

The Thyroid's Plastic Predicament: From Blood-Barrier Breach to Transcriptional Dysregulation

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Abstract

Background: Microplastics (MPs, 1–5 mm) and nano plastics (NPs, <1µm) are prevalent environmental contaminants with emerging evidence of endocrine disruption, particularly targeting the thyroid gland a master regulator of metabolism, development and homeostasis. **Objective:** This review synthesizes 2023–2025 evidence on MNPs thyroid toxicity, elucidating mechanisms from blood-thyroid barrier (BTB) penetration to transcriptional dysregulation, mammalian outcomes, associated chemical synergies, knowledge gaps and human health implications. **Methods:** Comprehensive analysis of peer-reviewed studies (n=45+) using PubMed/PMC/Scopus, focusing on in vivo/in vitro mammalian models, human biomonitoring and regulatory frameworks. **Results:** NPs breach fenestrated BTB capillaries via endocytosis, inducing ROS/NF-κB-mediated inflammation, TPO/NIS/Tg suppression, TBG/TTR displacement by additives (BPA/BPS/BPF, phthalates, PBDEs), THR antagonism and PAX8/CREB dysregulation. Rodent models show dose-dependent T3/T4 ↓ (40–60%), TSH ↑, follicular hypertrophy/fibrosis. Human data link placental MPs to neonatal T4 ↓ and occupational exposure to subclinical hypothyroidism. NPs exhibit 4–10× higher potency than MPs due to bioavailability/surface area. **Conclusions:** MNPs pose possible thyroid risk via multiple mechanisms justified by toxicokinetic, Pyrolysis GC-MS harmonization, longitudinal cohorts and precautionary policies. Pregnant women and children represent vulnerable populations.

Introduction

In the past few decades, the frequent use and dumping of plastic products have led to a surplus of plastic trash in the environment. Microplastics (MPs, 1 µm–5 mm) and nano plastics (NPs, < 1µm) are new pollutants that come from both commercial production and the breakdown of plastics. These small plastic particles may remain in the environment for extended periods of time and could be harmful to both human health and the ecosystem. MPs are categorized as primary or secondary according to the way they are

produced and their fragmented composition. Whereas secondary MPs are separated from bigger plastics by a variety of physical and biological processes, including UV radiation, temperature changes and wave action while primary MPs are mostly made into small sizes for commercial applications like personal care products. Plastic pollution is a global crisis in which thousands of species including marine life and human health, are affected and also destroying the ecosystem. Over 1 million marine life die annually due to plastic toxicity in the sea and oceans (Deng et al., 2025).

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Nowadays, humans mostly use processed and packaged foods which are the major source of MPs & NPs. Humans ingest those MPs & NPs through food which then causes toxicity in the body. To some extent water is also the source of plastic ingestion in the body. Later on, these MPs & NPs cause toxicity in the body which then causes serious illness. Plastic Pollution mostly affects our endocrine organs, which then disrupts our endocrine hormones. Endocrine hormones regulate various bodily functions like metabolism, growth, reproduction, mood and stress response. Human physiological processes such as growth retardation, decreased fertility, and immune system impairment may be disrupted by the consumption of microplastics, as shown in (Figure 1). The damage that MPs and NPs are causing on a global scale is extremely hazardous to the planet's health. Even though the precise proportion of plastic that is currently present is still up for debate, MPs and NPs have been found in both aquatic and terrestrial habitats. The top three countries in the world for plastic production are North America (19%), Europe (20%), and China (26%). Between 0.013 and 25 million metric tons of MPs/NPs could be carried through the marine atmosphere and released into the oceans each year (Wan et al., 2019).

The thyroid gland is a butterfly-shaped gland located just below the larynx (the voice box). It has two lobes on either side of the windpipe (Trachea) connected by the central isthmus. The thyroid secretes three hormones: T4, T3 and Calcitonin. T3 & T4 are iodine-containing derivatives of amino acid tyrosine. Breakdown of thyroid hormones occurs in the muscles, liver, and kidneys.

The latest research shows that these particles work in two different ways 1) Physical harming of thyroid follicles, 2) Chemical supplying endocrine disrupting compounds, e.g., bisphenols, phthalates, & flame retardants (Campanale et al., 2020). Their nanoscale capacity allows blood thyroid barrier to be perforated, where they disrupt receptor signaling ($TR\alpha/\beta$ antagonism), transportation (transthyretin relocation) and hormone production (e.g., by suppressing thioperoxides). The current research report states that exposure to polystyrene NP lowers serum T4 levels by 50% (Amerreh et al., 2019). This issue's urgency is intensified by three factors; Rapidly increasing Plastic Production (According to the Global Plastic Production Statista Research Department, the worldwide plastic production almost reached to the 413.8 million metric tons in 2023), Thyroid Hormone Sensitivity (It's lipid rich hormones and follicular structure make is unusually prone to lipophilic plasticizers) and Area of Diagnostic Weakness (Subcellular dysfunction carried on by NPs may go undetected by standard thyroid screenings).

New research has been initiated to investigate the possible relationship between exposure to MPs, NPs and thyroid dysfunction. This paper intends to investigate their

endocrine-disrupting potential and assess their specific impact on thyroid structure and functions based on current scientific information. This review article conveys a significant knowledge gap on how MPs & NPs directly impact thyroid functions at the cellular, molecular and systemic levels by dodging biological barriers.



