British Journal of Nutrition (2014), **111**, 785–797
doi:10.1017/S0007114513003279

Effects of potassium chloride and potassium bicarbonate in the diet on urinary pH and mineral excretion of adult cats

Nadine Paßlack[1]*, Thomas Brenten[2], Konrad Neumann[3] and Jürgen Zentek[1]

[1]*Department of Veterinary Medicine, Institute of Animal Nutrition, Freie Universität Berlin, Königin-Luise-Straße 49, 14195 Berlin, Germany*
[2]*Mars GmbH, Eitzer Straße 215, 27283 Verden, Germany*
[3]*Institute of Biometry and Clinical Epidemiology, Charité – Universitätsmedizin Berlin, Hindenburgdamm 30, 12203 Berlin, Germany*

(Submitted 2 January 2013 – Final revision received 22 August 2013 – Accepted 29 August 2013 – First published online 14 November 2013)

Abstract

Low dietary K levels have been associated with increasing renal Ca excretion in humans, indicating a higher risk of calcium oxalate (CaOx) urolith formation. Therefore, the present study aimed to investigate whether dietary K also affects the urine composition of cats. A total of eight adult cats were fed diets containing 0·31 % native K and 0·50, 0·75 and 1·00 % K from KCl or $KHCO_3$ and were evaluated for the effects of dietary K. High dietary K levels were found to elevate urinary K concentrations ($P<0·001$). Renal Ca excretion was higher in cats fed the KCl diets than in those fed the $KHCO_3$ diets ($P=0·026$), while urinary oxalate concentrations were generally lower in cats fed the KCl diets and only dependent on dietary K levels in cats fed the $KHCO_3$ diets ($P<0·05$). Fasting urine pH increased with higher dietary K levels ($P=0·022$), reaching values of 6·38 (1·00 % KCl) and 7·65 (1·00 % $KHCO_3$). K retention was markedly negative after feeding the cats with the basal diet (−197 mg/d) and the 0·50 % KCl diet (−131 mg/d), while the cats tended to maintain their balance on being fed the highest-KCl diet (−23·3 mg/d). In contrast, K from $KHCO_3$ was more efficiently retained ($P=0·018$), with K retention being between −82·5 and 52·5 mg/d. In conclusion, the dietary inclusion of $KHCO_3$ instead of KCl as K source could be beneficial for the prevention of CaOx urolith formation in cats, since there is an association between a lower renal Ca excretion and a generally higher urine pH. The utilisation of K is distinctly influenced by the K salt, which may be especially practically relevant when using diets with low K levels.

Key words: Cats: Dietary potassium: Urine composition: Urine pH

Several dietary risk factors have been discussed in the context of the formation of Ca oxalate (CaOx) uroliths in cats. These include factors that influence renal Ca and oxalate (Ox) excretion, urine pH and urinary concentrations of crystallisation inhibitors, e.g. citrate and glycosaminoglycans[1]. Besides the direct impact of dietary Ca[2,3] or exogenous and endogenous Ox[1,4,5] on urine composition, indirect effects of other dietary components, such as Mg, P and K, on the intestinal absorption and renal excretion of Ca and Ox have also been reported[1]. For instance, Ca and Mg can form a complex with free Ox in the intestine[6–8], resulting in reduced intestinal absorption and renal excretion of Ox[9–11], which has been demonstrated not only in human subjects, but also in dogs and rats. Moreover, other factors such as dietary P and fat can interact with Ca in the intestine and thereby prevent its complexation with Ox, which indirectly improves the intestinal absorption of Ox[12,13]. Furthermore, dietary K seems to play an interesting role in the formation of CaOx uroliths. Data from human studies demonstrate that high levels of dietary K reduce renal Ca excretion and thereby decrease the risk of the formation of Ca-containing crystals and stones[14,15]. In cats, low dietary K concentrations are also considered to be a risk factor for the formation of CaOx uroliths[2]. However, this observation has been made based on only epidemiological evaluations and, to our knowledge, no detailed experimental studies have been carried out.

It can be suggested that not only K concentrations, but also the anions in K salts affect the urine composition of cats. It is known that the inclusion of potassium citrate in diets can help to prevent the formation of CaOx uroliths by an alkalising effect on the urine and a resulting higher solubility of the crystals by modifying the urine pH and the increased fraction of Ca bound to the non-metabolisable citrate anions[16,17]. The physiological explanation for the impact of dietary K salts

Abbreviations: BW, body weight; CaOx, calcium oxalate; ME, metabolisable energy; Ox, oxalate.

* **Corresponding author:** Dr N. Paßlack, fax +49 3083855938, email nadine.passlack@fu-berlin.de

British Journal of Nutrition

on urine pH can be found in the respective counter-ion. If dietary K is associated with a metabolisable organic anion (e.g. HCO_3^-), K will be renally excreted and the organic anion will be oxidised to CO_2 and H_2O[18]. To maintain the acid–base balance, HCO_3^- will be produced and also be excreted in the urine, which leads to an increase in urine pH. In contrast, if dietary K is associated with a non-metabolisable anion (e. g. Cl^-), both K and the organic anion will be renally excreted[18], which may lead to the acidification of the urine. Therefore, dietary K salts, such as KCl and $KHCO_3$, may affect the acid–base balance and thereby urinary proton and base excretion in cats. Until now, to our knowledge, no detailed studies have been carried out to evaluate the effects of dietary K levels and salts on urine composition in cats. However, it is known that in cats[19], as in other species, renal K excretion increases with increasing levels of K in the diet[20].

Considering the above-mentioned observations of the effects of dietary K on urinary Ca concentrations in humans and that the alkalisation or acidification of the urine affects the solubility of urinary minerals[21], the present study aimed to investigate the effects of varying levels of KCl and $KHCO_3$ in a diet on urine pH as well as on urinary mineral and Ox excretion in cats. It was hypothesised that the expected effects are dependent not only on dietary K concentrations, but also on the anions present in the K salts. To evaluate the regulatory processes involved in mineral metabolism, the apparent digestibility of the dietary minerals and the concentrations of minerals in the blood were evaluated.

Materials and methods

Animal study

The experimental protocol was approved by the Animal Welfare Committee (Landesamt für Gesundheit und Soziales, Berlin, Germany, G 0157/10). A total of eight adult cats (European shorthair, four males and four females, 38 months old (mean; SEM: 2·00)) were included in the present study. The cats were housed in a room under constant light (12 h light–12 h darkness) and temperature (21°C) conditions. The cats were fed the experimental diets individually at 07.00 and 12.00 hours, and daily feed intake was recorded for each cat. The cats were housed in groups during a 9 d adaptation period and in metabolism cages during an 8 d collection period. The metabolism cages contained purpose-built cat litter boxes with plastic pellets as litter and connected urine collection containers to separate the urine from the faeces. Blood was collected from the cats in the morning (fasting) of the last day of each feeding period.

Diets and nutrient analysis

The cats were fed seven dry extruded diets during seven feeding periods. During each 17 d feeding period, all the cats were fed the same diet at the same time. The diets differed in the source of the added K (native concentration in the diet, KCl and $KHCO_3$) and in total K concentrations (0·31 % native K in DM and intended 0·50, 0·75 and 1·00 % K). The experimental diets were fed in the following order: diet containing only native K; diets with the added KCl; diets with the added $KHCO_3$. The results of the nutrient analysis of the diets are given in Table 1.

The concentrations of crude nutrients were measured according to the Weende analysis of feed[22]. This method was modified for the measurement of crude fat. In the first step, fat in the feed sample was extracted for 3 h using petroleum diethyl ether. The petroleum diethyl ether was consecutively vaporised for 1 h and 100°C in a compartment dryer. After cooling in a desiccator, the amount of crude fat was calculated by weighing the specimen cup with the feed sample before and after fat extraction. The measurement of the concentrations of minerals in the diets was carried out following the method used for the measurement of those in faecal samples (discussed below).

