Selenium status in adult cats and dogs fed high levels of dietary inorganic and organic selenium

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ABSTRACT: Cats (Felis catus) maintain greater blood Se concentrations compared with dogs (Canis familiaris) and, unlike dogs, show no signs of chronic Se toxicity (selenosis) when fed dietary organic Se (selenomethionine) concentrations of 10 μg/g DM. This study investigated the response of cats and dogs to high dietary concentrations of sodium selenite and organic Se to determine differences in metabolism between both species. In 2 consecutive studies, 18 adult cats and 18 adult dogs of with equal numbers of each sex were fed a control diet (0.6 μg Se/g DM) or the control diet supplemented to 8 to 10 μg Se/g DM from Na2SeO3 or organic Se for 3 wk. All animals were fed the control diet 1 mo before the start of the study and blood samples were taken on d 0 and 21. The Se balance was assessed during the final week and a liver biopsy was obtained on the final day of the study. Measurements included plasma Se concentrations, plasma glutathione peroxidise (GPx) activities, plasma Se clearance, Se intake, and urinary Se excretion. No clinical signs of selenosis were observed in the cats or dogs, and apart from Se clearance, form of Se had no effect on any of the measurements. Apparent fecal Se absorption was greater in the dogs fed both forms of Se, while greater plasma Se concentrations were observed in the cats on both the control and supplemented diet (P = 0.034). Cats fed the supplemented diets had lower hepatic Se concentrations (P < 0.001) and excreted more Se in urine (P < 0.001) compared with dogs. Furthermore, cats fed the Na2SeO3 supplement had greater Se clearance rates than dogs (P < 0.001). There was no effect of species on plasma GPx activity. We conclude that cats can tolerate greater dietary Se concentrations as they are more efficient at excreting excess Se in the urine and storing less Se in the liver.

Key words: cats, dogs, organic and inorganic selenium, selenium status

INTRODUCTION

Cats are known to maintain greater blood Se concentrations compared with other species. In 2 separate studies, concentrations of total Se in serum and plasma of cats were up to 10 times greater than in rats and humans (Forrer et al., 1991; Foster et al., 2001). Cats fed commercial diets maintained serum Se concentrations between 3.60 to 10.09 μmol/L, while dogs maintained reduced concentrations between 1.90 to 4.31 μmol/L (Forrer et al., 1991). Despite these increased blood Se concentrations, glutathione peroxidise (GPx) activity was not greater in cats compared with rats or humans and there was no change in GPx activity as a result of increasing Se intake above the minimum requirements (Foster et al., 2001; Wedekind et al., 2002, 2003; Todd et al., 2011). Todd et al. (2011) showed that Se either from Na2SeO3 or an organic source is effectively excreted by the kidneys to maintain plasma Se concentrations between 4.7 to 8.4 μmol/L, indicating that blood Se concentrations are closely regulated in cats.

Cats have been fed high levels (10 mg/kg) of Se for 6 mo with no clinical signs of toxicity (e.g., reduced

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food intake, reduced hair growth, and changes in serum chemistry profile; Wedekind et al., 2003). In contrast, dogs fed half this amount (5 mg/kg) showed a decreased feed intake (Wedekind et al., 2002) and reduced hair growth (Wedekind et al., 2002; Yu et al., 2006). The latter is in line with the study of Rhian and Moxon (1943) who studied chronic selenosis in dogs. Results showed 7.2 mg/kg of dietary organic Se or 10 mg/kg of sodium selenite were toxic as indicated by impaired growth and restricted food intake.

There have been no direct comparisons of the Se metabolism of cats and dogs fed similar dietary concentrations of Na₂SeO₃ or organic Se. Thus, the aim of this study was to investigate the response of cats and dogs to increased quantities (up to 8 to 9 mg/kg) of inorganic and organic dietary Se to gain insight into the Se metabolism of the 2 species and potentially determine differences in Se metabolism.

