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Abstract

Purpose: The high dependence on plastic in food processing and packaging introduces microplastics (MPs), nanoplastics (NPs), and endocrine-disrupting chemicals (EDCs) into the food chain, posing potential risks to public health, including immune alterations, inflammation, and metabolic disruptions linked to type 2 diabetes (T2DM). To assess the extent of plastic contamination in the food chain and its associated health effects, specifically focusing on the connection to T2DM.

Methodology: A systematic literature search was conducted across PubMed, Google Scholar, and governmental health agency publications from the past five years, using keywords related to MPs, NPs, EDCs, T2DM, and plastic policy.

Findings: MPs are widespread contaminants in the food chain, with major sources including marine organisms, bottled water, and packaging. Estimated annual human exposure ranges from 39,000 to 52,000 MP particles, with bottled water contributing up to 90,000 particles alone. MPs carry EDCs, such as bisphenols and phthalates, which are implicated in impaired insulin function and increased T2DM risk. Current filtration methods are insufficient to eliminate contamination, highlighting the need for stronger regulatory measures. While resources are spent on T2DM management, primary prevention remains inadequate.

Unique Contribution to Theory, Practice and Policy: This study highlights the significant threat of plastics use-particularly MPs, NPs and EDCs within the foodcahin. It implements several key findings including: Public health risk, regulatory and policy reform, economic consideration as well as research prioritization. Overall, this study mitigates the health risks associated with plastic contamination, stricter regulations on plastic use in food processing and packaging are imperative, alongside incentives for biodegradable alternatives. Future research should prioritize investigating the long-term effects of MP exposure and developing innovative strategies to reduce plastic contamination in the food chain.

Keywords: Diabetes, Endocrine, Food, Health, Microplastics, Nanoplastics, Endocrine Disrupting Chemicals

JEL Codes: *118*, *Q53*, *Q52*, *Q58*, *D62*

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INTRODUCTION

The escalating presence of microplastics (MPs), plastic particles <5 mm in size, poses a significant and increasingly recognized threat to human health [1]. While MPs are concerning generally, particles between 1 and 50 µm range are of particular interest due to their ability to get absorbed and accumulated in the body [2]. It is widely recognized that MPs, even at low concentrations, can enter biological systems through ingestion, inhalation, or dermal contact. However, ingestion remains the primary exposure route. Globally, humans on average are estimated to ingest tens of thousands of microplastic particles annually through various exposure routes. A widely cited study by Cox et al. (2019) estimated that annual human MP ingestion ranges from 39,000 to 52,000 particles, depending on age and sex, from food and beverages alone, with bottled water contributing up to an additional 90,000 particles per year for high consumers. [3]. Once ingested, MPs accumulate in various tissues and organs, including the lungs, liver, kidneys, spleen, and reproductive organs. Another study by Senathirajah et al. (2021) estimated that humans may ingest up to 5 grams of MPs weekly, although this figure represents an upper bound estimate and require validation. [4]. MPs exert localized cellular and tissue toxicity, primarily through inflammation. Moreover, they act as foreign bodies causing harmful effects by releasing chemicals that trigger the immune system [5]. Of particular interest is the role of EDCs carried by MPs. These EDCs, structurally similar to hormone receptors, alter the hormonal action of the endocrine glands, disrupting glucose metabolism by promoting oxidative stress, inflammation, and gut microbiota dysbiosis, contributing to insulin resistance and T2DM [6]. The consumption of plastic products has been increasing since their commercialization for household use in the 1950s. As their use expanded into the food industry for easier packaging, production, and disposability, plastics inevitably began to impact the environment, affecting agriculture and food sources [7]. This contamination extends beyond macroscopic level, entering the food chain in the form of MPs and NPs. MPs and NPs are small plastic particles that result from industrial manufacturing or the degradation of larger plastic particles. MPs and NPs differ in size, as NPs are smaller (<1µm) than MPs (<5mm).

Exposure to these particles exerts a range of adverse health effects, including oxidative stress, inflammation, hormonal disruption, liver damage, kidney dysfunction, and possibly cancer. Research has predominantly focused on the hepatotoxic and immuno-toxic effects of MPs/NPs, since these organs are especially vulnerable to environmental pollutants. Nonetheless, the potential link between MPs/NPs and T2DM remains understudied. Certain MPs and NPs, such as bisphenol A or phthalates, carry EDCs which suggest a plausible mechanism for increased insulin levels and T2DM risk [8]. This review aims to comprehensively examine the existing evidence on the potential association between MPs/NPs exposure and T2DM.

