THE RELATION OF HOSTILITY TO LIPIDS AND LIPOPROTEINS IN WOMEN: EVIDENCE FOR THE ROLE OF ANTAGONISTIC HOSTILITY[1]

Edward C. Suarez, Ph.D., Michael P. Bates, B.A., and Tina L. Harralson, Ph.D.
Duke University Medical Center

ABSTRACT

We examined the relation of antagonistic, neurotic, and cynical hostility to lipids and lipoproteins in 77 healthy women (aged 18-26) selected for having high (> 17) or low (< 12) scores on the Cook-Medley Hostility (Ho) scale. Fasting lipids were determined during the luteal phase of the menstrual cycle for oral contraceptive (OC) non-users (N = 41), and during pills 15-21 for OC users (N = 36). Factor scores for antagonistic and neurotic hostility were derived from a principal component of the Buss-Durkee Hostility Inventory, Spielberger's Anger Expression, and the NEO-Personality Inventory. High Ho scores were significantly associated with higher cholesterol. Antagonistic hostility significantly predicted cholesterol, low density lipoprotein cholesterol, triglycerides, and the ratio of cholesterol to high density lipoprotein cholesterol, with higher antagonistic hostility scores associated with higher levels. Neurotic hostility did not predict lipids. Results suggest a potential pathophysiological mechanism that may contribute to the association between hostility and coronary heart disease. Moreover, a measure of antagonistic hostility, relative to cynical and neurotic hostility, was the best predictor of lipid levels.

(**Ann Behav Med** 1998, *20*(2):59–63)

INTRODUCTION

Epidemiologic evidence suggests that hostility is associated with an increased risk of coronary heart disease (CHD) and severity of coronary artery disease (CAD) (1,2). In light of this evidence, researchers have taken steps toward identifying potential mechanisms underlying the link between hostility and CHD. Several hypotheses have been proposed implicating both behavioral and physiological mechanisms (1,2). One pathophysiological mechanism that has been explored is elevations in lipids and lipoproteins. Studies conducted in the 1960s and 1970s suggested a positive association between lipids and hostility (3–6). Despite these supportive early findings, declining interest led to few follow-up studies. Recent efforts, however, have again focused on determining the association between lipids and behavioral risk factors, such as hostility.

Recent studies that have examined the relationship between hostility and lipids have yielded equivocal results (7). A number of studies have reported positive correlations between lipids and various measures of hostility (8–15). Other studies, however, have failed to find an association (16–19). At this time, it is not known what factor(s) may contribute to the variability of the findings. Given the multidimensional nature of hostility (20,21), one possibility is that only certain aspects of hostility are associated with lipids. Preliminary evidence relating hostility dimensions to lipids supports the above hypothesis (22). Results of one study indicated that the expression of anger subscore from the Buss-Durkee Hostility Inventory (BDHI) (23) measuring the outward expression of anger was positively associated with lipids and lipoproteins (22). Given these observations, it may be that the lack of consistency is due to the degree to which any individual scale taps coronary-prone facets of hostility. Therefore, one alternative approach that circumvents the problems of using a single measure of hostility is to employ a number of scales and generate latent measures of the dimensions of hostility.

The goals of the current study were to determine the relation of Cook-Medley Hostility (Ho) and hostility dimensions to lipids and lipoproteins and to compare the relative strength of the associations of lipids to a single measure of hostility (i.e. Cook-Medley Ho) versus factor analytically-derived measures of hostility dimensions. We hypothesized that both the Cook-Medley Ho scale, a measure of cynical hostility (24), and the factor analytically-derived measure of antagonistic hostility would be positively and significantly associated with lipids and lipoproteins. Moreover, we hypothesized that, relative to Ho-derived hostility, the factor analytically-derived measure of antagonistic hostility would be a better predictor of lipids and lipoproteins.

METHODS

Subjects

Subjects were 77 healthy women (aged 18-26) who had Cook-Medley Ho scores above 17 (N = 44; high Ho group) or below 12 (N = 33; low Ho group). Subjects were preselected using *a priori* Ho cutoff scores representing the upper and lower thirds for women in this age group. Preselection on Ho scores was conducted so as to allow for the maximum difference in the level of hostility as assessed by the Ho scale. Racial breakdown of the sample was as follows: 72 Caucasians (94%), 3 Hispanics (4%), 1 Asian-American (1%), 1 Native American (1%). Thirty-six subjects (47%) were classified as oral contraceptive (OC) users, defined as OC use for at least six months prior to participation. The remaining 41 subjects (53%) reported no use of OC for at least the previous six months prior to participation in the study. One subject had missing lipoprotein data.

