Short- and longer-term health effects of air pollution and noise exposure related to transport behaviour: the MobiliSense Study

Web Appendix

Web appendix 1: Literature review of air pollution and noise effects on selected health outcomes

Respiratory symptoms

Studies of long-term exposure to air pollutants in adults have usually documented associations with respiratory symptoms.[1] A Swiss study documented positive associations between annual concentrations of nitrogen dioxide (NO_2) or particulate matter with an aerodynamic diameter of 10 µm or less (PM_{10}) and chronic phlegm production, chronic cough, breathlessness at rest, and dyspnea.[2]

Regarding studies of short-term effects, a work conducted in different European cities based on background concentrations over 24 hours measured from a central site and on a daily respiratory questionnaire completed over 6 months concluded that a high concentration of particulate matter with an aerodynamic diameter between 2.5 and 10 µm was positively associated with shortness of breath and wheezing.[3] As another example relevant to the present project devoted to exposures during trips, a Dutch study of 489 adults found that the exposure to black smoke over 24 hours (as an indicator of black carbon emitted by diesel engines) was associated with upper respiratory symptoms, in a more consistent way than the exposure to sulphate and PM_{10}.[4]

Several studies have focused on the effects of air pollutants on the exacerbation of symptoms among asthmatics or chronic obstructive pulmonary disease (COPD) patients. For example, a study of 75 asthmatic or COPD patients from the United Kingdom reported that high concentrations of NO_2 and ozone (O_3) over 24 hours were associated with wheezing or dyspnea within one or two days.[5] As another example, a study suggested that a higher concentration of PM_{10} was associated with dyspnea one day after.[6] However, certain studies also reported negative findings. Paradoxically, a study of 16 COPD patients that analysed, in

addition to the background concentrations of particulate matter with an aerodynamic diameter of 2.5 µm or less ($PM_{2.5}$), data from a personal monitoring of the exposure to $PM_{2.5}$ did not identify an association with any of the respiratory symptoms examined,[7] perhaps because of the weak number of participants.

A limitation of these studies is that paper questionnaires referring to relatively long recall periods (e.g., 24 hours, one week, one month) were used, implying reporting biases in the symptoms.

Lung function

A relatively weak number of studies have focused on the relationships between the long-term exposure to air pollutants and lung function. For example, a Swiss study showed that elevated annual concentrations of sulfur dioxide (SO_2), NO_2, and PM_{10} were associated with a lower forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), with stronger relationships observed for PM_{10}.[8] A longitudinal analysis from the same study subsequently documented that a decrease in the concentration of PM_{10} was associated with a lower decrease of lung function over time.[9] More recently, a French study reported that a higher concentration of PM_{10} over the preceding 12 months was associated with a lower FVC and FEV_1.[10] However, it is important to emphasize that, comparing different exposure areas with each other, these studies of the long-term effects of air pollutants are vulnerable to residual confounding.[11]

Apart from investigations of long-term exposure effects, studies of the effects of short-term exposure to air pollutants were conducted[12] based on repeated spirometry measurements. For example, an Italian study of 29 participants found that a higher daily concentration of NO_2 was related to a decrease in FEV_1 among asthmatics but not among coronary patients.[13] The strength of the associations identified in studies seemed to depend on the average concentration of pollutants over the territory, with stronger associations at higher average

concentrations. In a study of 16 COPD patients in Vancouver where the average concentration of PM_{10} was of 18 µg/m^3, each 10 µg/m^3 increase in the concentration of PM_{10} measured over one day was associated with a 3% larger decrease in FEV_1 between the morning and the evening measure.[7] In a study in which the average concentration of PM_{10} over 24 hours was above 150 µg/m^3, a 10 µg/m^3 increase in the concentration was related to a decrease of up to 7% in FEV_1.[14] However, a recent study also emphasized that even a "moderate" (compared to a "good") range of exposure to $PM_{2.5}$, NO_2, and O_3 over 24 hours according to the classification of the US Environmental Protection Agency was associated with a decreased FEV_1.[15]

