Published in final edited form as:
Bone. 2012 February ; 50(2): 576–579. doi:10.1016/j.bone.2011.04.012.

Relationship between abdominal fat and bone mineral density in white and African American adults☆, ,☆☆

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Abstract

Several studies have documented relationships between adipose tissue and bone mineral density (BMD); however, the degree to which there are racial differences in this relationship is not known. The purpose of this study was to examine the relationships between abdominal visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) and BMD among white and African American adults. The sample included 330 white women, 328 African American women, 307 white men, and 116 African American men 18–74 years of age. Dual-energy X-ray absorptiometry scans were used to measure BMD and computed tomography scans were used to measure abdominal VAT and SAT. Linear regression was used to assess the relationships between abdominal adiposity and BMD and to explore possible sex and race differences in the associations. In the total sample as well as in all sex-by-race groups, VAT and SAT were negatively related to BMD, after adjustment for lean body mass (LBM) and several covariates. The VAT model (including covariates) explained 33.3% of the variance in BMD and the SAT model (including covariates) explained 32.7% of the variance in BMD. Being African American, being male, and having high LBM were all associated with higher BMD. Race and sex interactions were not significant, indicating that the relationships were similar across race and sex groups. In conclusion, BMD was inversely related to abdominal VAT and SAT in white and African American adults after adjustment for LBM.

Keywords

Obesity; Osteoporosis; Race/ethnicity; Gender; Adiposity

Introduction

Recent population studies have shown that African Americans have higher bone mineral density (BMD) and higher rates of obesity than white Americans [1,2]. Conversely, white adults tend to have higher levels of abdominal visceral adipose tissue (VAT) than African American adults [3]. Several studies have documented a negative relationship between VAT and BMD [4–7]; however, the degree to which racial differences in BMD can be explained by differences in total or depot-specific adiposity is not known. Thus, the purpose of this

☆The Pennington Center Longitudinal Study is registered at ClinicalTrials.gov (identifier NCT00959270).

☆☆This research was supported by the Pennington Biomedical Research Center. PK is supported, in part, by the Louisiana Public Facilities Authority Endowed Chair in Nutrition. This work was partially supported by an NORC Center grant #2P30-DK072476-06 entitled “Nutritional Programming: Environmental and Molecular Interactions” sponsored by NIDDK.

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study was to examine the relationship between abdominal VAT and subcutaneous adipose tissue (SAT) and BMD among white and African American men and women.

Methods

Sample

The Pennington Center Longitudinal Study (PCLS) is an ongoing investigation of obesity, lifestyle, and the development of chronic diseases. The PCLS sample is comprised of volunteers who have participated in a variety of clinical studies, including diet interventions, weight loss, and other metabolic/physiologic studies conducted at the Pennington Biomedical Research Center (PBRC) [3]. The present investigation is limited to cross-sectional analyses of baseline data for participants who underwent whole-body dual-energy X-ray absorptiometry (DXA) scans and computed tomography (CT) scans of the abdomen over the period 2001–2010. In cases where participants had multiple baseline measurements, the earliest complete record was used. All DXA and CT measurements were conducted within 60 days of the initial screening visit (DXA mean, 20.9 days; CT mean, 25.6 days). The total sample includes 1081 adults 18 to 74 years of age, including 330 white women, 328 African American women, 307 white men, and 116 African American men. All procedures employed by the PCLS were approved by the PBRC Institutional Review Board, and all participants provided written, informed consent.

Measures

Height was measured in duplicate using a wall-mounted stadiometer, and weight in duplicate, using a digital scale after the volunteer removed outer clothing, heavy pocket items, and shoes. The average of the two measurements was used in all analyses, and the body mass index (BMI) was calculated as weight in kilograms per height in square meters. Bone-free lean body mass (LBM) and total body BMD were estimated by DXA using a Hologic QDR 4500A whole-body scanner (Bedford, MA, USA). In cases where the subject was too large to capture both arms within the scanning area, the left arm was not scanned and the right arm values were duplicated and substituted for the left arm. The results of a reliability study on 88 subjects measured 14 days apart at PBRC demonstrated good test–retest reliability for both total body BMD (mean CV±SD, 0.75%±0.52%) and LBM (mean CV±SD, 1.2%±0.8%).

