

Effects of an Angry Temperament on Coronary Heart Disease Risk

The Atherosclerosis Risk in Communities Study

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The objective of the study was to determine which component of an anger-prone personality more strongly predicts coronary heart disease (CHD) risk. Proneness to anger, as assessed by the Spielberger Trait Anger Scale, is composed of two distinct subcomponents—anger-temperament and anger-reaction. Participants were 12,990 middle-aged Black men and women and White men and women from the Atherosclerosis Risk in Communities Study who were followed for the occurrence of acute myocardial infarction (MI)/fatal CHD, silent MI, or cardiac revascularization procedures (average = 53 months; maximum = 72 months) through December 31, 1995. Among normotensive persons, a strong, angry temperament (tendency toward quick, minimally provoked, or unprovoked anger) was associated with combined CHD (acute MI/fatal CHD, silent MI, or cardiac revascularization procedures) (multivariate-adjusted hazard ratio = 2.10, 95% confidence interval: 1.34, 3.29) and with “hard” events (acute MI/fatal CHD) (multivariate adjusted hazard ratio = 2.28, 95% confidence interval: 1.29, 4.02). CHD event-free survival among normotensives who had a strong, angry temperament was not significantly different from that of hypertensives at either level of anger. These data suggest that a strong, angry temperament rather than anger in reaction to criticism, frustration, or unfair treatment places normotensive, middle-aged persons at increased risk for cardiac events and may confer a CHD risk similar to that of hypertension. *Am J Epidemiol* 2001;154:230–5.

coronary disease; prospective studies; stress; survival analysis

A recent analysis of the Atherosclerosis Risk in Communities Study (ARIC) cohort reported that normotensive persons who were highly predisposed to anger and free of coronary heart disease (CHD) at baseline were slightly greater than two times more likely to experience a CHD event than were their less-anger-prone counterparts (1). Proneness to anger was assessed by the Spielberger Trait Anger Scale, which is composed of two distinct subscales: anger-temperament and anger-reaction (2). Compared with persons who are prone to angry reactions, those who have a strong, angry temperament experience anger longer, more frequently, more intensely, and in a broader range of situations and express it more quickly, needing little or no

provocation. Persons prone to angry reactions, on the other hand, typically experience anger when frustrated, mistreated, or negatively evaluated by others. This study assessed the association between each trait anger component and CHD risk among persons enrolled in the ARIC cohort (3).

MATERIALS AND METHODS

ARIC is a large, population-based, prospective study of cardiovascular disease and its risk factors among residents aged 45–64 years in the US communities of Washington County, Maryland; suburban Minneapolis, Minnesota; Forsyth County, North Carolina; and Jackson, Mississippi. Baseline clinical examinations were conducted from 1987 to 1989 (visit 1), and follow-up examinations were given every 3 years thereafter (visits 2–4) (3). The population for this study was selected from the ARIC cohort who returned to visit 2 between 1990 and 1992 ($n = 14,348$). After exclusions, 12,990 persons remained for these analyses.

Using Spielberger’s trait anger-temperament and trait anger-reaction subscales (see Appendix), respondents rated the frequency of their experience with anger on a Likert-type scale as: almost never = 1, sometimes = 2, often = 3, and almost always = 4. Responses to the four items in each subscale were summed to yield a score.

Covariates analyzed were age, race/ethnicity, gender, waist-to-hip ratio, plasma low density lipoprotein and high

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Abbreviations: ARIC, Atherosclerosis Risk in Communities; CHD, coronary heart disease; MI, myocardial infarction.

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density lipoprotein cholesterol levels, hypertensive status, diabetes, alcohol drinking status, cigarette smoking status, and level of educational attainment.

Participants were followed from the date of their first clinic reexamination in ARIC through December 31, 1995. An incident CHD event was defined as acute myocardial infarction (MI)/fatal CHD, cardiac revascularization procedure (percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery), or silent MI (4). "Hard" events were restricted to MI/fatal CHD.

