Anti-perspirant deodorant particulate matter temporal concentrations during home usage.

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Highlights

- Accurate PM concentration measurements can be obtained using low-cost monitors
- Aerosol anti-perspirant deodorants have a long-lasting adverse effect on indoor air quality
- Poor ventilation maintains very high PM concentrations for > 2 hours after spray
- High PM concentrations are detected in rooms away from the point of aerosol use
- Anti-perspirant exposure should be considered in managing health conditions

Abstract

Particulate matter (PM) in ambient air is associated with many adverse health outcomes. Although many anthropogenic activities are associated with PM release in indoor settings, dispersion and persistence of PM is poorly understood. In this study, concentration, persistence and dispersion of $PM_{2.5}$ and PM_{10} released following aerosol antiperspirant use were measured in a bathroom environment under several door and window ventilation conditions, and in a nearby bedroom.

Daily mean concentrations were elevated in all experimental conditions compared to the control, but varied depending on the ventilation condition. The daily mean concentrations exceeded the WHO daily mean guideline values when there was little or insufficient ventilation in the bathroom, whereas ventilation through opening doors or windows prevents exceedances. After spraying, mean peak PM concentrations were lowest in the bathroom when the door and window were left open.

Introducing ventilation through opening the bathroom door and/or window reduced PM concentrations by > 93% 10 minutes after spray release, compared to reductions of 60% and 77% for PM2.5 and PM10, respectively, with no ventilation. Opening the bathroom window significantly increased peak PM concentration in the bedroom relative to leaving the window closed, suggesting increased dispersion of PM from bathroom to bedroom.

Keywords

Particulate matter, deodorant anti-perspirant, aerosol, exposure, respiratory health

1. Introduction

Particulate matter (PM) is a term that describes microscopic, inhalable particles, which are important components of outdoor and indoor pollution. PM can be classified by diameter: PM_{10}, $PM_{2.5}$, PM_1 and ultrafines, which describe particles <10 µm, <2.5 µm, <1 µm and <100 nm in aerodynamic diameter, respectively (World Health Organization, 2016).

Epidemiological and toxicological research has informed World Health Organization (WHO) guideline concentrations for maximum annual average (PM_{10} = 20 $\mu g/m^3$; $PM_{2.5}$ = 10 $\mu g/m^3$) and daily average (PM_{10} = 50 $\mu g/m^3$; $PM_{2.5}$ = 25 $\mu g/m^3$) exposure to ambient particulates. However, evidence has indicated that for PM there is no maximum safe level or threshold for particle exposure, to avoid adverse health effects (WHO, 2013), and the WHO is currently re-evaluating the guidelines for both $PM_{2.5}$ and PM_{10} (WHO, 2016).

The German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area (Maximale Arbeitsplatz-Konsentration (MAK) Comission, 2012) indicates that healthy individuals can clear particles >7 µm in diameter from the tracheobronchial compartment within 24 h and that particles >15 µm are deposited almost exclusively in the nose, pharynx or larynx (MAK Comission, 2012). Inhaled PM_{10} can penetrate into the lung and is deposited in the upper airways and nasal cavities (Fiordelisi et al., 2017), and is thought to induce upregulation of the pro-inflammatory cytokine interleukin (IL)-8, cytotoxic cell responses and deoxyribonucleic acid (DNA) damage through reactive oxygen species (ROS) generation (Van Den Heuvel et al., 2016). $PM_{2.5}$ and smaller particles penetrate deeper into the lung and can be deposited in the alveolar regions (Fiordelisi et al., 2017; James et al., 1991), where they trigger inflammatory responses, causing genetic mutations and epigenetic modifications that can result in lung carcinoma (Li et al., 2017; Miller et al., 2017). These fine and ultrafine particles have the potential to cross

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Abbreviations DNA: Deoxyribonucleic acid; IL: interleukin; MAK: Maximale Arbeitsplatz-Konsentration; PM: Particulate Matter; PPM: parts per million; ROS: reactive Oxygen Species; WHO: World Health Organizsation;

from lung alveoli to the blood stream, from where they can cause and exacerbate chronic cardiovascular disease and cause carcinomas that are distal to the lung (Arden Pope et al., 2011; Fiordelisi et al., 2017; Jalaludin and Cowie, 2014).

Inhalation exposure of PM is detrimental to health and contributes to early death (Lim et al., 2012; Royal College of Paediatrics and Child Health, 2016; WHO, 2018). In mice, sub-chronic low-level exposure to PM can induce an inflammatory response causing an adverse effect on lung health (Chan et al., 2019). Inhaled PM can cause acute respiratory irritation in humans (Wolkoff and Nielsen, 2017), and narrowing of airways, leading to the exacerbation of chronic respiratory diseases such as chronic obstructive pulmonary disorder (COPD) (Doneva et al., 2019; Morantes-Caballero et al., 2019) and asthma (Baldacci et al., 2015; Guarnieri and Balmes, 2014; Keet et al., 2018). Exposure to PM has also been associated with increased risk and mortality in cardiovascular disease (Arden Pope et al., 2011; Franklin et al., 2015; Künzli and Tager, 2005; Miller et al., 2017), increased risk for lung cancer (Arden Pope et al., 2011; Hamra et al., 2014; Künzli and Tager, 2005), decreased survival in Stage I breast cancer (DuPre et al., 2019), increased risk of type II diabetes incidence (Liu et al., 2019), increased risk of dementia (Peters et al., 2019) and an increased likelihood of low birth weight (Smith et al., 2017).

In the UK, air quality standards for outdoor air are enshrined in the Air Quality Standards regulations and Local Air Quality Management regime (Barnes et al., 2018), which are based largely on the WHO guideline levels. $PM_{2.5}$ is a notable exception as the WHO guideline for this pollutant is an annual average of 10 $\mu g/m^3$, whereas the UK (with the exception of Scotland) has adopted an annual average of 25 μ/gm^3. The WHO has indicated that as there is no convincing evidence for the difference in the level of risk associated with indoor as compared to outdoor sources of PM_{10} and $PM_{2.5}$, the outdoor air standards are equally applicable to indoor air (WHO, 2006). However, despite this, statutory pollutant concentration thresholds for the protection of health in indoor environments are not included in UK law, with the UK and Welsh Governments going no further than highlighting the risks and recommending precaution in their recently published Clean Air Plans (Department for Environment Food & Rural Affairs, 2019; Welsh Government, 2019).

Indoor air pollution is ranked 4th as a contributing factor towards global disease burden (Lim et al., 2012). Anthropogenic activities are known to have a significant impact on indoor air quality: smoking (Fiala et al., 2012; Glytsos et al., 2010; Rosen et al., 2015; Semple et al., 2015, 2013), cleaning (Abt et al., 2000; He et al., 2004; Kamens et al., 1991; Nazaroff and Weschler, 2004; Wolkoff et al., 1998), cooking (Abt et al., 2000; Géhin et al., 2008; He et al., 2004; Hussein et al., 2005), solid-fuel burning (Hussein et al., 2005; Salthammer et al., 2014), choice of furnishing (Jones, 1999), and use of personal care products, including deodorants (Lefebvre et al., 2012) have all been shown to contribute indoor PM concentrations. Aerosol anti-perspirant and deodorant use in developed countries is widespread and is increasing in proportion to non-aerosol alternatives ("Cosmetics Business," 2016). A self-reporting-based study found that > 91% of respondents reported regularly using deodorants, and of these, approximately 79% regularly use aerosol/spray forms of deodorant (Biesterbos et al., 2013). Aerosol deodorants are usually composed of the active ingredient(s), and a solvent & propellant mix, which, when released from the container, cause the contents to disperse into small particles (Rothe et al., 2011). This method of anti-perspirant delivery is highly inefficient, with only 11.4% of the product reaching the skin, and 89.6% entering the air where it can then be inhaled (Steiling et al., 2012).

Previous research into the size characteristics and persistence of particles released during the activation of aerosol-type sprays is limited. Particle size distribution studies of product released from household aerosol sprays showed an increase in particle size over time after spraying, possibly due to particle nucleation and coagulation (Afshari et al., 2005; Cuizas et al., 2015). Another common household aerosol, hair spray, has been shown to increase ambient $PM_{2.5}$ concentration by up to 11.6-fold (Glytsos et al., 2010). Therefore, it is reasonable to hypothesise that use of aerosol anti-perspirant could have a similar affect on indoor air quality. To the authors' knowledge, the impact and persistence of released anti-perspirant-associated PM in the indoor environment has yet to be determined.

The Dylos DC1100 Pro ("Dylos," 2019) is an optical particle monitor that records particle count number (PCN). Dylos instruments may be co-located with a calibrated instument capable of measuring PM concentrations for calculation of a conversion factor from PCN to PM concetration with R^2 values ranging from 0.58 to 0.99 (Dacunto et al., 2015; Holstius et al., 2014; Jones et al., 2016; Klepeis et al., 2013; Northcross

et al., 2013; Robert J. Vercellino et al., 2018; Semple et al., 2015, 2013; Sousan et al., 2016; Steinle et al., 2015),The relationship between PM count and PCN is dependent on the PM source and experimental conditions. Lower correlations were observed in field studies of environmental & occupational pollutants (Steinle et al., 2015, Jones et al., 2016, Holstius et al., 2014), compared to studies of PM release under laboratory conditions (Semple et al., 2013, Kleipis et al. 2013, Sousan et al., 2016, Vercellino et al., 2018) or in the home environment (Semple et al., 2015). The available evidence shows that Dylos optical particle counters are suitable for determining PM concentrations released from aerosol in an indoor home environment.

This study aimed to use a Dylos optical particle counter to determine concentrations of PM_{10} and $PM_{2.5}$ associated with the use of an aerosol anti-perspirant in a standard home environment by comparing peak PM concentrations to WHO daily mean guideline thresholds for PM_{10} and $PM_{2.5}$, and the time taken for the PM concentrations to return to room background levels in multiple ventilation conditions.

