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Gastrointestinal Parasites in Domestic Cats

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1. Introduction

With the domestication of animals, the contact between the latter and humans has intensified, favoring the occurrence of parasitic zoonoses (Brooker et al., 2004; Landmann et al., 2003; Katagiri et al., 2007; Thompson et al., 2008; Araújo et al., 2008). This is more evident in places where hygienic-sanitary conditions are poor (Ederli et al., 2008) and human or animal feces are present in the environment (Gatei et al., 2008; Smith et al., 2010; Sousa et al., 2010; Yoder et al., 2010).

Thus, large human conglomerates and environmental changes made by men have favored the occurrence of several emerging and re-emerging parasitic diseases (Prociv & Croese, 1996; MacCarthy & Moore et al., 2000).

Some parasites show low specificity to their host and may infect a great variety of animals (Tzipori, 1980; Xiao, 2010), causing even more severe infection in immunosuppressed individuals (Gatei et al., 2008; Alves et al., 2010).

Several etiological agents of zoonotic potential have been reported in domestic cats, constituting a severe public health problem (Robertson et al., 2002; Coelho et al., 2010; 2011a; 2011b; 2011c). Although several countries have adopted prophylactic and therapeutic measures, gastrointestinal parasites like helminths (Lima et al., 2006) and protozoa (Palmer et al., 2008) are commonly detected by means of different coproparasitological techniques in fecal samples from felines (Tzanes et al., 2008; Coelho et al., 2009).

Felines play an essential role in the epidemiology of parasites causing zoonoses, including *Ancylostoma caninum*, *Ancylostoma braziliense* (Coelho et al. 2011a), *Toxocara* spp., *Dipylidium caninum* (Abu-Madi et al., 2010; Mircean et al., 2010), and protozoa such as *Cryptosporidium* spp. and *Giardia* spp. (Apelbee et al., 2005; Bresciani et al., 2008).

Toxoplasma gondii is a protozoan capable of infecting a large number of animals and has felines as its definitive host. This parasite represents a great risk to the human population, causing diverse infection and mortality levels, especially among immunosuppressed people and pregnant women (Barbosa et al., 2007; Dubey, 2010).

Although the dog is considered the main urban reservoir for visceral leishmaniasis, the constant reports of this infection in felines have suggested that the latter play an important role in the cycle of this protozoan (Dantas-Torres, 2006; Coelho et al., 2011c).

2. Agents

Ancylostoma spp., *Toxocara* spp., *Dipylidium caninum*.

3. Epidemiology

Occurrence of gastrointestinal helminths in felines have been detected by means of parasitological necropsy in South Africa (Baker et al., 1989), Spain (Calvete et al., 1998), Egypt (Kalafalla, 2011) and Brazil (Ogassawara et al., 1986; Souza et al., 1982; Coelho et al., 2011a).

Analysis of fecal samples has been employed in epidemiological surveys in Iran (Sharif et al., 2010), the Netherlands (Overgaauw, 1997, Overgaauw & Boersena, 1998) and Brazil (Gennari et al., 2001; Labarthe et al., 2004; Coelho et al., 2009).

Ancylostoma was the most prevalent genus among the studied animals, which corroborates data in the literature (Serra et al., 2003, Funada et al., 2007). A large number of studies, however, have shown that the genus *Toxocara* sp. occurs at a higher frequency (Calvete et al., 1998, Ragozo et al., 2002), except for the study carried out by Bittencourt et al., 1996, in Espírito Santo do Pinhal, Brazil, where the proportion of these two helminths was the same (20%).

In a previous study, our team performed parasitological necropsy in 60 cats domiciled in Araçatuba Municipality, São Paulo State, Brazil, and sent to the Zoonosis Control Center of that municipality. The genus *Ancylostoma* spp. was most frequently detected. It must be highlighted that of all animals analyzed, 40 (86.96%) had *A. braziliense* and 11 (23.91%) had the species *A. tubaeforme*, and mixed infection by *A. braziliense* and *A. tubaeforme* occurred in 10 (21.74%) animals (Ishizaki et al., 2006).

Researchers like Campos et al. (1974), Ogassawara et al. (1986), Baker et al. (1989), Calvete et al. (1998) and Overgaauw & Boersema (1998) did not detect the genus *Toxocara* in a survey of the helminth fauna.

Predominance of the genus *Ancylostoma* over the remaining gastrointestinal parasites could be verified by our research group since 96% (49/51), 43.1% (22/51) and 19.6% (10/51) analyzed cats had eggs of *Ancylostoma* spp, *Toxocara* spp. and ovigerous capsules of *D. caninum*, respectively (Coelho et al., 2009).

