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Letters

Lowering Air Pollution Levels in Massachusetts May Prevent Cardiovascular Hospital Admissions

Numerous studies have found higher cardiovascular (CVD) risk associated with particulate matter smaller than 2.5 μm in diameter ($PM_{2.5}$) (1). Most studies have used classical regression methods, which are limited in their ability to establish causality. Alternatively, causal inference analyses in the potential-outcomes framework (2) formalize assumptions required to identify causal associations from an observational study while minimizing reliance on subjective specifications of unknown model forms. We estimated the number of cause-specific CVD hospital admissions—all CVD, myocardial infarction, ischemic stroke, and congestive heart failure—attributable to high levels of 2-day exposure to $PM_{2.5}$ using a causal modeling approach.

We constructed daily ZIP code counts of CVD hospital admissions of Medicare beneficiaries older than 65 years across Massachusetts (2000 to 2014). We obtained daily $PM_{2.5}$ exposure estimates from a satellite-based model (3). We defined a high-pollution day as 2-day average $PM_{2.5}$ exposure ≥75th percentile (≥10.6 μg/m^3). Under the potential-outcomes framework, each high-pollution ZIP code day has 2 potential outcomes—the number of admissions had it been a low- or high-pollution day—but only the outcome under high pollution is observed. We used matching to estimate the missing low-pollution potential outcomes. We stratified the sample by year, and within each year used propensity score matching to match each high-pollution ZIP code day to a low-pollution one with similar temporal and socioeconomic characteristics. Because the measured confounder values are roughly equal in each matched pair, the outcome in the matched low-pollution ZIP code day serves as an estimate of the missing potential outcome under low pollution. We estimated the $PM_{2.5}$-attributable hospital admissions by calculating the difference between the counts of admissions in the low- and high-pollution ZIP code days in each matched pair and summing across all pairs. Further details are available in Baccini et al. (4).