MATERIALS AND METHODS

This study was approved by the committee and conformed to the requirements of the Massey University Animal Ethics Committee (Massey University, 2003). The study was conducted as 2 identical trials run concurrently, one for cats and one for dogs, with no time between the trials.

Animals and Diets

Eighteen short-haired domestic cats (9 males and 9 females) and 18 harrier hounds (9 males and 9 females) were used for the study. Cats ranged from 1 to 4 yr of age and weighed between 2.80 and 4.96 kg (3.69 ± 0.12 kg); and dogs ranged from 2 to 8 yr of age and weighed between 16.0 and 27.0 kg (22.9 ± 0.6 kg) at the beginning of the trial. One month before each trial, all animals were fed the control diet, to standardize Se intake. The control diet (Table 1) used for both species was a commercial moist feline diet (Heinz Wattie’s, Hastings, New Zealand) that had passed a minimum adult feline maintenance feeding protocol according to AAFCO (2000) procedures and contained an analyzed Se content of 0.6 μg/g DM. Throughout the trial, animals were fed to meet their daily ME energy requirements calculated as 70 kcal/(kg BW·d) for cats and 110 kcal/[BW(0.75)-d] for dogs and had access to deionized water at all times.

Two different forms of Se were used to supplement the control diet: Na₂SeO₃, a 1% premix of Na₂SeO₃ and lime flour (Nutritech International, Ltd., Auckland, New Zealand) and a yeast-derived organic Se source (Sel-Plex, Alltech, Inc., Nicholasville, KY) containing selenomethionine, selenocysteine, and other selenoproteins and organo Se compounds. The trial design consisted of 3 groups of 3 animals, each assigned to one of the trial diets. The trial diets included the control diet (0.6 μg Se/g DM), and the 2 treatment diets supplemented with the respective form of Se to obtain a total Se concentration of 8 to 9 μg/g DM. Diets were freshly prepared on a daily basis as follows: an amount of control diet was mixed thoroughly in a 25-kg mixer (The Hobart Manufacturing Co., London, UK). A portion of the control diet was removed and the remaining portion was supplemented to contain 8 to 9 μg Se/g, using a sieve to disperse the powder. All diets were thoroughly mixed in the mixer before being fed to the animals.

Design

The trial consisted of a 2-wk adaptation period, where the animals were fed their respective treatment diets, and a subsequent 7-d collection period, in which samples were obtained for analysis. During the adaptation and collection periods, the animals were housed individually in metabolism cages to ensure they received the appropriate diet, monitor dietary intake,
Chemical Analyses

All diet, liver, and fecal samples were freeze-dried, ground to a fine powder using an electric grinder (Model CG-2; Breville, Oldham, UK), and mixed thoroughly before Se analysis. Hair was separated and removed from the feces after freeze-drying. All samples were analyzed in quadruplicate, except plasma and urine, which were analyzed in duplicate. Samples with replicates having a CV greater than 10% were subjected to further analysis until variability was reduced below this level. Total Se concentrations were analyzed using a fluorometric method (Simcock et al., 2005). Plasma GPx activities were assayed using a diagnostic kit and controls (Randox Laboratories, Ltd., Antrim, Northern Ireland) with an assay system (Cobas Fara II System; Roche, Basel, Switzerland). Plasma and urine creatinine samples were analyzed using a rate-blanked and compensated method (Creatinine Jaffé; Roche) with an analyzer (Roche Hitachi 912; Roche, Basel, Switzerland).

Calculation and Statistical Analyses

Fractional clearance of Se was calculated according to the equation:

\[
\text{Clearance (\%)} = \frac{((\text{Urinary Se} \times \text{Plasma Creatinine}) \times 100)}{\text{Plasma Se} \times \text{Urinary Creatinine}}
\]

Data were analyzed using SAS (SAS Inst., Inc., Cary, NC). Differences in plasma Se concentrations and GPx activities between treatment groups at the start of study (d 0) were tested using a 2-way ANOVA with treatment group and species as fixed factors. The interaction between treatment group and species was also included in the model. The effect of dietary treatment on both plasma measurements at d 21 of the study was analyzed using a 2-way ANOVA with diet, species, and the interaction between diet and species included in the model. Basal values of plasma Se concentration and GPx activity of animals at d 0 were included as a covariate.

