaly and upper abdominal discomfort. Some patients may also have nausea, vomiting, or respiratory signs such as wheezing, coughing and dyspnea. Pruritic rashes, chronic urticaria, lymphadenopathy, arthralgia, myalgia, angioneurotic edema and neurologic signs have also been reported. In adults, the most common symptoms are fever, weakness and enteric signs. The symptoms of toxocariasis may persist for months. Deaths are rare but have been seen in cases of myocarditis, eosinophilic meningoencephalitis or severe pneumonia.

The ocular form (ocular larva migrans) can cause a spectrum of ocular disease, including retinal granulomas, retinal detachment, uveitis, optic neuritis, keratitis, iritis, endophthalmitis, vitreous abscesses and hypopyon. The infection is usually unilateral, and a single larva is typically responsible for the symptoms; however, bilateral infections have also been reported. Symptoms may include leukokoria (white pupils), decreased visual acuity, strabismus, eye pain and “seeing lights.” The loss of vision may be progressive or sudden, and can be permanent. Concurrent systemic signs are uncommon.

In the covert form, antibodies to Toxocara are associated with a few systemic or localized symptoms that do not correspond to the other two syndromes. Abdominal pain is the most common sign. Other symptoms may include hepatomegaly, coughing, sleep disturbances, headaches, behavioral changes, weakness, pruritus, rash, asthma and respiratory distress. T. canis has also been implicated as a possible cause of idiopathic seizure disorders. The covert form is not always associated with eosinophilia. The symptoms of the covert form can last for months or years.

**Intestinal infections with adult worms**

Intestinal infections with adult T. canis and T. cati are very rarely reported, and the accuracy of some of these diagnoses has been questioned. Some cases were later identified as immature Ascaris worms rather than Toxocara, and others may have resulted when young children ingested whole worms that had been expelled by pets.

**Communicability**

Toxocara infections in humans are not contagious.

**Diagnostic Tests**

Human infections are often diagnosed by the clinical signs, opthalmoscopic examination, eosinophilia and other clinical pathology findings. Histopathology is occasionally done on biopsy or autopsy specimens but is not routinely used. Other parasite eggs found in the feces (Ascaris or Trichuris) suggest that the child has been exposed to feces-contaminated soil and support a diagnosis of toxocariasis.

Serologic tests, including ELISAs and an immunoblot assay, are sometimes used in humans. Some patients with ocular larva migrans may have low titers. Ouchterlony precipitation and polymerase chain reaction (PCR) tests have been developed but are not currently available in the U.S. Other tests described in the literature include gel immunodiffusion and immunoblotting (Western blotting).

**Treatment**

Anthelmintic drugs can be used to treat severe visceral larva migrans. Treatment may lead to severe hypersensitivity reactions caused by dying larvae, and anti-inflammatory medications such as corticosteroids are often given concurrently. Treatment of ocular disease may include surgery, laser photocoagulation, and/or drugs to decrease further damage to the eye.

**Prevention**

Prevention of human infections depends on the treatment and prevention of Toxocara infections in animals, the removal of feces before the eggs can become embryonated, good hygiene and public education.

To reduce human exposure, puppies and kittens should be dewormed. Adult animals may also need to be treated for patent infections. Canine feces should be removed from areas where children play before the eggs become embryonated. The feces should be burned, buried, or bagged and disposed of in the trash. There is no practical way to remove eggs from the soil once contamination has occurred.
Toxocariasis

Clinical Signs

Toxocara canis in dogs

Young puppies usually have the most severe signs of toxocariasis. The typical symptoms include poor growth, loss of condition and sometimes an enlarged abdomen (“potbelly”). Worms may be passed in the feces or vomited. Other possible symptoms are diarrhea, constipation, vomiting, flatulence, coughing or nasal discharge. Chronic enteritis can result in thickening of the intestinal walls or intussusception. In severe cases, puppies may die from obstruction of the gall bladder, bile duct or pancreatic duct, or rupture of the intestine and peritonitis. Intestinal infections with small numbers of parasites tend to be asymptomatic.

