Published in final edited form as:
Diab Vasc Dis Res. 2019 March ; 16(2): 153–159. doi:10.1177/1479164118813890.

Impact of race/ethnicity on insulin resistance and hypertriglyceridaemia

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Abstract

Objective: Insulin sensitivity affects plasma triglyceride concentration and both differ by race/ethnicity. The purpose of this study was to provide a comprehensive assessment of the variation in insulin sensitivity and its relationship to hypertriglyceridaemia between five race/ethnic groups.

Research design and methods: In this cross-sectional study, clinical data for 1025 healthy non-Hispanic White, Hispanic White, East Asian, South Asian and African American individuals were analysed. Insulin-mediated glucose disposal (a direct measure of peripheral insulin sensitivity) was measured using the modified insulin suppression test. Statistical analysis was performed using analysis of co-variance.

Results: Of the study participants, 63% were non-Hispanic White, 9% were Hispanic White, 11% were East Asian, 11% were South Asian and 6% were African American. Overall, non-Hispanic Whites and African Americans displayed greater insulin sensitivity than East Asians and South Asians. Triglyceride concentration was positively associated with insulin resistance in all groups, including African Americans. Nevertheless, for any given level of insulin sensitivity, African Americans had the lowest triglyceride concentrations.

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*Deceased, 12 February 2018.

Declaration of conflicting interests
The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Supplemental material
Supplemental material for this article is available online.

Conclusion: Insulin sensitivity, as assessed by a direct measure of insulin-mediated glucose disposal, and its relationship to triglyceride concentration vary across five race/ethnic groups. Understanding these relationships is crucial for accurate cardiovascular risk stratification and prevention.

Keywords

Insulin resistance; race; triglyceride; modified insulin suppression test

Introduction

The risk of cardiometabolic conditions including type 2 diabetes, dyslipidemia and atherosclerotic cardiovascular disease varies across race/ethnic groups.[1–4] While the reasons for this variability are not completely understood, it has been postulated that underlying differences in insulin sensitivity[5–8] may be partly responsible. Insulin sensitivity varies widely in apparently healthy individuals.[9–13] Although variability in insulin action does not seem to be unique to any given race/ethnic group, the degree of insulin sensitivity does appear to vary with race/ethnicity, at least in small studies mostly utilizing surrogate estimates of insulin sensitivity.[9,10,14,15] Decreased insulin sensitivity (insulin resistance) is also associated with increases in plasma triglyceride (TG) concentration, an emerging causal risk factor for atherosclerotic cardiovascular disease,[16,17] and there is evidence that the association between insulin resistance and TG levels may also vary as a function of differences in race/ethnicity.[18–20] Plasma TG levels may not only reflect insulin sensitivity but are likely to mediate negative consequences of insulin resistance. Given the importance of insulin resistance and compensatory hyperinsulinemia in the pathogenesis of cardiometabolic diseases, we sought to provide a more comprehensive evaluation of the impact of differences in race/ethnicity on insulin resistance and its associated dyslipidemia. This work differs from the many manuscripts that have addressed this issue in the past for three crucial reasons: (1) data are available from five different race/ethnic groups; (2) insulin resistance was quantified by a direct measurement of insulin-mediated glucose disposal, not a surrogate estimate; and (3) the groups were compared not only on their degree of insulin resistance but also on the relationship between insulin resistance and its closest lipid consequence – changes in plasma TG concentration.

Methods

Study design and patient population

Data for this cross-sectional study were obtained from a database containing clinical information for individuals who have previously participated in research studies at Stanford University (Stanford, CA) between 1991 and 2014. To be included, individuals had to have no anaemia (haemoglobin <10 g/dL) or cardiovascular, kidney or liver disease. Subjects were excluded if they had a history of diabetes as defined as fasting glucose ⩾126 mg/dL by the American Diabetes Association[21] or were taking medications that could affect carbohydrate metabolism. All individuals were categorized through self-identification by the following race/ethnic groups: non-Hispanic White, Hispanic White, South Asian, East Asian and African American.

Measurements

All procedures were performed in the Stanford General Clinical Research Center after fasting for 12 h. Subjects had body weight and height measured for calculation of body mass index (BMI; in kg/m^2). Plasma glucose was determined by the oxidase method (Analyzer 2; Beckman, Brea, CA). Lipoprotein concentrations were performed in the core laboratory at Stanford by standardized methods approved by the Centers for Disease Control and Prevention. Low-density lipoprotein cholesterol (LDL-C) concentration was calculated (except for 26 subjects whose LDL-C could not be calculated due to a TG concentration >400 mg/dL).

