Pollution

Air pollution and arrhythmia: the case is not over

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Commentary on the paper by Rich *et al* (see page 591)

In this issue, Rich *et al* present findings that strengthen the evidence for a causal role of air pollution in triggering arrhythmias among patients with implanted cardioverter defibrillators.¹ The use of objective health and exposure data in conjunction with the chosen case-crossover approach underscores the power of “quasi experimental” epidemiological research in this field. Based on only 139 ventricular arrhythmias (VA) recorded on implantable cardioverter defibrillators among 56 patients, the investigators found statistically significant associations between markers of acute exposure to air pollution and arrhythmias. Associations with the 24 hour mean concentrations prior to the event were stronger than with both shorter (6 and 12 hour) and longer (48 hour) time windows.

The mechanisms linking ambient pollution with arrhythmias remain to be elucidated. However, the plausibility and evidence for effects of pollutants on autonomic function are steadily increasing.² In a series of experiments, exposure of dogs to concentrated ambient particles led to cardiac and respiratory changes mediated via both the sympathetic nervous system and the vagus nerve.³ A recent controlled experiment in humans observed reduced cardiac vagal control after exposure to 200 ppb SO₂.⁴ These changes in autonomic function reflect increased susceptibility to cardiac arrhythmias.

The new study leaves open questions that reflect both limitations and challenges of the case-crossover approach.

Case-crossover analyses (if based on a small number of events) come with the same inherent assumption as panel studies, namely that exposure of a specific individual—rather than the total population average—can be characterised for the time window prior to the event, as well as for the chosen control day(s). Fixed site monitors do not necessarily accomplish this on the individual level. Moreover, the validity of this assumption grossly varies for different pollutants and time windows.^{5–6} This leaves us with the problem that comparisons of estimates across pollutants or time windows (see tables 2 and 3 in Rich *et al*) are of inherent interest but of questionable validity; for example, when measured at a single site, sulphur certainly reflects the exposure of a given subject to urban background pollution much better than more spatially heterogeneous pollutants such as NO₂ or elemental carbon, while in the case of ozone, the monitor station may entirely fail to characterise exposure.⁷ Thus associations between pollutants (or different time windows of exposure) and arrhythmias may differ not only for biological reasons but due to different degrees of misclassification.

Correlations between outdoor and personal concentrations give some insight into this differential misclassification. Janssen *et al* reported high outdoor-to-personal correlations (≥ 0.90) for 24 hour mean sulphur mass concentration, but much more subject specific and lower correlations for black smoke and PM_{2.5} mass.⁵ In the EXPOLIS

study, the correlation of personal and outdoor 48 hour mean CO concentrations ranged between 0.33 and 0.77 across five European cities, whereas correlations for lead (as a marker of traffic related PM) and potassium (as a marker for crustal PM) were 0.53 and 0.21 respectively.⁸ Therefore, extensive pollution speciation at a single “super site” monitor will not provide better insight in the health relevance of pollutants (or sources). Personal or close-to-personal exposure measurements are needed for that purpose. Rich *et al* also discuss how effects depend on wind direction—another promising approach to understand the health relevance of pollution from specific sources or air sheds.

Comparisons of effects across pollutants (table 2 in Rich *et al*) need to rely on comparable scales. This is usually achieved by scaling pollutants to comparable contrasts, for example to the highest versus lowest or to the interquartile range (IQR). The comparable exposure scale in the case-crossover design is the difference of pollutant concentrations between event and control day(s) rather than the ambient concentrations.⁹ The two distributions are not necessarily the same, and Rich and colleagues’ paper is the first air pollution case-crossover study to reveal both the ambient and the design relevant distribution of differences. Comparisons (across pollutants) are valid on the latter scale only. As seen in table 1 of Rich *et al*, IQRs were similar on both scales except for O₃ where the variability of the relevant term was far smaller than across the ambient concentrations. Thus, the interpretation of their results in table 2—based on ambient scale—would not change much.

