Particulate Air Pollution and the Rate of Hospitalization for Congestive Heart Failure among Medicare Beneficiaries in Pittsburgh, Pennsylvania

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The authors used a case-crossover approach to evaluate the association between ambient air pollution and the rate of hospitalization for congestive heart failure among Medicare recipients (aged ≥65 years) residing in Allegheny County (Pittsburgh area), Pennsylvania, during 1987–1999. They also explored effect modification by age, gender, and specific secondary diagnoses. During follow-up, 55,019 patients were admitted with a primary diagnosis of congestive heart failure. In single-pollutant models, particulate matter with an aerodynamic diameter of <10 μm (PM$_{10}$), carbon monoxide, nitrogen dioxide, and sulfur dioxide—but not ozone—were positively and significantly associated with the rate of admission on the same day. The strongest associations were observed with carbon monoxide, nitrogen dioxide, and PM$_{10}$. The associations with carbon monoxide and nitrogen dioxide were the most robust in two-pollutant models, remaining statistically significant even after adjusting for other pollutants. Patients with a recent myocardial infarction were at greater risk of particulate-related admission; otherwise, there was no significant effect modification by age, gender, or other secondary diagnoses. These results suggest that short-term elevations in air pollution from traffic-related sources may trigger acute cardiac decompensation in heart failure patients and that those with certain comorbid conditions may be more susceptible to these effects.

Congestive heart failure (CHF) affects approximately 5 million people in the United States (1). Age-adjusted incidence rates do not appear to be increasing (2, 3), but the prevalence is expected to increase as the population ages and heart failure mortality continues to decline (4). Hospital discharges for CHF have increased 164 percent over the past two decades from 377,000 in 1979 to 995,000 in 2001 (1). Thus, the identification of precipitating factors that lead to acute cardiac decompensation and subsequent hospitalization is of considerable public health interest.

Acute decompensation can be precipitated by pathologic, behavioral (5–9), and environmental factors such as extreme temperatures (5), smoking (10), and exposure to carbon monoxide and air particles with an aerodynamic diameter of less than 10 μm (PM$_{10}$) (11–14). Although a secondary diagnosis of CHF may affect pollution-related hospitalizations for ischemic heart disease (15, 16), subgroups of CHF patients more susceptible to environmental triggers have not been identified.

Accordingly, we evaluated the following specific hypotheses: 1) short-term elevations in PM$_{10}$ are associated with an increased rate of cardiac decompensation and subsequent hospitalization for CHF, and 2) the increase in the rate is more pronounced for older persons and those with specific...
comorbid conditions. We evaluated these hypotheses by using the case-crossover study design (17), which is well suited to assessing the effects of transient exposures on the subsequent risk of acute events (18). Although our primary hypotheses relate to particulate matter, we also evaluated the association between ambient measures of other criteria pollutants and the rate of hospitalization for CHF.

MATERIALS AND METHODS

Study population

We obtained information on hospital admissions from the Centers for Medicare and Medicaid Services, which provides financial reimbursement of inpatient hospital admission costs for most US citizens aged 65 years or older. Cases were defined as persons admitted from the emergency room with a primary discharge diagnosis of CHF (International Classification of Diseases, Ninth Revision (ICD-9), codes 428.0 and 428.1) between January 1, 1987, and November 30, 1999, and residing in Allegheny County (Pittsburgh area), Pennsylvania, as noted in the Medicare claims record. For patients admitted multiple times, we considered only those admissions that occurred more than 14 days since the last included admission. Pittsburgh was chosen because it is one of the larger cities for which daily air quality data are available for many years. This study was granted an exemption from institutional review board review by the Harvard School of Public Health Human Subjects Committee.

Daily integrated (24-hour) measures of PM$_{10}$ and hourly measures of ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide were obtained from the Aerometric Information Retrieval System of the US Environmental Protection Agency. The measurement methods used to determine ambient concentrations of each pollutant have been described elsewhere (19). Measures of PM$_{10}$ were available from 17 monitoring sites, ozone from four sites, carbon monoxide from three sites, sulfur dioxide from 10 sites, and nitrogen dioxide from two sites. We computed local daily mean concentrations of each pollutant by using an algorithm that accounts for monitor-specific means and variances, as described previously (20). We excluded from analysis 2 days on which PM$_{10}$ levels exceeded 150 μg/m$^3$, the current 24-hour Environmental Protection Agency standard.

