1. Full Title: Anger-Vital Exhaustion and Metabolic Syndrome
   Abbreviated Title: Anger, Exhaustion & Metabolic Syndrome

2. Writing Group:
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3. Timeline:
   Analysis to begin following Publications Committee approval. Manuscript anticipated for
   initial review by May 1998.

4. Rationale:
   A metabolic syndrome has been characterized linking several risk factors for diabetes and
   cardiovascular disorders. These factors include hyperinsulinemia, glucose intolerance, dyslipidemia, abdominal obesity, and hypertension (1-4). Although these abnormalities are common and frequently co-exist, they have been shown to cluster within individuals at a significantly greater rate than would be expected by chance. Hyperinsulinemia and insulin resistance have been forwarded as possible etiological factors underlying this syndrome (4,5).

   Metabolic and cardiovascular disorders may also be influenced by behavioral or psychological factors. Epidemiologic and clinical studies have identified several psychosocial risk factors (behavioral, social, and psychological) which appear to influence metabolic conditions as well as the incidence, course, and mortality associated with cardiovascular disorders. Psychosocial factors have recently been hypothesized to play a role in the pathogenesis of metabolic syndrome through repeated activation of neuroendocrine and endocrine axes, leading over time to a state of chronic hypersensitivity in these systems (6,7).
A large literature supports the role of chronic anger and hostility as a risk factor for coronary heart disease (CHD, 8). Anger and hostility also have emerged as the primary (toxic) components thought to underlie associations between Type A and CHD (9). The primary pathophysiological mechanism linking anger/hostility to CHD proposes heightened sympathetic reactivity involving elevated cardiovascular and neuroendocrine responses (10-12). Chronic mobilization of the neuroendocrine axis, as noted above, suggests a possible mechanism linking anger to metabolic syndrome. In a recent study, anger was found to be associated with metabolic syndrome (6).

Various syndromes of psychological distress have also been examined as risk factors for CHD. Depression has been linked to CHD in several studies, including incident of coronary artery disease (9) and post MI mortality (13,14). Vital exhaustion is another distress syndrome examined as a risk factor. Proposed by Appels and Mulder (15), vital exhaustion presumably results from exposure to prolonged and uncontrollable stress. This notion is similar to the “exhaustion stage” in Selye’s general adaptation syndrome and overlaps considerably with measures of depression. Vital exhaustion has been related to incident MI (15), recurrence of cardiac events (16), and severity of coronary artery disease (17). Vital exhaustion has also been associated with metabolic syndrome (6). As with anger, cardiovascular and neuroendocrine responses have been hypothesized as the pathophysiological mechanisms linking psychological distress to the development and progression of CHD(18).

The present study proposes to examine associations among two psychological variables assessed in ARIC and factors comprising the metabolic syndrome. The psychological variables of interest are trait anger and vital exhaustion.

5. Main Hypothesis:
The primary hypotheses are that trait anger and vital exhaustion (assessed by the Maastricht Questionnaire) will demonstrate an independent association with metabolic syndrome. Correlations will be calculated between all potential confounders and the presence of metabolic syndrome and between potential confounders and the psychological variables of interest. Based upon the results of this analysis, significant confounders will be controlled statistically. Age, sex, income, education, and diuretic use will be examined as potential effect modifiers. Confounder adjusted associations will be presented between the psychological variables and metabolic syndrome subtypes based on the number and combination of syndrome features.

6. Data Requirements
The primary variables will come from Visit 2: waste-to-hip ratio, fasting glucose, insulin, triglycerides, HDL cholesterol, prevalent hypertension, prevalent diabetes, trait anger, and vital exhaustion. Effect modifiers to be examined include: age, sex, income, education, and diuretic use. Potential confounders include: BMI,
ETOH, smoking, physical activity, and total calories. Participants taking psychotropic agents will be excluded.