METHODOLOGY

A systematic literature search was conducted across the following electronic databases: PubMed, PLOS Biology, Science Direct and Google scholar. Our research also included publications from trusted organizations, governmental health agencies, and educational platforms focused on environmental health and T2DM. We ensured that the information collected from websites was credible and relevant. To supplement peer-reviewed literature, we have also reviewed articles from trusted organizations; such as the World Health Organization (WHO), United Nations



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Environment Program (UNEP) and other scientific bodies. The search strategy employed a combination of keywords and MeSH terms, such as "microplastics", "nanoplastics", "diabetes mellitus", "endocrine-disrupting chemicals", "insulin resistance", "plastic policy", "bisphenol A", "phthalates", and "metabolic disorders". We used Boolean operators (AND, OR) to combine search terms.

Inclusion criteria were: (1) original research articles, review articles, and reports published in English within past 5 years (2018-2023); (2) studies exploring the health impacts of MPs/NPs on insulin resistance, T2DM and endocrine disruption in human or in vitro/in vivo models; and (3) web-based resources from reputable organizations, governmental health agencies, or peer-reviewed content aggregators.

Exclusion criteria were: (1) non-English publications; (2) non-credible content from non-reputable websites; (2) studies unrelated to human health or specified health outcomes; and (4) duplicate publications.

Two independent reviewers were involved in the screening process of the literature searched, who assessed the titles and abstracts of papers for relevance to our study. We obtained full text articles for the publications meeting our inclusion criteria. Discrepancies between the two independent reviewers during screening were resolved through adjudication by a third reviewer, who assessed the conflicting items and guided the final decision. This approach ensured objectivity and consistency throughout the review process. Each study was thoroughly explored, and data was extracted related to the study design, sample characteristics, methods employed to assess the exposure, measurements of primary outcomes related to T2DM and endocrine disruptions, and the key findings. Due to the diverse nature of the study included, we conducted a narrative synthesis to summarize the findings in the most appropriate manner.

Microplastic Contamination and Health Risks

Microplastic Contamination in the Food Chain

The study presents a detailed analysis of MP pollution along the food production chain, from agriculture through packaging and water, and shows its availability in different environmental and food matrices and the risk it poses to humans. MPs, such as polyethylene (PE), polypropylene (PP), and polystyrene (PS), are plastic materials that are brought into the food web through contaminated soil, water, and sediments. Sediment samples from different environmental settings reveal MP concentrations ranging from 10 to over 4,900 particles per kilogram. MPs can also be contained in bio-waste-based fertilizers, with concentrations ranging from 0 to 895 particles per kilogram. Marine organisms such as mussels, oysters, fish, and shrimp are vulnerable to MP contamination, with tissue concentrations ranging from 0.7 to 6.4 particles per gram with most of them located in the digestive tract [9]. Estimated average annual MP consumption is in the range of 25 to 32,000 particles for fish, 322 to 19,511 for crustaceans, and 500 to 32,750 for mollusks. Bottled water is also another important contributor, where particles pollute the water over 10,390 per liter, higher than tap water, based on which one uses the Polyethylene terephthalate (PET) or glass bottle [10]. To apply these numbers in a day-to-day example, an adult consuming seafood may ingest approximately 0.5 to 630 microplastic particles per week from fish alone. Furthermore, PET or nylon tea bags can leak hundreds of millions of MPs and NPs during infusion, highlighting



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the impact of packaging and processing materials as source of contamination. The fact that microplastics are still present in drinking water, seafood, and other foods, even though 70–83% of them are removed by water treatment facilities, indicates the limitations of current filtration methods. An estimated 39000 to 52000 particles are exposed to humans annually through food and water with bottled water alone accounting for up to 90000 particles [11][12]

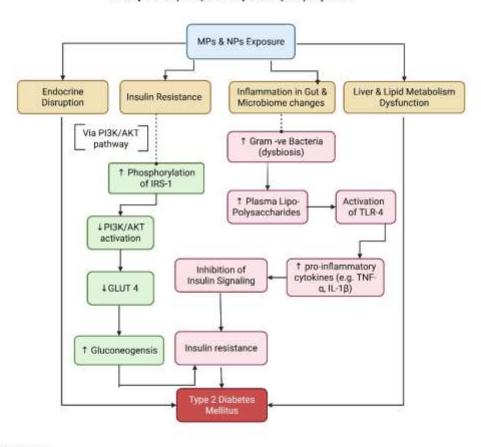
Microplastics have been detected in freshwater sources for drinking water in sizes ranging from less than 1 μm to 5000 μm . Most studies set a practical range of 1 to 750 μm , since particles above 100 μm are generally removed by standard filtration.[13] These observed size ranges help establish a foundation for risk assessment frameworks, such as the Threshold Microplastics Concentration (TMC), which is a recently introduced and scientifically tested concept. TMC allows for the calculation of the maximum number of microplastic particles per liter of water that may be considered safe based on conservative assumptions. Importantly, the TMC is a screening tool rather than a regulatory standard, enabling utilities and regulators to determine whether further water quality analysis or intervention is warranted.[13]

