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Residential indoor exposure to fine and ultrafine particulate air pollution in association with blood pressure and subclinical central haemodynamic markers of cardiovascular risk among healthy adults living in Perth, Western Australia

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Abstract
Despite that large percentages of individual daily time is spent in the home, few studies have examined the relationship between indoor particulate matter (PM) exposure in residential settings with subclinical indicators of cardiovascular risk. This cross-sectional study investigated associations between exposure to fine ($PM_{2.5}$) and ultrafine (UFP) PM in domestic indoor environments, with central blood pressure (BP) and component BP measures (pulse pressure, augmented pressure [AP], augmentation index [AIx], mean arterial pressure, pulse wave velocity [PWV]) in 40 non-smoking, otherwise healthy adults (58% women) living in Perth, Western Australia. Overall, in adjusted models, an interquartile range (IQR) increase in $PM_{2.5}$ was associated with a 3.2 mmHg (95% confidence interval [CI]: 0.99, 5.45) higher diastolic BP, and a 1.8 mmHg lower AP (95%CI: − 3.63, − 0.01) and 0.4 m/s PWV (95%CI: − 0.80, − 0.08), respectively. For the UFP fraction, an IQR increase was associated with a 5.2% higher AIx (95%CI: 0.51, 9.97) and a 0.6 m/s lower PWV (95%CI: − 1.00, − 0.11).
When stratified by sex, higher UFP concentrations were associated with higher DBP and lower PWV among women. Among men, higher UFP concentrations were associated with lower AP. Exposure to domestic indoor fine and ultrafine PM was associated with preclinical indicators of cardiovascular risk and some of these relationships were affected by sex. These findings contribute important evidence linking low-level residential indoor PM exposure with measurable impacts on cardiovascular physiology and may inform preventative recommendations as part of risk profiles for susceptible individuals.

Keywords Indoor air pollution · Ultrafine particles · Particulate matter · Blood pressure · Cardiovascular risk factors · Vascular function

Introduction

Exposure to particulate matter (PM) has been associated with cardiovascular (CV) morbidity and mortality (Brook et al. 2010; Corlin et al. 2018; Giorgini et al. 2016; Kelly and Fussell 2019; Landrigan et al. 2018; van Nunen et al. 2021). While a vast array of studies provides strong support for a causal relationship between exposure to PM smaller than 2.5 µm ('fine' PM; $PM_{2.5}$) and CV effects (Brook et al. 2010; Giorgini et al. 2016), there is increasing conversation about the notion that different sizes appear to carry different abilities to cause harmful effects, and systemic CV effects could be favoured by a smaller particle size.

The most studied PM size fractions linked to CV outcomes include ambient $PM_{2.5}$ and PM_{10} ('thoracic' PM with an aerodynamic diameter of ≤ 10 µm) (Vardoulakis et al. 2020). However, less is known about the impact of ultrafine particles (UFP; < 0.1 µm aerodynamic diameter) on CV effects and indicators of CV risk (Al-Kindi et al. 2020; Corlin et al. 2018; HEI Review Panel on Ultrafine Particles 2013). UFP are mainly derived from combustion

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processes and have strong spatial and temporal components in that they are typically short lived and are influenced by proximity to an emission source (Al-Kindi et al. 2020; HEI Review Panel on Ultrafine Particles 2013). Compared to the larger size fractions, explicit characteristics of UFP such as high particle numbers, high surface area to mass ratio, oxidative stress potential, ability to act as vectors for vapours and gases condensed on their surface, and probable systemic penetration may considerably increase their CV toxicity. However, this data is only now starting to emerge (Al-Kindi et al. 2020; Brook et al. 2010; Downward et al. 2018; Soppa et al. 2019).

Compared to ambient PM, less is known about residential indoor exposure to PM. It is well reported that most people spend over 80–90% of their daily time indoors with up to 60% spent in the home (Brasche and Bischof 2005; Klepeis et al. 2001; Leech et al. 2002). Residential sources of PM are abundant and concentrations are mostly related to the intrusion of airborne particles from outdoors, indoor emission sources including gas cooking and heating, and the use of aerosol sprays and cleaning products (Al-Kindi et al. 2020). The indoor home environment may therefore represent a major contribution to total personal PM exposure.

In the literature, most reported relationships are between ambient PM exposure and cardiovascular disease (CVD) endpoints such as heart disease and stroke, although findings have yielded mixed results (Brook et al. 2010; Chan et al. 2015; Choi et al. 2019; Downward et al. 2018; Giorgini et al. 2016; Honda et al. 2018). Furthermore, limited research has been undertaken which considers the relationship between PM and preclinical prognostic markers of CV risk, which may be involved in the pathogenesis of clinical CVD. These markers, including blood pressure (BP) and BP component measures (e.g., pulse pressure (PP), mean arterial pressure (MAP), augmented pressure (AP), augmentation index (AIx), pulse wave velocity (PWV)), quantify CV function and changes can indicate vascular disease, and independently predict CV events and mortality (Honda et al. 2018; Liao and Farmer 2014; Vlachopoulos et al. 2010).

