Acute Ambulatory Blood Pressure Response to Short-Term Black Carbon Exposure: The MobiliSense Sensor-Based Study

Sanjeev Bista, Giovanna Fancello, Basile Chaix

Acute ambulatory blood pressure response to short-term black carbon exposure: the MobiliSense sensor-based study

Sanjeev Bista[1], Giovanna Fancello[1], Basile Chaix[1]

[1] Sorbonne Université, INSERM, Institut Pierre Louis d'Epidémiologie et de Santé Publique IPLESP, Nemesis team, Faculté de Médecine Saint-Antoine, 27 rue Chaligny, 75012 Paris, France

Corresponding author: Sanjeev Bista, UMR-S 1136, Faculté de Médecine Saint-Antoine, 27 rue Chaligny, 75012 Paris, France.

Tel: +33 (0)1 44 73 86 64. E-mail: sanjeev.bista@iplesp.upmc.fr

Abstract

Documented relationships between black carbon (BC) exposure and blood pressure (BP) have been inconsistent. Very few studies measured both BC exposure and ambulatory BP across the multiple daily environments visited in the general population, and none adjusted for personal noise exposure, a major confounder. Our study addresses these gaps by considering 245 adults living in the Grand Paris region. Personal exposure to BC was monitored for 2 days using AE51 microaethalometers. Ambulatory BP was measured every 30 minutes after waking up using Arteriograph 24 monitors (n = 6772). Mixed effect models with a random intercept at the individual level and time-autocorrelation structure adjusted for personal noise exposure were used to evaluate the associations between BC exposure (averaged from 5 minutes to 1 hour before each BP measurement) and BP. To increase the robustness of findings, we eliminated confounding by unmeasured time-invariant personal variables, by modelling the associations with fixed-effect models. All models were adjusted for potential confounders and short-term time trends. Results from mixed models show that a 1-$\mu g/m^3$ increase in 5-minute averaged BC exposure was associated with an increase of 0.57 mmHg in ambulatory systolic blood pressure (SBP) (95% CI: 0.30, 0.83) and with an increase of 0.36 mmHg in diastolic blood pressure (DBP) (95% CI: 0.14, 0.58). The slope of the exposure-response relationship gradually decreased for both SBP and DBP with the increase in the averaging period of BC exposure from 5 minutes to 1 hour preceding each BP measurement. Findings from the fixed-effect models were consistent with these results. There was no effect modification by noise in the associations, across all exposure windows. We found evidence of a relationship between BC exposure and acute increase in ambulatory SBP and DBP after adjustment for personal noise exposure, with potential implications for the development of adverse cardiovascular outcomes.

1. Introduction

Short and long-term exposure to particulate matter with an aerodynamic diameter ≤2.5 μm ($PM_{2.5}$) is a potential contributor to cardiovascular morbidity and mortality (1). Possibly, oxidative stress, vasomotor dysfunction, the altered autonomic function of the heart, induction of systemic inflammation, and endothelial dysfunction mediate these effects on cardiovascular outcomes (2,3). Furthermore, it is well established that short-term exposure to $PM_{2.5}$ (averaged over several hours or days) affects blood pressure (BP) (4–7), with well-known biological pathways (2,8,9). $PM_{2.5}$ exposure from vehicle emissions had a stronger effect on BP than $PM_{2.5}$ from other sources (10,11). Therefore, investigating the particular source of $PM_{2.5}$ accountable for the documented relationship is an important topic for researchers to explore. There is some evidence that black carbon (BC), as a component of $PM_{2.5}$, may be related to cardiovascular health, with its particular chemical composition and diameter (12–14). Evidence suggests that exposure to BC is associated with increased heart rate variability and peripheral arterial tone (15,16), thus may also cause the variability in BP.

Despite this preliminary evidence, results on the association between exposure to BC and BP remain mixed, with some studies reporting positive associations (17–22) and others reporting no association (23–31). In most studies, BC exposure levels were either derived from a land-use regression (20,26), or from the nearby fixed monitoring station (19,21,22,27,30), possibly introducing exposure misclassification at the individual level (32). Three of the studies using personal monitors analyzed the exposure-response association between 24-hour averaged BC exposure and BP measurements (23,24,31), ignoring the fine-grained variability in BC exposure strongly related to a person's time-activity patterns (32) and acute BC exposure before the BP measurement.

To our knowledge, only three of the existing studies used ambulatory BP measurements (18,25,28); however, they reported mixed findings. One of them reported that diastolic blood pressure (DBP) was negatively associated with 4-minutes averaged BC exposure among women during cooking hours (25); another documented a positive association between BC exposure and both systolic blood pressure (SBP) and DBP for averaging periods ranging from 6 to 10 hours among hospitalized patients with metabolic syndrome (18); whereas the third one did not identify any association among hospitalized elderly people with coronary disease (28). None of them was conducted on the general population. Overall, most of these past studies have been conducted outside Europe (17,18,29–31,33,19–22,25–28), making the generalizability of this literature to Europe limited due to a confluence of environmental, lifestyle and genetic discrepancies (4).

To address these gaps, our study estimated the relationship between the short-term personal exposure to BC and the acute ambulatory BP response among adults from a European city. It is believed that a large number of repeated episodes of acute increase in BP may lead to hypertension (34) and other cardiovascular events in the future (35), especially among the susceptible population (36). Moreover, some studies show that ambulatory measured BP is more closely related to organ damage than isolated measurements in clinical settings (37). Nevertheless, the ambulatory BP outcomes should cautiously be interpreted in relation to the concurrent physical activity levels, considering that the relationship between ambulatory BP and physical activity varies widely between individuals (38).

We hypothesized that short-term personal exposure to BC (averaged over the previous hour or less) would elevate the ambulatory BP outcomes, even after taking into account the levels of

physical activity, ambient temperature, relative humidity, and personal noise exposure. To our knowledge, none of the studies exploring the BC-BP association has adjusted for concurrent noise exposure, although the confounding of traffic-related air pollutant effects by noise has been highlighted previously (39).

2. Methods

2.1 Study population

The MobiliSense study was conducted in the Grand Paris (the Paris City and some surrounding municipalities) in France from May 2018 to October 2020 (40). Participants were recruited following a two-stage stratified random sampling procedure. Briefly, stage 1 involved the random selection of neighbourhoods in the first and last quartiles of neighbourhood road traffic density in each quartile of neighbourhood income. The second stage involved the random selection of dwelling units in the pre-selected neighborhoods from the 2013 and 2014 population censuses by the National Institute of Statistics and Economic Studies. Overall, 31970 dwellings were selected from 234 neighborhoods. Postal mails were sent twice to invite residents from the selected dwellings, and 289 participants were recruited in the sensor-based MobiliSense study. Our selection analysis shows that the probability of participating in our study was higher among older and married people, among French than non-French people, among those with a higher educational level, for people living in Paris and the close suburb than in the far suburb, and for people living in individual houses rather than apartments, which has been detailed in our previous publication (41).

2.2 Personal exposure to BC

Personal exposure to BC was measured with aethalometers (MicroAeth AE51, AethLabs, CA, USA) from day 1 to day 4 of our study. This device has already been used by multiple studies before (18,23,28,29,31). The device was strapped to participants' shoulders with the inlet of the tube at the level of the neck and set to measure 10-second averaged BC concentration within participants' breathing zone. The optical and electronic instrumental noise can result in an error in BC measurement (42). Therefore, the correction of the erroneous high and low values was done using the Optimized Noise Reduction Averaging (ONA) algorithm (42), taking into account filter change, based on the R package "BlackCarbon" version 1.1 developed by Bista 2020 (43). The details on the processing steps and algorithm have been provided in our previous publication (41). Briefly, filter changes were identified from a ΔATN (coefficient of light attenuation) of at least -5 between two successive data points and were manually verified. The ONA processed BC concentrations that remained negative for longer than 5-minute sequences or the concentration of BC that remained absolutely constant for more than 4 hours, although the person was switching between micro-environments, were deleted from the analysis. Furthermore, extremely high values of BC (i.e., BC values >15 $\mu g/m^3$ corresponding to 0.56% of all BC measurements) were also excluded from the ONA processed data, as they may be spikes related to when the monitor was exposed to mechanical shocks or intense vibrations as it may be the case during intense physical activity (42).

Personal exposure to BC was averaged in 5-minute to 1-hour time windows prior to each ambulatory BP measurement.

2.3 Accelerometer data

Participants carried a tri-axial accelerometer, ActiGraph wGT3X+ (44), on their waist throughout the study period. ActiGraph is one of the valid commercial accelerometers permitting to assess levels of physical activities (45). While processing the data, the low-frequency extension option was applied instead of the normal filter in Actilife 6.13.3, to make it more adapted to slow-moving people (46). A value for the 3-axes count of 0 for at least 1 hour with a spike tolerance of 2 minutes of non-zero epochs, a default setting in Actilife, was selected to identify the non-wear time of the device (47). Vector magnitude (VM), as an indicator of physical activity, was aggregated in similar time windows as BC exposure according to the following equation:

$$VM = \sqrt{Axis\ 1^2 + Axis\ 2^2 + Axis\ 3^2}$$

2.4 Contextual variables

The GPS data collected with a BT-Q1000XT GPS receiver throughout the study, at a 5-second resolution, were processed just after the data collection with algorithms using the TripBuilder Web mapping application (48). These inbuilt algorithms assisted us in a) identifying the places visited by the participants in the study period; b) decomposing the trips between two visited places into trip segments commuted with unique modes; c) imputing information on the nature of each visited place relying on the geolocated places regularly visited by participants pre-assessed with the VERITAS application and on a database of points of interest (49); and d) imputing information on the mode of transport used in each trip segment based on speed, survey information from participants about the use of typical modes, and on the availability of public transport stations of the particular mode at the start and end of the subjected trip segment. The detail of the algorithms is discussed in our previous publication (50). Using the resulting

information displayed in the TripBuilder application, the start and end time of each trip stage and related mode information as well as visited places and their nature over the study period were verified and eventually corrected with the participants during a phone mobility survey. This mobility survey also permitted to graphically complement the trips lacking correct GPS data. From the GPS-based mobility survey data, we created a detailed timetable of places visited and trips covering the whole study period.

The proportion of time spent in the home, out of the home, and in motorized and non-motorized transport (walking, biking, skateboarding, etc.) were calculated for 5-minute to 1-hour periods preceding each BP measurement, using the timestamped information on visited places and trip segments (with information on modes) from the detailed timetable.

