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The Effects of Weight Loss on Relative Bone Mineral Density in Premenopausal Women

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Abstract

Heavier individuals have higher bone mineral density (BMD) than individuals of lower body weight, but it is unclear whether BMD changes in proportion to body weight during weight loss. This study compared BMD relative to body weight following a ~6 months weight loss program and a one year weight maintenance phase in premenopausal women and determined whether African American (AA) and European-American (EA) women's BMD respond similarly during weight loss. Premenopausal women (n=115, 34 ± 5 yrs.) were evaluated in an overweight state (BMI between 27–30 kg/m²), following an 800 kcal/day diet/exercise program designed to reduce BMI <25 kg/m², and one year following weight loss. Results indicated that BMD relative to body weight (Z-scores) increased after weight loss, but decreased during the one year weight maintenance phase. All one year follow up BMD Z-scores were increased (except L1) compared to baseline measurements (P < 0.05). These sites included the hip neck (+0.088, P=0.014), total hip (+0.099, P=0.001), L2 (+0.127, P=0.013), L3 (+0.135, P=0.014), and L4 (+0.199, P=0.002). AAs had significantly higher absolute BMD at all sites (P<0.05) compared to EAs, but no time by race interactions were evident during weight loss (except in L3). These results may indicate that weight loss is safe with regard to bone health for overweight premenopausal women.

Keywords

premenopausal women; bone; overweight; race

Introduction

Overweight individuals have a higher bone mineral density (BMD) than individuals of lesser body weight (1). Therefore, the general consensus has been that heavier individuals are more protected against bone fractures and osteoporosis than lighter individuals (2, 3, 4, 5, 6).

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Disclosure

The authors declare no conflict of interest.

Weight loss significantly decreases absolute BMD due to less loading on the bones, a decrease in parathyroid hormone causing renal calcium loss, and a decrease in extraovarial estrogen synthesis (7, 8). However, the decrease in absolute BMD due to weight loss may be countered by adaptive bone response to reduced stress on bones during normal activities. It is possible that risk of fractures may be reduced with weight loss if loss of bodyweight is proportionately more than loss of BMD. Supporting this, cross sectional studies have reported BMD to be proportional to lean mass rather than fat mass (9, 10, 11, 12). Therefore, if weight loss is primarily fat mass, it is likely that weight loss may improve BMD relative to body weight. Although absolute BMD decreases with weight loss, it is unclear whether BMD loss is proportional to weight loss.

When adjusting for body weight, lean mass is the major determinant of BMD rather than fat mass (9, 11, 12). Although cross-sectional studies have reported inverse relationships among fat mass and BMD, to date no longitudinal study has been performed to demonstrate this relationship among the same individuals. Beck et al. reported that femur BMD relative to body weight is lower in heavier postmenopausal women when compared to lighter individuals since lean mass is a smaller fraction in heavier individuals (9). Another cross-sectional study by Blum et al. comparing premenopausal women reported that for a given body weight, BMD of the total hip, lumbar spine, and total body were inversely related to percent body fat (10). A study by Janicka et al. on young men and women (13–21 yr) found that femur and spine BMD negatively correlated with fat mass once lean mass was adjusted for (11). In order to determine the efficacy for weight loss programs it is important to know what happens to relative bone density (relative to body weight) following weight loss.

When compared to European-Americans (EA), previous studies have attributed African Americans' (AA) higher BMD to higher peak bone mass, increased obesity rates, greater muscle mass, lower bone turnover rates, and longer periods of bone formation during adolescence (13). It is unclear whether the BMD response to weight loss is similar between AAs and EAs. A previous study comparing BMD between AAs and EAs reported that AA women have a 9% higher total hip BMD, a 15% higher femoral neck BMD, and a 64% lower risk of fracture when compared to EA women of similar ages (14). These findings have been reported by several other studies, suggesting a greater BMD among AA as compared to EA women (15, 13). However, to the authors' knowledge no study has evaluated the effects of weight loss on BMD between AA and EA women.

The purpose of this investigation was: 1) to compare BMD relative to body weight during and after weight loss in premenopausal women and 2) to determine whether AA and EA women's BMD respond similarly during weight loss and exercise training. We hypothesize that BMD values relative to body weight will improve as obese premenopausal women lose weight.

