25

Toxocariasis

AUTHORS DETAIL

Virginia Guadalupe García-Rubio[1], Juan José Ojeda-Carrasco[1], Liliana Aguilar-Marcelino[2] and Carlos Ramón Bautista Garfias[2]

[1]Centro Universitario Amecameca. Universidad Autónoma del Estado de México, Amecameca, México; [2]Centro Nacional de Investigación Disciplinaria en Salud Animal e Inocuidad, INIFAP, Jiutepec, Morelos, Mexico
*Corresponding author: vggarciar@uaemex.mx

Received: Sept 29, 2022 **Accepted: Dec 12, 2022**

INTRODUCTION

Among the infectious diseases transmissible between animal and human populations (zoonoses), dogs and cats are considered as a major reservoir to spread infection to the public since they can harbor diverse pathogens such as helminths. Human Toxocariasis is caused by nematode larvae of the *Toxocara* genus, one of the most prevalent parasites in the world (Mangaval et al. 2001; Luna et al. 2018) including *Toxocara (T.) canis* in dogs, to a lesser extent *T. cati* in cats (Fan et al. 2013), and possibly *T. vitulorum* in cattle and buffalo, as well as *T. leonina* in a wide range of carnivores. It is considered as a major helminth infection in many countries, especially in tropical regions (Fan et al. 2015); however, it can be found in industrialized countries, mainly in rural areas (Duréault et al. 2017). This zoonosis arises from disparities in healthcare and is associated with conditions of poverty and poor hygiene measures (Walsh and Haseeb 2012). According to the Centers for Disease Control and Prevention (CDC), in the US, toxocariasis is listed as one of the top five neglected diseases (Tyungu et al. 2020).

Parasite Morphology

The *Toxocara* genus belongs to the class Nematoda, order Ascaroidea, superfamily Ascaridoidea, and comprises of 21 species. *T. canis* and *T. cati* are the species most commonly involved in human toxocariasis. Taking this into consideration, the description of the morphology and characteristics of *T. canis* is made as a reference for the etiological agent of the disease in humans (Okulewicz et al. 2012).
Adult parasites have a cylindrical shape, elongated, and is ivory white in color. It is important to highlight that, externally, there are irregular transverse striae with eminent cervical wings, which are longer than wide. In addition to this, there are lips surrounding an oral orifice that is continuous with the esophagus; these lips, in turn, form a bulb with two lateral lobes separated by a canaliculus (Bowman 2020).
The adult specimens exhibit sexual dimorphism. The males measure between 4 and 10 cm in length by 2.5 mm in diameter; at the caudal end, they display an elongated finger-like appendage without caudal wings, with two series of about 20 to 30 small preanal papillae and five postanal papillae on each side of the tail, and they do not have a gubernaculum. In the case of females, they are larger, measuring 5-18 cm in length by 2.5-3 mm in diameter; the genital organs develop on either side of the vulvar region, which is located anteriorly. Females can expel around 200,000 eggs/day that measure 85-90 µm by 75 µm, are ovoid, and have a thick cover with small depressions, which favors their viability in the external environment for long periods of time even under unfavorable environmental conditions (Holland and Hamilton 2006).
At the time of oviposition, eggs are yellow in color due to the bile pigments discharged into the host's digestive tract. This coloration is not observed when the eggs are obtained by hysterectomy from a gravid female. There are four larval stages (L1 to L4), with L3 being the one found inside the embryonated eggs and is considered to be infective stage (Bowman 2020). Regarding the size of larval stages, L1 can measure up to 0.5 µm, L2 up to 500 µm, L3 up to 1.5 mm, and L4 up to 20 mm. The cuticle, nervous system and ganglion nuclei, as well as the excretory and digestive systems, are formed during the L1 stage, with few changes observed in the L2 phase in which virtually an increase in size is perceived. During the third larval phase (L3), the differentiation of the digestive apparatus and the genital apparatus occurs with the appearance of the lips and the genital outline. Finally, in the fourth larval stage (L4), both the lips and the sex of the nematode are differentiated, culminating in the adult stage with sexual maturity, growth, and expansion of the cervical wings (Despomier 2003).

Characteristics of the biological cycle of *Toxocara*

The biological cycle of *Toxocara* can be direct when it takes place in only one host or indirect when there is the participation of more than one host (more than one species); this cycle is represented in Fig. 1.
Non-embryonated eggs are expelled in the feces of the definitive host (canids: *T. canis* and felids: *T. cati*) into the environment, where they embryonate, reaching the L3 stage

Citation: García-Rubio VG, Ojeda-Carrasco JJ, Aguilar-Marcelino L and Garfias CRB, 2023. Toxocariasis. In: Aguilar-Marcelino L, Younus M, Khan A, Saeed NM and Abbas RZ (eds), One Health Triad, Unique Scientific Publishers, Faisalabad, Pakistan, Vol. 3, pp: 164-171. https://doi.org/10.47278/book.oht/2023.92

