Racial differences in calcium retention in response to dietary salt in adolescent girls\textsuperscript{1–3}

Karin Wigertz, Cristina Palacios, Lisa A Jackman, Berdine R Martin, Linda Doyle McCabe, George P McCabe, Munro Peacock, J Howard Pratt, and Connie M Weaver

ABSTRACT
Background: Sodium is an important determinant of urinary calcium excretion, and race is an important determinant of calcium retention. The effect of dietary sodium on calcium retention and the influence of race have not been studied in adolescence, the life stage during which peak bone mass is accrued.
Objective: The study reported here was undertaken to compare racial differences in calcium retention as a function of dietary salt intake.
Design: A total of 35 adolescent girls (22 black and 13 white) participated in two 20-d metabolic summer camps, separated by 2 wk, that simulated a free-living environment. The effect of changes in dietary sodium on calcium retention was tested in a randomized-order, crossover design with 2 concentrations of sodium—1.30 g/d (57 mmol/d) and 3.86 g/d (168 mmol/d)—and a constant calcium intake of 815 mg/d (20 mmol/d).
Results: Both race and sodium intake significantly affected calcium retention ($P < 0.01$). Calcium retention was significantly greater in black girls than in white girls, regardless of dietary sodium intake ($P < 0.001$). The high-sodium diet significantly reduced calcium retention in both whites and blacks ($P < 0.01$), primarily through a decrease in net calcium absorption. Black girls excreted significantly less calcium in the urine than did white girls, regardless of diet ($P < 0.05$).
Conclusions: Calcium retention is significantly greater in black girls than in white girls but is significantly reduced in girls of both races in response to salt loading. Am J Clin Nutr 2005;81: 845–50.

KEY WORDS Race, calcium retention, urinary calcium, dietary sodium, bone turnover, metabolic study, female adolescents

INTRODUCTION
An increase in dietary sodium promotes urinary calcium excretion (1–3). Epidemiologic studies support the relation between dietary sodium intake and urinary calcium excretion in both men (4, 5) and women (4–6), and they show the strongest relation in elderly men (7). Several short-term intervention studies in young men and women (8, 9), premenopausal women (10, 11), and postmenopausal women (10, 12–15) also found a similar relation of dietary sodium and urinary calcium excretion. The only study conducted to date in adolescents, a cross-sectional study, found a significant positive relation between urinary calcium and urinary sodium (16). Adolescence is an important period in which to study factors that influence calcium retention because almost 40% of adult peak bone mass is acquired during those years (17).

Race is also a major determinant of calcium retention. Black adolescents were shown to retain more calcium than did white adolescents who had the same calcium intake (18). Studies have shown that blacks have significantly higher bone mineral density and bone mineral content than do whites, both as adults (19, 20) and as children (21, 22), although not all studies agree with the latter finding (23). Racial differences in the effect of dietary sodium on calcium excretion and calcium retention have not been studied. The purpose of the current study was to ascertain the effect of dietary sodium on calcium retention, calcium homeostasis, and biochemical markers of bone turnover in black and white adolescent girls.

SUBJECTS AND METHODS
Subjects and protocol
A total of 40 girls (25 black and 15 white) were recruited to participate in two 20-d metabolic studies during the summer (June–August) of 1999. They were housed in a sorority house that had been temporarily converted into a metabolic unit. All meals and snacks were eaten under 24-h supervision. The 2 studies were separated by a 2-wk washout period, during which the girls returned home. The diet consisted of 815 mg calcium/d (20 mmol/d) and 1 of 2 intakes of sodium—1.30 g/d (57 mmol/d) or 3.86 g/d (168 mmol/d)—all of which are within the normal range for adolescents. The diet was constant in intakes of magnesium [227 mg/d (9 mmol/d)], potassium [2 g/d (51 mmol/d)], phosphorus [1100 mg/d (36 mmol/d)], protein (70 g/d), fat (73.6 g/d), and fiber (10 g/d). The 2 intakes of sodium were given in a random-order crossover design. Half of the girls received the low-sodium diet, and the other half received the high-sodium diet...

