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Progress in Research on Microplastics and Nano-plastics Cardiac Toxicity

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Abstract:

In recent years, research on the biological toxicity of microplastics and nano-plastics has emerged in the medical field, especially with rapid progress in studies related to cardiovascular diseases. However, it should be objectively noted that the amount of clinical data from international research remains limited, while domestic research in related fields with access to clinical data sources is extremely scarce, and in-depth clinical investigations and research advances are urgently needed. This review summarizes the progress of studies on the cardiovascular toxicity of microplastics. Based on domestic and international basic research and the factually limited clinical research, it elaborates on the mechanisms of cardiovascular toxicity induced by microplastics, their biomarkers, and detection methods. Emphasis is placed on detection methods for direct evidence of biological origin and biomarkers, aiming to provide a reference for subsequent data collection in clinical research stages.

Keywords: Microplastics, Cardiovascular Toxicity, Biomarkers, Detection Methods

1. Background:

As a low-cost, highly malleable, and easily industrially producible synthetic polymer material, plastics have been widely used in industrial production and daily life over the past few decades. Due to their high malleability, additives are incorporated during the synthesis process to modify their chemical composition and improve their physicochemical properties. In addition to polyethylene (PE) widely used in food packaging, modified plastic varieties such as polyvinyl chloride (PVC) for packaging pipes and artificial leather, and polybutylene terephthalate (PBT), an engineering plastic used to replace metal components in complex chemical environments to bear mechanical stress, have emerged. Plastics have actually become substances that humans have to come into contact with in production and daily life. However, plastics can break down into microplastics (Microplastics & Nano-plastics) through physical and chemical means during production and use. Microplastics refer to plastic particles with a diameter of less than 5 mm, which can be subdivided into microplastics (Microplastics, MPs) with a diameter of ≤ 5 mm and nano-plastics (Nano-plastics, NPs) with a diameter of ≤ 1000 nm [1]. Even though microplastic particles are extremely small, they are not chemical cleavage monomers and still retain the basic physicochemical properties of polymer materials such as plastics and their modified products. They are chemically stable, not easy to degrade in the natural environment, and due to their small size, they are easy to migrate with the natural cycle of water. Studies in the field of environmental science have shown that microplastics have long existed in soil, water, and atmosphere. pattern on the scalp. Hormonal factors, genetics, micronutrient deficiencies, microinflammation, and stress are all implicated in its development [6]. AGA is the most common type of alopecia, accounting for approximately 90% of all Living in such an environment, the human body is at risk of microplastic exposure. Previous biological sampling in the natural environment and basic research on experimental animals have shown that microplastics can contact the human body through the skin, respiratory tract, and digestive tract, and microplastics of certain specific sizes can enter organisms through biological membrane structures, and be transported to various parts of the body through the blood circulation, mainly accumulating in

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blood-rich tissues and organs such as the heart, blood, lungs, liver, kidneys, and placenta [2]. However, in fact, we should also note that domestic and foreign clinical research is not much, and the sample size of the data is still small. As a rapidly developing country, China's annual plastic output will continue to increase. The 2022 China Plastic Processing Industry Report shows that China's annual plastic output is 77716 kt, and the production output and consumption capacity will continue to increase [3]. At the same time, the prevalence and incidence of cardiovascular diseases in China are increasing year by year. It is urgent to carry out clinical investigations and obtain clinical evidence support to help understand and reduce the possible occurrence of adverse endpoint events of cardiovascular diseases caused by microplastic exposure in patients. This review summarizes the relevant literature and discusses the toxic mechanism of microplastics on the cardiovascular system, biomarkers, and clinical evidence detection methods, providing a reference for subsequent clinical research.

2. Distribution of Microplastics in the Cardiovascular System

The cardiovascular system consists of the heart that provides the power for blood circulation, arteries, veins, capillaries that transport blood and exchange substances with tissues, and the blood flowing in them. In previous clinical evidence, researchers have confirmed the existence and continuous accumulation of microplastics in the heart tissue and circulatory system of the cardiovascular system through heart tissue samples and incision of human thrombus[4].

