Effect of Vitamin D Supplementation on Blood Pressure in Blacks

John P. Forman, Jamil B. Scott, Kimmie Ng, Bettina F. Drake, Elizabeth Gonzalez Suarez, Douglas L. Hayden, Gary G. Bennett, Paulette D. Chandler, Bruce W. Hollis, Karen M. Emmons, Edward L. Giovannucci, Charles S. Fuchs, Andrew T. Chan

Abstract—Blacks have significantly higher rates of hypertension than whites, and lower circulating levels of 25-hydroxyvitamin D. There are few data about the effect of vitamin D3 (cholecalciferol) supplementation on blood pressure in blacks. During 2 winters from 2008 to 2010, 283 blacks (median age, 51 years) were randomized into a 4-arm, double-blind trial for 3 months of placebo, 1000, 2000, or 4000 international units of cholecalciferol per day. At baseline, 3 months, and 6 months, systolic and diastolic pressure and 25-hydroxyvitamin D were measured. The 3-month follow-up was completed in 250 (88%) participants. The difference in systolic pressure between baseline and 3 months was +1.7 mm Hg for those receiving placebo, −0.66 mm Hg for 1000 U/d, −3.4 mm Hg for 2000 U/d, and −4.0 mm Hg for 4000 U/d of cholecalciferol (−1.4 mm Hg for each additional 1000 U/d of cholecalciferol; P = 0.04). For each 1-ng/mL increase in plasma 25-hydroxyvitamin D, there was a significant 0.2-mm Hg reduction in systolic pressure (P = 0.02). There was no effect of cholecalciferol supplementation on diastolic pressure (P = 0.37). Within an unselected population of blacks, 3 months of oral vitamin D3 supplementation significantly, yet modestly, lowered systolic pressure. Future trials of vitamin D supplementation on blood pressure are needed to confirm these promising results, particularly among blacks, a population for whom vitamin D deficiency may play a more specific mechanistic role in the pathogenesis of hypertension. (Hypertension. 2013;61:779-785.) • Online Data Supplement

Key Words: black ■ blood pressure ■ hypertension ■ randomized controlled trial ■ vitamin D
Recruitment and Randomization
Participants in Open Doors to Health were invited to participate if they were aged 30 to 80 years, understood written and spoken English, self-identified as black,^1^1–14 and had permission from their primary care doctors. Participants were enrolled during winter to minimize the effects of sun exposure on vitamin D levels. We excluded individuals who had preexisting disorders of calcium metabolism and parathyroid function, type 1 diabetes mellitus, sarcoidosis, active malignancy (other than nonmelanoma skin cancer), or active thyroid disease. We also excluded those with cognitive impairment or who planned a vacation or extended travel to a sunny region during the supplementation phase of the study. Those taking vitamin D containing supplements were enrolled if they agreed to discontinue these medications for 6 months before enrollment and during the study.

A total of 328 participants were enrolled into the parent trial during the winters of 3 consecutive years, from 2007 to 2010 (Figure S1 in the online-only Data Supplement). In year 2 of the study, we amended the protocol to include BP measurements as an additional end point. Thus, to assess the effects of vitamin D supplementation on BP, we did not include the 45 participants enrolled in year 1 in whom we did not measure BP, leaving 283 participants enrolled in years 2 and 3 for this analysis (2008–2010).

Treatments
Participants were randomly assigned in a 1:1:1:1 ratio to 1 of 3 doses of cholecalciferol (vitamin D3): 1000 IU, 2000 IU, and 4000 IU; or placebo (Pharmavite LLC, Mission Hill, CA). All capsules also contained 200 mg of calcium. All capsules were indistinguishable, and both participants and research staff were blinded to treatment assignment. Study medications were initiated during early winter (November or December) and were taken orally once daily for 3 months (completed in February or March).

End Points and Follow-up
The primary end points of the study were the changes in systolic and diastolic BP (SBP and DBP) from baseline to the 3-month follow-up (at the end of randomized treatment). BP was also measured at the 6-month follow-up, 3 months after treatment was discontinued. At each follow-up assessment, trained study personnel obtained 3 BP readings at 5-minute intervals with participants in the seated position and feet flat on the floor. BP was determined with the OMRON HEM-907 device (Omron Healthcare Inc, Bannockburn, IL). This device has been validated^15–17^ and has been used in previous clinical trials. BP at each assessment was defined as the mean of the second and third readings.

