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Lipoprotein abnormalities are associated with insulin resistance in South Asian Indian women

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Abstract

South Asian Indians are at increased risk of coronary heart disease (CHD), possibly related to dyslipidemia characterized by high triglyceride (TG) and low high-density lipoprotein cholesterol (HDL-C) concentrations. The importance of differences in insulin resistance as compared to abdominal obesity in the development of this atherogenic lipoprotein profile is not clear, and the current cross-sectional study was initiated to examine this issue. Consequently, we defined the relationship between differences in insulin-mediated glucose uptake (IMGU), abdominal obesity, and various measures of lipoprotein metabolism known to increase CHD risk in 52 apparently healthy women of South Asian Indian ancestry. IMGU was quantified by determining the steady-state plasma glucose (SSPG) concentration during the insulin suppression test and abdominal obesity was assessed by measurement of waist circumference (WC), and the population was divided into tertiles on the basis of their SSPG results. Results indicated that although there were significant differences in SSPG, TG, and HDL-C values, there were no differences in age, blood pressure, total cholesterol, low-density lipoprotein cholesterol, body mass index, or WC between the highest and lowest tertiles. SSPG concentrations were significantly correlated with both log TG ($r = 0.44$, $P = .001$) and HDL-C ($r = -0.44$, $P < .001$) concentration, whereas TG and HDL-C concentrations were not significantly related to WC. Furthermore, the relationships between SSPG concentration and TG and HDL-C remained significant when adjusted for age and WC. Finally, a more extensive lipoprotein analysis indicated that the most insulin resistant tertile had higher TG concentrations, lower concentrations of HDL-C and HDL-C subclasses, and smaller and denser low-density lipoprotein particles than the most insulin sensitive tertile, despite the 2 groups not being different in age, BMI, or WC. These results indicate that a highly atherogenic lipoprotein profile seen in South Asian Indian women is significantly associated with insulin resistance independent of differences in WC.

1. Introduction

South Asian Indians have a higher mortality rate due to coronary heart disease (CHD) when compared with other ethnic groups [1], possibly related to an increased prevalence of insulin resistance and hyperinsulinemia, with dyslipidemia characterized by high triglyceride (TG) and low high-density lipoprotein cholesterol (HDL-C) concentrations [2,3]. There is evidence that high TG and low HDL-C concentrations are independently related to insulin resistance in individuals of both South Asian Indian and European ancestry [3-5]. On the other hand, more recent reports have emphasized the role that differences in regional fat distribution may play in the insulin resistance and dyslipidemia observed in South Asian Indians [6,7]. However, studies in which a specific measure of insulin-mediated glucose uptake (IMGU) has been used to define the relationship among insulin resistance, regional fat distribution, and dyslipidemia in South Asian Indians have enrolled relatively few participants, and the experimental population has consisted entirely, or primarily, of men [6,7]. Consequently, we believed it would be of interest to further pursue these issues, using a specific measure of IMGU, enrolling a larger number of participants, and focusing in this instance on women.

2. Methods

The study population consisted of 52 nondiabetic, apparently healthy women of South Asian Indian ethnicity,

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aged 30 to 64 years, who responded to print advertisements describing our studies on the role of insulin resistance in human disease. The Stanford Human Subjects Committee approved the protocols, and all subjects gave informed consent. Participants were in good health with normal physical examination findings and medical histories, were nondiabetic as defined by the American Diabetes Association [8], had normal liver/kidney function, and had no anemia. Volunteers meeting these criteria had their body mass index (BMI, kg/m^2) and waist circumference (WC, cm) determined as described previously [9], and those individuals with BMI of 23 kg/m^2 or greater were considered eligible for this study. The study was performed at the General Clinical Research Center of the Stanford University Medical Center.

Insulin-mediated glucose uptake was quantified by a modified version [10] of the insulin suppression test (IST) as described and validated by our research group [11,12]. After an overnight fast, an intravenous catheter was placed in each arm of the subject, one for the simultaneous 3-hour infusion of octreotide (0.27 μg/m^2 per minute), insulin (32 mU/m^2 per minute), and glucose (267 mg/m^2 per minute), and the other for the collection of blood samples every 10 minutes during the 150- to 180-minute period to measure plasma glucose and insulin concentrations. The values obtained during these last 30 minutes were then averaged to determine the steady-state plasma glucose (SSPG) and steady-state plasma insulin concentrations. Because steady-state plasma insulin concentrations are comparable in all individuals and glucose infusion is identical, the resultant SSPG concentration provides a direct measure of the ability of insulin to mediate the disposal of a given glucose load; the higher the SSPG, the more insulin resistant the individual.

