Ischaemic heart disease and fine particulate air pollution

C Arden Pope III

A large and growing body of research has provided increasingly compelling evidence that exposure to particulate matter (PM) air pollution contributes to cardiovascular disease (CVD) morbidity and mortality.[1] Overall, the evidence suggests that fine, combustion-related particulate pollution, measured as $PM_{2.5}$ (PM with an aerodynamic diameter of less than 2.5 μm), is most strongly associated with adverse CVD health outcomes. Studies have evaluated day-to-day changes in mortality risk associated with short-term (one to a few days) changes in PM air pollution as reviewed elsewhere.[1] One of the earliest of these studies[2] evaluated daily mortality counts in London for the winters of 1958–1972, a period of time with episodes of extremely high concentrations of air pollution. Elevations in air pollution were associated with increased risk of mortality and the exposure–response relationship was non-linear across the full range of exposures: relatively steep at lower levels of exposures and flattening out at higher levels of exposures.[2]

Over the subsequent decades, many studies have evaluated associations between short-term changes in air pollution and changes in risk of mortality, hospitalisation and other health endpoints, including ischaemic heart disease (IHD) events.[1] Many of these studies provide evidence that short-term exposure to PM pollution can play a role in triggering acute IHD events. Most recent studies have been conducted in countries with relatively clean air and observed exposure–response relationships that are near linear. A recent daily time-series mortality study that used data from 16 Chinese cities,[3] however, observed that risks of total, CVD and respiratory mortality were associated with PM_{10} (PM with an aerodynamic diameter of less than 10 μm, which includes $PM_{2.5}$ but also includes coarser PM with an aerodynamic diameter between 2.5 and 10 μm).

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Xie *et al*[4] report the results from an extremely interesting study of the temporal associations between short-term changes in air pollution and IHD events (including mortality). This is a highly pertinent and important study for several reasons including: the study used data from a large, prominent city (Beijing, China); the study included an exceptionally large range of pollution exposure; the study used central-site daily monitoring of $PM_{2.5}$; and the study used contemporary statistical analyses techniques that allowed for a robust evaluation of the shape of the exposure–response relationship. Xie *et al*[4] observed that the exposure–response relationship was relatively steep and nearly linear at the low range of exposure, largely consistent with contemporary studies from less polluted areas. However, they also observed that the exposure–response function levelled off at higher levels of exposure, similar to the pollution–mortality associations observed in historically highly polluted London.[2]

There is also evidence that long-term $PM_{2.5}$ exposure (years or decades) may contribute to the initiation and/or progression of CVD.[1] For example, a recent study[5] using data from the Los Angeles, California metropolitan area, evaluated associations between air pollution and progression of subclinical atherosclerosis based on changes in carotid artery intima-media thickness (CIMT). $PM_{2.5}$ and proximity to traffic-related sources of air pollution were associated with larger changes in CIMT. Also, the multi-ethnic study of atherosclerosis and air pollution reported associations between elevated $PM_{2.5}$ exposures and faster rates of CIMT progression.[6]

Prospective cohort studies have further demonstrated increases in risk of mortality associated with long-term exposures to $PM_{2.5}$. Overall, $PM_{2.5}$–mortality effect estimates of long-term exposures are much higher than estimates from short-term exposures. These studies also generally indicate that increased mortality risk is often most pronounced for CVD and IHD mortality.[1] One cohort study was conducted in four cities in Northern China.[7] Long-term exposure to PM_{10} air pollution was associated with increased CVD mortality, with the largest effects being with IHD mortality. Central estimates from various cohort studies suggest that a 10 $\mu g/m^3$ incremental increase in long-term exposure to $PM_{2.5}$ is associated with an approximately 7% excess risk of all-cause mortality and approximately a 12% excess risk of CVD mortality.[1] Interestingly, analyses that integrate information from studies of air pollution, secondhand cigarette smoke and active cigarette smoking also provide evidence that the exposure–response relationship between long-term exposure to $PM_{2.5}$ and CVD mortality is non-linear: relatively steep at lower ranges of exposure and flattening out higher levels of exposure.[8]

In conclusion, research reported by Xie *et al*[4] in this issue provides an intriguing and important contribution to the literature regarding the cardiovascular health effects of $PM_{2.5}$. It provides evidence that this air pollution contributes to CVD and the triggering of IHD events. It also provides evidence that the incremental effects of increased pollution decline at extremely high levels of pollution. Additional time-series and case-control studies in other cities of China, India and elsewhere with extremely high levels of pollution are needed to help confirm these results. Furthermore, cohort and related studies that evaluate effects of long-term exposure are also needed to help provide a more complete understanding regarding the shape of the exposure–response relationships across the full range of exposures, including highly polluted areas of the world.

Competing interests None.

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