2.1 Distribution of Microplastics in the Heart

Current literature and basic research on the distribution of microplastics in the heart cavity generally believe that the heart is regarded as the power source of a closed circulatory system, and the chambers of the heart are regarded as the channels of blood. The concentration of microplastics in the heart depends on the amount of blood in the heart, because the blood in the heart will eventually carry microplastics to peripheral blood vessels and organs. Therefore, the heart is not the area with the highest concentration of microplastics, but it is still slightly higher than the surrounding tissues and organs. Ding et al. [5] found that in red tilapia (Oreochromis niloticus) exposed to microplastics at concentrations of 1μg/L, 10μg/L, and 100μg/L, the concentration of microplastic particles in the intestinal part of red tilapia was the highest, followed by the gills and heart, and finally the peripheral blood vessels and organs. At the same time, observations on zebrafish found that the accumulation of microplastics in the gastrointestinal tract can also come from plastics ingested through drinking or breathing, but particles larger than 150μm cannot be absorbed and are excreted through feces[6]. Therefore, the accumulation of microplastics in the heart can still be regarded as a key area of concern. The damage to the structure and function of the chambers is mainly caused by the secondary changes caused by the damage to the myocardial tissue caused by the activation of the body's immune channels and ROS reactions triggered by microplastic particles, including myocardial fiber rupture and remodeling, and pyroptosis of cardiomyocytes[7]. Plastic particles do not directly damage the heart itself.

Moreover, structural variability damage occurs more in the heart during embryonic development. Bojic et al. [8]found that after human induced pluripotent stem cells (hiPSC) were exposed to 20nm and 400nm MPs for 24 hours, atrioventricular valve (AVV) dysplasia and extracellular matrix (ECM) dysfunction occurred. In addition, there is no literature or direct evidence to support that microplastic particles can directly damage the developed heart valve system and cause functional damage.

In particular, Yang et al.[9] observed clinically derived heart tissue samples (including myocardium, pericardial adipose tissue, etc.) and found that in the human heart, the left atrial appendage, epicardial adipose tissue, and pericardial adipose tissue are more likely to accumulate microplastic particles, which provides direct evidence of microplastics for patients undergoing heart surgery. It also suggests the possible impact of surgery on the introduction of microplastics and the potential impact of microplastics on human health after changes in cardiac structure and function.

2.2 Distribution of Microplastics in Blood Vessels

Arteries are the first vascular segment where blood is pumped out of the heart, divided into two pathways. The first pathway is that blood enters the aorta and then shunts through several secondary arteries of the aortic arch to supply blood to the whole body step by step. Marfella R et al.[10] collected atherosclerotic plaques from 304 patients who underwent carotid endarterectomy, incised them, and observed them after lysis. Microplastics were found in 181 patients' atherosclerotic plaque samples. The average level of microplastics in polyethylene and polyvinyl chloride was no less than 20μg per mg of atherosclerotic plaque sample. A follow-up of no less than 30 months was completed for 257 patients in all samples, and it was found that the risk of primary adverse events in patients with microplastic particles detected in these atherosclerotic plaques was higher than that in patients without detection, with an incidence rate of about 3.35 times (95% confidence interval, $P < 0.01$). In fact, microplastics have been confirmed to exist in this blood flow pathway, and it is initially found that their existence may lead to an increased risk of primary adverse endpoint events. The second pathway is the coronary artery. Liu et al. [11]collected coronary artery tissues from 4 patients with severe coronary atherosclerotic lesions and observed after lysis. The concentration of microplastics was 156.50 μg/g of arterial tissue samples, confirming that microplastics also exist in coronary arteries and lead to atheroscle-

rotic plaques. Furthermore, Liu et al. also collected aortic tissue samples from patients with carotid atherosclerotic plaques and found that the microplastic content in coronary atherosclerotic plaque tissue samples and arterial tissue samples from patients with carotid atherosclerotic plaques was higher than that in aortic tissue samples from patients without aortic plaque disease. The clinical evidence collection and observation experiments of the two teams have confirmed the widespread existence of microplastics in arteries and suggested that they are related to the occurrence and development of atherosclerotic plaques. At the same time, there is still a lack of clinical evidence for observing atherosclerotic plaque tissues and thrombi from other arterial segments, and more sample sizes and evaluation dimensions are needed for discussion.

