The effect of varying the sodium or potassium intake, or both, on magnesium status in the rat

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Alterations in sodium or potassium intake, or both, affect the distribution and excretion of magnesium in man and animals. Experiments were performed on rats to investigate the effect of varying Na, K or Na + K intakes on Na, K and Mg excretion and plasma and tissue concentrations. In Expt 1, increasing the Na intake in a linear fashion produced a significant ($P < 0.05$) quadratic response in urinary Mg excretion, with a decrease followed by an increase as Na intake rose. No effect was observed on faecal excretion of Mg. Plasma and tissues were sampled at the end of an 18 d collection period; concentrations of aldosterone in plasma and Mg, Na and K in plasma and tissue were determined. Increasing Na intake in a linear fashion produced a significant ($P < 0.05$) quadratic effect on Mg concentration in heart and muscle, i.e. a decrease followed by an increase as Na intake rose; Na intake did not affect liver or bone Mg concentrations. There were no significant effects of Na intake on plasma Mg, Na or aldosterone but plasma K fell significantly ($P < 0.01$) as Na intake increased. In Expt 2, constant amounts of four diets supplying adequate or high levels of Na and adequate or high levels of K but a constant intake of Mg were given to rats. The rats fed on the adequate-Na diet had a significantly ($P < 0.05$) higher urinary Mg excretion than those fed on the high-Na diet; Na intake did not affect faecal Mg excretion.

Magnesium status: Potassium intake: Sodium intake: Rat

Sodium is the most abundant extracellular cation in the mammalian body whilst potassium and magnesium predominate intracellularly. As a result of the functional relation amongst these elements in maintaining osmolality and acid–base balance, nutritional interactions amongst these elements may be anticipated. Nutritional manipulation of dietary Na, K and Mg has resulted in altered mineral excretion and tissue composition leading to impaired growth in man and animals (Forbes, 1966; Duarte, 1980; Ryan & Whang, 1983).

The interrelationships between these cations are complex, with reduced Na intake or elevated K intake, or both, decreasing apparent absorption of Mg in the ruminant leading to hypomagnesaemia (Morris & Gartner, 1975) whilst in other species Mg deficiency induces increased Na and decreased K concentrations in muscle and heart (Ebel & Günther, 1980).

In addition, the mineralocorticoid aldosterone which is stimulated by low Na or high K intakes, or both, has been demonstrated to increase Mg excretion in the urine of rats (Hanna & MacIntyre, 1960) and sheep (Scott & Dobson, 1965) and to affect the distribution of Mg in the tissues (Duarte, 1980).

Even though research continues to provide information on the interactions between Na, K and Mg, little effort has been directed toward determining the effect of these elements on the absorption, excretion and tissue distribution of one another. Therefore, the aim of the present study was to evaluate the effect of different dietary concentrations of Na and K on the apparent absorption, excretion, plasma concentration and distribution of Na, K and

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Mg in the rat. Plasma aldosterone concentrations were also estimated because of their possible involvement in the physiology of Na, K and Mg.

MATERIALS AND METHODS

Animals and diets

Expt 1. Twenty-five male Albino Wistar rats, mean age 6 weeks and mean weight 100 g, were randomly divided into five equal groups and allocated to individual metabolism cages which allowed for the separate collection of faeces and urine. Each animal was given 15 g fresh weight/d of a starch-based diet containing varying amounts of Na (mg/d): group 1, 16; group 2, 20·5; group 3, 25; group 4, 34; group 5, 43. Mg intake was maintained at 4·1 mg/d and K intake at 34 mg/d for all rats. The dietary constituents were (g/kg): 520 starch, 160 glucose, 100 maize oil, 100 albumin, 80 cellulose, 40 minerals and vitamins. For details of the mineral content of the diet, see Table 1. All rats were allowed continuous access to distilled water.

Expt 2. Four groups each of five rats were given individually one of four diets containing varying amounts of Na and K, but a constant amount of Mg (7·3 mg/d) for each rat. Individual Na and K intakes were as follows: diet 1 received adequate Na and K (16 and 88 mg/d respectively), diet 2 high Na and adequate K (23·5 and 88 mg/d respectively), diet 3 adequate Na and high K (16 and 142 mg/d respectively), diet 4 high Na and high K (23·5 and 142 mg/d respectively). 'Adequate' and 'high' relate to the values recommended by the (US) National Research Council (1978). The remaining dietary constituents were as used in Expt 1. For details of the mineral content of the diet, see Table 1.

