

## Anger Proneness Predicts Coronary Heart Disease Risk Prospective Analysis From the Atherosclerosis Risk In Communities (ARIC) Study

Janice E. Williams, PhD, MPH; Catherine C. Paton, MSPH; Ilene C. Siegler, PhD, MPH;  
Marsha L. Eigenbrodt, MD, MPH; F. Javier Nieto, MD, PhD; Herman A. Tyroler, MD

**Background**—Increased research attention is being paid to the negative impact of anger on coronary heart disease (CHD).

**Methods and Results**—This study examined prospectively the association between trait anger and the risk of combined CHD (acute myocardial infarction [MI]/fatal CHD, silent MI, or cardiac revascularization procedures) and of “hard” events (acute MI/fatal CHD). Participants were 12 986 black and white men and women enrolled in the Atherosclerosis Risk In Communities study. In the entire cohort, individuals with high trait anger, compared with their low anger counterparts, were at increased risk of CHD in both event categories. The multivariate-adjusted hazard ratio (HR) (95% CI) was 1.54 (95% CI 1.10 to 2.16) for combined CHD and 1.75 (95% CI 1.17 to 2.64) for “hard” events. Heterogeneity of effect was observed by hypertensive status. Among normotensive individuals, the risk of combined CHD and of “hard” events increased monotonically with increasing levels of trait anger. The multivariate-adjusted HR of CHD for high versus low anger was 2.20 (95% CI 1.36 to 3.55) and for moderate versus low anger was 1.32 (95% CI 0.94 to 1.84). For “hard” events, the multivariate-adjusted HRs were 2.69 (95% CI 1.48 to 4.90) and 1.35 (95% CI 0.87 to 2.10), respectively. No statistically significant association between trait anger and incident CHD risk was observed among hypertensive individuals.

**Conclusions**—Proneness to anger places normotensive middle-aged men and women at significant risk for CHD morbidity and death independent of the established biological risk factors. (*Circulation*. 2000;101:2034-2039.)

**Key Words:** coronary disease ■ stress ■ epidemiology

The deleterious effects of anger on cardiovascular health is increasingly gaining attention in the research literature. Evidence for an anger–coronary heart disease (CHD) association has been derived from studies that use different measures of anger, different CHD end points, and different study designs.<sup>1–5</sup> Trait anger, the focus of the present study, is a relatively stable personality trait that is manifested in the frequency, intensity, and duration of the anger experience.<sup>6</sup> Persons with high compared with low trait anger have rage and fury more often, more intensely, and with longer-lasting episodes. Persons with high trait anger, by virtue of their propensity toward anger and their long-term exposure to its pathophysiological sequelae, might be particularly susceptible to CHD. However, this association may be attenuated in persons under treatment for hypertension because many pharmacological agents used to treat this condition may reduce the expression of CHD and the physiological responses to stress.

Studies have linked this personality attribute to CHD risk factors. Johnson et al,<sup>7</sup> in cross-sectional analyses, showed

that trait anger was positively associated with the LDL/HDL ratio and triglyceride levels. Durel et al<sup>8</sup> reported a positive association between trait anger and blood pressure (systolic and diastolic) and between trait anger and cardiovascular reactivity. In a prospective analysis, Markovitz et al<sup>9</sup> demonstrated that increases in trait anger predicted increases in blood pressure from baseline to 3 years of follow-up. Although these studies support a positive relation between trait anger and CHD risk factors, there are no published studies of the association between trait anger and incident CHD events.

The prospective association between anger (of any type) and CHD was described initially by investigators from the Framingham Heart Study, who reported that suppressed anger independently predicted the 8-year incidence of CHD among both men and women.<sup>10</sup> More recently, Kawachi et al<sup>5</sup> examined whether problems controlling one's anger would predict CHD events among men in the Normative Aging Study. The investigators reported a 3-fold increase in CHD risk among individuals with the greatest difficulty controlling their anger as compared with those with the least. Addition-

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From the University of North Carolina (J.E.W., C.C.P., M.L.E., H.A.T.), Chapel Hill, NC; Duke University Medical Center (I.C.S.), Durham, NC; and Johns Hopkins University, Baltimore, Md (F.J.N.).

