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Mass spectrometry analysis reveals changes in patterns and correlations of nucleic acid epigenetic modifications in mouse tissues exposed to polystyrene microplastics

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ABSTRACT

Microplastics (MPs), which originate from plastic degradation, are becoming a significant environmental pollutant, and their prevalence is increasing rapidly. Humans can ingest MPs through various pathways and their presence has been detected in multiple human organs, raising concerns about the potential toxic effects associated with plastic consumption. Epigenetic modifications of nucleic acids play crucial roles in various biological processes, including gene expression and tumorigenesis. Previous studies have demonstrated that exposure to certain environmental pollutants can influence disease pathogenesis by affecting epigenetic factors, including modifications of nucleic acids. However, the impact of MPs on epigenetic modifications of nucleic acids remains largely unexplored. In this study, we systematically investigated the alterations in epigenetic modifications of DNA and RNA following exposure to polystyrene microplastics (PS-MPs). We utilized liquid chromatography-tandem mass spectrometry (LC-MS/MS) to simultaneously analyze two DNA epigenetic modifications of 5-methylcytosine (5mC) and 5-hydroxymethylcytosine (5hmC), along with twenty RNA epigenetic modifications from small RNA and nine epigenetic modifications from mRNA. We measured changes in the levels of DNA and RNA modifications across six tissues (heart, liver, spleen, lung, kidney, and intestine) in mice after PS-MPs exposure. The results indicated that exposure to PS-MPs significantly altered the landscape of epigenetic modifications in nucleic acids. Furthermore, we observed tissue-specific effects, suggesting that different organs respond uniquely to PS-MPs exposure. Additionally, the correlation patterns between DNA and RNA modifications changed following PS-MPs exposure. These findings provide valuable insights suggesting that PS-MPs exposure may alter the patterns of epigenetic modifications in nucleic acids, potentially leading to adverse health effects.

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While plastic products offer convenience, they contribute significantly to environmental issues [1,2]. The degradation of these plastics can result in the formation of microplastics (MPs), which are widely distributed within the environment [3]. MPs, defined as particles <5 mm in size, are emerging as a novel class of environmental pollutants, with their prevalence demonstrating a rapid increase [4,5].

In recent years, MPs have also been identified in various human organs, giving rise to concerns about the potential toxic effects associated with the ingestion of plastic [6,7]. Exposure to MPs can occur through multiple routes, including ingestion, inhalation, and dermal penetration (Fig. S1 in Supporting information) [8]. Ingestion is considered the primary route of exposure, as MPs have been found to be present in various food items, such as seafood, salt, and drinking water [8]. Additionally, MPs can also be ingested through accidental consumption of plastic debris or through the ingestion of microplastic-contaminated soil and dust [8]. Inhalation of microplastics can occur through the inhalation of airborne microplastic particles, which can be generated from

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the breakdown of larger plastic debris [8]. Dermal penetration, although less studied, may also occur through direct contact with microplastic-contaminated water or soil [8]. It has been assessed that the number of microplastic particles in common foods, relative to the recommended daily intake, equates to the average person ingesting between 39,000 and 52,000 microplastic particles annually through food and beverages [9]. In consideration of respirable plastic particles in the atmosphere, the total number of plastic particles entering the human body ranges from approximately 74,000 to 121,000 per year [9].

It has been demonstrated that MPs can cause damage to the human body in a manner analogous to that of atmospheric particulate pollutants. This damage occurs at the cellular level, where MPs penetrate cell membranes, induce inflammation, and increase the risk of death from a range of diseases, including cardiovascular and respiratory diseases, as well as cancers [10]. Additionally, animal studies have demonstrated that the inhalation of microplastics can result in the disruption of respiratory function, the activation of lung inflammation, the exacerbation of oxidative stress, and even the induction of pulmonary fibrosis [11]. Furthermore, microplastics can act as vectors for the transfer of exogenous harmful chemicals, proteins, and toxins that coexist with them, including potential pathogenic agents. For example, previous studies have shown that microplastics can act as a vector for the accumulation of various pollutants, including polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), phthalates, and pesticides, with a concentration factor of up to 10^6 times higher than the surrounding seawater [12].

