Cardiovascular impacts and micro-environmental exposure factors associated with continuous personal $PM_{2.5}$ monitoring

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Keywords: DEARS, exposure factors, cardiovascular health outcomes

Running head: Exposure factor health impacts

Abstract

The US Environmental Protection Agency's (US EPA) Detroit Exposure and Aerosol Research Study (DEARS) has provided extensive data on human exposures to a wide variety of air pollutants and their impact on human health. Previous analyses in the DEARS revealed select cardiovascular (CV) health outcomes such as increase in heart rate (HR) associated with hourly-based continuous personal fine particulate matter ($PM_{2.5}$) exposures in this adult, non-smoking cohort. Examination of time activity diary (TAD), follow-up questionnaire (FQ) and the continuous $PM_{2.5}$ personal monitoring data provided the means to more fully examine the impact of discreet human activity patterns on personal $PM_{2.5}$ exposures and changes in CV outcomes. A total of 329,343 minute-based $PM_{2.5}$ personal measurements involving 50 participants indicated that approximately 75% of these total events resulted in exposures < 35 $\mu g/m^3$. Cooking and car-related events accounted for nearly 10% of the hourly activities that were identified with observed peaks in personal $PM_{2.5}$ exposures. In-residence cooking often resulted in some of the highest incidents of one minute exposures (33.5 to 17.6 $\mu g/m^3$) with average peaks for such events in excess of 209 $\mu g/m^3$. $PM_{2.5}$ exposure data from hourly-based personal exposure activities (e.g., cooking, cleaning, household products) were compared with daily CV data from the DEARS subject population. A total of 1300 hourly-based lag risk estimates associated with changes in brachial artery diameter and flow-mediated dilatation (BAD, FMD, respectively), among others, were defined for this cohort. Findings indicate that environmental tobacco smoke (ETS) exposures resulted in significant HR changes between 3-7 hours following the event and exposure to smells resulted in increases in BAD on the order of 0.2 to 0.7 $mm/\mu g/m^3$. Results demonstrate that personal exposures may be associated with several biological responses, sometimes varying in degree and direction in relation to the extent of the exposure.

Introduction

Multiple studies have been conducted in the Detroit metropolitan area (Wayne County, Michigan) to understand human exposures to air pollutants and potential impacts on health (Lewis et al., 2005; Keeler et al., 2002; Rohr et al., 2010). Detroit is heavily influenced by emissions from mobile and point sources, including coke ovens, coal-fired power plants, iron/steel manufacturing, sewage sludge incineration, refineries and chemical plants (Hammond et al., 2008; Duvall et al., 2012). In addition, the Ambassador Bridge, a border crossing between Windsor, Canada and Detroit, is a potentially large diesel and automotive emissions source particularly during idling periods (Baxter et al., 2008). These sources and other factors have contributed to Detroit being designated as a nonattainment area for the $PM_{2.5}$ National Ambient Air Quality Standard (NAAQS) and in Wayne County having eight of the state's fifteen most polluted zip codes from industrial air pollution (Detroit Free Press, 2010).

While the ambient environment certainly impacts the residents of this area, researchers have found that people spend more than 85% of their time indoors (Klepeis et al., 2001). Therefore, individuals are routinely exposed to $PM_{2.5}$ air pollutants emanating from both ambient as well as indoor sources (Wallace et al., 2005; Williams et al., 2003). The indoor sources often contribute a significant percentage of one's total personal exposure (Olson et al., 2006; Wallace et al., 2005; Williams et al., 2000). Even so, human exposures to indoor $PM_{2.5}$ air sources are not well characterized relative to how they impact either chronic or acute health outcomes. Recently, an adult cohort undergoing extensive exposure and health monitoring reported seminal findings indicating that it was often the exposure to total fine particulate matter containing both ambient and non-ambient source origins (such as that potentially originating from the indoor and outdoor environments) that represented the highest risk estimates for a select panel of

cardiovascular outcomes (Brook et al., 2011a; Williams et al., 2012a). Acute (hourly) total personal $PM_{2.5}$ monitoring exposures, undifferentiated with respect to source contribution, have been reported to be linked to deleterious changes in heart rate and heart rate variability (Brook et al., 2011b; He et al., 2010; He et al., 2011). These novel findings did not report on the impact of either non-ambient sources or human activities on the resulting cardiovascular risk estimates.

The Detroit Exposure and Aerosol Research Study (DEARS), a three year air monitoring campaign conducted by the United States Environmental Protection Agency (EPA) provided such an opportunity for examining this research area. DEARS measurements have been used to examine the spatial variability of speciated fine and coarse particulate mass along with select air toxics to assess the suitability of using central-site monitor data in epidemiological and health studies (George et al., 2010; George et al., 2011; Rodes et al., 2010; Thornburg et al., 2010). Williams et al. (2009) provides a complete description of DEARS to include study objectives and design, methods and monitoring protocols.

In addition to the aforementioned objectives, a fundamental goal of DEARS was to determine the impact of human activities on personal exposure to air pollutants and expand upon the findings from previous studies. Wallace et al. (2005) found that outdoor particles contributed about half of the total personal exposures and indoor concentrations in a study of 37 health-impaired North Carolina residents. Analysis of data from the Relationship of Indoor, Outdoor and Personal Air (RIOPA) study found that the predictive power of a personal activity model for $PM_{2.5}$ mass was improved by incorporating personal activities in addition to outdoor $PM_{2.5}$ (Meng et al., 2009). Furthermore, McCormack et al. (2008) reported that common modifiable household activities, especially smoking and sweeping, contributed significantly to higher PM found in the bedrooms of inner-city Baltimore children.

Previous DEARS research has reported that short term (hourly) $PM_{2.5}$ personal exposures were significantly associated with increases in heart rate (HR) and that such events often occurred between 1 and 10 hours after the exposure event (Brook et al., 2011a). Similarly, it has also been reported from the DEARS that total personal exposures to various $PM_{2.5}$ mass components (i.e., iron and potassium) were far more often (61%) associated with various cardiovascular health effects than comparable ambient-based comparisons (Williams et al., 2012a). Such findings indicate that non-ambient source contributions had a significant impact on the cohort's overall health outcomes.

The current article will report on the association of select human activity patterns from the DEARS cohort and the impact of personal exposure activity factors on select cardiovascular health outcomes using continuous personal $PM_{2.5}$ mass monitoring. The objective for this effort is to provide an improved characterization of the impact of human exposures to non-ambient sources and how they might potentially contribute to observed cardiovascular health effects.

Methods

Participant recruitment

The DEARS was designed as a three year study (2004-2007) with two sampling seasons per year (one summer followed by one winter season) for a total of six seasons. A total of 136 participants were enrolled in the study where participants were only monitored during their year of recruitment (maximum 5 days in summer, 5 days in winter). More detailed descriptions of the DEARS recruitment process, participant retention, and full study demographics have been previously reported (Williams et al., 2009; Phillips et al., 2010). The seven participant inclusion criteria were: (1) non-smoking, (2) living in a non-smoking household, (3) ambulatory, (4) stationary (expected to live in the same dwelling for the next 9 months), (5) living in a detached

home, (6) age 18 or older, and (7) comprehend English or Spanish instructions. There were no health restrictions on enrollment other than being ambulatory, and no enrollment restrictions on occupation, socioeconomic status, sex, or ethnicity. All participants consented to the study protocol which had been formally reviewed and approved by RTI International, the University of North Carolina at Chapel Hill (EPAs IRB), the University of Michigan's IRB, and the US EPA's Human Subject Research Official. The randomly recruited participants were selected from a total of 6 census areas (enumeration monitoring areas-EMAs) which had a variety of industrial, regional, and local source impacts as required to meet the goals of the DEARS study design (EPA, 2012). Williams et al. (2009) and Duvall et al. (2012) have described each of the EMAs and the theorized and study-determined source impacts.