The association between each trait anger component and CHD risk was determined by Cox proportional hazards regression. Heterogeneity of effect was observed by hypertensive status only and only in the trait anger-temperament/CHD relation ($\chi^2_{(1)} = 9.26; p < 0.01$). Crude probabilities of CHD event-free survival were determined with the use of the Kaplan-Meier product limit method. Age- and gender-

adjusted survival curves were drawn by using probabilities obtained from Cox regression.

RESULTS

Tables 1 and 2 present cardiovascular risk factor profiles for trait anger-temperament and trait anger-reaction, respectively, by hypertensive status.

Among normotensive persons, the age-adjusted risk of combined CHD was 2.48 times greater among persons who reported having a strong, angry temperament compared with their counterparts who reported being less prone to an angry temperament (table 3). Although the magnitude of association was attenuated somewhat after multivariate adjustment, there remained a slightly greater than doubling of CHD risk among normotensive persons characterized as having a strong, angry temperament (probability value for linear

TABLE 1. Distribution of population characteristics for trait anger-temperament, by hypertensive status, the ARIC† Study, 1990–1992

	Normotensives			Hypertensives		
	AT† ≤ 8 (n = 8,021)	AT > 8 (n = 456)	P*	AT ≤ 8 (n = 4,231)	AT > 8 (n = 282)	P*
Age (years) (mean (SD))†	56.2 (5.6)	55.7 (5.8)	0.07	58.1 (5.6)	57.7 (6.1)	0.21
Male (%)	42.9	48.5	0.02	41.3	54.6	<0.01
Less than high school education	16.3	27.6	<0.01	27.8	38.3	<0.01
White (%)	82.7	81.8	0.64	61.1	61.0	0.98
Cigarette smoking (years) (mean (SD))	299.6 (412.1)	469.1 (517.0)	<0.001	280.0 (418.9)	429.5 (520.4)	<0.001
Drinks of alcohol (g/week)	35.6 (87.5)	45.8 (111.9)	0.02	36.7 (96.3)	57.8 (115.7)	<0.01
Diabetic (%)	6.8	8.4	0.19	18.4	20.4	0.39
Plasma LDL† cholesterol (mg/dl) (mean (SD))	131.9 (36.0)	133.3 (36.5)	0.45	135.0 (37.8)	135.9 (39.5)	0.71
Plasma HDL† cholesterol (mg/dl) (mean (SD))	51.0 (16.9)	48.7 (17.2)	<0.01	48.8 (16.6)	45.8 (15.2)	<0.01
Waist-to-hip ratio (cm) (mean (SD))	0.911 (0.084)	0.930 (0.079)	<0.001	0.944 (0.073)	0.960 (0.070)	<0.001

* Two-sample *t* test for the comparison of means or chi-square for the comparison of proportions.

† ARIC, Atherosclerosis Risk in Communities; AT, anger-temperament; SD, standard deviation; LDL, low density lipoprotein; HDL, high density lipoprotein.

TABLE 2. Distribution of population characteristics for trait anger-reaction, by hypertensive status, the ARIC† Study, 1990–1992

	Normotensives			Hypertensives		
	AR† ≤ 10 (n = 7,649)	AR > 10 (n = 828)	P*	AR ≤ 10 (n = 4,100)	AR > 10 (n = 413)	P*
Age (years) (mean (SD))†	56.3 (5.6)	55.2 (5.5)	<0.001	58.1 (5.7)	57.5 (5.6)	0.04
Male (%)	43.6	38.8	0.01	42.5	38.3	0.09
Less than high school education	17.1	15.0	0.01	27.8	34.1	0.06
White (%)	82.5	83.2	0.63	61.5	56.9	0.07
Cigarette smoking (years) (mean (SD))	303.1 (417.5)	360.0 (439.9)	<0.001	283.9 (420.8)	345.3 (487.2)	0.01
Drinks of alcohol (g/week)	34.9 (85.3)	47.4 (116.8)	<0.001	36.9 (94.3)	49.6 (126.3)	0.01
Diabetic (%)	6.8	7.1	0.73	18.3	20.0	0.42
Plasma LDL† cholesterol (mg/dl) (mean (SD))	132.2 (36.0)	130.1 (36.9)	0.11	135.3 (38.1)	132.5 (35.1)	0.15
Plasma HDL† cholesterol (mg/dl) (mean (SD))	50.8 (16.9)	51.3 (17.3)	0.42	48.5 (16.4)	49.5 (17.8)	0.27
Waist-to-hip ratio (cm) (mean (SD))	0.912 (0.084)	0.913 (0.087)	0.70	0.944 (0.073)	0.947 (0.073)	0.55

