Hyperinsulinism and Sex Hormones in Young Adult African Americans

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Hyperinsulinemia is a risk factor for cardiovascular disease, and is linked with non–insulin-dependent diabetes mellitus (NIDDM), hyperlipidemia, obesity, and hypertension. Sex hormones also play a role in the metabolic alterations associated with the risk for cardiovascular disease. A reduction in sex hormone–binding globulin (SHBG) may be predictive of future NIDDM particularly in women. The postmenopausal decline in estrogen is also associated with an increase in risk factor expression in women. Since African Americans experience a greater prevalence of NIDDM, obesity, and hypertension, conditions associated with hyperinsulinemia, the purpose of this study was to determine if alterations in sex hormone levels are associated with the plasma insulin concentration in young adult African Americans, and to determine if there are sex differences in the effect of insulin on lipids and sex hormones. In a sample of 221 nondiabetic African American men (n = 105) and women (n = 116) with a mean age of 31 years, we examined the relationship of the plasma insulin concentration with the body mass index (BMI), blood pressure, plasma lipids, and sex hormones, including free testosterone, estradiol, and SHBG. Plasma insulin increased with the BMI and other measures of adiposity ($P < .001$) in men and women. Significant correlations of insulin with plasma lipids were also present in both sexes. There was a significant inverse correlation of insulin with SHBG in both men ($r = .28$, $P = .007$) and women ($r = .27$, $P = .02$). There was a significant direct correlation of insulin with free testosterone in women ($r = .032$, $P < .001$). Stepwise multiple regression analyses with insulin as the dependent variable detected the BMI, triglyceride, and apolipoprotein A1 as significant contributors to the plasma insulin concentration in men. In women, the multiple regression model detected percent body fat, low-density lipoprotein (LDL) cholesterol, and free testosterone as significant contributors to plasma insulin. These data on young African Americans demonstrate a significant relationship between hyperinsulinemia and obesity, atherogenic lipid status, and lower SHBG. In the premenopausal women, the lower SHBG is linked with higher free testosterone, favoring a condition of relative androgen excess.

HYPERINSULINEMIA is strongly linked with cardiovascular diseases, including non–insulin-dependent diabetes mellitus (NIDDM), hyperlipidemia, obesity, and hypertension.[1] Both the prevalence and morbidity related to NIDDM, hypertension, and obesity are greater in African Americans compared with Caucasians.[2]

The relationship of sex hormones with insulin, particularly in women, has been investigated in epidemiologic studies. In women, the decline in ovarian steroidogenesis following menopause is associated with an increase in risk factors and expression of NIDDM, essential hypertension (EH), and atherogenic lipid status.[3] In studies on Mexican Americans and Caucasians, Haffner et al[4-6] did not detect an association of total testosterone or total estradiol with cardiovascular risk factors. However, in both premenopausal and postmenopausal women, there was an association of low plasma sex hormone–binding globulin (SHBG) with hyperinsulinemia and an atherogenic lipid profile. Data from other studies have detected a relationship of SHGB with plasma insulin and lipids.[7-9] An androgenic biochemical profile is also linked with NIDDM in Mexican American and Caucasian women.[7,10]

NIDDM, hypertension, and obesity are frequently associated with polycystic ovarian syndrome (PCOS), a condition of hyperandrogenism in premenopausal women. Hyperinsulinism is now considered the key mediator of the increased plasma testosterone and low SHBG in women with PCOS.[11,12] Interventions that decrease plasma insulin by increasing insulin sensitivity, including pharmacologic treatments[13-15] and weight reduction diets,[16,17] have demonstrated a reduction in plasma testosterone and an increase in SHBG.

The purpose of this study was to determine if alterations in sex hormone levels are related to hyperinsulinemia in young adult African Americans, and also to determine if there are differences between men and women in the relationship of the plasma insulin concentration with lipids and sex hormones.

