

# Hostility, the Metabolic Syndrome, and Incident Coronary Heart Disease

Raymond Niaura, John F. Todaro, and Laura Stroud  
Brown Medical School and The Miriam Hospital

Avron Spiro III  
Boston University School of Public Health and Boston Veterans  
Affairs Healthcare System

Kenneth D. Ward  
University of Memphis

Scott Weiss  
Brigham and Women's Hospital and Harvard Medical School

This investigation examined the impact of hostility and the metabolic syndrome on coronary heart disease (CHD) using prospective data from the Normative Aging Study. Seven hundred seventy-four older, unmedicated men free of cardiovascular disease were included in the study. The total Cook–Medley Hostility (Ho) Scale score, anthropometric data, serum lipids, fasting insulin concentrations, blood pressure, cigarette smoking, alcohol consumption, and total dietary calories were used to predict incident CHD during a 3-year follow-up interval. Multivariate analysis indicated that only Ho positively predicted and high-density lipoprotein cholesterol level negatively predicted incident CHD. Ho's effects on CHD may be mediated through mechanisms other than factors that constitute the metabolic syndrome.

*Key words:* hostility, metabolic syndrome, coronary heart disease

Both cross-sectional and prospective studies have identified hostility as an independent psychosocial risk factor for coronary heart disease (CHD), hypertension, and premature mortality (Barefoot, Dahlstrom, & Williams, 1983; Barefoot, Larsen, von der Lieth, & Schroll, 1995; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; MacDougall, Dembroski, Dimsdale, & Hackett, 1985; Shekelle, Gale, Ostfeld, & Paul, 1983). A quantitative analysis investigating the relationship between hostility and physical health found that hostility measured by structured interview (weighted mean  $r = .17$ ) or by the Cook–Medley Hostility (Ho) Scale and other cognitive experiential measures (weighted mean  $r = .08$ ) were predictive of CHD (Miller, Smith, Turner, Guijarro, & Hallet, 1996). Although the magnitude of this relationship appears small, it remains clinically significant given

the prevalence of CHD and the number of CHD-related deaths each year (Booth-Kewley & Friedman, 1987).

Although high levels of hostility are clearly associated with increased risk of CHD, less is known about the process(es) by which hostility confers such risk. Hostility may influence health behaviors that themselves confer risk; hostility may be associated with sociodemographic characteristics that are, in turn, associated with increased CHD risk; and hostility may be associated with aberrations in physiological states that hasten atherosclerosis. Numerous studies support, at least in part, each of these suppositions.

Hostility appears to be elevated in non-White men and individuals with lower socioeconomic status (Barefoot et al., 1991). In addition, hostility has been found to be associated with unhealthy behaviors, including increased cigarette use (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Matthews, Glass, Rosenman, & Bortner, 1977), positive smoking status (Siegler, Peterson, Barefoot, & Williams, 1992), greater alcohol consumption (Houston & Vavak, 1991; Leiker & Hailey, 1988; Shekelle et al., 1983), and increased caloric intake (Scherwitz et al., 1992). The positive relationship between hostility and physiological risk factors for CHD, such as waist-to-hip ratio (WHR; Niaura et al., 2000; Scherwitz et al., 1992), body mass index (BMI; Houston & Vavak, 1991; Siegler et al., 1992), hypertension (Barefoot et al., 1983, 1989; Irvine, Garner, Craig, & Logan, 1991), total cholesterol (Dujovne & Houston, 1991; Lundberg, Hedman, Melin, & Frankenhauser, 1989; Weidner, Sexton, McLellarn, Conner, & Matarazzo, 1987), fasting insulin and glucose (Niaura et al., 2000; Vitaliano, Scanlan, Krenz, & Fujimoto, 1996), and the ratio of total cholesterol to HDL cholesterol (HDL-C; Siegler et al., 1992), have also been documented.

