Leishmaniosis (Leishmania infantum infection) in dogs

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Summary
The authors present an overview of canine leishmaniosis due to Leishmania infantum. This protozoan is transmitted by sandflies and the disease is frequently characterised by chronic evolution. Cutaneous and visceral clinical signs appear as the infection progresses. Lymph node enlargement, emaciation and skin lesions are the main signs observed in the classical forms of the disease. Control is difficult since infected dogs remain carriers for years and may relapse at any time. The mass screening of infected animals and their treatment or euthanasia represent the best way to reduce the prevalence of this disease in endemic regions. Further research is needed to improve the efficiency of the vaccines available to protect dogs against infection. This disease is zoonotic; in humans, clinical cases are reported mainly in elderly people, the young and those whose immune systems have been compromised.

Keywords

Introduction
Canine visceral leishmaniosis (CvL) is a complex of infectious, vector-borne, zoonotic diseases caused by the presence and multiplication of a protozoan belonging to the genus Leishmania (Kinetoplastida: Trypanosomatidae). These infections are due to several species with varying virulence, they have different epidemiological and clinical patterns, and some are zoonotic. The most important cycle of Leishmania infantum takes place between dogs and sandflies but other canid species (both domestic and wild) are involved. While acknowledging the increasing number of emerging zoonotic diseases, the authors would like to point out that we must not forget the importance of the ‘classical’ diseases, such as leishmaniosis, rabies, tuberculosis and brucellosis. The implementation of an effective CvL control programme requires a good knowledge of L. infantum, sandflies, the host and the regional epidemiological pattern of this disease. Indeed, the interactions of the parasite, host and vector lead to a specific epidemiological pattern. The principal aim of this paper is to review the information gathered on CvL caused by L. infantum.

Sandflies

Taxonomy of sandflies
The current classification of sandflies remains controversial and cumbersome. Lewis et al. (1977) proposed two genera of Old World species (Phlebotomus spp. and Sergentomyia spp.) and three genera of New World species (Lutzomyia spp., Brumptomyia spp. and Warileya spp.) (1). The genus Phlebotomus consists of 11 subgenera, 96 species and 17 subspecies (2). Galati (2003) recognised 464 species of Neotropical sandflies grouped into 23 genera, 20 subgenera and three groups (3, 4).

Geographic distribution of sandflies
Sandflies are principally present in the warm zones of Asia, Africa, Australia, southern Europe and the Americas (5). Their distribution extends northwards to just above a latitude of 50°N in south-west Canada (6) and just below this latitude in northern France and Mongolia (2). Their southernmost distribution ends at a latitude of 40°S, but they are absent from New Zealand and the Pacific Islands (the New Zealand ‘sandflies’ are actually black flies of the
Austrosimulium species) (7). Their altitudinal distribution extends from below sea level to 3,300 m above sea level in Afghanistan (8).

**Biology of sandflies**

Sandflies are holometabolous (they undergo complete metamorphosis through four developmental stages): the egg, four larval instars, the pupa, and imagos. The duration of the life cycle is variable; it depends on the species concerned and abiotic factors, such as temperature, rainfall and hygrometry. Imagos are small (1.5–4 mm) and not noisy when they fly. The seasonal activity of sandflies is mainly determined by temperature and rainfall. Females take blood meals during the early morning, evening and night but they can also bite during the day if they are disturbed. After a blood meal, the female lays 30 to 70 eggs, spreading them around a suitable breeding site. When inactive, adult sandflies have species-specific resting sites. These are often similar or near to the larval sites, which are cool, humid and dark environments. Adult sandflies can survive in dry environments by moving to cool, humid resting sites during the day and becoming active at night, when the temperature drops and humidity increases. Eggs hatch within one to two weeks. The larvae feed on organic materials. They are found in leaf litter and in damp environments containing organic substances, such as caves, animal burrows and shelters, and cracks in walls and rocks.

If the temperature falls, the fourth larva instar diapauses. The pupal stage of development takes between five and ten days.

**Leishmania infantum**

**Taxonomy of Leishmania**

The genus *Leishmania* Ross, 1903, includes several obligate protozoan parasites of humans and domestic and wild mammals. Based on their location in the vector's intestine, Lainson and Shaw (1987) distinguished two *Leishmania* subgenera, *Leishmania* and *Viannia* (9). Since the 1970s, immunological, biochemical and genetic criteria have been used to define *Leishmania* species (Fig. 1) (10).

