American Stroke Association
A Division of American Heart Association
American Heart Association
Learn and Live

July 25, 2007

The Honorable Susan Fargo, Senate Chair
The Honorable Peter Koutoujian, House Chair
Joint Committee on Public Health
State House, Room 130
Boston, MA 02133

Dear Chairwoman Fargo and Chairman Koutoujian,

The American Heart Association supports House Bill 2227: An Act Relative to Reporting on Health Effects of Particular Matter. The American Heart Association is the largest voluntary organization in the world working to reduce disability and death from cardiovascular disease and stroke. Over the last decade, a growing body of epidemiological and clinical evidence has led to a heightened concern about the potential deleterious effects of ambient air pollution on health and its relation to heart disease and stroke. Of special interest are several environmental air pollutants that include carbon monoxide, oxides of nitrogen, sulfur dioxide, ozone, lead, and particulate matter. These pollutants are associated with increased hospitalization and mortality due to cardiovascular disease, especially in persons with congestive heart failure, frequent arrhythmias, or both. The well-established causal associations between active and passive smoking with heart disease and stroke support the plausibility of an adverse effect of PM on the cardiovascular system. Collectively, studies suggest that air pollution may accelerate the development of coronary atherosclerosis and worsen its sequelae. Some of these effects may occur over time, as with acceleration of the progression of atherosclerosis, or rather abruptly, as with the triggering of an arrhythmia or myocardial infarction by acute inflammatory responses, altered platelet adhesiveness, or perhaps vascular endothelial dysfunction.

Airborne PM consists of a heterogeneous mixture of solid and liquid particles suspended in air, continually varying in size and chemical composition in space and time. Primary particles are emitted directly into the atmosphere, such as diesel soot, whereas secondary particles are created through physicochemical transformation of gases, such as nitrate and sulfate formation from gaseous nitric acid and sulfur dioxide (SO2), respectively. The numerous natural and anthropogenic sources of PM include motor vehicle emissions, tire fragmentation and resuspension of road dust, power generation and other industrial combustion, smelting and other metal processing, agriculture, construction and demolition activities, residential wood burning, windblown soil, pollens and molds, forest fires and combustion of agricultural debris, volcanic emissions, and sea spray. Although there are thousands of chemicals that have been detected in PM in different locations, some of the more common constituents include nitrates, sulfates, elemental and organic carbon, organic compounds, biological compounds, and a variety of metals (eg, iron, copper, nickel, zinc, and vanadium).

An association between high levels of anthropogenic air pollutants and human illnesses has been known for more than half a century. The London fog incident of 1952 sparked the initial epidemiological research. As a result, a several-decades-long effort to reduce air pollution ensued and culminated in the Clean Air Act legislation of 1970. Despite improvements in air quality over the past few decades, associations between current ambient pollution levels and excess morbidity and mortality have been consistently detected. Although many pollutants may cause disease individually or in combination over the past decade, PM has become a major focus of research. During the past 15 years, the magnitude of evidence and number of studies linking air pollution to cardiovascular diseases has grown substantially. Observations related to the adverse health effects of short-term exposure are more numerous. In these studies, population-wide changes in acute outcomes (mortality, symptomatology, hospitalizations, and healthcare visits) are linked to short-term variations in ambient pollutant concentrations, most frequently through the use of population-based time-series analysis.

Until recently, the specific causes of the increased cardiovascular mortality due to long-term air pollution exposure have remained unclear. In the ACS study published this year, the investigators reported PM-mortality associations with the

specific cause of death. A statistically robust association between PM and overall cardiovascular mortality was confirmed for an increase in long-term exposure. The single largest increase in risk was for ischemic heart disease, which also accounted for the largest proportion of deaths. In addition, the risk for arrhythmia, heart failure, or cardiac arrest mortality was also increased. These findings suggest that air pollution promotes both ischemic and nonischemic cardiovascular events. Many studies provide strong evidence for the occurrence of adverse cardiovascular effects due to long-term air pollution exposure. However, many more studies have focused on short-term relationships between pollution exposure and adverse outcomes. The acute effects of air pollution are generally investigated by time series analyses of changes in health outcomes (eg, mortality) in relation to day-to-day variations in ambient air pollution concentrations. The finding indicated that the increase in cardiopulmonary mortality was not simply the result of “harvesting” (also called mortality displacement, which refers to the advancement of death by no more than a few days for severely ill individuals). Analyses of data from other locations also have indicated that the increased risks cannot be explained solely by harvesting and that longer lags are associated with higher relative risks of cardiopulmonary mortality. The higher relative risks demonstrated by using this statistical modeling may reflect the accumulation of both acute and subacute health effects over the longer lag periods. Just this year a study in the New England Journal of Medicine linked long term exposure to air pollution with incidences of increased risk of cardiovascular events in women.

The increase in relative risk for cardiovascular disease due to air pollution for an individual is small compared with the impact of the established cardiovascular risk factors. However, because of the enormous number of people affected, even conservative risk estimates translate into a substantial increase in total mortality within the population. The impact on cardiovascular disease therefore represents a serious public health problem. The latest draft of the US EPA Air Quality Criteria for Particulate Matter has confirmed the presence of an apparent linear dose-response relationship between PM and adverse events. On a global scale, the World Health Organization has estimated that 800 000 deaths occur per year and 7.9 million disability adjusted life-years are lost annually due to PM exposure. Given the burden of epidemiological evidence, the US EPA updated the National Ambient Air Quality Standards in 1997 to specifically include PM. The most current estimates by the EPA suggest that attainment of these standards would reduce total mortality by 23 000 deaths annually and cardiovascular hospital admissions by 42 000 per year in the United States. Nevertheless, 19% of all US counties with air-quality monitoring systems are presently not meeting these standards. House Bill 2227 will help to gain a better understanding of the level of exposure that the residents of the Commonwealth’s have to PM. The AHA will actively work to educate the public and public policy makers about the effects of air pollution on cardiovascular disease. The American Heart Association appreciates your time and consideration on this legislative matter.

Sincerely,

Allyson Perron
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