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Risk for Osteoporosis in Black Women

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Abstract. Models of involutional bone loss and strategies for the prevention of osteoporosis have been developed for white women. Black women have higher bone densities than white women, but as the black population ages there will be an increasingly higher population of black women with osteoporosis. Strategies should be developed to reduce the risk of black women for fragility fractures.

Dual energy X-ray absorptiometry measurements of the total body, femur, spine, and radius were performed on 503 healthy black and white women aged 20–80 years. Indices of bone turnover, the calcitrophic hormones, and radioisotope calcium absorption efficiency were also measured to compare the mechanisms of bone loss.

The black women had higher BMD values at every site tested than the white women throughout the adult life cycle. Black women have a higher peak bone mass and a slightly slower rate of adult bone loss from the femur and spine, which are skeletal sites comprised predominantly of trabecular bone. Indices of bone turnover are lower in black women as are serum calcidiol levels and urinary calcium excretion. Serum calcitriol and parathyroid hormone levels are higher in black women and calcium absorption efficiency is the same in black and white women, but dietary calcium intake is lower in black women.

Black and white women have a similar pattern of bone loss, with substantial bone loss from the femur and spine prior to menopause and an accelerated bone loss from the total skeleton and radius after menopause. The higher values for bone density in black women as compared with white women are caused by a higher peak bone mass and a slower rate of loss from skeletal sites comprised predominantly of trabecular bone. Low-risk strategies to enhance peak bone mass and to lower bone loss, such as calcium and vitamin D augmentation of the diet, should be examined for black women. The risk vs. benefits of hormonal replacement therapy should be determined, especially in older women.

Key words: Osteoporosis — Bone density — Race — Ethnicity — Fracture.

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Studies of involutional bone loss have been mostly limited to white women since they have the highest incidence of osteoporotic fractures. Comparative data for fracture incidence among the races is limited, but one study revealed the following age-adjusted fracture rates in black and non-Hispanic white women: 57.3 and 140.7 per 100,000, respectively [1]. The longevity of minorities is increasing and the proportion of older women in the U.S. population is increasing rapidly [2]. The relative protection from osteoporosis of black women has been overemphasized—older black women will increasingly represent a population at risk for osteoporotic fractures. Strategies for prevention of osteoporosis have been established for white women, whereas they remain to be proposed for black women [3, 4].

The comparability of values for bone density and the similarities in patterns of bone loss have been well established for white females of European origin [5–38]. Higher bone mineral density in American blacks as compared with whites has been found throughout the lifecycle, but most studies have not measured all skeletal sites of interest to establish whether the same model of bone loss may be applied to black and white women [39–46]. Cross-sectional and longitudinal data obtained in white women suggest a pattern of bone loss that differs for skeletal sites having different proportions of trabecular and cortical bone. Bone loss from the whole skeleton (80% cortical bone) and the distal radius is minimal prior to menopause in white women. There is, however, substantial premenopausal bone loss from sites having a greater proportion of trabecular bone (the proximal femur and lumbar spine) [5].

If there are racial differences in the pattern, magnitude, or determinants of bone mass, it may be inappropriate to extrapolate strategies for the prevention of osteoporosis, developed from data collected from white women, to other races. The possibility that such differences may exist is suggested by observations concerning racial differences in therapeutic drug responses, in bone density and bone turnover, in the vitamin D-endocrine system, and in body composition [47–57].

The purpose of the current study is to develop a comprehensive model to describe adult bone loss in black women and to compare these findings with observations in white women. Such a comparison could provide insight into which of the strategies recommended for the prevention of osteoporosis in white women are most promising for testing

in black women. Since our previous data collected on white women used older technologies (dual-energy photon absorptiometry as compared to X-ray absorptiometry), it was considered desirable to concurrently collect data on white women in order to have a valid comparison. Bone mineral density measurements by dual X-ray absorptiometry were made in 268 white and 235 black adult women. The specific questions concerning black and white differences we hoped to answer were: (1) Is the pattern of adult bone accretion and loss similar? (2) Are there differences in bone metabolism or in calcium-regulating hormones in black vs. white women? The long-term goal of this research is to propose strategies for prevention of osteoporotic fractures in black women.

