Parasites & Vectors

REVIEW

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LeishVet update and current recommendations on feline leishmaniosis

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Abstract

In 2015, the LeishVet group published a comprehensive review on feline leishmaniosis (FeL) caused by *Leishmania infantum*, highlighting the growing interest in the disease despite the limited availability of evidence-based studies at the time. Over the past decade, considerable advances have been made in various aspects of animal leishmaniosis, including FeL. Veterinary awareness has increased, and diagnostic support is more widely available. However, current knowledge on FeL still largely comes from case reports and small case series studies, with fewer analytical studies and a lower evidence level compared to canine leishmaniosis. In response, the LeishVet group reviewed recent progress and provided updated recommendations based on key international publications, clinical expertise and expert consensus. A series of questions was formulated to address the main aspects of FeL most relevant for veterinary practitioners, including etiology, transmission, epidemiology (including geographical distribution and risk factors), immunology, clinical presentations and clinicopathological findings, the role of coinfections and/or comorbidities, diagnosis, prognosis formulation based on clinical staging, treatment, monitoring and prevention of FeL. The answers are largely supported by visual aids (tables, figures, and algorithms). This updated review paper focuses mainly on *L. infantum* infection and disease in domestic cats as it is the species of *Leishmania* most frequently diagnosed in cats. Two specific questions have also been dedicated to infection in wild felids and *Leishmania* spp. other than *L. infantum*, for supporting veterinary specialists in wildlife and zoo medicine and taking in consideration that globalization opens new challenges to practitioners.

Keywords Cat, Clinical staging, Diagnosis, *Felis catus*, Infection, *Leishmania infantum*, *Leishmania* spp., Prevalence, Treatment and prevention, Wild felids

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Background

In 2015, the LeishVet group published a comprehensive review on feline leishmaniosis (FeL) caused by *Leishmania infantum*, highlighting the growing interest in the disease despite the limited availability of evidence-based studies at the time [1]. Key knowledge gaps included risk factors, clinical signs, diagnostic protocols, treatment options, prognosis, and prevention strategies in cats. Additionally, awareness among veterinary professionals regarding FeL has remained low. To address these issues, LeishVet developed a set of clinical recommendations based on a thorough review of the relevant international literature, the clinical expertise of its members and expert consensus discussions [1].

Over the past decade, considerable advances have been made in various aspects of animal leishmaniosis, including FeL [2–6]. While most of the data still focuses on canine leishmaniosis (CanL), it is now well recognized that feline infection is common in endemic regions and that infected cats can contribute to sand fly transmission [7]. Veterinary awareness has increased, and diagnostic support is more widely available. However, current knowledge on FeL largely comes from case reports and small case series, with fewer analytical studies and a lower evidence level compared to CanL. In response, the LeishVet group reviewed recent progress and has provided updated recommendations based on key international publications, clinical expertise and expert consensus. A series of questions has been developed on the topics considered most relevant for practitioners and the answers provided have been largely supported by visual aids (tables, figures, algorithms).

The aim of this review was to critically evaluate new data and findings on FeL published since the release of the previous LeishVet guidelines on this topic [1]. The review primarily focuses on infection and disease caused by *L. infantum* in domestic cats and provides updated LeishVet recommendations accordingly. In addition, specific sections address leishmaniosis in wild felids and infections caused by *Leishmania* spp. other than *L. infantum*, to support veterinary specialists working in wildlife and zoo medicine and to consider the emerging posed by globalization.

Methodology

The PubMed database was searched based on the following keywords and Boolean operators: ("Leishmani*"[All Fields]) AND ("cat"[All Fields] OR "cats"[All Fields] OR "catus" [All Fields] OR "feline*"[All Fields] OR "felid*"[All Fields] OR "felis"[All Fields]). Articles published between January 2015 and December 2025 were considered for initial analysis, without distinction between reviews, research articles, clinical trials and case series with confirmed diagnosis. The reference lists of the selected articles were consulted to look for additional references, this turn with no mandatory time limits. Thus, some articles published before 2015 were also included, as well as conference abstracts, online documents and personal communications from some LeishVet members. The LeishVet group reached their final recommendations after several rounds of discussion.

Etiology and transmission

Which species of *Leishmania* infect cats and what is their geographical distribution?

In total, eight species of the genus *Leishmania* have been found to infect cats in different locations around the world. *Leishmania infantum* is the only species present both in the Old (Africa, Asia and Europe) and New (Americas) Worlds and infects cats in areas of endemicity for human and canine disease (Figs. 1 and 2). *Leishmania major, Leishmania tropica* and *Leishmania tarentolae* have been found to occur naturally in the Old World only, and *Leishmania amazonensis*, *Leishmania braziliensis*, *Leishmania guyanensis* and *Leishmania mexicana* in the New World only, where the name *Leishmania chagasi* was often used previously and is now considered as a synonym of *L. infantum.* Except for *L. tarentolae*, all the *Leishmania* spp. that infect cats have proven a zoonotic character, i.e., they can also infect and cause disease in humans [8], apart from other mammals, including dogs or other felids [7]. In recent years, the most widely used method for characterizing *Leishmania* spp. has been the polymerase chain reaction (PCR) followed by DNA sequencing. PCR with restriction fragment length polymorphism (RFLP), high resolution melt analysis (HRM) and multilocus isoenzyme electrophoresis (MLEE) have also been used, although on a smaller scale.

Similar to what has recently been studied in dogs [9, 10], infection by *L. tarentolae*, a parasite normally hosted by lizards, has also just been described in cats in Italy. Colony or stray cats with a mostly complete outdoor lifestyle may be exposed to *L. tarentolae* in areas where *L. infantum* is endemic and some cats harbour both parasites [11]. The real impact of *L. tarentolae* (particularly in terms of possible serological cross-reactions during diagnosis or possibly repercussions on the immune response) remains to be determined.

How is *Leishmania* transmitted to and from cats?

Like other trypanosomatids, *Leishmania* spp. have coevolved with their arthropod hosts, i.e., sand fly vectors that transmit them to vertebrate hosts [12]. Sand flies can take their blood meal from a wide variety of vertebrates: mammals (including humans), birds, or reptiles [13], depending on the specific vector species feeding preferences [14, 15].

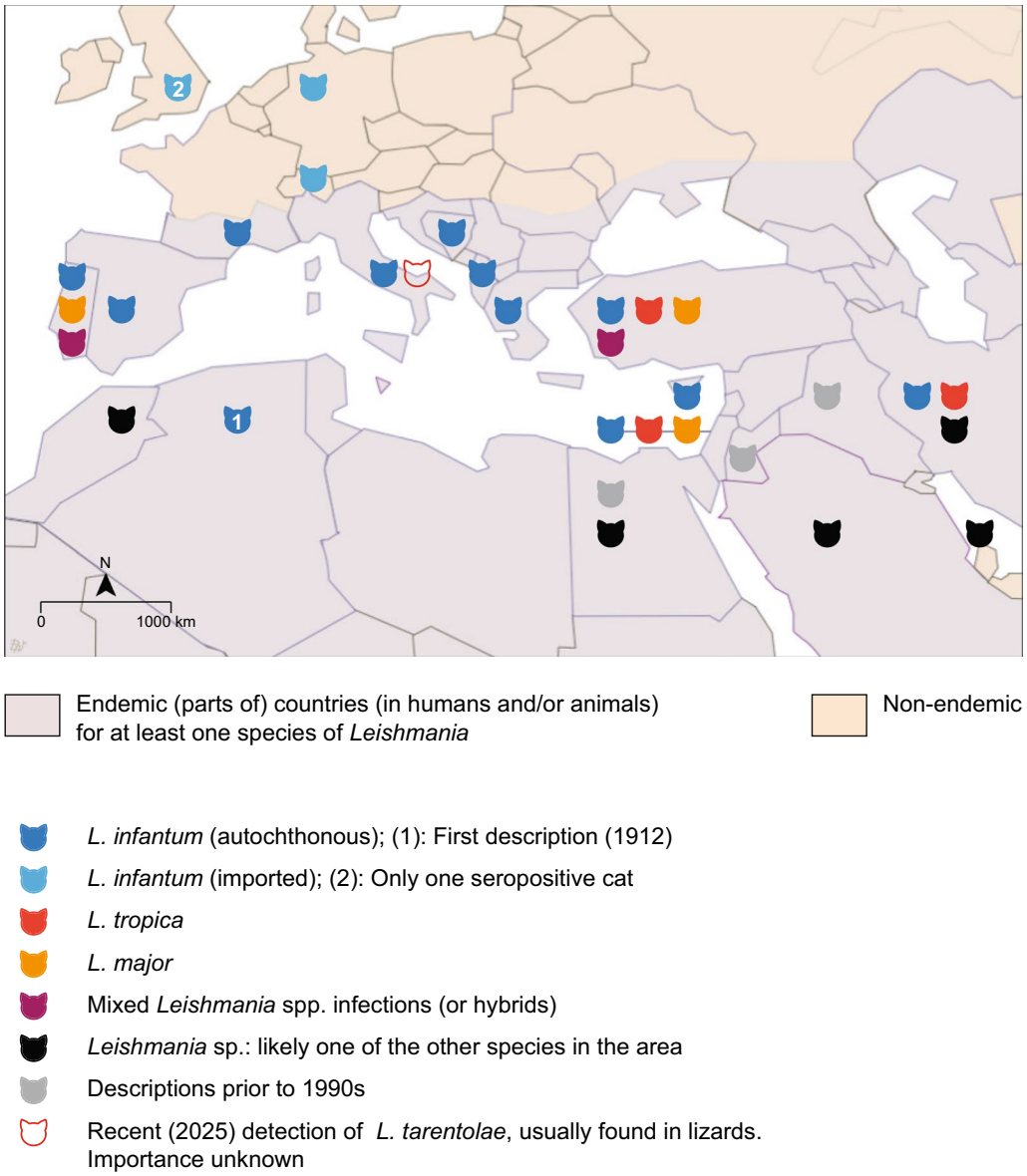


Fig. 1 *Leishmania* spp. detected in domestic cats (*Felis catus*) in the Old World (details in Additional file 1: Table S1)

Sand fly species which are competent vectors of *Leishmania* spp. can thus spontaneously feed on cats, as some studies have documented [16–19]. Xenodiagnostic studies have demonstrated the vector's ability to complete feeding on cats. These experiments have also proven that cats infected with *L. infantum* can effectively transmit the parasite to the sand fly as shown for *Phlebotomus perniciosus* in Europe [20] and for *Lutzomyia longipalpis* in South America [21–23]. Therefore, cats constitute potential sources of this parasite and may contribute to the global reservoir. No data are available on the infectiousness of healthy infected cats and information on other, non-vectorial transmission pathways in cats is currently very limited. These potential pathways are shown in Fig. 3, which allows for comparison with what is known in dogs. For example, *Leishmania* DNA can be detected in some tissues and blood of apparently healthy cats, and the risk of parasite transmission by blood transfusion has been suggested [24]. Other transmission modalities, including vertical or horizontal pathways, have been the subject of two investigations with a relatively small number of enrolled cats [25, 26], but they have not been proven in these vertebrate hosts, as is the case in dogs, mice or humans [27–30]. Nevertheless, the presence of DNA of *Leishmania* was documented in testes and

