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Review Article

Molecular Disruptive Potential of Phthalate Esters: Threats and Dynamics in Aquatic Ecosystem Resources

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Abstract

Phthalate esters are widely used plasticizers that enter aquatic ecosystems through industrial, agricultural, and domestic waste, posing risks to water quality and aquatic organisms. Their persistence and ability to disrupt molecular, endocrine, and physiological processes highlight the need to understand their environmental dynamics and ecological impacts. This review examined the molecular disruptive potential of phthalate esters in aquatic ecosystem resources by elucidating its sources, occurrence, exposure pathways, bioaccumulation, molecular disruptive mechanisms, and ecological impacts of phthalate esters (PAEs) in aquatic ecosystems. Information was synthesized from scientific literature, including journal articles, reports, and databases. Findings revealed that PAEs are ubiquitous contaminants originating from wastewater discharge, landfill leachates, agricultural runoff, and plastic degradation. Their persistence, hydrophobicity, and bioaccumulative properties facilitate uptake by aquatic organisms, causing oxidative stress, endocrine disruption, neurotoxicity, immunotoxicity, metabolic disorders, and transgenerational effects. Although biomagnification is generally limited, continuous exposure poses significant ecological risks. Interactions with microplastics and other environmental stressors may further enhance toxicity and ecosystem impacts. The review highlights important knowledge gaps regarding chronic exposure, mixture toxicity, and long-term ecological consequences. Overall, PAEs threaten aquatic biodiversity, ecosystem stability, and potentially human health, emphasizing the need for stricter regulations, improved waste and wastewater management, continuous environmental monitoring, safer alternatives to phthalate-based products, and further research to support effective risk assessment and pollution mitigation.

Key words: Phthalate esters, aquatic ecosystems, endocrine disruption, ecotoxicology, bioaccumulation, oxidative stress, environmental pollution

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INTRODUCTION

The rapid growth of plastic production has increased the release of phthalate esters (PAEs) into aquatic systems, where they act as persistent pollutants and endocrine disruptors. Widely used in PVC, packaging and personal care products, their production is projected to approach 500 million tons by 2050¹. As a result, PAEs are now ubiquitous in aquatic environments, where chronic low-dose exposure affects aquatic organisms¹. Their fate is controlled by hydrolysis, photolysis and microbial degradation, while uptake occurs mainly through ingestion, leading to bioaccumulation but limited biomagnification¹. High lipophilicity and low volatility enhance their persistence, with compounds such as DBP and DEHP frequently exceeding safety thresholds².

Fish provide a key pathway for human exposure, linking aquatic contamination to health risks. Toxic effects observed in aquatic organisms, particularly endocrine and metabolic disruption, mirror pathways associated with human disorders¹. In natural systems, PAEs interact with stressors such as temperature changes, hypoxia, pharmaceuticals and microplastics, producing complex effects that can intensify toxicity². Although phthalate esters are widely recognized as aquatic contaminants, knowledge of their molecular toxicity pathways and ecosystem-level consequences remains limited. Moreover, integrated studies linking endocrine disruption, oxidative stress, bioaccumulation and biodiversity loss under realistic environmental conditions are insufficient, particularly in developing regions. There exist available evidence indicates that phthalate esters have profound molecular impacts, including the modulation of gene expression, induction of oxidative stress, DNA damage and endocrine disruption. These findings are consistent with numerous studies highlighting the toxicological significance of phthalate ester exposure across aquatic organisms³.

