Magnesium retention from metabolic-balance studies in female adolescents: impact of race, dietary salt, and calcium

Cristina Palacios, Karin Wigertz, Michelle Braun, Berdine R Martin, George P McCabe, Linda McCabe, J Howard Pratt, Munro Peacock, and Connie M Weaver

ABSTRACT

Background: Previously, we showed that black girls retained more calcium than white girls did and that salt loading negatively affected calcium retention. Racial differences likely exist in other bone minerals also, such as magnesium, in response to salt loading during growth.

Objective: We studied racial differences in magnesium metabolism in response to dietary sodium and calcium during rapid bone growth.

Design: Twenty-seven white and 40 black girls (11–15 y old) were studied for 3 wk while they consumed low-sodium (1.3 g/d) and high-sodium (3.8 g/d) diets by using a randomized-order, crossover metabolic study with 3 dietary calcium intakes; the magnesium dietary intake was fixed at 230 mg/d. Urine and feces were collected during each 3-wk period in 24-h pools and analyzed for magnesium. A mixed-model ANOVA was used to determine the effect of race and dietary sodium with calcium intake as a covariate.

Results: Salt loading or calcium intake had no significant effect on urinary magnesium excretion. Blacks excreted significantly less urinary magnesium (mean ± SD: 83.8 ± 25.6 mg/d) than did whites (94.9 ± 27.3 mg/d; \(P < 0.05\)). No effects were observed in fecal magnesium excretion. Magnesium retention was higher with the low-sodium diet (50.1 ± 44.0 mg/d) than with the high-sodium diet (39.3 ± 49.8 mg/d) \(P < 0.05\), with no effects of race or calcium intake. Salt loading had no effect on biomarkers. Whites had higher 25-hydroxyvitamin D and insulin-like growth factor binding protein concentrations. Blacks excreted less urinary magnesium than did whites. Magnesium retention was similar between races but higher with the low-sodium diet. Kinetic studies are needed to fully explain magnesium homeostasis. This trial was registered at clinicaltrials.gov as NCT01564238.

Conclusions: Blacks excreted less urinary magnesium than did whites. Magnesium retention was similar between races but higher with the low-sodium diet. Kinetic studies are needed to fully explain magnesium homeostasis. This trial was registered at clinicaltrials.gov as NCT01564238.

INTRODUCTION

Magnesium is an important structural bone mineral, and ~60% of the total magnesium in the body is in bone (1). Magnesium also plays a role in energy metabolism and protein and nucleic acid synthesis and is a cofactor for >300 proteins, including the calcitropic hormones parathyroid hormone (PTH)\(^4\) and 1,25 vitamin D. An important role of magnesium in bone is in the decrease of the hydroxyapatite crystal size, which prevents larger, more-perfect mineral crystals that result in brittle bone and, therefore, improves bone quality (2). Also, magnesium acts as a buffer for the acid produced by the typical Western diet. Because bone undergoes continuous remodeling, an adequate supply of magnesium, as well as other nutrients important to bone, is needed to support bone formation. Individuals who undergo rapid growth, development, or repair have the greatest magnesium needs (3). Because 40% of the adult skeleton is formed during the adolescent growth spurt (4), magnesium is particularly important during this phase (5).

As children and adults, American blacks have higher bone mineral density (BMD) than American whites do (6–8). There are many aspects of mineral metabolism that may combine to contribute to differences in bone characteristics between races, but calcium is the most extensively studied (6–10). Metabolic balance studies showed that blacks absorb and retain calcium more efficiently than whites do during adolescence (10–12), which may explain the higher BMD usually reported in blacks. Also, it has been well documented that dietary sodium decreases calcium retention through increasing urinary calcium excretion in adults (13), which suggests that urinary calcium excretion is coupled with sodium excretion. In adolescents, we have shown a negative effect of dietary sodium on calcium retention because of increased urinary calcium excretion in girls while consuming diets low in calcium (800 mg/d) (11). We have also shown that the magnitude of this effect is racially dependent, such that black girls retained more sodium when they consumed a high dietary sodium (14) and retained more calcium than white girls did while consuming either a low- or high-sodium diet with calcium intake fixed at 800 mg/d (11). These results may be explained by the coupling of sodium and calcium reabsorption in the kidneys, in which a high-sodium diet results in a higher
urinary sodium excretion, which drags calcium along, and we hypothesized that whites have a possible genetic predisposition for a greater effect of salt on the kidneys (11). Because of the similar characteristics of the 2 divalent cations, magnesium and calcium share some of the same pathways of reabsorption in the kidneys, but the mechanisms are not well understood.

