

Indoor-Driven Urban–Rural InequalityInequalities in
MicroplasticTime-Weighted Exposure ExacerbatesHealth
Risks for Rural Residentsto PM_{2.5}-Bound Microplastics and
Plasticizers: A Case Study in Northernthe Guanzhong Plain,
China

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Abstract

Atmospheric microplastics (MPs) ~~and plasticizers~~ are emerging contaminants requiring systematic research on urban-rural exposure inequality urgently. This study examined MPs and plasticizers in PM_{2.5} across urban-rural ~~and indoor-outdoor~~ environments in Northern ~~China's~~ China's Guanzhong Plain by integrating indoor-outdoor time-activity weighted exposure. The 24 h time-weighted exposure concentrations for MPs and plasticizers were 3.6 and 6.8 times higher, respectively, in rural ~~areas~~ than in urban areas. Polyethylene terephthalate (PET) exhibited the greatest disparity in urban-rural MPs exposure. Plasticizer exposure was ~~overwhelmingly~~ dominated by phthalates, with di(2-ethylhexyl) phthalate (DEHP) reaching exceptionally high concentrations in rural indoor air ($\approx 600 \text{ ng m}^{-3}$), far exceeding urban levels (10.4 times). ~~Rural residents experienced consistently higher inhalation exposure to MPs and plasticizers, resulting in substantially elevated health risks, with non-carcinogenic and carcinogenic risks both 6.9 times higher than the urban populations. The volume-normalized oxidative potential (DTT_v) was significantly higher in rural than in urban environments (10.8 vs. 1.79 nmol min⁻¹ m⁻³) and strongly correlated with most MPs and plasticizer species ($r > 0.7$).~~ Source apportionment ~~revealed~~ indicated that contacting plastic products ~~accounted for contributed~~ 51.7% of ~~the~~ MPs and ~~plasticizers~~ plasticizer exposure in rural areas, nearly ~~double~~ twice the urban ~~value of contribution~~ (27.6%. ~~In contrast,~~ %), whereas transportation-related ~~sources~~ sources contributed only 5.99% in rural areas ~~but compared with~~ 22.6% in urban areas. ~~These results demonstrate clear urban-rural inequality in MPs~~ Rural residents had higher inhalation exposure and health risks, with non-carcinogenic and carcinogenic risks both 6.9 times higher than urban populations. PET, PE and PS induced reactive oxygen species (ROS) in A549 cells with potency exceeding that of several crustal metals in PM_{2.5}, with PET showing the highest oxidative stress potency, followed by PS and PE. The three polymers contributed 0.52% and plasticizers exposure 0.15% to PM_{2.5}-induced ROS in rural and related health effects urban samples, respectively. These findings reveal that indoor accumulation in

47 rural environments is a dominant driver of urban-rural inequalities in PM_{2.5}-bound MPs
48 and plasticizer exposure, highlighting the ~~need for~~importance of rural indoor exposure-
49 ~~based and equity-aware assessment frameworks~~ pathways and ~~interventions for air~~
50 source-specific mitigation strategies for emerging plastic-related air contaminants;
51 ~~especially for disadvantaged rural areas.~~

52
53 **Keywords:** Microplastics and plasticizers; Phthalates; Urban–rural difference;
54 Oxidative potential; Source analysis

1. Introduction

Systematic disparities exist between urban and rural areas in terms of population density, industrial structure, and lifestyle patterns, which profoundly shape their respective pollution emission characteristics and human exposure conditions (He et al., 2017; Song et al., 2024). Urban regions are typically characterized by higher population agglomeration, intensive industrial production, and transportation activities (Zhao et al., 2020). In contrast, rural areas often feature dispersed residential patterns, agriculture-dominated livelihoods, and relatively underdeveloped waste management infrastructure (Hart et al., 2005; Mihai et al., 2021). This spatial heterogeneity in socioeconomic conditions and environmental governance suggests that the sources, transport pathways, and exposure profiles of air pollutants may also exhibit significant spatial differentiation (Tomar et al., 2023).

As plastic production continues to increase and improper disposal becomes more common, contamination by microplastics (MPs) ~~and plasticizers~~ has substantially increased (Sharma et al., 2023). Atmospheric MPs typically refer to plastic particles in the air whose diameter is smaller than 5 mm (Arthur et al., 2009). Apart from the plastic daily necessities we encounter in our lives, MPs in urban areas may exhibit pollution patterns dominated by industrial and transportation sources (Järnskog et al., 2021; Qiu et al., 2020). While rural areas are more likely to experience MPs pollution from agricultural plastic products, such as greenhouse films and drip irrigation tapes, and improper waste management practices (Bonyadinejad et al., 2022). Previous studies have characterized atmospheric MP concentrations across different settings. Klein et al. (2023) reported higher deposition rates in urban than rural areas in northern Germany; D. Luo et al. (2024) found the highest MP concentrations in urban areas of the Qinghai-Tibet Plateau; and Liao et al. (2021) observed higher indoor than outdoor MP concentrations with urban levels exceeding those in rural areas. However, these studies lack a systematic evaluation of the differences in exposure to MPs between urban and rural populations.

Atmospheric MPs enter the human body primarily through respiration (Rahman et al., 2021). These particles reach the lungs and cause inflammation, which affects respiratory function (Amato-Lourenço et al., 2021; Saha & Saha, 2024). ~~They also enter the bloodstream~~MPs have been detected in human blood and ~~affect multiple organs, thereby posing a multifaceted range of health risks to humans (Saha & Saha, 2024).~~ Plasticizers are a class of chemicals used to enhance the properties of plastics, such as several tissues, including the lungs and placenta, suggesting their flexibility and malleability (Hahladakis et al., 2018), which are usually released into the atmosphere during the formation of MPs. These pollutants can enter the environment through various pathways, posing a potential threat to ecosystems for systemic translocation and human health (Samaei associated multiorgan health effects (Bastyans et al., 2022; Leslie et al., 2022; Nihart et al., 2025; Su Ragusa et al., 2022). Oxidative stress (2021). Reactive oxygen species (ROS) generation is regarded increasingly recognized as a key bridge pathway linking MPs exposure to cellular injury. Studies across multiple biological models have shown that links pollutant exposure to health effects (Lodovici & Bigagli, 2011; Zhou polypropylene (PP), polystyrene (PS), polyethylene terephthalate (PET), and polyethylene (PE) can elevate intracellular or mitochondrial ROS, leading to inflammatory signaling, lipid metabolic disruption, mitochondrial dysfunction, apoptosis, genotoxicity, and ferroptosis (Florance et al., 2022; K. Wang et al., 2023). Oxidative potential (OP) refers to the ability of a certain substance (such as particulate matter or MPs) to induce oxidative stress.; Ding et al., 2024; Lu et al., 2025; Wang et al., 2025; Wei et al., 2025). These studies indicate that diverse MPs can elicit ROS-mediated effects across multiple cell types and tissues. Therefore, it is critical to investigate scientific issues such as whether ~~MPs and the plasticizers can produce OP, and whether~~ there are significant differences in their effects in urban and rural environments. This can provide a systematic understanding of the health hazards and mechanisms of MPs ~~and the plasticizers~~.

To address these research gaps, by combining environmental measurements with

questionnaire-based time–activity data, we quantified inhalation personal exposure to MP and plasticizers and assessed its non-carcinogenic and carcinogenic risks in PM_{2.5} across typical urban and rural areas of the Guanzhong Plain, ~~northern~~Northern China. We also ~~evaluated the oxidation potential and its relationship with MPs and plasticizers to elucidate the exposure toxicity of these contaminants. Finally, a preliminary analysis was conducted to determine~~determined the source contribution and the main driving factor of the significant difference in exposure to indoor MPs and plasticizers. Finally, we evaluated ROS generation induced by representative MPs (PET, PE, and plasticizersPS) in A549 cells and estimated their contributions to PM_{2.5}-induced oxidative stress to characterize the potential toxicity of airborne MPs. This integrated framework provides new insights into urban–rural exposure inequality associated with airborne MPs and plasticizers and offers deeper understanding of the health hazards of emerging air pollutants.

2. Materials and ~~Methods~~methods

2.1 Sampling sites and PM_{2.5} sample collection

Indoor and outdoor atmospheric PM_{2.5} samples were collected from urban and rural areas in January 2024. The rural sampling site was Shibao Village, Yijun County, Tongchuan ~~City~~city, northern Guanzhong, Shaanxi Province, northwest China. The urban sampling site was Yanta District, Xi'an, the largest city in northwest China. The distance between these two sampling sites is 138.9 km. Fig. 1 depicts the locations of the two sampling sites and presents field photographs captured during the sampling period.

