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Abstract

Background: Epidemiological studies have demonstrated that air pollution exposure is associated with increases in cardiovascular morbidity and mortality. Exposure to air pollutants can influence cardiac autonomic tone and reduce heart rate variability, and may increase the risk of cardiac arrhythmias, particularly in susceptible patient groups.

Objectives: To investigate the incidence of cardiac arrhythmias during and after controlled exposure to air pollutants in healthy volunteers and patients with coronary heart disease.

Methods: We analysed data from 13 double-blind randomized crossover studies including 282 subjects (140 healthy volunteers and 142 patients with stable coronary heart disease) from whom continuous electrocardiograms were available. The incidence of cardiac arrhythmias was recorded for each exposure and study population.

Results: There were no increases in any cardiac arrhythmia during or following exposure to dilute diesel exhaust, wood smoke, ozone, concentrated ambient particles, engineered carbon nanoparticles or high ambient levels of air pollution in either healthy volunteers or patients with coronary heart disease.

Conclusions: Acute controlled exposure to air pollutants did not increase the short-term risk of arrhythmia in participants. Research employing these techniques remains crucial in identifying the important pathophysiological pathways involved in the adverse effects of air pollution, and is vital to inform environmental and public health policy decisions.

Introduction

Exposure to air pollution is a major public health concern and is associated with morbidity and mortality from cardiorespiratory diseases (Brook et al. 2010). Indeed, on a population level, exposure to combustion-derived particulate air pollution from traffic is recognized as a major trigger for myocardial infarction (Nawrot et al. 2011). With growing concern over the effects of exposure to air pollutants on the general public and susceptible patient populations, there is an increasing interest in defining the risks and underlying vascular and inflammatory mechanisms that may explain these observed associations.

The cardiovascular effects of air pollution are complex and include effects on vascular endothelial function, thrombosis, platelet function and atherogenesis, as well as changes in blood pressure and cardiac autonomic control (Langrish et al. 2012a). Indeed changes in autonomic control of the heart, measured by heart rate variability (HRV), have been widely studied in the air pollution literature and a recent meta-analysis of 18,667 subjects enrolled in 29 studies have demonstrated an inverse relationship between measures of HRV and exposure to particulate air pollution (Pieters et al. 2012). Reduced HRV represents a withdrawal of cardiac vagal tone or an increase in sympathetic tone, and is a predictor of poor prognosis in patients recovering from myocardial infarction and patients with cardiac failure (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996), and may increase the risk of cardiac arrhythmias in these at risk patients (Odemuyiwa et al. 1991). Recent evidence has linked activity of the autonomic nervous system and atrial electrical properties with the triggering of atrial arrhythmias such as atrial fibrillation and flutter (Arora 2012; Lo et al. 2011; Park et al. 2012). There is some limited epidemiological evidence linking exposure to air pollutants to both ventricular and supraventricular arrhythmias (Brook et al. 2010), although these associations are not consistent (Gold and Mittleman 2013).

In this study we explored our database of continuous electrocardiographic recordings during controlled exposure to a range of air pollutants to determine whether there is evidence of an increase in the short-term risk of arrhythmia.

Methods

Data were extracted from 13 consecutive randomized double-blind crossover studies including healthy volunteers and patients with coronary heart disease from 2004 to 2013 (Table 1). All trials were reviewed and approved by the appropriate local ethics review boards in Edinburgh, UK, Umeå, Sweden or Beijing, China. All subjects gave their written informed consent according to the Declaration of Helsinki. All healthy volunteers had a normal 12-lead electrocardiogram and cardiovascular response to exercise determined at screening. Patients with coronary heart disease were excluded if they had a history of arrhythmia, severe coronary disease without revascularisation, significant valvular heart disease or left ventricular systolic dysfunction, conduction abnormality on a resting 12-lead electrocardiogram, uncontrolled hypertension or an acute coronary syndrome within the previous 3 months. Subjects were exposed to a variety of air pollutants in controlled exposure studies or in ambient settings (with the use of a highly efficient facemask to provide a control (Langrish et al. 2009)). Detailed monitoring of personal exposure was performed and continuous electrocardiograms recorded to allow for the assessment of cardiac arrhythmia (Barath et al. 2010; Cruts et al. 2008; Langrish et al. 2012b; Langrish et al. 2009; Mills et al. 2007; Mills et al. 2005; Mills et al. 2011b; Mills et al. 2008).

In controlled exposure studies, subjects were exposed using a randomised double-blind controlled crossover design to either filtered air or to the experimental pollutant during intermittent exercise. The exposure time varied across these studies, and is documented below. Each study visit was separated from the next by at least 7 days.

Controlled and Ambient Exposure Generation

Diesel exhaust exposures

Controlled exposures to dilute diesel exhaust were performed in purpose-built exposure chambers in Umeå, Sweden (Mills et al. 2005) and in Edinburgh, UK through a collaboration with the National Institute for Public Health and the Environment, The Netherlands as described previously (Mills et al. 2011b). In Sweden, diesel exhaust was produced from a Volvo diesel engine (TD45, 4.5 L, 4 cylinders) under idling (Mills et al. 2005; Mills et al. 2011b) or city-cycle conditions (Barath et al. 2010). More than 90% of the exhaust was shunted away and the remainder mixed with filtered air and fed into a purpose-built whole-body exposure chamber at steady state concentration. Air was sampled in the breathing zone of the subject and analysed continuously for particle mass concentration, particle number concentration, oxides of nitrogen, carbon monoxide, and total hydrocarbons (Mills et al. 2005). In Edinburgh, diesel exhaust was produced from a diesel electricity generator (Deutz, 4 cylinder, 2.2L, 500 rpm), and was diluted as above before being fed into a modified body-box exposure chamber (Mills et al. 2011b). The exposures were standardized with a target particulate matter mass concentration of 300 $\mu\text{g}/\text{m}^3$. Subjects were exposed to the diesel exhaust and filtered air for 1 hour during intermittent exercise on a bicycle to generate an average minute ventilation of 20 L/min/m² body surface area.