The passage of the larvae through the liver and lungs can result in inflammation and respiratory distress of varying severity. Pneumonia can be seen soon after birth if the puppy was infected in utero; affected puppies may die within 2 or 3 days of birth. Severe infections can also cause ascites, fatty degeneration of the liver, secondary bacterial pneumonia or chronic stunting. Myocarditis is a rare complication.

Symptomatic infections are rare in adult dogs. High levels of liver enzymes may be seen during larval migration, and ocular signs, including orbital cellulitis and multifocal retinal disease, have been described. In sheep dogs, retinal disease is characterized by well-delineated areas of hyperreflectivity in the tapetal fundus, often accompanied by retinal hyperpigmentation and mild vitreal clouding. In severely affected animals, widespread hyperreflectivity and attenuation of the retinal blood vessels have been reported. Most dogs with retinal lesions do not seem to be visually impaired.

Toxocara cati in cats

Infected kittens tend to have less noticeable symptoms than puppies. Because kittens are infected only through the milk and not in utero, the larvae do not migrate through the trachea, and the kitten is also more mature when the parasite burden becomes heavy. Many infections in kittens are asymptomatic. In more severe cases, the clinical signs may include abdominal distension, a rough coat, diarrhea and possibly dehydration.

The effects of T. cati during tissue migration in cats (after ingestion of embryonated eggs) are unresolved. In one experimental infection, the primary lesions were eosinophilic pulmonary endarteritis and medial hyperplasia of the pulmonary arteries.

Toxocara vitulorum in ruminants

Common symptoms in calves include anorexia, abdominal pain, diarrhea or constipation, dehydration, steatorrhea, weight loss or poor weight gain, and a butyric odor on the breath. Uncommon sequelae include intestinal obstruction or perforation and intussusception. Coughing

Morbidity and Mortality

Larva migrans is not a reportable disease in the U.S.; however, some estimates suggest that 10,000 human cases occur each year. Most cases of toxocariasis are seen in children. Antibodies to Toxocara spp. have been reported in 4.6-7.3% of children in the U.S., 2.5% in Germany, and 83% in the Caribbean. Visceral larva migrans is most common in children between the ages of 1 to 7 years, particularly those who have a history of pica or frequently play in the dirt. Ocular larva migrans is mainly seen in children and young adults; infected children are usually older than those with visceral larva migrans.

The severity of the symptoms depends on the parasite burden, location of the larvae and duration of the infection. Most cases of visceral larva migrans are asymptomatic or mild and go unnoticed. Fatalities are rare but have occurred in cases with severe pneumonia, cardiac disease or neurologic disease. Damage to the eye can be permanent.

Infections in Animals

Species Affected

Dogs and wild canids, including foxes, coyotes, wolves, jackals, hyenas and dingoes, are the definitive hosts for T. canis. Cats are the definitive host for T. cati. Buffalo (Bubalus bubalis) and cattle are the major definitive hosts for T. vitulorum. This parasite has also been reported in lambs, bison and possibly goats. Fruit bats are the definitive hosts for T. pteropodis. A wide variety of mammals, including pigs, sheep, rodents, chickens, quail, non-human primates and humans, can serve as paratenic hosts for Toxocara spp.

Incubation Period

Puppies infected in utero can develop enteric signs within the first 2 to 3 weeks of life. Pneumonia and other symptoms of tissue migration can appear within a few days of birth. In kittens, the worms begin to mature in the intestines starting 4 weeks after birth. In experimentally infected calves, the incubation period for T. vitulorum has varied from 8 to 21 days.