Methods and Procedures

Subjects

One hundred and fifteen overweight (mean BMI of 27–30 kg/m²) premenopausal AA and EA women, 21–46 yr old, participated in a weight loss program until a BMI <25 kg/m² was

achieved. Subjects then began a one year weight maintenance phase. All subjects were physically untrained, engaging in only relatively sedentary physical activities. Institutional review board-approved informed consent was obtained before participation in the study, in compliance with the Department of Health and Human Services regulations for the protection of human research subjects.

Baseline and Weight Loss Assessment

Subjects were evaluated in the overweight state (prior to any intervention). Weight was stabilized for four weeks through dietary control. All testing was conducted following the weight stabilization period, and in the follicular phase of the menstrual cycle. During the weight stabilization period body weights were measured three to five times per week at the General Clinical Research Center (GCRC) at the University of Alabama at Birmingham. A macronutrient-controlled diet was provided during the final two weeks of weight maintenance. The energy content was appropriately adjusted to ensure a stable body weight ($\pm 1\%$ variation from initial body weight). All diets consisted of approximately $\sim 22\%$ of energy from fat, 23% from protein, and 55% from carbohydrate.

Subjects were randomized to one of three intervention groups: diet only, diet + aerobic training, and diet + resistance training. After discharge from the initial GCRC inpatient visit, the GCRC kitchen provided all meals for the period of weight reduction. A 3350 kJ (800 kcal) diet was provided, which was designed to meet all nutrient requirements excluding energy requirements. Subjects were maintained on the diet and/or aerobic or resistance training until a BMI $< 25 \text{ kg/m}^2$ was achieved. Subjects who successfully lost $>10 \text{ kg}$ and reached a BMI $< 25 \text{ kg/m}^2$ were entered into the one year weight maintenance phase of the study.

One Year Weight Maintenance Phase

Once subjects achieved a BMI of $< 25 \text{ kg/m}^2$ they began the one year weight maintenance program. This involved attending biweekly group cognitive/behavioral intervention sessions throughout the year, given by a behavioral interventionist who was trained and experienced in behavioral weight loss. The emphasis was on making lifestyle changes in diet and physical activity which favor weight control. Sessions were 90 minutes and addressed diet records, food shopping, label reading, food preparation, eating out, holidays, self-control techniques, weak-moment self-analysis, emotional and behavioral support techniques, and lifestyle physical activity.

Aerobic training

Aerobic training entailed continuous walking/jogging on a treadmill, cycling using a cycle ergometer, or stair stepping commencing with a warm-up of 3 min and 3–5 min of stretching. Subjects selected their mode of exercise at each exercise session. During the first week of training, the subjects performed 20 min of continuous exercise at 67% maximum heart rate. Each week after the first week, duration and intensity increased so that by the beginning of the eighth week, subjects exercised continuously at 80% of maximum heart rate for 40 min. Subjects were encouraged to increase intensity (either speed or grade) when average exercise heart rate was consistently below 80% of maximum heart rate during both

the weight loss and one year weight maintenance phases. After the exercise session, subjects cooled down for 3–5 min with gradually decreasing exercise intensity.

Resistance training

After a warm-up on the treadmill or bike ergometer for 5 min and 3–5 min of stretching, subjects performed the following exercises: squats, leg extension, leg curl, elbow flexion, triceps extension, lateral pull-down, bench press, military press, lower back extension, and bent leg sit-ups. One set of 10 repetitions was performed during the first four weeks, after which 2 sets of 10 repetitions were performed for each exercise with 2 min rest between sets. The training was progressive with intensity based on 80% of the maximum weight that an individual lifted one time (1 RM). Strength was evaluated every three weeks, and adjustments in training resistance were made based on the most current 1 RM in both the weight loss and one year weight maintenance phases. In both the aerobic and resistance exercise groups, subjects were expected to train three days/week during the weight loss and two days/week during the one year weight maintenance phase.