Despite the widespread occurrence of phthalate esters (PAEs) in aquatic environments and growing evidence of their toxicity, significant knowledge gaps remain regarding their molecular mechanisms of action, long-term ecological consequences and interactions with other environmental stressors such as microplastics, temperature fluctuations, hypoxia and heavy metals. Most existing studies focus on individual species under controlled laboratory conditions, with limited integration of oxidative stress, endocrine disruption, neurotoxicity, bioaccumulation, trophic transfer and ecosystem-level responses. Furthermore, information from developing regions, where monitoring and regulatory frameworks are often inadequate, remains scarce. These limitations hinder accurate ecological risk assessment and the

development of effective mitigation strategies. Addressing this topic is therefore essential to improve understanding of the pathways through which PAEs affect aquatic biodiversity, ecosystem stability and human health through contaminated aquatic resources³. Accordingly, this review aims to critically examine the sources, environmental occurrence, exposure pathways, molecular disruptive mechanisms, bioaccumulation patterns and ecological impacts of phthalate esters in aquatic ecosystems, while highlighting their implications for aquatic organisms and human health and identifying priorities for monitoring, risk assessment and pollution mitigation³.

Types of phthalate esters: Phthalate esters (PAEs) are diesters of 1,2-benzenedicarboxylic acid, with the ortho-isomer being the most commercially important. Common examples include dimethyl phthalate (DMP), diethyl phthalate (DEP), di-isobutyl phthalate (DIBP), dibutyl phthalate (DnBP), butyl benzyl phthalate (BBP) and di(2-Ethylhexyl) phthalate (DEHP). They are widely used as plasticizers in polyvinyl chloride (PVC) and other polymers to improve flexibility and durability and are also present in products such as cosmetics, adhesives, lubricants and packaging materials. PAEs are classified into low- and high-molecular-weight groups. Low-molecular-weight PAEs (e.g., DMP, DEP, DnBP) are mainly used as solvents and additives in personal care products and coatings, while high-molecular-weight PAEs, especially DEHP, are primarily used in PVC materials. Because they are not chemically bound to polymers, PAEs can leach into the environment during use and disposal, contributing to their persistence and endocrine-disrupting potential³.

Environmental release occurs through industrial activities, product usage and material degradation, leading to widespread contamination. Frequently detected compounds such as DEP, DBP and DEHP are common in both consumer products and environmental samples. Their low volatility, hydrophobicity and strong affinity for organic matter promote persistence and bioavailability, highlighting the need for continued monitoring and risk assessment, particularly in urban and industrial regions⁴.

Physical, chemical and environmental properties of phthalates: Phthalate esters (PAEs) are produced by esterification of phthalic anhydride with alcohols and their alkyl chain length strongly influences molecular weight and physicochemical behavior⁵. Structure variations determine properties such as viscosity and lipophilicity, which increase with chain length and enhance affinity for organic phases and biological tissues. PAEs are generally colorless, low-volatility, oily liquids at room temperature⁶. They are grouped

into low-molecular-weight (3-6 carbons), such as DMP, DEP, DnBP and DiBP, commonly used in cosmetics, pharmaceuticals, coatings and packaging and high-molecular-weight (7-13 carbons), such as BBzP, DEHP, DiNP and DiDP, mainly applied as plasticizers in PVC products⁴. Their physicochemical traits, including hydrophobicity, low volatility and stability, support widespread industrial use, with some formulations containing up to 40% phthalates. However, because they are not covalently bound to polymers, PAEs readily leach, volatilize and diffuse into the environment, where their fate is governed by solubility, vapor pressure and partitioning behavior. Consequently, they are now ubiquitous environmental contaminants, with persistence driven by high production volumes, poor waste management and incomplete wastewater removal, raising growing concerns for aquatic ecosystems and public health.

GENERAL STRUCTURE OF PHTHALATE ESTERS (PAEs)

Figure 1 illustrates the general chemical structure of phthalate esters (PAEs), which are diesters of phthalic acid. The structure consists of a benzene ring bonded to two ester functional groups (-COOR and -COOR) located at adjacent (ortho) positions⁴. The substituent groups R and R represent alkyl or aryl chains of varying lengths and configurations, which determine the physicochemical properties and toxicity of individual phthalate compounds. Common examples include dimethyl phthalate (DMP), diethyl phthalate (DEP), dibutyl phthalate (DBP) and di(2-Ethylhexyl) phthalate (DEHP). The hydrophobic nature of these side chains contributes to the persistence, bioaccumulation and environmental mobility of PAEs in aquatic ecosystems⁵. Due to their widespread industrial applications and endocrine-disrupting properties, phthalate esters are recognized as important environmental contaminants of global concern (Fig. 1).