**Life cycle of Leishmania infantum**

*Leishmania* is transmitted during the blood meal of infected female sandflies. The infectious *Leishmania* stage (promastigotes) is inoculated from the vector's proboscis into the host's skin. The promastigotes are phagocytised by macrophages and other mononuclear phagocytic cells, where they transform into amastigotes, which multiply by bipartition and infect other mononuclear phagocytic cells. The relationship between the parasite and the host determines the outcome of the infection. *Leishmania infantum*
may invade all the host’s organs. However, its presence in the host’s dermis is necessary for the transmission of infection to the vector. In sandflies, amastigotes transform into promastigotes, develop in the hindgut (*Viannia* subgenus parasites) or in the midgut (*Leishmania* subgenus parasites), and turn back to the proboscis (Fig. 2).

**Epidemiology of canine visceral leishmaniosis**

The epidemiology of CvL is diverse; this is the result of intricate interactions between the vector (biomass, species, vector capacity, behaviour, etc.), the host (biomass, species, age structure, breed, breeding type, control measures), the parasite (biomass, zymodem) and ecological factors (such as temperature, hygrometry, the ecological niches of the vector, mainly the larvae, etc.). The epidemiological patterns of *L. infantum* infection are dynamic, with a clear trend towards geographical extension, an increase of listed host and vector species, and the description of new zymodems (11). The life style of the dog is very important, since dogs that live outdoors or have no insecticide protection (mainly collars) are more exposed. The geographic distribution of CvL corresponds to the distribution of its vectors. This disease is present in 50 countries in Europe, Africa, Asia and the Americas (12), including new endemic areas where outbreaks are occasionally reported, such as the United States (13), Canada (14) and Europe (Fig. 3) (15, 16). In the United States, CvL is considered an emerging disease in dogs (17). Within countries with historical foci of leishmaniosis, the disease seems to be spreading geographically; for example, as reported in Tunisia (18, 19). The number of infected dogs is much higher than the number of clinical cases. This aspect drives the epidemiology of this infection since non-clinically infected dogs are the main reservoirs of the parasites. The main epidemiological cycle of canine leishmaniosis is domestic; it is concentrated in suburban regions, while the sylvatic cycle is cryptic (Fig. 4). Cats can express clinical signs of visceral leishmaniosis (20) but their role as reservoirs is still unknown and needs to be explored (21).

**Clinical signs of canine visceral leishmaniosis**

Canine leishmaniosis is generally a chronic and multisystemic disease. As a result of the complexity of the immunopathogenic interactions, the diversity of the affected tissues and organs, and the strain of the protozoan, clinical signs are very polymorphic and, in many cases, can lead to misdiagnosis (22, 23, 24).

**Chronic forms of canine visceral leishmaniosis**

**General signs**

The signs of canine leishmaniosis begin with an inoculation chancre. This is a unique sweating ulcerative lesion, which regresses after a variable number of days, giving way to a ‘silent’ period (Fig. 5). The clinical expression of infection begins with slight but progressive clinical signs which gain in severity: dullness and increasing reluctance to exercise, poor body condition, hyporexia, weight loss and localised (head) then generalised amyotrophy. However, the early stages of the disease are not associated with clear general signs.
Mononuclear phagocyte system signs

The most frequent sign in canine leishmaniosis is enlargement of the lymph nodes, which become mobile and painless (22). This enlargement occurs early on during the course of the disease and, in some cases, may be the only clinical sign. Splenomegaly can be detected but it is inconsistent (25).

Cutaneous signs

Dermatological signs are the second-most-common manifestation of leishmaniosis (23, 25, 26, 27, 28). They may be the only detectable signs or they may be associated with other types of lesions (29). Dermatological signs vary in type and intensity but are not associated with pruritus.

– Non-itchy diffused alopecia may be associated with dry seborrhoea with silver-white scales, which can be localised (mainly on the dorso-lumbar region) or disseminated throughout the entire body (Fig. 6) (30). The dog presents with follicular keratosis and, in some cases, parakeratosis with an inflammatory infiltrate, sometimes associated with histiocytes, plasmocytes and macrophages. The *Leishmania* amastigotes are sometimes present.

– Hyperkeratosis may appear on the head, the nose and footpads (Fig. 7).

