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Atmospheric Interactions and Cardiac Arrhythmias

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Although plausible pathophysiological mechanisms link air pollution to arrhythmogenesis, among them altered autonomic tone, repolarization abnormalities, oxidative stress, myocardial ischemia, and increased intracardiac pressure (Link and Dockery 2010), definitive conclusions have not been reached as yet. Langrish et al. (2014) analyzed 13 double-blind randomized crossover studies and found no significant risk of arrhythmia attributable to acute controlled exposure to air pollutants. Three issues related to meteorological factors probably either confound or modify the short-term association between air pollution and cardiac arrhythmia.

First, several meteorological elements, including air temperature, atmospheric pressure, relative air moisture, and wind speed and direction, also are implicated in triggering ventricular (Čulić et al. 2004, 2005) and supraventricular (Čulić et al. 2012, 2013) arrhythmias independent of physical and emotional stress. In the short term, those meteorological factors may facilitate arrhythmias in susceptible patients by increasing circulatory load and thromboinflammatory processes (Čulić 2014).

Second, these same meteorological elements substantially influence concentrations of sulfur dioxide, carbon monoxide, nitrogen dioxide, ozone, and suspended particulate matter (Bertaccini et al. 2012; Ilten and Selici 2008; Ito et al. 2007). In addition, the greatest ozone production and pollution results from stable, dry, hot weather with high atmospheric pressure and low wind (Vanos et al. 2014).

Air pollution may increase human vulnerability to the effects of temperature, and temperature extremes, in turn, influence population vulnerability to air pollution (Burkart et al. 2013; Ren et al. 2006). Vanos et al. (2014) reported that cardiovascular and respiratory mortality due to short-term exposure to gaseous air pollutants was significantly modified by weather types and season. Alberdi et al. (1998) reported that both relative air moisture and air temperature are strongly related to daily mortality even after controlling for air pollution and influenza. Keatinge and Donaldson (2001) suggested that prolonged cold weather with less wind and rain may produce false associations between mortality and certain air pollutants.

Finally, strong mutual interrelations exist among the above-mentioned meteorological elements. Alberdi et al. (1998) pointed out the strong inverse association they observed between relative air moisture and air temperature as an important problem for regression analysis.

Langrish et al. (2014) caution against definitive acceptance of air pollution as an independent trigger of cardiac arrhythmias. However, the studies included in their analysis had no data on meteorological factors. It is likely that interactive effects among air pollutants and meteorological elements bias each other's association with arrhythmias and other acute cardiac events. Therefore, further research of the health effects of atmospheric factors should continue in order to identify potentially harmful influences for the population as whole as well as for its vulnerable subgroups.

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Atmospheric Interactions and Cardiac Arrhythmias: Langrish et al. Respond

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We agree with Čulić's argument (2015) that environmental influences on human health are complex and likely multifactorial. Exposure to indoor and ambient urban air pollution has been estimated to contribute to 7 million premature deaths each year, predominantly from cardiovascular and respiratory conditions (Lim et al. 2012). Associations between exposure and cardiovascular mortality and morbidity have been demonstrated for nitrogen dioxide, sulfur dioxide, ozone, carbon monoxide, and particulate matter (Brook et al. 2010), although the associations are strongest for fine and ultrafine particulate matter (Hoek et al. 2013).

Air pollution is extremely complex and consists not of single components in isolation but rather combinations of components. These constituent components interact with one another in the environment, which may alter potential toxicity and the subsequent health impacts. Meteorological factors such as wind speed and direction, humidity, atmospheric pressure, and temperature play an important part both in determining an individual's exposure to ambient air pollution and in affecting the concentrations, chemical composition, and clearance of elements of the air pollution mixture. This is particularly true for the secondary pollutant ozone, which has a strong relationship with season and temperature (Langrish et al. 2010b). We agree that, in assessing the impact of ambient air pollution on public health, it is important to assess the air pollution mixture as a whole.

In our studies we used a controlled exposure facility to assess, in a robust and well-validated fashion (Langrish et al. 2010a), the contribution to potential arrhythmogenesis of individual air pollutants—diesel exhaust, wood smoke, ozone, and nitrogen dioxide—as well as ambient air pollution in Beijing,

China (Langrish et al. 2014). Exposure to neither air pollutants in isolation nor ambient Beijing air pollution was associated with cardiac dysrhythmia in either patients with coronary heart disease or healthy volunteers. As such, our studies do not address the influence of meteorological conditions on an individual's risk of cardiac arrhythmia; indeed, the meteorological conditions in Beijing were fairly constant throughout our studies (Langrish et al. 2009, 2012).

There is emerging evidence that cardiovascular morbidity and mortality is associated with meteorological and environmental conditions, and we agree with Čulić's statement that further research on the health impacts of atmospheric factors is important both for public health and for better understanding the interaction between urban air pollution and external influences.

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