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Effect of potato on acid–base and mineral homeostasis in rats fed a high-sodium chloride diet

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Excessive dietary NaCl in association with a paucity of plant foods, major sources of K alkaline salts, is a common feature in Western eating habits which may lead to acid–base disorders and to Ca and Mg wasting. In this context, to evaluate the effects of potato, rich in potassium citrate, on acid–base homeostasis and mineral retention, Wistar rats were fed wheat starch (WS) or cooked potato (CP) diets with a low (0·5 %) or a high (2 %) NaCl content during 3 weeks. The replacement of WS by CP in the diets resulted in a significant urinary alkalinisation (pH from 5·5 to 7·3) parallel to a rise in citrate and K excretion. Urinary Ca and Mg elimination represented respectively 17 and 62 % of the daily absorbed mineral in rats fed the high-salt WS diet compared with 5 and 28 % in rats fed the high-salt CP diet. The total SCFA concentration in the caecum was 3-fold higher in rats fed the CP diets compared with rats fed the WS diets, and it led to a significant rise in Ca and Mg intestinal absorption (Ca from 39 to 56 %; Mg from 37 to 60 %). The present model of low-grade metabolic acidosis indicates that CP may be effective in alkalinising urine, enhancing citrate excretion and ameliorating Ca and Mg balance.

Potato: Sodium chloride: Calcium: Citrate

In comparison with the prehistoric diet, the modern human diet shows an overabundance of NaCl associated with a low provision of alkaline salts of K, precursors of $KHCO_3$, which are ubiquitously present in plant foods (Eaton *et al.* 1996, 1997; Frassetto *et al.* 2001). As a consequence of a long-term low K supply and of a shift between the contemporary diet and the genetically determined metabolic machinery of man, a state of low-grade metabolic acidosis may take place (Frassetto *et al.* 1996; Sebastian *et al.* 2002) with several metabolic disturbances (Wiederkehr & Krapf, 2001).

There is evidence that long-term periods of dietary NaCl load may have adverse effects on bone metabolism and may be a risk factor for osteoporosis in postmenopausal women (Cohen & Roe, 2000). Excess in dietary NaCl is a well-known determinant of urinary Ca excretion, frequently in parallel to a bone-demineralising effect and an activation of bone resorption as shown in experimental animals (Goulding & Gold, 1986; Chan & Swaminathan, 1994; Creedon & Cashman, 2000) and human subjects (Shortt & Flynn, 1990, 1991; Matkovic *et al.* 1995; Itoh & Suyama, 1996; Lietz *et al.* 1997; Ginty *et al.* 1998).

Therapies that decrease urinary Ca excretion could potentially prevent the calciuric effect of affluent dietary NaCl. Alkaline salts of K (potassium bicarbonate, potassium citrate) have been shown to significantly reduce urinary Ca excretion (Lemann *et al.* 1989, 1991; Sakhaee *et al.* 1991; Morris *et al.* 1999; Frassetto *et al.* 2000) and improve bone status (Tucker *et al.* 1999; Marangella *et al.* 2004). There is also evidence that fruit and vegetable intake may be an effective means to counteract the acidity generated by the diet (Vormann & Daniel, 2001), reduce calciuria and consequently improve Ca balance (Appel *et al.* 1997; New, 2003). Rich in K (2 % of DM), essentially as potassium citrate, potato presents an interesting potential renal acid load of $-4{\cdot}0$ mmol/100 g edible portion (Remer & Manz, 1995) and has been recently recognised as an important factor for lowering net acid excretion (Prynne *et al.* 2004). Thus, potato intake could be taken into account in the prevention of mineral loss induced by chronic consumption of modern diets.

We designed the present study according to the hypothesis that supplementing the diet with exogenous alkali, in the form of cooked potato (CP), might counteract deleterious effects of a high-NaCl load on acid–base status and mineral excretion. To further investigate these points, rats were fed wheat starch (WS) or CP diets with low or high NaCl content during 3 weeks. In each group, the urinary acid–base profile was determined and Ca and Mg balance examined. These experiments were conducted in growing rats in condition of positive Ca and Mg balance.