Furthermore, very few studies have evaluated subclinical CV parameters including BP and BP component measures in relation to domestic indoor exposures to PM. Additionally, to our knowledge, of the few studies that were identified through literature search, all investigate very defined exposure settings such as those related to biomass fuel combustion in low- or middle-income countries (Alexander et al. 2015; Baumgartner et al. 2018, 2011; Baumgartner and Clark 2016; Clark et al. 2019; Young et al. 2019), or in high-income countries, different types of cookstove technologies (Fedak et al. 2019; Walker et al. 2020).

In this present study, we investigated the association between indoor residential exposure to fine and ultrafine PM, with functional intermediate measures of CV risk including BP and component measures of BP, in a population of healthy middle-aged adults living in Perth, Western Australia.

Methods

Study population and study session protocol

From March 2017 until May 2018, a cross-sectional sample of 40 adults living in 40 residences located in Perth, Western Australia, was recruited. Eligible participants were non-smokers aged between 35 and 69 years. Those who reported a history of CV morbidity or CVD took antihypertensive, antidiabetic, or lipid-modifying medications, or were unable to provide written consent, were excluded from the study. Participants with CVD were excluded as the study aimed to investigate subclinical markers of CV risk. Residences included in the study were smoke-free, standalone single-family dwellings, or group housing defined as sharing a common wall with a neighbouring property (e.g., apartments, flats, townhouses).

Data was collected in a two-stage process including the 'home stage' involving a 24-h continuous monitoring of indoor concentrations of $PM_{2.5}$ and UFP simultaneously with central ambulatory haemodynamic parameters. The second stage involved a clinic assessment to gather information on each participant's current health status including anthropometric measurements, to take blood samples to establish a lipid profile, and to determine central pulse wave velocity (PWV). Participants were offered the opportunity to select which stage was undertaken first; however, both stages were to be completed within a 14-day maximum time period. This time period was selected to accommodate equipment and investigator availability, and for the convenience of participants.

Further details of the main study protocol have been published elsewhere (Gilbey et al. 2019) and are described briefly below.

Written informed consent was obtained from all individual participants included in the study. The study protocol was approved by the Human Research Ethics Committee of Curtin University (HRE2016-0308).

Indoor air quality measurements

Each participating household was monitored for $PM_{2.5}$ and UFP using air monitoring instrumentation that was co-located in the main living area. Equipment was placed at a height of 1.5 m corresponding approximately to the breathing zone of a standing adult.

$PM_{2.5}$ concentrations were measured for 24 h using a DustTrak light scattering photometer (DRX 8533, TSI Inc.,

Shoreview, MN, USA). DustTrak is a real-time monitor that displays particle mass concentrations in units of micrograms per cubic metre (μg/m^3; g × 10^{-6}). Data was logged at 5-min intervals. The measuring range of the instrument is 1 μg/m^3 to 150 × 10^3 μg/m^3 with accuracy of ± 0.1% of the reading or 1 μg/m^3, whichever is greater.

A portable P-Trak 8525 (TSI Inc., Shoreview, MN, USA) was used to detect and count UFP numbers in real time with particle concentration being displayed in units of particles per cubic centimetre (particles/cm^3). This instrument has a limit of operation of 8 h at 21 °C and a measuring range of 0 to 5 × 10^5 particles/cm^3. This instrument detects and counts UFP ≤ 1 μm and was programmed to log data at 5-min intervals, for 6 h, from 4 pm onwards. This time period was selected as a likely time when cooking would be undertaken, which is identified as a significant source of indoor fine and UFP emissions (Bhangar et al. 2011; Buonanno et al. 2009; Gabdrashova et al. 2021; Liang et al. 2014; Torkmahalleh et al. 2017; Wan et al. 2011).

Data on ancillary parameters such as temperature (°C) and relative humidity (%RH) were logged at 30-min intervals using the Gray Wolf AdvancedSense Pro (Advanced Sense Pro. Gray Wolf Sensing Solutions, Shelton, CT, USA).

Central haemodynamic measurements

Simultaneous to IAQ measurements, central ambulatory haemodynamic parameters were measured non-invasively using a portable monitoring device (Oscar 2, Sun Tech Medical Inc., Morrisville, NC, USA) fitted to the left arm of each participant. This instrument was pre-programmed to obtain readings at 30-min intervals for the full 24 h. Data collected included systolic (SBP) and diastolic blood pressure (DBP) (mmHg), heart rate (beats per minute, bpm), along with component haemodynamic parameters including augmentation index (%; AIx), augmented pressure (mmHg; AP), pulse pressure (mmHg; PP), and mean arterial pressure (mmHg; MAP). The mean BP and haemodynamic parameter measurements were calculated as the mean of all readings throughout the 24-h monitoring period (Andreadis et al. 2016; O'Flynn et al. 2015). Measurements were deemed as valid following standardised protocol described by Parati et al. (2014) and O'Brien et al. (2013).

Questionnaires

Each participant was provided with a questionnaire to complete during the 24-h monitoring period. Participant demographics along with information on health and lifestyle behaviour was gathered by adapted version of the American Thoracic Society standardised indoor air quality (IAQ) and health questionnaire (Ferris 1978). In the health questionnaire, participants self-reported general demographic data including age, sex, address, and other health-related information including current and history of smoking (yes, no; never, or how long ago for quitting); alcohol intake (more or less than 2 alcoholic drinks per day); and current state of health, medical diagnoses, and medications.