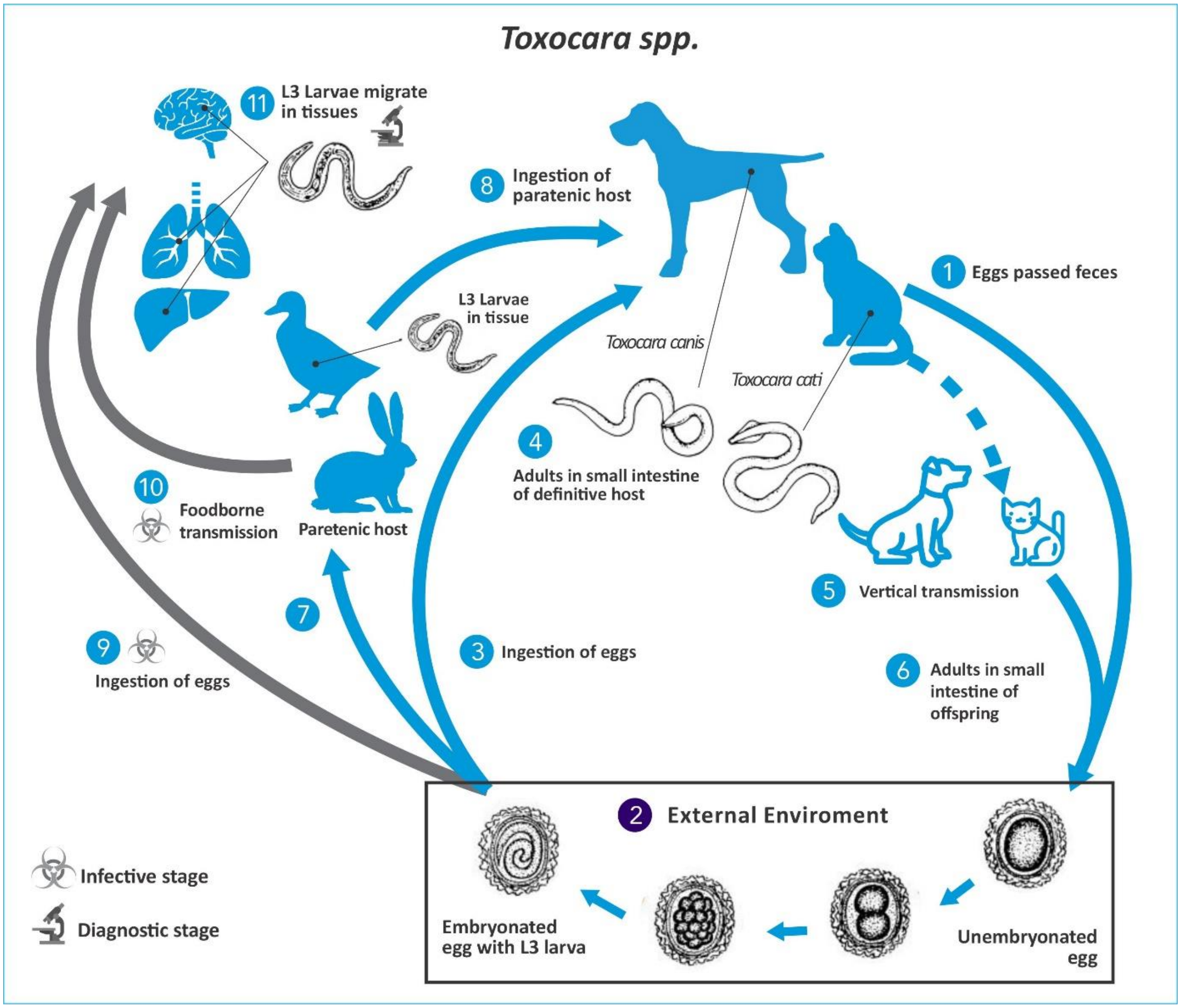

Fig. 1: Biological cycle of *Toxocara* spp.

embryo formation, the presence of O_2 and high relative humidity of 85-95% are essential requirements (Despommier 2003). It has been reported that at in a period of between one to four weeks, depending on environmental conditions. For an efficient process of temperatures between 12-18 °C, 54 days are required for the eggs to become infectious, while between 25-30 °C the time is reduced to 14 days. However, regardless of how long it takes for the eggs to embryonate and become infective, they can survive under optimal circumstances for at least one year (Chia-Kwung et al. 2003).
To complete the biological cycle, the definitive host must ingest the embryonated eggs, which hatch in the intestine, releasing the infective larvae that penetrate the intestinal wall. In the case of young animals, these larvae migrate and pass through different organs, reaching the lung via blood. When they reach the lumen of the bronchi, they are expelled with the secretion of mucus to the pharynx by coughing, and from this site, they are swallowed, passing through the gastrointestinal tract to settle definitively in the small intestine. In this organ, they mature, reach the adult stage, mate, and the females begin oviposition around 3 to 4 weeks after ingestion of the eggs. In adult dogs, infection by the oral route is also possible in the same way, culminating in the development of adult worms and the production of eggs; however, some L3 larvae remain encysted in the tissues, which justifies that in bitches with advanced gestation and due to hormonal influence these larvae are reactivated and migrate through the placenta (transplacental infection), reaching the fetal liver from where they pass to the heart through the suprahepatic vein and the vena cava, and from the heart through the pulmonary artery to the lungs. The pulmonary population of larvae is maximum between 3-5 days postinfection. Most of the larvae perforate the bronchial