Wood smoke exposures

Wood smoke was generated using a common Nordic wood stove (chimney stove) in a controlled incomplete combustion firing procedure (Unosson et al. 2013). Birch wood logs with a moisture content of 16-18% were inserted every 5-15 min to maintain a high burn rate with repeated air-starved conditions. The wood smoke was diluted with filtered air (highly efficient particle [HEPA] filter and activated carbon filter) in three steps and continuously fed into a controlled

environment exposure chamber (15.3 m³) to achieve a steady state concentration. The atmosphere in the chamber was monitored for gaseous pollutants using continuous measurement of nitrogen oxides (NO_x) and carbon monoxide (CO). PM₁ (particulate matter with an aerodynamic diameter of <1 µm) mass concentration was measured on-line using a tapered element oscillating microbalance (TEOM 1400 by Thermo Scientific) equipped with a PM₁ pre-cyclone. Integrated with the TEOM, a filter (Teflon) sampling line was used to determine the particle mass concentration gravimetrically. The exposures were standardized with a target particulate matter (PM₁) mass concentration of 300 µg/m³ for 3 hours (n=14) or 1,000 µg/m³ for 1 hour (n=15). As before, subjects were exposed to wood smoke during intermittent exercise to generate an average minute ventilation of 20 L/min/m² body surface area.

Ozone exposures

Ozone was generated using an ozone generator (500 MM, Fischer Labor und Verfahrens-Technik-Gmtt, Germany) and the ozone concentration was measured continuously in the breathing zone of the subject using a photometric ozone analyser (Dasibi Model 1108, Dasibi Environmental Corp., California, USA). During the exposures, ambient air was continuously drawn through the chamber at a ventilation rate of 30 m³/hour (Blomberg et al. 1999). Temperature and relative humidity were maintained at 20°C and 50% respectively. The exposures were standardized to an ozone concentration of 300 ppb for 75 min. Again subjects performed intermittent exercise during the exposure to maintain an average minute ventilation of 20 L/min/m² body surface area.

Concentrated ambient particle exposures

A Versatile Aerosol Concentration Enrichment System (VACES) concentrator within a mobile ambient particle concentrator exposure laboratory sited outside the Royal Infirmary of Edinburgh, Edinburgh, UK as used to deliver concentrated ambient particle (CAPs) exposures as

described previously (Mills et al. 2008). Incoming ambient air (500 L/min) was saturated with water vapour to increase the size of ultrafine and fine particles before being passed through five parallel virtual impactors, each operating at 100 L/min. This increase in size and therefore mass ensured the particles had sufficient momentum to pass through the impactors, exiting in the minor flow (5 L/min) in which the particle concentration is enriched by a factor of 10-20 fold. The outward minor flow from the five impactors (25 L/min) was desaturated by silica gel dryers to restore the particles to their original size, and diluted with filtered air prior to delivery into the human exposure chamber (50 L/min). Air was sampled in the breathing zone of the subject and analysed continuously for temperature, humidity, particle mass concentration, particle number concentration, oxides of nitrogen, carbon monoxide, sulphur dioxide and ozone (Mills et al. 2008). Subjects were exposed to CAPs at a target concentration of $200 \mu\text{g}/\text{m}^3$ for 2 hours during intermittent exercise to generate an average minute ventilation of $20 \text{ L}/\text{min}/\text{m}^2$ body surface area.

Exposure to engineered carbon nanoparticles

An aerosol of carbon nanoparticles was generated from graphite electrodes using an electric spark discharge generator (Palas CFG1000, Palas GmbH, Karlsruhe, Germany) in an atmosphere of pure argon. The output of the generator was mixed with filtered air, passed through an impactor with a cut-off of $0.1 \mu\text{m}$, and fed into a whole-body exposure chamber (Mills et al. 2011b). Subjects were exposed to the carbon particles for 2 hours with a target exposure of 4×10^6 particles/ cm^3 during intermittent exercise to generate an average minute ventilation of $20 \text{ L}/\text{min}/\text{m}^2$ body surface area.

Ambient exposures and personal monitoring

Subjects attended on two occasions in two randomized open-label controlled crossover studies, and were randomized to wear no mask or a highly efficient occupational facemask (Dust Respirator 8812, 3M, St Paul, USA) as described previously (Langrish et al. 2012b; Langrish et

al. 2009). The facemask visit was deemed the “control” visit for these analyses. Subjects were asked to walk for 2 hours in a city centre location in Beijing, China between 8 and 10 am. Exposure to ambient air pollutants was measured using portable monitoring equipment mounted in a backpack. Particle mass concentration (PM_{2.5}), number concentration, carbon monoxide, sulphur dioxide, temperature and humidity were recorded (Langrish et al. 2009). Physical activity was recorded using a global positioning system monitor within the backpack to ensure exercise performed on each visit was equivalent.

Continuous electrocardiograms and arrhythmia analysis

Continuous electrocardiograms were recorded from all subjects (Model 90217, Spacelabs Healthcare, UK) during the exposure and for the subsequent 24 hours, except one diesel exhaust study where the recordings were for 3 hours post exposure (n=10) and one wood smoke study where the recordings were for 8 hours post exposure (n=14). Data were analysed using the Pathfinder Digital 700 Series Analysis System (Delmar Reynolds, Spacelabs Healthcare, UK). Arrhythmias were identified using an automated algorithm and confirmed manually by trained operators.

Data analysis and statistics

The number of subjects with observed arrhythmia was determined during the pollutant and control exposure periods and compared using the Chi-squared test, and odds ratios were calculated. The numbers of arrhythmias per subject during pollutant and control exposure periods were compared using the Wilcoxon Matched Pairs Signed Rank Test. All analyses were performed using GraphPad Prism (Version 5 for Macintosh; GraphPad Software, San Diego, USA). Statistical significance was taken as a two-sided P value of <0.05. Data are expressed as median (interquartile range) or mean ± standard deviation as appropriate.