Materials and methods

Experimental diets

Rats were adapted to four different diets (Table 1) in which carbohydrates were provided by WS or by CP. Charlotte

Abbreviations: CP, cooked potato; WS, wheat starch.

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Table 1. Composition of experimental diets (g/kg dry matter)*

	Wheat starch		Cooked potato	
Ingredients†	Low salt	High salt	Low salt	High salt
Casein	200	200	140	140
Methionine	5	5	5	5
Maize oil	50	50	50	50
$CaCO_3$	5	5	5	5
$CaH(PO_4).2(H_2O)$	8·6	8·6	6·9	6·9
$MgSO_4.7H_2O$	9	9	–	–
KCl	3·72	3·72	–	–
PO_4H_2K	6·8	6·8	–	–
NaCl	5	20	5	20
Trace element mix	10	10	7·5	7·5
Vitamin mix	10	10	7·5	7·5
Wheat starch	687	672	–	–
Cooked potato	–	–	773	758
Na:K ratio	0·5	2·0	0·15	0·6

* All diets contained on a DM basis (g/kg diet): Ca, 3·7; Mg, 0·9; P, 3·1. The low-NaCl diets contained 2 g Na/kg whereas high-NaCl diets contained 7·9 g/kg. Cooked potato and wheat starch diets contained 15 and 4 g K/kg respectively. The mineral content of all diets was checked before the beginning of the experiment. The mineral content of the Charlotte potato cultivar was on a DM basis (mg/kg diet): Ca, 92; Mg, 257; K, 4934; P, 510.

† Casein and wheat starch were supplied by Louis François (Saint-Maur, France), maize oil by Huileries de la Lapalisse S.A. (Lapalisse, France), methionine and minerals by Sigma Chemical Co. (St Louis, MO, USA), and trace elements and vitamin AIN-93N mix without vitamin E by Dyets, Inc. (Bethlehem, PA, USA).

potatoes, a cultivar usually consumed in France, were obtained from the Jardins de Limagne (Aigueperse, France). Every day, about 1·5 kg potatoes was steamed for 30 min before mashing. Mashed potatoes were then mixed with a semi-purified mixture providing casein, methionine (0·5 %), maize oil, minerals, trace elements and vitamin mix. Each diet contained 20 % protein, namely 20 % casein in the WS diets and 14 % casein in the CP diets, potato providing 8 % protein on a dry-weight basis. WS and CP diets contained respectively 3·9 g K/kg and 13 g K/kg DM, K being exclusively provided by potato in the CP diets. High-salt diets contained 7·9 g Na/kg DM, whereas the others contained 2 g Na/kg DM. Thus, the Na:K ratio was 0·5 with the low-salt WS diet but only 0·15 with the low-salt CP diet, and it was 4-fold enhanced by the NaCl load in each case. All diets were balanced, on a dry-weight basis, in Ca (3·7 g/kg), Mg (0·9 g/kg) and P (3·1 g/kg), taking into account minerals provided by potato.

Animals

Male Wistar rats weighing approximately 180 g were used. They were from the colony of laboratory animals of the National Institute of Agronomic Research (INRA; Clermont-Ferrand/Theix, France). They were randomly allocated to four groups of eight rats and fed one of the experimental diets for 21 d, distributed as semi-purified moistened powder for the WS diets and as fresh CP, supplemented with a semi-purified mixture, for the CP diets. All the diets were balanced in nutrients, vitamins, Ca, Mg and P, taking into account the endogenous supply of potatoes. The animals were allowed free access to fresh food and water. Rats were housed two per cage and maintained in temperature-controlled rooms (22°C), with the dark period from 20.00 to 08.00 hours. They were maintained and handled according to the recommendations of the Institutional Ethics Committee (Clermont-Ferrand University, France), in accordance with decree no. 87–848. The body weight of rats was recorded twice per week during the experimental period. During the last 7 d of the experiment period, rats were transferred to metabolism cages; food intake and daily urine and faecal excretion were recorded over the last 4 d.

