



# Personal exposure to fine particulate matter (PM<sub>2.5</sub>) and self-reported asthma-related health

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## ABSTRACT

PM<sub>2.5</sub> (fine particulate matter  $\leq 2.5 \mu\text{m}$  in diameter) is a key pollutant that can produce acute asthma exacerbations and longer-term deterioration of respiratory health. Individual exposure to PM<sub>2.5</sub> is unique and varies across microenvironments. Low-cost sensors (LCS) can collect data at a spatiotemporal resolution previously unattainable, allowing the study of exposures across microenvironments. The aim of this study is to investigate the acute effects of personal exposure to PM<sub>2.5</sub> on self-reported asthma-related health.

Twenty-eight non-smoking adults with asthma living in Scotland collected PM<sub>2.5</sub> personal exposure data using LCS. Measurements were made at a 2-min time resolution for a period of 7 days as participants conducted their typical daily routines. Concurrently, participants were asked to keep a detailed time-activity diary, logging their activities and microenvironments, along with hourly information on their respiratory health and medication use. Health outcomes were modelled as a function of hourly PM<sub>2.5</sub> concentration (plus 1- and 2-h lag) using generalized mixed-effects models adjusted for temperature and relative humidity.

Personal exposures to PM<sub>2.5</sub> varied across microenvironments, with the largest average microenvironmental exposure observed in private residences ( $11.5 \pm 48.6 \mu\text{g}/\text{m}^3$ ) and lowest in the work microenvironment ( $2.9 \pm 11.3 \mu\text{g}/\text{m}^3$ ). The most frequently reported asthma symptoms, wheezing, chest tightness and cough, were reported on 3.4%, 1.6% and 1.6% of participant-hours, respectively. The odds of reporting asthma symptoms increased per interquartile range (IQR) in PM<sub>2.5</sub> exposure (odds ratio (OR) 1.29, 95% CI 1.07–1.54) for same-hour exposure. Despite this, no association was observed between reliever inhaler use (non-routine, non-exercise related) and PM<sub>2.5</sub> exposure (OR 1.02, 95% CI 0.71–1.48).

Current air quality monitoring practices are inadequate to detect acute asthma symptom prevalence resulting from PM<sub>2.5</sub> exposure; to detect these requires high-resolution air quality data and health information collected in situ. Personal exposure monitoring could have significant implications for asthma self-management and clinical practice.

## 1. Introduction

Exposure to air pollution is the leading environmental health threat, responsible for illnesses such as stroke, chronic obstructive pulmonary disease (COPD) and lung cancer (WHO, 2013), resulting in 7 million deaths globally each year (WHO, 2020). Though 99% of the global population breathe polluted air, the burden of morbidity and mortality

are not equally shared, with some groups (e.g., those with pre-existing disease such as asthma) most at risk (Royal College of Physicians, 2016).

Asthma is the most prevalent chronic respiratory disease (Chan et al., 2019), and over 368,000 people in Scotland receive treatment for asthma (Scottish Government, 2020a). The physiological and epidemiological links between exposure to air pollutants such as fine particulate matter (PM<sub>2.5</sub>) and acute asthma exacerbations, the longer-term

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deterioration of asthma, and the onset of asthma are well established (e.g., [Holst et al., 2020](#); [Kampa and Castanas, 2008](#); [Landrigan et al., 2018](#)), with air pollution exposure linked with oxidative stress, airway inflammation, hyperresponsiveness and, overtime, airway remodelling ([Guarnieri and Balmes, 2014](#)). Most of the epidemiological evidence that exists is based upon observational population-level health data, such as accident and emergency/emergency room visits, hospitalisations and medication administration (e.g., [Hales et al., 2021](#); [Hoffmann et al., 2022](#); [Priyankara et al., 2021](#); [Yadav et al., 2021](#)). While these approaches provide a useful perspective of health impact, the associations are spatiotemporally aggregated ([Su et al., 2017](#)) and can be influenced by underlying factors such as socioeconomic status and access to healthcare ([Williams et al., 2019](#)) and, therefore, may undermine epidemiological assessments linking health effects specifically with air pollution.

Recognising the issues associated with using aggregated health data, several studies have explored the links between air pollution exposure and individual-level asthma symptom prevalence (e.g., [de Camargo Matos et al., 2022](#); [Phaswana et al., 2022](#)) or medication (predominantly bronchodilator inhaler) use (e.g., [Su et al., 2017](#); [Williams et al., 2019](#)). However, these studies have been based on exposure assessments modelled from air quality data collected via ambient fixed-site monitoring (often at some distance from the participants' home addresses). Vitaly, such exposure assessment approaches fail to examine links with indoor or household exposures related to an individual's unique behaviours, such as cleaning and cooking ([AQEG, 2022](#)). Moreover, using modelled or surrogate exposure data can introduce bias into the personal exposure assessment ([Butland et al., 2019](#)).

Difficulties in accurately investigating personal exposure to air



**Fig. 1.** PurpleAir attached to customised backpack and powered by battery pack (inside). The PurpleAir was secured in place with Velcro to minimise agitating fibre particles and to keep the sensor as close as feasibly possible to 'breathing zone' height. When stationary for long periods, the participant was permitted to remove the PurpleAir from the backpack and keep it close-by.

pollution have, at least in part, been eased by the development of low-cost air quality monitors ([Chambers et al., 2018](#)). Low-cost monitors, owing to their small size, portability, low power requirements and high temporal resolution ([Loh et al., 2017](#); [Snyder et al., 2013](#)) can allow the assessment of exposure across microenvironments (e.g., [Steinle et al., 2015](#)). Such exposure data, paired with individual-level health data, can be used to assess linkages between microenvironmental air pollution exposures and health measures (e.g., [Hao et al., 2022](#); [Rabinovitch et al., 2016](#); [Turner et al., 2021](#)). However, such studies have, to date, mainly examined associations between exposures and asthma symptoms/medication use in prescribed microenvironments, been based on exposure metrics aggregated over relatively long timeframes, and/or based upon clinical measures (e.g., PEF, FEV<sub>1</sub>) which are not necessarily responsive to acute environmental change nor reflect patient wellbeing ([Juniper et al., 1996](#)). The aim of this study is to investigate the acute associations between personal exposure to PM<sub>2.5</sub> and self-reported asthma-related health.

