Article:
Argamino, Cristian Ryan A., Silva, Jéssica dos Santos, Kizhakkethil, Jishnu Pandamkulangara et al. (2026) Assessment of the occurrence of phthalate esters (PAEs) in indoor office PM2.5 from Brazil and the UK. Atmospheric Pollution Research. 102987. ISSN: 1309-1042

https://doi.org/10.1016/j.apr.2026.102987

Atmospheric Pollution Research xxx (xxxx) xxx

Contents lists available at ScienceDirect

Atmospheric Pollution Research

journal homepage: www.elsevier.com/locate/apr

Assessment of the occurrence of phthalate esters (PAEs) in indoor office $PM_{2.5}$ from Brazil and the UK

Cristian Ryan A. Argamino [a,b], Jéssica dos Santos Silva [c], Jishnu Pandamkulangara Kizhakkethil [a], Svetlana Stevanovic [b], Martyn Ward [d], Matthew McKenzie [e,f], Anna Bogush [a], Jacqueline F. Hamilton [d,g], Ricardo H.M. Godoi [c], Ivan Kourtchev [a,*]

[a] Centre for Agroecology, Water and Resilience (CAWR), Coventry University, Wolston Lane, Ryton-on-Dunsmore, CV8 3LG, UK
[b] School of Engineering, Deakin University, 75 Pigdons Road, Waurn Ponds, VIC, 3220, Australia
[c] Environmental Engineering Department, Federal University of Parana (UFPR), Curitiba, PR, Brazil
[d] Wolfson Atmospheric Chemistry Laboratories, Department of Chemistry, University of York, York, YO10 5DD, UK
[e] School of Life and Environmental Sciences, Deakin University, 75 Pigdons Road, Waurn Ponds, VIC, 3216, Australia
[f] Institute for Physical Activity and Nutrition, Deakin University, 75 Pigdons Road, Waurn Ponds, VIC, 3216, Australia
[g] National Centre for Atmospheric Science, University of York, York, YO10 5DD, UK

ARTICLE INFO

Keywords:
Indoor air quality
Aerosol
Plasticiser
Inhalation risk

ABSTRACT

Phthalate esters (PAEs) are ubiquitous synthetic chemicals, known pollutants and health hazards. Due to their adverse health effects, including those associated with inhalation exposure, PAEs have been relatively well studied in outdoor particulate matter with aerodynamic diameter <2.5 μm ($PM_{2.5}$). However, limited data exist on indoor emissions of PAEs, despite the fact that nearly 90 % of human daily activities occur indoors, indicating a potential gap in understanding human exposure to those pollutants. This pilot study aimed to perform a preliminary assessment on the presence of six priority PAEs and the alternative plasticiser bis-(2-ethylhexyl) adipate (DEHA) in indoor $PM_{2.5}$ from three offices in the UK and Brazil. DEHP (bis-(2-ethylhexyl) phthalate), was consistently detected in $PM_{2.5}$ (with concentrations up to 34 ng/m^3) from all studied offices. DEHP concentrations had consistently low airborne variability, especially within the occupied offices, suggesting that human activities could facilitate the continuous resuspension of DEHP-bound particles, maintaining a steady-state indoor concentration. Although mean DEHP levels in all studied offices were below the UK HSE workplace exposure limit, they represent a continuous chronic exposure source not accounted for by industrial safety standards. Reliance on oral reference values due to unavailability of inhalation reference standards for PAEs may underestimate risks due to route-specific metabolic differences. The study provides the first region specific data from the UK and Brazil suggesting indoor environments as contributors to $PM_{2.5}$-bound DEHP exposure, highlighting the need for more systematic and long-term monitoring initiatives.

1. Introduction

Air pollution has been associated with millions of premature deaths annually (WHO, 2024a). As humans spend approximately 90% of the time indoors, the assessment of indoor air quality plays a significant role in pollutant exposure. Indoor environments such as homes, schools, and offices can be significant sources of airborne pollutants, including particulate matter (PM) (Chatoutsidou et al., 2015).

Fine particulate matter ($PM_{2.5}$; aerodynamic diameter ≤2.5 μm) is a hazardous component of both indoor and outdoor air. $PM_{2.5}$ can penetrate deep into the lungs and translocate into the blood, inducing multisystem toxicity (WHO, 2021). $PM_{2.5}$ can be a vector for regulated persistent organic pollutants (POPs) and new and emerging pollutants (NEPs) (Barroso et al., 2019). Recent studies have reported that POPs and NEPs, such as phthalate esters, tend to mainly exist smaller airborne particles (e.g., $PM_{2.5}$) (Bahrami et al., 2025; Li and Wang, 2015), highlighting the importance of investigating these pollutants in fine particles, particularly within indoor environments.

* Corresponding author.
E-mail address: ivan.kourtchev@coventry.ac.uk (I. Kourtchev).

https://doi.org/10.1016/j.apr.2026.102987
Received 5 January 2026; Received in revised form 13 March 2026; Accepted 14 March 2026
Available online 15 March 2026

Phthalate esters (PAEs) are ubiquitous synthetic semi-volatile organic chemicals, classified as either POPs or NEPs. PAEs are widely used as plasticisers in construction materials (Abdi et al., 2021), furnishings (e.g. carpets, wallpaper) (Shi et al., 2018), and as stabilisers in personal care products (US EPA, 2000). Because PAEs are not covalently bound to the polymers, they can gradually migrate out of these materials and partition into indoor air (ECHA, 2025; Jamarani et al., 2018; Wang and Kannan, 2023).

Notably, six PAE compounds listed as priority pollutants by some regulatory agencies, e.g., the U.S. EPA, due to their reported toxicity: dimethyl phthalate (DMP), diethyl phthalate (DEP), di-*n*-butyl phthalate (DBP), benzyl butyl phthalate (BBP), bis-(2-ethylhexyl) phthalate (DEHP), and di-*n*-octyl phthalate (DOP) (US EPA, 2014, 2025b).

Emerging studies on prolonged exposure to PAEs (even at trace concentrations) have highlighted toxicological concerns due to their endocrine-disrupting properties and associations with adverse reproductive, developmental, and metabolic health outcomes (Argamino et al., 2025; Becker et al., 2004; Caldwell, 2012; Chang et al., 2021; Mariana et al., 2016). This growing evidence of toxicity emphasises the need to monitor PAEs in the environment, particularly in indoor settings, where exposure could be significant.

While there have been numerous reports on PAEs bound to outdoor $PM_{2.5}$ across several regions (e.g., Brazil, China, Czech Republic, Hong Kong, France, Puerto Rico) (Argamino et al., 2024; El Haddad et al., 2011; Feng et al., 2005; Kashyap and Agarwal, 2018; Long et al., 2025; Růžičková et al., 2016; Toro-Heredia et al., 2021; Zheng et al., 2000), data on indoor PM-bound PAEs remain limited. Existing studies on the indoor $PM_{2.5}$ fraction are geographically concentrated in Asia, particularly in China, with one study in Norway (Chen et al., 2018; Huang et al., 2023; Rakkestad et al., 2007; Wang et al., 2018, 2019; Zhang et al., 2014) (Table S1). For instance, concentrations of indoor $PM_{2.5}$-bound PAEs in Chinese residences and dormitories in Tianjin, Beijing, and Guangzhou, have been reported reaching up to 1 μg/m^3. To the best of our knowledge, airborne PAE data are unavailable for the UK and limited to total suspended particles (TSP) in Brazil (dos Santos et al., 2024), with no measurements of the inhalable $PM_{2.5}$ fraction.

The health effect of exposure to airborne pollutants is generally assessed using risk assessment approaches. Currently, risk assessments on $PM_{2.5}$-bound PAEs are typically limited to daily intakes or average daily doses, often emitting the evaluation of non-cancer and cancer risks in other monitoring studies (Hong et al., 2024; Huang et al., 2022; Li et al., 2019; Ma et al., 2014; Quintana-Belmares et al., 2018). Moreover, studies assessing the risks of PAEs exposure in $PM_{2.5}$ typically rely on the oral reference dose (RfD) due to the absence of published inhalation reference concentrations (RfC). For example, Zhang et al. (2019) used the oral RfDs for the assessment inhalation risk (e.g., hazard quotient (HQ)) of $PM_{2.5}$-bound PAEs in four metropolitan cities in China. This may lead to underestimated risks since pollutants (i.e., PAEs) could be metabolised differently via the ingestion route compared to the inhalation route (US EPA, 2024a). Considering current evidence, a critical knowledge gap exists regarding the role of $PM_{2.5}$-bound PAEs in indoor environments specifically regarding geographical representation beyond heavily industrial regions (e.g., China). Limited information on these airborne indoor pollutants restricts accurate exposure assessment and understanding of the potential health risks. For locations with no existing airborne PAE data, pilot studies are essential to establish baseline concentrations and to guide decisions on subsequent, larger monitoring efforts. While recent research indicates that indoor environments may contain a broader suite of PAEs (up to 10 PAEs and 14 alternative plasticisers) (Chen et al., 2025), this pilot study focuses on six PAEs designated as priority pollutants by the U.S. EPA (DMP, DEP, DBP, BBP, DEHP, and DOP) and the alternative plasticiser bis-(2-ethylhexyl) adipate (DEHA). These compounds were selected due to their high regulatory priority and known or suspected health risks, serving as a baseline for new measurements in previously unmonitored sampling sites. Consequently, this work aims to conduct a pilot study on $PM_{2.5}$-bound PAE pollution in indoor office settings at two locations (i.e., the UK and Brazil) with no prior measurement records, to establish preliminary data for future full-scale monitoring campaigns.

2. Methodology

2.1. Site description

Indoor $PM_{2.5}$ samples were collected from three offices labelled as Office 1, 2, and 3.

