

# Microplastics in human lung tissue from autopsy samples collected in 1991 and 2024: a comparative post-mortem study



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## Summary

**Background** Environmental accumulation and human exposure to microplastics MPs are increasing. It is not known whether there is a higher bioaccumulation of microplastics (MPs) in human lungs collected recently and compared with those collected decades ago. We aimed to assess changes in the presence, abundance, size, and types of MPs in human lung tissue collected 33 years apart, and to characterize histological patterns in these samples.

**Methods** We performed a comparative study of paraffin-embedded lung tissues collected post-mortem from two series of autopsy cases deceased either in 1991 (n = 42) or in 2024 (n = 57), both from the same tertiary care medical center. We assessed MPs prevalence, abundance, size, and polymer type using laser-direct infrared (LDIR) imaging. Particle count per gram of tissue was the primary endpoint. As supporting analysis, we evaluated MPs type and concentrations through pyrolysis–gas chromatography/mass spectrometry (Py-GC/MS) in a representative subset of samples. We also assessed collagen I staining to evaluate fibrosis and CD68 infiltration as an index of lung inflammation.

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**Findings** MPs were identified by LDIR in lung samples from 8/42 individuals (19%) deceased in 1991 and 44/57 (77%) deceased in 2024. MP burden per gram of tissue (median; Q1-Q3) increased from 12.9 particles (11.3–13.7) to 19.9 (15.5–28.2), with results corroborated by a two-part hurdle model analysis. Particle diameter decreased from 56.2  $\mu\text{m}$  (44.9–73.4) to 22.4  $\mu\text{m}$  (15.4–37.9). When considering the mean  $\pm$  SD of the relative abundance (%) of each plastic type/total MPs for each individual positive to MPs in the two groups, polymer composition shifted from polyethylene predominance ( $51.7 \pm 2\%$ ) in 1991 to a more heterogeneous mix including polyethylene terephthalate ( $28.4 \pm 3.1\%$ ), polyvinyl chloride ( $14.3 \pm 2.4\%$ ), and polystyrene ( $14.2 \pm 2.6\%$ ) in 2024. Py-GC/MS provided similar results in terms of MPs prevalence and relative abundance, while SEM suggested a pervasive presence of particles enriched in Sulphur in the 2024 group. Lung samples with evidence of MPs showed higher CD68 and collagen I abundance compared with those without evidence of MPs, independently of the case series.

**Interpretation** MPs contamination of human lung tissue was more frequently detected, showed greater polymer diversity, and was composed of smaller particles in post-mortem samples collected in 2024 compared with those collected in 1991. These observations are hypothesis-generating and should be interpreted in the context of the observational, cross-sectional, and retrospective nature of the study design.

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**Keywords:** Plastics pollution; Nanoplastics; Lung; Accumulation; LDIR; Py-GC/MS; Inflammation; Fibrosis

#### Research in context

##### Evidence before this study

Plastics waste in the environment promotes the formation of microplastics (MPs) and nanoplastics, *i.e.*, particles in the micro and nano range, which ambient levels are held to raise over time across different ecosystems. Whether the accumulation of MPs in human organs is increasing has been poorly investigated. We searched PubMed using terms such as “microplastics accumulation”, or “microplastics trend”, and “human” up to February 2026, with no restriction relative to language and not setting any inclusion or exclusion criteria. We found only two pertinent studies and we did not evaluate their quality. One study evidenced a higher burden of MPs in brain and liver samples collected from individuals deceased in 2024 and compared with subjects deceased in 2016, while the other study observed an increase in the prevalence of MPs in placentas collected in 2006, 2013 and 2021. To our knowledge, no study compared the prevalence and abundance of MPs in lung samples from people deceased 33 years apart.

##### Added value of this study

In this study, we provide evidence of a substantial increase in the prevalence and burden of MPs in human lung tissues collected from people deceased in 2024, compared with individuals deceased in 1991. We also observed a reduction in particles diameter and a shift in polymer composition to a more heterogeneous profile, suggesting evolving patterns of environmental exposure. Our study is the first in demonstrating a higher accumulation of MPs in the lungs when comparing samples with a temporal gap larger than any previously published study and using multiple technologies.

##### Implications of all the available evidence

Even though additional studies employing a greater number of samples, time points, and approaches in geographically diverse populations are needed to firmly establish a general trend in the accumulation of MPs in human organs, available evidence suggests an increasing bioaccumulation. Systematic biomonitoring of MPs also in living individuals is warranted to explore the long-term health consequences of exposure to plastic-related pollution.

#### Introduction

Plastics are pervasive in modern life. However, the wide and increasing use of plastics has resulted in extensive plastic pollution.<sup>1</sup> Plastic waste released into the environment degrades to produce microplastics (MPs) and nanoplastics, fragments smaller than 5 mm and 1  $\mu\text{m}$ , respectively. MPs are identified today in ecosystems

worldwide.<sup>2</sup> Recently, MPs have been detected in many human organs. In selected cases, they are associated with adverse clinical outcomes.<sup>3–11</sup>

Plastic production has increased almost six-fold since the early 1990s, reaching an annual rate of nearly 600 million metric tons at present.<sup>1,12</sup> This increasing output is associated with progressively rising levels of MPs in

multiple ecosystems.<sup>1,13,14</sup> Preliminary evidence suggests that human tissues derived from contemporary cohorts present higher levels of MPs compared to samples collected in the past.<sup>4,5,15</sup> However, some of these results are debated mainly due to possible methodological challenges in assessing MPs in human brain samples with pyrolysis–gas chromatography/mass spectrometry (Py-GC/MS).<sup>16</sup> The lung, continuously exposed to inhaled particles, is a critical site for assessing human exposure to pollution, representing a potential reservoir for airborne contaminants including MPs.<sup>17</sup> However, no study to date has compared the pulmonary burden, composition, and size of MPs in lung samples collected from people deceased three decades apart.

To address this question, we conducted a comparative analysis of post-mortem lung specimens from two autopsy case series collected in 1991 and in 2024, respectively. Using laser-direct infrared (LDIR) imaging, we assessed MPs number, size distribution, and polymer composition in the two case series. To partly support findings obtained from LDIR imaging, we analyzed a subset of 20 random samples, *i.e.*, 10 from each case series, with Py-GC/MS. We also tested this subset with scanning electron microscopy (SEM) as an ancillary and exploratory analysis. This orthogonal, multi-technique strategy provides converging evidence by combining mass-based polymer identification with particle-based chemical imaging and morphological characterization, consistent with recent recommendations to communicate analytical confidence in the identification of human MPs.<sup>18</sup> In addition, we compared collagen I deposition and CD68 abundance in positive vs. negative samples for MPs.