## 2. Materials and methods

### 2.1. Study design and participants

Thirty-seven non-smoking adults with asthma were recruited from across Scotland between February 2021 and July 2021. Eligibility criteria included that participants must be aged 18 or older, be a non-smoker, have been diagnosed with asthma by a healthcare professional and live in Scotland. This study was nested within a larger project involving semi-structured interviews, co-design of behavioural intervention and a follow-up monitoring campaign, with the same group of participants recruited to take part in all elements. A participant advisory group consisting of five individuals meeting the same eligibility criteria was consulted to refine the project design and pilot the methodology. This informed the design of crucial study elements such as diary templates (format and resolution) and customised backpack designs (described in more detail below). Data collection for the main study took place between September 2021 and September 2022. This study was reviewed and approved by the University of Stirling's General University Ethics Panel [GUEP 2021 2506 1892].

### 2.2. Personal exposure monitoring

Each participant monitored their personal exposure to fine particulate matter (particulate matter with an aerodynamic diameter  $\leq 2.5 \mu\text{m}$  (PM<sub>2.5</sub>)) using a PurpleAir PA-II-SD air quality sensor (PurpleAir, Draper, UT, USA; hereafter referred to as PurpleAir) attached to a customised backpack ([Fig. 1](#)). Data collection took place over one week per person to capture typical weekly variations in ambient PM<sub>2.5</sub> and to capture participants' weekly routines. The PurpleAir measures particles using Plantower PMS 5003 air quality sensors, in addition to relative humidity, temperature and barometric pressure (Bosch, Reutlingen, Germany). Laser counters take readings every 5 s, with averages logged to an SD card every 120 s. Prior to data collection, all 16 PurpleAirs used in this study were collocated for one-week for interunit comparability. Since collocation could not take place with a reference-grade monitor, it was accepted that the median value of all 16 sensors was the 'true' value. Each sensor was individually plotted against the 'true' value and subsequent equations were used to adjust sensor outputs. Statistical

**Table 1**

Personal exposure metrics. Calculations are based on calendar hour (i.e., between 18:00–19:00).

Personal exposure metric	Description
Hour mean	Mean value for each hour
Hour max	Maximum value for each hour
Within-hour range	Maximum value minus minimum value calculated for each hour
Within-hour increase	Maximum value minus minimum value calculated only where minimum timestamp precedes maximum timestamp

analyses were conducted on participant-hour natural log transformed PM<sub>2.5</sub> exposure data to counter skewness. Exposure variables examined included mean, maximum, within-hour range and within-hour increase (defined as the within-hour range calculated only where minimum timestamp precedes maximum timestamp) (Table 1). Hourly average PM<sub>2.5</sub> ambient air quality data were obtained from a fixed-site monitoring station closest to the participant's residential address for the sampling week. Data were obtained from the Air Quality in Scotland website (<https://www.scottishairquality.scot>).

Alongside personal exposure monitoring, participants were asked to complete a time-activity diary (see Supplementary Material) to support the pairing of PM<sub>2.5</sub> mass concentrations with activities and microenvironments. Time-activity diary templates were set at 1-h time intervals, with activity being a free-text response since it would not be possible to capture the entire range of possible activities. Microenvironment details were captured by check box, based on categories from previous studies (e.g., Steinle et al., 2015). These encompassed broad, general categories such as 'transport' and 'public building,' as well as more precise environments within the home (e.g., 'kitchen,' 'bedroom,' 'living room'). An 'other' option was provided for cases where required.

### 2.3. Asthma-related health

Participants were asked to keep a record of their asthma-related health for the duration of the monitoring week via a time-activity diary. Self-reported asthma symptoms were recorded hourly. This was designed as a free-text response to allow participants to describe their asthma symptoms in their own terms. These were reviewed by the researcher and subsequently grouped into broader terms (e.g., short of breath, out of breath, and struggling to catch my breath were grouped as breathless). Inhaler and other asthma medication use was recorded at hourly intervals with check box options (nil, preventer, reliever, other) and, where applicable, a space for time administered. To distinguish between routine or prescribed use of medication and when medication was used for the more immediate relief of asthma symptoms, participants were asked, "If you used your inhaler or asthma medication, why?" Again, this was an open-text response to realise the entire range of possible reasons.

### 2.4. Baseline survey/covariates

Participants were asked to complete a survey once during the baseline monitoring campaign. The survey was designed to capture contextual information as in previous work (e.g., Steinle et al., 2015), including personal information (i.e., age, gender), information about their neighbourhood, their home environment and building characteristics, other householders and their typical behaviours within the home. These data were included in the model selection process as potential confounders/candidate variables in adjusted models.

