Race-Dependent Health Risks of Upper Body Obesity

HILLARY J. DOWLING AND F. XAVIER PI-SUNYER

For Caucasian women, an excess of abdominal fat is a potent risk factor for the development of diabetes and cardiovascular disease. However, there is limited information regarding the health risks of upper body obesity for African-American women despite a higher prevalence of obesity and obesity-related diseases and a reportedly higher prevalence of abdominal fat accumulation. This study aimed to determine whether UBO, independent of total body fatness, is as potent a diabetic and CVD risk factor for black women as has been confirmed for white women. Diabetes and CVD risks and androgenic status were assessed in nondiabetic, premenopausal women of similar body fatness who differed by race (black or white) and body fat distribution (UBO or lower body obesity). In black women, high-density lipoprotein cholesterol was the only measurement adversely affected by abdominal fat; HDL cholesterol was significantly lower in the black UBO group (1.14 ± 0.05 mM) compared with the black LBO group (1.37 ± 0.08 mM). This contrasts markedly with our findings in white women. In confirmation of previous reports, white UBO women, compared with white LBO counterparts, had significantly higher glucose (967.6 vs. 709.2 mM/2 h) and insulin (120.5 vs. 52.1 pM/2 h) areas and significantly lower peripheral insulin sensitivities (0.99 vs. 2.95 × 10^{-4} $min^{-1}/\mu U/ml$). In addition, HDL cholesterol levels were significantly lower in the white UBO group (1.03 mM) compared with the white LBO group (1.49 mM), whereas plasma TG levels (white UBO, 1.72 vs. white LBO, 0.88 mM) and dBPs (white UBO, 84 vs. white LBO, 75 mmHg) were significantly higher. Finally, in the white UBO group compared with the white LBO group, SHBG levels were significantly lower (27.5 vs. 51.6 nM), whereas the free testosterone levels were higher (2.21 vs. 1.49%). We conclude that an equivalent degree of UBO, independent of total body fatness, is less detrimental for black women than for white women with regard to the risks of developing diabetes and CVD. *Diabetes* 42:537–43, 1993

Prospective, population-based studies (1,2) have confirmed that an excess of abdominal fat is a potent risk factor for the eventual development of obesity-related illnesses such as diabetes and CVD in Caucasian men (2) and women (3,4). This causal relationship between abdominal fat and disease has been examined extensively in Caucasians (5–9) and in several ethnic groups including Mexican Americans (10–12) and Japanese Americans (13). However, the importance of excessive abdominal fat as a risk factor among African Americans has not been thoroughly investigated (14,15), even though this ethnic group has the highest incidence of obesity and obesity-related diseases and may have a greater incidence of abdominal fat accumulation. Several national nutrition surveys in the United States have documented that the prevalence of obesity is greatest among African-American women, even after correcting for differences in socioeconomic status (16,17). In addition, anthropometric (suprailiac-to-triceps skinfold ratio) data from the Pittsburgh Healthy Women Study (18), a prospective longitudinal study from 1983 to 1985, suggest that black women, once they become obese, may have a more central or abdominal

From the Obesity Research Center, Department of Medicine, St. Luke's-Roosevelt Hospital Center, Columbia University College of Physicians and Surgeons, New York, New York.

Address correspondence and reprint requests to Dr. F. Xavier Pi-Sunyer, Director, Obesity Research Center, St. Luke's-Roosevelt Hospital Center, Amsterdam Avenue at 114th Street, New York, NY 10025.

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UBO, upper body obesity; LBO, lower body obesity; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BP, blood pressure; dBP, diastolic blood pressure; sBP, systolic blood pressure; BMI, body mass index; TG, triglycerides; OGTT, oral glucose tolerance test; IGTT, intravenous glucose tolerance test; WHR, waist-to-hip ratio; CV, coefficient of variation; FFM, fat-free mass; D_{ffm}, densities of fat-free mass; FSIGT, frequently sampled intravenous glucose tolerance test; S_I, insulin sensitivity; PCOS, polycystic ovary syndrome; LH, luteinizing hormone; FSH, follicle-stimulating hormone; SHBG, sex hormone-binding globulin; ANOVA, analysis of variance; LSD, least significant difference; DHEA-SO_4, dehydroepiandrosterone sulfate, NIDDM, non-insulin-dependent diabetes mellitus.

