British Journal of Nutrition (2006), **95**, 650–653

Nutrition Discussion Forum

Health effects of sulphate

The study of Brandolini *et al.* (2005) entitled 'Higher calcium urinary loss induced by a calcium sulphate-rich mineral water intake than by milk in young women' and published in the *British Journal of Nutrition* raises a critical point, as urinary Ca losses account for 50 % of the variability in Ca retention (NIH Consensus Development Panel, 1994). However, the article deserves comment regarding the authors' hypotheses in four main areas: (i) the unchanged intestinal absorption and faecal excretion; (ii) sulphate intake, metabolism and acidogenic effect; (iii) the potential effects of unbalanced nutrient intakes and the mechanisms proposed to explain the results on urinary Ca loss reported; and (iv) the long-term effects on bone of the $CaSO_4$-rich mineral water studied.

Unchanged intestinal absorption and faecal excretion

The authors correctly explained that the measurement of urinary Ca loss is sufficient when the intestinal absorption and the endogenous faecal loss do not vary. The assumption of an unchanged Ca intestinal absorption is not supported by studies showing that carbohydrate significantly increases intestinal absorption of minerals, including Ca (Bei *et al.* 1986; Heaney *et al.* 1989; Holbrook *et al.* 1989; Brink & Beynen, 1992, Sabatier *et al.* 2002). Guéguen expressed an opposite opinion when showing in growing pigs that 'calcium restriction may have enhanced some milk properties to stimulate calcium absorption' (Pointillart *et al.* 2000). The need to assess Ca absorption has been mentioned repeatedly (Barger-Lux *et al.* 1995; Heaney *et al.* 1999; Wood, 2000) in order to take into account large individual and daily variations in the absorption (Wolf *et al.* 2000; Wigertz *et al.* 2005). Decreased Ca absorption was shown to be an important risk factor for hip fracture (Ensrud *et al.* 2000) and Ca absorption efficiency accounts more for the variability in Ca balance than does Ca intake itself (Heaney, 2001). For sulphate, fractional Ca absorption was shown in women to be significantly higher from $CaSO_4$-fortified bread than from milk (Martin *et al.* 2002).

An inverse relationship between urinary Ca and endogenous faecal excretion was shown for faecal Ca loss, suggesting that faecal loss functions as an unregulated drain on the Ca economy (Heaney *et al.* 1999). A comparative study of isolated soya and milk proteins showed a higher endogenous faecal loss with soya and a lower urinary Ca excretion compared with milk protein, but bone metabolism and Ca retention were not significantly affected by the dietary regimen (Spence *et al.* 2005).

To assess 24 h urinary Ca, the subjects must be under rigorous metabolic balance and consuming a constant diet to reduce day-to-day variations among individuals (Peacock, 1988), particularly when small differences are expected. The unchanged intestinal absorption and faecal excretion, which would validate the measurement of calciuria, cannot be accepted in the experimental conditions of the study of Brandolini *et al.*

Sulphate intake, metabolism and acidogenic effect

Another of the authors' hypotheses is that drinking $CaSO_4$-rich water will increase urinary Ca excretion due to an acidification of urine by sulphate. It was indicated that milk provides four times less sulphate than water but this ratio seems underestimated, as 1 litre of mineral water and 0·4 litres of milk provide 1180 mg and 40 mg sulphate, respectively (Florin *et al.* 1993). Common foods, vegetables, beverages and cereal products contain high amounts of sulphate, sometimes in concentrations similar to or higher than those in the water studied (12 mmol/l or kg), so that total sulphate intakes may exhibit important day-to-day variations in the range of 7·5-fold. The analysis of urinary sulphate as a marker of sulphate absorption in evaluating the effects due to high intakes of sulphate is recommended (Institute of Medicine, 2004). The absence of data on sulphate intake and urinary excretion in Brandolini *et al.*'s study precludes formulation of any relationship between sulphate and the observed Ca loss, and – even more – the extrapolation of bone mineral mass loss over 30-year period and its eventual dramatic effect on bone fractures.