Sampling procedures

Rats were first anaesthetised with sodium pentobarbital (40 mg/kg) and maintained on a plate at 37°C. An abdominal incision was made, and blood was withdrawn from the abdominal aorta into heparinised tubes. After centrifugation at 10 000 ***g*** for 2 min, plasma was collected and stored at −20°C for analysis. After blood sampling, the caecum with its content was removed and weighed and samples of caecal contents were transferred into two microfuge tubes; one was immediately frozen at −20°C and the pH of the caecal contents was measured in the other one. Then, rats were killed by an overdose of pentobarbital.

Analytical procedures

For mineral determinations, 0·25 g dried samples (food and faeces) was dry-ashed (10 h at 500°C) and then extracted at 130°C in HNO_3–H_2O_2 (2:1) (Suprapur; Merck, Darmstadt, Germany) until discoloration. Final dilution was made in 0·1 g/l lanthanum oxyde solution (for Ca and Mg determinations). Mineral concentrations were determined for Ca and Mg by atomic absorption spectroscopy (AA800; Perkin-Elmer, Norwalk, CT, USA) in an acetylene–air flame at the following wavelengths: 422·7 nm (Ca); 285·2 nm (Mg). K and Na were determined by atomic emission spectroscopy at the following wavelengths: 766·5 nm (K); 589·5 nm (Na). P was determined spectrophotometrically using a commercial kit (Biomérieux SA, Lyon, France). Urines were 400-fold diluted with milli-Q water and anions were analysed using a DX320 Dionex chromatograph (Sunnyvale, CA, USA). The anions were separated on a 4 × 250 mm AS 11 column/AG 11 precolumn (flow rate 1 ml/min). An EG40 eluent generator controlled the elution with an OH^- gradient (0·5 to 35 mmol/l in 20 min) and the conductimetry detector was preceded by a ASRS self-regenerating suppressor. Peaks were identified and quantified by comparison with pure anion standards.

SCFA were measured by GLC in supernatant fractions of caecal contents (40 000 ***g***; 15 min) after acidification by H_3PO_4 (10 %), as described by Remesy & Demigne (1974).

Calculation and data analysis

Statistical analysis was performed using the XLStat software package (Addinsoft SARL, Paris, France). Results were expressed as mean values with their standard errors. Two-way ANOVA, coupled with the Newman–Keuls test, defined as $P<0·05$, was adopted to determine the main effects (potato and salt) and interaction.

Results

Food intake, body weight and plasma electrolytes

Food intake and body-weight gain did not differ significantly among the groups (Table 2). Arterial plasma concentrations of Na, K, Ca and Mg were unchanged.

Urine variables

As shown in Table 2, rats fed the WS diets excreted an acidic urine (about pH 5·5), whereas rats fed the CP diets excreted urine near pH 7·3. Urine alkalinisation by potato corresponded to a rise of cationic species excretion with a major contribution of K. Na excretion was about 5·5 mmol/d in the high-salt diets. K excretion (Table 2) was 4-fold greater in rats fed the CP diets than in rats fed the WS diets and it accounted for the near-totality of K intake. Urinary Ca excretion (Fig. 1 (A)) was significantly greater in rats fed the high-salt WS diet (0·11 mmol/d) than in those fed the low-salt WS diet. Moreover, potato reduced significantly Ca excretion down to 0·05 mmol/d in the high-salt CP diet compared with the high-salt WS group. Thus, Ca excretion represented about 17 % of the daily absorbed Ca in rats fed the high-salt WS diet compared with 3–5 % in those fed the low-salt WS and the CP diets. As shown in Fig. 1 (B), urinary Mg excretion in the WS diets was significantly increased, whereas potato consumption counteracted this effect. Mg excretion represented about 46 and 63 % of the daily absorbed Mg in rats fed the WS diet compared with 21–28 % in those fed the CP diets. Urinary ammonium (Table 2) was significantly increased in the high-salt WS diet compared with the others. Chloride excretion was about 2 mmol/d in rats fed the low-salt diets, and in the range of 6·15–6·62 mmol/d in diets supplemented with NaCl. Urinary citrate (Fig. 2 (A)) and α-ketoglutarate (Fig. 2 (B)) are recognised as extremely sensitive to acid–base conditions in kidneys. They were practically undetectable in acidic urines (control and NaCl diets), whereas they were present in substantial amounts in the CP diets, in the range of 150–300 μmol/d. Phosphate (Table 2) and oxalate (Fig. 2 (C)) excretions were markedly depressed with the CP diets.

