

Matters arising: Bioaccumulation of microplastics in decedent human brains

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In their recent article, Nihart and colleagues¹ investigated the presence of microplastics and nanoplastics (MNPs) in human kidney, liver, and brain tissues using pyrolysis gas chromatography–mass spectrometry (Py-GC–MS), attenuated total reflectance–Fourier transform infrared (ATR–FTIR) spectroscopy, and complementary electron microscopy techniques. They reported the presence of MNPs in all examined tissues, with notably higher concentrations in the brain compared to the liver and kidney. Specifically, median total MNP concentrations in brain tissue increased from 3,345 µg/g in 2016 to 4,917 µg/g in 2024. Polyethylene (PE) was the most abundant polymer type detected across all tissues, constituting approximately 75% of the total MNP mass in the brain, compared to 44–57% in the liver and kidney.

The considerable levels of MNPs reported in human tissues have raised concern among the scientific community, the public, regulators and various stakeholders. Given the multidisciplinary nature of MNP research in the human body, such findings, especially when published in high-impact journals, must be supported by robust evidence, particularly in light of the well-recognised challenges in accurately detecting and quantifying MNPs in physiological matrices. We critically examined two aspects of the study: (a) the ability of the methods used to reliably distinguish synthetic MNPs from biological substances such as lipids and proteins, and (b) the physical plausibility of the reported plastic concentrations in brain tissue.

Analytical reliability

Plastic extraction and digestion of the tissue

Before analysing MNPs in carbon-rich matrices such as human tissue, it is essential to employ a validated digestion protocol that ensures comprehensive removal of organic matter with high recovery rates and no degradation of the plastics themselves. In their study, Nihart et al. utilised a digestion method involving 10% potassium hydroxide (KOH) at 40 °C for 3 days, followed by centrifugation in polycarbonate tubes and an ethanol wash. However, this protocol has been widely reported by analytical studies to be ineffective in achieving complete removal of organic matter, particularly in lipid-rich tissues such as the brain ^{2, 3}. Consequently, it is generally considered unsuitable for the isolation of particles from tissue samples. As illustrated in their Figure S5, the digestion efficiency for brain tissue averaged below 95%, with several instances falling below 90% and one instance under 80%. This indicates that more than 5%, and in some cases over 10% of the brain tissue remained undigested, which can create a large number of particulates that could falsely be classified as MNPs. Analysing such incompletely digested samples for MNPs could lead to potential analytical interferences, as residual biological materials might affect the accuracy of detection and quantification. Additionally, 10% KOH is a strong base (pH value ~ 13-14) that potentially degrades, for example, PET particles, thus impacting the detection potential ⁴.

Controls for reliable MNPs detection in human tissues

Robust quality assurance and quality control (QA/QC) measures are critical for any analytical measurement. They are indispensable for substantiating the presence of MNPs in human tissues. However, the study by Nihart et al. (2025) does not report the use of key contamination control procedures such as procedural blanks or field blanks, which are essential to distinguish genuine plastic signals from widespread environmental contamination. Detecting MNPs in human tissues demands rigorous validation to ensure that detected particles are not analytical artefacts or resulting from pollution present during sampling and sample preparation ³. International standards, such as ISO 24187:2023 and OECD analytical guidance, recommend the systematic use of negative controls and procedural blanks, including open-air and reagent blanks, with results corrected for any background contamination. Furthermore, validation through spiking experiments with known polymer types, sizes and concentrations is critical for assessing recovery efficiency, limits of detection/quantification and identification accuracy, as well as to understand potential impacts of the extraction method on particle integrity. Without such validation, both underestimation and overestimation of MNP concentrations are possible. The absence of these foundational controls in the current study casts uncertainty on the reported MNPs concentrations and underscores the need for more rigorous analytical validation and responsible reporting when assessing human exposure to these emerging contaminants.

Py-GC-MS

In the study by Nihart et al., PE emerged as the predominant polymer type across all samples, irrespective of patient demographics or tissue origin. The consistent predominance of PE across all individuals, time frames, and organs is biologically implausible and may point to methodological artefacts or contamination during sampling or analysis. Py-GC-MS is not a selective detection method for PE if the particles are part of a matrix, rich in organic matter, e.g. containing lipids/proteins. There, the PE polymer signal can interfere with the matrix, as has been