Contamination can be decreased in public areas by restrictions on uncontrolled dogs and cats, collection of feces by dog owners, and prevention of animal access to areas such as children’s playgrounds. Puppies from 3 weeks to 3 months old excrete large numbers of T. canis eggs and appear to be the greatest hazard to humans. Cats shed T. cati, particularly between the ages of 2 and 6 months.

Good hygiene can help prevent infections or severe disease. Hands and raw foods should be washed before eating. Children should be taught not to eat soil, and to wash their hands after playing with pets or outdoor activities. Children should not be allowed to play in areas where animal feces are found. Families may also consider postponing the acquisition of a new pet until children are past the toddler stage.

Infections in Animals

Species Affected

Dogs and wild canids, including foxes, coyotes, wolves, jackals, hyenas and dingoes, are the definitive hosts for T. canis. Cats are the definitive host for T. cati. Buffalo (Bubalus bubalis) and cattle are the major definitive hosts for T. vitulorum. This parasite has also been reported in lambs, bison and possibly goats. Fruit bats are the definitive hosts for T. pteropodis. A wide variety of mammals, including pigs, sheep, rodents, chickens, quail, non-human primates and humans, can serve as paratenic hosts for Toxocara spp.

Incubation Period

Puppies infected in utero can develop enteric signs within the first 2 to 3 weeks of life. Pneumonia and other symptoms of tissue migration can appear within a few days of birth. In kittens, the worms begin to mature in the intestines starting 4 weeks after birth. In experimentally infected calves, the incubation period for T. vitulorum has varied from 8 to 21 days.

Last Updated: May 2005 © 2005
Toxocariasis

Treatment

Anthelmintics are effective for worms in the intestines, but hypobiotic larvae in the tissues are resistant to treatment. In dogs, parasites that renew their migration during pregnancy are susceptible to various drugs but treatment of pregnant animals is controversial. These drugs have not been tested in pregnant cats.

Heartworm preventative programs help to control *Toxocara* infections.

Prevention

Puppies and kittens should be dewormed to eliminate the shedding of eggs. Adult animals may also need to be treated for patent infections. Removal of feces and thorough cleaning is important in kennels. Cats and dogs allowed to hunt and eat rodents or other prey are more likely to be infected.

Contamination can be decreased in public areas by restrictions on uncontrolled dogs and cats, collection of feces by dog owners and prevention of animal access to public places such as children’s playgrounds. Puppies from 3 weeks to 3 months old excrete large numbers of *T. canis* eggs and are the greatest hazard. Cats shed *T. cati* particularly between the ages of 2 and 6 months.

*T. vitulorum* infections can be controlled by eliminating patent infections, which occur only in 3 to 10 week old calves. Sanitation is also important; calf feces should be removed to prevent the infection of adult ruminants by eggs.

There is no practical method to remove *Toxocara* eggs from the soil once contamination has occurred.

Morbidity and Mortality

**Toxocara canis** and **Toxocara cati**

Surveys have reported a wide range of prevalence for *T. canis* and *T. cati* in various study populations. In Western Europe, *T. canis* has been documented in 3.5-17% of dogs and *T. cati* in 8-76% of cats. In the U.S., *Toxocara* spp. have been reported in 2.79% of dogs and 10-85% of cats. Nearly all puppies born to infected bitches are infected.

The severity of the disease depends on the parasite burden. Although uncomplicated enteric infections are rarely fatal, puppies occasionally die from obstruction of the gall bladder, bile duct, pancreatic duct or intestine, or from pneumonia during larval migration. Untreated puppies may also have a decreased growth rate. Clinical disease in adult dogs is rare. Clinical signs in kittens and cats are less frequent and generally less severe than in dogs. Ocular toxocariasis is rare except in sheep dogs in New Zealand; in these dogs, a study found retinal disease in 39% of approximately 1450 dogs examined.

Communicability

Dogs, cats and ruminants with patent infections can transmit toxocariasis by contaminating the environment with eggs. The unembryonated eggs are not infectious when they are first shed.