Results

We identified 282 subjects (140 healthy volunteers and 142 patients with coronary heart disease; Table 1) who had been exposed to dilute diesel exhaust (n=117) (Barath et al. 2010; Cruts et al. 2008; Mills et al. 2007; Mills et al. 2005; Mills et al. 2011b), woodsmoke (n=29), ozone (n=15), concentrated ambient particles (n=29) (Mills et al. 2008), and engineered carbon nanoparticles (n=14) (Mills et al. 2011b) in controlled exposure studies and ambient air pollution (n=107) in Beijing, China (Langrish et al. 2012b; Langrish et al. 2009) (Table 2). The mean recording time was 22±5 hours, and there were over 12,500 hours of electrocardiographic data in total.

There was no difference between the incidence of arrhythmias or the number of arrhythmias (Table 3; Figure 1) in each subject following any exposure as compared to filtered air (or in the case of the ambient exposures, an exposure in the presence of a highly efficient facemask). Similarly, there was no difference in the incidence or number of arrhythmias in the healthy volunteer and patient subgroups when analysed independently (see Supplemental Material, Tables S1 and S2). One patient with coronary artery disease (73 year-old male with a past medical history of a previous myocardial infarction and hypertension, with a baseline blood pressure of 172/95 mmHg who was taking aspirin, benazepril, bisoprolol, and simvastatin) had an asymptomatic episode of non-sustained atrial fibrillation lasting 15 seconds whilst walking in central Beijing during his exposure visit (without a facemask). There were no other episodes of atrial fibrillation or flutter identified during the >12,500 hours of continuous electrocardiograms.

Discussion

We have compiled the single largest series of studies documenting continuous electrocardiographic monitoring in healthy volunteers and patients with stable coronary heart disease on appropriate medical therapy, who have been exposed to a diverse range of environmental air pollutants in acute controlled exposure studies. In over 12,500 hours of electrocardiographic data, we have identified no evidence to suggest an increased tendency to arrhythmia following brief controlled exposures. These data indicate that there is no significant risk of arrhythmia associated with controlled exposure to a wide range of air pollutants.

Air pollution and risk of arrhythmia

The epidemiological data linking exposure to air pollution and arrhythmia is limited. In a recent study conducted in Taipei, Taiwan, the total admissions to hospital with cardiac arrhythmia over a 4-year period (>16,000 hospital visits) were associated with daily increases in PM air pollution (Chiu et al. 2013), although the investigators provide no information on the type of arrhythmias observed. Among patients with implantable cardiac defibrillators, some studies have demonstrated an increase in ventricular arrhythmias with increasing exposure to particulate air pollutants (Dockery et al. 2005; Ljungman et al. 2008; Peters et al. 2000; Rich et al. 2006). In a recent study of elderly patients with coronary heart disease, in which 20% of subjects had a history of congestive cardiac failure, there was a small increase in the risk of non-sustained ventricular tachycardia measured on ambulatory electrocardiography with increasing exposure to PM air pollution (Bartell et al. 2013), although the same study did not find associations with changes in heart rate variability or supraventricular arrhythmias. The finding of an increased risk of ventricular arrhythmia is however not consistent and others have failed to show similar associations (Anderson et al. 2010; Metzger et al. 2007; Rich et al. 2004; Vedal et al. 2004). In recent long term follow-up from the Normative Aging Study, short term exposure to combustion-

derived PM air pollution (measured as black carbon) was associated with an increased risk of ventricular ectopy (Zanobetti et al. 2013). There is an association between air pollution exposure and the risk of hospitalization due to cardiac dysrhythmia (Colais et al. 2012; Santos et al. 2008; Tsai et al. 2009) and out-of-hospital cardiac arrest (Rosenthal et al. 2013), although this may be confounded by the strong association between exposure and the triggering of myocardial infarction (Nawrot et al. 2011) or decompensation of patients with cardiac failure (Atkinson et al. 2013; Shah et al. 2013).

Whilst air pollution exposure is robustly linked to changes in cardiac autonomic nervous system activity (Pieters et al. 2012), which in turn may alter atrial electrical properties and increase the risk of atrial arrhythmia (Arora 2012; Lo et al. 2011; Park et al. 2012), the association between air pollutant exposure and supraventricular arrhythmia is less robust. Among patients with coronary heart disease and elderly subjects, increasing exposure to particulate matter (PM) air pollution increased the incidence of asymptomatic runs of supraventricular arrhythmias in two observational studies (Berger et al. 2006; Sarnat et al. 2006), although in a recent robust case-crossover study of more than 10,000 admissions to hospital with atrial fibrillation there was no association with PM air pollution (Bunch et al. 2011). A more recent prospective analysis of patients with implantable cardiac defibrillators with established cardiac disease showed an increased risk of atrial fibrillation with acute increases in exposure to PM air pollution (Link et al. 2013). These contrasting findings may reflect the underlying individual susceptibility to arrhythmia of the patients recruited into the trials. Positive associations have generally been in patients with established cardiac disease, most notably cardiac failure who have structural abnormalities of the cardiac muscle and are generally at increased risk of developing cardiac dysrhythmias. Indeed, we have recently demonstrated an increased risk of hospitalization and death with increasing PM air pollution exposure in patients with heart failure (Shah et al. 2013).

We previously reported that among 32 healthy volunteers and 20 patients exposed to dilute diesel exhaust in controlled exposure studies, there were no increases in cardiac arrhythmia or changes in heart rate variability (Mills et al. 2011a), and the findings from this study are similar. Our screening procedures ensured all healthy volunteers were free from cardiac disease and we excluded patients with coronary heart disease who had resting electrocardiographic abnormalities or a history of arrhythmia. As such we have studied a relatively low-risk population. We cannot exclude an effect of exposure to air pollutants in patients with overt cardiac failure who have conditional susceptibility to developing arrhythmias.

