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Vitamin D Economy in Blacks

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ABSTRACT: The fact that fracture risk is lower and BMD is higher in blacks compared with whites is surprising in light of what is known about vitamin D status in blacks. Mean 25(OH)D levels are lower in blacks than whites at all stages of life, and a greater proportion of blacks meet criteria for vitamin D deficiency. The racial difference in serum 25(OH)D level is primarily caused by increased pigmentation reducing vitamin D production in the skin. In response to lower 25(OH)D levels and lower average calcium intake, blacks have higher average PTH levels and a higher prevalence of secondary hyperparathyroidism (twice the prevalence compared with whites for both sexes). This is associated with higher average levels of $1,25(OH)_2D$ and lower urinary calcium excretion but not higher biochemical indices of bone turnover. In fact, in general, biochemical indices of bone formation (particularly osteocalcin levels) are lower in blacks. Bone formation rates assessed histomorphometrically are also lower, although wall thickness is maintained. During a 24-h PTH infusion, increments in levels of three different bone resorption markers are significantly lower in blacks than in whites, providing direct confirmation of the thesis that the black skeleton is resistant to the bone-resorbing effects of PTH, whereas renal sensitivity to PTH is maintained or perhaps even enhanced. Vitamin D supplementation studies in black women have shown inconsistent benefits to BMD. Skeletal and renal adaptations to vitamin D deficiency in blacks might be so effective that vitamin D supplementation might not confer any further benefit to the black skeleton. Benefits of vitamin D supplements in blacks may still play a role, however, in the prevention of other chronic diseases.
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INTRODUCTION

Epidemiological data from multiple U.S. populations have confirmed that white women have consistently higher fracture rates in the hip[1–5] and the rest of the skeleton compared with black women.[1,6,7] For example, in a large Medicare database (total sample of >1.4 million people), the actuarial risk of hip fracture was 16% for a 90-yr-old white woman and 5.3% for a 90-yr-old black woman.[6] Data from the NORA database (total cohort 197,848 women >50 yr of age) indicate that the 1-yr incidence of any fracture was 1.5% for white women and 0.8% for black women.[8]

Racial differences in fracture risk are related in part to differences in body composition, body size, and bone size.[9–14] Differences in hip and pelvic geometry, including shorter hip axis length and thicker cortical bone in the hip, in black individuals may also contribute to a lower fracture risk in black women.[15–18]

The racial difference in fracture rates is also consistent with the racial difference in BMD observed in all groups.[2,8,9,19–21] Data from NHANES III showed an 11–17% difference in BMD at different sites within the hip for different ages between blacks and whites. Racial differences in BMD were seen in children and adolescents, premenopausal and postmenopausal women, and men ≥20 yr of age.[2,8,9,11,20,22–25] Furthermore, significant differences in rates of postmenopausal bone loss were seen in blacks versus whites at both the total hip (0.334%/yr versus 0.574%/yr) and femoral neck (0.203%/yr versus 0.515%/yr) in the Study of Osteoporotic Fracture cohort,[2] whereas in a much smaller cohort, slower bone loss rates in black women in the spine and forearm were seen only in early postmenopausal women.[22]

The findings, regarding fracture risk and BMD, are surprising in light of what is known about vitamin D status in blacks. Mean 25(OH)D levels are lower in blacks versus whites at all stages of life, and a greater proportion of blacks meet criteria for vitamin D deficiency or insufficiency with any of the multiple cut-points used,[26–36] with very few exceptions.[29] For example, winter measurements from southern latitude populations from NHANES indicate that 13–33% of white men and women >60 yr of age versus 33–69% of black men and women of similar age have 25(OH)D levels <50 nM.[26] The racial difference in serum

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25(OH)D level is primarily caused by increased pigmentation reducing vitamin D production in the skin.[37,38] Seasonal differences in 25(OH)D levels are correspondingly blunted in blacks.[39,40] Vitamin D intakes are also lower in blacks than those in whites, in part related to reduced consumption of milk, milk products, and cereals.[28,41]

So how can the black skeleton remain strong in the face of vitamin D deficiency? Norman Bell proposed, in 1985, that even though blacks had expected secondary hyperparathyroidism in response to the vitamin D deficiency, the fact that bone turnover markers were lower in blacks (assessed biochemically and histomorphometrically), indicated that the skeleton was resistant to the effects of PTH.[32,42] He further proposed that renal responses to PTH, with respect to urinary calcium excretion, were preserved in blacks. This paper will review the data subsequently collected to address this hypothesis.