The diets were formulated to fulfil the dietary recommendations for adult cats[23]. The concentrations of K in the basal diet (3·09 g/kg DM) and in the lowest-KCl (4·68 g/kg DM) and lowest-$KHCO_3$ (5·06 g/kg DM) diets were below or close to the recommendations of 5·2 g/kg DM (with 16·7 MJ metabolisable energy (ME)/kg DM), which was due to the

Table 1. Results of nutrient analysis of the experimental diets*

	Native K (%)†	K (%) added as KCl			K (%) added as $KHCO_3$		
Analysed composition	0·31	0·50	0·75	1·00	0·50	0·75	1·00
DM (g/kg)	930	930	926	935	919	925	927
Crude protein (g/kg DM)	307	310	311	317	315	311	314
Crude fat (g/kg DM)	101	117	103	103	114	113	109
Crude fibre (g/kg DM)	54·0	40·3	34·2	36·6	40·6	48·4	48·5
Crude ash (g/kg DM)	69·3	72·3	77·3	81·5	73·9	77·4	82·5
Ca (g/kg DM)	14·6	14·5	14·8	14·0	14·6	14·7	14·1
P (g/kg DM)	10·5	10·6	10·4	10·6	10·6	10·2	10·2
Na (g/kg DM)	8·03	7·94	8·04	8·24	8·46	8·31	8·60
K (g/kg DM)	3·09	4·68	7·45	9·25	5·06	7·32	9·53
Cl (g/kg DM)	8·99	10·8	15·1	12·4	11·4	9·89	11·8
Mg (g/kg DM)	0·87	0·90	0·90	0·89	0·88	0·88	0·88

* Ingredient list: poultry meal; rice; wheat whole grain; poultry fat; dried beet pulp; salt; yeast; marigold meal; minerals; vitamins.
† Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.

ingredients of the basal diet, but it allowed for the validation of the actual data on K requirements in cats[23].

Sample collection and pH measurements

The urine and faeces of the cats were collected at 07.00 and 12.00 hours. The measurement of urine pH was carried out on the first 2 d of the 8 d collection period. Therefore, the urine collection containers were treated with three drops of chlorhexidine–digluconate to prevent the overgrowth of bacteria in the urine. Urine pH was measured using the SevenMulti pH meter (Mettler-Toledo GmbH) when the cats were fasting (07.00 hours) and postprandially (12.00 hours). On the remaining 6 d of the collection period, the urine collection containers in the metabolism cages were treated with 2 ml of glacial acetic acid for the conservation of the urine. All the urine samples of each cat were filled into one large collection container and stored in a freezer (−20°C). Therefore, it was possible to determine the total urine volume during the collection period for each cat. The faeces of the cats were collected during the entire 8 d collection period and also stored in a freezer at −20°C until further analysis.

Preparation and analysis of the samples

The urine and faecal samples were prepared and analysed as described previously[3]. In short, the urine samples were acidified with hydrochloric acid (37 %) to adjust the pH to 2 (SevenMulti pH meter, Mettler-Toledo GmbH). Subsequently, the samples were filtered (Syringe Filter, Bulk, surfactant-free cellulose acetate, 0·2 μm, 25 mm non-sterile, Thermo Scientific) and stored at −80°C until the determination of the concentrations of urea, creatinine, anions and cations. The concentrations of urea and creatinine were measured using a HPLC method on an Agilent 1100 system with a UV detector (Agilent Technologies). The concentrations of urinary anions (sulphate and P as major anions; Ox and citrate as minor anions) were measured using an ion exchange HPLC system (Dionex DX-500; Dionex Corporation). The concentrations of urinary cations (Na, K and ammonium as major cations; Mg and Ca as minor cations) were measured using also an ion exchange HPLC system (Dionex DX-120; Dionex Corporation). The data obtained for urea, creatinine, anions and cations were analysed using Chromeleon Client, version 6.80 SP2 (Dionex Corporation).

The faecal samples were freeze-dried, ashed and subsequently mixed with hydrochloric acid (37 %) and distilled water in a beaker (except for the samples used for the determination of Cl concentrations). The beaker was covered and put into a warm sand bath for 50 min at 210–220°C. After cooling, the samples were transferred into flasks filled with distilled water using an ash-free filter. After this procedure, the concentrations of minerals in the samples were determined. For the determination of the concentrations of Cl, the faecal samples were also freeze-dried. Later, 2·5 g of these samples were weighed and transferred into a 50 ml graduated flask, mixed with 25 ml of distilled water and incubated in a shaker for 30 min at room temperature. Then, 1 ml of zinc sulphate × $7H_2O$ (35·8765 g/100 ml distilled water) and 1 ml of potassium hexacyanoferrate (15 g/100 ml distilled water) were added, and the graduated flask was filled with distilled water. After shaking, 10 ml of this solution were transferred into a 15 ml tube and centrifuged for 10 min at room temperature and 2000 ***g*** (Heraeus Multifuge 3 S-R, Thermo Scientific). Finally, the samples were filtered through 0·45 μm cellulose acetate membranes (VWR International), and the concentrations of Cl were measured using an ion exchange HPLC system (Dionex DX-500; Dionex Corporation).

The concentrations of P in the faecal samples were measured spectrophotometrically[24] (Ultrospec 2000, Pharmacia Biotech). The concentrations of Ca, Na, K and Mg in the faecal samples were measured by atomic absorption spectrometry (flame atomic absorption spectrometer type vario 6 with an autosampler AS 52, Analytik Jena AG).

Blood samples were stored at room temperature for 1 h before further preparation. While the EDTA tubes were stored in the refrigerator at 4°C, the serum tubes were centrifuged at 4°C and 1811 ***g*** for 10 min (Heraeus Megafuge 1.0R, Thermo Scientific). Serum was pipetted and stored in a 2 ml tube in the refrigerator before sending all the blood samples to an external laboratory (Laboklin, Bad Kissingen, Germany) or to the Clinic of Small Animals, Freie Universität Berlin (cooled transport). The concentrations of minerals (Ca, P, Na, K and Cl) in the blood samples were determined photometrically, and the complete blood count was carried out using an automatic method.

Statistical analysis

Data were analysed with SPSS 19 (SPSS, Inc.). For each outcome variable, a repeated-measures ANOVA with the within-subject factors K concentration and K salt (KCl and $KHCO_3$) was carried out. Polynomial contrasts (linear and quadratic) were separately calculated for the KCl and $KHCO_3$ treatments and for the arithmetic means of both treatments at K levels. In case of a significant interaction between dietary K salt and dietary K concentration, both the P values of the polynomial contrasts for KCl and $KHCO_3$ treatments are given and the contrasts for the averaged measurements are omitted. If the interaction is not significant, we only specify the contrasts for the averaged measurements. The data are presented as means with their standard errors. The level of significance was $\alpha = 0{\cdot}05$. Due to the exploratory nature of the study, we did not adjust the level of significance for multiple testing.

Results

Animal health, body weight, feed intake and urine volume

All the cats were healthy during the entire study period, and no effect of diets on body weight (BW) was observed (Table 2). Urine volume was elevated when the cats were fed the KCl diets (interaction: $P=0{\cdot}318$; salt: $P=0{\cdot}047$), and no significant dependence on dietary K levels could be observed. In both the KCl and $KHCO_3$ groups, the feed intake was elevated when supplementary K was fed during

British Journal of Nutrition

Table 2. Body weight (kg), urine volume (ml/kg body weight per d), urine pH and urine composition (mg/l) of cats fed diets with different potassium concentrations* and salts
(Mean values with their standard errors, *n* 8 per diet)