Materials and Methods

Participants

Participants were recruited from advertising in the local media and through a direct mail campaign. Exclusion characteristics consisted of any chronic illness, including hypertension, diabetes, obesity, any past history of illness or medication known to affect bone metabolism, and any use of oral contraceptives or hormonal replacement therapy or hysterectomy. After telephone screening, women were further rejected because of abnormal blood chemistries (multichannel chemistries, CBC, urinalysis, free thyroxine, TSH) or abnormal physical findings. The study was approved by the Institutional Review Board of Winthrop-University Hospital and written informed consent was obtained from each participant. A detailed history and physical questionnaire for risk factors was completed with the assistance of a nurse clinical research coordinator and physician. A 3-day diet history was obtained and was reviewed with the study dietician using food models to estimate portion size. A 24-hour food recall form was also completed with the assistance of the dietician, as was a recall for consumption of dairy products from different age groups. A 1-week physical activity log was completed and the Compendium of Physical Activity was used to estimate energy expenditure [58]. A BMI of 18–33 was considered acceptable for inclusion in the study.

A total of 1293 phone calls were received. Six-hundred five women were rejected and 131 decided not to participate. Five-hundred fifty-seven were examined, of which 54 were excluded. The most common causes for exclusion were undiagnosed diabetes, thyroid disease, and anemia. There were 148 black and 129 white premenopausal participants and 87 black and 139 white postmenopausal participants.

Laboratory Studies

After initial screening, which demonstrated a normal physical exam and routine laboratory studies, the participants collected a 24-hour urine for calcium, hydroxyproline, and creatinine after three days of a low hydroxyproline diet. They also had urine collected in the morning following an overnight fast. Blood was drawn for routine chemistries and for indices of bone turnover and various hormonal assays.

Serum parathyroid hormone was measured by the Allegro intact PTH immunoassay, purchased from Nichols Institute, San Juan Capistrano, CA [59]. The intraassay CV was 5.2% and interassay CV was 9.0%. Serum 1,25-dihydroxyvitamin D was measured by a radio-receptor binding assay using calf thymus receptor. The kit was manufactured by INCSTAR. Serum was extracted by a single column containing a C180H-activated matrix prior to the receptor assay [60]. The intraassay CV was 8.5% and the interassay CV was 17.3%. Serum 25-hydroxyvitamin D was measured by a radioreceptor assay purchased from INCSTAR. The intraassay CV was 4.1% and interassay CV was 7.0%. Estradiol and testosterone were measured by radioimmunoassay (purchased from Diagnostic Products Corp., Los Angeles, CA). Osteocalcin was measured by radioimmunoassay, purchased from INCSTAR (Stillwater, MN). Plasma and urinary cAMP were measured by radioimmunoassay (DuPont NEN, North Billerica, MA). Nephrogenous cAMP was determined as total cAMP excreted in the urine (nmol/dl glomerular filtrate) minus the cAMP filtered from the plasma (nmol/dl glomerular filtrate). Serum and urinary calcium were measured by atomic absorption spectrophotometry (Perkin–Elmer 560). Renal tubular reabsorption of calcium (RTRCa) was calculated using the equation: RTRCa = 1 – [Fasting urine calcium (mg/dl) × serum creatine (mg/dl)/serum ultrafilterable calcium (mg/dl) × fasting urine creatinine (mg/dl)]. Serum ultrafilterable calcium was calculated based on the assumption that 60% of the total serum is ultrafilterable. Serum and urinary inorganic phosphate were measured colorimetrically [61]. Urinary hydroxyproline was measured by the method of Prockop and Udenfriend [62]. Serum and urinary creatinine was determined by the method of Heinegard and Tiderstrom [63]. Serum total alkaline phosphatase was measured spectrophometrically with P-nitrophenylphosphate as substrate. Serum bone alkaline phosphatase was measured by the method of precipitation with wheat-germ lectin [64]. The serum total and bone alkaline phosphatase of the same patients were measured in the same assay. The intraassay CV was 4.7% and the interassay CV was 6.5%. Plasma LH and FSH were measured by RIA manufactured by Serono (Norwell, MA).

Calcium Absorption

Calcium absorption efficiency was based on a single measurement of serum specific activity after administration of an oral calcium tracer by the method of Heaney and Recker [65]. Participants fasted overnight and then drank 170 ml of orange juice containing 5 microcuries of ^{45}Ca and 200 mg of calcium as $CaCl_2$. Five hours later, an aliquot of blood was drawn and the serum calcium and radioactivity were determined.

Bone Mineral Density

Each participant had a total body scan and individual scans of the proximal femur, radius, and lumbar spine (L2–L4), using a Lunar Radiation Densitometer (Lunar Radiation, Madison, WI, Model DPX-L, Software program 1.3Y). The scan was run at medium speed. In addition, bone mineral density of the radius was measured on a Hologic Instrument (Hologic, Waltham, MA). The CV of each site measured was 1–1.5%.