The authors' hypothesis is based on previous animal studies showing that an acidogenic action of sulphate increased Ca excretion (Guéguen and Besançon, 1972; Whiting & Draper, 1980, 1981). In the first study either $CaSO_4$ or $CaCO_3$ was added to the diet of sheep, providing 80 % of Ca intakes. A significantly higher Ca excretion in urine but an unexplained lower bone resorption was observed with sulphate. There were no data on urinary sulphate excretion or on other parameters suggesting an acidification. The calculated daily consumption of sulphate was 12·8 g, corresponding to 16 g (166 mmol) for a man weighing 70 kg. This load is about thirteen times the amount of sulphate ingested by the volunteers in Brandolini *et al.*'s study. Studies performed in rats (Causeret & Hugot, 1958) and chickens (Waldroup *et al.* 1964) showed that $CaSO_4$ was as well used as $CaCO_3$ and calcium gluconate. Drinking water containing 5 g Na_2SO_4/l does not increase urinary Ca in heifers (Weeth & Hunter, 1971). The other rat studies mentioned (Whiting & Draper, 1980, 1981; Whiting & Cole, 1986, 1987) showed for large doses of sulphate (687 mmol; 66 g for a man weighing 70 kg) that urinary pH was significantly acidified, even more with chloride than for sulphate, and that after a transient increased Ca excretion, Ca balance was not significantly different between the sulphate and the control group. The authors concluded that their initial hypothesis that sulphate may produce a higher calciuric effect than chloride was not confirmed and was attributed to a lower absorption of sulphate (Whiting & Cole, 1987). In contrast with these animal studies, there was no change in urinary pH in the study of Brandolini *et al.* as well as in other studies on human volunteers.

In man, a high degree of correlation exists between the increase in urinary Ca and the increase in renal excretion of both total acid and

sulphate which accompanies increased levels of protein intakes or when sulphur-containing amino acids are added to the diet. However, it was shown that Ca reabsorption was not affected by urinary sulphate level (Block *et al.* 1980) and that there was no correlation between urinary Ca and urinary sulphate excretion in premature infants (Greer *et al.* 1986). In a cross-over study (Roux *et al.* 2004), the administration for 28 d of 1 litre of a mineral water rich in either $Ca(HCO_3)_2$ (606 mg Ca^{2+}; 2179 mg HCO_3^-; 4 mg SO_4^{2-}) or $CaSO_4$ (560 mg Ca^{2+}; 291 mg HCO_3^-; 1550 mg SO_4^{2-}) resulted in no significant changes in urine Ca:creatinine ratio and sulphate concentrations. Roux *et al*. commented that, 'it has been suggested that sulphate may induce an increase urine calcium excretion . . . but although non significant, the larger concentration of sulphate with the calcium sulphate-rich water did not lead to an excess of the urinary calcium'. These results obtained with a mineral water with a higher sulphate level do not agree with those of Brandolini *et al*. Ca utilization from 400 mg supplement as lactate, gluconate, sulphate and carbonate was reported in young women (Patton and Sutton, 1952). Total sulphate daily intake of 960 mg was similar to the $CaSO_4$ intake of the study of Brandolini *et al*. Ca balance performed over 8 weeks showed the highest mean retention of Ca with sulphate, and Ca utilization for all these salts was similar and within the range of that reported for milk. This well-controlled study confirmed the results obtained 6 years earlier on $CaSO_4$ (Schroeder *et al.* 1946). In these studies, not mentioned by Brandolini *et al.* high sulphate intakes did not impair Ca utilization and balance calculated from urine and faecal excretion.

It has been advised that bone and mineral investigators should look at the acid–base effects of diet and use appropriate methods to quantify these effects (Barzel and Massey, 1998). As studies suggest an acidogenic action of sulphate with unchanged urinary pH, the evaluation of the potential renal acid load or the renal acid excretion through either a direct or an indirect measurement appears necessary (Remer and Manz, 1995; Remer *et al.* 2003). Even in a study with subjects in a metabolic unit for 63 d with controlled diet, Ca excretion in urine did not always correlate with urinary pH, total acid or titratable acid, and that acid output was not directly related to the sulphate excretion (Jourdan *et al.* 1980). The exchange of only a few foods can markedly alter the daily intake of acid equivalent (Remer and Manz, 1995). It is also important to mention that natural mineral waters are not pure solutions of $CaSO_4$ and when using the same water (Contrex) as Brandolini *et al.* other authors (Wynckel *et al.* 1997) expected a risk of alkalosis resulting from its high bicarbonate content (377 mg/l) and not acidosis as suggested by Brandolini *et al*. Available studies in human subjects do not support the hypothesis that an excess of sulphate causes more Ca to be lost in the urine (Guéguen and Pointillart, 2000) through an acidogenic effect, and the observation of a daily increased excretion of 20 mg Ca excess per day in urine may be explained by other uncontrolled parameters of the study.