## Methods

### Study design

We performed a comparative autopsy-based study using lung tissue samples collected post-mortem from two independent case series of deceased individuals. The first comprised autopsies performed between January and December 1991 at the Institute of Legal/Forensic Medicine of University Hospital of Campania “Luigi Vanvitelli” and the second consisted of autopsies performed between January and September 2024 at the same institution using identical procedures. In both cases, corpses were kept at 5 °C and autopsies were performed with a standardized protocol based on a sequential examination of all organs. For both case series, only paraffin sections obtained from the posterior basal segment of the lower lobes were considered, in order to minimize variability related to regional exposure within the lung. Inclusion criteria for samples were: 1- being derived from autopsies with a minimum dataset of information relative to age and sex of the individual, and availability of data; 2- being derived from an autopsy performed within 48 h from the declaration of death, from corpses with no signs of decomposition, and not dead for pulmonary causes; 3- availability of the

appropriate segment of the lung and preserved histological structure. Data were retrieved from archived records for the 1991 series and from electronic databases for the 2024 cases and were used in an anonymized manner for data analysis. Data relative to employment were retrieved from archival records or, when unavailable, through phone interviews with surviving relatives. Risk of exposure to MPs was categorized as follows: no occupational exposure risk, low occupational exposure risk, and high occupational exposure risk, as recently suggested.<sup>19</sup> The study design is summarized in [Supplementary Fig. S1](#). We adhered to the STROBE guidelines checklist for reporting of data.

Lung tissues were fixed in 10% buffered formalin and embedded in paraffin through standard histopathologic procedures. The subsequent laboratory procedures were conducted under strict anti-contamination conditions, using non-plastic equipment. Blank paraffin sections and procedural blanks performed with the same methodology were subsequently prepared and tested as a quality-control measure. The Institutional Ethics Committee of the University of Campania “Luigi Vanvitelli” approved the study (Prot. 0002789/i).

### End points and sample size estimation

The primary endpoint was the total abundance of microplastic particles in lung tissue in the 2024 vs. the 1991 case series, as measured by LDIR. Secondary endpoints included MPs presence (yes/no) in the two series, mean particle diameter, and polymer composition. Py-GC/MS and SEM were used as supporting and exploratory technologies, respectively, in a randomly selected subset of samples. Stratified random sampling was performed by assigning a number to each case and then generating two separate sets, *i.e.*, one for 1991 and one for 2024, of random numbers using a web tool (<https://www.randomizer.org/>). Ancillary endpoints were the abundance of CD68 and Collagen I in MPs-positive vs. MPs-negative samples.

To estimate the sample size, a previous study suggested that brain samples collected post-mortem from individuals deceased either in 2016 or 2024 and analyzed with Py-GC/MS showed a mean difference of 1572 µg g<sup>-1</sup> with a standard deviation (estimated from quartiles through the formula  $Q3-Q1/1.35$ ) of 2222 µg g<sup>-1</sup>.<sup>5</sup> Considering an alpha of 0.05, a beta of 0.2, and an expected proportion of 70% vs. 30% of samples in the 2024 vs. 1991 case series, we calculated a sample size of 80 sections using the T statistic and non-centrality parameter, with the intention of comparing two means of a continuous measure. We increased such estimate by 20% and set a target of 96 samples.

### Detection and quantitation of microplastics by LDIR

To identify and quantify MPs in lung samples, formalin-fixed paraffin-embedded (FFPE) tissue blocks

were deparaffinized using graded isopropyl alcohol solutions following previously published protocols.<sup>20,21</sup> After deparaffinization, approximately 1.0 g of recovered tissue per case was allocated to LDIR analysis. For Py-GC/MS analysis, an additional ~0.5 g of deparaffinized tissue was independently processed.

Before LDIR analysis, samples were digested using an alkaline-oxidative protocol adapted from published LDIR procedures.<sup>22,23</sup> In brief, tissues were incubated in 10% potassium hydroxide (KOH) (1:30 w/v) at 50–60 °C for ~24–72 h to solubilize proteins and lipids, with duration adjusted based on digestion progress, followed by treatment with 30% hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) at 60 °C for up to 48 h to remove residual organic material. The hot digest was then filtered while still warm through gold-coated PET membrane filters (0.8 µm pore size, 25 mm diameter, 100/0 nm Au coating; Art. No. 201-12020-0825, Lab/Pharma membrane filters). The beaker was rinsed with ethanol, and the rinse was included in the filtration step to maximize particle recovery. Filters were air-dried, mounted on a dual-position holder compatible with the LDIR system (Agilent Technologies Inc.), and subsequently analyzed.

To target particles ≥10 µm, analyses were performed using an Agilent 8700 LDIR Chemical Imaging System (Agilent Technologies, USA) operated in particle analysis mode. This lower size threshold, below the conventional 20 µm limit of automated workflows, reflects methodological optimization rather than instrumental constraints and was achieved through the use of gold-coated membrane filters to enhance IR reflectance and reduce substrate interference, together with a stringent Hit Quality Index (HQI) acceptance threshold (≥0.80) to ensure reliable polymer identification at smaller particle sizes. This approach is consistent with previous studies extending LDIR detection towards ~10 µm under optimized conditions<sup>24</sup> and with manufacturer validation data demonstrating reliable identification below 20 µm (Optimizing Microplastic Characterization by LDIR-Agilent).

Particles were first located through rapid screening scans at 1800 cm<sup>-1</sup>, followed by acquisition of full infrared spectra in the range 975–1800 cm<sup>-1</sup> at a spectral resolution of 1 cm<sup>-1</sup>.<sup>25,26</sup> Spectra were matched against the Agilent Microplastics library (version Starter 2.1), supplemented with an in-house reference library for polymer identification. The validated particle detection range was 10–500 µm; particles with a hit quality index (HQI) ≥ 0.80 were accepted as microplastics. For each identified particle, polymer type and diameter were recorded. Further details on the analytical procedure are provided in the [Supplementary Material](#) (Extended Analytical Methods and Quality Control—Detection and Quantitation of Microplastics by LDIR).