We must emphasize the low positivity for *D. caninum*; the presence of this parasite is generally underestimated in surveys using coproparasitological tests since its diagnosis is made based on the presence of proglottids in fresh feces or adult forms in necropsy but rarely on the presence of ovigerous capsules in feces (Gennari et al., 1999). The percentages of infection by *D. caninum* are different according to the place of origin of animals. Souza et al. (1982) found prevalence of 51.42% in Rio Grande do Sul, whereas Blazius et al. (2005) obtained prevalence of 1.9% in Santa Catarina State, Brazil.

In São Paulo State, Brazil, Silva et al. (2001) observed that 100% (11/11) cats were positive for *Ancylostoma caninum*. In Minas Gerais State, Brazil, Mundim et al. (2004) verified that 90% (45/50) analyzed cats had eggs of *Ancylostoma* spp.

Environmental contamination by this helminth has been reported in several studies in Brazil (Côrtes et al., 1988; Santarém et al., 1998) and in the world (Shimizu, 1993; Uga, 1993; Şengür et al., 2005).

4. Physiopathogenesis

Parasite migration and spoliation of larvae of *A. braziliense* and *A. caninum* lead to a disease named cutaneous larva migrans (CLM) (Hunter & Worth 1945; Hanslik et al., 1998; Kwon et al., 2003; Caumes et al., 2004). High levels of intestinal lesions and is mainly related to the number of worms present in the intestinal lumen, as well as to the age of animals (Rey, 2001; Fortes, 2004).

5. Biology

Except for *D. caninum* which needs fleas as intermediate host, parasites belonging to the genera *Ancylostoma* and *Toxocara* areoelomic cavity of these insects and, when ingested by a mammal biologically defined as host, the parasite is released in the small intestine, where it establishes (Rey, 2001; Fortes, 2004).

6. Clinical signs

The number of adult parasites in the animals is a determinant for the infection severity and the manifestation of clinical signs. Dermatitis, eczema, itch, hypersensitivity and anemia are some of the diverse clinical manifestations shown by animals parasitized by *Ancylostoma* (Rey, 2001; Fortes, 2004).

Human toxocariasis may be associated with the formation of pyogenic abscesses (Rayes & Lambertucci, 1999), asthma (Tonelli, 2005), and several forms of ocular, hepatic and renal disorders (Jacob et al., 1994). These clinical signs are similar to those observed in domestic cats, especially in pups (Fortes, 2004).

Although *D. caninum* is considered slightly pathogenic, hypersensitivity, diarrhea, abdominal pain, as well as nervous manifestations and intussusceptions, may occur (Rey, 2001; Fortes, 2004).

7. Diagnosis

Diagnosis must be based on the animal history, including detailed anamnesis with special attention to the clinical manifestations that may be easily confused with those of other diseases. Thus, skin biopsy can also be performed to detect *Ancylostoma* (Acha & Szyfres, 2003), as well as serological tests to detect anti-*Toxocara* antibodies (Marchioro et al., 2011). In addition, coproparasitological tests have been shown highly effective in detecting these parasites (Hoffmann, 1987; Coelho, 2009).

Different prevalence levels can be found for these parasites according to the adopted diagnosis technique. In our study, parasitological necropsy was the “gold standard” test, while the techniques of flotation in saturated sodium chloride solution of 1.182 density (Willis, 1921) and spontaneous sedimentation in water showed different sensitivity and specificity levels (Coelho et al., 2011a).

This same difference was observed by our group in another study, in which fecal samples from 51 cats were analyzed, indicating the presence of eggs of *Ancylostoma* spp. in 96% samples according to the method of Willis and in 21.5% samples according to the technique of Faust. This study also indicated divergence between these techniques as to detection *Toxocara* eggs (43.1% by Willis and 9.8% by Faust) and *D. caninum* ovigerous capsules (19.6% by Willis and 5.8% by Faust (Coelho et al., 2009).

Thus, there is the need of associating different coproparasitological techniques in the laboratorial routine in order to increase the efficiency of the diagnosis of helminths and protozoa (Huber et al., 2004; Coelho et al., 2009).

Our group has worked to established an automated standard diagnosis method named Modified TF-Test®, which allows 3D computer analysis of parasitic structures present in the feces of animals by means of image recombination, leading thus to an important diagnostic innovation concerning helminths and protozoa affecting pets.