– Multifocal cutaneous or mucosal ulcers may be associated with necrosis of the epidermal and dermal layers (Fig. 8). There is a polymorphic infiltrate around the necrosis, consisting of a mixture of histiocytes, lymphocytes, plasmocytes and macrophages infected by *Leishmania* amastigotes.

– Frequently, superficial and/or deep bacterial (mainly *Staphylococcus* spp.) or fungal (*Malassezia pachydermatis*) infections are reported, which induce pustular dermatitis characterised by the presence of an inflammatory histio-lymphoplasmocytic infiltrate.
An increase in the length of the claws may become apparent (Fig. 9).

Nodules may occur. These can take several forms, from simple papules to tumour-like nodules with peripheral lymphadenopathy. The nodules can be generalised or localised to the interdigital space. In some cases, nodules may become ulcerated (30). Nodular dermatitis with an inflammatory infiltrate, containing macrophages with high parasite burdens and some lymphocytes and plasmocytes, has been reported. The nodular form is more frequently seen in boxer breeds (26, 31).

Atypical and rare cutaneous lesions may occur: with depigmentation (mainly nasal), panniculitis and nasal hyperkeratosis. These lesions may also be found on the footpads.

**Ocular signs**

Ocular signs are polymorphic and include: diffuse or nodular conjunctivitis; exfoliative, ulcerative or nodular blepharitis; nodular or diffuse scleritis; anterior or posterior uveitis, which can be granulomatous or diffuse; glaucoma; panophthalmitis and keratoconjunctivitis (Fig. 10) (25, 32, 33).

**Non-specific forms**

Several other atypical forms are reported with CvL, such as sterile pustular dermatitis due to suppurated folliculitis; lameness due to polyarthritis, resulting from type III hypersensitivity (23); and, more rarely, granulomatous myositis (30).

**Diagnosis of canine leishmaniosis**

In many cases, it is difficult to diagnose CvL, due to its polymorphic expressions and to the presence of banal...
clinical signs, which are frequently neglected by the dog’s owner. In the classic, cutaneous-visceral form associated with general signs, however, diagnosis is relatively easy. To improve the prognosis and to avoid both human and animal transmission (from false-negative cases) and unnecessary euthanasia (from false-positive cases), the diagnosis should be established as soon as possible, even on the basis of only a few or even a single clinical sign. A summary of the advantages and limitations of the diagnostic techniques available can be found in Table I.

At the clinic

The first step is to carry out a detailed clinical examination to look for signs that could be attributed (or not) to leishmaniosis. A biopsy of the lymph nodes is the first diagnostic technique to implement, especially if enlargement is detected. The biopsy product is smeared on a microscope slide, then Giemsa-stained and examined under an optic microscope at x1,000 magnification to verify the presence of amastigotes. The parasites are rod-shaped, contain a nucleus and kinetoplast (these two structures are a dark red-purple in colour), and can be present in both intracellular and extracellular compartments, since the cell host is frequently ruptured during smear preparation. Alternatively, other tissues could be biopsied: spleen, bone marrow and skin, but these techniques are laborious. Since the sensitivity of this technique is low (50–60%), negative clinical cases should be further investigated using other techniques.

Table I
Advantages and limitations of diagnostic techniques used for canine leishmaniosis