Personal exposure monitoring

Personal monitoring was performed continuously using a nominal 0900 hrs to 0900 hrs (± 2.5 hrs) time window from Tuesday morning through Sunday morning. The only time monitoring was not being performed was during field staff equipment refurbishment/data recovery which occurred between 0630 hrs and 1130 hrs each monitoring day. Participants wore the personal monitor (personal DataRAM 1000, or pDR, MIE, Inc., Bedford, MA) with the inlet in the breathing zone affixed to the vest along with other collocated passive and active samplers. The pDR measures particles in aerodynamic diameter from 0.3 to 10 μm, although the units are most sensitive to particles ranging from approximately 0.5 μm to 2 μm.

The pDR units were set to record one minute particle concentrations for 24-hr periods. For the first three DEARS seasons, the pDR was modified by adding a 2.5 μm PEM inlet and a short drying column upstream of the pDR's optical bench. However, the drying column resulted in sub-optimal performance of the unit and unexplainable concentration peaks. For the last three

158 seasons (winter 2005, summer 2006, winter 2007), the units were operated without the drying
159 column and only the data from these seasons were utilized in the present analysis. In addition, a
160 relative humidity algorithm was developed post-study which was applied to all data. This
161 application significantly improved the comparability of the nephelometric data versus personal
162 collocated $PM_{2.5}$ filter-based gravimetric samples. All data reported here have been treated and
163 normalized with respect to both relative humidity impacts and gravimetric-based normalization
164 factors. Full descriptions of personal nephelometric monitoring and subsequent continuous
165 monitoring data recovery and routine processing procedures have been described previously
166 (Wallace et al., 2005; Williams et al., 2009; Brook et al., 2011b).
167 Personal monitoring compliance (the percentage of time the participant wore the pDR as
168 per study protocol) was determined using a combination of both temperature and accelerometric
169 devices incorporated into the monitoring vest. We have established a required compliance rate
170 of at least 60% per monitoring event per individual as being needed to adequately assess a
171 participant's daily personal $PM_{2.5}$ exposure. The techniques associated with conducting personal
172 monitoring compliance and its importance in ensuring the highest data quality have been
173 reported (Rodes et al., 2010, Brook et al., 2011a; Brook et al., 2011b; Lawless et al., 2012). In
174 addition, all DEARS participants were monitored for personal environmental tobacco smoke
175 (ETS) exposures using a collocated filter-based collection method (Lawless et al., 2004;
176 Williams et al., 2009). A minimum personal exposure rate of 1.5 $\mu g/m^3$ of ETS-associated $PM_{2.5}$
177 constitutes exposure to this source (Rodes et al., 2010). DEARS personal monitoring data were
178 summarily categorized into sub-categories of personal monitoring compliance and ETS impacts.
179 ETS impacts on the DEARS health outcomes had already been established for both filter-based
180 and pDR based data (Brook et al., 2011a, Williams et al., 2012a). We report select findings

related to an "All Subjects" cohort that reflects the total sampling population regardless of ETS exposures or protocol compliance with respect to wearing the personal monitor as well as a "Vest-LowNicotine" cohort in which both full protocol compliance (wearing of the monitoring vest) and low ETS exposures occurred. Only findings presented in Table 5 reflect the more data restrictive subcohort. It is appropriate to consider the "All Subjects" cohort here for our primary reporting of human activity impacts on the local micro-environment (e.g., cooking, cleaning) as subjects typically indicated the vest to be in close proximity (same room) to them even when it was not being worn. As such, data findings from the total sampling population reflect a mixture of true personal exposures as well as some local micro-environment (non-personal) assessment. Examination of health outcomes without the potential confounding of ETS exposures in the "Vest-LowNicotine cohort" reported in Table 5 was believed to offer additional insight as to the impact of commonly encountered, non-ambient originating sources on health outcomes without potential confounding by ETS.

Personal pDR data recovery

The first and last 15 min of each monitoring day were excluded from this analysis. These periods typically involved the set up of instrumentation by field staff and may have led to elevated particle concentrations due to particle resuspension. Exposure peaks were identified by a change in the slope value of the resulting curve as the response grew above the baseline (peak start) and subsequently returned to a baseline value (peak stop). The resulting integrated area under the curve was then calculated and a mass concentration was determined. The procedures used to recover and process pDR data have been reported previously (Wallace et al., 2005).

Survey data

In addition to the personal PM measurements, participants kept an activity log based upon 15-minute intervals during which they indicated their locations, potential particle-generating activities (Candles, Car, Cooking Cleaning, Products, Smells, Smoke, Windows), and any noticeable exposure to environmental tobacco smoke (ETS). Full reporting of all of the DEARS surveys (daily TAD and FQ) is available on the DEARS website (EPA 2012). The 24 hr based daily activity logs were completed by each participant for each measurement day. The five highest peaks were matched to reported activities, and if no activity had been recorded at the time of a particular peak, the participant was asked if he or she could recall what activities were occurring at that time. Information concerning such events was recorded on the daily activity diaries and then cross-linked with the daily FQ. This latter survey tool, performed using a technician aided electronic form, collected information on a wide variety of potential source impacts, their duration, and the exact timing of the event on a minute by minute basis.

Survey and exposure data integration

$PM_{2.5}$ data were captured at the participant, location, and date-time level aspect. For each minute, a $PM_{2.5}$ mass concentration level was recorded. The minute data were post-collected processed by a novel SAS algorithm (Croghan and Williams, 2007) that located the beginning and ending of peaks. The time integrated mass concentration value ($\mu g/m^3$) and the maximum mass concentration ($\mu g/m^3$) values obtained during each peak reporting period (1 minute interval) were calculated. An example of how this SAS algorithm has been previously used for personal nephelometric measures has been reported (Wallace et al., 2005). The FQ data were at the event level. For each event, the start time and the duration of the event was recorded. There were often multiple events occurring at the same time. For example, when a participant was

cooking breakfast of eggs, toast, and coffee the different cooking methods (poaching eggs, toasting bread, brewing coffee) were stored as separate events.

Cardiovascular data collections

The University of Michigan conducted a companion cardiovascular health study simultaneously with the DEARS involving a subpanel of the full cohort (previously defined in Table 1). The general health and demographics of the total cohort have been reported earlier (Brook et al., 2011a; Brook et al., 2011b). Cardiovascular (CV) home study visits were performed at the participant's residence for up to five consecutive evenings, Tuesday through Saturday, between 1600 and 1900 hours. These visits took place on concurrent days while subjects wore the vest monitors. There were six CV outcomes: systolic and diastolic BP (SBP and DBP, respectively), heart rate (HR), brachial artery diameter (BAD, indicative of basal arterial tone), flow-mediated dilatation (FMD), and nitroglycerin-mediated dilatation (NMD, indicative of smooth muscle function). Participants were instructed to maintain their daily routine, including taking all medications, but to fast for at least 4 hr before the scheduled visits and to avoid unusual physical activity. During each visit, subjects rested supine for 10 min before automated BP and HR measurement (Omron 780 monitor; Omron Inc, Kyoto, Japan) were obtained in triplicate with a 1-min lapse between measures. The average of the second and third BP and HR recordings was used for analyses (Pickering et al., 2008). Detailed descriptions of the cardiovascular health study and its integration with the DEARS have been reported previously (Brook et al., 2011a,b; Williams et al., 2012a).