Unfortunately, these studies, most of them cross-sectional in design, do not tell researchers whether hostility's effect on CHD is mediated by its influence on any of these variables. Prospective

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Raymond Niaura, John F. Todaro, and Laura Stroud, Department of Psychiatry and Human Behavior, Brown Medical School, and Centers for Behavioral and Preventive Medicine, The Miriam Hospital, Providence, Rhode Island; Avron Spiro III, Department of Epidemiology, Boston University School of Public Health, and Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), Boston Veterans Affairs Healthcare System; Kenneth D. Ward, Center for Community Health, College of Arts and Sciences, University of Memphis; Scott Weiss, The Channing Laboratory, Brigham and Women's Hospital, Boston, and Department of Medicine, Harvard Medical School.

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Correspondence concerning this article should be addressed to Raymond Niaura, Centers for Behavioral and Preventive Medicine, The Miriam Hospital, Coro Building, Suite 500, One Hoppin Street, Providence, Rhode Island 02903. E-mail: raymond\_niaura@brown.edu

studies have established that hostility exerts an effect on CHD independent of the effects of so-called traditional risk factors, including age, tobacco use, hypertension, and total cholesterol. It remains possible, though, that the effects of hostility on CHD are partially mediated through its effect on these traditional risk factors. Studies that formally examine evidence for such mediation are lacking. Moreover, increased understanding of the etiology of CHD suggests that researchers must also consider other factors that may mediate hostility's effect on CHD, particularly those factors that are referred to collectively as *the metabolic syndrome*.

*The metabolic syndrome* refers to a clustering of disorders involving visceral obesity, insulin resistance, hyperglycemia, dyslipidemia, and hypertension (Kissebah & Krakower, 1994; Timar, Sestier, & Levy, 2000). The metabolic syndrome has been strongly implicated as an underlying disease process influencing the development of CHD and non-insulin-dependent diabetes (Bjorntorp, 1990a, 1990b; Lebovitz, 2001). Although recent studies continue to report a positive relationship between hostility-anger and incident CHD (Gallagher, Yarnell, Sweetnam, Elwood, & Stansfeld, 1999; Kawachi et al., 1998), none have examined this relationship while statistically covarying the complex of risk factors involved in the metabolic syndrome, specifically levels of fasting insulin and glucose, blood pressure, HDL-C, triglycerides (TRIGS), and visceral obesity (as measured by WHR). As noted above, studies have documented associations between hostility and these variables. Thus, it is conceivable that hostility might influence propensity toward CHD through its effects on one or more variables that constitute the metabolic syndrome.

The current study extends previous research examining the relationship between hostility and incident CHD in two ways. First, in addition to statistically controlling for the effects of behavioral variables (i.e., caloric intake, alcohol and tobacco use) and sociodemographic factors (i.e., age, educational attainment), the present study included physiological correlates of the metabolic syndrome. Second, because the relationship between hostility and incident CHD appears stronger among younger individuals (Dembroski, MacDougall, Costa, & Grandits, 1989; Lichtenstein, Pedersen, Plomin, DeFaire, & McClearn, 1989), the present study examined these relationships in a sample of older men participating in the Normative Aging Study (NAS). The aims of the present study were (a) to examine the relationship between hostility and sociodemographic, behavioral, and physiological risk factors for CHD, in particular those factors comprising the metabolic syndrome; and (b) to examine the degree to which hostility's effects on CHD are independent of, or mediated by, sociodemographic, behavioral, and metabolic syndrome variables.

## Method

### Participants

The NAS is a longitudinal study designed to examine biomedical and psychosocial changes involved in the normal aging process. This study involved a cohort of 2,280 men living in the greater Boston area who, at enrollment (1961–1970), were 21–80 years of age, predominantly White, had a high school education, and were free of any chronic medical conditions, such as cancer or diabetes mellitus. Volunteers were required to participate in regular examinations every 3 (age 52 or older) or 5 (younger than age 52) years. Since 1986, however, all men have been examined at 3-year intervals. Additional details of the sample and admission criteria have been described elsewhere (see Bosse, Ekerdt, & Silbert, 1984).

