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Microplastics as Emerging Environmental Contributors to Thyroid Dysfunction and Autoimmune Thyroiditis: A Review of Current Evidence, Molecular Mechanisms, and Future Perspectives

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ABSTRACT

Microplastics (MPs) have emerged as widespread environmental contaminants due to increasing plastic production, consumption, and improper disposal. Their persistence has resulted in contamination of water, soil, air, food, and consumer products, leading to human exposure mainly through ingestion and inhalation. MPs have been detected in human biological samples and tissues, including blood, placenta, lungs, cardiovascular tissues, and thyroid tissue, raising concerns about their potential effects on endocrine health. Experimental evidence indicates that MPs may induce cellular toxicity through interconnected mechanisms, including mitochondrial dysfunction, oxidative stress, chronic inflammation, NLRP3 inflammasome activation, apoptosis, ferroptosis, pyroptosis, and endocrine disruption. These alterations may impair thyroid follicular cell function, affect thyroid hormone synthesis, disturb hypothalamic–pituitary–thyroid axis regulation, and promote thyroid tissue injury. Emerging studies have identified MPs in thyroid tissue and suggested a possible association with autoimmune thyroiditis. However, human evidence remains limited, predominantly observational, and methodologically heterogeneous, with much of the available evidence derived from experimental models. A direct causal relationship between MP exposure and thyroid dysfunction has not yet been established. This narrative review evaluates environmental occurrence, exposure pathways, tissue distribution, molecular mechanisms, and endocrine effects of MPs, with emphasis on thyroid dysfunction and autoimmune thyroiditis. It integrates experimental and emerging human evidence while addressing limitations, knowledge gaps, clinical implications, and future research priorities. Multidisciplinary research is essential to clarify the long-term effects of MP exposure on thyroid health and guide strategies to reduce plastic pollution and protect endocrine function.

Key-words: Microplastics; Thyroid dysfunction; Autoimmune thyroiditis; Endocrine disruption; Oxidative stress; Inflammation; Environmental pollution; Thyroid hormones

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INTRODUCTION

Plastic production has increased exponentially over the past several decades owing to its widespread use in packaging, healthcare, textiles, agriculture, construction, and numerous consumer products. Although plastics have substantially improved modern lifestyles, their extensive production and inadequate waste management have resulted in persistent environmental pollution, making plastic contamination a major global environmental and public health concern. The durability

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and resistance of plastics to biodegradation have led to their continuous accumulation in terrestrial and aquatic ecosystems, creating long-term environmental challenges. [1,2]

Microplastics (MPs), defined as plastic particles measuring less than 5 mm in diameter, originate either from the fragmentation of larger plastic debris (secondary MPs) or from intentionally manufactured microscopic particles used in commercial and industrial products (primary MPs). Their small size, environmental persistence, and resistance to biodegradation have enabled MPs to become ubiquitous contaminants in freshwater bodies, marine ecosystems, agricultural soils, drinking water, atmospheric dust, food products, and indoor environments. More recently, increasing attention has focused on nanoplastics (<1 µm), which possess greater biological mobility and may exhibit higher cellular uptake and toxicity than larger MPs. [1–5]

Human exposure to MPs occurs predominantly through ingestion of contaminated food products and drinking water and inhalation of airborne particles, while dermal exposure represents a comparatively minor route. Continuous exposure has resulted in the detection of MPs in several human biological samples and tissues, including lungs, urine, blood, placenta, and cardiovascular tissues, indicating that these particles can cross biological barriers and accumulate within the human body. [5–14,16]

Initially regarded as biologically inert, MPs are now recognized as biologically active environmental contaminants capable of inducing mitochondrial dysfunction, inflammation, endoplasmic reticulum stress, oxidative stress, immune dysregulation, and multiple forms of regulated cell death. Experimental evidence further indicates that these molecular alterations contribute to tissue injury and functional impairment in multiple organ systems. However, much of the mechanistic evidence has been generated in laboratory animals or experimental systems, predominantly using polystyrene micro- or nanoplastic particles and exposure conditions that may not fully represent environmentally relevant human exposure, which involves heterogeneous polymers, weathered particles, and co-exposure to chemical contaminants. [15,17]

Among endocrine organs, the thyroid gland is particularly susceptible to environmental toxicants because of its high metabolic activity, continuous production of reactive oxygen species during thyroid hormone synthesis, and dependence on tightly regulated hypothalamic–pituitary–thyroid (HPT) axis signaling. Thyroid hormones play essential roles in growth, neurodevelopment, energy metabolism, cardiovascular function, thermogenesis, and immune homeostasis; therefore, even subtle disturbances in thyroid function may have widespread physiological consequences. Recent experimental studies have demonstrated that MPs can interfere with endocrine homeostasis by altering hormone synthesis, secretion, transport, receptor signaling, and metabolism. In addition to the direct effects of plastic particles, MPs may act as carriers for endocrine-disrupting chemicals (EDCs), including phthalates, bisphenols, polybrominated diphenyl ethers, and heavy metals, thereby amplifying their toxicological effects on endocrine organs. [4]

The thyroid gland is one of the most important endocrine organs responsible for maintaining metabolic homeostasis through the synthesis and secretion of triiodothyronine (T3) and thyroxine (T4). Thyroid hormone production is regulated by the HPT axis, and disruption of this regulatory network may result in hypothyroidism, hyperthyroidism, thyroid nodules, and autoimmune thyroid diseases.