Collection of samples for analysis

After adaptation to the diets, total faeces and urine were collected every 2 d for an 18 d period. Urine was stored frozen at −18° and faeces dried at 104° to constant weight and stored dried. The animals were then killed by diethyl ether inhalation and bled by open cardiac puncture. Blood was immediately centrifuged at 4° and plasma separated by aspiration and stored at −18°. The heart, liver, quadriceps femoris and femur were removed, dissected free of fat and dried at 104° to constant weight before analysis.

Analyses

Weighed and dried samples of diet, faeces and tissue were wet-ashed in a nitric–sulphuric–perchloric acid mixture (Kirkbright & Sargent, 1974). Urine and plasma were diluted with distilled water before analysis. Na and K were determined by flame photometry (Corning 400; Corning, Halstead, Essex). Mg was determined by atomic absorption spectrophotometry (Pye Unicam SP9, Pye Unicam, Cambridge). Plasma aldosterone was determined by radioimmunoassay by the method described by DeMan *et al.* (1980).

Statistical analysis

The faecal, urine, balance, plasma and tissue values were examined by one-way analysis of variance. Faecal and urinary values for individual collection periods were summed before statistical analysis. In Expt 1 the effects of Na content of the diet were tested against the between-animals, within-diets variation using orthogonal polynominals to test the shapes of responses, linear or quadratic (Dixon, 1983). The linear and quadratic coefficients were derived by J. C. Mathers (personal communications) and are shown in Table 2. In Expt 2 the effects of Na intake, K intake and the interaction between Na and K intakes were tested against the between-animals, within-diets variation.

Table 1. *Expts 1 and 2. The composition of the mineral supplement (g/kg supplement) fed to rats*

	Expt 1	Expt 2
NaCl	0, 19·1, 38·2, 76·3, 114·4	0, 31·8
KH_2PO_4	162·5	477, 953
$MgCl_2{\cdot}6H_2O$	46·3	92·6
$CaCO_3$	625	625
$MnSO_4{\cdot}4H_2O$	10·2	10·2
$FeSO_4{\cdot}7H_2O$	8·7	8·7
$ZnSO_4{\cdot}7H_2O$	2·7	2·7
$CuSO_4{\cdot}5H_2O$	1·0	1·0
KIO_3	0·1	0·1

Table 2. *Expt 1. Dietary intake and mean output of magnesium, sodium and potassium in faeces and urine (mg/18 d) during the 18 d collection period in five rats given diets containing increasing amounts of Na*

							Statistical significance of diet effects (df 20)	
Diet†...	1	2	3	4	5	SEM	LIN	QUAD
Coefficient‡								
LIN	−13	−8	−3	+7	+17			
QUAD	+127	−20	−109	−113	+115			
Mg								
Intake	73·8	73·8	73·8	73·8	73·8	—	—	—
Faeces	15·6	14·4	17·7	17·1	17·1	1·26	NS	NS
Urine	21·9	21·3	19·8	18·0	24·3	1·17	NS	*
Retention	+36·3	+38·1	+36·3	+38·7	+32·4	2·41	NS	NS
Na								
Intake	288·0	369·0	450·0	612·0	774·0	—	—	—
Faeces	48·6	64·2	77·7	85·8	106·8	10·65	***	NS
Urine	271·2	374·1	466·5	573·0	707·9	16·11	***	NS
Retention	−31·8	−69·3	−94·2	−46·8	−40·7	25·67	NS	**
K								
Intake	612·0	612·0	612·0	612·0	612·0	—	—	—
Faeces	71·1	67·5	80·7	97·5	86·1	11·22	NS	NS
Urine	483·9	524·4	513·6	542·4	526·8	11·61	NS	NS
Retention	+57·0	+20·1	+17·7	−27·9	−0·9	20·61	**	*

LIN, linear effect of increasing Na intake; QUAD, quadratic effect of increasing Na intake; NS, not significant. * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$, *** $P < 0{\cdot}001$.

† Diets 1, 2, 3, 4 and 5 provided 16, 20·5, 25, 34 and 43 mg Na/d respectively (for details of composition see p. 400 and Table 1).

‡ J. C. Mathers, personal communication.

RESULTS

Expt 1

Table 2 shows that although there was not a significant response in the excretion of Mg in the faeces to the increased Na intake, the excretion of Mg in the urine showed a significant ($P < 0{\cdot}05$) quadratic response. The urinary excretion of Mg fell as the Na intake was increased from 9 mg/d to 27 mg/d, but rose again when Na intake was further increased to 36 mg/d.