Office 1 (Coventry, UK; 362 m^3, unused/unoccupied) is a newly painted office and was not in use by personnel during the sampling period. Office 1 was equipped with an Air Handling Unit (AHU) that supplies and extracts fresh air and featured four fan coil units that provide recirculated air with heating and cooling capabilities. Additionally, the system included automatic windows that were controlled based on CO_2, external air temperature, and an internal free cooling set point. The Office 1 floor was fully carpeted (no details on age of carpet, which was not replaced during refurbishment). During sampling, the room had old computers (with insulated cables) and old office furniture (i.e., office chairs and desks) with plastic-made parts. Since office renovation was on-going, waste materials were in the office during the sampling period.

Office 2 (Coventry, UK; 36 m^3, used/occupied) was refurbished one year prior to sampling (e.g., new carpet, plasterboards, paint, office furniture) and was in use on weekdays. The office has a peak capacity of four occupants, with an observed average occupancy of two. Office 2 had limited ventilation through a small window near the ceiling of the room, which opened automatically depending on the set room temperature (e.g., 20 °C) and thus potentially diluting air concentrations of PAEs. The floor in Office 2 was fully carpeted, and the walls were covered with paint. The room had office furniture (i.e., office chairs, desks, cupboards) which had paint or coating, or were made of plastic (e.g. polyvinyl chloride (PVC)).

Office 3 (Rio Branco do Sul, Brazil; 33 m^3, used, occupied) was last refurbished before 2020 (specific year unknown) and was in use during the sampling period. The office has a peak capacity of ten occupants, with an observed average occupancy of three. There were no windows in Office 3 but the glass sliding door that was left open (to the corridor) during the sampling period. The office had ceramic tiles on the floor and walls were covered with paint. The room had office furniture (i.e., office chairs, desks, cupboards) which had paint or coating and parts (e.g., cupboard panels, chair backrest) made of plastic (e.g., polyvinyl chloride (PVC)).

A summary of the characteristics of the indoor sampling sites is provided in Table 1.

Table 1
Characteristics of indoor sampling sites.

Sampling Site	Size	Occupancy	Ventilation	Indoor Materials
Office 1 (Coventry, UK)	362 m^3	unused/ unoccupied	air handling unit; automatic windows	floor carpet (old), old computers with insulated cables, old office chairs and desks (with plastic parts)
Office 2 (Coventry, UK)	36 m^3	used/occupied (average occupancy: 2)	limited ventilation via small window	new floor carpet, plasterboards, paint; office furniture with plastic parts
Office 3 (Rio Branco do Sul, Brazil)	33 m^3	used/occupied (average occupancy: 4 occupants)	no windows; glass sliding door left open (leading to corridor)	ceramic floor tiles, painted walls, office furniture with plastic parts

2.2. Sample collection

Office 1 and Office 2 samples were collected on glass fibre filters (GFF) (Advantec, ⌀ = 47 mm) using MiniVol portable air sampler (Airmetrics, USA) placed ~ 1 m above floor level and operated at a rate of 10 L/min. GFFs were pre-baked in a furnace at 450 °C for 24 h before sample collection to remove potential organic contamination. For Office 1, four $PM_{2.5}$ samples were collected on glass fibre filters (GFF) for 24 h during weekdays and one sample was collected over the weekend (60 h) in November 2023. Office 2 was in use on weekdays from 9:00 a.m. to 5:00 p.m. Four $PM_{2.5}$ samples were collected on GFF for 16 h during weekdays (overnight), and one sample for 60 h over the weekend (Friday evening to Monday morning). After sampling, GFFs were stored in pre-washed 10 mL glass vials (Chromacol 10-HSV, P/N 03–341–956) with metal screw caps (Chromacol 18-MSC, P/N 03–341–957) and polytetrafluoroethylene (PTFE) septa (Chromacol 18-ST101, P/N 03–341–959) from Thermo Scientific (Waltham, MA).

Office 3 (indoor $PM_{2.5}$) samples were collected on quartz fibre filters (QFF) (PALL Life Sciences, Pallflex®, Tissuquartz, ⌀ = 37 mm) using a Harvard impactor placed ~ 1 m above floor level and operated at a rate of 10 L/min. QFFs were pre-baked in a furnace at 650 °C for 24 h before sample collection to remove potential organic contamination. After sampling, QFFs were folded in half, wrapped into aluminium foils, and stored in a freezer until shipment for analysis.

For all sampling sites, at least two field blanks were prepared by placing pre-baked filters into the sampler filter holder and drawing air for 2 min. Sampling information are presented in the Supplementary Information (Tables S2 and S3).

2.3. Sample extraction

Briefly, PM filter samples were spiked with 60 pg/μL DEHP-d4 and then extracted using ultrasonic agitation (2 × 20 min) in a 50:50 volumetric ratio of high purity (≥99.5%) dichloromethane (DCM): LC/MS grade methanol. The sample extracts were filtered using a pre-washed 0.45 mm PTFE syringe filter into a new pre-washed vial. Filter samples underwent a second extraction step by repeating the described steps above. Filtered sample extracts from the two extraction steps were combined and evaporated to dryness using ultrapure N_2. Dried extracts were resuspended in 200 μL 50:50 DCM: methanol, vortex-mixed and transferred into a 2 mL vial for GC-HRMS analysis.

2.4. Analysis of PAEs

The analysis of six PAEs and DEHA was performed using two gas chromatography–high resolution mass spectrometry (GC HRMS) methods (described in Sections 2.4.1 and 2.4.2) for the Brazilian and UK $PM_{2.5}$ sample sets, as instrument access was limited and each system required specific method adjustments due to hardware differences. Both methods were evaluated for performance and comparability, and the assessment criteria and results are presented in Section 2.5.2.

2.4.1. Analysis of Brazilian indoor samples (GC-Orbitrap-MS)

Determination of PAEs and DEHA in $PM_{2.5}$ from Office 3 (indoor) was performed using a previously developed and validated GC-Orbitrap-MS method (Argamino et al., 2024). A TRACE™ 1310 GC coupled to a Q Exactive™ Focus Hybrid Quadrupole-Orbitrap™ (GC QE Orbitrap) MS (Thermo Fisher, Bremen, Germany) was equipped with single taper ultra-inert liner with wool (4 × 6.5 × 78.5 mm) (Thermo Scientific, Cat No. 453A1925-UI) and a TG-5SilMS GC column (5% phenyl, 95% dimethylpolysiloxane; 30 m × 0.25 mm × 0.25 mm) (Thermo Scientific, P/N 26096-1420). Sample introduction involved a 1 μL injection volume into a GC split/splitless (SSL) inlet, operating in splitless mode to enhance detection sensitivity. The inlet temperature was set to 300 °C. Selected ion monitoring (SIM) mode with an optimised quadrupole isolation window setting (8.0 Da) was employed to improve detection sensitivity (Argamino et al., 2024). The GC oven temperature was initially set at 84 °C and held for 5 min before increasing to 180 °C at 25 °C/min. The temperature was further increased to 280 °C, at 5 °C/min, and held for 3 min. Lastly, the oven temperature was increased up to 300 °C at 10 °C/min and held for 3 min. The total method duration was set to 36.6 min. Auxiliary temperatures for transfer lines 1 and 2 were set to 280 °C. The total method duration was set to 36.6 min. The MS transfer line and the electron impact (EI) ion source temperatures were set to 300 °C. EI was performed at 70 eV energy, and filament delay was set to 2.8 min. MS resolution was set to 60000, and the mass range was at m/z 50–550. Selected ion monitoring (SIM) scan mode was employed. The automatic gain control (AGC) target was set to 1×10^6. Maximum injection time (IT) was set to auto, and spectrum multiplexing (MSX) count was set to 10. Isolation window (IW) was set to 8.0 Da. Data acquisition and processing were performed using the Thermo Scientific Xcalibur software (version 4.5).

2.4.2. Analysis of UK indoor samples (GC-QTOF-MS)

$PM_{2.5}$ samples collected from Office 1 and Office 2 in the UK were analysed using a GC-QTOF-MS (Agilent GC-QTOF-MS 7200, Santa Clara, California, United States). For the GC-QTOF-MS analysis, certain GC parameters (i.e., inlet temperature, injection volume, injection mode) and consumables (i.e., type of inlet liner and capillary column) were adapted from the GC-Orbitrap-MS method described in Section 2.4.1. (Argamino et al., 2024). Specifically, ultra-inert liners were installed (details in Section S1), and a 1 μL sample was introduced into a GC split/splitless (SSL) inlet, operating in splitless mode, with the inlet temperature set to 300 °C. Notably, the oven temperature program (Section S1) for the GC-QTOF-MS was adjusted for a shorter run time (approx. 19.8 min) compared to the GC-Orbitrap-MS (36.6 min) due to limited access to the GC-QTOF facility. The GC oven temperature was initially set at 80 °C and held for 3 min. The temperature was increased to 275 °C at 25 °C/min and held for 3 min. Lastly, the oven temperature was further increased to 320 °C at 15 °C/min, and held for 3 min. For the MS, the ion source (EI) temperature was set at 230 °C, and the quadrupole temperature was set at 150 °C. The auxiliary temperature for the transfer lines was set to 330 °C. Data acquisition and processing were performed using the Agilent MassHunter software (version B.07.01).

For the GC-QTOF-MS, this adjustment resulted in compromised detection of the DMP, DEP, DBP, BBP, DEHA, and DOP; therefore, the emphasis of this work was placed on DEHP that had reliable retention and peak shape characteristics as discussed in Section 2.5. Full scan (FS) mode was employed for the GC-QTOF-MS, utilising the uniform electromagnetic force applied to all ions accelerating simultaneously in a TOF mass analyser (Agilent Technologies, 2025; Santacruz et al., 2007). Quantifying MS ions and detailed data acquisition and processing information for are presented in Section S1.

2.4.3. Blank correction

Field blanks from each sampling site were extracted and analysed as described in Section 2.3. Blank-correction was performed by subtracting the detected PAE concentration values in the field blanks from the PM samples. Additional quality assurance and quality control (QA/QC) measures are described in Section 2.5.