Figure 1. Source of microplastics and nanoplastics

The Invasion: Breaching of MPs/NPs to Reach the Thyroid

Microplastics and nanoplastics are broad environmental toxic substances found in terrestrial, marine, freshwater and environmental ecosystems. Over 80% of MPs emerge from inland sources like urban runoff, agricultural practices, sewage waste and improper handling of plastic waste, whereas less than 20% originate directly from marine sources such as aquaculture and fishing (Yee et al., 2021). The thyroid gland in spite of its shielding vascular barrier is progressively susceptible to MPs & NPs penetration over multiple routes of exposure. Current research discloses that there are multiple pathways for thyroid accumulation respectively showing a size-dependent dissemination model that opposes the gland's defensive mechanism.

Route of uptake

Microplastics and nano plastics enter the living organism mostly through; Ingestion (dietary sources), and Inhalation (respiratory sources) showed in (Figure 2) are the major two ways from which MPs & NPs ingested in the body (Cary et al., 2023; Zhang et al., 2024). The primary way that humans are exposed to microplastics and nanoplastics is through ingestion. According to the studies MPs and NPs have been found in drinking water, shellfish, fruits, vegetables, table salt and honey (Barboza et al., 2018; Cox et al., 2019). These particles are eaten by marine creatures including fish, shellfish and plankton and they subsequently bioaccumulate down the food chain. When humans consume infected seafood or agricultural items that have been irrigated with tainted water, they are exposed (Galloway et al., 2017). Another significant factor is plastic packaging materials. Small plastic particles and additives can be released into food and beverages when food is heated in plastic containers

or when disposable bottles are used. Once ingested, microplastics and nanoplastics can cross the gastrointestinal barrier to the circulatory system. The gastrointestinal tract's particular integration as well as pH and cell types that affect the regulation of this translocation (Cary et al., 2023). Because of their size and surface quality small particles which are nanoparticles, have a greater probability than large particles of microplastics of bypassing biological barriers and entering the bloodstream where they may cause oxidative stress and inflammation (Barua et al., 2014; Stock et al., 2019).

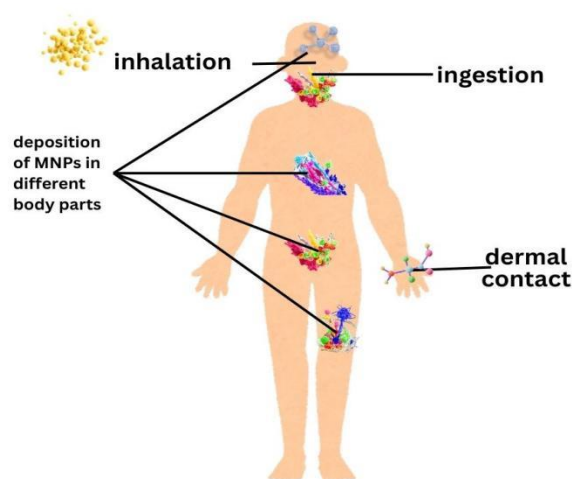


Figure 2. Source of uptake of microplastics and nanoplastics into human body.

Lymphatic/ blood dissemination

The inhalation of airborne microplastics has become a significant exposure route. Synthetic fibers and particles released from textiles, carpets, furniture, and plastic-based items are present in indoor and urban air (Dris et al., 2017). The quantity of MPs in the air indoors can be increased by routine tasks including using dryers, vacuuming, and wearing synthetic clothing. Depending on their size and form, inhaled particles may settle in the upper or lower respiratory tract (Vianello et al., 2019; Tan et al., 2024). According to certain study findings smaller nano plastics may be able to pass through the lungs' alveolar barrier and reach the bloodstream (Prata 2018). This may allow them to reach distant organs and potentially interact with endocrine glands such as the thyroid, although there is currently not sufficient evidence available in humans. The thyroid is one of the distant organs in which inhaled nano plastics accumulate which pass out through the respiratory tract and deviate from the respiratory epithelium via the process known as transcytosis. They can then enter the blood and lymphatic system and travel across. The proficiency of this

procedure is size dependent like nano plastics <100nm preferably bypass the barrier then microplastics <5mm (Zhang et al., 2024).

Dermal contact

Dermal exposure to microplastics and nanoplastics is a growing trail for systemic absorption especially nanoplastics (Patrick et al., 2024; Pileta et al., 2024). Despite the fact that the skin behaves as a hindrance to epidermal contact of microplastics & nano plastics can be caused by the use of personal care products like facial scrubs and body washers, cosmetics like makeup, soap, and shampoo and tainted water during bathing (Pileta et al., 2024). Whereas the piercing of those molecules via the flawless skin is observed as negligible, Impaired and weaken skin permits them to delve deeper (Menichetti et al., 2024). Nanoparticles may enter through sweat ducts, hair follicles, or pores in the skin even though human skin serves as a strong barrier (Schwabl et al., 2019). Additionally, the additives and adsorbed chemicals present on the surface of microplastics have the potential to leak and be transdermally absorbed which causes cytotoxicity, oxidative stress, inflammation, and skin barrier impairment which may lead to illness as well as skin cancer (Pileta et al., 2024).