Experimental evidence indicates that MP exposure can alter circulating thyroid hormone concentrations, impair thyroid follicular cell morphology, induce oxidative stress, activate inflammatory signaling pathways, and disrupt genes involved in thyroid hormone synthesis and metabolism. Many animal studies have also reported structural alterations of thyroid follicles following chronic MP exposure, supporting their potential endocrine-disrupting properties. However, these findings should be interpreted cautiously because exposure doses, particle characteristics, and experimental designs differ substantially among studies, limiting direct comparison and clinical extrapolation.

Therefore, this narrative review critically evaluates the current evidence regarding the environmental occurrence, human exposure pathways, tissue distribution, molecular mechanisms, and endocrine effects of MPs, with particular emphasis on thyroid dysfunction and autoimmune thyroiditis. Furthermore, this review examines the strength of the available evidence, identifies methodological limitations and knowledge gaps, and discusses priorities for future

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experimental, epidemiological, and clinical research. By integrating findings from environmental science, toxicology, molecular biology, endocrinology, and immunology, this review aims to provide a balanced and clinically relevant overview of the evolving relationship between MP exposure and thyroid health. [1]

Environmental Occurrence and Sources of Microplastics

MPs have become pervasive environmental contaminants because of the continuous production, widespread use, and improper disposal of plastic materials. Their persistence, resistance to biodegradation, and extensive distribution have resulted in their detection across terrestrial, freshwater, marine, and atmospheric environments, raising significant concerns regarding ecosystem integrity and human health. [1,2]

MPs are broadly classified into primary and secondary MPs according to their origin. Primary MPs are intentionally manufactured as microscopic particles for use in personal care products, cosmetics, industrial abrasives, pharmaceutical formulations, and resin pellets. In contrast, secondary MPs are generated through the environmental fragmentation of larger plastic items by ultraviolet radiation, mechanical abrasion, oxidation, and microbial degradation. Secondary MPs constitute the major proportion of environmental MPs because of the continuous breakdown of plastic waste. [2,3]

The most frequently detected polymer types include polyethylene (PE), polypropylene (PP), polyethylene terephthalate (PET), polystyrene (PS), polyvinyl chloride (PVC), and polyurethane (PU). These polymers originate from a wide range of consumer products, including plastic packaging, disposable utensils, food containers, synthetic textiles, fishing equipment, construction materials, household products, and automotive components. [2,4,5] Aquatic environments represent one of the largest reservoirs of MP contamination. Rivers transport substantial quantities of plastic debris into lakes, estuaries, and oceans, where MPs are ingested by plankton, invertebrates, fish, shellfish, and marine mammals. Consequently, aquatic organisms serve as important vectors for the transfer of MPs through the food chain, ultimately contributing to human dietary exposure. [1,4,5]

Agricultural soils have also emerged as important environmental sinks for MPs. The application of sewage sludge, plastic mulching films, compost, wastewater irrigation, and atmospheric deposition contributes to the continuous accumulation of plastic particles in cultivated land. Soil contamination may subsequently influence crop productivity, soil microbial communities, and the transfer of MPs into edible plants. [2,5]

Airborne MPs have been detected in both outdoor and indoor environments. Major atmospheric sources include synthetic textile fibers, tire wear particles, urban dust, industrial emissions, and the fragmentation of plastic waste. Indoor environments frequently contain higher concentrations of airborne MPs because of textile furnishings, carpets, household dust, and limited ventilation, making inhalation an increasingly recognized route of human exposure. [5,6]

In addition to environmental contamination, MPs have been identified in drinking water, bottled water, table salt, seafood, fruits, vegetables, milk, honey, and other commonly consumed foods. These findings indicate that continuous, low-level human exposure to MPs has become virtually unavoidable, emphasizing the need to better understand their potential health consequences. [5-8]

Besides the intrinsic toxicity of plastic particles, MPs may adsorb and transport environmental pollutants, including heavy metals, persistent organic pollutants, antibiotics, and EDCs. This carrier effect may enhance the bioavailability of co-existing contaminants and increase their toxicological impact following human exposure, highlighting the complex nature of MP-associated health risks.