body fat distribution compared with white women of similar BMI. These preliminary data support similar anthropometric findings reported in the first Health and Nutrition Examination Survey (17,19). In addition, Stevens et al. (20) have recently reported on changes in body weight and girths, over 25 years, in black and white adults. In women whose BMI remained unchanged during this interval, abdominal girths increased significantly more in black women (6.6 cm) than white women (2.8 cm). Given that black women face the greatest risk of becoming obese and, once obese, may deposit fat preferentially in the abdominal region, this study investigated whether UBO is as potent a risk factor for diabetes and CVD in black women as has been documented in white women. Diabetic and CVD risks, as well as androgenic status, were assessed in four groups of nondiabetic, premenopausal women who differed on the basis of race and body fat distribution. The women were carefully matched for age, menopausal status, degree of obesity, and fat patterning. This study's objective was to determine whether black UBO women are similar to their white counterparts, exhibiting glucose intolerance, hyperinsulinemia, peripheral insulin resistance, altered plasma lipids, elevated BP, and increased androgenic status compared with black LBO women.

Diabetes risk was assessed by levels of plasma glucose and insulin after an oral glucose challenge and by in vivo measurement of peripheral insulin sensitivity. CVD risk was assessed by levels of plasma TG, total cholesterol, HDL and LDL cholesterol, and sBP and dBP. In addition, androgenic status was determined by levels of plasma SHBG, total and free testosterone, and DHEA-SO_4 in blood samples obtained during the early follicular phase of the menstrual cycle.

RESEARCH DESIGN AND METHODS

We selected 42 women for study on the basis of their race, age, menopausal history, BMI (kg/m^2), percentage body fat, and body fat distribution. All subjects were in good health as determined by physical examination, medical history, and clinical screening tests that included an OGTT. Only those subjects with normal glucose tolerance according to the National Diabetes Data Group guidelines (21) were accepted. All subjects had to report stable body weights (mean ± 2.25 kg) for 6 mo. All but one were nonsmokers. The study was approved by the Internal Review Board of St. Luke's-Roosevelt Hospital Center, and all subjects gave their written consent.

Race. Potential subjects were premenopausal, nondiabetic, Caucasian (white), or African-American (black) women between the ages of 18 and 45 who were 130–150% of their ideal body weight (22). The extent of racial admixture was assessed by extensive personal interview. White women were eligible if their parents, grandparents, and great-grandparents were reported to be Caucasian; black women were eligible if their parents, grandparents, and great-grandparents were reported to be of African ancestry.

Anthropometric measurements. Body fat distribution was assessed by the minimum waist-to-maximum hip circumference ratio. The following circumferences were measured by the same trained observer according to the method of Ashwell et al. (23): *1*) midwaist, midway between the lower rib margin and the iliac crest; *2*) waist, umbilicus; *3*) minimum waist, minimum circumference between the lower rib margin and the iliac crest; and *4*) maximum hip circumference below the iliac crest. The CV for the circumference measurements was <1%. Women with a WHR of ≤0.76 were assigned to either a white or black LBO group, and those with a WHR of ≥0.85 were assigned to either a white or black UBO group. In this way, only those women with extremes of fat distribution were included, whereas those with intermediate distributions (0.76 ≤ WHR ≤ 0.85) were excluded.

TABLE 1
Clinical characteristics of study subjects

	Black women		White women	
	UBO	LBO	UBO	LBO
n	11	11	10	10
WHR	0.89 ± 0.01	0.72 ± 0.01	0.88 ± 0.01	0.71 ± 0.01
Age (yr)	36 ± 2	37 ± 2	40 ± 1	38 ± 2
BMI (kg/m^2)	33 ± 1	34 ± 1	35 ± 1	32 ± 1
Body fat (%)	44 ± 1	44 ± 1	43 ± 1	44 ± 1
FFM (%)	56 ± 1	56 ± 1	57 ± 1	56 ± 1

Data are means ± SE.

Body composition. Body fat content and FFM were determined by hydrodensitometry on subjects in the fasted state (CV for percentage of body fat by hydrodensitometry was <2%). In calculating percentage of body fat with the Siri equation, different D_{ffm} were used for the black (1.105 g/cm^3) and white (1.100 g/cm^3) women according to a recent report by Ortiz et al. (24). These investigators quantified the D_{ffm} by using a four-compartment model of body composition, and found it to be greater in black than white women primarily because of heavier bone mass.