### 2.5. Statistical analyses

Symptom prevalence (coded as a binary variable) and non-routine or exercise-related inhaler use were modelled as a function of different metrics of PM<sub>2.5</sub> personal exposure (see section 2.2) using mixed-effects logistic models with random intercepts for ID to account for person-level clustering within the data and repeated-measures design. Similarly, to test the association between symptom prevalence and environmental factors (temperature and relative humidity) distinctly from PM<sub>2.5</sub>, these were modelled as a function of same-hour, 1-h, and 2-h lag average PM<sub>2.5</sub>. Hourly aggregated PM<sub>2.5</sub> exposure data were also tested at 1-h and 2-h post-exposure to measure the potential continued impact of exposure on participants' self-reported health (Bancalari et al., 1999; O'Byrne, 2009). Odds Ratios (OR), a statistic which quantifies the strength of associations, are presented per interquartile range (IQR) increase in PM<sub>2.5</sub> concentration. The same analyses were repeated

substituting personal exposure data for data collected via fixed-site monitoring station. The correlation between both measures was tested using Spearman's correlation.

Model selection followed a stepwise selection approach, whereby variables were added and removed at different stages to achieve the

**Table 2**

Demographic and descriptive characteristics (n = 28). Urban/rural classifications are based on Scottish Government definitions (Scottish Government, 2020b). \*n = 27 due to nonresponse. Scottish Index of Multiple Deprivation (SIMD).

Participant characteristic	Statistic
<b>Age (years, mean (range))</b>	47.5 (24–74)*
<b>Gender (n (%))</b>	
Female	19 (67.9)
Male	9 (32.1)
<b>Other respiratory condition (n (%))</b>	
No	26 (92.9)
Yes	1 (3.6)
Missing	1 (3.6)
<b>Pregnant (n (%))</b>	
No	28 (100)
Yes	0 (0)
<b>SIMD Decile (n (%))</b>	
1	0 (0)
2	0 (0)
3	1 (3.6)
4	2 (7.1)
5	5 (17.9)
6	4 (14.3)
7	4 (14.3)
8	2 (7.1)
9	3 (10.7)
10	7 (25.0)
<b>Type of dwelling (n (%))</b>	
Apartment	10 (35.7)
Semi-detached house	4 (14.3)
Detached house	5 (17.9)
Detached bungalow	3 (10.7)
Detached cottage	2 (7.1)
Terraced house	3 (10.7)
Missing	1 (3.6)
<b>Number of residents (n, mean, (range))</b>	2.5 (1–5)
<b>Live with pets (n (%))</b>	
No	12 (42.9)
Yes	15 (53.6)
Missing	1 (3.6)
<b>Live with smoker (n (%))</b>	
No	27 (96.4)
Yes	0 (0)
Missing	1 (3.6)
<b>Have a solid fuel burner (n (%))</b>	
No	20 (71.4)
Yes	6 (21.4)
Missing	2 (7.1)
<b>Type of hob (n (%))</b>	
Gas	9 (32.1)
Other (electric, induction)	18 (64.3)
Missing	1 (3.6)
<b>In employment (n (%))</b>	
No	6 (21.4)
Yes	22 (78.6)
<b>Urban-rural Classification (n (%))</b>	
Large urban area	8 (28.6)
Other urban area	9 (32.1)
Accessible small town	1 (3.6)
Remote small town	2 (7.1)
Very remote small town	1 (3.6)
Accessible rural	5 (17.9)
Remote rural	0 (0)
Very remote rural	2 (7.1)
<b>Distance of home address from fixed-site monitor (km)</b>	
Max	125.0
Mean	20.6
Median	4.0
Min	0.1

best-fitting model (Chowdhury and Turin, 2020). To assess model fit, the Akaike information criterion (AIC), second-order Akaike information criterion (AIC<sub>c</sub>) and Bayesian information criterion (BIC) were considered. Final models included variables that were significant predictors and/or had a theoretical basis for their inclusion (Steyerberg and Vergouwe, 2014). All analyses were conducted using RStudio version 4.2.2 (R Core Team, 2022) using the packages lme4 (Bates et al., 2015), sjPlot (Lüdtke, 2018) and ggplot2 (Wickham, 2016).

### 3. Results

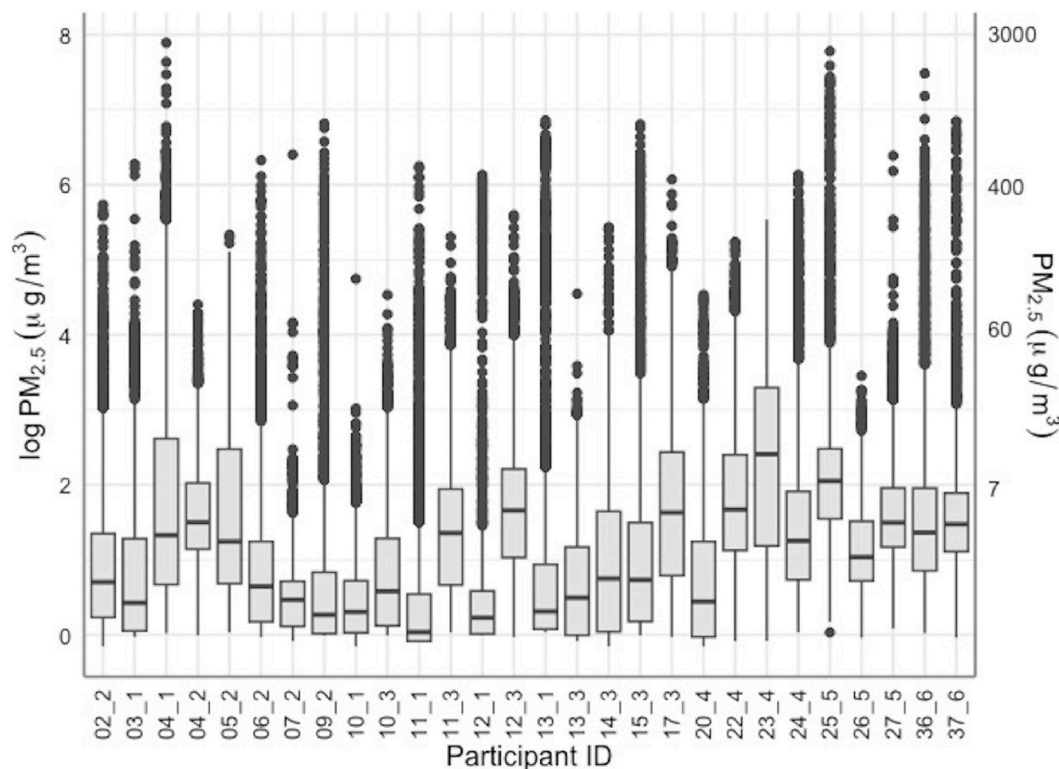
#### 3.1. Participant descriptive characteristics