The potential effects of unbalanced nutrient intakes

From the dietary records of all foods and drinks, Brandolini *et al*. calculated food intake in terms of energy, fibre, protein, Ca, Mg, Na and P, and from urine analyses, the urinary levels of Ca, Na and Mg. Intakes were significantly different for energy, protein, Na, P and Mg, as well as for fluid, when individuals received water or milk.

One of the significant differences in nutrient intakes reported in the study of Brandolini *et al*. is the higher P level in the milk period (848 *v*. 1243 mg/d; +46 %; $P=0{\cdot}0001$). P intakes do affect Ca absorption efficiency when Ca intake is low or adequate (Heaney & Recker, 1982; Spencer *et al.* 1984; Heaney, 2000*a*), but increase renal tubular reabsorption of Ca and thereby exert a hypocalciuric effect (Zemel *et al.* 1981; Zemel, 1988). Urinary phosphate was not reported in the study of Brandolini *et al.* but the daily loss of 20 mg Ca for a difference in P intake of 395 mg (5·0 mg Ca/100 g P) can be compared with a daily Ca loss of 51 mg for a difference in P intake of 897 mg (5·6 mg Ca/100 mg P; Fisler & Drenick, 1984). Thus the difference in P intake can account for all of the increased Ca loss in the Contrex period compared with the milk period, but this hypocalciuric effect of P is offset by the hypercalciuric effect of proteins (Lemann, 1999; Weaver *et al.* 1999; Massey, 2003).

Another difference is the unbalanced K intake, which is not discussed by Brandolini *et al.* although they mention that the simultaneous intake of P and K favours absorption and bone retention and that it is well known that K intake has an alkalogenic effect as shown in the potential renal acid load used to assess the acidity of foods and diets. The beneficial effects of dietary K on bone health are well established and its intake was able to explain 9 % of the variation in total-body bone mineral density and 12 % of the variation in spine bone mineral density in male subjects (Whiting *et al.* 2002). With a K concentration of 173 mg/100 g milk (Harmon, 1994), the ingestion of 0·4 litres of milk in the milk group leads to a higher K intake of 692 mg (17·7 mmol/d) in contrast with the water group, which receives only 3 mg K/d. A significant inverse relationship between the changes in urinary Ca and urinary K has been observed. From published studies (Lemann *et al.* 1993; Rafferty *et al.* 2005), one can expect a reduced urinary Ca excretion in the milk group ranging from 6·2 mg up to 17 mg (Sakhee *et al.* 1983; Buclin *et al.* 2001; Rafferty *et al.* 2005). Thus, increased urinary Ca loss reported in the water group may be explained exclusively by the higher K intake from milk.

Finally, fluid intake was apparently not controlled as shown by the significant 11·9 % higher urinary excretion of the Contrex group, confirmed by an 11·5 % significantly lower urinary creatinine concentration. Fluid intakes must be strictly controlled in studies performed on mineral balance (Whiting, 1990; Couzy *et al.* 1995; Wynckel *et al.* 1997; Guillemant *et al.* 2000; Heaney & Rafferty, 2001; Martini & Wood, 2002).

An increase in urine volume has been reported in children treated with K while the fluid intake was not controlled (Duff & Whiting, 1998) and the authors questioned the possible effects of an increase in Ca excretion caused by this higher urinary volume. In the study of Brandolini *et al.* the mean concentrations of Ca were 0·229 and 0·219 mmol/l for the Contrex and the milk group, respectively, and the excess diuresis of 160 ml urine in the Contrex group due to the uncompensated fluid intake of 1 litre of water and 0·4 litres of milk leads to an excess urinary excretion of 14·7 mg Ca. The unbalanced fluid intake may thus explain the Ca excess loss reported.

The higher K intake in the milk period and the higher water intake in the water period could explain a 30–40 mg lower calciuria observed by Brandolini *et al*. for the milk period.