We obtained National Weather Service data on daily mean ambient temperature, barometric pressure, and dew point from the EarthInfo CD-ROM entitled NCDC Surface Airways (EarthInfo, Inc., Boulder, Colorado) and calculated apparent temperature (an index of human discomfort) as described previously (21–23).

Study design and statistical analysis

We used the case-crossover study design to assess the effect of changes in daily mean concentrations of PM$_{10}$, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone on the rate of hospitalization for acute decompensated CHF. Our primary exposure of interest was mean daily PM$_{10}$ concentrations. Exposure during the case period was defined as mean PM$_{10}$ concentration either on the day of admission (lag 0) or 1–3 days preceding admission (lags 1–3). Control periods were chosen by using the time-stratified approach (24, 25) such that exposures during the case period were compared with exposures occurring on all other days of the same month (before or after the event day) that fell on the same day of the week as the case period (26).

We performed conditional logistic regression, stratifying on each hospitalization, to obtain estimates of odds ratios and 95 percent confidence intervals associated with an interquartile-range increase in the mean daily level of each pollutant. In this paper, effect estimates are reported as percent change in rate of hospitalization. In all analyses, we modeled the mean apparent temperature at lag 0 as a quadratic function, and mean apparent temperature at lag 1 and mean barometric pressure at lag 0 as linear functions of continuous variables. We modeled PM$_{10}$, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone as linear functions of continuous variables. We first evaluated the effect of PM$_{10}$ separately at lags of 0–3 days in single-pollutant models. On the basis of the results of this analysis, we selected a single lag for all subsequent analyses. Pollutants were first considered separately by using single-pollutant models and then jointly by using two-pollutant models. We assessed the assumption of linearity by using standard methods, including examining the shape of the dose-effect curve fitted with fractional polynomials and linear splines, and found the assumption of linearity over the range of our data to be reasonable.

We examined effect modification by considering categories of age (≥80 vs. 65–79 years as the referent), gender, and the presence of secondary diagnoses of atrial fibrillation (ICD-9 code 427.31), other cardiac arrhythmias (ICD-9 code 427, except 427.31), chronic obstructive pulmonary disease (ICD-9 codes 490–496), essential hypertension (ICD-9 code 401), type 2 diabetes (ICD-9 codes 250.x0 and 250.x2), acute myocardial infarction within the past 30 days (ICD-9 code 410), old myocardial infarction (ICD-9 code 412), angina pectoris (ICD-9 code 413), other forms of ischemic heart disease (ICD-9 codes 411 and 414), and acute respiratory infections (ICD-9 codes 460–466 and 480–487).

All reported $p$ values are based on two-sided tests. A $p$ value of <0.05 was considered statistically significant. All analyses were performed by using SAS statistical software, version 8 (SAS Institute, Inc., Cary, North Carolina).

RESULTS

There were 55,019 admissions from the emergency room with a primary discharge diagnosis of CHF among Medicare beneficiaries residing in Allegheny County (Pittsburgh area), Pennsylvania, between January 1, 1987, and November 30, 1999. Of these cases, 37.4 percent were patients admitted only once for CHF during the observation period, and 86.8 percent were patients admitted five or fewer times. Among patients admitted more than once, the median time between admissions was 154 days. Age on the day of admission ranged from 65 to 108 years (mean, 78.9; standard deviation, 7.78). Other characteristics of the cases are summarized in table 1.
The distribution of average daily concentrations of particulate and gaseous air pollutants is shown in table 2, and pairwise correlations between pollutants are shown in table 3.