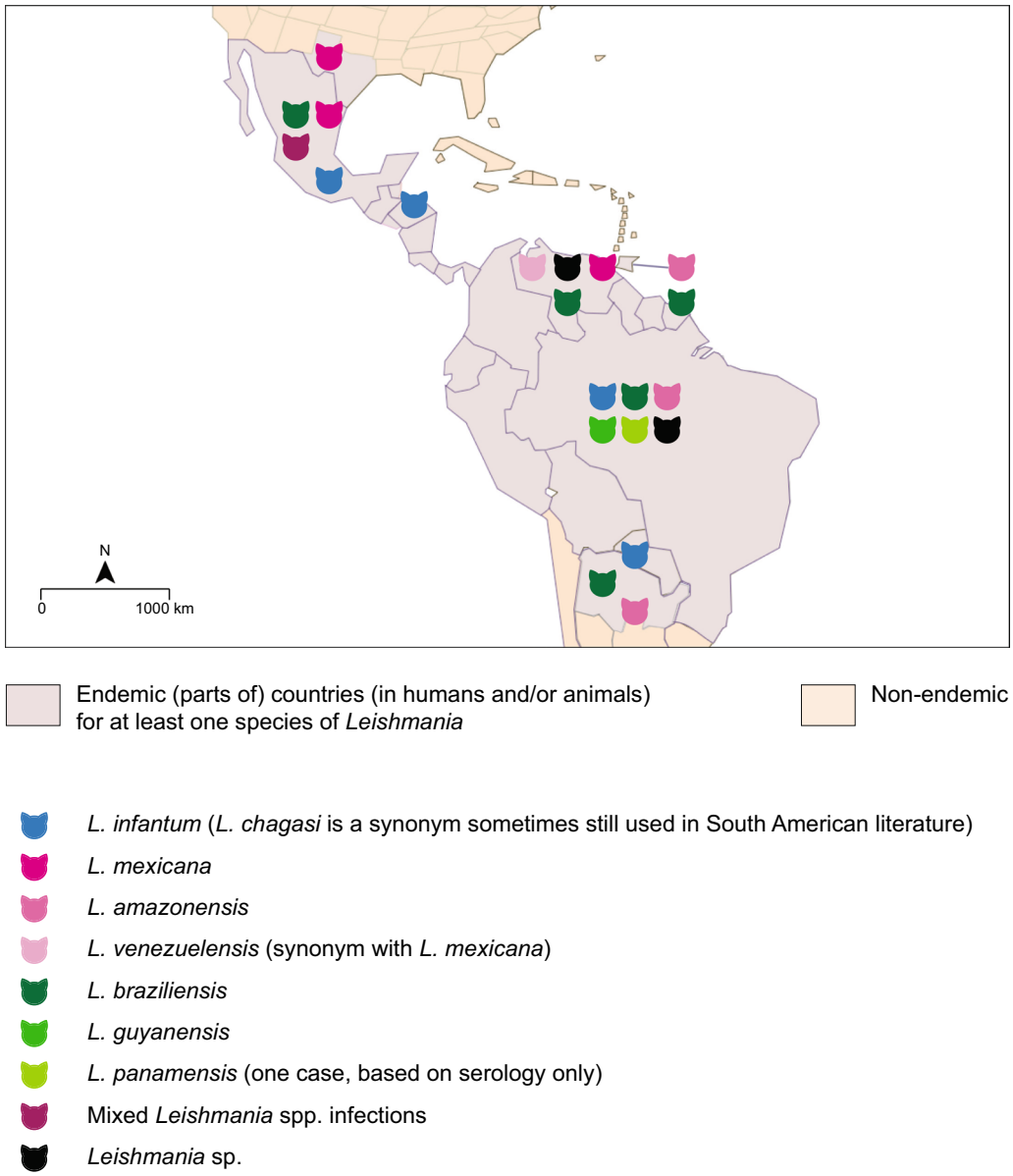


Fig. 2 *Leishmania* spp. detected in domestic cats (*Felis catus*) in the Americas (details in Additional file 2: Table S2)

epididymis of two males (the one tested was seropositive) and the ovaries and uterus of a seropositive female cat [26]. A recently published study showed the frequency of genital lesions of epididymitis in male cats, particularly those who were seropositive, as well as endometritis lesions in females. Furthermore, in pregnant female cats, at least one fetus tested positive for PCR, thus suggesting the hypothesis of transplacental transmission [25]. These results are similar to other observations in dogs in which the effectiveness of the venereal and vertical transmission routes has been well established [31], although the percentage of positive newborn kittens was low [25]. Although *L. infantum* is an inoculable pathogen, other modes of horizontal transmission have not yet been evaluated in cats. The frequency of fights in cats (scratches and bites with blood exchange) could play a role, as is already known for other infectious diseases. With all this taken into consideration, non-vectorial transmission in felines needs further investigations.

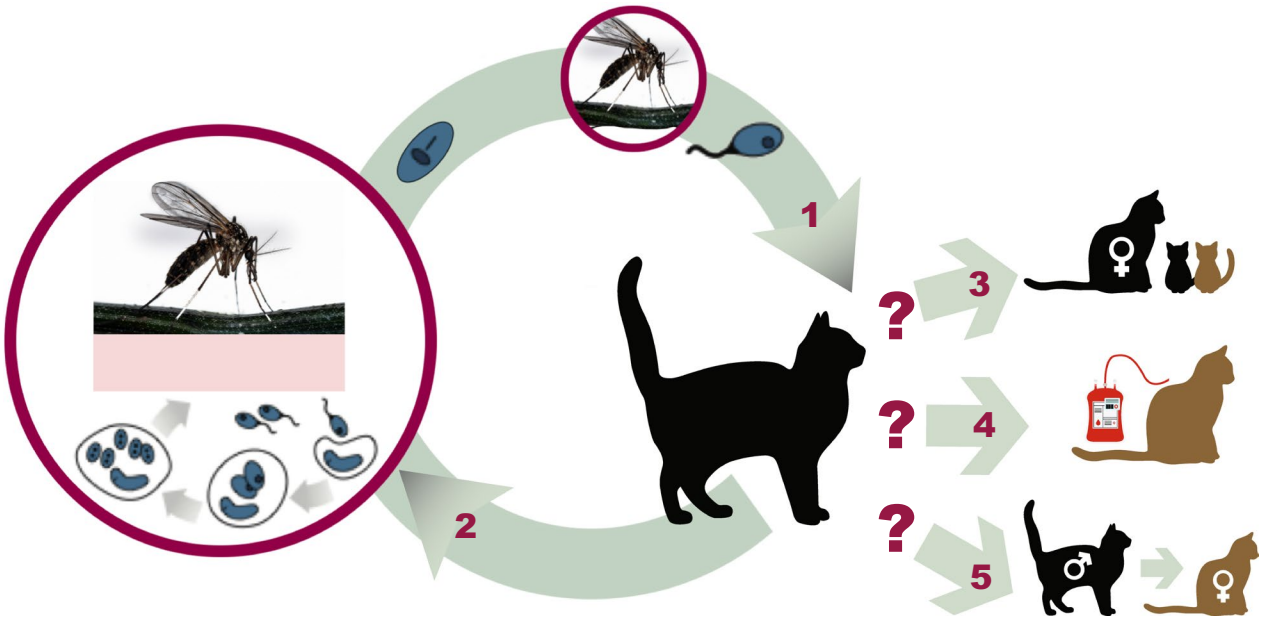


Fig. 3 Proven vectorial transmission of *Leishmania infantum* in cats and possible non-vectorial transmission modes. 1. Vectorial transmission through bites of infected sand flies. 2. Source of infection for sand fly vectors (experimentally proven with *Phlebotomus perniciosus* and *Lutzomyia longipalpis*). Cats contribute to the reservoir of *L. infantum*. 3. Vertical transmission (during pregnancy) is well demonstrated in dogs. PCR positive fetuses were detected in pregnant positive queens (serologically or by PCR). 4. The risk of transmission through blood transfusion has been demonstrated in both humans and healthy infected blood donor dogs. Although no such study is available in cats, positive blood PCR results from infected cats also suggest this risk. 5. Venereal transmission (not demonstrated in cats as it has been in dogs): genital tract *Leishmania* infections have been observed in both male and female cats. ?, Potential, but not clearly demonstrated for cats yet. PCR, polymerase chain reaction

Epidemiology including risk factors and geographical distribution

What is the prevalence of *L. infantum* infection in cats in endemic regions?

The detection of *L. infantum* infection in cats has been the subject of a growing number of studies over the past 10 years. These studies expand the information previously published [1].

Detection is mainly based on serological techniques (indirect fluorescent antibody test [IFAT], enzyme-linked immunosorbent assay [ELISA], direct agglutination test [DAT], western blot [WB] and Dual-Path Platform [DPP®] rapid immunochromatographic test) and the use of molecular techniques (including conventional PCR and quantitative real-time PCR [qPCR]) carried out on blood, bone marrow and lymph node aspirates, swabs (conjunctival or oral) or biopsies. The use of cytology (bone marrow and lymph nodes) is occasional and culture is rarely used.

Additional files 1 and 2: Tables S1 and S2 present the studies conducted over the last decade, representing over 100 publications covering 17 Old World and two New World countries. Figure 4 shows the number of studies conducted in each country, as well as the overall results.

Leishmanial infection has been found in cats in all regions where CanL is endemic. Seroprevalence in cats ranges from 0.0 to 75% and molecular levels of infection range from 0.0 to 60.7% in endemic regions of the Old World. Serological and molecular prevalence values are, respectively, from 0.0 to 74% and from 0.0 to 91.7% in South America.

The results vary greatly among studies, which are often difficult to compare. The measured prevalence values concern very disparate categories of cats, and the terminology used to categorize the cats is variable and not standardized (see Additional files 1 and 2: Tables S1 and S2). Furthermore, the number of cats enrolled in the studies varies from a few to several hundred, so the calculated prevalence cannot be considered representative of local feline populations in general. Finally, there are differences between the diagnostic methods used and, for example, the serological cut-off chosen, which can significantly influence the percentage of infected cats detected. In most studies, very low serological levels are neither sought nor used, which likely leads to a bias in the true infection values. It is interesting to note that the prevalence values obtained by serology (IFAT and/or ELISA) are higher than those obtained by detecting parasite DNA in the cat blood in approximately 75% of

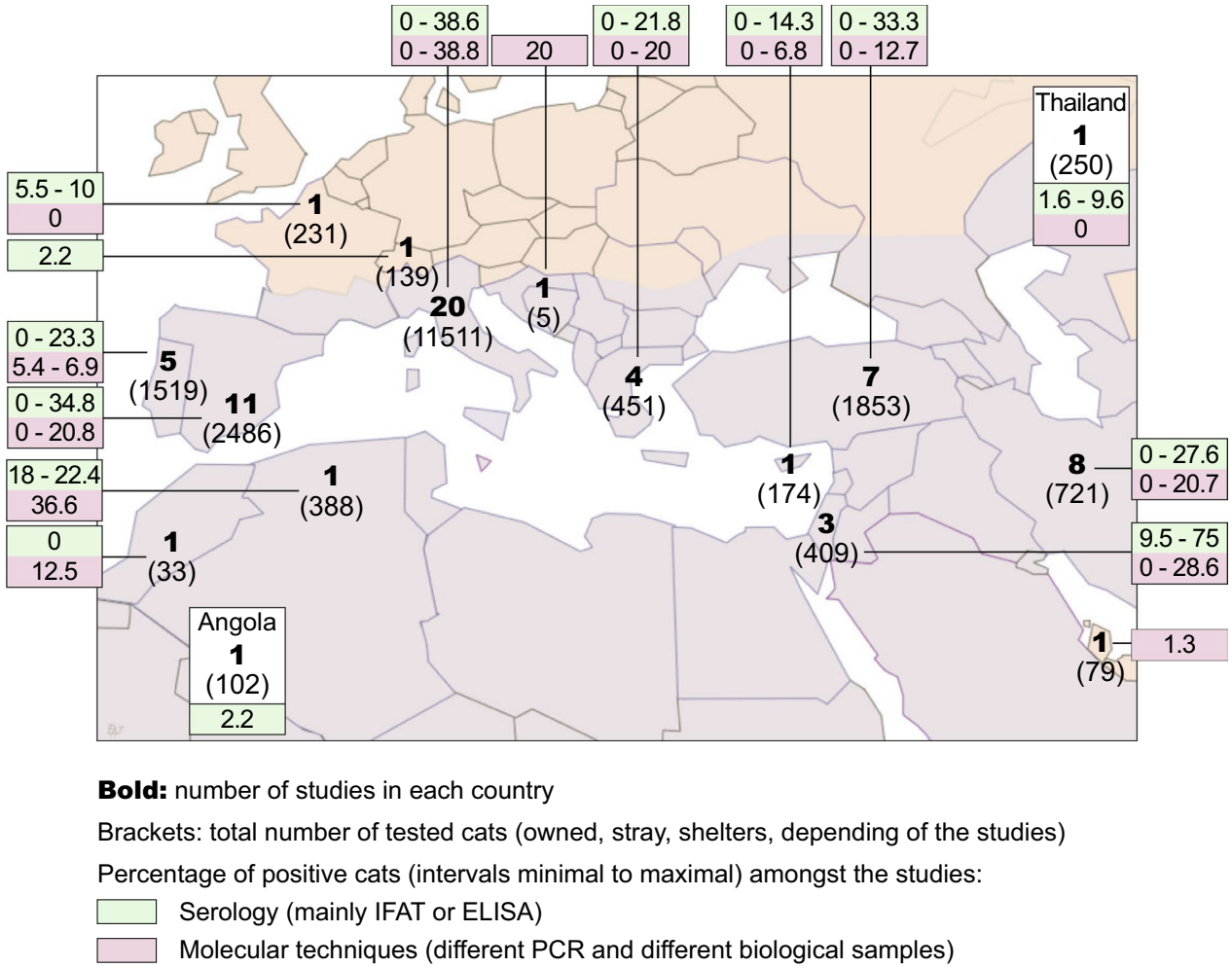


Fig. 4 *Leishmania* infection in cats from studies conducted during 2015–2025 in Southern and Eastern Europe, North Africa, the Middle East, and Asia (details in Additional files 1 and 2: Tables S1 and S2): ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody assay; PCR polymerase chain reaction

the studies using both approaches. Similarly, using IFAT, the prevalence values are higher than those obtained with ELISA in approximately 75% of studies using both techniques. Few studies have validated the serological techniques in cats using feline positive controls obtained from cats with clinical illness confirmed by isolation and negative control sera from a substantial number of cats from non-endemic areas [32–36]. Regarding the prevalence of disease, it is less frequently reported than in their canine counterparts. Just as the infection is often non-clinical in many wildlife species, immune responses that lead to natural feline resistance may account for the observed differences in the prevalence of infection in cats as compared with dogs [1]. Studies evaluating *Leishmania* specific cellular immunity in cats may be able to better estimate infection, but they are still poorly reported in cats [32, 37, 38].