To our knowledge, no study has previously tested racial differences on the effects of dietary sodium and calcium on magnesium handling in magnesium homeostasis. Because we have previously shown racial differences in sodium and calcium renal handling in girls with a low calcium intake (800 mg/d) (11, 14), it is plausible that different amounts of dietary sodium and calcium may also lead to differences in magnesium handling. Therefore, we conducted a secondary analysis to determine daily magnesium retention in black and white girls who consumed controlled diets that contained high and low amounts of sodium in a randomized crossover design for 3 wk with 3 calcium intakes. We hypothesized that the increase in calcium excretion previously observed with salt loading in girls who consumed the low calcium amount would also result in a higher magnesium excretion and lower magnesium retention.

SUBJECTS AND METHODS

Subjects

Black and white adolescent females, aged 11–15 y, participated in metabolic-balance studies to determine effects of sodium and calcium intakes on calcium retention (primary outcomes) and on other minerals (secondary outcomes). Full details of the metabolic-balance methods have been previously published (11, 14). The health of subjects was determined with a questionnaire, physical examination, and serum biochemistry. Applicants were excluded from the study if they were <11 or >15 y, had BMI <15th or >85th percentile for age, or had a history of postmenarcheal amenorrhea, pregnancy or abortion, eating disorders, or oral contraceptive or tobacco use. Race was determined by asking the race of parents and grandparents. Both parents and grandparents had to be white or black to be eligible in the study. Subjects completed six 24-h dietary recalls before the study, which were analyzed with Nutritionist IV Diet Analysis software (1995, version 3.5.1; First Databank Division). Pubertal development was evaluated by self-assessment of breast and pubic hair stage according to Tanner (15). Postmenarcheal age was defined as months after onset of menarche relative to the first day of the study. Black and white subjects were matched for weight and postmenarcheal age. All subjects were studied under protocols approved by the Purdue University Use of Human Subjects Research Committee, and subjects and their guardians gave informed consent before the study began.

Design

Subjects were residents in a Purdue University fraternity house, which was transformed during the summer of 1999 and 2000 into a metabolic unit for two 20-d balance sessions. Subjects were assigned to one of 3 calcium intakes (800, 1300, or 1800 mg/d), and each subject was studied under 2 sodium intakes (1.3 and 3.86 g/d) in a randomized crossover design. Magnesium intake was set at 230 mg/d, which is within the recommended intake for adolescent girls (200–300 mg/d) (3). The low calcium intake (800 mg/d) was chosen because it was the approximate usual median intake in most adolescents in the United States (16). The medium intake corresponded to the amount recommended by the Institute of Medicine (IOM) for calcium in adolescents of 1300 mg/d (17). The high intake corresponded to an amount above the intake for maximal retention (18) but well below the upper limit of 3000 mg/d as established by the IOM (17). Amounts of sodium were selected to be within the range of sodium intakes present in the population but, at the same time, to create sufficient difference in sodium intakes that were likely to alter the retention of other nutrients, such as calcium. Amounts are for sodium, not salt. The low sodium intake was approximately the recommended amount recommended by the IOM for adolescents (19), whereas the high sodium intake was chosen because it was the approximate usual intake in adolescents in the United States at the time of the study (16). The diet was also fixed for dietary potassium (2186 mg/d), phosphorus (1100 mg/d), protein (70 g/d), fat (73.6 g/d), and fiber (10 g/d), which represent usual intakes of these nutrients in this population (16).