Statistical analyses

Personal $PM_{2.5}$ exposure concentrations were transformed to logarithms due to positive skewness. The minute-based activity records were summarized by indicator functions for the presence of the activity in any given hour. $PM_{2.5}$ minute-based concentrations were averaged by the hour. A descriptive analysis was performed to examine the univariate relationship between $PM_{2.5}$ exposure concentrations and each of the nine sources separately. Multivariate linear regression was used to examine the relationship between $PM_{2.5}$ exposure concentrations and the nine sources simultaneously (West et al., 2007). The relationship between the six repeated cardiovascular health outcomes (24-hr based measurements) for each subject and the hourly reports of the presence of the nine sources was examined using a mixed linear model assuming each subject's intercept varied as a normal random variable (Fox 2012). Separate lag times between the time of recording the cardiovascular measure and time of exposure to source were examined from 1 to 23 hours. Additional predictors of cardiovascular outcomes that were included in the mixed model but did not vary over the repeated measurements within a subject were age, gender, race, body mass index, ambient temperature, and medication use.

Results

A statistical summary of DEARS participants' demographic data incorporated into the current analysis is shown in Table 1. Data from a total of 50 participants is reported. The age of one participant's home was not obtained. Participant ages ranged from 19 to 73 years with a mean of 42. The majority were African-Americans (55%) with the remainder comprised of Hispanics (35%) and Caucasians (10%). Approximately 25% of those who participated were men. Low male enrollment for the study could be due to work or other considerations. Close to 35% of the enrollees were employed outside the home and roughly 90% lived in single-family homes.

279 The pDR and survey databases for the winter 2006, summer 2006, and winter 2007
280 monitoring seasons resulted in ~ 250 person-days of personal exposure and time activity data.
281 Given the 1-minute sampling interval, $PM_{2.5}$ concentrations obtained from the unit changed
282 dramatically over the 24-hr sampling period, increasing while in the presence of a localized
283 source (cooking, sweeping, etc.) and decreasing when near an active process removing the
284 source (stove top exhaust fan). The PM concentration peaks obtained from the pDR varied by
285 both height (maximum concentration) as well as length of existence. Distribution and univariate
286 summary of the overall personal $PM_{2.5}$ exposures (by minute) is reported in Table 2. Out of the
287 329343 total events available for this analysis, about 97% of the events were positive (> 0
288 $\mu g/m^3$). Approximately 74% of the average $PM_{2.5}$ concentrations, excluding negative values,
289 were less than or equal to 35 $\mu g/m^3$. Some high exposure events were observed, with those
290 averaging 100 $\mu g/m^3$ or higher representing nearly 6% of all events.
291 An example of a participants' daily $PM_{2.5}$ exposure time series is shown in Figure 1. The
292 information was obtained from one participant in February 2007. Four distinguishable peaks
293 were identified for this particular person-day. The peaks were correlated with specific activities
294 using a combination of the FQs and the TADs. The highest peak for this person-day was 1200
295 $\mu g/m^3$, and the participant indicated exposure to in-vehicle second-hand smoke as the activity
296 occurring during this time period. Exposure to automotive emissions while at a gas station
297 during a period of heavy traffic was the correlating activity associated with the second highest
298 peak (889 $\mu g/m^3$). The two lowest peaks (367 $\mu g/m^3$ and 226 $\mu g/m^3$) were associated with
299 cleaning and transportation activities, respectively. This example serves as a representative time
300 series for a majority of the participants where the following was observed: (1) each person-day
301 consisted of four to six identifiable peaks attributed to specific activities as indicated in the FQ or

302 TAD, and (2) each person-day maintained a reasonable baseline concentration value on par with
303 typical indoor background levels.
304 Of the 24 time activity and exposure variables available for consideration, nine passed the
305 screening analysis for inclusion in the model: cooking activities, residential candle burning, ETS
306 exposure, vehicular travel, residential cleaning events, open windows, presence of smells of
307 unknown source, observation of visible smoke (aerosol) in the home and/or surrounding
308 neighborhood, and use of commercial chemicals/cleaners. Table 3 provides a statistical
309 summary of the pDR peaks associated with human activities by season. Peaks were identified in
310 the pDR data through statistical analysis if the following criteria were met: the increase in
311 concentration was at least 5 min in duration, the peak concentration was at least twice the
312 background concentration, and the concentration returned to background levels. The maximum
313 concentration represents the height of the peak while the duration is indicative of the source
314 generation time.
315 A total of 60 participants provided activity logs and corresponding pDR data where over
316 730 peaks were attributed to specific indoor activities. The greatest number of peaks (N=404)
317 were identified during Season 5, the only summer season, which had the largest number of
318 participants engaging in cooking, cleaning, and grooming activities along with higher candle
319 usage. Season 6 contained the lowest number of participants and the fewest peaks (N=144).
320 Cooking contributed to the highest number of peaks for all seasons ranging from 13% to 18% of
321 the total number of peaks. The peaks linked with cooking activities included several cooking
322 methods (grill, fry, bake, broil, boil toast) and three equipment types (oven, stove, microwave).
323 All cooking events, regardless of length, were included in the analysis. When considering

individual cooking methods, frying and grilling accounted for 59% of the total number of cooking peaks.

Two to seven percent of the identified peaks for each season were attributed to cleaning activities. Cleaning activities in this analysis included sweeping, mopping, vacuuming, and dusting events using any combination of standing liquid, spray or aerosol cleaners. Seventy-two percent of the total cleaning peaks were attributed to mopping and sweeping. Ferro et al. (2004) concluded that activities which disturbed dust reservoirs on furniture and textiles, such as dry dusting, resulted in high particulate exposures. Our analysis supports this finding since sweeping activities resulted in acute exposure peaks and the DEARS participants regularly used cleaners while dusting which reduced dust resuspension.

Fragrance (categorized as "smells" in the tables and figures) impacts on personal fine particle exposures varied by season based on fragrance and/or deodorizer type according to TAD analyses. A consistent number of peaks (3%-6%) were attributed to smells associated with sprays or aerosols in the winter and summer seasons; however, the findings indicate a seasonal relationship for smells associated with candle or incense use [11% (summer); 6.5% (avg. winter)]. Participants who used aerosol or spray deodorizers (e.g., Glade, Febreze) regularly used the products in both summer and winter seasons.

Figure 2 shows the percentage of time impacted by an indoor hourly source activity. For most of the source activities, the amount of activity time for a particular source is consistent among the seasons. However, the activity percentages for products (grooming) by season illustrate that the participants were more exposed to this source in Season 6. Indoor pollution impacts related to open windows during Season 5 resulted in the highest activity percentage (39%) for all sources. Given the fact that many of the DEARS participant homes did not have

central air conditioning, participants regularly kept their windows open for summertime cooling. Baxter et al. (2007) found higher indoor PM concentrations inside homes of lower socioeconomic status urban homes due to the entrance of ambient air into the indoor environment.