2.5. Quality control and quality assurance

2.5.1. Sample collection and preparation

The use of plastic-made materials was avoided to reduce PAEs contamination. Equipment used for sample collection (i.e., metal forceps) were wiped with methanol before use. Prior to sampling, the filters were baked (to remove organic contaminations) and then were individually wrapped in aluminium foil and stored in a sealed desiccator before transportation to the field. After sampling, samples were wrapped into a prebaked aluminium foil and stored in a freezer (−20 °C) until analysis. High purity reagents (>99%) were used in the sample

preparation and glassware were washed thrice with 50:50 DCM:MeOH prior to use.

2.5.2. Addressing potential biases caused by the use of different samplers, substrates, and analytical instrumentation

Potential biases in PAE analysis can be observed when using different sampling techniques, sampling substrates, and analytical instrumentation. To mitigate sampling biases, particularly those related to the gas-particle equilibrium of organic pollutants on filter substrates (Turpin et al., 2000), similar protocols were applied to both the Harvard impactor (quartz fiber filters, QFFs) and MiniVol sampler (glass fiber filters, GFFs) used for $PM_{2.5}$ collection. For instance, both QFFs and GFFs were pre-baked at 450 °C, and both samplers were equipped with $PM_{2.5}$ impactors operating at a consistent flow rate of 10 L/min. To further account for potential contamination, field blanks were collected. The background PAE concentrations in procedural blanks were then subtracted from sample values (e.g., mean DEHP concentration in blanks was 43 pg/μL, which is above the limit of quantitation (LOQ)).

Another potential consideration is the use of different types of samplers to collect PAEs in our study. Although, to the best of our knowledge, there is no information on sampling artefacts for PAEs associated with the specific samplers used in this study, Yanosky and Maclntosh (2001) reported that the Harvard Impactor produced results for $PM_{2.5}$ (mass) comparable to a reference sampler (i.e., the PQ200 Federal Reference Method $PM_{2.5}$ sampler) with no systematic bias. In contrast, the use of the MiniVol sampler led to slight underestimated $PM_{2.5}$ concentrations as sample mass increased. This sampling bias in the MiniVol can be addressed by employing field blank corrections (Hill et al., 1999). Further comparisons by Baldauf et al. (2001) indicated that the MiniVol can produce statistically comparable results for $PM_{2.5}$ mass with a Versatile Air Pollutant Sampler (VAPS). Baldauf et al. (2001) noted that the comparability of measurements was influenced by environmental factors, including ambient concentration, wind speed, temperature, and humidity. Baldauf et al. (2001) also observed passive collection of airborne PM at the MiniVol inlet during non-sampling periods, highlighting the need for field blanks to account for such artefacts. Although the studies on sampler comparison focused on $PM_{2.5}$ mass, their findings highlight the imperative of collecting and analysing field blanks for both the Harvard Impactor and MiniVol samplers (Hill et al., 1999).

When considering the quantification of semi-volatile organic compounds such as PAEs, a direct comparison of sampling biases and artefacts with studies focused on $PM_{2.5}$ mass is not straightforward. The measurement of semi-volatile organic compounds could be affected by both positive and negative artefacts, which can be associated with the sorption properties of target analytes on sampling media such as filters (Wang et al., 2015). For example, positive artefacts can arise from the adsorption of gas-phase compounds onto the filter, while negative artefacts can occur through the volatilisation of target compounds. For the quantification of 19 semi-volatile compounds, Wang et al. (2015) suggested employing back-up filters during sampling to correct for artefacts effectively. However, the same study noted that increasing the sampling time (>10 h) reduced sampling artefacts, as the gas-phase concentration gradient decreased within the QFF. While the absence of backup filters or denuders in this pilot study may lead to the adsorption of some gas-phase PAEs on the filter surface, the extended sampling durations (16–60 h) of more than 10 h would effectively reduce sampling artefacts as noted by Wang et al. (2015).

To account for potential analyte losses during extraction, all filter blanks and samples were spiked with the same amount of deuterated internal standard (60 pg/μL DEHP-d4) prior to ultrasonication. A previous method validation study demonstrated good extraction efficiencies (±20%) for PAEs from spiked filter substrates using a 50:50 (v/v) dichloromethane:methanol mixture (Argamino et al., 2024).

Analytical instrument performance was assessed for both GC-Orbitrap-MS and GC-QTOF-MS using the same system suitability test (SST) mixture (100 pg/μL PAE mix and 60 pg/uL DEHP-d4). Analyte concentrations in the SST were consistently within ±20% of the target (100 pg/μL). GC MS consumables (e.g., columns and type of liners) of the same specification and internal standard (DEHP-d4) were used across both systems. A shorter analysis time was employed in GC-QTOF-MS method compared to the GC-Orbitrap-MS method (described in Section S1) due to limited access to external facilities. Analyte identities in the GC-QTOF-MS full mass spectrum were confirmed against the National Institute for Standards and Technology Electron Impact MS (NIST EI-MS) database.

Quality assurance and quality control measures (QA/QC) were employed throughout the GC-QTOF-MS analysis. Following extraction, samples were reconstituted in glass vials with minimal headspace and immediately sealed to minimise evaporation and potential contamination. Reconstituted samples were analysed in a structured autosampler sample sequence, alongside method blanks, QC samples, and calibration standards. This sequence incorporated air blanks between sample injections to mitigate carryover and ensure data integrity. Replicate injections of PM samples were performed, with a criterion of %RSD<20 for acceptable precision.

While DMP, DBP, DEHA, DOP, and DEHP were detected above the LOQ in indoor $PM_{2.5}$ samples from Office 1 and 2 (analysed via GC-QTOF-MS), only DEHP consistently passed all QC checks throughout the entire sample analysis sequence, including those related to calibration linearity, blank levels, and precision of replicate injections. All target analytes passed QC checks in the GC-Orbitrap-MS analysis (Brazilian samples), however, only DEHP and DEHA were detected above the LOQ. DEHP was detected in all Brazilian samples, and DEHA was observed in only a single sample (1 ng/m^3). Therefore, this discussion focuses primarily on DEHP due to its high analytical detection confidence, and verified QC checks during indoor $PM_{2.5}$ sample analysis. Sample O2-5 (a weekend $PM_{2.5}$ sample from office 2) was excluded from the discussion due to poor precision of DEHP concentrations in replicate injections (RSD = 38%), indicating a deviation from the established QC criteria. LODs and LOQs are presented in the Supplementary Information (Table S4).

2.6. Human exposure risk assessment

The average daily dose for inhalation was calculated using Eq. (1):

$$ADD = \frac{C \times IR \times EF \times ED}{BW \times AT} \quad (1)$$

where C = median concentration (ng/m^3), IR = average inhalation rate (16 m^3/h), EF = exposure frequency (days/year), ED = exposure duration (year), BW = average body weight (85 kg for Office 1 and 2 (UK), and 80 kg for Office 3 (Brazil)), AT = averaging time (day). The average inhalation rate was based on the US EPA Exposure Factors Handbook (US EPA, 2011). Average body weight used in the calculations were obtained from data published by the national authorities from the UK (NHS, 2022) and Brazil (IBGE, 2014).

In the absence of chronic inhalation reference concentration (RfC) values for the inhalation exposure route, this work extrapolated inhalation RfCs based on US EPA guidelines (Eq. (2))(Monnot et al., 2023; US EPA, 1994):

$$\text{extrapolated RfC} = RfD \times \frac{BW}{IR} \quad (2)$$

where RfD = oral reference dose (Table 1), BW = average body weight (707 kg), and IR = average inhalation rate (16 m^3/h). The oral RfD value for DEHP (20 μg/kg-bw•day) was obtained from US EPA IRIS (US EPA, 2025a).

The HQ was calculated to estimate the human inhalation hazard of the PAEs detected in this work (Eq. (3))(ATSDR, 2025):

$$HQ = \frac{AAC}{RfC \text{ or } RfD} \quad (3)$$

AAC, RfC, and RfD values are presented in Table 1.

HQ values were interpreted as follows: HQ < 1 indicates that the exposure unlikely to cause adverse non-cancer effects. HQ > 1 suggests potential adverse health effects. However, it must be noted an HQ > 1 does not necessarily mean that adverse health effects are statistically likely to occur (US EPA, 2024b).

The Incremental Lifetime Cancer Risk (ILCR) was calculated to represent the incremental probability of an individual developing cancer over a lifetime due to exposure to DEHP, a probable carcinogen (Eq. (4)):

$$ILCR_{DEHP} = AAC \times IUR \times \frac{ED}{LY} \quad (4)$$

where IUR is the inhalation unit risk ($2.4x10^{-3}$ ng/m^3), ED is the lifetime exposure duration (about 48 years for both the UK and Brazil (ONS, 2018; World Bank, 2026), and LY is lifetime in years (80 years for the UK (WHO, 2024b); 72 years for Brazil (WHO, 2024c; OEHHA, 2025). The cancer risk threshold for DEHP is $1x10^{-6}$. ILCR values below $1x10^{-6}$ indicate negligible cancer risk, while exceedance of the value indicates significant cancer risk (ATSDR, 2025).

2.7. Statistical analysis

Statistical analyses were performed using GraphPad Prism (version 10.6.0). To test for significant differences in DEHP concentrations in indoor $PM_{2.5}$ samples (n = 14) collected from Offices 1, 2, and 3, a non-normal distribution was assumed given the limited power of normality tests for small sample sizes (GraphPad, 2025b; Motulsky, 2014). Consequently, the non-parametric Kruskal–Wallis test followed by Dunn's post hoc test was applied to assess statistically significant differences ($p < 0.05$). Due to the small sample sizes per group ($n \leq 5$), exact *p*-values were calculated rather than relying on the asymptotic Chi-square approximation (GraphPad, 2025a; Kruskal and Wallis, 1952).

3. Results and discussion

3.1. Occurrence of DEHP in indoor $PM_{2.5}$ collected from offices

DEHP, a regulated carcinogen, was consistently observed above the method LOQ in indoor $PM_{2.5}$ collected from the three office environments (Fig. 1; Table S5) with mean concentrations of 8.5 ± 9.0 ng/m^3 (Office 1, UK, unoccupied), 10.3 ± 2.9 ng/m^3 (Office 2, UK, occupied), and 28 ± 4.3 ng/m^3 (Office 3, Brazil, occupied).