Data Analysis

For each skeletal site, we determined the relationship of bone mass to age for black and white women separately. Baseline comparisons between the races for continuous variables were analyzed with the Student's *t* test and discrete variables were analyzed using chi-square. All statistical tests were two-tailed. For variables that were not normally distributed, logarithmic transformations were used. The changes in bone density with age were determined by linear regression of the various measures against age and age squared. If the quadratic coefficient was statistically significant, the changes were calculated separately for pre- and postmenopausal women. The changes were expressed as percentage change per year by dividing the slopes of the various bone density measurements by the calculated bone density at age 20 for premenopausal and for all women, and by the calculated bone density at age 50 for postmenopausal women. Since the changes are all less than 1% per year, this approximation seemed reasonable. The percent of white and black women at various ages who were osteopenic was determined by using the regression equations to estimate the bone density at appropriate ages in each race. Osteopenia was defined as a bone density below 2.5 standard deviations of white women between the ages of 20 and 40 years [66]. Bone mineral apparent density (BMAD) was calculated as follows: BMAD total body = BMD/(area/height, m), BMAD femur = BMD/area, BMAD spine = $BMD^{3/2}/BMC^{1/2}$, BMAD radius = BMD/area [67]. All analyses were run using SAS (SAS Institute, Inc., Cary, NC).

Table 1. Clinical characteristics

	Mean SE		
	Black $n = 235$	White $n = 268$	P value
Age	43.7 (0.87)	51.4 (0.90)	0.0001
Age at menarche	12.3 (0.1)	12.6 (0.08)	0.01
Age at menopause	50.1 (0.42)	51.0 (0.26)	
Number of children	1.5 (0.19)	2.1 (0.11)	0.01
Months breast feeding	5.9 (0.86)	4.8 (0.44)	
Height (cm)	164 (0.4)	164 (0.4)	
Weight (kg)	70 (0.8)	64 (0.6)	0.0001
Alcohol intake (mg/day)	4 (0.88)	10.8 (1.0)	0.0001
Education (median)	2-year college	4-year college	
Family income (median)	$40,000–49,999	$50,000–79,999	0.0001
% who fell/year	20	30	
Cigarette packs/year	199 (21.5)	290 (25.0)	0.01
Activity score			
Pre	1.9 (0.07)	2.4 (0.04)	0.0001
Post	2.2 (0.08)	2.5 (0.04)	0.01

Results

Clinical Characteristics (Table 1)

Race was self-declared but each participant completed a family history questionnaire that asked for race back to grandparents. All family members were declared white among the white participants. Among the black participants 7 women had 1/6 ancestors declared as nonblack and 7 had 2/6 declared as nonblack. The black participants were younger and heavier than the white participants. The age at menopause was the same for each group and menarche was 3 months earlier in black participants. Physical activity scores suggested slightly greater activity in the white population and less smoking and drinking in the black population.

Bone Density Values

The rate of loss for bone mineral density for each skeletal site for both races are given in Table 2. The quadratic term in the age + age^2 model was statistically significant only for the total body bone mineral and for the radial sites. These regions were further tested for pre- and postmenopausal differences, which were statistically significant. The regressions for the various sites on the hip and the spine showed no improvement using any model other than linear regression. All slopes were significantly different than zero.

It may be seen in Table 2 that both black and white women had bone loss that was fit by a linear model from the femur and spine, but the rate of loss from these sites was slower in black women. The regressions were also run with height and weight as covariates, which did not change the statistical significance of black/white differences in slopes. The data are also expressed in %/year, which is another convention commonly used in expressing rates of bone loss. This calculation is affected by the higher initial bone density in the black women, but remained significantly different between black and white women for the femoral neck and trochanter. The loss rates for BMAD of the spine and femur were not significantly different in black and white women, but mean BMAD values were higher in the black women.

The sites that were best fit by a curvilinear model showed an increase in BMD (except for the ultradistal site) in black women and a decrease in BMD in white premenopausal women. It was not possible to determine precisely the age of peak bone mass, but examining mean values grouped every five years suggested that premenopausal bone loss from the total body and ⅓ radius started later in the black women. The postmenopausal BMD loss rates were slightly, but not significantly, greater in the white women. When expressed as % change, only the ⅓ radius site was significantly different (higher loss in white women) (Table 3). There were no significant differences in BMAD loss rates between black and white women for the total body or radius and mean BMAD was higher in black women for the radius but not for the total body.

