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Guest Editorial

Clustering of Feline Coronaviruses in Multicat Households

Feline infectious peritonitis (FIP), caused by an inappropriate immune response to feline coronavirus (FCoV) infection, is currently said to be the leading infectious cause of death amongst pedigree cats and cats from shelters (Vennema et al., 1998). Yet little is known about this virus and the pathogenesis of FIP and, despite over 30 years of research, many questions remain unanswered. In this issue of the Journal, Kiss et al. (2000) have contributed one more part to the FCoV jigsaw. FCoV is incredibly difficult to isolate, which has hampered research, but Kiss et al., used molecular biological techniques to determine how many clinically healthy pet cats in Eastern Hungary were shedding virus and to examine the relatedness of the viruses found. FCoV was found in 36 of 113 (31.8%) cats sampled: this proportion was perhaps artificially increased by 15 of the cats coming from two Persian breeding catteries, 12 of which were positive. Eighty-eight of the remaining 98 cats were from single cat households, so if 24 were RT-PCR positive, then it would appear that around 24% of normal pet cats sheds FCoV (although again this proportion could be artificially increased if the undefined 10 all come from one household and a high proportion were shedding FCoV). The situation in the Persian breeding households serves to illustrate that FCoV shedding increases in situations where many cats are kept together, perhaps indoors, sharing litter trays, and where there is frequent exposure to other sources of FCoV through mating and possibly cat shows (Kiss et al., 2000).

FCoV is an RNA virus and therefore its huge genome is relatively unstable; this feature allows scientists to compare isolates from various sources for similarity. Kiss *et al.* showed that, as suspected but only hitherto demonstrated in one household by Herrewegh *et al.*, (1997), FCoVs from different households formed a cluster, indicating that they had probably originated from one coronavirus. In addition, this finding seems to confirm Dr Herrewegh's suspicion that infection with one

FCoV somehow conferred protection against other strains of FCoV (Herrewegh et al., 1997). It used to be thought that cats seropositive to FCoV were more, not less, likely to develop FIP. The theory was that antibody caused enhanced infection enabling the virus to enter macrophages more easily (Weiss & Scott, 1981) and it was noted that experimental infections of seropositive cats resulted in more of the seropositive cats than seronegative control cats developing FIP and did so more rapidly. Observations of naturally infected cats seemed to refute that finding (Addie et al., 1995): cats which had been exposed to FCoV infection appeared to be less, not more, likely to develop FIP. At last we have a possible explanation for that finding—that cats infected with one FCoV are resistant to superinfection by other FCoV strains. The consequences of these two schools of thought for practicing veterinary surgeons were exactly opposite—if one had to introduce a new cat into a household of seropositive cats it appeared from the experimental infections that it was safer to introduce a seronegative cat. Observation of naturally-infected animals showed that, in fact, it was safer to introduce a seropositive cat.

Neils Pedersen once said that more cats had died of FIP antibody tests than had ever developed FIP; likewise, a danger in the increased use of RT-PCR in FCoV infection is that it might be misused in an attempt to cull FCoV carriers. There is absolutely no justification for euthanasing healthy FCoV excretors, as they may well stop shedding FCoV the next week or the next month—a fact that does not emerge in the Kiss paper, since they were looking at a single timepoint. In this author's experience, the only sure way to identify a FCoV carrier (i.e. a cat which will shed FCoV for life) is by monthly faecal testing for at least a year showing uninterrupted virus shedding (unpublished data)

The advent of molecular biological techniques, and in particular the huge contribution by the group at Utrecht, has enabled research into FCoV infection to take off in a new way and, in particular, to enable more research into naturally occurring infections, which do seem to behave completely differently to laboratory infections for many reasons—virus strain, dose and mode of infection. I look forward to real progress being made in the fight against FIP in the coming years.

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REFERENCES

Addie, D.D., Toth, S., Murray, G.D. & Jarrett, O. (1995). The risk of feline infectious peritonitis in cats

naturally infected with feline coronavirus. *American Journal of Veterinary Research.* **56**, 429–34.

HERREWEGH, A.A.P.M., MAHLER, M., HEDRICH, H.J., HAAGMANS, B.L., EGBERINK, H.F., HORZINEK, M.C., ROTTIER, P.J.M. & de GROOT, R.J. (1997). Persistence and evolution of feline coronavirus in a closed catbreeding colony. *Virology* **234**, 349–63.

KISS, I., KECSKEMETI, S., TANYI, J., KLINGEBORN, B. & BELAK, S. (2000). Prevalence and genetic pattern of feline coronaviruses in urban cat populations. *The Veterinary Journal* 159, 64–70.

VENNEMA, H., POLAND, A., FOLEY, J. & PEDERSEN, N.C. (1998). Feline infectious peritonitis viruses arise by mutation from endemic feline enteric coronaviruses. *Virology*, 243, 150–7.

Weiss, R.C. & Scott, F.W. (1981). Antibody-mediated enhancement of disease in feline infectious peritonitis: comparisons with Dengue haemorrhagic fever. *Comparative Immunology, Microbiology and Infectious Diseases (Oxford)* 4, 175–89.

Book Review

Self assessment colour review of small animal abdominal and metabolic disorders

Tennant, B., London, Manson Publishing, 1999. £19.95 (soft) ISBN 1874545499

This book provides 196 questions on clinical cases in dogs and cats with abdominal and/or metabolic diseases. Questions are asked after presentation of a short history, provision of relevant clinical findings and sometimes histology results, serum chemistry findings and/or radiographs or endoscopic views. The cases have been prepared by 11 authors, specialists in small animal medicine from UK, USA and Ireland. The cases are presented in different ways, which is said to reflect the individual approach of particular authors. While an individual approach to cases can be commended, appropriate editing would have helped to present the cases in a uniform format.

The aim of the book is not only to test the ability of the reader, but also to be informative, with the target readership being all veterinary surgeons and students. Does the book succeed? In part, yes; but for this reviewer there was frustration with the format and the nature of some of the questions, with limited information provided in some answers. The cases are not grouped by systems, so that selection is random. If I wanted to test my knowledge on the urinary tract I could not focus on those questions without thumbing through the book, 'on search', or refer to the index and then go to different pages. Each question

had laboratory values, but all reference ranges were in tables at the back of the book. Reference values would have been better given with each table in the text, especially for less common tests (e.g. parathormone). The answers provided frequently did not provide a recipe for action: 'Monitor the urine' and 'Check the pH' are unhelpful statements and do not tell the reader what to look for or how to act on the pH value measured.

The cases are often not presented in a problem-oriented manner, or the focus for interpretation immediately refers to a radiograph, histopathology or other diagnostic results related to the case. This information would have been better given over the page so that the reader can think about the case without being 'led', and decide for themselves what tests they would select and why. However, the photographs are of excellent quality, although radiographs and ultrasonographs would have been more informative if they were 'explained' by arrows or other notations in the answer page.

The book provides testing questions, but an authoritative small animal medicine text also needs to be read in parallel in order to provide further information on the answer provided for many cases. Students especially would want to know why. The book has challenging questions, which would be useful in a practice library, especially for veterinary surgeons who are preparing for certificate or other examinations. For this reviewer, however, it fell short of its stated goals.

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