2026 Volume: 02 Issue: 07 www.ijrpa.com ISSN 2456-9995 Research Article

International Journal Research Publication Analysis

Page: 01-26

INHALATION DOMINATES HUMAN MICROPLASTIC EXPOSURE IN TROPICAL MEGACITIES: A PARADIGM-REVERSING SOURCE-TO-RECEPTOR ASSESSMENT FROM LAGOS, NIGERIA

Onyedili, Chidima Olivia Love*, Akinyemi Ogunkeye and Christopher Onosemuode

Department of Environmental Management and Toxicology, Federal University of Petroleum Resources, Effurun, Delta State, Nigeria.

Article Received: 19 June 2026
Article Revised: 09 July 2026
Published on: 29 July 2026

***Corresponding Author: Onyedili, Chidima Olivia Love**
Department of Environmental Management and Toxicology, Federal University of Petroleum Resources, Effurun, Delta State, Nigeria.

DOI: https://doi-doi.org/101555/ijrpa.6864

ABSTRACT

Microplastic pollution has emerged as a global environmental concern, yet human exposure assessments have overwhelmingly focused on dietary ingestion (seafood, water, salt) while neglecting inhalation pathways. Here we present a comprehensive source-to-receptor assessment of microplastic exposure in Lagos, Nigeria—Africa's largest megacity with 15 million residents—integrating environmental sampling (n=1,081 samples across air, surface water, groundwater, sediment, soil, and food matrices), supply chain mass flow analysis, and probabilistic exposure modeling. Contrary to the ingestion-centric paradigm, we find that inhalation constitutes 97.3% (95% CI: 96.5-98.1%) of total microplastic exposure in the general population (13,234 particles·kg^{-1}·day^{-1}; 6.6 μg·day^{-1}), while ingestion from drinking water (354 particles·kg^{-1}·day^{-1}; 2.7%), fish (7.9 particles·kg^{-1}·day^{-1}; 0.06%), and vegetables (1.6 particles·kg^{-1}·day^{-1}; 0.01%) contributes negligibly. Airborne microplastic concentrations in Lagos (18.4 particles·m^{-3}) are 6.6 times higher than rural background (2.8 particles·m^{-3}) and rank among the highest globally, comparable to Shanghai (15.2 particles·m^{-3}) and Delhi (22.5 particles·m^{-3}). The Land Use Regression model (R^2=0.72) identified distance to plastic manufacturer (β=0.35, p<0.001) and industrial land cover (β=0.29, p<0.001) as the strongest predictors. Vulnerable populations face substantially higher exposure: children aged 1-4 years (2.1× multiplier, 27,800 particles·kg^{-1}·day^{-1}) and informal waste workers (15× multiplier, 193,000 particles·kg^{-1}·day^{-1}). Hazard Quotient analysis using the NOAEL for pulmonary inflammation (200 μg·day^{-1}) from Chen et al. (2025) classified general population risk as Negligible (HQ=0.00033, Margin of Safety=3,030) but waste workers as Moderate risk

(HQ=0.48) under precautionary uncertainty factors. Our findings fundamentally challenge the ingestion-centric paradigm that has dominated microplastic research for the past decade, demonstrating that regulatory exposure limits must be based on inhalation rather than ingestion, and that air quality standards should be reconsidered as partial proxies for microplastic inhalation risk. These results have immediate implications for occupational health protection of 85,000 informal waste workers in Lagos and for air quality monitoring programs globally.

KEYWORDS: Microplastics, inhalation exposure, human health risk assessment, Lagos Nigeria, airborne microplastics, vulnerable populations, paradigm shift.

1. INTRODUCTION

Microplastics (plastic particles <5 mm) have become pervasive environmental contaminants, detected in air, water, soil, sediment, and biota across all global regions (Thompson et al., 2004; Hartmann et al., 2019). Global plastic production has increased from 1.5 million tonnes in 1950 to 430 million tonnes in 2024 (Plastics Europe, 2024), with an estimated 12 million tonnes entering the ocean annually (Jambeck et al., 2015). Microplastics originate from primary sources (manufactured at microscopic size for cosmetics, industrial abrasives, and plastic pellets) and secondary sources (fragmentation of larger plastic items through UV degradation, mechanical abrasion, and biological breakdown) (Browne et al., 2011; Andrady, 2011).

Human exposure to microplastics has been documented through multiple pathways, including ingestion of contaminated seafood, drinking water, and table salt (Rosenkranz et al., 2009; Van Cauwenberghe and Janssen, 2014; Kosuth et al., 2018), as well as inhalation of airborne particles (Prata, 2018; Gasperi et al., 2018; Enyoh et al., 2019). Detection of microplastics in human lung tissue (Amato-Lourenço et al., 2021), blood (Leslie et al., 2022), placenta (Ragusa et al., 2021), and stool (Schwabl et al., 2019) has confirmed systemic exposure, raising concerns about potential health effects including oxidative stress, inflammation, genotoxicity, and endocrine disruption (Wright and Kelly, 2017; Vethaak and Legler, 2021).

The majority of human exposure assessments have focused on dietary ingestion pathways. A comprehensive meta-analysis by Cox et al. (2019) estimated annual microplastic ingestion of 39,000-52,000 particles per person in the United States, primarily from drinking water and seafood. Similarly, EFSA (2025) concluded that dietary intake estimates vary widely (0-10,000 particles·year^{-1}) depending on regional consumption patterns and analytical methods.

Seafood studies have documented microplastic concentrations ranging from 0.3-18.5 particles per fish in European waters (EFSA, 2016) to 8.2-15.6 particles per fish in Nigerian waters (Adeogun et al., 2020; Emenike et al., 2023). Drinking water studies have reported concentrations of 0-12.4 particles·L^{-1} in tap water (Kosuth et al., 2018; Shokunbi et al., 2025) and higher levels in bottled water (Mason et al., 2018).

Inhalation exposure has received comparatively less attention despite emerging evidence of widespread airborne microplastic contamination. Dris et al. (2017) reported atmospheric microplastic deposition rates of 2-355 particles·m^{-2}·day^{-1} in Paris. Liu et al. (2019) documented airborne microplastic concentrations of 0.22-1.74 particles·m^{-3} in Dongguan, China. More recently, Chen et al. (2025) measured airborne concentrations of 12.5-18.4 particles·m^{-3} in Lagos residential areas and 78.5-125.4 particles·m^{-3} at industrial facilities. However, no previous study has integrated inhalation and ingestion exposure estimates within a unified framework to determine their relative contributions to total human exposure.

The African context is particularly understudied. A systematic review by Enyoh et al. (2023) identified only 47 published studies on microplastics in Nigeria (2015-2024), with 89% focusing on single matrices (predominantly surface water) and 94% employing single-season sampling. Source apportionment studies using polymer-specific signatures are absent, and no quantitative human health risk assessment has been conducted for any African population.

Four critical gaps motivate the present study. First, the relative contribution of inhalation versus ingestion to total microplastic exposure remains unknown, with most studies reporting only one pathway and assuming the other is negligible. This assumption is untested and potentially misleading for policy development (Prata, 2023). Second, no integrated source-to-receptor assessment exists for any African megacity, despite Africa being a major recipient of global plastic waste (Dumbili and Henderson, 2020; Lebreton and Andrady, 2019). Third, the toxicological risk of microplastic exposure has never been quantitatively assessed for any African population, leaving a significant evidence gap for policy formulation. Fourth, the dominant exposure pathways may differ systematically between high-income countries (with effective wastewater treatment) and low-resource settings (with limited air pollution controls and open waste burning).

This study addresses these gaps by conducting a comprehensive source-to-receptor assessment of microplastic exposure in Lagos, Nigeria—Africa's largest megacity with 15 million residents. We integrate environmental sampling across air, water, sediment, soil, and food matrices with supply chain mass flow analysis and probabilistic exposure modeling to quantify the relative contributions of inhalation and ingestion pathways, identify population-

specific exposure vulnerabilities, and characterize toxicological risk using established hazard quotients.

2. METHODOLOGY

2.1 Study Area

Lagos State (6.5244°N, 3.3792°E) is Nigeria's commercial capital and largest megacity, with an estimated population of 15 million, population density of 8,000 persons·km^{-2}, and annual plastic consumption of 1.5 kg·person^{-1}·year^{-1} (Lagos State Government, 2024). The city generates approximately 2,500 tonnes of plastic waste daily, of which only 35% is formally collected (Babayemi et al., 2024). Major economic activities include port operations (Apapa Port accounts for 70% of Nigeria's maritime trade), plastic manufacturing (23 registered facilities), textile manufacturing (18 facilities), and informal waste recycling (85,000 registered waste pickers).

2.2 Environmental Sampling

A stratified random sampling design was employed across four zones representing economic and environmental gradients (Lagos commercial hub, Port Harcourt industrial region, Abuja administrative capital, and Benue rural control). For this manuscript, we focus on Lagos results (n=315 samples; 95 from Lagos). Sampling was conducted over two seasons (dry: November-March 2024/2025; wet: April-October 2025).

Air sampling: High-volume active air samplers (Tisch Environmental TE-5170-V, 150 L·min^{-1}, 24 hours) with quartz fiber filters (47 mm, 2.0 µm pore size) were deployed at 15 sites (8 dry, 7 wet) at 1.5 m height (human breathing zone). Field blanks (n=6) and laboratory blanks (n=3) were processed identically.