All four groups (black and white UBO and LBO) were well matched for age, BMI, percentage of body fat, and percentage of FFM (Table 1).

OGTT. An OGTT was administered to subjects after an overnight, 12-h fast. Plasma glucose (CV <4%) (glucose analyzer, Beckman, Fullerton, CA) and insulin (25) (CV <12%) were measured in blood samples taken at time 0 and at 30, 60, 90, and 120 min after glucose ingestion (75 g of Koladex, Custom, Baltimore, MD). The integral glucose and insulin areas under the 2-h OGTT curves were estimated according to the following algorithm (derived by Mary Tai, Ed.D., Department of Nutrition, New York University). Area = $1/2(x_{30}y_0 + x_{30}y_{30}) + [1/2(x_{60} - x_{30})(y_{30} + y_{60}) + (x_{90} - x_{60})(y_{60} + y_{90}) + (x_{120} - x_{90})(y_{90} + y_{120})]$ where x is time of blood sampling in minutes and y is plasma glucose or insulin concentrations at the time noted.

FSIGT. Peripheral insulin sensitivity was measured in vivo according to the tolbutamide-modified FSIGT of Bergman (26). Subjects arrived at the Clinical Research Center at 0800 after an overnight fast. A venous catheter was placed in one arm for the injection of glucose (0.3

g/kg body wt; 50% dextrose injection) (Abbott, North Chicago, IL), administered at time 0, which was followed with the injection of tolbutamide (Orinase Diagnostic, Upjohn, Kalamazoo, MI) at 20 min. Subjects with a BMI <30 kg/m^2 received 300 mg of tolbutamide, whereas those with a BMI >30 kg/m^2 received 500 mg. A second venous catheter was placed in the other arm for frequent blood sampling (taken before glucose injection at −20, −15, −10, and −5 min, and postglucose injection at 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 24, 25, 27, 30, 40, 60, 70, 90, 100, 120, 140, 160, and 180 min). The catheters were kept patent with the slow infusion of saline. Plasma glucose and insulin were measured on all samples. The S_I index was calculated from the plasma insulin and glucose levels with the nonlinear mathematical model of glucose disappearance (MINMOD program, copyright R. N. Bergman, 1986). S_I calculations were performed by Drs. R. M. Watanabe and R. N. Bergman (Department of Physiology and Biophysics, University of Southern California School of Medicine, Los Angeles, CA).

Plasma sex hormones. To exclude subjects with PCOS and to determine whether the androgenicity previously observed in white UBO women is also evident in black UBO women, blood samples were obtained from subjects during the early follicular phase of the menstrual cycle (between days 4 and 7). Five blood samples were taken at 30-min intervals for 2 h and then pooled to control for the pulsatile manner in which hormones are secreted into the circulation. Serum LH and FSH were measured with immunoradiometric assay kits (Coat-a-Count FSH and LH IRMA, Diagnostic Products, Los Angeles, CA). Only those subjects with LH-to-FSH ratios of <2, an index of normal ovarian function, were accepted. In addition, SHBG (27,28), total testosterone (Diagnostic Products), free testosterone (29), and DHEA-SO_4 (Diagnostic Products) were measured by radioimmunoassay. The CV for these hormonal assays were ≤12%.

Fasting plasma lipids. Blood samples were taken from subjects after an overnight fast for the measurement of total and HDL cholesterol (assay kits 225 and 215–13, Diagnostic Chemicals, Monroe, CT) and TG (assay kit 210). LDL cholesterol was calculated as follows: LDL = total cholesterol − [HDL + (TG/5)]. The CVs were <1% for total cholesterol, <2% for HDL cholesterol, and <3% for TG.

BP. A total of 3 BP measurements were taken by the same trained observer using a sphygmomanometer after subjects had been seated comfortably for at least 30 min. The sBP was taken at the first Korotkoff sound and the dBP at the cessation of Korotkoff sound.