Some studies of short-term effects of air pollutants have focused on the size of the time window to consider to better identify associations with lung function. A Korean study that compared different strategies to proxy the individual residential exposure from measures performed at fixed monitoring stations showed that a high concentration of PM_{10} was associated with a reduced FVC, especially when concentrations were assessed over 24 hours two days before the spirometry test.[16] This finding is coherent with another study that reported that an elevated concentration of PM_{10} over the 37–60 hours preceding the spirometry assessment was associated with a decreased FVC and FEV_1.[17] However, it must be emphasized that certain studies did not observe a relationship between the exposure to air pollutants and lung function.[18]

Very few studies have established a direct link with the transport activity. One experimental (thus poorly generalizable) study of 60 participants demonstrated that walking for two hours in Oxford Street was associated with more important reductions in FVC and FEV_1 than that observed in the same participants when walking through Hyde Park.[12] Such an effect was particularly attributable to the higher exposure to ultrafine particles and black carbon (as markers of road traffic with diesel engines) in Oxford Street.

Limitation of these repeated measure studies of lung function is that they were often based on a low sample size.

Blood pressure

Air pollution effects: Relatively few studies investigated associations between the chronic exposure to air pollutants and blood pressure or the prevalence / incidence of hypertension. For example, in a US study, a higher exposure to nitrogen oxides (NO_x) but not to $PM_{2.5}$ was associated with a slight increase in the incidence of hypertension over 10 years.[19] Similarly, a German study reported that higher concentrations of $PM_{2.5}$ were associated with an increased blood pressure, which association persisted after adjustment for road traffic noise.[20]

A larger number of blood pressure studies considered short-term exposures to air pollutants. Transient elevations of blood pressure repeated daily over years could lead to chronic blood pressure increase; moreover, a transient increase in blood pressure can trigger cardiovascular events in vulnerable individuals[21] (as a potential explanation of the increased incidence of cardiovascular events during pollution episodes[22]). As an example, a US study reported that an increase of 10 $\mu g/m^3$ in the daily concentration of $PM_{2.5}$ was associated with a 3.2 mmHg higher systolic blood pressure, with still stronger effects in the area where the average concentration of PM2.5 was the highest.[22] However, certain studies did not observe such positive short-term association, or even documented negative associations.[23] These incoherent patterns may be attributable to the fact that blood pressure depends on both vascular resistance and the cardiac output, while the main hypothesis for air pollution effects is related to the first aspect (air pollution increases peripheral resistance and decreases elasticity of arterial walls). This is why our MobiliSense study also focuses on markers of arterial stiffness. Other sources of inconsistency include varying sources and composition of

suspended particles from one place to the other, differences in the susceptibility of populations, etc.[23]

Two studies found that the positive relationship between the short-term exposure to $PM_{2.5}$ and blood pressure was stronger in areas where road traffic was dense.[22, 24] An experimental study demonstrated that the effects documented on blood pressure were attributable to the organic carbon fraction of $PM_{2.5}$, mainly to fossil fuel combustion products of traffic sources.[25] Another study that relied on repeated measures did not find any association between the concentration of $PM_{2.5}$ and blood pressure but reported that a higher exposure to black carbon over the previous 7 days was related to an increased blood pressure.[26]

Aforementioned studies on blood pressure have assessed air pollutants with fixed monitoring stations, while very few studies were able to measure personal exposure.[27, 28] One study that measured concentrations of $PM_{2.5}$, organic carbon, and black carbon directly outside the residence showed that the strongest positive associations with blood pressure were documented for organic carbon, and for periods where the participants were at home and where measurement error was consequently the weakest.[29] However, some studies that relied on wearable monitors of $PM_{2.5}$ (carried in a backpack) did not permit to conclude that personal exposure was more strongly associated with blood pressure than background concentrations.[30, 31] One study[27] however showed that exposure to $PM_{2.5}$ measured with a wearable monitor was more strongly associated with blood pressure than the concentration of $PM_{2.5}$ measured outside each participant's residence; but that the concentration of black carbon outside the residence showed a still stronger association with blood pressure, suggesting that a priority for future research is to perform a personal monitoring of black carbon (which was not done in this study).