To be included in the present study, NAS participants were required to meet the following criteria: (a) completion of the Minnesota Multiphasic Personality Inventory (MMPI; Dahlstrom & Grant, 1960) in 1986, (b) completion of a comprehensive laboratory-based physiological assessment within 3 years following administration of the MMPI (see below), and (c) being free of any clinical evidence of cardiovascular disease, including use of cardiac (e.g., antihypertensives, lipid-lowering agents) and diabetic medications at the MMPI baseline assessment in 1986. Of the original NAS sample ( $N = 2,280$ ), 1,081 provided a complete and valid MMPI assessment. Twenty-eight percent ( $n = 307$ ) of the men who provided a valid MMPI assessment were excluded because they were taking one or more cardiac or diabetic medications at the baseline assessment in 1986, resulting in a final sample of 774 men for the present study.

### Procedures

On the night before the examination, participants refrained from eating or drinking after midnight and refrained from smoking after 8:00 p.m. The examination included blood pressure measurement, blood work (12-hr fasting serum levels of glucose, insulin, and lipids), an anthropometric evaluation (WHR and BMI), and assessment of health behaviors by standardized questionnaires (diet, alcohol intake, and smoking). Sociodemographic data, including educational attainment, were obtained on entry into the study.

### Measures

**Blood lipids.** Serum samples were analyzed for total cholesterol, HDL-C, LDL cholesterol (LDL-C), and TRIGS. Serum cholesterol was assayed enzymatically (SCALVO Diagnostics, Wayne, NJ). The HDL-C fraction was measured in the supernatant after precipitation of the LDL-C and very-low-density lipoprotein fractions with dextran sulphate and magnesium, using the Abbott Biochromatic Analyzer 100 (Abbott Laboratories, South Pasadena, CA). TRIGS concentration was measured using the Dupont ACA discrete clinical analyzer (Dupont Company, Biomedical Products Department, Wilmington, DE). LDL-C was estimated using Friedewald's formula (Friedewald, Levy, & Fredrickson, 1972).

**Fasting insulin.** Serum insulin concentration (INS) was determined by a solid phase [ $^{125}$ I]-radioimmunoassay (Diagnostic Products Corporation, Los Angeles, CA).

**Blood pressure.** Blood pressure was measured using a standard mercury sphygmomanometer with a 14-cm cuff. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured to the nearest 2 mm Hg. Both left- and right-arm pressures were measured in a sitting position, followed by right-arm pressures taken in a supine position, followed 30 s later by a second reading of right-arm pressures taken in a standing position. The palpatory method was used to check auscultatory systolic readings. The means of all systolic readings were used in analyses. There were no methodological differences in assessing blood pressure from one examination to another.

**BMI.** Weight was taken on a standard hospital scale with the participant dressed in undershorts and socks. Weight was measured to the nearest 0.5 lb and then converted to kilograms (kg). Height was measured standing in bare feet against a wall to the nearest 0.1 in. and then converted to meters (m). BMI was computed as  $(\text{kg}/\text{m}^2)$ .

**WHR.** With the participant standing, we measured abdomen circumference in centimeters at the level of the umbilicus and hip circumference in centimeters at the greatest protrusion of the buttocks. WHR was calculated as  $(\text{abdomen circumference})/(\text{hip circumference})$ .

**Health behaviors.** Behavioral risk factors assessed included alcohol and tobacco consumption and diet. Dietary data were obtained by means of a semiquantitative food frequency questionnaire (FFQ; Willett et al., 1985) that was mailed to each participant and completed before the examination. The FFQ lists food items with serving sizes and elicits information on

frequency of intake during the past year. Nutrient scores were computed by multiplying the frequency of intake by the nutrient content of the food items. Macronutrients examined in the present analyses were total energy intake (kcal/day) and alcoholic drinks per year (DPY). Information was also obtained on number of cigarettes currently smoked per week (CIGS).

**Hostility.** Hostility was measured with the Cook–Medley Ho Scale (Cook & Medley, 1954) taken from the MMPI Form AX (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) of the MMPI was administered, which includes items from both the MMPI and the MMPI-2.

**Sociodemographic risk factors.** Age in years was assessed at the time of the laboratory examination. Education was divided into four categories: less than high school, high school graduate (including attainment of a general education diploma), some college or college graduate (2 years of technical school or 4 years of college), and postcollege (some postgraduate or postgraduate).

