

# Hostile Behaviors Predict Cardiovascular Mortality Among Men Enrolled in the Multiple Risk Factor Intervention Trial

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**Background**—Hostility is associated with incident coronary disease in most large population-based studies, but little is known about its association with cardiovascular disease (CVD) mortality in high-risk individuals. The aim of this study was to assess the association of hostility with CVD mortality in the subsequent 16 years in the Multiple Risk Factor Intervention Trial (MRFIT) participants and to explore the influence of hostility in the subset that had a nonfatal CVD event during the trial.

**Methods and Results**—We coded the Structured Interview responses of 259 men who died of CVD during the 16 years of follow-up and 259 matching living control subjects. Signs of hostility were assessed by use of the Interpersonal Hostility Assessment Technique. Matching was based on center, intervention group, age, race, and interviewer; covariates included study entry diastolic blood pressure, cholesterol, smoking status, and nonfatal CVD event during the trial. High-hostile men were more likely to die of CVD than were low-hostile men. Adjusted odds ratio (OR) and 95% confidence intervals (CIs) were 1.61, 1.09 to 2.39. After the trial, high-hostile men who also had a nonfatal event during the trial were particularly likely to die of CVD, OR, 5.06, 1.42 to 8.22, compared with low-hostile men without a nonfatal event during the trial.

**Conclusions**—Hostility may be a risk factor for CVD mortality among high-risk men. Interventions aimed at anger management and stress reduction along with risk factor modification may be useful for hostile patients. (*Circulation*. 2004;109:66-70.)

**Key Words:** hostility ■ survival ■ etiology ■ risk factors ■ cardiovascular diseases

Hostility is multidimensional and includes mistrustful attitudes, aggressive behavior, and frequent angry feelings.<sup>1</sup> A recent review of the associations between psychosocial factors and risk for fatal coronary heart disease (CHD) or nonfatal myocardial infarction (MI) in prospective studies that included at least 500 healthy participants found 5 studies of hostility.<sup>2</sup> Three reported positive associations of CHD with hostile attitudes, 1 with feelings of anger, and 1 a null association. Also consistent with hostility as a risk factor are data suggesting that self-reported hostile attitudes are associated with atherosclerosis in the carotid and coronary arteries<sup>3,4</sup> and calcification in the coronary arteries,<sup>5</sup> and retrospective reports of anger are linked to the triggering of clinical coronary events.<sup>6</sup>

Less clear is the mortality experience of hostile coronary patients or those at highest risk for CHD with no frank disease. Hostile attitudes predicted recurrent nonfatal CHD but not CHD death among women who already had been diagnosed with CHD.<sup>7</sup> Among Finnish men who were hypertensive and had coronary disease, reports of anger were related to cardiovascular disease (CVD) and all-cause mortality.<sup>8</sup> In the Atherosclerosis Risk in Communities Study,

angry feelings predicted CHD but only among normotensives.<sup>9</sup> Finally, clinical ratings of overall potential for hostility based on behaviors displayed during a semistructured interview predicted a combined index of nonfatal MI (n=130) and CHD death (n=62) among high-risk men during the 7 years of the active phase of the Multiple Risk Factor Intervention Trial (MRFIT<sup>10</sup>).

The present study describes the association of hostility in relation to CVD mortality during the 7 years of the active phase of the trial and the subsequent 9 years of follow-up in MRFIT. Rather than basing the assessment of hostile behaviors on an overall clinical judgment as in the analysis of the occurrence of nonfatal MI and CHD death during the trial,<sup>10</sup> we used a scoring system called the Interpersonal Hostility Assessment Technique (IHAT), which uses highly specific criteria applied to each unit of verbal interaction, thereby reducing subjectivity of the ratings.<sup>11</sup> The mortality experience of men was evaluated in relation to whether death occurred during or after the trial and in light of the men's nonfatal CVD morbidity during the trial. Thus, the present article differs from the previous study of the MRFIT sample in 3 ways: (1) it focuses on CVD mortality as the outcome

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rather than on CHD death and nonfatal MI; (2) it examines the IHAT system, which previously had been associated with atherosclerosis documented by angiography in cross-sectional studies<sup>11,12</sup>; and (3) it compares the short-term (during the trial) and long-term (9 years after the trial) effects of hostility, which may be important in understanding mechanisms that might account for the effects of psychosocial risk.

