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Editorial

Plasticizers as emerging threats to human, animal and environmental health

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Plasticizers have become indispensable components of modern plastics because they improve flexibility, durability, and processing efficiency. Global plastic production now exceeds 400 million tonnes annually and continues to increase with industrialization and consumer demand (Geyer *et al*., 2017; UNEP, 2023). Among the most widely used plasticizers are bisphenol A (BPA) and phthalates, including di-(2-ethylhexyl) phthalate (DEHP), dibutyl phthalate (DBP), diethyl phthalate (DEP), diisobutyl phthalate (DIBP), benzyl butyl phthalate (BBP), and diisononyl phthalate (DINP). Because these compounds are physically incorporated rather than chemically bound to plastic polymers, they gradually migrate into food, water, soil, air, and living organisms throughout the product life cycle, from manufacturing to disposal (Net *et al*., 2015; Rochester and Bolden, 2015). Their widespread environmental occurrence has raised growing concerns regarding their implications for human, animal, and ecosystem health.

Plasticizer exposure has become nearly unavoidable. BPA and phthalates have been detected in drinking water, packaged foods, seafood, household dust, indoor air, cosmetics, medical devices, and agricultural soils. Biomonitoring studies have identified these chemicals and their metabolites in urine, blood, breast milk, placental tissue, amniotic fluid, and umbilical cord blood, indicating that exposure begins during fetal development and continues throughout life (Diamanti-Kandarakis *et al*., 2009; Gore *et al*., 2015; WHO, 2022; Liu *et al*., 2026). Because embryonic development, infancy, puberty, and pregnancy represent critical periods of hormonal regulation, even low-level exposure during these stages may produce lasting biological effects.

Plasticizers are recognized as important endocrine-disrupting chemicals (EDCs) capable of interfering with normal hormonal signaling. BPA structurally resembles estradiol and can bind to estrogen receptors while also interacting with androgen, thyroid hormone, glucocorticoid, and peroxisome proliferator-activated receptors. In contrast, phthalates primarily suppress steroidogenic enzymes involved in testosterone synthesis, resulting in impaired reproductive development and fertility (Zoeller *et al*., 2012; Gore *et al*., 2015). Since endocrine regulation governs growth, metabolism, immunity, neurological development, and reproduction, disruption of these pathways can affect multiple organ systems simultaneously.

Growing evidence indicates that the biological effects of plasticizers extend far beyond endocrine disruption. BPA and phthalates promote oxidative stress by increasing the production of reactive oxygen species while weakening endogenous antioxidant defenses, ultimately damaging lipids, proteins, DNA, and cellular membranes. Persistent oxidative stress contributes to mitochondrial dysfunction, apoptosis, fibrosis, and chronic inflammation. Plasticizers also activate inflammatory signaling pathways, including NF-κB, MAPK, and the NLRP3 inflammasome, leading to increased production of pro-inflammatory cytokines and tissue injury (Kahn

et al., 2020; Liu *et al*., 2026). These interconnected mechanisms help explain the diverse health effects associated with long-term exposure.

Recent studies have further demonstrated that plasticizers alter epigenetic regulation through changes in DNA methylation, histone modification, and microRNA expression, potentially producing long-lasting or even transgenerational effects (Akanbi *et al*., 2025). In addition, disruption of the intestinal microbiome has emerged as another important mechanism. BPA and phthalates reduce microbial diversity, increase intestinal permeability, and promote systemic inflammation, thereby contributing to obesity, diabetes, and non-alcoholic fatty liver disease (Dalamaga *et al*., 2024; Chi *et al*., 2024; Luque *et al*., 2025). These analyses suggested the complexity of plasticizer toxicity and emphasize that multiple biological pathways act together to promote disease.

Human epidemiological studies consistently associate plasticizer exposure with adverse health outcomes. BPA and phthalates have been linked to infertility, reduced semen quality, altered ovarian function, obesity, diabetes, cardiovascular disease, thyroid dysfunction, immune dysregulation, liver and kidney injury, and several hormone-dependent cancers. Prenatal exposure has also been associated with reduced birth weight, impaired neurodevelopment, behavioral disorders, attention deficit hyperactivity disorder (ADHD), and autism spectrum disorders, although the strength of evidence varies among outcomes (Rochester, 2013; Heindel *et al*., 2017; Trasande *et al*., 2024). Experimental studies further support these associations by demonstrating impaired spermatogenesis, ovarian dysfunction, hepatic steatosis, renal injury, and neuronal damage accompanied by alterations in oxidative stress, inflammatory mediators, and gene expression (Sujan *et al*., 2020; Mustari *et al*., 2023; Anjir *et al*., 2025; Puvvula *et al*., 2025).

The consequences of plasticizer contamination extend beyond human health. Domestic animals, wildlife, aquatic organisms, and livestock are continuously exposed through contaminated feed, drinking water, soil, and environmental dust. Chronic exposure in livestock has been associated with impaired growth, hormonal imbalance, oxidative stress, organ dysfunction, and reduced reproductive performance. Recent studies in Black Bengal goats demonstrated significant alterations in growth, hematological and biochemical parameters, hormonal profiles, semen quality, and reproductive development following environmentally relevant phthalate exposure, highlighting potential impacts on livestock productivity and food security (Hasan *et al*., 2024a; Hasan *et al*., 2024b). These studies reinforce the need to address plasticizer pollution as a One Health challenge linking human, animal, and environmental well-being.

