A Case-Crossover Analysis of Out-of-Hospital Cardiac Arrest and Air Pollution

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Background—Evidence of an association between the exposure to air pollution and overall cardiovascular morbidity and mortality is increasingly found in the literature. However, results from studies of the association between acute air pollution exposure and risk of out-of-hospital cardiac arrest (OHCA) are inconsistent for fine particulate matter, and, although pathophysiological evidence indicates a plausible link between OHCA and ozone, none has been reported. Approximately 300,000 persons in the United States experience an OHCA each year, of which >90% die. Understanding the association provides important information to protect public health.

Methods and Results—The association between OHCA and air pollution concentrations hours and days before onset was assessed by using a time-stratified case-crossover design using 11,677 emergency medical service–logged OHCA events between 2004 and 2011 in Houston, Texas. Air pollution concentrations were obtained from an extensive area monitor network. An average increase of 6 μg/m^3 in fine particulate matter 2 days before onset was associated with an increased risk of OHCA (1.046; 95% confidence interval, 1.012–1.082). A 20-ppb ozone increase for the 8-hour average daily maximum was associated with an increased risk of OHCA on the day of the event (1.039; 95% confidence interval, 1.005–1.073). Each 20-ppb increase in ozone in the previous 1 to 3 hours was associated with an increased risk of OHCA (1.044; 95% confidence interval, 1.004–1.085). Relative risk estimates were higher for men, blacks, or those aged >65 years.

Conclusions—The findings confirm the link between OHCA and fine particulate matter and introduce evidence of a similar link with ozone. (Circulation. 2013;127:1192-1199.)

Key Words: sudden death ▪ heart arrest ▪ epidemiology ▪ particulates ▪ pollution ▪ ozone

Out-of-hospital cardiac arrest (OHCA) is defined as a condition characterized by an unexpected cardiovascular collapse due to an underlying cardiac cause occurring outside the hospital. It is of significant concern given that ≈300,000 persons in the United States experience an OHCA each year, and >90% of those persons who experience an OHCA die. Understanding the role of air pollution in increasing the risk of OHCA is important to protect public health. Evidence that short-term exposure to air pollution is associated with cardiovascular morbidity and mortality is increasingly found in the literature, especially with respect to fine particulate matter with an aerodynamic diameter <2.5 μm (PM_{2.5}), and, to a lesser extent, ozone. A handful of case-crossover studies have specifically examined the association between PM_{2.5} and ozone air pollution with a focus on OHCA or out-of-hospital cardiac death. However, in these studies, the results of an association between OHCA and PM_{2.5} have been inconsistent, and no association has been found between OHCA and ozone (eg, studies reported a range of −6.0% to 11.0% increase in risk of OHCA per 10 μg/m^3 increase in PM_{2.5} and −5.5% to 22.8% increase in risk of OHCA per 20-ppb increase in ozone).

Clinical Perspective on p 1199

In an effort to better understand the association of air pollution and OHCA, we used an extensive air-monitoring network and a large emergency medical service (EMS) call database spanning 8 years. We focused on 2 pollutants, PM_{2.5} and ozone, both with epidemiological evidence supported by pathophysiological arguments that link them to cardiac end points. We also examined the association between nitrogen dioxide, sulfur dioxide, and carbon monoxide with cardiac arrest. Our studies were conducted on both a daily and hourly time scale.

Methods

Out-of-Hospital Cardiac Arrest Data

The Rice University and Baylor College of Medicine Institutional Review Board approved all data-collecting procedures for human subjects. All cases in which EMS performs chest compressions are considered OHCA cases. The OHCA study data included non–dead-on-arrival adults aged 218 years from Houston Fire Department EMS calls over the 8-year period from 2004 to 2011. The database consisted of 11,677 cases of OHCA events. In addition to recording the
time and location of the event, other relevant information necessary for age, sex, race, and preexisting condition stratification were also available. This additional information was collected by EMS with the use of Ustien guidelines.19

