1. Title: Dietary Antioxidants and Carotid Artery Plaque

2. Writing Group:
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3. Rationale:
This project is an extension of AWG 069 which examined dietary antioxidant consumption and carotid artery wall thickness at the baseline visit. In that analysis wall thickness was inversely related to both dietary vitamin E and vitamin C intake, the relationship with carotenoid intake was less clear. The associations seen for vitamins E and C were consistent with their ability to reduce LDL's susceptibility to oxidation, and the hypothesized role that LDL oxidation plays in the development of atherosclerotic disease. The goal of this analysis will be to determine whether or not there is a relationship between dietary antioxidants and the prevalence of carotid artery plaque. This determination will be done in the context of wall thickening, that is, the focus of the study will be to determine whether or not dietary antioxidants predict the presence or absence of plaque after adjusting for wall thickness. This analysis may prove informative if the antioxidants under study operate at other steps in the natural history of complex lesion formation in addition to any influence on LDL oxidation.

4. Main Hypothesis:
The hypothesis to be tested is that dietary antioxidants will be inversely associated with the presence of carotid artery plaque at Visit 1. We speculate that this association will persist after adjusting for the mean carotid artery wall thickness. The dietary antioxidants to be considered will be vitamin E, vitamin C and provitamin A carotenoids. In addition to the presence or absence of plaque, we will analyze the total number of plaques visualized and the presence or absence of shadowing.

5. Data:
This study will be done using V1 data. The following information will be required:
Demographics: age, gender, race, education, clinic.
Ultrasound variables: average wall thickness and derived variables for plaque, and plaque + shadowing. All diet and nutrition variables. The medication codes for the V1 medication survey.
Covariates: Complete smoking history, alcohol use, LDL and HDL cholesterol, replacement estrogen use, diastolic and systolic blood pressure, fasting plasma glucose, the presence of clinically manifest and symptomatic CHD at baseline. The analysis will use contingency table analysis and logistic regression to determine the relationship between dietary antioxidant intake and the prevalence of plaque adjusting for wall thickness and potential confounders.