Insulin-mediated glucose disposal was measured using the modified insulin suppression test.[22,23] After an overnight fast, individuals were administered a continuous infusion of octreotide acetate (0.27 $\mu g/m^2/min$), insulin (32 $mU/m^2/min$) and glucose (267 $mg/m^2/min$). Blood was sampled every 30 min until steady-state plasma glucose (SSPG) and steady-state plasma insulin (SSPI) levels were achieved. From 150 to 180 min of the infusion, blood was sampled at 10-min intervals. These final four results were used to determine SSPG and SSPI concentrations for each individual. Because octreotide acetate was used to inhibit endogenous secretion of insulin, each subject had a similar SSPI concentration. Therefore, the SSPG concentration for each individual represented an estimate of the ability of insulin to mediate disposal of infused glucose – that is, higher SSPG concentration reflected greater degree of peripheral insulin resistance.

Statistical analysis

Continuous variables were analysed using analysis of co-variance (ANCOVA). For racial/ethnic differences in insulin sensitivity, the Bonferroni correction was used for seven post hoc pairwise comparisons (non-Hispanic White vs Hispanic White, South Asian, East Asian and African American groups and African American vs Hispanic White, South Asian and East Asian groups). These specific comparisons were chosen a priori to focus on the groups that were most likely to differ based on prior literature and to decrease the number of tests. Categorical variables were analysed using the chi-squared test. Linear regression was performed to examine the relationship between insulin resistance and TG, LDL-C and high-density lipoprotein cholesterol (HDL-C) levels, again using the Bonferroni correction for the seven pairwise comparisons outlined above when appropriate. TG values were log-transformed to improve normality of distribution. For all statistical analyses, a two-sided p value $\leqslant 0.05$ was considered statistically significant. All analyses were done using SPSS (Version 24.0, IBM Corp., Armonk, NY). Results are expressed as means with standard deviations and count frequencies with percentages unless otherwise specified.

Results

Baseline characteristics

Table 1 summarizes the baseline characteristics of the study population by race/ethnicity and sex. There were 1025 individuals, of whom $n = 646$ (63%) were non-Hispanic White, $n = 91$ (9%) were Hispanic White, $n = 118$ (11%) were South Asian, $n = 109$ (11%) were East Asian and $n = 61$ (6%) were African American. Women comprised 60% of the total sample

population, and there was a similar sex distribution across all race/ethnic groups ($p = 0.1$). Only 11.8% of study participants were being treated with anti-hyperlipidemic agents. Of those, 93% were on statins, 6% on fibrates and 1% on ezetimibe.

Insulin resistance by race/ethnicity

The unadjusted SSPG levels for males and females in all five race/ethnic groups are provided in Table 2. After adjustments for age, sex and BMI, non-Hispanic Whites and African Americans had significantly lower SSPG levels (i.e. higher insulin sensitivity) than their South Asian and East Asian counterparts (Table 3).

Relationship between insulin resistance and TG, LDL-C and HDL-C concentration by race/ethnicity

Linear regression was used to determine the relationship between insulin resistance and natural log-transformed TG levels along with LDL-C and HDL-C levels after adjustments for age, sex and BMI. For all five groups, there was a significant, positive relationship between SSPG and TG levels on the log scale. Additionally, for a given level of insulin sensitivity, African Americans tended to have the lowest TG levels across all five race/ethnic groups, although these differences were only significant compared to non-Hispanic Whites (Table 4). There was no significant relationship between SSPG and LDL-C levels. While there was a significant, negative relationship between insulin resistance and HDL-C levels for all five groups, there was no race/ethnic difference in HDL-C level at a given level of insulin sensitivity. The statistical relationships between insulin resistance and natural log-transformed TG levels along with LDL-C and HDL-C levels remained the same after exclusion of patients on anti-hyperlipidemic agents, as demonstrated for TG levels in Supplemental Table 1.

The effect of race/ethnicity on log-transformed TG levels was estimated to be TG = e(3.913 + 0.002 × age – 0.00 4 × BMI + 0.003 × SSPG + 0.244 × sex [–0.257 if African American] OR [–0.011 if East Asian] OR [–0.097 if Hispanic White] OR [–0.026 if South Asian]).