Chemical Additives and Health Risks

Plastic packaging releases a range of toxic additives, including bisphenol A (BPA), phthalates polybrominated diphenyl ethers (PBDEs), tetrabromobisphenol A (TBBPA), and bisphenol S (BPS). These substances pose significant health risks to humans by leaching into food and environment, potentially leading to cancer and endocrine disruptions. Furthermore, MPs act as carriers for persistent organic pollutants (POPs) that build up in the food chain particularly in seafood. EDCs like phthalates and bisphenols (BPs) present in microplastics affect the function of pancreatic β-cells by disrupting the biochemical pathways required for β-cell adaptation to metabolic stress. Moreover, endoplasmic reticulum stress that affects insulin synthesis, mitochondrial dysfunction, elevated reactive oxygen species (ROS), and modifications in glucosestimulated insulin secretion (GSIS) as a result of altered electrical activity Ca2+ signaling and ion channel expression, all are among these disruptions. Insulin resistance, hyperinsulinemia, and βcell apoptosis result from these disruptions, which prevents β -cells from compensating for insulin resistance and speeds up the onset of T2DM. EDC exposure is associated with a higher populationlevel risk of T2DM through both direct and indirect effect, Indirectly EDCs can contribute to obesity, a significant risk factor for insulin resistance and T2DM. Directly they can impair insulin secretion and β-cell function. Furthermore, environmental pollutants can stimulate the release of proinflammatory cytokines from adipose tissue. This inflammation exacerbates β -cell impairment and strengthens the link between EDC exposure and T2DM pathogenesis [14,15].



Pathway to Type 2 Diabetes Mellitus Due to Microplastics(MPs) & Nanoplastics(NPs) exposure



Abbreviations

-we. Negative, TLR-4: Toll-Like receptor 4, TNFa: Tissue Necrosis Factor Alpha, IL-18: Interleukin-1 Beta, PI3K: Phosphatidylinositol 3-kinase, Akt: Protein kinase B; IRS-1: Insulin receptor substrate-1, GLUT 4: Glucose transporter type 4.

Figure 1: Simplified Pathway to Type 2 Diabetes Mellitus due to Exposure to Microplastics & Nanoplastics

Information Adapted from Hsiao, et al [16].

Regulatory Challenges and Public Health Implications

Current regulations on MPs in food are insufficient. Due to the lack of specific measures targeting the sources and pathways of MPs into the food chain. Key areas of contamination, such as marine or agricultural environments, lack a comprehensive framework for monitoring or limiting MPs.[17] Current efforts to manage plastic waste mainly focus on larger items, often overlooking the microscopic particles that arise from plastic degradation and their effects on human health.[18] Furthermore, waste management systems lack the advanced recycling and filtration technologies necessary to stop MP pollution, which allows these particles to continue to exist in ecosystems. Targeted policies, better infrastructure, and increased awareness are necessary for effective regulation to reduce MP contamination in food [19].



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Risks of Plastics in Food Packaging

The use of plastic materials in food packaging has raised significant concerns regarding their long-term health implications. Numerous studies have demonstrated that chemicals leached from plastic packaging—especially EDCs like BPA and phthalates—can enter food and beverages, potentially leading to chronic health conditions such as T2DM, obesity, and cardiovascular disease. These compounds interfere with hormonal pathways by mimicking or blocking endogenous hormones, disrupting metabolic and cellular functions even at low concentrations.

Empirical research has supported these associations. A landmark study by Lang et al. [20] using data from the U.S. NHANES found that adults with higher urinary BPA levels had significantly greater odds of developing cardiovascular disease and T2DM. This evidence underscores the potential metabolic toxicity of BPA, which is commonly found in polycarbonate plastics and the epoxy linings of food cans.

The leaching behavior of plastics varies significantly depending on the polymer type and environmental conditions. PET, widely used in water and soda bottles, is considered relatively stable under standard conditions. However, when exposed to heat or UV radiation, PET can release trace amounts of antimony, a potential carcinogen, as well as aldehydes and acetaldehyde, which may alter the organoleptic properties of food [21]. In contrast, polyvinyl chloride (PVC) often contains plasticizers like di-2-ethylhexyl phthalate (DEHP), which readily leach into fatty foods, especially under heat or prolonged contact. These phthalates are known to interfere with insulin signaling and lipid metabolism, contributing to metabolic syndrome.

PP, commonly used in yogurt containers and microwave-safe containers, exhibits more thermal stability and a lower tendency for additive migration compared to PET and PVC. However, it can still release oligomers and degradation products—particularly when heated—raising concerns about cumulative exposure through repeated use [22].