Correspondence to Janice E. Williams, PhD, MPH, Cardiovascular Disease Epidemiology Unit, School of Public Health, University of North Carolina, 137 E Franklin St, Suite 306, Chapel Hill, NC 27514. E-mail janice.williams@sph.unc.edu

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ally, they found a dose-response relation between the degree of difficulty with anger control and CHD risk and shorter CHD-free survival among participants with increasingly greater anger control problems.

The present study investigated, in a prospective design, the association of trait anger to CHD risk in a large bi-ethnic cohort of middle- to older-aged men and women enrolled in the Atherosclerosis Risk In Communities (ARIC) study.<sup>11</sup> We assessed whether there was a dose-response relation between level of anger and CHD risk, whether the probability of CHD-free survival differed depending on the level of anger, and whether the trait anger-CHD association differed by hypertensive status.

## Methods

### Study Population

The study participants were white and black men and women who completed the Spielberger Trait Anger Scale ( $n=14\,348$ ) at the second examination of the ARIC study. ARIC is a population-based prospective study of cardiovascular disease designed with both cohort and surveillance components. The ARIC participants, aged 45 to 64 years at baseline, were recruited from random probability samples in the US communities of Washington County, Maryland; suburban Minneapolis, Minn; Forsyth County, North Carolina; and Jackson, Miss.<sup>11</sup> Blacks were oversampled in Forsyth County, North Carolina, and exclusively sampled in Jackson, Miss. Since the baseline evaluations in 1987 to 1989, triennial clinical examinations and annual morbidity and mortality surveillance have been conducted.

Of the 14 348 individuals examined at the second visit (a return rate of 92.9% from baseline to the second clinic visit), 13 208 were free of clinically manifest CHD (defined as having no history of MI or cardiac revascularization procedures or ECG evidence of MI). Excluded from the analyses were 38 individuals with a racial/ethnic identity other than black or white; 40 with missing data on hypertensive status; and 144 with incomplete responses on the anger questionnaire. After these sequential exclusions, a total of 12 986 participants remained for the current analyses.

### Assessment of Trait Anger

During the second clinical examination, participants completed the Health-Life Profile, which contained the 10-item Spielberger Trait Anger Scale.<sup>6</sup> On this scale, respondents rated their typical experience with anger on a 4-point anchor: Almost Never=1, Sometimes=2, Often=3, and Almost Always=4. An overall anger score was obtained by summing each of the individual items. See the Appendix for the questionnaire items.

### Assessment of CHD Risk Factors

All physiological measurements and laboratory assessments were made by trained technicians who used standardized protocols. Three sitting blood pressure measurements were taken after a 5-minute rest period with the use of a random-zero sphygmomanometer. The average of the second and third readings was used as the measure of blood pressure reported in this study. Hypertension was defined as diastolic blood pressure  $\geq 90$  mm Hg, systolic blood pressure  $\geq 140$  mm Hg, or use within the past 2 weeks of medication to lower blood pressure. HDL cholesterol was measured enzymatically after precipitation of LDL-containing proteins using dextran-magnesium. LDL cholesterol was calculated with the use of the Friedewald formula. Diabetes was defined as a fasting serum glucose level  $\geq 140$  mg/dL and/or a history of diabetes, insulin therapy, or oral hypoglycemic medication use. Waist-to-hip ratio was the ratio of waist girth, abdominal circumference measured at the umbilicus, to hip girth, measured at the maximal gluteal protrusion. Data on alcohol consumption, cigarette smoking, and level of educational attainment were obtained by self-report. Participants were asked to bring all

medication (prescription and over-the-counter) taken within the 2 weeks immediately preceding the clinic visit. Each medication was coded by trained and certified interviewers with the use of a computerized medication classification system.