Using the collected demographic information, socioeconomic status (SES) was assigned using census-track data collected by the Australian Bureau of Statistics. Post codes were used to rank participant homes according to relative socio-economic advantage using the Australian Bureau of Statistic Socio-Economic Indexes for Areas (Australian Bureau of Statistics 2016).

Clinical assessment

All participants arrived at the clinical assessment facility having fasted for 12 h (other than water and regular medications). Participants were asked about demographic information, their current state of health, and recent illnesses prior to commencing the clinical assessment. Baseline measurements were then conducted following the same format for each individual. Participants' standing height and weight were measured in bare feet and wearing light clothing. Weight was measured by mechanical scale (SECA 762, SECA, Germany) and height using a stadiometre (S + M. Surgical and Medical Products, Australia). Waist and hip circumferences were measured using a non-stretch, retractable tape. Waist circumference was measured midway between the lowest rib and the iliac crest at the end of the participant's normal expiration. Body mass index (BMI) was calculated as kg/m^2 (Corlin et al. 2018).

While in a supine position, participants were instructed to rest for 10 minutes to equilibrate the examination environment. The brachial artery blood pressure was measured and entered into the SphygmoCor system (EM3XCEL, AtCor Medical Pty, West Ryde, Australia). Central arterial pressures were derived from arterial pulse waveforms obtained from the right femoral artery and concurrent direct applanation tonometry of the right common carotid artery. The device's proprietary software analysed pressure waveforms from the brachial artery and corresponding central aortic pressure was calculated using a generalised transfer function (McEniery et al. 2014). Transit distances were assessed by body surface measurements from the suprasternal notch to each pulse recording site (common carotid and femoral) (Mitchell et al. 2010). Carotid-femoral pulse wave velocity (PWV) was determined by measuring the time delay between the feet of the two waveforms arriving from the common carotid and femoral artery. All data was collected directly onto a laptop computer and processed with approved waveform analysis using a previously validated method (Butlin and Qasem 2017). Of the several methods used to quantify arterial stiffness, PWV is considered the reference

standard by the American Heart Association scientific statement (Townsend et al. 2015) and the European expert consensus document (Laurent et al. 2006).

A finger stick blood sample was collected using aseptic technique. The sample was immediately analysed for lipids (total cholesterol [TC], high density lipoprotein [HDL], low density lipoprotein [LDL], triglycerides [TG], and the cholesterol/HDL ratio) using appropriate reagent containing cassettes of the fully automated Alere biochemistry analysis system (Alere Afinion AS100, Waltham, MA, USA).

Statistical analysis

Means with standard deviations (SD) and counts with percentages were calculated for continuous and categorical variables, respectively. Normality was assessed for the outcome variables (SBP, DBP, AIx, AP, PP, PWV) using both visual inspection and Shapiro–Wilk test. Associations between indoor $PM_{2.5}$ and UFP and outcome variables were determined by Pearson correlation coefficients. Multiple regression analysis was performed to estimate the effects of $PM_{2.5}$ and UFP exposure on BP and each component of BP indicating CV function, separately.

While SphygmoCor collected additional data on component haemodynamic measures, 24-h ambulatory haemodynamic measures were preferentially used in the analysis of the data to account for unusual variations in readings which are sometimes seen with clinic measurements (e.g., 'white-coat hypertension'). Furthermore, ambulatory monitoring has been reported to be a more sensitive predictor of CV outcomes than haemodynamic measures collected during a clinic visit (Williams et al. 2018).

A staged process was used to select covariates. In the first stage, variables from a preliminary set of potential covariates identified by literature review (Baumgartner et al. 2018; Kephart et al. 2020) were included in the initial model if they were associated with the outcome ($p \leq 0.2$) in bivariate analysis (Corlin et al. 2018; Kephart et al. 2020). We also assessed the effect of adding individual variables that were identified as potentially important based on the literature but had not met the initial inclusion criteria (of $p \leq 0.2$). If these variables were not associated with the outcome and did not significantly change the effect estimates for UFP or $PM_{2.5,}$ they were excluded. Other established confounders known to be involved in the causal mechanisms of progressive CVD such as smoking, hypertension, dyslipidaemia, antihypertensive or lipid modifying medications, historical CV events, CVD and/or a medically diagnosed pre-diabetic or diabetic profile were effectively eliminated by the study design. The final model was adjusted for age, sex, SES, BMI, ratio of total cholesterol/high-density lipoprotein (TC/HDL), 24-h mean heart rate, 24-h average central MAP, 24-h mean indoor temperature, and relative humidity (RH). Further regression analysis was performed adjusting for age, SES, BMI, TC/HDL, 24-h mean heart rate, 24-h average central MAP, 24-h mean indoor temperature, and RH, with data stratified by sex to determine if effect estimates were altered.

To assist interpretability between UFP concentration and clinical outcome, UFP concentrations were scaled by 10^3 using the decimal scaling normalisation method described in other published literature (Corlin et al. 2018; Kephart et al. 2020). All statistical analyses were performed using the scaled UFP mean.