Procedures

Volunteers were recruited through advertisements placed in the campus newspapers and announcements posted on campus bulletin boards. Potential subjects were scheduled for a preliminary screening session during which they completed the 50-item true-false Ho scale and a health history and personal lifestyle habits questionnaire. Subjects were asked to participate only if

[1] Preparation of this manuscript was supported in part by grant HL-46283 from the National Heart, Lung, and Blood Institute.

Reprint Address: E. C. Suarez, Ph.D., Duke University Medical Center, Box 3926, Durham, NC 27710.

TABLE 1
Adjusted Group Means (SEM) for Lipids and Lipoproteins for Hostility

	High Ho (N = 44)	Low Ho (N = 33)	p
TSC	164.3 (4.1)	153.0 (4.5)	0.03
LDL	96.8 (3.6)	88.0 (3.6)	0.06
HDL	49.7 (1.8)	49.4 (1.6)	0.73
TRIG	89.2 (6.0)	82.8 (6.7)	0.24
RATIO	3.47 (0.14)	3.20 (0.12)	0.18

TABLE 2
Promax-Rotated Factor Loadings for Neurotic and Antagonistic Hostility

Scale	Neurotic Hostility (48.1% Variance)	Antagonistic Hostility (26.2% Variance)
Anger-in	*.84*	−.12
Anger-out	−.23	*.87*
Buss-Durkee Experience of Anger	*.86*	.20
Buss-Durkee Expression of Anger	.30	*.80*
NEO Agreeableness	−.33	*−.75*
NEO Neuroticism	*.79*	.37

their Ho score was above 17 or below 12 and reported no history of smoking, hypertension, or lipid disorders. Women were excluded if they reported changes in OC usage within the previous six months, irregular-menstrual cycles, or use of lipid-altering medications. To control for cyclical hormonal effects on lipids (25), non-users were scheduled for their laboratory testing during the luteal phase of their menstrual cycle (5-9 days prior to onset of menstruation), and OC users were scheduled during pills 15-21 of their cycle.

On the laboratory testing day, subjects reported to our offices between 8:00 and 9:00 a.m. after abstaining from food, caffeine, and medications for at least twelve hours prior to sample collection. A 5 ml blood sample was obtained by venous draw by a registered nurse who was blind to the subject's Ho group. Subjects then completed a number of paper-and-pencil questionnaires.

Psychological Questionnaires

Subjects completed the following questionnaires: BDHI, Spielberger's Anger Expression (AX), and the NEO-Personality Inventory (NEO-PI). The BDHI (23) is a comprehensive hostility inventory which yields seven subscales and a global hostility score. The BDHI also yields two subscores representing expression and experience of anger (26). Expression of anger represents the sum of the assault, indirect hostility, and verbal hostility subscales; experience of anger represents the sum of the resentment and suspicion subscales (26). Spielberger's AX (27) was designed to assess the frequency with which one expresses anger. It yields subscores for anger-in and anger-out, along with a total anger expression score. The NEO-PI (28) assesses personality along five trait dimensions. Two of these traits, neuroticism and agreeableness, appear to be related to hostility (29).

Lipid Assays

Blood samples were assayed for lipid and lipoprotein constituents by Duke University Medical Center Clinical Laboratories. Total serum cholesterol (TSC), high density lipoprotein (HDL) cholesterol, and triglycerides (TRIG) were determined enzymatically by an Instrumentation Laboratory analyzer (Monarch). The analyzer was calibrated with standard cholesterol calibrator (Sigma Diagnostics #C7921) for TSC and HDL and with precical calibrator serum and diluent (Boehringer Mannheim #620213) for TRIG. TSC and HDL were analyzed with cholesterol reagent (Boehringer Mannheim #816302). For HDL, larger proteins were precipitated out prior to analysis using dextran sulfate (Dextralip 50 #Y01307 Sochibo, SA) and magnesium chloride (Mallinckrodt #5958). TRIG was determined using triglyceride reagent (Sigma Diagnostics GPO-TRINDER #337-B). Low density lipoprotein (LDL) cholesterol and the ratio of TSC to HDL (RATIO) were derived arithmetically from TSC, HDL, and TRIG values.