The apparent fecal absorption of total Se in control animals, and Se absorption from Na₂SeO₃ and the organic source in treatment animals were expressed per unit BW and as a percentage of dietary intake. Dietary treatment effects on apparent fecal Se absorption, Se clearance, urinary Se excretion, and hepatic Se concentration at d 21 of the study were evaluated using a 2-way ANOVA, which was performed as described before. For each measurement, distribution of the
residuals was statistically evaluated and found to adhere to a normal distribution. When an interaction was observed, Bonferroni’s multiple-comparison test was performed. In all instances, differences were considered significant at a \( P = 0.05 \).

RESULTS

All animals remained healthy throughout both trials and fully recovered from the surgery. No signs of Se toxicity (e.g., vomiting, diarrhea, hair loss, and lameness) were observed. Average food intake during the collection period was 335 ± 10 g for the cats and 1,517 ± 31 g for the dogs. There was no difference in food intake per unit BW between the 3 groups within species. The actual Se concentrations in the experimental diets for the cats and dogs were 8.39 and 8.66 μg/g DM for the Na₂SeO₃ supplemented diets and 8.56 and 9.88 μg/g DM for the diets supplemented with the organic Se source. The analyzed Se concentration of the basal diet during the feline and canine study was 0.59 and 0.60 μg/g DM, respectively. All animals lost BW over the 3-wk collection period but diet did not affect the degree of BW loss. Cats lost 50 to 80 g (1.3 to 2.2%) and dogs lost 700 to 1,200 g (3 to 5.2%).

There was no difference in the mean plasma Se concentration and GPx activities of the cats or dogs in the different groups at the start of the study. However, plasma Se concentrations in the cats (average, 5.38 μmol/L) at the start were greater \( (P < 0.001) \) compared with those of the dogs (average, 3.50 μmol/L), while the GPx activities were not different between cats and dogs. At 21 d, the plasma Se concentration and GPx activity of the cats and dogs fed the control diet were not changed compared with the start of the study (data not shown). No interactions between diet and species were observed for plasma Se concentrations and GPx activity after 21 d of dietary treatment (Table 2). Plasma Se concentrations increased in animals fed the Se supplemented diets compared with those in the control group \( (P < 0.001) \). Overall, dogs had less plasma Se concentrations than cats \( (P = 0.034) \). Plasma GPx activities did not change in response to the increased dietary Se intake of the different forms in cats or dogs, and there were no differences in these activities between species.

Apparent fecal Se absorption expressed per unit of BW was not different between cats and dogs across the diets (Table 3). As expected, there was a diet effect in Se absorption with the control diet having a much lower absorption percentage compared with the supplemented diets for both species \( (P < 0.001) \). Apparent fecal Se absorption in percentage of the control diet and diets supplemented with Na₂SeO₃ and organic Se was greater in dogs compared with cats \( (P < 0.001) \). The calculated apparent fecal Se absorption from Na₂SeO₃ and organic Se source (excluding the control diet) for cats were 70.2 ± 2.9 and 69.6 ± 1.3%, while for dogs \( (P < 0.001) \), numerically greater values were observed (83.0 ± 1.4 and 78.5 ± 1.7%, respectively). The Se absorption was not different between the 2 sources.

