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Hostility during Late Adolescence Predicts Coronary Risk Factors at Mid-Life

Ilene C. Siegler,[1] Bercedis L. Peterson,[2] John C. Barefoot,[1] and Redford B. Williams[1]

Hostility, as measured by the Cook-Medley Hostility Scale of the Minnesota Multiphasic Personality Inventory, has been found to predict higher rates of both coronary heart disease and all-cause mortality. To evaluate one mechanism whereby hostility might contribute to health problems, the authors used regression models to determine whether hostility measured in college (1964–1966) predicted coronary risk factors assessed 21–23 years later (1987–1990) in 4,710 men and women. Of this group, 828 had lipids measured (1988–1991). Persons with higher hostility scores in college were significantly more likely at follow-up to consume more caffeine ($r = 0.043$), to have a larger body mass index ($r = 0.055$), to have higher lipid ratios ($r = 0.092$), and to be current smokers ($r = 0.069$) than those with lower hostility scores during college. Cross-sectional analyses found significant associations of contemporaneous hostility scores with the same four risk factors, as well as with alcohol consumption and hypertension (rs ranging from 0.043 to 0.117). These associations are large enough to have possible public health significance. We conclude that hostility may contribute to health problems through its influences on several coronary risk factors across the adult life span. *Am J Epidemiol* 1992;136:146–54.

body mass index; caffeine; cholesterol; exercise; hostility; hypertension; lipoproteins, HDL cholesterol; smoking

Hostility is a major focus of attention in research on the psychosocial precursors of coronary heart disease (1). Interest in hostility originated with work showing that hostility is a critical component of the Type A behavior pattern in predicting coronary heart disease risk (2–4). Measures of hostility based on both interview ratings and questionnaires correlate with coronary artery disease severity in cross-sectional studies (5–7), are associated with the progression of atherosclerosis in the common carotid artery (8) and, after controlling for known coronary heart disease risk factors, predict coronary heart disease incidence in prospective data (9–12). Hostility has also been related to other health outcomes, such as peripheral artery disease (13), functional health as rated by a physician (14), and premature mortality from all causes (11, 12, 14–16). Although

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Abbreviations: CARDIA, Coronary Artery Risk Development in Young Adults; HDL cholesterol, high-density-lipoprotein cholesterol; MMPI, Minnesota Multiphasic Personality Inventory; UNCAHS, University of North Carolina Alumni Heart Study.

[1] Behavioral Medicine Research Center and Department of Psychiatry, Duke University Medical Center, Durham, NC.

[2] Division of Biometry, Department of Community and Family Medicine, Duke University Medical Center, Durham, NC.

Reprint requests to Dr. Ilene Siegler, Box 2969, Duke University Medical Center, Durham, NC 27710.

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three prospective studies (17–19) have failed to find either crude or controlled associations between hostility and disease outcomes, the preponderance of evidence points to the importance of hostility as a psychological factor associated with coronary disease risk (20).

Three classes of related hypotheses might explain why higher levels of hostility are associated with adverse health outcomes. 1) Hostility might be a behavioral manifestation of an important biologic characteristic such as autonomic reactivity (2, 21). 2) Hostility may impact on other aspects of the psychosocial environment known to be important for health. For example, the behavior of hostile individuals might lead to more stressful encounters in daily life or undermine the functioning of their social support network (22, 23). 3) Hostile individuals may engage in high-risk health behaviors that may lead to a poorer risk factor profile (24, 25). This final class of explanations, the hostility-risk factor relation, is the focus of this paper.

Previous studies that have examined the relation between various measures of hostility and selected coronary heart disease risk factors have reported mixed results (26). In particular, previous research has found higher hostility associated with more smoking (10, 12), increased cholesterol (27–29), increased incidence of hypertension (11, 30), increased alcohol intake (12), decreased fitness (24), and increased body mass index (25). Other studies (10, 12, 17, 19, 25, 31) found no association between hostility and one or more of these same risk factors. However, no studies have reported that high levels of hostility are associated with reduced risk. There are many possible reasons for these different results, including the use of a variety of designs, samples, and hostility measures.

This study uses data from the University of North Carolina Alumni Heart Study (UNCAHS) to examine the hypothesis that hostility is associated with a broad array of risk factors. The UNCAHS allows a more definitive test than prior studies because the sample is sufficiently large, includes both men and women, and contains both prospective and cross-sectional data.