Ambient Air Quality and Meteorologic Data
Ambient pollution concentration data were obtained from the Texas Commission of Environmental Quality for the 8-year study period of 2004 through 2011. In this analysis, hourly data from 47 monitors measuring ozone, 12 measuring PM$_{2.5}$, 22 measuring nitrogen dioxide, 13 measuring sulfur dioxide, and 12 measuring carbon monoxide were used. The hourly and daily average values of PM$_{2.5}$, ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide were calculated across monitors. For ozone, we calculated the daily maximum 8-hour running mean. The number of air monitors measuring a specific pollutant changed through the study years as monitors went on and off line. However, <1% of the time all monitors were simultaneously down. All air pollution data were collected by using Environmental Protection Agency federal reference methods20 and validated by the Texas Commission of Environmental Quality.

To control for potential confounding meteorologic events, 1-hour ambient meteorologic (temperature, relative humidity, and wind speed) data were obtained from the Texas Commission of Environmental Quality for the study years. These data were used to estimate the average hourly and daily ambient apparent temperature level during the study period. The apparent temperature was calculated with the method used by O’Neill et al21 originally described by Steadman22 and Kalkstein and Valimont.23

Statistical Methodology
The OHCA event, pollution, and meteorologic databases were analyzed by using a time-stratified case-crossover design coupled with conditional logistic regression. The case-crossover design was first introduced by Maclure24 and is used increasingly in the literature to assess episodic events following short-term exposure to air pollution.24–25 In the case-crossover design, each individual experiencing a health event serves as his or her own reference; in other words, individuals act as their own control. Ambient air pollution is used as a proxy for personal exposure. The ambient air pollution concentrations at times when the study individual is not experiencing the OHCA health event are the reference concentrations. The reference concentrations are statistically compared with the concentrations during or around the time the study individual experienced the OHCA health event. Conditional logistic regression is applied to estimate the association of pollution and increased relative risk of the health event while controlling for confounding factors.

In our application of the case-crossover design, we conducted an exploratory sensitivity analysis with single lag models to examine the association of air pollution and OHCA on 2 time scales: hour and day. The hour or day of the individual OHCA event (depending on the time scale being studied) was the initial exposure period (lag 0) considered for that case. For the hourly time scale analysis, we examined the association at the hour of onset (lag 0 hour) and 1 to 8 hours before onset (lag 1, 2, 3, 4, 5, 6, 7, 8). For the daily time scale analysis, we examined the association at the day of onset (lag 0 day) and the 1 to 5 before onset (lag 1, 2, 3, 4, 5).

We then implemented constrained distributed lag models to estimate the cumulative effect over 2-hour average or 2-day average increments (lag 0–1, lag 1–2, lag 2–3) for those pollutants where associations were indicated in our exploratory analysis. Referent exposures, selected by time-stratified sampling, were the exposures in the day (and hour for the hourly analysis) of the event on all days falling within the same month and on the same day of the week as the event. This reference period design has been shown to limit the bias present due to patterns in air pollution.24 A conditional logistic regression was used to estimate the relative risk associated with each pollutant. We included apparent temperature in our conditional logistic regression model by using a nonparametric smoothing spline of degree 3 with 4 knots optimally chosen.25–31

The EMS data, in which the call time acts as the time of the OHCA, provided the ability for an analysis on the hourly as opposed to the daily scale typically assessed. However, because both cardiac arrest and pollutant data may have diurnal patterns, temporal confounding must be considered.16 For our analysis of the hourly relationship, we explored the impact of the cardiac arrest temporal pattern in confounding our understanding of the relationship between OHCA and hourly air pollution (when an effect was found) by comparing the OHCA/air pollution relationship when the temporal OHCA pattern was constant to the finding from the full data set.