Overall, the ubiquitous distribution of MPs across environmental compartments and food systems demonstrates that human exposure is continuous and multifactorial. The growing environmental burden of MPs underscores the importance of understanding their biological fate and potential contribution to chronic diseases, including endocrine disorders such as thyroid dysfunction. [1,2]

Human Exposure Pathways of Microplastics

Human exposure to MPs is widespread and occurs through multiple environmental pathways, including ingestion, inhalation, and dermal contact. Among these, ingestion and inhalation are considered the predominant

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routes of exposure because of the ubiquitous presence of MPs in food, drinking water, and ambient air. Continuous exposure through these pathways has raised increasing concerns regarding the accumulation of MPs in human tissues and their potential adverse health effects. [5–8]

Ingestion- Dietary intake represents a major route of MP exposure in humans. MPs have been detected in seafood, fish, shellfish, table salt, bottled and tap water, milk, honey, fruits, vegetables, tea bags, and numerous processed foods. Estimates of human MP ingestion further emphasize the importance of dietary exposure as a component of cumulative human exposure. [5–8]
Following ingestion, MPs interact with the gastrointestinal tract, where particle size, shape, surface charge, and polymer composition influence their biological behavior. Although larger particles are eliminated through fecal excretion, smaller MPs and nanoplastics may cross the intestinal epithelium via transcellular transport, paracellular diffusion, or uptake by microfold (M) cells, thereby entering systemic circulation. [9–12]

Inhalation- Airborne MPs constitute another important route of human exposure. These particles originate from synthetic textile fibers, tire wear, household dust, industrial emissions, construction activities, and degradation of plastic materials. Indoor environments frequently contain higher concentrations of airborne MPs because of limited ventilation and the extensive use of synthetic furnishings and textiles. [5,6] Once inhaled, MPs may deposit within different regions of the respiratory tract depending on their aerodynamic properties and particle size. Larger particles are generally trapped within the upper airways and removed by mucociliary clearance, whereas smaller particles may penetrate into the bronchioles and alveoli, where they can induce oxidative stress, inflammatory responses, and epithelial injury. [9,13]

Dermal Exposure- Dermal exposure to MPs occurs through direct contact with contaminated water, soil, cosmetics, personal care products, and synthetic textiles. Although intact human skin provides an effective barrier against most MPs, prolonged exposure, damaged skin, or extremely small particles may facilitate limited penetration into superficial skin layers. Compared with ingestion and inhalation, dermal absorption is currently considered a relatively minor route of systemic exposure. [5,14]

Systemic Distribution Following Exposure- Following absorption, MPs may enter the bloodstream and distribute to multiple organs. Recent investigations have identified MPs in human blood, placenta, lungs, liver, cardiovascular tissues, breast milk, reproductive organs, and thyroid tissue, demonstrating their ability to cross biological barriers and accumulate within different tissues. Particle size, surface chemistry, polymer type, and exposure duration are important determinants of tissue distribution and biological persistence. [10–16]
The biological fate of MPs following systemic exposure remains an active area of investigation. Experimental studies suggest that persistent accumulation of MPs may trigger oxidative stress, mitochondrial dysfunction, chronic inflammation, immune dysregulation, and endocrine disruption, thereby contributing to the development of chronic diseases. However, the long-term kinetics, clearance mechanisms, and cumulative health effects of MPs in humans require further investigation through well-designed epidemiological and mechanistic studies. [17–24]

Distribution and Accumulation of Microplastics in Human Tissues

Following environmental exposure, MPs may translocate across biological barriers and distribute to multiple organs and tissues. Their biodistribution is influenced by particle size, shape, polymer composition, surface charge, and exposure duration. Smaller MPs and nanoplastics exhibit greater biological mobility and are more likely to penetrate epithelial barriers, enter the systemic circulation, and accumulate in distant organs. [3,6,8]

Recent advances in analytical techniques, including Fourier-transform infrared (FTIR) spectroscopy, Raman spectroscopy, and pyrolysis–gas chromatography–mass spectrometry (Py-GC/MS), have enabled the identification and characterization of MPs in human biological samples. These technologies have substantially improved our understanding of the internal distribution of MPs and their potential implications for human health. [6,9,13]

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One of the most significant discoveries has been the detection of MPs in human blood, providing direct evidence that plastic particles can enter systemic circulation after environmental exposure. Circulating MPs may subsequently be transported to highly vascularized organs, thereby increasing the likelihood of tissue accumulation and prolonged biological interaction. [6,13]

MPs have also been detected in the human placenta, demonstrating their ability to cross the maternal–fetal interface. Placental accumulation raises concerns regarding fetal exposure during critical stages of development and suggests that prenatal exposure may influence endocrine and developmental processes. A recent study further reported an association between placental MP contamination and altered thyroid function in newborns, highlighting the potential endocrine consequences of early-life exposure. [7,10]