2.5 Noise exposure

Based on the findings that noise is positively related to heart rate variability parameters (51), hypertension (52) and blood pressure (53) and highly correlated with air pollution for sharing common sources, especially road traffic (54–56), we considered personal exposure to noise as a potential confounder for this study. A previous study suggested that the association between traffic-related air pollutants and cardiovascular responses could be confounded by noise exposure (39). On days 3, 4, 5 and 6, the SV 104A dosimeter (Svantek, Warszawa, Poland) attached to the belt, with a microphone fixed at the collar near the participant's ear, was used for monitoring personal noise exposure every second. The measured noise level was in A-weighted decibels [dB(A)], to capture the sound that corresponds to human hearing. Similar to BC exposure, noise data were aggregated using the notion of equivalent sound level (Leq) in the corresponding time windows ranging from 5-minute to 1-hour averaging periods. Leq is

defined as the constant sound that would have been generated with the same level of energy compared to the noise that was perceived during the time frame, expressed as dB and computed as follows (51):

$$\boldsymbol{L_{eq}} = 10\log \times \frac{1}{T}\int_{0}^{T} 10^{\frac{L(t)}{10}}\, dt$$

Leq : equivalent sound level

L(t) : noise level at time t

T: period length in seconds

2.5.1 Noise prediction for day 1

Personal noise exposure was not measured on the 1st day of our study, therefore, observed data from days 3 and 4 were used in predicting the noise exposure for day 1. Because of a very large number of data points at a lower level, both noise data (every 1 second) and BC measurements (every 10 seconds) were averaged at the minute level. BC concentration, mode of transport, the combination of departure place and arrival place (when the person was travelling), visited place id and activity type (when the person was at a place), week vs. weekend, time of the day (morning, noon, evening, and night) defined at the minute level were used in predicting noise exposure (variables related to departure and arrival places of trips and visited place ids are wide sets of binary variables specific to each participant). These variables were created using the detailed mobility survey timetable discussed above. Because we had a large number of data points at the minute level (more than 460000 for all participants) in the training dataset (days 3 and 4), we randomly selected 60% of the observations per combination of all categorical variables and ran the random forest (57) algorithm with only 50 trees (see additional detail on the random forest algorithm in Appendix 1). However, all the observations were retained for

those combinations having less than five minutes in cumulative duration. The R^2 of the random forest algorithm used in predicting noise exposure for day 1 was 81%. In summary, we were able to predict day 1 noise exposure for 237 participants using the random forest algorithm using the information from days 3 and 4.

2.6 Ambulatory blood pressure

Arteriograph 24 ambulatory blood pressure monitor (TensioMed, Budapest, Hungary) was used on day 1 and day 3 to measure participants' blood pressure from wakeup to bedtime. The device was set to measure it every 30 minutes during the wear-time, in a user-independent way with a single upper arm cuff. Details on its functioning and invasive validity are explained elsewhere (58).

2.7 Meteorological measurements

Values for ambient temperature and relative humidity were obtained for all the geographic locations continuously recorded with GPS receivers and the mobility survey (as explained in Appendix 2) by matching their timestamps to those of the hourly measurements of the closest Météo-France (https://meteofrance.com) weather monitoring station. Participants' locations were not constant during the monitoring, resulting in a change in their nearby station as a function of time. Adjusting for the absolute temperature and humidity measured at each station may not be appropriate, since there are deltas in temperature across stations depending on their location (e.g., near the traffic, in a park, etc.). Thus, as a method of data standardization between different weather stations and in order to make them comparable, z-scores were calculated for each station's measurements (temperature and humidity) retaining only the measurements taken within the whole study period (May 2018 to October 2020). To minimize misclassification in

personal exposure to temperature and humidity, we ignored those points which were more than 15 km away from the nearest station. These z-scores of temperature and relative humidity were aggregated in the corresponding time windows prior to each BP measurement as the BC personal exposures to adjust for in the regression models.

2.8 Other covariates

Age in our analysis was coded as a continuous variable and sex as a binary variable (male vs. female). Monthly alcohol drinks from the questionnaire, and body mass index (BMI) from measured height and weight were coded continuously. Educational attainment was coded into three categories, lower than Baccalauréat, equal to Baccalauréat, and higher than Baccalauréat. The employment status of participants was classified into five classes; retired, stable job, unstable job, unemployment, and other residual categories. Annual household income was divided by the number of units in the household, considering members younger than 14 years old as 0.5 units. Household income per consumption unit was divided into three categories based on tertiles. Individuals without income information were coded as missing (N = 6) so that we could retain all the observations for the main analysis. To control for neighbourhood socioeconomic effect on BP, the average of winsorised living standards (source: Insee, 2019) in both the residential neighbourhood and the neighbourhood where participants were when each BP measurement was taken were adjusted for in the analysis. Place of residence was further assessed through the distinction between Paris, close suburbs, and far suburbs.

2.9 Statistical analysis

2.9.1 Analytical sample

Non-wear time was verified for all devices and all participants via a paper diary and during a phone call after the period of observation. Periods of non-wear were deleted before the final processing. Reasons for excluding BP measurements from the present analysis (summarized in Figure 1) included the failure of any of the devices monitoring BC, noise, accelerometry, and BP or not taking part in the mobility survey on day 1 and/or day 3, non-wear of any device mentioned above, and closest meteorological station being 15 km away from participant's location. Overall, 6772 observations from 245 participants were retained for analysis.

2.9.2 Analyses

Linear mixed models were used to examine the temporal structure of the relationship between BC exposure averaged over the previous 5 minutes to 1 hour and ambulatory BP measurements. To account for the within-subject correlation of repeated outcome measures, a random intercept at the individual level was specified in all the models. A first-order autoregressive structure AR(1) was applied to account for temporal autocorrelation among residual errors within individuals (59). Short-term time trends over the day were accounted for with smoothing splines estimated for each individual using the lmeSplines package in R (60). All the models controlled for noise exposure, outdoor temperature and humidity related to the GPS track, physical activity (accelerometer vector magnitude), contextual variables (proportion of time being at home, in motorized vehicles or non-motorized transport), age, sex, monthly alcohol consumption, BMI, education attainment, employment status, the living standard of the residential neighborhoods and neighborhoods where BP was measured, and weekdays vs. weekend. The accelerometry, meteo variables, and contextual variables were considered in the same time window as BC exposure (from 5 minutes to 1 hour).

In a second analysis to increase the robustness of findings, we eliminated confounding by unmeasured time-invariant personal level variables, by modelling the associations with fixed-effect models (estimating effects within individuals) that control for one dummy variable related to each participant. Short-term time trends in the fixed-effect models were taken into account with 6-degree natural cubic splines (governed by Akaike Information Criterion, AIC), and only time-varying variables were adjusted for in the models.

Non-linear associations between short-term exposure to BC and BP were estimated in mixed models with 2nd to 10th-degree polynomials and natural cubic splines for each BC exposure window separately, keeping other aspects constant. We selected smoothers with 6-degree polynomial splines as these provided models with the lowest AIC. In separate models, piecewise linear regressions were estimated in order to obtain interpretable coefficients for the BC-BP association in two separate portions of the BC continuum. We selected the cutoff in BC for the two portions of the curve in the piecewise model that minimized the AIC for each BC exposure window. Furthermore, we conducted a sensitivity analysis by excluding 21 participants who reported being diagnosed as hypertensive by their physician. Finally, as another sensitivity analysis, considering that ambulatory blood pressure was collected only on day 1 and day 3, we examined the association between BC exposure and BP separately on day 1 and day 3 (stratification). Interaction between BC and noise exposure on BP has not been reported previously, however, a study documented that the effect of BC on heart rate variability was amplified at high noise levels (61). For the first time, as a sensitivity analysis, we assessed the BC effect modification by noise by adding a multiplicative interaction term of BC and noise exposure into the linear mixed-effect models. Since day 1 noise exposure was predicted, we tested this effect modification only for day 3 observations.

3. Results

3.1 Characteristics of participants

As shown in Table 1, among 245 participants, there were 56% of women. Participants had a mean age of 50 years (range: 33, 67 years). Twenty-six per cent were living in Paris and the rest in the suburbs; 68% had a higher diploma than Baccalauréat, while 6% had lower educational attainment than Baccalauréat; 66% of participants had a permanent job, 3% were unemployed and 12% were retired.

Table 1 also indicates the median value of the average exposure to BC in short periods preceding each BP measurement. On average, each individual contributed 28 BP measurements (range: 2, 59) over the study. The mean number of BP measurements per person per day was 14 (range: 2, 29). Of 245 participants, 21 were involved only on the 1st day and 8 were engaged only on the 3rd day of the study. The average ambulatory SBP and DBP among participants were 125.98 mmHg (SD: 18.00) and 71.42 mmHg (SD: 14.91) respectively.

3.2 Assessment of assumptions in regression models

The temporal autocorrelation coefficient (phi) for the residuals of the mixed effect model ranged from 0.34 to 0.37 for the models with SBP and from 0.23 to 0.25 for the models with DBP, justifying the need of taking into account the time autoregressive error structure within participants. The individual-level random effect residuals were normally distributed. The variance inflation factor was below 4.5 for the covariates in all the models.

3.3 Associations between air pollution and blood pressure

3.3.1 Mixed-effects models: linear associations

Estimates for the associations of BC exposure with BP from linear mixed models are reported in Table 2. A 1 $\mu g/m^3$ increase in BC exposure averaged over the preceding 5 minutes was associated with an increase of 0.57 mmHg in SBP (95% CI: 0.30, 0.83) and an increase of 0.36 mmHg in DBP (95% CI: 0.14, 0.58). The slope of the exposure-response relationship gradually decreased for both SBP and DBP with the increase in the size of the averaging period of BC exposure from 5 minutes to 1 hour preceding each BP measurement.

3.3.2 Fixed-effect models

Similar to the results from mixed models, we observed that a 1-$\mu g/m^3$ increase in BC exposure averaged over the 5 previous minutes resulted in the greatest changes of both SBP (0.42 mmHg; 95% CI: 0.17, 0.67) and DBP (0.31 mmHg; 95% CI: 0.09, 0.53), among all the exposure averaging windows (Table 2). As in the mixed models, the magnitude of the association gradually decreased for SBP when the size of the window for averaging BC exposure increased from 5 minutes to 1 hour before each BP measurement. Consistently with the mixed models, the effect estimates for the association between BC exposure and DBP were similar for 5-minute and 15-minute averaging periods and gradually decreased for longer averaging periods.

3.3.3 Non-linear associations and piecewise regression

Figures 2 and 3 represent the non-linear associations between BC exposure (averaged from 5 minutes to 1 hour prior to each BP measurement) and the ambulatory BP measurements. Considering these non-linear associations and the models that minimized AIC, a cutoff point of

5 μg/m^3 was selected for 5-minute averaged BC exposure and 4 μg/m^3 for 10-minute and 15-minute averaged BC exposures in the piecewise regressions. Results from the piecewise regressions in Figures 4 and 5 indicate that when the BC exposure was less than 5 μg/m^3, a unit increase in BC exposure during the previous 5 minutes was associated with a 0.99 mmHg increase in SBP (95%CI: 0.62, 1.36) and 0.51 mmHg increase in DBP (95% CI: 0.21, 0.81). When the BC exposure was above the cutoff point, 5-minute averaged BC exposure was no longer associated with both SBP and DBP. Similar trends were observed for both 10 and 15-minute averaged BC exposure: they were associated with SBP and DBP only below the cutoff value of 4 μg/m^3.

3.4 Sensitivity analysis

The effect estimates from the models excluding 21 hypertensive people (Table 3) were not very different in magnitude compared to the models including all the participants (Table 2).

On the first day of observation, BC exposure during the previous 5 minutes was associated neither with SBP [effect estimate for a 1 μg/m^3 increase in BC exposure: 0.10 mmHg (95% CI: -0.37, 0.57)] nor with DBP [-0.16 (95% CI: -0.55, 0.23)], whereas on the third day it was documented that a 1 μg/m^3 increase in average BC exposure during the previous 5 minutes lead to a 0.54 mmHg increase in SBP (95% CI: 0.21, 0.87) and a 0.37 mmHg increase in DBP (95% CI: 0.10, 0.64). We believe that this discrepancy between day 1 and day 3 may be attributable to the differences related to these days. On day 3, 84% of the 5-minutes observations preceding the BP measurements were from weekdays, whereas on day 1 it was just 49%. Given that day 1 was much more often during weekends, the percentage of previous 5-minute periods that were spent totally at home was higher for that day (79%), as compared to day 3 (48%). Thus there

were perhaps not enough observations out of home on day 1 (N = 667 vs. N = 1853 on day 3) for the effect of BC on BP to be detected. In coherence with this pattern, 4% of the 5-minute exposure windows were fully spent in motorized vehicles on day 3 vs. only 1% on day 1. Details on differences between day 1 and day 3 in terms of BC, BP, and other time-varying observations are presented in Appendix 3.