SUBJECTS AND METHODS

Population

The study was performed on 221 nondiabetic African American men (n = 105) and women (n = 116). Each participant was drawn from a cohort that has been under study in investigations of blood pressure and cardiovascular risk factors since adolescence. The sample at the time of study had an age range of 27 to 35 years, and is representative of the African American population of Philadelphia. Participants enrolled in this study included normotensives (blood pressure < 135 mm Hg systolic and <85 mm Hg diastolic) and borderline hypertensives (blood pressure > 135 and <150 mm Hg systolic or >85 and <96 mm Hg diastolic), based on repeated measurements of blood pressure. Known diabetics were excluded from enrollment. Women who were taking oral contraceptives or other exogenous estrogen preparations were excluded from the analysis in this study. Women enrolled in the study reported normal menstrual cycles and had no clinical signs of hyperandrogenism.

Procedures

Enrollment assessment consisted of anthropometric measurements (height, weight, skinfold thickness, and circumference of arm, hips, thigh, and waist) and blood pressure determination. Casual systolic (first phase) and diastolic (fifth phase) blood pressure measurements were obtained by auscultation with a mercury column sphygmomanometer in the sitting position following a 10-minute rest period. The average of two determinations was used as the blood pressure at the time of metabolic evaluation. From the anthropometric measurements,

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the body mass index (BMI), percent body fat, and fat-free mass were calculated.[18]

An oral glucose tolerance test (OGTT) was performed at 8:00 AM following a 12-hour fast. A fasting blood sample for serum lipids and glucose was obtained, and then a 75-g glucose solution (Glucola; Ames Laboratories, Elkhart, IN) was ingested. Blood samples were obtained at 30, 60, 90, and 120 minutes following ingestion of the glucose load, and were assayed for glucose and insulin concentrations. A serum sample was sent to the Lipid Research Laboratory, where total cholesterol, high-density lipoprotein (HDL) cholesterol, and total triglycerides were analyzed using standard enzymatic methods and an automated analyzer (Hitachi 704, Indianapolis, IN). HDL was isolated according to the method of Bachorik et al.[19] Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation.[20] Apolipoprotein A1, apolipoprotein B, and lipoprotein (a) were assayed turbidimetrically using commercial antibodies (Boehringer-Mannheim, Indianapolis, IN).

All female subjects were studied during the follicular phase of the menstrual cycle. Sex hormone levels were determined by radioimmunoassay on serum from samples obtained following an overnight fast. These assays included SHBG (DELFIA; Wallace, Turku, Finland), free testosterone, and estradiol (Diagnostic Products, Los Angeles, CA).

The plasma glucose concentration was analyzed with the glucose oxidase technique (Glucostat, model 27; Yellow Springs Instruments, Yellow Springs, OH). The plasma insulin concentration was determined with a solid-phase radioimmunoassay (Coat-A-Count; Diagnostic Products). The coefficients of variation for interassay and intraassay variability for glucose, insulin, and lipid assays were less than 5%.

Data Analysis

To examine the relationships of plasma insulin with sex hormones and other anthropometric variables, we first stratified the sample according to the fasting plasma insulin concentration. Men and women were grouped separately by tertiles of fasting insulin. Due to the known effect of obesity on the plasma insulin concentration, we also stratified men and women separately into three groups based on the BMI. Using the BMI criteria of Kumanyika,[21] men and women were classified separately as non-obese (low BMI, men < 27.8 and women < 27.3 Kg/m^2), obese (mid BMI, men 27.8 to 31.1 and women 27.3 to 32.2 Kg/m^2), and very obese (high BMI, men > 31.1 and women > 32.2 Kg/m^2). Comparison of group means was performed using a two-way ANOVA comparing the two genders and three insulin or BMI groups with a test for statistically significant interaction. P values less than .05 were considered statistically significant.