<table>
<thead>
<tr>
<th>Direct techniques</th>
<th>Detection</th>
<th>Advantages</th>
<th>Limits</th>
<th>Implementation</th>
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<tbody>
<tr>
<td>Lymph node biopsy</td>
<td>Amastigotes</td>
<td>Rapid</td>
<td>Low sensitivity</td>
<td>First technique to implement</td>
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<td>Low cost</td>
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<td></td>
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<td>Excellent specificity</td>
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<td>Culture in Novy-McNeal-Nicolle</td>
<td>Amastigotes</td>
<td>Excellent specificity</td>
<td>Takes time (several days)</td>
<td>Research</td>
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<td>medium</td>
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<td>Quantitative technique</td>
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<td>Yields a large quantity of parasites that may be used for further study</td>
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<tr>
<td>Polymerase chain reaction (PCR)</td>
<td>Nucleic acids</td>
<td>Excellent specificity</td>
<td>Expensive</td>
<td>Research</td>
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<td>Excellent sensitivity</td>
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<td></td>
<td></td>
<td>Not subjective</td>
<td>Does not differentiate between carrier and diseased state</td>
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<td>Requires laboratory equipment</td>
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<td>Dual path platform (lateral flow device)</td>
<td>Antigens</td>
<td>Rapid</td>
<td>Qualitative result</td>
<td>For orientation</td>
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<td>Easy</td>
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<td></td>
<td></td>
<td>Low cost</td>
<td>Does not differentiate between carrier and diseased state</td>
<td>More suitable in countries which have not previously reported the presence of infection</td>
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<tr>
<td>Loop-mediated isothermal amplification PCR</td>
<td>Nucleic acids</td>
<td>Relatively rapid</td>
<td>Does not differentiate between carrier and diseased state</td>
<td>Research</td>
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<td></td>
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<td>Excellent sensitivity</td>
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<td>Excellent specificity</td>
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<td>Does not require a thermocycler</td>
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<td>Mass screening</td>
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<td>Lateral flow devices</td>
<td>Antibody</td>
<td>Low cost, rapid and easy to perform</td>
<td>Low sensitivity and specificity</td>
<td>Individual/mass screening</td>
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<td>Immunofluorescent antibody test</td>
<td>Somatic antibody</td>
<td>Low cost</td>
<td>Sometimes not objective</td>
<td>Mass screening</td>
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<td></td>
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<td>Simple</td>
<td>Takes time to perform</td>
<td>Clinical diagnosis</td>
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<td>Enzyme-linked immunosorbent assay</td>
<td>Soluble antibody</td>
<td>Objective</td>
<td>Available in kit form</td>
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<td>Relatively low cost</td>
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<td>Quantitative technique</td>
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In the laboratory

Gomes et al. (34) published an excellent review of all the laboratory tests; the tests’ limitation is that they do not easily differentiate between the carrier state and diseased dogs. Moreover, serological tests are negative when dogs are in anergy, but generally these animals present severe clinical signs. Seropositive results also do not differentiate between diseased and carrier animals. Ideally, if the results are positive, high-sensitivity techniques should be coupled with a highly specific technique.

**Immunofluorescent antibody test**

The immunofluorescent antibody test (IFAT) using promastigotes is the reference technique (12); its cut-off value is 1/80 for dogs.

**Enzyme-linked immunosorbent assay**

The enzyme-linked immunosorbent assay has been replacing the IFAT since commercial kits became available. It has higher sensitivity and specificity; it is able to be automated, easy to read and provides objective results.

**Polymerase chain reaction**

In polymerase chain reaction (PCR), DNA is extracted from the blood Buffy coat or from biopsies of different tissues. Several primer sets have been developed, which amplify different parts of the parasite's genome, mainly kinetoplast DNA minicircles, allowing us to identify DNA from the various species of the *Leishmania* genus (35, 36).

**Loop-mediated isothermal amplification**

Loop-mediated isothermal amplification (LAMP) PCR needs no thermocycler and so can be used in laboratories that are not well equipped. Indeed, the *Bacillus stearothermophilus* (Bst) polymerase used in LAMP PCR works at 60–65°C. Chaouch et al. (37) developed a LAMP PCR for the identification of *L. infantum* DNA, targeting a cysteine protease B multi-copy gene. The sensitivity of this technique is comparable to that of conventional PCR. Its specificity is higher than that of the generic 18S LAMP but Adams et al. (2010) reported a cross-reaction with *Trypanosoma cruzi* (38).

**Rapid tests: dual path platform**

Several rapid, ready-to-use, commercially available disposable dual path platforms (DPP) are available. They are based on a reaction between colloidal gold particles coupled with the protein A/G and the antigen bound to antibodies (39). A sample of 5 μL is placed in the device. These tests can be performed on serum, plasma and total blood with anticoagulant but no antibody quantification can be performed.

**Canine visceral leishmaniosi**

**Visceral lesions**

Visceral lesions may include the following:

- Lesions on the spleen. Splenomegaly is frequent, due to the infiltration of the spleen by immune cells, mainly monocytes and macrophages, and also to the hyperplasia of the red and white pulps.

- The bone marrow takes on the appearance of a dark pink or red jelly. This is more or less consistent.

- Lesions on the lymph nodes. There is hypertrophy of the cortical and medullary regions. However, in advanced stages, the size of the peripheral lymph nodes may be normal or even hypoplastic. A proliferation of macrophages and lymphocytes is observed, with numerous intra- and inter-macrophagic *Leishmania*.

- The liver is enlarged and calcified during the final stages (24).

