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Chapter
Toxocariosis: From a One Health Perspective
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Abstract
Toxocariosis is a neglected zoonotic infection caused by the nematodes Toxocara canis or Toxocara cati. The distribution of the disease is worldwide and mainly affects dogs and cats, and its larval stage can cause human infection with serious repercussions on the health of its hosts. The infection causes a delay in the development, digestive disorders, nonspecific nervous manifestations, and occasionally death of some puppies and kittens associated with hyperparasitosis. In humans, the infection produces clinical syndromes known as visceral larva migrans (VLM), ocular larva migrans (OLM), neurotoxocariosis and covert toxocariosis. The close contact of people with their pets and the environmental conditions that favor the transmission of this disease place it within the context of one health. The One Health concept is defined as the collaborative efforts of multiple disciplines (medical personnel, veterinarians, researchers, etc.) that work locally, nationally, and globally to achieve optimal health for people, animals, and the environment, from this perspective, toxocariosis is a study model in which classic and recent knowledge of the medical and veterinary area must be combined for its full understanding, with a goal of establishing integrative criteria for its treatment, control, and prevention.

Keywords: Toxocara, one health, toxocariosis, zoonosis, visceral larva migrans, ocular larva migrans

1. Introduction
Toxocariosis is a neglected zoonotic disease transmitted from dogs and cats to humans. This is mainly caused by the presence and action of the nematode Toxocara canis (T. canis) and less frequently by Toxocara cati (T. cati syn. T. mystax). T. canis uses canines, mainly puppies, as its definitive host, and T. cati uses kittens. In addition, they use a wide variety of paratenic hosts, including pigs, sheep, rabbits, rats, mice, other mammals, chickens, and other birds. In humans, the infection is accidental, and the parasite behaves similarly as it does in paratenic hosts. Some invertebrates, such as earthworms and cockroaches, can also have Toxocara larvae in their tissues or their gut [1, 2].

Adult T. canis worms live in the small intestine of puppies. The females measure from 10 to 18 cm, and the males measured from 4 to 10 cm. At the anterior end, they have three small lips that do not protrude beyond the diameter of the body.
In recently delivered bitches, some larvae may reactivate during gestation migrate to the intestine, molt to L4 and L5 and become adult worms. Bitches can remain up to 60 days passing eggs in feces until the adult worms are eliminated spontaneously. This is one of the ways adult worms can develop in adult dogs [1].

Dormant larvae in the tissues of paratenic hosts can be reactivated when they are predated. If the predator is another paratenic host, the ingested reactivated larvae undergo a new somatic migration and become dormant in this new host. On the other hand, if the predator is an adult dog, the ingested reactivated larvae molt at L4 and L5 and develop into adult worms in the lumen of the small intestine without further somatic migration. In this way, dogs can spend a short time excreting eggs in the feces until the adult worms are eliminated spontaneously. This is another way that adult worms can develop in adult dogs [1].

The life cycle of *T. cati* is similar to that of *T. canis* except that prenatal transplacental infection in this parasite does not occur [6].

### 3. Epidemiology

#### 3.1 Dogs and cats

*T. canis* is the most common nematode in dogs in many regions of the world and *T. cati* in cats. In a meta-analysis study where data from more than 13 million dogs from 60 countries were included, the overall prevalence of *Toxocara* infection in dogs was found to be 11.1%. The prevalence was estimated in different World Health Organization regions: Eastern Mediterranean (19.2%), Africa (18.5%), South-East Asia (11.9%), North America (11.1%), South America (10.9%), Europe (10.8%) and Western Pacific (6.4%) [7].

In a second meta-analysis where data from 2,158,069 cats from 51 countries were included, an overall prevalence of *T. cati* of 17% was found. The prevalence was estimated in different World regions: African (43.3%), Eastern Mediterranean (21.6%), North America (18.3%), Europe (17.8%), Western Pacific (17.3%), South-East Asia (14.9%), and South American (12.6%) [8].

Transplacental transmission from bitches to their puppies is the most important form of *T. canis* infection in dogs. Not all somatic larvae in bitches are reactivated during the same gestation; thus, reactivation of larvae occurs in subsequent gestations. In addition, bitches become reinfected by ingesting persistent larvated eggs in an environment contaminated with fecal matter from their puppies. Transplacental transmission does not appear to occur in cats with *T. cati*, making lactogenic infection the most common form of infection in kittens [4, 6].

