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Estimated effects of air pollution and space-time-activity on cardiopulmonary outcomes in healthy adults: A repeated measures study

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ABSTRACT

Background: Exposure to air pollution is known to affect both short and long-term outcomes of the cardiopulmonary system; however, findings on short-term outcomes have been inconsistent and often from isolated and long-term rather than coexisting and short-term exposures, and among susceptible/unhealthy rather than healthy populations.
Aims: We aimed to investigate separately the annual, daily and daily space-time-activity-weighted effect of ambient air pollution, as well as confounding or modification by other environmental (including noise) or space-time-activity (including total daily physical activity) exposures, on cardiopulmonary outcomes in healthy adults.
Methods: Participants ($N = 57$: 54% female) had indicators of cardiopulmonary outcomes [blood pressure (BP), pulse (HR) and heart rate variability (HRV {SDNN}), and lung function (spirometry {FEV_1, FVC, SUM})] measured on four different mornings (at least five days apart) in a clinical setting between 2011 and 2014. Spatiotemporal ESCAPE-LUR models were used to estimate daily and annual air pollution exposures (including PM_{10}, PM_{Coarse}, but not Ozone {derived from closest station}) at participant residential and occupational addresses. Participants' time-activity diaries indicated time spent at either address to allow daily space-time-activity-weighted estimates, and capture total daily physical activity (total-PA {as metabolic-equivalents-of-task, METs}), in the three days preceding health measurements. Multivariate-adjusted linear mixed-effects models (using either annual or daily estimates) were adjusted for possible environmental confounders or mediators including levels of ambient noise and greenness. Causal mediation analysis was also performed separately considering these factors as well as total-PA. All presented models are controlled by age, height, sex and season.
Results: An increase in 5 μg/m^3 of daily space-time-activity-weighted PM_{Coarse} exposure was statistically significantly associated with a 4.1% reduction in total heart rate variability (SDNN; $p = 0.01$), and remained robust after adjusting for suspected confounders [except for occupational-address noise ($\beta = -2.7$, $p = 0.20$)]. An increase in 10 ppb of annual mean Ozone concentration at the residential address was statistically significantly associated with an increase in diastolic BP of 6.4 mm Hg ($p < 0.01$), which lost statistical significance when substituted with daily space-time-activity-weighted estimates. As for pulmonary function, an increase in 10 μg/m^3 of annual mean PM_{10} concentration at the residential address was significantly associated with a 0.3% reduction in FVC ($p < 0.01$) and a 0.5% reduction in SUM ($p < 0.04$), for which again significance was lost

Abbreviations: BP, blood pressure; HR, heart rate (pulse); FEV_1, forced expiratory volume in first second; FVC, forced vital capacity; SUM, largest sum of FEV_1 and FVC; PA, physical activity; PM_{10}, particulate matter of < 10 μm diameter; PM_{Coarse}, particulate matter of 10 to 2.5 μm diameter; $PM_{2.5}$, particulate matter of < 2.5 μm diameter; NO_2, nitrogen dioxide; NOx, nitrogen oxides; O_3, ozone

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when substituted for daily space-time-activity-weighted estimates These associations with pulmonary function remained robust after adjusting for suspected confounders, including annual Ozone, as well as total-PA and bioaerosol (pollen and fungal spore) levels (but not residential-neighborhood greenness {$\beta = -0.22$, $p = 0.09$; $\beta = -0.34$, $p = 0.15$, respectively}). Multilevel mediation analysis indicated that the proportion mediated as a direct effect on cardiopulmonary outcomes by suspected confounders (including total-PA, residential-neighborhood greenness, and occupational-address noise level) from primary exposures (including PM_{10}, PM_{Coarse}, and O_3) was not statistically significant.

Conclusion: Our findings suggest that increased daily space-time-activity-weighted PM_{Coarse} exposure levels significantly adversely affect cardiac autonomic modulation (as reduced total HRV) among healthy adults. Additionally, increased annual levels at the residential address of Ozone and PM_{10} significantly increase diastolic blood pressure and reduce lung function, respectively, among healthy adults. These associations typically remained robust when adjusting for suspected confounders. Occupational-address noise and residential-neighborhood greenness levels, however, were seen as mediators of cardiovascular and pulmonary outcomes, respectively. Total daily physical activity was not seen as a mediator of any of the studied outcomes, which supports the promotion of active mobility within cities.