Given the widespread use of these materials and their potential to affect human health through endocrine disruption and chemical migration, it is critical to re-evaluate food safety standards and packaging alternatives. This paper will further explore the mechanisms by which EDCs from plastics contribute to the pathogenesis of chronic diseases and highlight regulatory gaps that need to be addressed to mitigate public health risks.

Endocrine Disrupting Impact of Chemical Contaminants in Plastics

Food packaging materials frequently incorporate a wide range of synthetic polymers and chemical additives that can act as EDCs. These compounds interfere with the body's hormonal signaling systems, often by mimicking, blocking, or altering the synthesis and metabolism of endogenous hormones. Prominent among these are phthalates, bisphenols (such as BPA, BPS, and BPF), and perfluoroalkyl substances (PFAS), all of which have been extensively used in food contact materials due to their roles in enhancing plastic flexibility, durability, and resistance to heat and moisture. These substances were selected for focus because they are among the most ubiquitously detected in human biomonitoring studies, and because they show consistent associations with metabolic, reproductive, and developmental disturbances in both human and animal studies.

However, beyond these major groups, other chemicals present in food packaging materials also raise concern. For example, nonylphenol, a degradation product of nonylphenol ethoxylates used



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in plastics, is known for its estrogenic activity. Styrene, the monomer of polystyrene, can migrate into food under high-temperature conditions and has been shown to have potential neurotoxic and endocrine-disrupting effects. Additionally, benzophenones, used as UV filters and stabilizers in some plastic films and containers, can disrupt thyroid function and exhibit estrogenic and anti-androgenic properties. These lesser-known but biologically active compounds broaden the chemical scope of concern regarding plastic food packaging.

Migration of these chemicals from packaging into food occurs more readily under certain conditions — particularly heating, microwaving, mechanical stress, or contact with fatty, salty, or acidic foods. These interactions facilitate the release of free chemicals or unbound monomers from the polymer matrix. Once ingested, these EDCs can bind to hormone receptors (e.g., estrogen receptors, androgen receptors, PPAR γ) or interfere with nuclear transcription factors, disrupting gene expression and downstream cellular processes. This dysregulation has been implicated in a wide spectrum of health conditions, particularly those involving energy homeostasis, glucose regulation, and lipid metabolism.

For instance, bisphenols and phthalates have been shown to impair insulin receptor signaling and pancreatic β -cell function, resulting in reduced insulin sensitivity, elevated glucose levels, and a chronic state of low-grade inflammation [24][26][27]. These changes contribute directly to the development of T2DM and metabolic syndrome. Phthalates, in particular, can activate PPARs (peroxisome proliferator-activated receptors), leading to abnormal adipogenesis and lipid storage, further predisposing individuals to obesity and dyslipidemia.

In addition to these chemical contaminants, microplastics — tiny plastic particles formed through degradation or manufacturing processes — can be ingested through food and beverages. These particles can accumulate in the gastrointestinal tract and induce local immune activation, epithelial damage, and oxidative stress [23]. Microplastics also act as carriers for other toxic environmental chemicals (e.g., heavy metals, persistent organic pollutants), facilitating their entry into the body and compounding their biological effects. The accumulation of microplastics and their associated toxicants in human tissues may further disrupt immune and metabolic pathways, increasing susceptibility to cardiovascular diseases and chronic inflammation-related disorders [23][26].

Long-term, low-dose exposure to EDCs — particularly BPA, phthalates, and their analogues — has been strongly associated with subtle but persistent endocrine disruptions. Even at concentrations previously considered "safe," these substances can interfere with developmental programming, alter pubertal timing, impair reproductive hormone regulation, and promote fat accumulation through effects on estrogenic, androgenic, and glucocorticoid pathways. When combined with lifestyle and genetic factors, this chronic chemical exposure creates a biological environment that favors the onset and progression of T2DM, obesity, and other non-communicable diseases [24][26][27].

Cellular and Non-Cellular Mechanisms of Nanoplastics in Type 2 Diabetes Development

NPs contribute to the development of T2DM through various cellular and non-cellular mechanisms.



Cellular Mechanisms

After NPs are taken into the body, they tend to build up in important organs like the liver and pancreas. In vivo mouse studies, oral exposure to polystyrene nano plastics at doses of 1 mg/kg/day for 28 days has been shown to increase ROS, including oxidative stress. This stress disrupts the PI3K/Akt signaling pathway, which plays a major role in controlling how the body handles glucose. As a result, there's an increase in the phosphorylation of insulin receptor substrate-1 (IRS-1) at a specific site (serine 307), which lowers the activity of the Akt protein. Since Akt is crucial for helping insulin regulate glucose and fat metabolism, its reduced activity can lead to insulin resistance and high blood sugar levels. [32][33].