* Two-sample *t* test for the comparison of means or chi-square for the comparison of proportions.

† ARIC, Atherosclerosis Risk in Communities; AR, anger-reaction; SD, standard deviation; LDL, low density lipoprotein; HDL, high density lipoprotein.

TABLE 3. Hazard ratios and 95% confidence intervals for the association between trait anger-temperament and CHD* risk, the ARIC* Study, 1990–1995

	Spielberger trait anger-temperament scores					
	Normotensives			Hypertensives		
	Low (≤ 8)	High (> 8)		Low (≤ 8)	High (> 8)	
		Hazard ratio	95% CI*		Hazard ratio	95% CI
Population (no.)	8,021	456		4,231	282	
No. with incident events	167	23		213	13	
CHD events combined†						
Age-adjusted	1.00	2.48	1.60, 3.83	1.00	0.91	0.52, 1.60
Multivariate-adjusted‡	1.00	2.10	1.34, 3.29	1.00	0.70	0.39, 1.27
Acute MI*/fatal CHD						
Age-adjusted	1.00	2.78	1.61, 4.79	1.00	0.77	0.36, 1.64
Multivariate-adjusted	1.00	2.28	1.29, 4.02	1.00	0.58	0.25, 1.32

* CHD, coronary heart disease; ARIC, Atherosclerosis Risk in Communities; CI, confidence interval; MI, myocardial infarction; LDL, low density lipoprotein; HDL, high density lipoprotein.

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

‡ Adjusted for age, race/ethnicity, level of educational attainment, gender, waist-to-hip ratio, plasma LDL* and HDL* cholesterol levels, diabetes, years of cigarette smoking, and alcoholic drinks per week.

trend = 0.02). The age-adjusted risk of hard events was 2.78 times greater for normotensive persons who reported having a strong, angry temperament and 2.28 times greater when adjusted by the traditional CHD risk factors (p for linear trend = 0.04). There was a monotonic increase in CHD risk as a result of trait anger-temperament both for combined CHD and for hard events in the multivariate-adjusted models. Normotensive persons experienced a 68 percent greater risk of CHD (age-adjusted, hard events) for each four-unit increase in trait anger-temperament (95 percent confidence interval: 1.53, 1.84). In contrast, the association between trait anger-temperament and CHD risk among hypertensives was not statistically significant.

Results of the proportional hazards regression analysis for trait anger-reaction indicated a slight elevation in risk for combined CHD and for hard events, but none of these associations was statistically significant (table 4).

The log-rank test for equality over strata indicated that the CHD event-free survival functions were significantly different between normotensive persons who reported having a strong, angry temperament and their counterparts who reported being less prone to an angry temperament ($p < 0.001$) (figure 1). In contrast, the survival probability among hypertensives who reported having a strong, angry temperament was not significantly different from that of their counterparts who reported being less prone to an angry temperament ($p = 0.71$) (figure 2). Further, no significant difference in the probability of CHD event-free survival was observed between persons in the high and low anger-reaction subgroups ($p = 0.48$).