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during the first metabolic study; the order was reversed during the second metabolic study. Each study included 6 d of equilibration followed by a 14-d balance period.

Subjects were matched so that average postmenarchal age, height, and weight were equal between the 2 races. Menarcheal age on day 1 of the study was ascertained by using questionnaires. The girls who had not reached menarche by the time of the study were contacted later by telephone. Height was determined by using a scale (Detecto-Medic, Brooklyn, NY). Weight was measured by using a calibrated electronic scale. The criterion for race was that all 4 of a girl’s grandparents were of the same race.

Before the study, each participant completed six 24-h dietary recalls. The food records were evaluated by a dietitian and analyzed with the use of NUTRITIONIST IV DIET ANALYSIS software (version 4.1; First Databank Division, San Bruno, CA).

Exclusionary criteria included age < 10 y or > 15 y and a body mass index (in kg/m²) below the 15th or above the 85th percentile for age. Subjects with a history of postmenarchal amenorrhea, pregnancy, abortion, eating disorder, use of oral contraceptive, or tobacco use were also excluded. Health status was assessed from a questionnaire and physical examination.

Written informed consent was obtained from all studies. All subjects were studied under protocols approved by the Use of Human Subjects Research Committee of Purdue University.

Measurements

The diet consisted of 4-d cycle menus. Low-sodium soups made at the study site and Gatorade (low-sodium formula; Quaker Oats Company, Chicago, IL) were used to achieve the 2 intakes of dietary sodium. The high-sodium diet was achieved by adding salt to the low-sodium soups to provide 2 g sodium/d (87 mmol/d) and to low-sodium Gatorade to provide 0.86 g sodium/d (37 mmol/d). Duplicate composites of each day’s diet were prepared and stored at −10 °C before analysis of nutrients. Twenty-four-hour urine and fecal samples were analyzed separately for minerals. Samples and dietary composites were measured for calcium and sodium by using atomic absorption spectroscopy (5100 PC; Perkin Elmer, Norwalk, CT). Measurements of creatinine were used to correct the urine sample to a 24-h period and to determine compliance. Urinary creatinine was measured by using an automated colorimetric method on a spectrophotometer (Cobas Mira Systems; Roche Diagnostic Systems, F Hoffmann-LaRoche, Basel, Switzerland). Polyethylene glycol (MW ≈3400; Dow Chemical Co, Midland, MI) was used to monitor fecal compliance. Polyethylene glycol was analyzed by using a turbidimetric method (24). Two gelatin capsules containing 0.5 g polyethylene glycol were administered with each meal. Daily calcium retention was calculated as the 24-h calcium intake minus the 24-h urinary and fecal calcium excretion. For days when there were no stools, periods between stools were divided by the appropriate number of days. Body weight was recorded daily. Fat mass and lean body mass were determined by using dual-energy X-ray absorptiometry (Lunar Prodigy, Madison, WI). Tanner score was determined with subjects’ self-assessment of breast and pubic hair stage (25). Total-body 24-h sweat was extracted from clothing and collected from a body scrubdown procedure after 14 d of acclimatization and adaptation to the diet as previously described (26). Fasting serum was analyzed for calcium, sodium, 25-hydroxyvitamin D [25(OH)D], and 1,25-dihydroxyvitamin D [1,25(OH)₂D], osteocalcin, bone alkaline phosphatase, and parathyroid hormone as described previously (18). Insulin-like growth factor I and insulin-like growth factor–binding protein 3 were analyzed by radioimmunoassay as previously described (18). Fasting and 24-h urinary N-telopeptides of type I collagen were measured by enzyme-linked immunosorbent assay on a monoclonal antibody to human cross-linked N-telopeptides (Osteomark; Ostex International Inc, Seattle, WA).

Statistical analysis

A mixed-model analysis of variance was used in analyzing the data to allow for examination of the effects of race and diet and possible race × diet interactions (27). Statistical significance was set at $P < 0.05$. All statistical analyses were done by using SAS software (version 8.2; SAS Institute, Cary, NC). Of the 40 girls recruited to the study, 16 black and 7 white girls completed both balance periods, 6 black and 6 white girls completed one balance period only, 3 girls left after a few days because of homesickness, 1 black girl was sent home because of poor health, and 1 white girl was excluded after admitting that her racial background was mixed. For statistical inferences that include the available data (22 blacks and 13 whites).