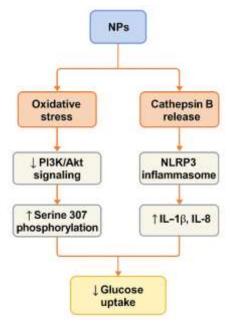


Figure 2: Flowchart showing how NPs Impact PI3K/Akt, IRS-1, NLRP3, and Glucose Uptake.

These effects have primarily been observed at moderately high concentrations, above typical daily environmental exposure, yet may become relevant in cumulative or hotspot exposure settings (e.g. contaminated food or occupational contact).

In addition, a type of nanoparticle called amino-modified polystyrene (PS-NH₂) can trigger inflammation. It does this by causing cathepsin B to leak into the cell's cytoplasm, which activates a protein complex called the NLRP3 inflammasome. In vitro experiments using mammalian cells exposed to 10-50 ng/mL for 24-48 hours demonstrated leakage of cathepsin B. This complex turns on inflammatory responses, leading to the release of cytokines like IL-1β and IL-8. This consistent low-level inflammation has been linked to insulin resistance. IL-1β, in particular, interferes with insulin signaling by promoting more phosphorylation of IRS-1, further disrupting the PI3K/Akt pathway and reducing glucose uptake by body tissues—ultimately resulting in high blood sugar [34].



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Non-Cellular Mechanisms

Nanoparticles, including metal-based, carbon-based, and polymeric types, have been increasingly associated with adverse effects on liver function and metabolic health. These particles can interfere with hepatic enzyme activity and activate signaling pathways such as Wnt/ β -catenin, which are implicated in the development of liver fibrosis and glucose intolerance [35].

These effects have been demonstrated in animal models exposed to 1-10 mg/kg/day for 30-60 days, a range that exceeds daily environmental exposure but may stimulate bio accumulative impact from chronic ingestion.

Given the liver's high blood flow and central role in detoxification, it is particularly susceptible to damage from nanoparticle exposure.

One of the primary ways NPs affect the liver is by disrupting lipid metabolism. Specifically, they impair the function of enzymes like acetyl-CoA carboxylase (ACC) and fatty acid synthase (FAS), leading to increased fat synthesis (lipogenesis). This metabolic shift contributes to the onset of non-alcoholic fatty liver disease (NAFLD) and promotes insulin resistance. Furthermore, chronic activation of the Wnt/ β -catenin pathway may result in abnormal cellular proliferation, tissue fibrosis, and impaired insulin signaling, thereby exacerbating glucose intolerance and metabolic dysregulation [35].

In addition to hepatic effects, certain nanoparticles—such as titanium dioxide (TiO₂), silver nanoparticles (AgNPs), and polystyrene—can significantly alter gut microbiota composition. These disruptions often reduce beneficial bacterial populations while encouraging the growth of harmful strains like *Escherichia coli*. As a Gram-negative bacterium, *E. coli* produces lipopolysaccharides (LPS), which can enter systemic circulation by compromising intestinal tight junctions—a phenomenon commonly referred to as "leaky gut." [36]

Once in the bloodstream, LPS activates Toll-like receptor 4 (TLR4) on immune and metabolic cells, initiating pro-inflammatory signaling pathways such as NF- κ B and JNK. These immune responses were reported at low to moderate doses indicating their relevence to chronic human dietry exposure.

This inflammation, in turn, interferes with insulin signaling by increasing serine phosphorylation of insulin receptor substrates (IRS), generating oxidative stress, and impairing mitochondrial function. These combined effects contribute to systemic insulin resistance and disturbances in glucose metabolism [36].