Statistical analysis. All statistical analyses were done with SAS (Carey, NC) on an IBM personal computer. Two-way ANOVA were used to examine the effects of race or body fat distribution or both on glucose tolerance, peripheral insulin sensitivity, plasma lipids, BP, and sex hormone levels. When a significant main effect of race or body fat distribution or both was observed, or when a significant race by body fat distribution interaction was observed, the LSD test was used for post hoc analysis (30). The LSD test identified which pair of group means were significantly different at $P < 0.05$. Data are means ± SE.

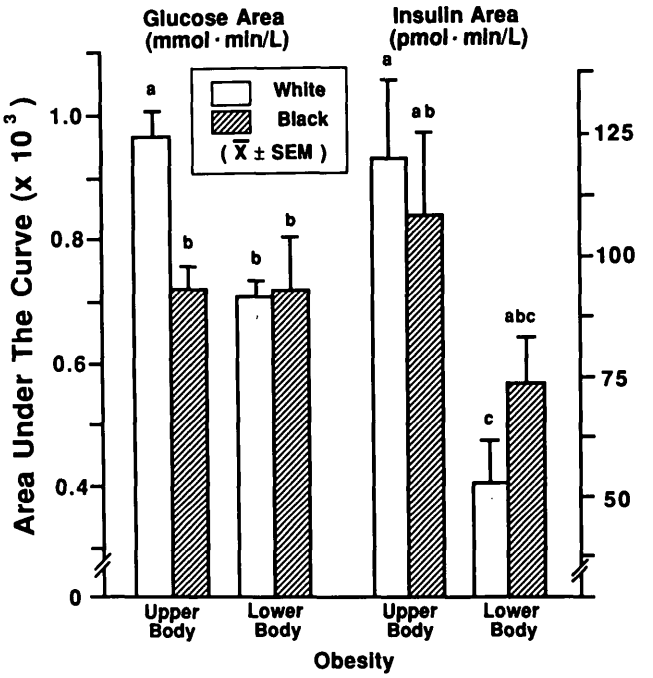

FIG. 1. Integral glucose (mM/2 h) and insulin (pM/2 h) areas (means ± SE) under OGTT curves are given for white and black UBO and LBO groups. Bars with different letters are significantly different from each other at $P < 0.05$, according to LSD post hoc analysis with appropriate mean square error term from two-way ANOVA.

RESULTS

OGTT. The integral glucose area under the OGTT curve (Fig. 1) for the white UBO group (968 ± 38 mM/2 h) was significantly greater by 36% compared with the white LBO group (709 ± 26 mM/2 h). By contrast, in black women, the glucose areas were similar for the UBO and LBO groups (717 ± 39 and 722 ± 81 mM/2 h, respectively). A racial difference in glucose areas was observed between the UBO groups; the area for the white UBO group was 35% greater than that of the black UBO group. However, the glucose areas for the white and black LBO groups were comparable.

The insulin area was significantly greater by 131% in the white UBO group (121 ± 16 pM/2 h) compared with the white LBO group (52 ± 9 pM/2 h). Black women exhibited a similar trend. The insulin area of the black UBO group (108 ± 17 pM/2 h) was 46% greater than that of the black LBO group (74 ± 9 pM/2 h); however, this difference did not reach statistical significance ($P = 0.085$). No racial differences in insulin areas were observed; insulin areas for the white and black UBO groups were similar, as were those of the white and black LBO groups.

Whole-body insulin sensitivity. The S_I index calculated from the IGTT quantifies the dependence of the fractional glucose disappearance on plasma insulin and thus estimates peripheral insulin sensitivity. The S_I index of the white UBO group (0.99 ± 0.16 × 10^{-4} $min^{-1}/\mu U/ml$) was significantly lower by 66% than that of the white LBO