In their recent case report, Ghio and colleagues (Ghio et al. 2012) described a 58 year old hypertensive female volunteer with frequent atrial ectopy who developed sustained atrial fibrillation/flutter during exposure to concentrated ambient particles (CAPs). The authors suggested a causal link, however atrial fibrillation (AF) is the most common cardiac arrhythmia in the general population and is associated with increasing age, the presence of hypertension, cardiac dysfunction and may be triggered by atrial ectopic beats originating from within the pulmonary veins (Haissaguerre et al. 1998). We suggest it is more likely that the investigators have simply witnessed an asymptomatic episode of AF in a subject at increased arrhythmic risk due to coexistent hypertension, age and frequent atrial ectopy and the occurrence of AF in the exposure chamber is likely to have been coincidence and simply the play of chance. The short (<0.25 min) single episode of asymptomatic atrial fibrillation in over 750,000 min of electrocardiographic recordings in our studies is similarly likely to be due to the play of chance in a patient at risk of arrhythmia due to poorly controlled hypertension and ischaemic heart disease.

Controlled exposure studies

Air pollution research is challenging due to the ever-changing ambient concentrations and composition of the air pollution mixture. Controlled exposure studies in both animal models and humans have been employed to address fundamental questions necessary to understand the association between air pollution exposure and acute cardiorespiratory effects (Langrish et al. 2010). These studies remain crucial when it comes to identifying important pathophysiological pathways involved in the adverse effects of air pollution. Whilst there is some limited evidence of an association between exposure to PM air pollution and the risk of arrhythmia, particularly in “at-risk” populations, the individual risk during a short controlled exposure study is likely to be extremely small. We have shown that such studies do not increase the short-term risk of arrhythmia in healthy volunteers and patient groups thought to have an increased susceptibility to the adverse effects of air pollution, such as those with chronic respiratory conditions and coronary heart disease.

Conclusions

Our data suggest that acute controlled exposures to air pollutants are safe and do not significantly increase the short-term risk of arrhythmia among individuals at low-risk of arrhythmia. Research employing these techniques, when scientifically and ethically justified, (National Research Council of the National Academies 2004; Rom et al. 2013) should continue and remains crucial in identifying pathophysiological pathways involved in the adverse effects of air pollution identified at the population level. These studies can be performed with minimal risk and have the potential for substantial societal benefit, informing environmental and public health policy decisions.

References

- Anderson HR, Armstrong B, Hajat S, Harrison R, Monk V, Poloniecki J, et al. 2010. Air pollution and activation of implantable cardioverter defibrillators in London. *Epidemiology* 21(3):405-413.
- Arora R. 2012. Recent insights into the role of the autonomic nervous system in the creation of substrate for atrial fibrillation: implications for therapies targeting the atrial autonomic nervous system. *Circ Arrhythm Electrophysiol* 5(4):850-859.
- Atkinson RW, Carey IM, Kent AJ, van Staa TP, Anderson HR, Cook DG. 2013. Long-term exposure to outdoor air pollution and incidence of cardiovascular diseases. *Epidemiology* 24(1):44-53.
- Barath S, Mills N, Törnqvist H, Lucking A, Langrish J, Söderberg S, et al. 2010. Impaired vascular function after exposure to diesel exhaust generated at urban transient running conditions. *Part Fibre Toxicol* 7:19.
- Bartell SM, Longhurst J, Tjoa T, Sioutas C, Delfino RJ. 2013. Particulate air pollution, ambulatory heart rate variability, and cardiac arrhythmia in retirement community residents with coronary artery disease. *Environ Health Perspect* 121(10):1135-1141.
- Berger A, Zareba W, Schneider A, Ruckerl R, Ibald-Mulli A, Cyrys J, et al. 2006. Runs of ventricular and supraventricular tachycardia triggered by air pollution in patients with coronary heart disease. *J Occup Environ Med* 48(11):1149-1158.
- Blomberg A, Mudway IS, Nordenhall C, Hedenstrom H, Kelly FJ, Frew AJ, et al. 1999. Ozone-induced lung function decrements do not correlate with early airway inflammatory or antioxidant responses. *Eur Respir J* 13(6):1418-1428.
- Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. 2010. Particulate Matter Air Pollution and Cardiovascular Disease. An Update to the Scientific Statement From the American Heart Association. *Circulation* 121:2331-2378.
- Bunch TJ, Horne BD, Asirvatham SJ, Day JD, Crandall BG, Weiss JP, et al. 2011. Atrial fibrillation hospitalization is not increased with short-term elevations in exposure to fine particulate air pollution. *Pacing Clin Electrophysiol* 34(11):1475-1479.
- Chiu HF, Tsai SS, Weng HH, Yang CY. 2013. Short-term effects of fine particulate air pollution on emergency room visits for cardiac arrhythmias: a case-crossover study in Taipei. *J Toxicol Environ Health A* 76(10):614-623.