Exposure to environmental pollutants or harmful substances can contribute to disease pathogenesis by influencing epigenetic factors that regulate gene expression [13–18]. Eukaryotic genomes carry a wide range of modifications [19–26]. 5-Methylcytosine (5mC) is the most prevalent DNA modification in mammals [27–30]. Due to its significant functions in biological processes, including gene expression, embryogenesis and tumorigenesis [31,32], 5mC is often regarded as the fifth base. It has been reported that exposure to microplastic particles results in a decline in DNA methyltransferase gene expression in zebrafish embryos, which likely resulted in a decline in DNA methyltransferase activity and a state of DNA hypomethylation, indicating that microplastic exposure affects the methylation levels in animal DNA [33]. In addition to 5mC, 5-hydroxymethylcytosine (5hmC) has attracted considerable attention and is now regarded as the sixth base of mammalian genomes [34,35]. Beyond being an intermediate in the oxidation of 5mC, 5hmC is also a stable epigenetic modification found in mammalian genomes [34,36,37]. An increasing body of evidence suggests that 5hmC plays a direct role in regulating gene expression in both physiological and pathological states [38,39]. However, there is still a lack of investigation into the effects of microplastic exposure on 5hmC levels in DNA.

In addition to DNA modifications, RNA is subject to a wide range of post-transcriptional modifications that exert a significant influence critical pathway, including RNA processing and metabolism [40,41]. RNA modifications are ubiquitous in a diverse range of organisms, including vertebrates, plants, yeast, bacteria, archaea, and viruses [42–45]. N^6-methyladenine (m^6A) is one of the most prevalent and extensively characterized RNA modifications, with a pivotal function in diverse physiological processes [46–49]. For example, m^6A is involved in the regulation of stem cell differentiation, animal growth and development, cancer progression, and immune responses [50,51]. A previous study showed that m^6A in cardiac long noncoding RNAs and circular RNAs exhibited disparate distributions and abundances in mice following exposure to MPs [52]. To date, over 160 chemically diverse RNA modifications have been identified in transfer RNA (tRNA), ribosomal RNA (rRNA), and messenger RNA (mRNA) [53,54]. Aberrant

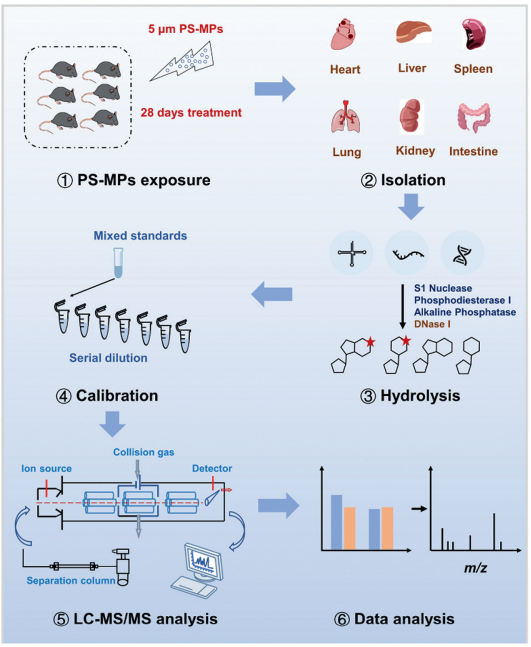


Fig. 1. Schematic illustration for PS-MPs exposure and mass spectrometry profiling of DNA and RNA modifications.

RNA modifications have been linked cellular dysfunction and are associated with multiple human diseases, including cancer [55–60]. However, little is known about whether microplastic exposure affects these epigenetic modifications in RNA.