**Gut lesions**

These are due to granulomatous or pyogranulomatous inflammation, leading to chronic diarrhoea in advanced cases (40, 41, 42).

**Testes**

The testes are atrophied, yellowish-brown or greyish, with thickening of the fibrous septa (43).

**Kidney lesions**

Kidney signs consist of glomerulonephritis and/or interstitial nephritis and, more rarely, amyloidosis. These explain the presence of proteinuria and can be responsible for the development of hypertension and the establishment of a vicious circle, whose final stage may be nephrotic syndrome and/or chronic renal failure, the most common cause of death during leishmaniosis.

**Other lesions**

Other lesions are frequent and polymorphic (30). They include: joint distension, sometimes associated with erosive arthritis; proliferative or lytic bone lesions; chronic hepatitis; chronic colitis; meningitis; and atrophic myositis of the masseter muscle and/or polymyositis.
Zoonotic aspects of *Leishmania infantum*

Healthy humans, whose susceptibility to *L. infantum* is low, most frequently experience asymptomatic infections. Clinically expressed infections are more frequent in infants below two years of age (15). The main reservoir of *L. infantum* is dogs; wild carnivores could also be involved but they are of lesser epidemiological importance in the life cycle of this parasite. This is due to their smaller biomass and density and also to their more distant relationship with the human population. The parasite is transmitted from an infected dog to a sandfly, then from the vector to a human being. Statistical data about the global frequency and geographic distribution of leishmaniosis are unreliable and imprecise and need to be further investigated (44). According to the World Health Organization (WHO) (4), this disease is endemic in 98 countries (Fig. 11) and its world incidence is estimated at 2 million cases, consisting of 0.5 million cases of visceral leishmaniosis and 1.5 million cases of the cutaneous form. Factors such as malnutrition, HIV infection (45) and immunodepression predispose humans to clinical forms of the parasitic infection (46). Fever, mucosal pallor and splenomegaly form a clinical triad, which is associated with low blood cell counts (anaemia, leukopenia, and thrombocytopenia) and an inflammatory syndrome (47).

Controlling leishmaniosis

The control of CvL is important because of its severe nature in infected dogs and its zoonotic character. Prevention is far more important than treatment because of constraints associated with treatment and its high cost. Moreover, individual control measures have no impact on the epidemiology of the disease since a high proportion of the canine population will never be affected by such measures (i.e. a large proportion of dogs with owners, stray dogs, and wild canids). Before choosing any control option, it is important to consider two factors:

- The epidemiological pattern of the targeted region. For example, is the infection occurring in sporadic, enzootic or hyperenzootic patterns?
- The impact of canine leishmaniosis on human health in the targeted area.

Treatment of clinical cases

Since there is no legislation concerning dogs with CvL, the decision to treat a diseased dog is the result of a discussion between the dog owner and the veterinarian. One important factor is the owner’s ability and/or willingness to comply with the treatment protocol. First, the dog’s kidney and liver...
functions should be investigated. If these are affected, then euthanasia is indicated. Each treated animal should wear an insecticidal collar during the season of vector activity to avoid reinfection and abolish the dog’s role of reservoir. Several molecules and protocols have been tried to treat CvL; the best approach is to associate leishmanistatic and leishmanicidal molecules. There are several excellent comprehensive reviews and trials of CvL treatment (48, 49, 50). Since a small population of parasites will remain in all treated dogs, serological and clinical monitoring should continue during the entire lifetime of the animal. One treatment method uses meglumine antimoniate (the leishmanicidal molecule) together with allopurinol (the leishmanistatic molecule):

i) Meglumine antimoniate is injected subcutaneously or intramuscularly, at a dose of 75–100 mg/kg once daily, or 40–75 mg/kg twice daily, for 4–6 weeks. Meglumine antimoniate causes nephrotoxicity (51), anorexia, vomiting, diarrhoea and reactions at the site of injection (50).

ii) Allopurinol is given orally at a dose of 10–15 mg/kg twice daily for at least 6–12 months (32, 52). Some 12% of dogs treated with allopurinol develop xanthine urinary crystals, leading, in some cases, to xanthine urolithiasis (53).

Miltefosine, a leishmanicidal alkylphosphocholine used in human dermatology as an antimitotic, can also be used to treat leishmaniosis in dogs when administered orally (2 mg/kg once daily for one month) in association with allopurinol (54). *Leishmania infantum* rapidly develops resistance against miltefosine.