What are the risk factors for infection with *L. infantum* in cats?

Prevalence studies based on serology, PCR or both have revealed several variables to be associated with positivity to *L. infantum*, both in the Old and New Worlds (Additional files 1 and 2: Tables S1 and S2). Those variables include:

1. Adult age [39–45].
2. Free-roaming or shelter conditions [41, 44, 46–49].
3. Male sex [40, 45, 47, 50, 51].
4. Sampling in winter [47, 52, 53].
5. Cohabitation with dogs [54] or with *Leishmania*-positive dogs [55].
6. Non-neutered status [43].
7. Lack of backyard cleaning and garbage collection [55].

8. Rural origin [45].
9. Coinfections with or seropositivity to some other pathogens, including: feline immunodeficiency virus (FIV) or feline leukemia virus (FeLV) [34, 40, 43, 47, 56, 57], feline coronavirus (FCoV) [58], severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), *Toxoplasma gondii*, *Dirofilaria immitis*, feline herpesvirus 1 (FHV-1) [59], *Hepatozoon* spp., "*Candidatus* Mycoplasma turicensis" [60] and *Mycoplasma* spp. [56].

The presence of antibodies to *P. perniciosus* salivary proteins in cats, a marker of exposure to sand fly bites [61], was also associated with positivity to *Leishmania* spp. by serology and PCR.

What is the epidemiological role of *L. infantum* infected cats?

Available data on the epidemiology of *L. infantum* infection in cats in endemic areas most often show that, if the same detection criteria are used, the prevalence of feline infection is lower than the prevalence detected in dogs [42, 62–65]. Furthermore, analyses of the contents of sand fly blood meals suggest that cats are usually not frequent hosts [14, 15].

The relative rarity of feline clinical cases compared to the occurrence of the disease in dogs in endemic areas also supports that view. However, infection levels are not negligible and may be locally equivalent [57] or even higher than those detected in dogs [66].

The chronicity of feline infections, combined with the ability to infect vectors, makes them excellent sources, clearly suggesting that the cat contributes to the reservoir pool of *Leishmania* to other hosts, including humans. Therefore, studies with infected and healthy cats should be extended to identify their role in sand fly infection.

What do we know about the immune response against *L. infantum* infection in cats?

There is substantial evidence that the differences in the spectrum of immunological responses observed in dogs and humans with leishmaniosis determine the wide range of clinical manifestations associated with the disease [67–69]. Dogs with a strong T helper (Th) 1-mediated immune response tend to show a granulomatous inflammatory pattern with fewer parasites in the skin, while those with a predominant Th2 response exhibit a perivascular to interstitial mixed infiltrate (predominance of macrophages, lymphocytes and plasma cells) with a higher parasitic load [69]. In contrast, the knowledge of how host immune responses might modulate the clinical and pathological spectrum in cats in response to *Leishmania* infection is very limited. Some studies suggest that cats may mount an effective immune response that limits parasite replication, resulting in milder or subclinical infections [37, 38, 70]. However, the histopathological cutaneous features of FeL are less well characterized, and it is currently unclear whether the formation of granulomas in cats reflects a similar immunological control of the infection as observed in dogs [69].

There is more information regarding the adaptive humoral response than cell-mediated immune response in cats. Different levels of parasite-specific antibodies and interferon-gamma (IFN-γ) are variably detected in infected cats [37]. The detection of antibodies against *L. infantum* antigen is well documented in cats naturally [71] or experimentally infected with this species [72]. Cats diagnosed with clinical FeL are antibody-positive (IgG) to *L. infantum* with variable levels of antibodies detected by quantitative methods (IFAT, ELISA and DAT) [1, 36]. Similar to dogs, the antibody level appears positively correlated with parasite load in blood [37, 38]. Cats induce parasite-specific Th1-mediated immune response producing IFN-γ [37] and testing positive by delayed hypersensitivity reaction type IV with a suspension of killed *L. infantum* promastigotes (Montenegro skin test or leishmanin skin test [70]). Lode et al. [38] detected higher serum concentrations of IFN-γ and interleukin (IL)-12 in apparently healthy infected cats compared to apparently healthy non-infected cats.

The states of *L. infantum* infection in which a specific Th1 immune response is found are not well established [37, 38, 70]. Again, similarly to dogs, antibody-negative cats are able to produce specific IFN-γ [37]. However, lower levels of adaptive immune response (specific antibodies and IFN-γ) compared to dogs are generally seen [63]. Finally, a correlation between specific IFN-γ production and any clinical manifestation has not been determined, as it has been shown, for instance, in papular dermatitis due to *Leishmania* infection in dogs [73].

Clinical presentations of FeL caused by *L. infantum*

Information about the spontaneous disease caused by *L. infantum* in cats is supported by cases and case series and non-analytical studies. This means that there is no consolidated evidence-based knowledge on FeL. According to current data, FeL remains rare, as a disease, even in areas where leishmaniosis is common in dogs and feline infection is frequent [66]. There is currently no consolidated evidence of risk factors favouring the progression to disease in infected cats. It cannot be excluded that the disease is still underdiagnosed because it is neglected—or even ignored—by many practitioners, and clinical suspicion is challenging when suggestive signs are lacking or the clinical presentation is overlooked or mixed with signs of a concurrent disease. Moreover, in some endemic

areas, the level of medical care for cats is generally lower compared to dogs.

FeL appears to be a highly variable disease, similar to its canine counterpart. The infection spreads systemically in cats and the disease can affect multiple organs and tissues. Cats may show either systemic or just regional clinical signs, which may progress over time [74–77].

The course of FeL is generally chronic and progressive, and the disease has been diagnosed in adult cats (age range of 2–21 years) with a median age of 8 years [78]. The incubation period could be long-lasting and possibly influenced by immunosuppressive therapies and other factors [20]. In non-endemic areas, as is the case with dogs, some cats can manifest signs of FeL years after their relocation from endemic countries [79].

What do we know about coinfections and other medical conditions?

A review of 63 clinical cases of cats with *L. infantum* infection revealed that 66% had co-morbidities [78]. The main coinfection was with FIV and several studies have identified FIV to be associated with feline *L. infantum* infection [43, 48, 57, 78]. FIV progressively dysregulates the cat's immune system and CD4+ Th lymphocyte cells are the main target for the viral infection [80]. These cells have a central role in adaptive immune defense mechanisms, and their impairment may enhance susceptibility of FIV-infected cats to *L. infantum* infection and also promote the progression of infection to disease. Moreover, some immune-mediated disorders (feline chronic gingivostomatitis [FCGS], chronic kidney disease [CKD]) and clinicopathological abnormalities in the complete blood count (CBC; anemia, leukopenia, pancytopenia) and serum proteins (hyperglobulinemia, hypergammaglobulinemia) have in their differential diagnosis lists both FIV and *L. infantum*. Understanding when these disorders are affected by FIV comorbidity is clinically relevant as they may improve with anti-*Leishmania* therapy.

Given the clinical overlap of cutaneous lesions between FeL and feline sporotrichosis (*Sporothrix brasiliensis*), this emerging zoonotic fungal disease should be considered, particularly in endemic regions with high prevalence, such as Brazil [81]. Other infections and medical conditions, also associated with immune suppression, have been sporadically reported as comorbidities in FeL cases. These also include treatment with corticosteroids in approximately 20% of the reported cases with a comorbidity [78]. A cat with chronic *L. infantum* infection developed clinical disease after cytarabin, an antineoplastic drug, and methylprednisolone were administered [20]. Any other long-term immunosuppressive treatment should be considered a potential risk factor for developing clinical FeL, similarly to what is known for severe feline toxoplasmosis. Acute and lethal toxoplasmosis cases have been associated with cyclosporine treatment in cats under immunosuppressive therapy for atopic dermatitis and immune-mediated hemolytic anemia (IMHA) [82, 83]. Additionally, cats treated with cyclosporine or cytarabin have also been diagnosed with FeL in Spain (personal communications: L. Solano-Gallego, G. Miró). Overall, coinfections or other comorbidities are present in the majority of cats with clinical FeL and are a major contributor to the pathogenesis and clinical outcome of feline *Leishmania* infection.

What are the most common clinical signs?

The most commonly reported clinical signs of FeL, which constitute the most frequent reason for veterinary consultation in cats, are dermatological (cutaneous and/or mucocutaneous). Additionally, lymphadenomegaly is often detected during physical examination, and these are the two main complaints in cats. Systemic signs (lethargy, anorexia and weight loss), ocular and oral disease are less frequently reported, as well as a list of rarer manifestations (Table 1). It is important to note that published case reports and case series often do not establish a direct association between specific clinical signs and *L. infantum* infection, so any definitive claim about the prevalence of clinical signs in FeL should be considered with caution. In fact, coinfections and comorbidities may occur and contribute to the disease as a whole. CKD has been reported in cats at the time of FeL diagnosis [74, 77, 84, 85], and signs of CKD (polyuria/polydipsia, anorexia, lethargy, weight loss, vomiting) may be the reason for seeking veterinary consultation. In other cases, CKD develops over time in followed-up cats [74, 84]. Mild forms of CKD (International Renal Interest Society [IRIS] stages 1–2) may be underdiagnosed when urinalysis with urinary protein-to-creatinine (UPC) ratio and serum symmetric dimethylarginine (SDMA) are not measured [86]. Importantly, apart from possibly inducing an immune-mediated renal disease, *L. infantum* amastigotes can be directly found in the kidney in association with macrophagic inflammation [87] and cultivated from the kidney of cats with FeL and CKD (M.G. Pennisi: personal communication).

What are the most common dermatological patterns seen?

As mentioned above, cutaneous involvement is reported in the majority of cats with leishmaniosis (approximately 70–75%) [78, 88]. The most frequent lesions observed are ulcers and nodules (ulcerated or not), which may occur singly or in combination (Figs. 7 and 6). These lesions may also be located at mucocutaneous junctions, including the eyelids (Fig. 5), nose, lips and anal area [78, 88–92]. In contrast to CanL, alopecia with scaling,

Table 1 Clinical and clinicopathological abnormalities reported in feline leishmaniosis (listed in decreasing order of frequency)

Clinical findings (common)	Clinicopathological abnormalities (common)
• Skin and mucocutaneous lesions • Lymphadenomegaly • Ocular lesions • Oral lesions • Systemic signs (lethargy, anorexia, weight loss)	• Hyperglobulinemia • Hypergammaglobulinemia • Mild-to-moderate non-regenerative anemia • Proteinuria
Clinical findings (uncommon)	**Clinicopathological abnormalities (uncommon)**
• Pale/icteric mucous membranes • Splenomegaly/hepatomegaly • Chronic upper respiratory signs • Cachexia • Polyuria/polydipsia • Fever • Chronic diarrhea • Mammary gland swelling • Subcutaneous swelling • Abortion	• Renal azotemia • Hypoalbuminemia • Monocytosis • Neutrophilia • Pancytopenia • Hemolytic anemia (very rare)



Fig. 5 Nodular dermatitis in a Sphynx cat with leishmaniosis



Fig. 6 Ulcerative dermatitis in a Persian cat with leishmaniosis (same patient as in Fig. 7)

footpad hyperkeratosis and onychogryphosis are not often observed in FeL [21, 77, 88, 93].