- Colais P, Faustini A, Stafoggia M, Berti G, Bisanti L, Cadum E, et al. 2012. Particulate air pollution and hospital admissions for cardiac diseases in potentially sensitive subgroups. *Epidemiology* 23(3):473-481.
- Cruts B, van Etten L, Tornqvist H, Blomberg A, Sandstrom T, Mills NL, et al. 2008. Exposure to diesel exhaust induces changes in EEG in human volunteers. *Part Fibre Toxicol* 5:4.
- Dockery DW, Luttmann-Gibson H, Rich DQ, Link MS, Schwartz JD, Gold DR, et al. 2005. Particulate air pollution and nonfatal cardiac events. Part II. Association of air pollution with confirmed arrhythmias recorded by implanted defibrillators. *Res Rep Health Eff Inst*(124):83-126; discussion 127-148.
- Ghio AJ, Bassett M, Montilla T, Chung EH, Smith CB, Cascio WE, et al. 2012. Case report: supraventricular arrhythmia after exposure to concentrated ambient air pollution particles. *Environ Health Perspect* 120(2):275-277.
- Gold DR, Mittleman MA. 2013. New insights into pollution and the cardiovascular system: 2010 to 2012. *Circulation* 127(18):1903-1913.
- Haissaguerre M, Jais P, Shah DC, Takahashi A, Hocini M, Quiniou G, et al. 1998. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 339(10):659-666.
- Langrish J, Frampton M, Blomberg A. 2010. Human exposure studies. In: *Cardiovascular effects of inhaled ultrafine and nanosized particles*, (Casseo F, Mills N, Newby D, eds). Hoboken, New Jersey: J Wiley & Sons Inc., 217-239.
- Langrish JP, Bosson J, Unosson J, Muala A, Newby DE, Mills NL, et al. 2012a. Cardiovascular effects of particulate air pollution exposure: time course and underlying mechanisms. *J Intern Med* 272(3):224-239.
- Langrish JP, Li X, Wang S, Lee MM, Barnes GD, Miller MR, et al. 2012b. Reducing personal exposure to particulate air pollution improves cardiovascular health in patients with coronary heart disease. *Environ Health Perspect* 120(3):367-372.
- Langrish JP, Mills NL, Chan JK, Leseman DL, Aitken RJ, Fokkens PH, et al. 2009. Beneficial cardiovascular effects of reducing exposure to particulate air pollution with a simple facemask. *Part Fibre Toxicol* 6:8.
- Link MS, Luttmann-Gibson H, Schwartz J, Mittleman MA, Wessler B, Gold DR, et al. 2013. Acute exposure to air pollution triggers atrial fibrillation. *J Am Coll Cardiol* 62(9):816-825.
- Ljungman PL, Berglind N, Holmgren C, Gadler F, Edvardsson N, Pershagen G, et al. 2008. Rapid effects of air pollution on ventricular arrhythmias. *Eur Heart J* 29(23):2894-2901.

- Lo LW, Chiou CW, Lin YJ, Lee SH, Chen SA. 2011. Neural mechanism of atrial fibrillation: insight from global high density frequency mapping. *J Cardiovasc Electrophysiol* 22(9):1049-1056.
- Metzger KB, Klein M, Flanders WD, Peel JL, Mulholland JA, Langberg JJ, et al. 2007. Ambient air pollution and cardiac arrhythmias in patients with implantable defibrillators. *Epidemiology* 18(5):585-592.
- Mills N, Törnqvist H, Gonzales M, Vink E, Robinson S, Söderberg S, et al. 2007. Ischemic and thrombotic effects of dilute diesel-exhaust inhalation in men with coronary heart disease. *N Engl J Med* 357(11):1075-1082.
- Mills N, Törnqvist H, Robinson S, Gonzales M, Darnley K, MacNee W, et al. 2005. Diesel exhaust inhalation causes vascular dysfunction and impaired endogenous fibrinolysis. *Circulation* 112:3930-3936.
- Mills NL, Finlayson AE, Gonzalez MC, Tornqvist H, Barath S, Vink E, et al. 2011a. Diesel exhaust inhalation does not affect heart rhythm or heart rate variability. *Heart* 97(7):544-550.
- Mills NL, Miller MR, Lucking AJ, Beveridge J, Flint L, Boere AJ, et al. 2011b. Combustion-derived nanoparticulate induces the adverse vascular effects of diesel exhaust inhalation. *Eur Heart J* 32(21):2660-2671.
- Mills NL, Robinson SD, Fokkens PH, Leseman DL, Miller MR, Anderson D, et al. 2008. Exposure to concentrated ambient particles does not affect vascular function in patients with coronary heart disease. *Environ Health Perspect* 116(6):709-715.
- National Research Council of the National Academies. 2004. *Research Priorities for Airborne Particulate Matter: IV. Continuing Research Progress*: The National Academies Press. Page 36.
- Nawrot TS, Perez L, Kunzli N, Munters E, Nemery B. 2011. Public health importance of triggers of myocardial infarction: a comparative risk assessment. *Lancet* 377(9767):732-740.
- Odemuyiwa O, Malik M, Farrell T, Bashir Y, Poloniecki J, Camm J. 1991. Comparison of the predictive characteristics of heart rate variability index and left ventricular ejection fraction for all-cause mortality, arrhythmic events and sudden death after acute myocardial infarction. *Am J Cardiol* 68(5):434-439.
- Park HW, Shen MJ, Lin SF, Fishbein MC, Chen LS, Chen PS. 2012. Neural mechanisms of atrial fibrillation. *Curr Opin Cardiol* 27(1):24-28.

- Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, et al. 2000. Air pollution and incidence of cardiac arrhythmia. *Epidemiology* 11(1):11-17.
- Pieters N, Plusquin M, Cox B, Kicinski M, Vangronsveld J, Nawrot TS. 2012. An epidemiological appraisal of the association between heart rate variability and particulate air pollution: a meta-analysis. *Heart* 98(15):1127-1135.
- Rich DQ, Kim MH, Turner JR, Mittleman MA, Schwartz J, Catalano PJ, et al. 2006. Association of ventricular arrhythmias detected by implantable cardioverter defibrillator and ambient air pollutants in the St Louis, Missouri metropolitan area. *Occup Environ Med* 63(9):591-596.
- Rich KE, Petkau J, Vedal S, Brauer M. 2004. A case-crossover analysis of particulate air pollution and cardiac arrhythmia in patients with implantable cardioverter defibrillators. *Inhal Toxicol* 16(6-7):363-372.
- Rom WN, Boushey H, Caplan A. Experimental human exposure to air pollutants is essential to understand adverse health effects. *Am J Respir Cell Mol Biol*. 2013; 49(5):691-6.
- Rosenthal FS, Kuisma M, Lanki T, Hussein T, Boyd J, Halonen JI, et al. 2013. Association of ozone and particulate air pollution with out-of-hospital cardiac arrest in Helsinki, Finland: Evidence for two different etiologies. *J Expo Sci Environ Epidemiol* 23(3):281-288.
- Santos UP, Terra-Filho M, Lin CA, Pereira LA, Vieira TC, Saldiva PH, et al. 2008. Cardiac arrhythmia emergency room visits and environmental air pollution in Sao Paulo, Brazil. *J Epidemiol Community Health* 62(3):267-272.
- Sarnat SE, Suh HH, Coull BA, Schwartz J, Stone PH, Gold DR. 2006. Ambient particulate air pollution and cardiac arrhythmia in a panel of older adults in Steubenville, Ohio. *Occup Environ Med* 63(10):700-706.
- Shah A, Langrish J, Nair H, McAllister D, Hunter A, Donaldson K, et al. 2013. Global association of air pollution and heart failure: a systematic review and meta-analysis. *Lancet* 382(9897):1039-1048.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. 1996. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation* 93(5):1043-1065.
- Tsai SS, Chiu HF, Wu TN, Yang CY. 2009. Air pollution and emergency room visits for cardiac arrhythmia in a subtropical city: Taipei, Taiwan. *Inhal Toxicol* 21(13):1113-1118.
- Unosson J, Blomberg A, Sandstrom T, Muala A, Boman C, Nystrom R, et al. 2013. Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. *Part Fibre Toxicol* 10(1):20.