Data Analysis

All statistical procedures were conducted using the SAS statistical package (30). Preliminary analyses of lipid and lipoprotein levels were performed using analysis of covariance (ANCOVA) with Ho group (high/low) as the between-subject factor and body mass index (BMI) and OC status as covariates. Antagonistic and neurotic hostility factor scores were generated using a principle component analysis with a promax rotation, an oblique transformation. The promax rotation, in contrast to an orthogonal transformation such as the varimax rotation, produces factors that are moderately correlated. The use of an oblique transformation allowed for a factor solution that is consistent with the notion that dimensions of hostility are moderately correlated and not independent (21). The following variables were included in the principal component analysis: expression and experience of anger from the BDHI, the anger-out and anger-in from the AX scale, and agreeableness and neuroticism from the NEO-PI. Factor scores, with means of 0 and standard deviations of 1, were computed as a linear combination of the standardized values of the variables (30). Factor scores were used as predictor variables in multiple regression analyses with BMI and OC use as covariates and lipids and lipoprotein levels as outcome variables.

RESULTS

Cook-Medley Ho Group

Preliminary analyses indicated no significant Ho group differences in BMI ($t[75] = -0.95$) or distribution of OC users and non-users (χ^2 [1] = 1.41). Results of ANCOVAs with OC use and BMI as covariates yielded a significant Ho group effect for TSC ($F[1, 73] = 5.14$, $p = .026$) and marginally significant for LDL cholesterol ($F[1, 73] = 3.75$, $p = .057$). For all analyses, OC use ($Fs > 6.8$) and BMI ($Fs > 3.35$) were significant predictors of TSC and LDL. Comparison of adjusted group means indicated that, relative to low Ho women, high Ho women had significantly higher TSC. High Ho women also exhibited higher levels of LDL cholesterol levels. Ho groups did not differ on levels of TRIG, HDL cholesterol, and the TSC/HDL ratio. Adjusted means for Ho groups are shown in Table 1.

Hostility Dimensions: Antagonistic and Neurotic Hostility

The principle component analysis yielded a two-factor solution accounting for 74.3% of the variance (see Table 2). Factor 1 (eigenvalue = 2.88), defined by positive loadings on anger-in, neuroticism, and experience of anger, was interpreted as the experience of hostility/anger or neurotic hostility. Factor 2 (eigenvalue = 1.57), defined by positive loadings for anger-out and expression of anger and a negative loading for agreeableness, was interpreted as the expression of hostility/anger or antagonistic hostility. The correlation between the two factor scores was −0.23 ($p < .05$). Results of t-tests indicated significant differences

between high and low Ho groups for both antagonistic hostility ($t[74] = -4.712, p < .001$; Ms [sd] = −.54[.88] and .41[.89], for high and low Ho group, respectively) and neurotic hostility ($t[72.5] = -5.34, p < .001$; Ms [sd] = −.57 [.66] and .44 [1], for high and low Ho group, respectively). A similar two-factor solution has been reported for men (31) and women (32) who were preselected on the basis of high and low Ho scores. In addition, Musante et al. (21) reported a similar factor structure using a sample of subjects who were not preselected on any criteria.

We tested whether preselection of subjects on Ho scores affected the factor solution by conducting independent principal components for the high and low Ho groups. We used each of the factor solutions to generate two sets of factor scores for both the high and low Ho groups. The stability of the factor solutions was determined by the degree to which factor scores derived from the high Ho factor solution correlated with scores derived from the low Ho factor solution for each Ho group. A variant factor structure would yield factor scores that would not be highly correlated. In contrast, an invariant factor structure would yield factor scores that would be highly correlated.