1. Introduction

Ambient air pollution exposure has been well-established as an adversary to public health (WHO Regional Office for Europe, 2015), especially airborne particulate matter (PM) of a size considered inhalable (Kim et al., 2015), through long-term studies. It is known to exacerbate underlying conditions of the cardiovascular (Hoek et al., 2013) and pulmonary (Langrish et al., 2012; Qian et al., 2013) systems. Short-term effects of exposure in healthy people, however, are less studied and have been shown as inconsistent in association (Panasevich et al., 2009), particularly for measures such as heart rate variability (HRV; reflecting acute changes in cardiac autonomic modulation, with reduced variability indicating heightened cardiovascular disease risk) (Cole-Hunter et al., 2016; Weichenthal, 2012). Moreover, it is believed that the major impact of air pollution exposure may be through eliciting subclinical and asymptomatic effects not easily detected or reported for investigation (WHO Regional Office for Europe, 2006).

Existing studies have shown daily variation in exposure to ambient PM_{10} and $PM_{2.5}$ (as respirable), and gaseous nitrogen dioxide (NO_2) and Ozone (O_3), to be linked to acute pulmonary (Ostro et al., 2001) and cardiovascular (Qian et al., 2013; Künzli et al., 2010; Chuang et al., 2007) health risk outcomes. Moreover, levels considered generally safe by regulatory authorities have been suggested to also increase the daily (Beelen et al., 2014) and even hourly (Wellenius et al., 2012) risk of adverse health outcomes. As well as variations in time, it is important to investigate more finely the ambient co-exposures and space-time-activity behaviors that occur in an urban environment, for the consideration of modification or modulation effect. This may be better done in real-world conditions rather than controlled human exposure studies. For example, exposure to PM in combination with Ozone (O_3) has been seen to increase diastolic blood pressure (BP; as with reduced HRV, such a change indicating a heightened risk) among healthy adults (Brook et al., 2009).

Noise, as a different form of pollution emitted with traffic, also needs to be considered in the complex, urban ambient mix. Health effects of ambient noise have been observed in combination with associated traffic-related air pollution components, confounding associations with health outcomes (Beelen et al., 2008; De Kluizenaar et al., 2007; Leon Bluhm et al., 2007). Other confounders may exist in an urban (built) environment, including public space (such as parks, or green spaces promoting greenness) and transport infrastructure. A consequence of urban greenness can be natural sources of aerosols ('bioaerosols'), of which daily level variations have been associated with adverse pulmonary outcomes (e.g. reduced lung function), after adjusting for air pollutants, in some populations (Zhang et al., 2012; DellaValle and Triche, 2012; Carracedo-Martinez et al., 2008). In addition, real-world (complex) exposures in experimental studies with traffic-related air pollution among healthy adults have shown mitigations in cardiovascular health outcomes through intermittent, moderate physical activity emulating active mobility (Matt et al., 2016; Cole-Hunter et al., 2016; Kubesch et al. 2015a). Considerations of the above exposures combined have been made in a recent health impact assessment which relies on evidence produced by experimental and epidemiology studies, which in turn rely on exposure-assessment methods such as modelling (Mueller et al., 2017).

A number of studies have used land-use regression modelling to estimate ambient air pollution exposure concentrations, including that of $PM_{2.5}$ and NO_2, at the residential addresses of study participants (Henderson et al., 2007; Eeftens et al., 2012b; Wang et al., 2013; Hoek et al., 2008). Noise exposure levels have also been modelled (Xie et al., 2011), and links to cardiovascular health outcomes have been made also considering air pollution co-exposure (Beelen et al., 2008). A recent review, however, has called for more personal methods (Steinle et al., 2013) such as time-activity diaries to determine micro-environment exposure sources and periods such as commuting (Morabia et al., 2010) or co-inhabiting with environmental tobacco smoke (Aquilina et al., 2010). Another recent review has called for more studies using traffic-related pollution indicators to properly assess confounding of health effects by noise or air pollutants (Tétreault et al., 2013).

Accordingly, the current study aimed to augment evidence on the effects of annual and daily space-time-activity-weighted estimates of ambient air pollution, as well as consider confounding or mediating effects of co-exposures including neighborhood noise, daily bioaerosol and total physical activity (alongside other space-time-activity) levels, on short-term intermediary cardiopulmonary health indicators (outcomes) among healthy adults.

2. Methods

2.1. Study design and protocol

The current analysis makes use of data collected in two separate (but methodologically comparable) experimental case-crossover studies, within the TAPAS (de Nazelle et al., 2011) and EXPOsOMICS (Vineis et al., 2016) projects, which ran from 2011 to 2014. As part of these experimental studies, repeated measures were taken of a set of baseline health markers on four occasions (sessions) by each participant at the same study clinic. Each of these measures took place in the morning between 07:00 to 08:00 h to minimise diurnal variation. The four sessions of a participant took place at least five days apart on either a Tuesday, Wednesday or Thursday to avoid weekend-influenced environmental quality parameters, e.g. air quality due to traffic congestion. The period of participation per participant lasted from one to four months, depending on availability of participants, study personnel and equipment.

See Fig. S1 for a flow chart of the study design.

Participants provided their home (residential) and work or school (occupational) addresses and were asked to fill out time-activity diaries