wall, reaching the air space, and from there, they move through the trachea to the pharynx, where they are swallowed. For this reason, three weeks after birth, puppies can already harbor sexually mature worms in the small intestine, capable of releasing eggs into the external environment (Oge and Ozbakiş-Beceriklisoy 2019). On the other hand, although it is more frequent in female cats than in female dogs, another form of transmission is by the lactogen (transmammary) route, either by the reactivation of encysted larvae or by infection of the mother during the beginning of pregnancy; both transplacental and transmammary infection are considered as the mechanisms of vertical transmission of the parasite (Gates and Nolan 2009). It has been reported that 98.5% of infections in puppies are prenatal and 1.5% occur during lactation (Gates and Nolan 2009).

Another route of entry of the infective larvae to the definitive host is the ingestion of paratenic hosts, fundamentally rodents in which the larvae are encysted in different tissues. In this case, the parasite cycle is completed in less time because these larvae do not migrate through the animal's tissues. Molting to the adult stage begins earlier, and egg production and shedding take place after a short prepatent period. Embryonated eggs with infective L3 can also be ingested by paratenic hosts. Although these eggs lose their cover and the larvae are free and move through different organs, they do not mature in the paratenic hosts. Human is among this type of host, also called an aberrant host, and it is generally due to the fact that they maintains playful or professional contact with the definitive hosts. Accidental ingestion of these eggs causes human toxocariasis due to the presence of larvae of this parasite. Contact between *T. canis* and man can also begin by ingestion of eggs containing the L3 larva, hatching can take place both in the stomach and in the small intestine since the stimuli required are very diverse, this can partly explain the wide range of paratenic hosts (Jasim and Hadi 2021).

The fundamental place of penetration of the larvae is the small intestine, particularly the ileum. The eggs that reach the colon and have not hatched are eliminated for the most part. It has been determined that the exact site of penetration is the Liewerkühn crypts, possibly because these are the areas with less motility during ingestion. It has also been reported that Paneth cells degranulate at the time of larval penetration. From the intestine they spread to the liver mainly through the portal route, although there is some evidence of intraperitoneal dissemination or direct passage through the lymphatics to the lung and spread through the systemic circulation to all parts of the body (Chen et al. 2018).

Transmission Mechanisms

Toxocara uses various sources of infection, adult parasites can reside on a wide range of domestic and wild definitive hosts, as shown in Fig. 2 (Holland 2017). Humans are mainly infected by accidentally ingesting embryonated eggs of the nematodes *T. canis*, *T. cati* and / or congeners, which