Despite a relatively small dataset, statistical analysis indicated a significant difference in DEHP concentrations among the three office groups ($p = 0.002$, Kruskal-Wallis). Dunn's post-hoc test yielded the following results: (1) Office 1 vs. Office 2: $p > 0.999$; (2) Office 1 vs. Office 3: $p = 0.011$; (3) Office 2 vs Office 3: $p = 0.049$.

The low concentration of other target PAEs and the detection of

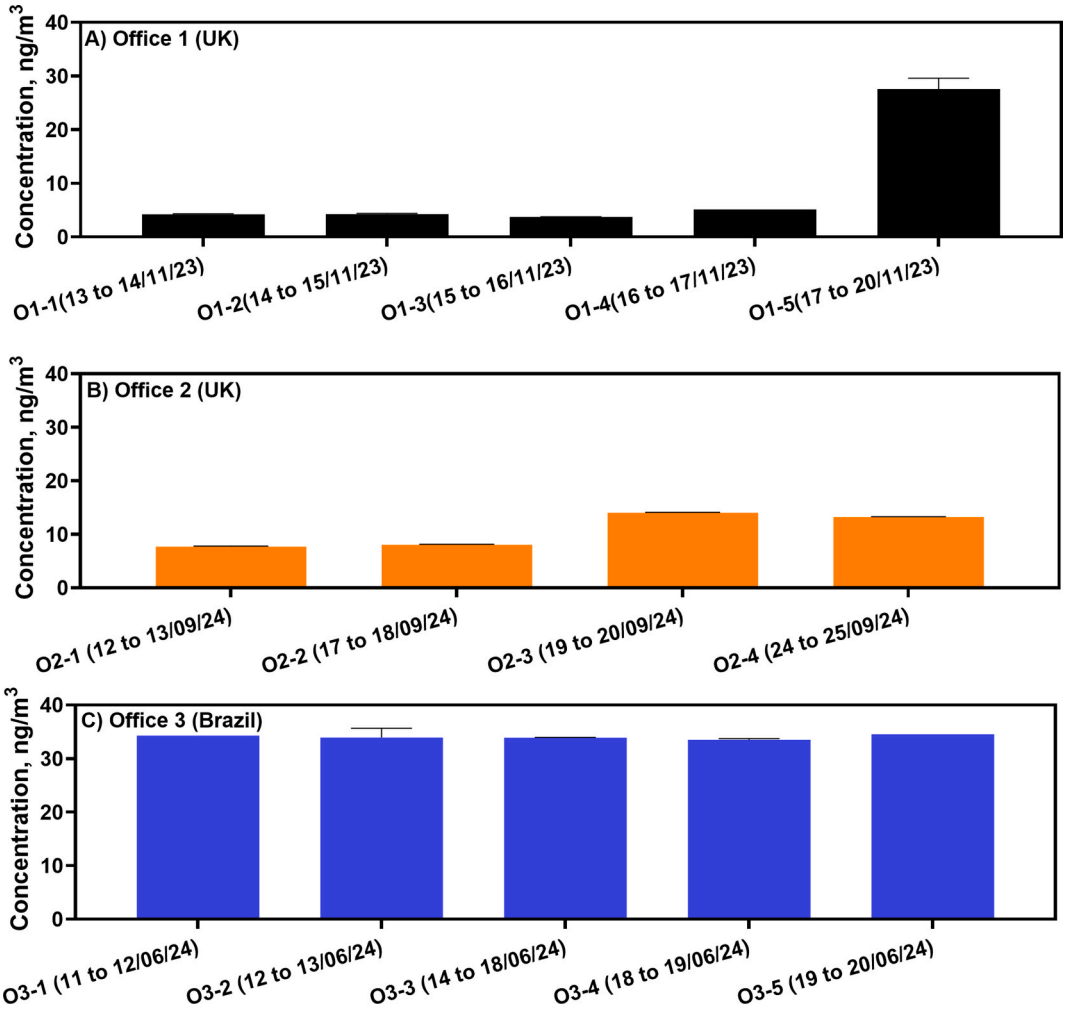

Fig. 1. DEHP concentrations in indoor $PM_{2.5}$ from Office 1(O1) (plot A), Office 2(O2) (plot B), and Office 3(O3) (plot C). Standard deviation bars correspond to the variation of concentrations within two repeat injections of each sample.

DEHP (above the LOQ) in indoor $PM_{2.5}$, could potentially be explained by physical and chemical properties such as vapour pressure and molecular weight. Among the target PAEs, DEHP had the lowest vapour pressure at 9.75 x 10^{-6} mm Hg (at 25 °C), and highest molecular mass (390.56 g/mol) therefore supporting its observation significantly above the LOQ in indoor $PM_{2.5}$.

The results of this work are supported by previous reports were DEHP was the most dominant plasticiser in indoor environments (Ait Bamai et al., 2014; Chen et al., 2022; Shinohara et al., 2024). For example, Chen et al. (2022) assessed the occurrence of six PAEs in $PM_{2.5}$ collected from 26 homes in Hong Kong (similar set of target pollutants as in the present work). DEHP was the most abundant PAE congener (85% of total PAE concentration) and its indoor emissions were attributed to household products, and continuous resuspension of PAE-contaminated house dust (Chen et al., 2022). Ait Bamai et al. (2014) assessed PAEs and DEHA levels in house dust from 128 homes in Sapporo, Japan. Among the target pollutants, DEHP was found at the highest concentration in both floor dust and multi-surface dust, reaching up to 44,000 μg/g. Ait Bamai et al. (2014) associated DEHP contamination in dust with PVC building materials, high relative humidity, and the age of the building (pre-2001). A recent study on PAEs in house dust from 100 homes in Nagoya, Japan reported that DEHP accounted for 85% of the total concentration of phthalates and non-phthalate plasticisers detected which were linked to floor carpets (Shinohara et al., 2024).

The higher concentrations and lower variability of DEHP were observed in indoor $PM_{2.5}$ within occupied offices (e.g., Office 2, Office 3) (SD < 5) compared to the unoccupied office (Office 1). The higher occupancy and activity in Offices 2 and 3 could potentially drive the constant resuspension of indoor particles and, including PM-bound DEHP, as observed in previous studies (Lunderberg et al., 2019; Qian et al., 2014; Rodes et al., 1991). Lunderberg et al. (2019) reported that human-related activities in a residential site substantially influenced the concentrations and dynamic behaviour of DEHP. The low variability in DEHP levels in the occupied offices could potentially suggest a steady-state concentration typical of SVOCs, which is reached when the rate of emission from a source material into an environment (e.g., indoor air) is balanced by its rate of removal (e.g., through ventilation, sorption by sinks) (Weschler and Nazaroff, 2008).

Conversely, a higher DEHP concentration was observed in the weekend sample collected from Office 1 (O1-5: 27 ng/m^3) compared to the weekday samples (O1-1 to O1-4, mean: 4 ng/m^3) (Fig. 1). This could be attributed to the build-up of PAEs in Office 1 over the weekend when ventilation may be reduced. The observed increase was not likely linked to sampling duration, as weekday concentrations consistently exceeded the LOQ.

Potential sources of DEHP indoors including building materials, furniture, and electronic equipment are present in the sampling locations. Office furnishings (e.g., floor construction materials) could act as long-term emission sources of PAEs as suggested in previous works (Lin et al., 2021). For example, Lin et al. (2021) reported DEHP concentrations of up to 4.5 μg/m^3 emitted from flooring materials (e.g., PVC tiles, plastic click flooring, and carpeted composite floors) in chamber experiments at room temperature (25 °C). Additionally, electronic and electrical equipment which contain PVC can release DEHP in indoor environments (Deng et al., 2021; Yang et al., 2020), and the heat generated during use can accelerate the release of DEHP (Clausen et al., 2012; Jeon et al., 2016).

Differences in the chemical composition of building materials and furniture, potentially influenced by differing regulatory frameworks in Brazil and the UK (Brazil Ministry of Health, 2019; Brazil Ministry of Health, 2021; Brazilian Institute of Geography and Statistics, 2025), could also contribute to the varying DEHP emissions in the sampling sites. To our knowledge, regulations currently limit DEHP concentration to less than 0.1% by weight for articles manufactured or imported into the UK (UK National Archives, 2025), while those in Brazil are more specific and focused on food contact materials and PVC articles (Brazil Ministry of Health, 2019; Brazil Ministry of Health, 2021; Brazilian Institute of Geography and Statistics, 2025).

Despite statistically significant differences, the direct comparison of indoor DEHP levels in the Brazil and UK offices is challenging due to variance in sampling and detection methods. The DEHP concentrations measured in this study (mean: 8.5 - 28 ng/m^3) were within the broad range reported for office environments across several regions (Table S5). The levels of DEHP were comparable to findings in various indoor environments (homes, offices, laboratories, schools, salons) in New York, USA (unspecified PM size; mean: 30 ng/m^3) (Rudel and Perovich, 2009; Tran and Kannan, 2015) and offices in Oslo, Norway ($PM_{2.5}$; mean: 5 ng/m^3) (Rakkestad et al., 2007), but were considerably lower than the concentration reported in certain highly contaminated offices, such as those recently furnished in Warsaw, Poland (PM_4; up to 1660 ng/m^3) (Szewczyńska et al., 2021) or in Beijing offices (China) ($PM_{2.5}$; up to 123 ng/m^3) (Chen et al., 2018). The variability across the reported studies highlights the significant influence of building characteristics, furnishing materials, occupant activities, ventilation strategies, and potentially geographical/regulatory contexts on indoor DEHP pollution levels.

3.2. Preliminary exposure risk assessment

The potential health risks associated with inhalation exposure to the DEHP measured in indoor $PM_{2.5}$ (Section 3.1) were assessed following U.S. EPA guidelines (details in Section 2.3). It must be noted that the primary challenge in this exposure assessment is the scarcity of experimentally derived, inhalation-specific toxicity values for DEHP.

