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Hostility and Fasting Glucose in African American Women

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Abstract

Objective—To examine whether the relationship of hostility (HOST) to fasting glucose indices is moderated by sex and race. HOST has been associated with abnormalities in glucose metabolism. Prior studies suggested that this association may be more prevalent in women and in African American (AA) individuals.

Methods—A total of 565 healthy AA and white (W) men and women (mean age = 33 ± 6 years) were assessed. HOST was measured by the 27-item version of the Cook Medley HOST Scale. The moderating effects of sex and race were evaluated for the associations of HOST to fasting glucose, insulin, and insulin sensitivity (HOMA-IR).

Results—Analysis showed a moderating effect of sex and race on the association of HOST to fasting glucose ($p = .03$), but not for insulin ($p = .12$). Analysis of HOMA-IR revealed a trend ($p = .06$) for the interaction. Stratified analyses by race and sex revealed a positive association between HOST and fasting glucose only in AA women, which remained significant after controlling for age and body mass index.

Conclusion—A relationship between HOST and fasting glucose was evident in AA women only, a group that has twice the risk of developing Type 2 diabetes compared with W women. Further studies are needed to elucidate the mechanisms by which HOST may affect glucose metabolism in AA women.

Keywords

hostility; African American; women; glucose; insulin

INTRODUCTION

Hostility (HOST) is a personality trait that is known to be a risk factor for cardiovascular disease (CVD) (1), although the mechanism has not been established. Several groups have found that HOST is related to parameters of glucose metabolism in nondiabetic individuals (2–6). In addition, HOST has recently been found to prospectively predict impaired fasting glucose levels and Type 2 diabetes mellitus (T2DM) (7). In a recent meta-analysis (8), Bunde and Suls concluded that HOST is significantly related to glucose levels and insulin resistance, whereas the association to fasting insulin is less consistent. Recent evidence suggested that the relationship between HOST and indices of glucose metabolism seems to

be more pronounced in women and in African American (AA) individuals suggesting that race and sex could moderate the HOST to glucose relationship. We have shown HOST to be significantly related to fasting glucose among AAs but not among white (W) men and women (5,9). Other studies have also found race differences in the relationship of HOST to glucose metabolism. For example, a study of young college students found a stronger association between HOST and fasting insulin in AAs than in Ws (10). We have also observed the association between HOST and glucose indices to be present in women but not men (5), a finding that has been replicated in other studies (11,12). Together, these results suggested that AA women may be especially susceptible to the deleterious effects of HOST on glucose metabolism. However, no previous study has examined the moderating effect of both sex and race on the association of HOST to glucose metabolism. The aim of the present study was thus to explore the moderating effects of race and sex in the association of HOST to fasting glucose, insulin, and insulin resistance.

METHODS

Participants

The study sample consisted of a combination of two very similar, but independently recruited groups of individuals. Both studies made specific efforts to recruit comparable proportions of AA men, AA women, W men, and W women, and included only healthy, nondiabetic subjects. As stated in our earlier paper (5), ethnicity was self-reported.

The first sample was recruited between 1998 and 2001 and was comprised of 165 participants (42 AA women, 32 W women, 39 W men, and 52 AA men; age range = 20–48 years; mean age = 34 ± 8.5 years). Participants were recruited via advertisements in the local media, fliers posted in supermarkets and other public locations, and outreach at public events (13). Preliminary results exploring race and sex effects on the association between HOST and fasting glucose indices have been presented on a subsample of 98 individuals (5). The second sample was recruited between 2004 and 2007. It consisted of 400 healthy individuals ($n = 101$ AA women, 118 W women, 99 W men, and 82 AA men; age range = 25–45 years, mean age = 33 ± 6.0 years), who were recruited through flyers distributed in and around Duke University Medical Center, and through local advertisements in the Durham community.