A case initially misdiagnosed in García-Torres et al. [78] as having papular dermatitis involved a cat presenting with three well-defined erythematous and ulcerated plaques, each approximately 1 cm in length and a few millimetres wide, lacking the characteristic "volcano-like" appearance typically observed in papular dermatitis associated with *Leishmania* infection in dogs [93, 94]. In cats, papular lesions typically evolve into plaques or nodules, particularly along the margins of the pinnae. This distribution could reflect the site of sand fly inoculation, as reported in dogs. Other cutaneous patterns associated with severe CanL, such as pustular or vasculitic ulcerative dermatitis, have not been documented in feline cases [93].

Pruritus is seldom a direct clinical feature of FeL. Although resolution of severe itching following antileishmanial therapy has been reported in one case [95], in most cases pruritus is attributed to concurrent diseases such as flea allergy dermatitis, pemphigus foliaceus or squamous cell carcinoma, as reviewed in Pennisi et al. [1]. Notably, transient pruritus involving the trunk has been described as an adverse reaction to allopurinol (AL) in a case of FeL [75].



Fig. 7 Nodular blepharitis in a Persian cat with leishmaniosis (same patient as in Fig. 6)

Which are the ocular manifestations observed in FeL?

Ocular disease in FeL (unilateral or bilateral) varies greatly and, taken as a whole, is frequently reported in published cases. Signs of anterior uveitis or panuveitis (red eye, iris "bombé", flare, hyphema, anisocoria, dyscoria and synechiae between iris and the cornea or the lens) are the most commonly observed ocular signs [74, 76, 79, 88, 96]. Conjunctivitis and single or multiple conjunctival nodules [77, 88, 96], keratitis [88, 96] and chorioretinitis [88] have also been reported. When FeL is not promptly diagnosed and treated, the inflammation may lead to panophthalmitis [79, 97]. In some cases, cytological and/or histological confirmation of a causative role of *L. infantum* in the ocular disease has been well documented [77, 79, 96, 97]. Unilateral conjunctival edema (lower and third eyelids chemosis) was observed in a cat with forehead granulomatous panniculitis caused by *L. infantum* [98]. The cause of the severe chemosis was not investigated and it could have been a consequence of the severe local swelling.

What are the oral manifestations detected?

Nodules occur in the gingival mucosa, the tongue and lips and amastigotes were seen in lesional biopsies from cats with leishmaniosis [1, 88, 99, 100]. Feline chronic gingivostomatitis is often observed in FeL [88, 96] and it could be representative of a complex interaction among different pathogens. FCGS is a severe and frequent immune-mediated oral disease affecting up to 26% of domestic cats [101]. Retroviral infections are among the risk factors for FCGS [102] and FIV is the more frequent coinfection detected in FeL [1, 57]. This severe chronic oral disease may be caused by the alteration of local and/or systemic immune response due to viral infections (e.g., FIV, feline calicivirus), but the consequences of interactions among viral pathogens and *L. infantum* have not been investigated. Moreover, other infections (e.g., feline calicivirus) are associated with FCGS and they were not investigated in cases of FeL with oral manifestations. Amastigotes are associated with macrophagic inflammation and have been detected in oral tissue of cats with leishmaniosis and FCGS (personal communication: M.G. Pennisi).

Which rare localizations have been reported?

Rare signs are associated with infrequent detection of the parasite in the respiratory and gastrointestinal tracts and other tissues as summarized below. Also, splenomegaly is sporadically reported, and *Leishmania* amastigotes were histologically detected in the spleen of necropsied cats [77, 95, 96, 103, 104]. Importantly, the cytological evaluation of fine needle aspirates in cats with splenomegaly may confirm the diagnosis [77, 95, 104].

Chronic upper respiratory tract disease is reported in some FeL cases and a causative role of *L. infantum* has been confirmed in some cats [74, 105, 106] or suspected because of clinical cure obtained with anti-*Leishmania* treatment [95]. Nodules were observed in the nasal cavity and the nasopharynx, and an ill-defined invasive mass extended to the rostral cranial cavity in a very severe case [74, 105, 106]. Chronic nasal discharge was the more frequent manifestation, and in severe forms signs of the obstructive syndrome (inspiratory dyspnea and stertor) were the main complaint. Interestingly, amastigotes were found in a bronchoalveolar lavage of an atopic cat with chronic respiratory disease treated with cyclosporine for 2 years (personal communication: G. Miró).

Chronic diarrhea was the main complaint in a cat with granulomatous gastroenteritis caused by *L. infantum* and the most severe lesions were detected in the duodenum [104].

Mastitis was reported in an old cat with a clinical recurrence of FeL under AL treatment. The diagnosis of *Leishmania*-associated mastitis was confirmed by cytology and PCR [76].

Regarding other rare signs seen, a clear association of the clinical signs observed and FeL has not been documented. This is the case of fever [88, 107] and icterus [85, 88]. FeL was diagnosed by cytological evaluation of an enlarged lymph node and antibody detection in a cat chronically treated with methylprednisolone and presented with multifocal neurologic signs [88]. The causative role of FeL in the neurological signs was not investigated, but clinical cure with anti-*Leishmania* treatment was reported [88].

Table 2 Differential diagnoses for each dermatological pattern of feline leishmaniosis, along with valuable clinical insights

Clinical pattern	Main clinical differentials[a]	Clinical insights
Cutaneous and mucocutaneous nodular dermatitis	Other infectious diseases (sporotrichosis, cryptococcosis, deep opportunistic mycosis, pseudomycetoma, mycobacteriosis, toxoplasmosis, dirofilariosis, protothecosis), eosinophilic granuloma complex, mosquito bite hypersensitivity, cutaneous xanthoma, neoplasia (mast cell tumor, lymphoma, feline progressive histiocytosis, feline sarcoid)	Nodules are frequently found on the head, ears, and limbs, initially presenting as erythematous, non-fluctuant and non-ulcerated lesions. Compared to CanL, feline lesions tend to be more localized rather than diffuse Unlike in other infectious skin diseases, purulent exudation is typically absent
Spontaneous ulcerative dermatitis	Viral infections (feline herpesvirus-associated dermatitis, calicivirosis), deep bacterial or fungal infections (sporotrichosis), idiopathic ulcerative dermatitis (located in the neck), adverse drug reactions (vasculitis, erythema multiforme), neoplasia (squamous cell carcinoma in white skin)	Ulcers often present as chronic, non-healing lesions, commonly located on the head, nose, ears, and limbs (symmetrical possible). Irregular borders and a granulating base. Unlike in other infectious skin diseases, purulent exudation is typically absent
Exfoliative (scaling) dermatitis	Dermatophytosis, cheyletiellosis, thymoma or non-thymoma associated exfoliative dermatosis, feline atopic skin syndrome, idiopathic or mucinotic lymphocytic folliculitis, idiopathic sebaceous adenitis, cutaneous epitheliotropic T cell lymphoma	Diffuse or patchy scaling, often affecting the head, neck, and limbs. The skin appears dry with fine to coarse scales, and mild to moderate alopecia may be observed

[a] Differential diagnoses for each dermatological pattern should be carefully considered based on prevalence of any disease in a particular geographical area, clinical presentation, lesion distribution, and associated cutaneous or extra-cutaneous signs

What clinicopathological findings may alert to the possibility of FeL?

Unfortunately, most data on clinicopathological findings in cats are limited to individual case reports where routine clinicopathological evaluation was performed [1, 74–77, 79, 85, 88, 95, 96, 98, 99, 104–109]. The most frequently reported laboratory abnormalities are displayed in Table 1 and include alterations compatible with chronic inflammation such as hyperproteinemia and hyperglobulinemia, typically associated with polyclonal hypergammaglobulinemia, and mild-to-moderate, normocytic, normochromic, non-regenerative anemia [74, 76, 88, 95, 96, 98, 99, 104, 105, 107, 108]. In a cat with severe chronic rhinosinusitis, a bi-clonal gammopathy on capillary serum protein electrophoresis was found and in the nasal lesion a high number of plasma cells associated with light chain type amyloid deposition were found [106]. In this severe case, a plasma cell dyscrasia concurrent with FeL was diagnosed, but the occurrence of a mature-type plasma cell tumor could not be totally excluded [106].

However, in some cases no alterations in the CBC were found [74, 96, 107, 108] and other hematological findings such as thrombocytopenia [76, 77, 96, 98], pancytopenia [110, 111], leukocytosis, neutrophilia, monocytosis, lymphocytosis [77, 79, 85, 88, 109], eosinophilia [105], and leukopenia [99] were reported but were less common. Hypoalbuminemia [88, 99, 106] as well as increased alpha-2 globulin levels [79, 88] were also seen. Other inflammatory markers, such as increased serum amyloid A [79, 106] and erythrocyte sedimentation rate [109] were observed. Additionally, less frequent biochemical abnormalities include increased bilirubin and elevated liver enzymes (alanine and aspartate transaminase) [1, 85, 88, 99, 106]. Renal involvement has been observed, both at the time of diagnosis and during follow-up, in some cases [1, 39, 78]. In particular, proteinuria (increased UPC ratio), increased serum SDMA and azotemia were detected [74, 85, 106, 107].

Differential diagnoses

FeL might be considered "a great imitator" in feline medicine. The differential diagnoses for the main clinical presentations of FeL observed in endemic areas are described in Tables 2 and 3. Comments on useful insights to help in the diagnostic process are included.

Many infectious and non-infectious diseases are considered in the differential, and it should be considered that they may coexist with FeL. The most common coinfections and medical conditions reported in cats with FeL are described under subheading 6.1 (What do we know about coinfections and other medical conditions?).

Diagnosis

What are the purposes for which *L. infantum* infection diagnosis is carried out?

The diagnosis of *L. infantum* infection in cats serves for two main purposes:

1. To confirm the disease in cats with compatible clinical signs and/or clinicopathological abnormalities.
2. To investigate the presence of the infection in apparently healthy cats and in cats with some other medical conditions.

It is important to note that, unlike in dogs, routine screening of clinically healthy cats for early detection of *L. infantum* infection to avoid disease progression is not commonly performed in endemic areas [112]. However, diagnostic investigation may be warranted in some scenarios (Fig. 8).

Cats that are candidates for relocation to non-endemic areas should be tested before leaving their country, irrespective of their health status. It is important to remember that seroconversion to *L. infantum* probably occurs in cats from weeks to months after infection. Cats travelling to endemic areas and those relocated should be tested again by quantitative serology after a reasonable amount of time (4–6 months) has elapsed since leaving the endemic area. Travel and relocation history of cats should always be carefully considered by practitioners at the annual health check-up consultation.

In the case of cats with risk factors for leishmaniosis, quantitative serological tests are recommended. For example, cats positive for antibodies to FIV and/or FeLV antigen, those requiring chronic immunosuppressive therapies (e.g., for FCGS, atopic syndrome, IMHA) and those suffering from malignant neoplasia (Fig. 8).

What are the main diagnostic techniques employed?

The main diagnostic techniques used for the diagnosis of FeL include quantitative serology by IFAT or ELISA carried out in diagnostic and research laboratories, and PCR for the detection of *Leishmania* spp. DNA [113, 114].