To evaluate daily intake, ADDs (ng/kg-bw•day) were calculated for typical office worker exposure scenarios based on median pollutant concentrations using Eq. (2). The ADDs varied by compound and location, consistent with observed concentration patterns in the sampling sites. For instance, DEHP ADD was highest in Office 3 (0.06 ng/kg-bw•day), reflecting the higher concentrations measured in the sampling site. Studies estimating the ADD from airborne indoor $PM_{2.5}$ concentrations are scarce. For instance, the calculated ADDs for DEHP in this work were notably lower than estimates from previous studies in highly polluted office environments in Beijing, China (DEHP ADD: 173 ng/kg-bw•day) (Chen et al., 2018).

To evaluate non-cancer risks, the HQ for DEHP was calculated using Eq. (3). However, due to the absence of an inhalation RfC, the assessment relied on toxicity values derived from oral exposure (oral RfDs) and extrapolated using RtR methods (Fadel et al., 2021; US EPA, 1994) (i.e., PAEs) (Section S2). This risk assessment approach is commonly used for studies on PAEs in $PM_{2.5}$ (note that reports are scarce of indoor $PM_{2.5}$) (Fadel et al., 2022; Guo et al., 2024; Hong et al., 2024; Munyaneza et al., 2023; Patnana et al., 2022). While this method provides a useful basis for preliminary assessment, further refinement may be required as more inhalation-specific data for airborne PAEs become available.

The validity of the RtR extrapolation method is anchored on specific criteria, where the pollutant should have predominant systemic effects, minimal route-dependent first-pass metabolism, and absence of significant portal-of-entry effects (US EPA, 2024a). PAEs may not meet these conditions due to potential direct respiratory tract effects (i.e., portal of entry toxicity) upon inhalation, which oral RfDs may not capture. For instance, PAEs can induce respiratory effects (i.e., chronic obstructive pulmonary disease (COPD), asthma, airway inflammation) (Bornehag et al., 2005; Hsu et al., 2012; Quirós-Alcalá et al., 2023), highlighting the potential importance of portal-of-entry toxicity for these pollutants via inhalation. The available oral RfDs for DEHP were based on liver toxicity (US EPA, 2017) and may not be relevant for predicting lung toxicity or reproductive effects resulting from inhalation exposure. There are also toxicokinetic differences between oral and inhalation exposure. For example, DEHP undergoes extensive hydrolysis following ingestion (i.e., first-pass metabolism in the liver), which is bypassed during inhalation

(Frederiksen et al., 2007). Consequently, significant uncertainty surrounds the validity of using current methods to estimate non-cancer risk from PAE inhalation, as the evidence for meeting the RtR extrapolation criteria is scarce.

Despite these limitations, the HQ for DEHP was calculated for illustrative purposes (presented in Table 1) and were less than 1. While these suggest non-hazardous levels after one year of exposure, consistent with some previous reports, extreme caution must be exercised when interpreting these findings due to the significant concerns regarding the appropriateness of the underlying toxicity values and the RtR extrapolation method. While not applicable in this work, it is worth noting that the cumulative hazard can also be assessed by calculating the hazard index (i.e., summation of HQs of multiple target pollutants). While this study focuses primarily on DEHP, PAEs may occur as mixtures in indoor environments, and exposure may therefore involve multiple PAEs simultaneously. Combined exposure may lead to additive or synergistic endocrine-disrupting effects, potentially increasing the overall health risk beyond what is estimated for individual compounds (Argamino et al., 2024; Chen et al., 2018; Huang et al., 2023; Miranowicz-Dzierżawska, 2023; Rakkestad et al., 2007; Wang et al., 2019; Zhang et al., 2014). For the carcinogenic risk assessment of DEHP via $PM_{2.5}$ inhalation, the estimated ILCR values were calculated based on the ADDs using Eq. (4). Since an Inhalation Unit Risk (IUR) for DEHP is unavailable (i.e., U.S. EPA), this assessment relied on the IUR for DEHP developed by California's OEHHA (2.4×10^{-3} ng/m^3) (OEHHA, 2025). The estimated ILCR values for Office 1, Office 2, and Office 3 were 5.66×10^{-11}, 1.41×10^{-10}, 4.40×10^{-10}, respectively. These risk estimates were below the 1×10^{-6} threshold often considered acceptable by regulatory bodies (ATSDR, 2025) and suggest that lifetime exposure to the measured indoor DEHP pose negligible carcinogenic risk. However, since the IUR value was extrapolated from oral toxicity benchmarks, concerns are also raised on the accuracy of these cancer risk predictions for DEHP. Similar to data limitations previously stated for ADDs, cancer risk assessments are scarce for DEHP in indoor $PM_{2.5}$ and the risk estimates in this work were lower those recently reported for average personal $PM_{2.5}$ exposures in Hong Kong adults (CR: $2x10^{-5}$) (Chen et al., 2022). Addressing risk assessment uncertainties requires acknowledging limitations in future reports and prioritising the development of experimentally derived inhalation RfCs for PAEs to enable accurate, pathway-specific non-cancer risk assessments for indoor and outdoor air exposures.

Risks estimated in this work potentially underestimate the human health and toxicological impacts of exposure to $PM_{2.5}$ bound-DEHP. DEHP is known to be cytotoxic to human lung cells, with mechanisms linked to inflammation, activation of cell death pathways, and disruption of cell cycle regulation (Argamino et al., 2025; Atia and Abdel-Gawad, 2019; Ma et al., 2018; Qin et al., 2021). DEHP exposure has been positively associated with respiratory illnesses including chronic obstructive pulmonary disorder (COPD), asthma, and airway inflammation (Bornehag et al., 2005; Hsu et al., 2012; Quirós-Alcalá et al., 2023). Moreover, exposure to DEHP, an endocrine disrupting compound (EDC), has been linked to obesity, and an increase in diabetes risk, even at low doses (Ernst et al., 2020; Yue et al., 2024). Lee et al., (2017) reported that *in vivo* (rat model system) fetal exposure to environmentally relevant doses of DEHP led to decreased levels of major steroid hormones in adult rats that could lead to the development of diseases.

This study focuses on inhalation exposure associated with $PM_{2.5}$-bound PAEs. $PM_{2.5}$ is a widely used metric for assessing particulate air pollution and related health risks and forms the basis of air quality guidelines established by the World Health Organization. However, total human exposure to airborne PAEs potentially includes gas-phase inhalation, dust ingestion, and dermal uptake (Chen et al., 2024). Considering other potential routes for airborne PAE exposure, it must be noted the measurements presented here represent a conservative, lower-bound estimate of the total indoor plasticiser exposure.

4. Conclusions

In this work, we performed a pilot investigation on the presence of six priority PAEs in $PM_{2.5}$, from selected indoor environments (offices) in Brazil and the UK. DEHP, a possible carcinogen and a widely restricted chemical, was the only target pollutant detected across all the sampling sites with high confidence. In contrast, concentrations of other target PAEs were either below the detection limit or failed to pass the quality control requirements for confident quantification. DEHP has a low vapour pressure and high molecular weight compared to other target PAEs, which could potentially explain its detection in office indoor $PM_{2.5}$. Airborne DEHP levels detected within occupied offices supports evidence from the literature that building materials and furnishings could act as persistent indoor sources. The low variability observed in DEHP levels across sampling days implies a steady-state concentration, a behaviour typical of SVOCs, which warrants further studies.

While the observed levels in Brazil (28 ± 4.3 ng/m^3) and the UK sites (9.3 ± 7.1 ng/m^3) fall within the range reported in other international studies, these findings highlight a continuous source of exposure for occupants. To the best of our knowledge, this study provides the first quantitative measurements of DEHP in indoor PM (specifically to sizes below 2.5 μm) from office environments in both Brazil and the UK.

Although the reported DEHP concentrations in this work were well below the UK occupational exposure limits (5.0 mg/m^3), such limits are designed for healthy adult workers and do not account for continuous, chronic exposure or the vulnerability of sensitive groups. Significant uncertainty surrounds non-cancer and cancer risk evaluations for airborne DEHP since inhalation-specific toxicity benchmarks (RfCs) are largely unavailable, driving the reliance on oral RfDs. More studies are needed to generate experimentally-derived inhalation toxicity benchmarks for PAEs, including DEHP, for more accurate risk assessments.

The findings in this pilot study demonstrate the value of expanding monitoring to a wider array of indoor settings, particularly in the UK and Brazil. Future studies should focus on how variables such as ventilation rates, room dimensions, indoor climate, and specific product standards influence the airborne concentrations of PAEs. Harmonised monitoring methods are crucial to support evidence-based policies regarding PAE usage in consumer products and to refine risk assessments for indoor exposure.

CRediT authorship contribution statement

Cristian Ryan A. Argamino: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Jéssica dos Santos Silva:** Writing – review & editing, Investigation, Formal analysis, Data curation, Conceptualization. **Jishnu Pandamkulangara Kizhakkethil:** Writing – review & editing, Investigation. **Svetlana Stevanovic:** Writing – review & editing, Writing – original draft, Supervision, Funding acquisition, Conceptualization. **Martyn Ward:** Writing – review & editing, Resources, Methodology, Formal analysis. **Matthew McKenzie:** Writing – review & editing, Supervision, Conceptualization. **Anna Bogush:** Writing – review & editing, Supervision, Conceptualization. **Jacqueline F. Hamilton:** Writing – review & editing, Resources, Conceptualization. **Ricardo H.M. Godoi:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization. **Ivan Kourtchev:** Writing – review & editing, Writing – original draft, Supervision, Resources, Project administration, Funding acquisition, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors wish to acknowledge Coventry University (CU) QR funding and Deakin University for providing a Cotutelle PhD studentship to Cristian Argamino. They also thank the CU Centre for Agroecology, Water and Resilience (CAWR) for financial support and Wolfson Atmospheric Chemistry Laboratories for providing technical support.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.apr.2026.102987.