The 2 higher calcium intakes (1300 and 1800 mg/d) were achieved by the consumption of calcium carbonate soft chew supplements (McNeil Nutritional). The high-sodium diet was achieved by adding salt to low-sodium soups, which provided 2 g Na/d, and to low-sodium sports drink (low-sodium formula; Quaker Oats Company), which provide 0.86 g Na/d. The diet required effort to achieve the low amounts of sodium. The dietary tolerance to low-sodium soups was good. We allowed subjects to use salt substitutes and pepper to improve the taste in low-sodium soups. Sodium and potassium contents of each shake were 5.5 and 0.8 mg, respectively. The analysis of daily meal composites resulted in a mean dietary calcium intake of 815 mg for the low calcium intake, 1370 mg for the medium calcium intake, and 1864 mg for the high calcium intake. The magnesium content of the diet was 231 ± 15 mg/d. There was a 2-wk washout period between sessions in which subjects returned home to their usual diet.

Measurements

Body weight was recorded daily in the morning by using an electronic scale (Health O Meter) while the subject wore night clothes and no shoes. Height was measured once without shoes by using a wall stadiometer. Blood pressure was measured every other day in a recumbent position by using a sphygmomanometer (Hawksely and Sons). Total body BMD, bone mineral content, total body calcium, fat mass, and lean body mass were measured by using dual energy X-ray absorptiometry (software version 4.3e; Lunar Corp).

We used a 4-d cycle menu with 3 meals and 2 snacks in the study. Food portions were maintained constant within each cycle menu and were equal for all subjects. Subjects were strictly supervised at all times to ensure compliance and to avoid consumption of other foods. Foods and beverages were prepared with deionized water and weighed to the nearest one-tenth of a gram on digital scales. Duplicates of each of the day’s meals were homogenized and analyzed to test for the variation in food batches.
Urine and feces were collected in separate acid-washed containers on a daily basis for 20 d for each metabolic session. Urine was pooled as 24-h samples and analyzed daily for creatinine to check compliance. Feces were also expressed per 24 h, and the completeness of collections was determined by using polyethylene glycol recovery (PEG) (molecular weight ~3400; Dow Chemical Co). Two gelatin capsules that contained 0.5 g PEG were administered with each meal.

Diet, fecal, and urine samples were measured for magnesium, calcium (11), sodium (14), and potassium (20). Samples were measured by using atomic absorption spectrophotometry (5100 PC; Perkin-Elmer) or inductively coupled plasma spectrophotometry (Optical Emission Spectrometer, Optima 4300DV; Perkin Elmer). Urinary creatinine was measured by using an automated colorimetric method (Roche Diagnostics). PEG was analyzed using a turbidimetric assay (21). Fasting serum was measured by using a monoclonal antibody to human crosslinked N-telopeptides of type I collagen were measured by using an enzyme-linked immunosorbent assay with a monoclonal antibody to human crosslinked N-telopeptides (Osteomark; Ostex International Inc). Results for most of these biomarkers while subjects consumed low calcium amounts have been previously published (10).

Retention calculation

Magnesium retention was calculated by subtracting urinary and fecal values from dietary magnesium. The first week of each session was regarded as the equilibration period to the diet.

Statistical analysis

For power calculations, we used the primary outcome of the main study, which was calcium retention; the secondary outcome was the retention of magnesium. Power estimates were based on the achievement of a 90-mg/d difference in calcium retention between the 2 intakes of sodium, particularly in white girls (22). A within-race difference by assuming a sodium impact of 90 mg Ca/d in 30 girls could be detected with a power of 0.99. Because the relation between urinary sodium and calcium in black girls was unknown at that time, we could not predict the power for girls when the study started.

All subjects who completed at least one intervention session were included in the analysis. Baseline characteristics of black and white subjects were compared by using Student’s t test. Primary response variables (magnesium in urine, feces, and retention) were analyzed by using a mixed-model ANOVA which distinguished between-subject variations from within-subject variation. Between-subject factors were race (black and white) and order (high sodium followed by low sodium, low sodium followed by high sodium); within-subject factors were sodium intake (low and high) and session (first and second 3-wk periods). Calcium intakes were considered in the model as a covariate. Preliminary analyses included these factors, and their interactions verified the absence of order and session effects. Therefore, primary analyses assessed effects of race, sodium intake, and interactions in the 3 factors. All analyses were performed with Statistical Analysis System software (version 9.2; SAS Institute). Statistical significance was set at \( P < 0.05 \).