Participants in both studies were required to have normal fasting glucose (<126 mg/dl), be able to read and write English, and be willing to provide their informed consent for participation. Participants were excluded who had medical conditions such as diabetes or a known liver, kidney or endocrinological dysfunction; history of substance abuse in the past year; history of stroke or significant head injury; terminal illness; current use of psychotropic medications or medications that could affect glucose metabolism (e.g., steroids, narcotics); and current pregnancy or breastfeeding.

Procedures

All participants were asked to refrain from eating or drinking after midnight and to avoid smoking after 8 PM on the day preceding the data collection. The physiological assessment incorporated an anthropometric evaluation including height and weight measurements. Blood samples were drawn in the morning, between 8 and 9 AM, to assess fasting insulin and glucose. One subject with evident T2DM (fasting glucose >126 mg/dl) was excluded from the data analysis. Both studies were approved by the Institutional Review Board at the Duke University Medical Center and informed consent was obtained from all participants.

Measures

HOST—HOST was measured using the 27-item version of the Cook Medley HOST Scale (14), which characterizes an attitude of cynicism and mistrust (15). Therefore, high HOST scores primarily reflect a cognitive predisposition to perceive threat with little emphasis on antagonistic emotions and behavioral tendencies. This hostile attitude does tend to lead to stressful interpersonal experiences and conflicts (16). Scores on the Cook Medley HOST Scale were found to be a better predictor of long-term health outcomes than scores for the original 50-item scale (1).

Glucose, Insulin, and Insulin Sensitivity

Plasma glucose concentration was run by a hexokinase method. Plasma insulin concentration was determined by radioimmunoassay (Millipore, Billerica, Massachusetts). Fasting insulin sensitivity was estimated using the Homeostatic Model Assessment (HOMA) (17). Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the formula: $\text{HOMA-IR} = \text{fasting glucose (mg/dl)} \times \text{fasting insulin } (\mu\text{U/ml})/405$.

Body Mass Index (BMI)

BMI was calculated as $\text{BMI} = \text{weight (kg)} / \text{height (m)}^2$.

Statistical Analysis

Positive skewness was evident in the distribution of values for both fasting insulin and HOMA-IR. Values for both variables were subjected to natural log (ln) transformations to achieve appropriate distributional properties for linear regression analysis.

Univariate general linear modeling was used to test for differences in HOST and between the samples as well as to test for differences in study variables between the race and sex groups. Moderating effects of race and sex on the HOST to glucose measures were tested, using multiple linear regression analysis. Regression models included the three experimental factors (HOST, race, and sex) and incorporated age as a covariate. Lower-order interactions (two-way interactions) were examined when the three-way interactions were not significant. In addition, stratified regression analyses by sex and race are presented for fasting glucose, insulin, and HOMA-IR. Because age and BMI have been associated to HOST (18,19) and BMI has been related to the glucose metabolism (20,21), the stratified regression analysis of the HOST to glucose and insulin associations are presented adjusted for age and in a second step adjusted for both age and BMI. Statistical analyses were performed using SPSS (version 15.0 for Windows, SPSS Inc., Chicago, Illinois).

Pooling of Study Samples

Comparisons of the two samples revealed no significant differences in mean age (33.4 ± 6.0 versus 34.0 ± 8.5 , $p = .36$) or Cook Medley HOST scores (9.6 ± 5.4 versus 10.1 ± 5.1 , $p = .31$). There was no evidence of a moderating effect of study sample on the HOST by sex by race interactions on glucose indices, with a four-way interaction analysis (sample $\times$ HOST $\times$ race $\times$ sex) testing the study effects on glucose, lninsulin, and lnHOMA-IR showing no significant interaction effect for glucose ($p = .92$), lninsulin ($p = .77$) or lnHOMA-IR ($p = .83$). Thus, the data from these two samples were pooled for subsequent analyses.

RESULTS

Sample Characteristics

Subject characteristics by sex and race groups are summarized in Table 1. The groups did not differ significantly in age. There were main effects of both sex and race for HOST with

higher scores among men as compared with women ($p < .001$), and among AAs as compared with Ws ($p < .001$). This is congruent with findings from population samples (18). There was a sex by race interaction for BMI ($p = .006$), with AA women having significantly higher BMI as compared with the other three groups.