Caecal variables

The CP diets significantly enlarged the caecum (+84 to +91 %) compared with the WS diets (Table 3). The caecal wall weight was relatively proportional to the caecal weight (about 30 %). The caecal pH was near neutrality in rats fed the WS diets whereas an acidification of the caecal pH (about 5·7) occurred in rats fed the CP diets. The caecal SCFA pool was larger in rats fed the CP diets, in the range of 447–491 μmol/caecum, than in those fed the WS diets (146–159 μmol/caecum). The acetate molar ratio was particularly high in rats fed the WS diets, and the CP diets increased the propionate molar ratio. The butyrate concentration in the caecum of low-salt WS fed rats was about 7·2 mmol/l and was slightly enhanced in rats fed the low-salt CP diet (11 mmol/l). The activation of caecal fermentations in rats fed the CP diets contributes to enhance DM faecal excretion.

Influence of diets on mineral absorption

The Ca and Mg intakes were very similar for all diets, in the range 1659 to 1817 μmol/d. However, apparent Ca absorption was significantly greater in rats fed CP diets, about 53–56 % of ingested Ca, than in those fed the WS diets, about 39 % (Table 4). In rats fed the WS diets, 37–41 % of the dietary Mg was apparently absorbed, whereas CP diets significantly enhanced apparent Mg absorption up to 60 %.

Table 2. Food intake, body weight, plasma electrolytes and urinary parameters in rats fed the wheat starch (WS) or cooked potato (CP) diets
(Mean values with their standard errors for eight animals per group)

	WS				CP						
	Low salt		High salt		Low salt		High salt		ANOVA *P* value		
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	CP	Salt	CP × salt
Food intake (g/d)	17·9	0·4	18·7	0·6	18·8	0·4	19·1	0·3	NS	NS	NS
Body-weight gain (g/d)	5·7	0·3	5·7	0·3	4·6	0·3	4·9	0·3	NS	NS	NS
Plasma											
K (mmol/l)	3·64	0·09	3·69	0·07	3·79	0·11	3·81	0·05	NS	NS	NS
Na (mmol/l)	133	0·4	133	1·8	129	1·6	129	1·0	NS	NS	NS
Ca (mmol/l)	2·61	0·06	2·77	0·09	2·81	0·08	2·74	0·10	NS	NS	NS
Mg (mmol/l)	0·75	0·01	0·8	0·03	0·81	0·02	0·76	0·02	NS	NS	NS
Urine											
pH	5·51[b]	0·03	5·53[b]	0·03	7·26[a]	0·02	7·36[a]	0·15	<0·001	NS	NS
Volume (ml)	16·0[c]	1·9	24·9[b]	1·9	48·3[a]	1·9	48·8[a]	2·7	<0·001	NS	NS
NH_4 (mmol/d)	1·11[b]	0·11	1·59[a]	0·10	0·96[b]	0·09	0·97[b]	0·10	NS	<0·05	NS
K (mmol/d)	1·38[b]	0·09	1·91[b]	0·25	6·8[a]	0·15	7·06[a]	0·26	<0·001	NS	NS
Na (mmol/d)	1·37[b]	0·09	5·81[a]	0·47	1.14[b]	0·03	5·30[a]	0·20	NS	<0·001	NS
Cl (mmol/d)	2·11[b]	0·10	6·62[a]	0·39	1·91[b]	0·21	6·16[a]	0·31	NS	<0·001	NS
PO_4 (mmol/d)	1·09[b]	0·05	1·28[a]	0·08	0·48[c]	0·03	0·56[c]	0·04	<0·001	<0·05	NS

[a,b,c] Mean values within a row with unlike superscript letters were significantly different ($P<0·05$).