Table 1: Research Articles and their Study Focus on T2DM and Microplastics

#	Title	Authors & Year	Type	Model/ System	Study Focus	Main Findings
[1]	Discovery and quantification of plastic particle pollution in human blood	Leslie et al. (2022)[1]	In vivo	Human blood	Plastic particles in blood	Detected plastic particles in 77% of human blood samples; suggests systemic exposure.
[2]	Polystyrene microplastics exacerbate systemic inflammation in high- fat diet-induced obesity	Lee et al. (2023)[2]	In vivo	Mice (high- fat diet)	Inflammation from microplastics	Polystyrene MPs increased systemic inflammation and worsened obesity-related parameters.
[3]	Impact of microplastic exposure on blood glucose levels and gut microbiota	Xu et al. (2024) [5]	In vivo	Mice (normal/HF D)	Glucose levels, gut microbiota	MPs altered gut microbiota and elevated glucose levels, worse in high-fat diet group.
[4]	Endocrine disruptors in plastics alter β-cell physiology and increase the risk of diabetes mellitus	Martínez- Pinna et al. (2023) [15]	In vitro	Pancreatic β-cells	β-cell dysfunction	Plastic endocrine disruptors impair insulin secretion and β-cell function.
[5]	Polystyrene nanoplastics with different functional groups and charges have different impacts on type 2 diabetes	Zhang et al. (2025) [32]	In vivo	Mice	Charged NPs & diabetes	Different charges on NPs led to varied diabetic outcomes and inflammation.
[6]	Amino-modified polystyrene nanoplastics induce NLRP3 inflammasome- dependent inflammation and apoptosis in mammalian cells	Li et al. (2023) [34]	In vitro	Mammalian cells	Inflammation/apopt osis	Amino-modified NPs triggered NLRP3 inflammasome and cell apoptosis.
[7]	Impact of the food additive titanium dioxide (E171) on gut microbiota—host interaction	Pinget et al. (2019) [36]	In vivo	Mice	Titanium dioxide effect	E171 altered gut microbiota and host immunity.
[8]	Inhalation of ambient ultrafine particles induces metabolic dysfunction	Xu et al. (2022) [37]	In vivo	Mice	Ultrafine particle inhalation	Induced gluconeogenesis via FoxO1 and caused metabolic dysfunction.
[9]	Protective role of sesame oil against oxidative stress	Yousef & Hussien (2015) [38]	In vivo	Rats	Oxidative stress by NPs	Al2O3 NPs caused oxidative stress, hormonal imbalance; sesame oil had protective effect.
[10]	Silver nanoparticles aggravate metabolic disturbances in diabetic mice	Yin et al. (2021)[39]	In vivo	Diabetic mice	Silver NPs & metabolism	AgNPs worsened metabolic state via Akt inhibition and oxidative stress.



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Chronic Disease Risk Caused by Long Term Exposure to Plastics Derived Chemicals

EDCs can increase the risk of cardiovascular disease by adversely affecting blood pressure, cholesterol levels and vascular function. Furthermore, PFAS, a common component in food packaging, have been linked with impairment in the immune response leading to a relative immunocompromised state therefore increasing susceptibility to infections. These effects on the immune system can also trigger autoimmunity and development of autoimmune diseases. MPs exacerbate metabolic dysfunction by causing cellular damage through oxidative stress, which can hider effective management of T2DM. Long term exposure to MPs can cause a cumulative effect which can accelerate the progression of chronic conditions such as T2DM, cardiovascular disease, chronic kidney disease and neuropathy [24][26].

Plastics persist for decades in the environment, and their increasing use leads to a significant build up in ecosystems and food chains, adding to the ongoing pollution and contamination of the environment. This increasing concentration of plastics in our surroundings and the absence of strict control when it comes to food packaging, has worsened the amount of harmful chemicals that can migrate to food production.

For example, California's Proposition 65 requires businesses to provide warnings about significant exposure to chemicals that's causes cancer, birth defects, or reproductive harm, including phalates and bisphenols used in plastics [40]. Similarly, the European Union's REACH regulation (Registration, evaluation, authorization, and restriction of chemicals. Strictly limits the use of certain EDCs and mandates chemical disclosure for manufacturers. [40,41]

In the U.S, the FDA's food contact substance notification program provides only limited oversight of plastic additives, and gaps remain in regulating low-dose chronic exposures, particularly regarding substances like PFAS.[42]

Stricter regulatory control is urgently needed to reduce the use of dangerous polymeric compounds in food production and packaging. To lessen possible health risks, it is important that strict regulatory control is implemented to reduce polymeric compounds in food production and packaging and manufacturers should disclose the chemicals used in food packaging to provide clarity and open up to the possibility of substituting harmful components with more reasonable alternatives [23][25][26]. The use of plastics in food packaging must be subject to stronger regulations to protect public health. By taking these steps, we can lessen the threats of plastic exposure and its effects on our health and environment.

The involvement of governing bodies is essential to help control this plastic "epidemic". Rules and regulations limiting or banning the use of single-use plastics, such as straws, cutlery and bags can help minimize the amount of plastic that end up in landfills and oceans.

Many countries have already implemented such policies: the EU's Single Use Plastics Directive (2019) bans several plastic items and promotes extended producer responsibility. Similarly, Canada and several U.S. states (including New York and Oregon) have enacted bans on plastic bags and packaging materials [44,45].

Promoting biodegradable or reusable alternatives could drastically cut down on plastic waste and reduce the risk of harmful chemicals leaching into food [27].