In this study, we aimed to conduct a comprehensive investigation into the alterations in epigenetic modifications of DNA and RNA following exposure to PS-MPs. To achieve this, we developed a liquid chromatography-tandem mass spectrometry (LC-MS/MS) method capable of simultaneously analyzing multiple naturally occurring modifications in DNA and RNA. We measured changes in the levels of these modifications across six tissues in mice after PS-MPs exposure. The animal model for microplastic exposure comprised a total of 12 mice, which were randomly divided into two groups. The mice were administered ultrapure water containing or not containing PS-MPs *via* oral gavage. The first group ($n = 6$) received ultrapure water and served as the control, while the second group ($n = 6$) was administered 30 mg/kg of body weight PS-MPs for a period of 28 days. Body weight was recorded at three-day intervals throughout the exposure period. Following 28-day exposure, the mice were sacrificed and the following tissues were collected and processed for DNA and RNA isolation: heart, liver, spleen, lung, kidney, and intestine (Fig. 1). All animal experiments were performed in compliance with the principles and guidelines for the use of laboratory animals and approved by the Care and Use Committee of Wuhan University (Ethical approval number: WP20230426).

We first assessed the influence of PS-MPs on body weights and organ indexes in mice following 28 days of continuous intragastric administration. The results revealed no notable discrepancies in body weights and organ indexes between the control group and the PS-MPs exposure group under current exposure conditions (Figs. 2A and B). These results align with previous studies showing that PS-MPs typically do not induce significant changes in body weights [61].

Subsequently, an investigation was conducted to ascertain whether PS-MPs induced tissue injury by examining liver and intestinal tissues in mice following PS-MPs exposure. Histopathological analysis revealed that liver tissue from the control group dis-

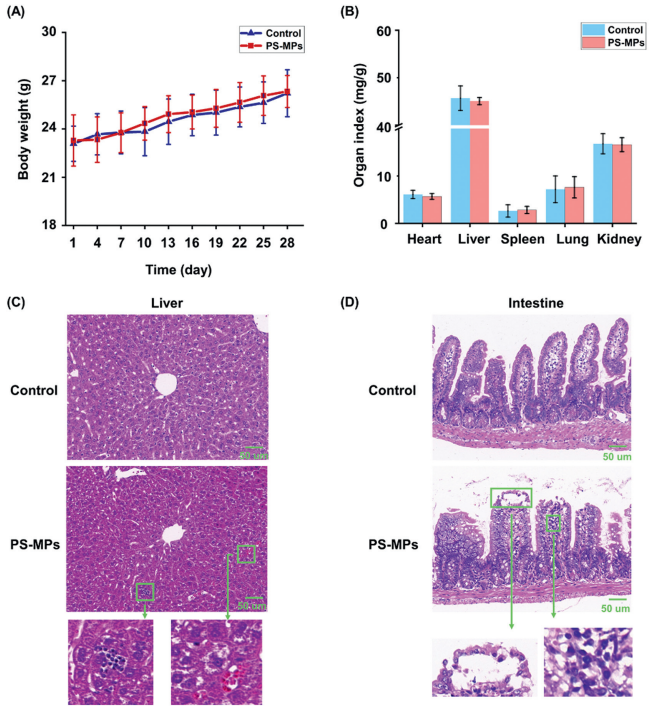


Fig. 2. (A) Effect of PS-MPs exposure on body weights. (B) Effect of PS-MPs exposure on organ indexes. (C) H&E staining of the liver tissue in the control and PS-MPs groups. (D) H&E staining of the intestine tissue in the control and PS-MPs groups. Scale bar: 50 μm.

played normal hepatic cells with intact cytoplasm, nucleus, nucleolus, and central vein (Fig. 2C). In comparison to the control group, the MPs exposure groups showed disorderly arranged liver cells in liver tissue (Fig. 2C). Inflammatory cell infiltration was observed in liver tissue of mice exposed to MPs (Fig. 2C). Regarding intestinal analysis, the overall structure of the intestinal tissue in the control group was observed to be normal, with neatly arranged intestinal villi (Fig. 2D). However, in the PS-MPs group, there was a slight abnormality in the structure of the intestinal tissue (Fig. 2D). The damage was primarily predominantly evidenced by the shedding of intestinal villi epithelium cytolysis and necrosis, with a minor infiltration of inflammatory cells (Fig. 2D).

