1. Title (length 26):
Cigarette Smoking and Carotid Atherosclerosis

2. Writing Group (list individual with lead responsibility first):
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3. Timeline:
To follow Ms. 002 (B-Mode Ultrasound: Associations with CHD Factors) in the analysis of traditional risk factors for carotid atherosclerosis. Some of the analyses can be done in conjunction with MS 002.

4. Rationale:
A review of recent articles (1-11) shows smoking to be a major risk factor for carotid atherosclerosis. For the most part, smoking has been considered as a dichotomous variable (ever vs. never or current vs. non-current). This is also the proposed strategy for MS 002. In addition, most previous studies have considered hospital-based populations. We propose to generalize these findings in two ways: (I) extension of findings to a population-based cohort, and (2) consideration of pack-years and time-since quitting variables in the prediction of carotid atherosclerosis. In addition, since the mechanism by which smoking is related to atherosclerosis is not well understood, we propose to explore the influence of potential mediators, such as white cell counts, platelets, fibrinogen, and alcohol use on this relationship.

5. Main Hypothesis:
Carotid far wall thickness will increase with pack years smoked. After controlling for pack years smoked and age, as well as other risk factors, carotid far wall thickness will decrease as time since quitting increases.

6. Data (variables, time window, source, inclusions/exclusions):
Variables will primarily consist of the same set used for MS.002. In addition, variables from the smoking questionnaire, as well as platelets, hematocrit, white cell count, alcohol, fibrinogen will be required. The time window, source, and inclusions/exclusions will be the same for those used for Ms. 002.

Keywords: Smoking, wall thickness, chemistry, alcohol

SELECTED REFERENCES