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Industry Responsibilities and Interventions in Management of Plastic Waste

Programs for Extended Producer Responsibility would make producers answerable for the complete packaging lifetime, from manufacture to disposal. To actively encourage the use of sustainable materials and significantly reduce waste, businesses can be forced to oversee the collection and recycling of their plastic packaging. Manufacturers can significantly decrease plastic pollution and the associated health hazards by implementing "take-back" initiatives or deposit return schemes [27][28][29].

Education regarding plastic consumption is important in lowering the demand for plastic and encouraging healthy and environmentally friendly options. Raising awareness about the negative health effects of plastic, including its associations with T2DM, hormone imbalance, and other chronic illnesses, will empower consumers to make better purchasing decisions. [28][29].

.Germany's Green Dot system (Der Grüne Punkt) is one of the earliest and most influential examples of Extended Producer Responsibility (EPR) in waste management. Introduced in 1991 under the Packaging Ordinance (Verpackungsverordnung), the system mandates that producers and distributors are responsible for the take-back and recycling of packaging materials after consumer use. [45] By 2019, Germany recycled or composted more than 70% of its packaging waste, in large part due to EPR schemes like the Green Dot. [46] The success of the system has led to its adoption or adaptation in over 30 countries, making it a global benchmark for EPR programs.[47]

The United Nations Environment program (2023) outlines that effective plastic reduction strategies require an integrated approach involving EPR enforcement, national targets, circular economy frameworks, and broad-based awareness campaigns. In South Africa, for example, modelling scenarios suggest that combining EPR with awareness campaigns and infrastructure investments could reduce plastic leakage by 63% by 2040. [48]

A key barrier to green transitions is the initial cost burden borne by firms or consumers. For example, transitioning from single-use plastics to biodegradable alternatives or implementing EPR systems increases upfront production and compliance costs. Without policy intervention, these costs can deter participation by both producers and consumers, leading to market failure.[49]



Table 2: Cost-Benefit Trade-offs in Sustainable Plastic Management Upfront Costs

Investment in R&D			Developing bioplastics and alternative materials involves high R&D costs. [50]
Reconfiguring processes	pro	duction	Adapting equipment to use recycled/reusable materials requires capital.[51]
Compliance	with	EPR	Firms incur costs in managing post-consumer waste and
regulations			fulfilling legal obligations. [52]

Long-term benefits:

Brand value	and	Consumers increasingly prefer and support eco-conscious brands.
consumer trust		[53]
Avoided environmental		Reduces risks of pollution fines, cleanup costs, and future liabilities.
& regulatory costs		[54]
Market access		Sustainable practices ensure compliance with international market
		standards.[55]

Incentives such as tax credits, grants, and preferential procurement policies play a crucial role in accelerating the industry's transition to sustainable plastic management. For instance, the World Bank (2021) emphasizes that financial incentives can help offset the higher initial costs of adopting circular practices, particularly for small and medium-sized enterprises (SMEs). [56] Similarly, Deutsche Gesellschaft für Internationale Zusammenarbeit (GIZ) highlights how performance-based subsidies in Southeast Asia have encouraged investments in recycling infrastructure and waste segregation systems [57]. Additionally, the International Resource Panel (IRP) notes that well-structured incentive schemes can stimulate innovation in biodegradable materials and promote collaboration across supply chains. These incentives not only lower financial barriers but also signal long-term regulatory and market trends, making green investments more attractive and less risky for businesses. [58]

Behavioral changes and Consumer Awareness to Reduce Plastic Exposure

The customers would find it easier to recognize products with sustainable, safe packaging if labels were clear. Products with labels like "plastic-free," "biodegradable," or "BPA-free" might help to choose items that complement their environmental and health values. According to the Health Belief Model, increasing consumers awareness of the potential health risks associated with plastic exposure and the benefits of safer alternatives can motivate behavioral change.[59] Similarly, the Theory of Planned Behavior suggests that clear labeling and social encouragement can strengthen consumers intentions to choose environmentally friendly products by shaping their attitudes, perceived control, and awareness of social expectations.[60] In order to satisfy growing consumer demand, businesses would therefore be encouraged to make investments in safer, environmentally friendly packaging [28][29]. In addition to awareness campaigns, taking small steps to reduce plastic consumption by humans is important. This includes avoiding single used plastics such as straws, cutlery, and takeout containers, and instead opting for reusable bags, water bottles, and



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silverware. These seemingly small changes when adopted widely can make a substantial difference. [29].

Reducing overall plastic use also can be achieved by informing customers about the advantages of selecting goods with little to no plastic packaging. Plastic exposure can be considerably reduced by buying products in bulk or those that are packaged in metal, glass, or paper. The demand for sustainable companies may rise if excessive packaging is made more widely known [28]. Furthermore, to cut down on plastic waste, consumers must be informed about appropriate recycling procedures. Customers should be aware of the kinds of plastic that are recyclable in their community and given clear local recycling requirements. Encouraging appropriate recycling practices guarantees that plastics are recycled instead of harming the environment or ending up in landfills [28]. The harm that plastic packaging poses to human health and the environment can be greatly decreased by combining legislative action with consumer education and personal accountability.

