Sodium Requirement of Adult Cats for Maintenance Based on Plasma Aldosterone Concentration¹,²

Shiguang Yu and James G. Morris³

Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis, CA 95616

ABSTRACT The sodium requirement of adult cats for maintenance was determined using a randomized block design of eight dietary sodium treatments (0.1, 0.4, 0.5, 0.66, 0.8, 1.2, 1.6 or 2.0 g Na/kg in a casein-lactalbumin-based purified diet) administered for periods of 4 wk. A total of 35 adult specific-pathogen-free domestic shorthaired cats (26 males and 9 females, 1.5–3 yr of age) was given an equilibration diet (2 g Na/kg) for 14 d before assignment (or reassignment) to the treatments. A total of 12 cats (8 males, 4 females) was randomly assigned to the lowest six levels of sodium, and four cats to the highest two sodium levels. Cats consuming the diet containing 0.1 g Na/kg had significantly elevated aldosterone concentration in plasma, and packed cell volume. In addition, these cats exhibited anorexia, body weight loss, reduced urinary specific gravity and sodium excretion, and had a negative sodium balance. However, adult cats did not develop polydypsia and polyuria reported in sodium-deficient kittens. Cats given the diet containing 0.66 g Na/kg did not have an increased packed cell volume, but aldosterone concentration in the plasma was significantly elevated. However, cats given diets containing ≥0.8 g Na/kg had plasma aldosterone concentrations ≤0.7 nmol/L (reference value for sodium-replete cats) and normal packed-cell volumes. A minimal sodium requirement of adult cats for maintenance of 0.8 g Na/kg diet (energy density = 22 kJ/g diet) or 0.4 mmol Na · kg body weight⁻¹ · d⁻¹ is proposed. J. Nutr. 129: 419–423, 1999.

KEY WORDS: • cats • sodium • aldosterone • requirement

In the absence of data from cats, a sodium requirement based on other mammals of 0.5 Na/kg diet was proposed by the National Research Council (1986) for growing kittens. Yu and Morris (1997) reported that this dietary concentration was inadequate and proposed a minimal sodium requirement of 1.6 g Na/kg diet (energy density = 22 kJ ME/g diet) for growing kittens, a value about three times that suggested by the National Research Council. The objective of this study was to determine the minimal sodium requirement of adult cats for maintenance based on changes in physiological measurements including aldosterone concentration in plasma.

MATERIALS AND METHODS

The experimental protocol adhered to the Guide for the Care and Use of Laboratory Animals (National Research Council 1985) and was approved by the Animal Use and Care Administrative Advisory Committee of the University of California at Davis.

Animals and their management. A total of 35 (26 males and 9 females) specific-pathogen-free domestic short-hair adult cats (1.5–3 yr of age) from the California Nutritional and Care Center of the University of California, Davis, was used. The cats were individually housed in stainless steel metabolic cages (60 × 60 × 60 cm) in temperature-controlled rooms (21 ± 2°C) with a light/dark cycle of 14:10. They had free access to the experimental diets (provided in stainless steel food bowls) and deionized water in plastic bottles.

Diets. Experimental diets were made by adding various amounts of sodium (as NaCl that does not alter acid-base status of cats) to a casein-lactalbumin-based purified basal diet at the expense of dextrose. The basal mixture contained (g/kg): casein (New Zealand Milk Products, Petaluma, CA), 222.5; lactalbumin (New Zealand Milk Products), 222.5; rendered animal tallow (Florisil Tallow, Dixon, CA), 305; sucrose, 100; corn starch (Melijel, Bridgewater, NJ), 90.5; taurine (Ajinomoto USA, Raleigh, NC), 1.5; choline chloride (International Mineral and Chemical, Terre Haute, IN), 3.0; vitamin mixture, 10 (Williams et al. 1987); and mineral mixture, 42.577 (containing CaHPO₄, 19.529; MgSO₄, 2.25; KCl, 10; K,HPO₄, 4.5; CaCO₃, 5.5; MnSO₄·H₂O, 0.019; FeSO₄·7H₂O, 0.47; NaF, 0.007; KI, 0.0015; ZnSO₄·7H₂O, 0.223; CuSO₄·5H₂O, 0.002; CrCl₃·6H₂O, 0.10; vitamin mixture, 10 (Williams et al. 1987); and mineral mixture, 42.577). The basal diet contained all nutrients (including chlorine) in amounts sufficient to meet the requirements of adult cats, with the exception of sodium. Experimental diets containing 0.10, 0.40, 0.50, 0.66, 0.80, 1.09, 1.50, 1.85, 2.87, 3.89, and 4.90 g NaCl/kg diet. Dietary sodium concentrations were verified by atomic absorption spectrophotometry. Diets were stored at 4°C after preparation until they were fed.