2. Materials and Methods

2.1. Deodorant product

The product used in this study was Sure Men Williams Racing Anti-perspirant Deodorant aerosol (250ml) manufactured by Unilever, Wirral CH63 3JW UK. Anti-perspirant ingredients as listed by the manufacturer were: butane, isobutane, propane, aluminium chlorohydrate, cyclopentasiloxane, PPG-14 butyl ether, parfum, disteardimonium hectorite, BHT, propylene carbonate, caprylic/capric triglyceride, gelatin crosspolymer, cellulose gum, benzyl salicylate, sodium benzoate, hydrated silica, aqua, sodium starch octenylsuccinate, maltodextrin, hydrolysed corn starch, silica, alpha-isomethyl ionone, benzyl alcohol, butylphenyl methylpropional, citronellol, geraniol, limonene, linalool.

2.2. Experimental location

Particle counts were recorded continuously over a period of 18 days in an upstairs (first floor) bathroom of a modern, detached four-bedroom house. The bathroom was constructed four years previously and contains a single inward opening standard panel door and a window with a single fixed casement pane and a single outward opening casement pane (Figure 1). The bathroom has a 102 mm extractor fan (Primeline model

169 PEF4020) permanently fitted into the ceiling that vents air outside via an external wall.
170 The fan has a run-on timer and has an extraction rate of 23 lh^{-1} (85 m^3h^{-1}). The
171 dimensions of the rooms are described in Table 1. Particle counts were also recorded
172 on separate days in a bedroom located opposite to the bathroom having a single
173 inward opening standard panel door directly opposite to the bathroom door.

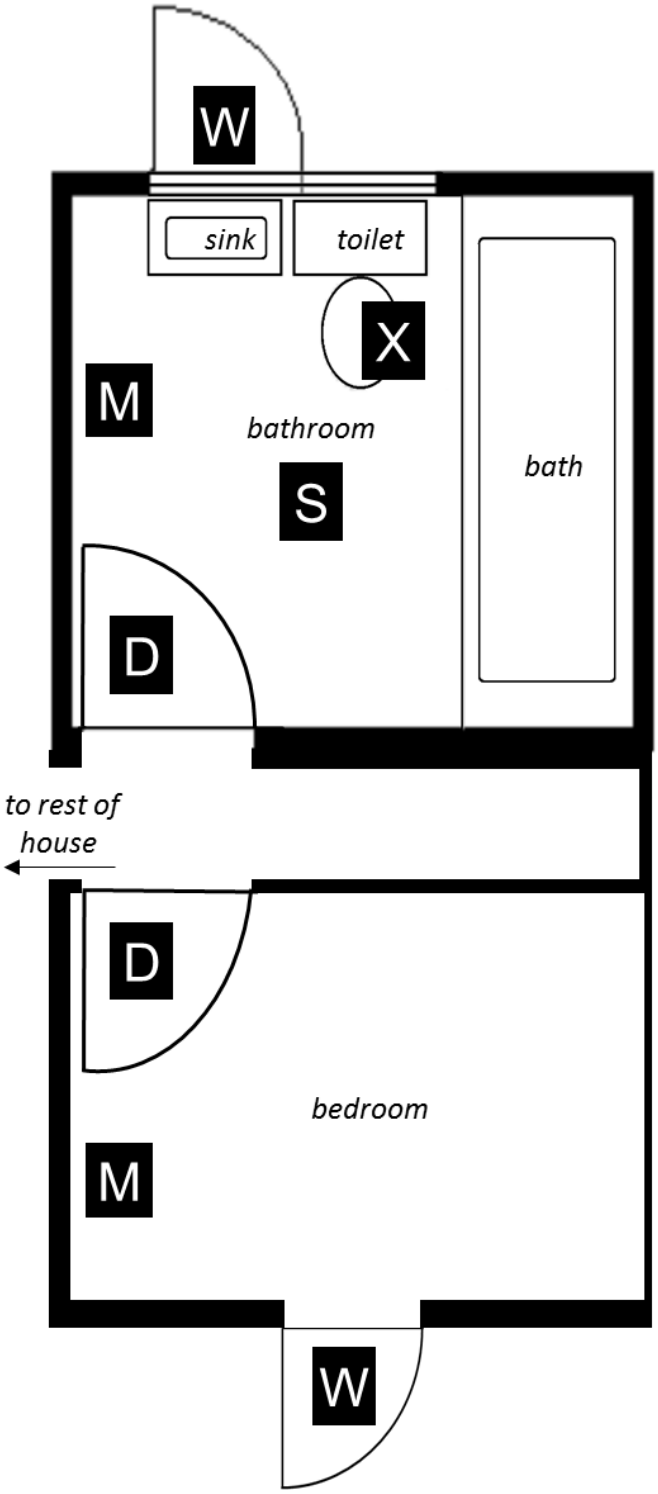


Figure 1 Location plan where experiment was conducted with positions of monitor in each room (M), spray (S), doors (D), windows (W) and extractor fan (X) shown (not to scale).

Room	Dimensions	Additional Information
Bathroom	2300 * 1900 * 2300 mm	Total vol.: 10.1 m^3
Standard-Panel Door	706 * 1907 mm	Total area: 1.4 m^2
Open-Panel Window	500 * 804 mm	Total area: 0.4 m^2
		1000mm from floor
Extractor Fan	100 * 100 mm	Extraction rate: 85 m^3h^{-1}
Bedroom	3300 * 3100 * 2300 mm	Total vol.: 23.5 m^2
Standard-Panel Door	706 * 1907 mm	Total area: 1.4 m^2

Table 1: Dimensions of experimental rooms

2.3. Experimental conditions

Particle counts for PM_{10} and $PM_{2.5}$ were measured in parts per million (PPM) using a Dylos DC1100 optical particle monitor with PC interface ("Dylos," 2019). PM concentrations were measured by a Trotec PC220 device with PC interface, which conforms to ISO 201501-4, and measures PM_{10} and $PM_{2.5}$ concentration (μgm^{-3}) with a 100% counting efficiency for particles greater than 0.45µm in size and a 5% coincidence loss (percentage error when counting large particle concentrations). An extensive literature search and review of information made available by manufacturers did not give absolute values of uncertainty measurements for either device. In order to factor in measurement uncertainty into our analysis, we estimated the uncertainty in the Trotec PC220 measurements as ±5% as per the coincidence loss rate reported by the manufacturer, and uncertainty in the Dylos DC1100 measurements was approximated at ±10%, taken as a central value of the coefficients of variation in Dylos monitor measurements as reported by Sousan et al. (2016). To account for this measurement uncertainty, replicates of each experimental condition were taken.

The Dylos monitor was connected to a Windows 10 tablet computer via serial port; timestamped data were recorded using CoolTerm software (Meier, 2019). Relative humidity and temperature (°C) were recorded using a DHT11 type sensor connected to an Arduino Uno microcontroller, and timestamped data were stored at minute intervals to a text file on the tablet computer. Manufacture accuracy estimates for humidity and temperature are ±5% relative humidity and ±2%°C respectively. All

particulate, temperature and humidity data were combined, using corresponding timestamps, in Microsoft Excel. Using correlation analysis, associations between minute changes in temperature and humidity and PM concentration were assessed for magnitude and significance.

Seven experimental conditions were tested to demonstrate the impact of ventilation on PM aerosol persistence following spraying of the deodorant. To mimic user experience, the spray was activated twice in the approximate centre of the room for a period of 3 seconds (6 seconds in total), as per the product instructions, at a height of approximately 1.5 m (to mimic axillary use). The Dylos monitor was placed at a height of 1.5m to mimic exposure at breathing height, at position 'M' in the bathroom (Figure 1). Five ventilation conditions in the bathroom were evaluated in triplicate:

1. No ventilation: window closed, bathroom door closed, extractor fan off
2. Ventilation: bathroom door fully open, window closed, extractor fan off
3. Ventilation: bathroom door fully open, window open 300mm from casement, extractor fan off
4. Ventilation: bathroom door closed, window open 300mm from casement, extractor fan off
5. Ventilation: bathroom door closed, window closed, extractor fan on

Baseline $PM_{2.5}$ and PM_{10} concentrations for all experimental conditions were established by measuring PM levels for 1 minute prior to aerosol spray. Following the spray, for all experimental condition replicates, 1-minute average particle counts were then recorded continuously for a time period of 180 minutes. To minimise air flow disturbance, following each aerosol spray, the experimenter immediately retreated downstairs ensuring that all doors on the ground floor were closed and thus became completely isolated from the experiment. Doors to all other rooms and windows located on the first floor remained closed during the experiment unless stated otherwise. Additional experimental conditions were tested to determine PM exposure in a nearby bedroom. The Dylos monitor was placed in the bedroom at 5m from the point of spray in the bathroom, in a direct line of sight, at a height of 1.5m, again to mimic breathing height (Figure 1). The conditions evaluated in triplicate were:

6. Bathroom and bedroom doors fully open, bathroom window closed, bathroom fan off

7. Bathroom and bedroom doors fully open, bathroom window open 300mm from casement, bathroom fan off.

The window in the bedroom remained closed throughout the experiment. All surfaces (including the floor) in the bathroom were cleaned with a damp cloth prior to every experimental replicate. Key parameters recorded in each condition were (i) peak PM concentration, (ii) time to peak PM concentration (minutes), (iii) initial rate of decline of suspended PM concentration, and (iv) time to return to threshold concentrations (minutes). The WHO guidelines for annual (10 μgm^{-3} $PM_{2.5}$: 20 μgm^{-3} PM_{10}) and daily average (25 μgm^{-3} $PM_{2.5}$: 50 μgm^{-3} PM_{10}) were used as the threshold concentrations. The annual average was chosen over the daily average due to the high likelihood that a product such as aerosol deodorant would be used daily, so any exposure to released PM is likely to be recurrent.

