ARIC MANUSCRIPT PROPOSAL FORM

Manuscript #508

1. Title:
Anger and the occurrence of CHD events

2. Writing Group:
(lead) Janice Williams, Ilene Siegler, Catherine Paton, Marsha Eigenbrodt, Herman Tyroler, Javier Nieto (to be confirmed)
Lead: Janice E. Williams
University of North Carolina
Department of Epidemiology
137 E. Franklin Street, Suite 306
Chapel Hill, NC 27516
(919) 966-3168 (telephone)
(919) 966-9800 (FAX)
35jwilliams@sophia.sph.unc.edu)

3. Timeline:
Proposal to publication committee - August,1997
Complete analyses - December, 1997
Submit first draft to publication committee - March, 1998
Submit to joumal - June, 1998

4. Rationale:
That the experience of anger is associated with CHD events is an hypothesis of long-standing. A growing literature derived from different study designs provides confirmatory evidence of an association. For example, in a case crossover study, Mittleman, Maclure, Sherwood et al (1995) assessed the triggering effect of anger on acute MI and found a two-fold increase in the onset of MI during the 2 hours prior to an event as compared to the usual experience of anger during the previous year. Similarly, Hecker, Chesney, Black, and Frautsch (1988) in a case-control study using data from the Western Collaborative Group Study (WCGS) found an almost two-fold increase in CHD incidence among individuals considered high in hostility (a concept often used interchangeably with anger). Follow-up data from this study showed those lowest in hostility with the lowest 10-year risk of CHD. In cross-sectional analyses, Williams, Haney, Lee, et al. (1980) found a 70% rate of clinically significant atherosclerosis documented angiographically among high-hostile individuals. Most recently, Kawachi, Sparrow, Spiro, Vokonas, and Weiss (1996) investigated prospectively the association of anger and CHD and observed an almost three-fold increase in the risk of CHD, a dose-response relationship in level of anger and CHD risk, as swell as shorter CHD-free survival among participants with higher levels of anger. However, despite the burgeoning

anger-CHD literature, few studies have examined this association prospectively, and none have done so in a population-based US population of white and African-American men and women. ARIC is such a population-based prospective study and an investigation of the anger-CHD relationship in the ARIC communities could provide a substantial contribution to the existing literature.

5. Main Hypothesis:
Trait anger (main exposure variable) is positively associated with the incidence and recurrence of CHD events (silent MI, fatal and nonfatal CHD, and cardiac procedures). The association will exist after controlling for the potential confounding effects of diastolic and systolic blood pressure levels, total serum cholesterol levels, BMI, smoking status, farnily history of CHD, alcohol consumption, depression, and age.

6. Analysis method and power calculation:
The main hypothesis will be tested using the proportional hazards regression model. The following is a table specifying the number of events required for a 5% significance level and 80% and 90% power at varying magnitudes of association and varying proportions of exposed and unexposed individuals in the study population. The method of Schoenfeld (1983) was used to calculate these quantities. In ARIC, 413 events occurred between Visit 2 (the date of administration of the anger questionnaire) and 1994 (the most recent event date).

To request table, please call (919) 962-2073 or the ARIC Student Assistant at (919) 962-3268

Based upon the above calculations, the proposed study has 80% and 90% power to detect a hazard ratio of 1.5 and above when the exposure prevalence ranges from l 5-50% in the population. The marginal condition is having 90% power when the exposure prevalence is 15%.

7. Variables:
Trait anger (measured using the Spielberger State-Trait Anger Scale and assessed in Visit 2); hypertensive status; diastolic and systolic BP; smoking status; CHD events between 1992 and 1994; family history of CHD; alcohol consumption; depression (assessed in Visit 2); age, lipid levels.

REFERENCES

Hecker HL, Chesney MA, Black GW, Frautschi N. Coronary-prone behaviors in the Western Collaborative Group Study. *Psychosom Med* 1988; 50:153-164.

Kawachi I, Sparrow D, Spiro A, Vokonas P, Weiss ST. A prospective study of anger and coronary heart disease. *Circulation* 1996; 94(9):2090-2095.

Mittleman MA, Maclure M, Sherwood JB, et al. Triggering of acute myocardial infarction onset by episodes of anger. *Circulation* 1995; 92:1720-1725.

Schoenfeld DA. Sample-size formula for the proportional-hazards regression model. *Biometrics* 1983; 39:499-503.

Williams RB, Haney TL, Lee KL, et al. TypeA behavior, hostility, and coronary atherosclerosis. *Psychosom Med* 1980; 42(6):539-549.