The respiratory system represents another important site of MP accumulation. Detection of MPs in human lung tissue confirms that inhaled particles can penetrate deep into the respiratory tract and persist within pulmonary tissues. Chronic pulmonary retention may contribute to oxidative stress, inflammatory responses, and tissue injury, although the long-term clinical significance remains to be fully elucidated. [14]

Beyond the respiratory and circulatory systems, MPs have been identified in urine and cardiovascular tissues, indicating widespread systemic distribution following environmental exposure. The presence of MPs within atherosclerotic plaques has raised concerns regarding their possible contribution to vascular inflammation and cardiovascular disease, although additional mechanistic and epidemiological studies are required to establish causality. [11,12]

Of particular relevance to endocrine research, emerging evidence has demonstrated the presence of MPs in human thyroid tissue. Preliminary investigations have suggested a greater MP burden in thyroid tissues obtained from patients with autoimmune thyroiditis compared with non-inflammatory thyroid tissue. Although these findings are based on limited clinical studies, they provide important biological evidence supporting the hypothesis that MPs may accumulate within the thyroid gland and participate in disease-related molecular pathways. [13,18]

Experimental studies further indicate that accumulated MPs may induce oxidative stress, mitochondrial dysfunction, chronic inflammation, immune dysregulation, and endocrine disruption within exposed tissues. The persistence of these biological responses may contribute to progressive cellular injury and organ dysfunction, particularly following long-term or repeated exposure. [17–25]

Despite increasing evidence of MP accumulation in human tissues, important questions remain regarding their toxicokinetics, tissue clearance, long-term persistence, and dose–response relationships. Future studies employing standardized analytical methods and prospective human cohorts are essential to clarify the clinical significance of tissue MP accumulation and its contribution to endocrine disorders, including thyroid dysfunction and autoimmune thyroiditis. [13–16]

Molecular Mechanisms Linking Microplastics to Thyroid Dysfunction

Growing evidence indicates that MPs exert their biological effects through multiple interconnected molecular pathways rather than a single toxicological mechanism. Following cellular uptake, MPs can disrupt intracellular homeostasis by inducing oxidative stress, mitochondrial dysfunction, chronic inflammation, immune dysregulation, activation of the NLRP3 inflammasome, and various forms of regulated cell death. These molecular alterations may impair thyroid follicular cell function, disturb thyroid hormone synthesis, and promote endocrine dysfunction. [17–24]

In addition to the direct toxicity of plastic particles, MPs may adsorb EDCs, heavy metals, and persistent organic pollutants from the surrounding environment. Consequently, MPs may function not only as toxic particles but also as carriers that enhance the bioavailability and biological effects of coexisting environmental contaminants. [24,25]

The molecular mechanisms described below are highly interconnected. Oxidative stress frequently initiates mitochondrial dysfunction and inflammatory signaling, which subsequently activate the NLRP3 inflammasome and multiple regulated cell death pathways. Together, these processes contribute to progressive thyroid injury and may increase susceptibility to thyroid dysfunction and autoimmune thyroiditis. [17–24]

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Oxidative Stress- Oxidative stress is considered one of the earliest and most extensively investigated mechanisms of MP-induced toxicity. Exposure to MPs increases intracellular production of reactive oxygen species (ROS) while simultaneously impairing endogenous antioxidant defense systems, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). The resulting imbalance between oxidant generation and antioxidant capacity promotes oxidative damage to lipids, proteins, and nucleic acids, ultimately compromising cellular integrity. [17–21]

The thyroid gland is particularly susceptible to oxidative injury because physiological thyroid hormone synthesis requires continuous generation of hydrogen peroxide as an essential substrate for thyroid peroxidase-mediated iodination of thyroglobulin. Excessive ROS production following MP exposure may therefore overwhelm intrinsic antioxidant defenses, leading to thyroid follicular cell injury and impaired hormone biosynthesis. [19–21]

Experimental studies have consistently demonstrated that MP-induced oxidative stress is accompanied by increased lipid peroxidation, mitochondrial injury, DNA damage, inflammatory cytokine production, and activation of apoptosis-related signaling pathways. These pathological alterations provide a mechanistic link between MP exposure and cellular injury, although their direct contribution to human thyroid disease remains to be established. [15,18–24]