The interaction term between BC and noise exposure (p-value > 0.05) did not show evidence of BC effect modification by noise exposure in any of the exposure windows.

4. Discussion

Recently, BC has been of increased interest in public health research as a marker of combustion-related air pollutants, as it is independently associated with health effects beyond those resulting from background $PM_{2.5}$ (62). Our results illustrate the potential effect of short-term personal exposure to BC on ambulatory BP. One $\mu g/m^3$ increase in BC exposure had the greatest estimated effect on BP when averaged over the previous 5 minutes compared to other exposure averaging windows, with an increase of 0.57 mmHg in SBP (95% CI: 0.30, 0.83) and an increase of 0.36 mmHg in DBP (95% CI: 0.14, 0.58). The magnitude of the exposure-response relationship gradually decreased for both SBP and DBP with the increase in the averaging period of BC exposure from 5 minutes to 1 hour preceding each BP measurement. When considering 30-minute exposure windows and longer, no association could be detected, suggesting a very acute and immediate blood pressure response. Findings from our study are consistent with the results from controlled human exposure (63) and semi-experimental studies (64), that higher exposure to traffic-related air pollutants has an increasing effect on BP within minutes of exposure. To our knowledge, only a single study to date, done in rural India,

explored the dose-response relationship between BC exposure over the previous 5 minutes and ambulatory BP (25). Although the methodology applied was consistent with our study, the findings were discrepant. An interquartile increase in previous 5-minute BC exposure decreased SBP by 0.1 mmHg (95% CI: -2.00, +1.90) and DBP by 0.80 mmHg (95% CI: -1.70, +0.10) (25). Similar to our work, studies conducted in a Chinese city and Los Angeles did not report associations between 1-hour averaged BC exposure and SBP or DBP (18,28). Likewise, a recent study in New Orleans documented no association between a 1 $\mu g/m^3$ increase in one hour of in-house BC exposure and clinically measured SBP (3.61 mmHg; 95% CI: -0.52, +7.76) (33) (which may be coherent with the absence of association in our study on day 1 when participants spent more time at home). A case-crossover study conducted among 30 healthy adult runners reported no association between a 1 $\mu g/m^3$ increase in 30-minute BC exposure and clinical BP [SBP: 1.67 mmHg (95% CI: −0.32, +3.65) and DBP: 1.16 mmHg (95% CI: −0.79, +3.12)], in a single pollutant model (17). Although with smaller point estimates, our result for the 30-minute time window is consistent with this study.

Other previous studies examining the association between BC exposure and clinical BP measurements investigated larger time windows for exposure than our study considering 1-hour exposure window at the most (19,20,31,21–24,26,27,29,30). These studies yielded mixed conclusions. A Canadian study averaging personal BC exposure over 3 hours documented no association between BC exposure and both SBP and DBP (29). A study reported positive associations between 24-hour averaged BC exposure and SBP and DBP (19), whereas others with the same averaging window reported null associations (20,23,24,27,31). A study in Boston found positive associations between BC exposure averaged over 5 days and SBP and DBP but reported that BC exposure was not associated with the outcomes when averaged for shorter time windows (30). Two studies documented positive associations of 7-day averaged BC

exposure with both SBP and DBP (20,21). Finally, two studies examined the long-term association of BC personal exposure with BP but had conflicting findings: one reported positive associations between annual BC exposure and SBP and DBP (26), whereas the other reported null associations with the same time patterns of exposure (20).

A range of factors may explain discrepancies in results between our and other previous studies. A first and foremost possible explanation is that we assessed associations between very short-term exposure to BC and ambulatory BP, in contrast to most other studies exploring effects after days, weeks and years of BC exposure on clinically measured BP (19,21,22,26,29–31). The only study exploring the dose-response association between previous 5-minute BC exposure and ambulatory BP monitored the BC exposure during cooking sessions using biomass (25), an uncommon source in our study area. It is well accepted that air pollutants from urban sources such as motor engines may produce distinct health effects than the air pollutants generated from incomplete biomass combustion (65). A second reason might be that the toxicological profile of BC widely depends on individual susceptibility, which may vary between populations (1). Third, our study was conducted among the healthy general public in their daily settings, whereas other studies were either only among women during cooking sessions (25), among runners during running sessions (17), with patients hospitalized with metabolic syndrome (18), or among older people (>65 years old) with coronary heart disease (28). Fourth, differences in the exposure range may explain the inconsistencies observed: in some other studies, the average personal exposure to BC was very high, e.g., 5.4 $\mu g/m^3$ (17), 5.08 $\mu g/m^3$ (18) or 40 $\mu g/m^3$ (geometric mean) (25), making the dose-response curve incomparable to our study where the mean BC exposure was not greater than 1.5 $\mu g/m^3$ across all exposure averaging periods. Fifth, in contrast to other studies, using ambient BC concentration from nearby monitoring stations (21,22,27,30) or land-use regression (20,26), we

used personally monitored BC exposure while establishing the dose-response relationship. There is evidence for the lack of agreement in exposure between station-measured air pollutants and personally monitored air pollutants (66). Some studies have highlighted that personal exposure to air pollutants may induce different responses in humans than background ambient levels due to variation in sources and chemical composition (67–69). In summary, differences in study design, period of exposure, methods used in exposure measurement, use of ambulatory BP vs. isolated clinical BP and study population make it hard to compare our findings with other studies.

Our results support the biological plausibility of the harmful effect of traffic-related particles on cardiovascular outcomes. Our main findings are compatible with the idea that acute changes in BP could play a role in explaining the existing relationship between short-term particle exposure and the onset of cardiovascular events (70,71), and the relationship between long-term exposure and the incidence of hypertension (72,73). Scientists have proposed several physiological mechanisms for the short-term and long-term effects of inhaled particles on BP (1,11,74,75). Regarding long-term effects, frequently repeated acute increases in BP may lead to hypertension (34) and other adverse cardiovascular conditions in the future (35). However, it is hard to empirically confirm that the acute cardiovascular responses experienced within a few minutes or hours after the pollution exposure and the chronic effects observed after several months or years of exposure share the same biological pathway (75), and that the latter is caused by the former. In order to further understand the mechanism underlying the relationship between BC and BP at different temporal structures, a study accessing both acute and chronic changes in BP over a longer period as a function of air pollutant exposure is needed, as we do with the first and second waves of MobiliSense.

4.1 Strengths and limitations

The strengths of our study include the use of personal monitors capturing both BC exposure and the time-activity patterns of participants (GPS receivers complemented by a mobility survey). Our sensor-based tracking approach is appropriate for studying air pollutant-induced health effects, reducing exposure misclassification. In fact, the use of personal exposure monitors, in contrast to extracting pollution data from stationary monitoring stations, is crucial for measuring pollutants with high spatial and temporal variability, as exposure misclassification leads to an underestimation of the association between combustion-related air pollutants and cardiovascular responses (76). The combination of air pollutant monitor, GPS receiver, and mobility survey enabled us to monitor participants in their real-life activity settings rather than in only a few preselected itineraries as in a controlled experimental study design. Another strength of our study is having an average of 28 BP measurements per person among a large number of participants (N = 245). Furthermore, in addition to controlling for time-varying confounders, the inclusion of fixed-effect analysis in our study increased the robustness of our findings, eliminating the confounding by unmeasured time-invariant personal level variables, for example, related to personal behavior.

Our study is the first to adjust for noise exposure while assessing the association between BC and BP, considering the large potential for noise to be a confounder due to road traffic (39). Also, our study is the only one assessing the dose-response relationship of BC with ambulatory BP measurements while averaging the personal exposure into small time windows (e.g., 5 minutes preceding each BP measurement) in an adult population observed in general settings.

Regarding limitations, as noise exposure on the first day of our study was predicted using random forest algorithms, there might still be residual confounding by noise in our results. However, the variability explained by our noise prediction model was likely high enough (81%) to capture the distribution of noise exposure. We expect that the non-differential misclassification of noise on 1st day may lead to an underestimation of the association of noise with blood pressure, and thus to an overestimation of the association between BC and BP; however, we did not observe an association between BC and noise in day 1 due to the particular nature of day 1, so we do not expect residual confounding by noise to be an important issue.

Since day 1 of our study was different in nature and had predicted noise exposure, we had to limit our sample to day 3 observations only when testing the BC effect modification by noise. Future sensor-based studies with a larger number of repeated measurements for a larger number of participants will have to confirm whether personal noise exposure modifies the association between air pollutant exposure and ambulatory BP or not. More broadly, the fact that day 1 of the study was different in nature than day 3, with day 1 being more frequently a weekend day, with participants more often at home and less often on a motorized trip, may be seen as a limitation of this study. Being at home has a major effect on BC exposure, as the sources of BC widely differ between micro-environments and geographical settings (e.g., lower exposure to vehicle emissions while being at home), implying differences in chemical composition and toxicology. It is well documented that exposure related to vehicle emissions has a stronger effect on BP than exposure from home-based sources (10,11).

Finally, our sample had a slight positive bias towards older people, urban citizens, and higher socioeconomic classes. Although we have adjusted for these factors in our final analyses with a sufficient number of observations in each subgroup (Table 1), our findings should be

generalized cautiously. It should be considered that by design we only recruited older adults (35-64 years) and focused on the metropolitan area of Paris which is specific to some extent.

5. Conclusion

This study stands as a first of its kind to access acute vascular impacts of exposure to BC in an adult population in all kinds of daily life settings considering participants' time-activity patterns and personal exposure. We documented some evidence of an association between exposure to BC and acute increases in BP in the Grand Paris region, which may have implications for the development of cardiovascular diseases. We found that the magnitude of this association between BC and BP decreased when BC exposure was averaged over longer time periods preceding each BP measurement. Our findings, therefore, suggest that changes in BP may be observed within a short time in response to exposure to air pollution, possibly implicating an autonomic nervous system response. These results highlight the importance of reducing anthropogenic environmental air pollution in urban settings in order to promote cardiovascular health among the adult population.

CRediT authorship contribution statement

Sanjeev Bista: Data processing, formal analysis, software, drafted the manuscript, methodology, interpreted the results and wrote the original draft. **Giovanna Fancello:** Methodology, software. **Basile Chaix:** Project conception, methodology, development of overall research plan, study oversight, funding acquisition, writing – review & editing and supervision.

Corresponding author: Sanjeev Bista

Declaration of competing interest

The authors have no relevant financial or non-financial interests to disclose.

Acknowledgement

This work was supported by the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation program (grant agreement 647000, 2014 ERC Consolidator grant, MobiliSense project). The funding institution had no role in the design of the study and collection, analysis, and interpretation of data and in writing the manuscript.

