1a. Full Title: The Moderating Effects of Social Support on the Association Between Negative Emotions and CHD Events, Carotid Arterial Wall Thickness, and Mortality

1b. Abbreviated Title: Social Moderators of CHD

2. Writing Group:

Lead: Thomas Mosley
Address: University of Mississippi Medical Center
Department of Medicine (Geriatrics)
2500 North State St.
Jackson, MS 39216-4505
Phone: (601) 984-5610 Fax: (601) 984-5783
Email: tmosley@medicine.umsmed.edu

A. Pat Dubbert  D. Herman Taylor
B. Tom Payne  E. Paul McGovern
C. Cecil Burchfiel  F. Mike Andrew

3. Timeline:

Analysis to begin following Publications Committee approval. Manuscript anticipated for initial review by August 2000.

4. Rationale:

Several negative emotions have been identified as possible risk factors for coronary heart disease (CHD), including anger and depression. These negative or "coronary-prone" emotions have been found to influence the incidence, morbidity, and mortality associated with CHD.

Anger and hostility, for example, have emerged as the primary (toxic) components thought to underlie associations between Type A and CHD (1). Several epidemiologic studies have found that chronic anger and/or maladaptive anger coping styles are associated with an increased risk of CHD-related morbidity and mortality (2-7). Anger has also been associated with all-cause mortality (3-5, 8-10).

Depression has also been linked to CHD in several studies, including incidence of coronary artery disease (1) and post-MI mortality (11,12). The construct of Vital Exhaustion, assessed in ARIC, overlaps considerably with that of depression. Proposed by Appels and Mulder (13), vital exhaustion presumably results from exposure to prolonged and uncontrollable stress.
Vital exhaustion has been related to incident MI (13), recurrence of cardiac events (14), and severity of coronary artery disease (15).

Researchers have proposed a variety of variables that may act as moderators of psychological risk factors, either augmenting or reducing their impact on disease. Moderators may include properties of the individual such as self-esteem and coping, as well as characteristics of the environment such as social support (16-18).

A considerable literature has established the particular importance of social support as a moderator of morbidity and mortality rates across a variety of diseases. Social support has been variously defined as quantity of social ties, access to practical or tangible assistance, or perceptions of emotional support. Low support (particularly emotional) and social isolation have been consistently related to cardiovascular disease risk, events, and all-cause mortality for men and women (19). Although social support may exert a direct (or main) effect on health outcomes, it may also function as a buffer, moderating the effects of negative emotions. From this perspective, individuals with poor social support would be at greatest risk from negative emotions. Conversely, individuals with adequate or high social support would be protected from the deleterious effects of negative emotions, and therefore only a weak relationship would be observed between emotions and health outcomes.

Few previous prospective investigations of CHD have collected multiple indices of negative emotions and social support. Thus, the relative contribution of these variables to CHD risk as well as the possible interaction between negative emotions and social support have not been comprehensively examined. ARIC provides a unique opportunity to address both of these shortcomings.

Overlap with other ARIC manuscripts: Previous manuscript proposals of psychological variables from ARIC have primarily focused on the main effect of a single variable on a single outcome (e.g., anger and MI, anger and arterial stiffness, anger and stroke, etc.). The current proposal acknowledges some overlap with manuscript #640 (The convergence of acute and chronic psychological factors and its impact on CHD risk), which has proposed to examine the combined influence of anger and vital exhaustion on CHD risk. However, the moderating role of social support on CHD risk associated with negative emotions, to our knowledge, has not been covered in previous manuscripts.

5. Main Study Questions:

What is the relative contribution of anger, vital exhaustion, and social support to the incidence and recurrence of CHD events (MI, silent MI, revascularization procedures), carotid arterial wall thickness, and mortality (CHD-specific, all-cause)?

Is risk (for CHD events, carotid wall thickness, and mortality) associated with negative emotions greater among individuals with low social support?

Does the buffering effect of social support on negative emotions vary by type of support (i.e., structural vs. functional)?
6. Data:

Study questions regarding risk of CHD events and mortality will be tested using Cox proportional hazards models, adjusted for the potential confounding effects of age, sex, ethnicity, income, education, systolic and diastolic blood pressure, total cholesterol, HDL, LDL, BMI, fibrinogen, prevalent disease (hypertension, diabetes), antihypertensive use, HRT (women), ETOH consumption, smoking, physical activity, and family history of CHD. TIA/stroke symptoms and FEV₁ will also be controlled in models of all-cause mortality. Multivariate linear regression models will be used to examine the relationship between negative emotions, social support, and carotid wall thickness. The regression models will be adjusted for traditional cardiovascular risk factors.

Visit 2 variables include: trait anger, vital exhaustion, social support (Lubben, ISEL), cardiovascular risk factors (listed above), and carotid arterial wall thickness. In addition, follow-up through 1995 for incident CHD events and mortality will be used.

References