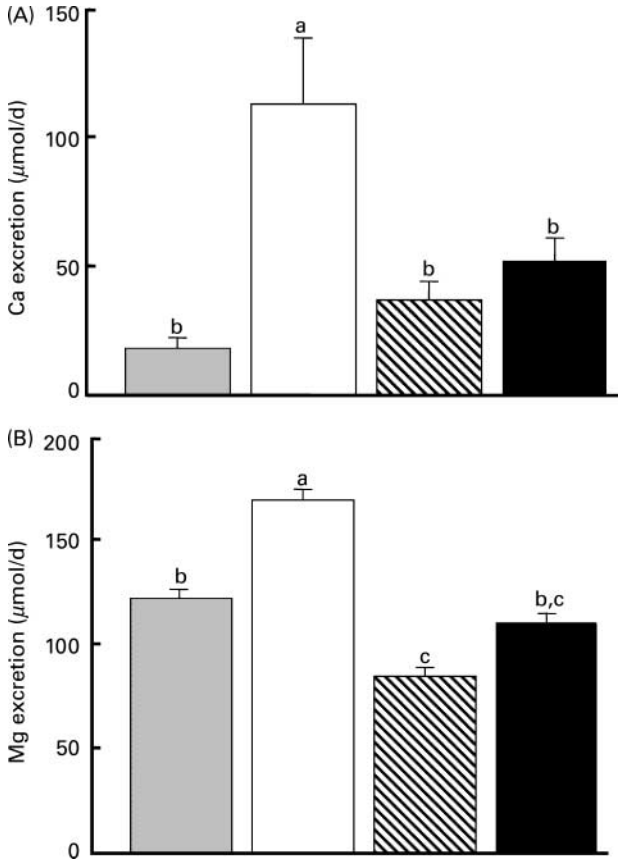

Fig. 1. Urinary Ca (A) and Mg (B) excretion (μmol/d) in rats fed low-salt wheat starch (WS; ▒), high-salt WS (□), low-salt cooked potato (CP; ▨) or high-salt CP (■) diets for 3 weeks. Values are means for eight rats per group, with standard errors represented by vertical bars. Urinary Ca excretion was affected by NaCl ($P<0{\cdot}01$) with a significant interaction between NaCl and CP ($P<0{\cdot}05$). Mg excretion was affected not only by CP ($P<0{\cdot}01$) but also by NaCl ($P<0{\cdot}01$). [a,b,c] Mean values with unlike letters were significantly different ($P<0{\cdot}05$).