The prepatent period for *T. canis* is usually 4 to 5 weeks after ingesting eggs, and 3 weeks in puppies infected before birth. Dogs excrete large numbers of *Toxocara* eggs; even a mildly infected dog will shed 10,000 eggs in each gram of feces. Most soil contamination occurs from puppies between the ages of 3 weeks and 3 months. A third to half of all dogs also shed *T. canis* eggs after giving birth. The prepatent period for *T. cati* is approximately 47 days after infection via the milk or prey, and 56 days after ingesting eggs. Cats shed *T. cati* particularly between the ages of 2 and 6 months. The prepatent period for *T. vitulorum* is usually 21 to 28 days. Most calves stop shedding eggs by the time they are 2 to 4 months old. Paratenic hosts can transmit the infection only if their tissues are eaten.

Diagnostic Tests

Patent infections in dogs, cats and ruminants can be diagnosed by fecal flotation. In fresh fecal samples, *Toxocara* eggs (approximately 85 µm x 75 µm) contain a single dense cell mass within a thick, brown outer shell. The shell contains a distinctive finely stippled brownish-yellow proteinaceous coat, best detected by moving the fine adjustment on the microscope. Eggs with an aberrant shape, size, or coat may be found. In adult dogs, eggs can be excreted intermittently or sporadically. Immature worms may be voided in feces or vomitus.

An enzyme-linked immunosorbent assay (ELISA) has been used in dogs to detect non-patent infections.

Morbidity and Mortality

**Toxocara canis and Toxocara cati**

Surveys have reported a wide range of prevalence for *T. canis* and *T. cati* in various study populations. In Western Europe, *T. canis* has been documented in 3.5-17% of dogs and *T. cati* in 8-76% of cats. In the U.S., *Toxocara* spp. have been reported in 2.79% of dogs and 10-85% of cats. Nearly all puppies born to infected bitches are infected.

The severity of the disease depends on the parasite burden. Although uncomplicated enteric infections are rarely fatal, puppies occasionally die from obstruction of the gall bladder, bile duct, pancreatic duct or intestine, or from pneumonia during larval migration. Untreated puppies may also have a decreased growth rate. Clinical disease in adult dogs is rare. Clinical signs in kittens and cats are less frequent and generally less severe than in dogs. Ocular toxocariasis is rare except in sheep dogs in New Zealand; in these dogs, a study found retinal disease in 39% of approximately 1450 dogs examined.

has been described in experimentally infected calves. A significant number of infections are fatal.

In adult cattle and buffalo, moderate experimental infections, probably comparable to natural infections, are asymptomatic. Feeding large doses of eggs results in fever, diarrhea and coughing, while very large doses have resulted in paralysis, conjunctivitis and opisthotonos.

**Toxocara in paratenic hosts**

There are few reports of larva migrans syndromes in animal paratenic hosts. Disseminated granulomatous disease due to *T. canis* was reported in a cat with a 19-day history of fever but no other clinical signs. Cats experimentally infected with *T. canis* had gross lesions throughout the body at necropsy, but remained asymptomatic.

Neurologic disease, cachexia and death have been reported in Mongolian gerbils. Rabbits experimentally infected with *T. vitulorum* developed liver and muscle damage, documented by changes in enzyme levels.

**Communicability**

Dogs, cats and ruminants with patent infections can transmit toxocariasis by contaminating the environment with eggs. The unembryonated eggs are not infectious when they are first shed.

The prepatent period for *T. canis* is usually 4 to 5 weeks after ingesting eggs, and 3 weeks in puppies infected before birth. Dogs excrete large numbers of *Toxocara* eggs; even a mildly infected dog will shed 10,000 eggs in each gram of feces. Most soil contamination occurs from puppies between the ages of 3 weeks and 3 months. A third to half of all dogs also shed *T. canis* eggs after giving birth. The prepatent period for *T. cati* is approximately 47 days after infection via the milk or prey, and 56 days after ingesting eggs. Cats shed *T. cati* particularly between the ages of 2 and 6 months. The prepatent period for *T. vitulorum* is usually 21 to 28 days. Most calves stop shedding eggs by the time they are 2 to 4 months old. Paratenic hosts can transmit the infection only if their tissues are eaten.