The effects of $PM_{2.5}$ and UFP exposure on the outcome variables (SBP, DBP, AIx, AP, PP, and PWV) were expressed as the mean change with 95% confidence interval (95% CI) of an outcome per increase in a pollutant interquartile range (IQR) concentration. A value of $p \leq 0.05$ was considered statistically significant. Analyses were performed using SPSS software (version 26.0, IBM Corp., Armonk, NY, USA).

Results

Characteristics of the study population

General characteristics of the study population are presented in Table 1. The age of study participants ranged from 35 to 69, and 23 (57.5%) were female. All participants were never smokers other than one 54-year-old male who had stopped smoking 19 years previously. The average age was 52.6 ± 10.9 years. Most participants fell into the healthy or overweight BMI category (85%) as defined by the World Health Organisation (WHO) (World Health Organisation 2019). Self-reported use of alcohol and medications along with the prevalence of comorbidities were low, and bivariate analyses demonstrated no meaningful associations between these covariates with $PM_{2.5}$ or UFP. The majority of participants (72.5%) lived in areas of higher socioeconomic advantage.

The mean ambulatory 24-h blood pressure readings for both systolic (118.1 ± 11.2 mmHg) and diastolic (70.0 ± 7.9 mmHg) blood pressure were below the target guidelines of 130/80 mmHg set by Australian and international committees (Chobanian et al. 2003; National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand 2012; Parati et al. 2014; Pickering et al. 2005).

Exposure characteristics

Exposure distributions are presented in Table 1. In Australia, guidelines have not been established for $PM_{2.5}$ indoors; however, the 24-h mean concentration was below the ambient air quality guideline (25 μg/m^3) and 12.5% of individual

Table 1 Characteristics of the study participants

					Percentiles		
		$N^{\dagger}$	*n* (%)	Mean (SD)	Min–max	25	50	75
Demographics								
Age [years]		40		52.6 (10.9)	35–69	42.2	54.0	64.0
Height [cm]		40		168.8 (9.3)	152.5–190.5	163.2	167.5	173.9
Weight [kg]		40		72.4 (14.0)	46–111	62.2	70.8	82.8
Female			23 (57.5)					
BMI [kg/m^2]		40		25.2 (3.4)	18.9–31.2	22.3	25.1	27.1
SES, decile		40						
	Low, 2–4		6 (15.0)					
	Medium, 5–7		5 (12.5)					
	High, 8–10		29 (72.5)					
Do you suffer from any chronic conditions? *n* (%)		40						
	None		29 (72.5)					
	Asthma, thyroid		11 (27.5)					
Medications		40						
	None		18 (45.0)					
	Vitamin supplements		12 (30.0)					
	Prescription medication		6 (15.0)					
	Combination vitamins and prescription		4 (10.0)					
Cardiovascular indicators								
Blood pressure								
	Systolic [mmHg]	32		118.1 (11.2)	91–132	105	111	117
	Diastolic [mmHg]	32		70.0 (7.9)	58–89	66	71	78
Haemodynamic indexes								
	Augmentation index [%]	38		39.3 (9.6)	17–59	32.7	38.5	46.2
	Augmented pressure [mmHg]	38		15.6 (4.0)	6–24	13.0	15.0	18.0
	Pulse pressure [mmHg]	38		38.5 (5.2)	30–54	35.0	38.0	41.2
	Mean arterial pressure [mmHg]	38		87.4 (8.6)	73–106	80.8	86.5	94.0
	Carotid-femoral PWV [m/s]	39		7.3 (1.3)	3.8–10.0	6.4	7.4	8.0
	Heart rate [bpm]	32		68.3 (8.3)	51–88	63.5	68.0	74.0
Blood lipids								
	Cholesterol [mmol/L]	40		5.1 (1.2)	2.6–7.7	4.1	5.3	5.9
	HDL [mmol/L]	40		1.5 (0.5)	0.7–2.6	1.2	1.4	2.0
	LDL [mmol/L]	40		3.1 (0.8)	1.5–5.0	2.4	3.1	3.5
	Triglycerides [mmol/L]	40		1.1 (0.7)	0.51–4.46	0.67	0.86	1.36
	Chol/HDL	40		3.6 (1.1)	2.2–7.1	2.6	3.2	4.3
Air pollution and ancillary measure exposures								
	$PM_{2.5}$ [μg/m^3], mean (SD)	25		15.7 (10.2)	3.0–34.0	7.5	12.0	23.0
	UFP [particles/cm^3], mean (SD)	40		11,256 (8744)	975–35,941	3452	9218	16,208
	Temperature [°C], mean (SD)	40		22.4 (2.8)	17.1–28.7	20.7	21.9	24.2
	Relative humidity, [%], mean (SD)	40		51.4 (7.4)	26.6–63.7	47.3	52.0	56.5

[†]Differences between the number of measurements and the number of participants is due to individual study not meeting test validity as outlined in the "Indoor air quality measurements" section or due to instrument malfunction

SD, standard deviation; *BMI*, body mass index; *SES*, socioeconomic status; *PWV*, pulse wave velocity; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein; *Chol/HDL*, cholesterol/HDL ratio; $PM_{2.5}$, particles with an aerodynamic diameter of ≤ 2.5 μm; *UFP*, ultrafine particles, particles with an aerodynamic diameter of ≤ 0.1 μm

households were exposed to a 24-h mean $PM_{2.5}$ concentration above this (National Environment Protection Council 2016). There are also no standards or guidelines for indoor concentrations of UFP; however, our recorded mean concentration is above the World Health Organisation 24-h recommended level with 47.5% individual homes breaching this guideline (World Health Organisation 2021). Forty homes were monitored for UFP concentrations; however, due to equipment malfunction, only 25 dwellings were monitored for $PM_{2.5}$.

