Environmental pollution has many facets, and the resultant health risks include diseases in almost all organ systems. Many infections are acquired by inhalation and ingestion of pathogens. Airborne diseases are spread when droplets of pathogens are expelled into the air due to coughing, sneezing or talking. Water-borne diseases are infectious diseases spread primarily through contaminated water.

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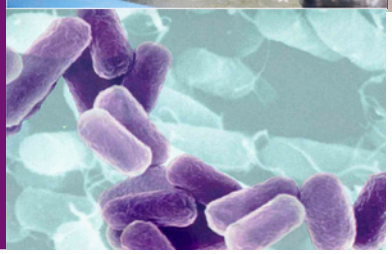

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Editorial Open Access

Air Pollution in Health and Diseases

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Cardiovascular disease (CVD) is the major cause of death in North America. Approximately 1 million people per year die of CVD in the United States which accounts for over 40% of all deaths in the US. Numerous advancements have been made in the field of health sciences, which have resulted in reduced deaths due to CVD. Recent statistical data suggest that CVD induced mortalities are on the rise again, and due to significant increase in the incidence of diabetes and obesity, the total burden of CVD in the future may even be greater. Numerous risk factors such as age, race, and lifestyle play an important role in the development of CVD; however, many patients with cardiac diseases show no obvious established risk. Furthermore, it has been shown that when genetically similar populations migrate to a new environment , the CVD risks are altered, suggesting that environmental factors may play an important role in the development of heart disease. Out of 350,000 sudden cardiac deaths each year in the United States, 60,000 deaths could be related to air (environmental) pollution. Ample evidence for links between environmental exposure and CVD has been accumulated over the years, the importance of these exposures as risk factors for the induction and development of CVD has only recently been seriously considered. Funding agencies such as the National Institute of Health (NIH), American Heart Association (AHA), and Health Effects Institute (HEI) have recognized the detrimental role played by air pollution in cardiac diseases and accordingly have increased the priority of research funding in an effort to understand the role of environmental pollutants in CVD.

The United States Environmental Protection Agency (USEPA) is required to set National Ambient Air Quality Standards (NAAQS) under the Clean Air Act and Ozone (O_3) is one of the six pollutants under consideration. Understanding the relationship between O_3 exposure, and disease, and mortality can significantly affect decisions related to the stringency of air pollution controls and thus has important implications for the impacts of these controls on human health. The concern is that while considering the benefits of air pollution standards, the mortality risks associated with O_3 exposure were excluded from the USEPAs analyses.

Health risks in human populations are increasingly being assessed by the use of empirical data from epidemiological studies. Although epidemiologic evidence for air pollution as an important and modifiable determinant of cardiovascular diseases is very strong, epidemiologic data have limitations of imprecise measurements. Air pollution epidemiologic research is challenged by the complexity of human exposure to environmental agents and by the difficulty of accurately measuring exposure. Residents are usually ubiquitously exposed to air pollution. In order to detect small effects of air pollution, both high statistical power and sophisticated study design are required. In addition, the characteristics of air pollutants vary and their concentrations change both spatially and temporally. Although everyone is susceptible to high concentration of pollution, its concentrations are not evenly distributed across populations. In addition, epidemiologic studies are limited by joint exposure to multiple pollutants and lack of clinical experimental models makes it difficult to systematically study the effects of individual pollutants and to demonstrate the individual risk. Due to such complexities, there are still many research questions to be addressed by future air pollution epidemiological studies. We believe that use of new tools such as geographic information systems, personal monitoring devices, and better measures of the full toxic air pollution mix may provide more refined estimates of the adverse health effects (diseases) that can be related to specific components of air pollution.

Epidemiological studies show statistical associations between health outcomes and exposure; they cannot establish a definite cause-effect relationship. Although the utility of animal studies is to establish this relationship, in some cases the evidence from animal experiments is of uncertain relevance for human populations. The interspecies dose extrapolations used to adjust for differences between humans and laboratory animals, and the extrapolations using statistical bioassay models for the high doses used in animal experiments to the much lower doses to which humans are likely to be environmentally exposed are the two major extrapolations required when animal data are used to estimate risks of human exposure.

We have established a systematic approach to researching the dose-dependent effects of single pollutant exposure using a controlled environment. Our current observations in normal adult rats are made from precise ozone measurements in the absence of other confounding factors such as PM or preexisting diseased states such as diabetes. We are the first to demonstrate a significant cardiovascular dysfunction in rats exposed to chronic levels of O_3. This cardiac injury was associated with a significant increase in circulating levels of cytokines, including TNF-α. Other laboratories have shown improved myocardial function in hearts extracted from rats exposed to low levels of O_3. Collectively, the studies described herein suggest that acute and chronic exposure to O_3 may have different effects on myocardial function. We hypothesize that the O_3 induced cardiac injury as demonstrated in our studies may be associated with the chronic effects of O_3 on organs ("Toxic rain"- due to prolonged exposure). On the contrary, in studies from other labs, when isolated hearts are exposed to O_3, the progressive toxicity of O_3 exposure would be absent and the O_3 associated effects in that case may be acute in nature.

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