**Incident CHD.** In the present study, 45 men experienced an initial occurrence of CHD between the MMPI baseline assessment in 1986 and the 3-year follow-up period. Diagnostic categories included in our definition of incident CHD were (a) myocardial infarction (MI;  $n = 24$ ), (b) angina pectoris ( $n = 23$ ), and (c) ischemic heart disease ( $n = 12$ ). The criteria for MI, angina pectoris, and ischemic heart disease were adapted from those used in the Framingham Heart Study (Shurtleff, 1974). A diagnosis of MI was made when supported by unequivocal electrocardiographic changes (i.e., pathologic Q waves), by a diagnostic elevation of serum glutamic–oxaloacetic transaminase and lactic dehydrogenase, and concurrent chest discomfort commonly evidenced in MI sufferers. Angina pectoris was diagnosed when participants reported experiencing recurrent chest discomfort that lasted for at least 15 min during periods of exertion and was remedied by rest or use of nitroglycerine. A classification of ischemic heart disease was defined as a horizontal or down-sloping ST-segment depression of  $> 1$  mm measured via a 12-lead electrocardiogram, without meeting established criteria for MI or angina pectoris.

### Data Analysis

Variables with nonnormal distributions (i.e., TRIGS, INS, CIGS, DPY) were log-transformed. Bivariate correlations were used to examine the relationship between hostility and sociodemographic (i.e., age, education), behavioral (i.e., total calories, DPY, CIGS), and physiological risk factors (i.e., WHR, BMI, SBP, DBP, INS, HDL-C, LDL-C). Next, to examine the multivariate relationship between hostility and incident CHD controlling for other variables, we used a series of linear logistic regressions with incident CHD as the dichotomous dependent variable (yes = 1; no = 0). Finally, we examined potential mediators of the relationship between hostility and incident CHD using the stepwise approach recommended by Baron and Kenny (1986).

## Results

### Sample Characteristics

Table 1 presents the sociodemographic, behavioral, and physiological characteristics of the sample collected at the time of the MMPI administration in 1986. Study participants were men, with an average age of 60.3 years ( $SD = 7.9$ ). With regard to behavioral characteristics, study participants demonstrated an average Ho score of 16.7 ( $SD = 7.8$ ). Approximately 65.5% of the sample currently drank alcohol (defined as  $> 1$  DPY), and 11.5% smoked cigarettes. Study participants, on average, consumed 1,995 calories per day, drank 567 DPY, and smoked 2.88 cigarettes per day. For those who reported drinking alcohol and smoking cigarettes, the average number of DPY and cigarettes per day were 645 and 25, respectively. In terms of anthropometric and physiological char-

Table 1  
*Descriptive Statistics*

Variable	<i>N</i>	<i>M</i> or %	<i>SD</i>
Ho	774	16.7	7.8
Age (years)	774	60.3	7.9
Education (%)			
Less than high school	64	8.3	
High school	190	24.5	
Some college	299	38.6	
College or postgraduate	210	27.1	
BMI ( $\text{kg}/\text{m}^2$ )	753	26.49	3.44
WHR	752	0.98	0.05
DPY	718	567	856
CIGS	772	2.88	9.12
Total calories (kcal/day)	708	1,995	626
HDL-C (mg/dL)	762	49.0	12.5
LDL-C (mg/dL)	761	158.5	35.8
TRIGS (mg/dL)	773	145.0	97.6
SBP (mm Hg)	735	127.6	15.6
DBP (mm Hg)	735	77.9	8.5
GLU (mg/dL)	774	101.5	16.0
INS (mU/mL)	705	0.98	0.23

*Note.* Ho = total Cook–Medley Hostility Scale score; BMI = body mass index; WHR = waist-to-hip ratio; DPY = drinks per year; CIGS = current cigarettes per day; HDL-C = HDL cholesterol; LDL-C = LDL cholesterol; TRIGS = triglycerides; SBP = systolic blood pressure; DBP = diastolic blood pressure; GLU = log-transformed fasting glucose; INS = log-transformed fasting insulin.

acteristics of the sample, study participants had an average WHR of 0.98 and BMI of 26.49. Average SBP was 127.6 and DBP was 77.9. Furthermore, average HDL-C and INS were 49.0 mg/dL and 0.98 mU/mL, respectively, for the sample.