RESULTS

Subject characteristics did not, for the most part, differ significantly between the black and the white girls, except that the whites were significantly ($P < 0.05$) older than the blacks (a result of selection to achieve similar postmenarchal ages in the race groups) and had a significantly higher habitual calcium intake (Table 1). Calcium retention with the low- and high-sodium diets was 453 ± 34 and 359 ± 34 mg/d, respectively, among the blacks and 235 ± 65 and 189 ± 72 mg/d, respectively.

### Table 1

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Blacks ($n = 22$)</th>
<th>Whites ($n = 13$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>12.5 ± 1.1</td>
<td>13.3 ± 1.0²</td>
</tr>
<tr>
<td>Habitual calcium intake before study (mg)⁷</td>
<td>606 ± 174</td>
<td>883 ± 405²</td>
</tr>
<tr>
<td>Habitual sodium intake before study (mg)⁷</td>
<td>2578 ± 606</td>
<td>2793 ± 565</td>
</tr>
<tr>
<td>Sweat calcium excretion (mg/d)⁴</td>
<td>54 ± 28</td>
<td>51 ± 15</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>158 ± 8</td>
<td>159 ± 9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>56 ± 13</td>
<td>56 ± 17</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.2 ± 3.9</td>
<td>21.9 ± 5.1</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>15.3 ± 8.2</td>
<td>17.1 ± 10.1</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>36.6 ± 5.9</td>
<td>35.9 ± 6.1</td>
</tr>
<tr>
<td>Total-body bone calcium content (g)</td>
<td>843 ± 207</td>
<td>805 ± 171</td>
</tr>
<tr>
<td>Tanner score</td>
<td>3.8 ± 1.0</td>
<td>3.6 ± 0.8</td>
</tr>
<tr>
<td>Postmenarchal age (mo)</td>
<td>5.8 ± 13.6</td>
<td>4.8 ± 16.5</td>
</tr>
</tbody>
</table>

¹ All values are $x ± SD$.
² Significantly different from blacks, $P < 0.05$ (mixed-model ANOVA).
³ $n = 20$ blacks, 10 whites.
⁴ To convert the values for sweat calcium excretion to mmol/24 h, multiply by 0.02495. These data were previously reported (26).
among the whites. The black girls retained 1.9 times more calcium than did the white girls, regardless of diet (Table 2 and Figure 1). The average racial difference in calcium retention was > 170 mg/d (4.2 mmol/d), regardless of sodium intake (Table 2, Figure 1). Calcium retention with high-sodium intake was 94 mg/d (2.4 mmol/d) and 46 mg/d (1.2 mmol/d) lower in the blacks and the whites, respectively, than it was with low-sodium intake. Average daily calcium retention in the blacks was significantly (P < 0.001) higher than that in the whites at both intakes of dietary sodium (Table 2 and Figure 1). Both the black and the white subjects had significantly (P < 0.01) more calcium retention with the low-sodium diet than with the high-sodium diet (Table 2 and Figure 1). Thus, both race and diet had a significant influence on calcium retention, but the effect of race was greater. No significant race × diet interaction was detected.

Mean 24-h urinary calcium excretion in the blacks and whites was 50 ± 9 and 69 ± 12 mg/d, respectively, with the low-sodium diet and 53 ± 10 and 107 ± 15 mg/d, respectively with the high-sodium diet (both: P < 0.05) (Table 2 and Figure 2). A significant race × diet interaction was also detected (P < 0.05). The higher sodium intake resulted in a significantly (P = 0.05) less urinary sodium excretion with the high-sodium diet (both: P < 0.05) (Table 2 and Figure 2). Significant effectsBlacks Whites Blacks Whites

### TABLE 2
Indexes of calcium metabolism and markers of bone turnover and calcium balance in the black and white adolescent girls