For glucose indices, there was a main effect of sex on fasting glucose, with men having significantly higher fasting glucose levels as compared with women ($p < .001$). This result is consistent with previous findings of sex differences in relation to fasting glucose (22). In addition, there was a sex by race interaction for fasting insulin levels ($p = .02$) as well as HOMA-IR ($p = .04$) with AA women having higher fasting insulin levels and HOMA-IR compared with all other groups.

Moderation by Race and Sex on the HOST to Fasting Glucose Indices Association

Age-adjusted models found a significant $\text{HOST} \times \text{race} \times \text{sex}$ interaction for fasting glucose ($p = .03$), but not for log-transformed ($\ln$) fasting insulin ($p = .12$). Analysis of $\ln\text{HOMA-IR}$ revealed a trend ($p = .06$) for the three-way interaction. Tests of the two-way interactions ($\text{HOST} \times \text{race}$ and $\text{HOST} \times \text{sex}$) for $\ln\text{insulin}$ and $\ln\text{HOMA-IR}$ were not significant ($p > .20$).

Stratified regression analysis controlling for age revealed a significant positive association between HOST scores and fasting glucose, $\ln\text{insulin}$, and $\ln\text{HOMA-IR}$ in AA women but not in the other groups (Table 2). Additional adjustment for BMI reduced the magnitude of the associations of HOST to insulin and HOMA-IR. This finding was expected, considering the close association of adiposity to insulin resistance (23). However, the relationship between HOST and fasting glucose remained significant even after adjustment for BMI in the AA women (Table 2).

DISCUSSION

Previous research suggested that the observed relationship between HOST and fasting glucose might be stronger in women and in AAs (5,9–11). The present study included a large sample that was balanced in terms of sex and race, and extends earlier findings by examining the combined moderating effects of race and sex. Results showed that a positive relationship between HOST and fasting glucose is present only in AA women, and is apparent independently in each of the samples that were pooled as well (data not presented). This effect is evident even after controlling for BMI and age. Whereas other studies have reported HOST to be associated with fasting glucose in samples including AA and W men and women (5,11,12), these studies were not designed to test for a sex by race interaction. Our results clearly showed that this effect is stronger in AA females than in these other groups. With both population-based epidemiological studies and national surveys showing that HOST is higher in AAs than in Ws (24,25), there is evidence suggesting that a substantial portion of AA women run the risk of being exposed to the detrimental effects of elevated HOST on glucose metabolism.

Our present finding is consistent with the notion that there is very little evidence of a consistent relationship between HOST, as measured by the Cook Medley Scale, and fasting glucose in men. In our previous study (5), the effect was reported in a mixed sample that was mainly female. After testing for a $\text{HOST} \times \text{gender}$ interaction in that study, it was evident that the HOST to fasting glucose effect was only present in women. However, the study lacked the power to test for a $\text{race} \times \text{gender} \times \text{HOST}$ interaction. Knox et al. (12) reported HOST and fasting glucose data on 2158 men and 2485 women. This study found an association of HOST to fasting glucose only in women with “high coronary artery disease risk.” There was no effect of HOST to glucose in the men, and no $\text{race} \times \text{gender}$ interaction

on the HOST to glucose effect was noted. Finally, Suarez (11) also found an effect of HOST to fasting glucose only in women, but did not test for a race $\times$ gender interaction. Thus, the present study confirms and extends previous findings by showing that the association of HOST to fasting glucose is particularly evident among AA women.

The apparent disproportionate effect of HOST on fasting glucose in AA women may be of clinical importance. AA women have an almost two-fold increased risk of T2DM as compared with their W counterparts (26,27). Although the effect size of HOST on fasting glucose in the present study is rather moderate, with HOST explaining about 5% of the variance in fasting glucose in these healthy AA women, the impact may still be clinically significant. Elevated fasting glucose levels even within the normal range greatly increases the risk for CVD morbidity (28) and mortality (29).