### Associations Between Hostility and CHD Risk Factors

The correlations among Ho and sociodemographic, behavioral, and physiological characteristics were examined (see Table 2). There was no relationship between Ho and the age of study participants; however, Ho was significantly and negatively correlated with level of education. Ho correlated positively with total caloric consumption, but no relationship was evidenced between Ho and current alcohol consumption or cigarette use. Furthermore, Ho correlated positively with both anthropometric measures, BMI and WHR; however, no relationship was observed between Ho and blood pressure (SBP and DBP), HDL-C, and INS.

### Predictors of Incident CHD

In this sample of older men, 5.8% ( $n = 45$ ) experienced at least one episode of incident CHD during their involvement in the NAS study between the 1986 baseline MMPI and assessment and the 3-year follow-up assessment. We computed hierarchical logistic regression models to examine odds ratios (ORs) and 95% confidence intervals (CIs) of incident CHD as a function of Ho; Ho adjusted for demographic variables; and Ho adjusted for demographic, behavioral, and physiological variables. In the first model, Ho was entered alone and significantly predicted incident CHD ( $OR = 1.04$ , 95%  $CI = 1.02$ – $1.09$ ). Next, Ho was entered in the model and adjusted for demographic variables (age and education). In this second model, there was a slight increase in the multivariate

Table 2  
Pearson Correlations Among Cook-Medley Hostility Scale Scores and Behavioral and Physiological Risk Factors

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. New CHD	—															
2. Ho		.11**														
3. Age			.09*													
4. Education			.02	-.07												
5. BMI				.17**	.05	.06	-.05	.04	-.05	-.13**	.03	.12**	.08*	.00	.05	.04
6. WHR					.12**	.14**	.07	.06	.15**	.00	.04	.07	-.03	-.04	.02	.05
7. DPY					-.13	-.04	-.15	-.18	.04	.02	-.13	-.11	.23	-.13**	.05	-.07
8. CIGS					-.11**	-.10**	-.05	-.13**	-.01	.13**	-.02	-.06	.07	.03	-.01	-.04
9. Total calories						.49**	.00	-.02	.02	-.20**	.04	.27**	.12**	.21**	.15**	.44**
10. HDL-C							.13**	.08*	.16**	-.08*	.06	.24**	.17**	.17**	.09*	.36**
11. LDL-C								.08*	.06	.17**	-.09*	.08*	.04	-.03	.08*	.00
12. TRIGS									.01	.13**	.01	.08*	.10**	-.15**	.00	.03
13. SBP									.06	.05	-.06	-.04	-.02	-.02	.00	-.03
14. DBP											-.07	-.44**	-.02	-.02	-.07*	-.18**
15. GLU												.07	-.03	.10**	.00	-.02
16. INS														.15**	.22**	.30**
														.60**	.15**	.14**
															.05	.23**
																.22**

Note. New CHD = incident CHD during follow-up after an initial baseline examination in which no CHD was detected; Ho = total Cook-Medley Hostility Scale score; BMI = body mass index; WHR = waist-to-hip ratio; DPY = drinks per year; CIGS = current cigarettes per day; HDL-C = HDL cholesterol; LDL-C = LDL cholesterol; TRIGS = triglycerides; SBP = systolic blood pressure; DBP = diastolic blood pressure; GLU = log-transformed fasting glucose; INS = log-transformed fasting insulin.

\*  $p < .05$ . \*\*  $p < .01$ .

OR for Ho (OR = 1.05, 95% CI = 1.01–1.09) and the effect for age (OR = 1.05, 95% CI = 1.01–1.09) but not education (OR = 0.81, 95% CI = 0.61–1.06). In the final model (see Table 3), which included Ho, sociodemographic variables, behavioral variables, and correlates of the metabolic syndrome, the OR for Ho was only marginally increased (OR = 1.06, 95% CI = 1.01–1.11), compared with the unadjusted OR, suggesting that for every 1-point increase in Cook-Medley Ho total scores, there was a 6% increase in the risk of incident CHD. Aside from Ho and HDL-C, none of the metabolic syndrome (i.e., HDL-C, INS, TRIGS, SBP, WHR), sociodemographic, behavioral, or other physiological variables significantly predicted incident CHD. The logistic regression model was rerun including those participants who were using diabetic medications ( $n = 18$ ) with no material change in the results. Another model was also run in which the fasting insulin and glucose variables were eliminated as separate terms and replaced with a term thought to represent insulin resistance (IR)— $IR = (\text{fasting insulin}/\text{fasting glucose})/22.5$ —derived from the HOMA method (Radziuk, 2000). This analysis did not alter the findings.