Capillaries are tiny blood vessels connecting arteries and veins, widely distributed in human tissues and organs, and function to provide a contact surface for gas exchange and material exchange between blood and tissues. Sun et al. [12]found in a study on zebrafish embryos that when embryos were placed in an environment of 100mg/L, thrombus formed in the tail capillaries and veins. The reason was that after microplastic particles in the tail capillaries caused mechanical obstruction, the blood flow speed slowed down. The obstructed blood vessels attracted the aggregation of coagulation factors and released active peptides, resulting in vascular edema and hypercoagulable blood, which extended to the venous blood flow with slow reflux, and finally increased blood viscosity, leading to thrombus formation. Lett Z et al.[13] studied that the activity of scramblases (substance transport structures composed of proteins located on the cell membrane structure) responsible for phospholipid translocation in PS-NPs regulated the functional interaction with adjacent tissues and coagulation cascade reaction, and enhanced red blood cell adhesion and thrombin generation.

As the core part of the entire cardiovascular system, blood has the ability to transport microplastics to the whole body, but microplastics themselves will also leave traces of their existence in the blood. Ballesteros S et al. [14]found in a study on whole blood samples that microplastic particles are not only floating in blood transport, but also absorbed by white blood cells and monocytes; further, Mohr.K et al. [15]exposed minnows to artificially synthesized microplastic particles (with diameters ranging from 80-170nm) and found that these plastic particles were absorbed by monocytes, granulocytes, and myeloid dendritic cells but not by lymphocytes. Heather A et al. [16]detected microplastics in the blood of 22 healthy volunteers, with an average value of 1.6μg/mL blood. The main component of microplastics was polyethylene (PET). However, microplastics actually exist in human blood and can be recognized by the immune system, but extensive detection has not been put into clinical application.

For the blood system, a recent case report of bone marrow transplantation mentioned that microplastics may interfere with the normal bone marrow hematopoietic function of the human body[17].

3. Toxic Mechanisms and Biomarkers of Microplastic-Induced Cardiovascular System Damage

3.1 Oxidative Stress Reaction of Reactive Oxygen Species

ROS reaction refers to a series of biochemical reactions involving reactive oxygen species (ROS). ROS mainly includes superoxide anion, hydroxyl radical, hydrogen peroxide, etc. These substances are naturally produced during cell metabolism and can act as signal molecules to participate in various physiological regulations. However, when the production of ROS is too much or too little, it may cause oxidative stress, leading to mitochondrial dysfunction, cell autophagy, cardiomyocyte necrosis and other adverse consequences. Zhang et al. [18]exposed chicken chicks to 5 μm, 1-100mg/L environment. After 42 days, through pathological examination, severe ultrastructural changes were found in the chicken heart at the histological level, including inflammatory cell infiltration, disappearance of mitochondrial cristae, and myocardial fiber rupture. The detection of physiological indicators found that MPs induced abnormal increase of antioxidant enzyme content and excessive production of ROS, thus triggering cell pyroptosis through the NF-κB-NLRP3-GSDMD axis, causing myocardial cell damage and aggravating myocardial inflammation. Ye et al. [19]confirmed this through observation in mice, and also mentioned the phenomenon of decreased cTnT and decreased heart weight index. Wei et al. [20]used 0.5mm microplastics in rats, and the concentration of 0.5-50mg/L was taken orally. The research team believed that PS-MPs first induced oxidative stress in rat hearts, triggered myocardial cell apoptosis, and then caused cardiac fibrosis and cell edema by activating the Wnt/β-catenin pathway. Wei et al. further studied and proved that PS-MPs cause inflammation and pyroptosis through oxidative stress, leading to myocardial fibrosis through ROS generation and nuclear factor κB (NF-κB) activation. PS-MPs activate the NLR family pyrin domain containing 3 (NLRP3)/-Caspase-1/Gasdermin D (GSDMD) pathway, and overexpress NLRP3, Caspase-1, IL-1β, IL-18, ASC, GSDMD, NF-κB, COX-2, iNOS, and IL-6. In addition, Zhang et al. [21]showed in the microplastic exposure experiment through inhalation that the longer the exposure time and the higher the exposure concentration of microplastic particles of the same size, the content of malondialdehyde (MDA) increased significantly, the activity of catalase (CAT) decreased, and the activities of glutathione peroxidase (GSH-Px) and superoxide dismutase (SOD) decreased in long-term exposure for more than 4 weeks. Further research

reflected that as indicators for evaluating myocardial damage, echocardiography, myocardial tissue microscopy, troponin, atrial natriuretic peptide, and myoglobin should also be included in the consideration of subsequent clinical research. In addition, excessive oxidative stress will also damage vascular endothelium. Vascular endothelial damage, as the initial event of atherosclerotic plaque, further promotes the aggregation of macrophages and coagulation factors in blood vessels, slows down blood flow, and finally contributes to the formation of atherosclerotic plaque and thrombus[22].

