

Dietary Iodine Level and Thyroid Function in the Cat^{1,2}

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EXPANDED ABSTRACT

Indexing Key Words:

- cats • thyroid disease • dietary iodine
- thyroxine

Feline hyperthyroidism was rarely reported until 1979 (Peterson 1984), but is now diagnosed frequently and is considered a common clinical condition in the cat. Hyperthyroidism is usually associated with nodular goiter and is not considered to be autoimmune in origin. Reasons for the sudden emergence of this condition have been sought. Because a large proportion of cats in the Western world are fed commercially produced rations, various investigators (Mumma et al. 1986) have focused on possible dietary causes. In particular, inappropriate dietary iodine concentrations have been examined.

Our investigations have tackled the problem in three ways. In our first study (Johnson et al. 1992), we measured the iodine content of 28 varieties of cat food (23 canned, 5 dried) commercially available in New Zealand. Food was purchased in a local supermarket, thoroughly blended and analyzed for iodine concentration using an automated method based on the ceric-arsenic acid reaction (Garry et al. 1973). We concluded that there was a considerable difference in the iodine concentrations of the various foods (range <1.48–167.0 $\mu\text{mol/kg}$ dry weight). Published recommendations of iodine levels in cat foods are variable and range between 1.1 and 23.6 $\mu\text{mol/kg}$ dry weight (Mumma et al. 1986); on this basis, 32% of cat foods we tested were out of the recommended range.

In a second study (Tarttelin et al. 1992) we measured serum fT_4 levels in cats fed diets of variable iodine content for a short time (2 wk). The subjects were 10 castrated 2-y-old cats. They were fed alternatively a control diet [mixture of canned diet and dry diet, 12.9 and 22.8 μmol iodine/kg dry matter (DM), respectively] and then canned test diets high, intermediate or low in iodine

(108.5, 17.5 and 0.8 $\mu\text{mol/kg}$ DM, respectively; the sequence of test diets was selected at random). After 1 wk and then at the end of the 2-wk feeding period, concomitant urine and jugular blood samples were taken. The fT_4 was measured by RIA and urinary iodine by the ceric-arsenic acid reaction (Garry et al. 1973).

Data were analyzed using analysis of variance. Student *t* tests were applied when the *F* ratio indicated significant differences existed between treatment group means. The paired serum fT_4 and the urinary iodine levels were subjected to regression analysis. Urine creatinine levels were measured, but the illustrated data are not transformed to iodine/creatinine ratios because statistical tests on both transformed data and untransformed data gave the same conclusions (mean \pm SEM creatinine levels in mmol/l : study periods 1, 3 and 5, 15.3 ± 5.3 ; period 2, 5.9 ± 2.0 ; period 4, 8.9 ± 3.3 and period 6, 8.5 ± 4.8).

Figure 1 illustrates some of the findings. There were highly significant differences in urinary iodine concentrations amongst the test-fed groups ($P < 0.001$, **Figure 1**). Urinary iodine concentrations at the end of the three control feeding periods were not significantly different. Serum fT_4 levels after control feeding were also not significantly different.⁴ However, serum fT_4 levels after feeding the high and medium level iodine test diet were significantly depressed when compared with controls ($P < 0.001$). Serum fT_4 levels after the

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⁴ Another group of 10 cats fed the control diet (but studied at a different time) showed similar levels of serum fT_4 when blood sampled monthly for 6 mo (grand mean \pm SEM thyroxine, 13.7 ± 2.8 pmol/l serum).

low iodine test diet were not different from those of the control period. When the serum fT_4 levels and the urinary iodine levels for each cat at each collection throughout the 12-wk study period were analyzed by regression analysis, a significant inverse linear relationship ($b = 0.59$; $P < 0.01$) was found. We concluded that the serum fT_4 level in cats is acutely responsive to changes in iodine intake, with high dietary iodine lowering serum fT_4 levels and vice versa.