										P (polynomial contrasts)†						
	Native K (%)	KCl (%)			$KHCO_3$ (%)					KCl		$KHCO_3$		K		
	0·31	0·50	0·75	1·00	0·50	0·75	1·00	SEM	Interaction	Linear	Quadratic	Linear	Quadratic	Linear	Quadratic	Salt
Body weight	3·96	3·73	3·82	3·88	3·88	3·90	3·96	0·17	0·167	–	–	–	–	0·663	0·112	0·265
Urine volume	11·8	11·7	13·1	15·0	11·5	12·1	11·6	0·65	0·318	–	–	–	–	0·526	0·736	0·047
Urine										–	–	–	–			
pH (fasting)	6·29	5·30	5·97	6·38	6·83	6·90	7·65	0·12	0·575	–	–	–	–	0·022	0·004	0·198
pH (postprandial)	6·33	5·34	6·28	6·31	6·84	6·81	7·40	0·13	0·374	–	–	–	–	0·629	0·649	0·114
Ca	25·5	24·9	29·2	23·8	20·2	22·4	20·1	1·46	0·795	–	–	–	–	0·401	0·853	0·183
P	2340	2749	2319	2001	2536	2209	2246	53·2	0·002	0·045	0·017	0·216	0·470	–	–	–
Mg	92·6	94·2	84·0	79·0	103	96·5	80·9	3·42	0·308	–	–	–	–	0·046	0·066	0·150
K	6373	7130	7517	7737	7008	7724	8058	82·1	0·208	–	–	–	–	<0·001	0·059	0·240
Na	6124	10 092	7907	6005	9794	10 313	8050	316	0·038	0·337	0·002	0·003	0·002	–	–	–
Urea	105	72·3	60·3	61·7	66·5	66·2	75·1	2·88	0·131	–	–	–	–	<0·001	0·003	0·392
Creatinine	2189	2257	1992	1814	2368	2306	2103	86·8	0·363	–	–	–	–	0·113	0·039	0·011
Sulphate	3094	2717	2412	2377	3153	2633	2820	87·5	0·420	–	–	–	–	0·005	0·216	0·061
Ox	93·1	100	86·4	80·5	102	150	122	5·05	0·001	0·132	0·334	0·021	0·001	–	–	–
Citrate	106	119	170	174	176	247	284	25·7	0·602	–	–	–	–	0·016	0·640	0·322
Ammonium	997	1204	1159	1080	1209	1068	793	28·6	0·023	0·388	0·039	0·007	0·003	–	–	–

Ox, oxalate.
* Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.
† The interaction term drives the other contrasts as follows: when the interaction is not significant, only *P* values for linear and quadratic contrasts for K (main effect means averaged over K sources) and the *P* value for salt are reported. When the interaction is significant, only *P* values for linear and quadratic contrasts for individual salts (KCl and $KHCO_3$) are reported.

the urine collection period (interaction: $P=0{\cdot}440$; linear contrast: $P=0{\cdot}026$ and quadratic contrast: $P=0{\cdot}018$) and during the faeces collection period (interaction: $P=0{\cdot}516$; linear contrast: $P=0{\cdot}016$ and quadratic contrast: $P=0{\cdot}02$) (Tables 3 and 4).

Urine pH

The interaction between dietary K salt and dietary K concentration was not significant for fasting urine pH ($P=0{\cdot}575$) (Table 2). Compared with the basal diet, which contained only native K, the dietary inclusion of 0·50 % K as KCl led to a decrease in urine pH from 6·29 to 5·30. However, an increase in the pH was observed with higher dietary K levels, added either as KCl or as $KHCO_3$ (linear contrast for K: $P=0{\cdot}022$; quadratic contrast for K: $P=0{\cdot}004$). The measurement of the postprandial urine pH indicated only a numerical increase with higher levels of K in the diets ($P>0{\cdot}05$).

Urine composition and renal mineral excretion

The dependence of urinary K concentrations on dietary K levels did not differ between the KCl and $KHCO_3$ groups (interaction: $P=0{\cdot}208$) (Table 2). We observed a highly significant linear increase in urinary K concentrations with higher dietary K levels ($P<0{\cdot}001$). Renal K excretion reached values of 116 mg/kg BW per d (1·00 % KCl) and 94·6 mg/kg BW per d (1·00 % $KHCO_3$) compared with 75·0 mg/kg BW per d after feeding the cats with the basal diet (Table 3); however, this increase was not statistically significant (P for interaction=0·467; linear contrast for K: $P=0{\cdot}128$). The ratio of renal K excretion: dietary K intake decreased with increasing dietary K levels (P for interaction=0·522; linear contrast for K: $P=0{\cdot}004$).

The interaction between dietary K salt and dietary K concentration was not significant for urinary Ca concentrations ($P=0{\cdot}795$) and renal Ca excretion ($P=0{\cdot}766$). However, while urinary Ca concentrations were unaffected by the dietary K concentration or dietary K salt ($P>0{\cdot}05$), renal Ca excretion was significantly higher in the KCl groups than in the $KHCO_3$ groups (salt: $P=0{\cdot}026$). Urinary Ox concentrations were generally higher in the $KHCO_3$ groups than in the KCl groups, and the dependence of urinary Ox concentrations on dietary K levels was different in the $KHCO_3$ and KCl groups (interaction: $P=0{\cdot}001$). In the $KHCO_3$ groups, we observed a sharp increase in urinary Ox concentrations between native and 0·75% dietary K levels and a moderate decrease between 0·75% and 1·00% K levels in the diets (linear contrast: $P=0{\cdot}021$ and quadratic contrast: $P=0{\cdot}001$), whereas in the KCl groups, no significant effect on urinary Ox concentrations was observed ($P=0{\cdot}132$ and $P=0{\cdot}334$ for linear and quadratic contrasts, respectively). The calculation of the daily renal Ox excretion indicated a significant interaction between dietary K salt and dietary K concentration ($P=0{\cdot}037$); however, the dependence of renal Ox excretion on dietary K levels did not reach significance either for the KCl groups or for the $KHCO_3$ groups.

The interaction between dietary K salt and dietary K concentration was significant for urinary P concentrations ($P=0{\cdot}002$). In only the KCl groups, urinary P concentrations

Table 3. Feed intake (g DM/kg body weight per d) during the urine collection period, renal mineral and oxalate (Ox) excretion (mg/kg body weight per d) and ratio of renal mineral excretion:mineral intake (%) of cats fed diets with different potassium concentrations* and salts
(Mean values with their standard errors, *n* 8 per diet)

	Native K (%)	KCl (%)			$KHCO_3$ (%)				*P* (polynomial contrasts)†						
									KCl		$KHCO_3$		K		
	0·31	0·50	0·75	1·00	0·50	0·75	1·00	SEM	Interaction	Linear	Quadratic	Linear	Quadratic	Linear	Quadratic	Salt
Feed intake	12·5	14·1	14·3	13·7	13·1	12·8	13·3	0·50	0·440	–	–	–	–	0·026	0·018	0·067
Renal Ca excretion	0·32	0·27	0·35	0·33	0·23	0·26	0·22	0·02	0·766	–	–	–	–	0·790	0·712	0·026
Renal Ca excretion:Ca intake	0·20	0·15	0·17	0·18	0·13	0·15	0·14	0·02	0·928	–	–	–	–	0·441	0·457	0·327
Renal P excretion	28·0	31·7	30·4	30·0	29·6	26·9	26·0	1·62	0·868	–	–	–	–	0·926	0·639	0·203
Renal P excretion:P intake	22·4	23·0	20·7	20·7	21·9	20·4	19·3	1·14	0·925	–	–	–	–	0·527	0·893	0·727
Renal Mg excretion	1·11	1·03	1·09	1·12	1·13	1·17	0·88	0·06	0·138	–	–	–	–	0·710	0·730	0·824
Renal Mg excretion:Mg intake	11·1	8·97	8·74	9·67	10·1	10·6	7·92	0·59	0·094	–	–	–	–	0·275	0·788	0·686
Renal K excretion	75·0	83·1	99·4	116	80·7	93·9	94·6	5·09	0·467	–	–	–	–	0·128	0·925	0·097
Renal K excretion:K intake	210	135	95·1	92·5	125	100	77·5	8·62	0·522	–	–	–	–	0·004	0·090	0·472
Renal Na excretion	72·0	116	103	94·6	112	124	101	6·76	0·590	–	–	–	–	0·248	0·088	0·455
Renal Na excretion:Na intake	78·3	112	90·8	81·4	102	116	93·7	5·84	0·318	–	–	–	–	0·634	0·105	0·414
Renal Ox excretion	1·05	1·23	1·21	1·28	1·20	1·88	1·44	0·11	0·037	0·596	0·775	0·204	0·192	–	–	–

*Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.
†The interaction term drives the other contrasts as follows: when the interaction is not significant, only *P* values for linear and quadratic contrasts for K (main effect means averaged over K source) and the *P* value for salt are reported. When the interaction is significant, only *P* values for linear and quadratic contrasts for individual salts (KCl and $KHCO_3$) are reported.