HOST has been shown to be a stable personality trait that is a risk factor for cardiovascular morbidity and mortality (16). However, the reliance on Cook Medley HOST as the primary personality measure may not have captured the full effects of HOST because of its emphasis on self-reports of cognitive processes. Other dimensions of HOST have been related to health outcomes as well. Hostility has also been related to depression and socioeconomic status, both of which have been shown to affect health outcomes (16). In addition, direct behavioral observation has been found to provide information about health risks that is not captured by self-reports (30). Therefore, broader assessments of antagonistic tendencies may produce evidence of more widespread impact of HOST on metabolic processes in this population. Finally, the present study cannot address the question of why AA women are particularly susceptible to the effects of HOST on glucose metabolism or whether this phenomenon represents genetic or sociocultural factors that differentiate AA women from AA men and W men and women.

HOST has previously been linked both to increased caloric intake (31) and to higher BMI scores (8), which suggests that increased adiposity could have been a plausible mediator of the relationship between HOST and fasting glucose in AA women in the present study. However, the association between HOST and fasting glucose remained significant even after adjusting for BMI. Nevertheless, these results do not exclude the possibility that adiposity plays a key role in the HOST-glucose relation for AA women. Greater waist circumference significantly increases the likelihood of multiple cardiovascular risk factors over and above BMI (32,33). In addition, it has been shown that abdominal fat, in particular visceral/omental adipose tissue, is the most sensitive to lipolytic stimulation (34). Preliminary results from a recent pilot study by our group showed that percent trunk fat (measured with dual X-ray densitometry technique), rather than BMI, mediated the HOST-glucose association in a different sample of AA women (35). Additional pathways in the link between HOST and glucose metabolism have been suggested, including increased adrenal medullary and cortical responding (6,36,37). Both epinephrine and cortisol are known to increase fasting glucose via effects on glycogenolysis and gluconeogenesis (38,39). Studies have also shown that AA women have increased catecholamine stimulated lipolysis (40) as well as increased β -receptor numbers in omental tissue (41) as compared with W women, potentially increasing free fatty acids that could disturb insulin sensitivity as well as glucose-stimulated insulin secretion (42).

In summary, the present study showed a significant positive association between HOST and fasting glucose among AA women that was not apparent in AA men and W men and women. This effect remained significant after adjusting for BMI and age. An additional study done by our group is presented in the current issue of the journal, which investigated the mechanisms by which the HOST to glucose metabolism association may be mediated in AA women.

Acknowledgments

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Glossary

AA	African American
BMI	body mass index
CVD	cardiovascular disease
HOMA-IR	homeostasis model assessment of insulin resistance
HOST	hostility
T2DM	Type 2 diabetes mellitus
W	white