RESULTS

Forty black girls and 27 white girls were enrolled in the study; 27 black and 19 white girls completed both low- and high-sodium intervention sessions, and 13 black girls and 8 white girls completed either one low- or one high-sodium session. There were no significant differences in subject characteristics between black and white girls except for habitual calcium intake, which was significantly higher in white than in black girls \( (P < 0.05) \); Table 1). Habitual intakes of both calcium and magnesium were lower than recommended by the IOM.

Magnesium excretion, retention, and absorption by race and dietary sodium are shown in Table 2. We did not find a significant effect of dietary sodium on urinary magnesium excretion,
and calcium intake as a covariate was not significant. However, we showed that blacks excreted significantly less urinary magnesium (83.8 ± 25.6 mg/d) than that of whites (94.9 ± 27.3 mg/d) independent of the sodium amount (P < 0.05). Fecal magnesium excretion was similar between black and white girls, with no significant effects of dietary sodium and no significant interactions between factors. Although no racial differences were observed in magnesium retention, an effect that was due to the salt loading was observed, whereas magnesium retention was higher with the low-sodium diet (50.1 ± 44.0 mg/d) than with the high-sodium diet (39.3 ± 49.8 mg/d; P < 0.05), with calcium as a covariate that was not significant.

With respect to serum and urine metabolites (Table 3), no effect because of salt loading was observed, but racial differences were detected in some of variables. Serum 25(OH)D was significantly higher in whites (37.4 ± 8.7 ng/mL) than in blacks (26.8 ± 8.3 ng/mL; P < 0.01). Serum 1,25(OH)2D concentrations were significantly higher in blacks (42.0 ± 12.7 pg/mL) than in whites (35.8 ± 8.4 pg/mL; P < 0.05), with a significant negative effect of calcium intake. PTH was also significantly higher in blacks (25.0 ± 9.3 ng/L) than in whites (21.1 ± 7.8 ng/L; P < 0.05). No significant differences were observed with urinary creatinine or N-telopeptide excretion as a result of salt loading or race, but calcium intake had a significant negative effect on creatinine and a positive effect on N-telopeptide excretion (P < 0.001). In addition, we explored the relation between hormonal and biochemical variables with magnesium metabolism, and no significant associations were observed.

Data for serum calcium, sodium, osteocalcin, bone alkaline phosphatase, IGF-1, and IGF binding protein 3 are available in the online supplementary table. Briefly, calcium intake had a positive significant effect on serum calcium (P < 0.05) and serum sodium (P < 0.001) but no differences due to salt loading or race. calcium intake had a negative effect on IGF-1, and IGF binding protein 3 (P < 0.001) but no differences as a result of salt loading. IGF binding protein 3 concentrations were significantly higher (4355 ± 1039 μg/L) in whites than in blacks (4017 ± 1159 μg/L; P < 0.01). No significant effects were

### Table 2

<table>
<thead>
<tr>
<th>Variables</th>
<th>Low-sodium diet (1.3 g/d)</th>
<th>High-sodium diet (3.8 g/d)</th>
<th>P-covariate (calcium intake)</th>
<th>Significant main effects and interactions*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary magnesium (mg/24 h)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>233 ± 15</td>
<td>233 ± 15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary excretion (mg/24 h)</td>
<td>Blasts (n = 33)</td>
<td>Whites (n = 21)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>231 ± 12</td>
<td>231 ± 77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fecal excretion (mg/24 h)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>95 ± 37</td>
<td>99 ± 48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Net magnesium absorption (mg/24 h)</td>
<td>Blasts (n = 33)</td>
<td>Whites (n = 21)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>137 ± 34</td>
<td>134 ± 48</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retention (mg/24 h)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>54 ± 38</td>
<td>42 ± 51</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*All values are means ± SDs. *Significant effects at P < 0.05 (ANOVA). There was no significant effect of order of session, and no significant interactions between factors were shown.