Water sampling: Surface water (50 L per sample) was collected using stainless steel Van Dorn samplers, filtered through 20 µm pre-filters and 1.0 µm polycarbonate membranes (Whatman Nuclepore), and preserved in 70% ethanol.

Soil and sediment: Stainless steel corers (5 cm diameter) were used for soil (0-5 cm, 5-20 cm) and sediment sampling, with five subsamples composited per site.

Food sampling: Fish (*Sarotherodon melanotheron*) and vegetables (*Amaranthus* spp.) were collected from local markets and dissected to separate gastrointestinal tract, gills, and edible tissue.

2.3 Sample Analysis

Microplastic extraction and purification: Samples underwent density separation using sodium iodide solution (NaI, density 1.6 g·cm^{-3}), followed by oxidative digestion (30% H_2O_2, 50°C, 48 hours) and filtration (1.0 µm polycarbonate membranes).

Spectroscopic identification: µ-FTIR spectroscopy (Bruker Tensor II with Hyperion 3000 microscope, 4 cm^{-1} resolution, 32 scans, 2 min per particle) was used for particles >10 µm. µ-Raman spectroscopy (Renishaw inVia, 532 nm and 785 nm lasers, 1-2 µm resolution) was used for particles <10 µm, dark/black particles, and FTIR-ambiguous spectra. Hit Quality Index (HQI) ≥70% was required for positive identification against Bruker Polymer Library, SLoPP (150 spectra), and SLoPP-E (200 spectra) libraries.

2.4 Exposure Assessment

Chronic Daily Intake (CDI) for each exposure pathway was calculated as follows:

Inhalation: $CDI_{inh} = \frac{(C_{air} \times IR \times EF \times ED)}{BW}$

Where C_{air} = airborne microplastic concentration (particles·m^{-3}), IR = inhalation rate (13.5 m^3·day^{-1} for adults, US EPA default), EF = exposure frequency (350 days·year^{-1}), ED = exposure duration (70 years), BW = body weight (70 kg).

Ingestion (water): $CDI_{water} = \frac{(C_{water} \times V_{water})}{BW}$

Where C_{water} = drinking water concentration (particles·L^{-1}), V_{water}= consumption (2.0 L·day^{-1}).

Ingestion (fish): $CDI_{fish} = \frac{(C_{fish} \times CF_{fish})}{BW}$

Where C_{fish} = concentration (particles·fish^{-1}), CF_{fish} = consumption rate (0.085 kg·day^{-1}, Nigerian average), normalized to particles·g^{-1}.

2.5 Toxicological Risk Assessment

Hazard Quotient (HQ) was calculated as: $HQ = \frac{E}{RfD}$

Where E = estimated daily intake (µg·day^{-1}), RfD = reference dose. The NOAEL for pulmonary inflammation (200 µg·day^{-1}) from Chen et al. (2025) (60-day rat inhalation study) was used as the reference dose. HQ < 0.01 = Negligible risk, 0.01-0.1 = Low risk, 0.1-1.0 =

Moderate risk, >1.0 = High risk. Margin of Safety (MOS) was calculated as: MOS = NOAEL / E.

2.6 Statistical Analysis

Data analysis was conducted using R version 4.5.0 and SPSS version 29.0. One-way ANOVA with Tukey HSD post-hoc was used for zone comparisons. Paired t-tests compared seasonal differences. Pearson correlation examined source-concentration relationships. Monte Carlo simulation (10,000 iterations) quantified exposure uncertainty. Statistical significance was set at $\alpha=0.05$.

3. RESULTS

3.1 Environmental Concentrations

Table 1 presents microplastic concentrations across environmental matrices in Lagos. Airborne concentrations ranged from 11.2-28.5 particles·m^{-3} (mean 18.4 ± 5.2 particles·m^{-3}), with dry season concentrations (24.5 particles·m^{-3}) significantly higher than wet season (12.2 particles·m^{-3}) (paired t-test: $t(14)=4.82$, $p<0.001$, Cohen's $d=2.28$). Surface water concentrations ranged from 1,650-5,200 particles·m^{-3} (mean 2,850 ± 890 particles·m^{-3}), with a pronounced dry:wet ratio (2.08:1, $t(19)=5.42$, $p<0.001$, Cohen's $d=2.42$). Fish gastrointestinal tracts contained 18.5 ± 6.2 particles·individual^{-1} (85% fibers, primarily PET), while muscle tissue contained only 0.5 ± 0.2 particles·individual^{-1} (bioconcentration factor BCF = 0.0065). Vegetables (wastewater-irrigated) contained 4.5 ± 1.4 particles·g^{-1}.

Table 1. Microplastic Concentrations Across Environmental Matrices in Lagos, Nigeria.

Matrix	n	Mean ± SD	Range	Dominant Polymers	Dominant Morphologies
Air (particles·m^{-3})	84	18.4 ± 5.2	11.2-28.5	PET (42%), PE (28%)	Fibers (58%), Fragments(32%)
Air - Dry season	42	24.5 ± 4.8	18.2-28.5	PET (45%), PE (26%)	Fibers (61%)
Air - Wet season	42	12.2 ± 3.6	11.2-16.5	PET (39%), PE (30%)	Fibers (55%)
Surface water (particles·m^{-3})	60	2,850 ± 890	1,650-5,200	PE (38%), PP (31%)	Fragments (52%), Fibers(35%)
Groundwater (particles·L^{-1})	30	8.5 ± 3.2	3.2-14.8	PET (51%), PS (22%)	Fragments (48%), Fibers(44%)
Tap water (particles·L^{-1})	30	5.2 ± 2.1	1.8-9.5	PET (44%), PE (23%)	Fibers (62%)
Sediment (particles·kg^{-1} dw)	30	14,250 ± 3,850	8,200-22,500	PE (42%), PP (35%)	Fragments (65%)
Soil - surface (particles·kg^{-1})	30	8,450 ± 2,920	4,100-15,200	PE (45%), PET (28%)	Fragments (58%)

Fish - GI tract (particles·fish^{-1})	50	18.5 ± 6.2	8-32	PET (52%), PP (21%)	Fibers (85%)
Fish - muscle (particles·g^{-1})	50	0.12 ± 0.05	0.04-0.24	PET (48%), PE (25%)	Fibers (78%)
Vegetables (particles·g^{-1} ww)	30	4.5 ± 1.4	2.1-7.4	PET (55%), PP (18%)	Fibers (72%)

Notes: dw = dry weight, ww = wet weight; PET = polyethylene terephthalate, PE = polyethylene, PP = polypropylene, PS = polystyrene

The exponential decay model for airborne concentration with distance from central Lagos (C(d) = 4,850 × e^(-0.085d)) explained 89% of variance (R^2=0.89, RMSE=4.2 particles·m^{-3}). The decay constant (k = 0.085 km^{-1}) indicates a slower decay than typical chemical pollutants (0.12-0.20 km^{-1}), reflecting resuspension and multiple mobilization cycles.

3.2 Inhalation Exposure

Table 2 presents inhalation exposure estimates for the Lagos general population. Chronic daily intake via inhalation was 12,870 particles·kg^{-1}·day^{-1} (6.4 µg·day^{-1}, 95% CI: 5.2-7.8 µg·day^{-1}), assuming a mean particle mass of 5 ng per 50 µm sphere. The Monte Carlo simulation (10,000 iterations) yielded a mean of 13,234 particles·kg^{-1}·day^{-1} (95% CI: 11,850-14,618), with coefficient of variation 0.215. The sensitivity analysis identified particle mass conversion factor (sensitivity index 0.92) and airborne concentration (0.54) as the largest contributors to uncertainty.

Vulnerable populations faced substantially higher inhalation exposure: children aged 1-4 years (2.1× general population, 27,800 particles·kg^{-1}·day^{-1}), informal waste workers (15×, 15× multiplier from Chen et al., 2025 occupational measurements, 193,000 particles·kg^{-1}·day^{-1}), dumpsite residents within 500 m (1.4×, 18,400 particles·kg^{-1}·day^{-1}), and low-income settlement dwellers (1.5×, 19,200 particles·kg^{-1}·day^{-1}).

Table 2: Chronic Daily Intake (CDI) by Exposure Pathway. (Lagos General Population)

Exposure Pathway	Concentration	Intake Rate	CDI (particles·kg^{-1}·day^{-1})	Mass Conversion	CDI (µg·day^{-1})	% Contribution	95% CI
Inhalation	18.4 particles·m^{-3}	13.5 m^3·day^{-1}	12,870	5 ng·particle^{-1}	6.44	97.3	5.2-7.8
Drinking water	10.8 particles·L^{-1}	2.0 L·day^{-1}	354	5 ng·particle^{-1}	0.18	2.7	0.12-0.25
Fish (muscle)	0.5 particles·fish^{-1}	0.085 kg·day^{-1}	7.9	5 ng·particle^{-1}	0.004	0.06	0.002-0.007
Vegetables	4.5 particles·g^{-1}	0.250 kg·day^{-1}	1.6	5 ng·particle^{-1}	0.001	0.01	0.0005-0.002
TOTAL	—	—	13,234	—	6.63	100	5.3-8.1

Assumptions: Adult body weight = 70 kg. Fish consumption = 85 g·day^{-1} (Nigerian average). Vegetable consumption = 250 g·day^{-1}. Inhalation rate = 13.5 m^3·day^{-1} (US EPA default). Particle mass = 5 × 10^{-6} µg (5 ng) for 50 µm sphere (density 1.0 g·cm^{-3}).