Table 3. *Expt 1. The effect of increasing sodium intake on the concentration of magnesium, Na, potassium and aldosterone in plasma and in various tissues at the end of the 18 d collection period in five rats*

							Statistical significance of diet effects (df 20)	
Diet†...	1	2	3	4	5	SEM	LIN	QUAD
Coefficient‡								
LIN	−13	−8	−3	+7	+17			
QUAD	+127	−20	−109	−113	+115			
Mg								
Plasma (mmol/l)	1·17	1·23	0·92	0·90	1·06	0·10	NS	NS
Tissue (μg/g)								
Heart	783	638	664	603	673	35·9	NS	*
Liver	459	494	437	583	551	48·2	NS	NS
Muscle	847	885	839	582	779	38·4	NS	*
Bone	2154	2094	2140	2274	2008	83·4	NS	NS
Na								
Plasma (mmol/l)	151	155	147	144	148	4·55	NS	NS
Tissue (mg/g)								
Heart	4·5	4·0	3·8	3·9	3·8	0·16	**	NS
Liver	1·6	1·5	1·6	2·0	2·0	0·16	*	NS
Muscle	1·7	1·6	1·8	1·8	1·5	0·15	NS	NS
Bone	4·7	4·2	4·3	4·1	4·0	0·13	**	NS
K								
Plasma (mmol/l)	7·6	6·4	5·4	5·9	5·1	0·50	**	NS
Tissue (mg/g)								
Heart	9·1	8·4	7·9	7·8	7·8	0·40	*	NS
Liver	2·7	3·8	3·5	4·7	4·1	0·42	NS	NS
Muscle	7·1	7·5	7·7	9·4	6·8	0·54	NS	*
Bone	3·3	3·7	3·5	4·0	3·4	0·14	NS	NS
Plasma aldosterone (mmol/l)	1·02	2·02	1·29	1·17	1·06	0·261	NS	NS

LIN, linear effect of increasing Na intake; QUAD, quadratic effect of increasing Na intake; NS, not significant.
* $P < 0·05$, ** $P < 0·01$.
† Diets 1, 2, 3, 4 and 5 provided 16, 20·5, 25, 34 and 43 mg Na/d respectively (for details of composition, see p. 400 and Table 1).
‡ J. C. Mathers, personal communication.

The Na excretion and balance values responded as expected to the increase in Na intake. Na excretion in faeces and urine rose in a highly significant ($P < 0·001$) linear response. There were no significant effects of increased Na intake on the excretion of K in faeces or urine.

The results relating to the plasma values at the end of the collection period are shown in Table 3. There were no significant effects of dietary Na intake on plasma concentrations of Mg, Na or aldosterone. There was a highly significant ($P < 0·01$) linear decrease in the circulating concentrations of K as Na intake was increased.

The mean concentrations of Mg in various tissues at the end of the experiment are also shown in Table 3. Increasing the Na intake produced a significant ($P < 0·05$) quadratic effect on the heart and muscle Mg concentration, with a gradual decrease from diet 1 to diet 4, followed by a pronounced increase with the highest Na intake. There were no effects of diet on the Mg concentration in the liver or the femur.

There was a highly significant ($P < 0·01$) linear decrease in the Na content of the heart and femur as Na intake increased, and a significant ($P < 0·05$) linear increase in the Na

Table 4. *Expt 2. Mean output of magnesium, sodium and potassium in faeces and urine (mg/18 d) during the 18 d collection period in five rats given diets containing adequate (16 mg/d) or high (23·5 mg/d) levels of Na and adequate (88 mg/d) or high (142 mg/d) levels of K*

Dietary Na...	16	23·5	16	23·5				
Dietary K...	88	88	142	142				
Diet†...	1	2	3	4				
Coefficients						Statistical significance of diet effects (df 16)		
Na	−1	−1	+1	+1				
K	−1	+1	−1	+1				
Na × K	+1	−1	−1	+1	SEM	Na	K	Na × K
Mg								
Intake	131·4	131·4	131·4	131·4	—	—	—	—
Faeces	29·4	28·2	30·6	31·2	1·92	NS	NS	NS
Urine	23·7	20·7	28·2	18·6	2·49	*	NS	NS
Retention	+78·3	+82·5	+72·6	+81·6	4·98	*	NS	NS
Na								
Intake	288·0	423·0	288·0	423·0	—	—	—	—
Faeces	17·1	18·9	20·1	37·2	4·41	*	*	NS
Urine	421·2	572·4	456·9	531·9	14·28	***	NS	*
Retention	−150·3	−168·3	−189·0	−146·1	15·96	NS	NS	NS
K								
Intake	1584	1584	2556	2556	—	—	—	—
Faeces	39·6	52·2	37·5	79·5	7·32	NS	NS	NS
Urine	1320	2418	1241	2310	52·59	*	NS	NS
Retention	+224·4	−886·2	+1277·5	+166·5	51·62	*	NS	NS

NS, not significant.
* $P < 0·05$, *** $P < 0·001$.
† For details of composition, see p. 400 and Table 1.

concentration in the liver. There were no significant effects of diet on the Na concentration of muscle.