References

1. Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux A V., et al. Particulate Matter Air Pollution and Cardiovascular Disease. Circulation [Internet]. 2010 Jun;121(21):2331–78. Available from: https://www.ahajournals.org/doi/10.1161/CIR.0b013e3181dbece1

2. Mills NL, Donaldson K, Hadoke PW, Boon NA, MacNee W, Cassee FR, et al. Adverse cardiovascular effects of air pollution. Nat Clin Pract Cardiovasc Med [Internet]. 2009 Jan 25;6(1):36–44. Available from: http://www.nature.com/articles/ncpcardio1399

3. Mills NL, Törnqvist H, Robinson SD, Gonzalez MC, Söderberg S, Sandström T, et al. Air Pollution and Atherothrombosis. Inhal Toxicol [Internet]. 2007 Jan 20;19(sup1):81–9. Available from: http://www.tandfonline.com/doi/full/10.1080/08958370701495170

4. Yang B-Y, Qian Z, Howard SW, Vaughn MG, Fan S-J, Liu K-K, et al. Global association between ambient air pollution and blood pressure: A systematic review and meta-analysis. Environ Pollut [Internet]. 2018 Apr;235:576–88. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0269749117338290

5. Wing S, Horton RA, Rose KM. Air Pollution from Industrial Swine Operations and Blood Pressure of Neighboring Residents. Environ Health Perspect [Internet]. 2013 Jan;121(1):92–6. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.1205109

6. Szyszkowicz M, Rowe BH, Brook RD. Even Low Levels of Ambient Air Pollutants Are Associated With Increased Emergency Department Visits for Hypertension. Can J Cardiol [Internet]. 2012 May;28(3):360–6. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0828282X11004429

7. Baumgartner J, Schauer JJ, Ezzati M, Lu L, Cheng C, Patz JA, et al. Indoor Air Pollution and Blood Pressure in Adult Women Living in Rural China. Environ Health Perspect [Internet]. 2011 Oct;119(10):1390–5. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.1003371

8. Levy BI, Ambrosio G, Pries AR, Struijker-Boudier HAJ. Microcirculation in Hypertension. Circulation [Internet]. 2001 Aug 7;104(6):735–40. Available from: https://www.ahajournals.org/doi/10.1161/hc3101.091158

9. Louwies T, Panis LI, Kicinski M, De Boever P, Nawrot TS. Retinal Microvascular Responses to Short-Term Changes in Particulate Air Pollution in Healthy Adults. Environ Health Perspect [Internet]. 2013 Sep;121(9):1011–6. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.1205721

10. Auchincloss AH, Diez Roux A V., Dvonch JT, Brown PL, Barr RG, Daviglus ML, et al. Associations between Recent Exposure to Ambient Fine Particulate Matter and Blood Pressure in the Multi-Ethnic Study of Atherosclerosis (MESA). Environ Health Perspect [Internet]. 2008 Apr;116(4):486–91. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.10899

11. Brook RD, Urch B, Dvonch JT, Bard RL, Speck M, Keeler G, et al. Insights Into the Mechanisms and Mediators of the Effects of Air Pollution Exposure on Blood Pressure and Vascular Function in Healthy Humans. Hypertension [Internet]. 2009 Sep 1 [cited 2021 Jul 30];54(3):659–67. Available from: https://www.ahajournals.org/doi/abs/10.1161/HYPERTENSIONAHA.109.130237

12. Magalhaes S, Baumgartner J, Weichenthal S. Impacts of exposure to black carbon, elemental carbon, and ultrafine particles from indoor and outdoor sources on blood pressure in adults: A review of epidemiological evidence. Environ Res [Internet]. 2018 Feb;161:345–53. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935117310289

13. Giorgini P, Di Giosia P, Grassi D, Rubenfire M, D. Brook R, Ferri C. Air Pollution Exposure and Blood Pressure: An Updated Review of the Literature. Curr Pharm Des [Internet]. 2015 Dec 21;22(1):28–51. Available from: http://www.eurekaselect.com/openurl/content.php?genre=article&issn=1381-6128&volume=22&issue=1&spage=28

14. Luben TJ, Nichols JL, Dutton SJ, Kirrane E, Owens EO, Datko-Williams L, et al. A systematic

review of cardiovascular emergency department visits, hospital admissions and mortality associated with ambient black carbon. Environ Int [Internet]. 2017 Oct;107:154–62. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0160412017300843

15. Bind M-A, Peters A, Koutrakis P, Coull B, Vokonas P, Schwartz J. Quantile Regression Analysis of the Distributional Effects of Air Pollution on Blood Pressure, Heart Rate Variability, Blood Lipids, and Biomarkers of Inflammation in Elderly American Men: The Normative Aging Study. Environ Health Perspect [Internet]. 2016 Aug;124(8):1189–98. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.1510044

16. Huang J, Deng F, Wu S, Lu H, Hao Y, Guo X. The impacts of short-term exposure to noise and traffic-related air pollution on heart rate variability in young healthy adults. J Expo Sci Environ Epidemiol 2013 235 [Internet]. 2013 Apr 17 [cited 2021 Jul 28];23(5):559–64. Available from: https://www.nature.com/articles/jes201321

17. Pun VC, Ho K. Blood pressure and pulmonary health effects of ozone and black carbon exposure in young adult runners. Sci Total Environ [Internet]. 2019 Mar;657:1–6. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0048969718348204

18. Zhao X, Sun Z, Ruan Y, Yan J, Mukherjee B, Yang F, et al. Personal Black Carbon Exposure Influences Ambulatory Blood Pressure. Hypertension [Internet]. 2014 Apr;63(4):871–7. Available from: https://www.ahajournals.org/doi/10.1161/HYPERTENSIONAHA.113.02588

19. Baumgartner J, Zhang Y, Schauer JJ, Huang W, Wang Y, Ezzati M. Highway proximity and black carbon from cookstoves as a risk factor for higher blood pressure in rural China. Proc Natl Acad Sci [Internet]. 2014 Sep 9;111(36):13229–34. Available from: http://www.pnas.org/cgi/doi/10.1073/pnas.1317176111

20. Schwartz J, Alexeeff SE, Mordukhovich I, Gryparis A, Vokonas P, Suh H, et al. Association between long-term exposure to traffic particles and blood pressure in the Veterans Administration Normative Aging Study. Occup Environ Med [Internet]. 2012 Jun;69(6):422–7. Available from: https://oem.bmj.com/lookup/doi/10.1136/oemed-2011-100268

21. Wilker EH, Baccarelli A, Suh H, Vokonas P, Wright RO, Schwartz J. Black Carbon Exposures, Blood Pressure, and Interactions with Single Nucleotide Polymorphisms in MicroRNA Processing Genes. Environ Health Perspect [Internet]. 2010 Jul;118(7):943–8. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.0901440

22. Mordukhovich I, Wilker E, Suh H, Wright R, Sparrow D, Vokonas PS, et al. Black Carbon Exposure, Oxidative Stress Genes, and Blood Pressure in a Repeated-Measures Study. Environ Health Perspect [Internet]. 2009 Nov;117(11):1767–72. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.0900591

23. Louwies T, Nawrot T, Cox B, Dons E, Penders J, Provost E, et al. Blood pressure changes in association with black carbon exposure in a panel of healthy adults are independent of retinal microcirculation. Environ Int [Internet]. 2015 Feb;75:81–6. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0160412014003286

24. Young BN, Clark ML, Rajkumar S, Benka-Coker ML, Bachand A, Brook RD, et al. Exposure to household air pollution from biomass cookstoves and blood pressure among women in rural Honduras: A cross-sectional study. Indoor Air [Internet]. 2019 Jan 15;29(1):130–42. Available from: https://onlinelibrary.wiley.com/doi/10.1111/ina.12507

25. Norris C, Goldberg MS, Marshall JD, Valois M-F, Pradeep T, Narayanswamy M, et al. A panel study of the acute effects of personal exposure to household air pollution on ambulatory blood pressure in rural Indian women. Environ Res [Internet]. 2016 May;147:331–42. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935116300640

26. Curto A, Wellenius GA, Milà C, Sanchez M, Ranzani O, Marshall JD, et al. Ambient Particulate Air Pollution and Blood Pressure in Peri-urban India. Epidemiology [Internet]. 2019 Jul;30(4):492–500. Available from: https://journals.lww.com/00001648-201907000-00005

27. Jansen KL, Larson T V., Koenig JQ, Mar TF, Fields C, Stewart J, et al. Associations between Health Effects and Particulate Matter and Black Carbon in Subjects with Respiratory Disease. Environ Health Perspect [Internet]. 2005 Dec;113(12):1741–6. Available from:

https://ehp.niehs.nih.gov/doi/10.1289/ehp.8153

28. Delfino RJ, Tjoa T, Gillen DL, Staimer N, Polidori A, Arhami M, et al. Traffic-related Air Pollution and Blood Pressure in Elderly Subjects With Coronary Artery Disease. Epidemiology [Internet]. 2010 May;21(3):396–404. Available from: https://journals.lww.com/00001648-201005000-00019

29. Weichenthal S, Hatzopoulou M, Goldberg MS. Exposure to traffic-related air pollution during physical activity and acute changes in blood pressure, autonomic and micro-vascular function in women: a cross-over study. Part Fibre Toxicol [Internet]. 2014 Dec 9;11(1):70. Available from: http://particleandfibretoxicology.biomedcentral.com/articles/10.1186/s12989-014-0070-4

30. Hoffmann B, Luttmann-Gibson H, Cohen A, Zanobetti A, de Souza C, Foley C, et al. Opposing Effects of Particle Pollution, Ozone, and Ambient Temperature on Arterial Blood Pressure. Environ Health Perspect [Internet]. 2012 Feb;120(2):241–6. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.1103647

31. Mirowsky JE, Peltier RE, Lippmann M, Thurston G, Chen L-C, Neas L, et al. Repeated measures of inflammation, blood pressure, and heart rate variability associated with traffic exposures in healthy adults. Environ Heal [Internet]. 2015 Dec 15;14(1):66. Available from: http://ehjournal.biomedcentral.com/articles/10.1186/s12940-015-0049-0

32. Dons E, Int Panis L, Van Poppel M, Theunis J, Willems H, Torfs R, et al. Impact of time–activity patterns on personal exposure to black carbon. Atmos Environ [Internet]. 2011 Jul;45(21):3594–602. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1352231011003359

33. Rabito FA, Yang Q, Zhang H, Werthmann D, Shankar A, Chillrud S. The association between short-term residential black carbon concentration on blood pressure in a general population sample. Indoor Air [Internet]. 2020 Jul 18;30(4):767–75. Available from: https://onlinelibrary.wiley.com/doi/10.1111/ina.12651

34. Timothy DJ, Kannan S, Schulz AJ, Keeler GJ, Mentz G, House J, et al. Acute Effects of Ambient Particulate Matter on Blood Pressure: Differential Effectsacross Urban Communities. Hypertension [Internet]. 2009 May 1 [cited 2021 Nov 10];53(5):853. Available from: /pmc/articles/PMC3593813/

35. Clement DL, De Buyzere ML, De Bacquer DA, de Leeuw PW, Duprez DA, Fagard RH, et al. Prognostic Value of Ambulatory Blood-Pressure Recordings in Patients with Treated Hypertension. N Engl J Med [Internet]. 2003 Jun 12 [cited 2021 Nov 10];348(24):2407–15. Available from: https://pubmed.ncbi.nlm.nih.gov/12802026/

36. Tofler GH, Muller JE. Triggering of acute cardiovascular disease and potential preventive strategies. Circulation [Internet]. 2006 Oct [cited 2021 Nov 10];114(17):1863–72. Available from: https://pubmed.ncbi.nlm.nih.gov/17060396/