To assess potential contamination, two blanks were included in every batch: (i) a procedural blank made of

paraffin-only sections without tissue processed through the full workflow, and (ii) a laboratory/ambient blank consisting of pre-cleaned filters placed open on the bench. Both controls were analyzed using the same conditions applied to the tissue samples. Both the procedural and laboratory blanks were devoid of microplastic particles above the spectral confidence threshold, indicating negligible background contamination under the adopted analytical conditions; instrumental background checks were performed at the beginning of each analytical session, and no significant spectral artefacts were detected. Detailed procedures are provided in the Extended Analytical Methods and Quality Control section of the [Supplementary Material](#).

### Detection and quantitation of MPs by Py-GC/MS

Identification and quantification analysis of MPs were based on an analytical method previously developed, validated, and applied to measure individual plastic polymer mass concentrations in the samples.<sup>3</sup> In detail, the abundance of 11 different MPs, including polyethylene (PE), polypropylene (PP), polystyrene (PS), acrylonitrile butadiene styrene copolymer, styrene-butadiene copolymer, polymethyl methacrylate, polycarbonate, polyvinylchloride (PVC), polyethylene terephthalate (PET), Nylon 6, and Nylon 66 were identified and quantified using a multishot microfurnace pyrolyzer (EGA/PY-3030D) with an autoshot sampler (AS-2020E; Frontier Lab, Japan) coupled to a GC-MS system (GC 8890, 5977C MSD; Agilent). Nylon 6 and Nylon 66 results were summed to obtain the estimates of polymer mass for polyamide (PA). Analyses were performed in double-shot mode, consisting of a first thermal desorption step (100–350 °C) designed to remove volatile/semi-volatile matrix interferences—including residual paraffin/waxy hydrocarbons potentially remaining after the pre-treatment—prior to polymer analysis, followed by a second pyrolysis step at 600 °C for 0.2 min to generate polymer-specific pyrolytic products. Separation was achieved on an Ultra Alloy® 5 capillary column (30 m × 0.25 mm, 0.25 µm) using helium (1.0 mL/min) with a 50:1 split ratio. The GC oven was programmed from 40 °C (5 min) to 340 °C at 20 °C/min (20 min hold). Mass spectrometric detection was conducted in EI mode (70 eV), with an ion source temperature of 230 °C and full-scan acquisition over m/z 35–450. The microplastic-silicon dioxide (MPs-SiO<sub>2</sub>) calibration standard (Lot: 22051001) was obtained from Frontier Laboratories Ltd. (Japan) and used as a plastic reference standard. This calibration standard includes 11 polymers and does not contain polyurethane, since not recommended by the manufacturer.

Prior to Py-GC/MS analysis, all samples underwent enzymatic and chemical digestion, adapted from previously published protocols with modifications, to remove interfering organic material and minimize

matrix-related interferences.<sup>27,28</sup> A portion of deparaffinized lung sample was accurately weighed and quantitatively transferred to a 20 mL glass vial pre-rinsed with dichloromethane. A freshly prepared 2.5% (w/v) Creon® enzyme solution was prepared in Milli-Q water, and the pH was adjusted to 8–10 by adding a few drops of saturated sodium carbonate (Na<sub>2</sub>CO<sub>3</sub>). Samples were adjusted to pH 9–10, treated with 10 mL of the enzyme solution, and incubated at 38 °C for 48 h. Following enzymatic digestion, 10 mL of 30% H<sub>2</sub>O<sub>2</sub> was added, and samples were further incubated at 60 °C for 24 h to remove residual organic material, including lipids and proteins. After digestion, samples were hot-filtered through 0.7 µm glass fiber filters (25 mm diameter; Whatman, Maidstone UK); filtration vessels were rinsed with Milli-Q water and combined with the filtrate. Filters were then folded, wrapped in aluminum foil, dried, and transferred to a stainless-steel pyrolysis cups (PY1-EC80F, Frontier Lab) for subsequent Py-GC/MS analysis. Polymer identification and quantification were performed using a well-established analytical workflow in which individual analytical standards of each target polymer were analyzed to define their characteristic pyrolysis products and diagnostic fragment ions ([Supplementary able S1](#)).<sup>27,29,30</sup> Data processing and spectral interpretation were conducted using MassHunter software (13.0.324.0 version) in combination with the NIST MS Search 2.4 mass spectral library from the National Institute of Standards and Technology.

To minimize the risk of plastic contamination, all sample handling was performed under a laminar fume hood at room temperature. Before analysis, work surfaces and glassware were thoroughly cleaned with 70% ethanol and covered with aluminum foil; glassware was additionally rinsed three times with analytical-grade dichloromethane. Contamination control measures included the routine analysis of instrumental blanks (pyrolysis cups containing quartz wool), periodic system cleaning runs (without pyrolysis cups), and procedural blanks processed alongside samples. Procedural blanks comprised (i) reagent blanks containing the full digestion mixture (Creon® solution, and H<sub>2</sub>O<sub>2</sub>) processed in parallel without tissue and (ii) ambient blanks consisting in unused GF/F filters exposed on the bench. These controls were used to verify the absence of cross-contamination and to monitor potential polymer background introduced during sample preparation, as detailed in the Extended Analytical Methods and Quality Control- Detection and Quantitation of MPs by Py-GC/MS section of the [Supplementary Material](#).

### Electron microscopy

Sections deparaffinized as described above were fixed with 2.5% glutaraldehyde in phosphate buffer and post-fixed in 0.5% osmium tetroxide in the same buffer and

then dehydrated in an acetone series and embedded in Epon Araldite resin. Sixty nanometer thick sections mounted on copper grids were coated for 10 s with platinum in a sputter coater (Jeol JFC-2300HR) and placed in an aluminum stub for SEM analysis (Jeol JSM-IT800) and imaged using back scattered electrons (SBED), when unusual particles were found, an energy-dispersive X-ray analysis (EDX) detector was used and X-ray spectra acquired as maps of elements and as a total content of element in an area in order to make a direct comparison of element content in particles and amorphous matrix.