3.2 Immune Inflammatory Response

Most of the microplastic particles in the body float in the blood and are directly captured by monocytes in the form of phagocytosis. However, the plastic particles themselves cannot be directly degraded in the human body. Monocytes-macrophages wrap the plastic particles and cannot metabolize them for a long time. They deposit on the basement membrane of the blood vessel wall and are recognized as foreign bodies by the immune system. The classical complement pathway is activated, attracting neutrophils and macrophages to gather. Neutrophils release proteolytic enzymes, elastase and other substances to damage the vascular endothelium. After vascular endothelial damage, coagulation factors gather and activate, and release active peptides, leading to local vascular edema and local blood hypercoagulable state. Objectively, microplastics activate the phagocytosis of macrophages, promote macrophages to accelerate phagocytosis of lipids through up-regulating the expression of MARCO, accumulate on the vascular endothelium, and promote the formation of atherosclerotic plaques and thrombi[23]. Li et al. [24] collected mouse feces after mice ingested PE microplastics and observed that the composition and diversity of intestinal microbiota changed. The intestinal microbiota in the group fed with high concentration of microplastics showed more microbial species, and the bacterial abundance and flora diversity increased. Different concentrations of microplastics can increase the secretion of pro-inflammatory cytokine IL-1α in serum, but different concentrations of microplastics will affect the secretion of specific cytokines. Microplastics reduced the percentage of Th17 and Treg cells in CD4+ cells, but the ratio of Th17/Treg cells remained unchanged. The impact of low concentration of microplastics was not significant, but high concentration of microplastics tended to induce intestinal inflammation by activating TLR4 signal, thus entering the circulatory system after being phagocytized by macrophages, and causing inflammation in blood-rich areas such as heart, spleen, and liver. Furthermore, Zhang et al. [25]conducted histopathological analysis on the heart tissue of mice ingesting microplastics, showing that the mouse myocardium was obviously broken, and the cardiomyocytes were arranged disorderly with a large number of inflammatory cell infiltration. After endothelial injury and myocardial pyroptosis, the research team detected the levels of markers in the NF-κ-B inflammation pathway. The mRNA and protein expressions of pro-inflammatory factors TNF-α, iNOS, COX-2, and IL-6 were significantly up-regulated, and IκB-α was significantly reduced[26]. The increase of IL-6 can be used as an indicator that the inflammatory reaction has begun[27].

3.3 Interfering with Lipid Metabolism

Microplastics interfere with lipid metabolism, causing lipid metabolism disorders and their secondary pathological changes, including hyperlipidemia and even coronary atherosclerosis. Pedro et al. [28]exposed zebrafish from embryo to larval development stage to nanoscale polystyrene particles (PS, diameter 22nm) and polymethyl methacrylate (PMMA, diameter 32nm) particles. It was found that in the samples exposed to PS, regardless of the diameter, the levels of lactate dehydrogenase (LDH) and isocitrate dehydrogenase (IDH) decreased, and the glycogen (Gly) level increased, that is, the levels of aerobic metabolism and anaerobic metabolism decreased, thus reducing the energy produced by zebrafish for lipids. At the same time, in terms of the trend of biomarkers, the higher the exposure concentration of microplastic particles and the smaller the diameter of the exposed particles, the more obvious the above indicators change; for PMMA, the change of Gly is not significant, and the levels of aerobic metabolism and anaerobic metabolism generally decrease. Although the aerobic metabolism increases and anaerobic metabolism decreases at the lowest exposure concentration of 0.01mg/L, the actual observation shows that there is no concentration dependence. The team think that microplastics first cause inflammatory reaction, then lead to mitochondrial cristae damage, which can not realize effective energy conversion, interfere with lipid metabolism, and lead to increased Gly level, decreased LDH and IDH levels. This speculation was confirmed in the study by Zhang et al. [29]on mice exposed to microplastics through inhalation (polystyrene microplastic particles, diameter 40nm, exposed to 0 μg/day, 16 μg/day, 40 μg/day, 100 μg/day for 12 weeks). Mice exposed to the above microplastic concentrations for only one week, the concentrations of three biomarkers released in serum, namely CTnT, NT-proBNP, and LDH, increased significantly, and the higher the concentration, the higher the concentration over time. Furthermore, the energy metabolism disorder of heart tissue is mainly caused by the damage to the structure of mitochondrial cristae, which slows down the metabolism and energy conversion of acetyl-CoA to lipids, leading to TCA cycle metabolism disorder. It happens that the function of cardiomyocytes depends heavily on ATP, and the abundance of mitochondria is the highest. The impact of energy