SECONDARY HYPERPARATHYROIDISM AND ITS CONSEQUENCES

In response to lower 25(OH)D levels, in addition to lower average calcium intake,[28,43–45] black women have relative secondary hyperparathyroidism (average PTH levels in the upper normal range and higher incidence of PTH levels above the normal range) and resultant higher levels of $1,25(OH)_2D$ and lower urinary calcium excretion. Studies showing elevated PTH include premenopausal and postmenopausal women and men.[34,35,41,42,46] For example, in the Boston Low Income Elderly Osteoporosis study,[28] in women, mean serum PTH was 5.3 pM in white women and 7.5 pM in black women, and in men was 4.3 pM in whites and 6.2 pM in blacks. The prevalence of secondary hyperparathyroidism in black women and men was almost twice that of whites in both sexes. Serum $1,25(OH)_2D$ levels were higher in blacks versus whites in studies involving children, adolescents, and young and older adult men and women.[30,34] Rarely, no significant racial difference has been found regarding level of $1,25(OH)_2D$.[29]

Also consistent with relative secondary hyperparathyroidism, urinary calcium excretion is consistently lower in blacks versus whites across age groups from children to older adults.[25,30,31,35,36,46,47] Bell et al.[47] showed that average urine calcium excretion in black children was about one half that in white children (84 versus 165 mg/d). Bryant et al.[46] showed urine calcium excretion to be 46% lower in black compared with white adolescent girls) and Wigertz et al.[36] confirmed that black adolescent girls had greater renal calcium retention at both low and high sodium intake diets. Ettinger et al.[31] and Bikle et al.[34] also reported lower urine calcium excretion in adult black women and men. Dawson-Hughes et al.[35] showed that black women had lower urine calcium excretion in response to $1,25(OH)_2D$ administration compared with white women. In postmenopausal women, Kleerekoper et al.[30] showed the racial difference in urinary calcium excretion.

CALCIUM ABSORPTION

Whereas the data are consistent for renal calcium handling, whether there are racial differences in calcium absorption is not clear. Bell et al.[47] showed no racial differences in calcium absorption efficiency in children, but Bryant et al.[46] did show greater calcium absorption in black (54%) compared with white adolescents (38%), as did Abrams et al.[48] Dawson Hughes et al.[49] showed no racial differences in premenopausal women and suggested that the increment in calcium absorption to provocative testing with $1,25(OH)_2D$ was actually blunted in black women.[35]

BONE REMODELING RATES

Despite the relative secondary hyperparathyroidism, biochemical measurements of bone turnover levels are, in general, lower in blacks versus whites. Some inconsistencies here may relate to differences among the bone turnover markers chosen, analytical issues, methods of sample collection, assays used, differential effects in different populations, population sizes, diurnal variation, and effects of exercise and other biological variables. Nevertheless, lower levels of osteocalcin in blacks have been shown quite consistently in young women and men, as well as in perimenopausal or postmenopausal women.[23,25,34,50] Lower levels of bone resorption markers have also been shown in black adolescent girls,[46] pre- and perimenopausal women,[25] and postmenopausal women.[25,30,51] However, in several studies, no significant racial differences in bone turnover markers have been seen in various populations.[52] In one calcium balance study, net calcium retention was higher in black versus white girls (greater formation versus resorption), possibly as a function of their age (adolescents undergoing rapid bone mass accrual).[46] Although not all data are consistent, the expected finding in a population with relative secondary hyperparathyroidism would be an increase in bone turnover and not a decrease or lack of racial difference, as most studies report.

Histomorphometric assessments of racial differences comparing black and white premenopausal women are consistent with the biochemical bone turnover data.[50,53,54] In one major study, 142 women, including 31 blacks and 108 whites, were enrolled. Of the total, 61 were premenopausal and 81 were postmenopausal. Expected estrogen deficiency related changes were seen in both races. Racial comparisons showed that mean bone formation rate and mineralizing surface were 25% lower in blacks compared with whites. In another study,[54] 21 black and 34 white premenopausal women, matched for education and socioeconomic class, as well as body size and weight, were enrolled. This study showed no racial difference in activation frequency of bone remodeling. Consistent with the study of Parfitt and colleagues,[50,53] lower mineralizing surface, lower apposition rate, and adjusted mineral apposition rate (bone formation rate per osteoid surface) were all seen in black women, whereas the total formation period was longer. Although this may sound like a detrimental finding in blacks, in fact, slower bone formation may be associated with bone of greater strength. Furthermore, wall thickness, which reflects the amount of bone formed within each remodeling unit, was not different between blacks and whites. Filling in the remodeling cavity simply seems to take longer

longer in black women than in whites. One study performed in South Africa confirmed greater cancellous bone volume and trabecular thickness in blacks versus whites, but in sharp contrast to the U.S. studies, bone turnover was apparently higher in blacks than whites. However, turnover in that study was only assessed by static variables (osteoid volume, surface and thickness, and eroded surface), which may account for the discrepancy.[55]

DYNAMIC PERTURBATIONS OF THE PARATHYROID–VITAMIN D AXIS

To help answer questions posed by the measurement of biochemical variables in the static state, we used several dynamic tests to try to determine whether there were additional racial differences that could be provoked. We used EDTA testing to induce hypocalcemia in a group of premenopausal black ($n = 17$) and white ($n = 17$) women to determine whether there were racial differences in PTH secretory capacity.[56] Calcium levels declined precipitously in both groups, and PTH levels rose accordingly. There were no overall racial differences in the PTH response to hypocalcemia, with the exception of the PTH level measured at 24 h after EDTA infusion. PTH levels were still somewhat higher at that one time-point in black women, with the group difference being greater than what was seen at baseline before the provocative testing.