We note that the insulin levels were approximately logarithmically normally distributed. ANOVA calculations and P values are based on logarithmic transformation of the plasma insulin values and the sum of plasma insulin values. The insulin data presented in the results are for the measured insulin values, not the logarithmic transformation of the insulin values. Insulin values measured during the OGTT were determined by examining the area under the curve of the four measurements using the trapezoidal rule to estimate the area. We found that the results using this method did not differ from the sum of the four insulin values.

All variables were examined in two sets (one for each gender) of bivariate correlation analyses using the Pearson correlation coefficient. Correlations and regression analyses were completed using the measured plasma insulin values. All variables that correlated significantly with the dependent variable (fasting insulin or sum of insulin levels during the OGTT) were entered into a stepwise multiple linear regression analysis to produce a regression model of the plasma insulin level (fasting or sum of insulin) on the best linear combination of the other measures. The regression models are based on the nonlogarithmically transformed sum of the plasma insulin concentration at fasting and 30, 60, and 120 minutes postbaseline.

The stepwise computer algorithm for the regression equation selects at the first step the single highest correlated variable with the dependent variable. At the second step, the algorithm selects the variable that produces the highest canonical correlation based on two independent variables with the dependent variable. Therefore, variables that are highly correlated with the first independent variable entered are usually not entered into the regression. The computer algorithm continues until there are no additional statistically significant ($P < .05$) increases in the prediction of the single dependent variable on the best linear combination of independent variables.

RESULTS

Data were analyzed on 105 men and 116 women who were not receiving any exogenous sex hormones. Table 1 provides the mean age, anthropometric measures, and blood pressure in each fasting plasma insulin group for men and women. All groups were closely matched by age, with a mean age of 31 years. Men were heavier and taller and had higher systolic blood pressure compared with women. Women had greater triceps skinfold thickness and percent body fat compared with men. Both systolic and diastolic blood pressure increased with increasing fasting insulin for both men and women. The mean values for sex hormones in each fasting insulin group are provided in Table 2. Sex hormone data demonstrated the expected differences between men and women. However, free testosterone increased with increasing fasting insulin in women, whereas there was a slight decrease in free testosterone with increasing fasting insulin in men. This resulted in a statistically significant interaction on free testosterone of sex $\times$ insulin group ($P < .001$). There was a decrease in mean SHBG from

Table 1. Characteristics of the Study Group

	Men			Women			Significance (P)		
Variable	Lowest Tertile (n = 35)	Middle Tertile (n = 35)	Upper Tertile (n = 35)	Lowest Tertile (n = 39)	Middle Tertile (n = 39)	Upper Tertile (n = 38)	Sex	Insulin Tertile	Sex × Insulin Tertile
Age (yr)	31.7 ± 3.6	30.4 ± 3.7	31.7 ± 5.1	31.2 ± 3.5	32.4 ± 3.4	32.5 ± 5.6	.18	.48	.20
BMI (kg/m^2)	23.9 ± 3.4	26.4 ± 4.0	32.6 ± 6.3	26.5 ± 5.5	33.7 ± 8.9	36.0 ± 10.1	.001	.001	.09
Height (cm)	178 ± 6	179 ± 8	176 ± 8	163 ± 7	164 ± 7	164 ± 9	.001	.39	.48
Weight (kg)	75 ± 9	85 ± 16	101 ± 22	71 ± 16	91 ± 26	96 ± 25	.73	.001	.18
Triceps skinfold	9.3 ± 4.4	13.8 ± 7.0	20.2 ± 4.9	20.5 ± 7.5	25.9 ± 8.7	26.7 ± 8.5	.001	.001	.05
Body fat (%)	16.9 ± 5.2	21.4 ± 5.9	27.2 ± 4.9	31.1 ± 6.8	36.4 ± 5.6	37.4 ± 5.2	.001	.001	.02
SBP (mm Hg)	121.2 ± 11.0	128.4 ± 15.5	130.7 ± 13.5	112.8 ± 12	124.3 ± 16	126.2 ± 16.8	.005	.001	.61
DBP (mm Hg)	76.6 ± 10.4	79.2 ± 14.2	83.7 ± 11.5	72.9 ± 10.7	79.8 ± 11.9	82.5 ± 15.4	.4	.001	.57