A potential multicollinearity among the various source factors could produce poor estimates of the effects of individual sources. Spearman correlation coefficients were analyzed between the nine source factors based on a total of 5597 hourly data. With the exception of the relationship between hourly use of candles and opening windows, no correlation coefficients above 0.2 were observed. Based on the near complete independence of the factors, a multiple linear regression model with all nine of the source factors was employed to describe their relationship with $PM_{2.5}$ exposures in log scale using hourly measurements. The exponential of the regression coefficients represent the expected changes in the $PM_{2.5}$ exposure by the selected source factor when all the other source factors are held constant. The estimated regression coefficients in both log and original scales are compared in Table 4.

Findings from Table 4 would indicate, as an example, exposure to cooking events would result in 93% increase in total personal hourly exposure concentrations. The highest source impact was observed for ETS exposure with an 128% increase in $PM_{2.5}$ exposure. Five activities (ETS, open windows, cooking activities, use of commercial chemicals/cleaners and residential candle burning) were found to contribute to the $PM_{2.5}$ exposure at the 5% significance level.

Modeling each source factor individually made only minor changes in the PM exposure estimates comparing to those estimates in Table 4 obtained by the multiple regression model. Overall, all indoor source activities were found to increase the $PM_{2.5}$ exposure at the 5% significance level. $PM_{2.5}$ exposures associated with each of the seven sources are displayed in

370 Figure 3. Note that Figure 3 shows positive hourly $PM_{2.5}$ exposures only, which cover about 97%
371 of the whole data.

372 More than 1300 different hourly lag risk estimates were developed for the six health
373 outcomes using the binary, hourly based source exposure scenario and the All Subjects cohort.
374 Even so, the total number of hourly lag-specific activities in which a significant relationship with
375 a deleterious health outcome was observed was relatively small. Tables S1 – S2 provide a
376 general summary of those in which a consistent trend (either positive or negative) risk estimate
377 was obtained across the various health outcomes from the "All Subjects" study population. Such
378 evidence had to be present in at least two of the total lag periods over the 24 hr period with a p-
379 value of ≤ 0.05 associated with a given health outcome to be discussed in any depth here.
380 Individual (single hourly lag outcomes) events did exist as a result of the analyses and are
381 reported here in Tables S1 – S2 as a matter of completeness.

382 Even though the DEARS cohort was recruited to be non-smoking and ETS-impacted data
383 had been removed from the dataset prior to the analyses (analytically determined ETS exposures
384 ≥ 1.5 $\mu g/m^3$), risk analyses indicated that ETS exposures were observed to significantly decrease
385 both SBP and DBP, resulting in changes of -18 $mmHg/\mu g/m^3$ (DBP - lag 2 hr) to as much as -33
386 $mmHg/\mu g/m^3$(SBP - lag 12 hr). Typically, statistically relevant and consistent (trend) changes in
387 SBP and DBP were observed to occur as early as 7 hr (SBP) with most of the effect peaking
388 following 12 hours from the event (DBP and SBP). It is noteworthy that there was an abrupt risk
389 estimate sign change for SBP (negative at 16 hr, positive at 17 hr) and of similar magnitudes.
390 SBP was also observed to be impacted by smoke and use of household products source activities
391 among others. Smoke exposures resulted in fast changes in SBP with observations for the 3 hr
392 lag being the most significant (34.2 $mmHg/\mu g/m^3$).

ETS exposures resulted in significant HR changes between 3-7 hours following the event. Results reported earlier (Brook et al., 2011b) on hourly-based total personal pDR exposures and HR changes for this cohort reported similar time lags of significance (1 through 10 hr). However, the risk estimates we obtained in the current effort are significantly higher than those associated with the original work (≥ 6.7 mmHg/$\mu g/m^3$ as compared to ~ 0.05 mmHg/$\mu g/m^3$). These differences might reflect the improved source-derived risk estimates as compared to the more general total personal exposures performed in the original work.

Results reported in Table 5 reflect the more restrictive cohort where full protocol compliance and low levels of ETS occurred. The effects of ETS and smoke were no longer as significant as they were previously in the All Subjects population for SBP. This analysis did reveal new associations with candle activities. This activity was associated with a decrease in SBP ranging in excess of -7.2mm/$\mu g/m^3$ over the course of 7-23 hours of lag. Candle activities were also associated with negative HR trends (1-6 hr lag). Car-related activities were associated with a late lag ($\geq$ 16 hr) increase of ~ 12 bpm. The effects of smells, smoke, and products were considerably reduced in the new treatment for BAD.

Discussion and Conclusions

This study investigated the association of select human activity patterns from the DEARS cohort and the impact of personal exposure activity factors on select cardiovascular health outcomes using continuous personal $PM_{2.5}$ mass monitoring. Collection of short duration (15 minute) time activity location data coupled with information from both the continuous PM data monitoring and a separate daily participant exposure questionnaire provided the means to examine the exposure scenario responsible for observed hourly-based health outcomes. Several

studies have identified near real-time cardiovascular responses due to exposures to certain PM sources (Peters et al., 2004; Brook et al., 2011b); however, this study lacked statistical power to conduct a high-time health outcome interpretation of the source exposures due to the unavailability of minute-by-minute data.

The data collected from this study confirmed that a combined exposure monitoring strategy (continuous personal PM monitoring, time activity location data, and daily participant exposure questionnaire) provides adequate data to determine potential source impacts in a real-world cohort. Of the nine personal exposure activities investigated for this study, cooking contributed to the highest number of personal exposure peaks. Cooking via frying and grilling accounted for roughly 60% of the total number of cooking peaks. Hence, potential indoor exposures to fine particles may be considerably higher for persons belonging to cultures where traditional practices include frequent consumption of fried foods (Ko et al., 2000).

Smell impacts on personal fine particle exposures showed the greatest variability among all personal exposure activities. The data indicated that participants who used aerosol or spray deodorizers (e.g., Glade, Febreze) regularly used the products in both summer and winter seasons. Bridges (2002) highlights the indoor air quality and environmental concerns associated with regular deodorizer use either manually or by way of metered aerosol deodorizer dispensing mechanisms. Religious, cultural and seasonal practices impacted participant candle and incense usage. Peaks occurring while participants burned incense were the highest of all smell-related peaks (max. peak = 608 $\mu g/m^3$). Chuang et al. (2012) found that the fine particle concentrations from burning candles and/or incense related to religious observances can produce $PM_{2.5}$ levels as high as 38.9 $\mu g/m^3$ and may pose significant risks in terms of respiratory health effects. Jetter et al. (2002) concluded that burning incense emits fine particulate matter in large quantities

compared to other indoor sources and that testing of the burned incense emissions revealed the presence of carbon monoxide (CO), nitric oxide (NO), and sulfur dioxide (SO_2). Higher summertime smell peaks may be attributed to citronella candle use to repel pests.

Window use was also found to be a significant contributor to indoor and personal $PM_{2.5}$ exposures. Given the numerous ambient air pollution sources in the Detroit metropolitan area and their potential individual air quality impact (Hammond et al., 2008), time periods when windows remained open for extended periods could promote occurrences where the indoor PM concentrations equaled or even exceeded outdoor concentrations. These results agree with findings from other studies; for instance, Ohura et al. (2009) reported that indoor concentrations of VOCs in China tended to be higher than outdoor concentrations.

Source impacts from smells were typically determined to result in increases in BAD on the order of 0.2 to 0.7 mm/$\mu g/m^3$. Smell impacts typically occurred after a 3 to 10 hr lag. As before, the strength of the estimate here for BAD changes is significantly higher than that observed in our original work for total personal $PM_{2.5}$ exposures (~0.30 mm/$\mu g/m^3$ as compared to 0.001 mm/$\mu g/m^3$). Exposure to smoke resulted in BAD events at the 6 and 7 hr time events and in the opposite direction (-0.39 to -0.71 mm/$\mu g/m^3$) to those associated with smells.