Long-term effects on bone of the calcium sulphate-rich mineral water studied

The authors wisely indicate that there is a need to confirm the dramatic long-term effects of sulphate on bone health that they

predicted. Their study was conducted in France and the $CaSO_4$-rich mineral water they studied (Contrex) is one of the most important bottled waters on the market. Ca^{2+} and SO_4^{2-} contents in mg/l of the main mineral waters sold in the France are: Evian (78, 10); Contrex (486, 1187); Vittel (202, 336); Volvic (10, 7). The high Ca^{2+} contents of SO_4^{2-}-rich waters explain why Contrex and Vittel are the only waters on the market contributing significantly to Ca intakes for the French population. This contribution was evaluated in the SU.VI.MAX (Supplémentation en VItamines et Minéraux AntioXydants study) cohort, which included 12 735 subjects followed for up to 8 years. Four matched case–control groups of 166 regular drinkers of Contrex, Vittel, Volvic and Valvert and tap water showed that Ca intake of regular drinkers of Contrex represents 18–20 % of total intake in men and women (Galan *et al.* 2002).

To evaluate the long-term impact of these mineral waters on bone mineral mass and the risk of fracture, the coordinators of the EPIDOS Study Group were contacted. This large, 2-year, multi-centre observational study of risk factors for hip fractures in 7575 women over 75 years of age was performed in five French towns between 1992 and 1994. Women receiving treatments likely to interfere with Ca metabolism were excluded and 4434 women aged 80·7 (SD 3·8) years were selected. Bottled water brands and the volume drunk every day were carefully recorded, enabling evaluation of the relationship between Ca intake from Contrex and Vittel and bone density. This study showed that after adjustment for the main variables influencing bone density, an increase of 100 mg Ca/d from the $CaSO_4$-rich waters Contrex and Vittel was associated with a 0·5 % increase in femoral bone density, while a non-significantly different but lower increase in bone density of 0·2 % was observed for Ca from other sources, including milk and dairy products (Aptel *et al.* 1999). The higher effect of Ca from water on bone is tentatively explained by the drinking pattern with fractional Ca absorptions inversely related with dose (Heaney & Recker, 1986) and a prolonged inhibition of parathyroid hormone secretion (Guillemant *et al.* 2002). From this long-term cross-sectional study, a woman with a daily consumption of 1 litre of a $CaSO_4$-rich mineral water containing 400 mg Ca would be estimated to have an equivalent bone density to that of a woman 7 years younger drinking a low Ca-content water (Aptel *et al.* 1999). This publication was not mentioned by Brandolini *et al*. but was listed by Heaney (2000*b*) in the 135 papers published between 1975 and 2000, the only one using mineral water as Ca source. These effects are due exclusively to the two main $CaSO_4$-rich mineral waters drunk in France, particularly those studied by Brandolini *et al*.

Conclusion

The rise in urinary Ca with milk has been suggested to be a reflection of absorption and not in any direction a negative effect (Heaney and Rafferty, 2001), and the same explanation applies for $CaSO_4$-rich mineral water. The main nutritional objective is to get adequate daily Ca intakes from food, supplements and also water, which is never proposed by US nutritionists (Weaver *et al.* 1999; Weinsier & Krumdieck, 2000) and may be appropriate for those people who cannot reach these recommendations by other means. The dramatically inadequate Ca consumption in different age groups is illustrated in the EPIDOS cohort of elderly women, where the range of dietary Ca intake was 49–3059 mg/d and 0–1584 mg/d was provided from water. A survey performed in children aged 8 years, the range of Ca intakes was 283–3754 mg/d (Jones *et al.* 2001). These studies demonstrate that the priority in Ca nutrition is to reach individual adequate Ca intakes before establishing the optimal conditions for its utilization.

Maurice J. Arnaud
Nestlé Ltd
Avenue Nestlé 55
CH-Vevey
Switzerland
Maurice.Arnaud@nestle.com
DOI: 10.1079/BJN20051652

References

Aptel I, Cance-Rouzeau A & Grandjean H for the EPIDOS Study Group (1999) Association between calcium ingested from drinking water and femoral bone density in elderly women: evidence from the EPIDOS cohort. *J Bone Miner Res* **14**, 829–833.