Phthalate esters as emerging aquatic contaminants:

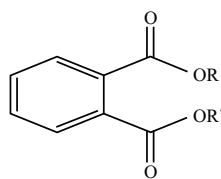
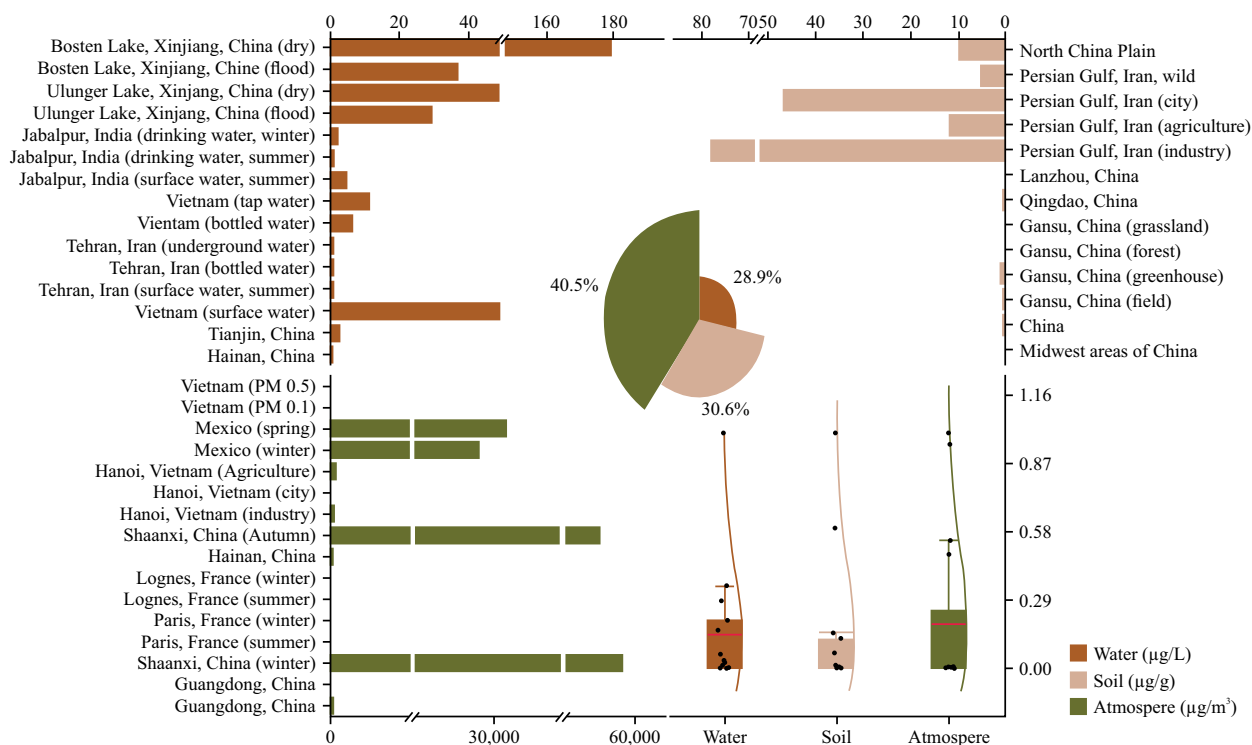
Phthalate esters (PAEs) originate primarily from consumer products and solid waste combustion, with weak polymer binding enabling their release into the environment. Consequently, PAEs are widely distributed across air, soil and aquatic systems, with major inputs from industrial and domestic wastewater, agricultural runoff and landfill leachates. Reported concentrations in aquatic environments range from 0.1×10^3 to 3.2×10^3 $\mu\text{g/L}$ ⁷. Commonly detected compounds include dimethyl phthalate (DMP), diethyl phthalate (DEP), dibutyl phthalate (DBP), benzyl butyl phthalate (BBP), dioctyl phthalate (DOP) and particularly di(2-Ethylhexyl) phthalate (DEHP), which is often dominant⁸. PAE concentrations exhibit

notable seasonal variability, typically increasing during colder periods due to reduced photodegradation and microbial activity. For example, surface waters in Jabalpur, India, showed concentrations rising from 410.48 $\mu\text{g/L}$ in summer to 7553.9 $\mu\text{g/L}$ in winter⁸. As natural water bodies serve as sources of drinking water, such contamination represents a direct pathway for human exposure.