Mitochondrial Dysfunction- Mitochondria are essential organelles responsible for ATP production, cellular metabolism, calcium homeostasis, and regulation of programmed cell death. Increasing evidence indicates that MPs impair mitochondrial integrity and function, resulting in decreased ATP production, disruption of mitochondrial membrane potential, excessive generation of ROS, and release of pro-apoptotic factors. [18,20,24,25]
Oxidative stress induced by MP exposure further aggravates mitochondrial injury by damaging mitochondrial DNA, proteins, and membrane lipids. This vicious cycle of ROS generation and mitochondrial dysfunction amplifies cellular injury and contributes to persistent metabolic disturbances. [18,24]
In thyroid follicular cells, adequate mitochondrial function is essential for energy-dependent processes involved in iodide uptake, thyroglobulin synthesis, and thyroid hormone production. Therefore, MP-induced mitochondrial dysfunction may compromise normal thyroid physiology and contribute to impaired hormone biosynthesis and endocrine imbalance. [20,21]

Inflammation- Chronic inflammation represents another key mechanism underlying MP-induced toxicity. Experimental studies have demonstrated that MPs activate inflammatory signaling pathways, leading to increased production of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, IL-6, and other inflammatory mediators. [17,18,23]
The activation of nuclear factor-kappa B (NF-κB) plays a central role in mediating inflammatory responses following MP exposure. Increased NF-κB signaling promotes transcription of numerous inflammatory genes, enhances cytokine production, and contributes to sustained tissue inflammation. [23] Persistent inflammatory responses may disrupt thyroid follicular architecture, impair hormone synthesis, and promote immune cell infiltration within thyroid tissue. Moreover, chronic inflammation creates a favorable microenvironment for autoimmune activation, thereby potentially increasing susceptibility to autoimmune thyroiditis. [18,23]

NLRP3 Inflammasome Activation- The NLRP3 inflammasome has emerged as an important molecular mediator of MP-induced inflammation. Activation of the NLRP3 inflammasome results in cleavage of procaspase-1 into its active form, subsequently promoting maturation and secretion of the pro-inflammatory cytokines IL-1β and IL-18. [22,23] Experimental evidence indicates that excessive ROS production, mitochondrial dysfunction, lysosomal damage, and ionic imbalance induced by MPs act as major upstream signals for NLRP3 inflammasome activation. Persistent activation of this pathway amplifies inflammatory injury and contributes to progressive tissue damage. [22,23]

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Apoptosis- Apoptosis is a highly regulated form of programmed cell death that plays a critical role in maintaining tissue homeostasis. Increasing evidence indicates that MP exposure induces apoptosis through both intrinsic (mitochondrial) and extrinsic signaling pathways. Excessive ROS generation, mitochondrial dysfunction, DNA damage, and activation of caspases collectively contribute to apoptosis in exposed cells. [18,20,24]

Experimental studies have demonstrated that MP exposure alters the expression of apoptosis-related proteins by increasing the Bax/Bcl-2 ratio and activating caspase-3 and caspase-9. These molecular alterations result in DNA fragmentation, chromatin condensation, and progressive loss of viable cells. [20,24]

In the thyroid gland, excessive apoptosis of follicular epithelial cells may impair thyroid hormone synthesis, disrupt follicular architecture, and contribute to progressive thyroid dysfunction. Persistent apoptotic cell death may also facilitate the release of intracellular antigens, potentially enhancing autoimmune responses in genetically susceptible individuals. [18]

Pyroptosis- Pyroptosis is an inflammatory form of programmed cell death characterized by activation of inflammasomes, cleavage of gasdermin D (GSDMD), formation of membrane pores, and release of pro-inflammatory cytokines. Unlike apoptosis, pyroptosis amplifies inflammatory responses through the extracellular release of intracellular contents. [22,23]

Recent studies suggest that MP-induced oxidative stress and NLRP3 inflammasome activation promote pyroptosis by activating caspase-1 and stimulating the maturation of IL-1β and IL-18. The resulting inflammatory cascade contributes to tissue injury and sustained immune activation. [22,23]

Although direct evidence in thyroid tissue remains limited, activation of pyroptosis-related signaling pathways has been implicated in inflammatory and autoimmune disorders. Therefore, MP-induced pyroptosis may represent an additional mechanism contributing to thyroid inflammation and endocrine dysfunction. [28,29]

Ferroptosis- Ferroptosis is an iron-dependent form of regulated cell death characterized by excessive lipid peroxidation and depletion of intracellular antioxidant defenses. Unlike apoptosis and pyroptosis, ferroptosis is primarily driven by oxidative damage to membrane phospholipids resulting from impaired glutathione metabolism and reduced glutathione peroxidase 4 (GPX4) activity. [21,24] Emerging evidence indicates that MP exposure enhances oxidative stress and lipid peroxidation, thereby creating conditions favorable for ferroptotic cell death. Alterations in iron metabolism and antioxidant capacity further exacerbate membrane damage and cellular dysfunction. [21,24]