## Methods

### Participants

MRFIT was composed of 12 866 men 35 to 57 years old without preexisting CHD. To qualify for the study, subjects had to rank in the upper 10% to 15% of risk for CHD on the basis of serum cholesterol, diastolic blood pressure, and cigarette smoking. Participants were randomly assigned to usual care or a special intervention group. The special intervention group received dietary instructions designed to alter eating patterns to reduce the intake of saturated fats and to reduce blood cholesterol levels, a smoking cessation program, and stepped-care drug therapy for high blood pressure. Details of the intervention program are available elsewhere.<sup>13</sup>

Participants at 8 study sites (n=3110) participated in a substudy assessing type A behavior via a semistructured interview developed by Rosenman.<sup>14</sup> The interview asked about work involvement, competitiveness, time urgency, anger, and anger expression, and the questions were asked in a formal, business-like fashion as opposed to a supportive, therapeutic interview. The interviews were administered and audiotaped by trained interviewers who met interview and assessment criteria, details of which are explained more fully elsewhere.<sup>15</sup>

During the course of the trial (mean, 7.1 years) and the posttrial period (an additional mean of 8.9 years), 259 men who had been interviewed died of CVD. Each of the case patients (cases) was randomly matched to control subjects (controls) alive throughout the follow-up period on the basis of age at baseline (within 1 year); group assignment, ie, usual care versus special intervention group; study site; race (all but 9); and interviewer (all but 3). The best-matched control was included for the 12 cases that did not match on race and interviewer.

### Measurement of Hostility

Hostility during the interview was assessed by use of IHAT.<sup>11</sup> There are 4 components to the assessment: irritation, indirect challenge, direct challenge, and hostile withhold/evade. Irritation was scored for irritated tone, impatience, or exasperation with the interviewer or interviewer, aroused reliving of negative events, condescension or snide remarks, harsh generalizations, and punched words with angry emphasis. Indirect challenge was scored for comments that indirectly challenged the question by implying that the answer was obvious or the interviewer was stupid for having asked it. Direct challenge was scored when the participant directly and explicitly challenged the question or the interviewer. Hostile withhold/evade was scored when the respondent avoided or refused to answer a question with associated hostile tone and intent not to answer. This category was scored only when it was clear that the participant was being difficult and not merely ambivalent about the answer.

Each component was scored during every speaking turn in the interview according to established criteria. The summary hostile behavior score is the total of the 4 components averaged across the number of questions asked during the course of the interview. This yields an average or per item score, thereby adjusting for variability in the length of the interviews. The total IHAT ratings in this sample were positively skewed (range, 0 to 0.85; median, 0.06), with 10.2% showing no hostile behaviors. Because of the small number of cases through year 16, we used a median split to provide nearly equal groups for comparison. However, we also conducted analyses dividing the participants into 4 nearly equal groups based on the quartiles of the distribution of hostility scores (ie, 0 through 0.0218, 0.0219 to 0.0570, 0.0571 to 0.13, and 0.14 to 0.90).

In the present study, once the primary assessors were trained to an acceptable level of interrater reliability, each rater scored half the sample, with 10% of the interviews being scored by both assessors. Interclass correlation for the 2 raters (T.L.H. and K.F.H.) scoring the interviews in common was 0.76. Raters were blind to the case/control status.

Total IHAT ratings were associated with previous clinical ratings of Potential for Hostility on the basis of an overall clinical judgment for those interviews with both ratings available in MRFIT,  $r(153)=0.32$ ,  $P<0.001$ . IHAT scores have been stable over a 4-year period in other samples (intraclass  $r=0.69$ <sup>16</sup>) and seem to measure a relatively stable predisposition to respond in a consistent manner.