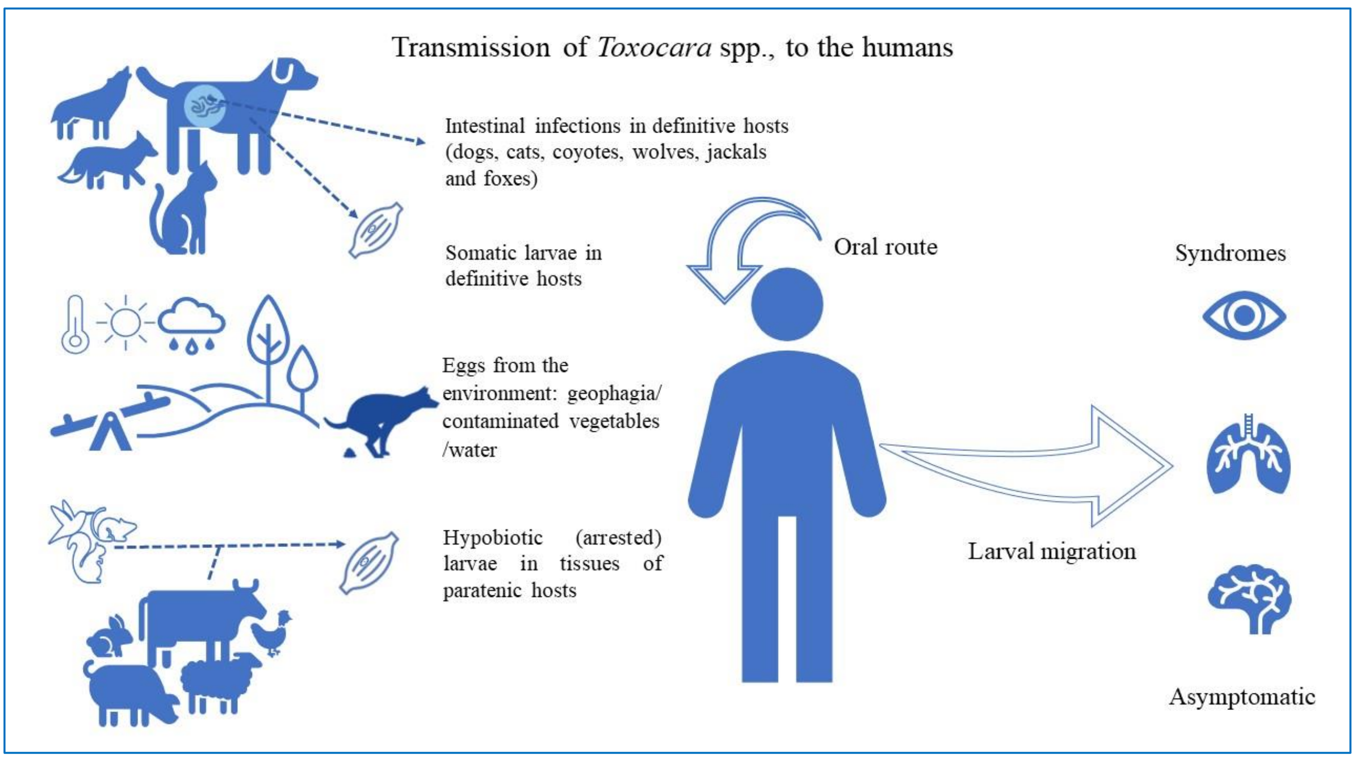

Fig. 2: Important epidemiological factors in the transmission of *Toxocara* spp., the four reservoirs of the parasite, important keys for its control.

contaminate raw vegetables and water, when carrying out recreational activities in parks, playgrounds, and sandboxes or through geophagy; to a lesser degree, by paratenesis, a type of transmission by the consumption of potentially infectious *Toxocara* larvae, encysted in the tissues of paratenic hosts, not sufficiently cooked; cattle, sheep, pigs, rabbits, chickens, and rodents serve as this type of host that can be food for both humans and definitive hosts and be reservoirs of *Toxocara* larvae (Alho et al. 2021). The transmission of *Toxocara* eggs present in the hair of dogs and, in some cases, of cats is an unlikely direct route of transmission to humans, as the eggs require an incubation period to become infective (Holland 2017; Maurelli et al. 2019).

In an extraordinary way, the infection of a patient after the ingestion of live slugs has been reported as an alternative therapy for esophageal reflux. In this particular case, the role of these as phoretic vectors has been hypothesized, transporting the infectious eggs in their mucus (Fellrath and Magnaval 2014).

Epidemiology

Dogs and wild canids, including foxes, coyotes, wolves, jackals, hyenas, and dingoes are the definitive hosts for *T. canis*, cats as definitive host for *T. cati* (Rostami et al. 2019a), buffalo (*Bubalus bubalis*), and cattle for *T. vitulorum* (Olmos et al. 2021). Puppies, kittens, and calves are the most important source of the adult parasite in the intestine, and therefore from very resistant eggs expelled to the outside that spread the infection; adult animals serve as reservoirs of the parasite, producing larvae that encyst in tissues, a role that should not be underestimated. Humans and other domestic and wild species serve as paratenic hosts; that is, species in which the biological cycle is not completed, however, serve the parasite to bridge an ecological gap in its life cycle. An infected mouse facilitates transmission of potentially infective larvae to dogs, cats, or foxes. In this regard, and despite the various investigations on this subject, the relative infective capacity of a variety of vertebrate and invertebrate hosts is unknown; it is very likely that they play a predominant role in disseminating infectious larval stages or helping the parasite to avoid unfavorable conditions in the absence of a definitive host (Holland and Hamilton 2006; Holland 2017; Olmos et al. 2021).

The prevalence of infection in dogs by *T. canis* shows wide variation worldwide, from 86 to 100% in puppies and from 1 to 45% in adult dogs; in the case of *T. cati*, 38.3% have been reported in Spanish feral cats, 79% in feral cats in Denmark, 91% in feral cats on farms in the United States, and 4.6% in the Netherlands (Fan et al. 2015). The presence of dogs and cats in urban public areas is common in many regions and contamination by their faeces significantly increases the risk of human infection by *Toxocara* (Traversa et al. 2014; Tyungo et al. 2020). The dissemination of these eggs in the environment depends on factors such as plant cover, wind, rain, displacement of definitive hosts and even the activities of birds, flies, beetles, earthworms, slugs, which indirectly determine the availability of eggs for susceptible hosts (Fan et al. 2015). Several investigations show that public spaces such as sandboxes and parks offer a continuous risk of acquiring toxocariasis. In Japan, in the city of Tokyo, 41.2% of sandboxes were found to be contaminated; in Kansas 6.6% and in Brazil 87.1%, in Portugal, 85.7% (Quattrocchi et al. 2012; Otero et al. 2018); in New York, from 29.6 to 66.7% (Tyungo et al. 2020). It is known that the viability of these eggs and their infectivity can be maintained for months and even years in adequate temperature and humidity conditions (Fan et al. 2013). The temperature, light, humidity, pH, the substrate and the vegetation can affect them; once these are eliminated in the faeces by the definitive hosts. These eggs are the main source of infection for humans due to contamination of water and food and possibly due to direct contact with dogs. In this regard, it has been reported through a systematic review and meta-analysis that eggs of *Toxocara* in different stages of development: non-viable (in all fur samples analyzed), viable/non-embryonated eggs (50.7 to 86%), embryonated (2 to 70.8%) and larvae (0.3 to 8.1%). These results suggest a low risk of infection by this route, in addition, emphasizing that these require adequate time and conditions to embryonate and reach the infectious larval stage 3 (Maurelli et al. 2019). Various studies worldwide have been carried out to find out the status of Toxocariasis in humans, despite this, it is not possible to compare the results because of different diagnostic tests, cut-off points, type of antigen, and type of population under study, in addition to this, diagnostic accuracy is significantly reduced due to cross-antigenicity, particularly in regions where polyparasitism is common (Smith et al. 2009).