Numeric values for sex are defined as ‘1’ if female and ‘2’ if male. Non-Hispanic White race is the reference group. As an example, the estimated TG concentration for a 50-year-old African American female with a BMI of 28 kg/m^2 and an SSPG level of 175 mg/dL and her equivalent non-Hispanic White counterpart would be 83 and 107 mg/ dL, respectively. Alternatively, a TG concentration of 150 mg/dL in a non-Hispanic White male would be equivalent to 116 mg/dL in an African American male of similar age, BMI and level of insulin resistance.

Discussion

In this study, we analysed the differences in insulin resistance and its relationship to hypertriglyceridaemia in 1025 healthy individuals from five race/ethnic groups using an intravenous, direct measure of insulin sensitivity. After adjusting for factors known to affect insulin resistance including BMI, we found that non-Hispanic Whites and African Americans had a similar degree of insulin resistance which was lower than that seen in their South Asian and East Asian counterparts. Additionally, for all five groups, there was a

positive relationship between insulin sensitivity and TG levels, although for a given level of insulin resistance, African Americans had lower TG concentrations than other race/ethnic groups.

While previous studies have examined variations in insulin sensitivity by race/ethnicity, they have been limited by the usage of surrogate measures of insulin sensitivity and/or comparison of only two to three race/ethnic groups.[9,14,15] The few studies that have used precise, quantitative measures of insulin-mediated glucose disposal have been carried out in considerably smaller sample sizes ($n < 44$) than the present[10] because direct measurements are costly, time-intensive and moderately invasive.

This analysis helps to confirm some prior findings. Our results in South Asians are similar to those reported by Raji et al.[24] and Laws et al.[10] In the former study, the authors found that 12 healthy South Asians had reduced glucose disposal rates (4.7 ± 0.4 vs 7.5 ± 0.3 mg/kg/min, $p < 0.0001$) based on the euglycemic-hyperinsulinemic clamp compared to age- and BMI-matched non-Hispanic Whites. In the latter study, the authors showed that 22 South Asian men and women had 60% higher SSPG levels based on the modified insulin suppression test than an equal number of men and women of European ancestry matched by age and BMI. The magnitude of difference in SSPG levels in this study was higher than ours (which was around 25%); however, their sample size was much smaller (22 vs 118 in ours). These findings are also in broad agreement with studies from Kanaya et al.[15] that estimated insulin resistance using the homeostasis model assessment of insulin resistance (HOMA-IR). They reported that South Asian individuals had a significantly higher degree of insulin resistance compared to African American, Hispanic White and non-Hispanic White individuals when adjusted for age, sex, BMI, waist circumference, smoking and alcohol use.

For other comparisons, however, our results may help arbitrate between prior results and explain discrepant findings when insulin sensitivity is directly measured versus estimated. In particular, there has been some disagreement in the literature about the degree of insulin sensitivity in African or African American populations. In a small study conducted by Goedecke et al.,[25] 15 Black South African women were noted to have the same degree of peripheral insulin sensitivity as 15 White South African women as measured by the euglycemic-hyperinsulinemic clamp. Pisprasert et al.[13] also showed that African American individuals had similar glucose disposal rates compared to Europeans as measured by the euglycemic-hyperinsulinemic clamp (which is closely correlated to the modified insulin suppression test[26,27]). Nevertheless, some studies have shown that surrogate estimates of insulin resistance may be higher in African Americans compared to their European counterparts. Haffner et al.[14] published results on the difference in insulin sensitivity between non-Hispanic Whites and African Americans as measured by the insulin sensitivity index obtained using a frequently sampled intravenous glucose test. In that study, African American subjects were reported to be more insulin resistant than non-Hispanic White subjects after adjustments for age, sex and BMI. This discrepancy may reflect differences in methodology. In that vein, despite having similar levels of insulin sensitivity by euglycemic-hyperinsulinemic clamp, Pisprasert and colleagues showed that African Americans appeared more insulin resistant when assessed by insulin sensitivity index, HOMA-IR and fasting insulin level.[13] Although our statistical power was somewhat limited, we found no

difference in insulin sensitivity between African American and non-Hispanic White volunteers which, when taken in context with other available data from reference-based measures, suggest that caution should be used in broadly assessing African American populations as insulin resistant.