Body Composition Measures

Total body composition, BMD, bone mineral content (BMC), and region areas of interest were determined by use of dual-energy X-ray absorptiometry (GE-Lunar Prodigy, Madison, WI, USA). BMD, BMC, and regional area measures included lumbar spine and the femoral bone. Subjects were positioned for the lumbar spine scan and then for the proximal femur scans, each scan lasting for about 30 seconds. The scans were analyzed using the GE-Lunar Prodigy enCORE 2002 software, version 6.10.029. Z score age, race and weight matched reference data from the GE Lunar Prodigy manual was used when comparing BMD relative to body weight. Z scores were calculated using more than 12,000 subjects from NHANES and Regional Lunar Reference data (16).

Section Modulus of the hip neck was determined by using previous calculations of Wang et al. (17). In the calculations, hip neck BMD and periosteal diameter were used to estimate resistance to bending (section modulus).

Statistics

Descriptive statistics were computed for each intervention group (diet only, diet + aerobic, and diet + resistance) at baseline, following weight loss, and one year following weight loss. All values are reported as means \pm SDs. A three (time) by three (group) by two (race) repeated measures ANOVA was performed on BMI, % body fat, body weight, and lean mass to calculate differences among exercise groups and AAs and EAs across the three time periods. Pearson's correlation analysis was used to examine associations between age and BMD measures at each time point.

A three (time) by three (group) by two (race) repeated measures ANOVA was performed on absolute BMD at the primary areas at risk for fracture: the hip neck, hip shaft, total hip, and the lumbar spine. An additional three (time) by three (group) repeated measures ANOVA was performed on BMD Z-scores. No significant differences were found for group or group by time interactions for absolute BMD or BMD Z-score, indicating that absolute BMD and

BMD Z-score changes were not significantly affected by training group. Therefore, all group data were pooled, and a time (repeated measures) by race ANOVA was performed on all variables of interest. Bonferroni corrected post hoc t tests were used to evaluate selected contrasts of interest. For all analyses, a *P* value <0.05 was deemed statistically significant.

To further analyze potential training group interactions, a time by group by race repeated measures ANOVA followed by Bonferroni corrected post hoc t tests were performed on absolute hip and spine BMC, regional areas of the hip and spine¹⁸ and hip neck section modulus.

Results

Because the age of the subjects could have been a potential confounder for changes in BMD during weight loss, a Pearson's correlation analysis was performed and indicated no relationship between age and BMD (hip: *P*=0.592, spine: *P*=0.809). Therefore, age was not considered in any further analysis. Descriptive statistics are displayed in Table 1. There was a significant time effect for all variables measured (*P*<0.001), but no significant race effect or time by race interaction. A significant time by group interaction (*P*=0.020) occurred for lean mass. Bonferroni corrected post hoc t tests showed significant losses in lean mass in both the diet only (after weight loss, *P*=0.009) and the diet + aerobic group (after weight loss, *P*<0.001; 1-year follow-up, *P*=0.004), but not for the diet + resistance group.

Absolute BMD and Bone Geometry

Hip—Significant main time effects from a repeated measures ANOVA existed for all hip BMD variables (hip neck *P*=0.007, hip shaft *P* = 0.023, and total hip *P*<0.001), such that absolute BMD decreased with weight loss (Table 2). No time by race interactions were found for any variable when comparing subjects at baseline, following weight loss, or following the one year weight maintenance phase, indicating that AA and EA women's hip BMD changed similarly with weight loss and regain. A race effect existed at all hip sites reflecting that AA women had higher BMD than EA women (hip neck *P*=0.030, hip shaft *P*=0.003, total hip *P*=0.028) (Table 2). Bonferroni corrected post hoc t tests indicated a significant decrease in hip neck and hip shaft BMD in AAs (*P*=0.023, *P*<0.001, respectively), and a significant decrease in total hip BMD in both AAs (*P*<0.001) and EAs (*P*=0.006) from baseline to 1-year following weight loss (Table 2).