37. Mancia G, Parati G. Ambulatory Blood Pressure Monitoring and Organ Damage. Hypertension [Internet]. 2000 Nov;36(5):894–900. Available from: https://www.ahajournals.org/doi/10.1161/01.HYP.36.5.894

38. Leary AC, Donnan PT, MacDonald TM, Murphy MB. The influence of physical activity on the variability of ambulatory blood pressure. Am J Hypertens [Internet]. 2000 Oct 1;13(10):1067–73. Available from: https://academic.oup.com/ajh/article-lookup/doi/10.1016/S0895-7061(00)01186-9

39. Allen RW, Adar SD. Are both air pollution and noise driving adverse cardiovascular health effects from motor vehicles? Environ Res [Internet]. 2011 Jan;111(1):184–5. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935110001866

40. Chaix B, Bista S, Wang L, Benmarhnia T, Dureau C, Duncan DT. MobiliSense cohort study protocol: do air pollution and noise exposure related to transport behaviour have short-term and longer-term health effects in Paris, France? BMJ Open [Internet]. 2022 Mar 31;12(3):e048706. Available from: https://bmjopen.bmj.com/lookup/doi/10.1136/bmjopen-2021-048706

41. Bista S, Dureau C, Chaix B. Personal exposure to concentrations and inhalation of black carbon according to transport mode use: The MobiliSense sensor-based study. Environ Int [Internet]. 2022 Jan;158:106990. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0160412021006152

42. Hagler GSW, Yelverton TLB, Vedantham R, Hansen ADA, Turner JR. Post-processing Method to Reduce Noise while Preserving High Time Resolution in Aethalometer Real-time Black Carbon Data. Aerosol Air Qual Res [Internet]. 2011 Oct [cited 2020 May 3];11(5):539–46. Available from: http://www.aaqr.org/doi/10.4209/aaqr.2011.05.0055

43. Bista S. BlackCarbon: Processing Raw Black Carbon Concentration. 2020;R package. Available from: https://cran.r-project.org/package=BlackCarbon

44. ActiGraph Corporation 2013. ActiGraph wGT3X-BT user manual [Internet]. Available from: http://actigraphcorp.com/wp-content/uploads/2015/04/wGT3X-BT_UsersManual_RevD_Q3_2014.pdf

45. Metcalf BS, Curnow JSH, Evans C, Voss LD, Wilkin TJ. Technical reliability of the CSA activity monitor: The EarlyBird Study. Med Sci Sports Exerc [Internet]. 2002 [cited 2020 Aug 7];34(9):1533–7. Available from: https://pubmed.ncbi.nlm.nih.gov/12218751/

46. WANNER M, MARTIN BW, MEIER F, PROBST-HENSCH N, KRIEMLER S. Effects of Filter Choice in GT3X Accelerometer Assessments of Free-Living Activity. Med Sci Sport Exerc [Internet]. 2013 Jan [cited 2020 Aug 5];45(1):170–7. Available from: https://pubmed.ncbi.nlm.nih.gov/22895373/

47. TROIANO RP, BERRIGAN D, DODD KW, MÂSSE LC, TILERT T, MCDOWELL M. Physical Activity in the United States Measured by Accelerometer. Med Sci Sport Exerc [Internet]. 2008 Jan;40(1):181–8. Available from: http://journals.lww.com/00005768-200801000-00025

48. Oliveira MGS, Vovsha P, Wolf J, Birotker Y, Givon D, Paasche J. Global Positioning System–

Assisted Prompted Recall Household Travel Survey to Support Development of Advanced Travel Model in Jerusalem, Israel. Transp Res Rec J Transp Res Board [Internet]. 2011 Jan;2246(1):16–23. Available from: http://journals.sagepub.com/doi/10.3141/2246-03

49. Chaix B, Kestens Y, Perchoux C, Karusisi N, Merlo J, Labadi K. An Interactive Mapping Tool to Assess Individual Mobility Patterns in Neighborhood Studies. Am J Prev Med [Internet]. 2012 Oct;43(4):440–50. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0749379712004667

50. Chaix B, Benmarhnia T, Kestens Y, Brondeel R, Perchoux C, Gerber P, et al. Combining sensor tracking with a GPS-based mobility survey to better measure physical activity in trips: public transport generates walking. Int J Behav Nutr Phys Act [Internet]. 2019 Dec 7;16(1):84. Available from: https://ijbnpa.biomedcentral.com/articles/10.1186/s12966-019-0841-2

51. El Aarbaoui T, Méline J, Brondeel R, Chaix B. Short-term association between personal exposure to noise and heart rate variability: The RECORD MultiSensor Study. Environ Pollut [Internet]. 2017 Dec;231:703–11. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0269749116320632

52. Chen S, Ni Y, Zhang L, Kong L, Lu L, Yang Z, et al. Noise exposure in occupational setting associated with elevated blood pressure in China. BMC Public Health [Internet]. 2017 Jan 23 [cited 2021 Nov 10];17(1):1–7. Available from: https://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-017-4050-0

53. Lee PJ, Park SH, Jeong JH, Choung T, Kim KY. Association between transportation noise and blood pressure in adults living in multi-storey residential buildings. Environ Int [Internet]. 2019 Nov;132:105101. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0160412019310529

54. Foraster M, Deltell A, Basagaña X, Medina-Ramón M, Aguilera I, Bouso L, et al. Local determinants of road traffic noise levels versus determinants of air pollution levels in a Mediterranean city. Environ Res [Internet]. 2011 Jan;111(1):177–83. Available from:

https://linkinghub.elsevier.com/retrieve/pii/S0013935110001878

55. Allen RW, Davies H, Cohen MA, Mallach G, Kaufman JD, Adar SD. The spatial relationship between traffic-generated air pollution and noise in 2 US cities. Environ Res [Internet]. 2009 Apr;109(3):334–42. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935108002752

56. Roswall N, Raaschou-Nielsen O, Ketzel M, Gammelmark A, Overvad K, Olsen A, et al. Long-term residential road traffic noise and NO2 exposure in relation to risk of incident myocardial infarction – A Danish cohort study. Environ Res [Internet]. 2017 Jul;156:80–6. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935117300634

57. Breiman L. Random Forests. Mach Learn 2001 451 [Internet]. 2001 Oct [cited 2021 Sep 3];45(1):5–32. Available from: https://link.springer.com/article/10.1023/A:1010933404324

58. Horváth IG, Németh Á, Lenkey Z, Alessandri N, Tufano F, Kis P, et al. Invasive validation of a new oscillometric device (Arteriograph) for measuring augmentation index, central blood pressure and aortic pulse wave velocity. J Hypertens [Internet]. 2010 Oct;28(10):2068–75. Available from: https://journals.lww.com/00004872-201010000-00015

59. Goldstein H, Healy MJR, Rasbash J. Multilevel time series models with applications to repeated measures data. Stat Med [Internet]. 1994 Aug 30;13(16):1643–55. Available from: https://onlinelibrary.wiley.com/doi/10.1002/sim.4780131605

60. Ball R, Galecki A. Package "lmeSplines" Title Add smoothing spline modelling capability to nlme. 2015 [cited 2021 Aug 25]; Available from: www.scionresearch.com

61. Huang J, Deng F, Wu S, Lu H, Hao Y, Guo X. The impacts of short-term exposure to noise and traffic-related air pollution on heart rate variability in young healthy adults. J Expo Sci Environ Epidemiol [Internet]. 2013 Sep 17;23(5):559–64. Available from: https://www.nature.com/articles/jes201321

62. Smith KR, Jerrett M, Anderson HR, Burnett RT, Stone V, Derwent R, et al. Public health

benefits of strategies to reduce greenhouse-gas emissions: health implications of short-lived greenhouse pollutants. Lancet [Internet]. 2009 Dec;374(9707):2091–103. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0140673609617165

63. Bellavia A, Urch B, Speck M, Brook RD, Scott JA, Albetti B, et al. DNA Hypomethylation, Ambient Particulate Matter, and Increased Blood Pressure: Findings From Controlled Human Exposure Experiments. J Am Heart Assoc [Internet]. 2013 May 20;2(3). Available from: https://www.ahajournals.org/doi/10.1161/JAHA.113.000212

64. Kubesch N, De Nazelle A, Guerra S, Westerdahl D, Martinez D, Bouso L, et al. Arterial blood pressure responses to short-term exposure to low and high traffic-related air pollution with and without moderate physical activity. Eur J Prev Cardiol [Internet]. 2015 May 17;22(5):548–57. Available from: https://academic.oup.com/eurjpc/article/22/5/548-557/5926708

65. Strak M, Hoek G, Godri KJ, Gosens I, Mudway IS, van Oerle R, et al. Composition of PM Affects Acute Vascular Inflammatory and Coagulative Markers - The RAPTES Project. Cormier SA, editor. PLoS One [Internet]. 2013 Mar 13;8(3):e58944. Available from: https://dx.plos.org/10.1371/journal.pone.0058944

66. Avery CL, Mills KT, Williams R, McGraw KA, Poole C, Smith RL, et al. Estimating Error in Using Ambient PM2.5 Concentrations as Proxies for Personal Exposures: A Review. Epidemiology [Internet]. 2010 Mar;21(2):215–23. Available from: https://journals.lww.com/00001648-201003000-00009

67. Choi Y-J, Kim S-H, Kang S-H, Kim S-Y, Kim O-J, Yoon C-H, et al. Short-term effects of air pollution on blood pressure. Sci Rep [Internet]. 2019 Dec 30;9(1):20298. Available from: http://www.nature.com/articles/s41598-019-56413-y

68. Padró-Martínez LT, Patton AP, Trull JB, Zamore W, Brugge D, Durant JL. Mobile monitoring of particle number concentration and other traffic-related air pollutants in a near-highway neighborhood over the course of a year. Atmos Environ [Internet]. 2012 Dec;61:253–64. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1352231012006760

69. Brook RD, Bard RL, Burnett RT, Shin HH, Vette A, Croghan C, et al. Differences in blood pressure and vascular responses associated with ambient fine particulate matter exposures measured at the personal versus community level. Occup Environ Med [Internet]. 2011 Mar 1;68(3):224–30. Available from: https://oem.bmj.com/lookup/doi/10.1136/oem.2009.053991

70. Farhadi Z, Abulghasem Gorgi H, Shabaninejad H, Aghajani Delavar M, Torani S. Association between PM2.5 and risk of hospitalization for myocardial infarction: a systematic review and a meta-analysis. BMC Public Health [Internet]. 2020 Dec 12;20(1):314. Available from: https://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-020-8262-3

71. Peters A, Dockery DW, Muller JE, Mittleman MA. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. Circulation [Internet]. 2001 Jun 12;103(23):2810–5. Available from: https://www.ahajournals.org/doi/10.1161/01.CIR.103.23.2810

72. Zhao M, Xu Z, Guo Q, Gan Y, Wang Q, Liu J. Association between long-term exposure to PM2.5 and hypertension: A systematic review and meta-analysis of observational studies. Environ Res [Internet]. 2022 Mar;204:112352. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0013935121016534

73. Lin H, Guo Y, Zheng Y, Di Q, Liu T, Xiao J, et al. Long-Term Effects of Ambient PM 2.5 on Hypertension and Blood Pressure and Attributable Risk Among Older Chinese Adults. Hypertension [Internet]. 2017 May;69(5):806–12. Available from: https://www.ahajournals.org/doi/10.1161/HYPERTENSIONAHA.116.08839