The adjusted CHD event-free survival function for normotensives who reported having a strong, angry temperament was not significantly different from that of their hypertensive counterparts ($\chi^2_{(1)} = 0.21$; $p = 0.65$) or from hypertensives who reported being less prone to an angry

TABLE 4. Hazard ratios and 95% confidence intervals for the association between trait anger-reaction and CHD* risk, the ARIC* Study, 1990–1995

	Spielberger trait anger-reaction scores		
	Low (< 10)	High (≥ 10)	
		Hazard ratio	95% CI*
Population (no.)	11,749	1,241	
No. with incident events	372	44	
CHD events combined†			
Age-adjusted	1.00	1.19	0.87, 1.63
Multivariate-adjusted‡	1.00	1.26	0.91, 1.75
Acute MI*/fatal CHD			
Age-adjusted	1.00	1.24	0.84, 1.84
Multivariate-adjusted	1.00	1.23	0.81, 1.87

* CHD, coronary heart disease; ARIC, Atherosclerosis Risk in Communities; CI, confidence interval; MI, myocardial infarction; LDL, low density lipoprotein; HDL, high density lipoprotein.

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

‡ Adjusted for age, race/ethnicity, level of educational attainment, gender, waist-to-hip ratio, plasma LDL* and HDL* cholesterol levels, diabetes, years of cigarette smoking, and alcoholic drinks per week.

temperament ($\chi^2_{(1)} = 0.05$; $p = 0.82$) (figure 3). These results indicate that high anger-temperament among normotensives confers a risk of CHD similar to that of hypertension. Statistically significant differences in survival were observed between hypertensive and normotensive

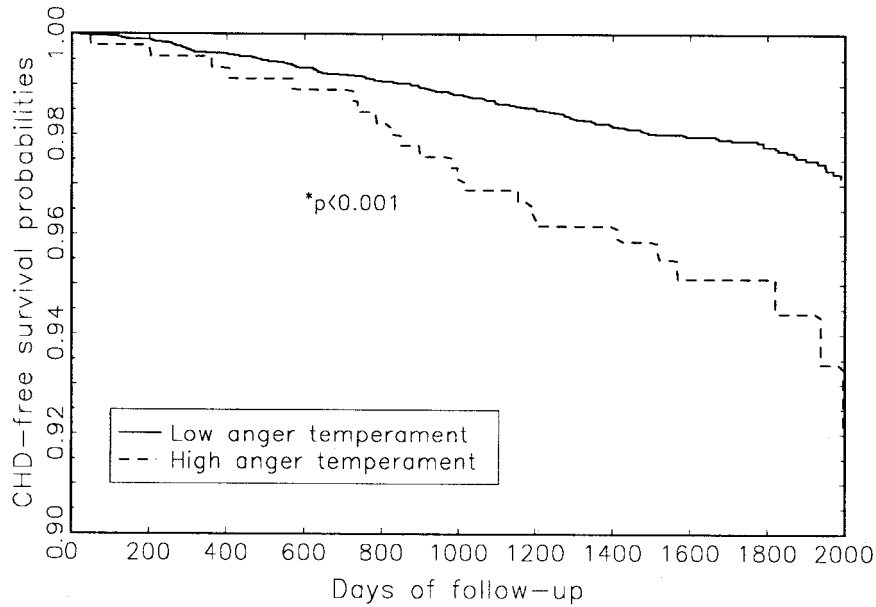


FIGURE 1. Crude coronary heart disease (CHD)-free survival probabilities among normotensives, by trait anger-temperament scores, the Atherosclerosis Risk in Communities Study, 1990–1995. *, log-rank test for equality over trait anger-temperament strata.

persons who reported being less prone to an angry temperament ($\chi^2_{(1)} = 65.15; p < 0.0001$).