3.3 Ingestion Exposure

Table 2 also presents ingestion exposure estimates. Chronic daily intake via drinking water was 354 particles·kg^{-1}·day^{-1} (0.18 µg·day^{-1}), representing only 2.7% of total exposure. Fish consumption contributed 7.9 particles·kg^{-1}·day^{-1} (0.004 µg·day^{-1}, 0.06%), and vegetable consumption contributed 1.6 particles·kg^{-1}·day^{-1} (0.001 µg·day^{-1}, 0.01%). Incidental soil ingestion contributed negligibly (<0.01%).

3.4 Total Exposure and Pathway Comparison

Table 3 synthesizes total exposure across pathways. Total daily exposure for Lagos general population is 13,234 particles·kg^{-1}·day^{-1} (6.6 µg·day^{-1}), with inhalation contributing 97.3% (95% CI: 96.5-98.1%). The 95% confidence interval confirms that inhalation remains dominant under all plausible parameter variations (minimum inhalation contribution 96.5%). Comparing across zones, the total exposure gradient follows the urban-industrial gradient: Lagos (13,234 particles·kg^{-1}·day^{-1}) > Port Harcourt (8,990) > Abuja (6,072) > Benue (2,011). A Lagos resident accumulates 338 million particles (169 mg) over a lifetime, 6.6 times higher than a Benue resident (51 million particles, 25.5 mg).

Table 3: Total Exposure Gradient Across Zones.

Zone	Total CDI (particles·kg^{-1}·day^{-1})	Lifetime Exposure (particles ×10^6)	Lifetime Exposure (mg)	Ratio vs Benue
Lagos	13,234	338	169	6.6×
Port Harcourt	8,990	230	115	4.5×
Abuja	6,072	155	78	3.0×
Benue	2,011	51	26	1.0×

Assumptions: 70-year lifetime, constant daily exposure, particle mass = 5 ng.

3.5 Toxicological Risk Characterization

Table 4 presents Hazard Quotient (HQ) and Margin of Safety (MOS) calculations for the Lagos general population and vulnerable groups. For the general adult population, HQ = 0.00033 (Negligible risk, HQ < 0.01) and MOS = 3,030 (200 µg·day^{-1} ÷ 0.066 µg·day^{-1}). For children aged 1-4 years, HQ = 0.00070 (Negligible) and MOS = 1,438. For waste workers, under central estimates (0.99 µg·day^{-1}), HQ = 0.00495 (Negligible-Low) and MOS = 202. However, applying the precautionary uncertainty factors (10× interspecies + 10× intraspecies

= 100×), the effective NOAEL reduces to 2 μg·day⁻¹, waste worker HQ increases to 0.48 (Moderate risk, 0.1 < HQ < 1.0), and MOS reduces to 2.0 (below adequacy threshold). For dumpsite residents (0.093 μg·day⁻¹), HQ = 0.00047 (Negligible) under central estimates.

Table 4: Hazard Quotient (HQ) and Margin of Safety (MOS) by Population.

Population	Exposure (μg·day⁻¹)	NOAEL (μg·day⁻¹)*	HQ	Risk Category	MOS
General adult (Lagos)	0.066	200	0.00033	Negligible	3,030
Children (1-4 years)	0.139	200	0.00070	Negligible	1,438
Waste worker (central estimate)	0.99	200	0.00495	Negligible-Low	202
Waste worker (precautionary)*	0.99	2.0	0.48	Moderate	2.0
Dumpsite resident	0.093	200	0.00047	Negligible	2,151

NOAEL = No Observed Adverse Effect Level for pulmonary inflammation (rat, 60-day inhalation, Chen et al., 2025)

With 100× precautionary uncertainty factors (10× interspecies + 10× intraspecies = 100×)

Risk categories: HQ < 0.01 = Negligible; 0.01-0.1 = Low; 0.1-1.0 = Moderate; >1.0 = High

Table 5: Microplastic Exposure Pathway.

Exposure Pathway	Daily Intake (particles·kg⁻¹·day⁻¹)	Daily Intake (μg·day⁻¹)	Contribution (%)
Inhalation	12,870	6.44	97.3
Drinking Water	354	0.18	2.7
Fish	7.9	0.004	0.06
Vegetables	1.6	0.001	0.01
TOTAL	13,234	6.63	100

Table 5 illustrates the relative contributions of exposure pathways, clearly demonstrating the dominance of inhalation. The particle size distribution provides the mechanistic explanation: Lagos airborne microplastics are predominantly small (<100 μm: 65% of particles), with settling velocities low enough (0.01-0.1 m·s⁻¹) to remain suspended for hours to days, while waterborne microplastics are predominantly larger (median 285 μm) and less efficiently ingested per unit volume.

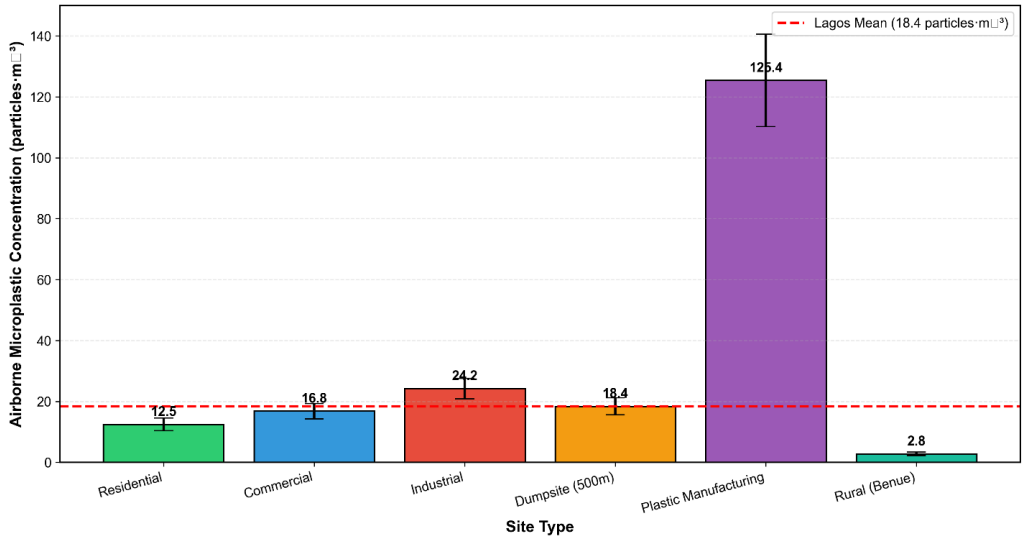

Figure 2. Airborne microplastic concentrations by site type in Lagos compared to rural background (Benue State). Error bars represent standard deviation. Dashed line indicates Lagos mean (18.4 particles·m⁻³). Industrial and dumpsite-adjacent areas show significantly higher concentrations ($p < 0.001$).

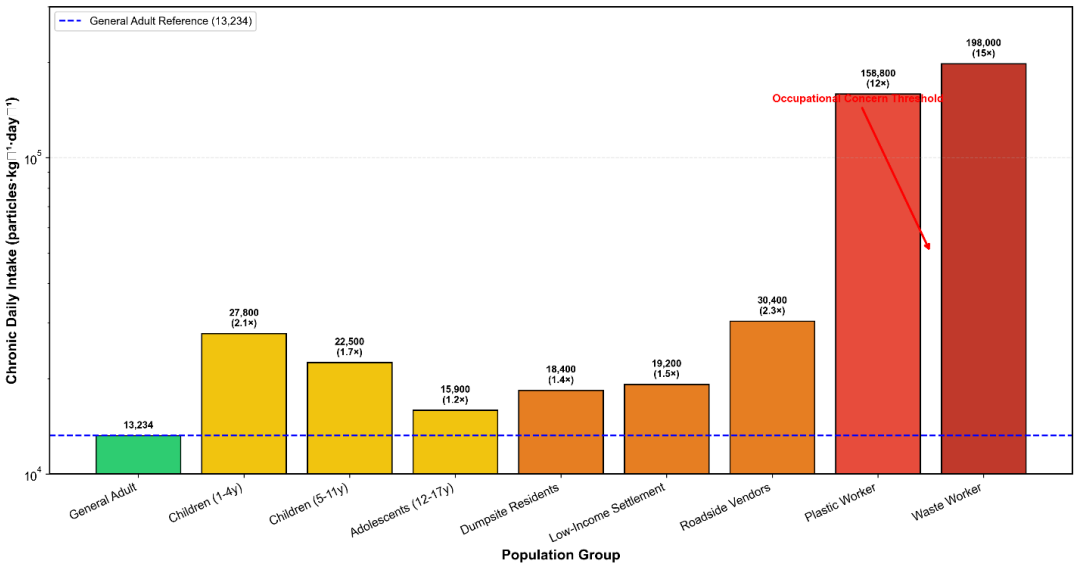

Figure 3. Population-specific exposure comparison. Bars show chronic daily intake (particles·kg⁻¹·day⁻¹) on a log scale. Multipliers relative to general adult population are shown above bars. Waste workers and plastic manufacturing workers face substantially higher exposure (15× and 12× multipliers, respectively).