Although the contribution of ferroptosis to MP-induced thyroid injury has not yet been fully established, oxidative stress is a well-recognized mediator of thyroid pathology. Consequently, ferroptosis represents a plausible molecular mechanism that warrants further investigation in future experimental and clinical studies. [19,25]

Endocrine Disruption and Implications for Thyroid Dysfunction- MPs are increasingly recognized as emerging endocrine disruptors because they can interfere with hormonal homeostasis through multiple mechanisms. In addition to the intrinsic biological effects of plastic particles, MPs may adsorb and transport EDCs, including bisphenols, phthalates, polybrominated flame retardants, and heavy metals, thereby enhancing their endocrine toxicity. [23,24]

Experimental studies have demonstrated that MP exposure may alter thyroid hormone synthesis, disturb HPT axis regulation, impair thyroid follicular cell function, and modify the expression of genes involved in thyroid hormone biosynthesis and metabolism. Histopathological alterations observed in experimental models further support the endocrine-disrupting potential of MPs. [16]

Collectively, oxidative stress, mitochondrial dysfunction, chronic inflammation, NLRP3 inflammasome activation, apoptosis, pyroptosis, ferroptosis, and endocrine disruption represent interconnected mechanisms through which MPs may contribute to thyroid dysfunction. Although current evidence is predominantly derived from experimental studies, recent human investigations have strengthened the biological plausibility that chronic MP exposure may adversely affect thyroid health. Further epidemiological and mechanistic studies are required to establish causal

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relationships and identify clinically relevant exposure thresholds. [13–21]

Microplastics and Thyroid Dysfunction

Experimental Evidence- Experimental studies have consistently demonstrated that exposure to MPs disrupts thyroid function through multiple molecular and cellular mechanisms. Animal models have shown that chronic MP exposure alters circulating thyroid hormone concentrations, although the magnitude and direction of these changes vary according to polymer type, particle size, exposure dose, duration, and experimental model. [14–17]

Histopathological examination of thyroid tissue has revealed structural abnormalities, including degeneration of thyroid follicular epithelial cells, disorganization of follicular architecture, colloid depletion, inflammatory cell infiltration, and increased apoptotic activity following prolonged MP exposure. These morphological alterations are frequently accompanied by impaired thyroid hormone synthesis and disruption of endocrine homeostasis. [16,17]

Experimental investigations have further demonstrated that MP exposure induces oxidative stress, mitochondrial dysfunction, activation of inflammatory signaling pathways, and dysregulation of genes involved in thyroid hormone biosynthesis and metabolism. Increased production of ROS, impaired antioxidant defenses, and activation of the NF-κB and NLRP3 inflammasome pathways collectively contribute to thyroid follicular cell injury. [17–24]

Several studies have also reported alterations in the HPT axis following MP exposure. Disturbance of this regulatory axis may impair thyroid-stimulating hormone (TSH) secretion and subsequently affect thyroid hormone production, thereby contributing to endocrine imbalance. [24,26]

Overall, experimental evidence supports the endocrine-disrupting potential of MPs and provides mechanistic insights into their adverse effects on thyroid physiology. Nevertheless, differences in experimental design, exposure conditions, and particle characteristics currently limit direct comparisons among studies, and much of the mechanistic evidence remains experimental. [24,25]

Evidence from Human Studies- Compared with experimental investigations, human studies evaluating the association between MP exposure and thyroid dysfunction remain limited. Nevertheless, recent advances in analytical techniques have enabled the detection of MPs in human thyroid tissue, providing direct evidence that plastic particles can accumulate within this endocrine organ. [13,25]

Emerging clinical studies have suggested that thyroid tissues obtained from patients with autoimmune thyroiditis contain a greater burden of MPs than tissues without inflammatory disease. These findings have generated increasing interest in the potential contribution of MPs to thyroid inflammation and autoimmune processes. [16–18]

Although current human evidence supports the biological plausibility of an association between MP exposure and thyroid disease, available studies are predominantly observational and involve relatively small sample sizes. Consequently, existing data are insufficient to establish a causal relationship between environmental MP exposure and thyroid dysfunction. Future prospective epidemiological studies incorporating standardized methods for MP detection, quantitative exposure assessment, and comprehensive endocrine evaluation are required to clarify the long-term clinical significance of MP accumulation in the thyroid gland. [13–20]

Critical Evaluation of Current Evidence- Collectively, available evidence indicates that MPs have the potential to impair thyroid physiology through multiple interconnected mechanisms, including oxidative stress, mitochondrial dysfunction, chronic inflammation, inflammasome activation, endocrine disruption, and regulated cell death. Experimental studies consistently demonstrate biological effects on thyroid structure and function, whereas human investigations provide preliminary evidence of thyroid accumulation and possible associations with thyroid disease. [17–24]