74. Brook RD, Weder AB, Rajagopalan S. "Environmental Hypertensionology" The Effects of Environmental Factors on Blood Pressure in Clinical Practice and Research. J Clin Hypertens [Internet]. 2011 Nov;13(11):836–42. Available from: http://doi.wiley.com/10.1111/j.1751-7176.2011.00543.x

75. Brook RD. You are what you breathe: Evidence linking air pollution and blood pressure. Curr Hypertens Rep [Internet]. 2005 Nov;7(6):427–34. Available from: http://link.springer.com/10.1007/s11906-005-0037-9

76. Suh HH, Zanobetti A. Exposure Error Masks the Relationship Between Traffic-Related Air Pollution and Heart Rate Variability. J Occup Environ Med [Internet]. 2010 Jul;52(7):685–92. Available from: https://journals.lww.com/00043764-201007000-00004

Table 1. Descriptive statistics for time variant and invariant variables of the study

	Mean (SD) or N (%)	**Median (2.5th percentile, 97.5th percentile)**
Personal characteristics		
Age	50 (8.69)	50 (35, 64)
Sex		
Female	137 (56%)	NA
Male	108 (44%)	NA
Hypertensive	21 (9%)	NA
Education		
Lower than Baccalaureat	16 (7%)	NA
Equal to Baccalaureat	62 (25%)	NA
Higher than Baccalaureat	167 (68%)	NA
Employment		
Stable	163 (66%)	NA
Unstable	12 (5%)	NA
Retired	31 (13%)	NA
Unemployment	8 (3%)	NA
Other	31 (13%)	NA
Residence		
Paris	64 (26%)	NA
Close suburb	178 (73%)	NA
Far suburb	3 (1%)	NA
Body mass index	25.28 (6.06)	24.04 (18.62, 39.00)
BC exposure averaged over several durations prior to BP measurements (µg/m3)		
Five minutes	1.14 (1.38)	0.77 (0.05, 5.11)
Fifteen minutes	1.14 (1.26)	0.87 (0.06, 5.27)
Thirty minutes	1.15 (1.20)	0.91 (0.07, 5.24)
One hour	1.17 (1.12)	0.95 (0.13, 5.00)
BP measurements		
BP measurements per day per person	14 (6)	14 (3, 26)
SBP (mmHg)	125.98 (18.00)	124 (98.10, 160.90)
DBP (mmHg)	71.42 (14.91)	71 (40.30, 100.00)

MobiliSense Study, 245 participants, 6772 BP measurements

NA: Not applicable

Table 2. Associations between a 1 μg/m^3 increase in personal exposure to black carbon averaged for 5 minutes to 1 hour preceding each BP measurement and ambulatory blood pressure

	SBP (mixed effect models) β (95% CI)	DBP (mixed effect models) β (95% CI)	SBP (fixed effect models) β (95% CI)	DBP (fixed effect models) β (95% CI)
Five minutes	0.57 (0.30, 0.83)	0.36 (0.14, 0.58)	0.42 (0.17, 0.67)	0.31 (0.09, 0.53)
Fifteen minutes	0.47 (0.17, 0.78)	0.36 (0.12, 0.60)	0.38 (0.09, 0.67)	0.31 (0.07, 0.55)
Thirty minutes	0.16 (-0.18, 0.49)	0.23 (-0.02, 0.48)	0.20 (-0.11, 0.51)	0.23 (-0.02, 0.48)
One hour	-0.02 (-0.41, 0.38)	0.08 (-0.21, 0.37)	0.03 (-0.32, 0.38)	0.12 (-0.15, 0.39)

MobiliSense Study, 245 participants, 6772 BP measurements

CI: confidence interval; BP: blood pressure; SBP: systolic blood pressure; DBP: diastolic blood pressure

Regarding time-varying variables, all models were adjusted for noise, physical activity, temperature, relative humidity, proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicle, other places), week vs. weekend, living standard of places where the BP measurements were taken and short-term time trend.

Mixed models were additionally adjusted for residence area, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, and living standard of residential area.

Table 3. Associations between a 1 μg/m^3 increase in personal exposure to black carbon averaged for 5 minutes to 1 hour preceding each BP measurement and ambulatory blood pressure (BP) (excluding 21 hypertensive participants)

	SBP (mixed effect models) β (95% CI)	DBP (mixed effect models) β (95% CI)	SBP (fixed effect models) β (95% CI)	DBP (fixed effect models) β (95% CI)
Five minutes	0.55 (0.28, 0.82)	0.34 (0.12, 0.56)	0.41 (0.14, 0.68)	0.30 (0.08, 0.52)
Fifteen minutes	0.40 (0.09, 0.71)	0.31 (0.06, 0.56)	0.35 (0.04, 0.66)	0.29 (0.05, 0.53)
Thirty minutes	0.09 (-0.26, 0.44)	0.16 (-0.11, 0.43)	0.16 (-0.17, 0.49)	0.19 (-0.06, 0.44)
One hour	0.01 (-0.40, 0.42)	0.05 (-0.26, 0.36)	0.05 (-0.32, 0.42)	0.10 (-0.19, 0.39)

MobiliSense Study, 224 participants, 6150 BP measurements

CI: confidence interval; BP: blood pressure; SBP: systolic blood pressure; DBP: diastolic blood pressure

Regarding time-varying variables, all models were adjusted for noise, physical activity, temperature, relative humidity, proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicles, other places), week vs. weekend, living standard of places where the BP measurements were taken and short-term time trend.

Mixed models were additionally adjusted for residence area, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, and living standard of residential area.

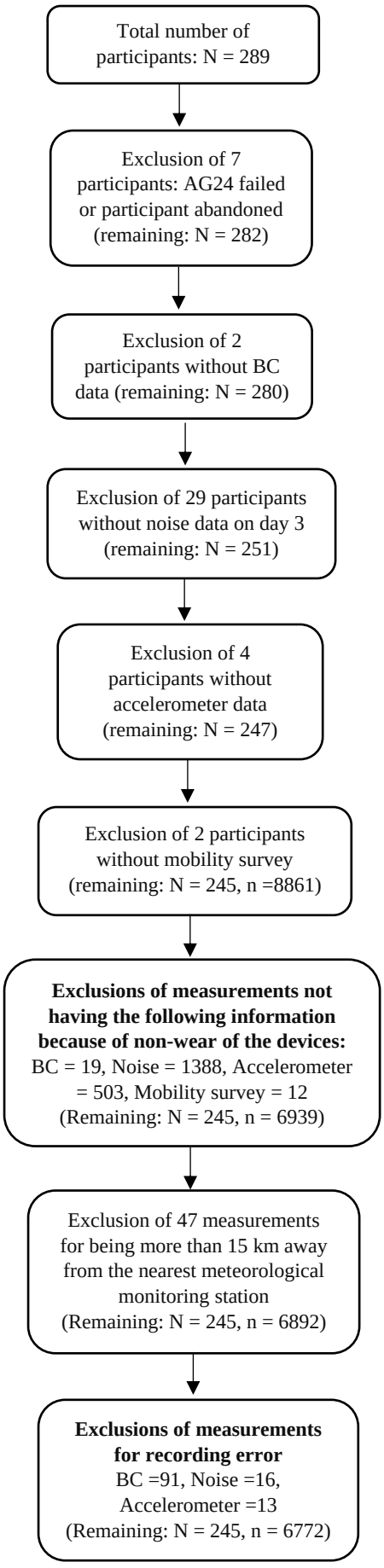

Fig 1. Data collection work flow

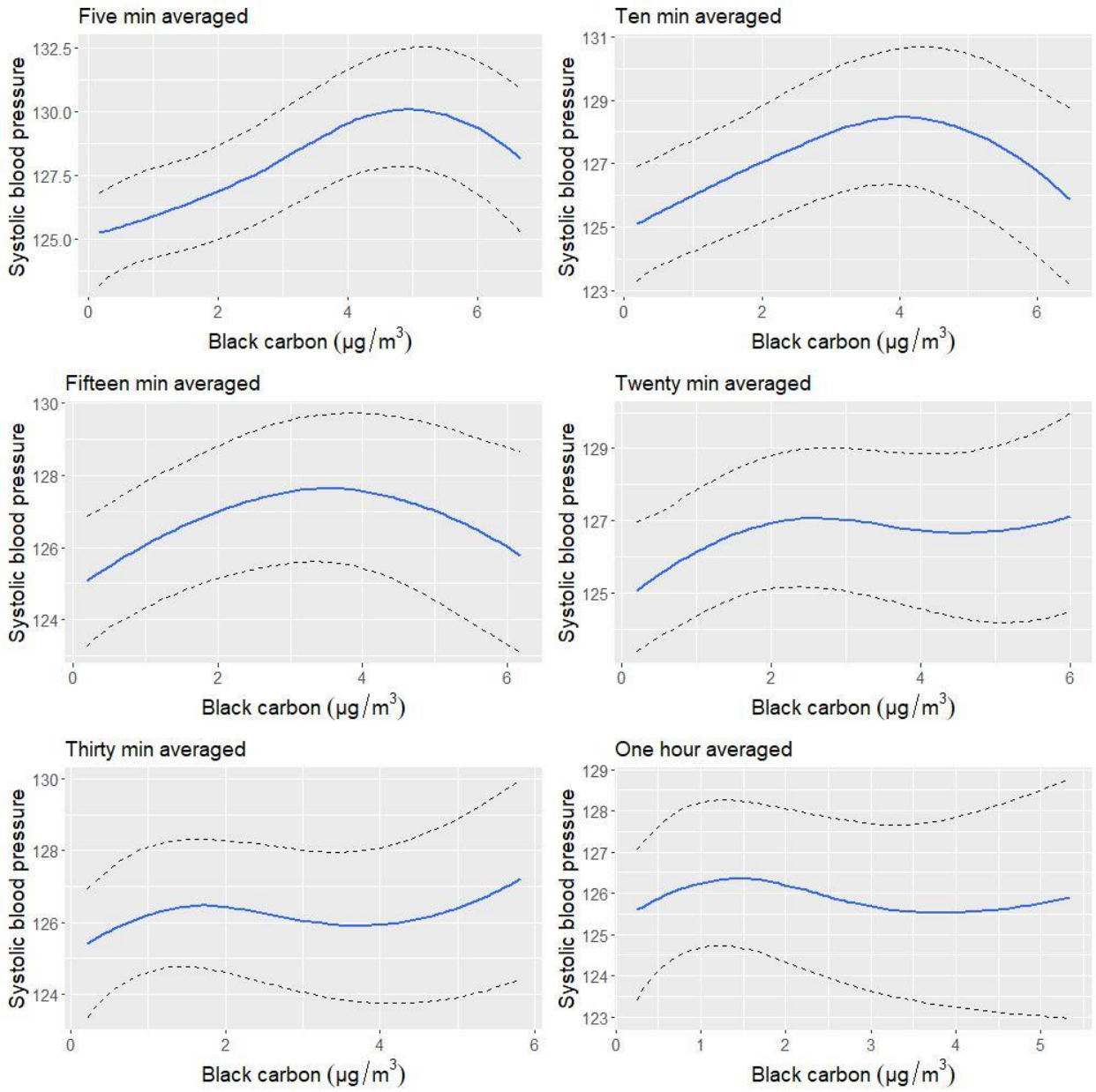

Fig 2. Polynomial splines with 6 degrees of freedom (solid line) and associated 95% confidence intervals (dotted lines) for the association between personal exposure to black carbon (μg/m^3) averaged over different time durations preceding blood pressure (BP) measurements and systolic blood pressure.