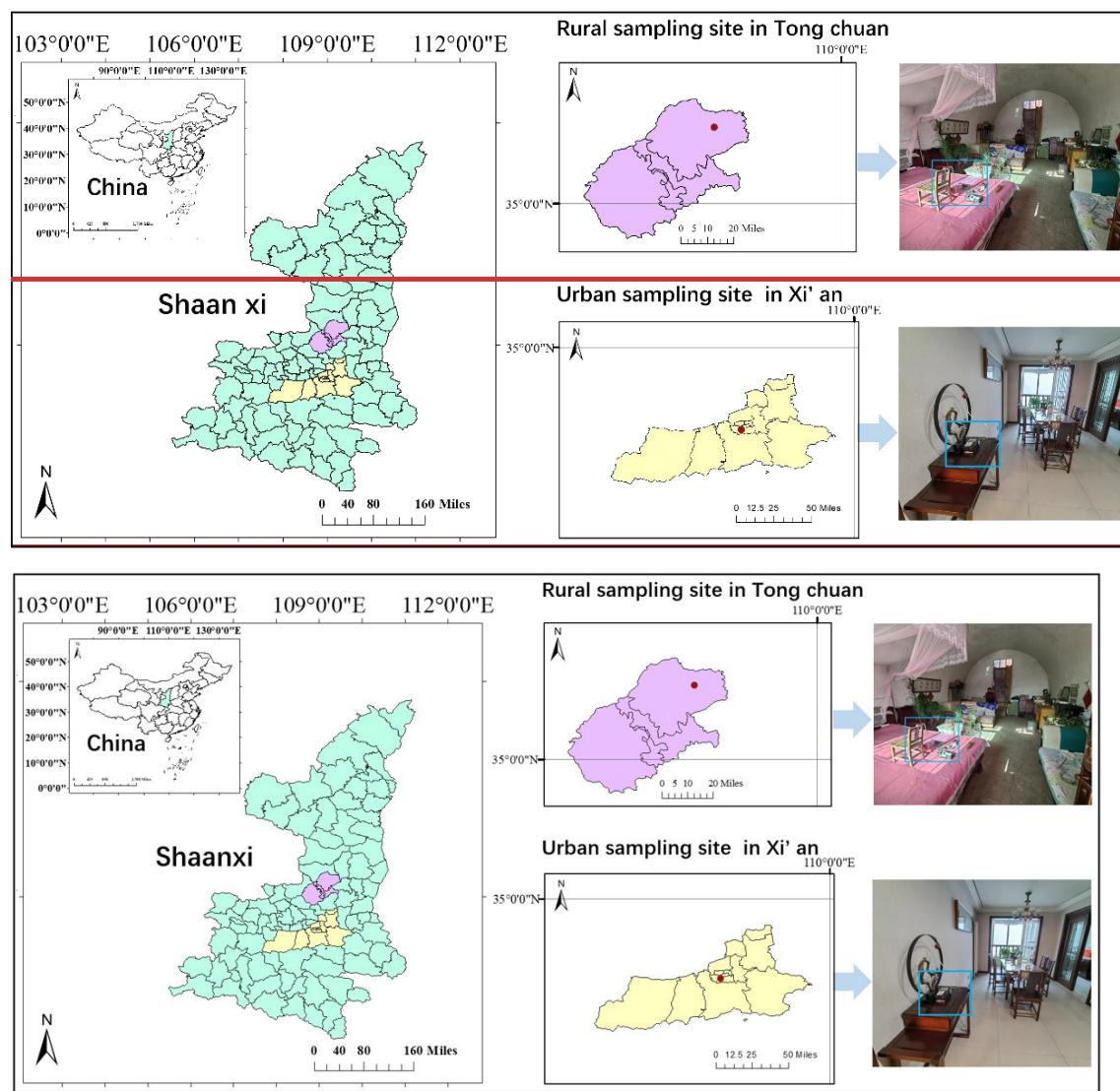


Fig. 1 Geographic locations and photographs of the urban and rural sampling sites.

PM_{2.5} samples were collected in indoor and outdoor settings by using an SKC sampler (PM_{2.5} model; SKC Inc., [USAUS](#)) equipped with a 10 L min⁻¹ flow pump (constant flow sampler; SKC) and a quartz filter membrane with 37 mm diameter and a pore size of 2.2 μm (Whatman, Buckinghamshire, UK). In cases of indoor sampling, the sampler was placed in the living room or bedroom and not in the kitchen to avoid interference from cooking fumes. Given that rural households are often large open-plan spaces combining sleeping, cooking, and living areas, efforts were made to place the sampler as far as possible from the cooking area. The indoor sampler was placed 1.5 m above the ground, corresponding to the average breathing height of humans. PM_{2.5}

147 samples were then continuously collected for 2 days in 10 rural households and 11
148 urban households, each over a 24 h period-, resulting in 19 rural indoor PM_{2.5} samples
149 and 22 urban indoor PM_{2.5} samples. Outdoor sampling was conducted in both areas on
150 3 separate days during the indoor sampling period for comparison with the indoor
151 samples. Rural sampling was conducted at the villagers' square in the village, whereas
152 urban sampling was conducted outdoors in residential areas. The outdoor sampling sites
153 were relatively open with no clear pollution sources. The outdoor sampler was similarly
154 placed approximately 1.5 m above the ground, with its filter membrane (with a diameter
155 of 47 mm) collected and replaced every 24 h. A total of three rural outdoor and three
156 urban outdoor PM_{2.5} samples were collected, for a total of 47 filter membranes.

157 **2.2 Questionnaire survey and weighted respiratory exposure calculation**

158 During the sampling period, we conducted a questionnaire survey for each adult
159 member of targeted household. These surveys captured daily routines of each
160 participant, including types of activities carried out indoors and outdoors, the time
161 required and frequency for these activities, frequency of plastic product use during
162 activities such as cooking, cleaning, shopping, and farming, window ventilation habits,
163 and laundry practices. Possibly due to the influence of the cold winter weather and the
164 similar age of the participants, the time spent outdoors and the frequency of such
165 activities were roughly the same, approximately 20.5 h in indoor settings and 3.5 h in
166 outdoor settings. The time-weighted calculations were conducted on the personal
167 exposure concentrations of MPs and plasticizers based on their concentrations in indoor
168 and outdoor microenvironments.

169 **2.3 Analysis of MPs and plasticizers in PM_{2.5}**

170 MPs were identified and quantified using pyrolysis gas chromatography mass
171 spectrometry (Py-GC/MS), a technique that thermally decomposes polymers into
172 characteristic marker compounds for unambiguous identification (Table S1) (Liu et al.,
173 2023). Filter samples were introduced into a Curie point pyrolyzer coupled online to a
174 GC/MS system, where rapid heating at 670°C generated diagnostic pyrolyzates that

were separated on a mid-polarity capillary column and detected in full scan mode. Quantification was based on calibration curves derived from authentic pyrolysis standards of each polymer.

Three classes of plasticizers, namely phthalate esters (PAEs), benzothiazoles (BTs), and bisphenol A (BPA), were quantified in PM_{2.5} samples. PAEs were analyzed by thermal desorption gas chromatography mass spectrometry following internal standard calibration with deuterated surrogates, avoiding solvent extraction and minimizing sample loss (Ho et al., 2019; Ho et al., 2008). BTs were extracted with an aqueous methanol mixture, purified through Oasis HLB solid phase cartridges, and quantified by ultrahigh performance liquid chromatography coupled to a triple quadrupole mass spectrometer operating in positive ion multiple reaction monitoring mode (Nuñez et al., 2020). BPA was determined following acidified methanol extraction and filtration, using high performance liquid chromatography with fluorescence detection at excitation and emission wavelengths of 275 and 313 nm, respectively (García-Prieto et al., 2008; Zhou et al., 2011). Detailed protocols, instrument parameters, quality assurance procedures, method detection limits and the detected components and their abbreviations are provided in our Supporting Information (Text S1 and Table S2). All analyses of MPs and plasticizers were subjected to duplicate testing every five samples to ensure data reliability, with a duplicate testing deviation of $\leq 20\%$.

2.4 Health effects assessment

2.4.2.4.1 Non-carcinogenic and carcinogenic risks

~~MPs and plasticizers inhalation exposure health risk were quantified using the United States Environmental Protection Agency's (US EPA) health risk assessment framework. Based on available toxicity parameters from the US EPA and previous studies (Esmailbeigi et al., 2023; Liu et al., 2023; Shi et al., 2019; K. Wang et al., 2025), nine substances were prioritized for risk screening: PS, PET, dimethyl phthalate (DMP), diethyl phthalate (DEP), butyl benzyl phthalate (BBP), di(2-ethylhexyl)~~

phthalate (DEHP), di-n-octyl-phthalate (DnOP), BPA, and BT. Noncarcinogenic risk was evaluated via the hazard quotient (HQ). Carcinogenic risk was assessed using the incremental lifetime cancer risk (ILCR). For specific calculation methods, see the Supplementary Information file (Text S3).

2.4.2 Determination of OP of PM_{2.5}

This study employed the dithiothreitol method to detect OP in PM_{2.5} samples (Y. Luo et al., 2024). Briefly, 4 mL of a sample extract obtained by ultrasonication was mixed with 1 mL of 1 mM DTT solution ($\geq 98\%$, Meryer; pH 7.4 buffer), resulting in a final concentration of 200 μM . At each time point (0, 5, 15, 30, 45, and 60 min), 0.5 mL of the DTT reaction mixture was added to amber centrifuge tubes preloaded with 0.5 mL of trichloroacetic acid (1%, w/v) to terminate the reaction. Subsequently, 25 μL of 10 mM 5,5'-dithiobis (2-nitrobenzoic acid) ($\geq 98\%$, Meryer) and 1 mL of 1 M Tris-HCl buffer solution were added to each centrifuge tube. After 200 μL of the solution was transferred to a 96-well plate, absorbance was measured at 412 nm using a microplate reader (FlexStation 3 Multi-Mode; Molecular Devices, San Jose, CA, USA). All procedures were conducted in the dark.

A preestablished standard curve was used to determine the concentration of DTT. The DTT consumption rate for each sample was calculated on the basis of absorbance measurements conducted at predetermined time points (unit: $\mu\text{M min}^{-1}$). According to the literature, the volume-normalized DTT consumption rate (DTT_v , $\text{nmol min}^{-1} \cdot \text{m}^{-3}$) characterizes overall OP under environmental exposure conditions, whereas the mass-normalized DTT consumption rate (DTT_m , $\text{pmol min}^{-1} \cdot \mu\text{g}^{-1}$) characterizes the intrinsic oxidative activity of PM_{2.5}. In this study, each sample was subjected to two replicate experiments (relative deviation below 5%), with the mean value taken as absorbance at that time point. At least one blank control was included in each experiment. A calibration curve was prepared using DTT solution at 15 concentration points (range: 1–750 $\mu\text{mol L}^{-1}$) for quantitative analysis, with linear regression coefficients (r^2) greater than 0.99.

2.5 Analysis of influencing factors and source apportionment

Indicators from the urban and rural household survey questionnaires were categorized based on sampling location and surrounding environment, daily living habits (use of air purification equipment, ventilation frequency, and cleaning practices), and exposure to plastic products. The frequency distribution of survey data was converted to calculate variable averages by using SPSS 26.0, yielding a comprehensive current status score of environment survey indicators (Table S3). Then further assessment of the main influencing factors that affect the concentrations and distributions of MPs and plasticizers in urban and rural areas in this study.

The U.S. Environmental Protection Agency (US EPA) Positive Matrix Factorization (PMF 5.0) model was conducted to explore the source of MPs and plasticizers in rural and urban PM_{2.5} indoor exposure sample (Norris et al., 2014). Eight MP components (PET, PP, PE, PS, Polymethylpolymethyl Methacrylate (PMMA), PVC, NR, SBR_BR) and five plasticizer components (2-hydroxybenzothiazole (HOBT), 2-(4-morpholinyl) benzothiazole (24MoBT), DEHP, DNOP, BPA) were used as source tracers, outputting to the model. The PMF model simplifies complex data with numerous variables into source types and species combinations representing source contributions. Based on preliminary observations and surveys of indoor environments during sampling, ~~supplemented by literature review, four source factors were identified in both rural and urban environments in this study. The reliability of the PMF method used in this study is detailed in Text S2 and Table S4 of the supporting information~~ three- to five-factor solutions were evaluated for both the rural and urban datasets. The number of factors was selected by jointly considering Q(Robust)/Q(True), scaled residual distributions, signal-to-noise ratios, agreement between observed and predicted concentrations, factor interpretability, and Bootstrap reproducibility. The four-factor solution provided the best overall balance between goodness of fit and factor stability. Bootstrap analysis was conducted for 100 runs, and the detailed diagnostic results are provided in Text S2 and Table S4 of the Supporting Information.