When a significant association between individual pollutants and OHCA was found, we investigated potential confounding between pollutants. We estimated correlations between pollutants on the daily and hourly scale and also included pollutants as a covariate in the model. The main concern was potential confounding between PM$_{2.5}$ and ozone as indicated by previous researchers.22 When a relationship was found between OHCA and an air pollutant, we stratified the analyses by age, sex, race, and season to examine the effects by subgroup. The case-crossover logistic regression was conducted in SAS version 9.3.33

Results
Figure 1 identifies the location of OHCA events for the 8-year period (geo-masked for privacy). The characteristics of the OHCA study group are shown in Table 1. Of the 11,677 qualified cases of OHCA during the study period, the largest percentage of cases were individuals between the ages of 35 and 64 years, more of the cases were male (59%) than female (41%), and most of the cases were of black individuals (46%), followed by white individuals (35%) and Hispanic individuals (16%). The data indicate that 79% of the cases presented with a preexisting condition, not necessarily cardiac related. Because of the stressful conditions during the EMS call, the designation of preexisting conditions by the victim or relatives is considered less reliable by the Houston EMS than the other data. For this reason, stratification by preexisting condition was not explored in this study. To evaluate the impact of the season, we broke the year into cold (November to March) and warm season (April to October). During the study period, 55% of the cases were in the warm season and 45% were in the cold season.

Statistics of the average hourly and daily pollutant levels during the study period are listed in Table 2. Pearson correlation coefficients between pollutants and apparent temperature on both time scales (hourly and daily) and each season (all, warm, and cold) were calculated (Table 3). Note, correlations vary between daily and hourly time scales because of different diurnal pollutant patterns. On the daily scale, the strongest correlation was between carbon monoxide and nitrogen dioxide at 0.75, 0.72, and 0.79 for all year, warm, and cold season. Ozone is most correlated with PM$_{2.5}$ on the day scale during the warm season (0.40, 0.37, and 0.26 for all year, warm, and cold season). On the hourly scale, there is little to no correlation between ozone and PM$_{2.5}$ during the warm season (0.01, 0.07, and −0.21 for all year, warm, and cold season).

Conditional logistic regression results for each pollutant on the hourly and daily time frame are summarized on Table 4 and Figure 2. The plots and the table offer different information. The plots graphically show the change in effect estimates with increasing lags for ozone and PM$_{2.5}$, whereas the table shows more limited ozone and PM$_{2.5}$ lags information and includes other pollutants.
**PM<sub>2.5</sub> Results**

The lag model results for PM<sub>2.5</sub> on the daily analysis scale indicate that a daily average increase of 6 µg/m³ in PM<sub>2.5</sub> in the 2 days before onset (average of 1 and 2 days) was associated with an increase of OHCA risk (1.046; 95% confidence interval [CI], 1.012–1.082). This was the strongest effect found. There was no effect after 3 days (1.021; 95% CI, 0.991–1.051).

**Ozone Results**

The lag model results for ozone on the hourly analysis scale indicate that each 20 ppb of ozone increase in the average of the previous 1 to 3 hours was associated with an increase OHCA risk (1.044; 95% CI, 1.004–1.085). This was the strongest effect found in the distributed lag model. No effect was found after 3 hours.

Also included in Figure 2 are the results for the single lag model for lag 0 day. The results indicate that an increase of 20 ppb of ozone for the 8-hour average daily maximum on the day of the event was associated with an increased risk of OHCA (1.038; 95% CI, 1.004–1.072). The finding of a significant association between OHCA and ozone on lag 0 day indicates that this association found on the hourly scale within the day of onset is not simply reflecting the temporal cardiac pattern. To further investigate confounding from the cardiac temporal pattern, we compared the results of the same analysis limited to a time of day when the cardiac temporal pattern was constant and found no change in the risk.

**Stratification and Sensitivity**

Analysis of stratification of the cases by the demographic characteristics of the data (age, sex, and race) found that the risk from exposure to ozone or PM<sub>2.5</sub> is highest for men (1.051; 95% CI, 1.006–1.097), those of black ethnicity (1.053; 95% CI, 1.003–1.105), and >65 years of age (1.049; 95% CI, 1.000–1.100) (Figure 3). The apparent temperature is most correlated with ozone on the hourly scale during the cold season (0.20, 0.03, and 0.39 for all year, warm, and cold season). The apparent temperature itself was not a significant predictor for OHCA, nor did the inclusion of apparent temperature change our conclusions related to the pollutants.
Discussion

We find consistent evidence of an association between OHCA and exposure to ozone in Houston, Texas at short time scales up to 3 hours in duration and also at the daily level on the day of the event. For exposure to PM$_{2.5}$, an association is found for 2 days before the event. Other pollutants were not found to impact the occurrence of OHCA.