The prevalence of predicted low Z-scores in the black population are provided for age 50 years, 65 years, and 80 years in Table 4. The scores were calculated from values for young (age 20–40 years) white women. It should be noted that at age 50 years, there are substantially more white women with a Z-score of −1 than for the black women. By age 80 years, there is a substantial number of black women who have sufficiently low bone density to be considered osteoporotic by the World Health Organization definition of greater than 2.5 SD below young adults [68]. As a result of cortical bone loss following menopause, most of the black population have osteoporotic values at the radius at age 80 years and 23% have low bone density of the femur.

Calcium Absorption

Calcium absorption efficiency is depicted in black and white premenopausal vs. postmenopausal women in Figure 1. Calcium absorption efficiency is lower in postmenopausal women. Although the absorption efficiency is similar in both races, because of their lower calcium intake, the amount of calcium actually absorbed is significantly different between black and white women. The value for calcium absorbed (Ca abs) is obtained as: Ca abs = % efficiency × dietary daily calcium intake (including calcium supplements).

Table 2. Rates of bone mineral density (BMD) loss for black and white women

Variable	Black		White		Black vs. white	
	Slope (SE) g/cm^2/ur	Slope (SE) %/yr	Slope (SE) g/cm^2/yr	Slope (SE) %/yr	P-value g/cm^2/year	P-value %/year
All women						
BMDNE	−0.0034 (0.00065)[a]	−0.32 (0.13)[a]	−0.0061 (0.00045)[a]	−0.59 (0.10)[a]	0.0001	0.02
BMDTR	−0.0012 (0.00063)[a]	−0.13 (0.15)[a]	−0.0033 (0.00045)[a]	−0.40 (0.13)[a]	0.0001	0.05
BMDWA	−0.0057 (0.00087)[a]	−0.53 (0.17)[a]	−0.0079 (0.00052)[a]	−0.79 (0.12)[a]	0.002	
BMDSP	−0.0037 (0.00077)[a]	−0.27 (0.12)[a]	−0.0057 (0.00062)[a]	−0.44 (0.12)[a]	0.004	
Premenopausal women						
TOTBMD	0.0008 (0.00077)	0.06 (0.06)	−0.0013 (−0.00072)	−0.11 (0.06)	0.01	0.01
BMD ⅓	0.0019 (0.00049)[a]	0.28 0.07)[a]	−0.0018 (0.00076)[b]	−0.24 (0.10)[b]	0.0001	0.01
BMDUD	−0.0002 (0.00050)	−0.04 (0.10)	−0.0003 (0.00041)	−0.07 (0.08)		
BMDMID	0.0011 (0.00044)[b]	0.18 (0.07)[b]	−0.0005 (0.00041)	−0.08 (0.07)		0.01
Postmenopausal women						
TOTBMD	−0.0037 (0.001144)[b]	−0.31 (0.08)[b]	−0.0050 (0.00095)[a]	−0.45 (0.08)[a]		
BMD ⅓	−0.0036 (0.00130)[c]	−0.52 (0.12)[c]	−0.0054 (0.00068)[a]	−0.82 (0.10)[a]	0.06	0.01
BMDUD	−0.0030 (0.00099)[c]	−0.64 (0.13)[c]	−0.0037 (0.00067)[a]	−0.84 (0.14)[a]		
BMDMID	−0.0044 (0.00080)[a]	−0.70 (0.08)[a]	−0.0049 (0.00062)[a]	−0.83 (0.10)[a]		

NE: Femoral neck, TR: trochanter, WA: Ward's triangle, SP: lumbar spine, BMD: bone mineral density, ⅓: ⅓ radial site, UD: ultra distral radius, MID: midradial sites, TOT: total; [a]P for slope difference from zero, [a]$P < 0.001$, [b]$P < 0.05$, [c]$P < 0.01$