metabolism disorder is more significant. At the same time, the metabolism level of citrate and propionate decreases after microplastics cause mitochondrial damage, and the hydrolysis amount of ATP decreases, further leading to the decrease of myocardial contractility and stroke volume.

4. Detection Methods for Clinical Evidence of Microplastic Particles

4.1 Visual and Optical Microscopy Method

Visual and optical microscopy observation of microplastics is the initial research method for microplastics. For larger microplastic particles (1mm-5mm in diameter), they can be directly identified, but under natural conditions, the shape, size, and industrial use of microplastic particles are different, and their colors are also different, so it is difficult to distinguish the type and content of plastics by naked eyes[30]. Although observation under fluorescence-stained optical microscope helps to improve the recognition success rate, due to the increasing diversity of microplastic particles in shape, size, and color, and the different affinity between fluorescent dyes and different types of plastic particles, the difficulty of direct recognition by human eyes is increased, so visual and optical microscopy is no longer the mainstream[31].

4.2 Raman Spectroscopy Method

Raman spectroscopy is an analytical method based on Raman scattering effect, which analyzes the scattering spectrum with different frequencies from the incident light to obtain information about molecular vibration and rotation, and is applied to molecular structure research. In recent years, it has been used for the identification of microplastics. The plastic component identification is realized by comparing the obtained spectral information of polymer functional groups with the standard library. Mistri M et al. [32]conducted qualitative and quantitative analysis on plastics in sardines in the ocean by μ-Raman spectroscopy, indicating that this method can actually be used for the identification of microplastic samples taken from organisms.

The advantage of Raman spectroscopy is that it is a non-destructive microplastic analysis method. Moreover, Raman spectrometer can also be combined with microscope (Micro-Raman). Because its laser beam is small, the spatial resolution is high, even up to 1μm, which can effectively distinguish the types of tiny particles such as microplastics. In addition, Raman spectroscopy is a non-contact measurement, which can reduce contact pollution of samples and facilitate other subsequent detections[33]. In the analysis process, large microplastic particles can be directly picked out, and microplastic samples smaller than 500 μm can be directly analyzed on the filter membrane, but there are some limitations. For example, Raman spectroscopy is sensitive to chemical pigments in microplastics, which may affect the analysis of polymers; impurities and matrix components in samples, especially additives with fluorescent properties, will interfere with the measurement results, bringing great difficulties to spectral analysis, so it is necessary to process the samples clean before analysis to ensure their purity[34].

4.3 Fourier Transform Infrared Spectroscopy Method

Fourier transform infrared spectroscopy (FT-IR) is a method for structural analysis according to the absorption characteristics of material molecules to infrared rays of different infrared wavelengths. At present, it has become an important tool for microplastic component analysis. Its principle is that due to the differences in molecular structure of different substances, when irradiated by infrared rays, the groups in the sample molecules absorb infrared radiation, causing the transition of molecular vibration and rotation energy levels, generating molecular absorption spectrum, thus obtaining information about specific functional groups and chemical bonds in the sample, and think the type and structure of compounds by comparing with the polymer spectrum library. Common compounds have their own characteristic infrared absorption spectra, just like fingerprints. The absorption peak of a single type of plastic is almost unique, and the identification accuracy is high. It has the advantages of fast analysis speed and no damage to samples, and has been widely used in the analysis of biological tissues and microplastics[35]. The identification of each suspected microplastic sample by FT-IR avoids the misjudgment caused by the subjective factors of operators and improves the accuracy. FT-IR has three modes: transmission, reflection, and attenuated total reflection, which have different application ranges in the field of microplastic analysis. The transmission mode is suitable for samples that are thin enough to ensure that infrared light can pass through. The detectable thickness of samples depends on the optical characteristics of plastics, such as transparent plastics can detect greater thickness. The reflection mode is suitable for opaque samples with a certain thickness. If the sample surface is irregular, it is easy to produce unstable spectra. Attenuated total reflection (ATR) mode is a surface contact analysis method, which is not sensitive to the thickness of the sample. It can analyze the composition of various irregular and uneven samples, and has a wide range of applications. However, ATR probe contacts the sample for analysis, which may damage fragile or highly weathered samples; in addition, the probe is easy to be polluted or even damaged by hard or sharp inorganic substances remaining on the sample surface, so it is necessary to ensure that the sample surface is clean during operation[36].