The distribution of PAEs in water systems is influenced by environmental conditions and infrastructure-related factors, including pipeline materials, residence time and storage practices⁹. Although conventional treatment processes reduce concentrations, complete removal is rarely achieved due to the persistence and chemical stability of PAEs⁹. In aquatic organisms, PAEs disrupt key biological functions, including growth, reproduction, metabolism and immunity. In primary producers, they inhibit growth through oxidative stress and reduced pigment content¹⁰. In invertebrates such as *Daphnia magna*, exposure impairs lipid metabolism and antioxidant capacity. In fish, PAEs induce oxidative damage and endocrine disruption, affecting reproduction and gene expression, as observed in species such as *Cyprinus carpio* and *Danio rerio*. These effects can scale to ecosystem-level impacts, including reduced population stability and altered trophic dynamics¹⁰. Molecular alterations may subsequently affect growth, reproduction, immune competence and survival of fish and other aquatic organisms. Furthermore, deteriorating water quality may increase the bioavailability and toxicity of environmental contaminants, including phthalate esters and heavy metals, thereby exacerbating molecular damage through mechanisms such as DNA strand breaks, lipid peroxidation, protein oxidation, endocrine disruption and mitochondrial dysfunction¹¹. Consequently, the study provides valuable baseline information for assessing how environmental conditions modulate contaminant-induced molecular responses in aquatic ecosystems. By establishing the status of key water quality parameters, the research contributes to predicting ecological risks, identifying vulnerable habitats and supporting effective water resource management strategies aimed at minimizing molecular toxicity and preserving the health, biodiversity and sustainability of aquatic resources under increasing anthropogenic pressure¹².

GLOBAL DISTRIBUTION OF PHTHALATE ESTERS IN ENVIRONMENTAL COMPARTMENTS

Figure 2 presents the concentrations and distribution patterns of phthalate esters (PAEs) in three major environmental compartments: Water, soil and atmosphere

Fig. 1: General chemical structure of phthalate esters⁶Fig. 2: Concentration and percentage of PAEs in the water, soil and atmospheric environments Worldwide¹²

across different geographical locations. The pie chart indicates that atmospheric samples account for the highest proportion of reported PAE contamination (40.5%), followed by soil (30.6%) and water (28.9%). Concentration ranges vary considerably among regions, reflecting differences in industrialization, urbanization, waste management practices and environmental conditions. Notably, elevated PAE levels were reported in atmospheric samples from China and Mexico, while significant contamination was also observed in soils and aquatic environments in China, Iran, India, Vietnam and France. The figure highlights the ubiquitous occurrence of PAEs and their widespread dispersion among environmental media, demonstrating their persistence, mobility and potential for ecological and human exposure. These findings underscore the global environmental concern associated with phthalate pollution and the need for effective monitoring and mitigation strategies.

Sources and environmental occurrence of phthalate esters:

Phthalate esters (PAEs) are ubiquitous contaminants found in air, water, soil and sediments, with di(2-Ethylhexyl) phthalate (DEHP) classified as a priority pollutant due to its persistence and toxicity¹². Regulatory limits such as 1.3 $\mu\text{g/L}$ in marine waters and 0.006 mg/L in drinking water reflect concern over their risks¹³. Their widespread distribution is mainly driven by industrial use and weak binding to polymer matrices, enabling continuous release through leaching, volatilization and degradation. Additional inputs arise from urban and industrial activities, including plastics, household products, indoor dust and atmospheric transport via particulate matter. In aquatic systems, major sources include industrial and municipal effluents, agricultural runoff, atmospheric deposition and wastewater treatment plants, where DEHP concentrations of 9-44 $\mu\text{g/L}$ have been reported with incomplete removal, leading to discharge into receiving waters and accumulation

in sediments and biota. Environmental levels vary widely across compartments, ranging from ng/m³ in air to ng-µg/L in water and ng-µg/g in soil, with DEHP often dominant in freshwater, marine and wetland systems. Due to high hydrophobicity, sediments act as major sinks, with concentrations reaching up to 100 µg/g, especially in low-flow systems like reservoirs that enhance pollutant retention¹⁴.