Table 3. Biochemical studies

	Mean SE							
	Premenopausal				Postmenopausal			
	Black		White	P	Black		White	P
Serum Ca, mg/dl	9.2	(0.06)	9.1 (0.03)		9.5	(0.03)	9.4 (0.03)	
Serum P, mg/dl	3.5	(0.04)	3.5 (0.04)		3.7	(0.04)	3.8 (0.03)	0.01
24-hour urine Ca, mg/day	57.9	(3.47)	109.2 (5.28)	0.0001	67.6	(4.44)	98.4 (4.84)	0.0001
Fasting urine Ca, mg/dl	8.8	(0.46)	10.4 (0.55)	0.02	8.5	(0.60)	9.3 (0.44)	
Tubular reabsorption Ca, fractional	0.99	(0.001)	0.98 (0.001)	0.0001	0.99	(0.001)	0.986 (0.001)	0.0001
Tubular reabsorption P, fractional	0.91	(0.003)	0.89 (0.004)	0.004	0.92	(0.003)	0.89 (0.004)	0.0001
Fasting hydroxyproline, mg/dl	2.8	(0.12)	2.7 (0.11)		2.8	(0.14)	2.3 (0.08)	0.01
Fasting cyclic AMP, nmol/mg	6.7	(0.27)	5.9 (0.23)	0.04	5.5	(0.26)	4.2 (0.16)	0.0001
Serum osteocalcin, ng/ml	2.8	(0.10)	3.4 (0.09)	0.0001	3.5	(0.14)	4.1 (0.09)	0.0002
Skeletal alkaline phosphatase, iu/l	11.0	(0.26)	11.2 (0.27)		14.6	(0.52)	15.2 (0.36)	
PTH, pg/ml	37.0	(1.15)	35.0 (1.12)		42.2	(1.45)	36.7 (1.08)	0.002
Nephrogenous C-AMP, nmol/dl	1.8	(0.06)	1.9 (0.06)		2.0	(0.08)	1.9 (0.08)	
25(OH)D, ng/ml	11.8	(0.63)	24.7 (1.05)	0.0001	15.1	(1.02)	29.4 (1.28)	0.0001
1,25$(OH)_2$D, pg/ml	33.2	(0.69)	31.0 (0.63)	0.02	34.5	(0.90)	32.0 (0.66)	0.02
Estradiol, pg/ml	80.5	(4.53)	84.3 (5.54)		12.1	(1.74)	9.4 (1.65)	
Testosterone, pg/ml	296	(10.8)	274 (9.4)		285	(13.0)	236 (5.6)	0.001
Creatinine clearance	69.0	(2.27)	70.4 (1.66)		69.5	(3.15)	59.4 (1.51)	0.004
Ca Abs Eff, %	32.5	(0.67)	32.0 (0.67)		28.4	(0.81)	26.5 (0.54)	

Laboratory Values

Selected biochemical studies are shown in Table 3. It may be seen that serum calcium was similar in black and white women, but serum phosphorus is lower in postmenopausal blacks. Urinary calcium excretion was lower in black women. Tubular reabsorption of calcium was higher in both premenopausal and postmenopausal black women. Parathyroid hormone and calcitriol levels were higher in black women and serum calcidiol was lower (Fig. 2). The fasting

Table 4. Comparison of prevalence of black and white Z-scores in osteopenic and osteoporotic ranges (Z-score from white 20–40 years)

	$Z \leq -1$ Age 50 years		$Z \leq -2.5$ Age 65 years		$Z \leq -2.5$ Age 80 years	
	Black	White	Black	White	Black	White
TOTBMD	1	34	1	23	18	64
BMDSP	5	40	2	17	7	29
BMD ⅓	1	16	13	73	95	99
BMDNE	8	59	3	29	23	56
BMDTR	6	35	1	10	3	17
BMDWA	6	61	7	40	47	66
BMDUD	5	17	5	29	34	78
BMDMID	1	13	11	45	69	96

NE: Femoral neck, TR: trochanter, WA: Ward's triangle, SP: lumbar spine, BMD: bone mineral density, ⅓: ⅓ radial site, UD: ultra distral radius, MID: midradial sites, TOT: total

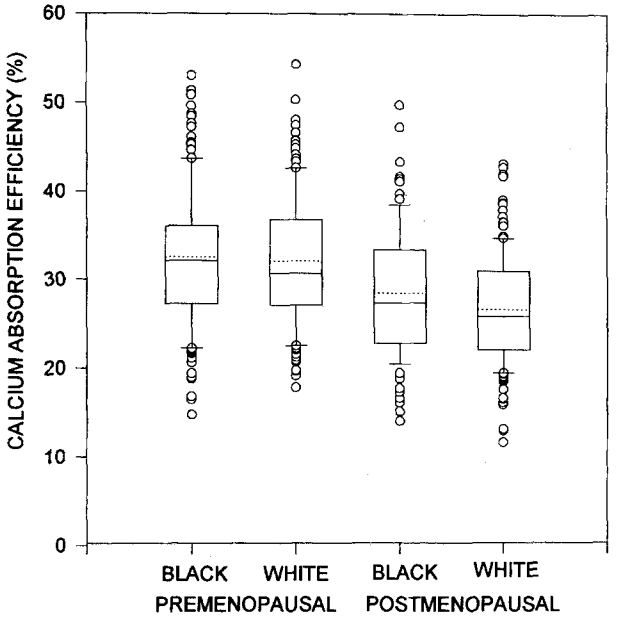

Fig. 1. Box plot of calcium absorption efficiency in black and white, pre- and postmenopausal women. The dashed lines in the plot represent the means, whereas the solid horizontal lines in the boxes represent the 25%, 50%, and 75% points of the cumulative distribution. The vertical lines extending from the boxes include from 10% to 90% of the cumulative distribution.