Plasticizer contamination has become a global public health concern because exposure occurs continuously through multiple environmental pathways. Biomonitoring studies have detected BPA and phthalate metabolites in the urine of more than 90% of examined populations, reflecting widespread exposure through food, drinking water, air, consumer products, and occupational settings. Although epidemiological studies cannot always establish direct causality, consistent associations have been reported between chronic plasticizer exposure and reproductive disorders, obesity, insulin resistance, type 2 diabetes, cardiovascular disease, thyroid dysfunction, and impaired liver and kidney function (Rochester, 2013; Heindel *et al*., 2017; Trasande *et al*., 2024). Early-life exposure is particularly concerning because fetal and childhood development depends on precisely regulated hormonal signaling. Prenatal exposure has been associated with reduced birth weight, impaired neurodevelopment, behavioral disorders, attention deficit hyperactivity disorder, and autism spectrum disorders. Experimental evidence further indicates that BPA crosses both the placental and blood–brain barriers, where it may alter neuronal development, synaptic plasticity, and neurotransmitter signaling through oxidative stress and dysregulation of the AMPK, HO-1, and Nrf2/Keap1 pathways (Figueirôa *et al*., 2025).

The impact of plasticizers extends well beyond human populations. Livestock, companion animals, wildlife, and aquatic organisms are continuously exposed through contaminated feed, forage, irrigation water, soil, and environmental dust. Such exposure threatens animal welfare, agricultural productivity, biodiversity, and food safety. Recent studies have demonstrated that chronic exposure to environmentally relevant phthalate mixtures in Black Bengal goats significantly altered growth performance, hematological and biochemical indices, electrolyte balance, hormonal profiles, and reproductive function. Male goats exhibited reduced testosterone concentrations, impaired semen quality, and abnormal reproductive organ development, whereas prenatal exposure adversely affected reproductive physiology in female offspring (Hasan *et al*., 2024a; Hasan *et al*., 2024b). Similar findings from laboratory animal studies have demonstrated hepatic injury, renal dysfunction, oxidative stress, reproductive toxicity, and endocrine disruption following BPA and phthalate exposure, reinforcing concerns that environmental contamination may compromise livestock productivity and ecosystem health.

Because complete elimination of plasticizer exposure is unrealistic, reducing health risks requires coordinated action involving governments, industries, researchers, healthcare professionals, veterinarians, farmers, and

consumers. Regulatory agencies should strengthen restrictions on high-risk plasticizers in food packaging, children's products, medical devices, and other consumer goods while harmonizing international safety standards. Importantly, future regulations should adopt a class-based approach that evaluates groups of structurally related plasticizers rather than regulating individual compounds, thereby reducing the likelihood of replacing one hazardous chemical with another of similar toxicity. Routine biomonitoring of plasticizers in food, drinking water, livestock feed, and environmental samples should also become an integral component of national food safety and environmental surveillance programs.

The development of safer alternatives represents another important priority. Advances in green chemistry provide opportunities to design biodegradable plasticizers with minimal endocrine-disrupting activity and reduced environmental persistence. However, new additives should undergo comprehensive pre-market toxicological evaluation, including endocrine, reproductive, developmental, and multigenerational toxicity testing, before commercial application. Such precautionary approaches can support industrial innovation while minimizing unintended health and environmental consequences.

Recent research has also highlighted promising supportive strategies to reduce plasticizer-induced toxicity. Plant-derived bioactive compounds with antioxidant and anti-inflammatory properties, including black seed oil, thymoquinone, and combined zinc and folic acid supplementation, have demonstrated protective effects against BPA- and phthalate-induced reproductive, hepatic, and renal damage in experimental animals by restoring antioxidant defenses, improving hormonal balance, and reducing tissue injury (Sujan *et al*., 2020; Mustari *et al*., 2022; Anjir *et al*., 2025). Although these findings are encouraging, well-designed clinical trials and livestock field studies remain necessary before such interventions can be recommended for routine use.

Future investigations should focus on several emerging priorities. Greater emphasis is needed on the combined toxicity of plasticizers with microplastics, pesticides, heavy metals, and other environmental contaminants because real-world exposure rarely involves a single chemical. Long-term epidemiological studies are required to clarify causal relationships between chronic low-dose exposure and disease. Integrating genomics, transcriptomics, proteomics, metabolomics, and epigenomics may facilitate the identification of early biomarkers of exposure and toxicity. In addition, further research should examine interactions between plasticizers, the gut microbiome, immune regulation, livestock health, and wildlife conservation to better understand the broader ecological consequences of plastic pollution.

Plasticizers have emerged as pervasive environmental contaminants with far-reaching consequences for human health, animal welfare, and ecosystem sustainability. Evidence from epidemiological, experimental, and mechanistic studies demonstrates that BPA, phthalates, and related compounds disrupt endocrine function, induce oxidative stress and inflammation, alter epigenetic regulation, and impair reproduction, metabolism, neurological development, and immune function. Addressing this growing challenge requires stronger regulatory policies, safer chemical alternatives, continued scientific innovation, and international collaboration. Integrating environmental protection with human and veterinary health under a One Health framework will be essential for reducing plasticizer exposure, safeguarding food systems, and protecting present and future generations.

Ethical approval and informed consent

Not applicable.

Data availability

Not applicable.

Conflict of interest

None to declare.

Author's contribution

Conceptualization, formal analysis, writing-original draft preparation, review and editing: Mohammad Alam Miah. The author has read and approved the final version of the published editorial.

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