NOTE. Tertiles are sex-based fasting insulin tertiles.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2. Sex Hormone Levels in the Study Group

Variable	Men			Women			Significance (*P*)		
	Lowest Tertile	Middle Tertile	Upper Tertile	Lowest Tertile	Middle Tertile	Upper Tertile	Sex	Insulin Tertile	Sex × Insulin Tertile
Free testosterone	22.5 ± 6.6	21.3 ± 5.9	18.0 ± 4.6	1.0 ± 0.6	1.3 ± 0.8	1.8 ± 1.6	.001	.03	.001
Estradiol	44.5 ± 16.4	46.8 ± 22.2	42.8 ± 19.2	94.7 ± 73.2	85.4 ± 58.6	73.9 ± 51.1	.001	.3	.47
SHBG	31.6 ± 23.1	27.9 ± 14.1	20.6 ± 14.8	47.1 ± 25.4	35.0 ± 19.0	34.4 ± 19.0	.001	.006	.49
T/E ratio	0.56 ± 0.21	0.54 ± 0.25	0.48 ± 0.20	0.02 ± 0.02	0.02 ± 0.02	0.03 ± 0.03	.001	.46	.20
Sample size for SHBG (n)	31	32	32	31	25	18			

NOTE. Tertiles are sex-based fasting insulin tertiles.
Abbreviation: T/E, free testosterone/estradiol.

the low to high insulin group for both men and women, and this change was statistically significant ($P = .006$).

The OGTT results for plasma glucose and insulin concentrations are presented in Table 3 for men and women according to BMI groups. The mean plasma insulin increased significantly from the low to high BMI group at each time point in the OGTT for both men and women, but there were no statistically significant sex differences. The mean plasma glucose was greater in the high BMI group. Although there were no sex differences in fasting plasma glucose, during the subsequent postglucose challenge, plasma glucose was higher in men versus women and these differences reached statistical significance.

Correlation analyses were applied on all men and all women with the plasma insulin levels during the OGTT. The results of these analyses using the sum of insulin levels during the OGTT as the dependent variable are provided in Table 4. There was a direct statistically significant correlation for the sum of insulin with the BMI and other parameters of adiposity for both men and women. The index of central obesity (the ratio of subscapular to triceps skinfold thickness) correlated significantly with plasma insulin in women ($r = .30$, $P < .001$) but not in men ($r = .07$, $P = .46$). The correlation coefficients of insulin with blood pressure measurements did not reach statistical significance for men or women.

There were statistically significant correlations for the sum of insulin during the OGTT with each of the lipid parameters in men. In women, plasma insulin correlated most significantly with LDL cholesterol ($P < .001$) and apolipoprotein B ($P = .004$). There were also statistically significant correlations for insulin with total cholesterol ($P = .02$) and triglycerides ($P < .03$). HDL cholesterol and apolipoprotein A1 in women in this sample did not correlate significantly with insulin.

The bivariate correlation of the sum of insulin during the OGTT with free testosterone was statistically significant in women ($r = .32$, $P < .001$) but not in men. These results in women are depicted in Fig 1. There were no statistically significant correlations for insulin with estradiol or the ratio of free testosterone to estradiol in men or women. There was a statistically significant correlation for insulin with SHBG in men ($r = -.28$, $P = .007$) and women ($r = -.27$, $P = .02$).

We then conducted stepwise multiple regression analyses of all variables on the sum of insulin as the dependent variable. The regression models are presented in Table 5 for men and women separately. In this study, there were some highly correlated independent variables such as the weight, height, and BMI. The regression algorithm is not disrupted or negated by this multicolinearity, but once one of a set of highly correlated parameters is entered into the model, usually the strongest correlate with the dependent variable, there is no additional predictive value for the others. In our analyses, we found that the BMI is highly correlated with other anthropometric measures such as weight and percent body fat. Once the BMI or percent body fat was entered into the regression model, neither weight nor other measures of adiposity added any additional predictive value.