Participants attended study visits at baseline, 3 months (at the end of randomized treatment), and 6 months (3 months after treatment discontinuation). At each study visit, a blood specimen was collected; height and weight were measured, and a brief questionnaire was administered. This questionnaire was designed to ascertain socioeconomic and demographic factors, medical information, use of nonstudy medications, dietary intake of vitamin D containing foods, use of supplements, physical activity, and smoking.

Compliance and Safety
We monitored adherence and compliance with biweekly telephone calls, monthly visits, electronic pill dispenser systems, and pill counts. To assess for toxicity, we informed participants of the potential for hypercalcemia, educated them on the warning signs and symptoms, and asked participants to call if he or she experienced any such signs or symptoms. We also screened for these problems during biweekly phone calls. Any concerning findings were noted and reported to the study physician, who made a determination about relatedness to vitamin D supplementation and the need for treatment discontinuation and further evaluation.

Plasma Vitamin D Levels
Blood samples collected at baseline, 3 months, and 6 months were separated, and plasma was stored in liquid nitrogen in the Dana-Farber Cancer Institute Clinical Research Laboratory (Boston, MA). After completion of the study, all plasma samples were sent as a single batch to the laboratory of Dr Bruce Hollis (Medical University of South Carolina, Charleston, SC), where 25(OH)D concentrations were measured using the Diasorin radiomunoassay. All laboratory personnel were blinded to treatment assignment. Masked quality control samples were also assayed; the mean coefficient of variation of 25(OH)D measurements was 9%.

Statistical Analysis
The trial was designed with a statistical power of 80% to detect differences in plasma 25(OH)D level of 5.3 ng/mL between treatment groups. Based on this planned sample size and accounting for exclusion of the 45 participants who already completed the study before modification of the protocol to include BP measurements, we estimated that the trial would have 80% power to detect a decrease in SBP of 4.1-mm Hg per 1000 IU/d of vitamin D3 supplementation, and 90% power to detect a 4.7-mm Hg decrease in SBP. Differences in the baseline characteristics of participants across the 4 treatment groups were compared using the Kruskal-Wallis test for continuous variables and a χ² test for categorical comparisons.

The primary end points were 3-month change in SBP at the end of treatment (ie, SBP at 3 months minus SBP at baseline) and 3-month change in DBP at the end of treatment (ie, DBP at 3 months minus DBP at baseline). For our primary analysis, we used linear regression with the dose of vitamin D3 (per 1000 IU/d) as the independent variable and the 3-month change in SBP (or 3-month change in DBP) as the dependent variables.

We performed a number of a priori secondary analyses. First, we analyzed the change in SBP and DBP according to the change in plasma 25(OH)D levels. Second, we analyzed the primary end point after excluding individuals who were taking antihypertensive medications at baseline. Third, we analyzed the effect of any vitamin D supplementation (all 3 treatment groups combined) compared with placebo on SBP and DBP. Fourth, we analyzed the persistence of treatment effects by examining the change in BP from the baseline to the 6-month examination, 3 months after treatment was discontinued.

We also performed 3 post hoc secondary analyses. Because a previous meta-analysis found that the effect of vitamin D supplementation on BP was greater in those individuals with higher baseline BP,^6^ we analyzed the primary end point after stratifying by baseline SBP (<120 mm Hg, ≥120 mm Hg). In addition, we determined the effect of vitamin D supplementation according to whether or not participants were vitamin D deficient at baseline (plasma 25(OH)D<20 ng/mL, ≥20 ng/mL). We tested whether the effect of cholecalciferol on BP was significantly different according to baseline SBP and plasma 25(OH)D by constructing interaction terms and including these terms in our linear regression models. We also performed a post hoc secondary analysis in which we reanalyzed the effect of vitamin D supplementation on SBP and DBP after adjusting for baseline SBP and DBP, respectively.

Results
Participant Characteristics
The current analysis comprises 283 participants who were enrolled and randomized after the inclusion of BP as an additional end point (Figure S1). The compliance rate with randomized therapy in the entire cohort was 96.6%. The 3-month
follow-up was completed in 250 of 283 participants (88%). These 250 individuals who had available BP measurements at baseline and at the end of treatment at 3 months were included in our primary analysis. Participants who did not have available BP measurements at 3 months were evenly distributed across the treatment groups.