References

1. Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG, Williams RB Jr. The Cook-Medley hostility scale: Item content and ability to predict survival. *Psychosom Med.* 1989; 51:46–57. [PubMed: 2928460]
2. Niaura R, Banks SM, Ward KD, Stoney CM, Spiro A 3rd, Aldwin CM, Landsberg L, Weiss ST. Hostility and the metabolic syndrome in older males: The normative aging study. *Psychosom Med.* 2000; 62:7–16. [PubMed: 10705906]
3. Raikkonen K, Keltikangas-Jarvinen L, Hautanen A. The role of psychological coronary risk factors in insulin and glucose metabolism. *J Psychosom Res.* 1994; 38:705–13. [PubMed: 7877125]
4. Vitaliano PP, Scanlan JM, Krenz C, Fujimoto W. Insulin and glucose: relationships with hassles, anger, and hostility in nondiabetic older adults. *Psychosom Med.* 1996; 58:489–99. [PubMed: 8902900]
5. Surwit RS, Williams RB, Siegler IC, Lane JD, Helms M, Applegate KL, Zucker N, Feinglos MN, McCaskill CM, Barefoot JC. Hostility, race, and glucose metabolism in nondiabetic individuals. *Diabetes Care.* 2002; 25:835–9. [PubMed: 11978677]
6. Zhang J, Niaura R, Dyer JR, Shen BJ, Todaro JF, McCaffery JM, Spiro A 3rd, Ward KD. Hostility and urine norepinephrine interact to predict insulin resistance: The VA normative aging study. *Psychosom Med.* 2006; 68:718–26. [PubMed: 17012525]
7. Shen BJ, Countryman AJ, Spiro A 3rd, Niaura R. The prospective contribution of hostility characteristics to high fasting glucose levels: the moderating role of marital status. *Diabetes Care.* 2008; 31:1293–8. [PubMed: 18460671]
8. Bunde J, Suls J. A quantitative analysis of the relationship between the Cook-Medley hostility scale and traditional coronary artery disease risk factors. *Health Psychol.* 2006; 25:493–500. [PubMed: 16846324]
9. Boyle, SH.; Surwit, RS.; Williams, RB.; Barefoot, JC. Hostility, race, and fasting glucose: The Vietnam experience study. Presented at the annual meeting of the Society of Behavioral Medicine; Baltimore, Maryland. 2004.
10. Cooper DC, Waldstein SR. Hostility differentially predicts cardiovascular risk factors in African American and white young adults. *J Psychosom Res.* 2004; 57:491–9. [PubMed: 15581654]
11. Suarez EC. Sex differences in the relation of depressive symptoms, hostility, and anger expression to indices of glucose metabolism in nondiabetic adults. *Health Psychol.* 2006; 25:484–92. [PubMed: 16846323]
12. Knox SS, Weidner G, Adelman A, Stoney CM, Ellison RC. Hostility and physiological risk in the National Heart, Lung, and Blood Institute family heart study. *Arch Intern Med.* 2004; 164:2442–8. [PubMed: 15596634]