To examine the nature of the association between Ho and CHD, we divided Ho into categories based on quintiles of Ho scores, yielding five hostility groups: < 20th percentile ( $n = 7$ ), 20th–39th percentile ( $n = 6$ ), 40th–59th percentile ( $n = 5$ ), 60th–79th percentile ( $n = 8$ ), and  $\geq 80$ th percentile ( $n = 19$ ; see Figure 1). Chi-square analysis indicated that significantly more episodes of incident CHD were observed in the upper quintile Ho group compared with lower quintile Ho groups,  $\chi^2(4, N = 45) = 14.44$ ,  $p < .01$ .

There was no evidence to support mediation of Ho's effects on incident CHD by any variable included in the present analysis. We

Table 3  
Predictors of Incident Coronary Heart Disease

Variable	OR (95% CI)	B	SE	Wald's $\chi^2$
Ho	1.06 (1.01–1.11)	0.06	0.02	6.33
Age	1.04 (0.99–1.09)	0.04	0.02	2.39
Education	0.80 (0.57–1.12)	-.23	0.17	1.71
BMI	1.09 (0.96–1.24)	0.09	0.07	1.81
WHR	0.59 (0.00–76.00)	-.53	4.82	0.01
DPY	1.00 (1.00–1.00)	0.00	0.00	0.56
CIGS	1.00 (0.97–1.04)	0.00	0.02	0.03
Total calories	1.00 (1.00–1.00)	0.00	0.00	1.77
HDL-C	0.95 (0.91–0.98)	-.06	0.02	7.45
LDL-C	1.00 (0.99–1.01)	0.00	0.01	0.42
SBP	1.02 (0.99–1.04)	0.02	0.01	2.07
GLU	1.00 (0.99–1.01)	0.00	0.00	0.56
INS	0.54 (0.08–3.50)	-.62	0.95	0.42
Constant		-6.03	4.82	1.56

Note.  $N = 774$ . Coronary heart disease coded as 1 = yes, 0 = no. Triglycerides and diastolic blood pressure were not included in the final model because their effects were interchangeable with HDL-C and SBP, respectively. In an alternative model, cigarette and alcohol use were dichotomized as current (yes, no), with no material change in the parameter estimates. OR = odds ratio; CI = confidence interval; Ho = total Cook-Medley Hostility Scale score; BMI = body mass index; WHR = waist-to-hip ratio; DPY = drinks per year; CIGS = current cigarettes per day; HDL-C = HDL cholesterol; LDL-C = LDL cholesterol; SBP = systolic blood pressure; GLU = log-transformed fasting glucose; INS = log-transformed fasting insulin.



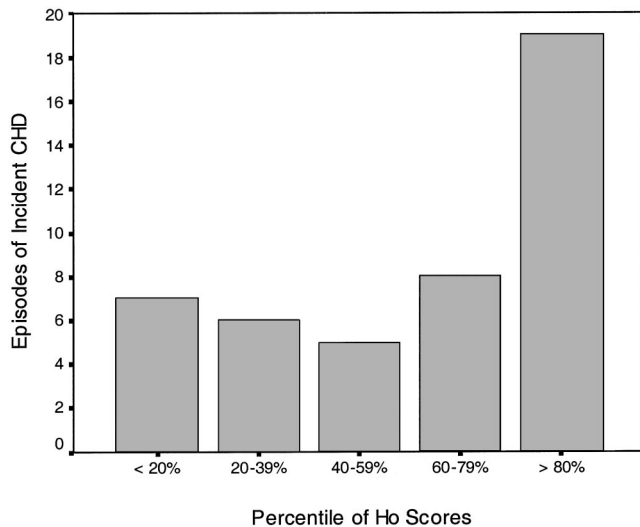


Figure 1. Episodes of coronary heart disease (CHD) across levels of hostility. Ho = total Cook–Medley Hostility Scale score.