MATERIALS AND METHODS

Study sample

In the UNCAHS, longitudinal data are being obtained on 5,814 men and 1,224 women who completed the Minnesota Multiphasic Personality Inventory (MMPI) (32) while attending the University of North Carolina in Chapel Hill during the period 1964–1966. In response to the baseline enrollment questionnaire, initially mailed from April 1, 1987, to 5,546 located living persons, 4,710 individuals (84.9 percent) joined the study by February 28, 1990. Only 253 persons (4.6 percent) refused to join the study, and 583 (10.5 percent) did not return the enrollment questionnaire by February 28, 1990. Participation in the UNCAHS is predicted by age, sex, and having a degree from the University of North Carolina but not by hostility (33). These results indicate that there is no selection bias in the prospective component of the study due to hostility. The UNCAHS participants are described in table 1. The sample group is largely composed of white, well-educated, white-collar professionals, most of whom reside in the southeastern United States.

Hostility

The Cook-Medley Hostility Scale (34) is a 50-item subscale of the MMPI. The majority of scale items reflect cynical attitudes and mistrust of others (15, 22, 35, 36). The initial hostility scores were calculated from the administration of the full MMPI during the 1960s. Only the 50 hostility items were readministered at the second follow-up, and these data are available on 3,841 persons. Hostility scores were not included in the analyses if the MMPI validity scales (L, F) indicated that the test administration was invalid (T scores > 70) or if more that 10 percent of the items were unanswered. This resulted in the exclusion of 274 persons at initial measurement and 57 persons at follow-up.

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TABLE 1. Characteristics of 4,710 participants in the University of North Carolina Alumni Heart Study*

Variable	% of sample
Age at initial testing (1964–1966)	
≤21	92.7
>21	7.3
Sex	
Male	81.8
Female	18.2
Race	
White	99.1
Black	0.6
Other	0.4
Highest degree	
High school degree	5.6
Technical degree	1.4
Baccalaureate	45.2
Masters	23.4
Doctorate	24.2
Current employment status	
Full-time employment	89.0
Part-time employment	5.3
Homemaker	2.6
Student	0.6
Other	2.5
Current location	
North Carolina	52.3
Other Southeastern	21.5
Mid-Atlantic	8.9
New England	3.0
Midwestern	4.4
Western	8.3
Not United States	1.5

* Note: Questionnaires were mailed April 1, 1987, and those received by February 29, 1990, are included in this report.

Assessment of risk factors and health behaviors

Reports of health behaviors and risk factors were obtained by questionnaire. Smoking status was defined by whether or not the subject was a current smoker of cigarettes, with subjects who smoked only pipes or cigars categorized as nonsmokers. Alcohol use was calculated as the number of servings of beer, wine, and liquor in the past week. Caffeine use was defined as the number of servings of beverages with caffeine (e.g., coffee, tea, cola) consumed per day. Exercise was the average number of hours a week the respondent exercised or played sports. Presence of hypertension was defined as an answer of "yes" to the question, "Has a doctor ever told you that you have high blood pressure?" Body mass index was calculated as weight (kg)/height $(m)^2$.

Lipid profiles

A lipid panel was offered as part of the second follow-up data collection (July 14, 1988, to April 17, 1991), and 831 subjects chose to have their lipids measured. Respondents were instructed to fast for 12 hours before having blood drawn at a nearby location of Roche Biomedical Laboratories. Total cholesterol and high-density-lipoprotein (HDL) cholesterol were measured directly with automated enzymatic methods (37), and the ratio of total cholesterol to HDL cholesterol was used as the lipids risk factor (38) in the present study. Participants who had their lipids measured did not differ from the rest of the UNCAHS sample on hostility, age, alcohol use, hours of exercise, or prevalence of hypertension. However, they did consume less caffeine ($p < 0.0001$), had a lower mean body mass index ($p < 0.0001$), were less likely to be smokers ($p < 0.0001$), and were more likely to be female ($p = 0.007$). These differences suggest that the mean lipid levels in the subsample may underestimate the lipid levels in the full UNCAHS sample, since people who do not smoke, who consume less caffeine, and who have lower body mass indices have been shown to have lower levels of lipids (39–41). This would lead to a conservative estimate of the association between hostility and lipid levels, because any restriction in range reduces the magnitude of a correlation coefficient.