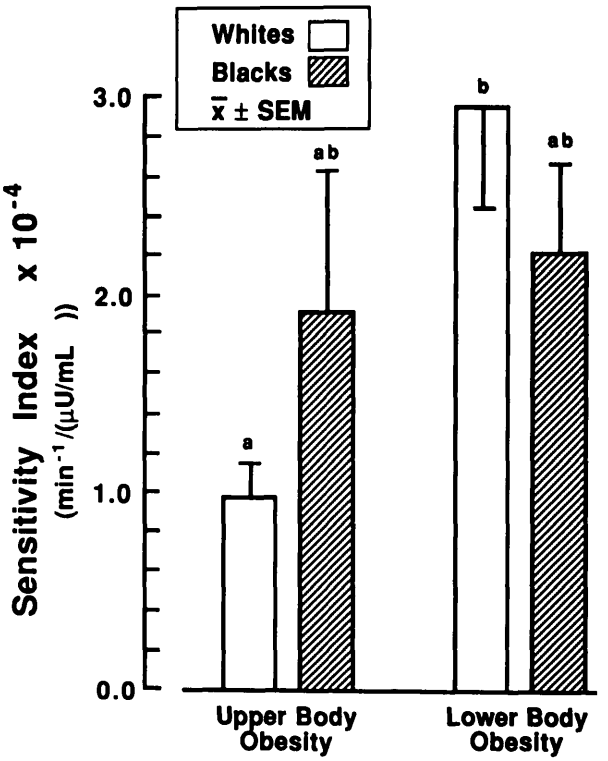

FIG. 2. Sensitivity index, ($min^{-1}/\mu U/ml$) an index of peripheral insulin sensitivity, was measured in vivo by using the FSIGT. Bars with different letters are significantly different at $P < 0.05$.

group (2.95 ± 0.48 × 10^{-4} $min^{-1}/\mu U/ml$) (Fig. 2). By contrast, in black women S_I index values for the UBO and LBO groups were similar (1.91 ± 0.69 and 2.21 ± 0.42 × 10^{-4} $min^{-1}/\mu U/ml$, respectively). No racial differences were observed in the S_I index; the white and black UBO groups had comparable S_I index values as did the white and black LBO groups.

Fasting plasma lipids. Total cholesterol values were moderate (<5 mM) and similar among all four groups (Fig. 3). Likewise, LDL cholesterol values were comparable among all groups.

In both races, HDL cholesterol values were significantly lower in the UBO groups compared with the LBO groups (white UBO, 1.03 ± 0.08 vs. white LBO, 1.49 ± 0.12 and black UBO, 1.14 ± 0.05 vs. black LBO, 1.37 ± 0.08 mM). No racial differences were observed; the white and black UBO groups had similar, low HDL cholesterol values, whereas the white and black LBO groups had comparable, high levels.

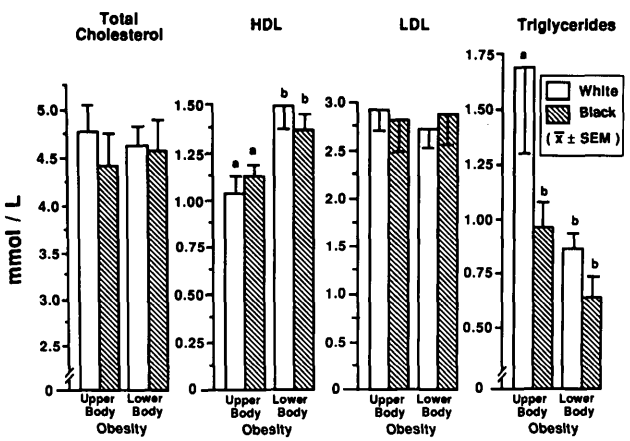

FIG. 3. Fasting plasma levels (means ± SE) of total, HDL, and LDL cholesterol and TG are shown. Bars with different letters are significantly different at $P < 0.05$.

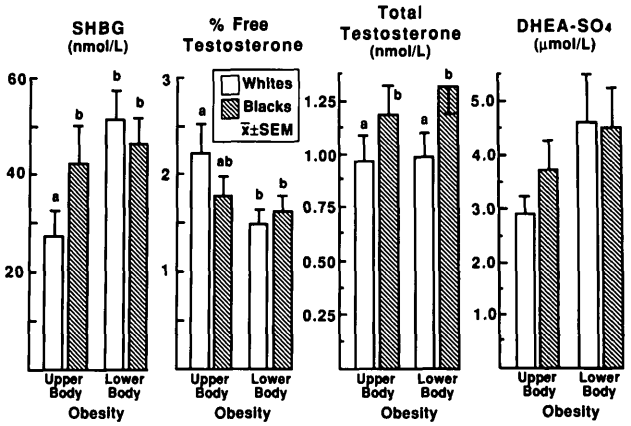

FIG. 4. Sex hormone levels were measured in pooled serum obtained during early follicular phase of menstrual cycle (days 4–7). Blood samples were taken at 30-min intervals over 2 h, and serum was then pooled. SHBG, free and total testosterone, and DHEA-SO_4 levels (means ± SE) are given. Bars with different letters are significantly different at $P < 0.05$.