**PM$_{10}$ and the rate of hospitalization for CHF**

Initially, we estimated the effect of PM$_{10}$ separately for lags of 0–3 days and found a 3.07 (95 percent confidence interval (CI): 1.59, 4.57; $p < 0.0001$) percent increase in the rate associated with an interquartile-range increase in PM$_{10}$ on the day of admission (lag 0). When we controlled for the effect at lag 0, PM$_{10}$ levels 1–3 days before the admission day were not associated with an altered rate of hospitalization. Additionally, the use of a 2-day moving average (average of lags 0 and 1) did not materially alter the results. Therefore, in subsequent analyses, we considered PM$_{10}$ levels on the day of admission only. Controlling for ambient temperature and barometric pressure rather than apparent temperature also did not materially alter the results. Including only the first observed admission for each patient yielded a qualitatively similar, but less precise effect estimate.

Daily concentrations of other air pollutants were highly correlated with PM$_{10}$ levels (table 3). We used two-pollutant models to evaluate potential confounding by simultaneous exposure to these co-pollutants (table 4). Controlling for either ozone or sulfur dioxide did not materially alter the estimated effect of PM$_{10}$. However, in two-pollutant models controlling for either carbon monoxide or nitrogen dioxide, the effect of PM$_{10}$ was negligible.

**Gaseous co-pollutants and the rate of hospitalization for CHF**

In single-pollutant models, daily fluctuations in ambient levels of carbon monoxide, nitrogen dioxide, and sulfur dioxide—but not ozone—were positively and significantly associated with the rate of hospitalization for CHF on the same day (table 4). In two-pollutant models, the effects of both carbon monoxide and nitrogen dioxide were not materially changed by adjustment for most other measured pollutants, including PM$_{10}$. Ozone was not significantly associated with the rate of hospitalization for CHF in either single-pollutant or two-pollutant models.

**Identification of susceptible subgroups**

The effect of PM$_{10}$ was more than threefold greater in patients with a secondary diagnosis indicating a myocardial

<table>
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<tbody>
<tr>
<td>No. of cases ($n = 55,019$)</td>
</tr>
<tr>
<td>Age $\geq$ 80 years</td>
</tr>
<tr>
<td>Male gender</td>
</tr>
<tr>
<td>White race</td>
</tr>
<tr>
<td>Primary diagnosis</td>
</tr>
<tr>
<td>ICD-9* code 428.0†</td>
</tr>
<tr>
<td>ICD-9 code 428.1‡</td>
</tr>
<tr>
<td>Secondary diagnoses</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>COPD*</td>
</tr>
<tr>
<td>Essential hypertension</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
</tr>
<tr>
<td>Other arrhythmias</td>
</tr>
<tr>
<td>Acute respiratory infections</td>
</tr>
<tr>
<td>Old myocardial infarction</td>
</tr>
<tr>
<td>Recent myocardial infarction</td>
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<tr>
<td>Angina pectoris</td>
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</table>

* ICD-9, International Classification of Diseases, Ninth Revision; COPD, chronic obstructive pulmonary disease.
† Congestive heart failure, unspecified.
‡ Left heart failure.

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<thead>
<tr>
<th>TABLE 2. Distribution of average daily concentrations of air pollutants in Allegheny County, Pennsylvania, 1987–1999</th>
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</thead>
<tbody>
<tr>
<td>Mean (SD*) &amp; Percentile</td>
</tr>
<tr>
<td>% of days missing</td>
</tr>
<tr>
<td>PM$_{10}$* ($\mu$g/m³)</td>
</tr>
<tr>
<td>Carbon monoxide (ppm)</td>
</tr>
<tr>
<td>Nitrogen dioxide (ppb)</td>
</tr>
<tr>
<td>Ozone (ppb)</td>
</tr>
<tr>
<td>Sulfur dioxide (ppb)</td>
</tr>
<tr>
<td>Temperature ($°C$)</td>
</tr>
<tr>
<td>Apparent temperature † ($°C$)</td>
</tr>
</tbody>
</table>