2.5 Health effects assessment

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3. Results and Discussion

2.5.2 Intracellular ROS assay of MP standards and PM_{2.5} extracts

Human A549 lung epithelial cells were used to evaluate intracellular ROS formation induced by PET, PE, PS, and PM_{2.5} extracts. PET and PE standards had nominal particle diameters of 1 μm , whereas the PS standard had a nominal diameter of 2.5 μm . The polymer standards were purchased from Jiangsu Zhongke New Materials Co., Ltd., China. The selection of PET, PE, and PS was primarily based on their environmental representativeness and toxicological relevance. All three were the main polymers detected in the PM_{2.5} samples in this study, with PET dominating the indoor samples and exhibiting the most significant urban-rural exposure differences; PS and PE also showed high detection levels. Furthermore, these three materials represent different chain structures: PET with an ester-based backbone, PE with a fully aliphatic chain, and PS with aromatic side chains. These structures can preliminarily reflect the differences in ROS induction among different polymer structures in a limited experimental system. Therefore, this study used them as representative microplastics for screening cellular ROS experiments.

A549 cells were cultured in Ham's F-12 medium supplemented with 10% heat-inactivated fetal bovine serum and 1% penicillin–streptomycin at 37 °C in a humidified 5% CO₂ atmosphere. The three polymer standards dispersed in culture medium by ultrasonication for 20 min followed by vortexing for 20 s immediately before cell exposure. Indoor PM_{2.5} filter punches collected from rural and urban households were pooled separately to generate representative samples for cellular experiments, followed by methanol extraction using ultrasonication (20 min × 3), filtration, evaporation under nitrogen, reconstitution in DMSO, and dilution to the desired concentrations. Cells were seeded in 96-well plates at a density of 8,000–10,000 cells well⁻¹ and cultured for 8 h before exposure. PET, PE, PS, rural PM_{2.5} extracts, urban PM_{2.5} extracts, and the reference compound tert-butylhydroquinone (t-BHQ) were serially diluted in cell culture medium. After 12 h of exposure, intracellular ROS generation was determined using the DCFH-DA assay.

In this study, a CCK-8 kit (Jiangsu Kaiji Biotechnology, China) was used to assess cell viability. The viability of all cells exceeded 80% after 24 h of drug exposure. ROS induction was expressed as the fold change relative to the negative control. Linear concentration–response models were fitted over the approximately linear portion of the response. The concentration producing a 1.5-fold increase in intracellular ROS relative to the control was defined as the EC_{1.5}. Lower EC_{1.5} values indicate greater ROS-inducing potency. All cell experiments were performed with six replicate wells.

2.5.3 CA-based estimation of MP contributions to PM_{2.5}-induced ROS

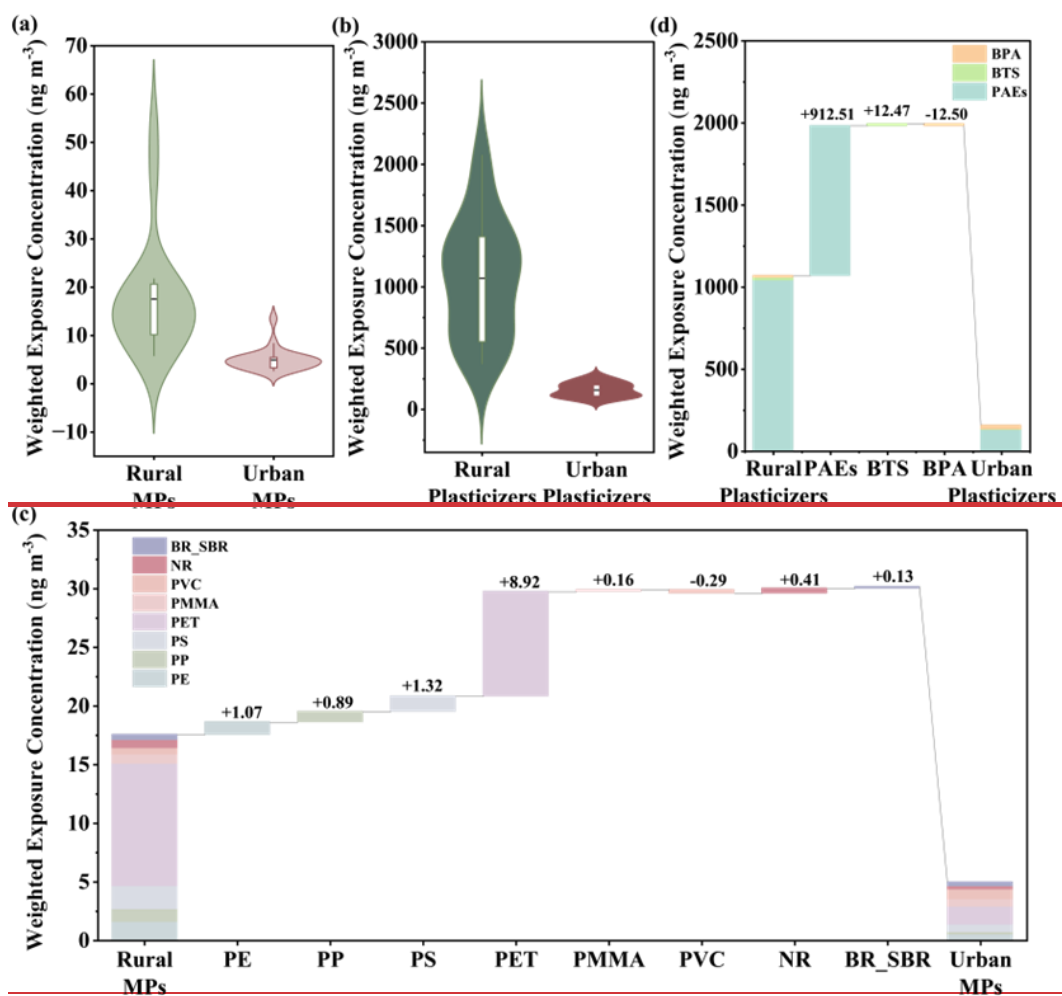
A concentration-addition-based (CA) equivalent approach was applied to estimate the contributions of PET, PP, and PS to the intracellular ROS response induced by PM_{2.5}. t-BHQ was used as the reference compound. For the specific calculation method, refer to the study by Jin et al. (Jin et al., 2019). The resulting contributions were interpreted as screening-level estimates based on the concentration-addition assumption.

3. Results and discussion

3.1 Urban–Rural Disparities in MPs and Plasticizers Exposure

A pronounced inequality in 24 h time-weighted personal exposure to MPs and plasticizers is evident between urban and rural settings (Fig. 2). For MPs, the exposure burden falls disproportionately on rural residents (Fig. 2a). The distribution of rural exposure is markedly broader and higher compared to the urban profile, with the mean concentration in rural areas (17.5 ng m^{-3}) being 3.6 times higher than in urban areas (4.94 ng m^{-3}). This ~~inequity~~equity is even more severe for plasticizers (Fig. 2b). Rural populations experience a substantially greater and more concentrated exposure burden, as shown by the dominant green violin plot, with a mean concentration (1070 ng m^{-3}) that is 6.8 times the urban mean (158 ng m^{-3}).

Analysis by contaminant type reveals a complex but consistent pattern of inequality (Fig. 2c & 2d). Rural areas showed higher time-weighted exposure than urban areas for seven microplastic types, excluding PVC. The urban-rural exposure disparity was greatest for PET. Rural populations endure significantly greater exposure to specific high-concern substances: PAEs and BTs are higher by $\pm 912 \text{ ng m}^{-3}$ and $\pm 12.5 \text{ ng m}^{-3}$, respectively. While BPA exposure is moderately lower in rural areas ($\pm 12.5 \text{ ng m}^{-3}$), this is overshadowed by the vast excess of PAEs. For MPs, rural inequality is driven by notably higher exposure to polymers like polyethylene terephthalate (PET, $\pm 8.92 \text{ ng m}^{-3}$). These stark disparities point to fundamentally different emission sources and exposure pathways. ~~The elevated rural burden is likely tied to agricultural practices, localized industry, and distinct waste management, whereas urban exposures stem from traffic, construction, and consumer product emissions. This evidence underscores a clear environmental inequality, where rural residents face a significantly higher aggregate burden of emerging contaminant exposure.~~ These stark urban-rural disparities in exposure suggest divergent source contributions and accumulation dynamics between the two settings. The underlying drivers—including indoor emission intensity, retention processes, and ventilation conditions—are further explored in Section 3.6.