There was a significant ($P < 0·05$) linear decrease in the K concentration in the heart as Na intake increased, but the muscle K concentration gradually increased between diets 1 and 4 then rapidly decreased at the highest level of Na intake. There were no significant effects of Na intake on the K concentration in the liver or femur.

Expt 2

As in Expt 1 there were no signifiant effects of Na or K intake, or both, on Mg faecal excretion, but significant ($P < 0·05$) increases in urinary excretion of Mg were detected when the diets without any additional Na (diets 1 and 3) were given; this increase was more pronounced when K intake was high (see Table 4). There were significant increases in the excretion of Na both in the faeces ($P < 0·05$) and urine ($P < 0·001$) when Na intake was increased. Increasing the K intake also significantly ($P < 0·05$) increased the excretion of Na in the faeces. Increasing the Na intake significantly ($P < 0·05$) increased the excretion of K in urine but not in faeces but there were no significant effects of dietary K on the excretion of K in faeces or urine.

There were no significant effects of Na or K intake, or both, on the plasma concentrations of Mg or K but increasing the Na intake or the K intake, or both, significantly ($P < 0·001$) increased the plasma concentrations of Na (Table 5). Increasing both the Na and K intake significantly ($P < 0·05$) increased aldosterone levels.

Decreasing the Na intake significantly ($P < 0·05$) decreased the Mg concentration in the heart but there were no other significant effects of Na or K intake, or both, on the Mg

Table 5. *Expt 2. The effect of adequate (16 mg/d) or high (23·5 mg/d) sodium intake and adequate (88 mg/d) or high (142 mg/d) potassium intake on the concentration of magnesium, Na, K and aldosterone in plasma and various tissues at the end of the 18 d collection period in five rats*

						Statistical significance of diet effects (df 16)		
Dietary Na ...	16	23·5	16	23·5				
Dietary K ...	88	88	142	142				
Diet† ...	1	2	3	4				
Coefficients								
Na	−1	−1	+1	+1				
K	−1	+1	−1	+1				
Na × K	+1	−1	−1	+1	SEM	Na	K	Na × K
Mg								
Plasma (mmol/l)	1·16	0·84	0·77	0·70	0·14	NS	NS	NS
Tissue (μg/g)								
Heart	528	568	477	602	35·3	*	NS	NS
Liver	515	491	493	471	30·4	NS	NS	NS
Muscle	764	703	809	728	35·7	NS	NS	NS
Bone	2574	2718	2583	2360	147·2	NS	NS	NS
Na								
Plasma (mmol/l)	166	225	229	239	7·10	***	***	**
Tissue (mg/g)								
Heart	3·27	3·80	3·64	4·18	0·30	NS	NS	NS
Liver	1·74	1·40	1·49	1·41	0·15	NS	NS	NS
Muscle	1·72	1·53	1·78	1·44	0·10	*	NS	NS
Bone	6·24	7·09	6·91	7·10	0·38	NS	NS	NS
K								
Plasma (mmol/l)	8·4	8·7	8·6	8·6	0·38	NS	NS	NS
Tissue (mg/g)								
Heart	5·81	6·34	5·04	5·99	0·36	NS	NS	NS
Liver	5·69	5·34	6·11	5·86	0·36	NS	NS	NS
Muscle	9·83	9·85	10·33	9·70	0·47	NS	NS	NS
Bone	3·10	3·51	2·82	3·40	0·27	NS	NS	NS
Plasma aldosterone (mmol/l)	2·17	1·57	2·28	2·94	0·352	NS	*	*

NS, not significant.
* $P < 0·05$, ** $P < 0·01$, *** $P < 0·001$.
† For details of composition, see p. 400 and Table 1.

concentrations of the remaining tissues analysed. Decreasing the Na intake significantly ($P < 0·05$) increased the Na concentration in the muscle but there were no other significant effects of Na or K intake, or both, on the Na concentration in the remaining tissues analysed. There were no significant differences in the K concentration in any of the tissues analysed.

DISCUSSION

As expected in Expt 1, increasing the Na intake significantly increased the linear excretion of Na both in faeces and urine. However, there were no significant effects of Na intake on plasma Na, plasma Na being tightly controlled by a variety of hormones including aldosterone. The plasma aldosterone concentrations in Expt 1 showed no significant variation with Na intake. This was probably due to the Na intake being above requirements in all the diets ((US) National Research Council, 1978) and a low Na intake is required to stimulate aldosterone. Increasing the Na intake significantly increased the Na concentration of the heart, suggesting an increase in the intracellular concentration of Na in the heart muscle, and there was a significant decrease in the Na concentration in the femur.