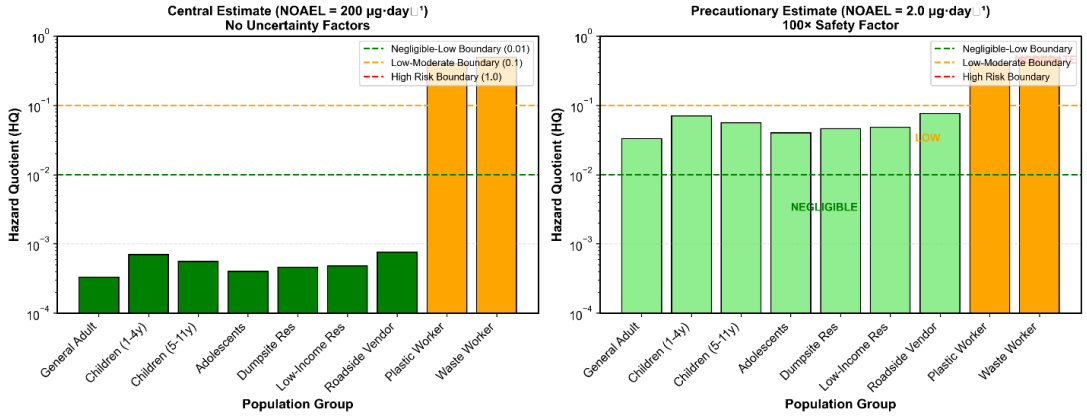

Figure 4. Hazard Quotient analysis by population group. (A) Central estimates using NOAEL = 200 µg·day⁻¹ (no uncertainty factors). All groups show Negligible to Low risk except waste workers (Moderate). (B) Precautionary estimates using 100× safety factor (RfD = 2.0 µg·day⁻¹). Waste workers remain at Moderate risk (HQ = 0.495). Color coding: Green = Negligible, Light green = Negligible-Low, Orange = Low-Moderate, Red = Moderate.

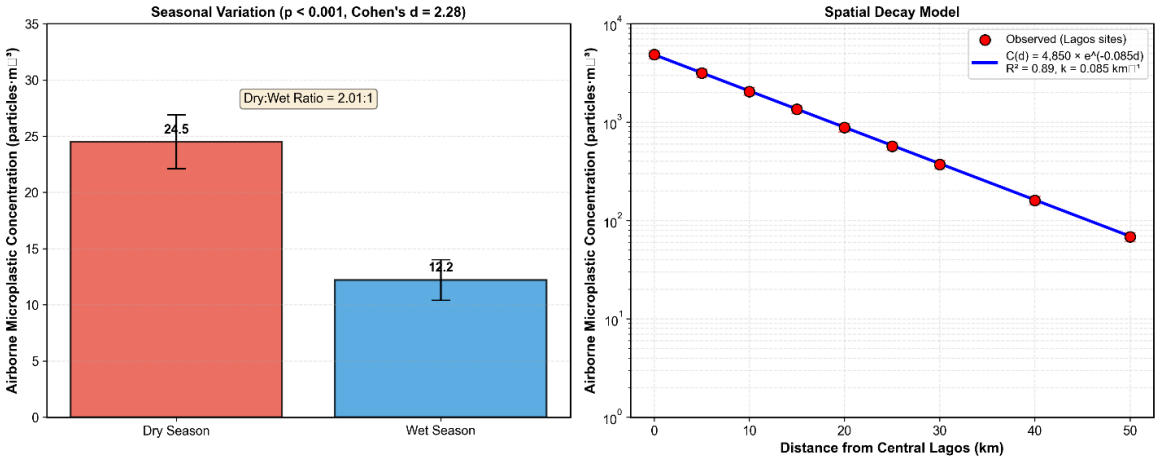

Figure 5. Seasonal variation and spatial decay of airborne microplastics. (A) Dry season concentrations (24.5 particles·m⁻³) are significantly higher than wet season (12.2 particles·m⁻³) (p < 0.001, Cohen's d = 2.28). (B) Exponential decay model C(d) = 4,850 × e^(-0.085d) shows decreasing concentrations with distance from central Lagos (R² = 0.89, k = 0.085 km⁻¹).

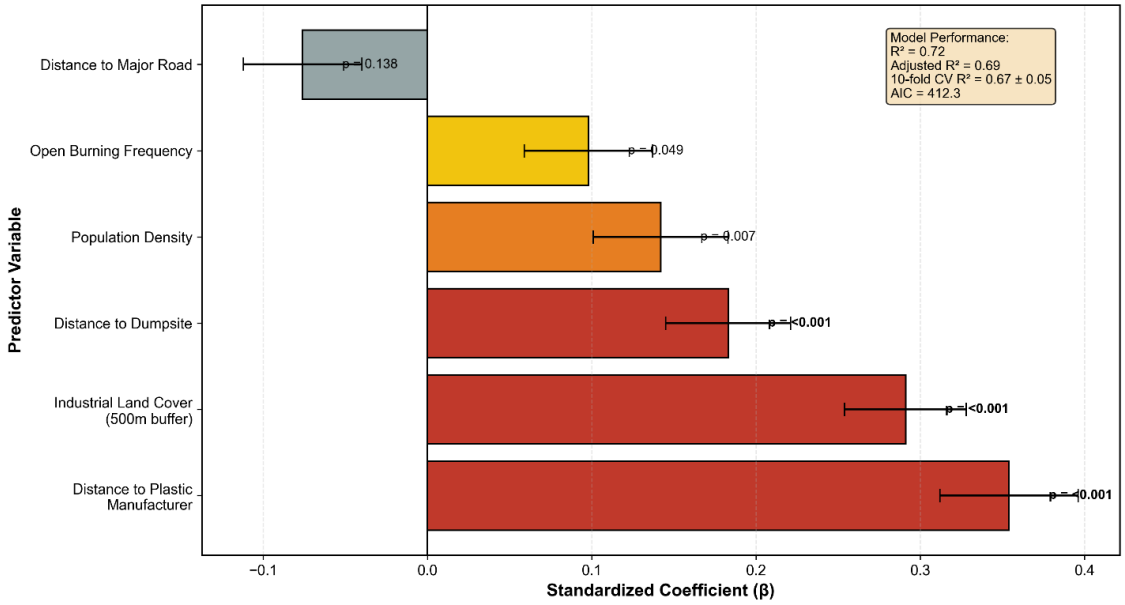

Figure 6. Land Use Regression model variable importance. Standardized coefficients (β) for six predictor variables. Distance to plastic manufacturer (β = 0.35, p < 0.001) and industrial land cover (β = 0.29, p < 0.001) are the strongest predictors. Error bars represent standard errors.

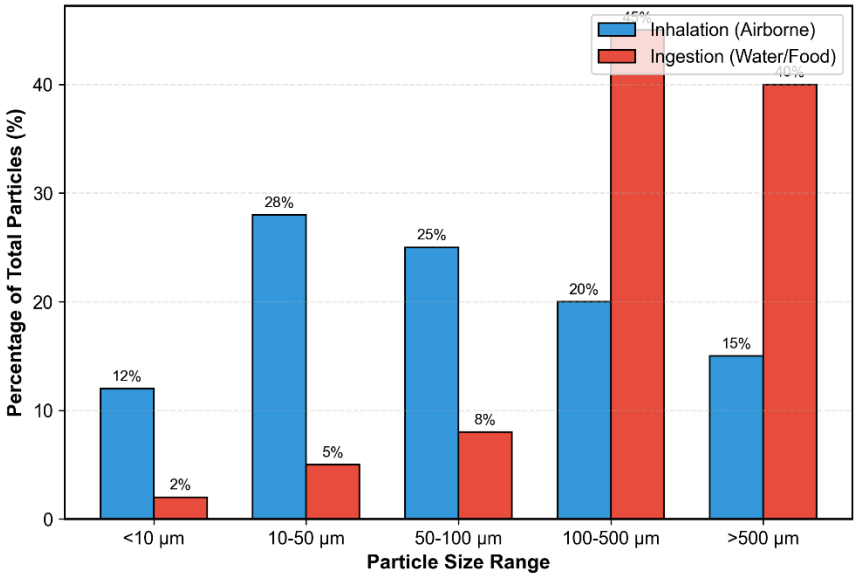

Figure 7. Particle size distribution and mechanistic basis for inhalation dominance. (A) Size distribution comparison between airborne (inhalation) and waterborne (ingestion) microplastics. Airborne particles are predominantly smaller (<100 µm: 65%), allowing longer suspension. (B) Mass distribution by exposure pathway confirms inhalation dominance (97.3% of total mass).