Size dependent penetration (NPs > MPs)

The biological effects of microplastics and nanoplastics are mostly determined by the size, shape, and surface chemistry of their particles. These characteristics indicate how readily they pass through biological barriers and build up in various organs (Yong et al., 2020; Yee et al., 2021). After ingestion, microplastics (1 μm –5 mm), which are comparatively bigger, typically stay in the gastrointestinal tract. The majority of them have little systemic absorption and are eliminated in feces (Schwabl et al., 2019). On the other hand, smaller pieces may enter intestinal epithelial cells through paracellular transport or endocytosis (Stock et al., 2019). Because of their incredibly small size and enormous surface area, nano plastics (less than 100 nm) have significantly higher bioavailability. The gut mucosa, alveolar membrane, blood–brain barrier and even the placental barrier are some of the biological barriers that these particles can pass through (Prata 2018; Yong et al., 2020). They may cause oxidative stress, inflammatory reactions and hormonal abnormalities in target organs such as the liver, kidneys, brain and endocrine glands once they enter the bloodstream (Lu et al., 2019). These findings indicate that nano plastics pose a greater potential risk to human health than larger microplastics due to their higher ability to penetrate tissues and interfere with cellular and endocrine functions.

Bioaccumulation and internal distribution

Because of their small size and durability, microplastics and nanoplastics can build up in tissues after they enter the body. MPs have been found in human blood, placenta and feces in recent investigations suggesting that they can go beyond the site of entry and circulate throughout the body (Ragusa et al., 2021). For example, it has been demonstrated in lab models that nano plastics can pass through biological barriers like the placental barrier, blood–brain barrier and intestinal epithelium (Yong et al., 2020). These results imply that MPs and NPs may build up in the thyroid gland and other endocrine organs. Once there they may cause oxidative damage, inflammation and enzyme inhibition which could disrupt hormone synthesis or receptor signaling. The risk of long-term physiological disruptions is increased by prolonged exposure and a poor rate of elimination.

The Thyroid Gland: Physiology and Endocrine Regulation

The thyroid gland is a butterfly-shaped endocrine organ that is situated in the anterior neck between the first thoracic (T1) and fifth cervical (C5) vertebrae. It is made up of two lateral lobes that are joined above the trachea by a thin isthmus (Khan and Farhana 2025). The superior and inferior thyroid arteries supply blood to the highly vascularized gland, allowing for the quick release of hormones into the bloodstream. Its primary function is to generate thyroid hormones that control growth, development, basal metabolic rate, and calcium homeostasis through parafollicular cells' release of calcitonin (Armstrong et al., 2023).

Hypothalamic-Pituitary-Thyroid (HPT) Axis

The physiological effects of thyroid hormones (THs) on brain development and function, growth, basal metabolic rate, thermogenesis, energy expenditure, muscular contraction, heart rate stimulation and the expression and activities of many proteins involved in lipid and carbohydrate metabolism are all regulated by the normal secretion of THs. The hypothalamic-pituitary-thyroid axis is a feedback loop that maintains hormonal balance and controls thyroid function. Thyrotropin-releasing hormone (TRH), which is released by the brain, triggers the anterior pituitary to release thyroid-stimulating hormone (TSH). TSH directly influences thyroid follicular cells to stimulate the production and release of hormones. TRH and TSH release are inhibited by circulating thyroid hormones (mostly T4 and T3), which closely regulate hormone levels and preserve homeostasis (Hersman and Beck-Peccoz 2023).

T3, T4 and TSH and their roles

Thyroxine (T4) and triiodothyronine (T3) are the two main hormones that the thyroid produces. T4 is primarily a

prohormone and accounts for approximately 90% of thyroid hormone production, although being less physiologically active. The active form that controls metabolic and developmental processes is T3, which is generated by both direct secretion and peripheral conversion from T4. The pituitary's release of TSH controls the synthesis of thyroid hormones and is a crucial clinical indicator of thyroid function (Armstrong et al., 2023).

Thyroid hormone synthesis, transport and action

Iodide ions are taken up by follicular cells through the sodium-iodide symporter (NIS), which initiates the creation of thyroid hormone. Thyroid peroxidase (TPO) oxidizes iodide and incorporates it into thyroglobulin to create iodinated tyrosine residues. T3 and T4 are produced by coupling processes inside the colloid, endocytosed back into follicular cells, processed, and released into circulation coupled to transport proteins like albumin, transthyretin, and thyroxine-binding globulin (Janssen & Janssen, 2017; Shahid et al., 2023). T3 enters cells at target tissues, attaches to nuclear thyroid hormone receptors (TR α , TR β), and modifies gene expression to control cellular development, differentiation, and metabolism. Because the thyroid hormones' systemic effect is essential for thermogenesis, neurodevelopment, and cardiovascular function, interruption of this route has serious consequences (Shahid et al., 2023).