For the high Ho group, factor scores generated from its own factor structure were highly correlated with scores derived from the factor solution of the low Ho group (Neurotic hostility [$r = 0.97, p < .001$]; Antagonistic hostility [$r = 0.87, p < .001$]). Similarly, for the low Ho group, factor scores generated from its own factor structure and from the factor solution of the high Ho group were also highly correlated (Neurotic hostility [$r = 0.90, p < .001$]; Antagonistic hostility [$r = 0.91, p < .001$]). These results indicate an invariant factor structure across Ho groups and suggest that preselection did not affect the overall factor structure. Although we did not have a subgroup of individuals with Ho scores in the intermediate range (i.e. 12–17), it is unlikely that the factor structure for a middle Ho group would differ significantly from the factor structures observed for high and low Ho groups, both of which were essentially identical.

A series of regression analyses was conducted for each lipid and lipoprotein constituent with BMI and OC use as covariates and antagonistic and neurotic hostility factor scores as predictor variables. Results indicated that antagonistic hostility was a significant predictor of TSC ($b = 8.08, p < .01$), LDL cholesterol ($b = 5.91, p < .05$), TRIG ($b = 11.48, p < .01$), and marginally significant for the TSC/HDL ratio ($b = .19, p = .057$). Neurotic hostility did not significantly predict any of the blood lipid constituents.

For illustrative purposes, antagonistic hostility factor scores were trichotomized in order to generate low ($N = 25$), medium ($N = 26$), and high ($N = 26$) antagonistic hostility groups. Group means for TSC, LDL cholesterol, TRIG, and RATIO were calculated and are shown in figures 1 and 2.

Lastly, we conducted a multiple regression to test directly the relative strength of the relations of Cook-Medley Ho and antagonistic hostility to lipids. TSC was used as the outcome variable since the Cook-Medley Ho significantly predicted only TSC. Regression analysis included antagonistic hostility factor score and Cook-Medley Ho group as predictor variables and OC use and BMI as covariates. Results indicated that antagonistic hostility was significantly associated with TSC ($b = 5.80; p = .045$). In contrast, the Ho group effect was not significant ($b = .09; p = .259$).

DISCUSSION

The primary aim of the current study was to examine the relations of dimensions of hostility to lipid and lipoprotein levels.

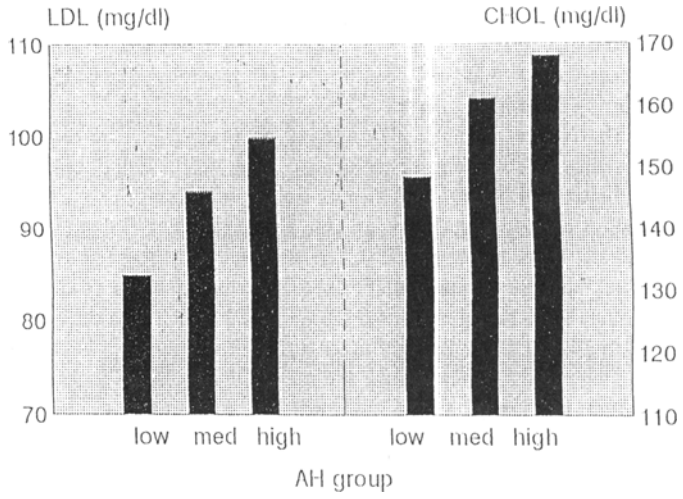

FIGURE 1: Means for LDL cholesterol and TSC for antagonistic hostility tercile groups.

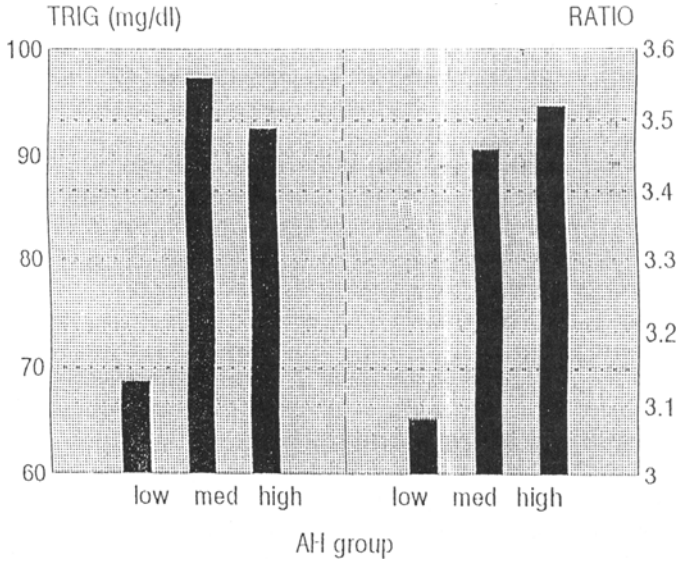

FIGURE 2: Means for triglycerides and the TSC-to-HDL ratio for antagonistic hostility tercile groups.