Overall, these studies highlight a broader issue of reliability for the use of surrogate measures of insulin resistance. In 490 non-diabetic volunteers, Yeni-Komshian et al.[12] studied the accuracy of several surrogate measures of insulin resistance compared to the modified insulin suppression test as a gold standard. They found that the total integrated insulin response to a 75 g oral glucose challenge (OGTT) was the most closely related to the modified insulin suppression test with a Pearson's correlation coefficient of 0.67, which meant that the total integrated insulin response to 75 g OGTT could only account for ~45% of the variability in true insulin resistance. Other surrogate measures of insulin sensitivity such as fasting insulin, fasting glucose/fasting insulin and HOMA-IR were found to have even lower correlation coefficients (0.61, −0.42 and 0.62, respectively). Consistent with this, Ingelsson and colleagues also found that correlations of various surrogate measures of insulin sensitivity based on fasting measures or OGTT with gold standard intravenous insulin sensitivity analyses are generally below 0.7.[28] These observations clearly demonstrate the limitations of using surrogate measures of insulin sensitivity to study race/ethnic differences in insulin resistance.

For a given level of insulin resistance, we found that African Americans have lower TG levels than non-Hispanic Whites. This is consistent with results of prior population-based studies that have shown that African American individuals tend to have lower TG concentrations than their non-Hispanic White counterparts.[29,30] Sumner and Cowie[18] also found that African Americans with insulin resistance defined by HOMA-IR had lower TG levels than comparable non-Hispanic and Hispanic Whites. Because of lower TG levels, African Americans were less likely to meet criteria for metabolic syndrome than their age-, sex- and BMI-matched non-Hispanic and Hispanic White counterparts.[31] In part, this difference in TG levels has been hypothesized to be due to increased lipoprotein lipase activity.[32] Despite these findings, African Americans are known to have higher cardiovascular risk than non-Hispanic Whites,[33] raising the concerns that the criteria commonly used for metabolic syndrome may underestimate cardiovascular risk when using standardized cutoffs without consideration of race/ethnicity. Quantification of the differences in TG concentration between race/ethnicity, as is done in this analysis, can help guide accurate risk estimation for metabolic syndrome and subsequent cardiovascular risk.

There are several limitations to this study. First, we had small sample sizes for a few race/ethnic groups, which limits our power to identify all statistically significant differences between groups. Second, we did not have data on physical activity, alcohol use and waist circumference, which may confound the SSPG and TG differences between race/ethnic groups.

Conclusion

Insulin sensitivity and its relationship to TG concentration varies among the five examined race/ethnic groups. Non-Hispanic Whites and African Americans have greater insulin sensitivity, as assessed by a direct measure of insulin-mediated glucose disposal, than other race/ethnic groups. Furthermore, at a given level of insulin resistance, African Americans have lower TG concentrations than non-Hispanic Whites. Nevertheless, there was a significant, positive relationship between TG and insulin resistance showing that TG levels do increase with worsening insulin resistance in African Americans, as with other race/ethnic groups. Understanding these differences is critical for assessing and mitigating cardiovascular risk, particularly in high-risk race/ethnic groups.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

V.R. performed the data analysis and wrote the manuscript. F.A. provided the data, assisted with data analysis and reviewed the manuscript. L.L. assisted with data analysis. G.R. contributed to the introduction and contributed to the research design. S.K. and E.I. provided critical review of the manuscript. J.W.K. contributed to the research design and reviewed and revised the manuscript. All authors agree with the contents of this manuscript.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Stanford Diabetes Research Center, P30DK116074.

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Table 1.

Baseline characteristics by race/ethnicity and sex.