Urinary Se excretion of the cats and dogs was low when the control diet was fed and increased up to 50-fold in cats and approximately 30-fold in dogs, when fed the supplemented diets (Table 3). Species differed in the urinary Se excretion with dogs fed the supplemented diets excreting less Se per day per kg BW than cats consuming these diets \( (P < 0.001) \). For both cats and dogs, the fractional clearance of Se from the plasma was greater in animals fed the 2 forms of Se than in the controls \( (P < 0.001) \). There were effects of diet, species, and the interaction between diet and species on Se clearance \( (P \leq 0.007) \). Cats had a greater clearance rate compared with dogs, with Se clearance of the cats fed the Na₂SeO₃ diet being greater \( (P = 0.021) \) compared with the organic Se fed cats. The Se clearance of the dogs fed the supplemented diets was not different. Both species had greater \( (P \leq 0.001) \) hepatic Se concentrations when fed the supplemented Se diets compared with those fed the control diet. There was no difference in the hepatic Se concentrations in animals fed the 2 forms of Se. However, concentrations of Se in the liver were greater in dogs compared with cats \( (P \leq 0.001) \).

DISCUSSION

The major signs of chronic Se toxicity or selenosis in animals include alopecia, hoof or nail necrosis and loss, and emaciation, while minor signs include

| Table 2. Plasma Se concentrations and glutathione peroxidise activity (GPx) in adult cats and dogs fed a control diet or the control diet supplemented with Na2SeO3 or organic Se1 for 21 d |
|-----------------|------------|-----------------|-----------------|
| Species | Diet | Total Se, μg/g DM | Plasma Se concentration, μmol/L | Plasma GPx2 activity, U/L |
| Cats | Control | 0.6 | 4.98 ± 0.20 | 12,611 ± 1,510 |
| + Na₂SeO₃ | 8.4 | 7.82 ± 0.18 | 13,751 ± 1,108 |
| + Organic | 8.6 | 9.13 ± 0.13 | 11,108 ± 591 |
| Dogs | Control | 0.6 | 4.15 ± 0.17 | 11,834 ± 864 |
| + Na₂SeO₃ | 8.7 | 7.21 ± 0.19 | 11,449 ± 904 |
| + Organic | 9.9 | 7.81 ± 0.50 | 10,638 ± 1,050 |
| P-value | Diet <0.001 | Species 0.034 | Diet × species 0.400 |


2GPx, glutathione peroxidise.
anorexia with concomitant BW loss and increased serum transaminases and alkaline phosphatase (Combs and Combs, 1986). In humans and some livestock, chronic seleniumosis has been reported when diets contained 5 μg Se/g DM (Koller and Exon, 1986). Selenium concentrations in commercial pet foods can reach >6 μg Se/g DM, with feline diets containing more Se compared with canine diets (Mumma et al., 1986; Simcock et al., 2005). Although there is one report describing acute Se toxicosis in the dog (Janke, 1989), to the knowledge of the authors, there are no reported clinical cases of chronic seleniumosis in cats and dogs in the literature. This lack of clinical cases may in part be related to low bioavailability of Se in commercial pet foods. Using a Se-depleted chicken bioassay, Wedekind et al. (1997) showed that Se bioavailability in moist and dry pet foods averaged between 17 and 25% relative to Na2SeO3. In subsequent studies, using the same methodology, greater relative bioavailabilities of canned (30%) and dry pet foods (53%) were reported (Wedekind et al., 1998). Todd et al. (2011) reported apparent fecal Se absorption of Na2SeO3, organic Se, and a commercial moist pet food for adult cats as 73.2, 80.0, and 25.3%, confirming the low bioavailability in moist commercial pet foods.