Vedal S, Rich K, Brauer M, White R, Petkau J. 2004. Air pollution and cardiac arrhythmias in patients with implantable cardioverter defibrillators. *Inhal Toxicol* 16(6-7):353-362.

Zanobetti A, Coull BA, Gryparis A, Kloog I, Sparrow D, Vokonas PS, et al. 2013. Associations between arrhythmia episodes and temporally and spatially resolved black carbon and particulate matter in elderly patients. *Occup Environ Med*. Epub ahead of print. Available at <http://dx.doi.org> doi: 10.1136/oemed-2013-101526.

Table 1. Baseline characteristics and medical history of subjects included in studies.

Parameter	Controlled Exposure: Diesel Exhaust	Controlled Exposure: Woodsmoke	Controlled Exposure: Ozone	Controlled Exposure: CAPs	Controlled Exposure: Engineered Carbon NPs	Ambient Exposure: Personal Monitoring
Healthy Volunteers						
<i>Baseline characteristics</i>						
Number	80	29	15	17	14	14
Male	74 (93%)	21 (72%)	15 (100%)	17 (100%)	14 (100%)	2 (14%)
Age, years	25 (18-24)	26 (20-35)	25 (22-30)	48 (21-69)	21 (20-44)	27 (20-45)
BMI, kg/m ²	24 ± 3	25 ± 4	N/A	25 ± 3	23 ± 2	21 ± 2
Pulse, bpm	65 ± 12	62 ± 13	68 ± 13	63 ± 10	74 ± 8	79 ± 3
SBP, mmHg	138 ± 17	122 ± 14	150 ± 15	139 ± 20	132 ± 12	113 ± 8
DBP, mmHg	72 ± 9	72 ± 8	74 ± 8	77 ± 8	68 ± 8	73 ± 6
Hemoglobin, g/dL	147 ± 11	146 ± 9	148 ± 9	144 ± 10	148 ± 11	
Creatinine, µmol/L	76 ± 12	78 ± 20	N/A	N/A	N/A	N/A
Patients						
<i>Baseline characteristics</i>						
Number	37	N/A	N/A	12	N/A	93
Male	33 (89%)	N/A	N/A	12 (100%)	N/A	80 (86%)
Age, years	63 (51-80)	N/A	N/A	59 (45-68)	N/A	63 (45-77)
BMI, kg/m ²	27 ± 3	N/A	N/A	28 ± 3	N/A	26 ± 3
Pulse, bpm	57 ± 8	N/A	N/A	53 ± 5	N/A	67 ± 10
SBP, mmHg	139 ± 20	N/A	N/A	138 ± 10	N/A	131 ± 17
DBP, mmHg	77 ± 8	N/A	N/A	80 ± 10	N/A	79 ± 10
Hemoglobin, g/dL	140 ± 11	N/A	N/A	148 ± 7	N/A	142 ± 11
Creatinine, µmol/L	78 ± 12	N/A	N/A	101 ± 12	N/A	76 ± 16

Parameter	Controlled Exposure: Diesel Exhaust	Controlled Exposure: Woodsmoke	Controlled Exposure: Ozone	Controlled Exposure: CAPs	Controlled Exposure: Engineered Carbon NPs	Ambient Exposure: Personal Monitoring
<i>Medical History</i>						
Previous MI	23 (62%)	N/A	N/A	7 (58%)	N/A	68 (73%)
Diabetes Mellitus	0 (0%)	N/A	N/A	0 (0%)	N/A	43 (46%)
Hypercholesterolaemia	25 (68%)	N/A	N/A	12 (100%)	N/A	40 (43%)
Hypertension	8 (22%)	N/A	N/A	4 (33%)	N/A	75 (81%)
Angina Pectoris	14 (38%)	N/A	N/A	5 (42%)	N/A	65 (70%)
<i>Medication Use</i>						
Aspirin	36 (97%)	N/A	N/A	12 (100%)	N/A	87 (94%)
Clopidogrel	3 (8%)	N/A	N/A	0 (0%)	N/A	16 (17%)
Beta-blocker	26 (70%)	N/A	N/A	11 (92%)	N/A	67 (72%)
Statin	32 (87%)	N/A	N/A	12 (100%)	N/A	73 (79%)
ACE Inhibitor	13 (35%)	N/A	N/A	1 (8%)	N/A	28 (30%)

NPs = nanoparticles; BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; MI = myocardial infarction; ACE = angiotensin converting enzyme. Data expressed as number (%), mean \pm standard deviation or median (interquartile range) as appropriate.

Table 2. Exposure parameters for air pollution exposures.