The majority of participants reported being non-Hispanic black; 6.7% reported Hispanic ethnicity. The median age of the entire cohort was 51 years (interquartile range, 44–59 years), and the median body mass index was 31.0 kg/m² (interquartile range, 26.7–36.2 kg/m²). The median BP was 122/78 mm Hg, and 41.7% of participants were taking antihypertensive medications. Baseline characteristics according to randomized treatment assignment are displayed in Table 1. There were no statistically significant differences in any of the participant characteristics across the 4 groups. However, there did appear to be a nonsignificantly higher baseline SBP among those assigned 4000 IU/d as compared with those assigned placebo.

### Effect of Supplementation on 25(OH)D Levels

The effect of the various doses of cholecalciferol supplementation on 25(OH)D levels after 3 months, as well as the persistence of the effect 3 months after cessation of supplements, is depicted in Figure S2A. At 3 months, median 25(OH)D levels rose to 45.9, 34.8, and 29.7 ng/mL, respectively, in participants assigned to 4000, 2000, and 1000 IU/d. By 6 months, median levels fell to 31.2, 27.0, and 21.2 ng/mL, respectively, in participants assigned to 4000, 2000, and 1000 IU/d. However, 25(OH)D levels at 6 months remained higher than the baseline levels.

### Primary Analysis

The primary efficacy analyses of vitamin D3 supplementation on SBP and DBP are shown in Table 2. For each additional 1000 IU/d of cholecalciferol, SBP was significantly decreased by 1.4 mm Hg (P=0.04). There was no effect of cholecalciferol supplementation on DBP, which fell by 0.5 mm Hg for each additional 1000 IU/d (P=0.37).

### A Priori Secondary Analyses

The change in plasma 25(OH)D levels associated with vitamin D3 supplementation was also associated with change in SBP but not DBP. For each 1 ng/mL greater increase in 25(OH)D level between baseline and 3 months, there was a significant 0.2-mm Hg decrease in SBP (P=0.02). In addition, for each 1-ng/mL greater increase in 25(OH)D level between baseline and 6 months, there was a significant 0.2-mm Hg decrease in SBP (P=0.05).