investigated in detail previously ⁵. For instance, PE as a polymer consists of a long carbon chain, which fragments under pyrolytic conditions according to its homologous series. Similar fragmentation patterns can arise from organic components within complex biological matrices, such as fatty acids, which also consist of long carbon chains and functional groups like carboxylic acids. An inexperienced analyst may not recognise subtle differences, so care should be taken to ensure that the retention times with their distances to neighbouring peaks (alkadienes, alkenes, alkanes) are uniform and that different markers or mass traces for PE should also be present. If possible, polymers should always be identified using several mass traces. The application of Py-GC-MS in the study by Nihart *et al.* lacks the specificity required for accurate identification of plastic polymers. Interference in the form of false positive signals can be expected from residual lipids in tissue samples that were not fully digested, as demonstrated in Figure S5 of the study by Nihart *et al.* Thus, the uniform dominance of PE particles observed by Nihart *et al.* likely originates from a misidentification of undigested lipids and other residual material and of earlier degradation of acid-sensitive polymer types during sample preparation ⁵. Detection of higher molecular weight ions specific to PE, but absent in lipids, is crucial to minimise the risk of falsely identifying lipids as plastic polymers. However, the authors reported the absence of these characteristic ions, suggesting that the observed signals likely originated from residual organic matter rather than PE, thereby indicating a potential misinterpretation of the results.

The human brain consists of ~60% fatty acids ⁶, while the liver and kidneys contain much less (usually < 5%) ^{7, 8}. Due to the limitations of Py-GC-MS and its application in the study, an inadequate lipid digestion protocol likely led to disproportionately high signals in the brain, up to an order of magnitude greater than in the liver or kidney, as reported by Nihart *et al.* These signals are most plausibly attributed to pyrolysis products of residual fatty acids and organic matter, rather than genuine plastic polymers. This, in turn, most likely falsely inflated the reported final MNPs concentrations in the work of Nihart *et al.*

Furthermore, Nihart *et al.* observed a positive (increasing) trend in the concentration of plastics over time, indicating bioaccumulation. We argue that this trend might also be an artefact of incomplete tissue digestion, a non-selective analysis protocol and different sampling conditions and storing time. For example, it has been reported that high body mass index (BMI) in humans and obesity increase the fat content in both the brain ⁹ and liver ¹⁰ tissues, but not in the kidneys ⁸. It is noteworthy that Nihart *et al.* reported a significant increase in plastic content over time in brain and liver tissues, but not in the kidneys (Figure 1c). This trend coincides with known increases in lipid accumulation in these organs, largely attributable to the global rise in obesity rates since the mid-20th century. Furthermore, differences in sampling conditions from a decade ago, as well as variations in storage duration, may have introduced technical and methodological artefacts that confound the interpretation of MNPs levels. Therefore, the observed signals may reflect the accumulation of lipids and other organic matter rather than the true bioaccumulation of plastic particles. These unaddressed methodological limitations in the study by Nihart *et al.* raise the possibility that the reported findings are artefactual rather than indicative of actual plastic burden in human tissues.

FT-IR

The authors claimed that PE predominance was confirmed using ATR-FTIR spectroscopy. They also noted that the prevalence of other polymers was less consistent, possibly due to differences in size distribution and limited sampling. However, a critical limitation arises from the fact that only nanosized particles, typically smaller than 200 nm, are capable of crossing the blood-brain barrier

(BBB), a well-documented phenomenon in the field of drug delivery to the brain as studied over the past several decades⁸. ATR-FTIR, including its most advanced forms, has a size-detection limit of approximately 500–1000 nm, i.e. it cannot reliably detect nanoscale plastics within this critical size range (1–200 nm) but may determine only possible agglomerates. This discrepancy raises significant concerns regarding the accuracy of the reported PE particle detection in the brain. Moreover, the authors used an identification threshold value of 60% (see Nihart et al. supplementary information, section 1.7), which is rather low. For instance, in organic-rich matrices, experts usually take a threshold of 80 or 90% for a positive match, considering match scores lower than false positives¹¹. If the authors had applied more stringent and widely accepted identification thresholds (e.g., 80–90%), as commonly used to minimise false positives, a substantial proportion of the reported identifications would likely not have met the criteria for positive polymer identification.

Electron microscopy

Polarisation wave or electron microscopy is often used to support the analysis of plastic particles when the type of plastics is known (e.g. using digestion protocols where plastic beads were intentionally added/ spiked in). However, microscopy alone without proper sample preparation cannot provide visual evidence of plastic presence in complex samples (i.e. tissues) without chemical confirmation of the same particles³. For example, many submicron refractory inclusions were presented by Nihart et al. (e.g. Figure 2), aiming "to provide visual evidence" of plastics present in human tissues. However, distinguishing MNPs from organic background in such complex matrices is extremely challenging due to the low density of MNPs and the potential for visual misidentification. Without robust chemical confirmation, definitive identification of these inclusions as plastics is not possible, reflecting a broader global challenge in the accurate detection and characterisation of MNPs. Therefore, relying solely on visual evidence without corroborating chemical analysis may lead to inaccurate conclusions regarding the presence of MNPs in biological tissues. This argument also applies to the "flake-like solid particulates" shown in Nihart et al. (Figure 2d), which appear surprisingly regular for environmental plastics. Even if the authors assume that the observed refractory inclusions are indeed plastics in origin and that their microscopy "provides visual evidence to support analytical chemistry", the evidence then contradicts their py-GC-MS data. For instance, Nihart et al. report an order of magnitude higher concentration of plastics in the brain than in the kidneys, yet the number of refractory inclusions (plastics) in the kidneys is multiple orders of magnitude higher than in the brain, see Figure 1.