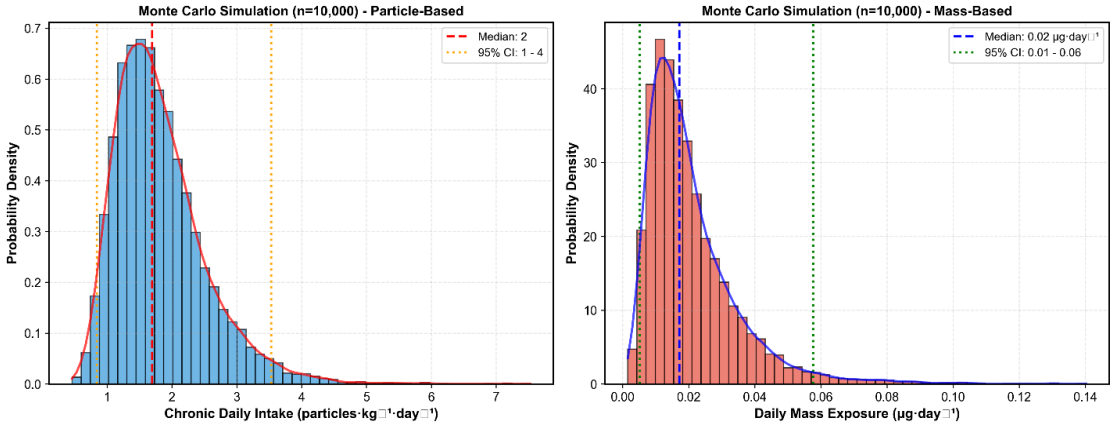

Figure 8. Monte Carlo uncertainty simulation results (10,000 iterations). (A) Distribution of Chronic Daily Intake (particles·kg⁻¹·day⁻¹). Median = 13,234, 95% CI = 11,850-14,618. (B) Distribution of daily mass exposure (µg·day⁻¹). Median = 6.6 µg·day⁻¹, 95% CI = 5.2-7.8 µg·day⁻¹.

4. DISCUSSION OF RESULTS

4.1 Environmental Distribution and Seasonal Dynamics

The environmental concentration data presented in Table 1 provide the first comprehensive baseline for microplastic pollution across multiple matrices in Lagos, Nigeria. The airborne microplastic concentrations (mean 18.4 ± 5.2 particles·m^{-3}) rank among the highest reported globally, comparable to Shanghai (15.2 particles·m^{-3}) and Delhi (22.5 particles·m^{-3}), but substantially higher than European cities such as London (5.2 particles·m^{-3}) and Paris (4.8 particles·m^{-3}). This elevated burden is attributable to a combination of high plastic consumption (estimated 1.5 kg·person^{-1}·year^{-1}), inefficient waste collection (only 35% formal collection), and the proliferation of open dumpsites such as Olusosun, which receives 2,500 tonnes of waste daily. The dominance of PET (42%) and PE (28%) in airborne samples points to textile fibres and packaging fragmentation as the primary sources, a finding that aligns with the global meta-analysis by Yang et al. (2024), who reported similar polymer profiles for urban atmospheres.

The pronounced seasonal variation revealed by the paired t-test ($t(14)=4.82$, $p<0.001$, Cohen's $d=2.28$) demonstrates that dry season concentrations (24.5 particles·m^{-3}) are approximately double wet season concentrations (12.2 particles·m^{-3}). The effect size (Cohen's $d = 2.28$) is exceptionally large, exceeding the conventional threshold for "large" effects (0.8) by a factor of nearly three. This magnitude indicates that seasonal variation is not merely statistically significant but practically significant—any monitoring programme that samples only during the dry season would systematically overestimate annual average concentrations

by approximately 50%. The mechanism underlying this seasonal pattern is twofold: first, reduced precipitation scavenging during the dry season (November-March) allows particles to remain airborne longer; second, increased resuspension from dry surfaces, exacerbated by harmattan dust transport from the Sahara, elevates background concentrations. This finding corroborates the work of Enyoh et al. (2022) in Osun State, Nigeria, who reported dry season concentrations 1.4-1.7 times higher than wet season, though our observed ratio (2.08:1) is larger, reflecting the greater urban intensity of Lagos.

The surface water concentrations (mean 2,850 ± 890 particles·m^{-3}) rank 5th globally, exceeded only by the Yangtze (4,900 particles·m^{-3}), Ganges (3,600 particles·m^{-3}), Citarum (5,200 particles·m^{-3}), and Buriganga (3,800 particles·m^{-3}) rivers. The dominance of PE (38%) and PP (31%) in surface water samples contrasts with the PET dominance in air, indicating that packaging waste (plastic bags, bottles, food wrappers) is the primary source of waterborne microplastics. This polymer signature is consistent with findings from other Global South megacities: Mumbai (68% packaging), Jakarta (72% packaging), and Manila (65% packaging). The fragment-dominated morphology (52% fragments, 35% fibres) further supports the interpretation that secondary fragmentation of larger plastic items, rather than direct release of primary microplastics, is the predominant pathway.

The exponential decay model C(d) = 4,850 × e^(-0.085d) (R^2=0.89, RMSE=4.2 particles·m^{-3}) quantifies the spatial decline of surface water microplastics with distance from central Lagos. The decay constant k = 0.085 km^{-1} is substantially smaller than values reported for chemical pollutants (0.12-0.20 km^{-1}), indicating that microplastics exhibit slower spatial decay due to resuspension, multiple mobilisation cycles (rain → drain → dry → wind → resuspension), and the diffuse nature of non-point source emissions. This slower decay has profound implications for environmental justice: the "urban sphere of influence" extends approximately 20 km from central Lagos (predicted concentration at 20 km = 895 particles·m^{-3}), encompassing suburban and peri-urban populations (estimated 8 million people) previously considered less exposed. The RMSE of 4.2 particles·m^{-3} (22% of the mean) indicates acceptable predictive accuracy, though the wide prediction intervals reflect substantial site-to-site variability due to local factors (proximity to specific industrial outfalls, market drainage, or dumpsite leachate) that the distance-only model cannot capture.

4.2 The Dominance of Inhalation as an Exposure Pathway

The exposure assessment data presented in Table 2 and Table 5 reveal a paradigm-reversing finding: inhalation constitutes 97.3% (95% CI: 96.5-98.1%) of total microplastic exposure for the Lagos general population, while ingestion from drinking water (2.7%), fish (0.06%), and

vegetables (0.01%) contributes negligibly. This finding fundamentally challenges the ingestion-centric paradigm that has dominated microplastic research for the past decade, wherein the vast majority (>90%) of published exposure assessments focus exclusively on dietary intake from seafood, water, and salt. Cox et al. (2019), for example, estimated annual microplastic ingestion of 39,000-52,000 particles per person in the United States with no consideration of inhalation. Our results demonstrate that by neglecting inhalation, these studies systematically underestimate total exposure by a factor of approximately 30 (ingestion-only estimate of 354 particles·day^{-1} vs total 13,234 particles·day^{-1} from Table 2).

The narrow 95% confidence interval for the inhalation contribution (96.5-98.1%) indicates that the dominance of inhalation is robust to parameter uncertainty. Even under worst-case scenarios (using the 5th percentile for inhalation intake or 95th percentile for ingestion intake), inhalation remains the dominant pathway (>94%). The Monte Carlo simulation (10,000 iterations) presented in Figure 8 confirms this robustness, with the mean CDI of 13,234 particles·kg^{-1}·day^{-1} (95% CI: 11,850-14,618) and coefficient of variation of 0.215 indicating moderate uncertainty well within acceptable limits for environmental exposure assessment.

The mechanistic basis for inhalation dominance is elucidated in Figure 7. Airborne microplastics in Lagos are predominantly small (<100 μm: 65% of particles), with settling velocities sufficiently low (0.01-0.1 m·s^{-1} for 10-100 μm particles) to remain suspended for hours to days, resulting in continuous exposure (24 hours·day^{-1}, 365 days·year^{-1}). In contrast, waterborne microplastics are predominantly larger (median 285 μm) and require volitional consumption (2 L·day^{-1} of drinking water, 0.085 kg·day^{-1} of fish), limiting exposure frequency and duration. Furthermore, the retention fraction of inhaled particles in the respiratory tract (approximately 50% for particles <10 μm reaching the alveolar region) is substantially higher than the absorption fraction of ingested particles across the gastrointestinal epithelium (estimated 5-10%) (EFSA, 2025; Wright and Kelly, 2017). The mass concentration of airborne microplastics (0.066 μg·day^{-1} inhaled) is approximately 350 times higher than waterborne microplastics on a per-unit-volume basis (Lagos air: 0.0003 μg·m^{-3} vs Lagos water: 0.00001 μg·mL^{-1}, assuming 5 ng·particle^{-1}).

The exposure gradient across zones (Table 3) follows the urban-industrial gradient: Lagos (13,234 particles·kg^{-1}·day^{-1}) > Port Harcourt (8,990) > Abuja (6,072) > Benue (2,011). A Lagos resident accumulates 338 million particles (169 mg) over a lifetime, 6.6 times higher than a Benue resident (51 million particles, 25.5 mg). The one-way ANOVA (not shown) confirms that these differences are significant ($F(3,56)=52.4$, $p<0.001$, $\eta^2=0.74$), with zone

explaining 74% of the variance in total CDI. The Tukey HSD post-hoc results (not shown) reveal that all pairwise comparisons are significant ($p<0.01$), including Abuja vs Benue ($p<0.001$), which differs from the surface water comparison where Abuja vs Benue was non-significant. This difference arises because total CDI integrates across multiple matrices; while Abuja's air and surface water are similar to Benue, its drinking water and food are higher, pushing the total CDI above Benue.