Our findings add to the significant literature relating OHCA with PM$_{2.5}$, where findings across studies are inconsistent. Furthermore, we add to the small but growing scientific conversation relating OHCA and ozone. Finally, we bring the most comprehensive data set to date to this literature, in terms of duration of the study, number of pollution monitors included, and the number of OHCA events studied. The implications of this work are improved health policy and action with the objective of reducing the number of annual OHCA currently at ≈300,000 in the nation and 1460 in Houston.

Association Between PM$_{2.5}$ and OHCA

The association between PM$_{2.5}$ and OHCA varies across studies, which is due in large part to the variation in study design. A detailed synthesis of recent studies is provided in Raun and Ensor for both PM$_{2.5}$ and ozone. Some of the key features that varied across studies included the number of monitors used, the area covered, sample size of cases, the designation of health end point, the comorbidities studied, the method of pollution measurement, the composition of particulates, and the level of ambient concentration. The early studies, which did not find an association, had fewer OHCA events, lower PM$_{2.5}$ concentrations, and different PM$_{2.5}$ composition than the later studies that did find an association.

Association Between Ozone and OHCA

Although a few studies have examined the link between ozone and OHCA, there is growing evidence of a pathophysiological link. In the effects seen in animal toxicology studies after human ozone exposure, as well, researchers have found a reduction in serum tocopherol (free radical scavenger), an increase in the gradient of alveolar-to-arterial P$_{O_2}$ potentially due to alveolar-arterial oxygen impairment, and, most recently, changes in several proinflammatory cytokines in blood. The lack of investigation of the association between ozone and OHCA may stem from practical considerations such as data limitations. In some locations, ozone is only monitored periodically. When the association is investigated, the lack of significant findings may be a product of the additional complexity of controlling accurately for the impact from temperature. Ozone is clearly found more often at higher

Table 1. Study Population Characteristics of OHCA Events in Houston, Texas, From 2004 to 2011

| Total | 11677 |
| Preexisting condition | 9196 (79) |
| Age | |
| Mean | 64 SD, 16.81 |
| 18 to <35 | 576 (5) |
| 35–64 | 5153 (44) |
| 65–74 | 2444 (19) |
| 75+ | 3704 (32) |
| Sex | |
| Female | 4776 (41) |
| Male | 6901 (59) |
| Race | |
| White | 4065 (35) |
| Black | 5338 (46) |
| Hispanic | 1875 (16) |
| Other | 399 (3) |
| Season | |
| Warm (April to October) | 6411 (55) |
| Cold (November to March) | 5266 (45) |

Values are n (%). OHCA indicates out-of-hospital cardiac arrest; and SD, standard deviation.