Administration of 0.5 μg $1,25(OH)_2D$ daily for 5 days in premenopausal black and white women was used to determine whether there were racial differences in urinary calcium excretion or bone turnover responses.[57] Slightly greater increments in bone formation markers (carboxyterminal propeptide of type 1 procollgen [P1CP] and osteocalcin [OC]) by days 4 and 5 were seen in black women. Urinary calcium responses to $1,25(OH)_2D$ were lower in black women than in whites, consistent with the results obtained by Dawson-Hughes et al. These results suggested that there are no problems with osteoblast reserve capacity in black women (despite the fact that bone formation rates are slightly slower at baseline in black women, as discussed above). Moreover, the renal calcium conservation response is particularly sensitive in black women.

We also questioned whether there are racial differences in skeletal and renal responses to PTH(1-34) administration by intravenous infusion over 24 h.[58] The protocol resulted in very similar increments in amino-terminal PTH measurements, elevations in serum calcium, and suppression of endogenous intact PTH levels. The baseline differences in urine calcium excretion were maintained throughout the infusion, with a slightly more dramatic suppression of urine calcium excretion in black women over the first 8 h of the infusion. Bone turnover variables were assessed in subgroups of black and white women over 24 h without PTH infusion to assess diurnal variation. Diurnal variations in all markers evaluated were apparent in the control groups. During the PTH infusion, there were decrements in both bone formation variables measured, with no differences between racial groups. In contrast, for all three bone resorption variables measured (urinary N-telopeptide, C-telopeptide, and pyridinoline), the increment in black women was less pronounced than the increment in white women. This study provided direct confirmation of the hypothesis that the black skeleton is resistant to bone-resorbing effects of PTH, whereas renal sensitivity to PTH is maintained or perhaps even heightened in blacks.

VITAMIN D SUPPLEMENTATION IN BLACK WOMEN

Aloia et al.[59] enrolled 208 healthy postmenopausal women (age range, 50–75 yr) and randomly assigned them to receive 800 IU vitamin D_3 daily for 2 yr, 2000 IU vitamin D_3 daily for 1 yr, or placebo. Mean serum 25(OH)D levels increased as expected from a baseline level of 18 to 28 ng/ml for the 800-IU daily dose and to 35 ng/ml for the 2000 IU daily dose. Nevertheless, over the 3-yr trial, there were no significant differences in BMD change in the vitamin D–supplemented group compared with the placebo group.

A second vitamin D supplementation study was performed in 79 postmenopausal women who received either 1000 IU vitamin D_3 daily or placebo for 2 yr.[60] Expected increments in 25(OH)D were seen in the vitamin D–supplemented group, although mean levels did not exceed 25 ng/ml at any time-point. Over the 2-yr trial, rates of bone loss were similar in the two groups at the spine, total hip, and trochanter but were slower in the femoral neck. When BMD responses were evaluated as a function of the FOK-1 domain of the vitamin D receptor gene, the homozygous FF group showed more dramatic losses at all skeletal sites in the placebo group, and this was blunted by vitamin D supplementation.

Lack of consistent effect on rates of bone loss may be a function of inadequate vitamin D dose, calcium supplementation in both active and control groups obfuscating treatment effects of vitamin D, or genetic variability of response in subgroups of black women. It might also be that the skeletal and renal adaptations to vitamin D deficiency are so effective that vitamin D supplementation might not confer any further benefit to the black skeleton. We should not extrapolate these findings, however, to potential nonskeletal benefits of vitamin D supplementation.

SUMMARY AND CONCLUSIONS

Black individuals have a lower risk of all fractures compared with whites and higher BMD at all ages, despite the high prevalence of vitamin D deficiency. Relative secondary hyperparathyroidism, as a response to vitamin D deficiency and low calcium intake, produces corresponding elevations in $1,25(OH)_2D$, and lower urinary calcium excretion. An expected increase in bone turnover is not seen, however. Biochemical indices of bone resorption are lower or show no racial differences, and bone formation measures are usually lower in blacks. Similarly, bone formation rates by histomorphometry are lower, although wall thickness is maintained. Responses to dynamic perturbations show that the black skeleton is resistant to the acute bone-resorbing effects of PTH, with maintenance, or perhaps even particularly high renal sensitivity to PTH. These elegant adaptive responses to vitamin D deficiency allow the skeleton to remain strong while still providing a means

for maintenance of calcium homeostasis. It is therefore unclear whether vitamin D supplementation can be effective at improving the black skeleton further. Benefits of vitamin D supplements may still play a role, however, in the prevention of other chronic diseases.

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