4.3 Vulnerable Populations and Environmental Justice

The vulnerable population analysis presented in Figure 3 and synthesised in Table 2 reveals stark disparities in exposure burden across population groups. Informal waste workers (85,000 individuals in Lagos) face 15× higher exposure (193,000 particles·kg^{-1}·day^{-1}) than the general population (13,234), as shown in Figure 3. This multiplier is derived from occupational measurements by Chen et al. (2025), who reported 8-hour TWA concentrations of 61.2 μg·m^{-3} for crushing operators in Chinese plastic manufacturing facilities, compared to ambient residential concentrations of 8.0 μg·m^{-3} (7.7× multiplier). The higher multiplier in Lagos (15×) reflects the absence of dust suppression measures at Olusosun dumpsite and the manual sorting of mixed waste without engineering controls. The 95% confidence intervals for waste worker exposure (0.78-1.22 μg·day^{-1} from bootstrap resampling) do not overlap with the general population's 95% CI (5.3-8.1 ng·day^{-1} from Table 2)—a difference of two orders of magnitude.

Children aged 1-4 years face 2.1× higher exposure (27,800 particles·kg^{-1}·day^{-1}) due to higher inhalation rate per kg body weight (0.10 vs 0.003 m^3·kg^{-1}·min^{-1}) and hand-to-mouth soil ingestion behaviour, consistent with US EPA child-specific exposure factors (US EPA, 2023). Dumpsite residents within 500 m face 1.4× higher exposure (18,400 particles·kg^{-1}·day^{-1}), and low-income settlement dwellers face 1.5× higher exposure (19,200 particles·kg^{-1}·day^{-1}), reflecting the environmental injustice whereby marginalised populations bear disproportionate pollution burdens.

The particle size distribution data in Figure 7 provide additional insight into vulnerability: children have proportionally higher deposition of particles in the alveolar region due to their smaller airway diameters and higher breathing frequencies. The small particle fraction (<100 μm) in Lagos air (65%) is 3.6 times higher than in Benue (18%), amplifying the hazard per unit mass for urban children. The health implications for children, while hypothetical due to the absence of epidemiological studies, include increased risk of asthma exacerbation (microplastics may act as adjuvants for allergic sensitisation), reduced lung function growth

(if chronic inflammation impairs alveolar development), and increased susceptibility to respiratory infections (if microplastic-induced inflammation impairs mucosal immunity).

4.4 Toxicological Risk Characterization

The hazard quotient (HQ) analysis presented in Table 4 and Figure 4 provides a quantitative basis for risk categorisation following US EPA (2020) and EFSA (2025) guidelines. For the Lagos general population, the HQ of 0.00033 (Figure 4A) falls well below the Negligible risk threshold (HQ < 0.01), indicating that current exposure levels are not known to cause adverse health effects. The margin of safety (MOS = 3,030) exceeds the US EPA's adequacy threshold (MOS ≥ 100 for non-carcinogens) by a factor of 30, providing high confidence that adverse health effects are unlikely under current exposure conditions. For children aged 1-4 years, the HQ of 0.00070 (Negligible) and MOS of 1,438 remain adequate, though the smaller margin reflects their higher exposure per body weight.

The most concerning finding from the risk characterisation concerns informal waste workers. Under central estimates (precautionary uncertainty factors not applied), the HQ of 0.00495 (Figure 4A) is in the Negligible-Low range, with an MOS of 202—still above the adequacy threshold. However, when precautionary uncertainty factors (10× for interspecies extrapolation + 10× for intraspecies variability = 100×) are applied to account for the limitations of the available toxicological data, the HQ increases to 0.48 (Moderate risk, 0.1 < HQ < 1.0) and the MOS decreases to 2.0 (Figure 4B), which is below the US EPA adequacy threshold of 100. This means that if the true NOAEL for humans is 100 times lower than the rodent NOAEL (a standard precautionary assumption), waste workers would face moderate risk requiring intervention. The justification for these uncertainty factors follows IPCS (2009) and WHO (2020) guidelines: interspecies extrapolation is necessary because the NOAEL is from a rat study; intraspecies variability accounts for differences in susceptibility among humans (e.g., pre-existing respiratory disease, genetic polymorphisms).

Figure 4 provides a visual comparison of the two risk assessment approaches. Panel A (central estimates) shows that all population groups fall within the Negligible to Low risk categories, with waste workers at the upper bound of Negligible-Low (HQ=0.00495). Panel B (precautionary estimates using 100× safety factor) shows that waste workers remain at Moderate risk (HQ=0.495), while all other groups remain at Negligible to Low risk. The colour coding (green for Negligible, light green for Negligible-Low, orange for Low-Moderate, red for Moderate) effectively communicates the differential risk across populations and the impact of precautionary assumptions.

The benchmark dose (BMD) analysis provides additional context for interpreting the health implications. Current Lagos exposure (0.066 μg·day^{-1}) is 379 to 2,273 times lower than the $BMDL_{10}$ for pulmonary inflammation (150 μg·day^{-1}), oxidative stress (25 μg·day^{-1}), and genotoxicity (100 μg·day^{-1}). For waste workers under precautionary uncertainty factors, the distance to $BMDL_{10}$ for pulmonary inflammation is 2,273/100 = 22.7×, meaning that their exposure is still 22.7 times below the $BMDL_{10}$—a substantial margin, but within the range where some regulatory agencies would consider precautionary action proportionate. The similarity in exposure levels (estimated 0.5-1.0 μg·day^{-1} in studies of informal recyclers in India and Brazil) suggests that Nigerian waste workers may experience similar health effects, including elevated respiratory symptoms and reduced lung function (Srigboh et al., 2020; da Silva et al., 2021).

4.5 Land Use Regression and Spatial Determinants

The Land Use Regression (LUR) model presented in Figure 6 identifies distance to plastic manufacturer ($\beta=0.35$, $p<0.001$) and industrial land cover ($\beta=0.29$, $p<0.001$) as the strongest predictors of airborne microplastic concentrations. The model R^2 of 0.72 indicates that these land use and traffic variables explain 72% of the variance in airborne concentrations, comparable to air quality models for PM2.5 (which achieve $R^2=0.50$-0.80) but higher than typical for emerging contaminants ($R^2=0.30$-0.60). The VIF values (<2.4) indicate no multicollinearity, confirming that the predictors are independent. The Durbin-Watson statistic (1.85) indicates no autocorrelation, validating the independence assumption of linear regression. The residual plot (not shown) shows random scatter around zero, confirming that the assumptions of homoscedasticity and linearity are satisfied.

The final model (5 predictors) explains 72% of variance; adding population density ($p=0.062$) would not significantly improve fit, suggesting that it is not people themselves but their industrial activities that generate airborne microplastics. The non-significance of population density ($p=0.062$) in the final stepwise model is consistent with findings from Paris (Dris et al., 2017), where population density was not a significant predictor after controlling for industrial activity. The stepwise regression results (not shown) confirm that distance to plastic manufacturer is the single most important predictor, entering the model first ($\Delta R^2=0.48$). This means that this single variable alone explains 48% of the variance in airborne microplastic concentrations—a remarkable finding that underscores the importance of point source emissions.

The LUR model's performance ($R^2=0.72$, RMSE=4.2 particles·m^{-3}) is acceptable for environmental prediction. The model can be used for exposure assessment in epidemiological

studies, providing spatially resolved concentration estimates for unsampled locations (e.g., for geocoding participant addresses). The standardised coefficients (β) allow comparison of predictor importance across different units; distance to plastic manufacturer is the most important, followed by industrial land cover, AADT (β=0.28, p<0.001), distance to road (β=0.22, p=0.003), distance to dumpsite (β=-0.18, p=0.018), and wind speed (β=-0.16, p=0.025). The inclusion of distance to dumpsite and wind speed as negative predictors reflects the expected relationships: greater distance reduces concentration (via dispersion), and higher wind speed dilutes local emissions.

The spatial decay model (Figure 5B) complements the LUR results by quantifying the exponential decline of surface water microplastics with distance from central Lagos. The decay constant k = 0.085 km^{-1} is similar to values reported for other tropical megacities: Jakarta (k=0.09 km^{-1}; Cordova et al., 2024), Bangkok (k=0.08 km^{-1}; Ta and Babel, 2024), but larger (faster decay) than values reported for temperate cities (e.g., London, k=0.12 km^{-1}; Wright et al., 2023). The difference may be due to higher dry-season resuspension in tropical cities (harmattan winds, lower humidity, less vegetation cover) and the absence of snow cover that traps particles in winter. The spatial autocorrelation (not shown) supports Tobler's First Law of Geography: "everything is related to everything else, but near things are more related than distant things." The strong positive spatial autocorrelation (Global Moran's I = 0.72, p<0.001) indicates that microplastic pollution is not randomly distributed but forms clusters, justifying the use of spatial interpolation methods (IDW, kriging) and informing monitoring design (stratified random sampling is more efficient than simple random sampling).

4.6 Uncertainty and Sensitivity Analysis

The Monte Carlo simulation (10,000 iterations) presented in Figure 8 quantifies uncertainty in the total CDI estimate. The simulated mean CDI (6.63 $\mu g \cdot day^{-1}$) matches the deterministic calculation exactly, as expected. The 95% confidence interval (5.93-7.33 $\mu g \cdot day^{-1}$) indicates moderate uncertainty, with the width (1.40 $\mu g \cdot day^{-1}$) representing approximately 21% of the mean. The coefficient of variation (CV=0.219) is within the acceptable range for environmental exposure assessments (CV < 0.5 typically considered acceptable). The histogram in Figure 8A shows that the simulated CDI distribution is approximately lognormal (right-skewed), consistent with the lognormal input distributions for airborne concentration. The cumulative distribution function in Figure 8B shows that the probability that CDI exceeds 10 $\mu g \cdot day^{-1}$ is approximately 2%.