Significant main time effects from a repeated measures ANOVA also existed in all hip BMC sites (hip neck *P*=0.012, hip shaft *P*<0.001, and total hip *P*=0.003). Time by race and time by exercise group interactions were not significant. Bonferroni corrected post hoc t tests indicated a significant decrease in hip shaft and total hip BMC at 1-year follow-up when compared to baseline in both AAs and EAs (*P*<0.05) (Table 3). For region areas at the total hip, a repeated measures ANOVA indicated a significant main effect across time (*P*=0.021). No other significant main effects were observed for the hip.

A repeated measures ANOVA indicated no significant difference in hip neck section modulus across time, nor were there time by race or time by group interactions (Figure 1).

Spine—A significant main time effect was observed for all BMD sites of the spine measured except for L4 (L1 $P < 0.001$, L2 $P < 0.001$, L3 $P = 0.008$) (Table 4). A time by race interaction was only observed in L3 ($P = 0.046$) (Table 4). A race effect existed at all spine sites ($P < 0.05$), reflecting that AA women had higher BMD than EA women. Bonferroni corrected post hoc t tests showed a significant decrease in BMD from baseline to weight loss in L1 among AAs ($P < 0.001$), L2 among AAs ($P = 0.002$) and EAs ($P = 0.023$), and L3 among EAs ($P = 0.048$). A significant decrease was also observed when comparing baseline measurements to one year following weight loss among EAs in L3 ($P = 0.007$).

Spine BMC significant main time effects from a repeated measures ANOVA occurred only in L1 ($P < 0.001$) and L3 ($P = 0.033$). No time by race or time by group interactions were significant. Bonferroni corrected post hoc t tests indicated a significant decrease in L1 BMC following weight loss when compared to baseline ($P < 0.001$ for AAs and $P = 0.044$ for EAs), and a significant increase in L3 in AAs at 1-year follow-up compared to baseline ($P = 0.018$) (Table 3). For region areas at the spine, L3 had a significant main effect ($P = 0.003$) with a Bonferroni corrected post hoc t test indicating the region area at L3 in EAs at one year follow-up was significantly higher than baseline ($P = 0.014$). No time by race or time by group interactions were significant for regions at the spine. Significant race effects did indicate that EAs had higher region areas at L1, L2, L3 compared to AAs ($P = 0.002$, $P = 0.002$, $P = 0.024$, respectively) (Table 3).

Body weight and relative BMD

In order to account for the potential confounding effect body weight has on bone density, a one way (time) repeated measures ANOVA was run on the Z-score corrected hip and spine variables. Race was not included in the analysis since Z-scores are standardized for race as well as weight. There was a significant main time effect for Z-scores at all hip sites (all $P < 0.001$). Bonferroni corrected post hoc t tests showed a significant BMD mean increase from baseline to immediately following the weight loss phase for all hip BMD sites ($P < 0.001$). In addition, all hip BMD sites were significantly higher than baseline at one year follow-up ($P < 0.05$) (Figure 2).

A significant time effect was observed for all spine sites (all $P < 0.015$). Bonferroni corrected post hoc t tests showed a significant increase between baseline and following weight loss for all spine sites ($P < 0.001$, except L1 which was $P < 0.05$). With the exception of L1, all spine bone density sites remained significantly increased at one year follow-up when compared to baseline ($P < 0.05$) (Figure 2).

Discussion

To the authors' knowledge, this is the first study to determine the effects of weight loss on absolute and relative (adjusted for weight) BMD in overweight AA and EA premenopausal women immediately after weight loss and one year following weight loss. Although AA women had higher bone densities than EA women, both groups changed bone density similarly across time. In addition, although the subjects tended to lose BMD during weight loss, they actually increased weight-adjusted bone density with weight loss, with the increase in weight-adjusted bone density persisting even one year following the weight loss.

Similar to previous studies (4, 18, 19, 20, 21, 22, 23) absolute hip and spine BMD decreased with weight loss. This was expected since there was a decrease of mechanical loading on the bones. However, contrary to previous studies, when comparing hip and spine BMD relative to body weight, BMD increased as body weight declined at all sites measured, suggesting that weight loss could decrease risks for fractures. Therefore, the results of this study suggest that it is safe and beneficial for overweight premenopausal women to lose weight since it improves BMD relative to body weight.