### Morbidity and Mortality Ascertainment

Before the end of the trial in February 1982, deaths were ascertained by use of next-of-kin interviews, routine follow-up of missed clinic visits, responses to postcards sent to participants, and searches of publicly accessible files of deceased persons. Cause of death was assigned by a 3-member panel of cardiologists not associated with MRFIT and blind to the participants' group assignment. Since February 1982, vital status has been ascertained by matching identifying information reported by participants at the time of enrollment with the National Death Index. The search of the National Death Index was for all deaths through December 1990 and is considered to be essentially 100% complete.<sup>17</sup> To determine cause of death, death certificates were collected and coded independently by 2 nosologists using the ICD-9.<sup>18</sup> Disagreements between the 2 nosologists were adjudicated by a third nosologist. The ICD-9 codes corresponding to the cause-specific mortality categories considered in this study are reported elsewhere.<sup>18</sup>

The following risk factors were selection factors for entering MRFIT and were considered as covariates: diastolic blood pressure defined as the average of 2 random-zero manometer readings, serum cholesterol concentration, and cigarette smoking (yes versus no) measured at screening. Further details regarding these assessments have been published elsewhere.<sup>19</sup>

As in previous MRFIT publications,<sup>19</sup> a nonfatal cardiovascular event during the trial was defined as angina by Rose questionnaire, intermittent claudication by Rose questionnaire, congestive heart failure, peripheral arterial occlusive disease, left ventricular hypertrophy by ECG, impaired renal function, accelerated hypertension, coronary artery bypass surgery, stroke, and ECG evidence or clinical evidence of MI.

### Statistical Analyses

Mortality was analyzed by use of dependent logistic regression models for matched case-control pairs. Additional analyses adjusted for the above covariates plus the occurrence of nonfatal CVD events during the trial. We also partitioned cases according to whether they had died during the trial or the posttrial period or whether cases and controls had a previous nonfatal CVD event during the trial and examined the effects of hostility for those with and without a previous nonfatal CVD event during the trial. Because of evidence that IHAT ratings may interact with smoking status in their relationship to CVD,<sup>12</sup> we tested for the interaction between hostility and smoking status.

## Results

Table 1 shows baseline characteristics and occurrence of a nonfatal CVD event during the trial for cases and controls. As expected, variables used to match cases and controls (ie, age at baseline, race, and MRFIT group assignment) did not differ significantly for cases and controls ( $P>0.50$ ). Cases were more likely to smoke and to experience a nonfatal CVD event during the trial. Table 2 shows the same characteristics according to high and low hostility scores. The only significant difference was that hostile men smoked more than nonhostile men.

**TABLE 1. Baseline Characteristics and Nonfatal CVD Events for Deceased Cases (n=259) and Control Subjects (n=259) Matched on Study Site, Age, Race, Intervention Group, and Interviewer**

Variable	Control Subjects	Patients	P
Mean age, y	48.8	48.6	0.97
No. Men by ethnicity			0.42
White	240	231	
Black	8	15	
Asian	7	9	
Other	4	4	
Group, % special intervention	46.1	47.7	0.76
Smoking status, % yes	51.9	69.9	<0.001
Mean serum cholesterol, mg/dL	244.6	249.8	0.11
Mean diastolic blood pressure, mm Hg	99.2	99.1	0.82
Occurrence of nonfatal CVD event during the trial, %			
Among cases who died during the trial and their controls	13.9	27.8	0.03
Among cases who died after the trial and their controls	17.2	32.2	0.001