Experimental design. A stratified random design with sex and body weight as criteria of classification was employed. A total of 12 cats (8 males and 4 females) was tested at each of the six lowest dietary sodium concentrations, and 4 cats at each of the two highest concentrations of sodium (2 males and 2 females for the diet containing 1.6 g Na/kg diet, and 3 males and 1 female for dietary sodium
concentration of 2.0 g Na/kg diet). Because 80 adult cats and metabolism cages were not available, the 35 cats in this study received more than one dietary treatment. As cats entered the study or came from a previous test diet, they underwent an equilibration period of 14 d, during which time they were given a sodium-replete purified diet (2 g Na/kg diet) and tap water. At the end of this period (d 0 or wk 0) and the end (d 28 or wk 4) of the experiment for each dietary treatment. To estimate the minimal sodium requirement, break points from the broken lines constructed using plasma aldosterone concentration (d 28) and packed cell volume (d 28) as a function of dietary sodium concentration were calculated using a nonlinear least square method (Robbins 1986). Pooled SEM for each variable is given if the variances between dietary groups were homogeneous. Otherwise, a separate SEM is presented for each dietary group. Probability levels \( P < 0.05 \) were considered significant for all tests.

RESULTS

Body weight, food and water intakes. Cats lost an average of 0.1 kg body weight over a 4-wk period when given the diet containing 0.1 g Na/kg diet, but maintained body weight during the experimental period when the dietary sodium concentration was \( \geq 0.4 \) g Na/kg diet (Table 1). Food intake was significantly reduced when dietary sodium concentration was \( \geq 0.4 \) g Na/kg diet, while both absolute water intake and the ratio of water intake to food intake were not affected by the sodium concentrations in the diets tested (Table 1). As expected, female cats had lower body weights and food and water intakes than male cats (separate data for male and female cats are not shown).

Urine production and urinary specific gravity. At the end of the experiment, dietary sodium concentration did not affect the quantity of urine produced, but urinary specific gravity was significantly lower in the adult cats consuming diets containing \( \geq 0.4 \) g Na/kg diet (Table 1). The cats fed the diet containing 0.5 g Na/kg diet had a lower urine production and greater urinary specific gravity at d 28 when compared to d 0 (Table 1).

Sodium and aldosterone concentrations in the plasma and packed cell volume. Sodium concentration in the plasma was

### Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>0.10</th>
<th>0.40</th>
<th>0.50</th>
<th>0.66</th>
<th>0.80</th>
<th>1.20</th>
<th>1.60</th>
<th>2.00</th>
<th>Pooled SEM</th>
<th>ANOVA²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight, kg</td>
<td>3.90</td>
<td>3.91</td>
<td>3.95</td>
<td>3.98</td>
<td>4.00</td>
<td>3.95</td>
<td>3.89</td>
<td>3.73</td>
<td>0.063</td>
<td>S</td>
</tr>
<tr>
<td>Food intake, g/d</td>
<td>3.00</td>
<td>3.80</td>
<td>3.92</td>
<td>3.87</td>
<td>3.98</td>
<td>4.02</td>
<td>3.93</td>
<td>3.82</td>
<td>3.64</td>
<td>0.067</td>
</tr>
<tr>
<td>Water intake, g/d</td>
<td>1.0</td>
<td>1.8</td>
<td>1.3</td>
<td>1.6</td>
<td>1.8</td>
<td>1.6</td>
<td>1.5</td>
<td>1.5</td>
<td>0.061</td>
<td>D</td>
</tr>
<tr>
<td>Specific gravity, kg/L</td>
<td>1.05</td>
<td>1.051</td>
<td>1.045</td>
<td>1.051</td>
<td>1.051</td>
<td>1.051</td>
<td>1.054</td>
<td>1.054</td>
<td>0.001</td>
<td>S</td>
</tr>
<tr>
<td>Plasma Na, mmol/L</td>
<td>137.1</td>
<td>139.9</td>
<td>133.4</td>
<td>136.8</td>
<td>136.5</td>
<td>136.9</td>
<td>135.8</td>
<td>1.91</td>
<td>1.91</td>
<td>1.91</td>
</tr>
</tbody>
</table>