13. Burroughs AR, Visscher WA, Haney TL, Efland JR, Barefoot JC, Williams RB Jr, Siegler IC. Community recruitment process by race, gender, and SES gradient: lessons learned from the Community Health and Stress Evaluation (CHASE) study experience. *J Community Health*. 2003; 28:421–37. [PubMed: 14620965]
14. Cook W, Medley D. Proposed hostility and pharisaic virtue scales for the MMPI. *J Appl Psychol*. 1954; 38:414–8.
15. Smith TW, Sanders JD, Alexander JF. What does the Cook and Medley hostility scale measure? Affect, behavior, and attributions in the marital context. *J Pers Soc Psychol*. 1990; 58:699–708. [PubMed: 2348365]
16. Smith TW, Glazer K, Ruiz JM, Gallo LC. Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *J Pers*. 2004; 72:1217–70. [PubMed: 15509282]
17. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985; 28:412–9. [PubMed: 3899825]
18. Barefoot JC, Peterson BL, Dahlstrom WG, Siegler IC, Anderson NB, Williams RB Jr. Hostility patterns and health implications: correlates of Cook-Medley hostility scale scores in a national survey. *Health Psychol*. 1991; 10:18–24. [PubMed: 2026126]
19. Siegler IC, Costa PT, Brummett BH, Helms MJ, Barefoot JC, Williams RB, Dahlstrom WG, Kaplan BH, Vitaliano PP, Nichaman MZ, Day RS, Rimer BK. Patterns of change in hostility from college to midlife in the UNC alumni heart study predict high-risk status. *Psychosom Med*. 2003; 65:738–45. [PubMed: 14508014]
20. Hu G, Lindstrom J, Valle TT, Eriksson JG, Jousilahti P, Silventoinen K, Qiao Q, Tuomilehto J. Physical activity, body mass index, and risk of type 2 diabetes in patients with normal or impaired glucose regulation. *Arch Intern Med*. 2004; 164:892–6. [PubMed: 15111376]
21. Wilson PW, Meigs JB, Sullivan L, Fox CS, Nathan DM, D'Agostino RB Sr. Prediction of incident diabetes mellitus in middle-aged adults: The Framingham offspring study. *Arch Intern Med*. 2007; 167:1068–74. [PubMed: 17533210]
22. Blaak E. Sex differences in the control of glucose homeostasis. *Curr Opin Clin Nutr Metab Care*. 2008; 11:500–4. [PubMed: 18542013]
23. Kahn BB, Flier JS. Obesity and insulin resistance. *J Clin Invest*. 2000; 106:473–81. [PubMed: 10953022]
24. Smith TW. Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychol*. 1992; 11:139–50. [PubMed: 1618168]
25. Shapiro D, Goldstein IB, Jamner LD. Effects of cynical hostility, anger out, anxiety, and defensiveness on ambulatory blood pressure in black and white college students. *Psychosom Med*. 1996; 58:354–64. [PubMed: 8827798]
26. Okosun IS. Racial differences in rates of type 2 diabetes in American women: How much is due to differences in overall adiposity? *Ethn Health*. 2001; 6:27–34. [PubMed: 11388083]
27. Brancati FL, Kao WH, Folsom AR, Watson RL, Szklo M. Incident type 2 diabetes mellitus in African American and white adults: The atherosclerosis risk in communities study. *JAMA*. 2000; 283:2253–9. [PubMed: 10807384]
28. Coutinho M, Gerstein HC, Wang Y, Yusuf S. The relationship between glucose and incident cardiovascular events. A metaregression analysis of published data from 20 studies of 95,783 individuals followed for 12.4 years. *Diabetes Care*. 1999; 22:233–40. [PubMed: 10333939]
29. Balkau B, Hu G, Qiao Q, Tuomilehto J, Borch-Johnsen K, Pyorala K. Prediction of the risk of cardiovascular mortality using a score that includes glucose as a risk factor. The DECODE study. *Diabetologia*. 2004; 47:2118–28. [PubMed: 15662552]
30. Barefoot, JC.; Boyle, SH. Hostility and anger. In: Leary, MR.; Hoyle, RH., editors. *Handbook of Individual Differences in Social Behavior*. New York: Guilford Press; 2009. p. 210–26.
31. Scherwitz LW, Perkins LL, Chesney MA, Hughes GH, Sidney S, Manolio TA. Hostility and health behaviors in young adults: The CARDIA study. Coronary artery risk development in young adults study. *Am J Epidemiol*. 1992; 136:136–45. [PubMed: 1415137]

32. Ghandehari H, Le V, Kamal-Bahl S, Bassin SL, Wong ND. Abdominal obesity and the spectrum of global cardiometabolic risks in US adults. *Int J Obes (Lond)*. 2009; 33:239–48. Epub 2008 Dec 9. [PubMed: 19050677]
33. Reeder BA, Senthilselvan A, Després JP, Angel A, Liu L, Wang H, Rabkin SW. The association of cardiovascular disease risk factors with abdominal obesity in Canada. *Canadian Heart Health Surveys Research Group. CMAJ*. 1997; 157(Suppl 1):S39–45. [PubMed: 9220953]
34. Arner P. Differences in lipolysis between human subcutaneous and omental adipose tissues. *Ann Med*. 1995; 27:435–8. [PubMed: 8519504]
35. Georgiades A, Surwit RS, Williams RB. Hostility and glucose indices in African American and Caucasian females: The mediating role of trunk fat. *Psychosom Med*. 2008; 70:A-69.
36. Pope MK, Smith TW. Cortisol excretion in high and low cynically hostile men. *Psychosom Med*. 1991; 53:386–92. [PubMed: 1924651]
37. Suarez EC, Kuhn CM, Schanberg SM, Williams RB Jr, Zimmermann EA. Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosom Med*. 1998; 60:78–88. [PubMed: 9492244]
38. Vicini P, Avogaro A, Spilker ME, Gallo A, Cobelli C. Epinephrine effects on insulin-glucose dynamics: The labeled IVGTT two-compartment minimal model approach. *Am J Physiol Endocrinol Metab*. 2002; 283:E78–84. [PubMed: 12067846]
39. Tonelli J, Kishore P, Lee DE, Hawkins M. The regulation of glucose effectiveness: How glucose modulates its own production. *Curr Opin Clin Nutr Metab Care*. 2005; 8:450–6. [PubMed: 15930973]
40. Bower JF, Vadlamudi S, Barakat HA. Ethnic differences in in vitro glyceride synthesis in subcutaneous and omental adipose tissue. *Am J Physiol Endocrinol Metab*. 2002; 283:E988–93. [PubMed: 12376326]
41. McConaughy MM, Sheets KA, Davis J, Privette J, Hickner R, Christian B, Barakat H. Differences in beta-adrenergic receptor densities in omental and subcutaneous adipose tissue from obese African American and Caucasian women. *Metabolism*. 2004; 53:247–51. [PubMed: 14767879]
42. Duez, H.; Lewis, GF. Fat metabolism in insulin resistance and type 2 diabetes. In: Feinglos, MN.; Bethel, MA., editors. *Type 2 Diabetes Mellitus: An Evidence-Based Approach to Practical Management*. Vol. 1. Durham, NC: Humana Press; 2008.