Through a systematic review and meta-analysis of five international databases for the period from 1980 to 2019, Rostami et al. (2019a) determined that one-fifth of the world's population (1.4 billion individuals) is exposed to *Toxocara*, its prevalence varies depending on the country and region (Table 1). However, it is highly prevalent in developing countries, in comparison with developed countries, also highlighting the importance of the clinical sequelae of the syndromes that the parasite develops.

Toxocariasis in Humans

More than 70 years before, toxocariasis was described for the first time in 1950 and it was considered a rare disease that mainly affected children (Magnaval et al. 2001). Currently, extensive knowledge has been generated about this helminthzoonosis, now it is known that a variety of clinical syndromes can develop including Visceral Larva Migrans (VLM), Ocular Larva Migrans or Ocular Toxocariasis (OLM), Neurotoxocariasis (NT), and Covert and Cutaneous Toxocariasis (CT) (Jasim and Hadi 2021). In endemic areas with high prevalence, *Toxocara* larvae have a severe medical and social impact because these produce significant morbidity that can have debilitating and long-lasting effects

Table 1: Estimates of seroprevalence of toxocariasis in people for the period from 1980 to 2019, by Regions of the World Health Organization. Source: (Rostami et al. 2019[a])

Region	Percentage (%)
African	37.7
South East Asia	34.1
Western Pacific	24.2
American	22.8
European	10.5
Eastern Mediterranean	8.2
Global seroprevalence	19.0

This prevalence is related to several risk factors for this important helminthozoonosis, which are summarized in Table 2.

Table 2: Predisposition factors to infection by *Toxocara* spp. Source: (Quattrocchi et al. 2012; Fan et al. 2015; Kyei et al. 2015; Rostami et al. 2019a; Tyungo et al. 2020; Quintero-Cusguen et al., 2021).

Etiological agent	
Large egg production capacity	
High resistance of the infecting phase to adverse environmental conditions	
Use of vertebrate and invertebrate hosts to maintain and spread in the environment	
Various routes of transmission	
Little knowledge of its pathogenicity mechanisms, possibility that strains of *T. canis* have specific tropisms	
Human	
Genetic factors	Cultural/socioeconomic factors
Susceptibility or resistance to infection by immune response	Pet ownership/ mainly puppies
	Geophagia/ nail biting/ history of dirt play
Being Hispanic, Black non-Hispanic	Poor hygiene/ not washing hands with soap before eating.
	Being male
Age (early age)	Consume raw meat/ non-potable water.
	Have a lower income level/live in extreme poverty
	Having a low level of education/ a lower human development index
	Garbage collectors/ farmers
	Immunocompromised
Environmental/geographic	
Increase in untreated/uncontrolled definitive hosts	
Polluted environment	
Countries with tropical and subtropical climates/ higher humidity, temperature, and rainfall.	
Rural environment	
Unhygienic environment	

that impair productive capacity and children development (Walsh and Haseeb 2012; Tyungo et al. 2020). When humans accidentally consume the infective larvae, these cannot develop into the adult form, so these migrate through the bloodstream to different organs, mainly the liver, heart, kidneys, brain, eyes, and muscles. The clinical manifestations depend on the intensity of the infection, the greater the number of infective eggs ingested, the greater the number of migrating larvae, and the immune system will detect them and develop a more energetic defense response (Kyei et al. 2015). This larval migration can last for months or years, causing tissue damage and causing local or systemic inflammatory reactions as a result of the death of these larvae, as well as type IV hypersensitivity reactions, mediated by Th1 cells and the development of eosinophilic granulomas; and type I hypersensitivity, with IgE production, eosinophilia and increased expression of cytokines IL-13, IL-5 and IL-4, due to a Th2 reaction (Quintero-Cusguen et al. 2021); which will manifest different symptoms according to the affected organ (VLM), sometimes waves of migratory larvae can be generated in the viscera. On the other hand, the migratory larvae can damage the retina by inducing granulomatous reactions that are responsible for the decrease or loss of vision (OLM). The larvae can migrate to the brain and spinal cord with associated neurological compromise and produce neurotoxocariasis (NT), resulting in the presentation of epilepsy, eosinophilic meningoencephalitis, myelitis, cerebral vasculitis and neuropsychological deficits, which is very serious as toxocariasis has been associated to reduced cognitive function, producing debilitating effects, in children from socioeconomically disadvantaged populations. Finally, one less severe syndrome called covert toxocariasis or common toxocariasis has been described, with skin manifestations such as chronic urticaria, chronic pruritus, and miscellaneous eczema (Jasim and Hadi 2021; Quintero-Cusguen et al. 2021).