2.4. Calculation of PM_{10} and $PM_{2.5}$ concentration from particle count

As the Dylos monitor only provides particle count data, a co-location study was performed with the Dylos and the Trotec PC220 device. The devices were placed adjacent to each other at the same height of 1.5m in the same bathroom under the same 5 experimental conditions (two replicates) and data collected at minute intervals for a duration of one hour which was the maximum recording time permitted by the Trotec. Due to the limited recording time of the Trotec, and the need to reset the device every hour, it would not have been practical to use this device over the required time frames, and human activity required for the device resetting process would have disturbed air flow. Timestamped Trotec PC220 data were retrieved and aligned with corresponding Dylos data. Using the aligned data, a standard equation was derived for conversion of $PM_{2.5}$ and PM_{10} particle count data concentration in μgm^{-3}

2.5. Statistical Analysis

Statistical significance of differences between the means measures of peak PM concentration (μgm^{-3}), time to peak PM concentration (minutes), time to return to threshold PM concentrations (minutes), and PM concentrations 10 minutes after peak PM concentration (μgm^{-3}) for each experimental condition and the no ventilation condition were assessed by t-test. Correlation was assessed using Pearson's coefficient.

3. Results

3.1. Temperature and Humidity Monitoring

Monitoring of temperature and humidity showed that a significant ($p < 0.05$) negative relationship exists between temperature and humidity across all experimental conditions. Whilst temperature and humidity remained relatively stable, with mean measurements of 21.80°C (SE ±0.03) and 67.05% (SE ±0.10) across all measurement periods, respectively, small changes were observed during the course of each measurement period. Humidity was significantly ($p < 0.05$) lower in the 'Fan only' experimental condition compared to all other ventilation conditions; all other conditions did not show significant differences in temperature or humidity from any other condition.

To ensure that uncertainty in humidity or temperature measurements would not lead to particulate matter measurement error, the rate of change in recorded temperature and humidity was evaluated across all experimental conditions, before investigating associations between temperature, humidity and PM concentration measurements. It was found that the mean rate-of-change in temperature and humidity measurements from minute-to-minute were very small, at 0.003°C (SE ± 0.008) and -0.024% (SE ± 0.032), respectively, markedly lower than the published uncertainty values for the DHT11 sensor of ± 2°C and ± 5%. Large fluctuations in temperature and humidity measurements from minute to minute could indicate unreliable measurements. Therefore, due to the lack of extreme fluctuations in minute-by-minute measurements, it was assumed that the sensor was providing reliable data on the temperature and humidity trends. Across all experimental conditions changes in temperature and humidity measurements were gradual.

Next, the impact of changes to the environmental conditions on PM concentration measurements was investigated. No statistically significant relationships were found when correlating the minutely rate-of-change values for temperature and humidity to the minutely rate-of-change PM concentration measurements in the control condition: only the control condition was examined to discount the effect of aerosol spray release on PM concentration.

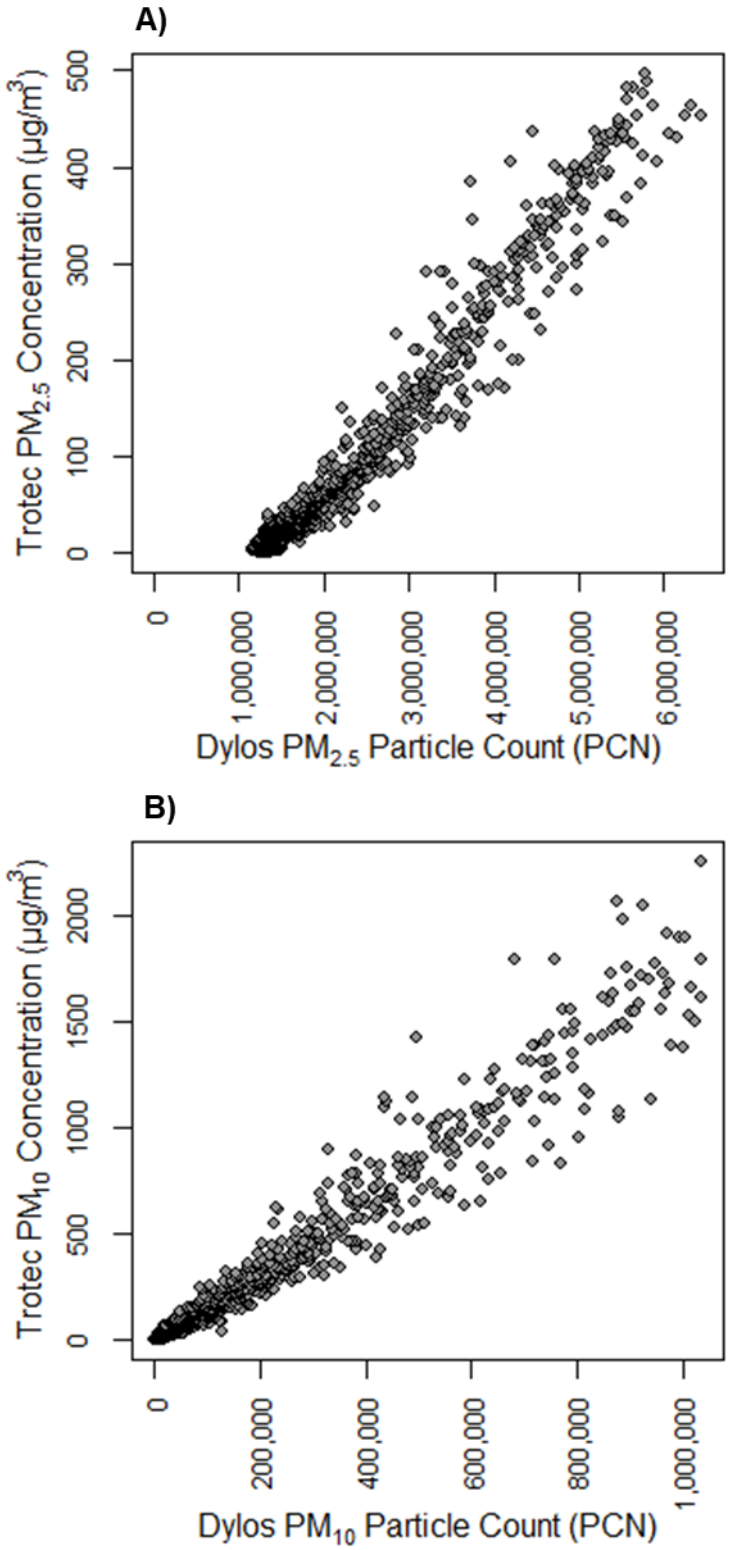
3.2. Calculation of PM_{10} and $PM_{2.5}$ concentrations from particle count data

The $PM_{2.5}$ and PM_{10} particle count and concentration (μgm^{-3}) data recorded by the Dylos and Trotec devices showed a strong linear relationship (Figure 2). This linear relationship was found to be statistically significant ($p < 0.05$) for both $PM_{2.5}$ and PM_{10} with R^2 values of 0.93 and 0.87, respectively.

The following linear equations were derived to convert particle count data to μgm^{-3}:

$[PM_{10}]\ \mu gm^{-3} = 0.0021 \times \text{particle count} - 43.92$ (i)

$[PM_{2.5}]\ \mu gm^{-3} = 0.00009 \times \text{particle count} - 114.24$ (ii)



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307 Figure 2: Co-located readings of PCN and PM concentration (μgm^{-3}) from the Dylos
308 and Trotec devices, respectively, for $PM_{2.5}$ (A), and PM_{10} (B). The Linear relationships
309 show a strong fit between the data from both devices with R^2 in excess of 0.85 for both
310 $PM_{2.5}$ and PM_{10}.

311

3.3. Background room PM concentrations

Mean background levels of $PM_{2.5}$ at 4µgm^{-3} (SE ± 0.12) and PM_{10} at 9 µgm^{-3} (SE ± 0.36) in the bathroom were shown to be consistently below the WHO daily mean threshold values of 25 and 50 µgm^{-3} respectively (Figure 3). Mean background levels in the adjacent bedroom were at similar levels to the bathroom for $PM_{2.5}$ at 4 µgm^{-3} (SE ± 0.26) but, whilst still well below the WHO threshold level, approximately doubled for PM_{10} at 15 µgm^{-3} (SE ± 0.97).

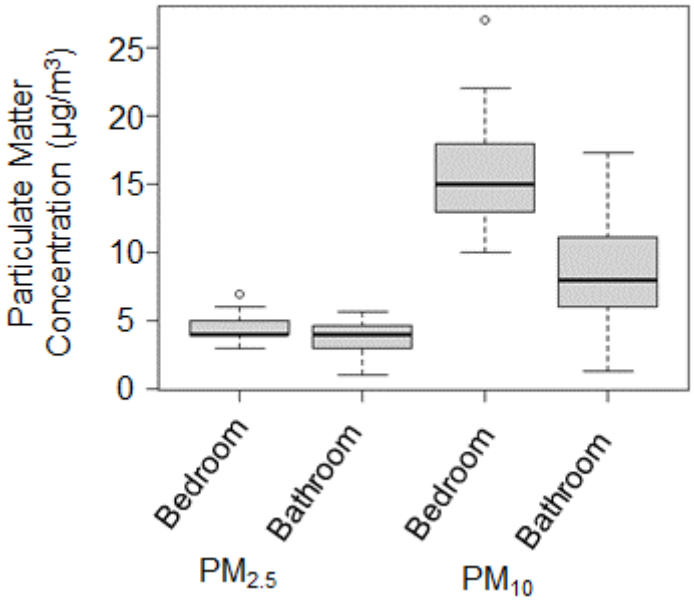


Figure 3: Distribution of background levels of $PM_{2.5}$ and PM_{10} in the bathroom and bedroom shown by box and whisker plots. Interquartile ranges for each particulate are shown by the whiskers, with outliers indicated by the circles; larger ranges were observed in PM_{10} measurements compared to $PM_{2.5}$ measurements. The mean distribution of PM concentrations appears to be higher in the bedroom compared to the bathroom, especially with regards to PM_{10}, but these differences were not found to be significant at the 95% level ($p > 0.05$).

