Background and Hypothesis

• Cardiovascular disease (heart disease and stroke)
  – Leading cause of death in US
  – Heart disease kills 30% of Californians

• Atherosclerosis is the primary cause of heart disease and stroke

• Atherosclerosis is an inflammatory disease
  – High LDL cholesterol responsible for 50% of the disease risk
  – Other harmful components interact with lipids to create inflammatory response

• Ambient levels of particle pollution (PM2.5) may contribute to atherosclerosis through an inflammatory response
Study Design*

- Baseline health data from two USC clinical trials on atherosclerosis (798 healthy adults >40 years)

- Baseline Carotid Intima-media Thickness (CIMT) and other clinical data (LDL cholesterol, blood pressure)

- Questionnaires on risk factors (lifestyle and demographics)

- Exposure assignment: ambient concentration of PM2.5 assigned to the ZIP code area of each subject’s residential address

Results

% Difference in CIMT per $10\,\mu g/m^3$ PM2.5 (95% CI)

- Total sample, N=798: 4.4 (0-9.0)
- Lipid lowering medication, N=109: 13.3 (0-28.5)
- Women >60, N=186: 15.7 (5.7-26.6)

% Difference in CIMT per $20\,\mu g/m^3$ PM2.5 (95% CI)

- Lowest to highest exposure: 12.1 (2.0-23.1)*

Statistically significant increase in CIMT with increasing pollutant levels (dose-response).

* unadjusted value
Research Implications

• Evidence is accumulating that…
  – “air pollution may accelerate the development of coronary atherosclerosis and worsen its sequelae.”
    – (American Heart Association)

• Increase in CIMT corresponds to 3-6% long-term increase risk for heart attack

• To account for short- and long-term effects of PM on health, prospective cohort studies will be needed

• Atherosclerosis results from a complex process and this response may be the result of a combination of various urban pollutants interacting with host factors