### Table 3

<table>
<thead>
<tr>
<th>Variables</th>
<th>Low-sodium diet (1.3 g/d)</th>
<th>High-sodium diet (3.8 g/d)</th>
<th>P-covariate (calcium intake)</th>
<th>Significant main effects and interactions*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25(OH)D (ng/mL)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>27.2 ± 6.9</td>
<td>37.4 ± 8.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,25(OH)2D (pg/mL)</td>
<td>Blasts (n = 33)</td>
<td>Whites (n = 21)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>41.6 ± 12.0</td>
<td>34.1 ± 8.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTH (ng/L)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>23.6 ± 7.6</td>
<td>21.5 ± 8.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine (mg/24 h)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>868 ± 248</td>
<td>891 ± 234</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h N-telopeptide (mmol BCE/nmol Cr)</td>
<td>Blasts (n = 35)</td>
<td>Whites (n = 24)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>341 ± 209</td>
<td>483 ± 202</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*All values are means ± SDs. *Significant effects at P < 0.05 (ANOVA). There was no significant effect of order of session, and no significant interactions between factors were shown. BCE, bone collagen equivalents; PTH, parathyroid hormone; 1,25(OH)2D, 1,25-dihydroxyvitamin D; 25(OH)D, 25-hydroxyvitamin D.

1 Data for 2 black girls receiving the low-sodium diet and 4 black girls and 1 white girl receiving the high-sodium diet were missing for 25(OH)D.
2 Data for 2 black girls and 1 white girl receiving the low-sodium diet and 5 black girls and 1 white girl receiving the high-sodium diet were missing for 1,25(OH)2D.
3 Data for 1 black girl and 1 white girl receiving the high-sodium diet were missing for PTH.
4 Data for 1 black girl receiving the low- and high-sodium diet were missing for urinary creatinine.
5 Data for 4 black girls and 1 white girl receiving the low-sodium diet and 2 black girls and 2 white girls receiving the high-sodium diet were missing for urinary 24-h N-telopeptide.
observed for serum concentrations of osteocalcin and bone alkaline phosphatase.

**DISCUSSION**

In this metabolic study, we showed that magnesium retention was greater during consumption of the low- compared with the high-sodium diet, as we had hypothesized. We also showed that blacks significantly excreted less urinary magnesium than did whites, but salt loading had no effect on the urinary magnesium excretion.

Racial differences in urinary magnesium excretion were not explained by differences in diet, because diet was completely and rigorously controlled. In addition, these differences were also not explained by differences in sweat because we previously reported no racial differences in magnesium excretion in sweat measured by a 24-h whole-body procedure (23). These differences may be explained in part by genetic differences in renal magnesium handling because the kidney acts as main regulation site for magnesium homeostasis (24). We have also previously shown racial differences in urinary sodium (14), potassium (20), and calcium (11) excretion between black and white girls.

We previously suggested that urinary calcium excretion is coupled with sodium excretion, possibly through the sodium-calcium exchanger, which is responsible for a portion of the calcium reabsorption (25). It is not known if this effect also occurs with magnesium. It is feasible that many controls for calcium may be shared with magnesium (26). The fine-tuning reabsorption of magnesium occurs in the thick ascending limbs and the distal convoluted tubule (27), whereas calcium and magnesium reabsorption are coupled via paracellular pathways (28). We had hypothesized that an increase in calcium and sodium excretion may result in an increase in magnesium excretion. However, in the current study, salt loading had no significant effects on urinary magnesium excretion. Although a sodium and magnesium cotransporter has not yet been identified in the kidney, magnesium reabsorption in the thick ascending limb and distal convoluted tubule is affected in part by the sodium and potassium ATPase activity (27). In addition, a sodium-dependent magnesium transport pathway that permits the entry of sodium into the cell and the extrusion of magnesium has been shown in other cell types (29, 30); therefore, more studies are needed to clarify such pathway.