3.4. Peak deodorant PM concentrations

Releasing aerosol deodorant in a household bathroom rapidly increased the concentration of $PM_{2.5}$ and PM_{10} above the background levels in all ventilation conditions tested, which gradually decreased over time (Figure 4). The largest mean increases from background concentrations, to 16430 µgm^{-3} PM_{10} (SE ± 4140) and 2372 µgm^{-3} $PM_{2.5}$ (SE ± 236), were observed in the 'No Ventilation' condition (Table

2). Ventilation through having a door and/or window open reduced the mean peak concentration for both $PM_{2.5}$ and PM_{10} in the bathroom, although this reduction was significant ($p < 0.05$) only in the 'Door and Window Open' condition, with mean peaks of 3856 μgm^{-3} PM_{10} (SE ± 522) and 921 μgm^{-3} $PM_{2.5}$ (SE ± 96). Mean PM concentrations were also found to be increased in a nearby bedroom. Monitoring in the bedroom with the bathroom window closed demonstrated average peak concentrations of 64 μgm^{-3} $PM_{2.5}$ (SE ± 20) and 313 μgm^{-3} PM_{10} (SE ± 95) (Table 2). After approximately 2 minutes these were significantly lower than those in the 'No Ventilation' condition ($p < 0.05$) but are still markedly higher than the WHO threshold values of 10 and 40 μgm^{-3}. Opening the bathroom window was found to significantly increase peak PM concentration (226 μgm^{-3} $PM_{2.5}$ (SE ± 137) and 926 μgm^{-3} PM_{10} (SE ± 633)) in the bedroom approximately 3-fold relative to leaving the window closed (64 μgm^{-3} $PM_{2.5}$ and 313 μgm^{-3} PM_{10}), suggesting increased dispersion of aerosol from bathroom to bedroom ($p < 0.05$) (Table 2).

3.5. Temporal patterns of deodorant PM concentration

For all conditions tested the time (in minutes) from spray to peak PM concentration was rapid. All conditions in the bathroom, with the exception of the 'Fan Only' condition, peaked within 1 minute; switching the extractor fan on appeared to delay the peak by 1 minute (Table 2). Peak PM concentration was achieved in the nearby bedroom on average 5 minutes (SE ± 2) after spray, which was accelerated, as stated above, to a mean time of 2 minutes (SE ± 1) by opening the bedroom window.

Both $PM_{2.5}$ and PM_{10} concentrations, 10 minutes after peak concentration, were found to be significantly reduced for all ventilation conditions with exception for the 'Fan Only' condition ($p < 0.05$). For PM_{10}, none of the ventilation conditions reached a concentration below the WHO daily mean threshold within 10 minutes. For $PM_{2.5}$, only the 'Bathroom Door and Window Open' condition reached a concentration below the WHO daily mean threshold within 10 minutes (Table 2) with different conditions still contributing to extremely high levels of PM at this timepoint despite showing apparent high percentage reductions. 'No Ventilation' led to a 59.74% and 76.88% decrease in $PM_{2.5}$ and PM_{10} after 10 minutes yet mean levels for $PM_{2.5}$ and PM_{10} were still almost 100 times the WHO threshold values. The largest percentage reduction from peak PM concentration at 10 minutes was achieved in the 'Bathroom Door and Window Open'

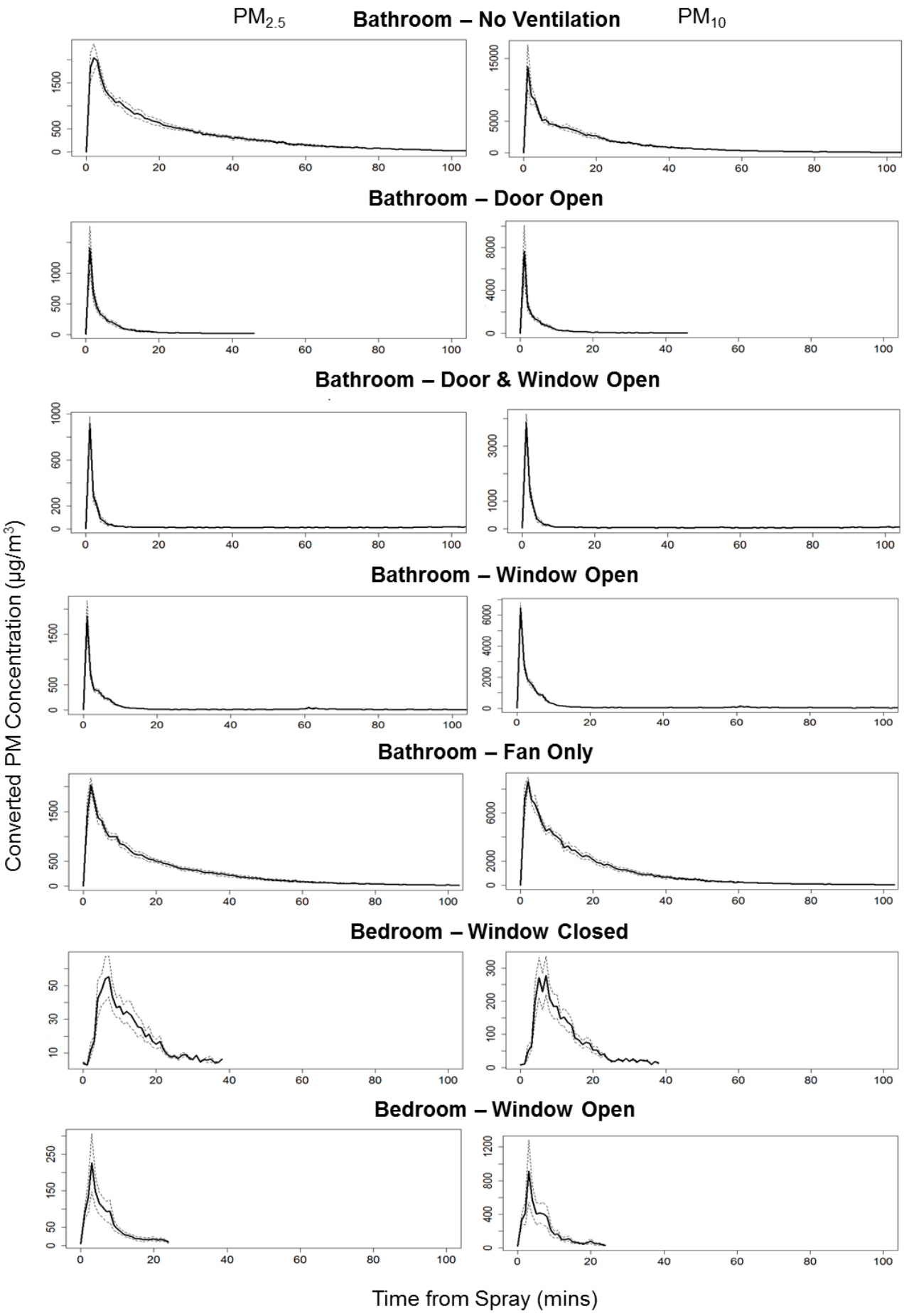
condition, with a reduction of 97.50% $PM_{2.5}$ and 98.31% PM_{10} and as stated above, achieved the PM concentrations closest to the WHO threshold values within 10 minutes. The percentage decreases in the bedroom were similar to those observed in the bathroom; with no ventilation ('Bedroom Window Closed') the percentage decreases within 10 minutes were 53.13% and 63.90% for $PM_{2.5}$ and PM_{10}, respectively, although the raw values of PM in the air were significantly lower than the 'No Ventilation' condition ($p < 0.05$). Likewise, 'Bedroom: bathroom Window Open' showed an 86.73% decrease for $PM_{2.5}$ and 89.09% decrease for PM_{10}, similar to the percentage decreases shown in the bathroom ventilation conditions.

PM concentrations were elevated in both rooms for a sustained period of time after spray release. The shortest mean time taken to return to below daily mean threshold, that is the time taken to return to concentrations of $PM_{2.5}$ below 25 μgm^{-3} and PM_{10} below 50 μgm^{-3} for a period of 3 or more minutes without a return to above these threshold values, was seen in the 'Bedroom: bathroom Window Open' condition, with 13 (SE ± 5) and 21 (SE ± 2) minutes for $PM_{2.5}$ and PM_{10} (Table 2). The shortest time to return daily mean threshold levels in the bathroom was achieved in the 'Bathroom Door Open' condition, with significantly reduced mean times of 37 and 50 minutes for $PM_{2.5}$ and PM_{10}, respectively ($p < 0.05$).

The 'Door and Window Open' condition saw a mean time of 44 (SE ± 35) and 104 (SE ± 44) minutes for $PM_{2.5}$ and PM_{10}, respectively, to return to below threshold levels which was due to a large degree of random fluctuation of both PMs. The mean concentration of $PM_{2.5}$ fell rapidly from a peak of 921 μgm^{-3} to 22 μgm^{-3} within 10 minutes. Monitoring over the following 180 minutes showed that $PM_{2.5}$ concentrations fluctuated around an average of 15 μgm^{-3} (SE ± 0.18), but did not exceed 22 μgm^{-3}, before reducing to below 10 μgm^{-3} an average of 140 minutes (SE ± 53) after spray. Mean PM_{10} concentrations showed a similar pattern, rapidly reducing from the mean peak concentration to 65 μgm^{-3} within 10 minutes, but fluctuated around an average of 43 μgm^{-3} (SE ± 0.81), with maximum and minimum values of 84 and 21 μgm^{-3}, before consistently returning to below the threshold values an average of 104 minutes (SE ± 44) after spray.

Use of the extractor fan in the 'Fan Only' condition did not significantly improve PM concentration reduction ($p > 0.05$); the temporal pattern of the mean PM

concentrations shows a slow and gradual decrease of PM, very similar to the ‘No Ventilation’ temporal pattern (Figure 4).