urine studies and the osteocalcin levels suggest lower bone resorption and bone formation in both pre- and postmenopausal black women. Serum testosterone was higher in the black women. This attained statistical significance in postmenopausal women and remained significant after adjustment for age. There were no differences in the routine clinical analyses, in thyroxine levels, or in estradiol levels.

Diet and Activity

Black women and white women recalled the following amounts of milk intake (ml, mean ± SE) age 6–12 years: 330 ± 23.7 vs. 570 ± 22.5; age 13–18: 285 ± 23.4 vs. 468 ± 21.9, respectively. The current intake of milk was 150 ± 21.6 vs. 267 ± 14.7. The white women also ate more yogurt and cottage cheese and increased their milk intake to a

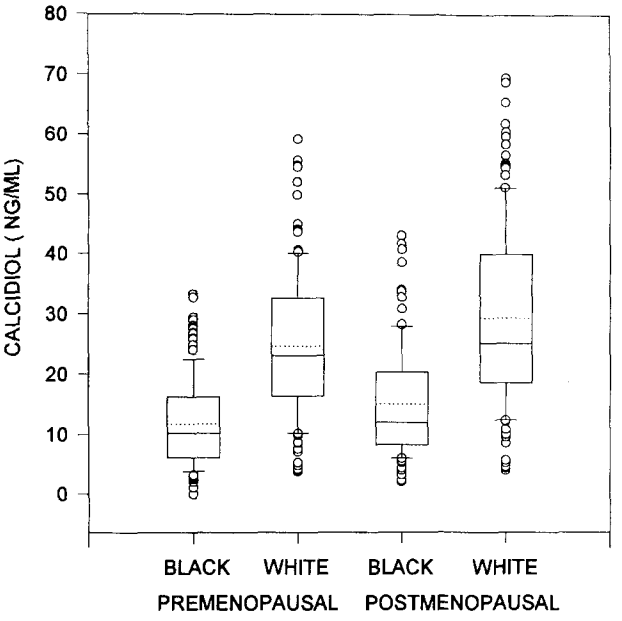

Fig. 2. Box plot of serum calcidiol in black and white, pre- and postmenopausal women. The dashed lines in the plot represent the means, whereas the solid horizontal lines in the boxes represent the 25%, 50%, and 75% points of the cumulative distribution. The vertical lines extending from the boxes include from 10% to 90% of the cumulative distribution.

greater extent during pregnancy and lactation. There was similar intake of greens, sardines, and bones. Dietary analyses are shown in Table 5.

When calcium supplements are included, average dietary calcium was 571 mg in black women as opposed to 736 mg in white women. (In postmenopausal women it was 511 mg vs. 713 mg, respectively). Eighty-one of the white women took calcium supplements as opposed to only 15 black women. Thus, the amount of calcium absorbed was less in black women. Moreover, a food frequency recall revealed that black women ingested 150 ml of milk per day as opposed to 270 ml for white women. Since the bioavailability of calcium from oxalate-containing vegetables may be less than from dairy products, there may be even less calcium absorbed in black women than estimated from dietary intake. Moreover, the ratio of protein to calcium has been considered a risk factor for bone loss and this ratio is higher in blacks than in whites. Activity scores were slightly lower in the black women.

Discussion

We examined a large population of healthy black and white women using dual X-ray absorptiometry, including the measurement of all the skeletal regions of interest. Our previous findings of the patterns of bone loss in white women were confirmed, suggesting that accelerated cortical bone loss occurs at menopause and that there is substantial premenopausal bone loss from the femur and spine, which are skeletal sites comprised predominantly of trabecular bone [5]. We have now shown that the same patterns of bone loss apply to both black and white women. Black women have a higher peak bone mass than white women and a slower rate of trabecular bone loss. Cortical bone loss after menopause is comparable in black and white women.