Regression analysis

After adjustment for age, sex, SES, BMI, heart rate, MAP, cholesterol/HDL ratio, and indoor temperature and relative humidity, several statistically significant associations were observed between $PM_{2.5}$ and UFP with BP and BP component measures.

An IQR increase in $PM_{2.5}$ (15.5 µg/m^3) was associated with a 3.2 mmHg (95% CI: 0.99, 5.45) higher DBP, and lower AP (95%CI: − 3.63, − 0.01) and PWV (95%CI: − 0.80, − 0.08) of 1.8 mmHg and 0.4 m/s, respectively. For the UFP fraction, an IQR increase was associated with a 5.2% higher AIx (95%CI: 0.51, 9.97) and a 0.6 m/s lower PWV (95%CI: − 1.00, − 0.11). An IQR increase in UFP was also marginally associated with a 1.4 mmHg (95% CI: − 0.25, 3.07) higher DBP. No relationship was observed between $PM_{2.5}$ or UFP, and SBP or PP.

The estimated effects of $PM_{2.5}$ and UFP on haemodynamic parameters are shown in Table 2.

When stratified by sex, UFP concentration was associated with higher DBP (β: 1.97 mmHg; 95% CI: 0.16, 3.79) among females, but not males (β: 0.02 mmHg; 95% CI: − 9.10, 9.14). Similarly, among females, an IQR increase in UFP concentration was associated with a 0.83 m/s lower PWV (β: − 0.83 m/s; 95% CI: − 1.50, − 0.16), but not for males (β: 0.45 m/s; 95% CI: − 1.36, − 1.36). There was a borderline, apparently favourable effect on AIx (β: − 7.69%; 95% CI: − 17.71, 2.32) and AP (β: − 2.75 mmHg; 95% CI: − 5.70, 0.20) among males, but not for females.

Although we observed higher SBP, we found no statistically significant associations with $PM_{2.5}$ or UFP when stratified by sex. Other than those already reported, we found no further consistent associations for men or women. Effect estimates and 95% confidence intervals for haemodynamic parameters after exposure to UFP stratified by sex are shown in Table 3.

Table 2 Effect estimates (95% confidence interval) of $PM_{2.5}$ and UFP on haemodynamic parameters

	$PM_{2.5}$, µg/m^3 ($n = 25$)	UFP, particles/cm$^{3\Delta}$ ($n = 40$)
	β (95% CI)	β (95% CI)
SBP, mmHg	− 1.56 (− 7.33, 4,22)	− 2.83 (− 5.67, 0.02)
p-value	0.54	0.81
DBP, mmHg	3.22 (0.99, 5.45)	1.41 (− 0.25, 3.07)
p-value	0.01	0.09
AIx, %	− 0.39 (− 8.93, 8.16)	5.24 (0.51, 9.97)
p-value	0.92	0.03
AP, mmHg	− 1.82 (− 3.63, − 0.01)	1.26 (− 0.71, 3.23)
p-value	0.05	0.20
PP, mmHg	− 3.68 (− 8.74, 1.38)	− 2.38 (− 5.49, 0.74)
p-value	0.13	0.34
PWV, m/s	− 0.44 (− 0.80, − 0.08)	− 0.56 (− 1.00, − 0.11)
p-value	0.02	0.02

Model adjusted for age, sex, SES, BMI, heart rate, MAP, cholesterol/HDL ratio, and indoor temperature and relative humidity

$PM_{2.5}$, particulate matter with an aerodynamic diameter of ≤ 2.5 µm; *µg/m^3*, micrograms per cubic meter; *UFP*, ultrafine particulate matter; $^{\Delta}$, concentrations scaled by 10^3; *β*, effect estimate; *CI*, confidence interval; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *AIx*, augmentation index; *AP*, augmented pressure; *PP*, pulse pressure; *PWV*, pulse wave velocity

Table 3 Effect estimates (95% confidence interval) of UFP on haemodynamic parameters stratified by sex

		UFP, particles/cm$^{3\Delta}$	
		β (95% CI)	*p-value*
SBP, mmHg	Male	− 0.85 (− 14.68, 12.97)	0.86
	Female	− 1.44 (− 4.69, 1,81)	0.33
DBP, mmHg	Male	0.02 (− 9.10, 9.14)	0.99
	Female	1.97 (0.16, 3.79)	0.04
AIx, %	Male	− 7.69 (− 17.71, 2.32)	0.09
	Female	3.24 (− 2.13, 8.61)	0.20
AP, mmHg	Male	− 2.75 (-5.70, 0.20)	0.06
	Female	0.42 (− 2.42, 3.27)	0.74
PP, mmHg	Male	− 1.65 (− 15.91, 12.61)	0.74
	Female	− 2.12 (− 5.54, 1.29)	0.19
PWV, m/s	Male	0.45 (− 1.36, − 1.36)	0.49
	Female	− 0.83 (− 1.50, − 0.16)	0.02