PM2.5
Bathroom – No Ventilation
PM10
Bathroom – Door Open
Bathroom – Door & Window Open
Bathroom – Window Open
Bathroom – Fan Only
Bedroom – Window Closed
Bedroom – Window Open
Converted PM Concentration (µg/m3)
Time from Spray (mins)

400

Figure 4: Mean (black) and ± 1 standard error (grey dashed) estimated concentration (μgm^{-3}) of PM_{10} and $PM_{2.5}$ for each ventilation condition. For all conditions, $PM_{2.5}$ and PM_{10} sharply increased immediately after spray, followed by a decline in PM concentration over time. Ventilation markedly reduced the time from peak PM concentration to baseline conditions. PM concentration increased in the adjacent bedroom, but there was a delay in reaching peak PM concentration.

The mean daily PM concentrations for each experimental condition, whereby data were averaged over the 24-hour period on the day that the experiment was performed, are also shown in Table 2. Whereas we report the mean in each case we only do this to provide a measurement of longer-term exposure as a comparator to WHO daily mean particulate thresholds using the same way that such a metric is calculated. Given the high concentrations of particulates post-spray in each condition and decrease back to baseline within a few hours, the data over 24 hours displayed an extreme right skew in each case caused by a high frequency of low concentration measurements once particulates had dispersed. Thus, we only show the measurements as statistical significance of mean daily differences between concentrations for each experimental condition with either control or the 'No Ventilation' condition over 24-hour periods was not determined. Mean daily $PM_{2.5}$ and PM_{10} concentrations were increased for all experimental conditions relative to the corresponding bathroom or bedroom control conditions, Furthermore, mean daily levels for the 'No Ventilation' and 'Fan Only'.

With no ventilation in the bathroom following spray the daily mean concentrations for $PM_{2.5}$ was 31 μgm^{-3} (SE ± 35) and for PM_{10} was 100 μgm^{-3} (SE ± 18). All ventilated conditions had a daily average mean PM level below the WHO daily mean threshold except 'Bathroom Fan Only' having daily mean concentrations of $PM_{2.5}$ was 25 μgm^{-3} (SE ± 3) and 94 μgm^{-3} (SE ± 15) for $PM_{2.5}$ and PM_{10} respectively (Table 2).

	Mean Peak PM Conc. (μgm^{-3})		Mean Time to Peak (mins)		PM Conc. 10 minutes after peak PM Conc. (μgm^{-3})		Decrease 10 Minutes after Peak PM Conc. (%)		Time to WHO Daily Mean Threshold (mins)		Mean Daily PM Conc. (μgm^{-3})	
	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}
No ventilation	2372 (236)	16430 (4140)	1 (0)	1 (0)	955 (181)	3799 (480)	59.74	76.88	116 (10)	116 (17)	31 (4)	110 (18)
Bathroom Door Open	1476 (559)	7917 (3986)	1 (0)	1 (0)	99* (22)	332* (59)	93.29	95.81	37* (18)	50 (19)	7 (1)	23 (6)
Bathroom Door and Window Open	921* (96)	3856* (522)	1 (0)	1 (0)	23* (7)	65* (21)	97.50	98.31	44 (35)	104 (44)	6 (1)	18 (3)
Bathroom Window Open	1853 (533)	6469 (569)	1 (0)	1 (0)	85* (17)	273* (39)	95.41	95.78	37* (20)	59 (24)	8 (1)	24 (5)
Bathroom Fan Only	2291 (49)	9698 (621)	2 (0)	2 (0)	830 (101)	3807 (584)	63.77	60.74	93 (10)	98 (7.8)	25 (3)	94 (15)
Bedroom: Bathroom Window Closed	64* (20)	313* (95)	5* (2)	5* (2)	30* (9)	113* (12)	53.13	63.90	13* (5)	21* (2)	5 (1)	17 (1)
Bedroom: Bathroom Window Open	226* (137)	926* (633)	3* (0)	3 (1)	30* (11)	101* (41)	86.73	89.09	15* (5)	19* (4)	5 (1)	19 (1)

Table 2: Mean measures, and standard error of mean (brackets), of peak PM concentration (μgm^{-3}), time to peak PM concentration, time to return to WHO daily mean threshold PM concentrations, and PM concentrations 10 minutes after peak PM concentration for each experimental condition. Significance levels denote comparison between each experimental condition with the 'No Ventilation' condition: *$p < 0.05$

4. Discussion

We have shown that background PM levels of upstairs rooms in a modern brick house were consistently below the WHO daily mean threshold values for $PM_{2.5}$ and PM_{10} (Figure 3), but were rapidly and persistently increased when a common anti-perspirant deodorant spray was released in accordance with the manufacturer's recommendations (Figure 4). Levels of PM released after spray in the 'No ventilation' condition was found to be above the WHO recommended daily mean threshold values for $PM_{2.5}$ and PM_{10} for a mean of 116 minutes, in each case (Table 2). Additionally, daily mean particulate concentrations in the "No ventilation" ($PM_{2.5}$ 31 μgm^{-3} & PM_{10} 110 μgm^{-3}) and "Fan only" ($PM_{2.5}$ 25 μgm^{-3} and PM_{10} 94 μgm^{-3}) bathroom conditions were not just increased compared to the bathroom control condition ($PM_{2.5}$ 4μgm^{-3} &

448 PM_{10} 9 μgm^{-3}) where no daily aerosol release was occurring but also greater than the
449 WHO daily mean guideline thresholds.

450 Use of a standard bathroom extractor fan did not significantly improve the removal of
451 PM from the air in the bathroom, highlighted by the observation that in all ventilation
452 conditions, with the exception of 'Fan only', a significant ($p < 0.05$) decrease in mean
453 PM concentration 10 minutes after peak concentration was achieved, compared to the
454 'No Ventilation' condition, suggesting that extractor fans of this type are not suitable
455 for controlling PM concentrations after aerosol anti-perspirant usage. This might be
456 unsurprising given the function of an extractor fan is to reduce moisture, but there may
457 be a public assumption that these installed devices can impact on particulate
458 reduction. The peak PM concentrations were delayed by one minute when the
459 extractor fan was on compared to all other bathroom conditions, and although the peak
460 concentration was decreased compared to no ventilation, the difference was not
461 significant. Additionally, the mean time taken to return to WHO daily mean threshold
462 PM concentrations, 93 (SE ± 10) and 98 (SE ± 8) minutes, was reduced for both $PM_{2.5}$
463 and PM_{10}, compared to 116 minutes (SE ± 10; SE ± 17) for $PM_{2.5}$ and PM_{10} in the no
464 ventilation condition, respectively, but these differences were not found to be
465 statistically significant. The mean PM concentrations observed 10 minutes after peak
466 PM concentration were very similar in the 'Fan Only' and 'No Ventilation' condition,
467 despite the higher initial peak observed with 'No Ventilation'. This suggests that
468 exceptionally high concentrations (> 10,000 μgm^{-3}) of PM may rapidly settle out of the
469 air or disperse even when no additional ventilation is provided, possibly due an
470 equilibrium effect, or due to the large amount of initial energy each particle will have
471 shortly after spray release, but settling or dispersion slows as this energy is lost.

472 Additional factors thought to contribute to the significant decreases in PM
473 concentration 10 minutes after the peak concentration for all other experimental
474 conditions are increased air exchange with other rooms in the house, or outside,
475 leading to faster removal of PM from the bathroom, and increased air volume available
476 to the aerosol to expand into when sprayed, limiting the initial peak concentration of
477 PM. For example, in the 'Door Open, Window Closed' condition, the immediate volume
478 of air the aerosol could expand into was markedly increased, compared to when the
479 door was shut. This could have directly led to a reduction in peak PM concentration

achievable, due to rapid dispersion of particles, possibly into other rooms including the bedroom.

It was expected that increased ventilation would reduce PM exposure through increased removal and dispersion leading to rapidly reduced PM concentrations. However, some of the ventilation conditions did not conform to this assumption. We hypothesise this to be primarily due to variation in indoor air flow and turbulence, which could be influenced by opening the door and/or window. One example highlighting this possibility was the finding that the 'Door and Window Open' condition did not show a statistically significantly reduction in the time to return to baseline PM concentrations compared to the control condition, with a mean return time of 44 and 104 minutes for $PM_{2.5}$ and PM_{10}, respectively, whilst the mean PM concentrations 10 minutes after peak PM concentrations were significantly reduced. In fact, the PM concentrations 10 minutes after peak concentration were lowest in this condition. This suggests a rapid removal of very high concentrations of PM, through dispersion and air exchange, but a long return to baseline time could suggest that PM is not settling out of the air in more turbulent conditions. A possible mechanism is the action of opening both a window and door could have created a through-draft, and increased the turbulence in the air, which could have prevented the PM from settling out of the air and could also have caused previously settled PM to resuspend.

Opening the bathroom window led to an increased mean peak PM concentration in the bedroom, of 226 μgm^{-3} $PM_{2.5}$ and 926 μgm^{-3} PM_{10}, compared to 64 μgm^{-3} $PM_{2.5}$ and 313 μgm^{-3} PM_{10} when the window was closed. This could also be due to the open window increasing airflow through the bathroom, into the bedroom, increasing the concentration of suspended PM particles in the bedroom. Despite this higher peak concentration, PM concentrations were similar in both conditions 10 minutes after peak concentration, and both $PM_{2.5}$ and PM_{10} concentrations had returned to baseline within 25 minutes in both conditions.

The PM concentration measurements reported in this study, obtained through conversion from particle count measurements are subject to an undefined level of uncertainty, which to the best of the study team's knowledge has not been widely reported. Estimated values for the uncertainty of each device, of ± 5% for the Trotec PC220 and ± 10% for the Dylos DC1100 were accounted for through repetition of each

experimental condition and removal of statistical outliers. Through reporting mean PM concentrations with standard errors, we show the variability present in our dataset, which could be interpreted as the range of possible PM concentrations present after aerosol deodorant usage. Even the lowest bounds of PM concentration ranges were shown to be higher than the WHO daily threshold values for PM exposure, which therefore could still represent a risk to exposed persons through long-term usage. The persistence of suspended PM above WHO threshold concentrations after spray release was also subject to this uncertainty. In low ventilation conditions (i.e. Fan only, or No Ventilation) the time to return to WHO threshold conditions was in excess of 90 minutes, with low inter-replicate variability reported (low standard errors), whereas in conditions with more ventilation (i.e. Window Open, Door Open) the standard errors were relatively higher. We hypothesise this was due to environmental conditions already discussed, rather than variability in the measurement procedures. Therefore, we suggest that measuring PM concentration with a low-cost Dylos monitor is a robust procedure.