8. Treatment

Although parasitic resistance to certain anthelmintics have been reported, the mebendazole, albendazole (Amato Neto et al., 1983) and ivermectin (Machado & El Achkar, 2003) remain showing good efficacy.

9. Agents

Cryptosporidium spp. and *Giardia* spp.

10. Epidemiology

Similarly to giardiasis, cryptosporidiosis is a cosmopolitan gastrointestinal disease caused by protozoa of the genus *Cryptosporidium*, widely distributed all over the world (Smith et al., 2006; Xiao & Fayer, 2008; Ballweber et al., 2009). It is considered a neglected disease of great public health importance due to its frequent occurrence (Alves et al., 2006; Savioli et al., 2006; Carvalho, 2009), difficult treatment (Schnyder et al., 2009; Rossignol, 2010) and singular epidemiological aspects such as its transmission mode, zoonotic potential (Mtambo et al., 1996; Monis & Thompson, 2003; El-Sherbini et al., 2006), and variation in subtypes with the geographical region (Hunter et al., 2008; Xiao, 2010).

On account of their low host selectivity, *Cryptosporidium felis* (Huber et al., 2007) and several other *Cryptosporidium* species have been described in cats, including *Cryptosporidium parvum* (Sargent et al., 1998) and *Cryptosporidium muris* (Pavlassek & Ryan, 2007).

Infection prevalence rates of 8.1% (19/235) for *Cryptosporidium* spp. were reported by Mtambo et al. (1992) in the United Kingdom. In Brazil, different *Cryptosporidium* infection rates were found in different states by Funada et al. (2007), 11.3% (37/327), Huber et al. (2002), 12.5% (6/48), and Coelho et al. (2009), 3.9% (2/51), using different coproparasitological techniques.

In Australia, Palmer et al. (2008) used molecular analyses and verified that *Giardia* Assemblages F and D are present in the feces of domestic cats. This is important since

infection by *Giardia duodenalis* assemblages are frequent in humans by assemblage B, while in pets assemblages C and D occur in dogs and assemblage F in cats (Monis & Thompson, 2003; Souza et al., 2007; Xiao & Fayer., 2008), there is also the possibility of cross infection by *Giardia* assemblages between animals and humans (Traub et al., 2004; Palmer et al., 2008; Feng & Xiao, 2011).

In our study, *Giardia* spp. was detected in 5.9% (3/51) fecal samples from domestic cats. Also in Brazil, Gennari et al. (1999) noted that 16.04% of 187 fecal samples from cats were positive for *Giardia* spp. In Australia, MacGlade et al. (2003) analyzed fecal samples from 40 cats and observed approximately 60% positivity prevalence for *Giardia*.

A similar occurrence was detected in Germany between 1999 and 2002, when fecal samples from 3164 cats were analyzed indicating that 51.6% had cysts of *Giardia* spp. (Barutzki & Schaper, 2003).

11. Physiopathogenesis

The pathophysiological mechanism of cryptosporidiosis consists in its intraenterocytic stage. This enteroinfection causes atrophy, fusion of intestinal villi and inflammation, which result in absorptive surface loss and unbalanced nutrient transport. It is not clear yet whether the parasite interferes with the cell function but it seems capable of inducing or inhibiting cell apoptosis (Chen et al., 1998; Dagci et al., 2002; Buret et al., 2003; Leav et al., 2003).

Histopathological analyses have revealed that cryptosporidiosis may lead to minimal inflammatory infiltration and villus blunting, while changes are more pronounced in immunosuppressed individuals, including greater inflammatory changes, epithelial cell barrier rupture with more extensive and intense inflammatory cell infiltration. Massive parasite infection in the enterocytes stimulates local inflammatory reaction, increasing the levels of prostaglandins, several cytokines, especially interferon. These inflammatory mediators change solute transport in the intestinal epithelial cell, leading to osmotic diarrhea (Leav et al., 2003).

Diarrhea due to poor absorption results of the interaction between parasitic products such as proteinases, which rupture the epithelial barrier, and the immune/inflammatory responses of the host, favoring deficient absorption of electrolytes and nutrients, combined with the hypersecretion of chlorine and water (Argenzio et al., 1990; Huang & White, 2006), inducing intestinal abnormalities, especially due to the activation of CD8+ lymphocytes in the intraepithelial compartment, with increased cytotoxic activity (Chai et al., 1999; Buret, 2009).