British Journal of Nutrition

Table 4. Feed intake (g DM/kg body weight per d) during the faeces collection period, amount of faeces, DM of the faeces and mineral concentrations in the faeces (mg/g DM) of cats fed diets with different potassium concentrations* and salts
(Mean values with their standard errors, *n* 8 per diet)

	Native K (%)	KCl (%)			$KHCO_3$ (%)					*P* (polynomial contrasts)†					
										KCl		$KHCO_3$		K	
	0·31	0·50	0·75	1·00	0·50	0·75	1·00	SEM	Interaction	Linear	Quadratic	Linear	Quadratic	Linear	Quadratic	Salt
Feed intake	12·4	14·2	14·2	13·5	13·3	12·8	13·2	0·50	0·516	–	–	–	–	0·016	0·002	0·073
Amount of faeces (g/d)	25·8	42·7	36·8	27·8	26·0	23·2	25·1	1·96	0·003	0·975	0·001	0·467	0·610	–	–	–
DM of faeces (%)	38·9	39·2	37·7	37·4	38·3	37·2	44·8	0·79	0·003	0·252	0·875	0·022	0·001	–	–	–
Amount of faeces (g DM/d)	9·54	16·3	13·7	9·84	9·44	8·11	10·7	0·66	< 0·001	0·457	0·003	0·375	0·041	–	–	–
Faecal																
Ca	54·4	41·5	46·9	53·2	53·7	54·1	51·2	1·09	0·001	0·782	0·004	0·267	0·415	–	–	–
P	26·3	25·1	25·0	25·5	26·3	26·6	26·0	0·34	0·778	–	–	–	–	0·737	0·710	0·089
Mg	3·64	2·92	3·21	3·51	3·76	3·64	3·47	0·07	0·013	0·888	0·007	0·271	0·221	–	–	–
K	3·04	3·26	3·62	3·67	2·91	3·26	3·72	0·14	0·442	–	–	–	–	0·044	0·591	0·254
Na	2·83	3·13	3·14	2·89	2·78	2·88	2·69	0·11	0·941	–	–	–	–	0·948	0·258	0·323
Cl	0·99	1·09	1·24	1·32	0·98	1·10	1·09	0·07	0·818	–	–	–	–	0·054	0·876	0·057

* Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.
† The interaction term drives the other contrasts as follows: when the interaction is not significant, only *P* values for linear and quadratic contrasts for K (main effect means averaged over K source) and the *P* value for salt are reported. When the interaction is significant, only *P* values for linear and quadratic contrasts for individual salts (KCl and $KHCO_3$) are reported.

increased significantly between native and 0·50 % K levels in the diets and decreased at higher dietary K levels (linear contrast: $P=0·045$ and quadratic contrast: $P=0·017$). However, neither the evaluation of renal P excretion nor that of the ratio of renal P excretion:dietary P intake indicated diet-dependent differences ($P>0·05$).

In the $KHCO_3$ groups, urinary Na concentrations increased between native and 0·75 % K levels in the diets and decreased between 0·75 and 1·00 % dietary K levels, whereas in the KCl groups, the increase was observed at 0·50 % dietary K levels (interaction: $P=0·038$). In both the KCl and $KHCO_3$ groups, the quadratic contrasts were significant ($P=0·002$). In addition, a linear increase was observed only in the $KHCO_3$ groups ($P=0·003$). The described effects could not be confirmed statistically for renal Na excretion ($P>0·05$), and additionally no group differences ($P>0·05$) were observed for the ratio of renal Na excretion:dietary Na intake.

No significant interaction between dietary K salt and dietary K concentration was observed for urinary Mg concentrations ($P=0·308$). Urinary Mg concentrations (means averaged over dietary K sources) increased between native and 0·50 % dietary K levels and decreased between 0·50 and 1·00 % dietary K levels significantly (linear contrast: $P=0·046$). However, no group differences were detected for renal Mg excretion or the ratio of renal Mg excretion: dietary Mg intake ($P>0·05$).

Urinary urea curves were similar for both the KCl and $KHCO_3$ groups (interaction: $P=0·131$). Initially, urea concentrations (averaged over dietary K sources) decreased, bottomed out at 0·75 % K levels in the diets and increased again. Both the linear decrease ($P<0·001$) and the quadratic effect ($P=0·003$) were highly significant.

Urinary creatinine concentrations were significantly higher in the $KHCO_3$ groups than in the KCl groups (P for interaction=0·363; P for salt=0·011). The urinary creatinine curve rose between native and 0·50 % dietary K levels, peaked out at 0·50 % K levels, and declined between 0·50 and 1·00 % K levels in the diets (quadratic effect: $P=0·039$).

No significant interaction between dietary K salt and dietary K concentration could be observed for urinary sulphate concentrations ($P=0·420$). The linear decrease in urinary sulphate concentrations with respect to dietary K levels was significant ($P=0·005$).

Urinary citrate concentrations exhibited no dependence on the dietary K salt ($P=0·322$), and there was no significant interaction between dietary K salt and dietary K concentration ($P=0·602$). Only the linear increase in urinary citrate concentrations (averaged over dietary K sources) was significant ($P=0·016$).

We observed a significant interaction between dietary K salt and dietary K concentration for urinary ammonium concentrations ($P=0·023$). In both the KCl and $KHCO_3$ groups, urinary ammonium concentrations increased between native and 0·50 % K levels in the diets and decreased between 0·50 and 1·00 % dietary K levels (quadratic effects: $P=0·039$ and $P=0·003$ for the KCl and $KHCO_3$ groups, respectively). In addition, we observed a significant linear decrease in the $KHCO_3$ groups (linear contrast: $P=0·007$).

Faecal parameters and faecal mineral excretion

The amount of faeces (g/d) was dependent on dietary K levels only in the KCl groups (interaction: $P=0·003$), and generally higher amounts were observed in the KCl groups than in the $KHCO_3$ groups (Table 4). In the KCl groups, the amount of faeces increased between native and 0·50 % dietary K levels and decreased between 0·50 and 1·00 % K levels in the diets (quadratic contrast: $P=0·001$). The amount of faeces on a DM basis reached a maximum at 0·50 % dietary K levels when added as KCl and subsequently decreased to levels similar to that of native K in the diets. In the $KHCO_3$ groups, the amount of faeces (g DM/d) decreased initially, bottomed out at 0·75 % dietary K levels and subsequently increased (interaction: $P<0·001$; quadratic contrasts: $P=0·003$ and $P=0·041$ for the KCl and KHO_3 groups, respectively).

Faecal DM concentrations decreased between native and 0·75 % dietary K levels in both the KCl and $KHCO_3$ groups. In the $KHCO_3$ group, we observed a sharp increase between 0·75 and 1·00 % K levels in the diets, whereas in the KCl groups, faecal DM concentrations decreased (interaction: $P=0·003$; linear and quadratic contrasts for the $KHCO_3$ groups: $P=0·022$ and $P=0·001$, respectively).

Faecal K concentrations increased with increasing dietary K levels (no interaction; linear contrast: $P=0·044$). In the KCl groups, the apparent digestibility of K decreased between native and 0·50 % dietary K levels and subsequently increased up to 1·00 % K levels in the diets, while it steadily increased in the $KHCO_3$ groups up to 0·75 % dietary K levels, followed by a small decrease (interaction: $P=0·003$; linear contrast for the KCl group: $P=0·001$, linear contrast for the $KHCO_3$ group: $P=0·003$, and quadratic contrast for the $KHCO_3$ group: $P=0·020$) (Table 5). An interaction between dietary K salt and dietary K concentration was observed for faecal K excretion ($P=0·003$). In the KCl groups, faecal K excretion increased between native and 0·50 % dietary K levels and subsequently decreased up to 1·00 % K levels in the diets. In the $KHCO_3$ groups, faecal K excretion levelled off between native and 0·75 % dietary K levels and increased between 0·75 and 1·00 % K levels in the diets (linear and quadratic contrasts for the KCl groups: $P=0·047$ and $P=0·017$, respectively; linear contrast for the $KHCO_3$ groups: $P=0·032$).

Faecal Ca and Mg concentrations were dependent on dietary K levels only in the KCl groups (interaction: $P=0·001$ and $P=0·013$). The faecal Ca and Mg curves for the KCl groups were V shaped and reached their minimum at 0·50 % dietary K levels (quadratic contrasts for the KCl groups: $P=0·004$ and $P=0·007$). Faecal P, Na and Cl concentrations were not affected by the dietary K salt or dietary K concentration ($P>0·05$).