It is important to note that all BMD sites relative to body weight after the one year weight maintenance phase (with the exception of L1) were significantly higher than baseline measurements. This suggests that dieting and/or exercising for as little as six months has positive long-term effects on relative BMD. This conclusion should encourage overweight individuals who previously have not been on any type of exercise plan and who may be wary about starting any such plan. If overweight individuals learn that an exercise program as short as six months in duration will have lasting, positive effects on their BMD, this knowledge may encourage them to not only begin an exercise program, but perhaps continue participation in such a program. Future studies are needed to determine how much weight loss is needed to onset a positive BMD effect. Also, it would be beneficial to know at what point weight loss is no longer effective on relative BMD.

It is unclear why BMD Z-scores remained significantly higher than baseline scores after a one year follow-up. It is important to note that during the one year follow-up, subjects attended biweekly behavioral interventions which informed them about the importance of diet and exercise. It is possible that by attending these sessions, subjects improved their eating habits which may have had a positive effect on their BMD. For example, consuming more calcium and vitamin D has positive effects on BMD (24). It is also possible that subjects may have been more physically active during the weight maintenance phase as compared to pre-enrollment activity levels. This increased activity may explain the higher BMD values because of increased mechanical loading on the bones (25). Furthermore, previous studies have found BMD to be in proportion to lean mass rather than fat mass (10). Results from the present study showed that subjects had a lower percent body fat after the one year follow-up when compared to baseline measures. Therefore, since lean mass would be a higher fraction in subjects at one year follow-up when compared to their baseline measurements, it makes sense that BMD would be increased relative to body weight.

While absolute bone loss did occur in total hip and hip shaft as evidenced by a significant drop in BMC, it is notable that absolute bone loss did not occur in any of the other sites of interest. However, it is puzzling that our results indicated no time by exercise group interactions in bone geometry. Beck et al. found that effects of exercise are more evident in bone structural geometry than in BMD (26). Beck's finding supports the proposition that mechanical loading induced by exercise mainly affects the outer bone surface, thereby more likely influencing BMC, regional area, and section modulus (26–28). Although bone loss was not occurring among our subjects, we were unable to detect a significant positive bone geometry effect in our study. However, the geometry estimate is a crude one and mechanically meaningful changes can be small. Our results suggest that exercise had little effect on bone geometry during weight loss which raises the possibility that rapid weight

loss (12kg) may reduce exercise benefits. More research is warranted to determine the impact of weight loss and exercise on bone geometry.

Similar to previous findings, AA's BMD was significantly higher than EA's at all sites (14, 16). A review by Aloia explored why AAs have higher BMD values when compared to EAs. Aloia noted first that AAs have longer periods of bone formation during adolescence than EAs, and second that AA girls seem to absorb more calcium during adolescence than EA girls since calcium absorption and renal calcium conservation is greater in AAs (16). Aloia also suggested the increased BMD in AAs could result from a tendency of AAs having higher body weights than EAs, which would naturally cause a heightened BMD. In the present study we cannot assume AAs' higher BMD is caused by increased body weights as both AAs and EAs had similar body weights and BMIs. Also, total body lean mass in both races were not significantly different.

When comparing time by race effects of absolute BMD in this study, no significant difference existed between AAs and EAs (except for L3). Since a significant difference did exist between races when comparing changes of L3 across time ($P=0.046$) more research is needed to determine if AA and EA's BMD respond differently during weight loss. EAs' L3 BMD decreased over time (baseline mean = 1.345 ± 0.022 g/cm², after weight loss mean = 1.329 ± 0.022 g/cm², one year follow-up mean = 1.323 ± 0.022 g/cm²), whereas AAs L3 BMD decreased during the weight loss program, but then increased back to baseline levels during the one year weight maintenance phase (baseline mean = 1.425 ± 0.020 g/cm², after weight loss mean = 1.413 ± 0.020 g/cm², one year mean = 1.425 ± 0.020 g/cm²). Although L3 was the only site showing a significant race by time interaction, the mean trends of the other spine BMD sites were similar. This suggests that, unlike EAs, AAs may regain absolute BMD a year following weight loss despite similar body fat percentage gains.

