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.

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.