Puppies are the main source of environmental contamination; they can excrete eggs in feces from 15 days of birth, and the greatest egg shedding occurs between 1 and 3 months of age, when they can eliminate more than a million eggs per day. Gradually, the worm burden in the intestine tends to decrease, and they stop shedding eggs before reaching 6 months of age. In addition, the larvae ingested by the lactogenic route gradually increase the worm burden and the elimination of eggs in the puppies. Puppies under three months of age are the only hosts that can develop adult worms in the intestine by ingesting larvated eggs, although apparently, this is not their main route of infection [9].

Adult *Toxocara* females are very prolific, producing between 25,000 and 85,000 eggs per day, and the presence of many females in the intestine of a puppy can mean the...
elimination of enormous numbers of eggs in the feces (>100,000/g). In the environment, when eggs are protected from direct sunlight and desiccation, they develop to the infective stage (L3 passive) 2–4 weeks after shedding. In earthen soils, the eggs can remain viable for many months, accumulating in the environment. Therefore, the soil in areas where dogs with toxocariosis commonly defecate is considered a permanent source of infection for animals and humans. In addition, rainwater can carry the eggs to distant places and accumulate them in large concentrations in some places [1]. A study that included 42,797 soil samples in 40 countries showed a global prevalence of *Toxocara* eggs in public places of 21%. The estimated prevalence rates in the different regions ranged from Western Pacific (35%), Africa (27%), South America (25%), South-East Asia (21%), the Middle East and North Africa (18%), Europe (18%) and the North and Central Americas (13%) [10].

Paratenic hosts infected by ingesting larvated eggs present in soil, food or water accumulate L3 in their tissues. If these are predated, they can be a source of infection for adult dogs. If predated by another paratenic host, the larvae can infect the new host, bypassing a definitive host.

### 3.2 Humans

Due to the great difficulty of identifying the physical presence of somatic larvae, the most common way to identify *Toxocara* infection in humans is by serological tests (ELISA and Western blot). Serologically, it is not possible to distinguish between a *T. canis* infection and a *T. cati* infection, and although *T. canis* infection has generally been considered to be the predominant infection in humans, the seroprevalence of *T. cati* has not been determined, which could have been underestimated [11]. The seroprevalence of *Toxocara* in humans varies in different regions of the world. A meta-analysis carried out in 2019 that included 265,327 participants in 71 countries showed an estimated global *Toxocara* seroprevalence rate of 19.0%. The pooled seroprevalence for regions was as follows: African (37.7%), South-East Asia (34.1%), Western Pacific (24.2%), American regions (22.8%), European regions (10.5%), and Eastern Mediterranean region (8.2%) [12]. Seroprevalence has been associated with different risk factors, such as age, contact with young dogs and kittens, socioec- onomic level, consumption of vegetables, and unboiled water, ethnicity, educational level, living in a rural area and pet ownership [13, 14]. The serological differences associated with the different ethnic groups in some countries may be the result of different contextual exposures linked, among other factors, to their socioeconomic level, segregation, and the environmental conditions in which the different ethnic groups live, and not necessarily due to a genetic predisposition [15].

The most common way of infection in humans occurs through the accidental ingestion of *Toxocara* larvated eggs, which can be found on soil in public parks, gardens, dirt floors, sandboxes, and vegetables irrigated with sewage, among others. Although people of any age can be infected, children are more frequently affected due to their habits of playing with pets and dirt, geophagia, and pica, in addition to their commonly poor hygiene habits [16–18]. Infection can also occur through the ingestion of somatic larvae present in raw or undercooked meat and viscera of cattle, pigs, and poultry, among others, which act as paratenic hosts ([Figure 1](#)).

Blattella *germanica* and *Periplaneta americana* cockroaches have recently been shown to be able to ingest and shed larvated *T. canis* eggs in their feces, suggesting that they could carry infective eggs from dog feces to kitchens where human food is prepared [2].