At this point, the potential mechanisms responsible for the observed biological changes that occurred in association with the various exposure sources must remain speculative. We have previously shown that personal-level exposure to $PM_{2.5}$ and ETS particulate components play a role in causing elevations in BP approximately in a 1-day lag period (Brook et al., 2011a). Many other human and animal studies have shown linkages between ambient $PM_{2.5}$ and endothelial dysfunction, vasoconstriction, and elevations in BP (Brook et al., 2004). In this post hoc analysis, we explored the association between six CV outcomes with several PM sources during numerous

time points over a 24 hour period. Given the numerous associations evaluated, a coherent unifying picture of the effect of each exposure source during the exposure period is difficult to establish. Nonetheless, the presented results demonstrate that various sources of exposure can be associated with many different biological responses, sometimes varying in degree and direction in relation to the acuity of exposure. In addition, this study provides no additional means of understanding the impact of the confounding effects of multiple activities on outcomes due to the data collection process. It has been shown by many studies that particulate exposure can rapidly affect the CV system via three broad pathways: altering autonomic nervous system balance, systemic pro-inflammatory changes that negatively impact vascular function and tone, as well as by direct effects of soluble components reaching the circulation (Brook et al., 2004). It is possible that the various PM sources impact the CV system through different mechanisms. It is also possible that they activate the generalized pathways in a differential manner depending upon the duration and acuity of exposures. In this context, we are not attempting to explain the total sum of the CV responses observed. However, the findings demonstrate in general that various sources of PM might possibly differentially impact the CV system in a manner that is different or that occurs beyond the effects observed simply by characterizing exposure by the 24-hour mean total $PM_{2.5}$ mass inhaled. Given the exploratory nature of these findings, more hypothesis-driven research would be useful.

Acknowledgements

The US Environmental Protection Agency through its Office of Research and Development funded and conducted the research described here under contract 68-D-00-012 (RTI International), EP-D-04-068 (Battelle Columbus Laboratory), 68-D-00-206 and EP-05-D-065 (Alion Science and Technology). It has been subjected to Agency review and approved for

publication. Mention of trade names or commercial products does not constitute an endorsement or recommendation for use. The US EPA acknowledges the staffs of Alion Science and Technology for preparation of sampling media and RTI International for overseeing field data collections. The DEARS cohort is thanked for their participation.

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Table 1. Demographics of DEARS participants and related statistics. Age of one home was not obtained.

Demographic	N[a]	Mean or % of total	Min	Max	SD
General					
Age (years)	49	41.9	19	73	13.9
Age of home (years)	49	69.6	8	120	21.5
Estimated daily time away from home (h)	50	4.6	0	12	3.6
Estimated one-way work commuting time (min)	18	15.0	0	45	11.8
Race					
African-American	27	54.0			
Caucasian	5	10.0			
Other, including Hispanics	18	36.0			
Gender					
Female	37	74.00			
Male	13	26.00			
Employment					
Outside home	18	36.00			
Not outside home	32	64.00			
Home description					
Detached house	44	88.00			
Attached house	4	8.00			
Apartment	2	4.00			

Table 2 is on next page. Ordering was modified to conserve page space.

Activity	Season 4	Season 5	Season 6
Participants (N)	20	27	13
Total Peaks	186	404	144
Candles			
Peaks (N)	21 (6)	53 (5)	9 (3)
Average (μg/m^3)	279.8	100.4	239.7
Range (μg/m3)	35 - 1162.7	31.4 - 599.3	30.5 - 755.4
Car			
Peaks (N)	11 (9)	34 (13)	12 (5)
Average (μg/m^3)	104.6	173.8	2011.4
Range (μg/m3)	42.7 - 354.7	33.6 - 802.7	34.0 - 16371
Cleaning			
Peaks (N)	6 (4)	14 (7)	2 (2)
Average (μg/m^3)	114.4	87.5	346.7
Range (μg/m3)	43 - 219.7	37.6 - 256.0	47.6 - 645.9
Cooking			
Peaks (N)	26 (13)	28 (18)	23 (10)
Average (μg/m^3)	430.6	1047.8	209.3
Range (μg/m3)	47.1 - 4613.1	31 - 17614.4	33.5 - 2556.6
ETS			
Peaks (N)	4 (3)	7 (5)	3 (1)
Average (μg/m^3)	194.7	388.6	5546.3
Range (μg/m3)	58.9 - 372.0	44 - 1499.0	79.9 - 16371
Products			
Peaks (N)	1 (1)	4 (4)	11 (2)
Average (μg/m^3)	74.9	627.6	403.4
Range (μg/m3)	-	72.2 - 844.4	30.5 - 2221.9
Smells			
Peaks (N)	-	10 (2)	6 (2)
Average (μg/m^3)	-	92	518.1
Range (μg/m3)	-	38 - 142.0	43.5 - 2221.9
Smoke			
Peaks (N)	2 (2)	-	-
Average (μg/m^3)	105.1	-	-
Range (μg/m3)	58.9 - 151.4	-	-
Windows			
Peaks (N)	15 (2)	224 (18)	-
Average (μg/m^3)	332.9	346.7	-
Range (μg/m3)	32.7 - 1350	31 - 17614.4	-

Table 3. Statistical Summary of pDR peaks by activity type. Peak values represent number of identified peaks and in ( ) the number of participants associated with these events.

Table 2. Distribution of personal minute-based $PM_{2.5}$ exposures.

$PM_{2.5}$ range[1] (ug/m^3)	Observations	% including non-positives*	% excluding non-positives**
≤ 0	11103	3.37	--
0 < PM ≤ 35	243959	74.07	76.66
35 < PM ≤ 100	54867	16.66	17.24
100 < PM ≤ 1000	18654	5.66	5.86
1000 < PM ≤ 5000	715	0.22	0.22
50000 > PM	45	0.01	0.01

* out of total of 329343 observations; ** out of total of 318240 observations

[1] Daily personal and ambient 24-hr $PM_{2.5}$ averages for population were 18 $\mu g/m^3$ and 16 $\mu g/m^3$, respectively.

Table 4. Multiple linear regression with all seven source factors. Each estimated partial regression coefficient indicates the expected change in $PM_{2.5}$ exposures by the source (hourly binary activities). Bold values indicate associations with p values ≤ 0.05.

	Estimate	Std.Error	P-value	exp(estimate)
(Intercept)	2.36	0.02	0.000	10.59
Cooking	**0.66**	**0.06**	**0.000**	**1.93**
Candles	**0.23**	**0.08**	**0.004**	**1.26**
ETS	**0.82**	**0.13**	**0.000**	**2.27**
Car	0.05	0.06	0.379	1.05
Cleaning	0.16	0.08	0.061	1.17
Windows	**0.80**	**0.04**	**0.000**	**2.23**
Products	**0.41**	**0.09**	**0.000**	**1.51**

Table 5. Cardiovascular changes associated with hourly lags from time of exposure for the cohort fully compliant with monitoring and experiencing low ETS exposures.