Cryptosporidium infection is auto-limiting for immunologically normal individuals. In immunodepressed humans, however, this disease is associated with high mortality and morbidity indexes (Hunter & Nichols, 2002), especially in HIV-positive (Cama et al., 2007), transplanted individuals (Dekinger et al., 2007) and children (Glaeser et al., 2004) showing deficient global count of T CD4+ lymphocytes (Assefa et al., 2009).

Parasitic infection by *Giardia intestinalis* is most frequently reported all over the world. It causes several intestinal, nutritional and general development disorders (Botero-Garcés et al., 2009; Singh et al., 2009).

Although giardiasis is an auto-limiting disease, it manifests in individuals mainly by means of acute diarrhea; however, asymptomatic chronic infections may occur, leading to malabsorption of vitamin A, B12 (Springer et al., 1997) and anemia due to iron deficiency (Ertan et al., 2002).

Children are most affected by this protozoan disease (Tellez et al., 1997; Thompson et al., 2000), especially in developing countries where hygienic-sanitary conditions are not adequate (Guimarães et al., 1995; Savioli et al., 2006), and domestic animals may produce cysts potentially infective for humans (Eligio-García et al., 2008).

In Colombia, Botero-Garcés et al. (2009) verified that 27.6% of the 2035 studied children were infected by *G. intestinalis* and part of them had significant body development deficit.

12. Biology

As to *Cryptosporidium* biology, sporulated oocysts are ingested by the host and, following exposure to the gastric juice and pancreatic enzymes, excystation occurs in the duodenum releasing four sporozoites. The latter are covered by microvilli located in a parasitophorous vacuole and start the asexual reproduction. In this event, they develop successive merogonies, releasing eight and four sporozoites, respectively (Fortes et al., 2004).

The four merozoites released from the second merogony originate the sexual stages, resulting in the genesis of microgametes and macrogametes, which unite to form the zygote. Sporulation occurs inside the oocyst, developing four sporozoites. In this event, oocysts of thin (capable of starting a new cycle inside the same host by means of retroinfection) and thick wall (highly resistant under environmental conditions and released in the feces) are formed. In healthy people, the infection generally remains in the gastrointestinal tract (TZIPORI & GRIFFITHS, 1998).

Considering the biological cycle of *Giardia*, we must highlight that in addition to producing trophozoites and cysts, this flagellate protozoan is capable of infecting a large number of domestic animals (Geurden et al., 2010), as well as men (Thompson & Monis, 2004); this microorganism is also highly evolved and with the capacity for recombination among their Assemblages (Cacciò & Sprong, 2010).

13. Clinical signs

In general, the clinical signs of parasitized animals consist in diarrhea (Fortes, 2004). Gastrointestinal disorders may manifest severely in immunosuppressed individuals (Assefa et al., 2009), while clinical manifestation variation, infection persistence and severity of symptoms are directly correlated to TCD4+ lymphocyte count (Gupta et al., 2008).

Similarly to cryptosporidiosis, giardiasis may develop varied symptoms, especially acute diarrhea, abdominal pain (Springer et al., 1997; Cimerman et al., 1999), anemia and loss in the energetic and protein values (Ertan et al., 2002; Gendrei et al., 2003).

14. Diagnosis

The diagnosis of *Cryptosporidium* spp. and *Giardia* spp. must always be made by associating two or more techniques in order to increase the diagnosis efficacy (Mtambo et al., 1992; Huber et al., 2004; Coelho et al., 2009).

The intermittent release of *Cryptosporidium* oocysts requires that coproparasitological tests be repeated, including new sample collection, even after a negative result (Huber et al., 2002; Brook et al., 2008; Huber et al., 2005).

15. Treatment

To treat cryptosporidiosis, nitazoxanide, trimethoprim-sulfamethoxazole and pyrimethamine can be used with certain efficacy once there is no immunosuppression associated. The treatment of giardiasis has included metronidazole, nitazoxanide, furazolidone, quinacrine and paramomycin (Petri Jr., 2003).

16. Agents

Toxoplasma gondii.

17. Epidemiology

In Brazil, Dalla Rosa et al. (2010) and Bresciani et al. (2007) proved by means of serological methods the occurrence of anti-*T. gondii* antibodies in 14.33% (43/300) and 25% (100/400) of the analyzed cats, respectively. Also in Brazil, prevalence rates of 35.4% (84/237) were found by Silva et al. (2002) and 26.3% (132/502) by Pena et al. (2006).

Lucas et al. (1998) and Garcia et al. (1999) suggested that toxoplasmic infection is predominantly more frequent among younger animals, confirming that the prenatal stage is predominant for acquiring this infection.