On the day of the IST, fasting blood samples were also obtained for determination of fasting lipid concentrations and lipoproteins, and the samples were stored frozen at −80°C. Plasma TG and HDL-C concentrations were determined in all subjects in the clinical laboratory of Stanford Medical Center. In a subset of patients, a more comprehensive lipoprotein analysis was obtained with the Vertical Auto Profile II (VAP-II) method, which is a density gradient approach that provides a measurement of cholesterol concentration in HDL-C, intermediate density lipoprotein cholesterol (IDL-C), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), and lipoprotein(a) [Lp(a)] in a single test [13]. The LDL subclass pattern is determined by the LDL peak maximum time, which is defined as the relative position of the LDL peak in the density gradient on a relative scale of 0 to 200 seconds, with 0 second corresponding to the beginning of the HDL (the most dense lipoprotein) peak and 200 seconds corresponding to the VLDL (the least dense lipoprotein) peak maximum. Small, dense LDL (pattern B) is defined by LDL maximum time of 115 seconds. These analyses were performed by Atherotech (Birmingham, AL), and VAP-II has been extensively validated with the Lipid Research Clinics β-quantification method performed at Northwest Lipid Research Laboratories at the University of Washington (Seattle, WA) [13]. It should be emphasized that this method provides a specific measure of narrow-density LDL-C (ND-LDL-C), in contrast to the commonly measured LDL-C defined by the National Cholesterol Education Program, which includes both Lp(a) and IDL-C.

Data are expressed as the mean ± SD. The initial analysis was performed by dividing the population into tertiles, based on their SSPG concentrations, and comparing the anthropometric metabolic variables of these 3 groups by analysis of variance. Pairwise comparisons between the highest and lowest, lowest and intermediate, and intermediate and highest tertiles were made using t tests. In addition, Pearson correlation coefficients were calculated between SSPG and lipid concentrations and obesity indices in the entire study population. Triglyceride values were log transformed to normalize the distribution for analysis purposes. Finally, a more intensive comparison of lipoprotein metabolism was made between the 16 most insulin resistant and 16 most insulin sensitive individuals by using t tests and χ^2 tests as appropriate. Multiple linear regression analyses were performed to evaluate the relative contribution of SSPG and WC to variations in TG and HDL-C concentrations. All statistical evaluations were performed with the Excel (Microsoft, Redmond, WA) and STATA 9 statistical software packages for Windows (StataCorp, College Station, TX). Statistical significance was assigned at $P < .05$.

3. Results

Table 1 compares the clinical characteristics of the 52 women enrolled in this study, divided into tertiles on the basis of their SSPG concentrations. By selection, SSPG concentrations increased from the most insulin sensitive to the most insulin resistant tertile. There were no differences in the ages, blood pressure, and total cholesterol or LDL-C levels of the 3 groups. However, both BMI and WC and TG and HDL-C concentrations varied as a function of SSPG tertile. Specifically, values for WC were significantly lower in the intermediate tertile than in the other 2 tertiles. However, neither BMI nor WC was significantly different between the most insulin sensitive (lowest SSPG) and the most insulin resistant (highest SSPG) tertiles. In the case of TG and HDL-C concentrations, values in the most insulin resistant third of the population were significantly different than in the other 2 groups.

To further evaluate the relationship between WC and TG and HDL-C concentrations compared with that between SSPG concentration and lipid measurements, simple Pearson correlation coefficients were determined. The results in the top left panel of Fig. 1 show that SSPG concentrations varied approximately 6-fold in the study population and that there was a statistically significant correlation (r = 0.44) between SSPG and log TG

Table 1
Characteristics based on upper, lower, and middle tertile SSPG values (mean ± SD)

	Lowest tertile (n = 17)	Intermediate tertile (n = 18)	Highest tertile (n = 17)	*P*
SSPG (range) (mg/dL)	85 ± 20 (54-112)	140 ± 20 (114-178)*	232 ± 48 (180-341)†,‡	<.001
Age (range) (y)	44 ± 10 (30-64)	43 ± 10 (31-64)	43 ± 8 (30-58)	.94
BMI (range) (kg/m^2)	27.9 ± 4.1 (23.7-37.5)	27.0 ± 2.3 (23.4-32.8)	30.1 ± 3.6 (26.0-37.4)‡	.04
WC (range) (cm)	92 ± 10 (78.5-116)	86 ± 6 (78.5-100)*	97 ± 11 (83-119)‡	.02
Systolic blood pressure (mm Hg)	115 ± 19	115 ± 18	116 ± 20	.99
Diastolic blood pressure (mm Hg)	69 ± 10	69 ± 7	69 ± 10	.99
Total cholesterol (mg/dL)	166 ± 25	177 ± 35	173 ± 32	.58
LDL-C (mg/dL)	101 ± 20	114 ± 30	103 ± 20	.24
HDL-C (mg/dL)	52 ± 12	51 ± 9	41 ± 8†,‡	.0016
TG (mg/dL)	84 ± 46	108 ± 41	153 ± 76†,‡	.0029