CT scans were performed with the participant lying in supine position with the arms over the head. A cross-sectional axial image was obtained at the level of the L4–L5 intervertebral space. The CT scans were performed at Baton Rouge General Medical Center, Baton Rouge, Louisiana, using a GE computed tomography scanner. Two different machines have been used over the course of data collection: GE LightSpeed Plus (2001–2007; n=976) and GE LightSpeed VCT (2007–2010; n=105). The CT scanner is calibrated to air (HU=0) daily. Image data were transferred to PBRC and commercially available software (Analyze; Analyze Direct, Rochester, MN, USA) was used to electronically measure areas of adipose tissue by selecting regions of interest defined by attenuation values (−30 to −190 HU for adipose tissue). Abdominal VAT and SAT cross-sectional areas (cm^2) were measured as previously described [3,8].

Age was computed from birth and observation dates. Self-reported smoking status was determined from questionnaire responses during screening, and participants were categorized as non-smokers, current smokers, or former smokers. Menopausal status (pre-menopausal/post-menopausal) was determined in women from their age and responses to questions regarding their reproductive history. Women aged 55+ years of age or those who

indicated that they can no longer have children because of achieving menopause were considered to be post-menopausal.

All continuous variables were normally distributed, with the exception of VAT, which was log-transformed prior to analysis. Associations among the independent variables were explored using Pearson correlations. Linear regression was used to assess the relationships between abdominal adiposity and BMD and to explore possible sex and race differences in the associations. Models included age, sex, and menopausal status (women only) as appropriate. Smoking status was initially included as a covariate; however, it was not a significant predictor in any models so it was dropped from subsequent analyses. Interaction effects were tested by including sex*VAT (or SAT), race*VAT (or SAT), and age*VAT (or SAT) terms in the models.

Results

Table 1 presents the descriptive statistics for the sample. The average age of the sample was 42.1 years (range, 18–74 years) and the average BMI was 31 kg/m^2 (range, 17–45 kg/m^2). Among the 4 sex-by-race groups, the correlations among the body composition variables ranged between 0.45 and 0.67 (all, $p<0.0001$) for SAT and VAT, between 0.37 and 0.51 (all, $p<0.0001$) for VAT and LBM, and between 0.47 and 0.67 (all, $p<0.0001$) for SAT and LBM. The results of the regression analyses for the prediction of BMD from VAT are presented in Table 2 and the results for SAT are presented in Table 3. In the total sample as well as in all sex-by-race groups, VAT was negatively related to BMD, after adjustment for several covariates (Model 1), with the exception of African American women ($p=0.06$). After further adjustment for SAT, VAT was no longer a significant predictor of BMD in white men ($p=0.18$). SAT was negatively associated with BMD in all groups except African American men ($p=0.11$). After further adjustment for VAT, SAT was no longer significantly associated with BMD in any group except for women ($p=0.04$).

In the total sample, the VAT model (including covariates) explained 33.3% of the variance in BMD (33.5% with the additional inclusion of SAT). Similarly, the SAT model (including covariates) explained 32.7% of the variance in BMD (33.5% with the additional inclusion of VAT). The variance explained in the 4 sex-by-race groups was lower, ranging from 13.9% to 21.0%. The variance in BMD explained by VAT (Table 2) ranged from 0.8% to 5.7%, which was reduced after the inclusion of SAT in the models (0.3% to 3.7%). The variance in BMD explained by SAT (Table 3) ranged from 0.9% to 2.0%, which was reduced after the inclusion of VAT in the models (<0.1% to 0.7%).