There are multiple reports of the presence of *Toxocara* eggs in the hair of dogs and cats, which is why it has been proposed that they are a source of infection for their...
The presence of adult worms causes intestinal irritation, which induces decreased absorption of nutrients and is responsible for diarrhea and vomiting observed in some young animals. The presence of adult worms in the intestinal lumen exerts a mechanical obstructive action on the normal flow of intestinal content. Microscopically, the presence of adult worms produces mucosal muscular hypertrophy, intestinal villus atrophy, and crypt hyperplasia [1].

Larval migration in mild or moderate infections in puppies generally does not produce obvious clinical signs; however, larval migration in severe infections produces respiratory signs such as tachypnea, cough, and runny nose. Nervous signs such as incoordination or convulsions are occasionally observed in puppies due to the passage of the larvae through the brain. In puppies with intense prenatal infection, the lesions produced by the passage of the larvae in the liver, lungs, or central nervous system can cause the death of the puppies in the first 2 weeks of life [28].

Mild to moderate adult worm infections in puppies are usually asymptomatic or cause mild digestive symptoms and growth retardation. In severe infections, dirty-looking bristly hair, rough skin, painful intestinal distention, vomiting (frequently with adult worms), bulging abdomen (mainly when they have just eaten), presence of large amounts of gas produced by intestinal dysbiosis, alternating periods of constipation and diarrhea with profuse mucus, decreased appetite and growth retardation, can be observed. The blood count shows eosinophilia and anemia. Occasionally, there may be the death of puppies due to aspiration of vomit and intestinal obstruction or rupture. The presence of large numbers of adult worms as a result of massive prenatal infections in puppies can cause complete obstruction of the intestinal lumen, intussusception of the small intestine, and death of the entire litter [9, 29, 30].

In kittens, there is no transplacental transmission; therefore, the development of adult worms occurs until almost 30 days of age and the beginning of the elimination of eggs at approximately 50 days of age. The clinical picture is similar to that described in dogs but less severe, diarrhea, vomiting, and loss of appetite predominate, and deaths are very rare. The highest incidence of *Toxocara cati* occurs between 2 and 6 months of age; in general, the worm burden is lower in kittens than in puppies and occurs when the kittens are older and therefore have a higher degree of development [9, 31].

4.2 Diagnosis of toxocariosis in dogs and cats

Sporadically, shed adult worms can be observed macroscopically in the vomit or feces of puppies. The detection of *Toxocara* eggs in feces is performed by coproparasitoscopic techniques, such as Faust or McMaster; however, this can only be done when there are adult stages in the intestine, mainly in puppies [3]. In the eggs, three external layers are observed, forming the shell; the outermost layer is albuminous, the middle layer is lipoid, and the inner layer chitinous. The shell has depressions on the surface, called pits, which give it an appearance similar to a golf ball. The egg measures 75–85 μm and has a protoplasmic mass that occupies the entire interior.

In adult dogs and paratenic hosts, infection by somatic larvae can be demonstrated by the detection of specific antibodies against excretion-secretion antigens using immunological techniques such as ELISA or Western blot; however, due to their cost, difficulty in obtaining the antigens, and their difficult implementation, these techniques are not widely used in the veterinary field [32].
5. Human toxocariosis

Human toxocariosis is a neglected worldwide zoonosis caused by nematodes of the genus *Toxocara* (*T. canis* and *T. cati*). Current data indicate that toxocariosis is an infection of global distribution whose importance has been significantly underestimated [12, 15, 33, 34]. Human toxocariosis occurs in four clinical forms: visceral larvae migrans (VLM) syndrome, ocular larvae migrans (OLM) syndrome, neurotoxocariosis and covert toxocariosis.

5.1 VLM syndrome

In the 1950s, second-stage larvae of *T. canis* (now known to be third-stage larvae) were identified in the tissues of several children associated with the presence of clinical signs and a pathology that has since been known as VLM [35]. The associated syndrome in these children was characterized by extensive eosinophilia, hepatomegaly, splenomegaly, hypergammaglobulinemia, and chronic cough with eosinophilic pulmonary infiltration. VLM is more common in children (1–5 years) than in adults because they are more exposed to the infection through the ingestion of larvated eggs of *T. canis*, favored by factors such as living with puppies, poor hygienic habits, and pica [14, 36].

