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Molecular survey of *Leishmania infantum* in the blood of dogs from French Guiana

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Purpose: Canine leishmaniasis is caused by *Leishmania infantum*, or also called in the New World *L. chagasi*. It is considered to be a major potentially fatal zoonotic infection where the domestic dog is the main reservoir. This infection is worldwide and reported with a higher incidence in tropical and subtropical areas such as South America where the number of infected dogs is estimated in the millions. No molecular data has been available on this disease in French Guiana. The aim of this study was molecular investigation of occurrence of leishmania in the blood of dogs in this region.

Methods & Materials: Since 2016, blood samples were collected on a total of 98 dogs from French Guiana and 26 other dogs coming from continental France, were sampled before and after a 4-month mission in the same region. The samples were tested by a qPCR to quantify *L. infantum* kinetoplast DNA. Some positive samples were confirmed and species were identified, after sequencing, by using two standard PCR systems: i) a pan-*Leishmania* system was designed, targeting 28S rRNA gene and ii) PCR generic primers to amplify a segment of the rRNA internal transcribed spacer 2 (ITS2) from multiple *Leishmania* species.

Results: The results show at least 4.08% (3/98) were positive to this pathogen and two (2/26) dogs returned positive although they were negative to begin with; one of them had an ulcer on the pastern. This last had 9×10^7 Leish/mL of blood, 1.3×10^{10} Leish/mL from ulcer swab and around 4.3×10^6 Leish/g of bone marrow. In general, the parasite load was from 2.5 Leish/mL to 8×10^{13} Leish/mL of dog's blood. Sequencing analyses identified *L. infantum* specie.

Conclusion: The detection of *L. infantum* in local dogs in French Guiana and in dogs from metropolitan France after coming back from French Guiana, provide evidence that this region is endemic for canine leishmaniasis. It highlights the need for active surveillance in canine population and implementation of control measures. Competent vectors in this region are yet to be identified.

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The role of IgG avidity determination in diagnosis of West Nile virus infection

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Purpose: West Nile virus (WNV) IgM antibodies have been shown to persist for up to 500 days or even longer in some patients. Therefore, additional tests are needed to confirm primary infection. The aim of this study was to analyze the value of IgG avidity in diagnosis of symptomatic and asymptomatic WNV infection.

Methods & Materials: A total 54 WNV IgM and/or IgG positive serum samples collected from 39 patients with neuroinvasive disease (meningitis/encephalitis/myelitis) and 15 asymptomatic persons were tested for WNV IgG avidity. Serological tests were performed using a commercial enzyme-linked immunosorbent assay (Euroimmun, Lübeck, Germany) and confirmed by virus neutralization test. Avidity index (AI) was calculated and expressed as percentage using the extinction values with and without urea treatment. The interpretation of AI results was determined as follows: AI < 40% = low avidity antibodies indicating acute primary infection; AI 40-60% = borderline avidity indicating recent (post-acute) infection; AI > 60% = high avidity antibodies indicating past WNV infection.

Results: WNV IgM antibodies were detected in 47/87.0% samples: 39/100% patients with neuroinvasive disease and 8/53.3% asymptomatic subjects. Recent WNV infection was documented by low/borderline AI in 44/93.6% IgM positive samples. In 3/6.4% IgM positive samples, high AI was detected indicating persisting IgM antibodies from a previous infection. All 7 IgM negative samples showed high AI. In 33/84.6% patients with WNV neuroinvasive disease tested within 30 days after onset of symptoms AI was low. Six patients (15.4%) tested 34-50 days after disease onset showed borderline AI (42-60%) indicating earlier maturation of WNV IgG antibodies.

Conclusion: The results of this study indicate that IgG avidity differentiates current/recent WNV infection from persistent IgM antibodies both in patients with WNV neuroinvasive disease and asymptomatic persons. Since many patients showed rapid avidity