A third study (Kyle et al. 1993) measured serum fT_4 levels in cats fed diets varying in iodine content for long periods (5 mo). We used two groups of eight cats aged between 1 and 2 y. All cats had been fed a control diet similar to that fed in the second study until the start of the test period. Then the cats were randomly assigned to a diet naturally low in iodine ($0.88 \mu\text{mol/kg DM}$) or the same diet with added iodine ($166.6 \mu\text{mol/kg DM}$). Our sampling, measurement and analytical procedures were similar to that described in the second experiment except that the diets were fed continuously for 5 mo and the sampling periods were monthly. Data are illustrated in Figure 2. Analysis of variance proved there were no significant differences between the mean levels of fT_4 of the two groups during the 5 mo of the study. Paired serum fT_4 levels and the urinary iodine levels for each cat at each collection showed no relationship when studied by regression analysis.

We were unable to measure the serum levels of thyroid stimulating hormone or triiodothyronine during our studies and so our knowledge of the response of the feline pituitary-thyroid system to changes in iodine intake is incomplete. Nevertheless, our results (as discussed in the second study) suggest that short-term feeding of cat food of widely differing iodine content results in a dramatic thyroid response as measured by serum free fT_4 . It is unclear, however, whether or not

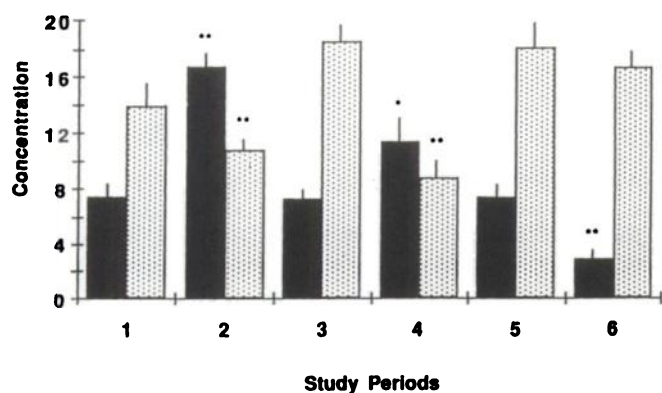


FIGURE 1 Mean concentration of urinary iodine (■, $\mu\text{mol/l}$) and serum fT_4 (□, pmol/l). Study periods 1, 3 and 5 were control periods, when the same diet was fed; 2, 4 and 6 illustrate periods when high, medium and low iodine diets were fed, respectively. Significant differences from the appropriate preceding control period are indicated by asterisks (* $P < 0.02$; ** $P < 0.001$).

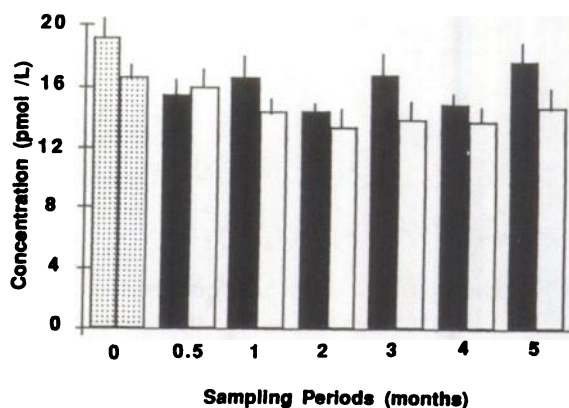


FIGURE 2 Mean serum fT_4 levels for two groups of cats after 5 mo on a high or low iodine diet. Standard errors of the mean are shown. Except for control period (0), when both groups were fed the same diet (□), diets are high iodine group (■) and low iodine group (□).

this can lead to permanent thyroid disease, such as nodular goiter with or without hyperthyroidism. The results of the third study support the concept that adaptive mechanisms tend to maintain the blood levels of thyroid hormone within the normal range in chronic states of high or low dietary iodine, and it is known that either state may eventually lead to goiter formation in humans and other animals (Delange and Ermans 1991, Naataki 1991). Because the development of goiter varies among individuals exposed to chronic excess or lack of iodine, it is likely that unknown genetic and environmental factors are influential in determining the final adaptive state.

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