DISCUSSION

In this study, the risk of CHD differed by trait anger subtype. In multivariate-adjusted analysis, compared with normotensive persons who reported being less prone to an angry

temperament, the risk of combined CHD among normotensives who reported having a strong, angry temperament was slightly more than two times as great; the risk of hard events was nearly two and one-third times as great. In addition, the probability of CHD event-free survival among normotensives who reported having a strong, angry temperament was not significantly different from that of hypertensives, regardless of their anger level. In contrast, the association between

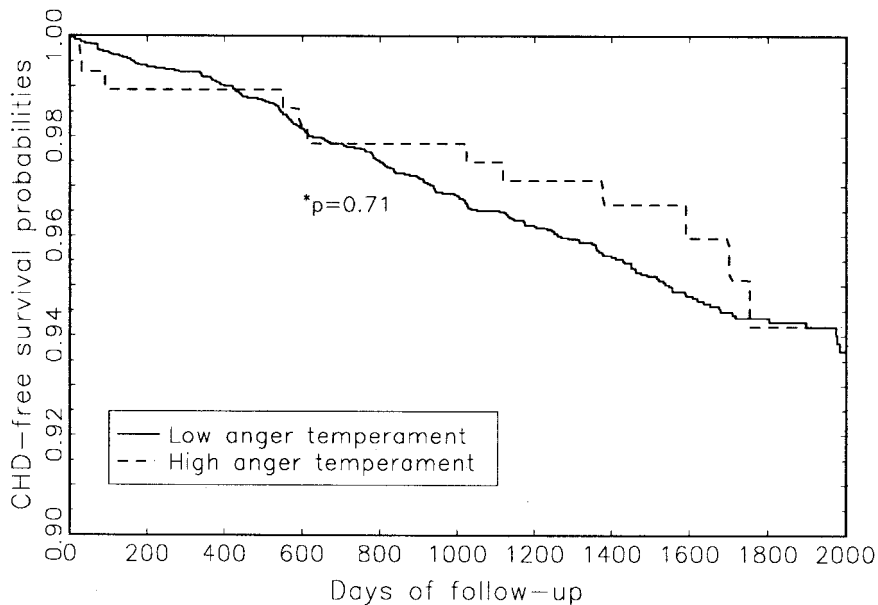


FIGURE 2. Crude coronary heart disease (CHD)-free survival probabilities among hypertensives, by trait anger-temperament scores, the Atherosclerosis Risk in Communities Study, 1990–1995. *, log-rank test for equality over trait anger-temperament strata.

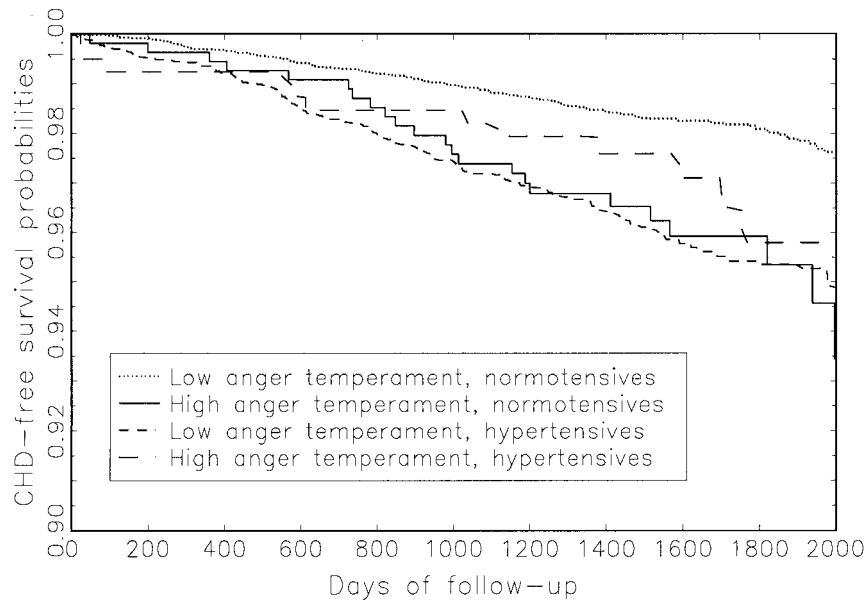


FIGURE 3. Adjusted coronary heart disease (CHD)-free survival probabilities, by hypertensive status and trait anger subscale scores, the Atherosclerosis Risk in Communities Study, 1990–1995.

trait anger-reaction and CHD risk was weak and was not statistically significant.