* SD, standard deviation; PM$_{10}$, particulate matter with an aerodynamic diameter of $<10$ μm.
† Defined as a person’s perceived air temperature by using the formula $AT = -2.653 + (0.994 \times Ta) + (0.0153 \times Td^2)$, where AT is the apparent temperature, Ta is the air temperature in degrees Celsius, and Td is the dew-point temperature in degrees Celsius.
infection within the past 30 days (rate ratio = 9.62, 95 percent CI: 3.14, 16.52 vs. rate ratio = 2.80, 95 percent CI: 1.29, 4.32; p homogeneous = 0.042). Similarly, the effect of carbon monoxide was approximately twofold greater in patients with a recent myocardial infarction, but this difference in the rate ratios did not reach statistical significance (rate ratio = 8.99, 95 percent CI: 3.34, 14.95 vs. rate ratio = 4.36, 95 percent CI: 3.12, 5.62; p homogeneous = 0.12). In contrast, the effect of neither PM10 nor carbon monoxide was significantly stronger for patients with a secondary diagnosis of an old myocardial infarction. The effect of either pollutant did not vary significantly by age, gender, or the presence of any other secondary diagnosis examined.

**DISCUSSION**

Heart failure patients may remain asymptomatic for extended periods if compensatory mechanisms and/or treatment is sufficient to balance the cardiac dysfunction. Factors that commonly disturb this balance include treatment noncompliance, uncontrolled hypertension, atrial fibrillation, acute respiratory infections, and myocardial ischemia or infarction (5–9). Although short-term increases in ambient particle levels have been associated with cardiovascular morbidity and mortality (27, 28), relatively few studies have examined their effect on CHF morbidity. We found that short-term increases in mean daily levels of ambient particles and/or other pollutants may also precipitate acute cardiac decompensation leading to hospitalization, most notably in patients with a recent myocardial infarction.

In single-pollutant models, PM10, carbon monoxide, nitrogen dioxide, and sulfur dioxide (but not ozone) were significantly associated with CHF admissions—carbon monoxide, nitrogen dioxide, and PM10 most strongly. Previous studies found similar results for these three pollutants (13, 14, 29) or for only carbon monoxide and nitrogen dioxide, but not PM10 (30). In two-pollutant models, associations with carbon monoxide and nitrogen dioxide were the most robust, as noted before (12, 14, 29). However, given the high correlation among ambient measures of PM10, carbon monoxide, and nitrogen dioxide, two-pollutant modeling is of limited value in disentangling their effects.

Generally, the effect estimates derived from single-pollutant models (table 5) in the current study are similar to those from previous reports. For example, previous estimates of the increase in CHF morbidity range from 2 percent to 7 percent (13, 14, 29, 30) for a 1-ppm increase in ambient carbon monoxide. Previous estimates of the effects of PM10 have also been similar (14, 30, 31) although not always statistically significant. For instance, Schwartz and Morris (12) found a 0.99 (95 percent CI: 0.37, 1.60) percent higher rate of admissions for CHF associated with a 10-μg/m3 increase in 2-day mean PM10. Hoek et al. (32) found that a 10-μg/m3 increase in the 7-day mean concentration of PM10 was associated with a 0.44 (95 percent CI: −0.51, 1.40) percent increase in the risk of CHF mortality. Two other studies (33, 34) reported an association between ambient

<table>
<thead>
<tr>
<th>Pollutant (IQR)</th>
<th>Single pollutant</th>
<th>Adjusted for PM10†</th>
<th>Adjusted for carbon monoxide</th>
<th>Adjusted for nitrogen dioxide</th>
<th>Adjusted for ozone</th>
<th>Adjusted for sulfur dioxide</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM10 (24 μg/m3)</td>
<td>3.07, 1.59, 4.57</td>
<td>NA†</td>
<td>−1.10, −3.02, 0.86</td>
<td>0.52, −1.46, 2.53</td>
<td>2.80, 1.29, 4.33</td>
<td>2.18, 0.37, 4.02</td>
</tr>
<tr>
<td>Carbon monoxide (0.55 ppm)</td>
<td>4.55, 3.33, 5.79</td>
<td>5.18, 3.49, 6.89</td>
<td>NA†</td>
<td>4.84, 3.06, 6.66</td>
<td>4.35, 3.08, 5.64</td>
<td>4.51, 3.15, 5.90</td>
</tr>
<tr>
<td>Nitrogen dioxide (11 ppb)</td>
<td>4.22, 2.61, 5.85</td>
<td>4.05, 1.83, 6.31</td>
<td>−0.37, −2.59, 1.89</td>
<td>NA†</td>
<td>3.73, 2.10, 5.39</td>
<td>3.79, 1.93, 5.67</td>
</tr>
<tr>
<td>Ozone (17 ppb)</td>
<td>−1.60, −3.77, 0.61</td>
<td>−1.96, −4.14, 0.27</td>
<td>0.13, −2.12, 2.44</td>
<td>−1.19, −3.38, 1.06</td>
<td>NA†</td>
<td>−1.41, −3.58, 0.81</td>
</tr>
<tr>
<td>Sulfur dioxide (11 ppb)</td>
<td>2.36, 1.05, 3.69</td>
<td>1.35, −0.27, 2.99</td>
<td>0.10, −1.35, 1.57</td>
<td>0.68, −0.82, 2.21</td>
<td>2.02, 0.68, 3.37</td>
<td>NA†</td>
</tr>
</tbody>
</table>