Serum TG were significantly elevated by 96% in the white UBO group (1.72 ± 0.39 mM) compared with the white LBO group (0.88 ± 0.07 mM). By contrast in black women, TG values were similar between the UBO and LBO groups (0.98 ± 0.10 and 0.66 ± 0.08 mM, respectively). The only racial difference in TG was observed between the UBO groups; TG for the white UBO group was significantly greater by 76% compared with the black UBO group, whereas TG were comparable between the white and black LBO groups.

Sex hormones. SHBG (Fig. 4) was significantly lower by 47% in the white UBO group (27.5 ± 5.0 nM) compared with the white LBO group (51.6 ± 5.2 nM). In contrast, SHBG was similar between the black UBO and LBO groups (42.7 ± 8.0 and 47.0 ± 5.0 nM, respectively). A racial difference was observed between the UBO groups only; SHBG was significantly lower by 36% in the white UBO group compared with the black UBO group, whereas levels were comparable between the white LBO and black LBO groups.

Free testosterone, expressed as a percentage of total testosterone, was significantly greater in the white UBO group (2.21 ± 0.26%) compared to the white LBO group (1.49 ± 0.12%). This was the only observed difference in the percentage of free testosterone. Levels for the black UBO and LBO groups were similar (1.77 ± 0.16 and 1.61 ± 0.13%, respectively). In addition, free testosterone levels were comparable for the white and black UBO groups and the white and black LBO groups.

Total testosterone levels showed a different pattern. Total testosterone was significantly greater by ~27% in black women (UBO, 1.18 ± 0.14 and LBO, 1.32 ± 0.14 nM) compared with white women (UBO 0.98 ± 0.12 and

LBO 0.99 ± 0.11 nM) independent of body fat distribution. Within each race, total testosterone was similar between the UBO and LBO groups.

DHEA-SO_4 was lower in the white UBO group (2.9 ± 0.3 μM) compared with the white LBO group (4.6 ± 0.9 μM); however, this difference did not reach significance ($P = 0.061$). In black women, DHEA-SO_4 was similar between the UBO and LBO groups (3.7 ± 0.5 and 4.5 ± 0.7 μM, respectively). Race did not affect DHEA-SO_4 levels; DHEA-SO_4 was comparable between the white and black LBO groups and between the white and black UBO groups.

BP. sBPs were moderate (<120 mmHg) and similar among all four groups (black UBO, 120 ± 3; black LBO, 118 ± 3; white UBO, 119 ± 3; and white LBO, 112 ± 3 mmHg). However, dBP was significantly higher by 11% in the white UBO group (84 ± 2 mmHg) compared with the white LBO group (75 ± 2 mmHg). By contrast, in black women, dBP was not significantly different between the UBO (85 ± 3 mmHg) and LBO (79 ± 5 mmHg) groups. dBP was also similar between white women and black women who were matched for body fat distribution.

DISCUSSION

Data from this study suggest that UBO, assessed by the WHR (≥0.85), is not as potent a risk factor for the development of diabetes and CVD in premenopausal black women as it is in white women of similar total adiposity.

When challenged with OGTT and IGTT, black UBO and LBO women had comparable glucose and insulin areas and peripheral insulin sensitivity, whereas white UBO women, in confirmation of previous reports (5–9), exhibited glucose intolerance, hyperinsulinemia, and significant insulin resistance compared with white LBO women. These data suggest that, at the level of total body fat studied, an upper body fat distribution has less of an impact on carbohydrate metabolism in black women than it does in white women.

CVD risk was assessed by levels of plasma HDL, LDL, and total cholesterol, TG, and BP. In both races, UBO was associated with a significantly lower HDL cholesterol, whereas total and LDL cholesterol were unaffected. TG, in black women, remained unaltered by extremes of body fat distribution, whereas in white women TG were nearly twofold greater in the UBO group compared with the LBO group. Thus, only low HDL cholesterol was associated with UBO in black women.