### CHD Events

Incident CHD events reported in this study occurred in the follow-up period from the participant's clinic visit in 1990 to 1992 through December 31, 1995, for a median of 53 months (maximum 72). An incident CHD event was defined as one of the following: acute MI/fatal CHD, silent MI, or cardiac revascularization procedure. Hospital CHD events were identified and validated through abstraction of death certificates and hospital discharge summaries. Data gathered on in-hospital events included presenting symptoms, ECG readings, cardiac enzyme levels, and other supportive diagnostic indicators. A maximum of three 12-lead ECG tracings were read by the University of Minnesota ECG Reading Center, with the use of the Minnesota code. The criteria for MI were based on a combination of chest pain, cardiac enzymes, ECG changes, and autopsy findings. Diagnoses were made with the use of a computerized algorithm and physician review system. Additional information was gathered on out-of-hospital deaths from the decedent's next of kin and appropriate physician's and/or the coroner's or medical examiner's report. The CHD diagnostic classification was assigned by 2 physicians on the ARIC morbidity and mortality classification committee; discrepancies in the diagnostic classification were adjudicated by a third panel member. A more detailed description of the quality control procedures used in the ascertainment of CHD events has been reported previously.<sup>12</sup>

### Statistical Analyses

The overall anger score (a numeric score of 10 to 40) was coded as a 3-level categorical variable in which scores of 22 to 40 defined high trait anger, 15 to 21 moderate anger, and 10 to 14 low anger. The cut-points were comparable to those used in previously published work with the Spielberger scale.<sup>13,14</sup> The main regression analyses were fit by the use of 3 different methods of handling missing values on the anger scale to determine the most appropriate strategy: (1) deleting all observations with missing values, (2) imputing a value of 1 for all missing values, and (3) imputing the average for a participant's nonmissing responses. Because the results from all 3 methods were similar, the analyses were restricted to observations with complete trait anger protocols. The association between trait anger and incident CHD was determined with the use of Cox proportional hazards regression, in which anger was entered into the model as an indicator variable. Heterogeneity of effect was examined by each of 11 covariates: age, race/ethnicity, sex, educational level, HDL cholesterol level, LDL cholesterol level, hypertensive status, diabetes status, smoking status, and alcohol drinking status; only interaction with hypertensive status was statistically significant ( $\chi^2_{(2,df)}=5.91$ ,  $P=0.05$ ). The regression analyses were stratified by hypertensive status to test the hypothesis that persons with hypertension would be less likely to demonstrate an association between trait anger and incident CHD. The models were fit separately for combined CHD and for "hard" events, adjusting first for age, and second for the additional covariates. To assess the presence of a linear trend in CHD risk, anger was entered into each regression equation as a continuous variable. Crude probabilities of CHD event-free survival were determined by the Kaplan-Meier product-limit method. When building the survival curves, deaths from all other causes were censored at the date of death.

## Results

A cardiovascular disease risk factor profile for the cohort at the first follow-up examination (1990 to 1992) is presented in Table 1. The mean Spielberger trait anger score was 16.0 (SD 3.7; range 10 to 40); 7.7% of the cohort scored in the high trait anger range, 55.2% in the moderate range, and 37.1% in the low range. High scorers were slightly younger, more likely to be men, and to have less than a high school

**TABLE 1. Distribution of Population Characteristics by Level of Trait Anger: ARIC Study, 1990 to 1992**

	Spielberger Trait Anger Scores			<i>P</i> *
	Low, 10–14	Moderate, 15–21	High, 22–40	
Participants, n (%)	4821 (37.1)	7165 (55.2)	1000 (7.7)	
Age, y, mean (SD)	57.3 (5.8)	56.6 (5.7)	56.3 (5.8)	<0.001
Male, n (%)	2018 (41.9)	3078 (43.0)	464 (46.4)	0.03
Less than high school education, n (%)	1058 (22.0)	1358 (19.0)	292 (29.2)	<0.01
White, n (%)	3481 (72.2)	5545 (77.4)	731 (73.1)	<0.01
Current smokers, n (%)	866 (18.0)	1706 (23.8)	311 (31.1)	<0.01
Current drinkers, n (%)	2500 (51.9)	4288 (59.9)	612 (61.2)	<0.01
Hypertensive, n (%)	1711 (35.5)	2434 (34.0)	367 (36.7)	0.09
Diabetic, n (%)	548 (11.4)	725 (10.2)	136 (13.6)	<0.01
Plasma LDL cholesterol, mg/dL, mean (SD)	133.1 (37.1)	133.2 (36.5)	132.1 (36.5)	0.66
Plasma HDL cholesterol, mg/dL, mean (SD)	50.4 (16.7)	50.0 (16.8)	49.0 (17.2)	0.05
Waist-to-hip ratio, mean (SD)	0.918 (0.083)	0.925 (0.081)	0.939 (0.081)	<0.001