LBM, sex, and race were significant predictors of BMD, such that being African American ($\beta=0.04$; $p<0.0001$), male ($\beta=0.04$; $p=0.0008$), and having high LBM ($\beta=0.0064$; $p<0.0001$) were positively associated with BMD, taking into account all other variables in the fully adjusted model. The race-by-VAT (or SAT) and sex-by-VAT (or SAT) interaction terms were not significant, and their inclusion in the models did not change the results. The interaction between age and SAT was not significant ($p=0.98$); however, the interaction between age and VAT was significant ($p=0.009$). The analysis was repeated in 2 age groups (<45 years and ≥45 years). VAT was negatively associated with BMD in the younger age group ($\beta=-0.054$; $p<0.0001$) but not in the older age group ($\beta=-0.002$; $p=0.86$).

Discussion

In this study, BMD was inversely related to abdominal VAT and SAT in white and African American adults after adjustment for LBM. Total body fat mass has been found to be positively associated with BMD in a limited number of studies [9]. However, a negative

relationship between VAT and BMD has also been demonstrated in other studies [4–7,10,11]. Our results confirm these latter findings in a large biracial cohort and demonstrate that the negative relationship between VAT and BMD is similar in white and African American men and women. Although the results are statistically significant, the magnitude of the association is relatively small as 0.8% to 5.7% of the variation in BMD was explained by VAT (Table 2).

The negative relationship between SAT and BMD in this study contrasts with studies that found a positive relationship [4,12–16]. However, these studies did not control for total body mass or LBM. Similar to our analysis, an inverse relationship between SAT and BMD was reported in postmenopausal women with type 2 diabetes after adjustment for body mass [11]. Thus, SAT may have an independent negative association with BMD after accounting for differences in total fat or overall body size.

There are several mechanisms that may explain the observed associations. The sheer weight of excess adiposity creates a mechanical load that is associated with increased BMD, which may explain the positive association between total body fat and BMD in several studies [9]. However, the results of this study and others that have examined associations with depot-specific body fat indicate that the relationship between adiposity and BMD is more complicated. There is substantial evidence that both bone remodeling and the distribution of adipose tissue are regulated through the hypothalamus and sympathetic nervous system [17]. Adipose tissue-derived hormones, including adiponectin, leptin, insulin/amylin/preptin, and adipocytic estrogens, appear to influence BMD [9]. In addition, obesity and its co-morbidities have been associated with sterile inflammation and the infiltration of myeloid and lymphoid cells [18,19]. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance [20]. The systemic release of inflammatory cytokines, such as interleukin 6, by adipose depots could contribute to bone loss and reduced BMD [21]. Finally, there is substantial evidence that mesenchymal stromal/stem cells (MSC) exhibit an inverse relationship with respect to adipocytic and osteoblast commitment [22]. The same mechanisms regulating MSC differentiation locally within the marrow microenvironment may act systemically between peripheral adipose depots and trabecular and cortical bone under conditions of subcutaneous or visceral obesity.

The strengths and limitations of this study warrant discussion. A major strength is the large biracial sample which includes quantitative measurements of BMD as well as direct measurement of abdominal obesity from CT. Furthermore, the sample is not limited to a narrow age range or to single sex or race which increases the ability to extrapolate the results. Similar to other studies, African Americans in this sample have higher BMD and lower levels of VAT than the white subjects. However, the sample is not representative of the general population as it is composed of research volunteers. Although the relationship between VAT and BMD was consistent across most race-by-sex groups, further research is required to determine the generalizability of the results reported in this study. Unfortunately, data on physical activity, dietary intake, socioeconomic status, and hormone replacement therapy use were not available, so the influence of these variables on the observed relationships could not be explored, and the cross-sectional nature of the analysis limits inferences about causality. The inclusion of information on these covariates would allow for the refinement of the relationships presented here. Finally, site-specific BMD measurements were not available, so the analysis relied on total body BMD. The hypothesized mechanisms suggest that the relationship between adipose tissue and bone may be due to systemic factors; however, differences in mechanical loading across different sites may influence the observed relationships. Future studies should examine site-specific BMD measurements to examine if these relationships are region-dependent.