Outcome	Activity	Lag (hour)	Total Obs	Non-zero Obs	Risk estimate	SE	P value
BAD	candles	11	94	5	0.44034	0.21436	0.04432
BAD	candles	12	94	5	0.44034	0.21436	0.04432
BAD	car	0	94	27	-0.24424	0.11579	0.0391
BAD	cleaning	6	94	11	0.57504	0.18804	0.00333
BAD	cooking	8	94	34	0.20447	0.07784	0.01092
BAD	products	5	94	11	0.44036	0.1343	0.00174
DBP	candles	11	96	5	-10.02789	3.4524	0.00509
DBP	candles	12	96	5	-10.02789	3.4524	0.00509
DBP	candles	20	96	9	-5.86602	2.1407	0.00801
DBP	candles	21	96	10	-7.21156	2.35749	0.00328
DBP	candles	22	96	13	-7.21156	2.35749	0.00328
DBP	candles	23	96	15	-4.87596	2.1369	0.02595
DBP	candles	7	96	12	-8.33988	2.90584	0.00561
DBP	car	10	96	17	-5.40153	2.45013	0.0312
DBP	cooking	1	96	39	4.3032	1.24811	0.00102
DBP	cooking	18	96	2	10.84	4.36531	0.01574
DBP	cooking	21	96	12	-4.023	1.96707	0.04509
DBP	cooking	22	96	23	-4.04429	1.38593	0.0049
DBP	windows	11	96	36	-4.25507	1.48987	0.00583
DBP	windows	12	96	20	-5.64067	1.86701	0.00365
DBP	windows	20	96	35	-3.30064	1.29443	0.01326
DBP	windows	21	96	43	-3.30064	1.29443	0.01326
DBP	windows	22	96	46	-2.82817	1.29842	0.03321
FMD	cleaning	13	89	1	8.74875	4.27571	0.04545
FMD	cleaning	19	89	2	9.55188	4.48252	0.0375
FMD	ets	6	89	5	12.6426	4.18268	0.00378
FMD	products	5	89	11	-5.65527	2.02437	0.00712
HR	candles	1	96	18	-10.14504	4.05275	0.01495
HR	candles	2	96	17	-8.82454	4.35757	0.04717
HR	candles	3	96	17	-8.82454	4.35757	0.04717
HR	candles	4	96	16	-8.82454	4.35757	0.04717
HR	candles	5	96	15	-8.82454	4.35757	0.04717
HR	candles	6	96	15	-8.82454	4.35757	0.04717
HR	car	16	96	4	11.10435	4.94518	0.02831
HR	car	19	96	5	12.09929	4.83695	0.01502
HR	car	20	96	8	11.96168	4.00022	0.00399
HR	car	21	96	8	9.3397	4.56545	0.04503
HR	cooking	18	96	2	17.85385	6.74525	0.01029
HR	cooking	4	96	45	5.59676	2.00911	0.00708
HR	ETS	3	96	6	12.40679	4.94984	0.01483
HR	products	0	96	14	-7.3972	3.62152	0.04535
HR	products	10	96	12	10.40859	3.32382	0.00265
HR	products	5	96	11	8.73885	3.42336	0.01317
HR	products	7	96	10	-7.09975	3.05314	0.02334
HR	products	9	96	14	8.02872	2.83331	0.0062
NMD	car	7	46	26	-7.4571	2.32641	0.00345
NMD	windows	12	46	20	-6.44918	3.00153	0.04079
SBP	candles	10	96	8	-16.4932	5.70719	0.0053
SBP	candles	11	96	5	-12.69826	5.51621	0.02471
SBP	candles	12	96	5	-12.69826	5.51621	0.02471
SBP	candles	20	96	9	-8.09728	3.43078	0.02143
SBP	candles	21	96	10	-7.91784	3.86718	0.04486
SBP	candles	22	96	13	-7.91784	3.86718	0.04486
SBP	candles	23	96	15	-7.24653	3.37889	0.03591
SBP	candles	7	96	12	-14.99983	4.43911	0.00126
SBP	candles	8	96	10	-16.4932	5.70719	0.0053
SBP	candles	9	96	9	-16.4932	5.70719	0.0053
SBP	cooking	1	96	39	6.13833	1.9621	0.00268
SBP	cooking	22	96	23	-4.52213	2.24233	0.04806
SBP	products	6	96	8	10.37459	4.49231	0.02427
SBP	windows	12	96	20	-8.66738	2.94358	0.00455

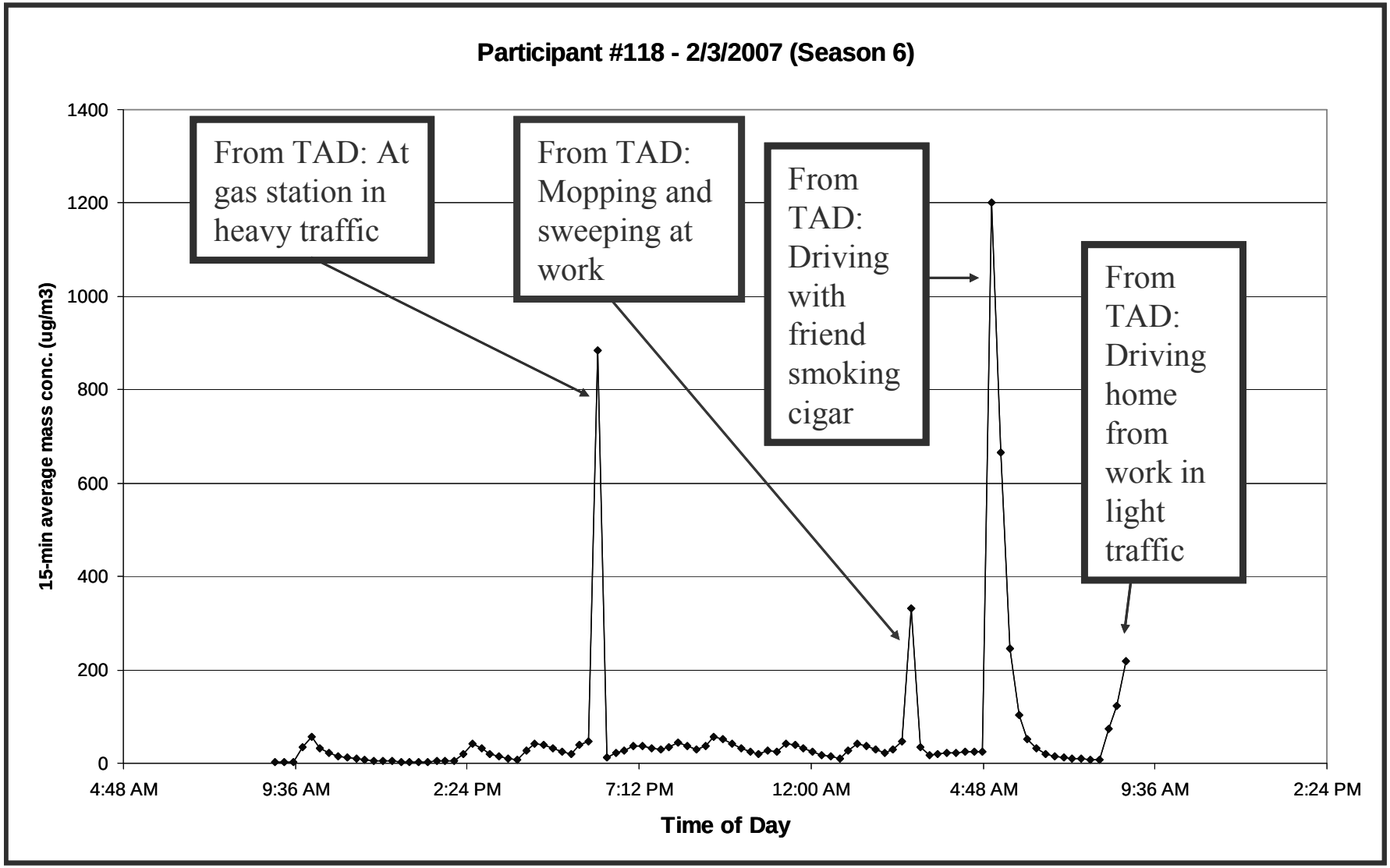

Figure 1. Example of pDR data combined with participant time activity data (TAD)