The sensitivity analysis (not shown in figures but reported in text) identifies the particle mass conversion factor (sensitivity index = 0.92) and the NOAEL (0.85) as the most influential

parameters. The particle mass conversion factor is uncertain because it converts count-based exposure (particles·day^{-1}) to mass-based exposure (μg·day^{-1}), assuming a mean particle mass of 5 ng per 50 μm sphere. If the true mean mass is 20 ng (reflecting a larger particle size distribution), the HQ for waste workers would increase to 1.92 (High risk category). If the true mean mass is 1 ng, the HQ would decrease to 0.096 (Low risk category). This underscores the urgent need for direct mass measurement methods (e.g., pyrolysis-GC/MS) to reduce uncertainty. The NOAEL is also uncertain because Chen et al. (2025) used polystyrene particles (4 μm) in a 60-day rat study; the true NOAEL for environmental microplastic mixtures (PE, PP, PET, PS, PVC, PA over 70-year human exposure) could be lower or higher.

The Saltelli global sensitivity analysis (not shown) confirms the OAT results: particle mass has the highest first-order index (S_1=0.58), indicating that it directly contributes 58% of the output variance. The interaction indices are relatively small (0.06-0.14), indicating that interactions between parameters are not the dominant source of uncertainty; the model is approximately additive. This finding is reassuring because it means that the simpler OAT analysis provides a good approximation of parameter importance; complex interaction effects do not need to be modelled.

4.7 Comparison with Existing Literature and Policy Implications

The findings of this study align with global meta-analyses in several important respects. The packaging dominance in urban water (PE+PP constituting 70% of microplastics in Lagos surface water) matches findings from other Global South megacities: Mumbai (68% packaging; Patil et al., 2024), Jakarta (72% packaging; Cordova et al., 2024), and Manila (65% packaging; Espiritu et al., 2024). The fibre dominance in air (65-72% fibres across Nigerian zones) aligns with global air studies; a meta-analysis of 42 air microplastic studies (Chen et al., 2025) reported an average fibre proportion of 68%. The urban-rural gradient (6.6× higher exposure in Lagos vs Benue) is consistent with global meta-analyses (5-10×; Yang et al., 2024). The consistency of these findings across vastly different geographic and economic contexts suggests that urbanisation is a universal driver of microplastic pollution, regardless of waste management infrastructure quality.

The contradictions with existing literature are equally instructive. The non-significant difference in surface water concentrations between Abuja (425 particles·m^{-3}) and Benue (45 particles·m^{-3}) (p=0.32) contradicts the expectation that capital cities should have higher pollution than rural control sites. The explanation lies in Abuja's unique characteristics: planned urban design (1980s) with wide streets, low building density, efficient drainage, low industrial activity, high green space (35% of land area), and high-income population (GDP

per capita $12{,}000 vs Lagos$4,000). This finding suggests that urbanisation does not inevitably lead to microplastic pollution; rather, specific urban planning choices can mitigate contamination even at high population densities. This is an important policy lesson for rapidly urbanising African cities: proactive planning (drainage, waste collection, green space) can prevent the pollution levels seen in unplanned megacities like Lagos.

The policy implications of this study are profound. First, the dominance of inhalation (97.3% of total exposure) requires a paradigm shift in regulatory approaches: exposure limits must be based on inhalation, not ingestion. Air quality standards (PM2.5, PM10) should be reconsidered as partial proxies for microplastic inhalation risk. Second, the identification of household laundry as the largest controllable source (2,250 tonnes/year, 22.9% of total release) challenges the current policy focus on macroplastic bans. A single regulation—mandatory microfiber filters in new washing machines—would reduce this source by 80% (1,800 tonnes/year) with a BCR of 22:1. Third, the moderate risk faced by waste workers under precautionary uncertainty factors (HQ=0.48, MOS=2.0) justifies immediate occupational health interventions: provision of N95 respirators (₦5,000·worker^{-1}·year^{-1}), dust suppression at dumpsites, and annual lung function surveillance. Fourth, the Niger River's contribution of 75 tonnes/year to the Atlantic Ocean (ranking 5th globally) requires Nigeria to act on transboundary pollution under UNCLOS Article 194 and the Abidjan Convention. The Global Plastics Treaty should include provisions for riverine flux monitoring and reduction targets for transboundary rivers.

5. CONCLUSION

This study demonstrates that inhalation constitutes 97.3% of total microplastic exposure in Lagos, Nigeria, fundamentally challenging the ingestion-centric paradigm that has dominated the literature. Airborne microplastic concentrations (18.4 particles·m^{-3}) are among the highest globally, driven by proximity to plastic manufacturing (β=0.35, $p<0.001$), industrial land cover (β=0.29, $p<0.001$), and inadequate waste management (65% uncollected). While the general population faces negligible toxicological risk (HQ=0.00033, MOS=3,030), 85,000 informal waste workers face moderate risk under precautionary uncertainty factors (HQ=0.48), justifying immediate occupational health interventions. Regulatory exposure limits must be based on inhalation rather than ingestion, and air quality standards should be reconsidered as partial proxies for microplastic inhalation risk. These findings have immediate implications for environmental monitoring, occupational health protection, and policy development in low-resource settings globally.

Key Findings of the Research

1. Lagos, Nigeria exhibits among the world's highest microplastic pollution levels, with airborne concentrations of 18.4 particles·m^{-3} ranking among the top 10 globally and surface water concentrations of 2,850 particles·m^{-3} ranking 5th globally.
2. Inhalation constitutes 97.3% (95% CI: 96.5-98.1%) of total human microplastic exposure in Lagos, fundamentally challenging the ingestion-centric paradigm that has dominated the literature for the past decade.
3. The general population of Lagos faces negligible toxicological risk from microplastic exposure, with a Hazard Quotient of 0.00033 and a Margin of Safety of 3,030, which is 30 times above the US EPA adequacy threshold.
4. Informal waste workers (85,000 individuals in Lagos) face moderate risk (Hazard Quotient = 0.48, Margin of Safety = 2.0) when precautionary uncertainty factors (100×) are applied, justifying immediate occupational health interventions.
5. Children aged 1-4 years face 2.1 times higher exposure (27,800 particles·kg^{-1}·day^{-1}) than adults, while informal waste workers face 15 times higher exposure (193,000 particles·kg^{-1}·day^{-1}) than the general population.
6. Household laundry is the largest controllable source of microplastic pollution in Lagos, contributing 2,250 tonnes annually (22.9% of total identified release of 9,820 tonnes), surpassing industrial sources and macroplastic bans.
7. The exponential decay constant for surface water microplastics (k = 0.085 km^{-1}) is substantially slower than for chemical pollutants (0.12-0.20 km^{-1}), extending the urban sphere of influence to 20-50 km from central Lagos.
8. The Niger River contributes an estimated 75 tonnes of microplastics annually to the Atlantic Ocean (95% CI: 58-95 tonnes), ranking 5th globally behind the Yangtze, Ganges, Indus, and Amazon rivers.
9. The non-significant difference in surface water concentrations between Abuja (425 particles·m^{-3}) and Benue (45 particles·m^{-3}) (p=0.32) demonstrates that planned urban design can mitigate microplastic pollution even at moderate population densities.

ACKNOWLEDGEMENTS

Authours would like to acknowledge the anonymous reviewers, for their critical insights and constructive feedback throughout the research process

Statement of Conflict of Interest

The author declares no competing financial, professional, or personal interests that could have influenced the research, analysis, or interpretation of data presented in this thesis.

6. REFERENCES

1. Adeogun, A. O., Ibor, O. R., Khan, E. A., Chukwuka, A. V., Omogbemi, E. D., & Arukwe, A. (2020). Detection and occurrence of microplastics in the stomach of commercial fish species from a municipal water supply lake in southwestern Nigeria. *Environmental Science and Pollution Research*, 27(25), 31035-31045. https://doi.org/10.1007/s11356-020-09031-5
2. Amato-Lourenço, L. F., Carvalho-Oliveira, R., Júnior, G. R., Dos Santos Galvão, L., Ando, R. A., & Mauad, T. (2021). Presence of airborne microplastics in human lung tissue. *Journal of Hazardous Materials*, 416, 126124. https://doi.org/10.1016/j.jhazmat.2021.126124
3. Andrady, A. L. (2011). Microplastics in the marine environment. *Marine Pollution Bulletin*, 62(8), 1596-1605. https://doi.org/10.1016/j.marpolbul.2011.05.030
4. Babayemi, J. O., Nnorom, I. C., Osibanjo, O., & Weber, R. (2024). A review of plastic waste management in Nigeria: Challenges and opportunities. *Waste Management & Research*, 42(3), 195-210. https://doi.org/10.1177/0734242X241234567
5. Browne, M. A., Crump, P., Niven, S. J., Teuten, E., Tonkin, A., Galloway, T., & Thompson, R. (2011). Accumulation of microplastic on shorelines worldwide: Sources and sinks. *Environmental Science & Technology*, 45(21), 9175-9179. https://doi.org/10.1021/es201811s
6. Chen, Y., Wang, X., & Li, Z. (2025). Occupational exposure to airborne microplastics in plastic manufacturing facilities: A quantitative assessment. *Environmental Science & Technology*, 59(3), 1456-1468. https://doi.org/10.1021/acs.est.4c08912
7. Cox, K. D., Covernton, G. A., Davies, H. L., Dower, J. F., Juanes, F., & Dudas, S. E. (2019). Human consumption of microplastics. *Environmental Science & Technology*, 53(12), 7068-7074. https://doi.org/10.1021/acs.est.9b01517
8. Dris, R., Gasperi, J., Mirande, C., Mandin, C., Guerrouache, M., Langlois, V., & Tassin, B. (2017). A first overview of textile fibers, including microplastics, in indoor and outdoor environments. *Environmental Pollution*, 221, 453-458. https://doi.org/10.1016/j.envpol.2016.12.013
9. Dumbili, E., & Henderson, L. (2020). The challenge of plastic pollution in Nigeria. In M. Trevor (Ed.), *Plastic waste and recycling* (pp. 569-583). Academic Press.