Consistent with our hypotheses, antagonistic hostility, characterized by the outward expression of anger in a verbal or physical manner and an antagonistic interpersonal style, was positively associated with TSC, LDL cholesterol, triglyceride, and the TSC-to-HDL ratio. In contrast, neurotic hostility was not associated with lipid or lipoprotein levels. In addition, Ho-defined cynical hostility was also positively correlated with TSC. However, when both the Ho scale and antagonistic hostility were entered into a single regression model, only antagonistic hostility, and not Ho, predicted TSC.

The results of the present study underscore the importance of examining potential differences in the associations between hostility dimensions and cardiovascular risk factors. The current findings are in line with recent observations indicating that only some aspects of hostility are associated with increased CHD risk. For example, Siegman et al. (26) found that severity of CAD was associated only with the BDHI expression of anger and not with the experience of anger subscore. As noted in the introduction, preliminary evidence has also indicated that the expression of

anger subscore from the BDHI, and not the experience of anger subscore, is predictive of blood lipids (22). Our results provide additional support to the notion that only certain aspects of global hostility are coronary prone.

The current findings also address themselves to the relative importance of construct measurement in determining the relationship between hostility and blood lipids. We find that a single measure of hostility, such as the Cook-Medley Ho scale, is moderately associated with TSC. In contrast, a factor analytically-derived measure of antagonistic hostility was a better predictor of not only TSC, but also TRIG, LDL cholesterol, and the ratio of TSC to HDL. Differences in the strength of these associations may be attributed to the fact that the Ho scale contains items measuring both neurotic and antagonistic hostility, with neurotic hostility more strongly associated with the Ho scale (31–33). Whatever the explanation for the observed relationships, the set of findings using the factor analytically-derived measure versus the results using the Ho scale argues for the use of multiple measures rather than single scales in future studies.

Given these findings, what can be said about possible behavioral and physiological mechanisms linking hostility to lipids? First, hostile individuals are characterized by personal habits that may contribute to elevations in lipids. In the Coronary Artery Risk Factor Development in Young Adult (CARDIA) study, persons with high Ho scores reported greater smoking and increased caloric intake, both known to have an effect on lipids (18). Physiological mechanisms such as stress-induced catecholamine hyperresponsivity may also link hostility to elevated lipids (34). Añimals studies have indicated that infusion of epinephrine leads to an increase in cholesterol (35). In our laboratory, we have observed positive associations between lipid levels and neuroendocrine responses to stress, but only in high Ho males (36) and females (37). It may be that elevated lipids in hostile women are attributed either to excessive behaviorally-induced catecholamine reactivity or to behavioral factors, or both.

Studies that have examined the relationship between hostility and lipids in women (e.g. 9,11,13,15,22,38), appear to have some degree of consensus. For the most part, these studies have reported positive associations between hostility and some lipid and lipoprotein constituents. The most consistent findings, however, have been reported in studies where the measure of hostility taps the outward expression of anger and/or an antagonistic interpersonal style. For example, Dujovne and Houston (9) found no association between the overall score on the BDHI and lipids. Follow-up analyses, however, showed that the expression of anger subscore, and not the experience of anger subscore, was positively associated with TSC, LDL, and the TSC-to-HDL ratio. The expression of anger subscore reflects the outward expression of anger in a verbal and/or physical manner. In contrast, Weidner et al. (15), using the SCL-90 paranoid ideation scale to measure hostility, only reported a significant association for LDL. The current findings replicate and extend previous observations by demonstrating that antagonistic hostility is the best predictor of lipids and lipoproteins. Moreover, the current observations directly compare the predictive validity of two different measures of hostility, the Cook-Medley Ho scale, and a factor analytically-defined measure of antagonistic hostility, with the results clearly indicating antagonistic hostility as the best predictor. Thus, one reason some studies fail to show a clear pattern of associations between lipids and a measure of hostility is the limitation of the scale to tap aspects of hostility that are coronary prone.