Diagnosis

In dogs and cats, the diagnosis is mainly carried out by coprological examination of eggs in faeces under the microscope (Gates and Nolan 2009; Okulewicz et al. 2012), by serological tests, such as ELISA (for antibody or antigen detection) and Western blot (Noordin et al. 2020) or by molecular methods, for example, PCR (Khademvatan et al. 2013; Öge and Özbakiş-Beceriklisoy 2019; Phoosangwalthong et al. 2022), and loop-mediated isothermal amplification (LAMP) technique (Azimian et al. 2021). In the case of humans, the diagnosis of Toxocara larvae may be accomplished by the detection of specific IgG antibodies against the parasite using serological tests (Zhan et al. 2015; Rostami et al. 2019b; Noordin et al. 2020) or by the detection of Toxocara larvae antigens by molecular assays (Despommier 2003; De et al. 2013).

Advantages and Disadvantages of Conventional Control

The conventional control of toxocariasis disease in humans has been done for decades through anthelmintic products, such as: 1) albendazole, 2) mebendazole, 3) thiabendazole, and 4) other drugs such as anti-inflammatory deugs (Chen et al. 2018).

The control advantage of these compounds is their easy application, speed, and efficiency (approximately in a range of 45 to 70%) depending on the compound. However, these compounds have side effects such as nausea, abdominal pain, and the most worrying reversible effects including hepatotoxicity, leukopenia, and alopecia caused mainly by albendazole in a dose of 400 mg orally for 5 consecutive days (Satou et al. 2005; Frazier et al. 2009).

The drug products (albendazole, mebendazole, thiabendazole) bind to free β-tubulin, which is an essential protein-like component of microtubules in helminths. These drugs have a great affinity for said component, which induces the inhibition of tubulin polymerization and the periphery of cytoplasmic microtubules. Additionally, anthelmintic compounds and mainly benzimidazoles alter the glucose metabolism of helminths, regarding the thiabendazole compound, it targets NADH oxidase reductase in helminths (Magnaval et al. 2022).

The disadvantages of the use of the aforementioned anthelmintic products are mainly anthelmintic resistance; however, another significant factor is the damage to beneficial organisms such as dung beetles that help keep grasslands clean, likewise they are used as biological models of environmental changes and have also been used to evaluate anthropogenic impacts on biodiversity due to the response to different levels of forest conversion and the eco-relationship of the presence of mammals (Sánchez-Hernández et al. 2022). In this context, the use and abuse of these products (macrocyclic lactones: ivermectin) have decreased the populations of these organisms in the Mexican southeast (Basto-Estrella et al. 2014), and in the Amazon and Pantanal, two regions of Brazil, the populations of these dung beetles have decreased by up to 50% to 70% due to the use of ivermectin, altering ecological niches (Correa et al. 2022). For this reason, it is urgent to implement sustainable alternatives to control toxocariasis.

Sustainable alternatives for Controlling Toxocariasis

Nowadays, multiomics tools, specifically proteomics, have shown potential for the generation of somatic and excretory-secretory proteins with specific functions for the invasion of pathogens in relation to the evasion or modulation of the immune system for the development of new generation vaccines. These proteins activates the host immune system (Zheng et al. 2020).

Totomoch-Serra et al. (2021) report the consolidation of cutting-edge technologies such as single cell analysis, immune repertoire analysis, multiple phenotyping, and spatial transcriptomics, which help to determine immune function and involvement in various infections by parasites such as toxocariasis.

On the other hand, in a study reported by Zhen et al. (2020), they used omic techniques such as genomics and transcriptomics and identified a number of genes that participate in the development of *Toxocara* and the interaction of the parasites and their hosts, and made the prediction and function of unknown genes by the comparison of other species. Omic sciences contribute to the development of new drugs, vaccines, and diagnostic tools for the sustainable control of toxocariasis worldwide.