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Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All</th>
<th>Placebo</th>
<th>1000</th>
<th>2000</th>
<th>4000</th>
<th>PValue</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>283</td>
<td>72</td>
<td>68</td>
<td>73</td>
<td>70</td>
<td>0.86</td>
</tr>
<tr>
<td>25(OH)D, ng/mL, Median (IQR)</td>
<td>15.7 (10.7–23.4)</td>
<td>16.3 (11.3–23.9)</td>
<td>16.3 (11.0–23.0)</td>
<td>14.5 (9.9–22.9)</td>
<td>15.6 (11.0–22.9)</td>
<td>0.86</td>
</tr>
<tr>
<td>SBP, mm Hg, Median (IQR)</td>
<td>122 (112–136)</td>
<td>120 (108–135)</td>
<td>123 (112–136)</td>
<td>121 (110–134)</td>
<td>128 (118–140)</td>
<td>0.07</td>
</tr>
<tr>
<td>DBP, mm Hg, Median (IQR)</td>
<td>78 (71–86)</td>
<td>78 (71–84)</td>
<td>80 (70–88)</td>
<td>75 (71–84)</td>
<td>78 (71–87)</td>
<td>0.47</td>
</tr>
<tr>
<td>Age, y, Median (IQR)</td>
<td>51 (44–59)</td>
<td>51 (44–59)</td>
<td>51 (43–59)</td>
<td>50 (43–57)</td>
<td>51 (46–60)</td>
<td>0.86</td>
</tr>
<tr>
<td>BMI, kg/m², Median (IQR)</td>
<td>31.0 (26.7–36.2)</td>
<td>31.1 (26.5–35.7)</td>
<td>30.8 (27.6–37.7)</td>
<td>30.5 (26.0–36.9)</td>
<td>31.2 (27.7–35.7)</td>
<td>0.82</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>185 (65.4)</td>
<td>46 (63.4)</td>
<td>50 (73.5)</td>
<td>46 (63.0)</td>
<td>43 (61.4)</td>
<td>0.53</td>
</tr>
<tr>
<td>Hispanic, n (%)</td>
<td>19 (6.7)</td>
<td>3 (4.2)</td>
<td>7 (10.3)</td>
<td>4 (5.5)</td>
<td>5 (7.1)</td>
<td>0.76</td>
</tr>
<tr>
<td>Work status, n (%)</td>
<td>106 (37.5)</td>
<td>26 (36.1)</td>
<td>26 (38.2)</td>
<td>29 (39.7)</td>
<td>25 (35.7)</td>
<td>0.76</td>
</tr>
<tr>
<td>Used</td>
<td>41 (14.5)</td>
<td>12 (16.7)</td>
<td>10 (14.7)</td>
<td>9 (12.3)</td>
<td>10 (14.3)</td>
<td>0.76</td>
</tr>
<tr>
<td>Retired</td>
<td>122 (43.3)</td>
<td>29 (40.2)</td>
<td>31 (46.3)</td>
<td>33 (45.2)</td>
<td>29 (41.4)</td>
<td>0.13</td>
</tr>
<tr>
<td>Education beyond high school, n (%)</td>
<td>89 (31.4)</td>
<td>20 (27.8)</td>
<td>25 (36.8)</td>
<td>23 (31.5)</td>
<td>21 (30.0)</td>
<td>0.71</td>
</tr>
<tr>
<td>Married, n (%)</td>
<td>88 (31.1)</td>
<td>24 (33.3)</td>
<td>25 (36.8)</td>
<td>21 (28.8)</td>
<td>18 (25.7)</td>
<td>0.68</td>
</tr>
<tr>
<td>Smoking status, n (%)</td>
<td>69 (24.4)</td>
<td>17 (23.6)</td>
<td>14 (20.6)</td>
<td>22 (30.1)</td>
<td>16 (22.9)</td>
<td>0.10</td>
</tr>
<tr>
<td>Current</td>
<td>52 (18.4)</td>
<td>8 (11.1)</td>
<td>15 (22.1)</td>
<td>11 (15.1)</td>
<td>18 (25.7)</td>
<td>0.03</td>
</tr>
<tr>
<td>Past</td>
<td>21 (7.4)</td>
<td>8 (11.1)</td>
<td>5 (7.4)</td>
<td>2 (2.7)</td>
<td>6 (8.6)</td>
<td>0.23</td>
</tr>
<tr>
<td>History of hypertension, n (%)</td>
<td>141 (49.8)</td>
<td>35 (48.6)</td>
<td>35 (51.5)</td>
<td>36 (49.3)</td>
<td>35 (50.0)</td>
<td>0.99</td>
</tr>
<tr>
<td>Takes antihypertensive medication, n (%)</td>
<td>118 (41.7)</td>
<td>34 (47.2)</td>
<td>29 (42.6)</td>
<td>27 (30.7)</td>
<td>28 (40.0)</td>
<td>0.71</td>
</tr>
<tr>
<td>Takes OCPS (if female), n (%)</td>
<td>16 (8.6)</td>
<td>3 (4.5)</td>
<td>3 (4.5)</td>
<td>4 (8.7)</td>
<td>6 (14.0)</td>
<td>0.30</td>
</tr>
<tr>
<td>Menopausal (if female), n (%)</td>
<td>106 (57.3)</td>
<td>30 (65.2)</td>
<td>26 (62.0)</td>
<td>23 (50.0)</td>
<td>27 (62.8)</td>
<td>0.33</td>
</tr>
</tbody>
</table>

BMI indicates body mass index; DBP, diastolic blood pressure; IQR, interquartile range; IU, international unit; OCP, oral contraceptive pill; SBP, systolic blood pressure; and 25(OH)D, plasma 25-hydroxyvitamin D level.

*These supplements were stopped before randomization.
We repeated our primary analysis after excluding those 118 individuals who were taking antihypertensive medications. Among the remaining 132 participants, each additional 1000 IU/d of cholecalciferol supplementation was associated with a decrease in SBP that was similar in magnitude to the whole population (1.2 mm Hg); however, this result was not statistically significant (P=0.24), possibly owing to the restricted sample size.