To our knowledge, this is the first study to determine racial differences in the effect of salt loading on magnesium retention. Higher magnesium retention was observed when subjects consumed the low- in comparison with the high-sodium diet. Because urinary magnesium excretion was not affected by salt loading, there were likely other mechanisms involved, possibly in absorption. The net intestinal magnesium absorption is negatively affected by phosphate intake (24), but studies have not shown that it is affected by calcium intake (31–34), and no study has tested salt loading. However, in the current study, fecal excretion was also not affected by salt loading. Thus, kinetic studies are needed to determine the full effects on metabolism.

The effect of sodium intake on magnesium retention shown in our study contrast with that in a study in 6 Japanese college women, whereas a relatively low sodium intake led to negative magnesium retention after 10 d (35), but only one amount of sodium was studied. Also, our results contrast with those of a study in white adolescent girls who consumed 1000 mg Ca/d and 190–195 mg Mg/d, whereas a negative magnesium retention (−1.8 to −6.8 mg/d) was reported, which represented and intake of 3.3–5.6 mg dietary Mg/kg body weight (31). In the current study, the mean magnesium retention in white girls was positive and averaged 35.3 ± 52.5 mg/d across sodium and calcium intakes, which represented 4.2 mg dietary Mg · kg body weight⁻¹ · d⁻¹. Similarly, a study in young adults who consumed ~3.6–4.2 mg Mg · kg body weight⁻¹ · d⁻¹ reported a positive magnesium balance (36). Another study in white adolescent girls that investigated the effect of 2 intakes of calcium (667 and 1667 mg/d) while they consumed 176 mg Mg/d showed that magnesium retention was similar between the 2 calcium intakes, which averaged 21 mg/d (33), which was similar to amount in the current report. Moreover, a high calcium intake (1800 mg/d) did not alter any aspect of magnesium kinetics, including absorption, excretion, or bone-turnover rates compared with a low calcium intake (800 mg/d) in another group of adolescent white girls studied previously by our group from a similar balance study (32). Contrasting results arise from reports that studied human subjects in a particular disease state or who did not participate in controlled balance studies (37, 38).

We did not find differences in biomarkers with salt loading or a relation between these biomarkers with magnesium metabolism. However, several racial differences were detected in some of the biomarkers. White girls had significantly higher concentrations of serum 25(OH)D and IGF binding protein 3 but significantly lower amounts of 1,25(OH)₂D and PTH compared with those of blacks. Similarly, other reports have also shown higher amounts of 25(OH)D (39, 40) and IGF binding protein 3 (41) and lower amounts of 1,25(OH)₂D (42) and PTH (43) in whites than in blacks.

The study had several strengths. Subjects were studied under highly supervised, controlled conditions. We tested different intakes of dietary calcium and sodium during the period of peak bone mass. The balance was calculated daily from 24-h urine and fecal collections. A limitation of the study was the short-term nature of this study, which precluded our ability to evaluate effects of dietary calcium and sodium on actual changes in bone mass. Instead, gains in bone mass could only be estimated from magnesium retention. A longer intervention is needed to test salt-loading effects on bone mineral accretion. In addition, kinetic studies are needed to fully explain magnesium homeostasis.

In conclusion, urinary magnesium excretion was lower in blacks than in whites. The low-sodium diet resulted in higher magnesium retention; however, calcium intake had no effect on magnesium metabolism. Therefore, salt loading not only had a negative effect on calcium retention, as we have previously reported in girls, but also on magnesium retention. A high-sodium diet during this critical period of peak bone mass may have negative implications on bone mass accretion and bone quality. In addition, a lower magnesium and calcium retention induced by a high-sodium diet could also have negative implications on blood pressure and cardiovascular disease later in life.

The authors’ responsibilities were as follows—CMW, BRM, JHP, MP, and GPM: designed the research; CP, KW, BRM, and MB: conducted the research; GPM, LM, and CP: analyzed data; CP, CMW, MP, GPM, and LM: wrote the manuscript; CP and CMW: had primary responsibility for the final content of the manuscript; and all authors: read and approved the final manuscript. None of the authors had a conflict of interest.
REFERENCES