Letter to the Editor

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Dear Drs. Hinchcliff and DiBartola,

We thank Drs. Steiner, Xenoulis and Suchodolski for their comments. The purpose of this study was to evaluate canine pancreatic lipase immunoreactivity (cPLI) concentrations, determined by Spec cPL and SNAP cPL, in dogs with hyperadrenocorticism (HAC) without clinical pancreatitis. Both assays were found to have poor specificity for clinical pancreatitis in dogs with HAC.

Drs. Steiner, Xenoulis and Suchodolski note that all possible clinical signs of pancreatitis were not included on the questionnaire used for initial evaluation of cases. The screening questionnaire asked for clinical signs which included anorexia or hyporexia, vomiting, diarrhea and abdominal pain. To avoid recruitment and collection bias, clinical signs of pancreatitis were limited to those that also occur with hypoadrenocorticism. As noted in the methods, however, one of the authors (DIM) contacted attending veterinarians for cases meeting initial inclusion criteria to obtain detailed case information, including additional clinical signs of disease present in each animal. Dogs with clinical signs of pancreatitis were then excluded from the study population.

We do not disagree that some dogs enrolled in the study could have had subclinical pancreatitis, as we discuss in the paper. It seems unlikely, however, that 55% of the dogs with HAC in this study also had asymptomatic pancreatitis. Although a few studies have identified HAC as a risk factor for severe clinical pancreatitis,[1,2] the authors are unaware of any studies showing an association between HAC and occult pancreatitis. Additionally, the clinical relevance of subclinical histologic inflammation of the pancreas remains unknown, as do the benefits (or lack thereof) of serial cPLI monitoring[3] and dietary intervention in dogs without clinical evidence of pancreatic disease.

Benign pancreatic hyperenzymemia is a fascinating biochemical phenomenon in healthy people.[4] These people have increased pancreatic enzymes on routine blood work without pancreatic disease,[4] based on clinical signs and advanced imaging results. These subjects have wide variations in serum pancreatic enzyme concentrations over time; at least one year must pass without development of pancreatic disease before the diagnosis of benign hyperenzymemia is made.[5] Amylase, lipase and trypsin are concomitantly increased in 90 – 95% of cases, but occasionally only one enzyme is increased.[5] Because lipase assays used in people are not pancreatic specific, the origin of increased lipase has not been definitively determined. It has been presumed to be pancreatic, however, based on the concomitant increases in amylase and trypsin in the vast majority of cases. The etiology of benign pancreatic hyperenzymemia remains unknown.

Respectfully, we did not conclude that cPLI concentrations are falsely increased by HAC, as averred by Drs. Steiner, Xenoulis and Suchodolski. Instead, we concluded that, due to their limited specificity for clinical disease in this population, cPLI test results should be interpreted with caution in dogs with HAC – particularly if other diagnostic test results do not support the diagnosis of clinical pancreatitis. We look forward to elucidation of the etiology of the association between HAC and abnormal cPLI results and its potential clinical relevance.

Sincerely,

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