References

Abdi, S., Sobhanardakani, S., Lorestani, B., Cheraghi, M., Panahi, H.A., 2021. Analysis and health risk assessment of phthalate esters (PAEs) in indoor dust of preschool and elementary school centers in city of Tehran, Iran. Environ. Sci. Pollut. Control Ser. 28 (43), 61151–61162. https://doi.org/10.1007/s11356-021-14845-y.

Agilent Technologies, 2025. Liquid Chromatography/Mass Spectrometry Fundamentals. Retrieved July 17, 2025 from. https://www.agilent.com/en/product/liquid-chromatography-mass-spectrometry-lc-ms/lcms-fundamentals/lcms-instrument-types.

Ait Bamai, Y., Araki, A., Kawai, T., Tsuboi, T., Saito, I., Yoshioka, E., Kanazawa, A., Tajima, S., Shi, C., Tamakoshi, A., Kishi, R., 2014. Associations of phthalate concentrations in floor dust and multi-surface dust with the interior materials in Japanese dwellings. Sci. Total Environ. 468–469, 147–157. https://doi.org/10.1016/j.scitotenv.2013.07.107.

Argamino, C.R.A., Pathania, D., Schultz, A., Bogush, A., Kourtchev, I., McKenzie, M., Stevanovic, S., 2025. Individual and binary exposure of short- and long-chain phthalate esters decreases viability and inhibits cellular respiration in human lung cells. Environ. Int. 202, 109603. https://doi.org/10.1016/j.envint.2025.109603.

Argamino, C.R.A., Sebben, B.G., da Costa, G., Towers, S., Bogush, A., Stevanovic, S., Godoi, R.H.M., Kourtchev, I., 2024. Development and validation of a GC Orbitrap-MS method for the analysis of phthalate esters (PAE) and bis(2-ethylhexyl)adipate (DEHA) in atmospheric particles and its application for screening PM2.5 from Curitiba, Brazil. Anal. Methods 16 (11), 1579–1592. https://doi.org/10.1039/D3AY02197A.

Atia, T., Abdel-Gawad, S., 2019. Pulmonary toxicity induced by exposure to phthalates, an experimental study. Inhal. Toxicol. 31 (9–10), 376–383. https://doi.org/10.1080/08958378.2019.1695025.

ATSDR, 2025. Calculating hazard quotients and cancer risk estimates. Retrieved 17 July, 2025 from. https://www.atsdr.cdc.gov/pha-guidance/conducting_scientific_evaluations/epcs_and_exposure_calculations/hazardquotients_cancerrisk.html.

Bahrami, A., Haghighat, F., Niu, J., Zhu, J., 2025. Phthalates particle-gas partitioning of inorganic particles: effect of particle size and phthalates adsorption competition. Build. Environ. 285, 113619. https://doi.org/10.1016/j.buildenv.2025.113619.

Baldauf, R.W., Lane, D.D., Marotz, G.A., Wiener, R.W., 2001. Performance evaluation of the portable MiniVOL particulate matter sampler. Atmos. Environ. 35 (35), 6087–6091. https://doi.org/10.1016/S1352-2310(01)00403-4.

Barroso, P.J., Santos, J.L., Martín, J., Aparicio, I., Alonso, E., 2019. Emerging contaminants in the atmosphere: analysis, occurrence and future challenges. Crit. Rev. Environ. Sci. Technol. 49 (2), 104–171. https://doi.org/10.1080/10643389.2018.1540761.

Becker, K., Seiwert, M., Angerer, J., Heger, W., Koch, H.M., Nagorka, R., Roßkamp, E., Schlüter, C., Seifert, B., Ullrich, D., 2004. DEHP metabolites in urine of children and DEHP in house dust. Int. J. Hyg Environ. Health 207 (5), 409–417. https://doi.org/10.1078/1438-4639-00309.

Bornehag, C.-G., Lundgren, B., Weschler Charles, J., Sigsgaard, T., Hagerhed-Engman, L., Sundell, J., 2005. Phthalates in indoor dust and their association with building characteristics. Environ. Health Perspect. 113 (10), 1399–1404. https://doi.org/10.1289/ehp.7809.

Brazil Ministry of Health, 2019. Collegiate board resolution - RDC no. 326, OF DECEMBER 3, 2019. Retrieved 20 June, 2025 from. https://www.in.gov.br/en/web/dou/-/resolucao-da-diretoria-colegiada-rdc-n-326-de-3-de-dezembro-de-2019-231272617.

Brazil Ministry of Health, 2021. College resolution board - RDC No. 529, 4 August 2021. Retrieved 20 June, 2025 from. https://anvisalegis.datalegis.net/action/UrlPublicasAction.php?acao=abrirAtoPublico&num_ato=00000529&sgl_tipo=RDC&sgl_orgao=RDC/DC/ANVISA/MS&vlr_ano=2021&seq_ato=000&cod_modulo=134&cod_menu=1696.

Brazilian Institute of Geography and Statistics, 2025. Cities and states: Rio Branco do sul. Retrieved 17 July, 2025 from. https://sidra.ibge.gov.br/tabela/5938#/n6/3304557/v/37/p/last%201/d/v37%200/l/v,p,t/resultado.

Caldwell, J.C., 2012. DEHP: genotoxicity and potential carcinogenic mechanisms—A review. Mutat. Res., Rev. Mutat. Res. 751 (2), 82–157. https://doi.org/10.1016/j.mrrev.2012.03.001.

Chang, W.-H., Herianto, S., Lee, C.-C., Hung, H., Chen, H.-L., 2021. The effects of phthalate ester exposure on human health: a review. Sci. Total Environ. 786, 147371. https://doi.org/10.1016/j.scitotenv.2021.147371.

Chatoutsidou, S.E., Ondráček, J., Tesar, O., Tørseth, K., Ždímal, V., Lazaridis, M., 2015. Indoor/outdoor particulate matter number and mass concentration in modern offices. Build. Environ. 92, 462–474. https://doi.org/10.1016/j.buildenv.2015.05.023.

Chen, J., Ward, T.J., Ho, S.S.H., Ho, K.F., 2022. Occurrence and risk assessment of personal PM2.5-Bound phthalates exposure for adults in Hong Kong. Int. J. Environ. Res. Publ. Health 19 (20). https://doi.org/10.3390/ijerph192013425. Article 13425.

Chen, Y., Lv, D., Li, X., Zhu, T., 2018. PM2.5-bound phthalates in indoor and outdoor air in Beijing: seasonal distributions and human exposure via inhalation. Environ. Pollut. 241, 369–377. https://doi.org/10.1016/j.envpol.2018.05.081.

Chen, Z., Gao, Y., Xia, F., Bi, C., Mo, J., 2024. Formation kinetics of SVOC organic films and their impact on child exposure in indoor environments. Sci. Total Environ. 912, 168970. https://doi.org/10.1016/j.scitotenv.2023.168970.

Chen, Z., Tian, E., Jiang, Y., Mo, J., 2025. Global perspectives on indoor phthalates and alternative plasticizers: occurrence and key transport parameters. J. Hazard Mater. 482, 136506. https://doi.org/10.1016/j.jhazmat.2024.136506.

Clausen, P.A., Liu, Z., Kofoed-Sørensen, V., Little, J., Wolkoff, P., 2012. Influence of temperature on the emission of Di-(2-ethylhexyl)phthalate (DEHP) from PVC flooring in the emission cell FLEC. Environ. Sci. Technol. 46 (2), 909–915. https://doi.org/10.1021/es2035625.

Deng, M., Han, X., Ge, J., Liang, X., Du, B., Li, J., Zeng, L., 2021. Prevalence of phthalate alternatives and monoesters alongside traditional phthalates in indoor dust from a typical e-waste recycling area: source elucidation and co-exposure risk. J. Hazard Mater. 413. https://doi.org/10.1016/j.jhazmat.2021.125322. Article 125322.

dos Santos, Cruz, M.N.d.S.d.l., Gioda, A., Siqueira, C.Y.d.S., 2024. Characterization of indoor air quality in a university library: implications associated with pollutant emissions from new and old books and chemicals. Environ. Foren. 1–17. https://doi.org/10.1080/15275922.2024.2432495.

ECHA, 2025. Phthalates. Retrieved 1 December, 2025 from. https://echa.europa.eu/hot-topics/phthalates.

El Haddad, I., Marchand, N., Wortham, H., Piot, C., Besombes, J.L., Cozic, J., Chauvel, C., Armengaud, A., Robin, D., Jaffrezo, J.L., 2011. Primary sources of $PM_{2.5}$ organic aerosol in an industrial Mediterranean city, Marseille. Atmos. Chem. Phys. 11 (5), 2039–2058. https://doi.org/10.5194/acp-11-2039-2011.

Ernst, J., Grabiec, U., Falk, K., Dehghani, F., Schaedlich, K., 2020. The endocrine disruptor DEHP and the ECS: analysis of a possible crosstalk. Endocr. Connect. 9 (2), 101–110. https://doi.org/10.1530/EC-19-0548.

Fadel, M., Ledoux, F., Afif, C., Courcot, D., 2022. Human health risk assessment for PAHs, phthalates, elements, PCDD/Fs, and DL-PCBs in PM2.5 and for NMVOCs in two East-Mediterranean urban sites under industrial influence. Atmos. Pollut. Res. 13 (1), 101261. https://doi.org/10.1016/j.apr.2021.101261.

Fadel, M., Ledoux, F., Farhat, M., Kfoury, A., Courcot, D., Afif, C., 2021. PM2.5 characterization of primary and secondary organic aerosols in two urban-industrial areas in the East Mediterranean. J. Environ. Sci. (China) 101, 98–116. https://doi.org/10.1016/j.jes.2020.07.030.

Feng, J., Chan, C.K., Fang, M., Hu, M., He, L., Tang, X., 2005. Impact of meteorology and energy structure on solvent extractable organic compounds of PM2.5 in Beijing, China. Chemosphere 61 (5), 623–632. https://doi.org/10.1016/j.chemosphere.2005.03.067.

Frederiksen, H., Skakkebaek, N.E., Andersson, A.-M., 2007. Metabolism of phthalates in humans. Mol. Nutr. Food Res. 51 (7), 899–911. https://doi.org/10.1002/mnfr.200600243.