In the present study, the calculated apparent fecal Se absorption from Na2SeO3 and the organic source was greater in dogs by approximately 12.4 and 6.9%, respectively, compared with cats. In addition, the dogs absorbed more Se from the basal diet compared with the cats (21.2 vs. 37.4%, respectively). Todd et al. (2011) reported a mean apparent fecal absorption of Se in the same control diet of 25.3 ± 3.0%. Selenite has been reported to be less efficiently absorbed compared with selenomethionine because of its passive mechanism of transport through the brush border membrane of the gastrointestinal tract (Wolffram et al., 1986) in contrast to the active transport for selenomethionine (Wolffram et al., 1989). In cats, no difference was found in the apparent fecal Se absorption between the 2 sources (73.9 and 73.2%). An almost identical apparent fecal Se absorption for Na2SeO3 of 73.2% was found in adult cats by Todd et al. (2011). The latter authors, however, reported a greater apparent fecal Se value for the same organic Se source of 80.0%. In dogs, a value of 86.3% was found for the Na2SeO3 and 81.1% for the organic Se source. Normal absorption percentages of Se from foods, organic and inorganic compounds, and milk based foods have been reported to be around 70% in humans and experimental animals, with organic forms of Se being slightly better absorbed than inorganic forms (Combs and Combs, 1986; Van Dael et al., 2001).

Once absorbed, selenite is taken up by the red blood cells, reduced to selenide, released to the plasma, bound to albumin, and transported to the liver (Suzuki, 2005). Selenium from selenocysteine can be liberated in the liver by selenocysteine-β-lyase and reduced to hydrogen selenide, which can be used for selenoprotein synthesis or metabolized to methylated excretory products. Selenide is assumed not only to be a common intermediate but also a checkpoint intermediate for utilization for selenoprotein (e.g., GPx, thioredoxin reductases, and iodothyronine deiodinases) synthesis, and the excretion of methylated Se (Suzuki, 2005). Selenomethionine can be metabolized into selenocysteine via the same transamination and transsulphuration pathway as methionine. In addition, selenomethionine can be directly used for body protein synthesis, providing a stable and safe storage mode for Se. The major urinary Se metabolite in rats and humans fed Se in the low-toxic range is 1β-methylseleno-N-acetyl-D-galactosamine or selenosugar B, while at greater dosages, Se is also excreted in the urine as trimethylselenonium (Suzuki, 2005). Selenosugar B is believed to be produced from the catabolism of selenoproteins in cells throughout the

### Table 3. Selenium absorption, Se excretion in urine, fractional Se clearance from plasma, and hepatic Se concentration in adult cats and dogs fed a control diet or the control diet supplemented with Na2SeO3 or organic Se1 for 21 d

<table>
<thead>
<tr>
<th>Species</th>
<th>Diet</th>
<th>Total Se, μg/kg DM</th>
<th>Apparent fecal Se absorption, μg/kg BW/d</th>
<th>%</th>
<th>Urinary Se excretion, μg/kg BW/d</th>
<th>Se clearance, %</th>
<th>Hepatic Se concentration, μg Se/g DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cats</td>
<td>Control</td>
<td>0.6</td>
<td>2.3 ± 0.5</td>
<td>21.2 ± 5.2</td>
<td>2.0 ± 0.3b</td>
<td>0.19 ± 0.02b</td>
<td>1.37 ± 0.05b</td>
</tr>
<tr>
<td></td>
<td>+ Na2SeO3</td>
<td>8.4</td>
<td>98 ± 5</td>
<td>70.2 ± 2.9</td>
<td>111 ± 3b</td>
<td>4.35 ± 0.36b</td>
<td>4.24 ± 0.59b</td>
</tr>
<tr>
<td></td>
<td>+ Organic</td>
<td>8.6</td>
<td>108 ± 2</td>
<td>69.6 ± 1.3</td>
<td>101 ± 4b</td>
<td>3.18 ± 0.13c</td>
<td>4.79 ± 0.37b</td>
</tr>
<tr>
<td>Dogs</td>
<td>Control</td>
<td>0.6</td>
<td>3.0 ± 0.3</td>
<td>37.4 ± 4.6</td>
<td>2.3 ± 0.2a</td>
<td>0.17 ± 0.02a</td>
<td>1.37 ± 0.13a</td>
</tr>
<tr>
<td></td>
<td>+ Na2SeO3</td>
<td>8.7</td>
<td>95 ± 3</td>
<td>83.0 ± 1.4</td>
<td>72 ± 5c</td>
<td>2.73 ± 0.25c</td>
<td>7.53 ± 0.38c</td>
</tr>
<tr>
<td></td>
<td>+ Organic</td>
<td>9.9</td>
<td>101 ± 2</td>
<td>78.5 ± 1.7</td>
<td>64 ± 3c</td>
<td>2.42 ± 0.35c</td>
<td>7.93 ± 0.53c</td>
</tr>
</tbody>
</table>