$n = 40$

Model adjusted for age, SES, BMI, heart rate, MAP, cholesterol/HDL ratio, and indoor temperature and relative humidity

UFP, ultrafine particulate matter; $^{\Delta}$, concentrations scaled by 10^3; *β*, effect estimate; *CI*, confidence interval; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *AIx*, augmentation index; *AP*, augmented pressure; *PP*, pulse pressure; *PWV*, pulse wave velocity

Discussion

This study investigated the associations between indoor domestic exposure to $PM_{2.5}$ and UFP, on BP and components of BP which quantify CV function involved in the development and progression of CVD. In a cohort of middle-aged adults, without clinical CVD, we found that exposure to indoor residential $PM_{2.5}$ and UFP is associated with

BP and some functional component measures of BP. Furthermore, we observed that some of these estimated effects were modified by sex.

In this current study, an IQR increase in domestic indoor $PM_{2.5}$ and UFP was associated with higher DBP; however, no effect was seen on SBP. Furthermore, increases in UFP exposure resulted in an estimated effect that was stronger and significant among women, but not for men. This finding agrees with the results of other similar studies. Lin et al. reported on associations between increased UFP with higher SBP and DBP which were stronger in women than in men (Lin et al. 2021). Corlin et al. found no association in adjusted models per IQR increase in UFP with SBP or DBP; however, it was concluded there was generally stronger positive, adverse associations among women. Among men, these authors also reported inverse or apparently favourable associations between UFP concentrations with SBP and PP (Corlin et al. 2018).

Conceivable explanations of sex differences may originate from sex-linked biological factors such as lung volume resulting in differential deposition and reactivity, and the transport of chemical agents (dependent on the toxicological profile of the PM) and systemic regulation influenced by hormones (Clougherty 2010; Lin et al. 2021).

Consistent with the current study, Brook and colleagues in a controlled human exposure study found no relationship between indoor concentrations of $PM_{2.5}$ with SBP and provided no explanation as to why these exposures elicited different and/or a greater effect on DBP compared to SBP. Brook et al. also demonstrated that $PM_{2.5}$ was capable of transiently raising DBP, however, only during the period of actual inhalation and concluded that perhaps the underlying haemodynamic changes responsible for the changes reflected a predominant vasoconstriction without changes in cardiac output or arterial compliance (Brook et al. 2009). In another recent controlled exposure study and in contrast to the current study, short-term exposure to $PM_{2.5}$ from different types of cookstoves was also observed to provoke transient alterations to SBP; however, no relationship was shown with DBP (Fedak et al. 2019; Walker et al. 2020). Choi and colleagues examined the hourly relationship between ambient $PM_{2.5}$ and BP and found no relationship between SBP and short-term exposure to $PM_{2.5}$ in 98,577 Korean adults (Choi et al. 2019). However, in a more recent European study of 132 healthy adults from Switzerland, the Netherlands, and Italy, 24-h measured residential UFP exposure was not associated with BP; however, an IQR increase in $PM_{2.5}$ was positively and unfavourably associated with a 1.4 mmHg rise in SBP (van Nunen et al. 2021). Similarly, in the longitudinal Boston Puerto Rican Health Study (USA), exposure to UFP was not associated with either SBP or DBP in 791 adults, while favourable associations were shown between SBP and PP with UFP among men (Corlin et al. 2018). Although the precise pathophysiologic mechanisms through which $PM_{2.5}$ and UFP exposure could result in biological changes that affect diastolic pressure more than systolic pressure remain speculative, some mechanisms are provided in the literature. For example, if exposure to air pollution causes increases in arterial stiffness as seen in Baumgartner and colleagues' work (Baumgartner et al. 2018), changes are more likely to be seen in systolic pressure (Fedak et al. 2019). More research is required, however, to clarify these pathways.

In the current study, although a significant relationship was not established between either size fraction of PM with PP, we did observe lower PP in the overall population and the same as Corlin et al. (2018), among males. Similar results were also demonstrated in a Taiwanese study by Chen and colleagues ($n = 9238$ non-smoking adults) with these authors concluding that PM_{10} lowers PP (Chen et al. 2012).

We observed mixed results between both PM size fractions with AIx and AP, although a consistent inversed and favourable association was shown with PWV. While these findings are in agreement with some other studies, the literature provides mixed results (Baumgartner et al. 2018; Brook and Rajagopalan 2009; Choi et al. 2019; Liang et al. 2014; van Nunen et al. 2021).