One possible limitation of the current study is that the study was conducted on young, healthy women. An important issue, therefore, is whether the findings can be generalized to older women. Preliminary evidence from one study has indicated that hostility measured during late adolescence predicts the TSC-to-HDL ratio assessed 21–23 years later (28). In the same study, hostility measured at midlife was associated with the lipid ratio. Findings by others also suggest that the current observations are not specific to this age group. As noted above, DuJovne and Houston (9) found an association between lipids and expression of anger subscore from the BDHI in a sample of subjects whose age ranged from 18 to 64. Thus, it is likely that the current findings are generalizable to an older population. Nevertheless, future studies should examine whether a similar pattern of associations is present in older women, as well as in men.

In conclusion, the results of the current study indicate that a factor analytically-derived measure of antagonistic hostility is associated with higher levels of TSC, LDL cholesterol, triglycerides, and the ratio of total cholesterol to HDL cholesterol. The findings provide empirical evidence to suggest that hostile women's increased risk of CHD may be due, in part, to elevations in blood lipids. Moreover, the findings suggest that only certain aspects of hostility, characterized by an outward expression of anger in a verbal or physical manner and an antagonistic interpersonal style, are potentially coronary prone.

REFERENCES

(1) Smith TW: Hostility and health: Current status of psychosomatic hypothesis. *Health Psychology.* 1992, *11:*139–150.
(2) Williams RB: A relook at personality type and coronary heart disease. *Progressive Cardiology.* 1991, *4:*91–97.
(3) Friedman M, Rosenman RH: Association of specific overt behavior pattern with blood and cardiovascular findings. *Journal of the American Medical Association.* 1959, *169:*1286–1296.
(4) Friedman M, Byers SO, Rosenman RH, et al: Coronary-prone individuals (Type A behavior pattern): Some biochemical characteristics. *Journal of the American Medical Association.* 1970, *212:*1030–1037.
(5) Jenkins CD, Rosenman RH, Friedman M: Components of the coronary-prone behavior pattern: Their relation to silent myocardial infarction and blood lipids. *Journal of Chronic Diseases.* 1966, *19:*599–609.
(6) Rosenman RH, Friedman M: Association of specific behavior pattern in women with blood and cardiovascular findings. *Circulation.* 1961, *24:*1173–1184.
(7) Niaura R, Stoney CM, Herbert PN: Lipids in psychological research: The last decade. *Biological Psychology.* 1992, *34:*1–43.
(8) Siegler IC: Hostility and risk: Demographic and lifestyle variables. In Siegman AW, Smith TW (eds), *Anger, Hostility and the Heart.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1994, 199–214.
(9) Dujovne VF, Houston BK: Hostility-related variables and plasma lipid levels. *Journal of Behavioral Medicine.* 1991, *14:*555–565.
(10) Fowkes FGR, Leng GC, Donnan PT, et al: Serum cholesterol, triglycerides, and aggression in the general population. *Lancet.* 1992, *340:*995–998.
(11) Lundberg U, Hedman M, Melin B, Frankenhaeuser M: Type A behavior in healthy males and females as related to physiological reactivity and blood lipids. *Psychosomatic Medicine.* 1989, *51:*113–122.
(12) Niaura R, Stoney CM, Ward K, et al: Hostility and cardiovascular disease risk in older males: The Normative Aging Study (Abstract). *Psychosomatic Medicine.* 1994, *56:*147.
(13) Scanlan JM, Myers CD, Werner JJ, et al: Hostility and cardiovascular risk factors: Interaction with sex and health habits (Abstract). *Psychosomatic Medicine.* 1994, *56:*147.