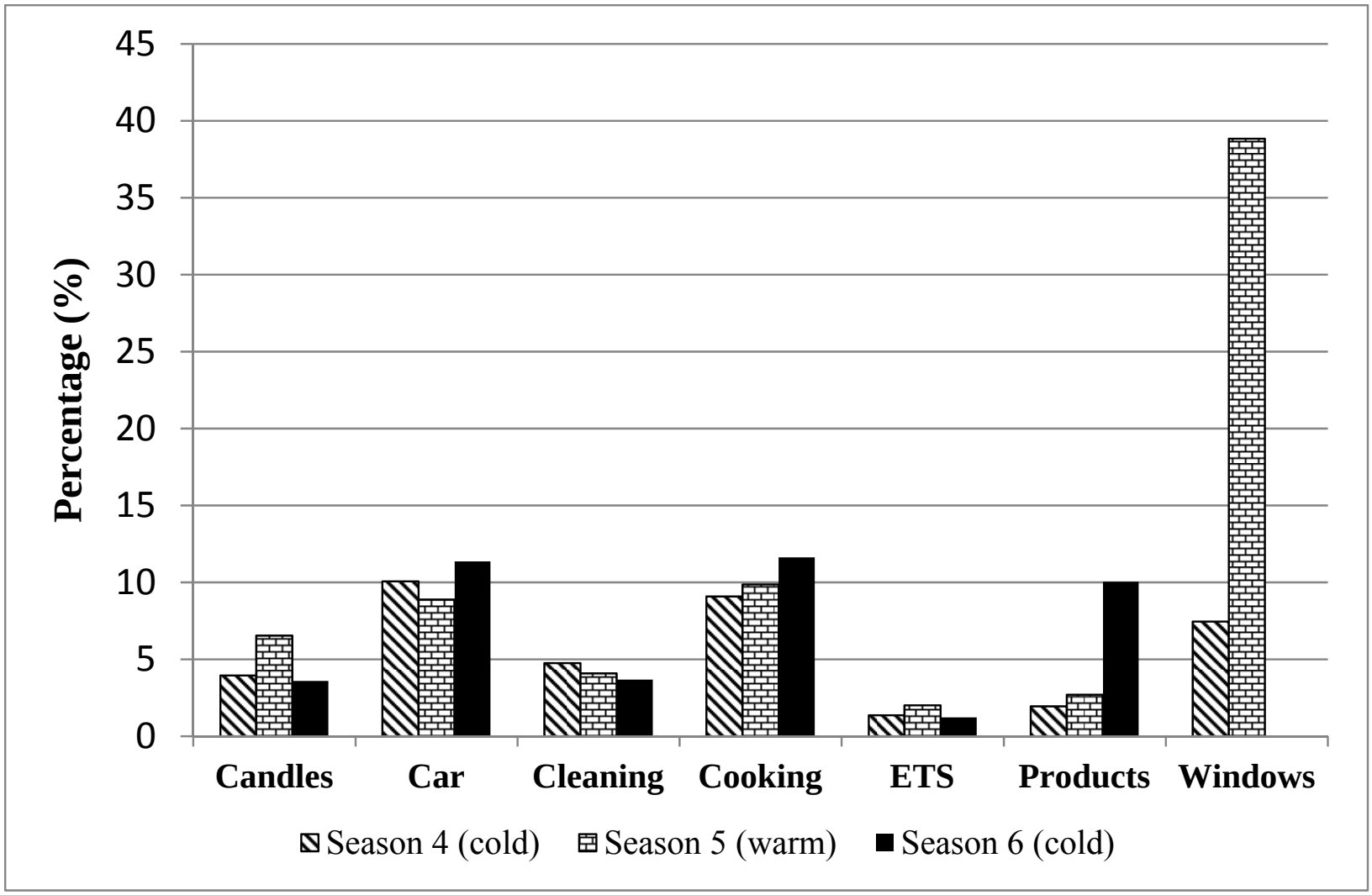

Figure 2. Percent of hourly activity by season for each source type

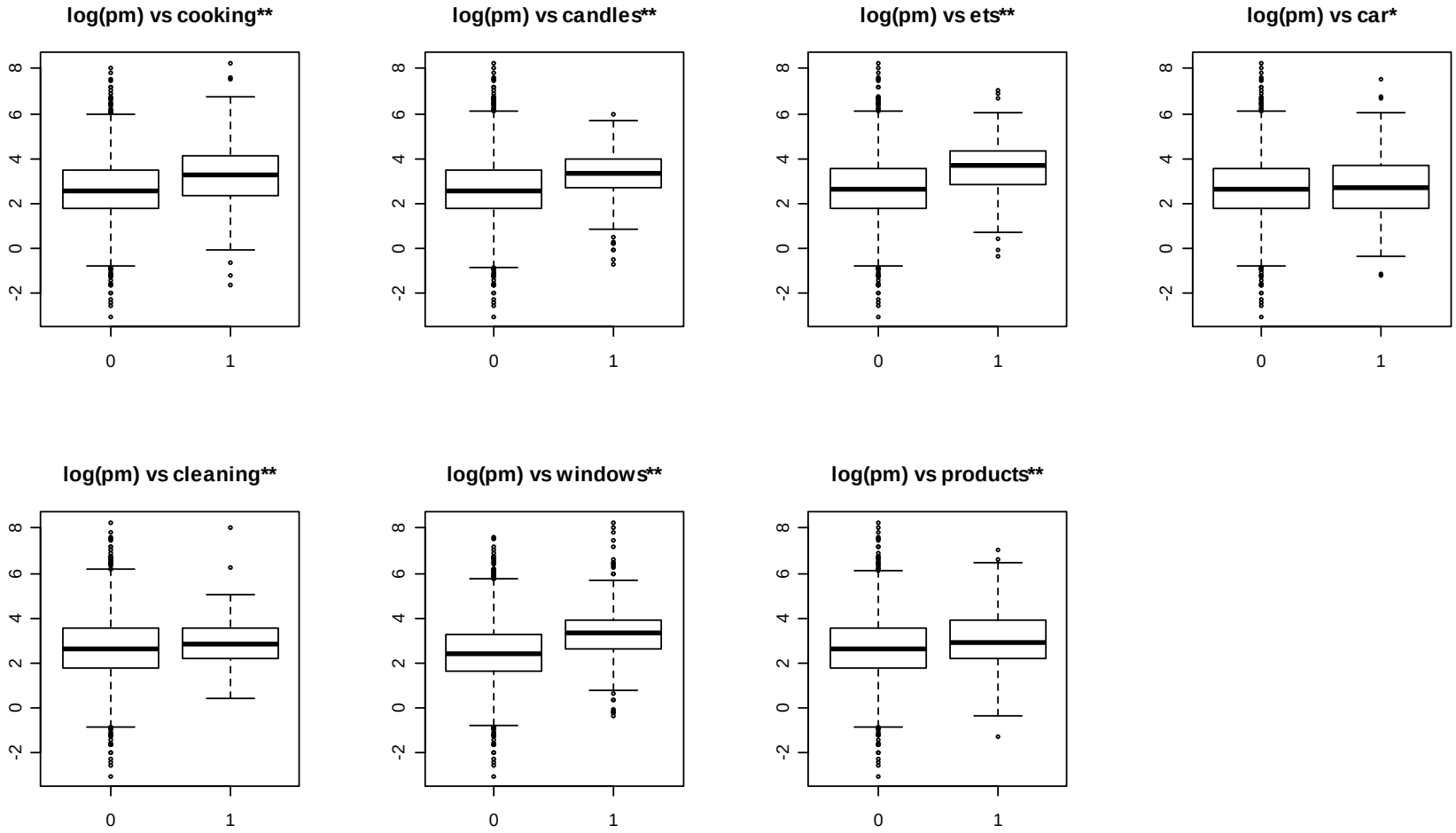

Figure 3. Comparison of hourly $PM_{2.5}$ exposure by source types based on a total of 5597 paired measurements across all seasons and all participants. The Y-axis represents the natural logarithms of $PM_{2.5}$ exposures in $\mu g/m^3$ (positive values only included), and the X-axis indicates hourly activities dichotomized.

Supplemental Materials

Table S1. Cardiovascular changes (SBP, DBP and HR) associated with hourly lags from time of exposure for the "All Subjects" cohort.

Outcome	Activity	Lag (hour)	Total Obs	Non-zero Obs	Risk estimate	SE	P value
SBP	car	11	253	14	-8.1257	2.4502	0.0011
SBP	car	15	253	3	16.2956	5.1965	0.0020
SBP	car	16	253	4	-13.2548	4.4042	0.0030
SBP	car	17	253	5	10.1415	3.9348	0.0107
SBP	car	18	253	5	10.1186	4.4168	0.0231
SBP	ETS	12	253	2	-33.0270	6.3423	0.0000
SBP	ETS	13	253	3	-17.4616	5.1009	0.0008
SBP	ETS	14	253	3	-21.4305	5.0480	0.0000
SBP	ETS	15	253	4	-19.4906	4.5494	0.0000
SBP	ETS	16	253	3	-19.4814	6.4337	0.0028
SBP	ETS	17	253	2	15.6492	5.8408	0.0080
SBP	ETS	7	253	3	-11.8198	4.8150	0.0150
SBP	products	19	253	5	-8.6178	4.3601	0.0495
SBP	products	2	253	17	-8.0413	2.6812	0.0031
SBP	products	20	253	6	-10.0934	4.0551	0.0137
SBP	products	21	253	8	-6.7798	3.3587	0.0449
SBP	smoke	1	253	1	18.9488	8.2970	0.0235
SBP	smoke	3	253	1	34.2058	8.1118	0.0000
SBP	windows	19	253	27	-4.4085	2.0233	0.0306
DBP	car	11	253	14	-4.2344	1.7293	0.0152
DBP	car	16	253	4	-7.3215	3.1131	0.0197
DBP	car	22	253	14	-3.7045	1.7188	0.0324
DBP	car	7	253	26	-3.2667	1.2626	0.0104
DBP	ETS	12	253	2	-17.9199	4.5677	0.0001
DBP	ETS	13	253	3	-10.4801	3.6167	0.0042
DBP	ETS	14	253	3	-13.5354	3.5706	0.0002
DBP	ETS	15	253	4	-11.4770	3.2546	0.0005
DBP	ETS	16	253	3	-11.9034	4.5168	0.0091
DBP	ETS	21	253	2	-8.8952	4.1141	0.0318
DBP	products	20	253	6	-6.5027	2.8379	0.0230
DBP	products	21	253	8	-5.6088	2.3371	0.0174
DBP	smells	17	253	1	12.8864	5.8017	0.0275
DBP	smoke	9	253	1	12.3308	5.8168	0.0353
DBP	windows	11	253	36	-3.1336	1.4046	0.0268