Twenty-eight participants were included in the final analyses, with nine participants excluded owing to sensor malfunction (n = 2), equipment non-return (n = 1), incomplete/illegible diary data (n = 5) and respiratory illness during the monitoring campaign (n = 1). Demographic and descriptive characteristics are presented in Table 2. After data cleaning (averaging periods only where >75% data were available), 143 participant days and 4032 participant hours remained.

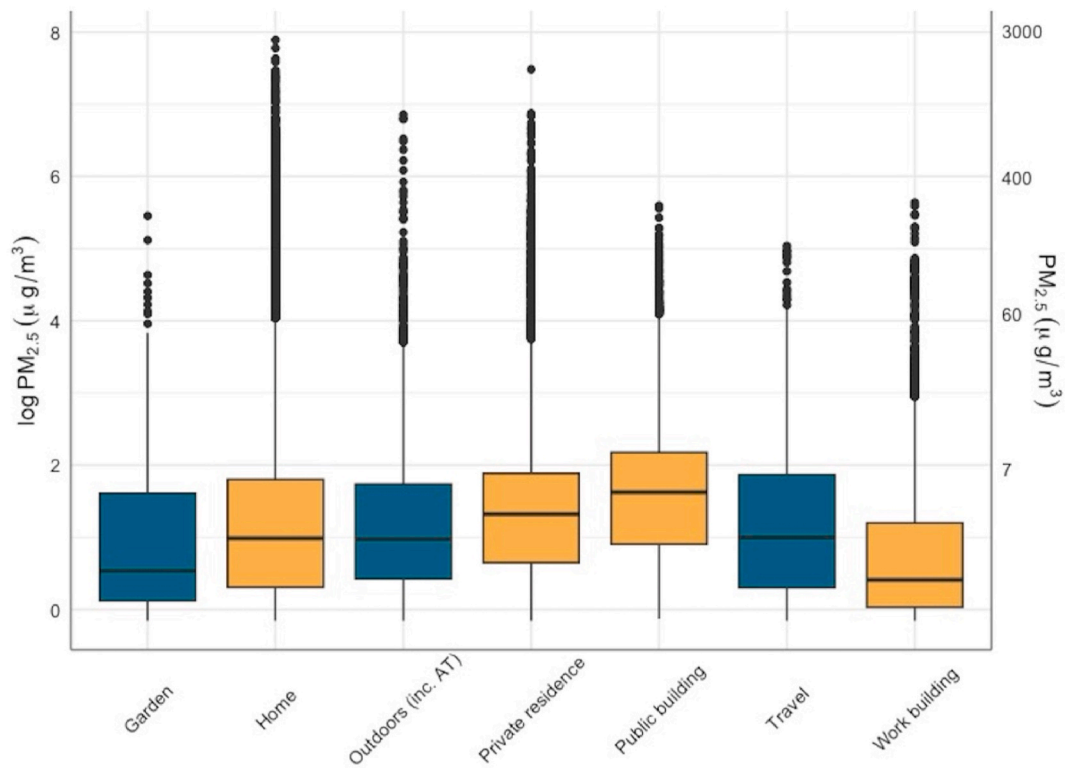
PM<sub>2.5</sub> personal exposures varied highly both within and between

participants, with seven-day averages ranging from  $1.0 \pm 2.5 \mu\text{g}/\text{m}^3$  to  $26.2 \pm 93.1 \mu\text{g}/\text{m}^3$  (Fig. 2). This variability was also reflected in microenvironmental exposure statistics with the greatest average microenvironmental exposure in the private residential microenvironment ( $11.5 \pm 48.6 \mu\text{g}/\text{m}^3$ ) and lowest in work buildings ( $2.9 \pm 11.3 \mu\text{g}/\text{m}^3$ ) (Fig. 3). Participants spent over 90% of participant-hours in indoor microenvironments, of which 80% of this time was spent in the home microenvironment (2904h). The least time was spent in the outdoor microenvironment (including 'garden' and active travel) (145h, 3.6% participant hours). Microenvironmental data were unavailable for 3.5% of the total monitoring period. Fixed-site monitor concentration average was  $5.0 \mu\text{g}/\text{m}^3$  ( $0.2\text{--}42.8\mu\text{g}/\text{m}^3$ ) during the participant measurement weeks.

Incidences of reliever inhaler use were low (n = 67) and varied between participants, with 12 participants recording no uses and one participant recording 15 uses over the monitoring week. Participants reported one or more symptoms on 451h during sampling (11% of participant-hours). This too was very variable between participants, with five participants experiencing/reporting no symptoms over the week and one participant reporting 151h with experience of symptoms.