In the dependent logistic model of deaths during the 16-year follow-up period, 60% of cases had high total hostility ratings, compared with 44% of the matched controls (OR, 1.67; 95% CI, 1.17 to 2.38). With the addition of covariates (Table 3, row 1), cases were still more likely to have high hostility ratings (above the sample median) relative to controls (OR, 1.61; 95% CI, 1.09 to 2.39). The interaction between hostility and cigarette smoking was nonsignificant ( $P=0.50$ ). Similar results were obtained in the quartile analysis for hostility. That is, without covariates, the odds of CVD mortality were 1.00, 0.97, 1.52, and 1.79 for the men in the first through fourth quartiles of hostility scores, respec-

tively ( $P=0.007$  for linear trend). With covariates, the odds of CVD mortality were 1.00, 0.93, 1.45, and 1.71 for the first through fourth quartiles of hostility scores, respectively ( $P=0.02$  for test for linear trend).

We next considered whether hostility effects varied by whether the death occurred during or after the trial and whether it was stronger in those with a previous nonfatal CVD event during the trial. (Nonfatal CVD events were not measured after the trial.) Among men who died during the trial and their controls, high hostility ratings were associated with during-trial CVD mortality (adjusted OR, 2.33; 95% CI, 1.03 to 5.30). The effect of hostility did not vary according to

**TABLE 2. Baseline Characteristics and Nonfatal CVD Events for Low and High-Hostile Men**

Variable	Hostility		P
	Low (n=238)	High (n=280)	
Mean age, y	49.1	48.7	0.42
No. men by ethnicity			0.20
White	222	249	
Black	5	18	
Asian	7	9	
Other	3	4	
Group, % special intervention	48.9	45.0	0.37
Smoking status, % yes	51	70	<0.001
Mean serum cholesterol, mg/dL	250.3	244.6	0.08
Mean diastolic blood pressure, mm Hg	99.4	98.9	0.48
Occurrence of nonfatal CVD event during the trial, %			
Among cases who died during the trial and their controls	22.5	19.2	0.61
Among cases who died after the trial and their controls	26.6	23.3	0.47

**TABLE 3. Odds of CVD Mortality (95% CI) and No. of Participants Deceased/Total No. in High and Low-Hostile Groups Adjusted for Covariates During the 16-Year Follow-Up Period and Trial and Posttrial Periods Separately**

Period	Hostility Group			
	Low		High	
	Odds Ratio	n Deceased/n Total	Odds Ratio	n Deceased/n Total
16-year follow-up	1.00	103/238	1.61 (1.09–2.39)	156/280
Trial only, nonfatal cardiovascular event				
No	1.00	20/62	2.92 (1.11–7.70)	37/63
Yes	2.77 (0.79–9.68)	11/18	3.10 (0.72–13.33)	11/15
Posttrial only, nonfatal cardiovascular event				
No	1.00	50/116	1.12 (0.67–1.85)	72/155
Yes	1.32 (0.61–2.83)	22/42	5.06 (1.42–8.22)	36/47

whether a previous CVD nonfatal event had occurred, ie, the interaction between hostility ratings and previous occurrence of a nonfatal CVD event was not significant among men who died during the trial and their controls ( $P=0.36$ ; Table 3, rows 2 and 3). The test for the interaction between smoking and hostility was significant ( $P=0.03$ ), with cigarette smoking having a larger impact on trial CVD mortality among low-hostile men than high-hostile men.

Among men who died after the trial and their controls, high hostility ratings tended to be associated with posttrial CVD mortality, but the effect was nonsignificant (adjusted OR, 1.44; 95% CI, 0.92 to 2.26). The interaction between hostility ratings and previous occurrence of a nonfatal CVD event was significant, however ( $P<0.03$ ; Table 3, rows 4 and 5). Compared with low-hostile men without a nonfatal CVD event during the trial, high-hostile men without a nonfatal event during the trial were not at greater risk for dying of CVD. However, high-hostile men with a nonfatal event during the trial were at high risk for dying of CVD during the posttrial period. No interaction of hostility and smoking status was obtained when considering men who died after the trial and their controls.