Estimated from models with a random intercept at the individual level and a temporal autocorrelation structure. All models were adjusted for noise, physical activity, temperature, relative humidity proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicles, other places), week vs. weekend, residence, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, living standard of residence area and places where the BP measurements were taken and short-term time trend.

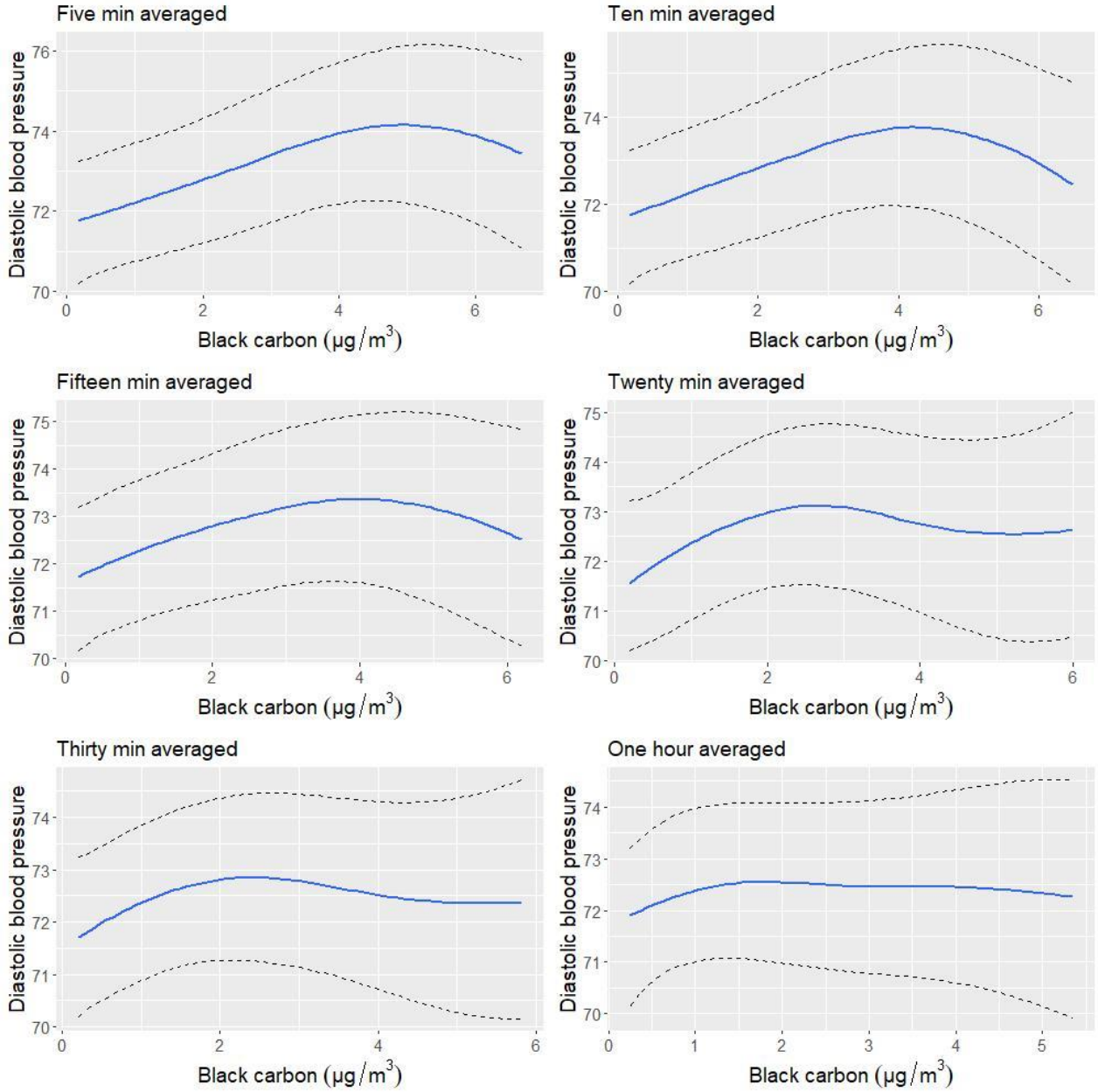

Fig 3. Polynomial splines with 6 degrees of freedom (solid line) and associated 95% confidence intervals (dotted lines) for the association between personal exposure to black carbon (μg/m^3) averaged over different time durations preceding blood pressure (BP) measurements and diastolic blood pressure.

Estimated from models with a random intercept at the individual level and a temporal autocorrelation structure. All models were adjusted for noise, physical activity, temperature, relative humidity proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicles, other places), week vs. weekend, residence, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, living standard of residence area and places where the BP measurements were taken and short-term time trend.

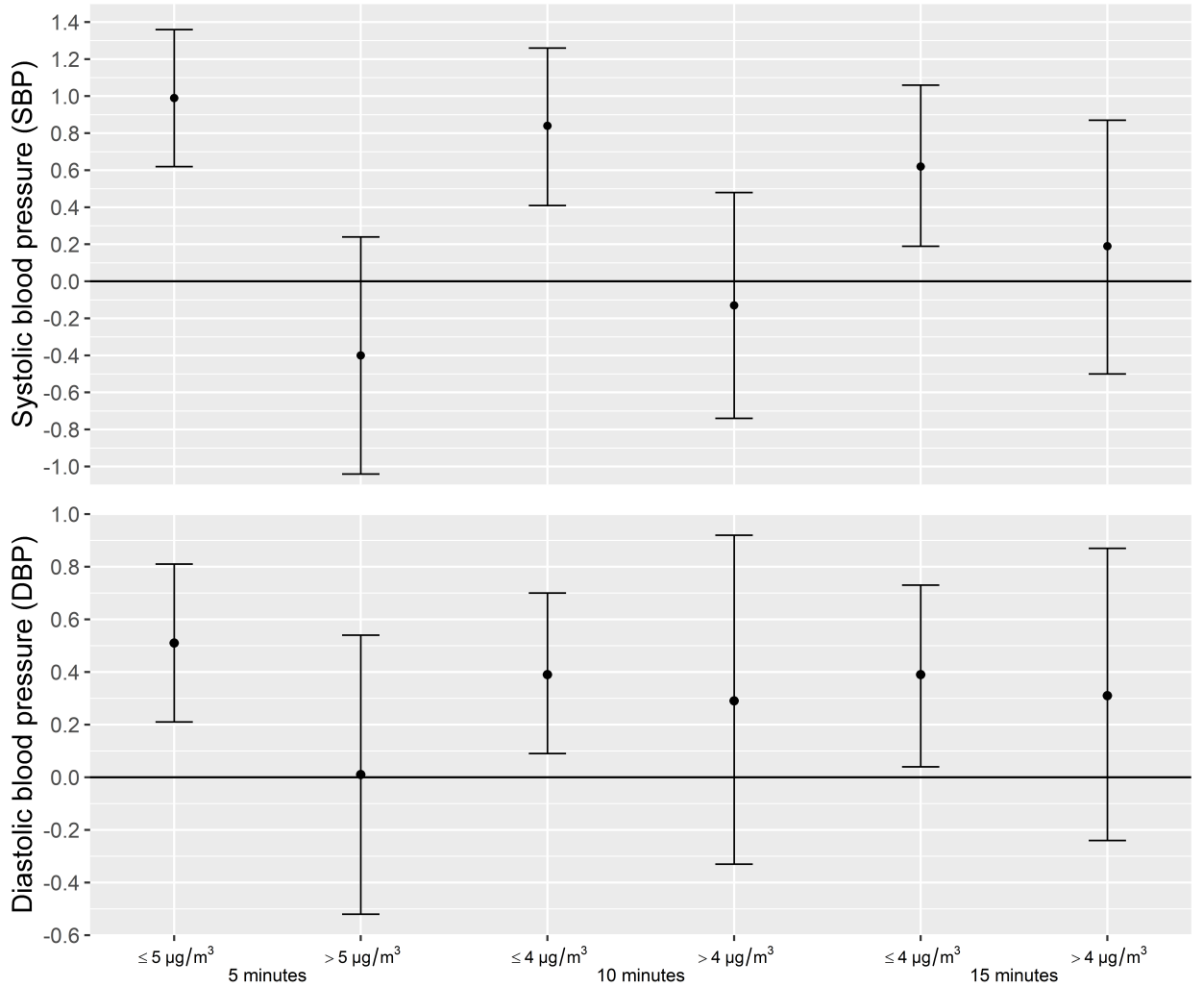

Fig 4. Estimates (and their 95% confidence intervals) from piecewise regression models for the associations between a 1-µg/m^3 increase in 5- to 15-minutes averaged black carbon exposure preceding ambulatory BP measurements and SBP and DBP, estimated from models with a random effect at the individual level and a temporal autocorrelation structure. All models were adjusted for noise, physical activity, temperature, relative humidity proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicles, other places), week vs. weekend, residence, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, living standard of residence area and places where the BP measurements were taken and short-term time trend.

Appendix

Appendix 1: Details on the random forest algorithm

Random forest was run from an R Package ‘randomForest’ (1) which implements Breiman's theory (2). The principle of the random forests method (2) relies on the decision tree method. In order to produce high generalizability, this method applies two sources of randomness to the classic decision tree method and simulates the process repeatedly, thereby leading to a forest of decision trees. The first source of randomness is that the approach considers only random samples (approximately 68%) of the independent variables while defining each knot of the tree. Secondly, sub-samples of the observations (68%) are randomly selected for growing each tree, and the remaining observations (32%), known as out-of-bag data, are used to generate predictions from the tree. With this technique, random forest overcomes overfitting, a common problem faced in classical regression analysis.

Appendix 2: ambient temperature and relative humidity associated with geographic locations recorded with GPS receivers and the mobility survey

Participants carried a BT-Q1000XT GPS receiver with them which collected GPS data every 5 seconds throughout the study period. Personal travel diary filled by participants for every trip, detailing transport modes taken, location as well as start and end times of the trip, allowed us to generate spatial coordinates for the trips for which GPS receiver did not function properly because of technical error or underground movement. Spatial coordinates of points for trip stages commuted through public transports that had no data from GPS receiver were generated from the information provided by the transport authority based on start and end location and particular mode and line taken (which was systematically verified during the mobility survey). Sometimes, during the survey, the research assistants had to report missing trips using two different tools: generation of a street network trip or manual drawing of a trip. In these two cases, spatial points along the trips were generated from these street network trips or manually drawn routes. Spatial points for those non-GPS trips were generated with ArcGIS 10.8.1. Firstly, lines were drawn from the points we had describing these trip segments, then points were generated every 10 meters along the polylines. Lastly, time stamps were added for these generated points using the start time and end time of the corresponding trip stage with the zoo R package (3). All the spatial points along the itineraries (GPS points and other sources of points described above) were used to retrieve the ambient temperature and relative humidity from the nearest Meteo France weather monitoring station, considering data of the corresponding hour.

Appendix 3: Comparison between day 1 and day 3

Detail on discrepancies between day 1 and day 3 in terms of blood pressure (BP), black carbon (BC) and other time-varying observations are provided here.