Carter et al. [67] have proposed that comparisons of bones of different sizes when using densitometry may be

Table 5. Daily dietary intake in black and white women

	Mean SE					
	Premenopausal			Postmenopausal		
	Black	White	*P*	Black	White	*P*
KCAL/WT (kg)	28.1 (0.9)	30.0 (0.7)		24.8 (1.0)	27.1 (0.7)	0.04
CHO/WT (g/kg)	3.4 (0.1)	3.8 (0.1)	0.02	3.1 (0.1)	3.5 (0.1)	0.02
Fat/WTKG (g)	1.1 (0.04)	1.1 (0.04)		0.9 (0.05)	0.9 (0.04)	
Protein/WT (g/kg)	1.2 (0.04)	1.3 (0.03)		1.1 (0.05)	1.2 (0.04)	
Protein/Ca (g/mg)	0.2 (0.006)	0.1 (0.004)	0.0001	0.2 (0.009)	0.1 (0.005)	0.0001
Calcium (mg)	606 (28)	761 (25)	0.0001	511 (25)	713 (28)	0.0001
Sodium (mg)	2502 (95)	2767 (84)	0.04	2136 (98)	2300 (104)	
Fiber (g)	12.5 (0.7)	13.7 (0.5)		15.8 (1.0)	16.9 (0.6)	
Vitamin D (μg)	2.2 (0.2)	3.0 (0.2)	0.002	2.3 (0.3)	3.1 (0.2)	0.02
Caffeine (mg)	118 (11)	230 (17)	0.0001	131 (16)	216 (14)	0.0001

WT: Body weight, CHO: carbohydrates

misleading because of inherent biases caused by bone-thickness differences. Using projected area to calculate volume, they introduced the concept of bone mineral apparent density (g/cm^3). This group, when studying black and white children, found that BMAD values were not different [67]. Our studies of whole body composition have demonstrated that, when expressed as % body weight, black women have a slightly larger musculoskeletal system than white women. However, Mazess et al. [69] have examined the use of BMC, BMD, and BMAD and have concluded that the best expression of data is with BMD. The BMD provided the best diagnostic sensitivity and the lowest precision error.

Fracture rates have been determined in longitudinal population studies in white women, establishing the use of bone mineral density measurements as predictors of fracture risk. Similar data are not available for the black population. However, because of the relationship of bone density to bone strength, it is reasonable, until prospective fracture data are available in the black population, to use the bone density in young normal white women and the SD from these values in assessing risk in black women. Such a calculation is presented in Table 4. It has been recommended that white women at menopause be considered for hormonal replacement therapy if their bone density values are 1 SD below the average for 20–40 year old women [70]. A standard deviation of less than 2.5 below the average of young adults has been selected by the World Health Organization as the densitometry definition of osteoporosis [68]. If one considers the values for bone density of the femur or spine to decide on the use of hormonal replacement therapy at menopause, roughly 8 times as many white women would be selected for this therapy, and only 5–8% of black women would be selected. Since the relative risk and benefit of prolonged hormonal therapy is unknown in black women, this finding is of importance.

As longevity increases in black women, there will be a higher number of elderly women who are osteopenic. Thus, as a result of menopausal and involutional bone loss, there are a substantial number of older black women with osteoporosis. The ratio of the percent of black to white osteoporotic women for the femoral neck is 1:7 at 50 years, but by age 80 years it is 1:2. From these values it is clear that it is important to prevent bone loss in black women even though they have a higher peak bone mass and a slower rate of trabecular bone loss.

Of course, factors in addition to bone mineral density may play a role in the risk for osteoporotic fractures. For instance, there is a difference between black and white women in the length of the femoral neck, which may be protective against hip fracture [71]. Moreover, there are black and white differences in body proportions.Body mass has emerged as a significant independent factor for hip fracture and black women have a higher body mass because of a larger musculoskeletal system and because of obesity.

In the hope of gaining insight that might lead to strategies for maximizing bone mass in black women, we studied indices of bone turnover, the calcitrophic hormones, and dietary intake. Bell et al. [72] reported that young adult blacks have a lower serum level of calcidiol, a higher calcitriol level, and lower urinary calcium excretion. They suggested that the lower calcidiol level was caused by less vitamin D3 from 7-dehydrocholesterol as a result of increased skin pigmentation. Increasing ultraviolet exposure in blacks results in equivalent calcidiol levels between blacks and whites; the lower levels of calcidiol in black women is a result of lower sunlight exposure in the northern latitudes.