GraphPad, 2025a. Interpreting results: Kruskal-Wallis test. Retrieved 1 December, 2025 from. https://www.graphpad.com/guides/prism/latest/statistics/how_the_kruskal-wallis_test_works.htm.

GraphPad, 2025b. Q&A: normality tests. Retrieved 1 December 2025 from. https://www.graphpad.com/support/faqid/959/.

Guo, W., Zhang, Z., Zhu, R., Li, Z., Liu, C., Xiao, H., Xiao, H., 2024. Pollution characteristics, sources, and health risks of phthalate esters in ambient air: a daily continuous monitoring study in the central Chinese city of Nanchang. Chemosphere 353, 141564. https://doi.org/10.1016/j.chemosphere.2024.141564.

Hill, J., Patel, P.D., Turner, J.R., 1999. Performance Characterization of the MiniVol PM2.5 sampler (Air Quality laboratory report, Issue. Retrieved 10 July, 2025 from. https://www.airmetrics.com/wp-content/uploads/2021/08/Performance-Characterization-of-MiniVol-PM2.5.pdf.

Hong, W.-J., Wang, X., Ding, J.-J., Jiang, J.-M., Li, M.-J., Ji, S., Sang, N., Guo, L.-H., 2024. Seasonal variation, source apportionment, and cancer risk assessment of PM2.5-bound phthalates: a case study in Taiyuan, China. Air Qual. Atmos. Health 17 (3), 455–467. https://doi.org/10.1007/s11869-023-01454-6.

Hsu, N.-Y., Lee, C.-C., Wang, J.-Y., Li, Y.-C., Chang, H.-W., Chen, C.-Y., Bornehag, C.-G., Wu, P.-C., Sundell, J., Su, H.-J., 2012. Predicted risk of childhood allergy, asthma, and reported symptoms using measured phthalate exposure in dust and urine. Indoor Air 22 (3), 186–199. https://doi.org/10.1111/j.1600-0668.2011.00753.x.

Huang, M., Zeng, Y., Luo, K., Lan, B., Luo, J., Zeng, L., Kang, Y., 2023. Inhalation bioacessibility and lung cell penetration of indoor PM2.5-bound PAEs and its implication in risk assessment. Environ. Pollut. 322, 121216. https://doi.org/10.1016/j.envpol.2023.121216.

Huang, Y.Q., Zeng, Y., Wang, T., Chen, S.J., Guan, Y.F., Mai, B.X., 2022. PM2.5-bound phthalates and phthalate substitutes in a megacity of southern China: spatioseasonal variations, source apportionment, and risk assessment. Environ. Sci. Pollut. Control Ser. 29 (25), 37737–37747. https://doi.org/10.1007/s11356-022-18784-0.

IBGE, 2014. PNS - National Survey of Health (2013). Retrieved 10 July 2025 from. https://www.ibge.gov.br/en/statistics/social/health/29541-2013-national-survey-of-health.html?edicao=19614&t=resultados.

Jamarani, R., Erythropel, H.C., Nicell, J.A., Leask, R.L., Marić, M., 2018. How green is your plasticizer? Polymers 10 (8), 834. https://doi.org/10.3390/polym10080834.

Jeon, S., Kim, K.-T., Choi, K., 2016. Migration of DEHP and DINP into dust from PVC flooring products at different surface temperature. Sci. Total Environ. 547, 441–446. https://doi.org/10.1016/j.scitotenv.2015.12.135.

Kashyap, D., Agarwal, T., 2018. Concentration and factors affecting the distribution of phthalates in the air and dust: a global scenario. Sci. Total Environ. 635, 817–827. https://doi.org/10.1016/j.scitotenv.2018.04.158.

Kruskal, W.H., Wallis, W.A., 1952. Use of ranks in one-criterion variance analysis. J. Am. Stat. Assoc. 47 (260), 583–621. https://doi.org/10.1080/01621459.1952.10483441.

Li, J., Wang, G., 2015. Airborne particulate endocrine disrupting compounds in China: compositions, size distributions and seasonal variations of phthalate esters and bisphenol A. Atmos. Res. 154, 138–145. https://doi.org/10.1016/j.atmosres.2014.11.013.

Lee, S., Martinez-Arguelles, D.B., Campioli, E., Papadopoulos, V., 2017. Fetal exposure to low levels of the plasticizer DEHP predisposes the adult male adrenal gland to endocrine disruption. Endocrinology 158 (2), 304–318. https://doi.org/10.1210/en.2016-1604.

Li, P.H., Jia, H.Y., Wang, Y., Li, T., Wang, L., Li, Q.Q., Yang, M.M., Yue, J.J., Yi, X.L., Guo, L.Q., 2019. Characterization of PM2.5-bound phthalic acid esters (PAEs) at regional background site in northern China: Long-Range transport and risk assessmen. Sci. Total Environ. 659, 140–149. https://doi.org/10.1016/j.scitotenv.2018.12.246.

Lin, W.-T., Chen, C.-Y., Lee, C.-C., Chen, C.-C., Lo, S.-C., 2021. Air phthalate emitted from flooring building material by the Micro-Chamber method: Two-Stage emission evaluation and comparison. Toxics 9 (9), 216. https://doi.org/10.3390/toxics9090216.

Long, F., Ren, Y., Ji, Y., Bai, X., Li, H., Wang, G., Yan, X., Chen, Y., Li, J., Zhang, H., Gao, R., Bi, F., Wu, Z., 2025. The characteristics of phthalate acid esters and bisphenol A in PM2.5 of a petrochemical city: concentrations, compositions, and health risk assessment in dongying. Environ. Pollut. 367, 125568. https://doi.org/10.1016/j.envpol.2024.125568.

Lunderberg, D.M., Kristensen, K., Liu, Y., Misztal, P.K., Tian, Y., Arata, C., Wernis, R., Kreisberg, N., Nazaroff, W.W., Goldstein, A.H., 2019. Characterizing airborne phthalate concentrations and dynamics in a normally occupied residence. Environ. Sci. Technol. 53 (13), 7337–7346. https://doi.org/10.1021/acs.est.9b02123.

Ma, J., Chen, L.-l., Guo, Y., Wu, Q., Yang, M., Wu, M.-h., Kannan, K., 2014. Phthalate diesters in Airborne PM2.5 and PM10 in a suburban area of Shanghai: seasonal distribution and risk assessment. Sci. Total Environ. 497–498, 467–474. https://doi.org/10.1016/j.scitotenv.2014.08.012.

Ma, Y., Guo, Y., Wu, S., Lv, Z., Zhang, Q., Xie, X., Ke, Y., 2018. Analysis of toxicity effects of di-(2 ethylhexyl) phthalate exposure on human bronchial epithelial 16HBE cells. Cytotechnology 70 (1), 119–128. https://doi.org/10.1007/s10616-017-0111-6.

Mariana, M., Feiteiro, J., Verde, I., Cairrao, E., 2016. The effects of phthalates in the cardiovascular and reproductive systems: a review. Environ. Int. 94, 758–776. https://doi.org/10.1016/j.envint.2016.07.004.

Miranowicz-Dzierżawska, K., 2023. Comparison of binary mixtures of dibutyl phthalate and diisobutyl phthalate cytotoxicity towards skin and lung origin cells in vitro 486, 153433. https://doi.org/10.1016/j.tox.2023.153433.

Monnot, A.D., Massarsky, A., Garnick, L., Bandara, S.B., Unice, K.M., 2023. Can oral toxicity data for PFAS inform on toxicity via inhalation? Risk Anal. 43 (8), 1533–1538. https://doi.org/10.1111/risa.14039.

Motulsky, H., 2014. Intuitive Biostatistics: a Nonmathematical Guide to Statistical Thinking. Oxford University Press, USA.

Munyaneza, J., Hossain, M.F., Jia, Q., Duan, Y., Xiu, G., 2023. Space-time characteristics of 16 PM2.5-bound phthalates (PAEs) in ambient air from Shanghai: profiles, sources, meteorological effects, and exposure risks. Aerosol Air Qual. Res. 23 (11). https://doi.org/10.4209/aaqr.220465. Article 220465.

NHS, 2022. Health survey for England, 2021. Retrieved 10 July 2025 from. https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2021/part-4-trends.

OEHHA, 2025. Di(2-ethylhexyl)phthalate. Retrieved 21 June, 2025 from. https://oehha.ca.gov/chemicals/di2-ethylhexylphthalate.

ONS, 2018. UK labour market: december 2018. Retrieved on. https://www.ons.gov.uk/employmentandlabourmarket/peopleinwork/employmentandemployeetypes/bulletins/uklabourmarket/december2018. (Accessed 12 March 2026).

Patnana, D., Chandra, B., Chaudhary, P., Sinha, B., Sinha, V., 2022. Optimized LC-MS/MS method for simultaneous determination of endocrine disruptors and PAHs bound to PM2. 5: sources and health risk in Indo-Gangetic Plain. Atmos. Environ. 290, 119363. https://doi.org/10.1016/j.atmosenv.2022.119363.

Qian, J., Peccia, J., Ferro, A.R., 2014. Walking-induced particle resuspension in indoor environments. Atmos. Environ. 89, 464–481. https://doi.org/10.1016/j.atmosenv.2014.02.035.

Qin, Y., Zhang, J., Avellán-Llaguno, R.D., Zhang, X., Huang, Q., 2021. DEHP-elicited small extracellular vesicles miR-26a-5p promoted metastasis in nearby normal A549 cells. Environ. Pollut. 272, 116005. https://doi.org/10.1016/j.envpol.2020.116005.

Quintana-Belmares, R.O., Krais, A.M., Esfahani, B.K., Rosas-Pérez, I., Mucs, D., López-Marure, R., Bergman, Å., Alfaro-Moreno, E., 2018. Phthalate esters on urban airborne particles: levels in PM10 and PM2. 5 from Mexico City and theoretical assessment of lung exposure. Environ. Res. 161, 439–445. https://doi.org/10.1016/j.envres.2017.11.039.