We developed an LC-MS/MS method with the objective of conducting a comprehensive investigation into the effects of PS-MPs exposure on DNA and RNA modifications across six major mouse tissues: heart, liver, spleen, lung, kidney, and intestine (Fig. 3). Under optimized analytical conditions, we effectively distinguished two DNA modifications (5mC and 5hmC) and twenty RNA modification (t^6A, Am, i^6A, m^1A, $m^{6,6}A$, m^6A, m^1I, I, m^5Um, m^5U, Um, m^5C, m^3C, Cm, $m^{2,2,7}G$, $m^{2,2}G$, m^2G, m^1G, m^7G and Gm) standards in the LC-MS/MS analysis (Figs. 3A and B, Table S1 in Supporting information). The chemical structures of these modified nucleosides are shown in Fig. S2 (Supporting information). Additionally, the retention times of detected peaks in the extracted-ion chromatograms from DNA and RNA in mouse tissues were consistent with the standards (Figs. 3A and B, Figs. S3-S7 in Supporting information). The same precursor ions and product ions (MRM mass transitions used for monitoring modifications, Table S2 in Supporting information) and consistent retention times between the nucleoside standards and detected peaks validated the detection of

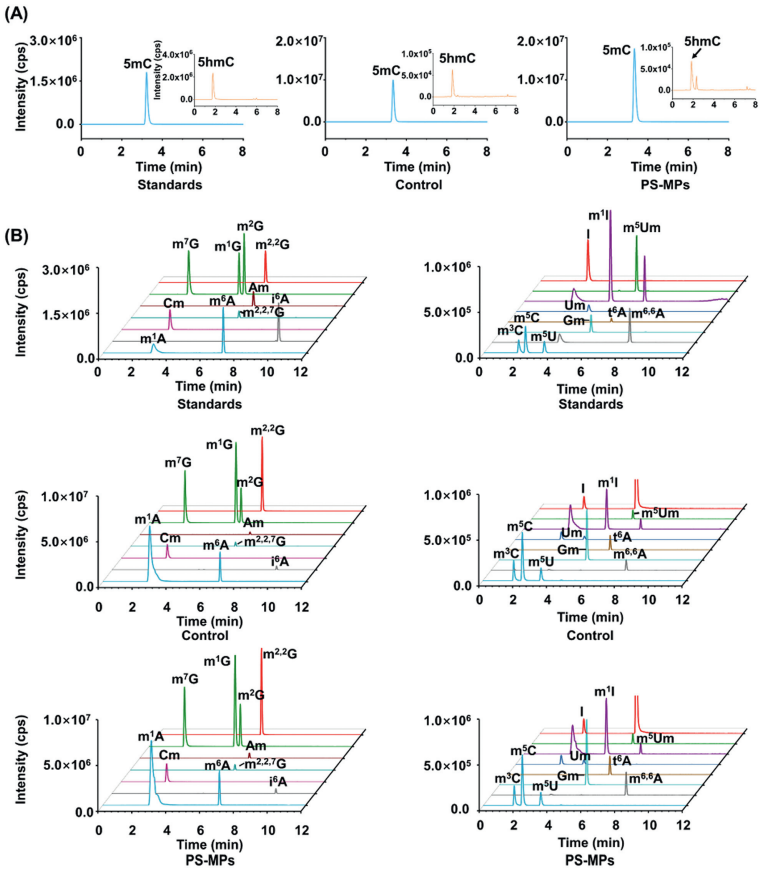


Fig. 3. The extraction-ion chromatograms of DNA and RNA modifications from mouse liver. (A) DNA modification standards, and DNA modifications detected from control group and PS-MPs exposure group. (B) RNA modification standards, and RNA modifications detected from control group and PS-MPs exposure group.

