

LETTER TO THE EDITOR

Dear Sir,

We would like to draw your readers' attention to our observations of an unexpectedly high prevalence of azotaemia in Birman cats.

Over several years we have observed that many young Birman cats referred to us for a variety of reasons had unexpectedly elevated urea and/or creatinine concentrations. In addition, several young Birman cats have been referred to us with renal failure. Some of the most severely affected cats have been under two years of age, with clinical signs developing shortly after routine neutering. Unfortunately, because of the low numbers involved and the lack of a specific diagnosis in many cases, it has not been possible to draw any firm conclusions as to the likely cause of the renal disease.

As a result of these observations, a small prospective survey of healthy Birman cats was undertaken to evaluate serum creatinine concentrations in a representative sample of these cats in the UK. The cats were recruited with the assistance of The Southern and South Western Birman Cat Club, and all were considered healthy on the basis of routine clinical examination. Where possible, a follow-up blood sample was collected eighteen months after the first.

Samples were collected from 106 Birman cats from 19 different households. They ranged in age from eight weeks to 12 years; 22 (21%) were between two and six months of age, 64 (60%) were between one and six years of age, and the remainder (19%) were between six and 12 years of age. Fifty percent were entire females, 14% neutered females, 23% entire males, and 13% neutered males (a high proportion were entire cats as many came from breeding households). The results of the creatinine determinations are shown in [Table 1](#) and reveal a surprisingly high prevalence of hypercreatininaemia. Of the cats less than six months of age 82% had creatinine concentrations above the reference range, while of the adult cats 35% had creatinine concentrations above the reference range.

Sixty-seven of the cats were reassessed 18 months after the first blood samples had been collected. Clinical information was available for all of these cats, but repeat blood samples were available for only 39. Two of the cats were reported to have died of renal failure (based on clinical and biochemical findings), one aged 10 years, the other at less than one year of age. Interestingly, only one of the three kittens that initially had elevated creatinine concentrations still had an elevated concentration at the second testing. Overall, of the cats that were sampled

Table 1. Serum creatinine levels in 106 healthy Birman cats

	Kittens (<6 months)	Adults
Age (median, range)	3 months (2–6)	4 years (1–12)
Number	22	84
Median creatinine ($\mu\text{mol/l}$)	100.5	148.0
Range	56–178	91–245
Inter-quartile range	86–114	124–176
Reference range ($\mu\text{mol/l}$)*	38–78	60–160
Mean ($\mu\text{mol/l}$) (standard deviation)	58 (10)	118 (19)
Number above reference range	18 (82%)	29 (35%)
Number >2 standard deviations above reference range	7 (32%)	12 (14%)

*Determined using 74 specific pathogen free (SPF) adult cats and 52 SPF kittens (8–18 weeks of age).

twice, 11 cats had elevated creatinine concentrations at the first sampling and 12 at the second sampling, but only six had elevated concentrations on both occasions. There was no significant difference in median creatinine concentrations (140 vs 143 $\mu\text{mol/l}$) between the two samplings (Wilcoxon signed rank test, $P=0.12$).

Despite the limitations of this small study it does provide evidence of an unexpectedly high prevalence of elevated serum creatinine concentrations in Birman cats in the UK, and especially in young Birman cats (less than six months old). However, the reason(s) for this finding remain unclear. It could reflect sub-clinical renal disease. In support of this, two of the cats, while apparently healthy at the time of first testing, developed progressive clinical and biochemical evidence of renal failure within a few months, and were euthanased (a post-mortem examination was not permitted in either case). Furthermore, evaluation of urea concentrations in all of the cats showed a similar pattern to the creatinine concentrations (data not shown). Unfortunately, the interpretation of this data was complicated by some of the cats not having been starved prior to sampling. However, it is also possible that the elevated creatinine concentrations do not reflect underlying renal disease. Since concurrent urinalysis was not undertaken and no attempt was made to directly quantify glomerular filtration rate, no conclusions can be drawn on the presence or absence of renal dysfunction. It is interesting that such a high proportion of the Birman kittens exhibited elevated creatinine concentrations and yet the prevalence was much lower in the adult cats. Although some young Birman cats have been seen at our clinic with confirmed renal failure resulting in early death or euthanasia, there have not been any suggestions that this is a major problem within the breed. Thus, while some of the cats sampled may indeed have underlying renal disease, it is possible that other reasons exist for the observations made in this study.

This preliminary study suggests that many young apparently healthy Birman cats exhibit

azotaemia. While the clinical significance of these findings remain unclear, and further studies are clearly warranted, it seems appropriate to suggest that evidence of azotaemia in an otherwise healthy Birman cat should not be over-interpreted as evidence of severe or progressive renal disease. It would however, seem sensible to monitor affected cats, to perform other additional tests of renal function, and to consider the possibility of renal dysfunction when undertaking anaesthesia, surgery or treatment in cats of this breed.

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