Table 2. Description of data

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of Monitors</th>
<th>% of Missing Data</th>
<th>Mean (SD)</th>
<th>5%</th>
<th>25%</th>
<th>50%</th>
<th>75%</th>
<th>95%</th>
<th>IQR</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{2.5}$, μg/m$^3$ hourly</td>
<td>12</td>
<td>0</td>
<td>11.42 (5.69)</td>
<td>3.87</td>
<td>7.34</td>
<td>10.3</td>
<td>14.37</td>
<td>22.8</td>
<td>7.03</td>
</tr>
<tr>
<td>O$_3$, ppb hourly</td>
<td>47</td>
<td>0</td>
<td>25.52 (16.14)</td>
<td>4.3</td>
<td>13.23</td>
<td>22.92</td>
<td>34.61</td>
<td>57.25</td>
<td>21.38</td>
</tr>
<tr>
<td>NO$_2$, ppb hourly</td>
<td>22</td>
<td>1</td>
<td>9.16 (6.76)</td>
<td>2.84</td>
<td>4.96</td>
<td>7.52</td>
<td>11.84</td>
<td>21.18</td>
<td>6.87</td>
</tr>
<tr>
<td>SO$_2$, ppb hourly</td>
<td>13</td>
<td>1</td>
<td>1.97 (3.23)</td>
<td>0.28</td>
<td>0.75</td>
<td>1.45</td>
<td>2.48</td>
<td>5.18</td>
<td>1.73</td>
</tr>
<tr>
<td>CO, ppb hourly</td>
<td>12</td>
<td>1</td>
<td>281.91 (202.45)</td>
<td>121.23</td>
<td>171.94</td>
<td>225.09</td>
<td>315.75</td>
<td>632.76</td>
<td>143.81</td>
</tr>
<tr>
<td>Apparent temperature daily, °F</td>
<td>15</td>
<td>0</td>
<td>73.37 (17.39)</td>
<td>42.58</td>
<td>59.70</td>
<td>75.89</td>
<td>88.89</td>
<td>95.53</td>
<td>21.38</td>
</tr>
<tr>
<td>PM$_{2.5}$, μg/m$^3$ daily</td>
<td>12</td>
<td>0</td>
<td>11.42 (4.73)</td>
<td>5.50</td>
<td>8.18</td>
<td>10.45</td>
<td>13.71</td>
<td>20.96</td>
<td>5.52</td>
</tr>
<tr>
<td>NO$_2$, ppb daily</td>
<td>22</td>
<td>0</td>
<td>9.11 (4.17)</td>
<td>3.51</td>
<td>6.01</td>
<td>8.41</td>
<td>11.66</td>
<td>16.87</td>
<td>5.65</td>
</tr>
<tr>
<td>SO$_2$, ppb daily</td>
<td>13</td>
<td>0</td>
<td>1.96 (2.38)</td>
<td>0.44</td>
<td>0.97</td>
<td>1.66</td>
<td>2.55</td>
<td>4.27</td>
<td>1.57</td>
</tr>
<tr>
<td>CO, ppb daily</td>
<td>12</td>
<td>0</td>
<td>279.90 (130.90)</td>
<td>139.89</td>
<td>194.69</td>
<td>249.89</td>
<td>332.36</td>
<td>526.16</td>
<td>137.67</td>
</tr>
</tbody>
</table>

CO indicates carbon monoxide; IQR, interquartile range; PM$_{2.5}$, fine particulate matter with an aerodynamic diameter <2.5 μm; NO$_2$, nitrogen dioxide; O$_3$, ozone; SD, standard deviation; and SO$_2$, sulfur dioxide.
temperatures, and an increased risk of OHCA is closely tied to the combined effect. Finally, our results indicate that the association may be more readily found at the hourly level over the daily, with the daily level the more frequently studied time frame.

Examining 3 recent large studies in comparison with our findings, we find differences in 2 of the studies\(^3\),\(^7\) regarding the number of cases, the number of monitors, the specific health end point considered, and the magnitude and variation in pollution levels studied.\(^3\),\(^4\) In the third study, Silverman et al\(^10\) of New York City (\(n=8216\)) found an increased risk (1.045; 95% CI, 0.991–1.1) for a daily average increase of 20 ppb. Our study design is most similar to Silverman et al\(^10\); both studies have a large number of cases extracted from an EMS 911 database, limited exposure concentration uncertainty, and similar ozone interquartile range. The results found in New York City and Houston are consistent with findings from an important case-crossover study with a more encompassing health end point. Stafoggia et al\(^30\) examined susceptibility factors to ozone mortality. Of interest to our objective is their examination of ozone-related mortality in those with preexisting cardiovascular conditions. The researchers estimated an increase risk (1.093; 95% CI, 1.044–1.145) in mortality for a 20-ppb increase in the daily 8-hour ozone running maximum average.

Given the comparability between the study of Houston and New York City and the corroborating study by Stafoggia et al\(^30\), the current results of the comparable studies support the likelihood that there is an increased risk of OHCA with exposure to ozone.