Mechanisms of Endocrine Disruption by MNPs and associated chemicals

The hypothalamic-pituitary-thyroid axis (HPT), the hypothalamic-pituitary-adrenal axis (HPA), and the hypothalamic-pituitary-gonadal axis are just a few of the hypothalamic axes that can be disrupted by MPs and their composite toxic additives because they can cross a variety of biological membranes, blood-brain barriers, and hormone receptors (Ullah et al., 2023). MNPs can enter the human body through the skin, food or inhalation. Numerous organ systems as well as samples of blood, breast milk, sputum, feces and urine have been reported to accumulate MNPs. These MNPs have the ability to transport and absorb dangerous substances such as EDCs, and they can be hazardous to the tissue and organs where they build up. Smaller particles have a bigger surface area and stronger absorptive capacity releasing more harmful compounds and a larger dose can carry more poisons. Toxicity is reliant on both size and dose (Tyc et al., 2025). As MPs get older in natural aquatic habitats, they may form a biofilm or eco-corona on their surface that may adhere to natural minerals and lower particle buoyancy. The distribution of MPs and related pollutants like PFASs in aquatic systems may be impacted by this mechanism which may increase their sinking and vertical transit. These pollutants are carried to far off places by air and oceanic currents. Furthermore,

MNPs' interactions with biotic and abiotic components in ecosystems demonstrate the wide range of ecological dangers they pose, affecting everything from terrestrial plants and soil microbial communities to marine life (Santhanam et al., 2024).

Blood thyroid barrier

The blood thyroid barrier is a selectively permeable barrier that controls solute exchange between the bloodstream and thyroid follicle cells. The blood thyroid barrier is made up of fenestrated capillaries with pores as small as 10 to 30 nm is separated from the epithelial cells by a thin basement membrane. Tight junctions connect the epithelial cells at the base of the thyroid follicles. This architecture allows for the selective passage of iodide and other precursors into the thyroid follicle. However, it prevents the passage of potentially harmful substances and large molecules into the colloid thereby protecting the machinery that synthesizes hormones within the colloid.

Evidence for MNPs crossing biological barriers

Due to their size (less than 100 nm) nano plastics can pass through the blood thyroid barrier (BTB). Research using rodent models has shown that polystyrene nanoparticles (50 nm) have accumulated within rat thyroid follicles after they were administered orally or intravenously. This accumulation was visualized with fluorescence microscopy as well as transmission electron microscopy (TEM). Because of their fenestrated nature the capillary beds of the thyroid gland are able to facilitate the passage of particles into the surrounding tissue space where they may be taken up by endocytic activity and subsequently incorporated into thyroid follicular cells. The ability of microplastics (>1 micron) to cross the blood thyroid barrier is very limited (Rasmussen et al., 2019; Jing et al., 2025).

Oxidative stress and inflammation

The most recent study to examine the effects of nanoparticle inhalation on the development and advancement of chronic obstructive pulmonary disease demonstrated that nanoparticle exposure produced oxidative stress, inflammation and a protease or antiprotease imbalance at both local and systemic levels (Anwar et al., 2023; Mughal et al., 2024). Through surface catalyzed production of reactive oxygen species (ROS) and activation through phagocytes MNPs can damage thyroid follicular cells. In rat thyrocytes treated with 100 nm PS-NPs there was an increase in Malondialdehyde (MDA) and a decrease in glutathione (GSH) which activated Nrf2 and inflammatory cascades (NF- κ B, IL-6, TNF- α). Chronic inflammation causes fibrotic scarring surrounding thyroid follicles (Zhang et al., 2024; Islam et al., 2025).

Interference with hormone synthesis

The chemical pollutants present in MNPs are responsible for inhibiting important enzymatic processes that are required to convert iodine into a form that is usable by the body including thyroid peroxidase (TPO) which is required for both converting iodide to iodine for hormone synthesis and for the production of thyroid hormones. In addition to disrupting enzyme function the MNPs also disrupt thyrotropin receptor (TSHR) signaling which is important for regulating the timing of when thyroid hormones are released. MNPs inhibits enzymes, TPO decreased by 45% in PS-NP treated cells after 72 hours at 10 μ g/ML., this inhibits iodide organification. NIS decreased by 32% in cells exposed to PS-NP which impaired iodide uptake. Signaling through TSHR and cAMP was disrupted which reduced the hormone release. BPA, Phthalates, Bisphenols leached from MNPs further inhibited these processes and affected hormone synthesis and their secretion (Jing et al., 2025).

Disruption of hormone transport proteins

Mostly thyroid hormones are carried by the blood in a bound state with proteins such as Thyroxine binding globulin (TBG), Transthyretin (TTR) and Albumin. The presence of metals in MNPs and the chemicals that are adsorbed into them will be able to alter the affinity for the proteins of each of the previously mentioned hormones. This alteration is expected to affect the transport of the hormones, the availability of the hormones to enter cells and the rate at which the hormones are removed from the body. All of these changes could contribute to an imbalance in the body's natural regulation of circulating thyroid hormone levels. Hormones are transported by 70% of T4 by TBG, 15% TTR and 10% albumin. PBDEs and BPA from the surface of MNPs competitively inhibit the binding of T4 to transthyretin (TTR), thyroxine binding globulin (TBG) and albumin. Therefore, it reduced the amount of free T4 available to be metabolized and used by cells. In vitro PS-MPs that were loaded with PBDEs caused a reduction in the binding of T4 to proteins of 28% (Wang et al., 2023; Gorini et al., 2025).