Baumgartner et al. (2018) found that increased $PM_{2.5}$ exposure was associated with no difference in PWV (−0.1 m/s; 95% CI: −0.4, 0.2) in 205 Chinese women (age range: 27–86 years), although consistent with the current study ($PM_{2.5}$: −0.44 m/s; 95% CI: −0.80, −0.08, UFP: −0.56, 95% CI: −1.00, −0.11), a relationship showing decreases in PWV was shown. In contrast, in our overall population, we observed a non-significant favourable relationship between $PM_{2.5}$ and AIx compared to Baumgartner et al., who found a modestly higher AIx (1.1%; 95% CI: −0.2%, 2.4%) among women, with increased concentrations of $PM_{2.5}$. When we stratified our analyses by sex, we also observed a moderately higher AIx in women, although the CI included zero (3.2%; 95% CI: −2.13, 8.61). These differences in direction and estimated effects between the two studies may potentially be explained by the considerably higher $PM_{2.5}$ concentrations (48-h averaging period; summer mean: 101.3 μg/m^3; winter mean: 218.5 μg/m^3) compared to the present study (24-h mean: 15.7 μg/m^3) and/or differences in study methodologies.

Complex physiological responses are involved in alterations to BP and component measures that are affected by vascular resistance and cardiac output (a product of stroke volume and heart rate). Evidence suggests PM may increase the resistance and decrease the compliance of vasculature via conceivable biological mechanisms involving endothelial dysfunction and vasoconstriction (Brook et al. 2009). Systemic inflammatory responses and oxidative stress leading to autonomic dysfunction are also thought to play a major role in the impact of PM exposure on CV health

(Bae et al. 2021; Bourdrel 2021; Kephart et al. 2020). Human and animal models have provided mechanistic evidence, whereby possible pathways are shown by which acute and chronic exposures to PM might disrupt haemodynamic balance favouring vasoconstriction, including autonomic imbalance and amplified release of various pro-oxidative, inflammatory, and/or haemodynamically functioning mediators (Bae et al. 2021; Brook et al. 2010; Giorgini et al. 2016). Additionally, it is thought some pro-oxidative elements of inhaled particles (e.g., nanoparticles, metal, organic compounds) are capable of translocating directly into the systemic circulation to facilitate direct adverse actions including systemic inflammatory responses (Brook et al. 2009). Together, these responses likely elicit the changes to autonomic vascular tone including arterial vasoconstriction and endothelial dysfunction, which are ultimately responsible for the rapid alterations in BP (and its components) reported to occur after PM exposure (Al-Kindi et al. 2020; Brook et al. 2010). Although it was beyond the scope of this current study to time-resolve exposure with response, the greater evidence supports rapid effects of particulate pollutants including fine and UFP (Baumgartner et al. 2018; Brook et al. 2010, 2009; Münzel et al. 2018), suggesting that the effects observed in our study may be the result of short-term or recent exposure. Support for this is garnered from controlled human exposure studies. Soppa and colleagues, investigating the impact of fine and UFP exposures on haemodynamic parameters in healthy adults, found that exposure to PM resulted in immediate and mostly transient effects on some indices (AIx and AP but not PWV) (Soppa et al. 2019). Furthermore, Brook et al. demonstrated that endothelial function can remain compromised for 24 h after exposure to concentrated ambient PM (Brook et al. 2009). In their review considering CV effects of particulate air pollution, Langrish et al. also linked short-term air pollution to acute and rapid effects on the arterial system, BP, and other functional CV endpoints (Langrish et al. 2012).

Lastly, legislation relating to air quality in high-income countries is traditionally based upon ambient outdoor concentrations which potentially leads to insufficient protection for individuals who spend most of their time indoors where concentrations of PM can be much higher than outdoor levels (Abdullahi et al. 2013).

Strengths and limitations

This study has several notable strengths above the contribution it adds to the currently limited body of evidence related to the impact of residential air pollution exposure in high-income countries on functional intermediate measures of CV risk.

Firstly, and importantly, this study benefitted from a relatively homogenous sample of apparently healthy, well-characterised, middle-aged adults. Households were also located in a geographical area where outdoor air pollution (contributing to total exposure) concentrations are relatively consistent and typically below accepted air quality standards.

Another strength of this study is that all clinical assessment and indoor environmental data were directly measured at an individual level. We did not rely on self-report or concentration estimates derived from modelling ambient air pollution potentially reducing the opportunity for introduced bias related to exposure and outcome misclassification. Additionally, this study relied on central ambulatory BP measures. Emerging data supports the superiority of ambulatory monitoring in comparison with repeated or one-off clinic-based measurements (Wilkinson et al. 2014).

We also recognise that this study has several limitations. While the findings of this research provide plausible evidence to support that exposure to present day, low-level concentrations of residential indoor $PM_{2.5}$ and UFP such as that encountered during typical daily activity might be capable of provoking adverse pathophysiological reactions known to promote CV events, this has only been shown in a small sample of apparently healthy, non-smoking adults. Additionally, the cross-sectional design restricts the establishment of a temporal relationship and provides no indication of the sequence of events. The observed impacts on outcomes at one point in time may have occurred before the onset of a pollution-mediated response. It is therefore not possible to evaluate the potential for causality in any of the reported associations. Moreover, our cross-sectional approach is sensitive to confounding from individual factors, and residual confounding by other unmeasured factors, such as diet and exercise levels, may have occurred. While most important variables were included in adjusted models, unknown and residual confounding cannot be eliminated as explanation for the observed associations.