### Immunohistochemistry

Deparaffinized and rehydrated sections were subjected to antigen retrieval by adding a buffer containing 10 mM sodium citrate with 0.05% Tween-20, at pH 6.0. Subsequently, the sections were washed in phosphate buffered saline (PBS) and then treated with a blocking solution consisting of fetal bovine serum with 0.1 g/mL saponin for 1 h at room temperature. Serial sections were then incubated with anti-CD68 (CONFIRM KP-1, 790–2931, Roche) or anti-collagen type I (Collagen I Polyclonal Antibody, Biotin, PA1-28530). Images were captured with personal computer-based 24-bit color image analysis system (IM500; Leica Microsystem AG) for each section. Quantification was performed in two manners: 1- by a pathologist blinded to the results of LDIR, using a semi-quantitative method to assign a score ranging from 0 to 3 (0, absent; 1, mild; 2, moderate; 3, marked); and 2- using a personal computer-based 24-bit color image analysis system (IM500; Leica Microsystem AG) to evaluate the results. For the latter approach, images were captured with identical exposure settings for each section and the results are expressed as the percentage of positivity of the parenchymal area.

### Statistical analysis

Continuous variables were assessed for normality through the Shapiro–Wilk test and expressed as median (IQR) or mean ± SD, while categorical variables as n (%). Relative polymer abundance in the two case series was calculated by first estimating the percentage of abundance for each type of plastics for each individual, *i.e.*, the number of MPs for the specific polymer type/total number of MPs, to then calculate the mean of the different percentages for each polymer in the two groups, considering only individuals positive for MPs. MPs prevalence, abundance, and size were compared between the two case series using the Mann–Whitney U test for continuous data the Fisher exact test for categorical variables, and the Fisher-Freeman-Halton test for contingency tables larger 2 × 2. Given the low prevalence of MPs in the 1991 group, the comparison of the LDIR-derived MPs burden was analyzed with a two-part hurdle model,<sup>31</sup> specifying logistic regression for

the detection probability  $P(Y > 0)$  and a Gamma GLM with log link for the conditional mean  $E[Y | Y > 0]$ . Uncertainty on the marginal mean  $E[Y] = P(Y > 0) \cdot E[Y | Y > 0]$  was quantified by a stratified bootstrap (10,000 resamples) and results were cross-validated against Fisher's exact and Mann–Whitney U tests. A comparison between MPs positive and MPs negative samples, independently of case series, was also performed. A two-tailed p value of  $<0.05$  was considered statistically significant. Statistical analyses were performed using GraphPad Prism, version 9.1.2, with the exception of the analyses of the hurdle model, which were performed with Python v3.13.

### Role of the funder

The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript, and decision to submit the manuscript for publication.

### Results

The final number of samples included in the study derived from 99 autopsy cases: 42 collected in 1991 and 57 in 2024 (Fig. 1A). The demographic and clinical characteristics of the two series are summarized in Table 1. The mean (SD) age was  $61 \pm 11$  years in 1991 and  $64.9 \pm 9.4$  years in 2024; 69.1% and 59.6% of participants were men, respectively. There were no differences in terms age, sex, smoking status, occupational exposure risk, causes and season of death between the two case series.

Representative LDIR micrographs for the two case series are shown in Fig. 1B while an example of particles detected for each polymer with the associated spectra is reported in Supplementary Fig. S2. MPs were identified in 8 of 42 lungs (19%) from 1991 and in 44 of 57 lungs (77%) from 2024 (Fig. 1C). Among those with evidence of MPs, the median (Q1–Q3) particle count increased from 12.9 (11.3–13.7) in 1991 to 19.9 (15.5–28.2) microplastics/g of tissue in 2024 (Fig. 1D). Given the low prevalence of MPs positivity in the 1991 case series, we also performed a results-weighted hurdle analysis. The detection probability showed an adjusted odds ratio of 14.4 (95% CI 5.4–38.6,  $p = 1.2 \times 10^{-7}$ ). Among detection-positive samples, the mean burden in 2024 was 2.62 times that in 1991 (95% CI 2.27–3.04,  $p < 0.001$ ;  $p = 8.5 \times 10^{-6}$ ), a result confirmed under lognormal sensitivity analysis. Combining the two components, the marginal mean burden was 10.6-fold higher in 2024 ( $E[Y] = 25.20$  vs. 2.37 particles/g; bootstrap 95% CI 6.1–25.3), and the joint year effect was significant (likelihood-ratio  $\chi^2 = 102.7$  on 2 df,  $p = 5.0 \times 10^{-23}$ ) (Supplementary Tables S2 and S3). When considering the mean  $\pm$  SD of the relative abundance (%) of each plastic type/total

MPs for each individual positive to MPs in the two groups, polymer composition shifted from PE ( $51.7 \pm 2\%$ ) and PP ( $27.8 \pm 2.7\%$ ) predominance in 1991 to a more heterogeneous pattern in 2024, *i.e.*, PET ( $28.4 \pm 3.1\%$ ), PVC ( $14.3 \pm 2.4\%$ ), PS ( $14.2 \pm 2.6\%$ ), PE ( $27.5 \pm 2.9\%$ ), and PP ( $13.5 \pm 3.1\%$ ) (Fig. 1E). Relative to morphology, particles detected in samples from 1991 had a larger diameter compared with those in 2024 samples, *i.e.*, median diameter was  $56.2 \mu\text{m}$  (44.9–73.4) in 1991 and  $22.4 \mu\text{m}$  (15.4–37.9) in 2024 (Fig. 1F). A representative example of the LDIR output for one sample from each case series is shown in Table 2. Moreover, Supplementary Fig. S3 presents the distribution of particle morphotypes among all MPs detected in positive samples from the 1991 and 2024 case series. Fragments were the predominant morphotype in both cohorts, whereas fibers, pellets, and spheres accounted for smaller proportions of the detected particles.

To corroborate findings obtained from LDIR imaging, we analyzed a subset of 20 random samples, *i.e.*, 10 from each case series, also with Py-GC/MS and SEM. The baseline characteristics of these cases were comparable with the rest of the population (Supplementary Table S4). Py-GC/MS corroborated the findings relative to the higher prevalence (Fig. 2A) and the more variegated repertoire (Fig. 2B) of MPs in samples derived from the 2024 and compared with the 1991 series, as evidenced by polymers mass concentrations of positive samples (Fig. 2C). Representative Py-GC/MS pyrograms of polymer detected in these samples are reported in Supplementary Fig. S4. Evaluation of these cases using the back-scattered electrons with SEM and derived spectral x-ray maps evidenced some particles showing a reduction in the presence of carbon and a greater abundance of sulfur (Fig. 2D and Supplementary Fig. S5). The presence of such particles was more pervasive in the 2024 case series.