These findings suggest that it is the intense, volatile aspect of proneness to anger that has the more catastrophic consequences for cardiovascular health; thus, it is angry temperament, not angry reaction, that is the more potent link to CHD. These findings seem logical, since temperament, with its constitutional underpinnings, is the more enduring component of trait anger, persisting across time and contexts (2). Therefore, by its nature, an angry temperament may be a more powerful initiator and sustainer of the pathophysiologic changes leading to CHD, particularly MI and sudden cardiac death.

One set of changes that may be initiated by an angry temperament is those mediated by the sympathetic adrenal-medullary system, resulting in cardiovascular hyperreactivity and an excess discharge of the catecholamines. These hemodynamic stresses and biochemical changes may contribute to the development and progression of atherosclerosis by causing endothelial injury (5) and by their close association with other processes involved in atherogenesis, e.g., vascular lipid uptake (6), increased platelet adhesion and aggregation (7), and activation of macrophages (8). Excessive levels of catecholamines can also cause direct damage to the heart muscle (9, 10) and produce potentially lethal cardiac arrhythmias (10, 11). Moreover, recent evidence suggests hostility/anger may influence CHD through coronary artery calcification, another aspect of atherosclerosis (12).

Another set of changes is suggested by the triggering hypothesis, which has been proffered as an explanation for the underlying mechanism linking anger to CHD (13–16). This hypothesis implicates anger as an initiator of the vascular and prothrombotic events that lead to atherosclerotic plaque disruption, occlusive thrombosis, and, ultimately, an

acute MI or sudden death. These events are the more proximal precipitants of (or “acute risk factors” for) MI, e.g., blood pressure surges in the arterial vessels, vasoconstriction, increased platelet adhesion and aggregability, increased blood coagulation, and increased fibrinogen concentration (15, 17–19). If the triggering mechanism is operative, then the current data suggest that, relative to the responses associated with anger caused by frustration, criticism, or unfair treatment, the biobehavioral sequelae of a fiery, explosive temper is a more powerful catalyst of atherosclerotic plaque rupture and its potentially lethal consequences.

This study suggests that a strong, angry temperament predisposes middle-aged, normotensive persons to a significantly greater risk of MI or sudden cardiac death than anger aroused in reaction to more circumscribed stimuli, e.g., frustration, criticism, or unfair treatment. These data also suggest that the tendency toward quick, unprovoked (or minimally provoked) anger may be as toxic to the cardiovascular system and have as detrimental a consequence as hypertension. Approximately 6 percent of the cohort reported having a strong, angry temperament, suggesting that this exposure is relatively infrequent. The adverse cardiovascular consequences of having a fiery temper, however, are quite dramatic.

Anger management may help to sever the link between this negative emotion and incident CHD. Results of at least two studies have demonstrated the efficacy of intervention (e.g., type A behavioral counseling, cardiac rehabilitation, and exercise training) to lower hostility/anger levels among patients with CHD (20, 21). These studies also showed that reducing hostility/anger could forestall the recurrence of events (20) and improve CHD risk factor profiles as well as overall quality of life (21). Similar studies are needed to determine the efficacy of intervention for the primary prevention of CHD.