Data analysis

The association of hostility scores with each of the risk factors was tested in separate statistical models controlling for age and sex. An alpha of 0.01 was used for assessing the statistical significance of all tests. Analyses

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of prospective data used the initial (1960s) hostility score as the predictor variable, while cross-sectional analyses used the hostility score from the second follow-up (1988–1991) as the predictor variable. General linear regression models (42) were used to test the association of hostility scores with the continuous dependent variables: caffeine consumption, alcohol consumption, hours of exercise, body mass index, and lipids ratio (total cholesterol/HDL cholesterol). Logistic regression models (43) were used to assess the association of hostility scores with the dichotomous dependent variables, hypertension and smoking status. Interactions of age and sex with hostility were tested in all models. Additionally, the analyses were done both including and excluding the 32 respondents with verified coronary heart disease; however, as the results were unchanged, only the analyses including the 32 cases are reported.

RESULTS

Descriptive statistics for the variables used in the regression analyses are presented in table 2. The unequal sample sizes are due to missing data on the variables of interest. The means of the risk factors observed among our participants are close to those that would be expected for a well-educated, middle-aged group (44–46). The lower hostility score at follow-up is in keeping with data showing age differences in hostility levels (47).

Analyses of prospective data

As shown in table 3, higher hostility scores at age 19 predicted greater caffeine consumption, a larger body mass index, a

TABLE 2. Data of the 855 women and 3,855 men in the University of North Carolina Alumni Heart Study*

Variables	Mean ± standard deviation	Percentiles		
		25	50	75
	Women			
Initial hostility (814)†	15.2 ± 7.7	9.6	14.1	20.0
Follow-up hostility (766)	13.3 ± 6.3	8.5	12.0	17.0
Age at follow-up (855)	42.9 ± 3.0	41.4	42.1	43.1
Caffeine (851)‡	2.9 ± 2.4	1.0	2.5	4.0
Body mass index (850)§	22.8 ± 4.3	20.1	21.6	24.1
Exercise (845)‖	2.7 ± 2.5	1.0	2.0	4.0
Alcohol (825)¶	3.7 ± 5.2	0	2.0	5.0
Lipid ratio (177)#	3.6 ± 1.2	2.9	3.3	4.0
Smoking status (855)**	0.159			
Hypertension (846)††	0.063			
	Men			
Initial hostility (3,678)	18.4 ± 7.7	12.8	18.0	23.7
Follow-up hostility (3,075)	14.3 ± 6.5	10.0	14.0	18.0
Age at follow-up (3,855)	42.7 ± 1.9	41.7	42.4	43.1
Caffeine (3,838)	3.6 ± 2.6	2.0	3.0	5.0
Body mass index (3,778)	25.2 ± 3.3	23.1	24.7	26.6
Hours of exercise (3,727)	3.7 ± 3.3	1.5	3.0	5.0
Alcohol (3,727)	7.3 ± 9.3	0	4.0	10.5
Lipid ratio (651)	4.7 ± 1.3	3.7	4.5	5.5
Smoking status (3,844)	0.175			
Hypertension (3,804)	0.118			

* Initial values were measured from 1964 to 1966. Follow-up data were collected from 1987 to 1991.
† Numbers in parentheses, number of respondents with valid data on the variable.
‡ Caffeine was measured in servings/day.
§ Body mass index was calculated as weight (kg)/height $(m)^2$.
‖ Exercise was measured in hours/week.
¶ Alcohol is servings of beer, wine, or liquor in the past week.
# Lipid ratio is serum total cholesterol/high-density-lipoprotein cholesterol.
** Smoking status is the percentage of persons smoking at follow-up.
†† Hypertension is the percentage of persons reporting a diagnosis of high blood pressure.