In conclusion, BMD was inversely related to abdominal VAT and SAT in African American and white men and women and the relationships did not differ by race or sex. Investigating potential biological and hormonal mechanisms by which adipose tissue influences BMD may provide insight into these observed clinical differences. Future research should examine how abdominal fat influences BMD in specific skeletal regions susceptible to fracture, such as femoral neck, lumbar spine, and hip. Clinical trials examining how change in abdominal fat alters BMD may reveal whether BMD loss can be attenuated with weight loss, especially in vulnerable populations including white adults and postmenopausal women. Exercise has been shown to reduce VAT in the absence of weight loss [23,24] and thus may be an important modality for improving outcomes in at-risk populations.

References

1. Looker AC, Melton LJ III, Harris TB, Borrud LG, Shepherd JA. Prevalence and trends in low femur bone density among older US adults: NHANES 2005–2006 compared with NHANES III. J Bone Miner Res. 2010; 25:64–71. [PubMed: 19580459]
2. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999–2008. JAMA. 2010; 303:235–41. [PubMed: 20071471]
3. Katzmarzyk PT, Bray GA, Greenway FL, Johnson WD, Newton RL Jr, Ravussin E, et al. Racial differences in abdominal depot-specific adiposity in white and African American adults. Am J Clin Nutr. 2010; 91:7–15. [PubMed: 19828714]
4. Gilsanz V, Chalfant J, Mo AO, Lee DC, Dorey FJ, Mittelman SD. Reciprocal relations of subcutaneous and visceral fat to bone structure and strength. Obstet Gynecol Surv. 2010; 65:3387–93.
5. Choi H, Kim K, Kim K, Hur N, Rhee Y, Han D, et al. Relationship between visceral adiposity and bone mineral density in Korean adults. Calcif Tissue Int. 2010; 87:218–25. [PubMed: 20631995]
6. Huang JS, Rietschel P, Hadigan CM, Rosenthal DI, Grinspoon S. Increased abdominal visceral fat is associated with reduced bone density in HIV-infected men with lipodystrophy. AIDS. 2001; 15:975–82. [PubMed: 11399979]
7. Bredella MA, Torriani M, Ghomi RH, Thomas BJ, Brick DJ, Gerweck AV, et al. Determinants of bone mineral density in obese premenopausal women. Bone. 2011; 48:748–54. [PubMed: 21195217]
8. Smith SR, Lovejoy JC, Greenway F, Ryan D, deJonge L, de la Bretonne J, et al. Contributions of total body fat, abdominal subcutaneous adipose tissue compartments, and visceral adipose tissue to the metabolic complications of obesity. Metabolism. 2001; 50:425–35. [PubMed: 11288037]
9. Reid IR. Relationships between fat and bone. Osteoporos Int. 2008; 19:595–606. [PubMed: 17965817]
10. Russell M, Mendes N, Miller KK, Rosen CJ, Lee H, Klibanski A, et al. Visceral fat is a negative predictor of bone density measures in obese adolescent girls. J Clin Endocrinol Metab. 2010; 95:1247–55. [PubMed: 20080853]
11. Yamaguchi T, Kanazawa I, Yamamoto M, Kurioka S, Yamauchi M, Yano S, et al. Associations between components of the metabolic syndrome versus bone mineral density and vertebral fractures in patients with type 2 diabetes. Bone. 2009; 45:174–9. [PubMed: 19446053]
12. Dolan SE, Carpenter S, Grinspoon S. Effects of weight, body composition, and testosterone on bone mineral density in HIV-infected women. J Acquir Immune Defic Syndr. 2007; 45:161–7. [PubMed: 17527091]
13. Yerges-Armstrong LM, Miljkovic I, Cauley JA, Sheu Y, Gordon CL, Wheeler VW, et al. Adipose tissue and volumetric bone mineral density of older Afro-Caribbean men. J Bone Miner Res. 2010; 25:2221–8. [PubMed: 20499353]
14. Kinjo M, Setoguchi S, Solomon DH. Bone mineral density in adults with the metabolic syndrome: analysis in a population-based U.S. sample. J Clin Endocrinol Metab. 2007; 92:4161–4. [PubMed: 17785365]