4.4 Pyrolysis-Mass Spectrometry Method

Thermal analysis technology studies various transformations and reactions of materials under temperature control, and is an important analytical testing method for studying the thermal decomposition process and reaction kinetics of polymers. Zhang Xiangnan et al. [37]proposed that in recent years, the combined use of pyrolysis (Pyr) with spectroscopy and gas chromatography-mass spectrometry (GC-MS) and other instruments has been used in the research of microplastics. The principle is that plastics are a kind of organic polymer compounds with very simple structure in industrial production. Their structural monomers are connected repeatedly through covalent bonds. The pyrolysis of thermoplastic plastics mainly follows the free radical pyrolysis mechanism. The energy obtained by heating reaches the bond energy of each atomic bond, and the molecular chain breaks to generate various small molecular compounds. Microplastic samples are heated in a strictly controlled environment to crack into volatile small molecules, which are separated and detected by a combined gas chromatography-mass spectrometer, and the composition and structure of polymers are inferred according to their pyrolysis products. Fisher et al. [38]carried out the research on qualitative and quantitative methods of microplastics by Pyr-GC-MS. After analyzing the characteristic pyrolysis products of polyethylene, polyvinyl chloride and other plastics and their mass spectrometry ions, quantitative markers were selected, and the linear relationship between markers and sample quality was explored (R^2=0.86-0.99), which first verified the feasibility of Pyr-GC-MS for qualitative and quantitative (mass) analysis of microplastics. However, this technology is once limited by the detection size of microplastics and the problem of sample loss caused by complicated steps after pyrolysis, which is not enough for widespread promotion. To this end, Zhang Xiangnan et al. [37]invented a device that directly combines a pyrolyzer with a mass spectrometry instrument, avoiding the pollution and loss of gas generated after pyrolysis (after using this device and supporting methods, the average recovery rate of cracked gas is not less than 94.6%), and the detection instrument is portable and low in power consumption, and developed a set of supporting map library and control program, which makes the qualitative analysis more clear and the quantitative analysis closer to the real level of samples.

After reviewing the literature on microplastics (micro-plastics & nano-plastics) in environmental science, biology, and related clinical research, the author found that the research on the toxicological effects of microplastics on the cardiovascular system, especially the heart, has made considerable progress in the animal field (such as zebrafish, mice, chickens, etc.), but there is a lack of sufficient and reliable clinical evidence sources, and this part of evidence needs to be enriched. For the toxicological effects on blood vessels, preliminary progress has been made in domestic and foreign clinical research, but the sample size is still small, which is not enough to form effective and universal clinical evidence. For the qualitative and quantitative study of the toxic effects of microplastics on the cardiovascular system, the detectable biomarkers mainly include (TNF-α, iNOS, COX-2, IL-6, LDH, IDH, Gly, MDA, cTn, ANP, etc.), but these biomarkers are still indirect biomarkers, and there is still a lack of in-depth clinical research.

In terms of qualitative and quantitative detection methods of microplastics in human body, visual and optical microscopy is gradually eliminated due to subjective errors and insufficient identification accuracy, and is not used as the mainstream microplastic qualitative and quantitative research method. Raman spectroscopy has high accuracy for plastic qualitative research, no contact and no damage to samples, but limited by the principle of the method, chemical dyes or fluorescent agents in microplastic samples may interfere with the test results, which still needs to be improved. Fourier transform infrared spectroscopy has the highest accuracy for qualitative and quantitative detection of microplastics, and does not contact samples, but has high requirements for the quality of samples themselves. Pyrolysis-mass spectrometry is a comprehensive use of classical microplastic detection methods, which has gradually become one of the important tools for qualitative and quantitative research of microplastics in recent years, and has great potential.

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