In humans, after ingestion of infective eggs, the larvae hatch in the small intestine and penetrate the intestinal wall, from which they are transported by the bloodstream to various organs, mainly the liver, heart, lungs, brain, muscle, and eyes [37]. In these organs, the larvae actively migrate, aided by proteases with which they cause tissue damage and exert a histophagous spoliating action (traumatic action). The migrating larvae do not continue their development; however, they remain dormant for several years, but they continue to secrete excretion-secretion antigens that induce an inflammatory response in some organs, such as the liver and spleen (hepatospleno-megaly), or are mediators of immunopathological alterations in other organs, such as the lung, where they produce eosinophilic pulmonary infiltration related to cough and persistent secretion [38].

Given the impossibility of carrying out studies in humans, experimental models have been developed in different species of paratenic hosts, such as primates [39], rabbits [40], rats [41], mice [42], and gerbils [43], where the sequence of pathophysiological and immunological events of VML have been studied. In these models, it has been observed that organ injuries can be acute or chronic. The acute phase is characterized by a severe inflammatory response that causes multifocal lesions with necrosis and vacuolization with polymorphonuclear infiltrate, mainly neutrophils with the presence of eosinophils in the liver and lungs. The chronic phase is characterized by the presence of granulomatous lesions with infiltrates of mononuclear cells, fibroblasts, and eosinophils, as well as the presence of fibrosis around the lesion with traces of calcification in the center of the lesions, which in some cases can be extensive. The main organs affected are the liver, lung, kidney, and brain (Figure 2). These lesions can be seen with or without the presence of the larva, which suggests the importance of the antigenic excretion-secretion products released by the larva in the tissues.

The clinical picture of VLM includes hyperleukocytosis (30,000–60,000 cells/mm³), eosinophilia (14–90%), abdominal pain, enlargement of lymph nodes, hepatomegaly, splenomegaly, increased ishemagglutinins and liver enzymes, intermittent fever, cough, and bronchospasm, among others [44–47]. The severity of the condition...
depends on the number of eggs ingested and the presence of larvae in critical places; although most patients recover and the signs subside with anthelmintic treatment, deaths from this infection have been reported [48, 49].

The diagnosis of VLM is based on the initial detection of antibodies against excretion-secretion antigens of *Toxocara canis* by ELISA and its confirmation by Western blot in patients with eosinophilia, with high concentrations of serum IgE or with suggestive clinical manifestations. [50–52]. It has been proposed that the confirmatory diagnosis can be validated with the identification of a larva from a biopsy or by some molecular tests, such as PCR, DNA hybridization and restriction fragment length polymorphism, or sequencing of *Toxocara* ribosomal DNA; however, it is still in the of experimentation in animal models and is not available for humans [53]. Different tools, such as ultrasound (US), contrast-enhanced ultrasound (CEUS), contrast-enhanced computed tomography (ceCT), contrast-enhanced magnetic resonance imaging (ceMRI) and positron emission tomography (PET), are currently used to obtain suggestive images of the main lesions in different human organs [49, 54].

![Lesions produced by *Toxocara canis* larvae in Mongolian gerbils (*Meriones unguiculatus*). A: lung with chronic granuloma. B: kidney with chronic granuloma with a larva trapped inside (L). C: larva in the pigmented layer of the retina with rupture of blood capillaries. D: larva in the brain with no apparent tissue reaction (photo credits: Dr. Alba-Hurtado).](image-url)
5.2 OLM syndrome

This syndrome was first described by Wilder in 1950, who found nematode larvae (unidentified at the time) in 24 of 46 pseudogliomas in eyes enucleated for endophthalmitis with apparent retinoblastoma [55]. Nichols later identified the larvae as *T. canis* in sections from four out of five of the eyes examined by Wilder [56]. Although, it is currently accepted that *T. canis* larvae are the main etiologic agent of OLM, it has also been shown that *T. cati* can cause ocular infections in humans [57–59].

OLM is a disease that generally occurs in young patients. In a systematic review and meta-analysis of studies published internationally, it was observed that the highest infection rate was detected in the 1–25 mean age group; within this range, the highest prevalence occurred between 11 and 20 years of age and was higher in men than in women [34]. It has been shown that having contact with dogs, ownership of dogs or cats, exposure to soil, and consuming raw/undercooked meat can be risk factors for OLM [12, 26, 34, 60].