Microplastics: Human society is increasingly described as living in a “plastic-dominated era”, reflecting the massive global production and accumulation of plastics¹⁵. By 2015, about 8.3 billion metric tons had been produced, with 6.3 billion tons becoming waste, much of which has entered the environment due to poor management. Although plastics are resistant to degradation, they fragment into microplastics through mechanical, chemical and photodegradation processes, making microplastic pollution a major global concern comparable to climate change and ocean acidification¹⁶. These particles induce physical and physiological stress in aquatic organisms, including reduced reproduction, energy depletion, oxidative stress and metabolic disruption and also act as carriers of hydrophobic pollutants, enhancing contaminant transfer and toxicity. For instance, microplastics associated with PAHs can cause biochemical and physiological damage in marine mussels, while degradation products and additives intensify combined chemical pollution¹⁷. Phthalate esters (PAEs), widely used plasticizers, frequently co-occur with microplastics across environmental compartments, increasing the risk of combined exposure. However, their interaction and joint toxicity remain poorly understood. Microplastics have been detected in freshwater, marine systems, wastewater, soils and even food and drinking water, confirming their entry into food webs and human exposure pathways. They accumulate mainly in the gastrointestinal tract of organisms such as zooplankton, fish, earthworms and mammals, where they can disrupt gut microbiota and metabolism. Despite this, combined effects of microplastics and PAEs on gut health and microbial ecology remain insufficiently studied, highlighting a key gap in environmental toxicology¹⁸.

Wastewater discharge: The widespread use of phthalate esters (PAEs) has improved the flexibility and performance of many consumer and industrial products, supporting modern living standards¹⁸. However, because they are not chemically bound to polymers, PAEs readily leach into the environment and are now widely detected in water, soil, sediment and air¹⁹. Many compounds exhibit estrogenic activity, leading to endocrine disruption in exposed organisms, while some may

also interact with hemoglobin and affect blood physiology. China alone accounts for over 25% of global PAE consumption, with commonly detected compounds including DMP, DEP, DBP and DEHP²⁰. High groundwater concentrations have also been reported, with DEP and DBP reaching 147 µg/L and 50 µg/L, respectively²⁰. The PAEs enter sewage systems through industrial production, product leaching, household and personal care products, atmospheric deposition and infrastructure-related sources. Although wastewater treatment plants using activated sludge can partially reduce their levels via adsorption and microbial degradation, complete removal remains limited²¹.

Landfill leachate: Landfills are not inert disposal sites but long-term chemically and biologically active systems that generate landfill leachate when rainfall percolates through waste, mobilizing contaminants. Among these, phthalate esters (PAEs) are of growing concern due to their persistence, toxicity and continuous release from degrading plastic waste, with leachate acting as a major transport pathway. As a result, landfills function as both reservoirs and point sources of PAEs, especially in areas with high plastic input²². Reported concentrations in leachate (114-303 µg/L) for compounds such as DEHP, DBP and BBP are far higher than in natural waters, identifying landfills as strong contamination hotspots. PAE behavior in landfills is influenced by waste composition, landfill age, temperature and microbial activity, with interactions involving microplastics and organic matter further enhancing sorption-desorption dynamics and reinforcing landfills as active transformation zones. Once released, PAEs can migrate into soils, groundwater and surface waters, particularly where containment systems are inadequate, raising concerns due to their endocrine-disrupting potential even at low concentrations²³. A major limitation is the incomplete removal of PAEs during treatment, as biological and advanced oxidation processes often fail to achieve full mineralization and may generate toxic intermediates²⁴.