This was confirmed in our study, in which 15.7% (11/70) cats were seroreactive for *T. gondii*, which occurred mainly in young animals (Coelho et al., 2011b). Association between sex and breed with occurrence of infection by *T. gondii* was not verified by Bresciani et al. (2007); Pinto et al. (2009) and Dalla Rosa et al. (2010).

18. Physiopathogenesis

Soon after the ingestion of environmental oocysts or tissue cysts, the parasite causes systemic infection, resulting in bradyzoite production (Dubey, 2010). It must be highlighted that toxoplasmosis manifests more severely in immunosuppressed individuals, especially those showing TCD4 lymphocyte count lower than 100 cells per mm³ (Hoffmann et al., 2007).

19. Biology

As to *T. gondii* biology, it is important to emphasize that this parasite has zoonotic potential (Dubey, 2010), showing oocysts capable of contaminating the environment and remaining infective for long periods (Elmore et al., 2010).

The occurrence of these protozoan diseases has been correlated to management, environment (Modolo et al. 2008), livestock by-products (Hiramoto et al., 2001) and even dissemination through water (Jones & Dubey, 2010).

20. Clinical signs

Infection by *T. gondii* can cause several lesion levels in the host, including the asymptomatic forms, in addition to retinochoroiditis (Alves et al., 2010), cerebral lesions, psychiatric disorders (Torrey & Yolken, 2003; Youken et al., 2009) and disseminated forms (Barbosa et al., 2007).

It is an opportunistic infection, common in immunosuppressed patients, being the most common cause of secondary infection of the central nervous system, causing the occurrence of severe encephalitis (Collazos, 2003; Pradhan et al., 2007).

Experimental infections in cats are often asymptomatic, few animals get sick and deaths rarely occur (Omata et al., 1990; Sato et al., 1993). However, Dubey et al. (1996) and Elmore et al. (2010) report the occurrence of some lesions in neonates.

Experimental infections in cats are frequently asymptomatic, a few animals become ill and deaths are rare (Omata et al., 1990; Sato et al., 1993). Abortion and neonatal mortality have been described for pregnant cats orally inoculated with *T. gondii* tissue cysts (Powell et al., 2001).

21. Diagnosis

In addition to clinical manifestations, fecal analyses and molecular techniques (Elmore et al., 2010), several serological techniques have been the main methods employed for toxoplasmosis diagnosis (Camargo, 1964; Lappin et al., 1989; Dubey et al., 2004; Coelho et al., 2011b).

In humans, behavioral changes (Zhu, 2009), encephalic lesions (Zajdenweber et al., 2005) and ocular (Alves et al., 2010) may indicate presence of infection.

22. Treatment

Toxoplasmosis treatment includes sulfonamides, trimethoprim, pyrimethamine, ponazuril, clindamycin and their associations can be successfully employed (Mitchell et al., 2006; Dabritz et al., 2007).

23. Agents

Leishmania spp.

24. Epidemiology

The occurrence of leishmaniasis in domestic cats has been reported in a large number of countries (Mancianti, 2004; Maia et al., 2008; Silva et al., 2008). In our study, only the species *Leishmania* (*L.*) *chagasi* was found in the analyzed cats (Coelho et al., 2011c), which could be associated or not with other diseases (Coelho et al. 2010). Also in Brazil, Savani et al. (2004) and Silva et al. (2008) found *Leishmania* (*L.*) *infantum*. The latter has been equally described in cats in France (Ozon et al., 1998), Italy (Pennisi et al., 2004), Spain (Ayllon et al., 2008) and Iran (Hatan et al., 2010).

The cutaneous form of *Leishmania* (V.) *braziliensis* was described in two cats from Rio de Janeiro State, Brazil (Schubach et al., 2004), while *Leishmania* (L.) *amazonensis* was described in Mato Grosso do Sul State (Souza et al., 2005). Craig et al. (1986) detected the occurrence of *L. mexicana* in cats from Texas, USA.

Studies of animal epidemiology have evidenced several infection prevalence levels according to the employed method and the study site. In our study, the analyzed tissue samples were from 52 domestic cats with 5.76% positivity. Similarly, Rossi et al. (2007) detected 6.7% positivity for *Leishmania* spp. among 200 analyzed cats.