15. Tarquini B, Navari N, Perfetto F, Piluso A, Romano S, Tarquini R. Evidence for bone mass and body fat distribution relationship in postmenopausal obese women. Arch Gerontol Geriatr. 1997; 24:15–21. [PubMed: 15374132]

16. Wang MC, Bachrach LK, Van Loan M, Hudes M, Flegal KM, Crawford PB. The relative contributions of lean tissue mass and fat mass to bone density in young women. Bone. 2005; 37:474–81. [PubMed: 16040285]

17. Rosen CJ, Bouxsein ML. Mechanisms of disease: is osteoporosis the obesity of bone? Nat Clin Pract Rheumatol. 2006; 2:35–43. [PubMed: 16932650]

18. Weisberg SP, McCann D, Desai M, Rosenbaum M, Leibel RL, Ferrante AW Jr. Obesity is associated with macrophage accumulation in adipose tissue. J Clin Invest. 2003; 112:1796–808. [PubMed: 14679176]

19. Yang H, Youm YH, Vandanmagsar B, Ravussin A, Gimble JM, Greenway F, et al. Obesity increases the production of proinflammatory mediators from adipose tissue T cells and compromises TCR repertoire diversity: implications for systemic inflammation and insulin resistance. J Immunol. 2010; 185:1836–45. [PubMed: 20581149]

20. Xu H, Barnes GT, Yang Q, Tan G, Yang D, Chou CJ, et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. J Clin Invest. 2003; 112:1821–30. [PubMed: 14679177]

21. Kern PA, Ranganathan S, Li C, Wood L, Ranganathan G. Adipose tissue tumor necrosis factor and interleukin-6 expression in human obesity and insulin resistance. Am J Physiol Endocrinol Metab. 2001; 280:E745–51. [PubMed: 11287357]

22. Gimble JM, Zvonic S, Floyd ZE, Kassem M, Nuttall ME. Playing with bone and fat. J Cell Biochem. 2006; 98:251–66. [PubMed: 16479589]

23. Ross R, Dagnone D, Jones PJ, Smith H, Paddags A, Hudson R, et al. Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men. A randomized, controlled trial. Ann Intern Med. 2000; 133:92–103. [PubMed: 10896648]

24. Ross R, Janssen I, Dawson J, Kungl AM, Kuk JL, Wong SL, et al. Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial. Obes Res. 2004; 12:789–98. [PubMed: 15166299]

Table 1

Descriptive characteristics of 1081 white and African American adults from the Pennington Center Longitudinal Study. Results are presented as means (SD).

	White women	African American women	White men	African American men
N	330	328	307	116
Age (y)	46.6 (13.0)	38.9 (11.9)*	43.0 (14.4)**	36.2 (13.9)*,**
Body mass index (kg/m^2)	31.5 (4.7)	30.9 (5.3)	31.2 (5.1)	29.3 (4.8)*,**
Fat mass (kg)	34.5 (8.5)	32.0 (9.6)*	28.3 (10.1)**	21.4 (9.5)*,**
Lean body mass (kg)	47.2 (6.0)	48.4 (6.9)*	67.2 (8.3)**	68.2 (8.8)**
Visceral adipose tissue (cm^2)	144.7 (71.7)	93.3 (52.4)*	160.3 (81.5)**	94.6 (63.0)*
Subcutaneous adipose tissue (cm^2)	463.6 (127.1)	448.9 (146.9)	351.3 (145.5)**	283.8 (154.1)*,**
Bone mineral density (g/cm^2)	1.083 (0.093)	1.136 (0.097)*	1.169 (0.099)**	1.240 (0.098)*,**

* Significant race difference, within sex (p<0.05).