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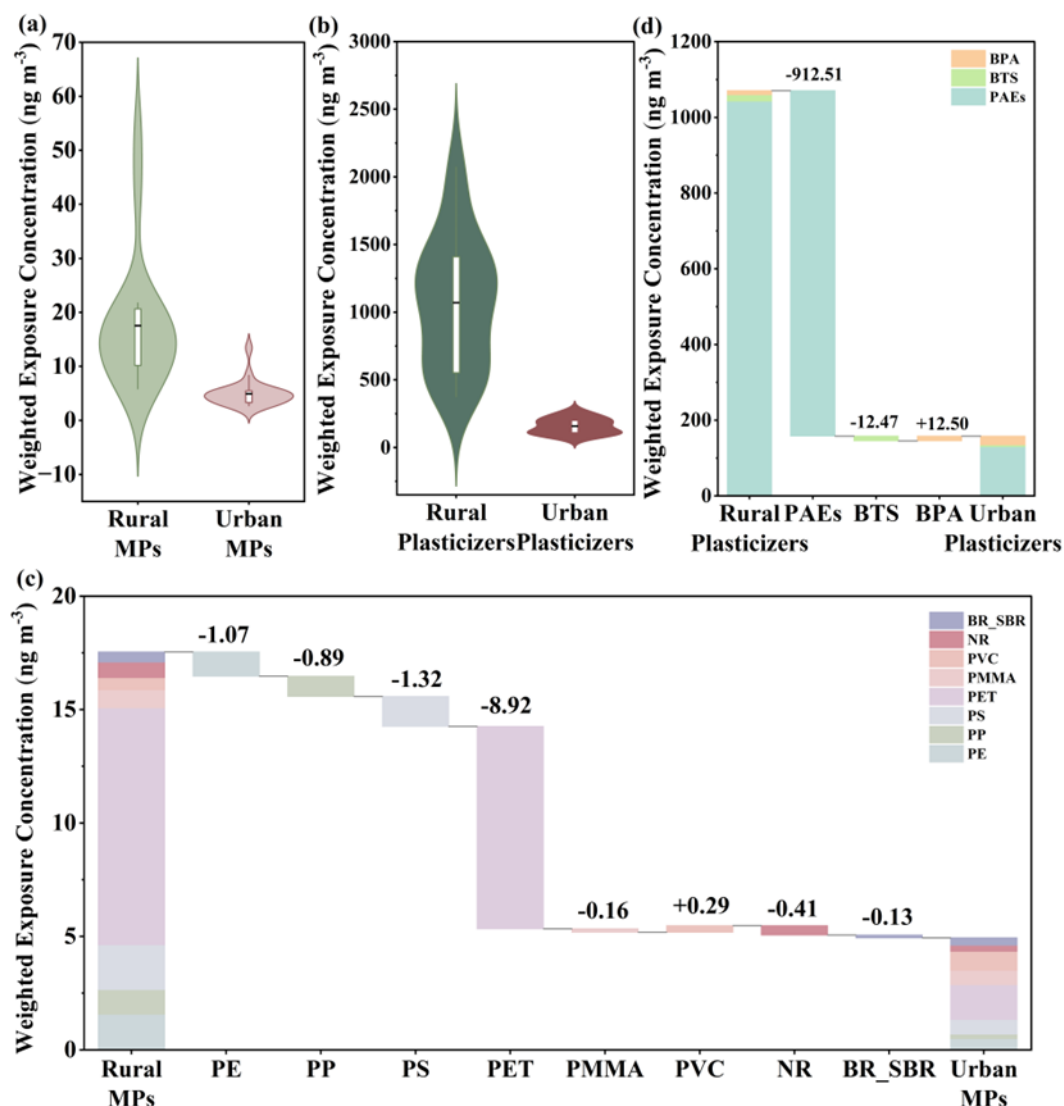
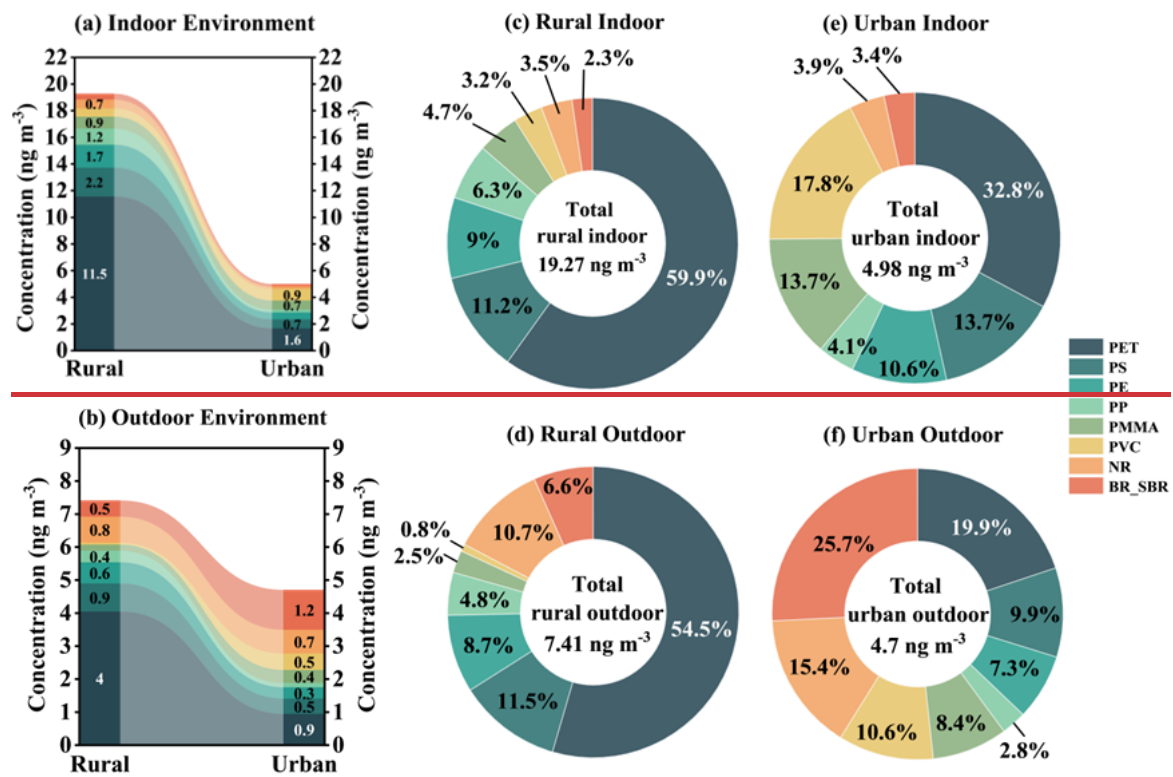


Fig. 2 Urban-rural inequality in time-weighted personal exposure to (a) total MPs, (b) total plasticizers, (c) eight types of MPs, and (d) three types of plasticizers. (In Fig. 2c and 2d, rural values are used as the reference; negative values indicate that weighted exposure concentration in rural areas is higher than in urban areas).

3.2 Chemical composition differences of MPs

As shown in Fig. 3, PET had the highest mass concentration among all eight MPs. MPs exposure was consistently higher in rural versus urban settings (Fig. 3a and 3b). The disparity was most pronounced indoors, where rural concentrations (19.3 ng m⁻³) exceeded urban levels (4.98 ng m⁻³) by a factor of 3.9. In contrast, outdoor concentrations were lower and the disparity reduced, with rural areas (7.41 ng m⁻³) showing only 1.6 times the exposure of urban areas (4.70 ng m⁻³). With the exception

of urban outdoor environments, PET ranked first in terms of mass concentration in the other three sampling environments, ranging from 32.8% in urban indoor environments to 59.9% in rural indoor environments. A study conducted in Sri Lanka reported that PET was the dominant MP in both indoor and outdoor environments (Perera et al., 2022). In the present study, PET, PS, PVC, and PE were the primary MPs detected in urban and rural indoor environments, accounting for 46.4%, 12.5%, 10.5%, and 9.8%, respectively, of the total mass concentrations of MPs (Figs. 3c and 3e). Vehicle-tyre wear markedly influenced the relative abundances of NR and SBR (Rauert et al., 2021). When averaged across urban and rural sites, NR rose from 3.7% indoors to 13.1% outdoors, while SBR increased from 2.9% indoors to 16.2% outdoors. In urban outdoor environments, the proportions of NR and SBR reached 15.4% and 25.7%, respectively, making them the most abundant MPs in these environments. This underscores the importance of closely monitoring and controlling the concentrations of these MPs in urban outdoor environments.



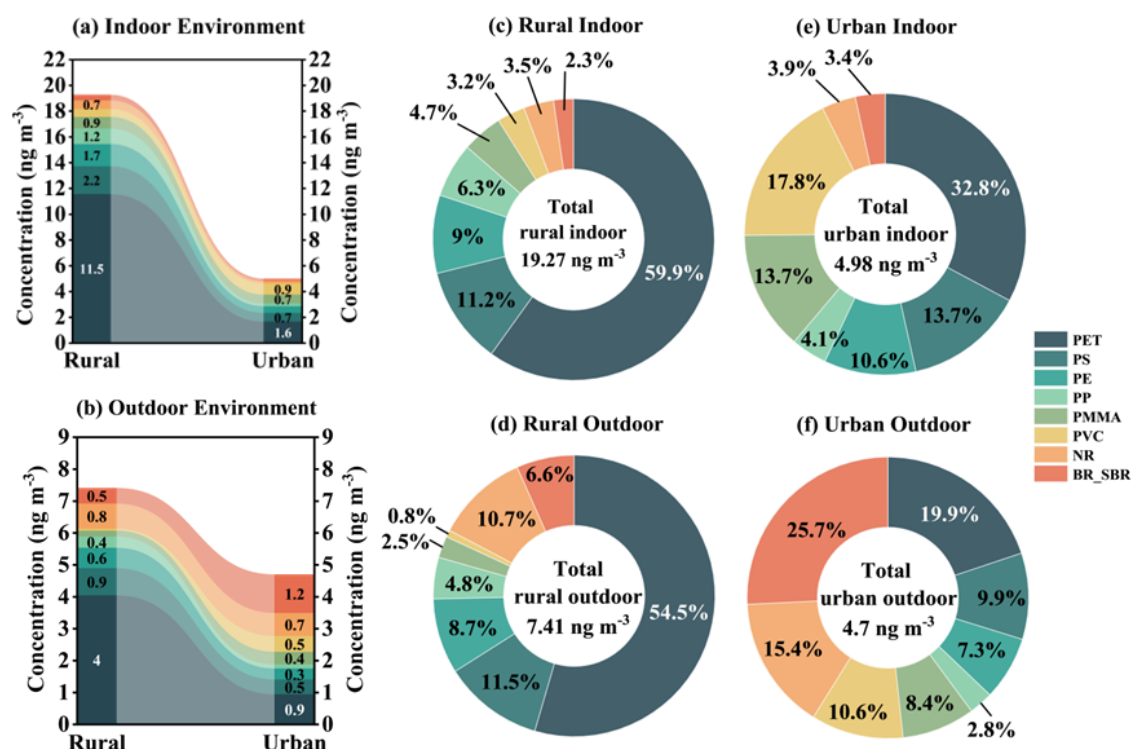


Fig. 3 (a-b) MPs concentration comparison between rural and urban environments; (c-f) the proportions of MPs in different environments.

3.3 Characteristics of plasticizers

In this study, we examined three types of plasticizers: PAEs (six species), BTs (nine species), and BPA. Overall, PAEs had the highest mass concentration among the plasticizers, with an average mass concentration of 457.0 ng m⁻³ (Fig. 4). The average mass concentrations of BPA and BTs were both less than 30 ng m⁻³. In indoor environments, both PAEs and BTs exhibit higher concentrations in rural areas than in urban areas. In outdoor environments, the concentration of PAEs in cities is slightly higher than that in rural areas, while the concentration of BTs in cities is approximately twice that in rural areas. Unlike the completely opposite concentration characteristics of PAEs and BTs between urban and rural areas in indoor environments, the concentration of BPA is consistently higher in cities than in rural areas in both indoor and outdoor environments.

For PAEs, it was noteworthy that the mass concentrations of DNOP and DEHP were extremely high. Especially in rural indoor environments, the concentration of DEHP was close to 600 ng m⁻³, while that of DNOP was approximately 450 ng m⁻³. The average mass concentrations of the remaining four components—dibutyl phthalate

(24.9 ng m⁻³), BBP (18.1 ng m⁻³), DMP (6.17 ng m⁻³), and DEP (5.58 ng m⁻³)—were all relatively low, with none of them exceeding 30 ng m⁻³. In all sampling environments, the mass concentrations of these six PAEs exhibited uniform spatial variation patterns, with the highest concentrations observed in rural indoor environments and the lowest concentrations observed in urban indoor environments. These results indicate that rural residents are exposed to higher levels of PAEs pollution.