Although sBP was not affected by fat patterning in either race, dBP was significantly elevated in white but not black UBO women. This observation supports previous epidemiological findings. Blair and et al. (19) examined the relationship between BP and body fat distribution (assessed by triceps and subscapular skinfolds) in white and black women using data from the First Health and Nutrition Examination Survey (1971–1973). These investigators found that, although the subscapular skinfold had no effect on sBP in either race, it was associated with a greater increase in dBP in white women than in black women. Thus, this study confirms and extends these findings and suggests that the effect of body fat distribution on dBP is race dependent.

These findings, which suggest that UBO is less of a risk factor for black than for white women, are not consistent with the findings of Svec et al. (14). These investigators, whose data was obtained solely from hospital medical records, reported that older, heavier black women had a higher WHR and a greater incidence of diabetes and hypertension compared with younger, thinner black women. Unfortunately, that study did not control for age and body weight, which severely confounds the data; possibly, age or adiposity contributed to the diabetes and hypertension in these black women independent of their body fat distribution. Indeed, Landin et al. (31) have demonstrated the importance of controlling for body weight and adiposity in studies of fat patterning. They found that the degree of obesity significantly influences the expression of metabolic risk factors associated with a high WHR.

Saad et al. (32) observed that the relationship between insulin resistance (and its accompanying hyperinsulinemia) and hypertension is dependent on race; the association was evident in whites but not in blacks or Pima Indians. These investigators suggest that the underlying mechanisms that link insulin resistance to the development of hypertension are active only in whites. However, Falkner et al. (33) observed that lean, young black men with borderline hypertension (135/85 mmHg) had lower rates of insulin-mediated glucose uptake compared with normotensive (112/70 mmHg) black men. They suggest that a relationship does exist between peripheral insulin resistance and hypertension in lean black men. The findings of this study support those of Saad et al. Together, they suggest that certain metabolic relationships (insulin resistance to hypertension and UBO to insulin resistance) appear to be dependent on race. In addition, Banerji and Lebovitz (34) report that 59% of black patients with NIDDM have normal insulin sensitivity by euglycemic insulin clamp and a low risk profile for CVD. Thus, even in diabetic patients (not the case in our study), insulin sensitivity may be better, and CVD risk factors less than would be anticipated, suggesting a possible racial dimorphism.

UBO was not associated with increases of certain androgenic indexes in black women as it was in white women. Although SHBG was decreased and the percentage of free testosterone was increased in white UBO women in confirmation of previous reports (35,36), these levels were not altered by extremes of body fat distribution in black women. Whether the higher levels of total testosterone observed among black women are clinically important is unclear, because it is the free, unbound testosterone that is biologically active. However, to our knowledge, this is a novel finding and may merit further study in light of the fact that Ortiz et al. (22) have recently reported that black women have greater skeletal muscle and bone mineral mass than white women of similar BMI.

Why UBO obesity is less detrimental to black women than to white women is unclear. Perhaps the mechanisms mediating the effects of excessive abdominal fat are somehow attenuated or absent. Alternatively, black

women may have smaller amounts of intra-abdominal fat for a given WHR. In our study, UBO was assessed indirectly by using the anthropometric measure of WHR rather than directly, with computed tomography. The WHR was used because this simple ratio has been shown to correlate significantly with both the total amount of intra-abdominal fat and the ratio of intra-abdominal to subcutaneous fat as measured directly by computed tomography (23). In our study, the UBO white and black groups, although having comparable WHRs, may have had differing amounts of intra-abdominal fat, which is the depot considered most responsible for mediating the effects of UBO (8). Possibly, black women, for a given WHR, have more subcutaneous and less intra-abdominal fat than white women, which may help to explain our findings. To test this hypothesis, future studies should include computed tomographic scans of abdominal fat depots.

In conclusion, data from this study suggest that, in women whose body fat is 43–44%, UBO is less detrimental for black women than for white women with regard to the risks of developing diabetes and CVD. Whether or not this applies to women with a greater or lesser percentage total body fat will need to be determined.

We would like to caution against misinterpreting these findings; they do not imply that obesity among black women is insignificant. Indeed, the national Health and Nutrition Examination Surveys reveal that obesity takes a tremendous toll on the health and longevity of black women. These data do suggest, however, the intriguing possibility that the mechanisms responsible for mediating the deleterious effects of UBO, although functioning in white women, are attenuated or absent in black women.

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