Serological techniques should be validated by reference laboratories, but cut-offs and other thresholds of positivity may differ between methods. The interpretation of results should be clearly explained by the laboratories. It is important to remember that very low to low values are mainly suggestive of infection but not necessarily of disease. Based on current evidence, high levels of antibodies are conclusive of a diagnosis of FeL and can be defined as a fourfold (at least) increase of an established IFAT cut-off titer or high levels already defined by other serological techniques, as quantitative ELISA, from a reference

Table 3 Differential diagnoses for the main non-dermatological patterns of feline leishmaniosis (FeL), along with valuable clinical insights

Clinical pattern	Main clinical differentials[a]	Clinical insights
Lymphadenomegaly and/or splenomegaly	Other infectious diseases [FIV, FIP (particularly abdominal lymph nodes), salmonellosis (abdominal lymph nodes), bartonellosis, mycobacteriosis, systemic mycoses, toxoplasmosis], neoplasia (lymphoma, metastatic neoplasia), allergic and immune-mediated diseases, adverse drug reactions (phenobarbital, GS-441524), idiopathic conditions (eosinophilic sclerosing fibroplasia, pyogranulomatous lymphadenitis, hemophagocytic syndrome, plexiform vasculopathy)	Lymphadenomegaly is mild to severe and usually generalized. Lymph nodes are firm, smooth, and non-painful Splenomegaly is mild to moderate and diffuse texture change can be observed at ultrasound examination (honeycomb)
Ocular signs due to acute or chronic uveitis and to their sequelae included panophthalmitis	Other infectious diseases (FIP, FIV, and FeLV infections, toxoplasmosis, bartonellosis, cryptococcosis,), immune-mediated and idiopathic uveitis, reflex uveitis, neoplasia (lymphoma, melanoma)	Bilateral or unilateral red eye, iris bombé, flare, hyphema, anisocoria, dyscoria, synechiae between iris and cornea or the lens, loss of vision
Mucosal nodules	Neoplasia (extra-medullary plasma cell tumor, fibrosarcoma, lymphoma, melanoma, hemangioma), lipogranulomatous conjunctivitis, epitheliotropic mastocytic conjunctivitis, oral eosinophilic granuloma, oral polyps	Solitary or multiple conjunctival, gingival and tongue nodules and plaques
Chronic upper respiratory tract disease	Other infectious diseases (cryptococcosis, bordetellosis and mixed bacterial infections), neoplasia (lymphoma, carcinoma, sarcoma), polyps, lymphoplasmacytic rhinitis	Unilateral or bilateral nasal discharge, stertor, inspiratory dyspnea, facial deformity
Chronic diarrhea	Other infections (protozoal, bacterial, mycotic), immune-mediated diseases (chronic inflammatory enteropathy, triaditis), neoplasia (lymphoma)	Small bowel diarrhea, weight loss
Chronic renal disease [b]	Inherited (polycystic kidney disease), other infections (FIP, FIV, FeMV, *Leptospira*), poisoning (ethylene glycol, lily) neoplasia (lymphoma, carcinoma)	Polyuria-polydipsia and dehydration, dysorexia, weight loss, lethargy, vomiting

FeLV, feline leukemia virus; FeMV, feline morbillivirus; FIP, feline infectious peritonitis; FIV, feline immunodeficiency virus

[a] Differential diagnoses for each non-dermatological pattern in FeL should be carefully considered based on signalment, history, clinical presentation, appropriate clinical pathological and imaging findings

[b] Chronic renal disease may be a comorbidity of FeL as other differentials included in this table (e.g., FIV, FeLV infections)

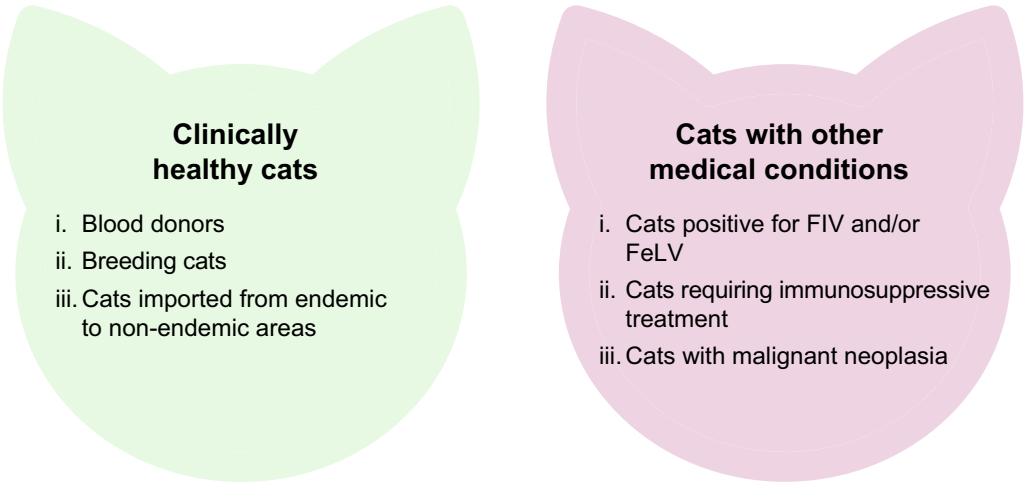


Fig. 8 Clinical situations that require diagnostic investigation for *Leishmania* infection in cats with no signs suggestive of feline leishmaniosis. FeLV, feline leukemia virus; FIV, feline immunodeficiency virus

laboratory. To our best knowledge there are currently no commercial serological tests for feline *Leishmania* infection which have been externally validated and published in a peer-reviewed journal, and rapid tests for dogs are not considered accurate for use in cats [115].

PCR techniques are frequently used on blood, conjunctiva, lymph node, skin and bone marrow aspirates. Blood PCR is considered less sensitive than other sampling locations [52]. Parasitemia in infected cats is typically even lower than in dogs [63, 66], and it is recommended to test lesional tissues when available and several tissues from the same cat by PCR to increase the likelihood of diagnosis [116]. The main PCR techniques used for diagnosis are kinetoplast DNA PCR and the ribosomal internal transcribed spacer 1 (ITS1) PCR [66, 114], with qPCR being widely available and recommended. The combination of multiple diagnostic tools is recommended for the best detection. It is important to identify the species of *Leishmania* in geographical locations where more than one of these species are endemic.

Cytological specimens on stained preparations from lesional tissues or biological fluids can also be very informative about the type of pathological process, such as the type of inflammation compatible with this infection. Moreover, cytology is the fastest method to differentiate infectious agents, such as *Sporothrix* spp. and *Cryptococcus* spp., among others, from *Leishmania* spp. Therefore, amastigotes can be visualized by cytology; however, cytology might often be unrewarding if the parasite load is low [116, 117].

Histopathology of the skin or other affected organs may also be helpful in the detection of the type of inflammatory response in the evaluated tissue and, in some cases, also for the detection of *Leishmania* amastigotes, although more difficult to identify than in cytology. However, specific immunohistochemistry (IHC) is usually necessary to detect *L. infantum* infection in affected tissues [92].

What is the diagnostic approach for a cat suspected of having leishmaniosis?

The clinical diagnostic approach for cats showing clinical signs compatible with leishmaniosis is displayed in Fig. 9. The initial step should be to perform cytological examination of lesional tissues or body fluids. The cytological findings are very informative to find out the type of inflammation as well as to detect *Leishmania* amastigotes. If amastigotes are found, FeL diagnosis is confirmed (direct diagnosis). If the inflammatory pattern is suggestive of FeL but no amastigotes are detected, diagnosis may rely on molecular techniques (e.g., PCR), to confirm or rule out infection in cytological specimens, or on histology with *Leishmania* IHC. In some cases, cytology may yield non-specific findings, prompting consideration of alternative diagnoses. Nevertheless, in any case, CBC, serum biochemistry profile, serum electrophoresis, urinalysis with UPC ratio, and quantitative serology should always be done.

What are the cytological findings that are suggestive of FeL?

The inflammatory infiltrates observed in tissue cytology samples (such as aspirates and impression smears) from organs like the skin, eye, oral and nasal mucosa, liver, spleen and kidney are commonly composed of neutrophils, macrophages or a combination of both

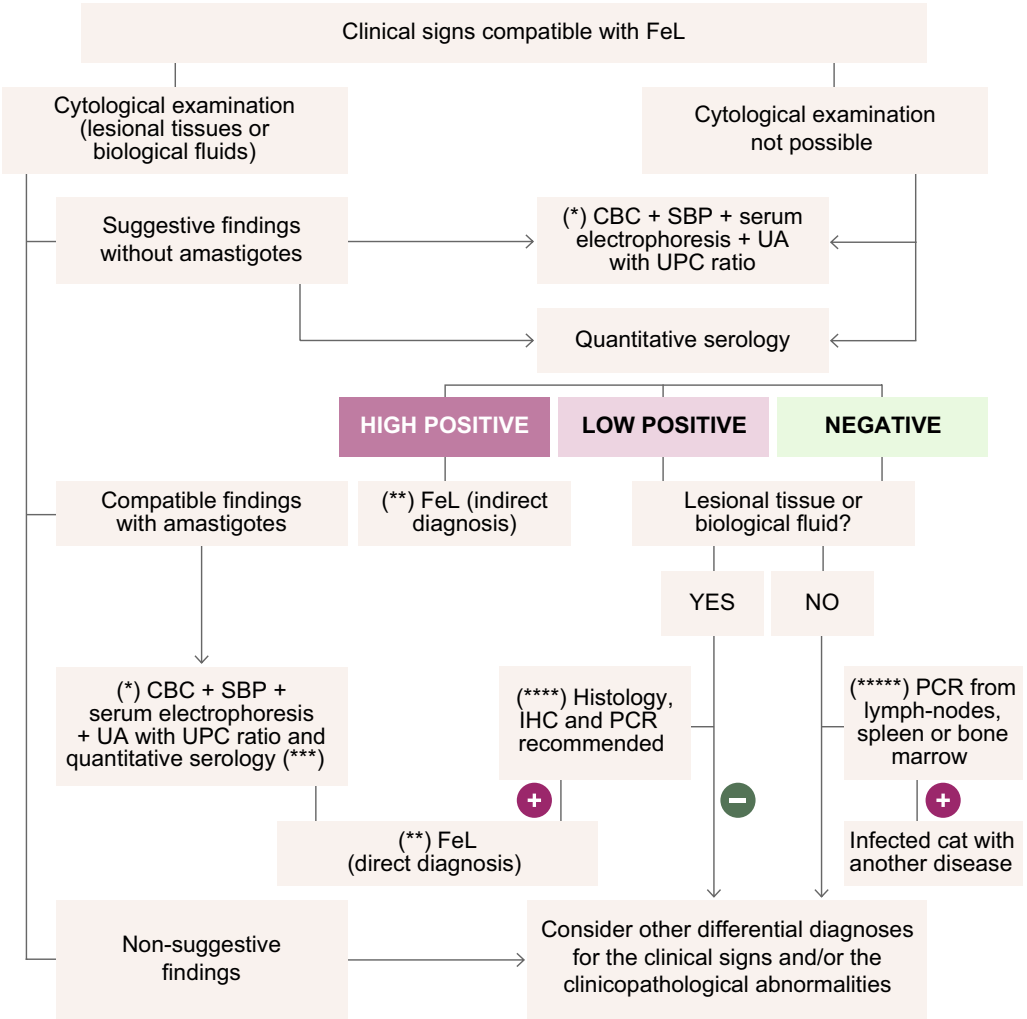


Fig. 9 Flow chart for the diagnostic approach to cats with clinical signs compatible with feline leishmaniosis (FeL). (*) To better check health status and evaluate clinicopathological abnormalities suggestive of FeL. (**) Conventional anti-*Leishmania* treatment and monitoring for resolution of clinical signs and clinicopathological abnormalities. (***) Quantitative serology is recommended for the post-treatment follow-up. (****) A cat will be considered with leishmaniosis if histopathological findings are compatible with leishmaniosis and IHC or PCR are positive. It is not common to visualize amastigotes in histology and, therefore, IHC is always recommended. If IHC is negative, PCR is performed to confirm or rule out infection. (*****) Detection of infection based on clinical suspicion. CBC, complete blood count; IHC, immunohistochemistry; PCR, polymerase chain reaction; SBP, serum biochemical profile; UA, urinalysis; UPC, urinary protein-to-creatinine

(neutrophilic-macrophagic inflammation), but can also include lymphocytes and plasma cells (lympho-plasmacellular inflammation). Macrophages can contain amorphous to fibrillar pinkish cytoplasmic inclusions due to amyloid accumulation [106]. Additionally, lymphoid reactive hyperplasia is frequently seen in lymphoid organs such as lymph nodes and spleen, where variable numbers of *Leishmania* amastigotes may be detected [1].