Noise effects: Regarding long-term effects, according to the World Health Organisation,[32] associations were consistently documented between the residential exposure to road traffic noise and hypertension. Most studies relied on noise maps derived from noise dispersion models. For example, a Swedish study of 1953 participants showed a positive association between road traffic noise (at the residence, from noise maps) and self-reported physician-diagnosed hypertension, with a stronger association documented with incidence than with prevalence (based on a retrospective questionnaire), and with a stronger association when the analyses were restricted to individuals who had lived for a long time at their residence.[33] A Swedish cross-sectional study of 667 subjects found that the adjusted odds of self-reported physician-diagnosed hypertension were 1.38 times larger for each 5 dB(A) increase in road traffic noise exposure (dispersion model and expert classification) at lower overall noise levels than in other studies.[34] Interestingly, the association was stronger among participants who had lived at the address for >10 years and among those not having triple-glazed windows, living in an old house, and having the bedroom facing a street. Other studies performed direct noise measurements in selected study sites to assess long-term exposures. For example, a cross-sectional study conducted in Taiwan among 321 males and 499 females residing nearby four main roads of Taichung along which measurements were performed reported an adjusted dose-response increase across noise exposure groups in the prevalence of self-reported physician-diagnosed hypertension.[35]

Regarding other sources than road traffic, certain studies have documented relationships between air traffic noise and hypertension or blood pressure,[36-38] while few were able to take into account railway traffic noise or the multi-exposure to noise of different transport modes. For example, a Swiss study of 6450 participants found that a cross-sectional measure of systolic blood pressure increased by 0.6 or 0.8 mmHg for each 10 dB(A) increase in daytime or night-time exposure to railway noise, while associations with road traffic noise were only

documented among participants with diabetes.[39] Of interest for the present project interested in the multi-exposure to noise and air pollutants, the adjustment for outdoor annual concentrations of NO_2 and PM_{10} did not result in weaker associations of road or railway traffic noise with blood pressure. Also of interest for the innovative joint assessment of objective and subjective noise levels in the present project, the European HYENA study found that the positive association between aircraft noise and the prevalence of hypertension was stronger among participants who reported being annoyed by aircraft noise.[40]

Only few studies in real-life, non-occupational settings have relied on wearable noise sensors. Pointing to the limitations of resting blood pressure, a study of 60 young adults in Taiwan that simultaneously measured ambulatory blood pressure and personal noise exposure over 24 hours found that each 5 dB(A) increase in noise exposure was associated with a transient increase of 1.15 and 1.16 mmHg in systolic and diastolic blood pressure during daytime and of 0.74 and 0.77 mmHg during night-time.[41] A German study of 632 adolescents and 482 adults documented an association between night-time noise exposure (personal dosimetry over 24 hours) and hypertension (measured blood pressure), but did not report any association with the subjective assessment of noise from a diary (the study, however, did not examine interactions between objective and subjective noise assessments).[42]

Regarding limitations, first, most studies of long-term effects of noise have used a cross-sectional design (no incidence data on hypertension) and relied on self-reports of physician-diagnosed hypertension. Second, few studies of air pollution effects were based on repeated measures of resting blood pressure,[43] and repeated measure studies have either recruited a small number of participants (much smaller than 100) or collected a limited number of measures per individual ($n \approx 3$[22, 26]). Third, among repeated measure studies, extremely few have relied on ambulatory monitoring of blood pressure,[29, 41, 44] and none has examined both

resting and ambulatory blood pressure as we do (while each assessment may have its own strengths). Fourth, few studies have examined pulse pressure, central rather than brachial blood pressure (as more predictive of target organ damage and morbidity / mortality), and aortic pulse wave velocity or the augmentation index (as markers of arterial stiffness[45]) in relation to air pollutants.[23, 46] Finally, the air pollution and noise studies that compared participants with each other (rather than repeated measures with each other) have insufficiently controlled for confounding factors related to individual and environmental characteristics.[47]

Heart rate variability

Researchers focus on heart rate variability to investigate how the sympathetic and parasympathetic branches of the autonomous nervous system modulate heart rate. An alteration of the autonomous regulation of heart rate may be one of the pathophysiological mechanisms through which air pollution[48, 49] (as also confirmed by toxicological studies[50]) and noise[51, 52] increase cardiovascular mortality. Indeed, studies have shown that reduced heart rate variability may be associated with an increased incidence of myocardial infarction in the general population, and with a poor prognosis in heart disease patients.[53-55]