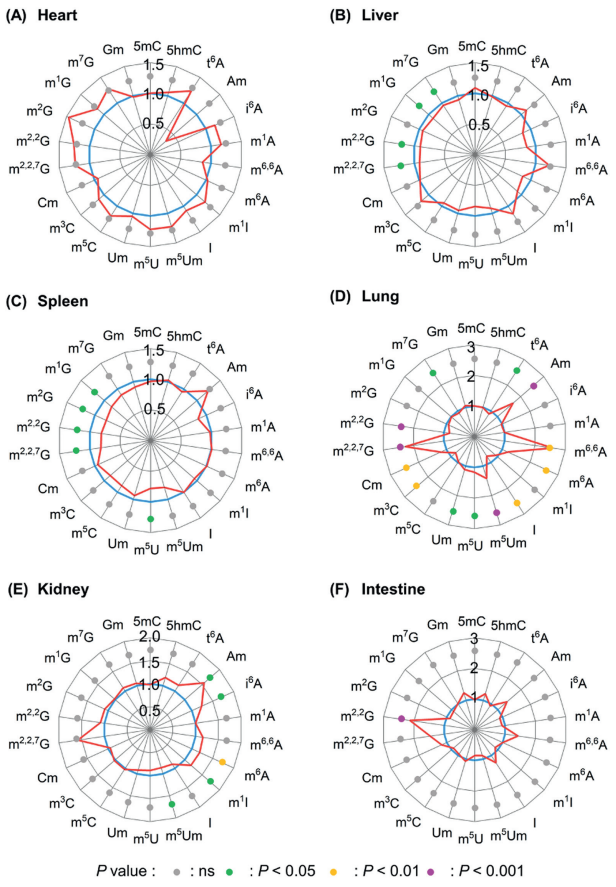


Fig. 4. Comparison of DNA and RNA modifications levels from mouse tissues. (A) Heart. (B) Liver. (C) Spleen. (D) Lung. (E) Kidney. (F) Intestine. Shown in blue and red represent the modifications from the control and PS-MPs groups, respectively. The average level of each DNA and RNA modification from six mouse tissues in PS-MPs group was normalized to the control group.

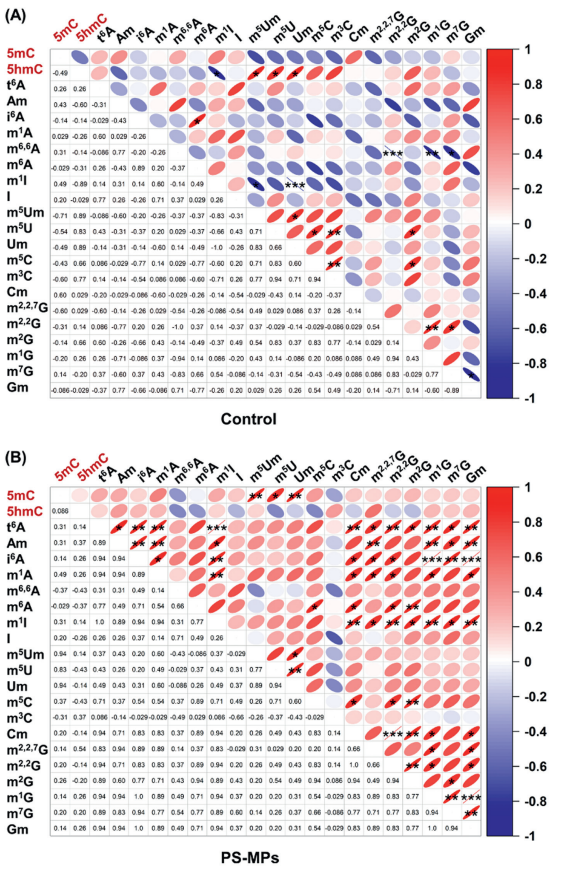


Fig. 5. Correlation analysis between DNA and RNA modifications in liver from the control and PS-MPs groups. (A) The control group. (B) The PS-MPs group. $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

the two DNA modifications and twenty RNA modifications in the mouse tissues. Subsequently, calibration curves were constructed for the purpose of quantifying these modifications. The results exhibited satisfactory linearity with coefficients of determination (R^2) exceeding 0.99 (Tables S3 and S4 in Supporting information). Overall, the developed LC-MS/MS method enabled us to comprehensively and accurately assess DNA and RNA modifications in tissues collected from both control and PS-MPs exposure groups.