Pairwise comparisons (for SSPG, BMI, WC, HDL, and TG).
* $P < .05$, lowest tertile compared with intermediate tertile (placed as superscript in intermediate tertile).
† $P < .05$, lowest tertile compared with highest tertile (placed as superscript in highest tertile column first).
‡ $P < .05$, intermediate tertile compared with highest tertile (placed as superscript in highest tertile column second).

concentrations ($P = .001$). In contrast, the relationship between TG concentrations and WC ($P = .38$) was not statistically significant. The results in the lower left panel of Fig. 1 indicate that the relationship between SSPG and HDL-C ($r = -0.44$) was also statistically significant ($P = .001$), whereas WC was not significantly related to HDL-C concentration ($P = .34$).

Although neither WC nor BMI (data not shown) were associated with TG or HDL-C concentrations, the 2 indices of obesity were themselves significantly correlated ($r = 0.77$, $P < .001$), and both BMI ($r = 0.45$, $P < .001$) and WC ($r = 0.33$. $P = .04$) were related to SSPG concentration.

Multiple linear regression analysis was performed to evaluate the independence of the relationship between SSPG and TG and HDL-C concentration. When adjusted for differences in age and WC, the relationships between SSPG and log TG and HDL-C concentrations remained statistically significant (log TG, $P = .003$; HDL-C, $P = .002$). BMI was also tested as a marker of obesity in these analyses and, similar to WC, was not significantly related to either TG or HDL-C concentration.

Finally, a more complete analysis of the differences in lipid and lipoprotein metabolism between the 16 most insulin resistant and 16 most insulin sensitive individuals is seen in Table 2. These results show that the SSPG concentrations were 2.2-fold increased in the most insulin resistant women, associated with higher TG concentrations, lower HDL-C, lower HDL-C_2, and HDL-C_3 concentrations, an increase in the prevalence of atherogenic, small, dense LDL and decreased time to peak LDL, indicating smaller LDL particle size. However, there were no differences between the total cholesterol and LDL-C concentrations of the 2 groups. Consistent with the results in Table 1, there were no differences in the ages, BMI, or WC of the 2 groups

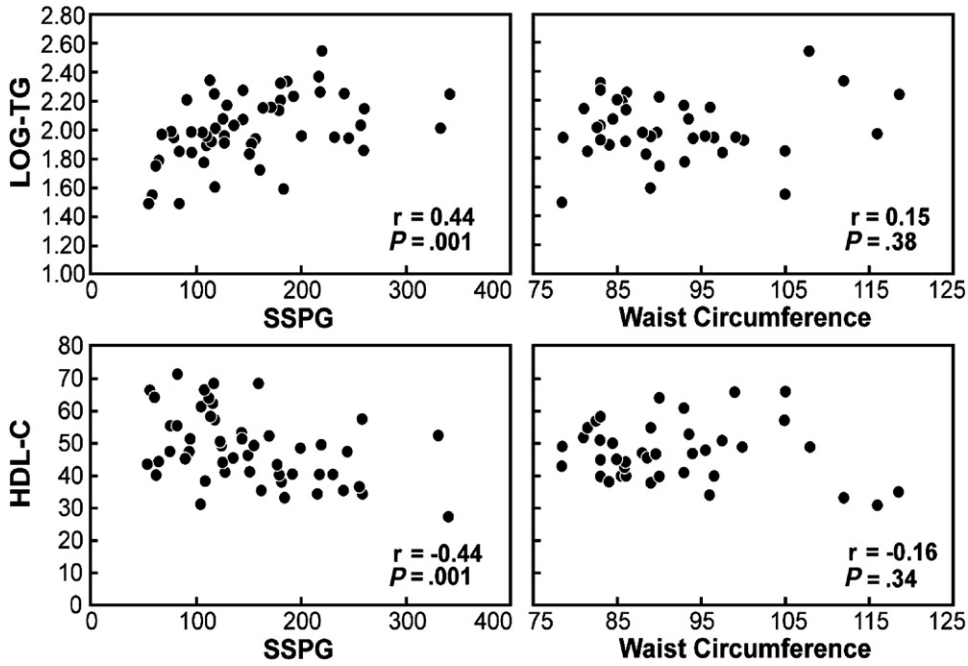

Fig. 1. Correlation coefficients (r values) between SSPG and WC and TG and HDL-C concentration.