Modulation of thyroid hormone receptors and signaling pathways

Thyroid hormone receptor interference caused by MNP bound endocrine disrupting chemicals can be either antagonist or agonist at thyroid hormone receptors (TR α and TR β). This type of interference with TRs is necessary for normal regulation of gene expression that is essential for thyroid hormone function which causes a variety of metabolic and developmental problems. The Ki values for

binding to TR α and TR β are similar for BPA analogues (BPS, BPF) and T3 (10^{-7} to 10^{-8} M). BPA analogues act as both partial agonists and antagonists on the Thyroid Hormone Receptors. Impairment of cell growth (proliferation) and hormone secretion through the disruption of signaling pathways (PI3K, AKT, MAPK and ERK) initiated by thyrotropin receptor (TSHR) also occurs due to exposure to PS-NPs (Jing et al., 2025; Stanojević & Dolenc, 2025).

Transcriptional dysregulation

MNP exposure is known to have an effect on the regulation of certain transcription factors in the thyroid such as PAX8 and CREB that are involved in the control of thyroid development, hormone gene expression and cellular differentiation. The disruption of these regulatory pathways could lead to a compromise in thyroid function and the initiation of a disease process. PAX8 is the master regulator of thyroid which has been shown to be decreased by 40% after 48 hours of PS-NP exposure thereby decreasing the potential of the Tg and TPO promoters. The phosphorylation of cAMP response element binding protein was decreased with the result being a decrease in TSH induced activation thereby resulting in the disruption of genes responsive to hormones. Thyroid specific genes like sodium iodide symporter (NIS), thyroglobulin (Tg) and TSH receptor show downregulation while type 2 deiodinase (DIO2) shows compensatory upregulation (Jing et al., 2025).

Evidence from Mammalian Studies on Micro & Nanoplastics (MNPs) and Thyroid Disruption

This section offers a thorough summary of data from mammalian models showing how microplastics (MPs) and nanoplastics (NPs) damage the thyroid. Using only peer-reviewed research published between 2023 and 2025, it investigates hormone disruptions, histopathological modifications, molecular changes, cellular toxicity, limited human data and crucial dose-response correlations (Ullah et al., 2023; Islam et al., 2025).

In Vivo Animal Studies

Thyroid hormone disruption caused by MNP is confirmed by numerous animal investigations. Serum T3, free T3 (FT3), free T4 (FT4), and total T4 levels dramatically dropped in a dose-dependent manner in rats exposed to polystyrene nano plastics (PS-NPs) at 1–10 mg/kg/day for five weeks, whereas TSH increased compensatory, indicating HPT axis suppression. Zebrafish exposed to PS-MPs also displayed embryonic thyroid abnormalities and decreased T4 and T3. These results imply that MNPs affect peripheral deiodination and hormone production (Ullah et al., 2023; Zhang et al., 2025).

Dose-dependent damage is revealed by thyroid histopathology. Mice exposed to PS-NP (0.1–1.0 mg/L) showed inflammatory infiltrates, follicular cell hypertrophy, colloid depletion, and epithelial disruption. Increased dosages caused vascular congestion and fibrosis. NP increase within follicular cells and colloid gaps was verified by transmission electron microscopy, which correlated with structural deterioration. These alterations resemble traditional hypothyroid disease, but they also have additional inflammatory characteristics specific to particle poisoning (Zhang et al., 2024).

Thyroid-specific genes are identified by transcriptomic studies. Exposure to PS-NP increased the expression of the TSH receptor (TSHR) and deiodinase genes while decreasing the expression of thyroglobulin (Tg), thyroid peroxidase (TPO), and sodium-iodide symporter (NIS). Changes in binding and expression were seen for the transcription factors CREB (hormone signaling) and PAX8 (thyroid development). There have also been reports of epigenetic alterations, such as DNA methylation of HPT axis promoters. Iodide absorption and hormone coupling pathways were found to be compromised by metabolomics (Aloisi & Poma, 2025).

In Vitro studies

Primary thyroid follicular cells and associated cell lines (such as FRTL-5 rat thyrocytes) when exposed to PS-NPs (10-100 μ g/mL), show dose and time-dependent toxicity (Aloisi & Poma, 2025). The significant findings include: Oxidative stress (elevated ROS, lipid peroxidation and Nrf2 pathway activation within 24 hours), Mitochondrial dysfunction (reduced membrane potential, ATP depletion and cytochrome c release leading to apoptosis), Endoplasmic Reticulum Stress (upregulated GRP78 & CHOP, impairing protein folding and hormone processing), Signaling Disruption that involve inhibition of Camp/ PKA pathway and PI3K/Akt signaling critical for cell survival and hormone secretion (Anwar et al., 2023). Polystyrene produced more ROS than polyethylene terephthalate, indicating polymer-specific effects. Uptake was affected by surface charge; cationic NPs exhibited three times more internalization by clathrin-mediated endocytosis (Aloisi & Poma, 2025).

Human epidemiological data

Recent research shows that exposure to MNPs may cause reproductive toxicity in mice. This toxicity can be noticed by oxidative stress, hormonal changes, reproductive issues and testis and ovarian damage (Ali et al., 2023). Prenatal thyroid programming effects were suggested by the correlation between decreased neonatal T4 and T4/T3 ratios and microplastic pollution in human placentas (Zhang et al., 2025). Detection of microplastics and nano plastics in 80%

of human blood samples correlates with subclinical hypothyroidism (high TSH, low FT4) in workers exposed to plastics. Thyroid nodule prevalence was 2.3 times higher with TSH profiles were altered due to high levels of plastic pollution. As cross-sectional evidence is insufficient for causation, there’s an urgent need for longitudinal cohort studies (Tyc et al., 2025).