BTs were present at relatively low mass concentrations, with individual levels remaining below 6 ng m⁻³, representing the lowest concentrations among the three classes of plasticizers analyzed. Within this group, HOBT, BT, 2-benzothiazolyl-N-morpholinyl sulfide (OBS), and 24MoBT showed relatively higher mass concentrations in rural indoor and urban outdoor environments.

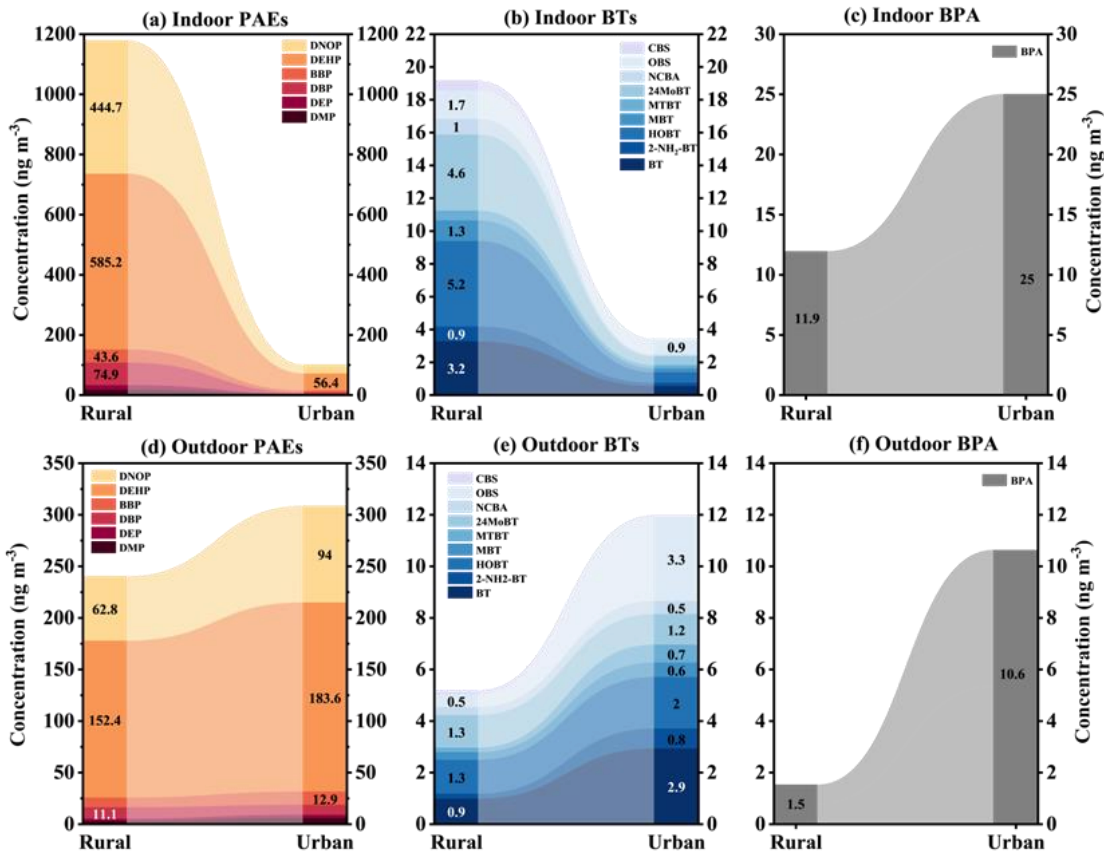


Fig. 4 Mass concentrations of plasticizers (a and d: PAEs; b and e: BTs; c and f: BPA) in indoors and outdoors of rural-urban environments.

3.4 Health effects of MPs and plasticizers in PM_{2.5}

3.4.1 Noncarcinogenic and carcinogenic risks

As shown in Table 1, except for BPA, the ADDs of 24 h time-weighted personal

exposure to PS, PET, DMP, DEP, BBP, DEHP, and DnOP in rural areas were always higher than those in urban areas. **3.4** Specifically, the ADD of DEHP was $1.43 \times 10^{-4} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $2.05 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, indicating significantly higher exposure in rural areas. In addition, the ADD of BBP was $1.06 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $2.08 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, underseoring substantially greater rural exposure levels. Moreover, the ADD of DnOP was $1.07 \times 10^{-4} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $1.07 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, pointing to a markedly elevated rural exposure burden. In contrast to these results, the ADD of BPA was lower in rural areas ($2.85 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$) than in urban areas ($6.28 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$). Taken together, these findings indicate differences in exposure levels to MPs and plasticizers between urban and rural areas, with rural areas carrying a higher risk of exposure.

As shown in Table 1, the HQ in urban and rural areas was between 6.60×10^{-8} and 2.66×10^{-3} . In addition, the HI calculated in rural areas (9.99×10^{-3}) was approximately 6.9 times higher than that in urban areas (1.44×10^{-3}). Although the noncarcinogenic risks of all MPs were below 1.0, DEHP (rural settings: 71.6%, urban settings: 71.1%) and DnOP (rural settings: 26.7%, urban settings: 18.6%) significantly contributed to the calculated HI, and their health risks remain a concern, particularly in rural indoor environments.

Table 1 Noncarcinogenic risk of time-weighted respiratory exposure to MPs and plasticizers in urban and rural environments.

	RfD ($\text{mg kg}^{-1} \text{ d}^{-1}$)	Rural			Urban		
		ADD ($\text{mg kg}^{-1} \text{ d}^{-1}$)	HQ	HQ/TH (%)	ADD ($\text{mg kg}^{-1} \text{ d}^{-1}$)	HQ	HQ/TH (%)
PS	0.02	5.40×10^{-7}	2.70×10^{-5}	0.27	1.78×10^{-7}	8.90×10^{-6}	0.62
PET	0.1	2.86×10^{-6}	2.86×10^{-5}	0.29	4.20×10^{-7}	4.20×10^{-6}	0.29
DMP	10	3.80×10^{-6}	3.80×10^{-7}	0.00	6.60×10^{-7}	6.60×10^{-8}	0.00
DEP	0.8	3.76×10^{-6}	4.70×10^{-6}	0.05	3.91×10^{-7}	4.88×10^{-7}	0.03
BBP	0.2	1.06×10^{-5}	5.28×10^{-5}	0.53	2.08×10^{-6}	1.01×10^{-5}	0.72

DEHP	0.02	1.43×10^{-4}	7.15×10^{-3}	71.61	2.05×10^{-5}	1.03×10^{-3}	71.05
BnOP	0.04	1.07×10^{-4}	2.66×10^{-3}	26.68	1.07×10^{-5}	2.68×10^{-4}	18.59
BPA	0.05	2.85×10^{-6}	5.71×10^{-5}	0.57	6.28×10^{-6}	1.26×10^{-4}	8.69
HI	↓	↓	9.99×10^{-3}	↓	↓	1.44×10^{-3}	↓

As shown in Table 2, DEHP accounted for more than 99% of the total carcinogenic risk (Σ ILCR) in both rural and urban environments, with BT and BBP making relatively low contributions to such risk. The Σ ILCR value for the rural samples was calculated as 2.00×10^{-6} , which exceeds the internationally recognized risk threshold (1.0×10^{-6}), indicating that MPs and plasticizers in rural environments pose a clear carcinogenic risk through respiratory exposure pathways, which warrants immediate attention. By contrast, the Σ ILCR value for the urban samples was calculated as 2.88×10^{-7} , indicating an acceptable degree of risk. The carcinogenic risk caused by individual exposure to MPs and plasticizers in $PM_{2.5}$ in rural areas was about 7 times higher than that in urban areas. These results also suggest that DEHP is the primary driver of carcinogenic risk for MPs and plasticizers. Further research is required to examine the specific exposure pathways and pollution sources of DEHP.

Table 2 Carcinogenic risk of time-weighted respiratory exposure to MPs and plasticizers in urban and rural environments.

		EC ($\mu\text{g}\cdot\text{m}^{-3}$)	IUR ($\text{m}^3\cdot\mu\text{g}^{-1}$)	SF ($\text{kg}\cdot\text{d}\cdot\text{mg}^{-1}$)	ILCR	ILCR/ Σ ILCR (%)
Rural	BT	3.372×10^{-3}	1.8×10^{-7}	↓	6.07×10^{-10}	0.03
	BBP	↓	↓	0.00019	2.01×10^{-9}	0.10
	DEHP	↓	↓	0.014	2.00×10^{-6}	99.87
	Σ ILCR	↓	↓	↓	2.00×10^{-6}	↓
Urban	BT	1.001×10^{-3}	1.8×10^{-7}		1.80×10^{-10}	0.06
	BBP	↓	↓	0.00019	3.96×10^{-10}	0.14
	DEHP	↓	↓	0.014	2.87×10^{-7}	99.80
	Σ ILCR	↓	↓	↓	2.88×10^{-7}	↓

3.4.2 Effects of MPs and plasticizers on OP in $PM_{2.5}$

DTT_v of $PM_{2.5}$ in rural areas showed that the mean and median values were 10.8

(± 12.6) and $4.86 \text{ nmol min}^{-1} \text{ m}^{-3}$, respectively, ranging between 1.19 and $37.2 \text{ nmol min}^{-1} \text{ m}^{-3}$, which were significantly higher than the mean ($1.79 \pm 1.47 \text{ nmol min}^{-1} \text{ m}^{-3}$) and median ($1.31 \text{ nmol min}^{-1} \text{ m}^{-3}$) values measured in urban areas (Fig. 5a). Previous research demonstrated that DTT_v exhibits significant spatial heterogeneity across the southeastern United States, with elevated levels in urban and roadside zones linked to cardiovascular and respiratory diseases (Fang et al., 2016; Fang et al., 2015). The substantially higher DTT_v observed in rural areas of this study suggests that rural populations may face comparable or greater health risks from oxidative stress induced by airborne pollutants. The mean DTT_m value was still higher in rural areas ($34.7 \pm 27.9 \text{ pmol min}^{-1} \mu\text{g}^{-1}$) than in urban areas ($27.8 \pm 20.4 \text{ pmol min}^{-1} \mu\text{g}^{-1}$), although the median values of the two areas were somewhat close (41.72 vs. $20.23 \text{ pmol min}^{-1} \mu\text{g}^{-1}$) (Fig. 5b), indicating that MPs and plasticizers in rural settings exhibited more stable oxidative activity and quality-related toxicity. DTT_m characterizes the inherent oxidizing capacity of chemically active species in particulate matter and is governed by chemical composition (particularly structural features such as peroxide content in organic compounds) rather than by concentration or size distribution, as evidenced by strong correlations between DTT_m and specific molecular functionalities (Jiang et al., 2019; Wang et al., 2018). MPs and plasticizers exhibit higher levels and greater variability in terms of environmental exposure OP (DTT_v) and intrinsic OP (DTT_m) in rural areas than in urban areas, which may be attributable to differences in pollution sources and particle composition between urban and rural areas. This once again demonstrates the difference in toxicity risk caused by exposure to PM_{2.5} between urban and rural areas.