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TABLE 3. Initial hostility (Ho) score, as predictor of risk factors, controlling for age and sex in the University of North Carolina Alumni Heart Study*

Variables	Test of Ho effect		Predicted values of dependent variable		Pearson's partial correlation of dependent variable with Ho
	F	*p*	At 20th percentile of Ho = 11	At 80th percentile of Ho = 24.5	
Caffeine (4,472)†, ‡	8.40	0.0038	3.43	3.63	0.043
Body mass index (4,416)§	13.41	0.0003	24.61	24.95	0.055
Exercise (4,457)‖	6.85	0.0089	3.37	3.59	0.039
Alcohol (4,339)¶	4.73	0.0297	6.38	6.88	0.033
Lipid ratio (792)#	6.66	0.0100	4.41	4.62	0.092
Smoking (4,481)**	21.43††	<0.0001	0.148	0.193	0.069
Hypertension (4,435)‡‡	0.10††	0.7531	0.105	0.108	0.004

* Initial values were measured from 1964 to 1966. Follow-up data were collected from 1987 to 1991.
† Numbers in parentheses, number of respondents used in the analysis.
‡ Caffeine was measured in servings/day.
§ Body mass index was calculated as weight (kg)/height $(m)^2$.
‖ Exercise was measured in hours/week.
¶ Alcohol is servings of beer, wine, or liquor in the past week.
# Lipid ratio is serum total cholesterol/high-density-lipoprotein cholesterol.
** Smoking status is the percentage of persons smoking at follow-up.
†† χ^2 value (not *F*).
‡‡ Hypertension is the percentage of persons reporting a diagnosis of high blood pressure.

greater probability of being a current smoker, a larger lipid ratio, and, somewhat surprisingly, more hours of exercise per week. Initial hostility scores did not significantly predict alcohol consumption or hypertension.

Effect sizes for the prospective association of hostility with each of the risk factors are illustrated in table 3 by partial Pearson correlation coefficients (controlling for age and sex). Since the dependent variables were not normally distributed, partial Spearman rank-order correlation coefficients were also calculated and were essentially the same (not shown). Table 3 also illustrates effect sizes with predicted risk factor values at the 20th and 80th percentiles of hostility (values of 11 and 24.5, respectively) as calculated from the regression models. For example, table 3 shows that an increase from the 20th to the 80th percentile of hostility is associated with a predicted increase in caffeine use of 0.2 servings per day.

Effect sizes for the association of hostility with the dichotomous dependent variables, smoking and hypertension, can be estimated with odds ratios calculated from the beta coefficients of the logistic models. Thus, the odds of being a smoker are 1.38 times greater (95 percent confidence interval 1.20–1.58) for subjects at the 80th percentile of hostility than for those at the 20th percentile. The odds ratio for the association of hostility with hypertension status, 1.03, was not significantly different from 1.

Finally, all but one of the interactions of age and sex with hostility were clearly nonsignificant, i.e., *p* values greater than 0.50. The association of hostility with smoking status was nonsignificantly larger in the women than in the men ($p = 0.02$). Overall, therefore, the associations of hostility with risk factors appear to be substantially the same in women and men.

Analyses of cross-sectional data

As in the prospective data, the cross-sectional data (see table 4) also showed hostility (measured at approximately age 42) to be significantly associated with caffeine consumption, body mass index, current smoking status, and lipid ratio. The association of hostility with alcohol consumption and hypertension, which were nonsignificant in the prospective data, achieved statistical significance in the cross-sectional data, such that the more hostile subjects drank more alcohol

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TABLE 4. Follow-up hostility (Ho) score as predictor of risk factors controlling for age and sex in the University of North Carolina Alumni Heart Study*

Variables	Test of Ho effect		Predicted values of dependent variable		Pearson's partial correlation of dependent variable with Ho
	F	*p*	At Ho = 11	At Ho = 24	
Caffeine (3,824)†, ‡	50.00	<0.0001	3.35	3.95	0.114
Body mass index (3,774)§	31.71	<0.0001	24.52	25.18	0.091
Exercise (3,814)‖	1.29	0.2568	3.52	3.40	−0.018
Alcohol (3,714)¶	10.16	0.0014	6.37	7.29	0.052
Lipid ratio (827)#	11.20	0.0006	4.44	4.75	0.116
Smoking (3,833)**	11.80††	0.0010	0.150	0.192	0.053
Hypertension (3,783)‡‡	7.02††	0.0081	0.090	0.117	0.043

* Initial values were measured from 1964 to 1966. Follow-up data were collected from 1987 to 1991.
† Numbers in parentheses, number of respondents used in the analysis.
‡ Caffeine was measured in servings/day.
§ Body mass index was calculated as weight (kg)/height $(m)^2$.
‖ Exercise was measured in hours/week.
¶ Alcohol is servings of beer, wine, or liquor in the past week.
# Lipid ratio is serum total cholesterol/high-density-lipoprotein cholesterol.
** Smoking status is the percentage of persons smoking at follow-up.
†† χ^2 value (not *F*).
‡‡ Hypertension is the percentage of persons reporting a diagnosis of high blood pressure.

and were more likely to report hypertension. Conversely, the prospective positive association of hostility with hours of exercise disappeared in the cross-sectional data.