TABLE 1
Study Group Characteristics Stratified by Sex and Race (Mean $\pm$ Standard Deviation Values)

	African American		White		<i>p</i> for Sex $\times$ Race Interaction
	Women (<i>n</i> = 143)	Men (<i>n</i> = 134)	Women (<i>n</i> = 150)	Men (<i>n</i> = 138)	
Cook Medley HOST	10.5 $\pm$ 5.2	11.9 $\pm$ 0.5.5 *	7.1 $\pm$ 4.5	9.7 $\pm$ 4.9 **	.20
Age (years)	33.2 $\pm$ 7.1	33.0 $\pm$ 7.1	33.5 $\pm$ 6.5	34.5 $\pm$ 6.8	.33
BMI (kg/m ²)	29.2 $\pm$ 6.9	27.5 $\pm$ 5.2	25.6 $\pm$ 5.1	26.5 $\pm$ 4.2	.006
fasting glucose (mg/dl)	84.3 $\pm$ 8.5	86.0 $\pm$ 8.1 *	82.3 $\pm$ 6.8	87.6 $\pm$ 8.1	.07
Fasting insulin (μ U/ml)	16.0 $\pm$ 10.1	12.8 $\pm$ 9.1	12.8 $\pm$ 6.9	12.6 $\pm$ 8.1	.02
HOMA-IR	3.4 $\pm$ 2.5	2.8 $\pm$ 2.3	2.7 $\pm$ 1.6	2.8 $\pm$ 2.0	.04

* *p* < .001 for comparison between men and women;

** *p* < .001 for comparison between African American and white.

HOST = hostility; BMI = body mass index; HOMA-IR = homeostasis model assessment of insulin resistance.

TABLE 2

Results From Regression Analysis on the Association of HOST to Glucose, Insulin, and HOMA-IR, Stratified by Gender and Race

	<u>African American</u>		<u>White</u>	
	Women	Men	Women	Men
Fasting glucose				
Adjusted for age	0.22 **	−0.12	0.02	0.05
Adjusted for age and BMI	0.19 *	−0.06	0.01	0.04
lnfasting insulin				
Adjusted for age	0.17 *	−0.04	0.02	0.08
Adjusted for age and BMI	0.12	0.05	−0.03	0.04
lnHOMA-IR				
Adjusted for age	0.19 *	−0.06	0.02	0.09
Adjusted for age and BMI	0.13	0.04	−0.03	0.05

BMI = body mass index; HOMA-IR = homeostasis model assessment of insulin resistance; ln = natural log transformation.

*
 $p < .05$;

**
 $p < .006$.