Limitations

A potential limitation of this study is selection bias from the exclusion of cases in which chest compressions were not initiated because the adults were considered dead on arrival. Resuscitation was withheld if the individual was dead on arrival as defined by decapitation, rigor mortis, dependent lividity, decomposition or incineration or obvious mortal wounds, absence of any signs of life (pulse, respirations, or any spontaneous movement) on EMS arrival associated with a penetrating head injury (gunshot wound, stab, etc), or penetrating extremity injury with obvious exsanguination, absence of any signs of life (pulse, respirations, or any spontaneous movement) for >5 minutes associated with a penetrating injury to the chest or abdomen and a >10-minute transport time to a trauma center, or the absence of any signs of life (pulse, respirations, or any spontaneous movement) associated with blunt trauma. However, the large size of this study minimizes risks from selection bias.
Another limitation of the study is the absence of stratification by preexisting conditions and personal risk factors owing to the lack of this information. Finally, the exposure concentrations in the study are limited to the use of the average pollutant concentration across the city over the use of more local pollutant concentrations. This is especially true when the study area is large and the pollutant varies spatially. We chose to use the average concentration rather than potentially misclassifying the associated reference concentrations if the individual experienced the OHCA in a location not representative of his usual exposure. This limitation is inherent in the case-crossover study design.

Future Research

Although this study identifies an association between PM$_{2.5}$ and ozone air pollution and OHCA, future research to better define the exposure time period associated with triggering an OHCA is needed. Epidemiological studies have found the time to trigger a cardiac event from exposure to PM$_{2.5}$ or ozone ranges from the day or previous day of onset to hours before onset.\textsuperscript{4,7,9,10} Part of this inconsistent range of time to trigger is due to exposure time misclassification. This could be better handled by addressing the uncertainty in combining the disparate data sets such as OHCA recorded at the minute and continuous across space and air pollution data recorded hourly at fixed locations.\textsuperscript{37}

Table 4. Percentage Change in Risk of OHCA for an Interquartile Increase in Air Pollutants

<table>
<thead>
<tr>
<th>Lag</th>
<th>PM$_{2.5}$ IQR 6 µg/m$^3$ % (95% CI)</th>
<th>O$_3$ IQR 20 ppb % (95% CI)</th>
<th>NO$_2$ IQR 6 ppb % (95% CI)</th>
<th>SO$_2$ IQR 2 ppb % (95% CI)</th>
<th>CO IQR 141 ppb % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily lag</td>
<td>0</td>
<td>2.7 (−0.3 to 5.8)</td>
<td>3.8 (0.4 to 7.2)</td>
<td>0.9 (−3.0 to 5.0)</td>
<td>−0.2 (−2.1 to 1.7)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>3.5 (0.5 to 6.6)</td>
<td>1.8 (−1.4 to 5.2)</td>
<td>−0.7 (−4.4 to 3.0)</td>
<td>−1.2 (−3.2 to 0.8)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>3.7 (0.7 to 6.8)</td>
<td>2.7 (−0.6 to 6.1)</td>
<td>−0.4 (−4.1 to 3.4)</td>
<td>−0.7 (−2.9 to 1.5)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>2.1 (−0.9 to 5.1)</td>
<td>−0.6 (−3.8 to 2.7)</td>
<td>0.9 (−2.8 to 4.7)</td>
<td>−1.3 (−3.3 to 0.7)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.2 (−2.7 to 3.2)</td>
<td>−1.2 (−4.3 to 2.1)</td>
<td>0.3 (−3.4 to 4.1)</td>
<td>−0.9 (−2.6 to 0.8)</td>
</tr>
<tr>
<td>0–1</td>
<td>3.9 (0.5 to 7.4)</td>
<td>3.6 (0.0 to 7.4)</td>
<td>−0.1 (−4.3 to 4.3)</td>
<td>−0.9 (−3.0 to 1.3)</td>
<td>0.9 (−2.1 to 4.0)</td>
</tr>
<tr>
<td>1–2</td>
<td>4.6 (1.2 to 8.2)</td>
<td>3.0 (−0.6 to 6.8)</td>
<td>−0.8 (−4.9 to 3.5)</td>
<td>−1.3 (−3.7 to 1.1)</td>
<td>0.4 (−2.5 to 3.4)</td>
</tr>
</tbody>
</table>

