1. Title: Family CHD History and Anthropometry

2. Writing Group:
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3. Timeline:
   Begin analyses during Summer 1995, draft by Fall 1995

4. Rationale

   Familial studies have estimated that 25% of the variance in body mass index across generations can be explained by genetics, with very little of this heritability accounted for by subcutaneous fat accumulation. A central fat deposition pattern is associated with elevated levels of triglycerides, insulin, and glucose, and with increased prevalence of NIDDM and hypertension compared with either general obesity or a peripheral fat pattern. Elevations in these cardiovascular risk factors are believed to be the result of excess release of free fatty acids from visceral depots directly into the portal circulation. Limited information is available, however, as to how a familial predisposition toward central fat deposition is associated with cardiovascular endpoints. We propose to compare markers of abdominal fat distribution in ARIC participants with a high familial CHD risk versus those with a lower familial CHD risk.

5. Main hypotheses:

   1) Waist circumference, waist-to-hip ratio, and body mass index are higher in participants with an elevated family risk score.
   2) Waist and waist-to-hip ratio remain independently associated with family CHD risk after adjusting for general obesity (estimated by body mass index).
   3) The above associations are found in all four race/gender groups (black females, black males, white females, white males).

6. Data:

   Data are from ARIC visit 1. Obesity and fat-patterning (BMI, WHR, and waist circumference) will be estimated using family risk score as the independent variable, derived from all 4 ARIC field centers, and assessed in race/gender specific models. To determine the association of familial aggregation of CHD risk with central adiposity, models predicting WHR and waist circumference will be adjusted for BMI. Potential covariates that will be controlled for after the univariate modeling include age, pack-years of smoking, ethanol intake, physical activity, and education. LDL, HDL, triglycerides, insulin, glucose, diabetic prevalence and hypertensive prevalence will be examined as potential intervening variables, both by inclusion/exclusion in the multivariable models and by factor analysis to assess clustering of metabolic factors with central obesity. Univariate results will also be compared with estimates obtained using FRSFH31, the validated familial risk score, from two ARIC/FHS sites.