

Recent achievements in research at the UCD School of Veterinary Medicine

Exploring relationship between neutrophil activation and states of CanL infection

Canine leishmaniosis (CanL) is a widespread zoonotic disease caused by the protozoan parasite Leishmania infantum, transmitted primarily through the bite of female Phlebotomine sand flies. Dogs, acting as the main reservoir, harbour the parasite within various myeloid lineage cells. Clinical manifestations of CanL range from subtle to severe systemic disease, with the interplay between the host's immune response and the parasite being crucial in determining disease outcome. While the adaptive immune response has traditionally been the focus of research, recent evidence suggests a significant role of the innate immune response, particularly neutrophils, in controlling Leishmania infection. Interferons (IFNs) are proteins known to be crucial in regulating immune responses. Among them, IFN-y has been widely reported to induce a protective response against leishmania infection. Additionally, recent research indicates its ability to influence myeloid cell activity, including neutrophils, in various ways.

This study delved into the involvement of neutrophils in CanL, focusing on neutrophil activation through the nitroblue tetrazolium (NBT) test, and its association

Research team

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with different stages of Leishmania infantum infection, antibody production, and IFN- γ levels. The results unveiled significantly higher neutrophil activation in dogs with mild, self-limiting disease compared to healthy seronegative dogs and those in advanced disease stages. Moreover, healthy seropositive dogs displayed elevated neutrophil activation, suggesting a link between increased neutrophil activity and either exposure to Leishmania without disease development or mild, self-limiting disease, emphasising the role of neutrophil activity in disease control. Additionally, a significant positive association was observed between IFN- γ production and heightened NBT test results, suggesting a potential relationship.

This study highlighted the critical role of neutrophils in controlling Leishmania infection. Full paper in: Vet. Sci. 2023, 10, 572. https://doi.org/10.3390/vetsci10090572

Gallbladder wall thickness in fasted dogs without signs of hepatobiliary disease

Ultrasound examination is commonly used to estimate gallbladder wall thickness in dogs, aiding in the diagnosis of gallbladder disease. However, there are no published reference values available for dogs supported by published measurement data. The aim of this study was to establish the normal thickness of the gallbladder wall in dogs presented to a referral hospital and requiring abdominal ultrasound examination.

This was a cross-sectional observational study, recruiting dogs requiring abdominal ultrasound examination for reasons unrelated to primary hepatobiliary disease. A standard ultrasonographic sequence of gallbladder wall images was recorded for later review.

Inclusion criteria were normal ultrasonographic hepatobiliary, pancreatic and small intestinal findings. Dogs were excluded if they had inadequate medical records, a previous history of hepatobiliary, gastrointestinal or pancreatic disease likely to impact the biliary system (e.g., nausea, chronic vomiting, jaundice, diarrhoea), unexplained increases in liver enzyme activities, hypoalbuminaemia, or ascites.

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As a result of the study, the upper limit for gallbladder wall thickness in 53 fasted (8 hours) dogs less than 40 kg was determined to be 1.30mm (90 per cent confidence interval, 1.19-1.41).

In conclusion, the normal gallbladder wall thickness in dogs is considerably lower than previously reported. Use of this new value is likely to significantly increase the sensitivity for diagnosis of gallbladder disease in dogs. However, additional studies are required to determine the potential effects of body weight, and to determine the optimal cut-off to distinguish between healthy and diseased gallbladders.

The full paper is available at: https://onlinelibrary.wiley.com/doi/10.1111/jvim.16810