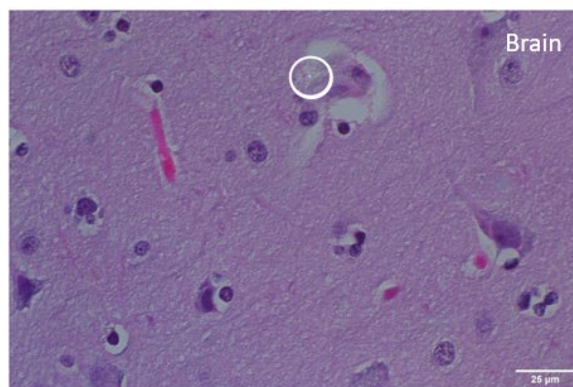
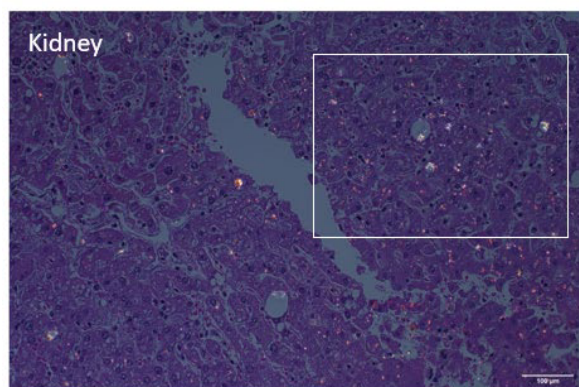


Figure 1. Microscopic presentation of plastics (refractory inclusion) inclusion shown by Nihart et al., aiming to "provide visual evidence to support analytical chemistry". Note that there are many refractory inclusions in the kidney tissue (left) and only one in the approximately same magnification in the brain (right). However, the py-GC-MS experiments showed an order of magnitude higher concentration in the brain compared to kidney tissue.

Biological plausibility: converting particle mass to particle number

In their study, Nihart et al. reported the presence of flake-like particles, typically less than $0.4\ \mu\text{m}$ in length, in human brain tissue. To theoretically evaluate these findings, we estimated the number of 100 nm PE and PP MNs that would be present in a gram of brain tissue based on the reported size range of 100–200 nm and their flake-like morphology. From the TEM image provided in Figure 2d of their publication (reproduced here as Figure 2), it appears that the width of the particles is approximately one-tenth of their length. Based on this observation, we assumed a minimum particle thickness of 5 nm for our calculations.

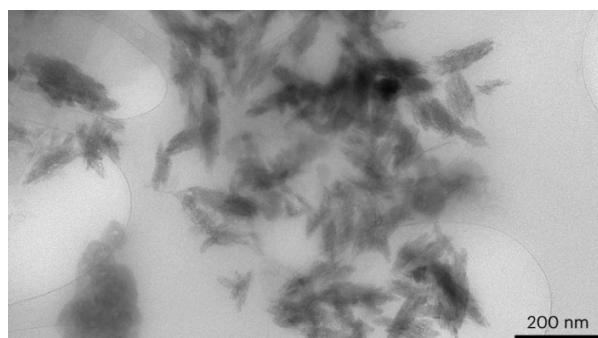


Figure 2: Transmission electron microscopy (TEM) image of shard-like nanoplastic particles observed in human brain tissue. Adapted from "Bioaccumulation of microplastics in decedent human brains – overlooked analytical issues," by Nihart et al., 2024.¹

The reported mass concentration was approximately 10,000 $\mu\text{g/g}$ weight of total plastic particles in the brain for both years (2016 and 2024), with 75% PE and 10% PP. This means there are approximately 0.0075 g of PE and 0.001 g of PP per gram of brain tissue. To consider a more conservative estimate, we assume 0.006 g of PE and 0.001 g of PP per gram of brain tissue.