Quirós-Alcalá, L., Belz, D.C., Woo, H., Lorizio, W., Putcha, N., Koehler, K., McCormack, M.C., Hansel, N.N., 2023. A cross sectional pilot study to assess the role of phthalates on respiratory morbidity among patients with chronic obstructive pulmonary disease. Environ. Res. 225, 115622. https://doi.org/10.1016/j.envres.2023.115622.

Rakkestad, K.E., Dye, C.J., Yttri, K.E., Holme, J.A., Hongslo, J.K., Schwarze, P.E., Becher, R., 2007. Phthalate levels in Norwegian indoor air related to particle size fraction. J. Environ. Monit. 9 (12), 1419–1425. https://doi.org/10.1039/B709947A.

Rodes, C.E., Kamens, R.M., Wiener, R.W., 1991. The significance and characteristics of the personal activity cloud on exposure assessment measurements for indoor contaminants. Indoor Air 1 (2), 123–145. https://doi.org/10.1111/j.1600-0668.1991.03-12.x.

Rudel, R.A., Perovich, L.J., 2009. Endocrine disrupting chemicals in indoor and outdoor air. Atmos. Environ. 43 (1), 170–181. https://doi.org/10.1016/j.atmosenv.2008.09.025.

Růžičková, J., Raclavská, H., Raclavský, K., Juchelková, D., 2016. Phthalates in PM2.5 airborne particles in the Moravian-Silesian Region, Czech Republic. Perspectives in Science 7, 178–183. https://doi.org/10.1016/j.pisc.2015.11.029.

Santacruz, C.P., Håkansson, P., Barofsky, D.F., Piyadasa, C.K.G., 2007. A constant-momentum/Energy-Selector time-of-flight mass spectrometer. J. Am. Soc. Mass Spectrom. 18 (1), 92–101. https://doi.org/10.1016/j.jasms.2006.08.020.

Shi, S., Cao, J., Zhang, Y., Zhao, B., 2018. Emissions of phthalates from indoor flat materials in Chinese residences. Environ. Sci. Technol. 52 (22), 13166–13173. https://doi.org/10.1021/acs.est.8b03580.

Shinohara, N., Oguri, T., Takagi, M., Ueyama, J., Isobe, T., 2024. Evaluating the risk of phthalate and non-phthalate plasticizers in dust samples from 100 Japanese houses. Environ. Int. 183, 108399. https://doi.org/10.1016/j.envint.2023.108399.

Szewczyńska, M., Dobrzyńska, E., Pośniak, M., 2021. Determination of phthalates in particulate matter and gaseous phase emitted in indoor air of offices. Environ. Sci. Pollut. Control Ser. 28 (42), 59319–59327. https://doi.org/10.1007/s11356-020-10195-3.

Toro-Heredia, J., Jirau-Colón, H., Jiménez-Vélez, B.D., 2021. Linking PM2. 5 organic constituents, relative toxicity and health effects in Puerto Rico. Environ. Chall. 5, 100350. https://doi.org/10.1016/j.envc.2021.100350.

Tran, T.M., Kannan, K., 2015. Occurrence of phthalate diesters in particulate and vapor phases in indoor air and implications for human exposure in Albany, New York, USA. Arch. Environ. Contam. Toxicol. 68 (3), 489–499. https://doi.org/10.1007/s00244-015-0140-0.

Turpin, B.J., Saxena, P., Andrews, E., 2000. Measuring and simulating particulate organics in the atmosphere: problems and prospects. Atmos. Environ. 34 (18), 2983–3013. https://doi.org/10.1016/S1352-2310(99)00501-4.

UK National Archives, 2025. Commission regulation (EU) 2018/2005. Retrieved from 17 July, 2025 from. https://www.legislation.gov.uk/eur/2018/2005#text%3Dphthalate#match-1.

US EPA, 1994. METHODS FOR DERIVATION OF INHALATION REFERENCE CONCENTRATIONS AND APPLICATION OF NHALATION DOSIMETRY. Retrieved 17 July, 2025 from. https://www.epa.gov/sites/default/files/2014-11/documents/rfc_methodology.pdf.

US EPA, 2011. Exposure Factors Handbook, 2011 Edition. Retrieved 10 May, 2025 from. https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.

US EPA, 2014. Priority Pollutant List. Retrieved 10 July, 2025 from. https://www.epa.gov/sites/default/files/2015-09/documents/priority-pollutant-list-epa.pdf.

US EPA, 2017. Di (2-ethylhexyl)phthalate (DEHP) CASRN 117-81-7 | DTXSID5020607. Retrieved 10 July, 2025 from. https://cfpub.epa.gov/ncea/iris2/chemicallanding.cfm?substance_nmbr=14.

US EPA, 2024a. Appendix B: Route-to-Route extrapolation of inhalation benchmarks. Retrieved 17 July 2025 from. https://semspub.epa.gov/work/11/175214.pdf.

US EPA, 2024b. EPA's air toxics screening assessment. Retrieved 10 July, 2025 from. https://www.epa.gov/system/files/documents/2024-05/airtoxscreen_2020-tsd.pdf.

US EPA, 2025a. Integrated risk information System. Retrieved 10 July 2025 from. https://www.epa.gov/iris.

US EPA, 2025b. Toxic and priority pollutants under the clean water Act. Retrieved 10 July, 2025 from. https://www.epa.gov/eg/toxic-and-priority-pollutants-under-clean-water-act#priority.

US EPA (US Environmental Protection Agency), 2000. Dimethyl Phthalate. US EPA. Retrieved 9 May 2025 from. https://www.epa.gov/sites/default/files/2016-09/documents/dimethyl-phthalate.pdf.

Wang, X., Bi, C., Xu, Y., 2015. Modeling and analysis of sampling artifacts in measurements of gas-particle partitioning of semivolatile organic contaminants using filter-sorbent samplers. Atmos. Environ. 117, 99–109. https://doi.org/10.1016/j.atmosenv.2015.06.053.

Wang, J., Dong, Z., Li, X., Gao, M., Ho, S.S.H., Wang, G., Xiao, S., Cao, J., 2018. Intra-Urban levels, spatial variability, possible sources and health risks of PM2.5 bound phthalate esters in Xi'an. Aerosol Air Qual. Res. 18 (2), 485–496. https://doi.org/10.4209/aaqr.2017.09.0333.

Wang, Y., Ding, D., Shu, M., Wei, Z., Wang, T., Zhang, Q., Ji, X., Zhou, P., Dan, M., 2019. Characteristics of indoor and outdoor fine phthalates during different seasons and haze periods in Beijing. Aerosol Air Qual. Res. 19 (2), 364–374. https://doi.org/10.4209/aaqr.2018.03.0114.

Wang, W., Kannan, K., 2023. Leaching of phthalates from medical supplies and their implications for exposure. Environ. Sci. Technol. 57 (20), 7675–7683. https://doi.org/10.1021/acs.est.2c09182.

Weschler, C.J., Nazaroff, W.W., 2008. Semivolatile organic compounds in indoor environments. Atmos. Environ. 42 (40), 9018–9040. https://doi.org/10.1016/j.atmosenv.2008.09.052.

WHO, 2021. WHO global air quality guidelines: particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. Retrieved 9 May, 2025 from. https://www.who.int/publications/i/item/9789240034228.

WHO, 2024a. Ambient (outdoor) air pollution. Retrieved from 17 July, 2025 from. https://www.who.int/news-room/fact-sheets/detail/ambient-(outdoor)-air-quality-and-health.

WHO, 2024b. Health data overview for the United Kingdom of Great Britain and Northern Ireland. Retrieved 12 March, 2026 from. https://data.who.int/countries/826.

WHO, 2024c. Health data overview for the Federative Republic of Brazil. Retrieved 12 March, 2026 from. https://data.who.int/countries/076.

World Bank, 2026. Labor force, total - Brazil Data. from. https://data.worldbank.org/indicator/SL.TLF.TOTL.IN?locations=BR. (Accessed 12 March 2026).

Yang, Harris, S.A., Jantunen, L.M., Kvasnicka, J., Nguyen, L.V., Diamond, M.L., 2020. Phthalates: relationships between air, dust, electronic devices, and hands with implications for exposure. Environ. Sci. Technol. 54 (13), 8186–8197. https://doi.org/10.1021/acs.est.0c00229.

Yanosky, J.D., Maclntosh, D.L., 2001. A comparison of four gravimetric fine particle sampling methods. J. Air Waste Manag. Assoc. 51 (6), 878–884. https://doi.org/10.1080/10473289.2001.10464320.

Yue, S., Zheng, W., Fan, C., Wang, C., Zhao, Y., Yuan, Q., Zhao, M., 2024. Mechanistic insights into di-2-ethylhexyl phthalate (DEHP)-induced metabolic disruption: integrating gut hormone secretion and metabolomics in colonic organoids. Environ. Sci. Technol. Lett. 11 (9), 940–947. https://pubs.acs.org/doi/abs/10.1021/acs.estlett.4c00593.

Zhang, L., Wang, F., Ji, Y., Jiao, J., Zou, D., Liu, L., Shan, C., Bai, Z., Sun, Z., 2014. Phthalate esters (PAEs) in indoor PM10/PM2.5 and human exposure to PAEs via inhalation of indoor air in Tianjin, China. Atmos. Environ. 85, 139–146. https://doi.org/10.1016/j.atmosenv.2013.11.068.

Zhang, X., Wang, Q., Qiu, T., Tang, S., Li, J., Giesy, J.P., Zhu, Y., Hu, X., Xu, D., 2019. PM2.5 bound phthalates in four metropolitan cities of China: concentration, seasonal pattern and health risk via inhalation. Sci. Total Environ. 696, 133982. https://doi.org/10.1016/j.scitotenv.2019.133982.

Zheng, M., Fang, M., Wang, F., To, K.L., 2000. Characterization of the solvent extractable organic compounds in PM2.5 aerosols in Hong Kong. Atmos. Environ. 34 (17), 2691–2702. https://doi.org/10.1016/S1352-2310(99)00521-X.