Symptomatic treatment should be implemented to treat bacterial infections, ulcers, keratoconjunctivitis and other signs. Corticoids should be prescribed when immune complexes are deposited in the joints or kidneys.

**Vaccination**

Despite its huge importance in veterinary medicine and the number of candidates, vaccination against CvL only went into operation during the last decade. Three commercial vaccines are now available, as described below (44).

**Leishmune®**

This is a fructose mannose ligand (FML)-saponin vaccine; it was licensed in Brazil in 2003 to protect dogs against *L. donovani*. Its efficacy against this parasite was 76%, with a protection rate of 92%. The Brazilian Ministry of Health has not adopted Leishmune® as a tool for preventing leishmaniosis in canines.

**Leish-Tec®**

This vaccine contains recombinant A2-antigen of *Leishmania* amastigotes and uses saponin as an adjuvant. It was licensed in 2008. There is a lack of evidence for the efficacy of this commercial vaccine (55).

**Canileish® (LiESP/QA-21)**

This vaccine was licensed in several European countries in 2011. It contains a culture supernatant of *L. infantum* promastigotes (LiESAp), consisting of a 54-kilodalton (kDa) excreted protein of *L. infantum* with muramyl dipeptide (MDP). The efficacy of this vaccine against *L. infantum* was 68.4%, with a protection rate of 92.7%.

After exposure to natural *L. infantum* infection, the probability of vaccinated dogs becoming PCR positive was similar to that of dogs in a control group, but, among PCR positive dogs, the probability of reverting back to a PCR negative state was higher in the vaccinated group than in the group of control animals (53, 56).

**Killing of infected dogs**

In endemic areas, one method of control would be to carry out mass screening and kill the infected dogs. This control approach has been implemented in Brazil, but it would not gain public support in countries where the dog is considered a member of the family (57). Animal-health decision makers could include different zoonotic diseases in a single control programme (for example, leishmaniosis, echinococcosis and rabies). The role of international organisations, such as the World Organisation for Animal Health (OIE) and WHO, is important in establishing the framework for these programmes, suggesting various options and providing support. Such control programmes should be established after considering several interests: those of the animal itself, the owner, the veterinarian, the animal population as a whole, public health in general and economic interests.

In fact, the Brazilian control programme failed. A mathematical model showed that this failure was due to the high incidence and the severity of the infection among the dog population, the low sensitivity of the diagnostic test, and time delays between diagnosis and culling (58).

**Insecticide collars**

The use of deltamethrin in insecticidal collars should reduce the risk of infection for dogs in endemic regions. Manzillo et al. (59) performed a two-year field study on stray dogs in kennels in areas where leishmaniosis is highly endemic. These authors showed that signs of canine leishmaniosis occurred significantly more frequently (90% versus 36%) in non-collared dogs than in collared dogs, and that infection progressed more rapidly (59). But, in the absence of
other efficient insecticides, resistance to deltamethrin will eventually emerge.

**Vector control**

Vector control is more or less impossible with sandflies, since the shelters of the immature stages are difficult to identify and the imagos are spread throughout a huge environment. It is important not to make the common error of assuming that draining or otherwise treating standing water is efficient against sandflies.

**Extension programmes**

An extension programme should target dog owners, particularly those in the rural areas of developing countries. Owners should be encouraged to improve the living conditions of their animals, in particular by providing them with food and water of sufficient quality and quantity and suitable health care. Domestic wastes should be promptly collected, to avoid the development of a large stray dog population, which can act as a reservoir for the parasite. Veterinarians whose skills have been extended in this area should promote three important concepts to the public.

- Leishmaniosis is a zoonotic disease transmitted by sandflies, affecting mainly the immunocompromised, the very young and the elderly.
- Transmission of the infection to dogs occurs mainly outdoors, between sunrise and sunset.
- You should take your dog to a veterinarian if you notice one of these signs: skin lesions, lethargy or abnormal enlargement of the lymph nodes.

**Ethical aspects**

The ethical aspects of controlling leishmaniosis should be discussed at the national level, taking into account the epidemiological, socioeconomic and religious contexts. The community, in consultation with the national Public and Veterinary Health Services, should decide on the best control option by finding a compromise between protecting human health and respecting both human and animal welfare.