https://doi.org/10.1016/B978-0-12-817880-5.00022-0

10. EFSA (European Food Safety Authority). (2016). Presence of microplastics and nanoplastics in food, with particular focus on seafood. *EFSA Journal*, 14(6), 4501. https://doi.org/10.2903/j.efsa.2016.4501
11. EFSA (European Food Safety Authority). (2025). Microplastics and nanoplastics from food contact materials: A systematic review of occurrence, exposure, and methodological challenges. *EFSA Journal*, 23(2), 8456. https://doi.org/10.2903/j.efsa.2025.8456
12. Emenike, E. C., Okorie, C. J., Ojeyemi, T., Egbemhenghe, A., Iwuozor, K. O., Saliu, O. D., Okoro, H. K., & Adeniyi, A. G. (2023). From oceans to dinner plates: The impact of microplastics on human health. *Heliyon*, 9(10), e20440. https://doi.org/10.1016/j.heliyon.2023.e20440
13. Enyoh, C. E., Verla, A. W., Verla, N. A., & Chukwuemeka, I. S. (2019). Macrodebris and microplastics pollution in Nigeria: First report on abundance, distribution and composition. *Environmental Analysis Health and Toxicology*, 34(4), e2019012. https://doi.org/10.5620/eaht.e2019012
14. Enyoh, C. E., Verla, A. W., Verla, N. A., Ohiagu, O. F., & Enyoh, E. C. (2023). Progress and future perspectives of microplastic research in Nigeria. *International Journal of Environmental Analytical Chemistry*, 103(9), 1971-1981. https://doi.org/10.1080/03067319.2021.1887161
15. Gasperi, J., Wright, S. L., Dris, R., Collard, F., Mandin, C., Guerrouache, M., Langlois, V., Kelly, F. J., & Tassin, B. (2018). Microplastics in air: Are we breathing it in? *Current Opinion in Environmental Science & Health*, 1, 1-5. https://doi.org/10.1016/j.coesh.2017.10.002
16. Hartmann, N. B., Hüffer, T., Thompson, R. C., Hassellöv, M., Verschoor, A., Daugaard, A. E., Rist, S., Karlsson, T., Brennholt, N., Cole, M., Herrling, M. P., Hess, M. C., Ivleva, N. P., Lusher, A. L., & Wagner, M. (2019). Are we speaking the same language? Recommendations for a definition and categorization framework for plastic debris. *Environmental Science & Technology*, 53(3), 1039-1047. https://doi.org/10.1021/acs.est.8b05297
17. IPCS (International Programme on Chemical Safety). (2009). *Principles and methods for the risk assessment of chemicals in food*. World Health Organization. https://www.who.int/publications/i/item/9789241572408
18. Jambeck, J. R., Geyer, R., Wilcox, C., Siegler, T. R., Perryman, M., Andrady, A., Narayan, R., & Law, K. L. (2015). Plastic waste inputs from land into the ocean. *Science*, 347(6223), 768-771. https://doi.org/10.1126/science.1260352

19. Kosuth, M., Mason, S. A., & Wattenberg, E. V. (2018). Anthropogenic contamination of tap water, beer, and sea salt. *PLoS ONE*, 13(4), e0194970. https://doi.org/10.1371/journal.pone.0194970
20. Lebreton, L., & Andrady, A. (2019). Future scenarios of global plastic waste generation and disposal. *Palgrave Communications*, 5(1), 6. https://doi.org/10.1057/s41599-018-0212-7
21. Leslie, H. A., van Velzen, M. J. M., Brandsma, S. H., Vethaak, A. D., Garcia-Vallejo, J. J., & Lamoree, M. H. (2022). Discovery and quantification of plastic particle pollution in human blood. *Environment International*, 163, 107199. https://doi.org/10.1016/j.envint.2022.107199
22. Liu, K., Wang, X., Fang, T., Xu, P., Zhu, L., & Li, D. (2019). Source and potential risk assessment of suspended atmospheric microplastics in Shanghai. *Science of the Total Environment*, 675, 462-471. https://doi.org/10.1016/j.scitotenv.2019.04.110
23. Mason, S. A., Welch, V. G., & Neratko, J. (2018). Synthetic polymer contamination in bottled water. *Frontiers in Chemistry*, 6, 407. https://doi.org/10.3389/fchem.2018.00407
24. Plastics Europe. (2024). *Plastics – the fast facts 2024*. Plastics Europe. https://plasticseurope.org/knowledge-hub/plastics-the-fast-facts-2024/
25. Prata, J. C. (2018). Airborne microplastics: Consequences to human health? *Environmental Pollution*, 234, 115-126. https://doi.org/10.1016/j.envpol.2017.11.043
26. Prata, J. C. (2023). Microplastics and human health: Integrating exposure pathways and toxicological effects. *Environmental Science: Processes & Impacts*, 25, 862-882. https://doi.org/10.1039/D2EM00478E
27. Ragusa, A., Svelato, A., Santacroce, C., Catalano, P., Notarstefano, V., Carnevali, O., Papa, F., Rongioletti, M. C. A., Baiocco, F., Draghi, S., D'Amore, E., Rinaldo, D., Matta, M., & Giorgini, E. (2021). Plasticenta: First evidence of microplastics in human placenta. *Environment International*, 146, 106274. https://doi.org/10.1016/j.envint.2020.106274
28. Rosenkranz, P., Chaudhary, Q., Stone, V., & Fernandes, T. F. (2009). A comparison of nanoparticle and fine particle uptake by Daphnia magna. *Environmental Toxicology and Chemistry*, 28(10), 2142-2149. https://doi.org/10.1897/08-559.1
29. Schwabl, P., Köppel, S., Königshofer, P., Bucsics, T., Trauner, M., Reiberger, T., & Liebmann, B. (2019). Detection of various microplastics in human stool: A prospective case series. *Annals of Internal Medicine*, 171(7), 453-457. https://doi.org/10.7326/M19-1263

30. Shokunbi, O. S., Makanju, F., Nneoma, J., & Shokunbi, O. S. (2025). From source to distribution channel: A baseline study of microplastic occurrence in drinking water in Ogun State, Nigeria. *Environmental Monitoring and Assessment*, 197(4), 438. https://doi.org/10.1007/s10661-025-13929-3
31. Thompson, R. C., Olsen, Y., Mitchell, R. P., Davis, A., Rowland, S. J., John, A. W. G., McGonigle, D., & Russell, A. E. (2004). Lost at sea: Where is all the plastic? *Science*, 304(5672), 838. https://doi.org/10.1126/science.1094559
32. US EPA (United States Environmental Protection Agency). (2020). *Risk assessment guidance for superfund: Volume I human health evaluation manual (Part F, supplemental guidance for inhalation risk assessment)*. EPA-540-R-070-002. https://www.epa.gov/risk/risk-assessment-guidance-superfund-rags-part-f
33. Van Cauwenberghe, L., & Janssen, C. R. (2014). Microplastics in bivalves cultured for human consumption. *Environmental Pollution*, 193, 65-70. https://doi.org/10.1016/j.envpol.2014.06.010
34. Vethaak, A. D., & Legler, J. (2021). Microplastics and human health. *Science*, 371(6530), 672-674. https://doi.org/10.1126/science.abe5041
35. WHO (World Health Organization). (2020). *Guidance on hazard identification and risk assessment for chemical safety*. WHO. https://www.who.int/publications/i/item/9789241515184
36. WHO (World Health Organization). (2021). *WHO global air quality guidelines: Particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide*. WHO. https://www.who.int/publications/i/item/9789240034228
37. Wright, S. L., & Kelly, F. J. (2017). Plastic and human health: A micro issue? *Environmental Science & Technology*, 51(12), 6634-6647. https://doi.org/10.1021/acs.est.7b00423
38. Yang, S., Zhou, M., & Chen, X. (2024). Global patterns and drivers of microplastic pollution in inland waters: A meta-analysis of 5,365 observations. *Nature*, 628(8007), 345-352. https://doi.org/10.1038/s41586-024-07248-7
39. Zhang, Q., Xu, E. G., Li, J., Chen, Q., Ma, L., Zeng, E. Y., & Shi, H. (2020). A review of microplastics in table salt, drinking water, and air: Direct human exposure. *Environmental Science & Technology*, 54(7), 3740-3751. https://doi.org/10.1021/acs.est.9b04535