Exposure	Group	PM _{2.5} (µg/m ³)	PM ₁₀ (µg/m ³)	Particle Count (x10 ⁴ /cm ³)	CO (ppm)	NO _x (ppm)	NO (ppm)	NO ₂ (ppm)	Ozone (ppm)	THC (ppm)
Diesel Exhaust										
60 mins (n=64)	HV	N/A	307	79	6.57	4.08	4.45	1.10	N/A	3.0
60 mins (n=37)	Patients	N/A	294	103	3.82	3.29	2.46	0.83	N/A	2.8
120 mins (n=16)	HV	N/A	363	120	3.50	0.60	0.40	0.20	N/A	N/A
Ambient										
120 mins (n=14)	HV	86	N/A	2.4	N/A	N/A	N/A	N/A	N/A	N/A
120 mins (n=93)	Patients	89	N/A	4.4	N/A	N/A	N/A	N/A	N/A	N/A
Wood smoke										
60 mins (n=15)	HV	895 (PM ₁)	N/A	N/A	15.32	N/A	0.53	N/A	N/A	N/A
180 mins (n=14)	HV	314 (PM ₁)	N/A	N/A	26.00	0.41	N/A	N/A	N/A	N/A
Ozone										
75 mins (n=15)	HV	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.30	N/A
Concentrated Ambient Particles										
120 mins (n=29)	HV & Patients	N/A	190	9.9	0.02	0.01	0.01	0.01	N/A	N/A
Engineered Carbon Nanoparticles										
120 mins (n=14)	HV	N/A	70	387	N/A	N/A	N/A	N/A	N/A	N/A

PM_{2.5} = particulate matter with a mean aerodynamic diameter of ≤2.5 µm; PM₁₀ = particulate matter with a mean aerodynamic diameter of ≤10 µm; CO = carbon monoxide; NO_x = oxides of nitrogen; NO = nitric oxide; NO₂ = nitrogen dioxide; THC = total hydrocarbons. Data expressed and mean or median as appropriate.

Table 3. Occurrence and number of arrhythmias in all subjects (n=282) included in the study. Odds ratio of arrhythmia occurring following pollutant “exposure” as compared to non-exposed “air” controls.

Exposure and arrhythmia	Unexposed: No. subjects with documented arrhythmia	Exposure: No. subjects with documented arrhythmia	Odds Ratio (95% CI)	P Value	Unexposed: No. events per subject [median (IQR)]	Exposure: No. events per subject [median (IQR)]	P value
Diesel Exhaust (n=117)							
Pause	2	1	0.50 (0.04-5.55)	0.56	0 (0-0)	0 (0-0)	>0.99
Dropped Beat	36	37	1.04 (0.60-1.81)	0.89	0 (0-1)	0 (0-1)	0.72
VT	1	0	0.33 (0.01-8.20)	0.32	0 (0-0)	0 (0-0)	>0.99
Salvo	1	4	4.11 (0.45-37.32)	0.18	0 (0-0)	0 (0-0)	0.56
Triplet	1	3	3.05 (0.31-29.80)	0.31	0 (0-0)	0 (0-0)	>0.99
Couplet	9	11	1.25 (0.50-3.13)	0.64	0 (0-0)	0 (0-0)	>0.99
Bradycardia	60	57	0.90 (0.54-1.51)	0.69	1 (0-57)	0 (0-35.5)	0.30
SVT	2	2	1.00 (0.14-7.22)	1.00	0 (0-0)	0 (0-0)	>0.99
Atrial Fibrillation	0	0	N/A	N/A	N/A	N/A	N/A
Bigeminy	7	3	0.41 (0.10-1.64)	0.20	0 (0-0)	0 (0-0)	0.96
Trigeminy	4	4	1.00 (0.24-4.10)	1.00	0 (0-0)	0 (0-0)	0.50
VE	58	54	0.87 (0.52-1.46)	0.60	1 (0-4)	0 (0-4.5)	0.75
SVE	61	65	1.15 (0.69-1.92)	0.60	1 (0-6)	0 (0-4.5)	0.90
Ambient (n=107)							
Pause	0	0	N/A	N/A	N/A	N/A	N/A
Dropped Beat	1	2	2.02 (0.18-22.62)	0.56	0 (0-0)	0 (0-0)	>0.99
VT	1	2	2.02 (0.18-22.62)	0.56	0 (0-0)	0 (0-0)	>0.99
Salvo	1	2	2.02 (0.18-22.62)	0.56	0 (0-0)	0 (0-0)	>0.99
Triplet	1	0	0.33 (0.01-8.20)	0.32	0 (0-0)	0 (0-0)	>0.99
Couplet	9	4	0.42 (0.13-1.42)	0.15	0 (0-0)	0 (0-0)	0.24
Bradycardia	25	21	0.8 (0.42-1.54)	0.51	0 (0-0)	0 (0-0)	0.82
SVT	2	5	2.57 (0.49-13.57)	0.25	0 (0-0)	0 (0-0)	0.45

Exposure and arrhythmia	Unexposed: No. subjects with documented arrhythmia	Exposure: No. subjects with documented arrhythmia	Odds Ratio (95% CI)	P Value	Unexposed: No. events per subject [median (IQR)]	Exposure: No. events per subject [median (IQR)]	P value
Atrial Fibrillation	0	1	3.03 (0.12-75.34)	0.32	0 (0-0)	0 (0-0)	>0.99
Bigeminy	16	18	1.15 (0.55-2.40)	0.71	0 (0-0)	0 (0-0)	0.80
Trigeminy	4	5	1.26 (0.33-4.84)	0.73	0 (0-0)	0 (0-0)	0.50
VE	87	86	0.94 (0.48-1.86)	0.86	7 (1-66)	7 (1-83)	0.52
SVE	86	88	1.13 (0.57-2.25)	0.73	5 (1-28)	6 (1-28)	0.25
Concentrated Ambient Particles (n=29)							
Pause	1	0	0.32 (0.01-8.24)	0.31	0 (0-0)	0 (0-0)	>0.99
Dropped Beat	5	5	1.00 (0.26-3.91)	1.00	0 (0-0)	0 (0-0)	0.73
VT	0	0	N/A	N/A	N/A	N/A	N/A
Salvo	1	0	0.32 (0.01-8.24)	0.31	0 (0-0)	0 (0-0)	>0.99
Triplet	2	4	2.16 (0.36-12.85)	0.39	0 (0-0)	0 (0-0)	>0.99
Couplet	2	3	1.56 (0.24-10.10)	0.64	0 (0-0)	0 (0-0)	>0.99
Bradycardia	16	14	0.76 (0.27-2.13)	0.60	2 (0-33)	0 (0-28.5)	0.42
SVT	0	0	N/A	N/A	N/A	N/A	N/A
Atrial Fibrillation	0	0	N/A	N/A	N/A	N/A	N/A
Bigeminy	2	1	0.48 (0.04-5.64)	0.55	0 (0-0)	0 (0-0)	>0.99
Trigeminy	1	2	2.07 (0.18-24.24)	0.55	0 (0-0)	0 (0-0)	>0.99
VE	22	26	2.76 (0.64-11.96)	0.16	5 (0.5-34.5)	4 (2-28.5)	0.93
SVE	23	23	1.00 (0.28-3.56)	1.00	2 (1-8)	4 (1-13)	0.06
Wood smoke (n=29)							
Pause	2	0	0.19 (0.01-4.06)	0.15	0 (0-0)	0 (0-0)	0.50
Dropped Beat	7	9	1.41 (0.44-4.51)	0.56	0 (0-0.5)	0 (0-1)	0.17
VT	0	0	N/A	N/A	N/A	N/A	N/A
Salvo	0	0	N/A	N/A	N/A	N/A	N/A
Triplet	0	0	N/A	N/A	N/A	N/A	N/A