\*One-way ANOVA test for comparison of means or  $\chi^2$  test of association.

education compared with participants who were moderate or low scorers. Further, high scorers were more likely to be smokers and drinkers, with slightly lower HDL cholesterol levels and higher waist-to-hip ratios than moderate or low scorers. A total of 416 individuals had CHD events in the 72-month follow-up period (median 53). The absolute risk of incident CHD in the population as a whole was 7.4 events per 1000 person-years; the attributable risk for the high anger state was 4.0 events per 1000 person-years.

In the entire cohort, the age-adjusted hazard ratio (HR) of CHD (95% CI) was 1.73 (95% CI 1.25 to 2.40) for high versus low trait anger and 1.12 (95% CI 0.91 to 1.38) for moderate versus low anger. The corresponding multivariate-adjusted HRs were 1.54 (95% CI 1.10 to 2.16) and 1.10 (95% CI 0.88 to 1.36). Similarly, the age-adjusted HR for “hard” events was 1.75 (95% CI 1.17 to 2.64) for high versus low trait anger and 1.04 (95% CI 0.79 to 1.35) for moderate versus low anger. The corresponding multivariate-adjusted HRs were 1.63 (95% CI 1.07 to 2.48) and 1.08 (95% CI 0.82 to 1.43).

No racial/ethnic differences were found in the risk of incident CHD as the result of trait anger when this association was adjusted for the traditional CHD risk factors. Among whites, the age-adjusted relative hazard for combined CHD and for “hard” events was statistically significant, but when adjusted for the additional covariates, these results did not remain significant [1.51 (95% CI 0.88 to 2.59) and 1.50 (95% CI 1.01 to 2.23)]. Among blacks, neither the age- nor the multivariate-adjusted results were significant.

In contrast to the racial/ethnic comparisons, there were statistically significant differences in the hazard ratios for the association of trait anger and incident CHD among normotensive and hypertensive cohort members. Results of the proportional hazards regression models for both groups are presented in Table 2. Among normotensive individuals, the

risk of combined CHD and of “hard” events increased monotonically as a function of trait anger. For combined CHD, the age-adjusted HR indicated that high anger scorers were 2.61 times more likely to have an event than low anger scorers. In addition, a 40% greater risk was observed among moderate compared with low anger scorers (probability value for linear trend 0.0006), although this HR was marginally significant. The relative risks were attenuated slightly after multivariate adjustment, but the patterns of risk and significance levels remained strong. The age-adjusted relative risk of high versus low anger for “hard” events was higher than that for combined CHD and similar for moderate versus low anger (probability value for linear trend 0.01). Again, the multivariate-adjusted point estimates for this subgroup were attenuated but remained significant (probability value for linear trend 0.02). These results indicate that the risk of “hard” events among high anger normotensive individuals, excluding those with only revascularization or silent myocardial infarction (MI), was nearly 3 times that of their low anger counterparts. Among hypertensive individuals, none of the HRs was statistically significant. In both CHD event categories, the pattern of risk for the age- and multivariate-adjusted models was similar.