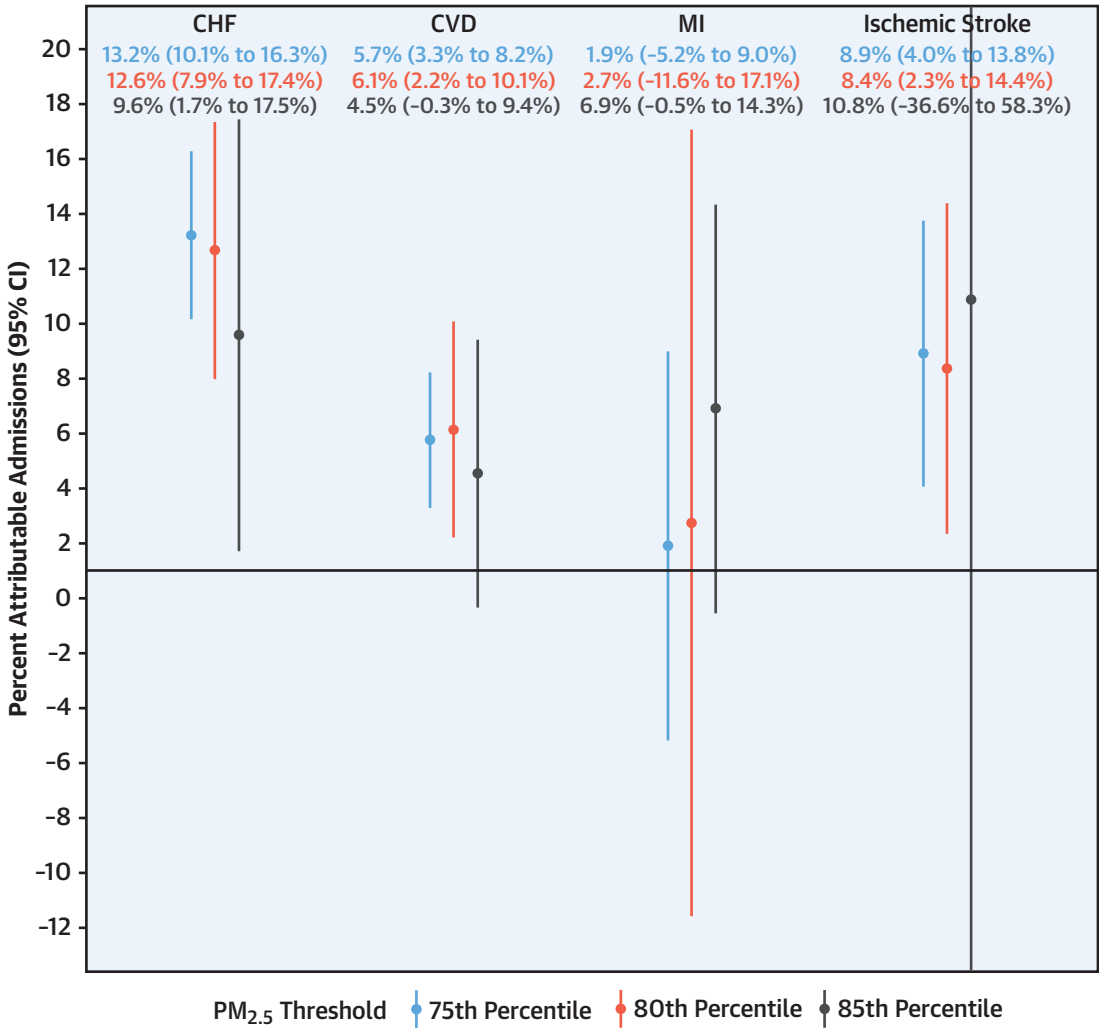
We observed 886,874 high-pollution ZIP code days in the study period. All confounder distributions were well balanced after matching. The mean $PM_{2.5}$ exposure was 6.9 μg/m^3 and 15.5 μg/m^3 in low- and high-pollution ZIP code days. A total of 10,087 (95% confidence interval [CI]: 5,727 to 14,447) CVD, 2,844 (95% CI: 1,286 to 4,410) ischemic stroke, and 7,449 (95% CI: 5,726 to 9,172) congestive heart failure admissions were attributed to high $PM_{2.5}$ exposure. These numbers correspond to 5.7% (95% CI: 3.3% to 8.2%) of the CVD, 8.9% (95% CI: 4.0% to 13.8%) of the ischemic stroke, and 13.2% (95% CI: 10.1% to 16.3%) of the congestive heart failure admissions that occurred during these high-pollution days. No association was observed with myocardial infarction: 568 admissions (95% CI: −1,553 to 2,690), which corresponds to 1.9% (95% CI: −5.2% to 9.0%). As a sensitivity analysis, the threshold for high-pollution days was changed to the 85th (13.2 μg/m^3) or the 95th (18.6 μg/m^3) percentile while keeping the original definition of a low-pollution day (<75th percentile). The percentage of $PM_{2.5}$-attributable admissions in high-pollution days remained similar (**Figure 1**).

We found significant numbers of CVD admissions among the elderly population in Massachusetts that were attributable to short-term exposure to $PM_{2.5}$. These findings agree with other studies that found significant increases in hospital admissions associated with $PM_{2.5}$ exposure (3). An advantage of this analysis is that the estimated effects are less sensitive to modeling choices regarding confounders. This causal evidence is important for clinicians who can play a major role in reducing the CVD burden attributed to $PM_{2.5}$, through interventions and health education tailored to patients at risk (5).

This study also has several limitations. First, we analyzed a binary rather than a continuous exposure. Second, residual confounding may still be present. Third, the results may not be generalizable to a younger population. Lastly, there is a possibility of exposure measurement error.