OLM is generally observed in the absence of clinical signs and symptoms of VLM; it is considered to occur in people initially exposed to a small number of larvae, so they do not mount a significant immune response (many patients with a clinical diagnosis of OLM are seronegative to *Toxocara*), and the larvae migrate freely through various organs and accidentally reach the eye [61, 62]. Observations in experimental models and some clinical evidence indicate that *Toxocara* larvae infect the eye by migrating through capillaries or directly from the brain through the optic nerve [63–66].

The lesions detected in the eyes of patients diagnosed with OLM have been granulomas located near the optic disc or intraretinal (see Figure 2C), posterior and peripheral retinochoroiditis, panuveitis, optic papillitis, uveitis, retinal deformation or detachment, idiopathic epiretinal membranes, infiltration of inflammatory cells in the humor vitreous, hemorrhagic lesions and neuroretinitis as a sequel to migration of larvae in the retina [60, 67–69]. The main clinical manifestations include poor visual acuity, vision loss, strabismus, leukorrhea, eye irritation, and endophthalmitis [58, 70]. In most cases, lesions occur in only one eye, although there are reports of bilateral conditions [70].

The initial diagnosis of OLM is based on clinical signs and observation of lesions with an ophthalmoscope in the fundus examination. Confirmation of the diagnosis can be made by the detection of antibodies against excretion-secretion antigens of *T. canis* by ELISA in the vitreous humor of the affected eye and the study of the lesions by ultrasound biomicroscopy (UBM) and optical coherence tomography (OCT) [71, 72].

5.3 Neurotoxocariosis

The first report of the presence of an encapsulated larva of *T. canis* in the brain of a child was in 1951; originally, the larva was identified as probably *Ascaris lumbricus* [73]; later, this larva was identified as *T. canis* [74]. The damage produced by *Toxocara* larvae in the central nervous system (CNS) of humans has been widely discussed by many authors. The pathology depends on the number of larvae, the location of the larvae in the nervous system, the time postinfection, the immune response, and some intrinsic factors of the host. Most cases of neuro toxocariosis have been attributed to the presence of *T. canis* and, less frequently, to *T. cati*; however, the latter cannot be...
ruled out in some neurological infections. In experimental models, a greater tendency for
*T. canis* to migrate to the CNS than *T. cati* has been observed [75].

In humans, many *Toxocara* infections in the CNS can go unnoticed and do not
produce manifestations; therefore, their frequency is unknown. Some autopsy studies
have shown the presence of larvae in the leptomeninges, gray and white matter of the
cerebrum, cerebellum, thalamus, and spinal cord, unrelated to previous neurological
signs [76].

In experimental models, it has been shown that *T. canis* larvae in the CNS can
produce areas of necrosis, loss of Purkinje cells, glial nerve fibers and nerve sheaths,
granulomatous lesions, hemorrhagic and exudative lesions, vasculitis with eosinophilic
and lymphocytic infiltration, gliosis and hemosiderosis. Some larvae can be observed
without any response around them (see Figure 2D) [76, 77].

The clinical pictures of neurotoxocariosis in humans rarely occur simultaneously
with signs of VLM. Most clinical manifestations occur in adult men with an average
age of 35–42 years. Clinical signs associated with neurotoxocariosis may be indica-
tors of different neurological disorders, such as myelitis (sensation disorders such
as tingling sensation or hypoesthesia to specific dermatomes; motor disorders such
as sphincter disturbances and conus medullaris syndrome; autonomic disturbances
such as bladder and bowel dysfunction, and erectile failure), encephalitis (focal
deficits, confused state, seizure and cognitive disorders) or meningitis (headaches,
stiff neck/neck pain, nausea or vomiting, and Kernig’s/Brudzinski’s sign). Fever may
occur on some occasions, although this is not a constant sign [76, 78].

The association between *T. Canis* seropositivity and cognitive development is
controversial and has been widely discussed by several authors. Some authors, such
as Magnaval et al. [79], found no association between seropositivity and any recog-
nizable neurological syndrome; however, other authors have shown an association
between seropositivity and lower cognitive development in children; however, due to
incomplete controls and low sample size, the results are not clear [80–83].