It is interesting to observe that BMD sites were higher in AAs, whereas regional area sites of the spine were higher in EAs. Although AAs had higher BMDs, while EAs had higher region areas of the spine, neither group encountered bone loss since BMC remained relatively constant (29). If this finding is replicated in future studies, we will have reason to believe that neither race is at greater risk for fracture.

Strengths of the study were using a longitudinal design and controlling for diet and exercise. A limitation was the lack of information on the micro-architecture of bone in subjects, which could also play a role in predicting bone fractures (30).

In conclusion, weight loss induced by dieting, whether with or without exercise, increases BMD at the hip and spine relative to body weight. AAs have higher absolute BMD compared to EAs, and also may retain absolute BMD better than EAs during weight loss, but EAs have higher regional areas at the spine. More importantly, a weight loss program, as short as six months, has a significant long term effect on BMD Z-scores.

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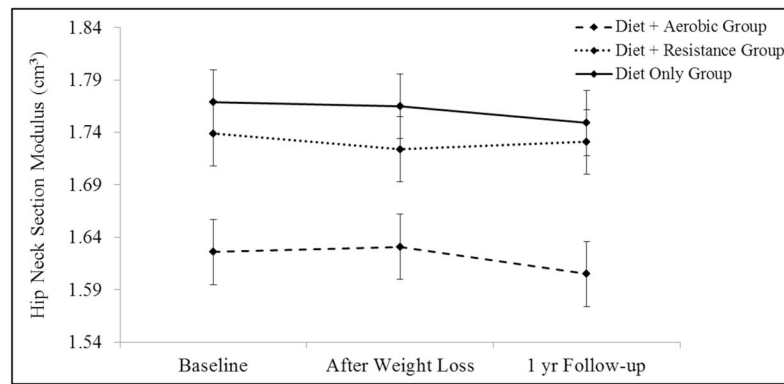


Figure 1. Group differences in hip neck resistance to bending (section modulus) across time
 A time by group by race repeated measures ANOVA was run followed by Bonferroni corrected post hoc t- tests to analyze hip neck section modulus. No significant difference in hip neck section modulus existed across time in any group or either race, nor were there significant time by group or time by race interactions. Values are means \pm standard error.

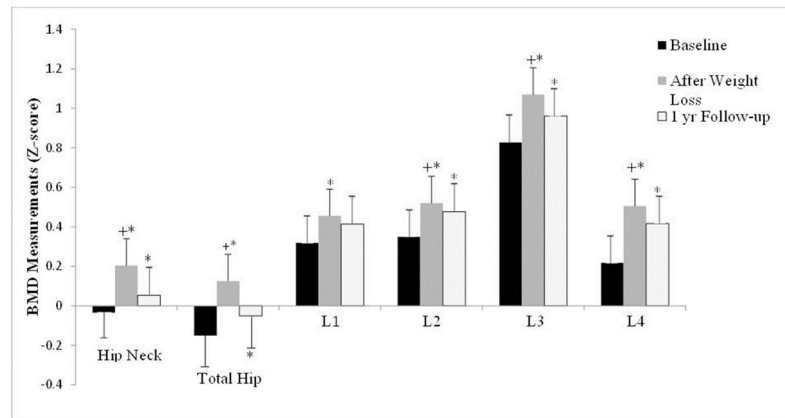


Figure 2. Changes in hip and spine BMD relative to body weight across time

A repeated one way ANOVA was run separately for each site. Main effect P values for each site were significant: hip neck ($P < 0.001$), total hip ($P < 0.001$), L1 ($P = 0.015$), L2 ($P < 0.001$), L3 ($P < 0.001$), and L4 ($P < 0.001$). Bonferroni corrected post hoc t tests indicated that BMD significantly increased after the weight loss program in all sites measured. All one year follow-up measurements, except L1, were significantly higher when compared to baseline measurements. +* indicates a significant difference of $P < 0.001$ from baseline. * indicates a significant difference of $P < 0.05$ from baseline.

Table 1

Descriptives. A 3(time) by 3 (group) by 2 (race) repeated measures ANOVA was run on BMI, % body fat, body weight, and lean mass to calculate differences among AAs and EAs across the three time periods. There was a significant time effect for all variables measured but no race effect or time by race interaction.

