

DETERMINATION OF FELINE SERUM AMYLOID A (SAA) BY MEANS OF A COMMERCIALY AVAILABLE HUMAN SAA TURBIDIMETRIC IMMUNOASSAY

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Introduction

Serum Amyloid A (SAA) has been recognized as an acute phase protein in cats with a marked increase within 8 hours after the occurrence of an inflammatory stimulus. Maximum concentration is reported to be reached after 24-48 hours and a fast normalization was observed when no further stimulus was present. These features suggest feline SAA to be useful as a marker for inflammation as also sustained by previous studies. However, at the present no automated commercially available assay is validated with a practicability expected by modern laboratories for routine diagnostic purposes. The objective of this study was to evaluate whether feline SAA could be measured reliably using a turbidimetric immunoassay (TIA) designed for the determination of human SAA.

Materials and Methods

A commercially available TIA for human SAA (Eiken Chemical Co, Tokyo, Japan [Lot no. 47007]) was used for determination of feline SAA performed on an automated chemical analyzer (ADVIA 1650, Bayer, Newbury, UK) according to recommendations of the manufacturer for human SAA determination. Intra- and interassay imprecision were investigated by multiple measurements of feline serum samples and serum pools, respectively, and assay inaccuracy was assessed by investigation of linearity under dilution. Overlap performance was investigated by comparison of SAA levels of (A) clinically healthy cats (n=56) and cats with a clinical diagnosis of an inflammatory disease (n=13) and (B) cats with (n=31) and without (n=72) an acute phase response (as evidenced by an elevated level of alpha1-acid glycoprotein determined by a feline specific immunodiffusion assay [Ecos Institute, Miyagi, Japan]), respectively.

Results

The observed intra- and interassay imprecision were between 2.1–9.9% and 7.0–12.5%, respectively. Dilutions were measured in a linear and proportional manner indicating no significant inaccuracy. A significant difference ($P<.05$, non-parametric) in SAA concentration (median [range]) was observed between both (A) healthy cats (0.4 mg/L [0.0-3.8 mg/L]) and cats with a diagnosed inflammatory disease (46.6 mg/L [3.3-150.6 mg/L]) and (B) between cats with (21.3 mg/L [0.4-150.6 mg/L]) and without (0.4 mg/L [0.0-60.4 mg/L]) a present acute phase response.

Discussion and Conclusion

The observed analytical performance seemed acceptable for clinical purposes. In the overlap performance, the expected difference in SAA levels according to the presence or absence of systemic inflammation was detected. Thus, feline SAA could be determined reliably by use of the human SAA TIA. The high practicability of the assay, being commercially available and automated should facilitate the implementation of feline SAA measurements for routine diagnostic purposes.