Table 2
Specialized lipoprotein characteristics of insulin-sensitive and -resistant groups (mean ± SD)

	Insulin sensitive, SSPG ≤130 (n = 16)	Insulin resistant, SSPG ≥170 (n = 16)	*P*
Age (y)	46 ± 11	43 ± 8	.2
BMI (kg/m^2)	28.7 ± 3.8	28.4 ± 2.2	.4
WC (cm)	93 ± 10	93 ± 10	.5
SSPG (mg/dL)	98 ± 21	219 ± 52	<.0001
Systolic blood pressure (mm Hg)	119 ± 19	113 ± 19	.2
Diastolic blood pressure (mm Hg)	71 ± 11	69 ± 9	.3
Total cholesterol (mg/dL)	173 ± 19	174 ± 28	.5
LDL-C (mg/dL)	103 ± 13	110 ± 24	.2
Pattern B (%)	19	69	.02
Time to peak LDL-C (s)	117 ± 3	114 ± 3	.002
HDL-C (mg/dL)	53 ± 9	44 ± 9	.003
HDL-C_2 (mg/dL)	13 ± 3	10 ± 3	.006
HDL-C_3 (mg/dL)	40 ± 6	34 ± 7	.004
TG (mg/dL)	93 ± 42	151 ± 70	.004
hsCRP (mg/dL)	2.8 ± 2	4.1 ± 5	.2
Lp(a) (mg/dL)	7.0 ± 3	4.9 ± 2	.006

that differed so dramatically in insulin sensitivity and lipoprotein profile.

Finally, although not the goal of the study, the high-sensitivity C-reactive protein (hsCRP) and Lp(a) concentrations of the most insulin sensitive and resistant groups were also compared. These data also appear in Table 2 and indicate that, whereas there were no differences in the hsCRP concentrations of the 2 groups, the insulin-sensitive individuals had significantly higher values for Lp(a).

4. Discussion

The results provide substantial evidence that IMGU was significantly associated with high TG and low HDL-C concentrations in the 52 South Asian Indian women enrolled in this study and that this relationship was independent of differences in abdominal obesity as assessed by WC. Thus, the findings in Table 1 show that the most insulin resistant third of the population had SSPG concentrations approximately 3-fold higher than the most insulin sensitive tertile, associated with significantly higher plasma TG and lower HDL-C concentrations. These differences were seen, although these 2 groups were not different in age, BMI, or WC.

The findings in Table 1 are buttressed by the cross-sectional results shown in Fig. 1 indicating that SSPG concentrations were significantly related to both TG and HDL-C concentrations, whereas this was not true of measurements of WC. Furthermore, the relationship between SSPG and TG and HDL-C concentrations remained statistically significant when adjusted for differences in age and WC. It should be noted that these findings were seen in a population enriched in degree of adiposity.

The atherogenic lipoprotein profile associated with insulin resistance in South Asian women is not limited to a high TG and low HDL-C concentration as shown in Table 2. Thus, in addition to a high TG and low HDL-C, the 16 most insulin resistant women in whom we had a complete lipoprotein profile had significantly lower HDL-C_2 and HDL-C_3 concentrations, as well as a shift to smaller and denser LDL particles compared with the 16 most insulin sensitive subjects. Again, these differences were seen although the 2 groups were similar in age, WC, and BMI.

Although these data support the view that insulin resistance, not abdominal obesity, is primarily responsible for the characteristic dyslipidemia seen in South Asian individuals [2,3], it must be pointed out that we used WC as a surrogate marker of abdominal obesity, rather than directly measuring visceral fat. In that context, it is necessary to discuss our findings in light of 2 other studies [6,7], in which quantitative measurements were made of IMGU, lipoprotein concentrations, and visceral fat content. In the case of Banerji et al [6], results in 20 South Asian Indian men indicated that whereas both IMGU and visceral adipose tissue volume were significantly correlated with plasma TG concentrations ($r = -0.45$ and 0.46, respectively), only visceral adiposity was independently associated with TG concentration on multivariate analysis. In contrast, only insulin resistance, not visceral adiposity, was significantly correlated with HDL-C concentration. Raji et al [7], in their study of 25 South Asians (18 men and 7 women), also found significant correlations between IMGU and both TG ($r = -0.47$) and HDL-C ($r = 0.54$) concentrations, but did not attempt to see if these relationships were independent of differences in various measures of obesity.