also examined whether the relationship between Ho and incident CHD may have been moderated by other behavioral, physiological, or sociodemographic variables. A series of two-way interaction terms (Ho with each variable) was entered into the model sequentially after main effects. Only the interaction between Ho and WHR made a significant contribution to the model, Wald's  $\chi^2(4, N = 45) = 3.87, p = .05$ . More specifically, an increased frequency of incident CHD occurred in older men with high levels of Ho but lower levels of WHR.

### Discussion

Results of the present study are consistent with other studies demonstrating that Ho is associated with and predicts incident CHD above and beyond the influence of traditionally assessed CHD risk factors, including blood lipid profiles, sociodemographic characteristics, and certain behavioral factors, such as alcohol consumption and cigarette use (Barefoot et al., 1983; Shekelle et al., 1983). The current study, however, extends these findings by demonstrating that Ho predicted incident CHD above not only traditional risk factors but also factors related to the metabolic syndrome. Specifically, older men with the highest levels of Ho (i.e., those in the upper quintile) were at the greatest risk for incident CHD, independent of the effects of fasting insulin, BMI, WHR, TRIGS, and blood pressure. Of the variables commonly associated with the metabolic syndrome, only HDL-C levels predicted incident CHD. This finding is consistent with other studies suggesting that increased levels of HDL-C are associated with lower risk of CHD morbidity (Assman & Schulte, 1992; Burchfiel et al., 1995). Although Ho and HDL-C were associated with incident CHD, HDL-C, age, and most other variables assessed did not appear to mediate or moderate the relationship between Ho and incident CHD. This suggests that in older healthy men, Ho and HDL-C can be regarded as independent risk factors for incident CHD.

Inconsistent with previous findings, the interaction between Ho and WHR predicted incident CHD. In older men from the NAS, it

appears that an increased proportion of incident CHD was experienced by those with higher levels of Ho and lower levels of WHR. It should be noted that this relationship was only marginally significant and should be interpreted with caution, especially given the increased risk of false-positive results with tests for multiple interaction effects.

There were a number of limitations to this study that should be reported. First, the nature of the sample (i.e., older men, primarily White, normal health status) may have obscured relationships between metabolic risk factors and CHD that may have been evident in a more general and representative population sample. Therefore, generalizability of our results to younger adults, women, and other ethnic minority populations is limited. Second, power to detect significant effects may have been low because of the relatively low incidence of CHD; a longer follow-up may have afforded this opportunity. Third, we did not include measures of perceived stress, stress hormones, and other CHD risk factors that may have assisted with the interpretation of the interrelationships between demographic, behavioral, and physiological variables. Fourth, the magnitude of the associations between Ho and other variables may be viewed as small; however, this is generally consistent with the results of other studies (e.g., Kaye, Folsom, Jacobs, Hughes, & Flack, 1993) and may have significant implications for older men presenting with high levels of Ho and low HDL-C.

Future studies examining the relationship between Ho and CHD should focus on uncovering the physiological and psychosocial mechanisms underlying these relationships. For example, it is possible that Ho predisposes an individual toward CHD through other mechanisms not measured in this study (e.g., cardiac arrhythmia, imbalance in sympathetic–parasympathetic nervous system activity, cardiovascular and endocrine–neuroendocrine responses to stress, coronary artery vasospasm, clotting factors). In addition, future studies should continue to examine the relationship between Ho and incident CHD in diverse populations (i.e., race, sex, socioeconomic groups) because much of the research in this area has been focused on White men. Studies examining cultural, racial, and gender differences with respect to Ho and incident CHD may elucidate additional physiological, behavioral, and/or genetic mechanisms underlying these relationships. Finally, the confluence of findings that Ho predicts incident CHD suggests that we should continue to investigate the efficacy of providing psychosocial interventions for individuals with high levels of hostility (Burell et al., 1994; Friedman et al., 1986).

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