Faecal Ca, P, Na and Mg excretion was dependent on dietary K levels only in the KCl groups (interaction: $P=0·047$, $P=0·005$, $P=0·041$ and $P=0·007$) and reached its maximum at 0·50 % K levels in the diets (quadratic contrasts for the KCl groups: $P=0·027$, $P=0·008$, $P=0·009$ and $P=0·023$). Comparable results were obtained for faecal Cl excretion, which depended on dietary K levels only in the KCl groups and reached its maximum at 0·50 % K levels in the diets (interaction: $P=0·025$; linear contrast: $P=0·024$ and quadratic contrast: $P=0·016$).

The apparent digestibility of Ca was dependent on dietary K levels only in the $KHCO_3$ groups (interaction: $P=0·047$) and reached its maximum after the inclusion of 0·75 % K in the diets (quadratic contrast for the $KHCO_3$ groups: $P=0·037$).

The dependence of the apparent digestibility of P on dietary K levels was quite different in both the KCl and $KHCO_3$ groups (interaction: $P=0·001$). For the KCl groups, we observed a V-shaped pattern, whereas in the $KHCO_3$ groups, the apparent digestibility of P increased between native and 0·75 % K levels in the diets and decreased between 0·75 and 1·00 % dietary K levels (quadratic contrasts: $P=0·005$ and $P=0·022$ for the KCl and $KHCO_3$ groups, respectively). Comparable results were obtained for the apparent digestibility of Na, which was different in both the KCl and $KHCO_3$ groups (interaction: $P=0·024$). In the KCl groups, after a steep decline between native and 0·50 % dietary K levels, we observed a steady increase in the apparent digestibility of Na between 0·50 and 1·00 % K levels in the diets (quadratic contrast: $P=0·004$). In the $KHCO_3$ groups, a moderate increase between native and 0·75 % dietary K levels was observed, followed by a decrease between 0·75 and 1·00 % K levels in the diets (quadratic contrast: $P=0·042$).

The apparent digestibility of Mg was affected by the dietary K concentration and dietary K salt (P for interaction=0·007); however, the dependence of the apparent digestibility of Mg on dietary K levels did not reach significance for the KCl or $KHCO_3$ groups.

In the KCl groups, the apparent digestibility of Cl decreased between native and 0·50 % dietary K levels and subsequently increased, whereas in the $KHCO_3$ groups, an increase between native and 0·50 % K levels in the diets was followed by a small decrease (interaction: $P=0·002$, linear and quadratic contrasts for the KCl groups: $P=0·014$ and $P=0·013$, respectively, and quadratic contrast for the $KHCO_3$ groups: $P=0·015$).

Mineral retention

No interaction between dietary K concentration and dietary K salt was observed for K retention in the cats ($P=0·640$). The balance was markedly negative after feeding the cats with the basal diet (−197 mg/d) and the 0·50 % KCl diet (−131 mg/d). Feeding the cats with the 0·75 % KCl and 1·00 % KCl diets led to a better, but still negative balance, with values up to −23·3 mg/d. The inclusion of 0·50 % $KHCO_3$ in the diets also led to a negative K retention (−82·5 mg/d), but it became positive with the inclusion of 1·00 % $KHCO_3$ (52·5 mg/d). Overall, an increase in the values of K retention was observed with higher dietary K levels; however, this increase was stronger after the inclusion of $KHCO_3$ in the diets (P for salt=0·018; linear contrast for K: $P=0·002$).

Ca retention was consistently positive, with values between 50·4 and 259 mg/d. However, a dependence on dietary K levels could only be observed in the $KHCO_3$ groups, where the curve had reached its maximum after the inclusion of 0·75 % K in the diets (interaction: $P=0·036$; quadratic contrast for the $KHCO_3$ groups: $P=0·025$). The dependence of P retention on dietary K levels was quite different in both the KCl and

Table 5. Faecal mineral excretion (mg/kg body weight per d), apparent digestibility of minerals (%) and mineral retention (mg/d) of cats fed diets with different potassium concentrations* and salts (Mean values with their standard errors, *n* 8 per diet)

										P (polynomial contrasts)†						
	Native K (%)	KCl (%)			$KHCO_3$ (%)					KCl		$KHCO_3$		K		
	0·31	0·50	0·75	1·00	0·50	0·75	1·00	SEM	Interaction	Linear	Quadratic	Linear	Quadratic	Linear	Quadratic	Salt
Faecal Ca excretion	132	196	172	140	137	118	146	8·20	0·047	0·998	0·027	0·516	0·285	–	–	–
Apparent digestibility of Ca	26·2	9·11	17·6	26·2	31·2	36·9	21·8	2·33	0·047	0·737	0·109	0·709	0·037	–	–	–
Ca retention	164	50·4	114	184	217	259	154	16·3	0·036	0·439	0·071	0·939	0·025	–	–	–
Faecal P excretion	66·2	123	95·5	69·6	68·5	58·1	73·5	5·54	0·005	0·356	0·008	0·576	0·201	–	–	–
Apparent digestibility of P	50·3	23·2	36·1	52·7	53·3	55·3	45·1	2·33	0·001	0·097	0·005	0·413	0·022	–	–	–
P retention	126	0·40	66·5	163	169	169	127	13·2	0·005	0·092	0·001	0·959	0·112	–	–	–
Faecal Na excretion	7·49	17·6	11·8	8·52	7·78	6·73	7·98	1·01	0·041	0·545	0·009	0·909	0·381	–	–	–
Apparent digestibility of Na	92·8	86·3	89·9	92·7	93·5	94·0	93·2	0·63	0·024	0·349	0·004	0·625	0·042	–	–	–
Na retention	43·5	−93·6	−3·78	33·4	−35·4	−92·8	−20·9	22·2	0·247	–	–	–	–	0·601	0·068	0·476
Faecal Mg excretion	8·73	14·3	12·4	9·22	9·56	7·80	9·77	0·64	0·007	0·877	0·023	0·576	0·353	–	–	–
Apparent digestibility of Mg	17·1	−4·27	4·09	23·4	20·3	29·4	15·1	2·98	0·007	0·278	0·070	0·885	0·064	–	–	–
Mg retention	1·66	−6·06	−3·57	5·94	4·27	8·36	3·66	1·26	0·018	0·124	0·055	0·392	0·105	–	–	–
Faecal K excretion	7·86	17·5	14·9	10·7	8·11	7·54	10·7	1·00	0·003	0·047	0·017	0·032	0·172	–	–	–
Apparent digestibility of K	79·8	76·1	86·6	91·9	88·7	92·3	91·6	1·20	0·003	0·001	0·109	0·003	0·020	–	–	–
K retention	−197	−131	−55·6	−23·3	−82·5	−37·6	52·5	20·4	0·640	–	–	–	–	0·002	0·618	0·018
Faecal Cl excretion	2·74	6·12	5·35	4·03	2·87	2·69	3·34	0·41	0·025	0·024	0·016	0·214	0·223	–	–	–
Apparent digestibility of Cl	97·8	96·5	97·2	98·1	98·3	98·1	98·0	0·19	0·002	0·014	0·013	0·570	0·015	–	–	–

* Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.

† The interaction term drives the other contrasts as follows: when the interaction is not significant, only *P* values for linear and quadratic contrasts for K (main effect means averaged over K source) and the *P* value for salt are reported. When the interaction is significant, only *P* values for linear and quadratic contrasts for individual salts (KCl and $KHCO_3$) are reported.

$KHCO_3$ groups (interaction: $P=0{\cdot}005$). The value of P retention was close to 0 after feeding the cats with the 0·50 % KCl diet, which was a marked decrease when compared with that observed after feeding the basal diet, where P retention of 126 mg/d was observed. However, the 0·75–1·00 % KCl diets led to an increase in P retention up to 163 mg/d (quadratic contrast: $P=0{\cdot}001$). In the $KHCO_3$ groups, we found no significant dependence of P retention on K levels in the diets.

The interaction between dietary K salt and dietary K concentration was significant for Mg balance ($P=0{\cdot}018$). However, the dependence of Mg balance on K levels in the diets did not reach significance for the KCl or $KHCO_3$ groups. The data obtained for Na retention indicated no dependence on the dietary K salt or dietary K concentration.