**Fig. 2.** Natural log transformed PM<sub>2.5</sub> personal exposure collected via PurpleAirs over the monitoring week by participant. Whiskers extend to the minimum and maximum values. The lower end of the box represents Q1 (lower quartile) and the upper end of the box Q3 (upper quartile). The median value is denoted by the black line inside the box. Black dots represent outliers.



**Fig. 3.** Natural log transformed PM<sub>2.5</sub> personal exposure by the microenvironment. Whiskers extend to the minimum and maximum values. The lower end of the box represents Q1 (lower quartile) and the upper end of the box Q3 (upper quartile). The median value is denoted by the black line inside the box. Black dots represent outliers. Yellow boxes denote indoor environments, and blue boxes denote outdoor environments. Outdoor includes active travel (AT). Mean values: Garden = 5.0 µg/m<sup>3</sup>; Home = 9.0 µg/m<sup>3</sup>; Outdoors = 8.6 µg/m<sup>3</sup>; Private residence = 11.5 µg/m<sup>3</sup>; Public building = 8.2 µg/m<sup>3</sup>; Travel = 5.1 µg/m<sup>3</sup>; Work building = 2.9 µg/m<sup>3</sup>. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

**Table 3**

Odds Ratio and 95% CI for the associations between personal exposure to PM<sub>2.5</sub> and symptom prevalence. Statistically significant estimates (p < 0.05) are highlighted in bold. Personal exposure adjusted model includes temperature and humidity and fixed-site adjusted model includes residential distance from monitoring station.

	Odds Ratio (95% CI) per IQR increase in PM <sub>2.5</sub> personal exposure					
	Adjusted			Unadjusted		
	Same-hour	1h lag	2h lag	Same-hour	1h lag	2h lag
Hour mean	<b>1.29 (1.07–1.54)</b>	<b>1.24 (1.04–1.49)</b>	1.09 (0.90–1.31)	<b>1.24 (1.04–1.48)</b>	<b>1.27 (1.06–1.51)</b>	1.12 (0.93–1.35)
Hour max	<b>1.32 (1.12–1.55)</b>	<b>1.28 (1.09–1.51)</b>	1.12 (0.95–1.32)	<b>1.30 (1.12–1.52)</b>	<b>1.31 (1.12–1.54)</b>	1.15 (0.98–1.36)
Within hour range	<b>1.12 (1.04–1.20)</b>	<b>1.12 (1.04–1.20)</b>	1.07 (0.99–1.14)	<b>1.12 (1.05–1.19)</b>	<b>1.13 (1.06–1.20)</b>	<b>1.08 (1.01–1.16)</b>
Within hour increase	1.06 (0.96–1.17)	<b>1.14 (1.04–1.26)</b>	<b>1.13 (1.02–1.24)</b>	1.05 (0.95–1.15)	<b>1.14 (1.04–1.26)</b>	<b>1.13 (1.02–1.24)</b>
Fixed-site PM <sub>2.5</sub> concentration	1.10 (0.89–1.34)	1.05 (0.86–1.29)	0.97 (0.79–1.20)	1.10 (0.89–1.34)	1.05 (0.86–1.29)	0.97 (0.79–1.20)

### 3.2. Personal exposures and asthma-related health

The most frequently reported single asthma symptoms, wheeze, chest tightness and cough, were reported on 3.4%, 1.6% and 1.6% of participant-hours, respectively. Symptom prevalence-exposure models included temperature and humidity as covariates with other variables dropped due to insignificance. Symptom prevalence odds ratios for hourly PM<sub>2.5</sub> exposure metrics are summarised in Table 3. Associations between PM<sub>2.5</sub> personal exposure and asthma symptom prevalence

showed similar temporal trends for mean, maximum and range exposure metrics, with same-hour personal exposure associated with the greatest OR for symptom prevalence (Table 3; Fig. 4). With a 1-h lag effect for the same exposure metrics, OR for symptom prevalence decreased but remained positively and significantly associated (Table 3). However, with a 2-h lag effect, no significant (albeit consistently positive) associations were observed. Discordantly, symptom prevalence was not found to be significantly associated with same-hour increase personal exposure but was found to be positively and significantly associated with