Table 3. OGTT Results

Variable	Men			Women			Significance (*P*)		
	Low BMI (n = 60)	Mid BMI (n = 22)	High BMI (n = 23)	Low BMI (n = 41)	Mid BMI (n = 24)	High BMI (n = 51)	Sex	BMI	BMI × Sex
Plasma insulin (μU/mL)									
Fasting	7.6 ± 5.7	12.5 ± 8.5	20.1 ± 9.9	9.7 ± 8.8	15.2 ± 12.3	16.1 ± 8.7	.77	.001	.16
30 min	59.5 ± 41.1	106.1 ± 67.3	157.0 ± 77.2	80.3 ± 50.2	106.8 ± 66.6	149.4 ± 98.2	.26	.001	.14
60 min	73.8 ± 49.0	98.5 ± 55.3	180.3 ± 74.0	74.4 ± 56.6	91.3 ± 44.5	153.0 ± 91.0	.25	.001	.58
120 min	40.3 ± 49.6	53.9 ± 43.3	118.9 ± 78.2	56.5 ± 50.7	79.3 ± 79.4	119.2 ± 93.3	.01	.001	.21
Approximate area under the insulin curve									
Sum of insulin levels	181 ± 115	271 ± 133	476 ± 187	221 ± 138	292 ± 156	438 ± 245	.56	.001	.19
Plasma glucose									
Fasting	92.1 ± 7.6	96.2 ± 9.8	100.2 ± 10.3	90.9 ± 14.6	93.6 ± 11.0	94.6 ± 28.9	.26	.06	.73
30 min	155.9 ± 24.3	155.9 ± 28.4	172.7 ± 22.1	139.5 ± 30.7	139.9 ± 22.7	150.2 ± 39.7	.001	.03	.93
60 min	161.1 ± 32.6	153.4 ± 29.8	182.1 ± 33.3	139.5 ± 44.1	143.6 ± 36.9	163.5 ± 48.4	.004	.01	.29
120 min	104.1 ± 28.9	108.8 ± 27.8	135.7 ± 34.3	120.4 ± 37.7	119.6 ± 34.2	134.6 ± 55.3	.04	.008	.49

NOTE. ANOVA calculations and *P* values are based on logarithmic transformation of the plasma insulin values and the sum of plasma insulin values.

Table 4. Correlates of Plasma Insulin (sum of insulin) for Men and Women

Correlate	Men (n = 105)		Women (n = 116)	
	Pearson *r*	*P*	Pearson *r*	*P*
Free testosterone	−.16	.11	.32	.001
Estradiol	−.05	.61	−.03	.74
SHBG*	−.28	.007	−.27	.02
T/E ratio	−.08	.42	.14	.15
Height	−.18	.06	−.04	.66
Weight	.51	.001	.35	.001
Triceps skinfold	.44	.001	.32	.001
BMI	.59	.001	.37	.001
% body fat	.52	.001	.43	.001
Total body fat	.54	.001	.41	.001
Fat-free mass	.41	.001	.26	.004
Centrality index	.07	.46	.30	.001
SBP	.16	.10	.18	.06
DBP	.13	.19	.11	.24
MBP	.15	.13	.14	.13
Total cholesterol	.25	.01	.22	.02
HDL	−.32	.001	−.22	.20
LDL	.31	.001	.30	.001
Triglycerides	.53	.001	.20	.03
Apo A1	−.29	.003	−.06	.52
Apo B	.48	.001	.29	.004

Abbreviations: T/E, ; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBP, mean blood pressure; Apo, apolipoprotein.

*Sample sizes for SHBG were n = 95 and n = 74 for men and women, respectively.