How can diagnostic imaging aid in diagnosing and monitoring FeL?

Abdominal ultrasonography is a basic investigation tool useful for completing the diagnostic pathway in suspected cases of FeL, particularly when abnormalities are evident on physical examination of the abdomen (e.g., splenomegaly, hepatomegaly, thickened intestine wall, kidney masses). These clinical findings can be produced by other causes that have to be distinguished from FeL and may also be present as a comorbidity with FeL (see Differential diagnoses, Table 3). In the case of the spleen, apart from increased size, a honeycomb appearance has been observed [74, 95, 104]. Ultrasound-guided fine-needle aspirates of spleen, lymph nodes and other organs with focal or diffuse lesions can be obtained for cytological and PCR investigations. In cases of macrophagic and lymphocytic gastroenteritis due to *L. infantum* infection,

abdominal ultrasonography has shown increased intestinal thickness and this finding respectively led to digestive system endoscopy [104] and exploratory laparotomy [118] for taking multiple biopsies in both cases.

In cats with chronic upper respiratory tract disease, advanced diagnostic imaging and nasal endoscopy are required to investigate anatomical abnormalities and to perform guided biopsies of nodular or infiltrative lesions (see Differential diagnoses, Table 3). Computed tomography (CT) of the head was used in two cats with severe dyspnea due to rhinopharyngeal obstructive disease caused by FeL [105, 106]. A nasal cavity with nasopharyngeal invading mass, sinusitis and enlarged regional lymph nodes were found [105, 106]. In one case, the osteolytic destruction of the nasal turbinates, nasal septum and the cribriform plate were very severe, and CT images documented an infiltrative mass in the rostral cranial cavity [105]. CT examination demonstrated impressive improvement obtained by anti-*Leishmania* treatment in this case [105]. Rhinoscopy-guided biopsies of invasive masses and nodules can provide samples for culture, PCR, cytological and histological assessment needed for diagnosing FeL in these atypical cases [74, 105, 106]. In one case, histopathology evidenced a mixed histiocytic and lymphoplasmacytic rhinitis with a high number of plasma cells associated with lakes of eosinophilic congophilic substance suggestive of light chain type amyloid [106].

What is the diagnostic approach for skin lesions in cats with suspected FeL?

A systematic and evidence-based approach to the diagnosis of FeL with dermatological manifestations should begin with an assessment of its likelihood within the differential diagnosis list (Table 2). This prioritization should be guided by key diagnostic features of the clinical manifestations and the known prevalence of FeL in the relevant geographic area.

Cutaneous cytology represents a simple, rapid and minimally invasive first-line diagnostic tool that should be performed on any skin lesion amenable to sampling. The detection of *Leishmania* parasites in skin samples via cytology can be effectively achieved through fine-needle aspiration or puncture of papules, plaques or nodules, direct impression smears of ulcerative areas or skin surface underneath a crust [92] or by delicate scraping the raised edges of ulcers (personal communication: L. Ordeix, P. Bourdeau). When cytological findings are suggestive of FeL but do not reveal amastigotes, and clinicopathological abnormalities suggestive of leishmaniosis are present, *Leishmania* PCR from cytological specimens [119] or histopathological confirmation by skin biopsy plus *Leishmania* IHC and/or qPCR [120] are necessary to definitively diagnose FeL or to explore other differential diagnoses, whatever the results of serology may be.

Dermatopathological analysis of *L. infantum*-associated cutaneous lesions in cats typically reveals an inflammatory infiltrate composed of macrophages, lymphocytes, plasma cells and variable numbers of neutrophils [89, 121–123]. The infiltrate generally follows a nodular (granulomatous) to diffuse pattern in the dermis and/or panniculus [98, 122, 124], although in a few cases a granulomatous perifolliculitis with a high number of lymphocytes and plasma cells and a lichenoid lymphoplasmocytic interface dermatitis at the dermoepidermal junction have been described, like in dogs [89]. Epidermal changes may also include orthokeratotic or parakeratotic hyperkeratosis, particularly in cases of exfoliative dermatosis, along with variable epidermal acanthosis, which may be ulcerated [89, 123]. Interestingly, a case of granulomatous panniculitis clinically characterized by poorly demarcated, painless swelling of the forehead has been described [98]. Macrophages within the infiltrate may contain intracytoplasmic *Leishmania* amastigotes in cats [92], although in cases with less common dermatopathological patterns such as perifolliculitis or lichenoid interface dermatitis, the parasitic load may be low [89]. In such cases, *Leishmania*-specific IHC and/or PCR on skin biopsies can be employed to assess the presence of *Leishmania* in the lesion [92, 120, 125]. Notably, qPCR is considered the most sensitive method for detecting *Leishmania* in the skin of diseased dogs [126]. Nevertheless, a positive qPCR result may also occur in infected cats presenting with unrelated dermatological conditions [89, 123, 127, 128]. Therefore, for the diagnosis of disease, qPCR findings should always be interpreted in the context of the accompanying clinicopathological data.

Should blood donor cats be tested for *L. infantum* infection?

We know that blood transfusion-borne transmission of the infection has been documented in recipient dogs and humans in *L. infantum*-endemic areas [30]. This may occur also in cats. In fact, some epidemiological studies have reported variable percentages of antibody and/or blood PCR positivity in apparently healthy cats from endemic areas [42, 61, 129]. The quality control requirements of some veterinary blood banks operating in endemic areas do not include *L. infantum* among the infectious agents to be tested in feline blood donors and do not exclude donation from shelter and outdoor cats. However, apart from emergencies, LeishVet recommends testing for *L. infantum* in clinically healthy cats being screened for blood donation. We have to consider that the transfused blood will be received by fragile individuals whose immunological status could be impaired,

Table 4 Staging system for confirmed feline leishmaniosis (FeL) in domestic cats (*Felis catus*)

Stage	Clinical signs	Laboratory findings	Quantitative serology[b]	Comorbidities	Prognosis	Therapy[c]
I Mild	Mild (e.g., mild lymphadenomegaly, solitary skin/mucocutaneous lesion, conjunctivitis)	None	Negative-to-low	None	Good	None or short-term treatment with allopurinol (e.g., 3 months). Monitoring required always
II Moderate	Mild-to-moderate (e.g., multifocal skin/mucocutaneous lesions, generalized lymphadenomegaly, lethargy, weight loss, ocular and/or oral disease)	Mild-to moderate non-regenerative anemia, hyperproteinemia with hypergammaglobulinemia, IRIS stages 1 and 2 renal disease[a]	Low-to-high	A. None B. Yes (e.g., FIV, latent FeLV, diseases requiring systemic corticosteroid therapy, malignant neoplasia)	Good to guarded for both sub stages	a. Allopurinol (at least 6 months) b. Allopurinol (6–12 months) + management of concomitant comorbidities Adding meglumine is optional [d] Follow IRIS recommendations
III Severe	The same as in moderate disease but in advanced stage (particularly due to renal disease)	Moderate to severe non-regenerative anemia, pancytopenia, IRIS stage 3 and stage 4 renal disease[a]	Generally high	Usually yes (e.g., end stage FIV infection, dual retroviral infections, immunosuppressive therapies)	Guarded to poor	Management of concomitant comorbidities, follow IRIS recommendations and consider long term allopurinol Adding meglumine is optional [d]

FeL, feline leukemia virus, FIV, feline immunodeficiency virus

[a] Renal disease is a comorbidity that may be related to *Leishmania infantum* infection. International Renal Interest Society (IRIS) stages (https://www.iris-kidney.com/iris-guidelines-1): stage 1: creatinine < 1.6 mg/dL; stage 2: creatinine 1.6–2.8 mg/dL; stage 3: creatinine 2.9–5.0 mg/dL; stage 4: creatinine > 5.0 mg/dL

[b] Quantitative serology (ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody assay); classification of the degree of antibody level should be provided by referral laboratories

[c] Therapy of FeL is scientifically neglected (there is not enough scientific evidence). So, these indications are based on review of the scientific literature and LeishVet's experience

[d] To consider adding meglumine antimoniate if during monitoring there is no response or worsening of the clinical FeL

and this may be a risk factor for developing FeL. Also, the transfused blood, even in a non-fragile animal, may induce a chronic infection status that, years later, may progress to disease.

A good compromise between costs and safety is to perform tests for excluding *L. infantum* infection (quantitative serum antibody detection and blood PCR) after confirming the negative status of candidate donors for other worldwide spread blood-transmitted pathogens [130].

May we formulate prognosis based on a clinical staging system?

Given the limited number of long-term cohort studies on infected cats and the wide range of cofactors that may influence the progression of infection and based on both the available literature and the authors' clinical experience, the LeishVet group proposes a simplified staging system, compared to the one used in CanL [30], to support case evaluation and management. However, it is essential to regard this clinical staging system as a preliminary framework, subject to future revisions and refinements as new, high-quality scientific and clinical evidence becomes available.

Table 4 presents a proposed clinical staging system of FeL, based on three stages, ranging from mild to severe disease. For each stage, key clinical signs, clinicopathological and serological findings, relevant comorbidities, prognostic considerations and therapeutic recommendations are outlined.

Prognosis is related to the clinical stage, with Stage I having a more favourable prognosis, due to the mild/localized clinical signs, the absence of clinicopathological alterations and comorbidities, and negative or low levels of anti-*Leishmania* antibodies, which suggests a good cellular immune response. The worst prognosis is related to advanced phase disease (Stage III), complicated by severe comorbidities (e.g., CKD, severe non-regenerative anemia, pancytopenia) and progressive retroviral coinfections. Stage II clinical cases have a non-predictable (i.e., good to guarded) prognosis because of the presence of various factors that conditionate the evolution of the disease, independently of the treatment. Cats on Stage IIb have a worse prognosis than cats classified as Stage IIa, because of the occurrence of coinfections and comorbidities.

Treatment and monitoring

What are the most used drugs for FeL treatment?

Treatment efficacy clinical trials in FeL are not yet available, with the most important evidence derived from the description of their use for single clinical cases. To date, the treatment of FeL remains empirical as pharmacokinetic studies of anti-*Leishmania* drugs in cats are still lacking. A recent review on FeL [131] examined 28 clinical cases reported in the literature, in 21 of which it was possible to have complete data on the clinical outcome after treatment. These clinical cases had been diagnosed from 2009 to 2023, with extremely variable clinical approach and follow-up that makes comparison difficult. Additionally, one-third were affected by concomitant immune suppressive infectious diseases. Fourteen cats were infected by *L. infantum*, while three hosted cutaneous *Leishmania* spp. (2 cases: *L. mexicana*; 1 case: *L. amazonensis*), the remaining four did not include a confirmative and definitive demonstration of the species of *Leishmania*. This review confirms that AL as a single treatment is the most used drug in the therapy of FeL, followed by the combination of meglumine antimoniate (MA) and AL [1, 77, 88, 95, 107, 131]. The dose and duration of treatment as well as side effects are summarized in Table 5. MA treatment is considered well tolerated in cats at a dosage of 50 mg/kg, but local and systemic side effects have been reported in dogs for this drug, like pain at the injection site, cellulitis, hepatic, cardiac and renal toxicity, weakness, and pancreatitis [67, 132, 133], which should not be excluded in cats because of the limited numbers of feline cases followed up during treatment. A clinical response to a treatment specifically targeted and exclusively active on *Leishmania* can provide information on a possible role of the parasite in clinical cases; however, LeishVet does not recommend "ex juvantibus" diagnosis (i.e., making inferences about *Leishmania* spp. as the cause of suspected clinical FeL based solely on an observed favourable response to treatment).

Miltefosine (MIL), a drug that is used in combination with AL for CanL treatment, was employed in one case of FeL, because of adverse effects due to AL treatment. The authors reported the occurrence of acute kidney injury after MIL treatment course, in a cat that was already affected by CKD [74]. In Brazil, a cat with leishmaniosis treated sequentially with marbofloxacin, MIL and AL showed clinical improvement only after 5 months of AL monotherapy, but remained seropositive (IFAT), cytologically and molecularly positive to *L. infantum*, and also infectious to *L. longipalpis* [134]. Nevertheless, the administration of MIL using the commercial product licensed for CanL (Milteforan®, Virbac Laboratory, Carros, France) caused the formation of Heinz bodies in red blood cells of two out of three healthy cats treated for 14 days in a safety study, because the formulation contains propylene glycol as excipient [135].

