AIRGENE STUDY GROUP

AIR POLLUTION AND INFLAMMATORY RESPONSE IN MYOCARDIAL INFARCTION SURVIVORS: GENE-ENVIRONMENT INTERACTION IN A HIGH RISK GROUP (AIRGENE)

INTRODUCTION

Epidemiological research during the last decade has indicated that exposure to air pollution at the levels presently measured in European urban environments is associated with an increase in mortality and also with a variety of health conditions, including emergency room visits and hospital admissions for respiratory and cardiovascular diseases. Particulate matter (PM) appears to be the air pollutant most consistently associated with adverse health outcomes.

Although the toxicological mechanism has not yet been established, the small size fraction of ambient aerosols, measured as PM10 (particles with an aerodynamic diameter less than 10μm) or PM2.5 (less than 2.5μm), rather than the larger particles is considered to be responsible for most of the health effects. The number of concentrations of ultrafine particles (0.01 to 0.1μm) is hypothesised to be of particular concern.

It is widely accepted that cardiovascular disorders, especially coronary heart disease, are the most prevalent chronic health conditions affecting both sexes in the western world, entailing enormous health care costs.

STUDY SETTING

Study area: Athens, Augsburg, Barcelona, Helsinki, Rome, Stockholm
Study period: May 2003 – April 2004
Study population: 1200 myocardial infarction survivors
Measurements: Ultrafine particle concentrations will be measured at a central site in each city and traditional air pollution concentrations will be obtained from the local air hygiene networks. Inflammatory markers and single nucleotide polymorphisms will be determined in a core laboratory.
Statistical analyses: Time-series methods; gene-environment interactions will be analysed for subgroups

AIMS OF THE STUDY

- To assess the inflammatory response in association with ambient concentrations of air pollution in myocardial infarction survivors in 6 European cities.
  - To determine dose-response relationships between air pollutants and biomarkers of systemic inflammation in myocardial infarction survivors.
  - To compare the inflammatory response of ultrafine particles to the response of traditional air pollutants.
- To define susceptible subgroups of myocardial infarction survivors based on genotyping.
  - To determine the role of the variation in genes of inflammatory responses by assessing the gene-environment interactions for air pollution exposures.
  - To provide insight into the mechanisms leading from exposure to ambient air pollution to early biological effects in high-risk populations.

APPLICATION OF RESULTS

To provide information that will facilitate the development of appropriate public health strategies to reduce the negative effects of ultrafine particles and traditional air pollutants on the exacerbation of cardiovascular disease.

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