Exposure and arrhythmia	Unexposed: No. subjects with documented arrhythmia	Exposure: No. subjects with documented arrhythmia	Odds Ratio (95% CI)	P Value	Unexposed: No. events per subject [median (IQR)]	Exposure: No. events per subject [median (IQR)]	P value
Couplet	0	0	N/A	N/A	N/A	N/A	N/A
Bradycardia	18	18	1.00 (0.35-2.90)	1.00	3 (0-113)	5 (0-87.5)	0.32
SVT	0	0	N/A	N/A	N/A	N/A	N/A
Atrial Fibrillation	0	0	N/A	N/A	N/A	N/A	N/A
Bigeminy	1	0	0.32 (0.01-8.24)	0.31	0 (0-0)	0 (0-0)	>0.99
Trigeminy	1	0	0.32 (0.01-8.24)	0.31	0 (0-0)	0 (0-0)	>0.99
VE	19	15	0.56 (0.20-1.62)	0.29	0 (0-1)	0 (0-1)	0.07
SVE	17	14	0.66 (0.23-1.86)	0.43	3 (1-10.5)	2 (0-7.5)	0.79
Engineered carbon nanoparticles (n=14)							
Pause	0	0	N/A	N/A	N/A	N/A	N/A
Dropped Beat	14	12	0.17 (0.01-3.94)	0.14	6 (2.75-14)	6.5 (3.25-13.75)	0.88
VT	0	0	N/A	N/A	N/A	N/A	N/A
Salvo	0	0	N/A	N/A	N/A	N/A	N/A
Triplet	0	0	N/A	N/A	N/A	N/A	N/A
Couplet	0	0	N/A	N/A	N/A	N/A	N/A
Bradycardia	7	6	0.75 (0.17-3.32)	0.70	0.5 (0-64)	0 (0-26)	0.74
SVT	0	0	N/A	N/A	N/A	N/A	N/A
Atrial Fibrillation	0	0	N/A	N/A	N/A	N/A	N/A
Bigeminy	0	0	N/A	N/A	N/A	N/A	N/A
Trigeminy	0	0	N/A	N/A	N/A	N/A	N/A
VE	7	9	1.80 (0.40-8.19)	0.45	0.5 (0-7.25)	1 (0-5.5)	0.64
SVE	9	10	1.39 (0.28-6.84)	0.69	2.5 (0-5)	2 (0-5)	0.42
Ozone (n=15)							
Pause	1	1	1.00 (0.06-17.63)	1.00	0 (0-0)	0 (0-0)	>0.99
Dropped Beat	5	8	2.29 (0.52-10.01)	0.27	0 (0-1)	1 (0-4)	0.23

Exposure and arrhythmia	Unexposed: No. subjects with documented arrhythmia	Exposure: No. subjects with documented arrhythmia	Odds Ratio (95% CI)	P Value	Unexposed: No. events per subject [median (IQR)]	Exposure: No. events per subject [median (IQR)]	P value
VT	0	0	N/A	N/A	N/A	N/A	N/A
Salvo	0	0	N/A	N/A	N/A	N/A	N/A
Triplet	0	0	N/A	N/A	N/A	N/A	N/A
Couplet	0	0	N/A	N/A	N/A	N/A	N/A
Bradycardia	12	11	0.69 (0.12-3.79)	0.67	18 (1-271)	14 (0-33)	0.12
SVT	0	0	N/A	N/A	N/A	N/A	N/A
Atrial Fibrillation	0	0	N/A	N/A	N/A	N/A	N/A
Bigeminy	0	0	N/A	N/A	N/A	N/A	N/A
Trigeminy	0	0	N/A	N/A	N/A	N/A	N/A
VE	12	8	0.29 (0.06-1.44)	0.25	2 (1-3)	1 (0-2)	0.21
SVE	9	12	2.67 (0.52-13.66)	0.23	1 (0-3)	1 (1-3)	0.86

VT = ventricular tachycardia; SVT = supraventricular tachycardia including atrial fibrillation; VE = ventricular ectopic beat;

SVE = supraventricular ectopic beat; bradycardia defined as HR <50 bpm. Data expressed as number or median (interquartile range) as appropriate.

P values and odds ratios from Chi-squared analysis and Wilcoxon Matched-Pairs Signed Rank Test as appropriate.

Figure legend

Figure 1. Forrest plot showing the risk of arrhythmias during and after exposure to air pollutants compared to a control air exposure (or in the presence of a highly efficient facemask for the ambient exposures). Data expressed as odds ratio (95% confidence intervals), and dotted line plotted at an odds ratio of 1. $P > 0.05$ for all (Chi-squared analysis).

VT = ventricular tachycardia; SVT = supraventricular tachycardia; AF = atrial fibrillation; VE = ventricular ectopy; SVE = supraventricular ectopy; CAPs = concentrated ambient particles; NPs = nanoparticles.

Figure 1.