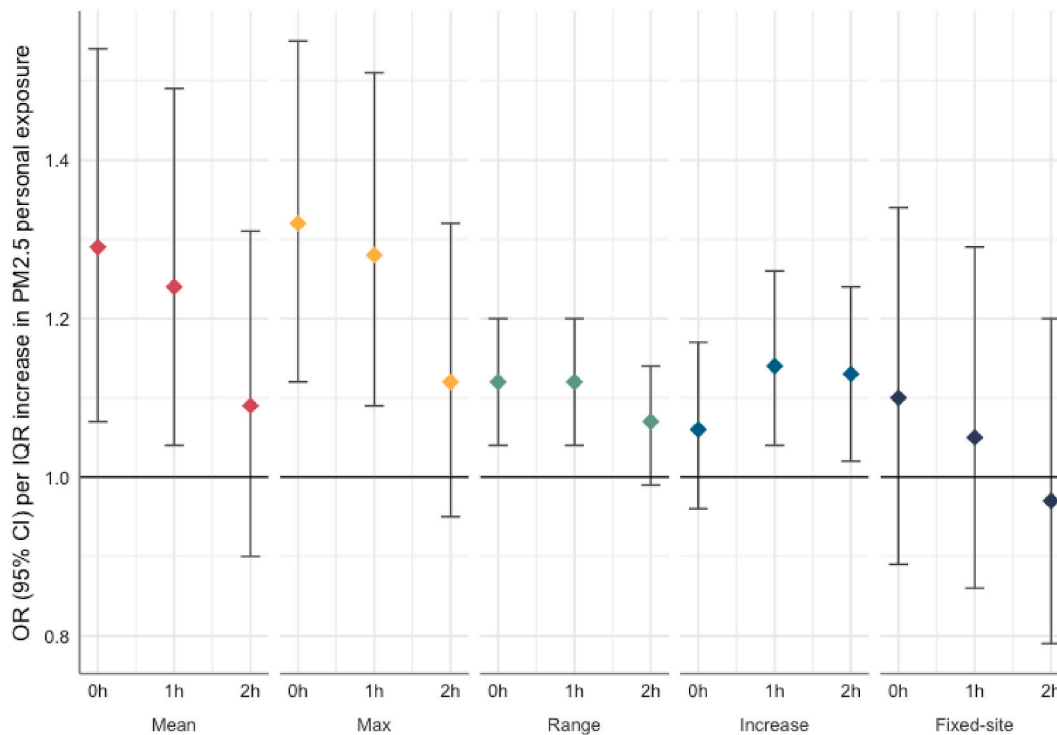


Fig. 4. Odds Ratio and 95% CI for the associations between personal exposure to PM<sub>2.5</sub> and symptom prevalence for same-hour (0h), 1h and 2h lag.

Table 4

Odds Ratio and 95% CI for the associations between personal exposure to PM<sub>2.5</sub> and reliever inhaler use. Statistically significant estimates ( $p < 0.05$ ) are highlighted in bold. Personal exposure adjusted model includes temperature and humidity and fixed-site adjusted model includes residential distance from monitoring station.

	Odds Ratio (95% CI) per IQR increase in PM <sub>2.5</sub> personal exposure					
	Adjusted			Unadjusted		
	Same-hour	1h lag	2h lag	Same-hour	1h lag	2h lag
Hour mean	1.02 (0.71–1.48)	0.81 (0.53–1.24)	0.76 (0.49–1.17)	1.03 (0.72–1.49)	0.83 (0.55–1.25)	0.78 (0.51–1.19)
Hour max	1.11 (0.81–1.53)	0.85 (0.58–1.23)	0.78 (0.53–1.15)	1.12 (0.82–1.52)	0.86 (0.60–1.23)	0.80 (0.55–1.16)
Within hour range	1.11 (0.98–1.27)	0.92 (0.76–1.12)	0.92 (0.76–1.11)	1.11 (0.97–1.26)	0.92 (0.76–1.11)	0.92 (0.76–1.11)
Within hour increase	1.00 (0.81–1.23)	1.08 (0.88–1.33)	1.00 (0.81–1.24)	1.00 (0.82–1.23)	1.08 (0.88–1.32)	1.00 (0.82–1.24)
Fixed-site PM <sub>2.5</sub> concentration	0.92 (0.61–1.38)	0.85 (0.56–1.29)	0.88 (0.58–1.34)	0.92 (0.61–1.39)	0.85 (0.56–1.29)	0.88 (0.58–1.34)

a 1-h and 2-h lag effect. Testing the association between symptom prevalence and temperature and relative humidity revealed significant negative associations, with a one-unit increase in average temperature associated with a decrease of 0.03 in the log-odds of experiencing symptoms ( $p < 0.05$ ) and a one-unit increase in average humidity associated with a decrease of 0.03 in the log-odds of experiencing symptoms ( $p < 0.05$ ). Both symptom prevalence with a 1-h and 2-h lag was not significantly associated with average temperature and relative humidity.

Inhaler use exposure models also contained temperature and humidity as potential covariates within the model. Across PM<sub>2.5</sub> personal exposure metrics for same hour, positive but not statistically significant associations with reliever inhaler use were observed (Table 4; Fig. 5). For hour mean, maximum and range metrics with a 1-h and 2-h lag

effect, a negative but, again, not statistically significant association was observed (Table 4; Fig. 5).

### 3.3. Fixed-site monitoring data and asthma-related health

Models using fixed-site air quality data included distance from the closest fixed-site station as a control variable. Despite hourly averaged fixed-site and personal exposure PM<sub>2.5</sub> data being moderately correlated (Spearman  $r = 0.44$ ,  $p < 0.05$ ), there were no significant associations between asthma symptom prevalence and fixed-site ambient levels of PM<sub>2.5</sub> (Table 3; Fig. 4). Neither a 1-h nor 2-h lag effect was found to be associated. Additionally, no significant associations were observed for reliever inhaler use and fixed-site ambient PM<sub>2.5</sub> (Table 4; Fig. 5).