Similar to dogs and CanL, FeL treatment has not demonstrated to provide parasitological cure in the treated cats. Sick cats which received FeL treatment showed a good clinical result in roughly 50% of cases, with a

Table 5 Dose, duration of administration and reported side effects of drugs used for treating feline leishmaniosis (FeL) based on clinical cases reported in the literature

Drugs	Dose and duration	Adverse effects	References
Allopurinol[a]	10 mg/kg, PO, q12h[b] Duration not standardized[c]	AKI Increased liver enzymes Pruritus Head and neck dermatitis	[84, 95] [123] [75] [74]
Meglumine antimoniate[a]	50 mg/kg, SC, q12h or q24h, for 4 weeks	AKI	[74]

To date, the treatment of FeL remains empirical regarding drugs, dosage and duration

AKI, acute kidney injury; BID, twice a day; PO, *per os*; q12h, every 12 h; q24h, every 24 h; SC, subcutaneous; SID, once a day

[a] The two drugs should be used in combination. A fatal case of AKI has been reported in a cat treated with both drugs [88]

[b] Allopurinol has also been administered SID or BID with lower or higher dosages

[c] Usually at least 6–12 months and until clinical cure

decrease in parasitic load. Relapses and recurrences are always possible, as well as the possible need for euthanasia practiced in about 30% of cases in one study [131].

Should healthy infected cats be treated with anti-*Leishmania* drugs?

In principle, as in dogs, clinically healthy infected cats demonstrate an effective immune response and therefore leishmanicidal treatments should be limited for use in sick cats. However, these healthy infected cats should be monitored (Fig. 10). There is no evidence-based data on the recommended frequency of monitoring the course of *L. infantum* infection derived from longitudinal studies. Nevertheless, we recommend to follow up antibody positive and/or PCR positive cats with no compatible signs of FeL, with the aim of early detection of disease progression, by performing clinical and serological monitoring with variable frequency, based on clinical criteria and occurrence of risk factors. In case of healthy cats infected by *L. infantum*, monitoring will be included in the annual health check. In case of comorbidities (e.g., neoplasia, FeLV) and/or risk factors (e.g., FIV, administration of intense or prolonged immunosuppressive therapy) it is advisable to perform

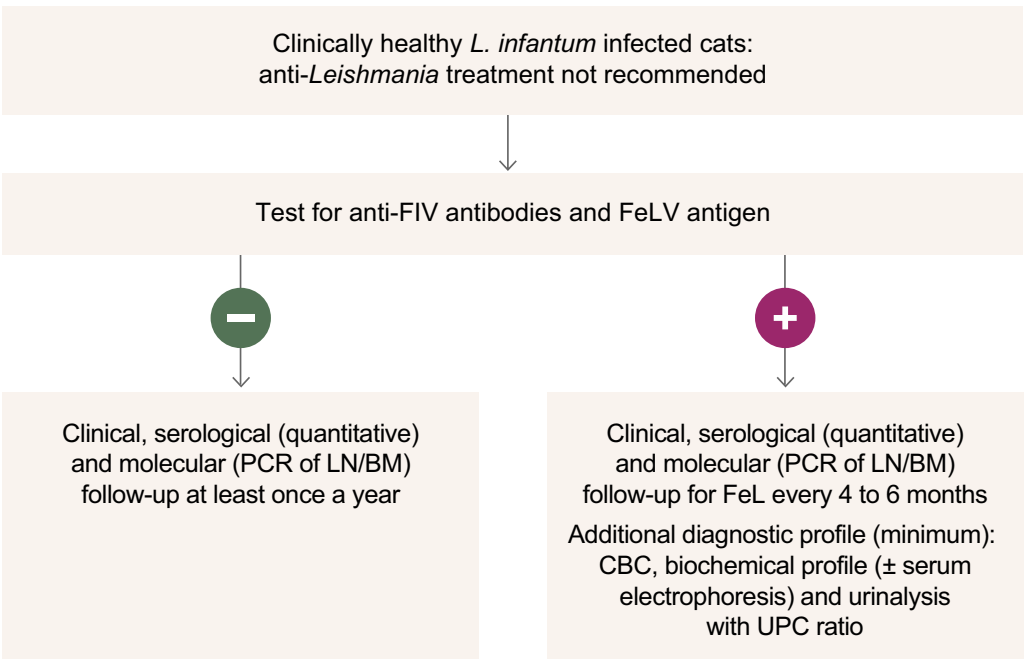


Fig. 10 Management of cats with serological and/or PCR-positive positivity for *Leishmania infantum* and no clinical signs and clinicopathological abnormalities compatible with feline leishmaniosis (FeL; clinically healthy infected cats). BM, bone marrow; CBC, complete blood count; FeLV, feline leukemia virus; FIV, feline immunodeficiency virus; LN, lymph node; PCR, polymerase chain reaction; UPC, urinary protein-to-creatinine

monitoring every 4 to 6 months (i.e., 2–3 times/year), depending on the patient's clinical status. A fourfold increase (i.e. two titres) of an established IFAT cut-off titer (or high levels already defined by other serological techniques, as quantitative ELISA) should raise suspicion of transitioning from a healthy infected to a sick state, and a thorough evaluation of clinical signs and clinicopathological alterations should be carried out.

Studies of cat treatment with immunomodulatory active substances that can help to prevent disease progression in healthy infected animals are lacking and future studies are needed.

How to monitor cats under treatment?

As in dogs, it is necessary to monitor sick cats under treatment by running a CBC, serum biochemical profile, serum protein electrophoresis, and urinalysis with UPC ratio in combination with quantitative serology and PCR as well as any supplementary diagnostic procedure required (e.g., imaging examination). The frequency of clinical check-ups is higher initially, aiming at early recognition of drug adverse events and lack of treatment efficacy, and depends on clinical staging after diagnosis. Later on, it is continued as required by the cat's clinical status (Fig. 11).

Which preventive measures are available for FeL and do we need to apply them to all cats living in endemic areas?

Prevention of FeL relies primarily on minimizing exposure to sand flies, as there are currently no drugs or vaccines specifically approved for this purpose in cats. Preventive measures should therefore be recommended for cats living in endemic areas, particularly for those with outdoor access, to reduce the risk of *L. infantum* transmission. Vector control through the application of topical insecticides with repellent or antifeeding activity represents the most effective strategy. A collar containing a combination of 10% imidacloprid and 4.5% flumethrin has been evaluated successfully in dogs [136–138] and cats [139], significantly decreasing the likelihood of *L. infantum* infection in cats under field conditions, and

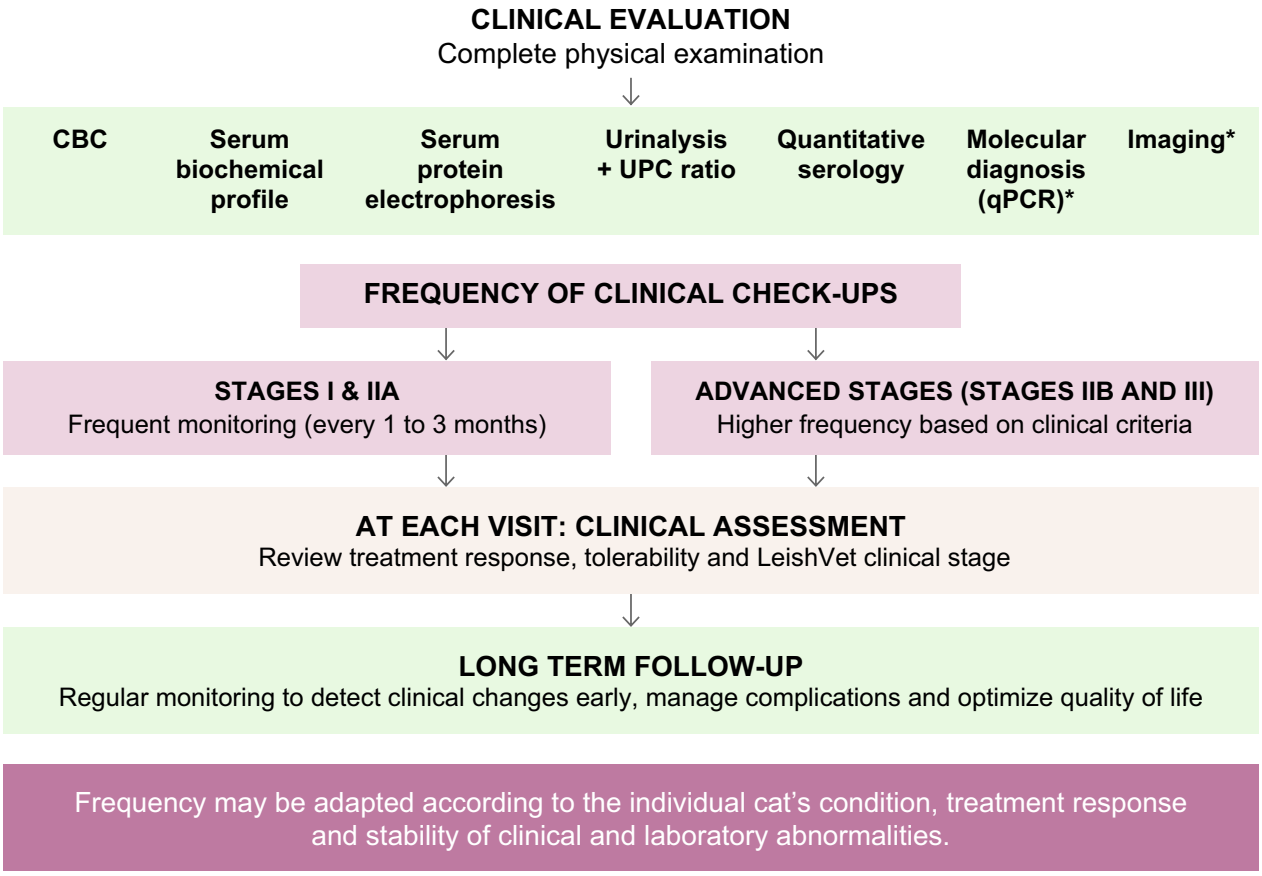


Fig. 11 Flowchart for the monitoring of cats undergoing treatment for leishmaniosis. * To perform as clinically required. CBC, complete blood count; qPCR, quantitative real-time polymerase chain reaction; UPC, urinary protein-to-creatinine

it is currently considered the only prophylactic tool supported by evidence for this purpose in this species; however, this flea and tick collar is not specifically registered for the prevention of FeL (off-label use). The efficacy of repellent products is time-dependent and the recommended intervals of application should be respected when evaluating their effectiveness. Apart from the flumethrin collar, pyrethroid-based formulations are generally toxic to cats and products commonly used in dogs must be strictly avoided, as well as close contact with dogs treated with pyrethroid spot-ons during the first 24 h after application [140]. Additional preventive measures aimed at reducing sand fly exposure are recommended in endemic areas, including keeping cats indoors or restricting outdoor activity during peak vector activity (from dusk to dawn) and installing fine-mesh window screens to limit vector entry [141]. Maintaining good general health through adequate nutrition and regular veterinary care, including appropriate endo- and ectoparasite control, may support immune competence and potentially reduce the risk of infection or disease progression. Prevention of retroviral infections such as FIV and FeLV is also important, as these infections may increase susceptibility to progressive *L. infantum* infection and possibly the severity of clinical leishmaniosis [1, 47, 57, 80, 142].

What diseases are caused in cats by *Leishmania* spp. other than *L. infantum*?

Feline leishmanial disease caused by species other than *L. infantum* is shown in Table 6. Figures 1 and 2 show the geographical distribution of these species, and Additional files 1 and 2: Table S1 and S2 show studies where these species were detected in cats. Moreover, some studies in the Old World detected *L. major* [143–146] and *L. tropica* [143, 145, 147, 148] infections in domestic cats. In most of these studies, no clinical data from infected cats were provided, and the causative role of these species in clinical signs reported was not investigated.