Table A1. Mean and range of blood pressure (BP) and 5-minute averaged black carbon exposure prior to BP measurements in day 1 and day 3			
	Day 1	Day 3	P-value from Wilcoxon test
Systolic blood pressure	125.1 (74.0, 278.0)	126.4 (82.0, 268.0)	0.013
Diastolic blood pressure	71.2 (26.0, 169.0)	72.7 (28.0, 144.0)	<0.001
Black carbon (μg/m3)	1.06 (0, 13.17)	1.21 (0.01, 14.05)	0.115
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements			

Table A1 illustrates that there was only a slight difference between the BP measurements taken on day 1 and day 3 of the study, whereas BC exposure over 5 minutes prior to each BP measurement was not different across day 1 and day 3, based on a Wilcoxon test.

Table A2. Number (percentage) of blood pressure (BP) measurements during weekdays and weekend in day 1 and day 3			
	Weekdays	Weekend	P-value from Fisher's Exact test
Day 1	1588 (49.95%)	1591 (50.05%)	<0.001
Day 3	3039 (84.58%)	554 (15.42%)	
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements			

Table A3. Number (percentage) of 5-minute episodes before blood pressure (BP) measurements spent in different contexts in day 1 and day 3					
	Home	Non-motorized travel	Motorized travel	Visited place other than home	Mixed context
Day 1	2512 (79.0%)	56 (1.7%)	36 (1.1%)	482 (15.1%)	93 (3.0%)
Day 3	1740 (48.4%)	89 (2.4%)	141 (3.9%)	1462 (40.6%)	161 (4.5%)

P-value from Fisher's Exact test*	Ref	<0.001	<0.001	<0.001	<0.001
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Each context was compared with the home micro-environment.					

The slight difference in the concentration of BC exposure in 5-minute episodes between day 1 and day 3 noted in Table A1, although not statistically significant, is well explained by Tables A2 and A3. About 50% of the measurements of BP on day 1 were recorded on weekends, whereas only 15% were during weekends on day 3. As a result, 79% of the 5-minute episodes on day 1 were spent at home and only 1% in motorized transport, while only 48% of the 5-minute episodes were at home and as much as 4% were in motorized transport on day 3. It is suggested in the literature (4,5) that BC generated from vehicle emission have different effect on human health than BC produced from household sources due to variation in chemical composition.

Table A4. Mean (range) of black carbon exposure in 5-minute episodes before blood pressure (BP) measurements according to the context and micro-environment, in day 1 and day 3					
	Home	Non-motorized travel	Motorized travel	Visited place other than home	Mixed context
Day 1	1.03 (0.00, 13.17)	3.71 (0.14, 10.79)	1.89 (0.28, 10.35)	0.74 (0.00, 7.79)	1.85 (0.08, 7.24)
Day 3	1.11 (0.02, 11.47)	4.12 (0.01, 13.66)	2.32 (0.10, 9.83)	0.88 (0.01, 11.11)	2.27 (0.01, 14.05)
P-value from Wilcoxon test*	0.002	0.695	0.120	0.195	0.134
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.					

On day 3, although hard to conclude statistically, the 5-minute averaged BC exposure was slightly higher in terms of point estimates compared to that on day 1 in all the contexts (Table A4). It has been emphasized above that day 1 was much more on weekends compared to day 3. Weekends mean a lower vehicle density in the streets, which might be the reason for the lower concentration of BC in the different micro-environments, as diesel vehicles are a major source of BC.

Table A5. Mean (range) of systolic blood pressure across different micro-environments assessed in the 5-minute period before blood pressure (BP) measurements in day 1 and day 3					
	Home	Non-motorized travel	Motorized travel	Visited place other than home	Mixed context
Day 1	123.6 (78.0, 278.0)	131.2 (106.0, 169.0)	137.6 (74.0, 200.0)	128.8 (95.0, 184.0)	135.8 (99.0, 275.0)

Day 3	124.1 (82.0, 211.0)	129.3 (95.0, 244.0)	137.2 (98.0, 266.0)	127.5 (83.0, 268.0)	132.9 (99.0, 218.0)
P-value from Wilcoxon test*	0.532	0.378	0.683	0.022	0.357
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.					

Table A6. Mean (range) of diastolic blood pressure across different micro-environments assessed in the 5-minute period before blood pressure (BP) measurements in day 1 and day 3

	Home	Non-motorized travel	Motorized travel	Visited place other than home	Mixed context
Day 1	70.1 (31.0, 169.0)	73.1 (53.0, 107.0)	75.9 (26.0, 134.0)	75.8 (32.0, 120.0)	74.0 (34.0, 116.0)
Day 3	70.8 (36.0, 128.0)	72.8 (45.0, 135.0)	76.2 (28.0, 144.0)	74.5 (37.0, 144.0)	74.8 (35.0, 125.0)
P-value from Wilcoxon test*	0.106	0.173	0.366	0.013	0.780
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.					

Table A5 and A6 illustrate that the mean systolic and diastolic BP measured in visited places other than home were slightly higher on day 1 compared to day 3. Other places than home visited on day 1 and 3 may be different, given that day 1 is more often on week-ends and day 3 more often during the week.

Table A7. Mean (range) of vector magnitude across different micro-environments assessed in the 5-minute period before blood pressure (BP) measurements in day 1 and day 3.

	Home	Non-motorized travel	Motorized travel	Visited place other than home	Mixed context
Day 1	91.9 (0.0, 1110.5)	71.2 (0.7, 692.8)	592.5 (73.2, 1226.5)	67.7 (0.0, 626.0)	314.7 (0.6, 951.6)
Day 3	86.6 (0.0, 1082.0)	59.1 (0.0, 818.1)	489.6 (7.0, 1122.0)	59.2 (0.0, 754.9)	280.5 (2.2, 906.6)
P-value from Wilcoxon test*	0.086	0.501	0.040	0.036	0.156
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.					

Table A7 shows that the mean level of physical activity was higher in day 1 compared to day 3 in motorized travel episodes and in visited places other than home contexts. Even when statistical tests did not demonstrate a difference, point estimates were systematically higher on day 1.

Table A8. Mean (range) of systolic blood pressure across quartiles (Q1 to Q4) of vector magnitude assessed in the 5-minute period before blood pressure (BP) measurements in day 1 and day 3				
	Q1 (0.00 to 6.88)	Q2 (6.89 to 46.83)	Q3 (46.84 to 142.20)	Q4 (142.21 to 1226.51)
Day 1	120.3 (82.0, 171.0)	124.9 (78.0, 201.0)	125.2 (79.0, 212.0)	129.2 (74.0, 278.0)
Day 3	121.8 (83.0, 244.0)	124.9 (83.0, 178.0)	127.9 (82.0, 268.0)	131.9 (86.0, 266.0)
P-value from Wilcoxon test*	0.200	0.816	0.010	0.008
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.				

Table A9. Mean (range) of diastolic blood pressure across quartiles (Q1 to Q4) of vector magnitude assessed in the 5-minute period before blood pressure (BP) measurements in day 1 and day 3.				
	Q1 (0.00 to 6.88)	Q2 (6.89 to 46.83)	Q3 (46.84 to 142.20)	Q4 (142.21 to 1226.51)
Day 1	68.9 (33.0, 114.0)	72.26 (37.0, 140.0)	71.6 (35.0, 136.0)	72.0 (26.0, 169.0)
Day 3	71.07 (39.0, 129.0)	72.5 (37.0, 118.0)	73.5 (36.0, 144.0)	73.9 (28.0, 144.0)
P-value from Wilcoxon test*	0.001	0.675	0.024	0.016
MobiliSense Study Day 1: 237 participants, 3179 BP measurements Day 3: 224 participants, 3593 BP measurements *Separate statistical test was run for each context to compare the outcome across day 1 and day 3.				

Tables A8 and A9 show that there was a slight difference in mean BP between day 1 and day 3, with higher point estimates of BP in most of the quartiles of physical activity, and higher BP levels in day 3 in some of the quartiles as confirmed by the statistical test.

Table A10. Associations between a 1 μg/m^3 increase in personal exposure to black carbon averaged over the 5 minutes preceding BP measurements and ambulatory blood pressure (BP), stratified on day 1 and day 3

	SBP (mixed effect models) β (95% CI)	DBP (mixed effect models) β (95% CI)	SBP (fixed effect models) β (95% CI)	DBP (fixed effect models) β (95% CI)
Day 1	0.16 (-0.31, 0.63)	-0.09 (-0.48, 0.30)	0.07 (-0.40, 0.54)	-0.16 (-0.55, 0.23)
Day 3	0.69 (0.36, 1.02)	0.47 (0.20, 0.74)	0.54 (0.21, 0.87)	0.37 (0.10, 0.64)

MobiliSense Study
Day 1: 237 participants, 3179 BP measurements
Day 3: 224 participants, 3593 BP measurements
CI: confidence interval, SBP: systolic blood pressure, DBP: diastolic blood pressure
All models were adjusted for noise, physical activity, temperature, relative humidity, proportion of time spent in different contexts (home, motorized vehicle and non-motorized vehicles, other visited places than home), week vs. weekend, living standard of places where the BP measurements were taken, and short-term time trend.
Mixed models were additionally adjusted for residence area, age, sex, household income per member, education, employment, monthly alcohol consumption, body mass index, and living standard of residential area.

While stratifying the analyses by day 1 and day 3, the systolic and diastolic BP were associated with 5-minute averaged BC exposure only in day 3, after adjusting for all potential confounders. This discrepancy observed between day 1 and day 3 may very well stem from the differences documented above between the 2 days. Day 1 was more often during weekends and had very high number of observations recorded at home, while there were few observations in motorized vehicle and non-home micro-environments. As explained above, BC exposure from vehicle emission has higher documented effect on human health compared to BC from other sources (4,5). About 50% of the BP measurements on day 3 were taken when the participants were out of their home and BC exposures recorded across all the micro-environments were generally higher on day 3 than on day 1. These reasons may explain the discrepancies that we observed in the patterns of associations between day 1 and day 3.

References

1. Liaw A, Wiener M, Adele C. Package “randomForest” Title Breiman and Cutler’s Random Forests for Classification and Regression. CRAN [Internet]. 2018 [cited 2021 Sep 10]; Available from: https://www.stat.berkeley.edu/~breiman/RandomForests/
2. Breiman L. Random Forests. Mach Learn 2001 451 [Internet]. 2001 Oct [cited 2021 Sep 3];45(1):5–32. Available from: https://link.springer.com/article/10.1023/A:1010933404324
3. Zeileis A, Grothendieck G, Ryan JA, Ulrich JM, Andrews F. Package “zoo” Title S3 Infrastructure for Regular and Irregular Time Series (Z’s Ordered Observations). 2021 [cited 2021 Aug 16]; Available from: https://zoo.r-forge.r-project.org/
4. Auchincloss AH, Diez Roux A V., Dvonch JT, Brown PL, Barr RG, Daviglus ML, et al. Associations between Recent Exposure to Ambient Fine Particulate Matter and Blood Pressure in the Multi-Ethnic Study of Atherosclerosis (MESA). Environ Health Perspect [Internet]. 2008 Apr;116(4):486–91. Available from: https://ehp.niehs.nih.gov/doi/10.1289/ehp.10899
5. Brook RD, Urch B, Dvonch JT, Bard RL, Speck M, Keeler G, et al. Insights Into the Mechanisms and Mediators of the Effects of Air Pollution Exposure on Blood Pressure and Vascular Function in Healthy Humans. Hypertension [Internet]. 2009 Sep 1 [cited 2021 Jul 30];54(3):659–67. Available from: https://www.ahajournals.org/doi/abs/10.1161/HYPERTENSIONAHA.109.130237