PE: $6000\ \mu\text{g/g} = 6000 \times 10^{-6}\ \text{g/g} = 0.006\ \text{g/g}$

PP: $1000\ \mu\text{g/g} = 1000 \times 10^{-6}\ \text{g/g} = 0.001\ \text{g/g}$

Since the flakes are thin platelets, we estimate their volume using:

$$V_{\text{flake}} = \text{Length} \times \text{Width} \times \text{Thickness}$$

Length = 100 nm = $100 \times 10^{-7}\ \text{cm}$

Width = 10 nm = $10 \times 10^{-7}\ \text{cm}$

Thickness = 5 nm = $5 \times 10^{-7}\ \text{cm}$

By assuming material densities of 0.92 g/cm³ for PE and 0.90 g/cm³ for PP, we calculated the mass of a single flake-like particle based on an estimated size of 100 nm in length, 10 nm in width, and 5 nm in thickness.

$$Mass = V \times Density$$

$$Mass, PE = 4.6 \times 10^{-18} \text{ g}$$

$$Mass, PP = 4.5 \times 10^{-18} \text{ g}$$

From the obtained mass of a single flake, we calculated the estimated number (N) of flakes per gram of brain tissue:

$$N = \frac{Total\ mass}{Mass\ of\ one\ flake}$$

$$N, PE = 1.3 \times 10^{15} \text{ flakes per g}$$

$$N, PP = 2.22 \times 10^{14} \text{ flakes per g}$$

These calculations assume uniform distribution and no aggregation of the flakes. Such high particle counts raise questions about the plausibility of these concentrations within the physical constraints of brain tissue, considering the limited intracellular and extracellular space available. This discrepancy underscores the need for careful interpretation of the reported data and highlights the importance of rigorous methodological validation in studies assessing MNPs accumulation in human tissues, particularly tissues protected by barriers such as the BBB.

Regulatory consequences

Policy advisors and regulatory bodies such as the European Chemicals Agency (ECHA), the U.S. Environmental Protection Agency (EPA), and the Food and Drug Administration (FDA) rely on scientifically robust data to assess the (eco)toxicological risks of MNPs, evaluated using the Klimisch (human health) or CRED (environmental health) scores to confirm their reliability. However, if unsupported claims about the translocation of plastic particles into the human brain are introduced into the risk narrative, they could misguide policy discussions and result in inappropriate regulatory responses, including unjustified exposure limits or premature calls for life-cycle-wide plastics legislation¹². Such potentially ‘sensational’ scientific findings may develop into a major catalyst for stringent plastic policies and drive citizens’ fear; subsequent denial of the findings could have far-reaching consequences extending beyond science: trust in regulatory processes, public institutions, and scientific integrity itself may be undermined. If a newly evolved narrative about MNPs in the human brain turns out to be inaccurate, it will not only negatively impact regulators and their efforts to effectively address the hazards posed by MNPs; it will also erode trust in scientific institutions and fuel social trends that are already sceptical of science, thereby hindering the establishment of science-based policies in the future.

In times of prevailing post-truth politics¹³, science must therefore remain a reliable source of information and work to prevent misinformation to the greatest extent possible. Misleading conclusions about human plastic exposure and the translocation of particles into the brain could either unnecessarily increase public fear or divert attention from actual health risks, fostering

scepticism toward legitimate environmental health concerns. Finally, journals and researchers could come under ethical scrutiny for publishing studies without the proper controls and thus influencing regulatory frameworks without solid scientific backing ¹⁴.

Concluding remarks

Given these fundamental flaws/reporting omissions at each step of the analyses, the reported results and conclusions are, at best, unsupported, prone to significant false positives and, as such, do not have quantitative or qualitative value. The claim of Nihart et al. that they provided a "robust" detection does not hold, as none of the three methods were used by established protocols for MNP analysis, nor did they adhere to basic quality control practice (see this publication ⁵). All these observations make the conclusions of Nihart et al. questionable.

Recent evaluations of Py-GC/MS methods suggest that comparisons against pristine polymer standards may underestimate the true mass of aged or weathered plastic particles, which are chemically degraded and structurally altered. Moreover, spatial variability in plastic concentrations across brain regions could challenge assumptions made when extrapolating localised measurements to the whole organ. These factors underscore the need for careful interpretation of absolute concentration values, especially when data originate from single brain regions or are compared to standards not representative of environmentally aged polymers.

As demonstrated in our analysis, several methodological issues in the study by Nihart et al. raise serious concerns. These include incomplete tissue digestion (especially in lipid-rich brain tissue), potential misidentification of residual biological material as PE, and a striking mismatch between the number of reported plastic particles and the physical capacity of brain tissue to accommodate them. If such findings are taken at face value without further validation, they risk becoming influential in shaping regulatory narratives, despite their considerable scientific uncertainty.

Ethics declarations

The author declares no competing interests.

Contributions

F.A.M and DM conceived the work. All authors contributed to the writing of the article.

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