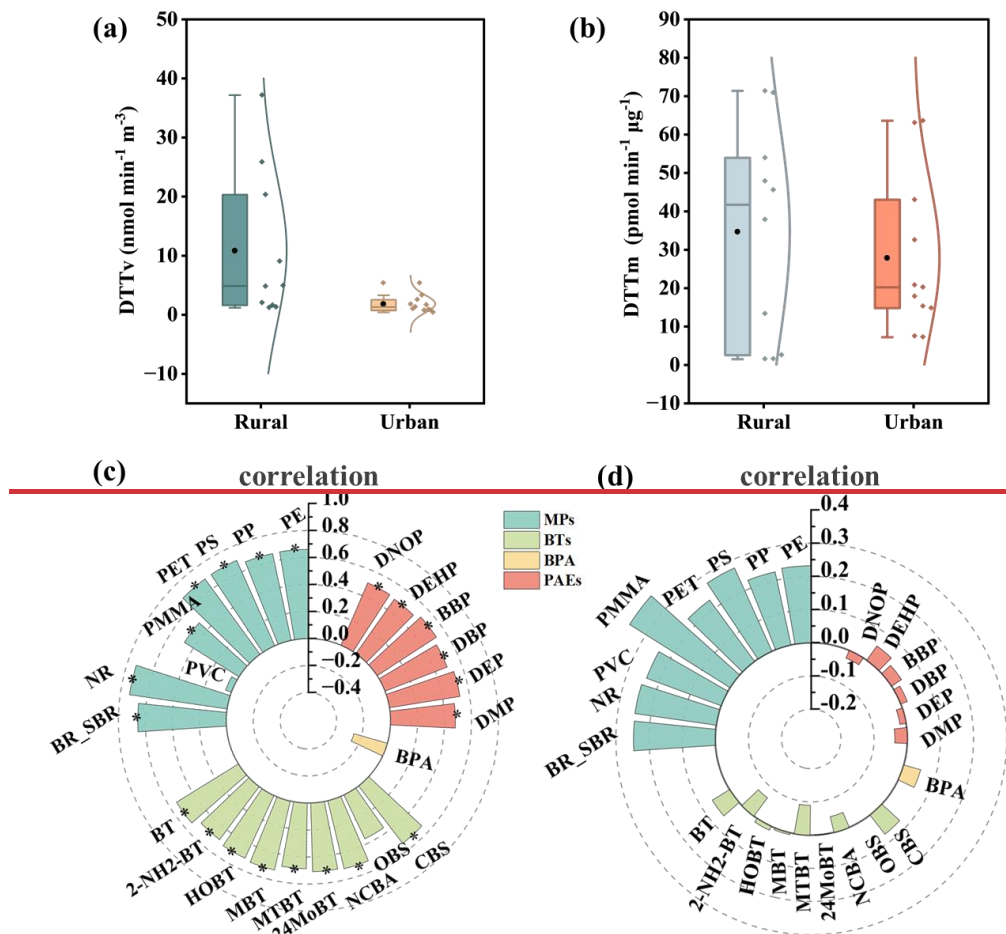


Fig. 5 Comparison of (a) DTT_v and (b) DTT_m of 24 h time-weighted urban and rural PM_{2.5}. Correlation between (c) DTT_v, (d) DTT_m and MPs/plasticizers in urban and rural environments (*P < 0.05).

Fig. 5c and 5d also depicted the Pearson correlations of DTT_v and DTT_m with MPs and plasticizers. In this study, most MPs exhibited a significant positive correlation with DTT_v ($r > 0.65$), especially NR ($r > 0.7$), followed by PS and PET ($r > 0.69$). Additionally, all MPs showed a weak positive correlation with DTT_m ($0.2 < r < 0.4$). Wei et al. (2025) reported that MPs induced the production of mitochondrial reactive oxygen species (ROS), which triggered autophagy dependent ferroptosis, thereby exacerbating inflammatory responses in chronic obstructive pulmonary disease. Jeon et al. (2023) examined PP and PS as MPs and reported that the pathogenic factor in THP-1 macrophages was the intrinsic ROS-generating potential of MPs. Florance et al. (2022) discovered that sulfated PS nanoparticles induced mitochondrial oxidative stress,

leading to lipid metabolism disorders and oxidative damage in human macrophages. Ding et al. (2024) indicated that PS particles primarily accumulated in rat gastric tissue, triggering oxidative stress, mitochondrial damage, and genotoxicity. Lu et al. (2025) reported that PET induced oxidative stress, lipid accumulation, and apoptosis in hepatocytes, leading to structural damage and functional abnormalities in the liver. Wang et al. (2025) discovered that high levels of exposure to PE resulted in the upregulation of proinflammatory genes in follicular fluid and increased ROS in oocytes, ultimately affecting oocyte quality.

As shown in Fig. 5c, most of the BTs exhibited strong positive correlations with DTT_v , with correlation coefficients approaching 1.0. These results indicated that these substances efficiently drove DTT consumption, which in turn promoted OP generation. By contrast, PAEs exhibited a weak positive correlation with DTT_v , and BPA exhibited a negative correlation with DTT_v , suggesting their weak contribution to the OP of particles mediated by DTT_v . This inconsistency with the noncarcinogenic and carcinogenic effects of PAEs described in Section 3.5.1 indicates that PAEs exhibit diverse toxic mechanisms, with their health impacts potentially mediated through endocrine disruption, genotoxicity, or oxidative stress (Wang et al., 2023). Environmental media, exposure scenarios, limitations of a single assessment method all influence the expression of toxicity. Therefore, future evaluations of the health risks of MPs and plasticizers should integrate multidimensional approaches, optimize methods for specific scenarios, and construct a multilevel evidence chain to enhance accuracy. As shown in Fig. 5d, the correlation coefficients between plasticizers and DTT_m significantly decreased, with the radial range decreasing to -0.1 to 0.1 . This phenomenon underscores the limitations of DTT_m in characterizing the intrinsic activity of OP. In summary, DTT_v sensitively captures the environmental OP risks of MPs and BTs, whereas DTT_m lacks sensitivity in intrinsic activity analysis. The differences between these two indicators demonstrate the complementarity of the environmental exposure and intrinsic activity perspectives in OP evaluation, providing a reference for

~~the multidimensional analysis of OP induced by MPs and plasticizers in PM_{2.5}.~~

3.5 Influencing factors and sources of MPs and plasticizers

Substantial disparities in the dominant factors influencing MPs and plasticizers were observed between rural and urban households in this study (Table S3). Among rural households, contact with plastic products exhibited the highest composite score (mean: 1.80), followed by sampling location and surrounding environment (1.53), and daily living habits (1.50). In urban households, in contrast, daily living habits ranked first (1.73), followed by contact with plastic products (1.54), while sampling location and surrounding environment contributed the least (1.45). These survey-based findings suggested that MPs and plasticizers in rural areas were more strongly associated with direct plastic usage and external environmental factors, whereas MPs and plasticizers in urban areas were more closely linked to lifestyle-driven behaviors, such as using more personal cleaning and care products, beauty products, printed materials, etc.

To further explore the differences in exposure to MPs and plasticizers between rural and urban areas, source apportionment analysis was conducted using PMF models, and almost identical four contributing factors were identified, as shown in Fig. ~~6-5~~. Although the four-factor PMF solution showed generally satisfactory diagnostic performance and Bootstrap reproducibility, the source results should be interpreted at the level of broad source patterns rather than as uniquely resolved emission sources. Seven of the eight factors had Bootstrap mapping rates above 80%, while rural factor 3 had a mapping rate of 76%, indicating greater uncertainty in this factor. In addition, several polymers and additives may be shared among different consumer and household sources.

Factor 1, characterized by high and balanced loadings across multiple polymers (e.g., PE, PP, PS) and plasticizer species (e.g., HOBt, DEHP), was attributed to the general impact of contacting plastic products- (Liao et al., 2021). Factor 2 exhibited pronounced contributions from NR and BR_SBR, markers of tire wear, and was assigned to transportation-related sources- (Miller et al., 2022; Panko et al., 2019; Pan

et al., 2012). Factor 3 displayed elevated loadings of PVC and PMMA, polymers commonly used in construction and interior finishing materials, (Li et al., 2021), and thus represented home decoration sources. Factor 4 (Others) represented residual sources where plasticizer concentrations substantially exceeded those of microplastic components, reflecting the release of plasticizers from non-plastic consumer products such as recycled paper, coated containers, paints, inks, personal care products, thermal paper, and industrial additives- (Loganathan & Kannan, 2011).

Quantitative source contributions further underscored urban–rural heterogeneity. In rural areas, plastic products (factor 1) accounted for 51.7% of the total source contribution, nearly double the urban value of 27.6%, establishing this category as the predominant source in rural environments. This divergence likely reflects higher intensity of plastic use in rural areas, where plastic items may undergo more frequent reuse, accelerating wear and tear and consequently releasing greater amounts of MPs (Licciardello, 2024). It may also stem from lower waste management standards in rural environments and the limited availability of plastic alternatives. Conversely, transportation (factor 2) contributed only 5.9% in rural areas but 22.6% in urban samples, directly implicating tire wear particles and rubber additives as major urban sources, consistent with dense traffic networks and high vehicle ownership in urban areas. The questionnaire survey revealed that no target households in rural or urban areas had engaged in renovation activities within the past three years. Home decoration materials (factor 3) contributed modestly in both settings, with 4.2% in rural and 9.1% in urban areas; the slightly higher urban share may be attributable to greater density of synthetic building materials. Others (factor 4) accounted for substantial proportions in both rural and urban areas, at 38.2% and 40.7% respectively, with a marginally higher urban value that may reflect more intensive consumption of processed goods, cosmetics, and printed materials for urban residents. Critically, this reveals that plasticizers enter the environment independently of plastics, complicating their source attribution and mitigation.

Collectively, these results delineate fundamentally distinct contamination regimes in rural and urban indoor environments. Rural MPs and plasticizers pollution is dominated by an externalized product-oriented pathway, driven by reliance on single-use plastics and inadequate waste infrastructure, resulting in a use-end-dominated pattern characterized by direct consumer plastic inputs. Urban MPs and plasticizers pollution, by contrast, exhibits a multi-source composite regime shaped by the interplay of lifestyle-driven consumption, infrastructure-related emissions notably from tire wear, and dynamic releases from the built environment including aging PVC and PMMA from construction materials. This pronounced structural heterogeneity in source apportionment provides a robust scientific foundation for spatially differentiated and precisely targeted mitigation strategies of MPs and plasticizers.