Calculated endogenous potassium losses

Based on the data obtained for daily K intake (mg/kg BW per d) and renal and faecal K excretion (mg/kg BW per d), the regression analysis was as follows: $y = 0{\cdot}475x + 49{\cdot}457$ (renal K excretion) and $y = 0{\cdot}078x + 4{\cdot}098$ (faecal K excretion). Therefore, the extrapolation to 0 indicates endogenous renal K losses of 49·5 mg/kg BW per d and endogenous faecal K losses of 4·1 mg/kg BW per d.

Blood parameters

No group differences were observed in the blood counts of the cats (data not shown). Although some group differences were detected for mineral concentrations in the blood (Table 6), these differences were only small, and all the blood parameters were within the normal range for cats.

An interaction between dietary K salt and dietary K concentration was observed for Cl concentrations in the blood of the cats ($P<0{\cdot}001$). In both the KCl and $KHCO_3$ groups, an increase in Cl concentrations was followed by a decrease, while the highest Cl concentrations were attained at 0·50 and 0·75 % dietary K levels added as KCl and $KHCO_3$, respectively (linear contrasts: $P<0{\cdot}001$ (KCl groups) and $P=0{\cdot}011$ ($KHCO_3$ groups); quadratic contrasts: $P<0{\cdot}001$ (KCl groups) and $P=0{\cdot}001$ ($KHCO_3$ groups)). However, the total concentrations among all the groups varied between 108 and 121 mmol Cl/l, implicating a lower physiological relevance.

K concentrations in the blood depended on dietary K levels only in the KCl groups (interaction: $P=0{\cdot}005$). The curve exhibited a decline between native and 0·75 % dietary K levels and rose between 0·75 and 1·00 % K levels in the diets, slightly exceeding the level of native dietary K (quadratic contrast: $P=0{\cdot}010$).

Na levels in the blood of the cats depended on dietary K levels only in the $KHCO_3$ groups (interaction: $P<0{\cdot}001$). In these groups, a slight increase was observed between native and 0·75 % dietary K levels, followed by a moderate decrease (linear and quadratic contrasts for the $KHCO_3$ groups: $P=0{\cdot}001$ and $P<0{\cdot}001$, respectively). Ca concentrations in the blood exhibited only small variations (2·54–2·77 mmol/l). In both the KCl and $KHCO_3$ groups, Ca concentrations in the blood rose between native and 0·75 % dietary K levels,

Table 6. Mineral concentrations (mmol/l) in the blood of cats fed diets with different potassium concentrations* and salts
(Mean values with their standard errors, *n* 8 per diet)

	Native K (%)	KCl (%)			$KHCO_3$ (%)				*P* (polynomial contrasts)†						
									KCl		$KHCO_3$		K		
	0·31	0·50	0·75	1·00	0·50	0·75	1·00	SEM	Interaction	Linear	Quadratic	Linear	Quadratic	Linear	Quadratic	Salt
Ca	2·57	2·54	2·68	2·66	2·68	2·77	2·57	0·03	0·235	–	–	–	–	0·293	0·017	0·121
Phosphate	1·32	1·34	1·31	1·43	1·39	1·43	1·40	0·02	0·363	–	–	–	–	0·051	0·980	0·119
Na	153	153	155	153	154	154	149	0·31	<0·001	0·561	0·118	0·001	<0·001	–	–	–
K	4·68	4·43	4·02	4·78	4·70	4·74	4·63	0·06	0·005	0·953	0·010	0·633	0·386	–	–	–
Cl	111	121	117	108	113	114	109	0·60	<0·001	<0·001	<0·001	0·011	0·001	–	–	–

* Total concentrations of K in DM: 0·31 % and intended 0·50, 0·75 and 1·00 %.
† The interaction term drives the other contrasts as follows: when the interaction is not significant, only *P* values for linear and quadratic contrasts for K (main effect means averaged over K source) and the *P* value for salt are reported. When the interaction is significant, only *P* values for linear and quadratic contrasts for individual salts (KCl and $KHCO_3$) are reported.

British Journal of Nutrition

peaked at 0·75 % K levels in the diets and declined subsequently (interaction: $P=0·235$; quadratic contrast for K: $P=0·017$). The data obtained for phosphate concentrations in the blood of the cats indicated no dependence on the dietary K salt or dietary K concentration ($P>0·05$).

Discussion

The initial consideration of the present study was that a different K intake from two sources, KCl and $KHCO_3$, would affect renal K excretion, urine pH and urinary Ca concentrations. Except for urinary Ca concentrations, this assumption was confirmed by the results of the present study. The results have practical relevance, because so far the significance of dietary K has been studied in cats only rarely and different salts and their impacts on the metabolism have not been studied extensively.

An important background for the present study was provided by observations made in human subjects, which indicate that the K concentration in the diet affects renal Ca excretion[14,15] and should, therefore, be considered as a risk factor for the formation of Ca-containing uroliths. Although some authors have suggested that this correlation also applies for cats[2], to our knowledge, no detailed studies have been carried out to evaluate the impact of varying dietary K levels on the feline urine composition. It also seems important to consider the anion in the dietary K salt as a potential risk factor for the formation of uroliths, as the solubility of minerals is affected by the alkalisation or acidification of the urine[21]. The present study demonstrates that renal Ca excretion in cats is, contrary to that in humans, unaffected by dietary K levels. However, we observed a generally higher renal Ca excretion when K was added as KCl to the diets than as $KHCO_3$ ($P=0·026$). This finding is in accordance with data obtained from studies carried out in human subjects, where dietary $KHCO_3$, but not KCl, decreased urinary Ca excretion[14]. The authors of these studies suggest that the reduction in renal Ca excretion by generally higher dietary K levels may be mediated by a higher phosphate retention and an associated inhibition of calcitriol synthesis in the kidneys, which results in a lower intestinal Ca absorption[14,15]. It has also been suggested that a higher dietary K intake may lead to an enhanced Ca retention and an inhibition of bone resorption[15]. In the present study, we observed marked higher P and Ca retentions in the $KHCO_3$ groups than in the KCl groups up to a concentration of 0·75 % K in the diets, but not after the dietary inclusion of 1·00 % K as $KHCO_3$. Therefore, the underlying mechanism that led to the lower renal Ca excretion in the $KHCO_3$ groups compared with that in the KCl groups remains unclear and needs further evaluation. It should also be taken into consideration that urinary Ca concentrations were not significantly lower after the dietary inclusion of K as $KHCO_3$. However, with respect to the prevention of CaOx urolith formation, the present results indicate that dietary $KHCO_3$ would be more favourable than KCl, since there is an association with a generally lower renal Ca excretion.

The absent effect of dietary K levels on renal Ca excretion in cats is in contrast to the findings of studies carried out in human subjects[14,15] and may possibly be explained by differences in their metabolism: in these studies[14,15], it has been hypothesised that the lower renal Ca excretion, induced by higher dietary K levels, could be a result of a natriuretic effect of K, which leads to a decrease in the extracellular fluid volume. This volume contraction has been demonstrated to be associated with lower Ca excretion in humans, although the underlying mechanisms have not been fully clarified. In the present study, no diuretic effect was observed when the cats were fed diets containing increasing concentrations of KCl and $KHCO_3$, but the urine volume was higher after the KCl diets were fed ($P=0·026$), indicating an effect of Cl^- as the non-metabolisable anion in the dietary K salt. A higher urine volume in combination with the observed unaffected urinary Ca concentrations can be considered to be beneficial for the prevention of CaOx urolith formation, although the total renal Ca excretion was higher in the KCl groups than in the $KHCO_3$ groups.