Although there are some discrepancies in previous studies, the added evidence from our study establishes that black women, as compared with white women, have slightly lower indices of bone turnover before and following menopause. Lower bone turnover in black women has also been found on histomorphometry of bone biopsy samples [73]. The etiology of the lower bone turnover is unknown, may be genetic, and may occur primarily in trabecular bone since loss rates are different in the two races at skeletal sites that are comprised predominantly of trabecular bone. Statistically significant lower serum phosphorus and higher PTH levels were found only in the postmenopausal women in our study, but TRP was significantly higher in both premenopausal and postmenopausal black women.

The higher PTH levels with lower bone turnover has been interpreted to indicate relative skeletal resistance to PTH in black women. This is in conflict with studies using PTH infusion, which suggest similar skeletal and renal responses [74]. Our black population also had a higher tubular reabsorption of phosphorus, supporting increased PTH levels. It is possible that small differences in skeletal sensitivity to PTH are not demonstrable as an acute effect of PTH infusion. Interestingly, black participants in the PTH infusion studies also had increased baseline serum PTH levels and reduced calcidiol levels.

Deficiency of dietary calcium intake and low calcidiol levels could result in relative secondary hyperparathyroidism with compensatory increased calcitriol levels, reduced serum phosphate, and reduced urinary calcium excretion. Our data support this hypothesis in that calcidiol levels and dietary calcium were lower in black women.

Increasing calcium intake in black children up to 1600 mg per day does not suppress serum levels of calcitriol [75]. Since calcium intake has been found to be a major determinant of peak bone mass in white women, it appears likely that black girls might benefit to even a greater extent from an increased calcium intake since their calcium intake is lower. Lower calcium intake in the black population has been documented in the NHANES studies [76]. In latitudes where ultraviolet exposure is not maximal, it might also be necessary to administer vitamin D supplementation to ensure that calcitriol levels do not decline and because 250H vitamin D may directly influence bone mass accretion [75]. Prospective controlled studies of the influence of calcium and vitamin D on bone mass in each stage of the lifecycle of North American black women should be performed.

It should also be noted that the difference in calcium intake between the two races should account for only about 16 mg difference in urinary calcium excretion. The one percent difference in tubular reabsorption of calcium may at first appear clinically insignificant, but since approximately 10,000 mg of calcium is filtered daily, a 1% difference could explain the lower urinary calcium in blacks. The difference in renal tubular calcium reabsorption was not caused by dietary sodium since sodium intake was similar in both groups in our population. Whether the differences in TRCa represent a genetic difference or are a result of different PTH levels remains to be determined.

Our participants are not truly representative of African-Americans (nor of white women) in the entire U.S. population. They were recruited from a middle-class population and the recruitment methods did not provide a random population sample. Moreover, the race of participants was self-declared so that the black population did not have a 100% African ancestry. However, the higher socioeconomic status of our black women as compared with the general American population minimizes ethnic differences, making environmental factors, including diet, less influential. We present the clinical characteristics of our participants only for purposes of description of the population. Population-based fracture studies of black women are needed. A case-control study of hip fracture showed that thinness, previous stroke, use of walking aids, and alcohol intake were associated with increased risk and estrogen therapy was protective [77].

In addition to examining the role of increasing calcium intake (and vitamin D) as a strategic option for the prevention of osteoporosis in black women, the value of hormonal replacement therapy must be addressed. Unfortunately, there is little information available concerning the risks and benefits of replacement therapy in black women. The four largest trials studying cardiovascular benefits did not include black women, and the relative risk of hormonal therapy influencing breast cancer is unknown [78]. The concept has emerged that initiating hormonal replacement therapy in older women (rather than at menopause) may be a wise strategy, since older women continue to lose bone; epidemiologic studies suggest the protective effect of estrogen started in later life [79]. This strategy is even more germane to black women since they develop osteoporosis in significant numbers at a later age than white women. Starting hormonal therapy at age 65 years in black women could prevent osteopenia of the spine and hip and would minimize the length of hormone exposure. The risk of wrist fracture is not reduced sufficiently, but there is less disability from wrist fractures than from fractures of the spine or hip. The risks and benefits of hormonal replacement therapy in elderly black women remains to be determined.

In conclusion, black women have a larger whole body mineral mass than white women because of their higher peak bone mass and lower bone turnover. However, the pattern of bone loss is similar in black and white women. The benefit of increased dietary intake of calcium and vitamin D should be evaluated in black women. Whether provision of calcium with vitamin D will reverse the relative secondary hyperparathyroidism and will enhance bone density remains to be demonstrated. Finally, the risks and benefits of providing hormonal replacement therapy, particularly in the elderly, should be examined in black women.

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