We then compared the characteristics between samples positive ( $n = 52$ ) vs. those negative ( $n = 47$ ) for MPs, independently of the case series, and found no differences (Supplementary Table S5). However, samples with evidence of MPs, compared with those without, showed a higher abundance of both Collagen I and CD68 (Fig. 3).

### Discussion

This comparative autopsy study provides the first direct evidence of a higher abundance of MPs accumulation in human lungs from individuals deceased 33 years apart. Indeed, the prevalence of lungs containing detectable MPs and their mean particle abundance increased substantially.

Only one previous study has assessed the levels of MPs in samples from autopsies performed in two different calendar years, comparing individuals deceased either in 2016 or in 2024.<sup>5</sup> In that study,

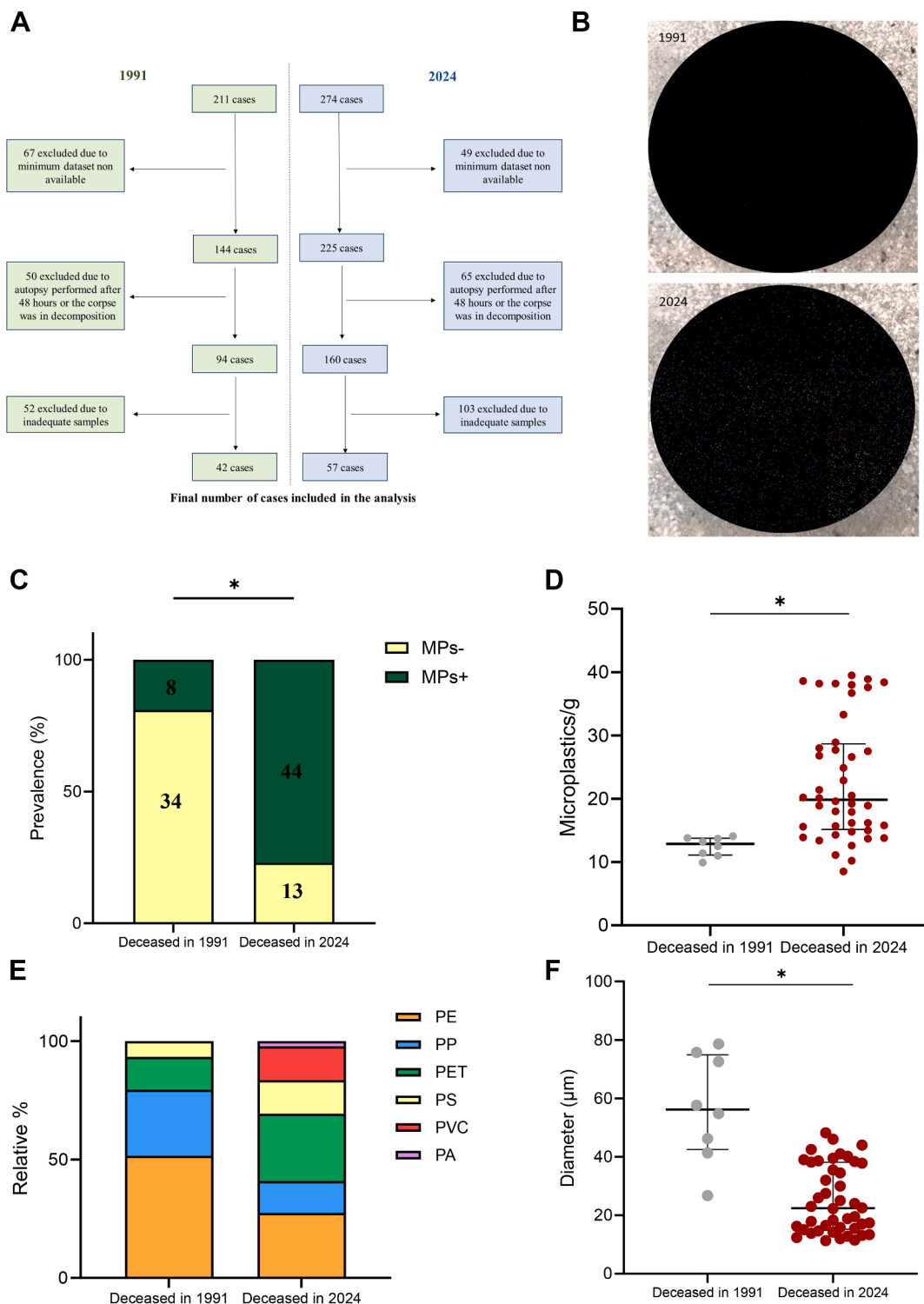


**Fig. 1: Microplastics prevalence, abundance, size, and type in 1991 and 2024 case series according to LDIR.** (A) Flowchart of samples inclusion for the 1991 and 2024 case series. (B) Representative LDIR images for the 1991 and 2024 case series. (C) Prevalence of microplastics (MPs) in the two case series;  $*p < 0.001$ , Fisher's exact test. (D) Number of microplastics per gram of tissue in positive samples from cases of 1991 vs. 2024;  $*p < 0.001$ , Mann-Whitney test. (E) Relative distribution (%) of the different polymers identified in positive samples from the 1991 and 2024 case series. (F) Particle diameter of microplastics detected in positive samples from the 1991 and the 2024 case series;  $*p < 0.001$ , Mann-Whitney test. PE: Polyethylene, PP: Polypropylene, PET: Polyethylene terephthalate, PS: Polystyrene, PVC: Polyvinyl chloride, PA: Polyamide.