** Significant sex difference, within race (p<0.05).

Table 2

Results of regression analyses for the prediction of total body bone mineral density from abdominal visceral adipose tissue (VAT[a]) in 1081 white and African American adults from the Pennington Center Longitudinal Study.

	Model 1[b]					Model 2[c]				
	Model	VAT				Model	VAT			
	R^2 (%)	β	SE	R^2 (%)[d]	*p*-Value	R^2 (%)	β	SE	R^2 (%)[d]	*p*-Value
Total sample	33.3	−0.034	0.007	1.6	<0.0001	33.5	−0.027	0.008	1.6	0.0003
Women	25.4	−0.035	0.009	1.6	0.0002	25.9	−0.027	0.010	0.8	0.008
Men	23.3	−0.039	0.010	2.6	0.0002	23.4	−0.033	0.011	1.4	0.005
White	30.0	−0.032	0.009	1.3	0.0006	30.3	−0.025	0.010	0.6	0.02
Women	19.4	−0.050	0.014	3.1	0.0005	20.1	−0.041	0.015	1.9	0.006
Men	15.2	−0.029	0.013	1.4	0.03	15.6	−0.020	0.015	0.5	0.18
African American	33.4	−0.032	0.010	1.6	0.001	33.5	−0.027	0.012	0.8	0.02
Women	20.5	−0.024	0.013	0.8	0.06	21.0	−0.015	0.014	0.3	0.30
Men	17.6	−0.048	0.017	5.7	0.006	17.6	−0.047	0.021	3.7	0.03

[a] VAT has been log transformed prior to analysis.

[b] Model 1 includes age and lean body mass as covariates. The model for the total sample also includes sex and race as covariates, and the race-specific models include sex, and sex-specific models include race as covariates. Models in women also include menopausal status as a covariate.

[c] Model 2 includes SAT in addition to covariates from Model 1.

[d] Partial R^2 attributable to VAT.

Table 3

Results of regression analyses for the prediction of total body bone mineral density from abdominal subcutaneous adipose tissue (SAT) in 1081 white and African American adults from the Pennington Center Longitudinal Study.

	Model 1[a]					Model 2[b]				
	Model	SAT				Model	SAT			
	R^2 (%)	β	SE	R^2 (%)[c]	*p*-Value	R^2 (%)	β	SE	R^2 (%)[c]	*p*-Value
Total sample	32.7	−0.000093	0.000023	1.0	<0.0001	33.5	−0.000050	0.000026	0.2	0.05
Women	25.1	−0.000102	0.000030	1.3	0.0008	25.9	−0.000068	0.000032	0.5	0.04
Men	22.0	−0.000098	0.000036	1.4	0.007	23.4	−0.000037	0.000042	0.1	0.38
White	29.7	−0.000090	0.000029	1.1	0.002	30.3	−0.000058	0.000032	0.4	0.08
Women	18.2	−0.000115	0.000042	1.9	0.006	20.1	−0.000074	0.000044	0.7	0.09
Men	15.1	−0.000086	0.000042	1.2	0.04	15.6	−0.000054	0.000048	0.4	0.26
African American	32.7	−0.000092	0.000038	0.9	0.02	33.5	−0.000042	0.000043	0.1	0.33
Women	20.7	−0.000092	0.000044	1.1	0.04	21.0	−0.000068	0.000050	0.5	0.17
Men	13.9	−0.000121	0.000075	2.0	0.11	17.6	−0.000003	0.000091	<0.1	0.97

[a] Model 1 includes age and lean body mass as covariates. The model for the total sample also includes sex and race as covariates, and the race-specific models include sex, and sex-specific models include race as covariates. Models in women also include menopausal status as a covariate.

[b] Model 2 includes log-transformed VAT in addition to covariates from Model 1.

[c] Partial R^2 attributable to SAT.