The probability of CHD event-free survival was significantly lower among hypertensive than normotensive individuals ( $\chi^2_{(1\ df)}=71.6$ ,  $P<0.0001$ ). Among normotensives, the results of the log likelihood ratio tests indicated that CHD event-free survival differed significantly among high, moderate, and low anger scorers ( $\chi^2_{(2\ df)}=13.7$ ,  $P=0.001$ ) (Figure 1). The probability of CHD event-free survival was significantly lower among individuals reporting high trait anger compared with those reporting low anger ( $\chi^2_{(1\ df)}=15.2$ ,  $P=0.0001$ ) and compared with those reporting moderate anger ( $\chi^2_{(1\ df)}=8.0$ ,  $P=0.005$ ). Moderate anger scorers were marginally different from low scorers ( $\chi^2_{(1\ df)}=3.5$ ,  $P=0.06$ ).

**TABLE 2. Hazard Ratios (95% CI) for Association Between Trait Anger and CHD by Hypertensive status: ARIC Study, 1990 to 1995**

	Spielberger Trait Anger Scores							
	Normotensives				Hypertensives			
	Low, 10–14	Moderate, 15–21	High, 22–40	<i>P</i> *	Low, 10–14	Moderate, 15–21	High, 22–40	<i>P</i> *
Population, n	3110	4731	633		1711	2434	367	
CHD events combined†								
Individuals with incident events, n	53	110	27		89	116	21	
Age adjusted	1.00	1.40 (1.01–1.94)	2.61 (1.64–4.16)	0.0006	1.00	0.96 (0.73–1.27)	1.14 (0.71–1.84)	0.73
Multivariate adjusted‡	1.00	1.32 (0.94–1.84)	2.20 (1.36–3.55)	0.02	1.00	0.95 (0.71–1.28)	1.08 (0.67–1.76)	0.73
“Hard” CHD events								
Individuals with incident events, n	31	63	18		60	71	13	
Age adjusted	1.00	1.36 (0.89–2.10)	2.97 (1.66–5.31)	0.005	1.00	0.88 (0.62–1.24)	1.05 (0.58–1.92)	0.85
Multivariate adjusted	1.00	1.35 (0.87–2.10)	2.69 (1.48–4.90)	0.02	1.00	0.93 (0.65–1.34)	1.00 (0.54–1.86)	0.98

\*Probability value for linear trend.

†Acute MI/fatal CHD, cardiac revascularization procedures, or silent MI.

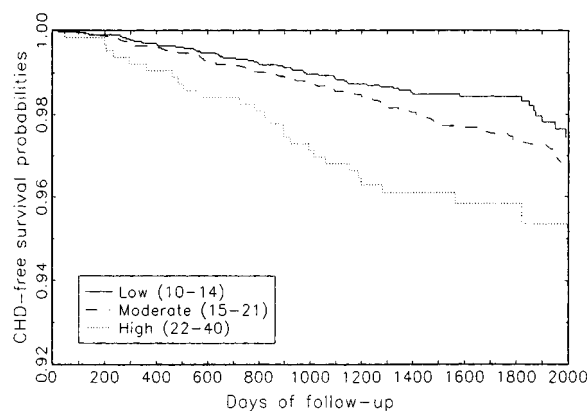
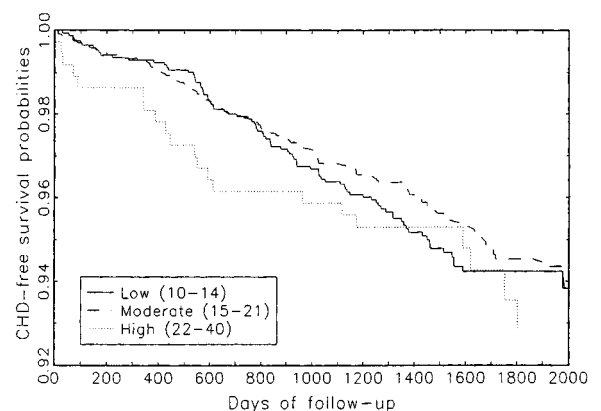
‡Adjusted for age, race/ethnicity, level of educational attainment, sex, waist-to-hip ratio, drinking status (never, current, former), smoking status (never, current, former), plasma LDL and HDL cholesterol levels, and diabetes status.

High, moderate, and low anger scorers among hypertensive individuals did not differ significantly in their probability of CHD event-free survival ( $\chi^2_{(2\ df)}=0.8$ ,  $P=0.66$ ) (Figure 2).