Besides the impact of dietary K on urinary Ca excretion, some observed side effects may be particularly interesting in the context of the formation of CaOx stones in cats: as a low urine pH (<6·29) is considered to be a risk factor for the formation of CaOx uroliths[25,26], the alkalising effects of $KHCO_3$ in the diets on urine pH should be noted. The inclusion of 0·50 % K as KCl in the diets led to a decrease in urine pH (fasting: 5·30; postprandial: 5·34) than when fed the basal diet with 0·31 % native K (fasting pH: 6·29; postprandial pH: 6·33), but higher levels of KCl led to an increase in urine pH. Although the fasting pH of 6·38 and the postprandial pH of 6·31 (1·00 % KCl diet) were still in the acidic range, dietary KCl had no dose-related acidifying effects on the urine. It was initially suggested that increasing levels of Cl in the diets would result in increased urinary Cl concentrations and, therefore, in a decrease in urine pH. In this context, it has to be taken into consideration that the present study design cannot provide data on renal Cl excretion. For the analysis of urinary cations and anions, the urine samples were acidified with hydrochloric acid. Thus, the measurement of urinary Cl concentrations could be falsified, even after subtraction of the added Cl. Therefore, only faecal Cl concentrations can be considered for an evaluation of the excretory mechanisms of dietary Cl. However, the results demonstrate that faecal Cl concentrations were not affected by dietary Cl concentrations, with mean values of 1·09–1·32 mg/g DM. Moreover, faecal Cl excretion increased initially after the inclusion of 0·50 % K as KCl in the diets than when fed the basal diet, but subsequently decreased with higher dietary KCl levels. It can, therefore, be assumed that renal Cl excretion increased with increasing levels of Cl in the KCl diets, as expected. The surprising increase in urine pH brought about by the higher levels of KCl in the diets, however, could be explained on the basis of the data obtained for K retention: when the cats were fed the basal diet with only 0·31 % K in DM, K retention was markedly negative with a value of −197 mg/d. It can be hypothesised that the cats were in a K deficiency state, resulting in an intracellular influx of the available, intestinal absorbed K to maintain the water and electrolyte balance and a consecutively reduced renal excretion of K. In contrast,

British Journal of Nutrition

the basal diet contained 8·99 g Cl/kg DM, which is distinctly above the recommended allowance of 0·96 g/kg DM[23]. Therefore, excess Cl consumed with the feed may be renally excreted, resulting in the low urine pH. Feeding the cats with the 0·50 % KCl diet also resulted in a clearly negative K retention of −131 mg/d, and a deficient supply with K can, therefore, be supposed. Thus, as in the case of the basal diet, the available K was probably transported into the cells and only at a low percentage renally excreted. As opposed to this, the Cl concentration in the diet was higher than that in the basal diet and therefore an increased renal Cl excretion can be assumed, resulting in a further decrease in urine pH. Both 0·75 % KCl and 1·00 % KCl diets led to a more balanced K retention, indicating that the cats received sufficient K, which resulted not only in the transport of K into the cells but also in an increased renal K excretion. Although Cl was still excreted into the urine in high amounts, it can be hypothesised that the increased renal excretion of K led to the small but measurable increase in urine pH.

The unavoidable net losses of K were about 54 mg/kg BW per d. As the demonstrated apparent digestibility of K was at an average of 86·7 %, a gross requirement of 62·3 mg K/kg BW per d can be assumed. Based on an average daily feed intake of 13·4 g DM/kg BW, the minimum of 4·65 g K/kg DM (with 16·1 MJ ME/kg DM) could be recommended for cat food, which is near to the actual recommendations of 5·2 g K/kg DM with 16·7 MJ ME/kg DM[23] (≙ 5·0 g K/kg DM with 16·1 MJ ME/kg DM). In this context, the data obtained for K retention in the cats demonstrated that K from $KHCO_3$ was more efficiently retained than K from KCl (P=0·018). In particular, feeding the cats with the 0·75 % $KHCO_3$ and 1·00 % $KHCO_3$ diets led to a better or positive K balance (−37·6 and 52·5 mg/d) than feeding the KCl diets. This aspect may have practical relevance especially when using diets with low K concentrations, which could result in K deficiency in cats if K is added to the diets as KCl.

The increasing urinary K concentrations with increasing dietary K levels were accompanied by a decrease in urinary Na concentrations. This observation indicates that renal K excretion in cats is mediated by an exchange of Na in the kidneys, which has already been described in studies carried out in human subjects[27–29]. The authors of these studies suggested that renal K secretion is mediated by a stimulation of the renal Na, K-ATPase, a higher tubular flow and an increased aldosterone secretion.

Urinary Ox concentrations were generally lower when the cats were fed the KCl diets than when fed the $KHCO_3$ diets, and a dependence on dietary K levels could only be demonstrated for the $KHCO_3$ groups. Urinary Ox concentrations increased with the inclusion of 0·50–0·75 % K as $KHCO_3$ and moderately decreased after feeding the cats with the 1·00 % $KHCO_3$ diet. However, these differences were less clear when comparing the daily renal Ox excretion among the groups. Based on the current knowledge, it remains unclear why the salt of dietary K affected the urinary Ox concentrations of the cats. In general, intestinal Ox absorption or endogenous Ox formation could have been modified. Considering that cat food normally contains only small amounts of Ox, the feline endogenous Ox synthesis is more relevant for the urinary Ox concentrations of cats. However, this aspect has only rarely been investigated in cats so far[1]. Studies carried out in human subjects and rodents have demonstrated that endogenous Ox is mainly formed by metabolic conversions of sugars, amino acids and glycolate[1]. In addition, pyridoxine is known to be a cofactor for the enzyme AGT1[30], which catalyses the transamination of glyoxylate to glycine. Considering that glyoxylate is a precursor of Ox, pyridoxine deficiency can indirectly enhance endogenous Ox synthesis and consecutively urinary Ox excretion. This relationship has already been demonstrated in kittens[31,32]. Moreover, pyridoxine is a cofactor for several enzymes, which are important for the synthesis of citrate. Thus, pyridoxine deficiency can lead to reduced urinary citrate concentrations and therefore to a higher risk of CaOx precipitation[33]. Furthermore, ascorbate has been identified to increase urinary Ox concentrations in human subjects[34–38]. The underlying mechanism is a non-enzymatic oxidation of ascorbate to Ox, which is accelerated when the urine is alkaline. Although ascorbate has not been demonstrated to be an important precursor for the formation of Ox in cats[39], the observed higher urinary Ox concentrations in the present study were associated with a higher urine pH when the cats were fed the $KHCO_3$ diets than when fed the KCl diets. Therefore, it might be that the alkaline urine pH led to an accelerated breakdown of ascorbate to Ox in the urine. This hypothesis is supported by the fact that none of the other mentioned relevant factors influencing endogenous Ox synthesis (sugars, amino acids, glycolate and pyridoxine) differed between the present experimental diets. Based on practical relevance, it can be concluded that a high urine pH, as observed after the dietary inclusion of increasing $KHCO_3$ levels, is accompanied by higher urinary Ox concentrations than after feeding the basal or KCl diets, which could be considered as an antagonistic effect in the prevention of CaOx urolith formation. However, it has been assumed that CaOx precipitation is reduced in an alkaline urine, possibly mediated by an increase in the activity of crystallisation inhibitors such as citrate and chondroitin sulphate[40]. In general, a higher urine pH stimulates renal citrate excretion[1], which could be confirmed by the present results. Thus, the demonstrated increase in urinary Ox concentrations with higher levels of $KHCO_3$ in the diets than when fed the basal and KCl diets seems to be of subordinate importance in the context of the formation of CaOx uroliths, while the observed increase in urine pH could have a greater significance as a preventive factor.

Mineral concentrations in the faeces of the cats as well as the measured blood parameters were not or only sparsely affected by the varying K concentrations or K salts in the diets. Therefore, it can be concluded that dietary K especially affects the urine composition in cats, indicating that K homeostasis is mainly regulated by the kidneys. In the context of the prevention of CaOx stone formation in cats, the present results indicate that the dietary inclusion of $KHCO_3$ might be more favourable than that of KCl as K source, since there is an association between a generally higher urine pH and a lower renal Ca excretion. Unlike in humans, no correlation

between dietary K levels and urinary Ca excretion was observed in cats. The present data can further confirm the actual recommendations for dietary K concentrations in cat food; however, when added as KCl, the utilisation of K is obviously restricted and could result in a K deficiency in case of marginal K levels in a diet.

Acknowledgements

The present study did not receive any specific funding.

The authors would like to thank Professor Barbara Kohn, Clinic of Small Animals, Freie Universität Berlin, for the analysis of the blood samples.

The authors' contributions are as follows: N. P., T. B. and J. Z. designed and organised the study; N. P. carried out the analysis of urine and faecal samples and wrote the manuscript; N. P. and K. N. carried out the data analysis; J. Z. revised the manuscript.

None of the authors has any conflicts of interest.