*Means with different superscripts within row are different (P < 0.05).

body and released into the blood stream to be excreted
in the urine (Suzuki, 2005). To the knowledge of the
authors, there is no information in the literature whether
cats and dogs metabolize Se to selenosugars.

At the start of the study, there was no difference
between the groups within species in plasma Se
concentration and GPx activity. Plasma Se concentrations
of the cats fed the control and supplemented diets reported
here (5.17 to 9.13 μmol/L, respectively) are similar to
plasma (3.95 to 8.70 μmol/L, respectively; Foster et al.,
2001) and serum (3.60 to 10.09 μmol/L, respectively;
Forrer et al., 1991) Se concentrations reported in cats
fed commercial feline diets. Todd et al. (2011) fed adult
cats diets supplemented with Na2SeO3 and organic Se to
contain up to 2.2 μg Se/g DM and reported that plasma
Se concentrations ranged between 4.5 and 8.4 μmol/L.
To the knowledge of the authors, there are no reports of
chronic or acute selenosis in cats in the literature, from
which a plasma Se concentration under selenonotic
conditions can be obtained. In the present study, the
cats maintained plasma Se concentrations within the
ranges normally observed when fed experimental
diets containing lower dietary Se concentrations and
commercial petfoods. The plasma Se concentration of
the dogs fed the control diet (3.42 μmol/L) was also
within the range of serum Se concentrations normally
observed for dogs fed commercial diets (1.90 to 4.31
μmol/L; Forrer et al., 1991). However, the dogs fed the
supplemented diets had average plasma Se concentrations
of 7.21 and 7.81 μmol/L, which are values well outside
the latter range, indicating that these animal were unable
to maintain plasma Se concentrations within the range.
Wedekind et al. (2002) and Yu et al. (2006) reported
that dogs fed 7.2 μg Se/g from selenoferous corn and 10
μg Se/g as added Na2SeO3 were toxic. In cats, Smith et al.
(1937) supplemented a diet (raw lean beef and milk) with
Na2SeO3 or subcutaneously injected cats with Na2SeO3
to provide 0.02. 0.10, or 0.25 mg Se/(kg BW·d) for up to
188 d. Although the experimental details of this study are
minimal and no details regarding signs of selenosis were
reported, reassessment of the data of the cats completing the
study showed a linear increase in urinary Se excretion per
unit BW with increasing dietary Se intake.

The low fractional clearance of Se in cats and dogs
fed the control diet indicates that homeostatic Se control
mechanisms effectively conserved Se. In animals fed
very high concentrations of Se, dimethylselenol can be
formed, which is excreted through the lungs and results
in a garlic odor. In the present study, no characteristic
garlic smell on the breath of the cats and dogs was
observed. The Se clearance was less in dogs compared
with cats and greater concentrations of Se were found
in the liver of dogs. The latter was probably not caused
by the hepatic Se concentrations at the start of the study
as the feline and canine control groups had an identical
average hepatic Se concentration. López-Alonso et al.
(2007) reported average liver Se concentrations in 59
Spanish domestic dogs to be 0.686 mg/kg. Samples were
obtained at euthanasia or routine necropsy in veterinary
hospitals, with the greatest recorded value of 2.55 mg
Se/kg. The average liver Se concentration found in dogs
in the control group was greater (1.37 μg Se/g) in the
present study, but within the range recorded by López-
Alonso et al. (2007).