Barger-Lux MJ, Heaney RP, Lanspa SJ, Healy JC & DeLuca HF (1995) An investigation of sources of variation in calcium absorption efficiency. *J Clin Endocrinol Metab* **80**, 406–411.

Barzel US & Massey LK (1998) Excess dietary protein can adversely affect bone. *J Nutr* **128**, 1051–1053.

Bei X, Wood RJ & Rosenberg ICH (1986) Glucose polymer increases jejunal calcium, magnesium, and zinc absorption in humans. *Am J Clin Nutr* **44**, 244–247.

Block GD, Wood RJ & Allen LH (1980) A comparison of the effects of feeding sulfur amino acids and protein on urine calcium in man. *Am J Clin Nutr* **33**, 2128–2136.

Brandolini M, Guéguen L, Boirie Y, Rousset P, Bertière M-C & Beaufrère B (2005) Higher calcium urinary loss induced by a calcium sulphate-rich mineral water intake than by milk in young women. *Br J Nutr* **93**, 225–231.

Brink EJ & Beynen AC (1992) Nutrition and magnesium absorption: a review. *Prog Food Nutr Sci* **16**, 125–162.

Buclin T, Cosma M, Appenzeller M, Jacquet AF, Decosterd LA, Biollaz J & Burckhardt P (2001) Diet acids and alkalis influence calcium retention in bone. *Osteoporos Int* **12**, 493–499.

Causeret J & Hugot D (1958) Influence du taux calcique du regime alimentaire sur l'utilisation physiologique du calcium de divers sels. I. – Étude comparative du carbonate, du sulfate et du lactate de calcium. *Ann Zootech* **7**, 69–77.

Couzy F, Kastenmayer P, Vigo M, Clough J, Munoz-Box R & Barclay DV (1995) Calcium bioavailability from a calcium- and sulfate-rich mineral water, compared with milk, in young adult women. *Am J Clin Nutr* **62**, 1239–1244.

Duff TL & Whiting SJ (1998) Calciuric effects of short-term dietary loading of protein, sodium chloride and potassium citrate in prepubescent girls. *J Am Coll Nutr* **17**, 148–154.

Ensrud KE, Duong T, Cauley JA, Heaney RP, Wolf RL, Harris E & Cummings SR (2000) Low fractional calcium absorption increases the risk for hip fracture in women with calcium intake. *Ann Intern Med* **132**, 345–353.

Fisler JS & Drenick EJ (1984) Calcium, magnesium, and phosphate balances during very low calorie diets of soy or collagen protein in obese men: comparison to total fasting. *Am J Clin Nutr* **40**, 14–25.

Florin THJ, Neale G, Goretski S & Cummings JH (1993) The sulphate content of foods and beverages. *J Food Compost Anal* **6**, 140–151.

Galan P, Arnaud MJ, Delabroise A-M, Preziosi P, Bertrais S, Franchisseur C, Maurel M, Favier A & Hercberg S (2002) Contribution of mineral waters to dietary calcium and magnesium intake in the French SU.VI.MAX cohort. *J Am Diet Assoc* **102**, 1658–1662.

Greer FR, McCormick A & Loker J (1986) Increased urinary excretion of inorganic sulfate in premature infants fed bovine milk protein. *J Pediatr* **109**, 692–697.