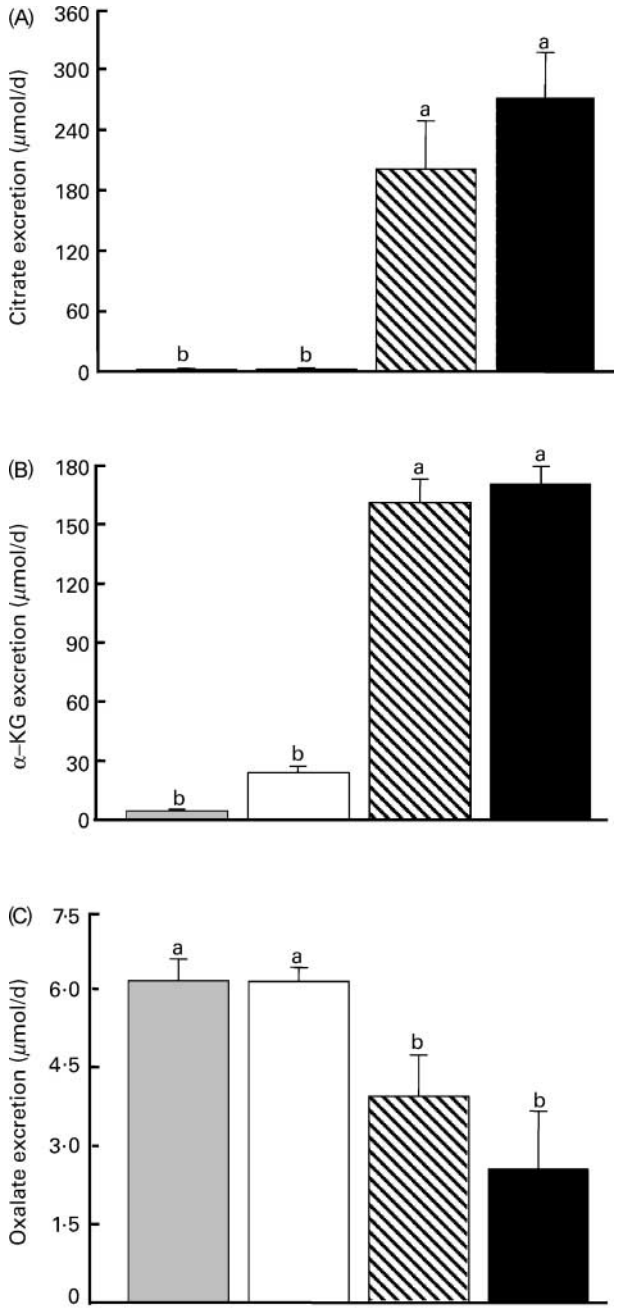

Fig. 2. Urinary citrate (A), α-ketoglutarate (α-KG; B) and oxalate (C) excretion (μmol/d) in rats fed low-salt wheat starch (WS; ▒), high-salt WS (□), low-salt cooked potato (CP; ▨) or high-salt CP (■) diets for 3 weeks. Values are means for eight rats per group, with standard errors represented by vertical bars. Citrate, α-KG and oxalate excretion were affected only by CP ($P<0{\cdot}001$). [a,b] Mean values with unlike letters were significantly different ($P<0{\cdot}05$).

Discussion

The present study was conducted to determine whether potato consumption is effective on acid–base homeostasis and to prevent mineral losses induced by a high salt load.

The present results indicate that CP diets delivered a substantial alkali load, with a significant rise in urinary pH and a decrease in ammonium excretion as previously shown with potassium citrate or potassium bicarbonate administration in men and experimental animals (Sakhaee *et al.* 1991; Sabboh *et al.* 2005).

The present results also confirm that short-term intake of salt supplements (20 g/kg) significantly increases urinary Ca and Mg excretion, as shown in experimental animals (Whiting & Cole, 1986; Shortt & Flynn, 1991; Chan & Swaminathan, 1994; Creedon & Cashman, 2000) and human subjects (Itoh & Suyama, 1996; Lietz *et al.* 1997; Ginty *et al.* 1998). It is well known that when dietary NaCl is increased, the fractional reabsorption of Na is decreased, leading to a parallel reduction in Ca reabsorption (Shortt & Flynn, 1990, 1991). The dependence of urinary Ca excretion on Na excretion has been attributed to the existence of linked or common reabsorption pathways for these cations in the convoluted portion of the proximal tubule and in the loop of Henle (Antoniou *et al.* 1969). Furthermore, it has been shown that by replacing dietary NaCl with an equimolar amount of dietary $NaHCO_3$, it is possible to significantly reduce hypercalciuria (Kurtz & Morris, 1985; Lemann *et al.* 1991; Maurer *et al.* 2003). There is evidence that Cl in excess has to be eliminated extensively by kidneys to maintain a plasma Na:Cl ratio of about 1·4. Thus, the unmetabolisable anion Cl, representing fixed

Table 3. Caecal variables in rats fed the wheat starch (WS) or cooked potato (CP) diets
(Mean values with their standard errors for eight animal per group)

	WS				CP				ANOVA *P* value		
	Low salt		High salt		Low salt		High salt				
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	CP	Salt	CP×salt
Caecum (g)	2·34[b]	0·28	2·30[b]	0·24	4·31[a]	0·27	4·47[a]	0·33	<0·001	NS	NS
Caecum wall (g)	0·77[b]	0·02	0·77[b]	0·05	1·30[a]	0·07	1·27[a]	0·06	<0·001	NS	NS
Caecal pH	7·02[a]	0·07	7·02[a]	0·03	5·69[b]	0·12	5·75[b]	0·10	<0·001	NS	NS
SCFA pool (μmol/caecum)	146[b]	30	159[b]	23	491[a]	39	447[a]	56	<0·001	NS	NS
Molar ratio* (Ac:Pr:Bu)		76:17:7		74:20:6		60:33:7		59:37:4			
DM excretion (g/d)	0·49[b]	0·03	0·52[b]	0·05	0·99[a]	0·03	1·07[a]	0·04	<0·001	NS	NS

Ac, acetate; Pr, propionate; Bu, butyrate.
[a,b] Mean values within a row with unlike superscript letters were significantly different ($P<0·05$).
*Molar ratio (example for Ac) = (Ac × 100)/(Ac + Pr + Bu).

acidity, might itself play a role in mineral excretion (Massey & Whiting, 1996; Barzel, 1997).