In conclusion, if an intervention had lowered the short-term exposure to $PM_{2.5}$ in Massachusetts, a significant number of CVD hospital admissions would

FIGURE 1 **Percentage of Cause-Specific CVD Hospital Admissions Attributed to High-Pollution Days, Comparing Different $PM_{2.5}$ Thresholds**



Shown are the proportions of admissions (with 95% confidence intervals [CIs]) in high-pollution days that are attributed to particulate matter smaller than 2.5 μm in diameter ($PM_{2.5}$), of the total count of admissions in these days, comparing different percentiles of 2-day average $PM_{2.5}$ as thresholds: the 75th (10.6 μg/m^3, marked in **blue**), the 80th (13.2 μg/m^3, marked in **red**), and 85th (18.6 μg/m^3, marked in **grey**). For all analyses, low-pollution days were days with exposure levels<10.6 μg/m^3. Days that were not within the category of low- or high-pollution days were removed from the analyses. The figure shows similar associations between $PM_{2.5}$ and all the admission causes across the different thresholds. CHF = congestive heart failure; CVD = cardiovascular disease; MI = myocardial infarction.

have been prevented. The Environmental Protection Agency Ambient Air Quality Standard was set to protect the public from the harmful effects of $PM_{2.5}$ pollution. However, our findings suggest that for the elderly population in the United States, harmful CVD consequences of $PM_{2.5}$ exposure can still occur even for those exposed to $PM_{2.5}$ level well below the 24-h 35 μg/m^3 Ambient Air Quality Standard set by the Environmental Protection Agency.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC* author instructions page.

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Decade-Long Temporal Trends in U.S. Hypertension-Related Cardiovascular Mortality

The 2017 American College of Cardiology/American Heart Association guideline proposes an aggressive approach to hypertension (HTN) management (1). The NHANES (National Health and Nutrition Examination Survey) study suggested that HTN-related cardiovascular disease (CVD) deaths in the United States significantly decreased between NHANES 1 (1971 to 1992) and NHANES 3 (1988 to 2006) (2). However, more recent studies have suggested that the rate of decline in overall CVD deaths has slowed since 2011—potentially related to the growing burden of diabetes, obesity, and heart failure (HF) (3). The HF-related CVD deaths show an inflection point: mortality rates decreased from 1999 to 2012 and then increase from 2013 to 2017 (4). Recent temporal trends in HTN-related CVD deaths are unknown; therefore, we examined these trends and identified subgroups at particular high risk.

We analyzed age-adjusted mortality rates attributed to HTN-related CVD using death certificates from the Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research from 2007 to 2017 (5). This data includes underlying and contributing causes of death from nationally standardized death certificates in the United States (3,4). Mortality rates were age-adjusted based on the 2000 U.S. census as the default population. HTN-related CVD mortality was defined as death where CVD (I00 to I99) was the underlying cause of death and HTN (I10 to I15) was the contributing cause of death. HTN (I10 to I15) included essential HTN, hypertensive heart and/or renal disease, and secondary HTN. Subgroup analysis included sex, U.S. census regions, age (45 to 55, 55 to 64, and 65 to 74 years), and urbanization status. Urbanization status was determined by the 2013 National Center for Health Statistics urban-rural classification scheme for counties. Urban was defined as large central metro or large fringe metro. Rural was defined as micropolitan or noncore.

Age-adjusted death rates (per 100,000) due to HTN-related CVD significantly increased over time (18.3 [95% confidence interval (CI): 18.1 to 18.4] in 2007 vs. 23.0 [95% CI: 22.9 to 23.2] in 2017; $p < 0.001$ for decade-long temporal trend). Age-adjusted death rates increased by 72% in rural and 20% in urban populations ($p < 0.001$ for time trend), and increased in each region. Age-adjusted death rates increased for all age groups over time, with the absolute highest rates of death in the oldest patient groups (age 65 to 74 years) at every time point. Although HTN-related CVD death rates were overall higher among men compared with women for all time points (2017 men: 25.7 [95% CI: 25.4 to 25.9] vs. women: 20.3 [95% CI: 20.1 to 20.5]), both sexes showed significant rates of increasing death over time ($p < 0.001$).

The highest death risk was identified in the rural south, which demonstrated an age-adjusted 2.5-fold higher death rate compared to other regions ($p < 0.001$) (**Figure 1**). The urban south also demonstrated increasing HTN-related CVD death rates over time: age-adjusted death rates in the urban South increased by 27% compared to all other urban regions ($p < 0.001$). But the absolute mortality rate (2017: rural south: 38.3 [95% CI: 37.1 to