When the final multiple *R* value for the men is squared, 47% of the variation in the sum of insulin is explainable by the three independent variables. The BMI accounts for 35%, triglyceride accounts for 8%, and apolipoprotein A1 accounts for the remaining 4% of the variance in the dependent variable. For the women, the multiple R^2 equals 28% of the variance. Percent body fat accounts for 19%, LDL cholesterol accounts for 5%, and free testosterone accounts for the remaining 4% of the variance in the dependent variable. Sex hormones did not contribute to the model in men. However, in women, free testosterone does contribute significantly to the variance in insulin levels.

Table 5. Stepwise Linear Regression Models

Step/Variable	Slope	SE of Slope	β	R^2	Overall F Ratio	Overall *P*	F Change *P*
Men (n = 105)							
BMI	12.57	2.47	0.41	.59	56.3	<.001	<.001
Plasma triglycerides	1.08	0.27	0.33	.66	40.2	<.001	<.001
Apolipoprotein A1	−1.10	0.41	−0.20	.69	30.9	<.001	.008
Women (n = 116)							
% body fat	11.06	2.86	0.33	0.43	26.0	<.001	<.001
Plasma LDL	1.48	0.56	0.22	0.48	17.1	<.001	.010
Free testosterone	40.89	16.49	0.21	0.53	14.0	<.001	.015

DISCUSSION

Hyperinsulinemia is considered a significant risk factor for cardiovascular disease. In this study on young adult African Americans, plasma insulin correlates most strongly with the BMI, as well as other measures of adiposity, in both men and women. Relative hyperinsulinemia is an index of insulin resistance,[1] and obesity is a well-established component of the insulin resistance syndrome in Caucasians and other ethnic groups.[22-24] In the young men, strong correlations were present between the sum of insulin concentrations during the OGTT and each of the plasma lipid parameters. In the young women, strong correlations were present between insulin and LDL cholesterol and apolipoprotein B, and the correlations for insulin with total cholesterol and triglycerides also reached a level of statistical significance. Overall, the correlations for hyperinsulinemia with plasma lipids in this sample of young African Americans are also consistent with the dyslipidemia of hyperinsulinemia and insulin resistance.[23]

We detected an inverse relationship of SHBG with plasma

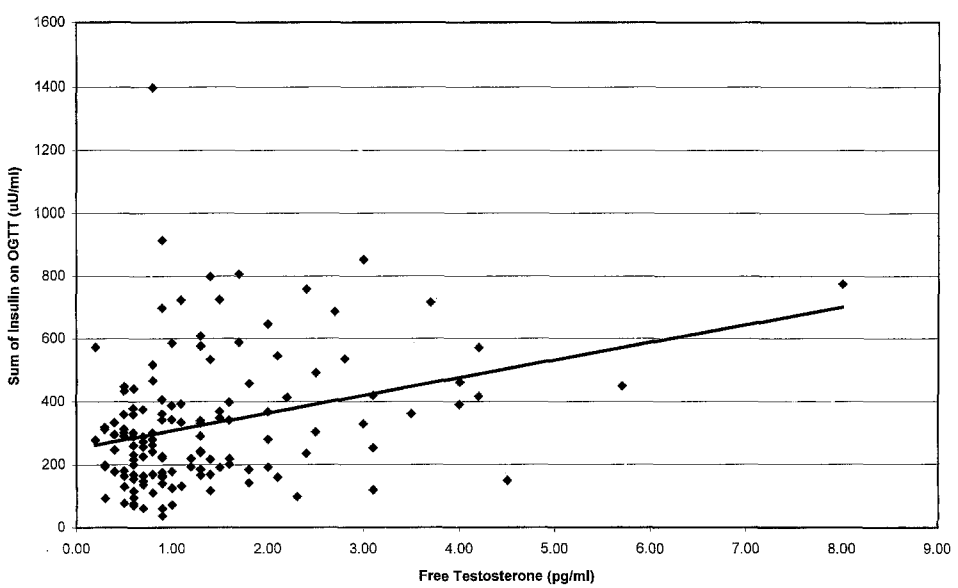

Fig 1. Correlation of the sum of insulin during the OGTT with plasma free testosterone in the women (statistically significant, $r = .31$, $P < .001$). For the men (not shown), the correlation was not significant ($r = .16$, $P = .12$).