We also examined the effect of any dose of cholecalciferol supplementation (ie, all 3 treatment groups combined) on 3-month change in BP compared with placebo. In these analyses, cholecalciferol supplementation at any dose lowered SBP by 4.4 mm Hg, an effect which approached statistical significance (P=0.07).

To assess whether the effect of vitamin D3 supplementation persisted after discontinuation of supplementation, we examined the change in BP at the 6-month examination, 3 months after study treatment was discontinued. We did not observe any significant effect of randomized treatment on either SBP or DBP at the 6-month examination in comparison with the baseline examination (Figure S2B and S2C). For each 1000 IU/d additional cholecalciferol supplementation, the 6 month SBP was decreased by 0.4 mm Hg (P=0.56) and DBP was decreased by 0.2 mm Hg (P=0.75).

Post Hoc Secondary Analyses
We repeated our primary analysis after stratifying by baseline SBP (<120 mm Hg, ≥120 mm Hg) and baseline plasma 25(OH)D (<20 ng/mL, ≥20 ng/mL). The effect of cholecalciferol on BP was identical among those whose baseline SBP<120 mm Hg or ≥120 mm Hg. In contrast, the effect of each additional 1000 IU/d of cholecalciferol on SBP was larger among those whose baseline plasma 25(OH)D was <20 ng/mL (a 2.2 mm Hg decrease; P=0.03) than among those whose baseline plasma 25(OH)D was ≥20 ng/mL (a 0.4 mm Hg decrease; P=0.66). However, these effects were not statistically different from each other (P interaction=0.33).

We observed a clinically but not statistically significant difference in baseline SBP among the treatment groups (Table 1). Thus, we repeated our primary analysis after adjusting for baseline BP. In these models, each 1000 IU/d additional cholecalciferol dose nonsignificantly lowered systolic pressure by 0.7 mm Hg (P=0.29).

Discussion
To our knowledge, this is the largest randomized, double-blind, placebo-controlled trial examining the effects of cholecalciferol supplementation among black individuals. On an intent-to-treat basis, supplementation with cholecalciferol for 3 months significantly lowered SBP, although no effect on DBP was observed. In addition, a greater increase in plasma 25(OH)D level in response to supplementation was significantly associated with a larger decrease in SBP. The magnitude of the effect was greater with higher doses of cholecalciferol, but overall was clinically modest (a 1.4-mm Hg decrease in SBP for every 1000 IU/d given).

Our findings suggest that, among blacks, vitamin D supplementation may play a role in lowering BP. However, these conclusions are tempered by our observation that the baseline SBP was greatest among participants treated with the highest dose of cholecalciferol, raising the possibility that the effect of cholecalciferol could have been attributable, in part, to regression to the mean. However, all 3 groups treated with cholecalciferol had declines in SBP (Figure S2B), and there was a significant decrease in SBP associated with increasing 25(OH)D levels in response to supplementation.

To date, >10 trials have evaluated the effect of vitamin D therapy on BP,21–36 4 of which were designed specifically to evaluate BP as a primary end point.30,31,35,36 Of these 4 trials, our results are supported by a placebo-controlled trial which showed that daily intake of 800 IU of cholecalciferol significantly reduced SBP by 7 mm Hg after 8 weeks of treatment among 148 individuals.35 In contrast, a single dose of cholecalciferol 100,000 IU did not lower BP after 5 weeks in a placebo-controlled trial of 189 individuals with vitamin D deficiency.36 This trial was limited by its short duration, relatively small sample size, and modest 7-ng/mL rise in 25(OH)D levels in response to supplementation.30