It has also been shown that acidosis may also affect Mg reabsorption through a direct effect of protons on distal Mg transport (Dai *et al.* 1997, 2001). The present observations strengthen the view of a major role of urinary Mg excretion in the regulation of acid–base homeostasis; and its role may be more relevant than that of Ca, as shown previously (Sabboh *et al.* 2005). The present study also shows that, by replacing WS with CP in a high-salt diet, it is possible to counteract the hypercalciuric and hypermagnesuric effects of NaCl. Many studies have shown that K supplementation reduces whereas K deprivation elevates urinary Ca excretion (Lemann *et al.* 1989, 1991). Moreover, the addition of oral potassium citrate to a high-salt diet may prevent a rise of urine Ca excretion (Sellmeyer *et al.* 2002). $KHCO_3$ is well known to reduce urinary Ca excretion by the neutralisation of endogenous acid production (Sebastian *et al.* 1994; Frassetto *et al.* 2000). As K organic salts metabolism yields virtually to $KHCO_3$, they have the potential to neutralise endogenously produced acidity (Sellmeyer *et al.* 2002). They could thus stimulate the renal tubular reabsorption of cations since urinary Ca and Mg excretion seems to commensurate with endogenous acid production (Dai *et al.* 1997, 2001; Lemann, 1999; Yeh *et al.* 2003). Furthermore, K seems to have a direct impact on the kidney to promote Ca reabsorption (Brunette *et al.* 1992).

Potato has also to be considered as a source of fibres which may influence the food efficiency of the diet. The CP diet led to a rise in faecal DM excretion, which could explain the difference in the weight gain between the CP and WS diets. Moreover, the microbial breakdown of potato fibres in the large intestine produces large amounts of SCFA accompanied by a lowering of the caecal content pH as previously shown (Mathers & Dawson, 1991). It is well known that acidic fermentation may influence the intestinal absorption of minerals, especially Ca and Mg, by increasing their solubility and their transport across the cell membrane (Nellans & Goldsmith, 1981; Lutz & Scharrer, 1991; Trinidad *et al.* 1993; Coudray *et al.* 2003). It is also conceivable that there are probably interesting interactions between fermentable fibres and potassium citrate which might have additive alkalinising consequences.

These observations are particularly important since a negative Ca balance generally results in a compensatory release of minerals (Ca, Mg) from bone tissue, liable to alter the structural integrity of the skeleton, leading in man to various forms of osteopenia and osteoporosis (Buclin *et al.* 2001). The skeleton has also been involved in the buffering of an acid load through the release of hydroxyde (OH^-) and phosphate (PO_4^{3-}) anions, along with Ca cations (Ca^{2+}). In this context, consumption of plant foods rich in K alkaline salts, such as potato, may be of interest in the maintenance of mineral reserves and, in the long term, of bone mass (New *et al.* 1997; Macdonald *et al.* 2004, 2005).

Phosphate is the major buffer system in urine and its excretion is increased during acidosis, probably as a result of a decrease of the preferentially transported form (HPO_4^{2-}) together with a direct effect of luminal pH on the apical phosphate carrier in the proximal tubule (Ambuhl *et al.* 1998). Sebastian *et al.* (1994) have shown that administration of

Table 4. Daily total intake (TI), faecal excretion (FE) and apparent absorption of calcium and magnesium in rats fed wheat starch (WS) or cooked potato (CP) diets

(Mean values with their standard errors for eight animal per group)