1 Values are means for the cats in each dietary group (n = 12; 8 males and 4 females for dietary Na concentrations \( \leq 1.20 \) g Na/kg diet; n = 4; 2 males and 2 females for 1.60 g Na/kg diet) and 3 males and 1 female for 2.00 g Na/kg diet). All cats were given a purified diet containing 2.0 g Na/kg diet during wk 0.
2 Two-way ANOVA significance (P < 0.05) with diet (D) and sex (S) as main effects.
3 Mean of wk 0 or 4 of the experiment.
4 Significantly different from wk 0 or d 0 (P < 0.05 paired Student’s t test).
5 Kruskal-Wallis test, S: significant sex effect.
6 Significantly different from wk 0 or d 0 (P < 0.05 paired Student’s t test).
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not affected by the dietary treatment (Table 1). However, both aldosterone concentration in the plasma (Fig. 1) and packed cell volume (Fig. 2) were significantly elevated in cats at d 28 when dietary sodium concentration was ≤0.5 g Na/kg diet. There was no difference in packed cell volume between d 21 and 28 for any groups. Plasma aldosterone concentrations of cats given diets containing ≥0.5 g Na/kg diet were not different at d 21 and 28 for each dietary treatment, indicating that these cats were stable in aldosterone secretion. However, aldosterone concentration tended (P > 0.05) to be higher at d 28 than d 21 when dietary sodium concentrations were 0.4 and 0.1 g Na/kg diet (Fig. 1), indicating an exacerbation of sodium deficiency. Calculated break points for plasma aldosterone concentration (d 28) and packed cell volume (d 28) were 0.57 and 0.69 g Na/kg diet, respectively.

Sodium balance. Sodium output in feces (about 0.5 mmol/d) was not significantly affected by dietary sodium concentrations ranging from 0.1 to 2.0 g Na/kg diet. Sodium output in urine was directly related to dietary sodium concentration. Cats given diets containing ≤0.4 g Na/kg had negative sodium balances [sodium intake − (fecal sodium output + urinary sodium output)].

DISCUSSION

Mineral requirements based on balance studies have the inherent problem that small unaccounted losses lead to cumulative errors and an underestimation of the actual requirement. In contrast, plasma aldosterone concentration is responsive to sodium balance and extracellular fluid volume of the animal and is more likely to reflect the actual status of the animal. Therefore, we regard the setting of a requirement based on sodium balance in the presence of an elevated plasma aldosterone concentration as irresponsible. Plasma aldosterone has been directly used to estimate the sodium requirements of growing kittens (Yu and Morris 1997) and indirectly the sodium requirements of cattle (Morris and Gartner 1971) and sheep (Morris and Petersen 1975) through the effect of aldosterone on the parotid salivary gland secretion of sodium and potassium. Use of aldosterone requires that a “normal” value for sodium-replete animals be defined, which we have done (Yu and Morris 1998). To ensure that the cats were sodium-replete before being given the experimental diets, we used an initial equilibration diet that contained 2 g Na/kg of dry matter. This concentration of sodium exceeds the requirement for growing kittens, but does not contain a large excess. Also the concentration conforms to the AAFCO (1998) adult minimal maintenance recommendation for sodium but is less than that present in most commercial cat foods. A random survey gave a mean value of 4.5 ± 0.2 g Na/kg, (n = 6). It is also less than the average daily sodium intake of American adults (3.28 g/day, Nutrition.org 1998) which corresponds to a dietary concentration of about 6.5 g Na/kg of dry matter.