Future Directions and Knowledge Gaps in Plastic Exposure and Metabolic Health

The negative health impacts of plastics and their potential to increase T2DM risk are a major concern, as highlighted by the findings of this study. Despite emerging evidence linking plastic exposure—particularly through microplastics (MPs) in food—to metabolic disruption, critical knowledge gaps and research limitations persist. Notably, there is a scarcity of long-term prospective human studies that can directly establish a causal link between chronic plastic exposure and the onset of T2DM [30]. Much of the current evidence is derived from cross-sectional studies or animal models, which limit the ability to draw definitive conclusions in human populations.

Furthermore, the heterogeneity in food types, processing methods, and packaging materials complicates the assessment of exposure levels and outcomes, making it difficult to develop standardized risk models [31]. This variability contributes to the ongoing uncertainty in quantifying the real-world impact of plastic-related EDCs on metabolic health. Additionally, current research often does not adequately address the interactions between plastic exposure and other environmental or behavioral risk factors for T2DM, such as diet quality, socioeconomic status, or physical activity.

Beyond scientific challenges, regulatory inertia and industry lobbying also play a substantial role in slowing progress. Despite growing scientific consensus on the potential harms of EDCs and microplastics, regulatory bodies have been slow to implement stricter limits or enforce bans, often citing insufficient "causal" evidence or relying on outdated safety thresholds. Economic incentives—such as the low cost, convenience, and profitability of plastic production—further delay the transition to safer alternatives. In some cases, chemical and plastic industry lobbying has influenced policymaking by challenging proposed regulations or promoting doubt about the risks of certain compounds. This resistance limits the implementation of effective public health measures and hinders funding for independent long-term research.

To address these issues, future research should prioritize standardized exposure assessment tools, prospective human cohort studies, and mechanistic investigations into how micro- and nanoplastics disrupt metabolic regulation. Moreover, there is a growing need to evaluate not only



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the risks associated with plastics but also the safety and feasibility of alternative materials used in food packaging. Encouragingly, shifting public awareness and policy pressure may create momentum for more proactive regulation and investment in safer technologies [30][31].

CONCLUSION AND RECOMMENDATIONS

The excessive reliance on plastics in food processing and packaging demonstrated significant dangers on public health, particularly through the contamination of food with MPs and the associated EDCs. These compounds disrupt metabolic pathways, contribute to insulin resistance, and increase the risk of chronic conditions such as T2DM. Despite the presence of these significant risks today, current regulations and policies regarding plastic use remain insufficient.

To overcome this burden, a multifaceted approach must be put into action as soon as possible. This includes not only making law for mandatory disclosure of plastic additives in food packaging but also taxing new plastic production to encourage using recycled or biodegradable materials. Harmful plastic particles are present in varying concentrations in the sea, air, and significantly in the food we consume. It is readily apparent that the vast majority of food items today are wrapped in plastic; water, juice, and dairy products are also bottled in plastic. Previously, vegetables and fish might have been considered safer alternatives; however, plastic chemicals have now permeated all aspects of our food chain. Plastic derived chemicals are spreading in oceans and being deposited into marine animals. Moreover, they appear in soil due to the heavy reliance on plastic in farming. Current guidelines and regulations are inadequate to limit or halt this pervasive contamination. This highlights the need for a 'One Health' approach, which sees the strong connection between a healthy environment and healthy people. While billions of dollars are invested in research to prevent and treat T2DM and manage its complications, very little of this money goes to making sure our food is healthy and clean by dealing with plastic pollution. Increasing awareness among health policy makers and the general population may be promising in reducing these burdens. Enforcing strict regulations on food production companies and providing incentives can limit the pollution of food with plastic. We encourage the use of alternative biodegradable materials in food production to overcome this public health issue. Addressing the issue of plastic contamination in our food systems demands a coordinated, multistakeholder approach. A successful approach should involve governments, industry leaders, researchers, public health agencies, environmental organizations, and consumers. Policymakers must collaborate with scientific communities to develop evidence-based regulations. Moreover, industries should be held accountable for transparency in their material use and encouraged to invest in sustainable alternatives. Public health institutions and educators can play a crucial role in raising awareness and promoting behavior change. knowledgeable consumers can drive demand for safer food packaging practices. Through this collective effort we can create a resilient food system that protects human health and preserves environmental integrity. Future research should investigate the long-term health effects of plastic in more detail and suggest innovative methods to break the contamination chain. For example, better filters for food, safer packaging from plants, and natural ways to clean up polluted places.



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