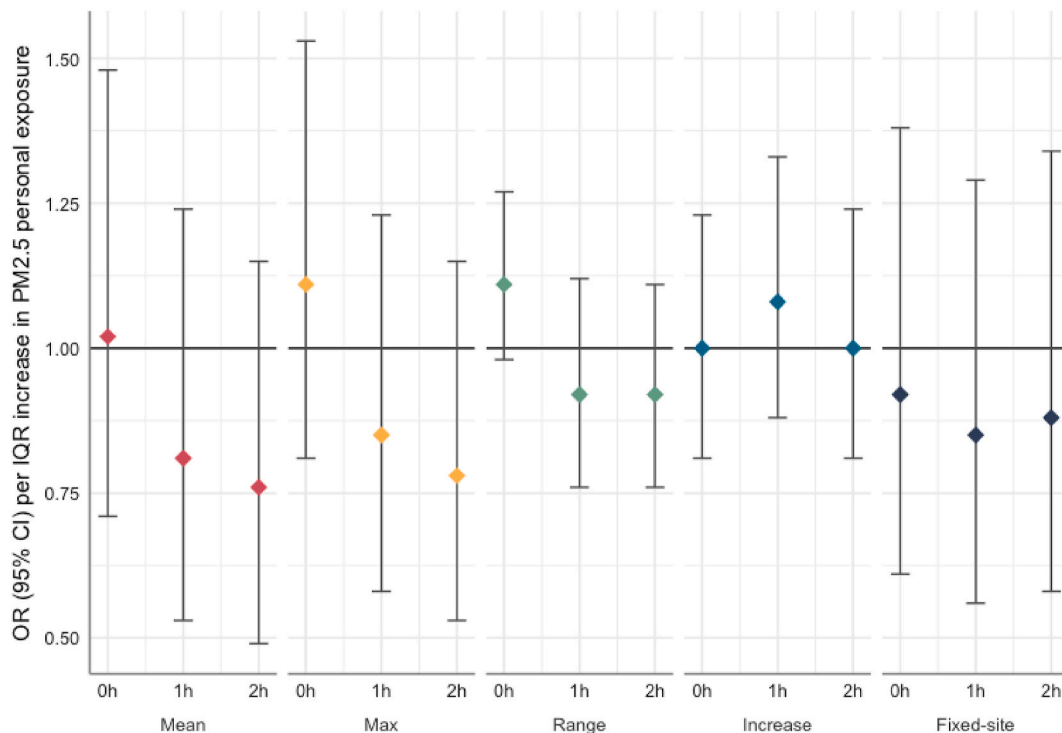


Fig. 5. Odds Ratio and 95% CI for the associations between personal exposure to  $PM_{2.5}$  and reliever inhaler use for same-hour (0h), 1h and 2h lag.

#### 4. Discussion

Using directly measured hourly  $PM_{2.5}$  personal exposure data paired with individual-level health data, this study has investigated the acute associations between personal exposure to  $PM_{2.5}$  and self-reported asthma-related health.

The association between increased  $PM_{2.5}$  exposure and increased asthma symptom prevalence has been relatively well demonstrated within the literature; however, these studies tend to examine temporally aggregated air quality data ranging from daily (e.g., Šcibor et al., 2022) to biweekly (e.g., Mirabelli et al., 2018) to annual exposure data (e.g., Doiron et al., 2017). Adding to this previous work, findings from this study have revealed significant positive associations between same-hour and 1-h lag personal exposure to  $PM_{2.5}$  and symptom prevalence. Results show that same-hour personal exposure to  $PM_{2.5}$  was most strongly associated with symptom prevalence, decreasing with each hour lag. By the 2-h lag, the association remained positive but was no longer statistically significant, suggesting that short-term exposures play an important role in acute asthma symptom prevalence. Previous immunological work on allergen-induced asthma has shown that the release of inflammatory mediators resulting in bronchoconstriction occurs within 15 min of exposure and lasts between one and three h (Bancalari et al., 1999; O'Byrne, 2009). We have shown that personal exposure monitoring using high-frequency sampling, paired with hourly-monitored health outcome data, can support detection of acute asthma symptom prevalence associated with personal exposure to  $PM_{2.5}$ . Such an approach, from an asthma-management perspective, could be a fundamental step in identifying activities and/or microenvironments associated with increased exposure thus has potential to inform decision making and drive behaviour changes.

This study has demonstrated that high temporal resolution fixed-site monitoring data are unable to detect acute environmental changes inherent to personal exposures and its impacts on asthma-related health. Personal exposures have often been estimated based upon fixed-site monitoring or modelled data (e.g., Su et al., 2017; Williams et al., 2019). To compare the health effect estimates using both approaches,

hourly averaged  $PM_{2.5}$  data retrieved from the closest fixed-site monitoring station to the participants' home addresses were used as a surrogate for directly monitored personal exposure data collected via PurpleAir. Using this surrogate approach, no significant associations were found for same-hour, 1-h or 2-h lag. This contrasts with much of the literature which has found significant associations using fixed-site data (e.g., de Camargo Matos et al., 2022; Phaswana et al., 2022). This may be due to the acute (one week per participant) nature of this study since correlations between fixed-site monitoring data and personal exposure data have been shown to increase with time (Hutcheon et al., 2010; Strand et al., 2007). However, regardless of timeframe, fixed-site data cannot (and are not designed to) detect the inherent heterogeneity in personal exposures that arise from individual behaviours, particularly within indoor environments (i.e., cooking behaviours, second-hand tobacco smoke exposure, home heating behaviours) (McCarron et al., 2022).

Fixed-site monitoring, by design, monitors ambient air quality predominantly influenced by outdoor sources, such as vehicle and industrial emissions. A subsidiary finding from this study arising from time-activity monitoring is the proportion of time participants, adults with a diagnosis of asthma, spent across different microenvironments, spending only 3.6% of their time in outdoor spaces and, as many other studies have found, more than 90% of their time indoors (e.g., Mazaheri et al., 2018). Results from this study reveal that greatest exposure to  $PM_{2.5}$  occurs in residential buildings (i.e., when visiting friends/relatives or in participants' own homes (which could also explain the null finding from the fixed-site data)). As such, using fixed-site data as a proxy for personal exposure may lead to exposure misclassification (De Hartog et al., 2010). Evangelopoulos et al. (2021) discuss that, in most cases, using ambient concentrations as a proxy for personal exposure will overestimate exposure since not all ambient pollution will ingress indoors where people spend most of their time. Though true, this downplays the importance of indoor exposure, considering that indoor microenvironments contribute substantially to personal exposures due to prolonged duration and closer proximity to sources. Moreover, Habre et al. (2014) found that indoor sources account for almost three-quarters