Air pollution effects: The strongest evidence for a relationship between an increased exposure to air pollutants and reduced heart rate variability has been reported for particulate matter.[43] A meta-analysis (18667 participants from 29 studies) of the relationship between particulate matter and heart rate variability suggests that an increased concentration of $PM_{2.5}$ is associated with a reduced heart rate variability, as demonstrated by indicators of both the time domain and the frequency domain.[56] Even if certain studies have reported stronger air pollution effects among people with cardiovascular diseases,[57] this meta-analysis did not observe that

the association became weaker when studies with cardiovascular disease patients were excluded. Studies have often measured air pollutant exposure over 24 hours. For example, a work that assessed the concentration of $PM_{2.5}$ from a fixed monitoring station reported that considering exposure windows of 1 hour to 4 hours did not yield a stronger association than an exposure window of 24 hours.[58] It should be noted, however, that certain studies did not identify associations between an increased exposure to air pollutants and reduced heart rate variability, or even reported associations in the opposite direction.[59-61]

Regarding air pollutants from traffic sources, a study of 28 elderly subjects reported that a high concentration of $PM_{2.5}$ (assessed from fixed monitoring stations) was associated with reduced heart rate variability, but that the concentration of black carbon (used as a marker of particles from road traffic) resulted in stronger associations and with a larger number of indicators of heart rate variability.[62] A high concentration of carbon monoxide (CO) was also associated with reduced heart rate variability, but the association with CO had entirely disappeared after adjustment for black carbon, a marker of particles from road traffic.

Noise effects: The literature on heart rate variability is scarcer for noise than for air pollution effects. A German study of 110 individuals (326 electrocardiogram recordings) observed that increases in sound pressure below 65 dB(A) were associated with changes in heart rate variability suggestive of an elevation in sympathetic tone and parasympathetic withdrawal, while elevations in sound pressure above 65 dB(A) were primarily associated with increased sympathetic activity.[51] Of relevance for the present project is a Chinese randomised crossover study of participants successively spending time in a traffic centre and in a park. These participants underwent personal monitoring of noise and traffic-related air pollutants ($PM_{2.5}$, CO, and black carbon).[52] The study found that higher noise levels were associated with reduced heart rate variability, resulting from an increased sympathetic activation and a

decreased parasympathetic modulation. It also reported that noise levels modified the relationships between air pollutants and heart rate variability.

Regarding limitations, first, previous repeated measure studies of heart rate variability have relied on small sample sizes. For example, of the 25 repeated measure studies identified in the aforementioned meta-analysis of air pollution effects,[56] one study included 100 participants, 3 studies between 50 and 100 participants, and 21 studies less than 50 participants. Second, a number of studies did not combine indicators of heart rate variability from both the time domain and the frequency domain, although certain did.[56, 63]

Web appendix 2: List of confounders for the regression analyses

On the basis of our precise literature review for each exposure–outcome relationship, we will take into account – to adjust for confounding or as modifying factors – the following variables into the models (varying or not over time; list of factors to be adapted to the exposure and health variables examined): demographic characteristics (age, sex, country of birth, cohabitation, etc.); socioeconomic characteristics (education, employment status, occupation, income, wealth, etc.); health characteristics (body mass index, waist circumference, heart rate,[51, 64] personal history of diseases, medication use,[22] etc.); health behaviour (physical activity and body posture assessed with accelerometry,[29, 51, 64] tobacco and alcohol consumption, etc.); contextual characteristics defined at the residence, at the different places visited over the observation period, and along trip itineraries (socioeconomic level, building density, population density, traffic density,[22, 24] etc.); detailed characteristics of the dwelling; temperature, relative humidity or apparent temperature,[23] and atmospheric pressure[58, 63]; estimated incidence of influenza or influenza-like illness[4]; pollen and mould in the air (French Aerobiology Network); hour, day of the week, and season of measurement; and conditions of measurement of blood pressure at rest.

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