Individuals with chronic disease and care management teams should consider the impact of common household activities, such as long-term daily aerosol spraying, on disease and put in place measures to reduce that impact. We have shown that daily mean particulate concentrations can be increased above the daily and annual WHO threshold values for particulate exposure. Long-term exposure at these increased concentrations through sustained use of aerosol product could contribute towards worsening of respiratory and cardiovascular disease symptoms, and short-term exposure of high PM levels may also contribute towards exacerbations of these conditions. Amongst health care professionals, there is an increasing awareness of the role of indoor pollutants in respiratory health and management of long-term chronic respiratory conditions. This is reflected, for example, in the development of information for COPD patients in optimising their home environment to avoid exposure to indoor pollution (National Institute for Health and Care Excellence, 2020). Currently, there is little advice given regarding aerosol anti-perspirants and how the patients could mitigate the potential risk they pose. This study shows that improving ventilation during activities that release high levels of PM, such as spraying anti-perspirants, is a significant action that could be undertaken to reduce personal exposure and return PM concentrations below WHO threshold concentrations significantly faster. We also

show that ventilation in nearby rooms, which are not isolated from the spray, can increase the exposure of that room to aerosolised PM, and should therefore be avoided to minimise exposure. Therefore, when using sprays which can release high levels of aerosolised PM, such as anti-perspirant, the room should be isolated from other rooms in the house through closing the door and ventilation through opening the window for at least 1 hour should be performed to minimise PM exposure and maximise PM removal.

Persistent exposure to high household PM concentrations released from aerosols such as through daily aerosol deodorant usage may increase a person's likelihood that their personal daily exposure exceeds the WHO guideline threshold values for average daily PM exposure. Our findings are especially important in the context of increasing rates of aerosol-type deodorant usage in the UK. Such exposure could contribute towards an increased risk for, or more rapid progression of, respiratory and cardiovascular diseases. Indeed, indoor sources of air pollution may, in fact, be more important in a health context than outdoor sources, due to the increased duration of exposure. Several studies have shown that people tend to spend the majority of their time indoors (Klepeis et al., 2001; Wallace et al., 2006). Kleipeis et al. (2001) estimated that Americans spend upwards of 90% of their time indoors and 70% of their time at home. Indeed, given the current pandemic has led to a large increase in home-working, it is likely that the proportion of time spent indoors has increased. In addition, for inhalation pathways, many studies have also shown that the proportion of indoor emissions inhaled is much higher than the proportion of outdoor emissions inhaled, due to the lower rates of ventilation and air exchange that occur indoors as opposed to outdoors (Nazaroff and Weschler, 2004; Thatcher and Layton, 1995; Wallace et al., 2006).

Limitations & Future Directions

The authors consider this study to show good evidence for PM persistence following aerosol antiperspirant usage, but note that the findings have certain limitations: 1) only one type of antiperspirant product was tested; 2) only one type of room was used for spray release; 3) the experimental conditions did not truly replicate those of the 'living' home environment with inhabitants opening & closing doors/windows, which could potentially disturb settled PM; and 4) the Dylos monitor provides an average PCN for

the previous minute of monitoring, higher resolution monitoring may be necessary to more accurately measure the peak PM concentrations following spray release, as the PM concentrations are likely to rapidly change immediately after spray. This is not anticipated to have serious implications for this work, as the peak concentrations observed in all experimental conditions are orders of magnitude greater than the WHO threshold values, and a major goal of this work was to demonstrate the persistence of PM following aerosol antiperspirant release.

Although we tested only one anti-perspirant product, a review of similar products on the market from various manufacturers shows highly similar compositions (supplementary Table 1), so it is reasonable to generalise these findings to other commercially available anti-perspirant aerosol products. Limonene, for example is used in many anti-perspirant deodorants to mask odours but is known to react indoors with ozone that has entered from outside and form sub-micron secondary particles via the ozone-limonene reaction (Wainman et al., 2000). Thus, in addition to the delaying impacts that opening a bathroom window has on PM reduction observed in this study it is important to consider potential secondary particle formation when doing so. Further studies should focus on qualifying and quantifying temporal presence and concentrations of particles formed from commonly used anti-perspirant ingredients.

It is possible that rooms of different sizes, with additional windows or doors may cause different PM dispersion behaviours, for example a room with two windows on opposite walls may have a strong through-draft which rapidly clears released PM. Therefore, a further study could examine PM concentration and persistence following aerosol release in a multitude of rooms

In future, there should be close examination of the relationships between aerosol exposure and chronic disease exacerbation risk, by quantifying daily and mean PM exposures associated with aerosol usage and subsequent disease adverse events.

Conclusions

This work provides novel evidence for the short-term PM concentration peaks and long-term persistence of PM released following aerosol antiperspirant use, and highlights the risk posed to individuals with chronic disease from such exposure. It adds to the body of work identifying the risks associated with release of PM in indoor settings and provides evidence for governments to develop and implement new policy.

Personal care products, such as aerosol deodorants, can have a marked impact on indoor air quality, with levels of $PM_{2.5}$ and PM_{10} remaining elevated above the WHO thresholds for up to 3 hours after product use if inadequate ventilation is achieved, and no or poor ventilation can lead to daily average particulate concentrations in excess of the WHO daily mean exposure threshold limits for both PM_{10} and $PM_{2.5}$. We have shown that spraying aerosol deodorant can also cause a sustained increase in PM in other rooms within the house when internal doors are left open. Daily use of aerosol anti-perspirants, coupled with long periods of time spent indoors, could be a major contributing factor to an individual's annual exposure to PM. However, exposure can be limited through appropriate measures such as opening a window and isolating the room in which the spray was released. Furthermore, removing PM from the air through only the use of extractor fans is not sufficient to significantly reduce exposure. This is an important area for further research and development of advice for patients of chronic respiratory disease to help them reduce exposure to harmful PM.

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Data Availability

Datasets related to this article can be obtained by contacting the corresponding author.

References

Abt, E., Suh, H.H., Catalino, P., Koutras, P., 2000. Relative Contribution of Outdoor and Indoor Particle Sources to Indoor Concentrations. Environ. Sci. Technol. 34, 3579–3587.

Afshari, A., Matson, U., Ekberg, L.E., 2005. Characterization of indoor sources of fine and ultrafine particles: a study conducted in a full-scale chamber. Indoor Air 15, 141–150.

Arden Pope, C., Burnett, R.T., Turner, M.C., Cohen, A., Krewski, D., Jerrett, M., Gapstur, S.M., Thun, M.J., 2011. Lung cancer and cardiovascular disease

639 mortality associated with ambient air pollution and cigarette smoke: Shape of
640 the exposure-response relationships. Environ. Health Perspect. 119, 1616–
641 1621. https://doi.org/10.1289/ehp.1103639

642 Baldacci, S., Maio, S., Cerrai, S., Sarno, G., Baïz, N., Simoni, M., Annesi-Maesano,
643 I., Viegi, G., 2015. Allergy and asthma: Effects of the exposure to particulate
644 matter and biological allergens. Respir. Med. 109, 1089–1104.
645 https://doi.org/10.1016/j.rmed.2015.05.017

646 Barnes, J., E.T., H., Chatterton, T.J., Longhurst, J.W.S., 2018. Policy Disconnect: A
647 critical review of UK Air quality policy in relation to EU and LAQM responsibilities
648 over the last 20 years. Environ. Sci. Policy 85, 28–39.

649 Biesterbos, J.W.H., Dudzina, T., Delmaar, C.J.E., Bakker, M.I., Russel, F.G.M., Von
650 Götz, N., Scheepers, P.T.J., Roeleveld, N., 2013. Usage patterns of personal
651 care products: Important factors for exposure assessment. Food Chem. Toxicol.
652 55, 8–17. https://doi.org/10.1016/j.fct.2012.11.014

653 Chan, Y.L., Wang, B., Chen, H., Ho, K.F., Cao, J., Hai, G., Jalaludin, B., Herbert, C.,
654 Thomas, P.S., Saad, S., Oliver, B.G.G., 2019. Pulmonary Inflammation Induced
655 by Low Dose Particulate Matter Exposure in Mice. Am J Physiol Lung Cell Mol
656 Physiol 317. https://doi.org/10.1152/ajplung.00232.2019

657 Cosmetics Business [WWW Document], 2016. URL
658 https://www.cosmeticsbusiness.com/news/article_page/Global_deodorants_mar
659 ket_shows_marginal_growth/119993 (accessed 7.2.20).

660 Cuizas, D., Prasauskas, T., Krugly, E., Sidaraviciute, R., Jurelionis, A., Sediukyte, L.,
661 Kauneliene, V., Wierzbicka, A., Martuzevicius, D., 2015. Characterization of
662 indoor aerosol temporal variations for the real-time management of indoor air
663 quality. Atmos. Environ. 118, 107–117.