insulin in men. However, in the premenopausal women, there was a very strong direct correlation for free testosterone with insulin, as well as an inverse correlation for SHBG with insulin. Our multiple regression model also detected a significant contribution of free testosterone to hyperinsulinemia. We have previously reported a significant correlation for SHBG with insulin sensitivity in premenopausal African American women.[25] Thus, SHBG decreases with progressive insulin resistance in this population. The correlation of insulin with free testosterone and SHBG reported here indicates that hyperinsulinemia in women is related to higher androgen activity. It is of note that free testosterone was highest and SHBG lowest in the high BMI, or most obese, group of women.

Previous studies have reported a similar relationship of fasting insulin with androgen activity in women.[9,26] Haffner et al[10] reported that lower SHBG is predictive of NIDDM in women but not in men. The data from our cross-sectional examination of African Americans demonstrate that plasma insulin is inversely correlated with SHBG in both men and women. Further, the OGTT data indicate that with increased BMI, there is an increase in plasma glucose and insulin, indicative of progressive impairment in glucose tolerance in both men and women.

In a recent study, Larsson and Ahren[27] found that postmenopausal women with impaired glucose tolerance have higher androgen activity than women with normal glucose tolerance, and the androgen activity correlates with the degree of glucose intolerance. The correlations for hyperinsulinemia with lower SHBG and higher free testosterone in the most obese premenopausal African American women suggest a heightened risk for development of NIDDM.

SHBG synthesis is modulated by sex hormones. The binding of sex hormones with plasma proteins, including SHBG, determines tissue uptake and subsequent activity of sex hormones in vivo. SHBG levels are higher in women compared with men[28] and higher in children compared with adults, and decrease at puberty in both sexes.[29,30] Women with clinical androgen excess, including those with PCOS and idiopathic hirsutism (IH), have low SHBG compared with unaffected women.[31,32] Our sample excluded women with known PCOS or IH. None of the women reported here had clinical signs of PCOS, and all had regular menses. Data from our sample suggest that very obese premenopausal African American women, who do not meet the criteria for hyperandrogenism syndromes, have a relative androgen excess that is associated with other metabolic risk factors for NIDDM.

Insulin resistance accompanied by hyperinsulinemia is a common feature of PCOS.[33,34] In women with PCOS, hyperinsulinemia may contribute to hyperandrogenism by increasing ovarian androgen production and decreasing SHBG.[35-37] Dunaif et al[15] demonstrated that an improvement in insulin sensitivity with a decrease in hyperinsulinism during treatment with the insulin-sensitizing agent troglitazone also results in a decrease of androgen excess in PCOS. They reported that the decrease in plasma insulin resulted in an increase in SHBG with a concurrent decrease in free testosterone, changes that are consistent with the concept that insulin is a direct negative regulator of hepatic SHBG production.[14,38] From the data on our sample of premenopausal obese women without PCOS, it is possible that the hyperinsulinemia may have a direct effect on ovarian androgen production. However, the strong relationship of hyperinsulinemia with lower SHBG in men and women would suggest that the impairment of hepatic SHBG production by hyperinsulinemia accounts for the relative androgen excess observed in the obese women.

Data from this study on young adult African American men and women demonstrate a significant relationship of hyperinsulinemia to an atherogenic lipid profile and lower SHBG, as well as obesity. These findings are consistent with reports on Caucasians and Mexican Americans.[4-6] In these premenopausal women, lower SHBG is accompanied by higher free testosterone, favoring a condition of relative hyperandrogenism. Together, this constellation of metabolic parameters suggests that very obese premenopausal African American women are at high risk for NIDDM and associated cardiovascular disease.

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