The largest trial (Women’s Health Initiative, n=36,282) was designed to evaluate fracture and cancer risk in a population of largely vitamin D–insufficient women; cholecalciferol (400 IU/daily) with calcium was not associated with changes in BP or incident hypertension after 7 years of follow-up.38 However, the dose of cholecalciferol used was low and not expected to significantly increase 25(OH)D levels,37,39 the rate of medication noncompliance was high, and 60% of women assigned to placebo also consumed supplemental vitamin D.
Three meta-analyses combining most of these previous trials failed to observe an overall lowering of BP associated with vitamin D supplementation. However, vitamin D supplementation was associated with significant decreases in BP in meta-analyses limited to trials comprising only hypertensive individuals or to trials using higher doses of vitamin D (>1000 IU/daily). A major limitation of these previous studies was the limited representation by black individuals. Compared with whites, blacks have lower circulating 25(OH)D levels and a higher prevalence of vitamin D deficiency. Moreover, increased BP among blacks is believed to be more strongly mediated by inappropriately elevated activity of the renin-angiotensin system (RAS), which is known to be associated with hypertension and cardiovascular disease. The most well-supported mechanism by which vitamin D may affect BP is its role as a negative regulator of the RAS. The development of vitamin D receptor null mice has facilitated numerous experiments illuminating the relation among vitamin D, the RAS, and hypertension. Vitamin D receptor null mice, for example, have significant elevations in renin activity and circulating plasma angiotensin II concentrations, and have hypertension and cardiac hypertrophy that is attenuated when RAS antagonists are administered. Recently, human mechanistic studies have shown that lower levels of 1,25(OH)2D and 25(OH)D are associated with higher plasma renin and angiotensin II concentrations, and that lower 25(OH)D levels are associated with higher renin activity and circulating plasma angiotensin II concentrations, and have hypertension and cardiac hypertrophy that is attenuated when RAS antagonists are administered.

Our study has several strengths. First, to our knowledge, this is the first randomized, double-blind, placebo-controlled trial to examine the specific effect of vitamin D supplementation on BP among blacks. Second, we used a range of cholecalciferol doses that were sufficient to raise plasma 25(OH)D levels to normal among a population that was largely vitamin D–deficient or insufficient at baseline. Third, participants were treated only during the winter months to minimize the influence of seasonal variations on circulating 25(OH)D levels. Last, our compliance with study medications was >95% and ascertainment of BP was performed in a standardized format with a research quality device. Our study also has several limitations. First, despite randomization, the treatment groups were imbalanced with respect to baseline SBP. The effect of supplementation on change in systolic pressure was attenuated, and no longer significant, after adjusting for baseline systolic pressure. This raises the concern that significant BP-lowering effect that we observed may have been partly explained by regression to the mean; however, the direction of the effect is toward a benefit from vitamin D supplementation, and the lack of significance could be attributable to limited power. In addition, we did observe significant decreases in SBP with increasing plasma 25(OH)D levels, supporting a true causal association. Second, although we designed our intervention for 3 months to minimize the influence of major seasonal variation in 25(OH)D levels, it is possible that a longer duration of treatment may be required to fully assess the effect of supplementation on BP. Third, ≈40% of the participants in our study were taking antihypertensive medications at baseline, which could potentially mask the effects of supplementation. However, this would tend to attenuate our observed associations. Moreover, our results were similar among those who did and did not take antihypertensive medications. Finally, our study did not evaluate the mechanisms that may underlie the effects of vitamin D supplementation.

**Perspectives**

In this randomized, placebo-controlled trial, cholecalciferol supplementation significantly lowered SBP in blacks. If confirmed, our results may also partly inform future therapeutic strategies among blacks for whom vitamin D deficiency may play a more specific mechanistic role in the pathogenesis of hypertension.

**Sources of Funding**

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**Disclosures**

The authors of this article had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Statistical analyses were performed by an author (D.L. Hayden), whose affiliations are listed.

**References**

47. Tomashitz A, Pilz S, Ritz E, Grammer T, Drechsler C, Boehm BO, März W. Independent association between 1,25-dihydroxyvitamin


**Novelty and Significance**

**What Is New?**

- Although various randomized trials have examined whether or not vitamin D supplementation can decrease blood pressure in predominantly white populations, this had not been tested in blacks.

**What Is Relevant?**

- Low levels of 25-hydroxyvitamin D are consistently associated with increased blood pressure or a higher risk of developing hypertension.
- If this association was causal, vitamin D deficiency may, in part, explain the discrepant hypertension risk among blacks in the United States.

**Summary**

We found that, compared with placebo, vitamin D supplementation modestly but significantly reduced systolic blood pressure. In addition, greater increases in 25-hydroxyvitamin D levels during the study period produced larger reductions in systolic blood pressure.

If confirmed, these results may inform future therapeutic strategies to reduce blood pressure or prevent hypertension among blacks.
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