Percentages superior to those obtained in our study were found in Portugal by Maia et al. (2008), who observed 30.4% (7/23) felines carrying leishmaniasis. In Greece, Diakou et al. (2009) verified that 3.87% (11/284) cats had anti-*Leishmania* antibodies. Similarly, in Spain, Solano-Galego et al. (2007) analyzed anti-*Leishmania infantum* antibodies from 445 cats and observed seroreactive prevalence in 6.29% of these animals.

25. Physiopathogenesis

After parasite replication, there is formation of perivascular congestion, mononuclear and neutrophil inflammatory infiltrate (Schubach et al., 2004) with secondary bacterial (Coelho et al. 2010) and fungal infections (Ozon et al., 1998) at the lesion sites. Lesions may be localized or systemic, affecting different organs, and may be associated with FIV/ FeLV; in these cases, the most severe form of the disease occur (Pennisi et al. 2002; 2004).

26. Biology

This heteroxenic protozoan has mammals as its definitive hosts and dipterans of the genera *Lutzomyia* and *Phlebotomus* as intermediate hosts and vectors (Fortes, 2004). As the dog is considered the main urban reservoir of this disease although there are frequent reports of this infection in cats, the role of felines in the biological cycle of this parasite is not well defined yet (Dantas-Torres et al. 2006).

However, xenodiagnosis studies carried out by Maroli et al. (2007) proved that sand flies are capable of acquiring the infection from naturally infected cats.

27. Clinical signs

Skin lesions are more frequent among felines. Infected animals may show vegetative lesions, dermatitis and ulcers (Coelho et al. 2010a); healthy animals may also carry this parasite (Coelho et al., 2010b), and in some cases the disseminated form may occur (Ozon et al., 1998).

Weight loss, pale mucosae, dehydration, systemic lymphadenomegaly and hepatomegaly, and ocular lesions are the main manifestations (Pennisi et al. 2004).

Laboratorial changes are irregular and may include pancytopenia (Marcos et al., 2009), hyperleukocytosis (Ozon et al., 1998), and discreet or no biochemical alteration (Souza et al., 2009).

28. Diagnosis

Diagnosis is based especially on serological (Mancianti, 2004), parasitological (Bresciani et al., 2010), molecular analyses (Coelho et al. 2010b), isolation in culture medium (Simões-Matos et al., 2004), and clinical manifestations (Dantas-Torres et al., 2006).

Clinical tests in places where the disease is endemic have shown that some infected animals remain seronegative (Ferrer et al., 1999). The serological titer shown by the animal is not related to the presence of symptoms and their intensity (Lima et al., 2003). On the other hand, PCR sensitivity and specificity are very high and this technique can detect the DNA of parasites in patients that remain clinically healthy for many years (Ferrer et al., 1999).

In a previous study, our research group suggested that antibody production in response to *Leishmania* spp. in felines is very low, which led to no serological reactions by means of IFA and ELISA (Serrano et al., 2008).

29. Treatment

Treatment may be based on allopurinol, meglumine antimoniate and ketoconazole (Pennisi et al., 2004). Rüfenacht et al. (2005) reported the use of griseofulvin, itraconazole, ketoconazole, selamectin, lufenuron, cephalexin and prednisolone for leishmaniasis treatment in cats.

30. Final considerations

The high occurrence of endoparasites observed among domestic and stray animals evidences the zoonotic potential of these helminth and protozoan diseases, suggesting greater concern about the therapeutic and prophylactic measures feasible to the feline population.

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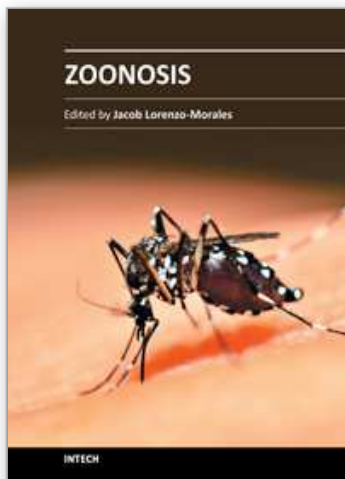
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Zoonotic diseases are mainly caused by bacterial, viral or parasitic agents although "unconventional agents" such as prions could also be involved in causing zoonotic diseases. Many of the zoonotic diseases are a public health concern but also affect the production of food of animal origin thus they could cause problems in international trade of animal-origin goods. A major factor contributing to the emergence of new zoonotic pathogens in human populations is increased contact between humans and animals. This book provides an insight on zoonosis and both authors and the editor hope that the work compiled in it would help to raise awareness and interest in this field. It should also help researchers, clinicians and other readers in their research and clinical usage.

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