

ATTACHMENT

INFORMATION PAPER ON PROPOSED POLICY AMENDMENT FOR CONDITIONS FOR THE IMPORTATION OF DOGS

***Leishmania* species**

INTRODUCTION*

Leishmaniasis is caused by the vector-borne protozoan parasite, *Leishmania*. Various forms of clinical manifestations of human leishmaniasis have been described and divided into three entities: visceral leishmaniasis (VL, kala azar), cutaneous leishmaniasis (CL, oriental sore, uta, pian bois, chiclero's ulcer) and mucocutaneous leishmaniasis (MCL, espundia). In the New World, leishmanioses are caused by *L. braziliensis* complex (MCL and CL), *L. mexicana* complex (CL), *L. peruviana* (CL) and *L. chagasi* (VL and CL); in the Old World, leishmanioses are caused by *L. donovani* (VL), *L. infantum* (VL and CL), *L. tropica* (CL), *L. major* (CL) and *L. aethiopica* (CL). *Leishmania infantum* and *L. chagasi* have been found to be identical by biochemical genotyping and should be regarded as synonyms. The diseases are mainly zoonoses with a few exceptions. Canine leishmaniasis (CanL) is a chronic viscero-cutaneous disease caused by *L. infantum* (= *L. chagasi*), of which the dog acts as the source reservoir. In some instances, parasites belonging to *L. braziliensis* complex, *L. major* and *L. tropica* have been isolated from this host. The vectors of leishmanioses are phlebotomine sandflies belonging to the genera *Lutzomyia* (New World) and *Phlebotomus* (Old World).

*(ref: OIE Manual of Diagnostic Tests and Vaccines)

ISSUES FOR CONSIDERATION

Leishmania spp. are intracellular protozoan parasites that cause significant disease in humans and dogs. It also affects other canids, some rodents, marsupials, sloths, anteaters and hyraxes. Dogs, humans and, less commonly, some non-domesticated mammals are reservoir hosts. Transmission is normally by phlebotomine sandflies (Order Diptera, Family Psychodidae, Subfamily Phlebotominae) but may result from direct contact with blood or secretions. Leishmaniasis in humans and dogs is widespread through Africa, southern Europe and Asia, and in South and Central America. Major epidemics occur in the Middle East and South America. Endemic foci are found in the Mediterranean Basin countries, India, Africa, parts of China and other areas of Asia.

The distribution of diseases caused by *Leishmania* spp is changing. *Leishmania infantum* infection has become established in Foxhounds in the United States of America and Canada, with transmission not thought to be by sandflies. The endemic range in Europe is shifting north and westwards from Mediterranean countries. In the United Kingdom, follow-up of pets which have become ill following short-stay travel to European Union countries under the Pet Travel Scheme (PETS) has revealed a low incidence of leishmaniasis (as well as other vector transmitted diseases). Autochthonous (locally acquired) leishmaniasis is considered exotic to the United Kingdom.

Leishmania spp. are transmitted by phlebotomine sandflies of the genera *Phlebotomus* (Old World) or *Lutzomyia* (New World). These genera have not been identified in Australia. Australian phlebotomines have been reclassified into three genera - *Australophlebotomus*, *Idiophlebotomus* and *Sargentomyia*. The likely feeding hosts of Australian phlebotomine sandflies are small mammals (such as rabbits and bats) and some reptiles.

Leishmania spp. may cause cutaneous disease or a severe visceral disease in humans and dogs, with dogs as reservoir hosts for the disease. In southern Europe, *Leishmania* is an important opportunistic infection in human HIV/AIDS patients and other immunocompromised individuals.

The internal form of the disease in humans is called “kala-azar”, and frequently leads to death after a chronic illness with weight loss, lymphadenopathy, anorexia, and muscle wasting. The parasite is spread through the body via macrophages and infects the bone marrow, liver and spleen. Renal failure is common in dogs and syndromes involving the respiratory or gastrointestinal tract are common in humans. Dogs exhibit both cutaneous and visceral forms of the disease, called canine visceral leishmaniasis (CVL).

Parasitaemia may occur intermittently throughout the course of the disease. Parasites may be detected by direct examination of stained blood smears, or by polymerase chain reaction (PCR) testing of bone marrow, spleen or lymph node tissue, or blood. Highest sensitivity is obtained from splenic biopsy which is, however, considered unsuitable for general import testing.

Antibody testing is more sensitive than direct detection methods but false negatives occur. Serological diagnostic methods may underestimate the prevalence of infection because of the timing of testing, impaired immune response, or sequestering of the parasite in the spleen, lymph nodes, liver and bone marrow. Seroconversion generally occurs from 10 days to 6 months post-infection depending on the immune status of the dog. Fifty to eighty percent of seropositive dogs are asymptomatic but, in the Old World (Europe, Asia and Africa), most infected dogs eventually develop the disease.

Both symptomatic and asymptomatic infected dogs may be infective to sandfly vectors. The presence of infected dogs in non-endemic areas could lead to the establishment of new endemic cycles if competent vectors of visceral leishmaniasis are present. Treatment of infected dogs may produce clinical improvement but not parasitological cure. Dogs may still be infective to vectors while undergoing treatment. There is no effective vaccine available for dogs at present.

A study in Brazil has shown that it is possible for the tick *Rhipicephalus sanguineus* to be infected by *L. chagasi* (*infantum*) naturally from dogs, and that this tick may be a possible vector. Further studies are needed to demonstrate the tick’s vector competency. *Rhipicephalus sanguineus* is widespread in Australia.

A number of wild species and rodents in endemic countries can be infected. Infections in wild animals other than canids are generally mild or inapparent. Leishmaniasis in cats has not been commonly diagnosed or reported and there is little evidence that cats play a role in the maintenance or spread of the disease. Infected cats show mainly cutaneous symptoms, but visceral leishmaniasis occurs occasionally. Recent literature indicates the need for further research on the role of the cat as a reservoir host in endemic areas.

FURTHER READING

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