<table>
<thead>
<tr>
<th>Index or marker</th>
<th>Low-sodium diet</th>
<th></th>
<th>High-sodium diet</th>
<th></th>
<th>Significant effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blacks</td>
<td>Whites</td>
<td>Blacks</td>
<td>Whites</td>
<td></td>
</tr>
<tr>
<td>Serum calcium (mg/dL)²</td>
<td>9.84 ± 0.11</td>
<td>9.64 ± 0.13</td>
<td>9.69 ± 0.09</td>
<td>9.82 ± 0.15</td>
<td>White &gt; black&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium (mEq/L)²</td>
<td>141.3 ± 0.6</td>
<td>141.6 ± 1.2</td>
<td>142.0 ± 0.6</td>
<td>142.8 ± 0.8</td>
<td>Black &gt; white&lt;sup&gt;4&lt;/sup&gt; High-sodium &gt; low-sodium&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>25(OH)D (ng/mL)</td>
<td>26.6 ± 1.9</td>
<td>39.7 ± 2.7</td>
<td>29.8 ± 2.9</td>
<td>37.1 ± 3.3</td>
<td></td>
</tr>
<tr>
<td>1,25(OH)₂D (pg/mL)</td>
<td>45.8 ± 3.0</td>
<td>33.2 ± 3.0</td>
<td>49.5 ± 3.5</td>
<td>41.0 ± 2.9</td>
<td></td>
</tr>
<tr>
<td>Osteocalcin (µg/L)²</td>
<td>39.2 ± 5.7</td>
<td>49.0 ± 11.2</td>
<td>43.8 ± 5.2</td>
<td>58.8 ± 11.3</td>
<td></td>
</tr>
<tr>
<td>Bone alkaline phosphatase (U/L)²</td>
<td>66.8 ± 7.0</td>
<td>66.6 ± 5.3</td>
<td>66.7 ± 6.4</td>
<td>64.8 ± 11.1</td>
<td></td>
</tr>
<tr>
<td>Parathyroid hormone (ng/L)²</td>
<td>22.7 ± 1.8</td>
<td>20.4 ± 3.3</td>
<td>25.0 ± 2.3</td>
<td>22.4 ± 3.3</td>
<td></td>
</tr>
<tr>
<td>Insulin-like growth factor I (µg/L)²</td>
<td>506 ± 22</td>
<td>453 ± 34</td>
<td>559 ± 18</td>
<td>473 ± 50</td>
<td></td>
</tr>
<tr>
<td>Insulin-like growth factor–binding protein 3 (µg/L)²</td>
<td>4662 ± 209</td>
<td>5145 ± 310</td>
<td>4756 ± 291</td>
<td>4984 ± 401</td>
<td></td>
</tr>
<tr>
<td>Urine Calcium excretion (mg/24 h)</td>
<td>50 ± 9</td>
<td>69 ± 12</td>
<td>53 ± 10</td>
<td>107 ± 15</td>
<td>White: high-sodium &gt; low-sodium&lt;sup&gt;6&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium excretion (mg/24 h)</td>
<td>870 ± 66</td>
<td>958 ± 41</td>
<td>2671 ± 158</td>
<td>3485 ± 124</td>
<td>High-sodium: white &gt; black&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine (mg/24 h)</td>
<td>938 ± 54</td>
<td>1054 ± 56</td>
<td>992 ± 64</td>
<td>1002 ± 63</td>
<td></td>
</tr>
<tr>
<td>24-h N-telopeptide (nmol BCE/ mmol Cr)²</td>
<td>281 ± 35</td>
<td>345 ± 19</td>
<td>317 ± 41</td>
<td>285 ± 63</td>
<td></td>
</tr>
<tr>
<td>Feces Calcium excretion (mg/24 h)</td>
<td>334 ± 28</td>
<td>496 ± 75</td>
<td>382 ± 34</td>
<td>518 ± 68</td>
<td>White &gt; black&lt;sup&gt;3&lt;/sup&gt;</td>
</tr>
<tr>
<td>Net calcium absorption (mg/24 h)</td>
<td>471 ± 28</td>
<td>311 ± 74</td>
<td>421 ± 38</td>
<td>301 ± 71</td>
<td>Black &gt; white&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium excretion (mg/24 h)</td>
<td>140 ± 25</td>
<td>84 ± 15</td>
<td>199 ± 33</td>
<td>96 ± 14</td>
<td>High-sodium: black &gt; white&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Calcium retention (mg/24 h)</td>
<td>453 ± 34</td>
<td>235 ± 65</td>
<td>359 ± 34</td>
<td>189 ± 72</td>
<td>Black &gt; white&lt;sup&gt;4&lt;/sup&gt; Low-sodium &gt; high-sodium&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>1</sup> All values are x ± SEM; n in brackets. 25(OH)D, 25-hydroxyvitamin D; 1,25(OH)₂D, 1,25-dihydroxyvitamin D; BCE, bone collagen equivalents. To convert the values for serum calcium to mmol/L, multiply by 0.2495. To convert the values for serum sodium to mmol/L, multiply by 1. To convert the values for 25(OH)D to nmol/L, multiply by 2.495. To convert the values for 1,25(OH)₂D to pmol/L, multiply by 2.6. To convert the values for osteocalcin to nmol/L, multiply by 0.2495. To convert the values for parathyroid hormone to ng/L, multiply by 1. To convert the values for insulin-like growth factor I to nmol/L, multiply by 0.131. To convert the values for insulin-like growth factor–binding protein 3 to nmol/L, multiply by 0.035. To convert the values for urinary and fecal calcium and calcium net absorption (dietary calcium minus fecal calcium excretion) to mmol/24 h, multiply by 0.02495. To convert the values for urinary creatinine to mmol/24 h, multiply by 0.00884. To convert the values for urinary and fecal sodium to mmol/24 h, multiply by 0.0435.