In this context, Walsh and Haseeb [84], conducted one of the most conclusive stud-
ies; they analyzed a sample of 3,949 children representative of the US child population.
Seropositive to *T. canis* children scored significantly lower on the Scale for Children-
Revised (WISC-R) and Wide Range Achievement Test-Revised (WRAT -R) than sero-
negative children. Moreover, this relationship was independent of socioeconomic status,
ethnicity, sex, rural residence, cytomegalovirus infection and blood lead levels. These
results show another facet of the importance of toxocariosis as a neglected infection.

The diagnosis of neurotoxocariosis is difficult because there is no characteristic
clinical syndrome. Due to the lack of confirmatory diagnostic tests and the nonspe-
cific nature of its symptoms, neurotoxocariosis is probably underdiagnosed. As there
is no universally accepted criterion for the diagnosis of this syndrome, a comprehen-
sive diagnosis must be considered that must include the broad spectrum of neuro-
logical manifestations (signs of meningitis, encephalitis, myelitis, and/or cerebral
vasculitis), together with high titers of antibodies against *Toxocara* in cerebrospinal
fluid and/or blood, eosinophilia in blood and/or cerebrospinal fluid, suggestive radio-
logical images, the presence of risk factors and clinical and radiological improvement
after anthelmintic therapy [54, 78, 85].

5.4 Covert toxocariosis
Taylor et al. [86] proposed the term covert toxocariosis to describe a new clinical
entity of human toxocariosis. It is currently considered that covert toxocariosis is
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characterized by nonspecific symptoms and signs that are not associated with the VLM, OLM, or neurotoxocariosis. Clinical manifestations include asthma, acute bronchitis, pneumonia, wheezing with or without Loeffler’s syndrome, chronic urticaria or eczema, lymphadenopathy, myositis, and pseudorheumatoid syndrome, with or without eosinophilia.

The excretion-secretion antigens produced by T. canis during migration are strong stimulants of Th2-associated immune responses and the consequent induction of IL-4, IL-5, IL10, and IL-13. This cytokine profile induces an increase in the level of specific IgE-antibodies and eosinophilia, which are effectors to kill some larvae. These same effectors contribute to airway hypersensitivity and inflammation, associating chronic T. canis infection with allergic disorders such as asthma, allergic rhinitis, atopic dermatitis, and urticaria [87–89].

Asthma is a lung disease characterized by an exacerbation of the immune response in the airways to a variety of external stimuli, which produces inflammation, bronchospasm, and obstruction of the airways, which are reversible spontaneously or with treatment. Since years ago, several epidemiological and experimental studies have shown a significant relationship between Toxocara infection and the development of asthma, mainly in children [90–92]. Meta-analysis studies, where extensive collections of published data were made, have confirmed this association. Li et al. [93] using data from 723 asthmatic patients and 807 controls found a significantly higher prevalence of T. canis infection in patients with asthma than in controls (OR 3.36, P < .001). Aghaei et al. [87] using data from 872 asthmatics and 4597 non-asthmatics children, found an increased risk for asthma in children with Toxocara infection seropositivity (OR, 1.91; 95% CI, 1.47–2.47). The exact mechanisms by which Toxocara infection induces asthma and other allergic disorders remain unclear. Toxocara larval migration has been associated with an intense immune response, which causes strong allergic inflammation involving the intestine, muscle, liver, kidney, and lung [43, 94]. An animal model study (mice) showed that previous infection with T. canis intensified the ovalbumin-induced allergic airway inflammatory response associated with elevated eosinophil counts and IgE antibody levels in bronchoalveolar fluid and increased expression of IL-4 mRNA in the lung [92]. Several authors have described skin manifestations associated with toxocariosis and the risk of seropositive patients presenting skin lesions [95]. Significant associations have been observed between Toxocara seropositivity and pruritus (OR = 4.1, P < 0.1) and chronic urticaria (OR = 6.9, P < 0.0001) [96, 97]. Some of these patients presented with symptoms of VLM or OLM; however, the majority had no signs of previous Toxocara infection. Similar to neurotoxocariosis, there is no specific diagnosis for the clinical symptoms of cutaneous toxocariosis, so the participation of T. canis as a producer of skin alterations has probably been underestimated.