	Non-Hispanic White	Hispanic White	South Asian	East Asian	African American	*p* value
N						
Male	273	35	34	42	23	-
Female	373	56	84	67	38	-
Age (years)						
Male	51 ± 10	46 ± 10	47 ± 11	51 ± 9	47 ± 8	<0.01
Female	52 ± 10	48 ± 9	41 ± 11	46 ± 12	48 ± 9	<0.01
BMI (kg/m^2)						
Male	29.9 ± 4.4	32.8 ± 5.6	27.0 ± 4.3	28.1 ± 3.7	30.8 ± 5.0	<0.01
Female	29.6 ± 5.3	32.6 ± 7.5	28.0 ± 3.9	28.7 ± 5.6	33.3 ± 7.9	<0.01
TC (mg/dL)						
Male	190 ± 44	185 ± 39	198 ± 41	201 ± 48	174 ± 38	0.13
Female	199 ± 36	197 ± 32	175 ± 35	202 ± 42	184 ± 33	<0.01
TG[a] (mg/dL)						
Male	168 ± 11	158 ± 29	194 ± 30	195 ± 27	112 ± 36	0.03
Female	126 ± 5	122 ± 14	114 ± 11	151 ± 12	100 ± 16	0.12
HDLC (mg/dL)						
Male	42 ± 11	39 ± 9	39 ± 9	41 ± 13	43 ± 13	0.37
Female	53 ± 14	50 ± 15	48 ± 11	52 ± 14	49 ± 15	0.02
LDLC (mg/dL)						
Male	117 ± 36	115 ± 28	118 ± 28	130 ± 50	109 ± 34	0.19
Female	121 ± 31	122 ± 24	104 ± 28	122 ± 33	115 ± 31	<0.0l
FPG (mg/dL)						
Male	99 ± 10	103 ± 9	95 ± 12	101 ± 10	97 ± 8	0.01
Female	97 ± 10	96 ± 8	94 ± 9	98 ± 9	96 ± 10	0.11

BMI: body mass index; TC: total cholesterol; TG: triglyceride; HDLC: high-density lipoprotein cholesterol; LDLC: low-density lipoprotein cholesterol; FPG: fasting plasma glucose.

Data are shown as mean ± standard deviation. The value of $p \leqslant 0.05$ is considered statistically significant.

[a]Statistical analysis was performed on natural log-transformed TG values.

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Table 2.

SSPG by sex and race/ethnicity (unadjusted).

	Non-Hispanic White	Hispanic White	South Asian	East Asian	African American	*p* value
Male	154 ± 4	204 ± 12[a,b]	183 ± 12	166 ± 11	141 ± 14	<0.01
	(146, 163)	(181, 226)	(160, 206)	(145, 187)	(113, 169)	
Female	150 ± 4	179 ± 10[a]	167 ± 8	186 ± 9[a]	176 ± 12	<0.01
	(142, 157)	(160, 199)	(151, 183)	(168, 204)	(152, 199)	

SSPG: steady-state plasma glucose; SE: standard error.

Data shown as mean ± SE and 95% confidence interval (in parentheses). SSPG values are given in mg/dL. The value of $p \leqslant 0.05$ is considered statistically significant.

[a]Statistically significant difference compared to non-Hispanic White individuals.

[b]Statistically significant difference compared to African American individuals.

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Table 3.

SSPG by race/ethnicity (adjusted for age, sex and BMI).

Non-Hispanic White	Hispanic White	South Asian	East Asian	African American	*p* value
151 ± 2	169 ± 7	189 ± 6[a,b]	188 ± 6[a,b]	145 ± 8	<0.01
(147, 156)	(156, 182)	(177, 201)	(176, 199)	(129, 160)	

SSPG: steady-state plasma glucose; BMI: body mass index; SE: standard error.

Data shown as mean ± SE and 95% confidence interval (in parentheses). SSPG values are given in mg/dL. Covariates in the model are evaluated at the following values: age, 49 years; sex, 1.4 (where sex = '1' if female and '2' if male); and BMI, 29.8 kg/m2. The value of $p \leqslant 0.05$ is considered statistically significant.

[a]Statistically significant difference compared to non-Hispanic White individuals.

[b]Statistically significant difference compared to African American individuals.

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Table 4.

TG level by race/ethnicity (adjusted for age, sex, SSPG and BMI).

Non-Hispanic White	Hispanic White	South Asian	East Asian	African American	*p* value
148 ± 5	124 ± 14	131 ± 13	157 ± 136	107 ± 17[a]	<0.01
(137, 158)	(97, 152)	(106, 156)	(132, 182)	(74, 140)	

TG: triglyceride; SSPG: steady-state plasma glucose; BMI: body mass index; SE: standard error.

Data shown as mean ± SE and 95% confidence interval (in parentheses). TG values are displayed in mg/dL; statistical analysis performed on naturallog transformed TG levels. Covariates in the model are evaluated at the following values: age, 49 years; sex, 1.4 (where sex = '1' if female and '2' if male); SSPG, 161 mg/dL; and BMI, 29.8 kg/m2. The value of $p \leqslant 0.05$ is considered statistically significant.

[a]Statistically significant difference compared to non-Hispanic White individuals.