Dose response and particle size considerations

According to dose response correlations higher MNP exposure typically leads to thyroid disruption yet threshold levels are still unclear. Nano plastics pose higher toxicological risks due to their increased bioavailability, superior cellular uptake and ability to bypass biological barriers. Because they can penetrate deeper into tissues smaller microplastics have a more damaging impact on cells and tissues than bigger ones, as shown in Table 1.

Table 1. Dose response and particle size consideration.

Effect category	Microplastics (1-5000µm)	Nanoplastics (<100nm)
Bioavailability	Low; largely retained in the gastrointestinal tract and excreted	High systemic penetration via endocytosis and translocation across biological barriers
T4 Hormone reduction	Decrease of approximately 15-25%	Decrease of approximately 40-60%
Cellular uptake	Primarily via macrophage phagocytosis	Direct thyrocytes uptake through clathrin-mediated endocytosis
Histopathology	Mild colloid depletion	Follicular disruption and fibrosis
Gene dysregulation	Downregulation of Tg and TPO	Downregulation of NIS, TSHR & PAX8; Upregulation of DIO2 (compensatory response)
Threshold dose	>3 mg/kg associated with over toxicity	0.1-1 mg/kg is associated with subtle HPT axis suppression
Surface area ratio	Baseline surface interaction	Approximately 1000 x higher surface area, enhancing EDC adsorption

Discussion

In real world settings micro and nano-plastics are rarely pure polymers instead they frequently contain strategically added additives (such as plasticizers and flame retardants) and have the ability to adsorb pollutants from dust, water, sediment and biota. These chemical co-exposures are crucial because many of the same compounds or which are known as suspected thyroid disruptors and MNPs can affect the transport, bioavailability and biological effects (Gorini et al., 2025). Many structural analogs (e.g., BPS, BPF and BPAF) are replacing bisphenol A (BPA) which is widely used in polycarbonate plastics and epoxy resins including applications with food contact potential. Recent mechanistic evidence indicates that BPA or its analogs can disrupt thyroid function by interfering at multiple levels such as altering thyroid related gene expression (including synthesis related genes like NIS, TPO and TG) and by interacting

with receptor mediated signaling pathways that regulate endocrine function. According to animal and cell-based evidence some analogs may have endocrine disrupting potency equal to or greater than BPA. This is significant because “regrettable substitution” may maintain thyroid risk even as BPA gradually disappears (Stanojević & Dolenc, 2025). Phthalates are polymer additives found in many consumer and commercial items that are used to give flexibility (most notably in PVC). Because they are not covalently bonded to polymers, they can migrate or spill into foods and environmental media. According to a 2025 review that focuses on the thyroid outcomes, there is mounting evidence that phthalates (and more recent alternative plasticizers) can affect thyroid function by interfering with hormone synthesis, transport, metabolism and action with

consumption of contaminated food being a significant exposure pathway. The same review points out that binding proteins like thyroxine-binding globulin (TBG) play a major role in thyroid hormone transport and that observed changes in hormone ratios (like TT4/fT4 or TT3/fT3) in relation to phthalate exposure are consistent with interference at the level of binding proteins and feedback regulation in the HPT axis (Gorini et al., 2025). PBDEs, or legacy brominated flame retardants, are persistent in the atmosphere, water resources, soil, animals and human bodies. It is fat soluble and has historically been used in daily essentials like plastic products, building materials, electronic equipment and in textiles. There is evidence that PBDEs disrupt the thyroid through a variety of mechanisms such as competitive binding to thyroid hormone transport proteins (like TTR and TBG) and induction of hepatic enzymes (like UGTs) that raise thyroxine clearance and disrupt the homeostasis of circulating thyroid hormones (Wang et al., 2023).

Plastic particles in the environment may absorb a wide range of co-contaminants such as metals, endocrine disruptors and persistent organic pollutants which means that exposure frequently happens as a mixture rather than as a single chemical. Lab studies show that as microplastics age and their surfaces change they become more effective at grabbing and releasing BPA in biological systems proving that weather plastics act as carriers whose toxic payload depends on their age and the conditions of the body. The thyroid axis is prone to disruption at multiple checkpoints (synthesis enzymes, binding proteins, transporters, receptors metabolism) so mixtures transported or concentrated by MNPs can possibly affect thyroid function through many converging processes rather than a single target (Saraceni et al., 2025).

Mixture toxicity is a major problem since MNPs may increase internal exposure to contaminants and additives by concentrating them at cell membranes or within tissues and transferring them across exposure interfaces (gut/lung). The direction of interaction is not confirmed though. *In vitro* study investigating BPA absorbed microplastics discovered that adsorption/desorption was highly dependent on MP surface chemistry and reported that synergy was not always seen under their particular experimental conditions which highlights the fact that results could depend on polymer type, aging, functional groups and exposure design. Recent findings show that BPA and its substitutes can have synergistic or additive effects via multiple pathways like receptor signaling and epigenetic pathways highlighting the need to evaluate mixtures rather than evaluating chemicals in isolation (Ullah et al., 2023).