Guéguen L & Besançon P (1972) Influence des sulfates sur le métabolisme phospho-calcique I. – Utilisation du sulfate de calcium par le mouton. *Ann Biol Anim Biochim Biophys* **12**, 589–598.
Guéguen L & Pointillart A (2000) The bioavailability of dietary calcium. *J Am Coll Nutr* **19**, 119S–136S.
Guillemant J, Le H-T, Accarie C, Tezenas du Montcel S, Delabroise A-M, Arnaud MJ & Guillemant S (2000) Mineral water as a source of dietary calcium: acute effects on parathyroid function and bone resorption in young men. *Am J Clin Nutr* **71**, 999–1002.
Guillemant J, Accarie B, de la Guéronnière V & Guillemant S (2002) Calcium in mineral water can effectively suppress parathyroid function and bone resorption. *Nutr Res* **22**, 901–910.
Harmon RJ (1994) Physiology of mastitis and factors affecting somatic cell counts. *J Dairy Sci* **77**, 2103–2112.
Heaney RP (2000*a*) Dietary protein and phosphorus do not affect calcium absorption. Am J Clin Nutr **72**, 758–761.
Heaney RP (2000*b*) Calcium, dairy product and osteoporosis. *J Am Coll Nutr* **19**, 83S–99S.
Heaney RP (2001) Nutrition and risk for osteoporosis. In *Osteoporosis*, 2nd ed, vol. 1 pp. 669–699 [R Marcus, D Feldman and J Kelsey, editors]. San Diego, CA: Academic Press.
Heaney RP & Rafferty K (2001) Carbonated beverages and urinary calcium excretion. *Am J Clin Nutr* **74**, 343–347.
Heaney RP & Recker RR (1982) Effects of nitrogen, phosphorus, and caffeine on calcium balance in women. *J Lab Clin Med* **99**, 46–55.
Heaney RP & Recker RR (1986) Distribution of calcium absorption in middle-aged women. *Am J Clin Nutr* **43**, 299–305.
Heaney RP, Smith KT, Recker RR & Hinders SM (1989) Meal effects on calcium absorption. *Am J Clin Nutr* **49**, 372–376.
Heaney RP, Recker RR & Ryan RA (1999) Urinary calcium in perimenopausal women: normative values. *Osteoporos Int* **9**, 13–18.
Holbrook JT, Smith JC & Reiser S (1989) Dietary fructose or starch: effects on copper, zinc, iron, manganese, calcium and magnesium balance in humans. *Am J Clin Nutr* **49**, 1290–1294.
Institute of Medicine (2004) Sulfate. In *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, pp. 424–448 Washington, DC: The National Academies Press.
Jones G, Riley MD & Whiting S (2001) Association between urinary potassium, urinary sodium, current diet, and bone density in prepubertal children. *Am J Clin Nutr* **73**, 839–844.
Jourdan M, Glock C, Margen S & Bradfield RB (1980) Sulphate, acid-base, and mineral balances of obese women during weight loss. *Am J Clin Nutr* **33**, 236–243.
Lemann J (1999) Relationship between urinary calcium and net acid excretion as determined by dietary protein and potassium: a review. *Nephron* **81**, Suppl. 1, 18–25.
Lemann J, Pleuss JA & Gray RW (1993) Potassium causes calcium retention in healthy adults. *J Nutr* **123**, 1623–1626.
Martin BR, Weaver CM, Heaney RP, Packard PT & Smith DL (2002) Calcium absorption from three salts and $CaSO_4$-fortified bread in premenopausal women. *J Agric Food Chem* **50**, 3874–3876.
Martini L & Wood RJ (2002) Relative bioavailability of calcium-rich dietary sources in the elderly. *Am J Clin Nutr* **76**, 1345–1350.
Massey LK (2003) Dietary animal and plant protein and human bone health: a whole foods approach. *J Nutr* **133**, 862S–865S.
NIH Consensus Development Panel (1994) Optimal calcium intake. *JAMA* **272**, 1942–1948.
Patton MD & Sutton TS (1952) The utilization of calcium from lactate, gluconate, sulfate and carbonate salts by young college women. *J Nutr* **48**, 443–452.
Peacock M (1988) Renal excretion of calcium. In *Calcium in Human Biology*, pp. 125–169 [BEC Nordin, editor]. London: Springer-Verlag.
Pointillart A, Coxam V, Sève B, Clin C, Lacroix CH & Guéguen L (2000) Availability of calcium from skim milk, calcium sulfate and calcium carbonate for bone mineralization in pigs. *Reprod Nutr Dev* **40**, 49–61.
Rafferty K, Davies KM & Heaney RP (2005) Potassium intake and the calcium economy. *J Am Coll Nutr* **24**, 99–106.