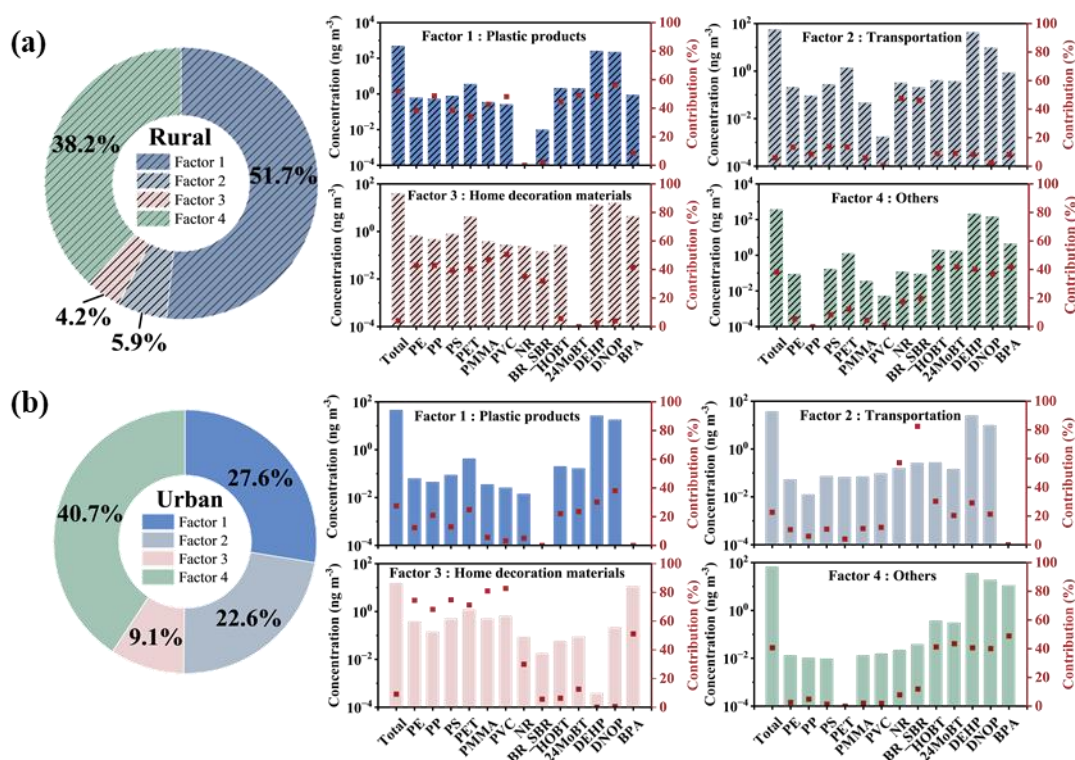


Fig. 65 Source contribution and profiles for MPs and plasticizers in PM_{2.5} in rural and urban areas based on the PMF model.

3.5 Health effects of MPs and plasticizers in PM_{2.5}

3.5.1 Noncarcinogenic and carcinogenic risks

As shown in Table 1, except for BPA, the ADDs of 24 h time-weighted personal

exposure to DMP, DEP, BBP, DEHP, and DNOP in rural areas were always higher than those in urban areas. Specifically, the ADD of DEHP was $1.43 \times 10^{-4} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $2.05 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, indicating significantly higher exposure in rural areas. In addition, the ADD of BBP was $1.06 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $2.08 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, underscoring substantially greater rural exposure levels. Moreover, the ADD of DNOP was $1.07 \times 10^{-4} \text{ mg kg}^{-1} \text{ d}^{-1}$ in rural areas and $1.07 \times 10^{-5} \text{ mg kg}^{-1} \text{ d}^{-1}$ in urban areas, pointing to a markedly elevated rural exposure burden. In contrast to these results, the ADD of BPA was lower in rural areas ($2.85 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$) than in urban areas ($6.28 \times 10^{-6} \text{ mg kg}^{-1} \text{ d}^{-1}$). Taken together, these findings indicate differences in exposure levels to plasticizers between urban and rural areas, with rural areas carrying a higher risk of exposure.

As shown in Table 1, the HQ in urban and rural areas was between 6.60×10^{-8} and 2.66×10^{-3} . In addition, the HI calculated in rural areas (9.93×10^{-3}) was approximately 6.9 times higher than that in urban areas (1.43×10^{-3}). Although the noncarcinogenic risks of all plasticizers were below 1.0, DEHP (rural settings: 72.01%, urban settings: 71.7%) and DNOP (rural settings: 26.83%, urban settings: 18.76%) significantly contributed to the calculated HI, and their health risks remain a concern, particularly in rural indoor environments.

Table 1 Noncarcinogenic risk of time-weighted respiratory exposure to MPs and plasticizers in urban and rural environments.

		<u>Rural</u>			<u>Urban</u>		
	<u>RfD</u> ($\text{mg kg}^{-1} \text{d}^{-1}$)	<u>ADD</u> ($\text{mg kg}^{-1} \text{d}^{-1}$)	<u>HQ</u>	<u>HQ/HI</u> (%)	<u>ADD</u> ($\text{mg kg}^{-1} \text{d}^{-1}$)	<u>HQ</u>	<u>HQ/HI</u> (%)
<u>DMP</u>	<u>10</u>	<u>3.80×10^{-6}</u>	<u>3.80×10^{-7}</u>	<u>0.004</u>	<u>6.60×10^{-7}</u>	<u>6.60×10^{-8}</u>	<u>0.005</u>
<u>DEP</u>	<u>0.8</u>	<u>3.76×10^{-6}</u>	<u>4.70×10^{-6}</u>	<u>0.05</u>	<u>3.91×10^{-7}</u>	<u>4.88×10^{-7}</u>	<u>0.03</u>
<u>BBP</u>	<u>0.2</u>	<u>1.06×10^{-5}</u>	<u>5.28×10^{-5}</u>	<u>0.53</u>	<u>2.08×10^{-6}</u>	<u>1.01×10^{-5}</u>	<u>0.73</u>
<u>DEHP</u>	<u>0.02</u>	<u>1.43×10^{-4}</u>	<u>7.15×10^{-3}</u>	<u>72.0</u>	<u>2.05×10^{-5}</u>	<u>1.03×10^{-3}</u>	<u>71.7</u>
<u>DNOP</u>	<u>0.04</u>	<u>1.07×10^{-4}</u>	<u>2.66×10^{-3}</u>	<u>26.8</u>	<u>1.07×10^{-5}</u>	<u>2.68×10^{-4}</u>	<u>18.8</u>
<u>BPA</u>	<u>0.05</u>	<u>2.85×10^{-6}</u>	<u>5.71×10^{-5}</u>	<u>0.57</u>	<u>6.28×10^{-6}</u>	<u>1.26×10^{-4}</u>	<u>8.77</u>
<u>HI</u>	<u>↓</u>	<u>↓</u>	<u>9.93×10^{-3}</u>	<u>↓</u>	<u>↓</u>	<u>1.43×10^{-3}</u>	<u>↓</u>

As shown in Table 2, DEHP accounted for more than 99% of the total carcinogenic risk (Σ ILCR) in both rural and urban environments, with BT and BBP making relatively low contributions to such risk. The Σ ILCR value for the rural samples was calculated as 2.00×10^{-6} , which exceeds the internationally recognized risk threshold (1.0×10^{-6}), indicating that MPs and plasticizers in rural environments pose a clear carcinogenic risk through respiratory exposure pathways, which warrants immediate attention. By contrast, the Σ ILCR value for the urban samples was calculated as 2.88×10^{-7} , indicating an acceptable degree of risk. The carcinogenic risk caused by individual exposure to MPs and plasticizers in $PM_{2.5}$ in rural areas was about 7 times higher than that in urban areas. These results also suggest that DEHP is the primary driver of carcinogenic risk for MPs and plasticizers. Further research is required to examine the specific exposure pathways and pollution sources of DEHP.

Table 2 Carcinogenic risk of time-weighted respiratory exposure to MPs and plasticizers in urban and rural environments.

		<u>EC</u> ($\mu\text{g m}^{-3}$)	<u>IUR</u> ($\text{m}^3 \mu\text{g}^{-1}$)	<u>SF</u> (kg d mg^{-1})	<u>ILCR</u>	<u>ILCR/</u> <u>ΣILCR</u> (%)
Rural	BT	3.37×10^{-3}	1.8×10^{-7}	\	6.07×10^{-10}	0.03
	BBP	\	\	0.00019	2.01×10^{-9}	0.10
	DEHP	\	\	0.014	2.00×10^{-6}	99.9
	ΣILCR	\	\	\	2.00×10^{-6}	\
Urban	BT	1.00×10^{-3}	1.8×10^{-7}		1.80×10^{-10}	0.06
	BBP	\	\	0.00019	3.96×10^{-10}	0.14
	DEHP	\	\	0.014	2.87×10^{-7}	99.8
	ΣILCR	\	\	\	2.88×10^{-7}	\

3.5.2 Intracellular ROS and CA-based contribution to MPs

The $EC_{1.5}$ values of the three MPs ranged from 1.58×10^{-5} to $1.03 \times 10^{-4} \text{ mol L}^{-1}$, corresponding to the following potency ranking: PET > PS > PE. PET exhibited the lowest $EC_{1.5}$ ($1.58 \times 10^{-5} \text{ mol L}^{-1}$) and an approximately 6.5-fold greater molar effect potency than PE ($1.03 \times 10^{-4} \text{ mol L}^{-1}$). These differences suggest that repeat-unit chemistry and associated surface properties may influence the oxidative potential of

different polymers. Table 3 compares the EC_{1.5} for three representative MP polymers and several PM_{2.5}-associated metals and polycyclic aromatic hydrocarbons (PAHs).

The ROS-inducing potencies of the different substances were highly heterogeneous, with EC_{1.5} values spanning more than six orders of magnitude. The carcinogenic PAHs, such as dibenzo[a,l]pyrene (DBaP) and benzo[a]pyrene (BaP) and the redox-active transition metals, like Mn(II) and Cu(II) showed the high-potency of ROS, whereas the relatively redox-inactive crustal elements Rb(I), Mg(II), Al(III), and Ti(IV) exhibited substantially lower potencies. The three MPs molar effect potencies were approximately 1.3-2.2 orders of magnitude lower than those of Cu(II) and Mn(II). PET and PS were generally approximately one to two orders of magnitude more potent than Rb(I), Mg(II), Al(III), and Ti(IV), whereas PE was only approximately 2- to 15-fold more potent than these four elements.