It should be remembered that our experimental population, consisting of 52 women, was quite different from that of the 2 studies discussed above. In contrast, only 7 women were represented in the total of 45 subjects studied by Banerji et al [6] and Raji et al [7]. Despite this fairly dramatic gender difference in patient population, a surprising number of generalizations seem to be justified. In the case of HDL-C concentrations, the evidence to date is unanimous that the more insulin resistant South Asian Indians are, the lower their HDL-C concentration will be; this relationship is true of men and women and is independent of abdominal obesity as estimated visceral adipose tissue volume or WC.

The situation is more controversial regarding plasma TG concentrations. Although there is general agreement that measures of IMGU and TG concentrations are significantly correlated in both South Asian Indian men and women, Banerji et al [6] have concluded that in men the increase in TG concentration is a function of more visceral adiposity, whereas our findings in women suggest that the dyslipidemia is secondary to insulin resistance. There are 2 obvious explanations for the discrepant

conclusion concerning the relationship between insulin resistance, abdominal obesity, and plasma TG concentration. At the simplest, it could be argued that the difference is related to gender; we studied women, Banerji et al studied men [6]. Alternatively, our conclusion that abdominal obesity is not a significant predictor of plasma TG concentration could be attributed to our use of a surrogate estimate of abdominal obesity, WC, rather than a direct measure of visceral obesity. This latter possibility might explain the lack of a significant relationship in Fig. 1 between WC and SSPG concentrations, but it seems less likely to account for the findings in Tables 1 and 2. Despite having similar values for WC, the insulin-resistant third of the population had significant differences in many aspects of lipoprotein metabolism compared with the most insulin sensitive third, including higher TG and lower HDL-C concentrations. To attribute all of these differences in lipoprotein metabolism to variations in visceral fat volume, it would be necessary to postulate that there was a substantial increase in the visceral fat/subcutaneous fat ratio in insulin-resistant compared with the insulin-sensitive individuals. Although this possibility cannot be ruled out, it seems unlikely given that the 2 groups were not different in ethnicity, age, sex, and overall obesity, and had identical values for WC. Furthermore, there is published evidence that measurements of WC and visceral fat are highly correlated in women ($r = 0.87$) [14]. Thus, it seems more likely that our inability to detect a relationship between TG and WC in South Asian Indian women, compared with the findings of Banerji et al in men [6], is more likely due to gender differences, rather than that WC is an inadequate marker of visceral fat content.

It must be acknowledged that our findings are also in conflict with the notion of a "hypertriglyceridemic waist," as popularized by Pouliot et al [15], Lemieux et al [16,17], and St-Pierre et al [18]. The overall thrust of these and related manuscripts is that an increase in visceral adiposity is the cause of insulin resistance, hyperinsulinemia, and an atherogenic lipoprotein profile similar to that of the insulin-resistant women in our study, and increased risk of CVD and type 2 diabetes mellitus. It would be inappropriate in the context of this manuscript to discuss in detail all of the contributions of these authors, but a few comments are necessary. In the first place, to the best of our knowledge, their studies have not focused on South Asian Indians. Second, fasting hyperinsulinemia has been used as a surrogate estimate of insulin resistance in these studies, a measurement that accounts for only approximately one third of the variability in actual measurements of IMGU [19]. Finally, in most instances WC was used as a marker of visceral obesity, similar to our study. Thus, our conclusion in South Asian Indian women, in whom we have used a specific technique to measure IMGU, that insulin resistance, not abdominal obesity, accounts for a high TG and low HDL-C, cannot be dismissed when put into the context of the notion of a hypertriglyceridemic waist.

Finally, the hsCRP and Lp(a) results deserve some comment. It has been suggested that inflammatory changes may contribute to the development of insulin resistance [20], and it is clear from the current study that despite a more than 2-fold increase in SSPG concentration that the hsCRP concentrations in the insulin-resistant group were not different from the values in insulin-sensitive subjects. On the other hand, Lp(a) concentrations were higher in insulin-sensitive individuals, and this observation is consistent with the previous report of a negative correlation between Lp(a) and estimate of insulin resistance in South Asians; in contrast, there was no association between Lp(a) and insulin resistance in whites [21]. Furthermore, there is also evidence that Lp(a) levels are higher in South Asian Indians when compared to whites [22]. The heterogeneity of this genetic risk factor among different ethnic groups deserves further study.

In conclusion, the results of this study demonstrate that the presence of insulin resistance in South Asian Indian women is significantly associated with a highly atherogenic lipoprotein profile, consisting of higher TG concentrations, lower concentrations of HDL-C and HDL-C subclasses, and smaller and denser LDL particles. These changes appear to be independent of differences in WC, and women who are equally abdominally obese can have widely divergent lipoprotein profiles.

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