In Brazil, a study has been carried out on the *in vitro* evaluation of ovicidal fungi isolated from the soil (*Acremonium*, *Aspergillus*, *Bipolaris*, *Fusarium*, *Gliocadium*, *Mucor*, and *Trichoderma*) on *T. canis* eggs, obtaining promising results after 14 days post-confrontation (fungus-egg interaction) (De Souza Maia Filho et al. 2012). Another biocontrol agent that has been evaluated is the fungus *Trichoderma (T.) virens* on *T. canis* eggs. The results of the mentioned study showed that the number of larvae obtained in the different organs was lower in the group of animals that were infected with the embryonated eggs of *T. canis* exposed to the fungus *T. virens* compared to the group of animals that received embryonated eggs without exposure to the fungus *T. virens*. The fungus *T. virens* showed potential as a biocontrol agent on *T. canis* eggs (De Souza Maia Filho et al. 2016).

Some authors have suggested the immunological control of Toxocariasis as a possible alternative (Barriga 1988; Jaramillo-Hernández et al. 2020). It must be considered that *Toxocara* eggs contaminate a wide variety of food, so there must be strict control of aliments destinated to human consumption (Bolicar-Mejia et al. 2014; Chen et al. 2018; Healy et al. 2022). In this context, it has been indicated that contact of young people (under 18 years old) with dogs and cats is a significant risk factor for Toxocariasis (Fitz et al. 2022). The development of new molecular tools has been suggested to facilitate the diagnosis and new control approaches to Toxocariasis in humans (Guangxu et al. 2017, Azimian et al. 2021).

Conclusion

Toxocariasis is a worldwide zoonotic issue that is increasing year by year. The major reasons behind its spread include climate change, people not following basic sanitary measures to dispose off dog feaces properly and ignorance of this issue by the health authorities. In the years to come, better control measures for toxocariasis must be implemented under the One Health scheme if success is pursued.

REFERENCES

Alho et al., 2021. Human Toxocariasis in Portugal- An overview of a Neglected Zoonosis over the Last Decade (2010-2020) Infectious Disease Reports 13: 938-948.

Azimian et al., 2021. Molecular evaluation of *Toxocara* species in stray cats using loop-mediated isothermal amplification (lamp) technique as a rapid, sensitive and simple screening assay. Veterinary Medicine Science 7: 647-653.

Barriga O, 1988. A critical look at the importance, prevalence and control of Toxocariasis and the possibilities of immunological control. Veterinary Parasitology 29: 195-234.

Basto-Estrella G et al., 2014. Dung beetle (Coleoptera: Scarabaeinae) diversity and seasonality in response to use of macrocyclic lactones at cattle ranches in the Mexican neotropics. Insect Conservation and Diversity-7: 73–81.

Bolicar-Mejia A et al., 2014. Toxocariasis in the Americas: burden and disease control. Current Tropical Medicine Reports 1: 62-68.

Bowman DD, 2020. The anatomy of the third-stage larva of *Toxocara canis* and *Toxocara cati*. Advances in Parasitology 109: 39-61.

Chen J et al., 2018. Toxocariosis: a silent threat with a progressive public health impact. Infectious Diseases of Poverty 7: 59.

Chia-Kwung F et al., 2003. Infectivity and pathogenicity of 14-month-cultured embryonated eggs of *Toxocara canis* in mice, Veterinary Parasitology 113: 2.

Correa M.A.C et al., 2022. Ivermectin impacts on dung beetle diversity and their ecological functions in two distinct Brazilian ecosystems. Ecological Entomology 1: 13.

De N et al., 2013. Molecular diagnosis of an ocular Toxocariasis patient in Vietnam. Korean Journal of Parasitology 51: 563-567.

De Souza Maia Filho F et al., 2012. Fungal ovicidal activity on *Toxocara canis* eggs. Revista Iberoamericana Micology. 30(4): 226-230.

De Souza Maia Filho, F et al., 2016. *Trichoderma virens* as a biocontrol of *Toxocara canis*: *In vivo* evaluation. Revista Iberoamericana Micology DOI: 10.1016/j.riam.206.06.004.

Despommier D, 2003. Toxocariasis: Clinical aspects, Epidemiology, Medical Ecology, and molecular aspects. Clinical Microbiology Reviews 16: 265-272.

Duréault A et al., 2017. Toxocariasis, a neglected disease in Switzerland? Revue Médicale Suisse.13(558): 815-819.

Fan CK et al., 2013. Factores que afectan la manifestación de la enfermedad de toxocariosis en humanos: genética y medio ambiente. Parasitología Veterinaria 193(4): 342-352.

Fan CK et al., 2015. Cerebral Toxocariasis: Silent Progression to Neurodegenerative Disorders? Clinical microbiology reviews 28(3): 663–686.

Fellrath JM and Magnaval JF, 2014. Toxocariasis after slug ingestion characterized by severe neurologic, ocular and pulmonary involvement. Open Forum Infectious Diseases 1: 1–3.

Fitz BMYF et al., 2022. Dog and cat contact as risk factor for human Toxocariasis: systematic review and meta-analysis. Frontiers in Public Health 10: 854468.

Frazier M et al., 2009. Treatment of ocular toxocariasis with albendezole: a case report. Optometry. 80: 175-180.