For studies of IAQ, home measurements are a common method for estimating exposure (Ferguson et al. 2020). Although there are known issues with monitoring devices in the home (incorrect set-up, tampering, altered participants behaviours, etc.), it is assumed that the extent of uncertainty in the method of IAQ assessment is confined to the reliability of the measuring equipment. Direct measurements offer protection against issues associated with self-reported data; however, impacts on exposure and/or outcome data can also arise due to the way participants interact with monitoring equipment. For example, although smokers were excluded from this current study, in literature reporting on parental-reported smoking prevalence in the home, due to the social stigma attached to in-home smoking, particularly where children are present, parents may not be forthcoming in revealing their smoking habits. This effect was demonstrated in a

study by Jurado et al. (2004) who reported the presence of cotinine, the predominant nicotine metabolite, in the urine of 14% of children whose parents identified as a non-smoker. In the context of the current study, effect estimates may have been influenced by modified participant behaviours deriving from understood knowledge of indoor air pollutant sources and/or haemodynamic measures.

Similarly, in-home exposures were collected rather than personal exposures. While using residential pollutant concentrations to characterise an individual's exposure has benefits over ambient estimates, personal exposure assessments combined with time activity measures are the ideal method to accurately classify an individual's true exposure. Future studies may benefit from comprehensive qualitative inputs comprehensively outlining participant behaviours over the monitoring period.

Finally, due to instrumental limitations, UFP concentrations could only be measured over a 6-h period. The timing of the monitoring of UFP concentrations in this study coincided with the traditional evening cooking period and therefore likely included peak levels of UFP concentrations and therefore the highest exposure periods. Cooking is a well-established source of UFP (Gabdrashova et al. 2021; Wan et al. 2011) and the findings of this study could well be presenting the 'worst case scenario'. Additionally, instrument malfunction resulted in only 25 measurements being obtained for $PM_{2.5}$, which might have influenced some of the outcomes observed in this study. To make meaningful statements about indoor exposure across a population, considerable sample sizes are required to ensure the sample population accurately reflects the population of interest (Ferguson et al. 2020). To reduce potential experimental bias effects of small sample sizes, larger studies are recommended. While monitoring studies such as the current study provide invaluable empirical evidence of exposure, they require significant resources, are time consuming, and can be cost prohibitive. Other study designs such as those using modelling strategies have the advantage of being able to examine larger numbers of different scenarios and isolate specific factors that contribute to exposure and subsequent outcomes. However, they also come with large modelling uncertainties. While the reported results are an important contribution to this understudied area of research, further studies are recommended to corroborate the findings of this current work, using study designs that capture measurements representative of actual exposure and with larger sample sizes.

Finally, it is generally considered that PM air pollution plays an important role in contributing to sub-optimal BP and component outcomes; the evidence available, however, is inconsistent. The specific reasons for these inconsistencies between studies of PM air pollution exposure and associated health outcomes is not clear; however, it might be partly explained by differences in study methodology including study design and sample size, regional characteristics, population characteristics, averaging times for exposure, and the selection of monitoring instrumentation. Although infrequently acknowledged, these various disparities and lack of standardisation in experimental conditions are common limitations and hamper comparability of reported finding between studies.

Conclusion

In this study, we identified adverse associations between domestic exposures to indoor $PM_{2.5}$ and UFP and some components of BP.

These findings are important and add to the evidence that exposures to domestic low-level concentrations encountered during routine daily exposure indoors may have a measurable impact on CV physiology and may be involved in the promotion and pathogenesis of CVD. Furthermore, these responses could also conceivably occur in an augmented manner where there are pre-existing CV risk factors or conditions (such as dyslipidaemia, diabetes) that adversely affect the ability to offset against established physiological dysfunction such as autonomic system imbalances, reduced arterial compliance, or alterations to vascular tone due to air pollution–mediated inflammation (Clark et al. 2019; Corlin et al. 2018; Huynh et al. 2020; Rajagopalan et al. 2018; van Nunen et al. 2021; Zanoli et al. 2017).

With such large periods of daily time known to be spent in indoor domestic environments, understanding this association will assist with informing policy considerations and mitigation efforts aimed at reducing domestic indoor air pollution levels and the impact of exposure to these pollutants to CV health. Furthermore, it may also inform preventative recommendations as part of risk profiles for susceptible individuals.

Currently the body of evidence is insufficient and data supporting a progression of the markers studied with PM (and other pollutants) is limited. While the sample size of the study is relatively small, the findings are an important contribution to this expanding area of research on indoor air quality and its relationship with CV health. Further studies incorporating longitudinal designs and/or larger-scale controlled clinical outcome trials are required to corroborate our findings and expand the current levels of understanding.

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Author contribution SEG, CMR, YZ, MJS, RRH, and KBR contributed to the study conception and design. Material production and data collection were performed by SEG. SEG and YZ did the statistical analysis. The first draft of the manuscript was written by SEG. SEG, CMR, YZ, MJS, RRH, and KBR read and approved the final manuscript.

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Data availability The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Human Research Ethics Committee of Curtin University (HRE2016-0308).

Consent to participate All participants declared their written consent to participate in this study.

Conflict of interest The authors declare no competing interests.

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