Based on CA equivalent approach, the three MP polymers together accounted for approximately 0.52% of the PM_{2.5}-induced ROS response in rural samples and 0.15% in urban samples (Table S5). PET made the largest contribution, mainly because of its substantially higher environmental abundance. Although the direct ROS contribution of three MPs was limited, their toxicological significance may extend beyond their intrinsic oxidative activity. Environmental MPs possess strong sorption capacities for hydrophobic organic contaminants (e.g., PAHs and per-and polyfluoroalkyl substances (PFAS)) and heavy metals, allowing them to function as vectors for co-contaminants through the “Trojan horse” effect (Zhang & Xu, 2022). The subsequent desorption of these contaminants following inhalation and cellular uptake may increase their local bioavailability and amplify oxidative stress, inflammation, and other toxic responses compared with exposure to MPs or co-contaminants alone (Hahladakis et al., 2018; Prata, 2018; Amato-Lourenço et al., 2021). Therefore, assessing the health risks of MPs based solely on their intrinsic toxicity may underestimate their potential impacts under environmentally realistic co-exposure conditions (Hu et al., 2022).

Table 3 Comparison of EC_{1.5} values for ROS induction between MPs and typical

PM_{2.5}-bound metals and PAHs.

<u>Category</u>	<u>Substance</u>	<u>EC_{1.5} (mol L⁻¹)</u>	<u>Source</u>
<u>Metal</u>	<u>Rb(I)</u>	<u>1.51×10⁻³</u>	<u>Chen et al., 2025</u>
<u>Metal</u>	<u>Mg(II)</u>	<u>3.98×10⁻⁴</u>	<u>Chen et al., 2025</u>
<u>Metal</u>	<u>Al(III)</u>	<u>3.70×10⁻⁴</u>	<u>Chen et al., 2025</u>
<u>Metal</u>	<u>Ti(IV)</u>	<u>2.29×10⁻⁴</u>	<u>Chen et al., 2025</u>
<u>MPs</u>	<u>PE</u>	<u>1.03×10⁻⁴</u>	<u>This study</u>
<u>MPs</u>	<u>PS</u>	<u>2.11×10⁻⁵</u>	<u>This study</u>
<u>MPs</u>	<u>PET</u>	<u>1.58×10⁻⁵</u>	<u>This study</u>
<u>Metal</u>	<u>Cu(II)</u>	<u>8.28×10⁻⁷</u>	<u>Chen et al., 2025</u>
<u>Metal</u>	<u>Mn(II)</u>	<u>6.03×10⁻⁷</u>	<u>Chen et al., 2025</u>
<u>PAHs</u>	<u>BaP</u>	<u>1.60×10⁻⁸</u>	<u>Chen et al., 2025</u>
<u>PAHs</u>	<u>DBaP</u>	<u>1.20×10⁻⁹</u>	<u>Chen et al., 2025</u>

Note: EC_{1.5} represents the concentration required to induce a 1.5-fold increase in intracellular ROS relative to the control; lower EC_{1.5} values indicate greater oxidative stress potency. Molar concentrations of PET, PE, and PS are expressed as repeat-unit-equivalent concentrations, with corresponding repeat-unit molar masses of 192.2, 28.05, and 104.2 g mol⁻¹, respectively.

3.6 Dominated drivers underlying urban–rural exposure inequality

The indoor and outdoor concentration distributions (Figures 3 and 4) further indicated that pollutant concentrations were substantially elevated in the rural indoors, with an rural–urban concentration ratio of 3.9 for MPs and a nearly order-of-magnitude gap for DEHP and other typical PAEs; however, urban outdoor PAEs were slightly higher than rural levels, and urban MPs were slightly lower. This inversion demonstrates that differences in indoor source emission strength, combined with indoor accumulation and retention, constitute the core mechanism driving urban–rural exposure inequality. PMF source apportionment revealed that the core structural difference in urban–rural exposure to MPs and plasticizers lies in the contribution gap from plastic-product-related sources: these sources accounted for 51.7% of the total in rural areas, roughly 1.9 times the urban value (27.6%).

The combined evidence from PMF source apportionment, indoor–outdoor concentration patterns, questionnaire responses, and field observations suggests that household activities and residential characteristics jointly drive the substantially higher indoor burden of MPs and plasticizers in rural households. The aging and resuspension of everyday plastic items contribute significantly: rural households tend to use plastic commodities (storage containers, tableware, table covers, etc.) for longer periods with higher reuse rates, leading to more severe material degradation and sustained release of MPs and plasticizers (Yu et al., 2023; Wang et al., 2024). Frequent turning of heavy winter clothing and synthetic bedding facilitates resuspension of fibrous MPs, while prolonged food storage in plastic bags and containers adds to plasticizer volatilization, together forming a baseline of indoor sources (Hernandez et al., 2017; Vassilenko et al., 2021; Wang et al., 2024; Bi et al., 2024). According to the questionnaire information, 60% of rural households in this survey burn plastic waste (such as apple bags, recycled mulch film, etc.) in indoor stoves (usually used as kindling for solid fuel) or nearby yards during the winter heating season. the incomplete combustion generates flue gases rich in MPs and plasticizers. Burning indoors directly retains the emissions in living spaces, while courtyard burning allows the fumes to infiltrate indoors through door/window gaps and ventilation openings (Velis & Cook, 2021; Wu et al., 2021; Růžicková et al., 2023).

Beyond source strength differences, the residential characteristics of rural areas in winter markedly reduce pollutant dispersion and thus amplify urban–rural exposure disparities. During the sampling period in January 2024, the average daily outdoor temperature for rural households was -5°C, and the average indoor temperature was 9°C (without central heating); while the average daily outdoor temperature for urban households was -1°C, and the average indoor temperature was 21°C. Inadequate ventilation dilution is a primary factor: the cold winter climate in the Guanzhong Plain prompts rural families to substantially reduce window opening for thermal insulation. Questionnaire responses showed that the composite score for “daily living habits”

(including ventilation frequency, cleaning practices, and use of air purifiers) was lower in rural areas (1.50) than in urban areas (1.73), indicating generally lower ventilation and cleaning frequencies. Meanwhile, winter heat-retention practices in rural area in order to resist the cold, such as sealing doors and windows and installing door curtains, increase building airtightness and reduce natural ventilation and outdoor air infiltration, thereby lowering air exchange rates and weakening pollutant dilution and removal (Ghasemi et al., 2020; Jahanbin & Semprini, 2022; Mukai et al., 2009; Wallace et al., 2002; Zhang et al., 2022). The low air exchange rate prevents indoor-emitted MPs and plasticizers from being diluted, leading to continuous accumulation in enclosed spaces. Moreover, the interconnected cooking, living, and sleeping areas in many rural houses limit the effectiveness of kitchen exhaust for whole-house dilution (Feng et al., 2025). In addition, decentralized heating in rural homes can generate buoyancy-driven indoor airflow, altering the transport, dispersion, and decay of deposited particles; stronger near-floor airflow and turbulence may increase particle resuspension.

In summary, the superposition of source strength and retention effects collectively elevates the indoor inhalation exposure burden of rural residents and provides a mechanistic explanation for the marked urban–rural differences in health risks. The stronger indoor sources and reduced pollutant removal in rural households resulted in substantially higher inhalation exposure, which was reflected in the greater ROS-inducing potential of rural PM_{2.5} samples and the elevated non-carcinogenic and carcinogenic risks associated with MPs and plasticizers compared with urban areas. Based on these contrasting source and exposure patterns, source-informed exposure-reduction priorities may differ between the investigated rural and urban settings. For the rural households, potential measures include reducing the repeated handling and indoor storage of disposable or aged plastic materials, improving plastic-waste collection and separation, avoiding uncontrolled waste burning, increasing ventilation when outdoor conditions permit, and adopting lower-emission alternatives for food storage and agricultural materials. For the investigated urban environment, priorities

may include reducing traffic-related non-exhaust emissions, particularly tire-wear particles, and strengthening the control of emissions associated with consumer products and building materials. These recommendations represent source-informed priorities rather than quantitatively validated mitigation efficiencies.

4. Conclusion

~~The~~This study revealed significant urban–rural ~~inequality~~inequalities in exposure to atmospheric MPs and plasticizers ~~was observed in northern~~in the Guanzhong Plain ~~in Northern~~ China ~~in this study~~. 24 h time-weighted MPs exposure concentrations in rural areas were approximately 3.6 times higher than in urban areas, while plasticizer exposure was even greater, 6.8 times. ~~Notably, PAEs levels, especially DEHP, were markedly elevated in rural indoor air, reaching approximately 600 ng m⁻³.~~Tire wear-derived polymers, including NR and SBR, were major contributors to the MPs in outdoor environments. MP and plasticizer ~~pollution~~exposure in rural areas ~~was~~ primarily ~~stems from the repeated~~associated with plastic product-related sources, ~~suggesting that frequent use and indoor accumulation of plastic-containing products and weak waste management systems, forming a pollution pattern dominated by direct input of plastic consumer goods represent key pathways contributing to rural exposure.~~ In contrast, urban pollution exhibits multi-source and complex characteristics, resulting from the combined effects of various human activities. Critically, the carcinogenic risk from MPs and plasticizers in rural environments exceeded the recommended limit and was about 7 times higher than in urban settings, ~~with DEHP accounting for the majority (>99%) of the risk. DTT_v of PM_{2.5} was significantly higher in rural areas, indicating greater potential to induce oxidative stress under real exposure conditions. It correlated strongly with MPs such as PET, PS, PP, and with benzothiazoles; whereas DTT_m showed limited sensitivity due to compositional and concentration effects in PM_{2.5}. This distinction highlights the complementary roles of exposure-driven (DTT_v) and intrinsic activity (DTT_m) perspectives in OP evaluation.~~ The in vitro tests further ~~indicated that PET, PE and PS have certain efficacy in inducing ROS. Their contribution~~

to the ROS of PM_{2.5} is approximately 0.52% (in rural areas) and 0.15% (in urban areas).

Although there are some limitations in this study, such as ~~the~~ relatively small sample size ~~and research area~~, ~~the constrained geographic scope~~, the lack of physical methods to determine the morphology of MPs, ~~and the winter-only sampling season which may limit the generalizability of our findings to other seasons (e.g., summer with different ventilation and source patterns)~~, this is the first ~~systematic~~ study of atmospheric MPs and plasticizers conducted in both indoor and outdoor environments across urban and rural areas in ~~northern~~Northern China. ~~The substantial~~Our findings demonstrate that urban-rural inequalities ~~exist between urban and rural residents in terms of~~PM_{2.5}-bound MPs and ~~plasticizers~~plasticizer exposure ~~are primarily driven by elevated indoor accumulation in rural environments. The results further support prioritizing source-informed exposure-reduction measures in rural households, particularly those targeting indoor pollutant accumulation and emissions associated health effects. The higher environmental health burden borne by rural populations underscores the urgent need to integrate equity considerations into environmental policy~~with the use and ~~risk assessment frameworks, and to implement targeted interventions to protect rural community health~~storage of plastic-containing products.

Supplementary data

Supplementary data in this manuscript can be found in Supporting Information.

Data availability

Data will be made available on request.

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.

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