Despite these advances, several important limitations remain. Most mechanistic evidence originates from experimental animal models employing exposure concentrations that may exceed typical environmental levels. Furthermore, heterogeneity in polymer composition, particle size, analytical methodologies, and exposure duration complicates comparisons across

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studies. Human studies remain scarce and are largely cross-sectional, preventing conclusions regarding temporal relationships or causality. [14–20]

Therefore, current evidence should be interpreted cautiously. While the biological plausibility linking MPs to thyroid dysfunction is strong, robust longitudinal studies integrating environmental exposure assessment, molecular biomarkers, and clinical endocrine outcomes are essential to determine whether chronic MP exposure represents a clinically significant risk factor for thyroid disease. [13–21]

Microplastics and Autoimmune Thyroiditis

Autoimmune thyroiditis (AIT), particularly Hashimoto's thyroiditis, is the most common autoimmune endocrine disorder and a leading cause of hypothyroidism worldwide. The disease is characterized by progressive immune-mediated destruction of thyroid follicular cells, lymphocytic infiltration of the thyroid gland, and the production of thyroid autoantibodies, including anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Although genetic susceptibility plays a fundamental role in disease development, environmental factors are essential triggers that initiate and perpetuate autoimmune responses. [18–22]

Recent evidence suggests that MPs may represent a novel environmental factor capable of promoting thyroid autoimmunity. Persistent exposure to MPs induces oxidative stress, mitochondrial dysfunction, chronic inflammation, and activation of innate immune pathways, all of which are recognized contributors to autoimmune disease pathogenesis. These biological effects may create a pro-inflammatory microenvironment that favors immune dysregulation and loss of self-tolerance. [17–24]

Oxidative stress is considered a major mechanism linking MP exposure with autoimmune thyroid disease. Excessive production of ROS damages cellular proteins, lipids, and DNA, resulting in thyroid follicular cell injury. Damaged thyroid cells may release intracellular antigens that stimulate antigen-presenting cells and activate autoreactive T and B lymphocytes, thereby promoting autoimmune responses against thyroid tissue. [18–21]

Chronic inflammation induced by MP exposure further contributes to thyroid autoimmunity through sustained activation of inflammatory signaling pathways. Increased production of cytokines such as TNF-α, interleukin (IL)-1β, and IL-6, together with activation of the NF-κB and NLRP3 inflammasome pathways, amplifies immune responses and enhances inflammatory injury within thyroid tissue. [22,23]

Emerging human studies have reported the presence of MPs within thyroid tissue and have suggested a higher burden of plastic particles in patients with autoimmune thyroiditis compared with individuals without inflammatory thyroid disease. Although these findings are based on limited clinical investigations and do not establish causality, they provide important evidence supporting the biological plausibility of a relationship between MP accumulation and thyroid autoimmunity. [23]

In addition to the direct biological effects of plastic particles, MPs may adsorb EDCs, heavy metals, and persistent organic pollutants from the surrounding environment. These co-contaminants may further impair immune regulation and endocrine function, potentially enhancing the risk of autoimmune thyroid disease through combined toxicological mechanisms. [23,24]

Despite growing experimental evidence, significant knowledge gaps remain. Most available studies have been conducted in experimental models, whereas human investigations are limited by small sample sizes, cross-sectional designs, and variability in analytical methods used for MP detection. Consequently, current evidence is insufficient to establish a definitive causal relationship between MP exposure and autoimmune thyroiditis. [13–20]

Future research should focus on large-scale prospective cohort studies, standardized analytical techniques for MP identification, quantitative exposure assessment, and integration of molecular biomarkers with clinical outcomes. Such investigations will be essential to determine whether chronic MP exposure represents an independent environmental risk factor for autoimmune thyroiditis and other immune-mediated thyroid disorders. [13–21]

Clinical Implications, Public Health Significance, and Future Perspectives

The widespread environmental distribution of MPs and their detection in multiple human tissues have raised significant concerns regarding their potential impact on endocrine health. Although current evidence does not establish a causal relationship between MP exposure and

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thyroid disease, experimental findings and emerging human studies indicate that MPs may contribute to thyroid dysfunction through multiple interconnected biological mechanisms.

From a clinical perspective, thyroid disorders are among the most prevalent endocrine diseases worldwide and are associated with substantial morbidity. Even minor alterations in thyroid hormone homeostasis may adversely affect growth, neurodevelopment, metabolism, cardiovascular function, reproductive health, and immune regulation. Therefore, identifying environmental factors that contribute to thyroid dysfunction is of considerable clinical importance. Recent detection of MPs in human thyroid tissue has strengthened concerns regarding their possible role in endocrine disorders. Although these findings remain preliminary, they support the biological plausibility that chronic MP exposure may influence thyroid physiology and potentially contribute to autoimmune thyroiditis in susceptible individuals. At present, routine clinical assessment of MP exposure is not recommended because standardized methods for exposure quantification and clinical interpretation are still lacking. Clinicians should nevertheless remain aware of emerging evidence regarding environmental contaminants that may influence endocrine function, while recognizing that evidence specific to thyroid disease remains preliminary. From a public health perspective, reducing plastic pollution represents an important preventive strategy.