Plasma aldosterone and packed cell volume significantly increased in cats when the concentration of sodium in the diet decreased and were the most sensitive indices of sodium status. These measurements were also the most sensitive indices of sodium deficiency in growing kittens (Yu and Morris 1997). Sodium-deficient adult cats also exhibited anorexia, body weight loss, hyponatremia, decreased urinary specific gravity and negative sodium balance, but not the polydypsia and polyuria observed in growing kittens.

Water intake is controlled by the thirst center in the brain that responds to volume and pressure changes of the extracellular fluid (Koeppen and Stanton 1996), and is stimulated by elevated angiotensin II concentration in the plasma (McKinley et al. 1992). Water intake of most mammals is closely correlated with food intake. Cats consuming dry cat food drink about 1.5–2.0 mL of water per gram of food consumed (Burger et al. 1980, Kane et al. 1981). Water intake was about 1.7 g/g of food intake for cats given the diet containing ≤0.5 g Na/kg even though plasma aldosterone concentration and packed cell volume were significantly elevated. Increased aldosterone concentration was probably the result of activation of the renin–angiotensin–aldosterone system (Koeppen and Stanton 1996).
1996) while elevated packed cell volume probably reflected decreased volume of plasma and extracellular fluid though we did not measure plasma osmolality. Apparently, the reduced volume of extracellular fluid of cats fed the sodium-deficient diet (0.1 g Na/kg) did not stimulate water intake.

Independent of sodium intakes, adult cats had fecal sodium output of about 0.5 mmol/d which was not significantly different due to treatment (Table 2). These losses were similar to the fecal sodium losses of growing kittens (0.6 mmol/d) fed similar purified diets (Yu and Morris 1997), suggesting the obligate fecal sodium loss of cats fed the purified diets. Finco et al. (1989) reported fecal sodium losses of 1 and 1.5 mmol/d in male adult cats fed commercial dry cat food, when sodium intakes were 17 and 39 mmol/d, respectively. Compared to purified diets, commercial dry cat food diets usually have higher amounts of indigestible matter that resulting in greater fecal volume, fecal water and hence fecal sodium excretion, resulting in reduced apparent sodium absorption (Partridge 1975).

Definition of a nutrient requirement requires that it be based on one or more physiological responses, and generally, the response with the highest requirement determines the requirement. Of the measurements taken, plasma aldosterone concentration and packed cell volume were the most responsive to dietary sodium concentration. Similar packed cell volumes and plasma aldosterone concentrations between d 21 and 28 for each dietary treatment (≥0.5 g Na/kg diet for aldosterone) suggested stabilized responses to dietary sodium concentrations. These variables were used to construct broken lines as a function of dietary sodium concentration. The estimated break points for plasma aldosterone concentration and packed cell volume at d 28 were 0.57 g Na/kg (asymptotic SEM = 0.012; 95% CI 0.54, 0.59) and 0.69 g Na/kg diet (asymptotic SEM <0.001; 95% CI 0.69, 0.69), respectively.

Packed cell volume of cats given the diet containing 0.66 g of Na/kg was 1.12 ± 0.2 mmol/L, which is above the reference level we reported in sodium-replete adult cats (0.7 mmol/L, n = 148). Only when cats were given diets containing ≥0.8 g of Na/kg, were plasma aldosterone concentrations ≥0.7 mmol/L. Therefore, if plasma aldosterone concentration is taken as the criterion of sodium adequacy, a minimal dietary sodium concentration of 0.8 g of Na/kg diet (energy density = 22 kJ/g diet) is necessary. As the mean body weight and food intake of cats fed diets containing ≥0.8 g Na/kg were 3.85 kg (d 28) and 42 g/d (wk 4, Table 1), the minimal sodium requirement of adult cats for maintenance is equivalent to 0.4 mmol Na·kg body weight−1·day−1.

Cats have a higher sodium requirement for maintenance than the value of 0.5 g of Na/kg diet proposed by the National Research Council (1986) based on the requirements of other mammals. As both sodium-depleted and repleted cats do not exhibit sodium appetite (Yu et al. 1997) and will not select foods on the basis of their sodium content to correct a deficiency, it is essential that feline diets contain adequate concentrations of sodium.

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LITERATURE CITED