(14) Sloane RB, Habib A, Eveson MD, Payne RW: Some behavioral and other correlates of cholesterol metabolism. *Journal of Psychosomatic Research.* 1991, *5:*183–186.
(15) Weidner G, Sexton G, McLellarn R, et al: The role of Type A behavior and hostility in an elevation of plasma lipids in adult women and men. *Psychosomatic Medicine.* 1987, *49:*136–145.
(16) Almada SJ, Zonderman AB, Shekelle RB, et al: Neuroticism and cynicism and risk of death in middle-aged men: The Western Electric Study. *Psychosomatic Medicine.* 1991, *53:*165–175.
(17) Niaura R, Herbert P: Hostility and blood lipids in healthy adults. The 12th Annual Meeting of the Society of Behavioral Medicine. Washington, DC: March, 1991.
(18) Scherwitz LW, Perkins LL, Chesney MA, et al: Hostility and health behaviors in young adults: The CARDIA Study. *American Journal of Epidemiology.* 1992, *136:*136–145.
(19) Shekelle RB, Gale M, Ostfeld AM, Paul O: Hostility, risk of coronary heart disease, and mortality. *Psychosomatic Medicine.* 1983, *45:*109–114.
(20) Barefoot JC, Lipkus IM: Assessment of anger-hostility. In Siegman AW, Smith TW (eds), *Anger, Hostility and the Heart.* Hillsdale, NJ: Lawrence Erlbaum Associates, 1993, 43–66.
(21) Musante L, MacDougall JM, Dembroski TM, Costa PT: Potential for hostility and dimensions of anger. *Health Psychology.* 1989, *8:*343–354.
(22) Dujovne VF, Houston BK, Dujovne CA: Type A behavior, anger expression, and plasma lipid levels: A preliminary study. *Coronary Artery Disease.* 1992, *3:*1171–1176.
(23) Buss AH, Durkee A: An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology.* 1957, *42:*155–162.
(24) Smith TW, Frohm KD: What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley Ho scale. *Health Psychology.* 1985, *4:*503–520.
(25) Kim HJ, Kalkhoff RK: Changes in lipoprotein composition during the menstrual cycle. *Metabolism.* 1979, *28:*663–668.
(26) Siegman AW, Dembroski TM, Ringel N: Components of hostility and the severity of coronary artery disease. *Psychosomatic Medicine.* 1987, *49:*127–135.
(27) Spielberger CD, Johnson EH, Russell SF, et al: The experience and expression of anger: Construction and validation of an anger expression scale. In Chesney MA, Rosenman RH (eds), *Anger and Hostility in Cardiovascular and Behavioral Disorders.* Washington, DC: Hemisphere Publishing Company, 1985, 5–30.
(28) Costa PT, McCrae RR: *The NEO Personality Inventory.* Odessa, FL: Psychological Assessment Resources, 1985.
(29) Dembroski TM, Costa PT: Coronary-prone behavior: Components of the Type A pattern and hostility. *Journal of Personality.* 1987, *55:*212–235.
(30) SAS Institute, Inc: *SAS User's Guide: Statistics, Version 5 edition.* Cary, NC: SAS Institute, Inc., 1985.
(31) Suarez EC, Williams RB: The relationships between dimensions of hostility and cardiovascular reactivity as a function of task characteristics. *Psychosomatic Medicine.* 1990, *52:*558–570.
(32) Suarez EC, Harlan E, Peoples MC, Williams RB: Cardiovascular and emotional responses in women: The role of hostility and harassment. *Health Psychology.* 1993, *12:*459–468.
(33) Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG, Williams RB: The Cook–Medley Hostility Scale: Item content and ability to predict survival. *Psychosomatic Medicine.* 1989, *51:*46–57.
(34) Brindley DN, McCann BS, Niaura R, Stoney CM, Suarez EC: Stress and lipoprotein metabolism: Modulators and mechanisms. *Metabolism.* 1993, *42:*3–15.
(35) Dimsdale JE, Herd JA, Hartley LH: Epinephrine-mediated increases in plasma cholesterol. *Psychosomatic Medicine.* 1983, *45:*227–232.
(36) Suarez EC, Williams RB, Kuhn CM, et al: Biobehavioral basis of coronary-prone behavior in middle-aged men. Part II. Serum cholesterol, the Type A behavior pattern, and hostility as interactive modulators of physiological reactivity. *Psychosomatic Medicine.* 1991, *53:*528–537.
(37) Suarez EC, Harralson TL, Bates MP, Tyrey M, McNeer TCC: Neuroendocrine responses are associated with lipids in hostile women (Abstract). *Psychosomatic Medicine.* 1995, *57:*92.
(38) Siegler IC, Peterson BL, Barefoot JC, Williams RB: Hostility during late adolescence predicts coronary risk factors at midlife. *American Journal of Epidemiology.* 1992, *136:*146–154.