**Diagnostic Tests**

Patent infections in dogs, cats and ruminants can be diagnosed by fecal flotation. In fresh fecal samples, *Toxocara* eggs (approximately 85 µm x 75 µm) contain a single dense cell mass within a thick, brown outer shell. The shell contains a distinctive finely stippled brownish-yellow proteinaceous coat, best detected by moving the fine adjustment on the microscope. Eggs with an aberrant shape, size, or coat may be found. In adult dogs, eggs can be excreted intermittently or sporadically. Immature worms may be voided in feces or vomitus.

An enzyme-linked immunosorbent assay (ELISA) has been used in dogs to detect non-patent infections.
**Toxocara vitulorum**

*T. vitulorum* is a major cause of buffalo calf mortality in the tropics, where up to 100% of these calves can be infected. Mortality rates of 30-40%, and up to 80%, have been described in uncontrolled infections. The growth rate of infected calves is also decreased, resulting in economic losses. Some but not all studies have found *T. vitulorum* infections to be less prevalent in cattle than buffalo. The disease is also widely believed to be less severe in cattles; however, this has not been proven and is controversial.

**Post Mortem Lesions**  
[Click to view images]

In enteric disease, adult worms are found in the intestines. Chronic *T. canis* enteritis can result in mucoed enteritis, thickening of the intestinal walls or intussusception. Other complications may include obstruction of the gall bladder, bile duct or pancreatic duct, or rupture of the intestine and peritonitis.

*T. vitulorum* adults are found packed into a section of the duodenum, rather than distributed along the small intestine. Rarely, adult worms have been found in the bile ducts.

**Lesions due to Toxocara larvae**

In dogs, *T. canis* larvae can cause petechial hemorrhages and pneumonia during their migration through the lungs, and larvae may be found in the pleural cavity and diaphragm. Severe inflammation may be seen in the liver, and ascites and fatty degeneration of the liver have been reported. There are rare reports of myocarditis and pulmonary artery thrombi. Granulomas containing larvae are occasionally found in the kidney cortex of young dogs, often as an incidental finding. Ocular lesions including retinal disease and orbital cellulitis have also been described.

In cats naturally or experimentally infected with *T. canis*, multifocal, circumscribed white to gray nodules (eosinophilic granulomas) have been found in the liver, lungs, kidneys, heart (epicardium and myocardium), spleen, diaphragm, intestinal serosa and other tissues. Eosinophilic arteritis and bronchiolitis, and medial hypertrophy and hyperplasia of the pulmonary arteries were also seen in these cats. Eosinophilic pulmonary endarteritis and medial hyperplasia of the pulmonary arteries, without granulomas in other tissues, were reported in cats experimentally infected with *T. cati*.

In sheep experimentally infected with *T. canis*, multifocal white nodules have been reported in the liver, lungs, kidneys and spleen. Eosinophilic granulomas and lung lesions including diffuse interstitial pneumonitis were found on histologic examination.

---

**Internet Resources**

Centers for Disease Control and Prevention (CDC)  

CDC Guidelines for Veterinarians: Prevention of Zoonotic Transmission of Ascarids and Hookworms of Dogs and Cats  

Companion and Exotic Animal Parasitology  
[http://www.ivis.org/advances/Parasit_Bowman/toc.asp](http://www.ivis.org/advances/Parasit_Bowman/toc.asp)

Material Safety Data Sheets – Canadian Laboratory Center for Disease Control  

Medical Microbiology  

The Merck Manual  

The Merck Veterinary Manual  

---

**References**


Toxocariasis


* Link defunct as of 2012