In Central and South America, leishmaniosis caused in cats by *Leishmania* spp. other than *L. infantum* is mostly a dermatological disease. It is caused by species included in the subgenus *Leishmania* represented by the *L. mexicana* complex (*L. mexicana, L. amazonensis*) and in the subgenus *Viannia* represented by the *L. braziliensis* complex (*L. braziliensis* and *L. guyanensis*) [120]. Due to the limited number of published cases, detailed clinical descriptions of cats infected by these *Leishmania* spp. is scarce. The majority of cases were caused by *L. mexicana* [120, 149–153]. The rarity of diagnosis and detailed descriptions in cats means that comprehensive clinical data are not available, and our current knowledge of the disease presentation is based on isolated case reports or few case series [120, 149–160]. The cutaneous lesions observed (mainly nodular-ulcerative dermatitis), diagnostic techniques used and drugs administered are reported in Table 6. Interestingly, no cats tested for FIV and FeLV were found positive. Some cats were followed up, and the clinical course was variable with frequent clinical recurrences and new additional lesions occurring [149–152, 157, 159]. Spontaneous cyclic progression of lesions caused by *L. mexicana* was described as follows: a small erythematous papule evolves to a brownish edematous nodule resembling a "clown nose" that ulcerates and then may temporarily heal with crusted scaly caps [151]. Life expectancy was, however, good, even in cats where treatment failed or was not given [149, 151, 152]. Excisional biopsy and cryosurgery resulted in curative outcomes in some followed up cats [150, 153, 160]. Severe neutropenia was described as an adverse drug reaction to AL treatment (15 mg/kg, q24h) in a single cat [152]. Recurrence of signs was partly controlled by the oral administration of *Artemisia annua* (wormwood) extract in this cat [152].

Currently, no studies are available about the preventive efficacy of a collar containing flumethrin and imidacloprid against *Leishmania* spp. other than *L. infantum* at individual and population level in cats.

What is the importance of other felid species in the epidemiology of FeL?

Several wild felid species, including the European wildcat, Iberian lynx, lion, ocelot, jaguar, tiger, serval and the sand cat, have been reported as infected or seropositive for *Leishmania* spp. and could therefore be considered potential hosts or reservoirs [7, 161, 162]. The first reports in this context include detection of *Leishmania* DNA by qPCR in tissues (liver, spleen and skin) of European wildcats (*Felis silvestris silvestris*) from northern Spain [163]; and subsequent studies have confirmed additional positive wildcats from the same areas [164, 165]. The published records of Iberian lynx (*Lynx pardinus*) are from Sobrino et al. [166], who found PCR-positive spleen and blood samples in one of four sampled lynxes from Spain. Lima et al. [167] detected *L. infantum* infection by molecular ($n = 4$) and serological ($n = 11$; ELISA and IFAT) diagnosis in a sample of 41 lynxes in southern Portugal. Moreover, a large-scale epidemiological study run in the Iberian Peninsula by Barbero-Moyano et al. [168] evaluated the circulation of *L. infantum* in free-ranging ($n = 487$) and captive ($n = 296$) Iberian lynx populations, detecting a prevalence of 10% by quantitative serology

Table 6 Description of skin lesions, other clinicopathological findings, diagnostic methods used and treatment in cats diagnosed with leishmaniosis caused by *Leishmania* spp. other than *Leishmania infantum* in the Western Hemisphere

	Cutaneous lesions (number of cats)	Other clinicopathological findings	Diagnostic methods employed	Treatment	Reference
L. amazonensis	a. Alopecic nodule on the nose and many nodules on the ears and digital regions ($n = 1$))	a. None	a. Nodule cytology and isolation	a. None	a. [155]
	b. Multiple alopecic nodules on the ears ($n = 1$)	b. None	b. Nodule cytology and isolation	b. Azithromycin and Allopurinol	b. [158]
	c. Swollen alopecic nose and multiple nodules on the ear ($n = 1$)	c. Hyperproteinemia, hyperglobulinemia	c. Nodule cytology, histology, PCR and antibody detection (IFAT)	c. Allopurinol	c. [159]
L. braziliensis	a. Interdigital vegetative lesion ($n = 1$)	a. None	a. Lesional cytology and PCR	a. None	a. [175]
	b. Swollen alopecic nose and facial ulcerative dermatitis (n=1t); swollen alopecic nose and vegetation inside a nostril ($n = 1$)	b. None	b. Lesional cytology (one cat), isolation	b. None	b. [154]
	c. Nasal ulcer and nodules in the ears ($n = 1$)	c. None	c. Nodule swab PCR	c. None	c. [156]
	d. Ulcerated nodule at medial canthus ($n = 1$)	d. None	d. Lesional PCR	d. None	d. [176]
L. guyanensis	Small papules (2 mm) on the nasal planum ($n = 1$)	None	Histopathology, IHC and PCR from papular sample	Surgical removal of papules	[160]
L. mexicana	a. Multiple alopecic nodules on the left ear ($n = 1$)	a. None	a. Cytology and isoenzyme characterization	a. Pinnectomy	a. [149]
	b. One or multiple nodules, variably ulcerated on the pinnae and less commonly on the muzzle and periorbital skin ($n = 8$)	b. Non-reported	b. Cytology (1/1), histopathology, (5/8) PCR (5/8)	b. Surgical removal (3/8); Allopurinol (1/8)	b. [150]
	c. Ulcerated nodule on the inner surface of the marginal skin pouch of the right pinna ($n = 1$)	c. None	c. Cytology, histopathology, PCR	c. Pinnectomy	c. [157]
	d. Solitary or multiple nodules on the nose ($n = 3$), ears ($n = 1$), ears and nose ($n = 1$), ears, nose, tail, and lower limbs ($n = 1$)	d. None	d. Cytology (4/4), histopathology (4/5), IHC (5/5); ELISA (2/6); WB (4/5); lesional PCR (2/5)	d. No	d. [120]
	e. Globous, alopecic, erythematous nodules in the nasal planum and the nares (clown nose) variably ulcerated ($n = 12$); similar lesion coexisting on ear and tail (1/12) and ear and extremities (1/12)	e. Weight loss (5/12)	e. Cytology, PCR	e. Meglumine antimoniate (4/4) in combination with allopurinol (1/4)	e. [151]
	f. Erythematous, thickened, crusted margin of right pinna and ulcerated exophytic nodule at caudal right tarsus ($n = 1$)	f. Moderate leukopenia, mild thrombocytopenia and hyperglobulinemia	f. Cytology, PCR (tarsal lesion)	f. Marbofloxacin, imiquimod (topical), artemisinin partly in association	f. [152]
	g. Erythematous nodules on the nasal planum (one cat)	g. None	g. Lesional cytology and histopathology	g. Cryosurgery	g. [153]

ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; IHC, immunohistochemistry; PCR, polymerase chain reaction; WB, western blot

(IFAT) and/or qPCR. Cases in captive large felids have also been documented. Libert et al. [169] reported leishmaniosis in a breeding group of Barbary lions (*Panthera leo leo*) at Montpellier Zoological Park, where one adult animal showed compatible clinical signs (epistaxis, colitis, lameness, paw ulcers) and was PCR-positive in blood. It improved after a short course of allopurinol combined with marbofloxacin, while another group member was clinically healthy but seropositive (IFAT) and PCR-positive. Iatta et al. [170] reported that among 20 tigers (*Panthera tigris*) tested from a wildlife park in Italy, nine were serologically positive (IFAT) and five PCR-positive; infected animals showed dysproteinemia with hypergammaglobulinemia. In Brazil, PCR–RFLP on lymph node and/or blood samples detected *Leishmania* infection in five pumas (*Puma concolor*), one jaguar (*Panthera onca*) and one lion (*Panthera leo*) at the zoological park of the Federal University of Mato Grosso [171, 172]. Also in Brazil, at the Belo Horizonte Zoological Garden, two ocelots (*Leopardus pardalis*), one Siberian tiger (*Panthera tigris altaica*), one jaguar and one lion were found *Leishmania*-positive in serum samples by a commercial ELISA (Safetest® Diagnostics), an in-house ELISA and/or three commercial rapid tests, i.e. DPP®, rKDDR® immunochromatographic kit and SNAP Leishmania IDEXX® [173]. Nevertheless, these serological tests have not been validated for use in large felines. Finally, it is worth mentioning that earlier reports described infection with *Leishmania donovani* in sand cats (*Felis margarita*) from Saudi Arabia [174], and also in servals (*Leptailurus serval*) [161].

Despite these reports, the epidemiological role of wild felids in *Leishmania* transmission remains unclear. Most infected individuals appear to be subclinical, and parasite loads reported in felids are often lower and more variable than those typically seen in canid reservoir hosts. While domestic cats have been shown to infect sand fly vectors under xenodiagnosis, there is no information on the capacity of transmission to vectors by wild felids (and hence contributing substantially to wild or zoonotic cycles). Therefore, wild felids may act mainly as spill over or incidental hosts in many settings. Still, in particular ecological contexts (e.g., where sylvatic cycles are intense), they could contribute to maintaining or bridging *Leishmania* transmission.

Conclusions

These update and current recommendations on FeL are intended to provide useful information for feline practitioners on risk factors, clinical signs, diagnostic protocols, treatment options, prognosis and prevention measures in cats. In areas of endemicity, the prevalence of *L. infantum* infection in cats is generally lower than in dogs, and leishmaniosis as a disease is also generally less frequent in cats than in dogs. Nevertheless, similar to its canine counterpart, FeL appears to be a highly variable disease, with affected cats potentially showing regional or systemic clinical manifestations. Veterinary awareness has increased, and diagnostic support is more widely available, aimed at confirming the disease in clinically suspected cats or detecting infection in apparently healthy animals. Coinfections or other comorbidities are present in the majority of clinical FeL cases and are a major participant in the pathogenesis and outcome of infection. The LeishVet group proposes a simplified preliminary staging system, compared to the one used in CanL, to support case evaluation and management. The treatment of FeL remains empirical as pharmacokinetic studies of anti-*Leishmania* drugs in cats are still lacking. Studies of feline treatment with immunomodulatory substances that can help to prevent disease progression in healthy infected animals are lacking and future studies are needed. The role of cats should be considered in contributing to the *Leishmania* reservoir for other hosts, including humans. Prevention of sand fly bites by the application of topical insecticides containing molecules with repellent or antifeeding effects is an effective strategy to reduce the risk of *Leishmania* transmission to and from cats.

Abbreviations

AL	Allopurinol
CanL	Canine leishmaniosis
CBC	Complete blood count
CKD	Chronic kidney disease
CT	Computed tomography
DAT	Direct agglutination test
DPP	Dual-path platform
ELISA	Enzyme-linked immunosorbent assay
FCGS	Feline chronic gingivostomatitis
FeL	Feline leishmaniosis
FeLV	Feline leukaemia virus
FIV	Feline immunodeficiency virus
IMHA	Immune-mediated hemolytic anemia
IFAT	Indirect fluorescent antibody test
IFN-γ	Interferon-gamma
IHC	Immunohistochemistry
IRIS	International Renal Interest Society
MA	Meglumine antimoniate
MIL	Miltefosine
PCR	Polymerase chain reaction
qPCR	Quantitative real-time PCR
RFLP	Restriction fragment length polymorphism
SDMA	Symmetric dimethylarginine
Th	T helper
UPC	Urinary protein-to-creatinine
WOAH	World Organization for Animal Health

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13071-026-07642-4.

Additional file 1: Table S1. Prevalence of *Leishmania* spp. infection in cats (*Felis catus*) in Old World countries (data published from 2015 to 2025), using serological and/or molecular techniques. Data related to the period of sampling, the characteristics of studied cats and variables associated with positivity are included. Except when otherwise stated, the involved species was confirmed or presumed to be *Leishmania infantum*

Additional file 2: Table S2. Prevalence of *Leishmania* spp. infection in cats (*Felis catus*) in the New World (data published from 2015 to 2025), using serological and/or molecular techniques. Data related to the period of sampling, the characteristics of studied cats and variables associated with positivity are included. Except when otherwise stated, the involved species was confirmed or presumed to be *Leishmania infantum*

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Availability of data and materials
Data supporting the main conclusions of this study are included in the manuscript.

Declarations

Ethics approval and consent to participate
Not applicable.

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Competing interests
The authors declare no competing interests.

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