The effects of PS-MPs exposure on DNA and RNA modifications were assessed using the established LC-MS/MS method. The quantification results indicated no significant changes in the levels of 5mC and 5hmC in DNA across the six tissues: heart, liver, spleen, lung, kidney, and intestine (Fig. 4 and Fig. S8 in Supporting information). The trends in modification changes of small RNA (<200 nt) varied across different tissues. In particular, the levels of Am, $m^{6,6}A$, m^6A, m^5Um, m^5U, Um, Cm, $m^{2,2,7}G$ in the lung, as well as Am, i^6A, m^6A and m^1I in the kidney, and $m^{2,2}G$ in the intestine, were significantly increased in the PS-MPs group compared to the control group (Fig. 4 and Figs. S9-S15 in Supporting information). Additionally, we observed significant decreases in certain modifications: $m^{2,2,7}G$, $m^{2,2}G$, m^1G and m^7G in the liver (Fig. 4 and Fig. S10); m^5U, $m^{2,2,7}G$, $m^{2,2}G$, m^2G and m^1G in the spleen (Fig. 4 and Fig. S11); and t^6A, I, m^3C, $m^{2,2}G$ and m^7G in the lung (Fig. 4 and Fig. S12), along with m^5Um in the kidney (Fig. 4 and Fig. S13), when comparing the PS-MPs group to the control group.

Transfer RNAs (tRNAs) typically exhibit a length in size from 70 to 90 nucleotides [62–64], constituting the majority (~90%) of small RNA [65]. Modifications in tRNAs are vital for the maintenance of their distinctive clover-leaf structures and ensuring accurate translation of genetic information [62]. These modifications are instrumental in the performance of various tRNA function, including intracellular localization, codon decoding fidelity, and structural stability [66]. Following exposure to PS-MPs, the levels of m^6A and Am were elevated in the lung and kidney tissues. Am has been shown to enhance the accuracy and efficiency of protein synthesis by stabilizing tRNA binding to ribosomes [67]. Similarly, m^6A on tRNA has been reported to enhance the stability of tRNA and improve the decoding ability of mRNA [68]. Consequently, disruptions in tRNA modifications induced by PS-MPs exposure may affect protein synthesis, potentially leading to dysregulation of cellular homeostasis and impacting cell function.

It is established that a number of epigenetic marks operate in concert to regulate gene expression and cellular processes [69–71]. However, the relationship between DNA modifications and RNA modifications in mouse tissues, along with their alterations following PS-MPs exposure, remains unexplored. In our study, we identified significant correlations between specific modifications and their changes after PS-MPs exposure (Fig. 5 and Figs. S16-S20 in Supporting information).

In the control group of the liver, the level of 5hmC showed positive correlations with m^5Um, m^5U and Um. Additionally, there was a positive correlation between i^6A and m^6A, as well as between m^5Um and Um. The level of m^5U was positively correlated with m^5C, m^3C and m^2G, while m^5C showed positive correlations with m^3C and m^2G. The level of $m^{6,6}A$ showed negative correlations with $m^{2,2}G$, m^1G and m^7G (Fig. 5A). After exposure to PS-MPs, the correlations between these modifications changed significantly. In liver, the level of 5mC exhibited positive correlations

with m^5Um, m^5U and Um. Additionally, the level of t^6A showed positive correlations with Am, i^6A, m^1A, m^1I, Cm, $m^{2,2,7}G$, $m^{2,2}G$, m^2G, m^1G, m^7G and Gm. The level of Am also demonstrated positive correlations with i^6A, m^1A, m^1I, $m^{2,2,7}G$, m^1G, m^7G and Gm (Fig. 5B).

Exposure to PS-MPs significantly altered the landscape of epigenetic modifications in nucleic acids. A comparison of the correlations of modifications in tissues between the control group and the PS-MPs exposure group revealed a significant increase in the number of modifications that exhibited strong correlations in both liver and intestine tissues following PS-MPs exposure. In contrast, the number of modifications showing significant correlations decreased in the heart, spleen, lung, and kidney tissues after PS-MPs exposure. These results underscored the tissue-specific effects of PS-MPs exposure on epigenetic modifications, indicating that distinct organs may exhibit disparate responses to PS-MPs. This suggests that PS-MPs exposure can have distinct effects on different tissues, and not all tissues are equally affected. The accumulation of PS-MPs in the body may vary across organs, leading to differential toxic responses. To further clarify the mechanisms underlying the observed changes in modification patterns across different organs, evaluating the levels of oxidative stress and inflammation in different tissues may provide valuable insights.