Remer T & Manz F (1995) Potential renal acid load of foods and its influence on urine pH. *J Am Diet Assoc* **95**, 791–797.
Remer T, Dimitriou T & Manz F (2003) Dietary potential renal acid load and renal net acid excretion in healthy, free-living children and adolescents. *Am J Clin Nutr* **77**, 1255–1260.
Roux S, Baudoin C, Boute D, Brazier M, de la Guéronnière V & de Vernejoul M-C (2004) Biological effects of drinking-water mineral composition on calcium balance and bone remodeling markers. *J Nutr Health Aging* **8**, 380–384.
Sabatier M, Arnaud MJ, Kastenmayer P, Rytz A & Barclay DV (2002) Meal effect on magnesium bioavailability from mineral water in healthy women. *Am J Clin Nutr* **75**, 65–71.
Sakhee K, Nicar M, Hill K & Pak CYC (1983) Contrasting effects of potassium citrate and sodium citrate therapies on urinary chemistries and crystallization of stone-forming salts. *Kidney Int* **24**, 348–352.
Schroeder LJ, Cahill WM & Smith AH (1946) The utilization of calcium in soybean products and other calcium sources. *J Nutr* **32**, 413.
Spence LA, Lipscomb ER, Cadogan J, Martin B, Wastney ME, Peacok M & Weaver CM (2005) The effect of soy protein and soy isoflavones on calcium metabolism in postmenopausal women: a randomised crossover study. *Am J Clin Nutr* **81**, 916–922.
Spencer H, Kramer L & Osis D (1984) Effect of calcium on phosphorus metabolism in man. *Am J Clin Nutr* **40**, 219–225.
Waldroup PW, Ammerman CB & Harms RH (1964) The utilization by the chick of calcium from different sources. *Poult Sci* **43**, 212–216.
Weaver CM, Proulx WR & Heaney R (1999) Choices for achieving adequate dietary calcium with a vegetarian diet. *Am J Clin Nutr* **70**, 543S–548S.
Weeth HJ & Hunter JE (1971) Drinking of sulfate-water by cattle. *J Anim Sci* **32**, 277–281.
Weinsier RL & Krumdieck CL (2000) Dairy foods and bone health: examination of the evidence. *Am J Clin Nutr* **72**, 681–689.
Whiting SJ (1990) Effect of prostaglandin inhibition on caffeine-induced hypercalciuria in healthy women. *J Nutr Biochem* **1**, 201–205.
Whiting SJ & Cole DEC (1986) Effect of dietary anion composition on acid-induced hypercalciuria in the adult rat. *J Nutr* **116**, 388–394.
Whiting SJ & Cole DE (1987) The comparative effects of feeding ammonium carbonate, ammonium sulphate, and ammonium chloride on urinary calcium excretion in the rat. *Can J Physiol Pharmacol* **65**, 2202–2204.
Whiting SJ & Draper HH (1980) The role of sulfate in the calciuria of high protein diets in adult rats. *J Nutr* **110**, 212–222.
Whiting SJ & Draper HH (1981) Effect of a chronic acid load as sulfate or sulfur amino acids on bone metabolism in adult rats. *J Nutr* **111**, 1721–1726.
Whiting SJ, Boyle JL, Thompson A, Mirwald RL & Faulkner RA (2002) Dietary protein, phosphorus and potassium are beneficial to bone mineral density in adult men consuming adequate dietary calcium. *J Am Coll Nutr* **21**, 402–409.
Wigertz K, Palacios C, Jackman LA, Martin BR, McCabe LD, McCabe GP, Peacock M, Pratt JH & Weaver CM (2005) Racial differences in calcium retention in response to dietary salt in adolescent girls. *Am J Clin Nutr* **81**, 845–850.
Wolf RL, Cauley JA, Baker CE, Ferrell RE, Charron M, Caggiula AW, Salamone LM, Heaney RP & Kuller LH (2000) Factors associated with calcium absorption efficiency in pre- and perimenopausal women. *Am J Clin Nutr* **72**, 466–471.
Wood RJ (2000) Searching for the determinant of intestinal calcium absorption. *Am J Clin Nutr* **72**, 674–676.
Wynckel A, Hanrotel C, Wuillai A & Chanard J (1997) Intestinal calcium absorption from mineral water. *Miner Electrolyte Metab* **23**, 88–92.
Zemel MB (1988) Calcium utilization: effect of varying level and source of dietary protein. *Am J Clin Nutr* **48**, 880–883.
Zemel MB, Schuette SA, Hegsted M & Linkswiller HM (1981) Role of sulfur-containing amino acids in protein-induced hypercalciuria in men. *J Nutr* **111**, 545–552.