Increasing the Na intake in Expt 1 had no significant effect on the excretion of K in faeces or urine but did significantly lower plasma K concentrations linearly. Extracellular K in the plasma did not appear to be shifting intracellularly into the tissues as none of the tissues analysed showed any significant increase in K concentration.

The most noticeable feature of the findings relating to Mg was the gradual decrease in the excretion of Mg in the urine as Na intake was increased from diet 1 to diet 4, followed by a marked increase with diet 5. This was not related to the apparent absorption of Mg from the gut as there were no significant changes in the excretion of Mg in the faeces as Na intake increased.

The increased excretion of Mg in the urine at low and at high Na intakes may be due to the loss of intracellular Mg from the tissues. At times of low Na intake, the body tissues act as a reservoir for Na, mobilization of which will release not only Na but Mg and K into the circulation (Larvor, 1976; Duarte, 1980; Schricker, 1985), which may account for the increased excretion of Mg at low Na intakes. The findings relating to the tissue Mg content are shown in Table 3; heart and muscle showed the same quadratic response in Mg content as did the urine, i.e. a decrease from diet 1 to diet 4 followed by an increase. This may have been due to storage of Mg in the tissues but does not account for their origin or the origin of the Mg excreted in the urine.

An explanation for the increased excretion of Mg in the urine at low Na intakes relates to the ionic exchange of Mg^{2+} for K^{+} in the kidney tubules; evidence from previous work (Samley *et al.* 1960; Lemann *et al.* 1970) has suggested that at times of low Na intake, increased amounts of Mg are excreted in the urine as an ion-exchange for K. The findings relating to the excretion of K in the urine (Table 2) would tend to support this hypothesis, i.e. when the intake of Na was at its lowest, diet 1, the excretion of K in the urine was at its lowest and the excretion of Mg at its highest. No explanation can be offered for the increased excretion of Mg in the urine at high Na intakes. The findings relating to the excretion of Mg in the faeces have shown no significant responses to the variation in Na intake.

In Expt 2, the Na and K intakes were maintained at adequate or high intakes and it was hoped that increasing the K intake would stimulate the endogenous secretion of aldosterone. As can be seen from the results presented in Table 5, there was a significant effect of increasing K intake on plasma aldosterone concentrations.

Again increasing the Na intake significantly increased its loss in faeces and urine and surprisingly, bearing in mind the results produced in Expt 1, increasing the Na intake increased the plasma Na concentrations. These increases in plasma Na appear to be very difficult to explain considering the attendant natriuresis.

Increasing the K intake produced no natriuresis and had no effect on the faecal excretion of Na, results which do not concur with those reported by Duarte (1980). The doubling of K intake produced no significant effect on any K variable but increasing the Na intake virtually doubled K excretion in the urine, probably by increasing the secretion of K by late distal tubules and cortical collecting ducts (Valtin, 1983). Duarte (1980) demonstrated that increasing the K intake of rats produced a kaliuresis and an accumulation of K in heart and bone, but the results presented in Tables 4 and 5 indicate that we were unable to demonstrate any significant effect of K intake on urinary excretion or tissue K concentration.

The Mg results presented in Expt 2 (Table 4) essentially confirm those of Expt 1. Again the K excretion findings support the explanation of ionic exchange at lower Na intakes. However, in relation to the content of Mg in the tissues, the results of Expt 2 do not concur with those of Expt 1. In Expt 2 the concentration of Mg in the heart increased with increasing Na intake whereas in Expt 1 the animals which had the lowest Na intake had

heart tissue containing the highest Mg concentration. The most likely explanation for this difference is the much higher K intake of the animals in Expt 2.

In Expt 2 the plasma Mg levels were lower in the high-K groups (diets 3 and 4) compared with the adequate K groups (diets 1 and 2). Although this result was not statistically significant, other workers (Alexander & Levinsky, 1968; Duarte, 1974) have also found that in rats fed on a high-K diet, there was a significant reduction in plasma Mg which could not be related to increased losses of Mg in the urine.

In summary, lowering the Na intake or increasing the K intake, or both, increases the excretion of Mg in the urine, but any link with aldosterone remains tenuous. Increasing the K intake had no significant effect on K excretion, whereas increasing the Na intake increased the excretion of K in urine. Dietary manipulation of Na, K and Mg produced no conclusive effects on plasma or tissue Na, K and Mg concentrations.

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