| Variable                   | Deceased in 1991 (n = 42) | Deceased in 2024 (n = 57) | p-value |
|----------------------------|---------------------------|---------------------------|---------|
| Age (years)                | 63.5 (54.3–67.8)          | 65 (59–71)                | 0.111   |
| Males, n (%)               | 29 (69.1)                 | 34 (59.6)                 | 0.401   |
| Females, n (%)             | 13 (30.9)                 | 23 (40.4)                 |         |
| Smoking, n (%)             | 31 (73.8)                 | 35 (61.4)                 | 0.281   |
| Occupational exposure risk |                           |                           |         |
| Missing, n (%)             | 4 (9.5)                   | 2 (3.5)                   | 0.711   |
| High risk, n (%)           | 15 (35.7)                 | 23 (40.4)                 |         |
| Low risk, n (%)            | 9 (21.4)                  | 13 (22.8)                 |         |
| No risk, n (%)             | 14 (42.9)                 | 19 (36.8)                 |         |
| Nationality                |                           |                           |         |
| Italian, n (%)             | 39 (92.9)                 | 50 (87.7)                 | 0.518   |
| Other, n (%)               | 3 (7.1)                   | 7 (12.3)                  |         |
| Manner of death            |                           |                           |         |
| Natural cause, n (%)       | 29 (69.1)                 | 37 (64.9)                 | 0.830   |
| Non-natural cause, n (%)   | 13 (30.9)                 | 20 (35.1)                 |         |
| Mechanism of death         |                           |                           |         |
| Cardiac cause, n (%)       | 15 (51.7)                 | 19 (51.4)                 | >0.999  |
| Cerebral cause, n (%)      | 6 (20.7)                  | 7 (18.9)                  |         |
| Others, n (%)              | 8 (27.6)                  | 11 (29.7)                 |         |
| Place of death             |                           |                           |         |
| Home, n (%)                | 29 (69)                   | 38 (66.7)                 | 0.844   |
| Hospital, n (%)            | 2 (4.8)                   | 5 (8.8)                   |         |
| Others, n (%)              | 11 (26.2)                 | 14 (24.5)                 |         |
| Season of death            |                           |                           |         |
| Spring, n (%)              | 9 (21.4)                  | 14 (24.6)                 | 0.941   |
| Summer, n (%)              | 10 (23.8)                 | 15 (26.3)                 |         |
| Autumn, n (%)              | 12 (28.6)                 | 13 (22.8)                 |         |
| Winter, n (%)              | 11 (26.2)                 | 15 (26.3)                 |         |

Continuous variables are presented as mean (SD), while categorical variables are presented as number (n) and the relative percentage (%).

**Table 1: Demographic and clinical characteristics of the two case series.**

Py-GC/MS data suggested an increased MPs concentration in liver and brain samples collected more recently but not in the kidney.<sup>5</sup> MPs abundance was not influenced by age, sex, ethnicity, or cause of death, similar to the results provided here. Our data, spanning a larger time range and focusing on a tissue directly exposed to air levels of pollutants, corroborates and extends those findings. Indeed, the use of an additional and different technology, *i.e.*, LDIR, in samples derived from a different organ might mitigate the concerns related to the technical and possibly other limitations relative to the use of Py-GC/MS in human brain samples.<sup>16</sup> Indeed, although our sample preparation for Py-GC/MS included steps to reduce residual biological material, potential matrix interference (*e.g.*, from incompletely removed lipids) cannot be entirely excluded and may represent a source of analytical variability. In addition, we employed Py-GC/MS only in a subset of samples, with only two positive cases in the 1991 series. While such an approach corroborated the increase in prevalence and polymer type diversity, the resulting limited statistical power, *i.e.*, only two positive samples in the 1991 case series, impeded quantitative

comparison of mean total polymer concentration between the two case series. Thus, Py-GC/MS findings should accordingly be interpreted as supportive rather than confirmatory in this respect. Given that particles in 2024 were smaller, it may be possible that overall mass according to Py-GC/MS is not increased when considering only positive samples. Finally, we compared case series of people deceased in two different calendar years, thus having only two time points. For all these reasons, further studies employing an even larger range of analytical techniques and a higher number of time points and samples are needed to establish that MPs accumulation in human organs is higher in people deceased more recently.

Previous studies have reported of MPs in human lungs, in samples derived from both living and deceased individuals.<sup>32–34</sup> One study performed with lung samples from autopsy cases collected in 2021 and using a different methodology provided results similar to those presented here for the 2024 series in terms of both prevalence of MPs positivity and types of polymer detected.<sup>32</sup> More broadly, studies with lung samples suggest PE, PET, and PP as the most common MPs

| Particle ID                              | Diameter ( $\mu\text{m}$ ) | Area ( $\mu\text{m}^2$ ) | Aspect ratio | Morphotype | Polymer | HQI    |
|------------------------------------------|----------------------------|--------------------------|--------------|------------|---------|--------|
| Representative sample — 1991 case series |                            |                          |              |            |         |        |
| A38                                      | 164.29                     | 21200.0                  | 1.227        | Fragment   | PE      | 0.973  |
| A3                                       | 49.35                      | 1912.5                   | 0.469        | Fragment   | PE      | 0.876  |
| A57                                      | 45.66                      | 1637.5                   | 3.185        | Fiber      | PE      | 0.820  |
| A272                                     | 36.35                      | 1037.5                   | 1.695        | Fragment   | PE      | 0.869  |
| A342                                     | 34.09                      | 912.5                    | 1.688        | Fragment   | PE      | 0.826  |
| A368                                     | 33.38                      | 875.0                    | 1.528        | Pellet     | PE      | 0.835  |
| A456                                     | 31.41                      | 775.0                    | 0.458        | Fragment   | PE      | 0.881  |
| A460                                     | 30.64                      | 737.5                    | 1.000        | Pellet     | PP      | 0.832  |
| A495                                     | 30.38                      | 725.0                    | 0.750        | Fragment   | PP      | 0.878  |
| A518                                     | 29.85                      | 700.0                    | 1.348        | Fragment   | PP      | 0.840  |
| A519                                     | 29.85                      | 700.0                    | 0.900        | Fragment   | PET     | 0.829  |
| A525                                     | 29.85                      | 700.0                    | 0.595        | Fragment   | PS      | 0.851  |
| Representative sample — 2024 case series |                            |                          |              |            |         |        |
| A1003                                    | 20.73                      | 337.5                    | 2.000        | Fragment   | PP      | 0.809  |
| A963                                     | 21.11                      | 350.0                    | 1.500        | Fragment   | PP      | 0.847  |
| A930                                     | 21.11                      | 350.0                    | 0.429        | Fragment   | PE      | 0.834  |
| A815                                     | 22.57                      | 400.0                    | 2.000        | Fragment   | PET     | 0.809  |
| A1905                                    | 22.21                      | 387.5                    | 1.000        | Fragment   | PET     | 0.803  |
| A1012                                    | 20.34                      | 325.0                    | 1.231        | Fragment   | PP      | 0.838  |
| A995                                     | 20.73                      | 337.5                    | 0.568        | Fragment   | PVC     | 0.823  |
| A1028                                    | 20.34                      | 325.0                    | 2.062        | Fragment   | PET     | 0.800  |
| A888                                     | 21.85                      | 375.0                    | 1.526        | Fragment   | PS      | 0.827  |
| A916                                     | 21.48                      | 362.5                    | 0.800        | Pellet     | PVC     | 0.8340 |
| A872                                     | 21.85                      | 375.0                    | 2.158        | Fragment   | PS      | 0.843  |
| A1036                                    | 20.34                      | 325.0                    | 1.250        | Pellet     | PE      | 0.830  |
| A957                                     | 21.11                      | 350.0                    | 1.750        | Pellet     | PS      | 0.813  |
| A927                                     | 21.11                      | 350.0                    | 0.435        | Fragment   | PE      | 0.809  |
| A978                                     | 20.73                      | 337.5                    | 0.429        | Fragment   | PA      | 0.819  |
| A597                                     | 25.54                      | 512.5                    | 0.234        | Fiber      | PET     | 0.842  |
| A699                                     | 23.94                      | 450.0                    | 1.750        | Fragment   | PET     | 0.828  |
| A298                                     | 35.46                      | 987.5                    | 2.065        | Fragment   | PE      | 0.814  |
| A104                                     | 57.12                      | 2562.5                   | 1.524        | Fragment   | PE      | 0.809  |