## Discussion

The present article tested the hypothesis that hostility levels would predict subsequent CVD mortality in high-risk men during the 16 years of follow-up of MRFIT and explored whether the pattern of associations varied by length of follow-up and occurrence of a previous nonfatal event. The study confirmed the primary hypothesis and showed that men who had died of CVD had higher levels of hostility than matched living controls. Matching was based on study site, age, intervention group, race, and interviewer, and analyses adjusted for smoking status, diastolic blood pressure, and total cholesterol level at study entry and occurrence of nonfatal event during the trial. Subanalyses by quartiles of hostility suggested that the men in the highest quartile had the highest risk for CVD death. This is one of few studies showing that hostility levels prospectively predict CVD mortality in high-risk individuals.

Exploratory analyses also suggested that potency of hostility as a risk factor weakened over time among the men who did not have a nonfatal event during the trial. Conversely, hostility was a strong risk factor for CVD death after the trial among those men who had a nonfatal event during the trial. These men had 5 times the risk compared with living low-hostile controls. The explanation for these findings may be a result of selective survival. Those who were most vulnerable to the detrimental effects of hostility may have had fatal or nonfatal events early. Those hostile individuals who survived into the posttrial period without a CVD event may have been relatively hardy. Conversely, men who remained at risk for CVD in the long term were already compromised in their function by a nonfatal CVD event, suggesting that hostility may trigger a CVD death in vulnerable individuals.

Several negative studies in the literature used a measure of hostile attitudes, as opposed to other aspects of hostility.<sup>20,21</sup> Although hostile attitudes do predict CVD incidence in some studies,<sup>22,23</sup> it is not a complete measure of the underlying behavioral concept. In our study, we measured additional aspects of hostility, including signs of irritation, arrogance, uncooperativeness, and angry feelings by use of the IHAT.<sup>11</sup> Assessments based on observation of interview behaviors have advantages over self-report questionnaires such as those used to measure hostile attitudes. They avoid a number of self-presentation biases that affect questionnaire responses and identify hostile tendencies in individuals not aware of them, factors that probably account for the low correlations observed between self-report and interview-based assessments.<sup>24</sup> We suggest that future studies of hostility should use a comprehensive assessment that allows for better characterization of the multiple aspects of hostility.

Why might hostility predict cardiovascular death? It is well documented that hostile persons are likely to smoke, have a poor diet, be obese, and take little exercise, even early in life.<sup>25,26</sup> Although important, these factors probably did not account for our results as we studied a high-risk group of men. Hostile people have elevated ambulatory blood pressure during daily life, in both patient and nonpatient groups,<sup>27,28</sup> and exhibit

elevated blood pressure and heart rate in response to annoying circumstances presented in laboratory settings.<sup>29</sup> Preliminary data suggest that hostile persons also exhibit diminished vagal modulation of heart function,<sup>30</sup> increased platelet reactivity,<sup>31</sup> and endothelial dysfunction.<sup>32</sup> To the extent that hostile people encounter more anger-producing situations or interpret ambiguous situations negatively, they may be prone to abrupt sympathetic activation and its consequences in the vulnerable heart.

The present findings have several clinical implications. First, patients should be informed about the potential health consequences of anger and hostility. There are no clinical trials of anger management interventions with CVD events as the outcome. However, several clinical trials using stress management techniques in combination with other modalities do show reduced hostility and reduced risk of new clinical events in coronary patients.<sup>33,34</sup> Second, the association of hostility with unhealthy behaviors occurs early in the natural history of atherosclerosis.<sup>25,26</sup> Concerted efforts to promote exercise, prevent weight gain, and reduce the likelihood of smoking may be especially beneficial in hostile young adults. Third, mental health services frequently concern anger management issues, with treatment often being successful.<sup>35</sup> Thus, patient referral can be a useful option for the practicing cardiologist.

In sum, the present study found that men who died of CVD during the 16 years of follow-up were more likely to be hostile at study entry compared with matched living controls. Hostility may be an important risk factor for CVD death in high-risk men.

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