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REFERENCES

- Williams JE, Paton, CC, Siegler IC, et al. Anger proneness predicts coronary heart disease risk: prospective analysis from the Atherosclerosis Risk in Communities (ARIC) Study. *Circulation* 2000;101:2034-9.
- Spielberger CD, Jacobs G, Russell S, et al. Assessment of anger: the State-Trait Anger Scale. In: Butcher JN, Spielberger CD, eds. *Advances in personality assessment*. Vol 2. Hillsdale, NJ: Lawrence Erlbaum Associates, 1983:161-89.
- ARIC Investigators. The Atherosclerosis Risk in Communities (ARIC) Study: design and objectives. *Am J Epidemiol* 1989;129:687-702.
- White A, Folsom AR, Chambless LE, et al. Community surveillance of coronary heart disease in the Atherosclerosis Risk in Communities (ARIC) Study: methods and initial two years' experience. *J Clin Epidemiol* 1996;49:223-33.
- Kaplan JR, Pettersson K, Manuck SB, et al. Role of sympathoadrenal medullary activation in the initiation and progression of atherosclerosis. *Circulation* 1991;84(suppl 6):VI23-32.
- Born GVR. Recent evidence for the involvement of catecholamines and of macrophages in atherosclerotic processes. *Ann Med* 1991;23:569-72.
- Anfossi G, Trovati M. Role of catecholamines in platelet function: pathophysiological and clinical significance. *Eur J Clin Invest* 1996;26:353-70.
- Adams DO. Molecular biology of macrophage activation: a pathway whereby psychosocial factors can potentially affect health. *Psychosom Med* 1994;56:316-27.
- Prichard BN, Owens CW, Smith CC, et al. Heart and catecholamines. *Acta Cardiol* 1991;46:309-22.
- Schomig A. Catecholamines in myocardial ischemia. Systemic and cardiac release. *Circulation* 1990;82(suppl 2):II13-22.
- Schomig A, Richardt G. Cardiac sympathetic activity in myocardial ischemia: release and effects of noradrenaline. *Basic Res Cardiol* 1990;85(suppl 1):9-30.
- Iribarren C, Sidney S, Bild DE, et al. Association of hostility with coronary artery calcification in young adults: The CARDIA Study. *JAMA* 2000;283:2546-51.
- Mittleman MA, Maclure M, Sherwood JB, et al. Triggering of acute myocardial infarction onset by episodes of anger. *Circulation* 1995;92:1720-5.
- Gabbay RH, Krantz DS, Kop WJ, et al. Triggers of myocardial ischemia during daily life in patients with coronary artery disease: Physical and mental activities, anger and smoking. *J Am Coll Cardiol* 1996;27:585-92.
- Muller JE, Abela GS, Nesto RW, et al. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. *J Am Coll Cardiol* 1994;23:809-13.
- Tofler GH, Stone PH, Maclure M, et al. Analysis of possible triggers of acute myocardial infarction (The MILIS Study). *Am J Cardiol* 1990;66:22-7.
- Fuster V, Badimon L, Badimon JJ, et al. The pathogenesis of coronary artery disease and the acute coronary syndromes. *N Engl J Med* 1992;326:310-18.
- Wenneberg SR, Schneider RH, Walton KG, et al. Anger expression correlates with platelet aggregation. *Behav Med* 1997;22:174-7.
- Jern C, Eriksson E, Tengborn L, et al. Changes of plasma coagulation and fibrinolysis in response to mental stress. *Thromb Haemost* 1989;62:767-71.
- Friedman M, Thoresen CE, Gill JJ, et al. Alteration of type A behavior and its effect on cardiac recurrences in post myocardial infarction patients: summary results of the recurrent coronary prevention project. *Am Heart J* 1986;112:653-65.
- Lavie CJ, Milani RV. Effects of cardiac rehabilitation and exercise training programs on coronary patients with high levels of hostility. *Mayo Clin Proc* 1999;74:959-66.

APPENDIX**SPIELBERGER TRAIT ANGER-TEMPERAMENT SUBSCALE**

- 1) I am quick tempered.
- 2) I have a fiery temper.
- 3) I am a hotheaded person.
- 4) I fly off the handle.

SPIELBERGER TRAIT ANGER-REACTION SUBSCALE

- 1) I get angry when I am slowed down by others' mistakes.
- 2) I feel annoyed when I am not given recognition for doing good work.
- 3) It makes me furious when I am criticized in front of others.
- 4) I feel infuriated when I do a good job and get a poor evaluation.