For the Na2SeO3 and organic Se
supplemented dogs in the present study, Se retention
was 20 and 33 μg/(kg BW·d), respectively, which is in
line with the Se clearance and hepatic Se concentrations.
The data showed that cats did not retain as much Se as
dogs. In fact, for the Na2SeO3 supplemented cats, the
excretion recorded was greater than the intake, a result
which is difficult to explain. For cats fed the organic Se
supplement diet, little difference was found between the
intake and excretion [7 μg/(kg BW·d)]. The hepatic Se
clearance, however, was not different between the
cats on the supplemented diets.

There is some indication that cats can tolerate greater
concentrations of dietary Se. Wedekind et al. (2002) and
Yu et al. (2006) reported the initial signs indicative of the
onset of selenosis, decreased feed intake, and reduced
hair growth in studies where adult dogs were fed 5 mg
Se/kg diet in the form of selenomethionine for 26 wk.
In comparison, Wedekind et al. (2003) fed adult cats up to
10 mg Se/kg diet in the form of selenomethionine for 26
wk and observed no clinical signs of Se toxicity (e.g.,
reduced food intake, reduced hair growth, or changes in
serum chemistry profile). Sixty years earlier, Rhian and
Moxon (1943) studied chronic Se poisoning in dogs and
fed up to 20 mg/kg Se from Na2SeO3 and seleniferous
corn (Rhian and Moxon, 1943) and reported reduced food
intake, severe BW loss or growth retardation, coarse and
loose hair, abnormalities of the nervous system, ascites,
and liver necrosis. Rhian and Moxon (1943) concluded
that dogs fed 7.2 μg Se/g from seleniferous corn and 10
μg Se/g as added Na2SeO3 were toxic. In cats, Smith et al.
(1937) supplemented a diet (raw lean beef and milk) with
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minimal and no details regarding signs of selenosis were
reported, reassessment of the data of the cats completing the
study showed a linear increase in urinary Se excretion per
unit BW with increasing dietary Se intake.

It is attractive to postulate that the differences
observed in the response of cats to greater dietary
concentrations of Na2SeO3 and organic Se compared
with dogs (and potentially other animals) may be related
to the unique sulphur-containing AA metabolism of
cats. Cysteine dioxygenase and cysteine sulfuric acid
decarboxylase activity in cats are low and most of sulfur
AA catabolism does not occur via the cysteine sulfuric
acid but via the desulfurization pathway. Yielding energy
from cysteine has been more advantageous for cats
throughout evolution as taurine synthesis was ensured by
As such, the greater dietary intake of cysteine (and selenocysteine) may have resulted in the development of a highly effective pathway to eliminate both H₂S and H₂Se from the body. Although likely, at present it is unknown if cats and dogs synthesize selenosugars. If so, it would be interesting to study Se speciation in the urine of cats and dogs to provide more insight into the Se metabolism of both species.

The absence of reports of chronic selenosis in the literature of cats and dogs fed commercial diets, which can contain up to 6.1 μg Se/g DM (Mumma et al., 1986; Simcock et al., 2005), can be explained in part by a reduced Se bioavailability in commercial pet foods, as well as a greater renal Se clearance by cats. The present study showed that cats were able to tolerate greater dietary Se concentrations by more efficiently excreting excess Se in the urine, instead of storing Se in the liver. Dogs had a decreased Se clearance compared with cats, and, as canine foods have been shown to contain less Se compared with feline commercial foods (Simcock et al., 2005), the absorbable Se concentrations in commercial petfoods would likely be too low to cause chronic selenosis. If Se absorption of a 6.1 μg Se/g DM commercial petfood was similar to Se in Na₂SeO₃ and the organic source (approximately 80%), it could result in clinical signs associated with selenosis. The reason for the reduced bioavailability of Se in commercial petfoods compared with unprocessed Na₂SeO₃ and the organic Se source should be investigated to understand factors affecting Se bioavailability to ensure safe consumption of petfoods by especially dogs in the future.

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