DBP	windows	19	253	27	-2.9674	1.4112	0.0368
HR	car	20	253	8	6.6056	2.6000	0.0119
HR	cooking	4	253	45	4.4991	1.2877	0.0006
HR	ETS	3	253	6	6.7604	2.9422	0.0227
HR	ETS	4	253	1	16.2445	7.2565	0.0263
HR	ETS	7	253	3	8.5232	4.1418	0.0410
HR	products	5	253	11	7.4621	2.4325	0.0025
HR	smells	23	253	2	11.6153	5.0607	0.0228
HR	smoke	22	253	1	16.2445	7.2565	0.0263
HR	windows	23	253	48	3.2498	1.4401	0.0252

Table S2. Cardiovascular changes (BAD, FMD and NMD) associated with hourly lags from time of exposure for the "All Subjects" cohort.

Outcome	Activity	Lag (hour)	Total Obs	Non-zero Obs	Risk estimate	SE	P value
BAD	candles	0	232	20	0.1472	0.0736	0.0472
BAD	products	0	232	14	0.1978	0.0820	0.0170
BAD	products	1	232	12	0.2453	0.0973	0.0126
BAD	products	12	232	3	-0.3631	0.1438	0.0125
BAD	products	14	232	1	-0.4987	0.2494	0.0471
BAD	products	3	232	15	0.1665	0.0811	0.0416
BAD	products	5	232	11	0.2948	0.0842	0.0006
BAD	products	6	232	8	0.2209	0.0992	0.0272
BAD	products	8	232	13	0.2269	0.0924	0.0150
BAD	smells	0	232	5	0.3669	0.1437	0.0115
BAD	smells	10	232	2	0.3964	0.1981	0.0469
BAD	smells	3	232	5	0.3866	0.1354	0.0048
BAD	smells	4	232	7	0.2094	0.1044	0.0465
BAD	smells	5	232	7	0.3614	0.1183	0.0026
BAD	smells	6	232	6	0.4131	0.1288	0.0016
BAD	smells	7	232	5	0.2753	0.1299	0.0355
BAD	smells	8	232	5	0.5391	0.1538	0.0006
BAD	smells	9	232	3	0.7327	0.1897	0.0002
BAD	smoke	6	232	2	-0.3904	0.1705	0.0233
BAD	smoke	7	232	2	-0.7080	0.1916	0.0003
FMD	candles	2	221	17	-3.2475	1.4964	0.0314
FMD	candles	3	221	17	-3.2475	1.4964	0.0314
FMD	ETS	19	221	1	11.8172	4.6609	0.0122
FMD	products	0	221	14	-3.2697	1.4712	0.0276
FMD	products	1	221	12	-4.0394	1.7240	0.0203
FMD	products	5	221	11	-4.5520	1.5689	0.0042
FMD	smells	10	221	2	-9.4687	3.6218	0.0098
FMD	smells	8	221	5	-6.8030	2.8195	0.0169
FMD	smells	9	221	3	-7.9058	3.3621	0.0199
FMD	smoke	7	221	2	13.5899	3.5884	0.0002
FMD	smoke	8	221	1	11.8172	4.6609	0.0122
NMD	car	7	109	26	-6.4399	1.8373	0.0008
NMD	cleaning	1	109	9	7.5176	2.6388	0.0057
NMD	cleaning	8	109	16	-7.2476	2.5657	0.0061
NMD	cooking	11	109	13	8.2117	4.0989	0.0488
NMD	windows	12	109	20	-3.6395	1.8232	0.0496