Gates M and Nolan T, 2009. Endoparasite prevalence and recurrence across different age groups of dogs and cats. Veterinary Parasitology 166: 153-158.

Guangxu et al., 2017. Human toxocariasis. Lancet Infectious Disease18(1): 14-24.

Healy S et al., 2022. Brain food; rethinking food-borne toxocariasis. Parasitology 149: 1–9.

Hernández-Sánchez G et al., 2022. The dung beetles (Coleoptera: Scarabaeidae: Scarabaeinae) of Quintana Roo, Mexico: Annotated checklist of species and new records. Revista Chilena de Entomología. 48(2): 415-434.

Holland CV and Hamilton C, 2006. The significance of cerebral toxocariasis. In *Toxocara* the Enigmatic Parasite (ed. Holland, C. V. and Smith, H. V.), CABI Publishing Oxfordshire, UK. pp. 58–73.

Holland CV, 2017. Knowledge gaps in the epidemiology of *Toxocara*: the enigma remains. Parasitology 144: 81-94.

Jaramillo-Hernández D et al., 2020. Toxocariasis and *Toxocara* vaccine: a review. Orinoquia 24: 79-95.

Jasim SY and Hadi AM, 2021. A review study for Toxocariasis. GSC Biological and Pharmaceutical Sciences 16(03): 191–199

Khademvatan S et al., 2013. Using feces of stray cats: a study from Southwest Iran. PLOS ONE 8(6): e65293.

Kyei G et al., 2015. Sero-Epidemiology of *Toxocara canis* Infection in Children Attending Four Selected Health Facilities in the Central Region of Ghana. Ghana Medical Journal 49(2):77–83.

Magnaval JF et al., 2001. Highlights of human toxocariasis. The Korean Journal of Parasitology 39(1): 1–11.

Magnaval JF et al., 2022. Therapy and prevention of human Toxocariasis. Microorganism. 10(2): 241.

Maurelli MP et al., 2019. The Presence of *Toxocara* Eggs on Dog's Fur as Potential Zoonotic Risk in Animal-Assisted Interventions: A Systematic Review. Animals 9(10): 827.

Noordin R et al., 2020. Serodiagnostic methods for diagnosing larval Toxocariasis. Advances in Parasitology 109: 131-152.

Öge H and Özbakiş-Beceriklisoy G, 2019. Detection and identification of *Toxocara canis* in infected dogs using PCR. Helminthologia 56: 118-123.

Okulewicz et al., 2012. *Toxocara canis*, *Toxocara cati* and *Toxascaris leonina* in wild and domestic carnivores. Helminthologia 49: 3–10.

Olmos LH et al., 2021. Presence of *Toxocara vitulorum* in lactating calves from the town of Guachipas, province of Salta Revista. Medicina Veterinaria 102(2): 10–13

Otero D et al., 2018. Environmental contamination with *Toxocara* sp. eggs in public parks and playground sandpits of Greater Lisbon, Portugal. Journal of Infection and Public Health 11: 94-98

Phoosangwalthong et al., 2022. *Toxocara canis* and *Toxocara cati* in Stray Dogs and Cats in Bangkok, Thailand: Molecular Prevalence and Risk Factors. Parasitologia 2: 88–94.

Quattrocchi et al., 2012. Toxocariasis and epilepsy: systematic review and meta-analysis. PLoS Neglected Tropical Diseases.6(8): e1775.

Quintero-Cusguen P et al., 2021. Toxocariosis. Acta Neurológica Colombiana 37(1) supl.1: 169-173.

Rostami A et al., 2019a. Seroprevalence estimates for toxocariasis in people worldwide: A systematic review and meta-analysis. PLoS Neglected Tropical Diseases 13(12): e0007809.

Rostami A et al., 2019b. Human toxocariasis – A look at a neglected disease Through an epidemiological "prism". Infection, genetics and Evolution 74: 104002.

Satou T et al., 2005. *Toxocara canis*: search for a potential drug amongst beta-carboline alkaloids-*in vitro* and mouse studies. Experimental Parasitology 110: 134-139.

Smith H et al., 2009. How common is human toxocariasis? Towards standardizing our knowledge. Trends in Parasitology 25(4): 182-188.

Totomoch-Serra A et al., 2021. Proteomics as a tool in vaccine development against Toxocarisis. Revista Chilena Infectology 38(5): 727-728.

Tyungu DL et al., 2020. *Toxocara* species environmental contamination of public spaces in New York City. PLoS Neglected Tropical Diseases 14(5): e0008249.

Walsh MG and Haseeb MA, 2012. Reduced cognitive function in children with toxocariasis in a nationally representative sample of the United States. International Journal for Parasitology 42: 1159–1163.

Zhan B et al., 2015. Identification of immunodominant antigens for the laboratory diagnosis of toxocariasis. Tropical Medicine and International Health 20: 1787-1796.

Zheng W et al., 2020. *Toxocara* “omics” and the promise it holds for medicine and veterinary medicine. Advances in Parasitology 109: 89-108.