Time Point	Changes Across Time			P Value	
	Diet Only Group N=30	Diet + Aerobic Group N=37	Diet + Resistance Group N=48	Time	Race
Age					
Baseline	35.09±5.87	34.71±6.56	33.26±5.98	-----	0.725
After Weight Loss	-----	-----	-----		
1 yr Follow-up	-----	-----	-----		
Height (cm)					
Baseline	165.08±5.89	163.60±6.52	165.51±6.52	-----	0.402
After Weight Loss	-----	-----	-----		
1 yr Follow-up	-----	-----	-----		
BMI (kg/m²)					
Baseline	28.28±1.39	28.32±1.57	28.06±1.10	<0.001	0.127
After Weight Loss	23.97±0.85 ⁺ *	23.64±1.12 ⁺ *	23.93±0.97 ⁺ *		0.188
1 yr Follow-up	26.16±2.06 ⁺ *	25.46±2.31 ⁺ *	25.46±3.85 ⁺ *		0.357
% Body Fat					
Baseline	44.60±3.50	45.61±3.24	44.62±3.97	<0.001	0.209
After Weight Loss	35.17±4.96 ⁺ *	34.95±3.86 ⁺ *	33.72±5.02 ⁺ *		0.162
1 yr Follow-up	40.62±5.21 ⁺ *	39.87±6.15 ⁺ *	39.22±5.36 ⁺ *		0.140
Body Weight (kg)					
Baseline	78.49±6.44	75.60±5.80	77.46±7.86	<0.001	0.936
After Weight Loss	66.02±5.72 ⁺ *	63.16±5.42 ⁺ *	65.96±6.88 ⁺ *		0.165
1 yr Follow-up	72.46±7.52 ⁺ *	68.05±8.18 ⁺ *	71.36±7.62 ⁺ *		0.189
Lean Mass (kg)					
Baseline	46.33±3.74	43.79±4.05	46.49±5.55	<0.001	0.229
After Weight Loss	44.59±4.36 ⁺ *	42.22±3.98 ⁺ *	45.83±5.87		0.503
1 yr Follow-up	46.12±3.86	41.99±4.06 ⁺ *	45.58±5.37		0.020

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Values are means \pm standard deviation.

μ Time by race interaction.

β Time by group interaction.

+ * indicates a significant difference of $P < 0.001$ from the baseline phenotypes.

* indicates a significant difference of $P < 0.05$ from the baseline phenotypes.

Table 2

Absolute hip BMD changes across time

A repeated measures ANOVA was run separately on each hip BMD site followed by Bonferroni corrected post hoc t tests. AAs had a significantly higher BMD at all sites. Absolute BMD values decreased across time.

Time Point	Absolute Hip BMD Changes Across Time (g/cm ²)			P Value	
	AA ^a N=63	EA ^b N=52	Time	Race	TXRC ^c
Hip Neck			0.007	0.030	0.963
Baseline	1.089±0.148	1.034±0.115			
After Weight Loss	1.085±0.139	1.030±0.119			
1 yr Follow-up	1.076±0.146 *	1.023±0.121			
Hip Shaft			0.023	0.003	0.338
Baseline	1.316±0.158	1.235±0.133			
After Weight Loss	1.318±0.154	1.233±0.132			
1 yr Follow-up	1.305±0.159+*	1.194±0.224			
Total Hip			<0.001	0.028	0.438
Baseline	1.102±0.133	1.048±0.113			
After Weight Loss	1.105±0.130	1.047±0.112			
1 yr Follow-up	1.092±0.133+*	1.038±0.114*			

^a African Americans.

^b European-Americans.

^c Time by race interaction.

+* indicates a significant difference of P<0.001 from the baseline BMD measurement.

* indicates a significant difference of P<0.05 from the baseline BMD measurement.

Table 3
Absolute hip and spine BMC and region area changes across time

A time by group by race repeated measures ANOVA was run on BMC and region areas of the hip and spine followed by Bonferroni corrected post hoc t tests.