<sup>2</sup> Values were measured at the end of each study session by mixed-model ANOVA.

<sup>3</sup> P < 0.01.

<sup>4</sup> Significant race × diet interaction, P < 0.05.

<sup>5</sup> These data were previously reported (28).

<sup>6</sup> P < 0.001.
Blacks had significantly \((P < 0.01)\) lower concentrations of fecal calcium, which resulted from higher net intestinal calcium absorption, than did whites, regardless of diet (Table 2). Blacks had significantly \((P < 0.01)\) higher fecal sodium excretion than did the whites when consuming the high-sodium diet, but not when consuming the low-sodium diet, as previously reported by Palacios et al (28) (Table 2). There were no significant differences in 24-h sweat calcium excretion in this study (Table 1) that were due to diet or race, as previously reported (26). The mean habitual dietary calcium intake (Table 1), calculated from six 24-h dietary recalls before the study, was 277 mg/d (7 mmol/d) lower in the blacks than in the whites \((P < 0.05)\). The mean habitual dietary calcium intake in the adolescent girls during the period of peak bone accretion. Urinary calcium excretion in the adolescent black girls did not increase in response to increased sodium intake to the extent that it increased in the adolescent white girls. Blacks retained > 170 mg/d (4.2 mmol/d) more calcium than did whites \((P < 0.001)\), regardless of diet. If our study had been continued for 1 y and if
the acute effects were sustained, the racial difference in total-body calcium retention would become ≈62 g (1.6 mmol), whereas bone densitometry in our study found a radial difference of 38 g/d (1.0 mmol). The smaller racial difference in observed total-body calcium can be explained by the lower calcium intakes of the black girls when consuming their habitual diets. The peak rate of bone accretion in white girls occurs between the ages of 12 and 14 y (29); the mean age of the white girls in this study was at the midpoint of this range (ie, 13 y). Both races retained less calcium at high intakes of dietary sodium, but the racial difference in calcium retention was nearly twice the difference due to diet. The greater retention of calcium by the black adolescent girls than by the white adolescent girls was due to greater absorption of calcium, less excretion of urinary calcium, and greater rates of bone formation than of bone resorption (18). The study reported here showed less fecal calcium excretion in the blacks than in the whites, which is consistent with the former group’s higher calcium absorption. The racial difference in calcium retention among adolescents leads to significantly higher bone mass in black adults than in white adults (19, 20).