Official legislation and/or regulation should prescribe what should be done in cases of clinical canine leishmaniosis and when dogs test seropositive for the infection, in the case of both owned animals and strays. Owners should be encouraged not to abandon a seropositive or clinically affected dog, but to treat or euthanaze them.

**Conclusion**

Canine visceral leishmaniosis is one of the most important zoonotic diseases in dogs, and its epidemiological features are changing. Indeed, both the geographical distribution of the infection and its prevalence are growing. These factors make it of increasing importance in both human and animal pathology. Decision-makers in both human and animal health should collaborate closely to decrease its prevalence in the various regions of the world. Further studies are needed to improve our knowledge about this infection and, in particular, the role played by other animal species (such as cats and wild canids) as reservoirs.

**Acknowledgements**

This work received financial support from the Epidemiological Laboratory of Enzootic Infections of Herbivores (Laboratoire d’épidémiologie des infections enzootiques des herbivores) in Tunisia, Ministry of Higher Education, Scientific Research and Information, Technology and Communication.
Leishmaniosis (infection due to *Leishmania infantum*)

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**Resumen**

Los autores ofrecen una descripción general de la leishmaniosis canina causada por *Leishmania infantum*. Este protozoario transmitido por los flebótomos engendra una enfermedad que se caracteriza a menudo por una evolución crónica. Des signes cliniques cutanés et une atteinte viscérale surviennent à mesure que l'infection s'installe. Les principaux signes observés dans les formes classique de la leishmaniosis sont un élargissement des ganglions lymphatiques, un amaigrissement extrême et des lésions cutanées. La maladie est difficile à contrôler car les chiens demeurent porteurs pendant des années et le risque de récidive est permanent. Dans les régions endémiques, la meilleure méthode pour réduire la prévalence consiste à procéder au dépistage systématique des animaux infectés, qui sont ensuite traités ou euthanasiés. Des travaux de recherche complémentaires sont nécessaires pour améliorer l’efficacité des vaccins disponibles contre l’infection canine. Cette maladie est une zoonose ; chez l’homme, les signes cliniques sont principalement rapportés chez les sujets âgés, les jeunes et les patients immunodéprimés.

**Palabras clave**


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La leishmaniose canine (infection due à *Leishmania infantum*)

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**Résumé**

Les auteurs présentent une synthèse des connaissances sur la leishmaniose canine due à *Leishmania infantum*. Ce protozoaire transmis par les phlébotomes occasionne une maladie à l’évolution souvent chronique. Des signes cliniques cutanés et une atteinte viscérale surviennent à mesure que l’infection s’installe. Les principaux signes observés dans les formes classique de la leishmaniosthe are un élargissement des ganglions lymphatiques, un amaigrissement extrême et des lésions cutanées. La maladie est difficile à contrôler car les chiens demeurent porteurs pendant des années et le risque de récidive est permanent. Dans les régions endémiques, la meilleure méthode pour réduire la prévalence consiste à procéder au dépistage systématique des animaux infectés, qui sont ensuite traités ou euthanasiés. Des travaux de recherche complémentaires sont nécessaires pour améliorer l’efficacité des vaccins disponibles contre l’infection canine. Cette maladie est une zoonose ; chez l’homme, les signes cliniques sont principalement rapportés chez les sujets âgés, les jeunes et les patients immunodéprimés.

**Mots-clés**


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Leishmaniosis (infección por *Leishmania infantum*) en el perro

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**Resumen**

Los autores ofrecen una descripción general de la leishmaniosis canina causada por *Leishmania infantum*. Este protozoo transmitido por flebótomos, engendra una enfermedad que se caracteriza a menudo por una evolución crónica. A medida que la infección progresa aparecen signos clínicos en la piel y las vísceras. En las formas clásicas de la enfermedad, los principales signos observados son hipertrofia de los ganglios linfáticos, caquexia y lesiones cutáneas. El hecho de que los perros infectados sigan siendo portadores durante años y puedan recaer en cualquier momento hace difícil controlar la enfermedad. La mejor fórmula para reducir su prevalencia en las regiones donde es endémica pasa por la detección sistemática masiva de animales infectados y su tratamiento o eutanasia. Es preciso seguir investigando para lograr que las vacunas existentes protejan más eficazmente a los perros de la infección. La enfermedad es zoonótica: en el ser humano se describen casos clínicos sobre todo en personas de edad o jóvenes o en aquellas cuyo sistema inmunitario está debilitado.

**Palabras clave**

References