of PM<sub>2.5</sub> mass within the home. This highlights the spatial inadequacies of using fixed-site data as a proxy for personal exposure and upon which to assess epidemiological impacts. Furthermore, current health-based guidance concerning exposure to air pollution is based upon data monitored and modelled using fixed-site data and primarily focuses on outdoor avoidance behaviours to reduce exposure to outdoor air pollution. While the avoidance of triggers is critical for asthma control (Papaioannou et al., 2015), such advice ignores the significant contributions of highly variable indoors sources, as demonstrated in this study and, owing to the small proportion of time individuals spend in outdoor microenvironments, is unlikely to have a significant impact. Papaioannou et al. (2015) suggest that personalised management is key to achieving asthma control. Research is needed to test the feasibility of personal exposure feedback as an asthma-management strategy.

As this and other studies have demonstrated, exposure to PM<sub>2.5</sub> can trigger the precipitation of asthma symptoms. Despite personal exposure to PM<sub>2.5</sub> being positively associated with symptom prevalence, the same results were not found for inhaler use. It was hypothesised that since reliever inhalers are the “first line of defence against asthma exacerbation” (Williams et al., 2019, pg. 5250), an association may exist between inhaler use and PM<sub>2.5</sub> exposure. Conversely, results from this study suggest that there are no significant associations between hourly personal exposure and inhaler use for any metric of personal exposure and for any (analysed) lag effect. Our results conflict with similar works in this field (e.g., Ścibor et al., 2022; Su et al., 2022; Williams et al., 2019) with suggestions for this disparity previously discussed. The null result is potentially fuelled by a relatively small sample size and a high number of inhalers use non-events within the sample, meaning our analysis may be underpowered to detect significant associations. 43% of participants did not report any use of their reliever inhaler during the study, which, in itself, is insightful. Non-adherence is a measured phenomenon, with Price et al. (2013) reporting that between 40 and 60% of people with asthma are non-adherent to their medication and note that there are several multifaceted reasons for this. The invisible nature of air pollution, along with phenomena such as the home or neighbourhood ‘halo effect’ (Bickerstaff and Walker, 2001; Hofflinger, 2019), may influence how individuals appraise the threat (Rogers, 1983) that air pollution poses to their asthma-related health thus making them less likely to use their inhaler than when triggers are more perceivable (i.e., pet dander, cold weather). This indicates that a refocus of air pollution within clinical practice may be required, ensuring patients are aware of air pollution as an asthma trigger and subsequently promoting appropriate use of reliever inhalers to alleviate all asthma symptoms, not only those with obvious, visible triggers.

The monitoring methodology is both a strength and a limitation of this study. While the use of low-cost sensors allows for the collection of air quality data across microenvironments giving a more accurate indication of personal exposure, reliability rests on the correct use of the device (i.e., close to breathing zone height, always in the same environment as the participant). Though participants were trained on using the sensor (via a printed guidebook and online videos), there is no way to assess compliance during data collection. A few participants had missing air quality data for short periods of their monitoring campaign as a result of batteries running out of charge or power cables disconnecting. Simple alterations to the monitoring equipment, such as fixed cables, could overcome this simple issue and result in a more complete dataset. Additionally, time-activity diaries were used to collect data central to analysis within this study. While these were developed alongside a participant advisory group and considered to be the most ‘accessible’ format for recording information, issues with time-activity diaries have been discussed extensively in the literature. Inaccuracies, missing data, recall error, incompletion and the burdensome nature of data collection may hinder the reliability of data collected (Broderick et al., 2003; Jordan et al., 2006; Sternfeld et al., 2012). Future research should examine the agreement between diaries and ancillary environmental data collected via low-cost sensors to assess accuracy.

Additionally, the generalisability of the results from this study are limited. Though care was taken to recruit a broadly representative sample, since this study relied on voluntary participation, it will suffer from selection bias, with people more concerned or impacted by air pollution more likely to volunteer, and those with greater capacity (i.e., time, energy) more able to participate. We struggled to recruit people from Scottish Index of Multiple Deprivation (SIMD) deciles one and two, representing the most deprived areas, where there is likely different environmental susceptibility to air pollution (e.g., housing, access to healthcare, smoking behaviours, occupational exposures, modes of travel) (Royal College of Physicians, 2016). On a broader scale, ambient air quality in Scotland (which plays a role in personal exposure) is generally very good in comparison to other countries, and the underlying factors that influence household exposures are very different globally.

In conclusion, we have found compelling evidence which suggests that high-resolution data (both spatially and temporally) is required to detect the impact of PM<sub>2.5</sub> exposure on acute asthma-related health impacts. We have demonstrated that current monitoring practices are inadequate to assess these acute impacts, and we suggest that personal exposure data can be better used both in asthma self-management and clinical practice as an effective asthma-management strategy.

### Ethical approval

Ethical approval for this study was granted by the University of Stirling’s General University Ethics Panel [GUEP 2021 2506 1892].

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### CRediT author statement

**AMcC:** Conceptualisation, formal analysis, data curation, writing-original draft, visualisation. **SS:** Conceptualisation, writing-review & editing. **CB:** Conceptualisation, writing-review & editing. **VS:** Conceptualisation, writing-review & editing. **CG:** Conceptualisation, writing-review & editing. **HP:** Conceptualisation, writing-review & editing.

### Declaration of competing interest

None.

### Data availability

Data will be made available on request.

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### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.socscimed.2023.116293>.



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