* In models controlling for barometric pressure and apparent temperature.
† IQR, interquartile range; PM10, particulate matter with an aerodynamic diameter of <10 μm; CI, confidence interval; NA, not applicable.

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Increment</th>
<th>Increase in rate (%)</th>
<th>95% CI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{10}^*$</td>
<td>10 $\mu$g/m$^3$</td>
<td>1.27</td>
<td>0.66, 1.88</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>1 ppm</td>
<td>8.43</td>
<td>6.14, 10.77</td>
</tr>
<tr>
<td>Nitrogen dioxide</td>
<td>10 ppb</td>
<td>3.83</td>
<td>2.37, 5.30</td>
</tr>
<tr>
<td>Ozone</td>
<td>10 ppb</td>
<td>–0.95</td>
<td>–2.23, 0.36</td>
</tr>
<tr>
<td>Sulfur dioxide</td>
<td>10 ppb</td>
<td>2.14</td>
<td>0.95, 3.35</td>
</tr>
</tbody>
</table>

* CI, confidence interval; PM$_{10}^*$, particulate matter with an aerodynamic diameter of <10 $\mu$m.

particles and CHF mortality, but differences in exposure metrics for ambient particles prevent direct comparison to the current study.

Our effect estimate for PM$_{10}^*$ from the current study is similar to that of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), a large multicity study. With a 10-$\mu$g/m$^3$ increase in same-day PM$_{10}^*$, we found a 1.27 (95 percent CI: 0.66, 1.88) percent increase in CHF hospitalizations, while the National Morbidity, Mortality, and Air Pollution Study found a 0.84 (95 percent CI: 0.51, 1.18) percent increase in total cardiovascular disease hospitalizations among Medicare beneficiaries aged 65 years or older in Pittsburgh and a 1.02 (95 percent CI: 0.76, 1.27) percent increase in 14 cities combined (26).

Agent(s) responsible for the observed associations

In studies carried out in single cities, it is not possible to clearly distinguish the effects of multiple pollutants that co-vary strongly over time. Therefore, the effect we are attributing to ambient particles may be mediated at least in part by gaseous co-pollutants.

Three pieces of evidence support ambient particles as the responsible agent. First, ambient measures of gaseous pollutants at a central site are poorly correlated with personal exposure to those pollutants. For example, one study (35) found this to be true of nitrogen dioxide. Instead, ambient measures of both nitrogen dioxide and carbon monoxide were strongly and significantly associated with personal measures of exposure to particulate matter with an aerodynamic diameter of less than 2.5 $\mu$m and even more so with personal exposure to elemental carbon, a surrogate for traffic-related particles. Second, several large multicity studies have concluded that gaseous co-pollutants do not confound the association between PM$_{10}^*$ and daily deaths (28, 36, 37), and it may in fact be inappropriate to control for them. Third, biologic plausibility is evidenced by animal toxicologic and human panel studies, which suggest that particulate-related deceleration of heart failure patients may be mediated by relative increases in sympathetic nervous system activity (38, 39), inappropriate changes in vasmotor tone (40), or triggering or exacerbation of myocardial ischemia (41, 42). In contrast, carbon monoxide has been shown to have vasodilatory (43) and antiarrhythmic (44) properties in laboratory animals, both of which would be expected to reduce the risk of acute decompensation.