Many plastics associated chemicals that are related to thyroid disturbance are environmentally persistent and have the potential to bioaccumulate particularly hydrophobic substances like PBDEs which may persist in indoor dust and

food webs long after use limitations are implemented. Phthalates are produced in large quantities, used extensively and continuously released (by leaching or abrasion) which is why they maintain chronic exposure pressure even though they may not be as persistent as PBDEs in all environments. Some phthalates may also show slow degradation under natural conditions in specific settings. Chronic low dose thyroid relevant exposures can persist even when point sources decrease because MNPs themselves are physically persistent and their extended residence durations offer a continuous platform for chemical sorption/desorption cycles (Wang et al., 2023).

A major research priority is the development of synchronized and authentic methods for MNPs detection and quantification. FTIR (Fourier-transform infrared spectroscopy) and Raman are commonly used methods to evaluate tiny particles from different environmental samples for the quantification and identification of MPs. The alternate method which is using now a days is pyrolysis GC-MS which makes it better for detecting low concentrations and tiny plastic particles providing a more accurate way to measure mass of specific plastic types. Because pyrolysis GC-MS is still facing obstacles regarding matrix interference, inconsistent calibration and absence of standardized protocols for data processing. Recent research recommended that there is a need for clear standardization procedures and the use of AI driven data analysis to achieve more consistent and accurate polymer identification and quantification (Jing et al., 2025).

The accurate identification of nano plastics in tissues and blood at environmentally relevant levels is an ongoing analytical problem. The literature identifies this as a key factor in the uncertainty surrounding nano-plastic risk assessment when compared to microplastics. Recent studies on detection methods highlight integrating complementary tools which are spectroscopy for particle characterization and MS-based approaches for polymer mass to boost confidence especially in biological materials. Thyroid dysfunction can be detrimental because thyroid hormones control adult metabolism, brain function and fetal/child development. Even small changes in T3/T4/TSH levels can have significant long term health effects. Although direct human data is still insufficient. Experiments and review of literature eventually connect micro and nano plastics to endocrine relevant pathways which influence the current approach to risk assessment (Jahedi et al., 2025). For fetal growth and neurodevelopment, adequate maternal thyroid hormone availability is crucial. Interruption during pregnancy is linked to unfavorable developmental trajectories, such as poor development and poor growth outcomes. Since MNPs can transport hormone disrupting additives and can also stimulate oxidative or inflammatory stress on their own. Interference with thyroid function is

viewed as a credible mechanism by which exposure to these particles may help generate the developmental toxicity effects seen in experimental studies (Zhang et al., 2025).

Thyroid disruption can lead to weight loss fluctuations, dyslipidemia and other metabolic disorders because thyroid hormones affect basal metabolic rate, lipid metabolism, carbohydrate metabolism and energy expenditure. Although population level causation confirmation is still unclear. Metabolic dysfunction is listed among the likely outcome categories supported by animal and cell studies in recent comprehensive evaluations of MNP health effects (Li et al., 2023).

Thyroid imbalance is mechanistically linked to neurocognitive risk especially when exposure occurs during pregnancy or early childhood because thyroid regulation is closely linked to the development of the central nervous system and adult brain function. According to recent reviews thyroid mediated mechanisms may interact with neurotoxicity and neuroendocrine consequences in experimental micro and nano plastics research either directly through hormone signaling or indirectly through inflammation and oxidative stress (Ali et al., 2023).

Pregnancy constitutes a particularly vulnerable period because the developing fetus relies partially on the mother's thyroid hormone particularly during the gestation period. Because micro and nano plastics are pervasive in the environment entering the body through the food we eat, the water we drink and the air we breathe. Laboratory studies have demonstrated that MNPs can interfere with hormone pathways. Pregnancy is commonly highlighted as a priority group for exposure reduction and future epidemiologic research (Zhang et al., 2023).

Infants and young children are at risk to micro and nano plastics because their developing endocrine and nervous systems are more prone to hormonal disruption and they often receive higher doses per kilogram of body weight due to elevated intake routes, hand to mouth actions, plastic toys and the routine use of plastic feeding bottles which can leach millions of microplastic particles during formula preparation and sterilization. Toddlers spend several hours a day playing with toys and frequently put them in their mouths, they ingest particles that have detached from the toy surface. Studies show that plastics are the common source of MPs in toddlers as the frequency of hand to mouth events is 30 to 70 contacts per hour, which can lead to ingestion of millions of particles each day. Although current research does not yet provide enough quantitative data to precisely estimate thyroid-related risks in this age group. The collected evidence is sufficient to justify precautionary research programs and systemic monitoring of infants and toddlers for MNP exposure (Bhattacharyya et al., 2025).

Some occupational groups and communities such as those who work with plastics, dust or fibers can experience high

levels of micro and nano plastics making these populations the key targets for biomonitoring and early risk identification (Jahedi & Fard, 2025).

