OZONE AND PARTICULATE AIR POLLUTION TRIGGER DIFFERENT TYPES OF OUT-OF-HOSPITAL CARDIAC ARREST

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Background and Aims: Out-of-hospital cardiac arrest (OHCA) has been previously associated with exposure to air pollution. But it is not clear if it acts by precipitating myocardial infarction or by other mechanisms. We studied the effects of particulate and gaseous air pollution on the risk of OHCA due to myocardial infarction (MI) vs. the risk of OHCA due to other cardiac causes, in Helsinki 1998-2006.

Methods: Helsinki Emergency Medical Services provided OHCA data. Cardiac cause was identified from prior symptoms, ECGs, hospital and autopsy reports. Pollutant data was obtained from central ambient monitors. A case-crossover analysis determined hazard ratios (HRs) for hourly lagged exposures (Lag 0 - 3) and daily lagged exposures (Lag 0d - 3d).

Results: Among OHCAs due to MI (OHCA_MI), interquartile increases in pollutant exposure were associated with elevated HRs for PM2.5 (Lag 0, 1.16 95% CI:1.04, 1.28; Lag 1, 1.16 CI: 1.04, 1.28; Lag 2, 1.14 CI:1.03, 1.26; Lag 0d 1.21 CI: 1.07, 1.37) and NO (Lag 0d, 1.06 CI: 1.01, 1.12). Among OHCAs not identified as due to MI (OHCA_Other), elevated HRs were associated with ozone exposure (Lag 0d, 1.30 CI: 1.03, 1.64; Lag 1d, 1.41 CI: 1.11, 1.79; Lag 2d, 1.37 CI: 1.08, 1.73). HRs for OHCA_MI and ozone were nonsignificantly less than one; HRs for OHCA_Other and PM2.5 were close to one and nonsignificant. HRs for OHCA_MI associated with PM2.5 compared to HRs for OHCA_Other and ozone showed opposite trends with sex and season.

Conclusions: The results suggest that air pollution triggers OHCA via two distinct modes: one associated with particulates and leading to MI and one associated with ozone involving etiologies other than MI, e.g. arrhythmias.