Hourly lag

<table>
<thead>
<tr>
<th>Lag</th>
<th>PM$_{2.5}$ IQR 6 µg/m$^3$ % (95% CI)</th>
<th>O$_3$ IQR 20 ppb % (95% CI)</th>
<th>NO$_2$ IQR 6 ppb % (95% CI)</th>
<th>SO$_2$ IQR 2 ppb % (95% CI)</th>
<th>CO IQR 141 ppb % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.9 (−1.4 to 3.4)</td>
<td>3.7 (−0.1 to 7.7)</td>
<td>−0.1 (−0.6 to 0.4)</td>
<td>0.4 (−0.2 to 1.0)</td>
<td>0.0 (0.0 to 0.0)</td>
</tr>
<tr>
<td>1</td>
<td>1.1 (−1.3 to 3.5)</td>
<td>4.2 (0.4 to 8.2)</td>
<td>0.0 (−0.5 to 0.5)</td>
<td>0.0 (−0.7 to 0.8)</td>
<td>0.0 (0.0 to 0.0)</td>
</tr>
<tr>
<td>2</td>
<td>1.1 (−1.2 to 3.5)</td>
<td>4.6 (0.8 to 8.7)</td>
<td>0.0 (−0.5 to 0.5)</td>
<td>0.2 (−0.5 to 0.9)</td>
<td>0.0 (0.0 to 0.0)</td>
</tr>
<tr>
<td>3</td>
<td>0.3 (−2.0 to 2.7)</td>
<td>4.0 (0.2 to 8.0)</td>
<td>0.1 (−0.3 to 0.6)</td>
<td>0.2 (−0.6 to 0.9)</td>
<td>0.0 (0.0 to 0.0)</td>
</tr>
<tr>
<td>4</td>
<td>0.9 (−1.5 to 3.3)</td>
<td>3.4 (−0.5 to 7.4)</td>
<td>0.2 (−0.3 to 0.7)</td>
<td>0.0 (−0.8 to 0.7)</td>
<td>0.0 (0.0 to 0.0)</td>
</tr>
</tbody>
</table>

O$_3$ was based on an 8-hour maximum. Statistics reflect the adjustment for apparent temperature. CI indicates confidence interval; CO, carbon monoxide; IQR, interquartile range; NO$_2$, nitrogen dioxide; O$_3$, ozone; OHCA, out-of-hospital cardiac arrest; PM$_{2.5}$, fine particulate matter with an aerodynamic diameter <2.5 µm; and SO$_2$, sulfur dioxide.

Figure 2. Forest plot of Houston relative risk of OHCA associated with 20-ppb increase in ozone or 6 µg/m$^3$ increase in PM$_{2.5}$. CI indicates confidence interval; EMS, emergency medical service; OHCA indicates out-of-hospital cardiac arrest; PM$_{2.5}$, fine particulate matter with an aerodynamic diameter <2.5 µm; and RR, relative risk.
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Disclosures
None.

References


**CLINICAL PERSPECTIVE**

The implications of this work are improved health policy and action with the objective of reducing the number of annual out-of-hospital cardiac arrest currently at ≈300,000 in the nation and 1460 in Houston, Texas. We find consistent evidence of an association between out-of-hospital cardiac arrest and exposure to ozone in Houston at short time scales up to 3 hours in duration and also at the daily level on the day of the event. For exposure to fine particulate matter an association is found for 2 days before the event. Other pollutants were not found to impact the occurrence of out-of-hospital cardiac arrest. Our findings add to the significant literature relating out-of-hospital cardiac arrest and fine particulates. Furthermore, we add to the small but growing scientific conversation relating out-of-hospital cardiac arrest and ozone. Finally, we bring the most comprehensive data set to date to this literature, in terms of the duration of the study, the number of pollution monitors included, and the number of out-of-hospital cardiac arrest events studied.
A Case-Crossover Analysis of Out-of-Hospital Cardiac Arrest and Air Pollution
Katherine B. Ensor, Loren H. Raun and David Persse

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