	WS				CP				ANOVA *P* value		
	Low salt		High salt		Low salt		High salt				
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	CP	Salt	CP × × salt
Ca											
TI (μmol/d)	1659	38	1798	65	1738	37	1817	32	NS	NS	NS
FE (μmol/d)	1016[a]	90	1117[a]	135	764[a]	24	862[a]	55	<0·05	NS	NS
TI−FE (μmol/d)	643[b]	72	681[b]	104	974[a]	44	955[a]	66	<0·001	NS	NS
Apparent absorption (%)	38·7		37·9		56·0		52·6				
Balance (μmol/d)	625[b]	71	568[b]	126	936[a]	51	902[a]	67	<0·001	NS	NS
Mg											
TI (μmol/d)	649	15	683	26	680	14	693	12	NS	NS	NS
FE (μmol/d)	383[a]	35	428[a]	50	277[b]	10	300[b]	17	<0·001	NS	NS
TI−FE (μmol/d)	267[b]	24	256[b]	35	404[a]	14	393[a]	13	<0·001	NS	NS
Apparent absorption (%)	41·1		37·5		59·4		56·7				
Balance (μmol/d)	145[b]	24	96[b]	41	319[a]	16	280[a]	14	<0·001	NS	NS

[a,b] Mean values within a row with unlike superscript letters were significantly different ($P<0·05$).

potassium bicarbonate improved P balance. In this view, phosphate excretion was significantly reduced in rats fed the potato diets, suggesting a better retention of this anion.

Citrate excretion was strongly enhanced in rats fed potato diets. Addition of fruits and vegetables to the diet have been shown to increase citrate excretion and to decrease calcium oxalate relative saturation in stone formers (Meschi *et al.* 2004). A significant decrease in net acid excretion was also observed upon potassium citrate supplementation, and this was parallel to a significant increase of urine citrate (Marangella *et al.* 2004). It appears that the major effect of dietary citrate on urinary citrate excretion is attributable to its metabolism in alkali (Sakhaee *et al.* 1991). Citrate is a characteristic renal metabolite which is recognised as a 'window on renal metabolism' (Simpson, 1983). Renal handling of citrate takes place in the proximal tubule across an Na-dependent dicarboxylate transporter (NADC-1) that reabsorbs a variety of Krebs cycle intermediates such as α-ketoglutarate and succinate (Pajor, 1999). Decrease in luminal pH has an effect on citrate reabsorption, probably due to a change in concentration of the preferentially transported ionic species, citrate^{2-} (Brennan *et al.* 1988), and in NADC-1 abundance (Aruga *et al.* 2000). Citrate metabolism in renal tubular cells is also affected by urinary pH, with adaptive changes in the activity of cytosolic ATP citrate lyase and mitochondrial aconitase (m-aconitase) (Melnick *et al.* 1996, 1998). Previous studies have also indicated that urinary citrate could act as a potent endogenous stone formation inhibitor by chelating Ca and inhibiting Ca precipitation, as well as Ca oxalate crystallisation (Harvey *et al.* 1985). Thus, the alkali load provided by potato consumption associated with the increase in urinary pH leads to the rise in urinary citrate excretion which may contribute to prevent the risk of kidney-stone diseases. Potato contains small amounts of oxalate, which is partially absorbed, whereas urinary oxalate might come mainly from endogenous synthesis. Potato consumption seems effective to lower this endogenous production.

In conclusion, potato consumption is often associated with bad food habits (high intake of fats and animal products, low intake of fruits and vegetables). In itself, potato, consumed with few fats and within the scope of a balanced diet, may exert, through its high micronutrient content (potassium citrate, vitamin C, fibres), a diversity of interesting effects on health (Ruano-Ravina *et al.* 2002). In the present study, potato appears to be effective in alkalinising urine and ameliorating Ca and Mg balance, in part by counteracting various adverse effects of a high NaCl load proper to major Western diets, such as excessive mineral wasting. Increasing the consumption of plant foods, sources of alkali agents mainly constituted by organic salts of K and Mg, may prove to be a practical means to maintain cation reserves in the body and, in the long term, to protect bone, notably in individuals at risk of developing osteoporosis (New *et al.* 1997; Macdonald *et al.* 2004, 2005). In this context further studies, in human subjects, should be conducted in order to identify the extent of the effect of potato on bone health (New, 2003).

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