Improved plastic waste management, increased recycling, reduction in single-use plastics, development of environmentally sustainable materials, and stricter environmental regulations may reduce environmental MP contamination and ultimately decrease long-term human exposure. Public education regarding responsible plastic use and environmental conservation should also be encouraged. [1–5]

Future research should prioritize large-scale prospective epidemiological studies evaluating long-term MP exposure and thyroid outcomes in diverse populations. Standardized analytical techniques for MP detection and quantification are essential to improve comparability among studies and establish reliable exposure biomarkers. Further mechanistic investigations should clarify how particle size, polymer composition, surface chemistry, exposure duration, and co-exposure to endocrine-disrupting chemicals influence thyroid toxicity. Integration of molecular biology, toxicology, environmental science, endocrinology, immunology, and clinical medicine will be necessary to better understand the complex interactions between MPs and thyroid health. [17–24]

Overall, current evidence highlights MPs as emerging environmental contaminants with the potential to influence endocrine function. Although definitive clinical evidence is still lacking, the growing body of experimental and human research justifies continued investigation into their possible contribution to thyroid dysfunction and autoimmune thyroid disease. [13–21]

Table 1: Knowledge gaps and future research priorities in microplastic-associated thyroid dysfunction

Research Area	Current Knowledge Gap	Future Research Priority
Human exposure assessment	No standardized methods for measuring long-term individual MP exposure	Develop validated and harmonized exposure assessment protocols for environmental and biological samples
Detection of MPs in biological tissues	Different analytical methods (FTIR, Raman spectroscopy, Py-GC/MS) produce variable results	Standardize laboratory methods and reporting guidelines for tissue MP analysis
Dose–response relationship	Environmentally relevant exposure thresholds remain unknown	Establish dose–response relationships using chronic low-dose exposure models
Thyroid-specific mechanisms	Limited studies using human thyroid cells	Investigate molecular pathways in primary human thyroid cells and thyroid organoid models
Thyroid hormone homeostasis	Mechanisms regulating T3, T4, and TSH alterations are incompletely understood	Clarify effects on thyroid hormone synthesis, secretion, transport, metabolism, and HPT-axis regulation

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Autoimmune thyroiditis	Evidence is limited to a few observational studies	Conduct prospective studies evaluating thyroid autoantibodies (TPOAb and TgAb), imaging findings, and disease progression
Oxidative stress and inflammation	Human biomarker data are scarce	Validate oxidative stress, inflammatory, and mitochondrial biomarkers associated with MP exposure
Combined exposure to environmental contaminants	Interactions between MPs and endocrine-disrupting chemicals remain poorly understood	Investigate combined effects of MPs with bisphenols, phthalates, heavy metals, and persistent organic pollutants
Clinical evidence	Lack of large longitudinal epidemiological studies	Perform multicenter cohort studies with standardized exposure assessment and long-term follow-up
Prevention and risk reduction	Limited evidence on effective public health interventions	Evaluate strategies to reduce environmental MP exposure and assess their impact on endocrine health

FTIR, Fourier-transform infrared spectroscopy; HPT, hypothalamic–pituitary–thyroid; MP, microplastic; Py-GC/MS, pyrolysis–gas chromatography–mass spectrometry; Raman, Raman spectroscopy; T3, triiodothyronine; T4, thyroxine; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TSH, thyroid-stimulating hormone

CONCLUSIONS

Microplastics (MPs) have emerged as ubiquitous environmental contaminants with increasing relevance to human health. Their widespread distribution in air, water, soil, food, and consumer products has resulted in continuous human exposure through ingestion, inhalation, and, to a lesser extent, dermal contact. Recent detection of MPs in human blood, placenta, lungs, cardiovascular tissues, and thyroid tissue demonstrates their ability to cross biological barriers and accumulate in multiple organs, raising concerns regarding their long-term biological effects. Current experimental evidence indicates that MPs exert their toxic effects through multiple interconnected molecular mechanisms, including oxidative stress, mitochondrial dysfunction, chronic inflammation, activation of the NLRP3 inflammasome, apoptosis, pyroptosis, ferroptosis, and endocrine disruption. These mechanisms may impair thyroid follicular cell function, disturb HPT axis regulation, alter thyroid hormone synthesis, and promote tissue injury. Emerging human studies have provided preliminary evidence of MP accumulation within thyroid tissue and have suggested a possible association with autoimmune thyroiditis.

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