Time Point	BMC (g)		Regional Area (cm ²)	
	AA ^a	EA ^b	AA ^a	EA ^b
Hip Neck				
Baseline	5.151±0.805	4.900±0.674	4.730±0.285	4.758±0.341
After Weight Loss	5.195±0.837	4.884±0.668	4.802±0.597	4.758±0.319
1 yr Follow-up	5.094±0.777	4.859±0.686	4.733±0.281	4.762±0.359
Hip Shaft				
Baseline	18.048±2.261	17.216±2.018	13.726±0.645	13.993±0.737
After Weight Loss	18.128±2.183	17.163±1.890	13.762±0.651	13.971±0.672
1 yr Follow-up	17.929±2.217*	17.007±1.963*	13.755±0.624	13.936±0.708
Total Hip				
Baseline	35.491±5.248	31.961±4.316	30.340±2.164	30.593±1.792
After Weight Loss	33.279±4.737	31.863±4.292	30.090±1.937	30.545±1.797
1 yr Follow-up	33.079±5.126*	31.667±4.378*	30.238±2.004	30.607±1.844
L1				
Baseline	14.533±2.665	14.088±2.487	11.089±1.111	11.794±1.116
After Weight Loss	13.994±2.420+*	13.680±2.490*	10.986±1.166	11.680±1.103
1 yr Follow-up	14.398±2.709	13.742±2.738	11.094±1.280	11.712±1.205
L2				
Baseline	16.503±2.970	16.384±2.872	11.991±1.260	12.761±1.169
After Weight Loss	16.341±2.996	16.180±2.595	12.065±1.279	12.894±1.240
1 yr Follow-up	16.733±2.973	16.153±2.869	12.146±1.237	12.761±1.295
L3				
Baseline	19.144±3.506	18.707±3.508	13.432±1.481	13.794±1.630
After Weight Loss	19.160±3.195	18.804±3.257	13.557±1.383	14.192±1.732
1 yr Follow-up	19.527±3.328*	18.851±3.587	13.644±1.335	14.182±1.668*
L4				
Baseline	21.413±3.910	20.936±4.068	15.710±1.829	16.242±2.072
After Weight Loss	21.608±3.490	20.562±3.710	15.986±1.569	16.310±2.022
1 yr Follow-up	21.327±3.769	20.396±3.980	15.638±1.897	16.138±2.066

Values are means ± standard deviation.

^a African Americans.

^b European-Americans.

^c Race effect: European-American values are significantly higher (P<0.05) than African American values.

+* indicates a significant difference of P<0.001 from baseline.

* indicates a significant difference of P<0.05 from baseline.

Table 4

Absolute spine BMD changes across time

A repeated measures ANOVA was run separately on each spine BMD site followed by Bonferroni corrected post hoc t tests. AAs had a significantly higher BMD at all sites. No TXR effect existed except at L3.

Time Point	Absolute Spine BMD Changes Across Time (g/cm ²)			P Value	
	AA ^a N=63	EA ^b N=52	Race	Time	TXR ^c
L1					
Baseline	1.308±0.163	1.194±0.142		<0.001	0.235
After Weight Loss	1.275±0.150 ⁺ *	1.175±0.141			
1 yr Follow-up	1.294±0.158	1.175±0.150			
L2					
Baseline	1.378±0.169	1.278±0.152		<0.001	0.102
After Weight Loss	1.353±0.164*	1.256±0.137*			
1 yr Follow-up	1.374±0.163	1.258±0.146			
L3					
Baseline	1.425±0.166	1.348±0.148		0.008	0.046
After Weight Loss	1.413±0.169	1.329±0.137*			
1 yr Follow-up	1.425±0.167	1.323±0.148*			
L4					
Baseline	1.354±0.170	1.271±0.140		0.182	0.055
After Weight Loss	1.352±0.162	1.253±0.131			
1 yr Follow-up	1.365±0.163	1.255±0.146			

Values are means ± standard deviation.

^a African Americans.

^b European-Americans.

^c Time by race interaction.

⁺ * indicates a significant difference of P<0.001 from the baseline BMD measurement.

* indicates a significant difference of P<0.05 from the baseline BMD measurement.