Because of heterogeneity of effect by hypertensive status, additional analyses were conducted to better understand the possible impact of medication use and blood pressure levels on the results obtained. As expected,  $\chi^2$  analyses indicated that significantly more hypertensive than normotensive individuals used  $\beta$ -adrenergic antagonists ( $\beta$ -blockers), calcium channel blockers, ACE inhibitors, diuretics, and other antihypertensive medication ( $P=0.001$ ). Hypertensive individuals were examined to determine whether medication use was related to trait anger status or to the incidence of CHD. High, moderate, and low anger scorers were significantly different from one another in their use of antianxiety ( $P<0.01$ ) and antidepressant ( $P=0.033$ ) medication but not in their use of antihypertensive agents (probability value range 0.16 to 0.78). A similar pattern was observed among normotensive individuals. Further, the association between medication use

and the incidence of CHD among hypertensive individuals was statistically significant for calcium channel blockers [HR=1.84 (95% CI 1.36 to 2.50)] and ACE inhibitors [1.58 (95% CI 1.16 to 2.15)] but not for  $\beta$ -blockers [1.29 (95% CI 0.96 to 1.73)], aspirin [0.88 (95% CI 0.65 to 1.19)], diuretics [0.97 (95% CI 0.74 to 1.26)], other antihypertensives [0.93 (95% CI 0.65 to 1.32)], antianxiety agents [0.72 (95% CI 0.40 to 1.27)] or antidepressants [1.38 (95% CI 0.83 to 2.29)]. Entering medication use in the multivariate-adjusted regression model did not substantially affect the HRs [0.99 (95% CI 0.58 to 1.67) and 0.87 (95% CI 0.64 to 1.19)] for high and moderate trait anger, respectively. Medication data were complete for 87.9% of the study participants.

The anger-CHD relation was examined among participants who were not taking antihypertensive medication but in whom high blood pressure levels (systolic blood pressure  $\geq 140$  or diastolic blood pressure  $\geq 90$ ) were observed ( $n=1094$ ). The multivariate-adjusted HR comparing high to low anger scorers was 0.65 (95% CI 0.22 to 1.93), indicating

**Figure 1.** CHD event-free survival probabilities among normotensive individuals by trait anger scores.**Figure 2.** CHD event-free survival probabilities among hypertensive individuals by trait anger scores.



no significant association between CHD and trait anger. In contrast, a significant anger-CHD association was observed among normotensive individuals who were not taking anti-hypertensive medications [2.35 (95% CI 1.43 to 3.86)].

### Discussion

In the entire cohort, high trait anger was associated with an increased risk of both combined CHD and of "hard" events. Among normotensive individuals, a high level of trait anger was associated with a slightly 2-times-greater risk of combined CHD and with a near 3-times-greater risk of "hard" events compared with a low level of anger. A dose-response relation was observed between anger and CHD in both event categories. CHD event-free survival was progressively shorter in low, moderate, and high anger groups. No significant association was found between trait anger and CHD risk among hypertensive individuals.

The precise biological mechanism and process by which anger influences CHD is yet to be clarified. One hypothesis is that the heightened sympathetic arousal and catecholamine secretion induced by anger are damaging to the heart and its vasculature and also play a role in the development of atherosclerotic lesions. Excessive circulating catecholamines (eg, epinephrine and norepinephrine) are known to cause direct damage to the endothelium and heart muscle<sup>15,16</sup> and to disrupt the electrical rhythm of the heart.<sup>17</sup> Further, studies have confirmed that the presence of catecholamines is associated with increased platelet adhesion and aggregation,<sup>18</sup> lipid mobilization,<sup>19</sup> and activation of macrophages,<sup>20</sup> each of which has been implicated in the complex process of atherogenesis, stemming from endothelial injury and ending in the formation of atherosclerotic lesions.<sup>21</sup>