Recent studies have indicated the presence of coordinated regulation among an array of epigenetic markers, such as the relationships between DNA methylation and histone marks [72], as well as between RNA modifications and histone marks [69,70]. Nevertheless, the regulatory behaviors and relationships among different modifications have been less frequently investigated. Prior research has identified certain correlations between RNA modifications, for instance, the tRNA C38 m^5C modification, which depends on the initial attachment of tRNA 34 queuosines [73]. Our results suggest that there may be widespread coordinated regulation among RNA modifications. It seems plausible to suggest that a coordinated regulatory network exists to govern RNA modifications, in order to maintain tissue-specific homeostasis. By identifying specific RNA modification patterns in different tissues and exploring the relationships among these modifications, we can gain deeper insights into the intricate regulatory functions and mechanisms underlying tissue specificity. Additionally, significant modifications in the correlations between these modifications were observed in response to PS-MPs exposure, indicating that PS-MPs exposure can influence the coordinated regulatory processes among distinct epigenetic marks. This finding provides a basis for further research into the intricate interplay between DNA and RNA modifications in response to PS-MPs exposure.

Previous studies have demonstrated that microplastics accumulating in the liver may result in impaired function [74]. In the present study, we detected and quantified nine modifications in liver mRNA, including m^6A, m^6Am, m^1A, m^5C, Am, Um, m^7G, Gm, and Cm. The results demonstrated a notable reduction in m^1A levels following PS-MPs exposure ($P < 0.05$, Fig. 6A). We quantitatively analyzed the expression levels of the methyltransferase genes *Trmt6* and *Trmt61a*, which are responsible for the formation of m^1A, as well as the demethylase gene *Alkbh3* (Fig. 6B, Table S5 in Supporting information). However, the results revealed no significant changes between the control and PS-MPs exposure groups. It is postulated that there may be undiscovered m^1A-modifying enzymes that contribute to the reduction in m^1A levels in mRNA following exposure to PS-MPs. Furthermore, future research on assessing the activities of these enzymes after exposure to PS-MPs may offer valuable insights into the underlying mechanisms. m^1A plays crucial roles in development and disease, including the regulation of growth, development, stress responses, and tumorigenesis [75,76]. m^1A sites are predominantly enriched in the 5′ untranslated regions (UTRs) of mRNAs, where they are essential for

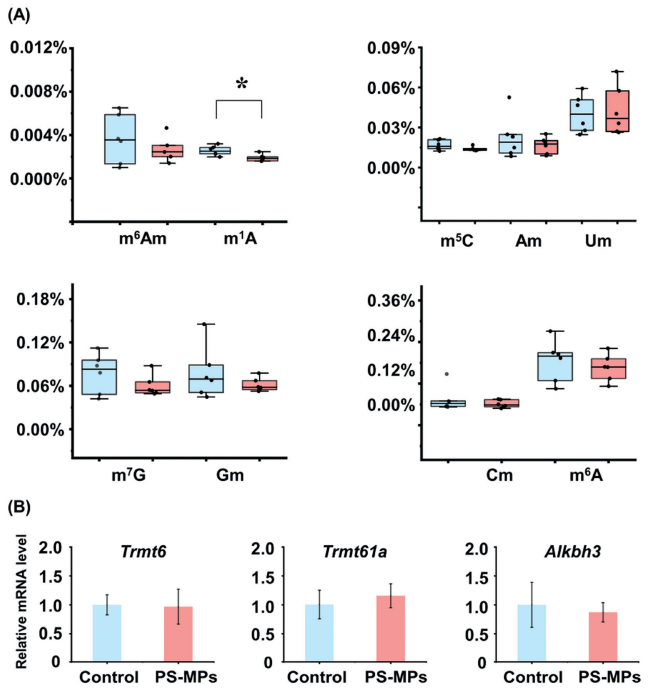


Fig. 6. The measured levels of modifications in mRNA and expression of m^1A-modifying enzymes in mouse liver tissues upon PS-MPs exposure. (A) The measured levels of modifications in mRNA from mouse live tissues between control and PS-MPs groups. (B) The relative expression levels of *Trmt6*, *Trmt61a*, and *Alkbh3* genes from mouse live tissues between control and PS-MPs groups. Data are presented as the means ± SD ($n = 6$). Data analysis was conducted using an unpaired two-tailed Student's *t*-test to determine statistical significance. $^*P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