Reduced calcium retention with increasing dietary salt was likely due largely to reduced calcium absorption in both races and partially due to increased urinary excretion of calcium in whites. The higher net calcium absorption with lower salt intake in our study did not achieve significance because of the variation in fecal calcium, and the effect of dietary salt on calcium absorption should be quantified with more sensitive calcium tracer techniques. The earlier cross-sectional study in adolescents used 24-h urine collection in 370 adolescent white girls to predict the effect of urinary sodium and urinary calcium (16). When we used the same linear regression equation as was used in that study, the difference in urinary calcium calculated from the obtained difference in urinary sodium excretion in our study [Δ 2.56 g (111 mmol)], which was also described by Palacios et al (28), was predicted to be 52 mg (1.3 mmol). Thus, predicting the effect of dietary salt on urinary calcium from cross-sectional analysis overestimates the actual effect in white adolescents by 1.4-fold.

In contrast, in the current study, the blacks excreted significantly less urinary calcium while consuming a high-sodium diet than the whites. The difference in urinary calcium excretion between low-sodium and high-sodium diets [Δ 2.56 g (111 mmol)] was 3 mg/d (0.1 mmol/d) in the blacks. With the use of the same linear regression equation (16), the predicted effect of dietary salt on urinary calcium would have overestimated the effect even more in the blacks. The finding that increased dietary sodium led to increased urinary calcium in the white but not in the black adolescent girls indicated a possible genetic predisposition in whites for a greater effect of salt on the kidneys.

The greater sodium retention in blacks consuming a high-sodium diet that was observed in the current study was described previously by Palacios et al (28). We found that the higher sodium retention reflected in the lower urinary sodium output was not due to differences in fecal or sweat sodium excretion. We hypothesized that, because we observed no weight gain or increase in blood pressure to have resulted from the unexpectedly high sodium retention, sodium might have been temporarily deposited into the bones during this short pubertal growth period, as previously suggested by Palacios et al (28). Such an occurrence would be similar to results with calcium that are due to the higher rate of bone turnover found in blacks (18). In our study, the lack of a racial difference in urinary sodium excretion with the low-sodium diet, accompanied by significantly less urinary calcium excretion in the blacks, suggests that racial differences in calcium retention and sodium retention and excretion are unrelated. However, at high sodium intakes, we did observe significant effects of race (P < 0.001) and diet (P < 0.01) on calcium retention.

Although, before the current study, blacks appeared to consume less calcium before the study and had a higher 1,25(OH)₂D concentration than did whites, these variables did not predict calcium retention in these subjects and therefore do not offer an explanation for the racial difference in calcium retention that we found. In a previous study of calcium balance, we predicted a cumulative adult racial difference of 12% in the bone mass of black and white women (18), which would be consistent with the 10–13% higher adult bone density previously reported in black women than in white women (19, 20). If dietary salt were reduced from the higher to the lower intakes used in the current study [Δ 2.56 g Na/d (111 mmol Na/d)], the expected additional annual bone gain from the baseline total-body bone calcium content due to increased calcium retention would be 4% for black girls and 2% for white girls during their peak growth spurt. Bailey et al (29) estimated that modification of another lifestyle factor, physical activity, could increase total-body bone mineral content by 11% at 1 y after the peak in accretion rates (at age 12.5 y) in white girls. Although race (the strongest predictor of bone mass), diet, and physical activity play important roles in maximizing bone mass during peak bone accretion, dietary sodium may also affect bone development, but in whites more than in blacks.

We thank Ania Kempa-Steczek, Ron McClintock, and the Camp Calcium staff for excellent technical assistance and the subjects for their cooperation in this study.

CMW, MP, JHP, and GPM were responsible for the design of the study. KW, CP, LAJ, and BRM were responsible for the conduct of the study and data collection. GMP, KW, CP, BRM, and LDM were responsible for data analysis. KW, CMW, MP, and JHP were responsible for manuscript preparation. None of the authors had a personal or financial conflict of interest.

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