Evidence supports a direct relation between the rupture of atherosclerotic plaque, occlusive thrombosis, and the onset of acute MI.<sup>22,23</sup> Increased attention is being paid to possible triggers of the sequence of events leading to MI because it has been demonstrated that these acute coronary syndromes do not occur randomly.<sup>24,25</sup> Anger has been examined and found to be one such trigger.<sup>1,26</sup> The relation is understood from the framework of the triggering hypothesis in which anger can be viewed as a trigger of "acute risk factors" that in turn disrupt vulnerable atherosclerotic plaques.<sup>27</sup> Acute risk factors are the physiological responses (eg, vasoconstriction, increased arterial pressure, increased platelet adhesion and aggregability, increased plasma coagulation, and fibrinolysis) that occur in close temporal proximity to an event and are responsible for the disruption of atherosclerotic plaque and the formation of an occlusive thrombus.<sup>27</sup> When the disruption causes injury to the intimal and medial layers of the artery (type III lesion), occlusive thrombosis results and, within a brief period of time, acute MI or sudden death.<sup>27,28</sup>

The observed lack of association between anger and CHD risk among hypertensive individuals might have been due to the cardioprotective effects of medications that lower CHD morbidity and mortality rates. As expected, hypertensive individuals were found to use significantly more  $\beta$ -blockers, ACE inhibitors, calcium channel blockers, diuretics, and other antihypertensive agents than normotensive individuals. These medications, by preventing physiological responses

such as vasoconstriction, thrombus formation, cardiac arrhythmias, and plaque disruption, might have forestalled a CHD event in hypertensive individuals who took them regularly. Two previous studies of the anger-CHD relation examined possible effect modification by the use of  $\beta$ -blockers and obtained contradictory results.<sup>1,5</sup> One study found a lower risk of CHD among regular users and the second did not. Effect modification by aspirin use was also examined in those studies. In both, CHD risk was lower among aspirin users than among nonusers. Though we did not assess effect modification of CHD risk by aspirin use, the groups of hypertensive individuals and normotensive individuals in our study had similar proportions of individuals who took aspirin regularly.

We examined participants who were not taking antihypertensive medication but in whom blood pressure levels were high. Among them, we found no significant CHD risk associated with anger. Therefore, an alternative explanation for the lack of an observed anger-CHD association among hypertensive individuals may be that proneness to anger confers little or no additional CHD risk among individuals with high blood pressure, despite their medication use.

A strength of the present study is its prospective design. Because participants were free of clinically manifest CHD at baseline, the temporal ambiguity in the anger-CHD relation was virtually eliminated. Another strength of this study is that it is population-based, consisting of both black and white participants. To our knowledge, this is the first bi-ethnic, prospective study of the relation of anger to CHD, and in our study no racial/ethnic differences were observed. In addition, a relatively large number of events occurred in the observation period, and these events were validated according to standardized criteria. The classification of disease was based on data abstraction from medical records and discharge summaries, which, admittedly, is a process subject to error depending on the completeness and accuracy of data contained in the records. Another potential weakness of this study is the relatively marginal probability value of the hypertension interaction ( $P=0.05$ ) in reference to the multiple stratifications that were conducted. However, this interaction is rather strong (despite the marginal probability value), as indicated by the nearly 3-fold increase in incident CHD risk in normotensives, while the association is entirely absent in individuals with hypertension.

In conclusion, these findings suggest that in addition to any adverse personal and social consequences that may accrue from having frequent and intense anger, there may be unfavorable cardiovascular-related outcomes as well. Anger proneness as a personality trait may place normotensive middle-aged men and women at significant risk for CHD morbidity and death independent of the established biological risk factors.

### Appendix

#### Spielberger Trait Anger Scale

- (1) I am quick tempered.
- (2) I have a fiery temper.
- (3) I am a hotheaded person.
- (4) I get angry when I am slowed down by others' mistakes.

- (5) I feel annoyed when I am not given recognition for doing good work.
- (6) I fly off the handle.
- (7) When I get angry, I say nasty things.
- (8) It makes me furious when I am criticized in front of others.
- (9) When I get frustrated, I feel like hitting someone.
- (10) I feel infuriated when I do a good job and get a poor evaluation.

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