maintaining protein translation [40]. Consequently, the decreased level of m^1A in mRNA following exposure to PS-MPs may lead to aberrant protein translation, ultimately disrupting normal biological processes. This result indicates that, in addition to modifications in small RNA (<200 nt), PS-MPs exposure can also influence alterations in mRNA modifications. However, further investigation is required to elucidate the precise mechanisms underlying these changes. Future research into the mechanisms of the alteration of m^1A in mRNA induced by PS-MPs exposure may provide valuable insights into the broader implications of microplastics on RNA epigenetic modifications and their effects on gene expression and cellular function.

PS-MPs may influence epigenetic modifications by inducing oxidative stress, which can be triggered by the production of intracellular reactive oxygen species (ROS) [77]. The elevated levels of ROS may compromise the function of enzymes involved in DNA and RNA modification, ultimately leading to alterations in epigenetic marks and impacting gene expression [78]. Moreover, PS-MPs can trigger immune responses or activate inflammatory pathways, resulting in the release of pro-inflammatory factors that can, in turn, influence the epigenetic modifications of nucleic acids. Certain modifications to DNA and RNA may be catalyzed by the same enzyme or family of enzymes. For instance, TET dioxygenases are involved in the oxidation of 5mC in DNA and m^5C in RNA [79,80]. Alterations in the activity or expression of these enzymes due to PS-MPs exposure could potentially lead to changes in both DNA and RNA modifications. Furthermore, there may be a cascade of effects between specific modifications. Changes in DNA methylation patterns may influence the occurrence of RNA modifications by altering chromatin structure or the binding of transcription factors, which in turn can affect RNA modifications. To gain a deeper understanding of the toxicological effects of PS-MPs, future re-

search could employ comprehensive genome-wide analyses of DNA methylation and hydroxymethylation, as well as transcriptome-wide analyses of key RNA modifications. This approach may offer valuable insights into the underlying mechanisms of PS-MPs' toxicological effects, shedding light on the complex interactions between PS-MPs exposure and epigenetic regulation.

In summary, we conducted a comprehensive analysis using LC-MS/MS to investigate the impact of PS-MPs exposure on the landscape of epigenetic modifications in nucleic acids within mouse tissues. We detected and quantified two modifications in DNA, twenty modifications from small RNAs (<200 nt), and nine modifications from mRNA. The results demonstrated significant alterations in RNA modifications across various mouse tissues resulting from PS-MPs exposure, with different organs exhibiting unique responses. Additionally, the correlation patterns between DNA and RNA modifications changed following PS-MPs exposure. It is noteworthy that there was a significant decrease in m^1A level in mRNA from live tissues after PS-MPs exposure. The findings of this study highlight the impact of PS-MPs on epigenetic modifications in DNA and RNA, representing the first comprehensive evaluation of microplastic effects on nucleic acid epigenetic modifications. This study highlights the broader implications of microplastics on epigenetic modifications and their potential effects on gene expression from an epigenetic perspective.

Declaration of competing interest

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

CRediT authorship contribution statement

Tian Feng: Validation, Methodology, Formal analysis, Data curation, Writing – original draft, Conceptualization. **Shu-Yi Gu:** Validation, Methodology, Formal analysis, Data curation. **Lu-Fei Shi:** Validation, Formal analysis, Data curation. **Yao-Hua Gu:** Formal analysis, Writing – original draft, Conceptualization, Supervision, Methodology, Investigation. **Bi-Feng Yuan:** Project administration, Conceptualization, Methodology, Investigation, Writing – review & editing, Funding acquisition, Supervision, Formal analysis.

Acknowledgments

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.cclet.2025.111546.

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