Table 2: Particle-level LDIR output for one representative positive sample from the 1991 case series and one from the 2024 case series.

detected, possibly reflecting their widespread use.<sup>35</sup> On the other hand, the lungs are a transient environment, with established trafficking and clearance mechanisms that depends on the functional status of this organ. To this respect, also the findings relative to higher abundance of CD68 and Collagen I in samples positive for MPs should be interpreted with a caution. Indeed, the relationship observed here is correlational and does not establish causality. Even though samples from people dead for pulmonary causes were excluded and manifest lung diseases were not observed, subclinical lung conditions or even diseases might have been present. As a result, the possibility that inflammation and fibrosis occurred first might be also conceived, with a resulting compromised clearance at those sites then leading to particles build-up. It is thus not possible to determine whether the histological findings are attributable to MPs exposure, pre-existing lung disease, or their interaction. Thus, the relative results should be considered exploratory and hypothesis-generating

rather than mechanistic evidence of MPs-associated pathology. In addition, given the destructive nature of the technologies used here to assess MPs, future studies should employ optical PTIR, a technology that allows the visualization of the particles within the tissue,<sup>36,37</sup> coupled with staining for cellular events in serial sections, to firmly associate the presence of MPs with lung inflammation and fibrosis, as recently demonstrated for human colon samples.<sup>21</sup>

Beyond the limitations discussed above, it is important to note that occupational exposure studies report a range of pulmonary diseases in workers exposed to different plastics.<sup>35</sup> Whether exposure to ambient levels of MPs in the general population and the related accumulation in the lungs are pathogenic phenomena is now being actively explored.<sup>19</sup> To this regard, the use of autopsy samples impedes the exploration of associations between MPs and diseases, given the lack of this latter information. Similarly, even though we detected no differences in the prevalence of workers

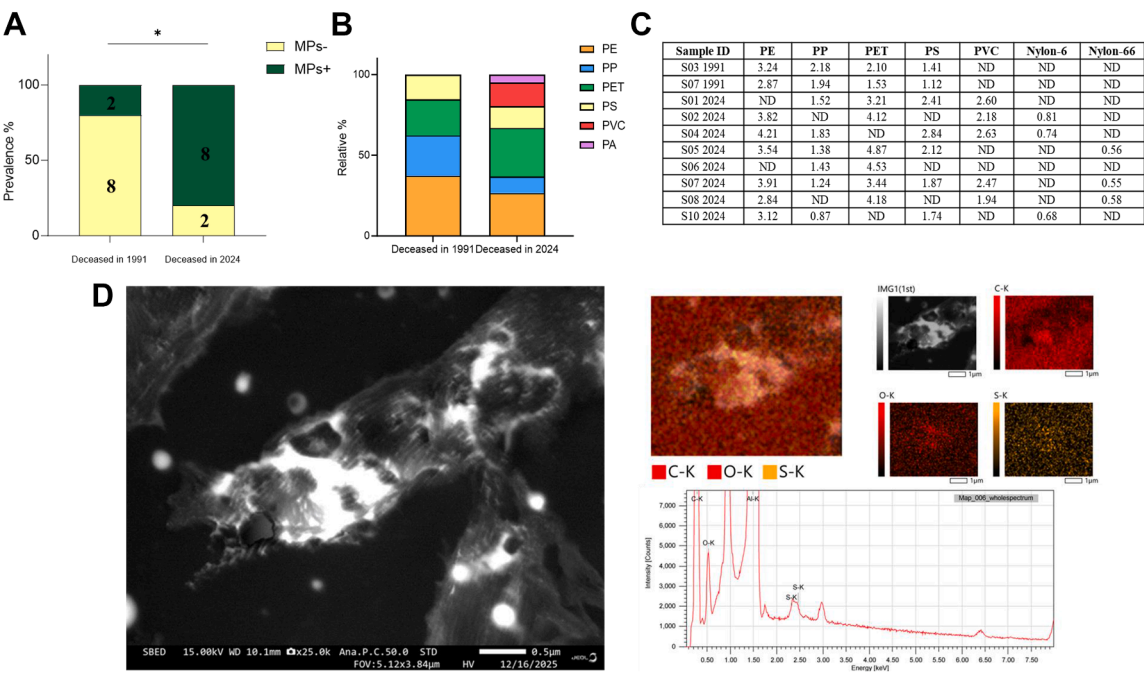


**Fig. 2: Analysis with pyrolysis-gas chromatography/mass spectrometry and scanning electron microscopy in a subset of 20 samples.** (A) Prevalence of microplastics (MPs) in the 1991 and 2024 case series according to Py-GC/MS data; \* $p < 0.05$ , Fisher's exact test. (B) Relative distribution (%) of the different polymers identified in positive samples from the 1991 and 2024 case series using the same technology. (C) Table showing polymer-specific mass concentrations ( $\mu\text{g/g}$ ) determined by Py-GC/MS in all positive samples, *i.e.*, 2 of the 1991 and 8 of the 2024 case series; acrylonitrile butadiene styrene copolymer, styrene-butadiene copolymer, polymethyl methacrylate, and polycarbonate are not presented in the table since they were all Not Detectable (ND). (D) Example of a particle identified with SEM in a sample from the 2024 case series, with the relative EDX spectrum suggesting a high abundance of sulphur.