664 Dacunto, P.J., Klepeis, N.E., Cheng, K.C., Acevedo-Bolton, V., Jiang, R.T., Repace,
665 J.L., Ott, W.R., Hildemann, L.M., 2015. Determining PM2.5 calibration curves for
666 a low-cost particle monitor: Common indoor residential aerosols. Environ. Sci.
667 Process. Impacts 17, 1959–1966. https://doi.org/10.1039/c5em00365b

668 Department for Environment Food & Rural Affairs, 2019. Clean Air Strategy 2019.

669 Doneva, M., Petrova, G., Petrova, D., Kamusheva, M., Petkova, V., Tachkov, K.,
670 Pencheva, V., Georgiev, O., 2019. Chronic obstructive pulmonary disease
671 exacerbations and progression in relation to ambient air pollutants exposure. J.
672 Thorac. Dis. 11, 2490–2497. https://doi.org/10.21037/jtd.2019.05.50

673 DuPre, N.C., Hart, J.E., Holmes, M.D., Poole, E.M., James, P., Kraft, P., Laden, F.,
674 Tamimi, R.M., 2019. Particulate matter and traffic-related exposures in relation
675 to breast cancer survival. Cancer Epidemiol. Biomarkers Prev. 28, 751–759.
676 https://doi.org/10.1158/1055-9965.EPI-18-0803

677 Dylos [WWW Document], 2019. URL http://www.dylosproducts.com/dcbaopaqm.html
678 (accessed 9.10.19).

679 Fiala, S.C., Morris, D.S., Pawlak, R.L., 2012. Measuring indoor air quality of hookah
680 lounges. Am. J. Public Health 102, 2043–2045.
681 https://doi.org/10.2105/AJPH.2012.300751

682 Fiordelisi, A., Piscitelli, P., Trimarco, B., Coscioni, E., Iaccarino, G., Sorriento, D.,
683 2017. The mechanisms of air pollution and particulate matter in cardiovascular
684 diseases. Heart Fail. Rev. 22, 337–347. https://doi.org/10.1007/s10741-017-
685 9606-7

686 Franklin, B.A., Brook, R., Arden Pope, C., 2015. Air pollution and cardiovascular
687 disease. Curr. Probl. Cardiol. 40, 207–238.
688 https://doi.org/10.1016/j.cpcardiol.2015.01.003

689 Géhin, E., Ramalho, O., Kirchner, S., 2008. Size distribution and emission rate
690 measurement of fine and ultrafine particle from indoor human activities. Atmos.
691 Environ. 42, 8341–8352. https://doi.org/10.1016/j.atmosenv.2008.07.021

692 Glytsos, T., Ondráček, J., Džumbová, L., Kopanakis, I., Lazaridis, M., 2010.
693 Characterization of particulate matter concentrations during controlled indoor
694 activities. Atmos. Environ. 44, 1539–1549.
695 https://doi.org/10.1016/j.atmosenv.2010.01.009

696 Guarnieri, M., Balmes, J.R., 2014. Outdoor air pollution and asthma. Lancet 383,
697 1581–1592. https://doi.org/10.1016/S0140-6736(14)60617-6

698 Hamra, G.B., Guha, N., Cohen, A., Laden, F., Raaschou-Nielsen, O., Samet, J.M.,

699 Vineis, P., Forastiere, F., Saldiva, P., Yorifuji, T., Loomis, D., 2014. Outdoor
700 particulate matter exposure and lung cancer: A systematic review and meta-
701 analysis. Environ. Health Perspect. 122, 906–911.
702 https://doi.org/10.1289/ehp.1408092

703 He, C., Morawska, L., Hitchins, J., Gilbert, D., 2004. Contribution from indoor
704 sources to particle number and mass concentrations in residential houses.
705 Atmos. Environ. 38, 3405–3415. https://doi.org/10.1016/j.atmosenv.2004.03.027

706 Holstius, D.M., Pillarisetti, A., Smith, K.R., Seto, E., 2014. Field calibrations of a low-
707 cost aerosol sensor at a regulatory monitoring site in California. Atmos. Meas.
708 Tech. 7, 1121–1131. https://doi.org/10.5194/amt-7-1121-2014

709 Hussein, T., Hämeri, K., Heikkinen, M.S.A., Kulmala, M., 2005. Indoor and outdoor
710 particle size characterization at a family house in Espoo-Finland. Atmos.
711 Environ. 39, 3697–3709. https://doi.org/10.1016/j.atmosenv.2005.03.011

712 Jalaludin, B., Cowie, C., 2014. Particulate air pollution and cardiovascular disease -
713 It is time to take it seriously. Rev. Environ. Health 29, 129–132.
714 https://doi.org/10.1515/reveh-2014-0031

715 James, A.C., Stahlhofen, W., Rudolf, G., Egan, M.J., Nixon, W., Gehr, P., Briant,
716 J.K., 1991. The Respiratory Tract Deposition Model Proposed by the ICRP Task
717 Group. Radiat. Prot. Dosimetry 38, 159–165.

718 Jones, A.P., 1999. Indoor air quality and health. Atmos. Environ. 33, 4535–4564.

719 Jones, S., Renee Anthony, T., Sousan, S., Altmaier, R., Park, J.H., Peters, T.M.,
720 2016. Evaluation of a low-cost aerosol sensor to assess dust concentrations in a
721 swine building. Ann. Occup. Hyg. 60, 597–607.
722 https://doi.org/10.1093/annhyg/mew009

723 Kamens, R., Lee, C. te, Wiener, R., Leith, D., 1991. A study of characterize indoor
724 particles in three non-smoking homes. Atmos. Environ. Part A, Gen. Top. 25,
725 939–948. https://doi.org/10.1016/0960-1686(91)90136-U

726 Keet, C.A., Keller, J.P., Peng, R.D., 2018. Long-term coarse particulate matter
727 exposure is associated with asthma among children in medicaid. Am. J. Respir.
728 Crit. Care Med. 197, 737–746. https://doi.org/10.1164/rccm.201706-1267OC

729 Klepeis, N.E., Hughes, S.C., Edwards, R.D., Allen, T., Johnson, M., Chowdhury, Z.,
730 Smith, K.R., Boman-Davis, M., Bellettiere, J., Hovell, M.F., 2013. Promoting
731 Smoke-Free Homes: A Novel Behavioral Intervention Using Real-Time Audio-
732 Visual Feedback on Airborne Particle Levels. PLoS One 8, 1–17.
733 https://doi.org/10.1371/journal.pone.0073251

734 Klepeis, N.E., Nelson, W.C., Ott, W.R., Robinson, J.P., Tsang, A.M., Switzer, P.,
735 Behar, J. V., Hern, S.C., Engelmann, W.H., 2001. The National Human Activity
736 Pattern Survey (NHAPS): A resource for assessing exposure to environmental
737 pollutants. J. Expo. Anal. Environ. Epidemiol. 11, 231–252.
738 https://doi.org/10.1038/sj.jea.7500165

739 Künzli, N., Tager, I.B., 2005. Air pollution: From lung to heart. Swiss Med. Wkly. 135,
740 697–702. https://doi.org/2005/47/smw-11025

741 Lefebvre, M.A., Meuling, W.J.A., Engel, R., Coroama, M.C., Renner, G., Pape, W.,
742 Nohynek, G.J., 2012. Consumer inhalation exposure to formaldehyde from the
743 use of personal care products/cosmetics. Regul. Toxicol. Pharmacol. 63, 171–
744 176. https://doi.org/10.1016/j.yrtph.2012.02.011

745 Li, J., Li, W.X., Bai, C., Song, Y., 2017. Particulate matter-induced epigenetic
746 changes and lung cancer. Clin. Respir. J. 11, 539–546.
747 https://doi.org/10.1111/crj.12389

748 Lim, S.S., Vos, T., Flaxman, A.D., Danaei, G., Shibuya, K., Adair-Rohani, H.,
749 Amann, M., Anderson, H.R., Andrews, K.G., Aryee, M., Atkinson, C., Bacchus,
750 L.J., Bahalim, A.N., Balakrishnan, K., Balmes, J., Barker-Collo, S., Baxter, A.,
751 Bell, M.L., Blore, J.D., Blyth, F., Bonner, C., Borges, G., Bourne, R.,
752 Boussinesq, M., Brauer, M., Brooks, P., Bruce, N.G., Brunekreef, B., Bryan-
753 Hancock, C., Bucello, C., Buchbinder, R., Bull, F., Burnett, R.T., Byers, T.E.,
754 Calabria, B., Carapetis, J., Carnahan, E., Chafe, Z., Charlson, F., Chen, H.,
755 Chen, J.S., Cheng, A.T.-A., Child, J.C., Cohen, A., Colson, K.E., Cowie, B.C.,
756 Darby, S., Darling, S., Davis, A., Degenhardt, L., Dentener, F., Jarlais, D.C. Des,
757 Devries, K., Dherani, M., Ding, E.L., Dorsey, E.R., Driscoll, T., Edmond, K., Ali,
758 S.E., Engell, R.E., Erwin, P.J., Fahimi, S., Falder, G., Farzadfar, F., Ferrari, A.,
759 Finucane, M.M., Flaxman, S., Fowkes, F.G.R., Freedman, G., Freeman, M.K.,
760 Gakidou, E., Ghosh, S., Giovannucci, E., Gmel, G., Graham, K., Grainger, R.,