References

1. Dijcker JC, Plantinga EA, van Baal J, *et al.* (2011) Influence of nutrition on feline calcium oxalate urolithiasis with emphasis on endogenous oxalate synthesis. *Nutr Res Rev* **24**, 96–110.
2. Lekcharoensuk C, Osborne CA, Lulich JP, *et al.* (2001) Association between dietary factors and calcium oxalate and magnesium ammonium phosphate urolithiasis in cats. *J Am Vet Med Assoc* **219**, 1228–1237.
3. Passlack N & Zentek J (2013) Urinary calcium and oxalate excretion in healthy adult cats are not affected by increasing dietary calcium levels. *PLoS ONE* **8**, e70530.
4. Dijcker JC, Hagen-Plantinga EA & Hendriks WH (2012) Changes in dietary macronutrient profile do not appear to affect endogenous urinary oxalate excretion in healthy adult cats. *Vet J* **194**, 235–239.
5. Dijcker JC, Hagen-Plantinga EA, Everts H, *et al.* (2012) Dietary and animal-related factors associated with the rate of urinary oxalate and calcium excretion in dogs and cats. *Vet Rec* **171**, 46–52.
6. Stevenson AE, Blackburn JM, Markwell PJ, *et al.* (2004) Nutrient intake and urine composition in calcium oxalate stone-forming dogs: comparison with healthy dogs and impact of dietary modification. *Vet Ther* **5**, 218–231.
7. Morozumi M, Hossain RZ, Yamakawa K, *et al.* (2006) Gastrointestinal oxalic acid absorption in calcium-treated rats. *Urol Res* **34**, 168–172.
8. Penniston KL & Nakada SY (2009) Effect of dietary changes on urinary oxalate excretion, and calcium oxalate supersaturation in patients with hyperoxaluric stone formation. *Urology* **73**, 484–489.
9. Liebman M & Chai W (1997) Effect of dietary calcium on urinary oxalate excretion after oxalate loads. *Am J Clin Nutr* **65**, 1453–1459.
10. Liebman M & Costa G (2000) Effects of calcium and magnesium on urinary oxalate excretion after oxalate loads. *J Urol* **163**, 1565–1569.
11. Holmes RP, Goodman HO & Assimos DG (2001) Contribution of dietary oxalate to urinary oxalate excretion. *Kidney Int* **59**, 270–276.
12. Masai M, Ito H & Kotake T (1995) Effect of dietary intake on urinary oxalate excretion in calcium renal stone formers. *Br J Urol* **76**, 692–696.
13. Naya Y, Naya Y, Ito H, *et al.* (2002) Association of dietary fatty acids with urinary oxalate excretion in calcium oxalate stone-formers in their fourth decade. *BJU Int* **89**, 842–846.
14. Lemann J Jr, Pleuss JA, Gray RW, *et al.* (1991) Potassium administration reduces and potassium deprivation increases urinary calcium excretion in healthy adults [corrected]. *Kidney Int* **39**, 973–983.
15. Lemann J Jr, Pleuss JA & Gray RW (1993) Potassium causes calcium retention in healthy adults. *J Nutr* **123**, 1623–1626.
16. Osborne CA, Kruger JM, Lulich JP, *et al.* (1995) Disorders of the feline lower urinary tract. In *Canine and Feline Nephrology and Urology*, pp. 625–680 [CA Osborne and DR Finco, editors]. Baltimore: Williams and Wilkins.
17. Pietrow PK & Karellas ME (2006) Medical management of common urinary calculi. *Am Fam Physician* **74**, 86–94.
18. Poupin N, Calvez J, Lassale C, *et al.* (2012) Impact of the diet on net endogenous acid production and acid–base balance. *Clin Nutr* **31**, 313–321.
19. Dow SW, Fettman MJ, Smith KR, *et al.* (1990) Effects of dietary acidification and potassium depletion on acid–base balance, mineral metabolism and renal function in adult cats. *J Nutr* **120**, 569–578.
20. Von Engelhardt W & Breves G (editors) (2010) *Physiologie der Haustiere (Physiology of Companion Animals)*. Stuttgart: MVS Medizinverlag Stuttgart GmbH & Co. KG.
21. Markwell PJ, Buffington CT & Smith BH (1998) The effect of diet on lower urinary tract diseases in cats. *J Nutr* **128**, 2753S–2757S.
22. Naumann C & Bassler C (2004) *Die chemische Untersuchung von Futtermitteln 3. Aufl., 5. Ergänzungslieferung (Chemical Feed Analyses, Vol. 3)*. Darmstadt: VDLUFA-Verlag.
23. National Research Council (NRC) (2006) *Nutrient Requirements of Dogs and Cats*. Washington, DC: The National Academic Press.
24. Gericke S & Kurmies B (1952) Colorimetrische Bestimmung der Phosphorsäure mit Vanadat-Molybdat (Colorimetric determination of phosphoric acid with vanadate molybdate). *Fres Zeitsch Anal Chem* **137**, 15–22.
25. Kirk CA, Ling GV, Franti CE, *et al.* (1995) Evaluation of factors associated with development of calcium oxalate urolithiasis in cats. *J Am Vet Med Assoc* **207**, 1429–1434.
26. Osborne CA, Lulich JP, Thumchai R, *et al.* (1995) *Etiopathogenesis and Therapy of Feline Calcium Oxalate Urolithiasis. Proceedings of 13th ACVIM Forum*. Blacksburg, VA: ACVIM pp. 487–489.
27. Field MJ, Stanton BA & Giebisch GH (1984) Differential acute effects of aldosterone, dexamethasone, and hyperkalemia on distal tubular potassium secretion in the rat kidney. *J Clin Investig* **74**, 1792–1802.
28. Giebisch G (1998) Renal potassium transport: mechanisms and regulation. *Am J Physiol* **274**, F817–F833.
29. Woda CB, Bragin A, Kleyman TR, *et al.* (2001) Flow-dependent K^+ secretion in the cortical collecting duct is mediated by a maxi-K channel. *Am J Physiol Renal Physiol* **280**, F786–F793.
30. Takada Y, Mori T & Noguchi T (1984) The effect of vitamin B_6 deficiency on alanine:glyoxylate aminotransferase isoenzymes in rat liver. *Arch Biochem Biophys* **229**, 1–6.
31. Bai SC, Sampson DA, Morris JG, *et al.* (1989) Vitamin B-6 requirement of growing kittens. *J Nutr* **119**, 1020–1027.
32. Bai SC, Sampson DA, Morris JG, *et al.* (1991) The level of dietary protein affects the vitamin B-6 requirement of cats. *J Nutr* **121**, 1054–1061.

33. Teerajetgul Y, Hossain RZ, Yamakawa K, *et al.* (2007) Oxalate synthesis from hydroxypyruvate in vitamin-B6-deficient rats. *Urol Res* **35**, 173–178.
34. Auer BL, Auer D & Rodgers AL (1998) Relative hyperoxaluria, crystalluria and haematuria after megadose ingestion of vitamin C. *Eur J Clin Invest* **28**, 695–700.
35. Baxmann AC, De O G Mendonca C & Heilberg IP (2003) Effect of vitamin C supplements on urinary oxalate and pH in calcium stone-forming patients. *Kidney Int* **63**, 1066–1071.
36. Chai W, Liebman M, Kynast-Gales S, *et al.* (2004) Oxalate absorption and endogenous oxalate synthesis from ascorbate in calcium oxalate stone formers and non-stone formers. *Am J Kidney Dis* **44**, 1060–1069.
37. Massey LK, Liebman M & Kynast-Gales SA (2005) Ascorbate increases human oxaluria and kidney stone risk. *J Nutr* **135**, 1673–1677.
38. Moyad MA, Combs MA, Crowley DC, *et al.* (2009) Vitamin C with metabolites reduce oxalate levels compared to ascorbic acid: a preliminary and novel clinical urologic finding. *Urol Nurs* **29**, 95–102.
39. Yu S & Gross K (2005) Moderate dietary vitamin C supplement does not affect urinary oxalate concentrations in cats. *J Anim Physiol Anim Nutr* **89**, 428–429.
40. Tiselius HG (1981) The effect of pH on the urinary inhibition of calcium oxalate crystal growth. *Br J Urol* **53**, 470–474.