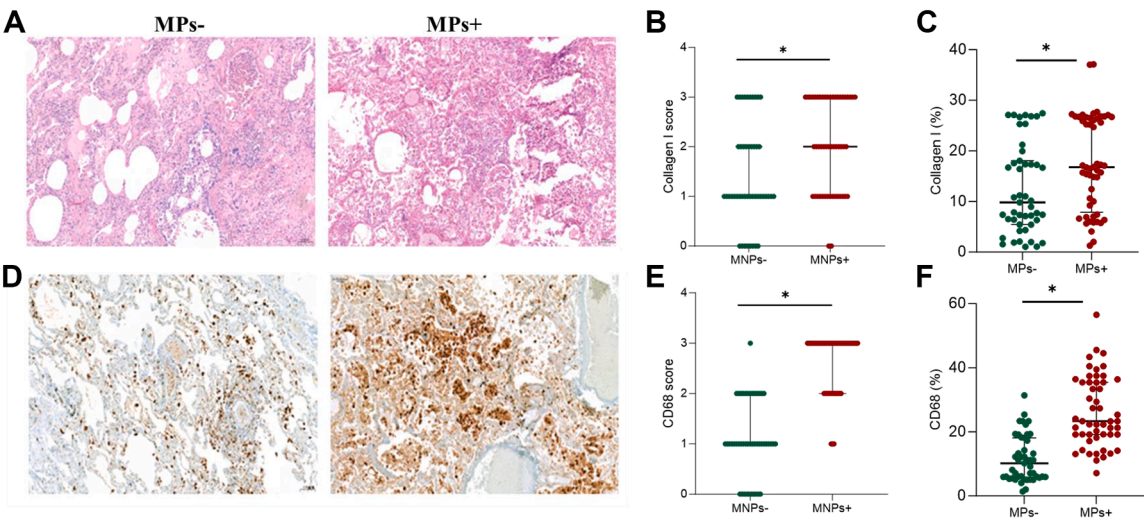


**Fig. 3: Collagen type I and of CD68 in positive vs. negative samples for microplastics.** Representative images of collagen type I (A) and CD68 (D) staining in the group of samples with evidence of microplastics (MPs+) and the group with no evidence of microplastics (MPs-), along with the relative quantitation through pathologist-derived scores (B and E) or through percentage of positive area (C and F). Means  $\pm$  SD are shown. \*  $p < 0.01$ ; Mann-Whitney test.

with a high occupational exposure risk, such information was obtained through interviews of the surviving relatives for the majority of the 1991 cases and it thus may be prone to bias. More broadly, the use of autopsy case series entails a consistent risk of residual confounding, because detailed clinical and demographic information on individual patients was not available and since environmental, dietary, occupational, and healthcare histories are inherently different when comparing individuals living 33 years apart. In addition, given that we did not test autopsy samples derived from other centers our findings might not be geographically generalizable. Indeed, local, rather than planetary, phenomena might explain the observations provided here.<sup>38</sup> Given the limit of detection with LDIR, we did not collect data relative to the specific accumulation of particles <10 µm, *i.e.*, those more likely reaching the alveoli and the blood. Similarly, the use of ~0.8 µm and ~0.7 µm filters, respectively for LDIR and Py-GC/MS analyses, precludes the retention of particles below such thresholds, possibly excluding a submicron fraction likely relevant in the respiratory tract. Thus, the reported particle counts do not represent overall pulmonary plastic burden but rather only the detectable fraction within the possible size range, an observation with obvious implications for the biological interpretation of the results.

An additional limitation relates to the interpretation of the findings obtained by SEM-EDX. In particular, the detection of sulfur-containing particles cannot be considered specific evidence of MPs. Indeed, sulfur is a common constituent of airborne pollution and may originate from multiple environmental sources with different chemical compositions. Therefore, the presence of sulfur-containing particles *per se* does not represent a proof of MPs contamination. These results must be considered ancillary findings rather than corroborative evidence for MPs. Furthermore, the use of samples with preserved histological structure might have introduced a survivor bias, especially for the 1991 group. Although the comparability of HQI distributions, particle size data, and paraffin composition analyses collectively support the analytical integrity of the cross-series comparison, the possibility that long-term storage of archival FFPE material differentially affected particle recovery, fragmentation, polymer degradation, or extraction efficiency cannot be fully excluded and represents a residual source of uncertainty in the interpretation of the observed differences. Finally, given the limited availability of previous studies with a similar design,<sup>5</sup> sample size was calculated to estimate a difference in the abundance but not in the prevalence of MPs. In addition, numerical values for calculation derived from the only study with a similar design, which employed Py-GC/MS<sup>5</sup> and not LDIR for the analysis, forcing a cross-method extrapolation. Thus, the study was not formally powered for its

primary endpoint. Furthermore, the sensitivity analysis performed with the two-part hurdle model showed a low deviance. Also, much of the apparent difference between groups observed in this analysis was driven by prevalence rather than by differences among positive samples. These aspects, coupled by the fact that Py-GC/MS and SEM were performed only in a subset of 20 cases, might have influenced the statistical power of the results presented. Larger studies are warranted to strengthen the robustness of the findings.

## Conclusion

In summary, results of our study suggest that MPs contamination of the human lung is more prevalent in samples collected recently compared with those obtained three decades ago. Given the observational and retrospective nature of the study, these findings should be considered hypothesis-generating and need confirmation in future studies with larger sample sizes and multiple time points. However, these observations might suggest the need for systematic biomonitoring of MPs also in living individuals.

## Contributors

RM, FP, RLG, VP, AM, CS, AC, PL, PP, MB, GP, and PI conceived the idea, designed and supervised the study, analyzed data, and wrote the manuscript. LG, GF, TS, LVM, MM, EM, GD, AF, FF, IP, GF, AF, CPC, SH, AF, SG, GF, CL, GM, ACh, FO, BA, ND, and MLB collected samples, recovered data, performed the experiments, provided background expertise, and/or reviewed the manuscript for intellectual content.

## Data sharing statement

The dataset analyzed during the current study is available from the corresponding authors on reasonable request.

## Declaration of interests

PJL received study and travel support from the Minderoo Foundation, and consulting fees from the Centre Scientifique de Monaco.

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## Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.lanepe.2026.101804>.

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