As before, predicted risk factor values are presented at hostility values of 11 and 24.5. Since hostility scores were lower in the second follow-up than in the initial data collection, these values correspond, not to the 20th and 80th percentiles of hostility, but to the 37.8th and 93.4th percentiles. The odds ratios associated with the smoking and hypertension effects were, respectively, 1.34 (95 percent confidence interval 1.13–1.60) and 1.34 (95 percent confidence interval 1.08–1.67).

DISCUSSION

The major finding in this study is that a higher level of hostility, assessed in the 1960s, is associated with a pattern of risk factors in 1988–1991 that could place an individual at greater risk of developing coronary heart disease and other health problems. Those persons with higher hostility scores in college were more likely 21–23 years later to consume more caffeine, to have a larger body mass index, to have higher lipid ratios, and to be current smokers than those with lower hostility scores during college. The relations of hostility to these risk indicators were not modified by sex.

While the results in this paper demonstrate the existence of statistically significant relations between hostility and risk factors, one must ask if the magnitudes of the relations are of public health significance. Effects that on the surface appear to be small can often be of great importance. For example, the Physician's Aspirin Study (48), heralded as demonstrating the beneficial effects of aspirin on coronary risk, found a correlation between treatment and outcome of only 0.034.

There are several reasons to believe that the associations observed in the UNCAHS are meaningful. First, even small effects can have a very large impact if present over an extended period of time (49). Second, since risk factors often interact synergistically, the impact of multiple risk factors can be greater than that determined by simply adding up the risks of each individual factor (50). Third, our sample includes well-educated, middle-aged, white adults, who would be

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expected to be more health conscious then the average person. The association of hostility with coronary heart disease risk factors may be even stronger in a more heterogeneous sample. The cross-sectional Coronary Artery Risk Development in Young Adults (CARDIA) Study (51), which included both blacks and whites with a wider range of education, did find a larger impact of hostility on smoking.

Data from the UNCAHS and the CARDIA Study can also help to identify potential mechanisms relating hostility to body mass index and lipid levels. Hostility scores were associated with higher caloric intake but not with body mass index in the 18- to 30-year-old CARDIA Study subjects (51). Similarly among UNCAHS participants, hostility and body mass index in college were not significantly correlated ($r = 0.02$; $p = 0.45$). However, because hostility was a significant predictor of lipids and body mass index in the 1980s, our results suggest that the association of hostility with increased caloric intake, as documented in the CARDIA Study, may lead to lipid and body mass index increases at mid-life.

At the neurobiologic level, prior research has found low brain serotonin associated with increased aggressive behavior (52), alcohol use (53), food consumption (54), and smoking (55). We speculate, therefore, that a functional deficiency of serotonin may be responsible for the observed associations between hostility and ingestive behaviors.

A potential limitation of this study is that the data (except the lipids) are based on self-report. Mail surveys with similar populations have conducted special validity studies on the self-report of body mass index (56) and hypertension (57). Results indicate that, while such self-reports are generally accurate, respondents tend to underestimate their weight, and some individuals may not know that they have elevated blood pressure. To the extent that risk factors are underreported, an information bias results such that the probability of finding a significant association is reduced (58, 59). Therefore, the associations between hostility and the risk factors found in this study sample are probably conservative estimates. Even so, bias might occur if reporting accuracy was related to hostility. However, there are no studies in the literature suggesting a relation between hostility and accuracy of self-reports.

While the lipid data are not subject to any potential self-report limitation, they are only available on a subset of the UNCAHS. Because that subset differed from the total sample on characteristics that have been associated with lower lipids, the potential limitations on the generality of the findings should be kept in mind.

These findings, with the data from the CARDIA Study, lead us to conclude that hostility may contribute to health problems through its influences on several coronary risk factors across the adult life span.

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