Most consistent with our data is the interpretation that elevations in a combination of ambient particles, carbon monoxide, and nitrogen dioxide are responsible for the association. In urban areas, the primary source of carbon monoxide is motor vehicle emissions, which also contribute significantly to ambient levels of nitrogen dioxide and PM$_{10}^*$. Thus, motor vehicle emissions may be the responsible exposure. This interpretation is supported by recent epidemiologic studies showing that living in proximity to major roadways is associated with increased rates of cerebrovascular (45), cardiopulmonary (46), and all-cause (47) mortality. Additionally, a study of 14 US cities (48) found that the magnitude of the association between PM$_{10}^*$ and cardiovascular hospital admissions increased significantly as the proportion of particles from highway vehicles increased. As discussed above, toxicologic evidence points to particles as the most likely culprit. However, toxicologic studies that examine the cardiovascular effects of traffic-related pollutant mixtures are still needed.

If causal, the results of the current study are noteworthy. Among those aged 65 years or older, the rate of hospitalization with a primary diagnosis of CHF is estimated as 222 per 10,000 person-years (49). If we take this as the underlying rate of hospitalization in the target population at the median PM$_{10}^*$ concentration and assume that PM$_{10}^*$ is an adequate marker of the pollution episodes responsible for the observed associations, then the daily PM$_{10}^*$ levels over the median value resulted in an approximately 1.5 percent excess rate of admission. Although this relative increase in rate is small, the absolute increase in hospital admissions may be quite high given that 995,000 hospitalizations for CHF took place in the United States in 2001 (1).

Identification of susceptible subgroups

Previous studies suggest that CHF patients are at greater risk of pollution-related hospitalization for ischemic heart disease (15) and acute myocardial infarction (16), as well as nonaccidental mortality (50, 51). However, to our knowledge, subgroups of CHF patients at increased risk of pollution-related acute decompensation have not been identified previously.

We did not confirm, for either PM$_{10}^*$ or carbon monoxide, recent reports that patients with diabetes may be particularly susceptible to the effects of ambient particles (16, 52–54), presumably because of increased endothelial dysfunction. The possibility that patients with a history of myocardial infarction may be at greater risk for pollution-related events was evaluated among Medicare beneficiaries in Cook County, Illinois (54). The association between PM$_{10}^*$ and all-cause mortality was more than twice as large among patients with versus without a history of myocardial infarction. We found that the association between PM$_{10}^*$ and the rate of admission for CHF was nearly threefold greater among patients with a recent myocardial infarction than those without. A smaller increase in the association, not statistically significant, was observed for carbon monoxide.
Enhanced susceptibility was much less pronounced among patients with an old infarct, suggesting that the remodeling myocardium may be more susceptible to pollution.

Limitations

First, particle concentrations were measured by using PM$_{10}$, whereas particulate matter with an aerodynamic diameter of less than 2.5 μm is the currently regulated measure based on studies suggesting their greater toxicity. Second, the use of ambient PM$_{10}$ levels rather than personal exposure measures, while highly correlated with indoor and personal PM$_{10}$ levels (55), will result in some nondifferential misclassification, tending to underestimate risk. Third, admission dates may have differed from the date of symptom onset, resulting in some exposure misclassification and underestimation of the true relative risk. Fourth, disease misclassification is also possible as a result of diagnostic or coding errors, although these errors are likely unrelated to pollution levels. While the direction of the resulting bias is expected to be toward the null, the magnitude is expected to be small.

A potential limitation inherent to research using administrative data sets is the lack of information on chronic risk factors for disease. However, a strength of the case-crossover design used in this study is that self-matching ensures that, within strata, there is no variability in—or confounding by—chronic risk factors, whether measured or unmeasured.

Summary and implications

Short-term increases in a combination of particulate air pollution, carbon monoxide, and nitrogen dioxide were associated with acute decompensation and subsequent hospitalization of heart failure patients, especially those with a recent myocardial infarction. This and other studies suggest that motor vehicle emissions are the responsible exposure. Because of the large number of hospitalizations for CHF, even a modest increase in rate can account for a large number of admissions, which may be preventable.

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REFERENCES