Forastiere *et al* conducted a case-crossover analysis of air pollution effects on cardiac arrest.¹⁰ The published results were also based on IQR of the *ambient concentrations*. Table 1 presents the IQR of the difference between case and control exposure. In the original table, effects (as odds ratios (OR) per IQR) appear clearly stronger for particle number count (PNC). However, if assessed on the scale that allows direct comparisons, the effect sizes of PNC, PM₁₀, and CO are similar. The example demonstrates that the use of the relevant exposure term is crucial to appropriately investigate, interpret, and compare effects.⁹

In summary, Rich *et al* further support the notion that air pollutants trigger arrhythmias. The relevance of different pollutants and time windows of exposure remain to be elucidated, and case-crossover studies may play an important role in this. The use of personal exposure assessment and of appropriate exposure terms will be critical for valid

Table 1 Associations (OR) of ambient pollutants with cardiac arrest shown for the interquartile range (IQR) of ambient concentrations (non-comparable scale) and of the concentration differences between event and control days (comparable scale in case-crossover studies)

Pollutant	Non-comparable scale		Comparable scale	
	IQR	OR	IQR	OR
PM ₁₀ (µg/m ³)	29.7	1.048	29.9	1.048
NO ₂ (µg/m ³)	18.5	1.023	18.3	1.023
CO (mg/m ³)	1.2	1.065	0.8	1.045
PNC (number/1000 cm ³)	27.8	1.076	18.2	1.049

From Forastiere *et al*.¹⁰

comparisons across pollutants, constituents, and time windows of exposure.

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Organisational justice

Injustice at work and health: causation or correlation?

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Commentary on the paper by Ferrie *et al*
(*Occup Environ Med*, July 2006)*

Organisational justice has emerged in recent years as a determinant of workers' health, joining the growing list of other psychosocial aspects of the work environment, including job strain, effort-reward imbalance, and job insecurity. In a series of studies carried out mainly among Finnish workers, perceptions of organisational justice have been linked to poor self-rated health, minor psychiatric disorders, and sickness absences.^{1,2} In the July issue of this journal, Ferrie and colleagues provide an independent test of low organisational justice as a predictor of psychiatric morbidity within a well established cohort, the British Whitehall II study.³ What do these studies add to the literature on the psychosocial work environment, and do we have sufficient evidence to implicate organisational justice as a causal influence on workers' health?

Initial studies in this area were cross-sectional and involved self-reported

outcomes, so that reverse causation and common method bias could not be ruled out. In a longitudinal follow up of Finnish hospital workers, Kivimäki and colleagues² checked for the possibility of reverse causation by comparing the changes in perceptions of justice between initially healthy employees versus those with baseline health problems. Although the interaction term between time and baseline health was reported to be statistically non-significant, it was also evident that workers with health problems reported lower perceptions of justice compared to healthy co-workers at both the baseline and at the follow up.² The new study by Ferrie *et al* examined the effects of change in relational justice over time in relation to the onset of psychiatric morbidity. A favourable change in perceptions of justice was associated with a reduction in psychiatric morbidity, whereas an adverse change increased the risk. These results bolster the case that reverse causation is not the major explanation for the observed associations. Nevertheless, if health declines and changes in perceptions of justice are

contemporaneous, it is difficult to completely rule out reverse causation, even with longitudinal change analysis. As Ferrie *et al* acknowledge, controlled experiments in the work setting would help, although it is not clear exactly what "treatment" should be designed to increase perceptions of justice.

Common method variance is a cause for concern when both the exposure and outcome variables are self-reported. Future studies of organisational justice and health would be strengthened by incorporating biomarkers and other endpoints that are not perceived or self-reported. Alternatively, common method variance could be addressed by aggregating individual responses to questions about organisational justice up to the work group or firm level. It makes theoretical sense to conceptualise and measure justice as an organisation-level characteristic, as opposed to individual-level perceptions. Following this logic, investigators should focus on the contextual influence of organisational justice on workers' health within a multi-level analytical framework, i.e. "exposure" to aggregated perceptions of justice assigned to individual workers nested within different workplaces. (A parallel argument could be made about investigating the health effects of other work environment characteristics, such as job strain and effort-reward imbalance.)

A further noteworthy finding from existing studies of organisational justice and health is that the associations with endpoints tend to be attenuated (in several instances to statistical non-significance) after controlling for other psychosocial aspects of the work environment, including decision authority, effort-reward imbalance, and social

* Ferrie JE, Head J, Shipley MJ, *et al.* Injustice at work and incidence of psychiatric morbidity: the Whitehall II study. *Occup Environ Med* 2006;**63**:443–50.