6. Comprehensive control of toxocariosis

The main role in the control of toxocariosis falls on the veterinarian, who is responsible for the diagnosis and deworming programs in dogs and cats, as well as the awareness and health education of pet owners so that they are aware of the threat of this and other infectious diseases from pets to humans. Periodic deworming of dogs and cats is an effective strategy to reduce the worm burden and, therefore, the number of eggs in the environment [98]. Puppies and kittens must be dewormed (piperazine, ivermectin, mebendazole, pyrantel, and febantel, among others) at
one month of age, and the treatment should be repeated at least twice in 15 days. In
adult dogs, coproparasitoscopic examinations (Faust technique) should be carried
out every 6 months, and positive dogs should be dewormed, with special care for
dogs with known predatory habits. There are no effective antiparasitic agents against
somatic larvae of *Toxocara* sp. In adult female dogs and cats, therefore, to reduce
transplacental and/or lactogenic transmission to their puppies, it is necessary to
reduce the number of infective eggs in the environment where they live. The main way of infection in humans is the ingestion of infective eggs (L3 passive)
that contaminate their environment. The fecal of dogs and, to a lesser extent, of cats
in the soil of public parks, gardens, ridges, and rural areas, among others, is the cause
of the gradual accumulation of infective eggs of *Toxocara* sp. in these places. Due to
its high resistance, there are no chemical products capable of inactivating these eggs
in the soil without seriously affecting other organisms and damaging the ecosystem.
Therefore, one of the most important strategies for the control of environmental
contamination is the immediate collection of dog feces eliminated during walks and
its subsequent disposal in the drainage. It should always be considered that puppies
and kittens are the main egg eliminators; however, adult dogs can also eventually
eliminate eggs [99].

One of the risk factors most frequently associated with human toxocariosis is
ownership of dogs or cats. For this reason, it is necessary to wash the floors daily with
soap and water inside the houses or patios where the dogs live and defecate to detach
the infective eggs from the surfaces and achieve their mechanical dragging to the
drainage, considering that the infective eggs resist most commercial disinfectants. In
addition, due to the possible presence of infective eggs attached to pet hair, it is neces-
sary to periodically bathe and brush dogs and cats to avoid the presence of
*Toxocara* eggs or other parasites in the hair.

Drainage water contaminated with *Toxocara* eggs can reach places where vegeta-
bles are grown or there may be dogs that defecate in these places, so vigorous washing
of vegetables with drinking water is essential, especially those that are eaten raw
and are grown at ground level (lettuce, cabbage, carrots, and strawberries, among
many others) to reduce the risk of ingestion of infective eggs by humans. Another
source of infection in humans is the ingestion of raw or undercooked meat or viscera
of paratenic hosts infected with somatic larvae (chickens, pigs, cattle, and ducks,
among others) in traditional dishes, so it is suggested that this type of dish is cooked
with meat from animals raised in conditions free of the parasite. Cooking meat kills
somatic larvae. At the government level, it is necessary to implement educational
campaigns for the management of pet feces, knowledge of this and other zoonotic
diseases, and the control of feral dogs and cats.

In summary, toxocariosis is a complex disease that, for its comprehensive control
from a one health perspective, requires the knowledge of researchers and different
health professionals. The veterinarian is the professional responsible for the diagno-
sis, control, and prevention of toxocariosis in pets that act as definitive hosts of the
parasite (dogs and cats), as well as in domestic species that can act as paratenic hosts
(chickens, pigs, beef, rabbits, etc.). From the perspective of human health, the joint work of a very wide variety of
health professionals is required to achieve an early and accurate diagnosis of the

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disease or at least a firm suspicion of the condition. Among these are parasitologists, infectologists, pediatricians, allergists, ophthalmologists, neurologists, dermatologists, and epidemiologists, who are sensitized and trained to cover the entire clinical spectrum that human toxocariosis can produce. In addition, highly trained laboratory personnel are required for the parasitological, immunological, and molecular diagnosis of toxocariosis in animals and humans.

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Conflict of interest

The authors declare no conflict of interest.

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