761 Grant, B., Gunnell, D., Gutierrez, H.R., Hall, W., Hoek, H.W., Hogan, A., H Dean
762 Hosgood, I., Hoy, D., Hu, H., Hubbell, B.J., Hutchings, S.J., Ibeanusi, S.E.,
763 Jacklyn, G.L., Jasrasaria, R., Jonas, J.B., Kan, H., Kanis, J.A., Kassebaum, N.,
764 Kawakami, N., Khang, Y.-H., Khatibzadeh, S., Khoo, J.-P., Kok, C., Laden, F.,
765 Lalloo, R., Lan, Q., Lathlean, T., Leasher, J.L., Leigh, J., Li, Y., Lin, J.K.,
766 Lipshultz, S.E., London, S., Lozano, R., Lu, Y., Mak, J., Malekzadeh, R.,
767 Mallinger, L., Marcenes, W., March, L., Marks, R., Martin, R., McGale, P.,
768 McGrath, J., Mehta, S., Mensah, G.A., Merriman, T.R., Micha, R., Michaud, C.,
769 Mishra, V., Hanafiah, K.M., Mokdad, A.A., Morawska, L., Arian, D.M., Murphy,
770 T., Naghavi, M., Neal, B., Nelson, P.K., Nolla, J.M., Norman, R., Olives, C.,
771 Omer, S.B., Orchard, J., Osborne, R., Ostro, B., Page, A., Pandey, K.D., Parry,
772 C.D.H., Passmore, E., Patra, J., Pearce, N., Pelizzari, P.M., Petzold, M.,
773 Phillips, M.R., Pope, D., III, C.A.P., Powles, J., Rao, M., Razavi, H., Rehfuess,
774 E.A., Rehm, J.T., Ritz, B., Rivara, F.P., Roberts, T., Robinson, C., Rodriguez-
775 Portales, J.A., Romieu, I., Room, R., Rosenfeld, L.C., Roy, A., Rushton, L.,
776 Salomon, J.A., Sampson, U., Sanchez-Riera, L., Sanman, E., Sapkota, A.,
777 Seedat, S., Shi, P., Shield, K., Shivakoti, R., Singh, G.M., Sleet, D.A., Smith, E.,
778 Smith, K.R., Stapelberg, N.J.C., Steenland, K., Stöckl, H., Stovner, L.J., Straif,
779 K., Straney, L., Thurston, G.D., Tran, J.H., Dingenen, R. Van, Donkelaar, A.
780 van, Veerman, J.L., Vijayakumar, L., Weintraub, R., Weissman, M.M., White,
781 R.A., Whiteford, H., Wiersma, S.T., Wilkinson, J.D., Williams, H.C., Williams, W.,
782 Wilson, N., Woolf, A.D., Yip, P., Zielinski, J.M., Lopez, A.D., Murray, C.J.L.,
783 Ezzati, M., 2012. A comparative risk assessment of burden of disease and injury
784 attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a
785 systematic analysis for the Global Burden of Disease Study 2010. Lancet 380,
786 2224–2260. https://doi.org/10.1016/S0140-6736(12)61766-8. A

787 Liu, F., Chen, G., Huo, W., Wang, C., Liu, S., Li, N., Mao, S., Hou, Y., Lu, Y., Xiang,
788 H., 2019. Associations between long-term exposure to ambient air pollution and
789 risk of type 2 diabetes mellitus: A systematic review and meta-analysis. Environ.
790 Pollut. 252, 1235–1245. https://doi.org/10.1016/j.envpol.2019.06.033

791 Maximale Arbeitsplatz-Konsentration Comission, 2012. List of MAK and BAT Values
792 2012 Commission for the Investigation of Health Hazards of Chemical
793 Compounds in the Work Area.

794 Meier, R., 2019. CoolTerm.

795 Miller, M.R., Raftis, J.B., Langrish, J.P., McLean, S.G., Samutrtai, P., Connell, S.P.,
796 Wilson, S., Vesey, A.T., Fokkens, P.H.B., Boere, A.J.F., Krystek, P., Campbell,
797 C.J., Hadoke, P.W.F., Donaldson, K., Cassee, F.R., Newby, D.E., Duffin, R.,
798 Mills, N.L., 2017. Inhaled Nanoparticles Accumulate at Sites of Vascular
799 Disease. ACS Nano 11, 4542–4552. https://doi.org/10.1021/acsnano.6b08551

800 Morantes-Caballero, J.A., Alberto, H., Rodriguez, F., 2019. Effects of air pollution on
801 acute exacerbation of chronic obstructive pulmonary disease: A descriptive
802 retrospective study (pol-AECOPD). Int. J. COPD 14, 1549–1557.
803 https://doi.org/10.2147/COPD.S192047

804 National Institute for Health and Care Excellence, 2020. Chronic Obstructive
805 Pulmonary Disease in over 16s: Diagnosis and Management.

806 Nazaroff, W.W., Weschler, C.J., 2004. Cleaning products and air fresheners:
807 Exposure to primary and secondary air pollutants. Atmos. Environ. 38, 2841–
808 2865. https://doi.org/10.1016/j.atmosenv.2004.02.040

809 Northcross, A.L., Edwards, R.J., Johnson, M.A., Wang, Z.M., Zhu, K., Allen, T.,
810 Smith, K.R., 2013. A low-cost particle counter as a realtime fine-particle mass
811 monitor. Environ. Sci. Process. Impacts 15, 433–439.
812 https://doi.org/10.1039/c2em30568b

813 Peters, R., Ee, N., Peters, J., Booth, A., Mudway, I., Anstey, K.J., 2019. Air Pollution
814 and Dementia: A Systematic Review. J. Alzheimer's Dis. 70, S145–S163.
815 https://doi.org/10.3233/JAD-180631

816 Robert J. Vercellino, Sleeth, D.K., Handy, R.G., Min, K.T., Collingwood, S.C., 2018.
817 Laboratory Evaluation of a Low-cost, Real-time, Aerosol Multi-Sensor. J Occup
818 Env. Hyg 15, 559–567. https://doi.org/10.1016/j.physbeh.2017.03.040

819 Rosen, L., Zucker, D., Hovell, M., Brown, N., Ram, A., Myers, V., 2015. Feasibility of
820 measuring tobacco smoke air pollution in homes: Report from a pilot study. Int.
821 J. Environ. Res. Public Health 12, 15129–15142.
822 https://doi.org/10.3390/ijerph121214970

823 Rothe, H., Fautz, R., Gerber, E., Neumann, L., Rettinger, K., Schuh, W., Gronewold,

824 C., 2011. Special aspects of cosmetic spray safety evaluations: Principles on
825 inhalation risk assessment. Toxicol. Lett. 205, 97–104.
826 https://doi.org/10.1016/j.toxlet.2011.05.1038

827 Royal College of Paediatrics and Child Health, 2016. Every breath we take: The
828 lifelong impact of air pollution. Report of a working party.

829 Salthammer, T., Schripp, T., Wientzek, S., Wensing, M., 2014. Impact of operating
830 wood-burning fireplace ovens on indoor air quality. Chemosphere 103, 205–211.
831 https://doi.org/10.1016/j.chemosphere.2013.11.067

832 Semple, S., Apsley, A., MacCalman, L., 2013. An inexpensive particle monitor for
833 smoker behaviour modification in homes. Tob. Control 22, 295–298.
834 https://doi.org/10.1136/tobaccocontrol-2011-050401

835 Semple, S., Ibrahim, A.E., Apsley, A., Steiner, M., Turner, S., 2015. Using a new,
836 Low-Cost air quality sensor to quantify Second-Hand smoke (SHS) levels in
837 homes. Tob. Control 24, 153–158. https://doi.org/10.1136/tobaccocontrol-2013-
838 051188

839 Smith, R.B., Fecht, D., Gulliver, J., Beevers, S.D., Dajnak, D., Blangiardo, M.,
840 Ghosh, R.E., Hansell, A.L., Kelly, F.J., Ross Anderson, H., Toledano, M.B.,
841 2017. Impact of London's road traffic air and noise pollution on birth weight:
842 Retrospective population based cohort study. BMJ 359, 1–13.
843 https://doi.org/10.1136/bmj.j5299

844 Sousan, S., Koehler, K., Thomas, G., Park, J.H., Hillman, M., Halterman, A., Peters,
845 T.M., 2016. Inter-comparison of low-cost sensors for measuring the mass
846 concentration of occupational aerosols. Aerosol Sci. Technol. 50, 462–473.
847 https://doi.org/10.1080/02786826.2016.1162901

848 Steiling, W., Buttgereit, P., Hall, B., O'Keeffe, L., Safford, B., Tozer, S., Coroama, M.,
849 2012. Skin exposure to deodorants/anti-perspirants in aerosol form. Food
850 Chem. Toxicol. 50, 2206–2215. https://doi.org/10.1016/j.fct.2012.03.058

851 Steinle, S., Reis, S., Sabel, C.E., Semple, S., Twigg, M.M., Braban, C.F., Leeson,
852 S.R., Heal, M.R., Harrison, D., Lin, C., Wu, H., 2015. Personal exposure
853 monitoring of PM2.5 in indoor and outdoor microenvironments. Sci. Total

Environ. 508, 383–394. https://doi.org/10.1016/j.scitotenv.2014.12.003

Thatcher, T.L., Layton, D.W., 1995. Deposition, resuspension, and penetration of particles within a residence. Atmos. Environ. 29, 1487–1497. https://doi.org/10.1016/1352-2310(95)00016-R

Van Den Heuvel, R., Den Hond, E., Govarts, E., Colles, A., Koppen, G., Staelens, J., Mampaey, M., Janssen, N., Schoeters, G., 2016. Identification of PM10 characteristics involved in cellular responses in human bronchial epithelial cells (Beas-2B). Environ. Res. 149, 48–56. https://doi.org/10.1016/j.envres.2016.04.029

Wainman, T., Zhang, J., Weschler, C.J., & Lioy, P.J., 2000. Ozone and limonene in indoor air: a source of submicron particle exposure. Environ. Health Perspect. 108, 1139–1145. https://doi.org/10.1289/ehp.001081139

Wallace, L., Williams, R., Rea, A., Croghan, C., 2006. Continuous weeklong measurements of personal exposures and indoor concentrations of fine particles for 37 health-impaired North Carolina residents for up to four seasons. Atmos. Environ. 40, 399–414. https://doi.org/10.1016/j.atmosenv.2005.08.042

Welsh Government, 2019. Clean Air Plan for Wales: Healthy Air, Healthy Wales.

Wolkoff, P., Nielsen, G.D., 2017. Effects by inhalation of abundant fragrances in indoor air – An overview. Environ. Int. 101, 96–107. https://doi.org/10.1016/j.envint.2017.01.013

Wolkoff, P., Schneider, T., Kildesø, J., Degerth, R., Jaroszewski, M., Schunk, H., 1998. Risk in cleaning: Chemical and physical exposure. Sci. Total Environ. 215, 135–156. https://doi.org/10.1016/S0048-9697(98)00110-7

World Health Organization, 2018. Air Pollution and Child Health: Prescribing Clean Air: IN PRESS.

World Health Organization, 2016. WHO Expert Consultation: Available evidence for the future update of the WHO Global Air Quality Guidelines (AQGs).

World Health Organization, 2013. Review of evidence on health aspects of air pollution – REVIHAAP, World Health Organization Regional Office for Europe.

World Health Organization, 2006. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: Global update 2005. https://doi.org/10.1016/0004-6981(88)90109-6