One major difficulty is that micro and nano plastics are not a uniform unit. They comprise a diverse array of particle sizes, forms (such as fibers, fragments and spheres), polymer types, degrees of environmental aging and attached chemical. This variability complicates the development of universal exposure metrics and holds the ability to apply consistent dose response models across different MNP populations. Although many animal studies and cell culture experiments have well known harmful effects of micro and nano plastics. But there is little direct evidence that the levels of MNPs normally encountered in that environment cause particular diseases in humans. The lack of real-world epidemiological data makes it difficult to establish reliable reference doses or formal guideline values (Jahedi & Fard, 2025; Ali et al., 2025).

The currently continuous data gaps especially for nano plastics the European parliament has given a mandate to EFSA (European Food Safety Agency) which was accepted in December 2025, to perform an updated scientific assessment of the health risks posed by microplastics in food, water and air. This task is expected to be completed by 2027 and the results of the study will be expected to have a significant effect in dealing with microplastics. The EPA (Environmental Protection Agency) which is the federal agency in the US, is responsible for protecting human health and the environment. The EPA's US microplastics research program is actively refining human health testing methods, utilizing *in vitro* assays, computational toxicology and approaches to assess partial uptake and clearance which are the crucial steps that must be completed before a quantitative risk assessment can be fully established.

Many researchers describe that MNPs thyroid concerns as biologically realistic with growing experimental support in light of the current research data. However, they advise more thorough exposure assessment, standardized hazard testing and long-term human investigations before a conclusive risk threshold can be developed (Ali et al., 2025).

Knowledge Gaps and Research Challenges

Measuring the actual risks of micro and nano plastics remains a massive challenge because exposure is difficult to measure with accuracy. Studies use inconsistent materials and endpoints and the smallest particles (nano plastics) are still analytically challenging to identify in real world samples. According to the recent research it will remain challenging to compare data from different labs and research or convert them into recommendations for human health in the absence of enhanced standardization and analytical techniques (Cunningham et al., 2023).

Limitations of current study

One significant drawback is the over reliance on simplified models (single polymer types like polystyrene spheres) which might not accurately describe irregular, aged, mixed polymer particles that humans come into contact with. There is limited data on nano plastics compared to microplastics which makes it uncertain to generalize toxicity and behavioral findings from larger particles down to nanoscale (Ali et al., 2025).

Numerous studies on particle exposure use inconsistent parameters like particle counts, total mass and nominal concentrations which makes it difficult to compare results. In reality human exposure involves a complex mixture of varying sizes, shapes including fragments and fibers, and weathered surfaces that have degraded over time. Limited toxicokinetic data is another major drawback; it is difficult to link exterior exposure to internal organ dose because absorption, distribution, metabolism and elimination make it challenging for analysis (Cunningham et al., 2023). Many toxicological studies emphasize general physiological markers of distress like inflammation and oxidative stress instead of specific endocrine-relevant results for cross-study comparison and alignment with regulatory safety frameworks. Furthermore, some research involves high concentrations and limited exposure time, which are useful for identifying pathways but might not accurately represent long-term low-dose human exposure (Ali et al., 2025).

Need for standardized methodologies

Standardization is frequently cited as a significant hindrance. Studies vary in particle preparation, dispersion techniques, measured and reported by using particle number, mass, surface area and polymer-specific markers. Standardized test protocols and shared templates are essential for authoritative evidence, as demonstrated by OECD methodologies for nanomaterial testing and the mutual acceptance of data concept (Rasmussen et al., 2019). Ongoing debates continue to modify toxicokinetic recommendations for nanomaterials. According to recent method-focused articles, consistent sample preparation, calibration, and data processing rules are necessary to make results repeatable and comparable across labs, even for commonly used quantification instruments like Pyrolysis GC-MS (Jing et al., 2025).

Conclusion

There is increasing evidence that micro and nano plastics can accumulate in tissues and interact with biological pathways related to endocrine function making them prevalent pollutant that humans are exposed to through ingestion, inhalation and dermal contact. These effects are size dependent and frequently indicate higher potency for

nano plastics. We have strong laboratory evidence that MNPs causes thyroid disruption but it is hard to prove that same MNPs cause thyroid disruption in humans because measuring real world exposure is challenging. Recent reviews on plastic related health risks highlight that lack of evidence is not proof of safety and they encourage preventative measures in addition to better evidence production to fill research gaps. Future research directions include standardized MNP materials and dosage metrics (mass, number, surface area) with transparent reporting, conducting long term low dose exposure studies mimicking environmental scenarios, and well powered human trials with repeated thyroid endpoints and enhanced biomarker screening. To better detect and measure plastics we need to continue improving and standardizing Py-GC-MS methods for enhanced accuracy acknowledging that current methods for analyzing polymers in biological samples still face limitations regarding accuracy, interference and sensitivity. Based on existing research policies should focus on reducing unnecessary plastic exposure (from food packaging, industrial releases and indoor dust), requiring better disclosure of additives and updating safety assessments to include mixtures and nano plastics.

Author Contributions

Sammar Zaitoon, Janita Ashiq, Saira Muqadas, Saima Malik, Ghazala Nayab: Methodology, writing – original draft and visualization (Figures 1 and 2). Tahreem Riaz, Feroza, Fatima Abid, Kinza Fatima: Literature search, data curation: Tahseen Akhtar, Kashaf Masood, Fahmeeda Zafar: Writing – review & editing and formal analysis. Imran Mukhtar: Supervision, Conceptualization, project administration and final validation.

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Conflicts of Interest

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