Agricultural runoff: Phthalic acid esters (PAEs) in agricultural soils are increasingly concerning due to their persistence, widespread distribution and risks to environmental and human health. Their levels vary depending on industrial proximity, soil management and use of plastic-based agricultural inputs. A major source is plastic mulching films made of PVC and PE, which contain plasticizers such as DEHP and DBP that leach into soils under UV radiation, temperature changes and microbial activity, with accumulation increasing where disposal and recycling are inadequate²⁵. Another key pathway is treated wastewater and sewage sludge, which

introduce residual PAEs from domestic, industrial and plastic waste sources. Common compounds include DEHP, DBP and DnOP, with concentrations influenced by application rates and treatment efficiency²⁶. Additional inputs arise from agrochemicals, where DEP and DMP may occur as impurities or additives, leading to gradual buildup in intensively farmed soils. Atmospheric deposition from industrial emissions, vehicle exhaust and plastic burning further contributes to widespread contamination, including in remote rural areas. Agricultural plastics such as irrigation pipes, greenhouse films and storage materials also release DEHP and DBP under heat and UV exposure. Together, these pathways drive PAE accumulation, raising concerns about persistence, bioaccumulation and transfer into crops, with especially high levels reported in intensively cultivated regions such as China²⁷. Continuous monitoring is therefore essential for protecting soil quality and food safety. Phthalate esters may have long term effects on all aquatic organisms. Indiscriminate usage and handling of DEP in areas close to fish farms and aquatic environment should be discouraged. It is recommended that additional research should be done on how the fish's blood and tissue react biochemically and genetically to DEP intoxication²⁸. Phthalates generally have the capacity to disrupt vital metabolic and homeostatic processes, corroborating its status as persistent environmental pollutants with ecotoxicological relevance²⁹.

SOURCES AND PATHWAYS OF PHTHALATE ESTER (PAEs) CONTAMINATION IN AGRICULTURAL SOILS

Figure 3 illustrates the major sources and environmental pathways through which phthalate esters (PAEs) enter and accumulate in agricultural soils. Atmospheric deposition from industrial emissions, wastewater irrigation, application of organic fertilizers, pesticide usage and plastic mulching or greenhouse membranes are identified as key contributors. PAEs released from these sources infiltrate the soil profile and migrate into deeper layers through leaching and transport processes. Due to their hydrophobic nature and persistence, PAEs can adsorb onto soil particles, accumulate over time and potentially be taken up by crops, thereby entering terrestrial food chains. The figure highlights the complex interactions between anthropogenic activities and environmental processes that govern the distribution, persistence and ecological risks of PAEs in agricultural ecosystems. Understanding these contamination pathways is essential for developing effective strategies to control PAEs pollution and protect soil quality, food safety and environmental health²⁹.

Molecular disruptive mechanisms of phthalate esters on aquatic organisms: Phthalate esters (PAEs) have been shown to disrupt community stability and population dynamics in aquatic ecosystems by impairing growth, development, reproduction and metabolic processes in exposed organisms³¹. These organism-level effects cascade across ecological scales, ultimately altering ecosystem structure and function. A key impact is the inhibition of photosynthesis in algae and phytoplankton, leading to reduced primary productivity and limiting the energy base available to higher trophic levels. Since primary production underpins aquatic food webs, such reductions impair energy transfer efficiency across trophic levels³¹. Consequently, declines in zooplankton populations, combined with PAE-driven bioaccumulation, can reduce energy flow to higher consumers and decrease top predator biomass, weakening overall food-web stability³¹. Additionally, reduced abundance of benthic organisms may weaken their regulatory control over phytoplankton, increasing the risk of algal blooms and further ecosystem imbalance³¹.

The PAEs also interfere with reproductive processes in fish, resulting in reduced fertility, impaired reproductive success and potential transgenerational effects. These disruptions can compromise recruitment and threaten aquatic biodiversity, thereby increasing pressure on fisheries resources³². Through combined effects at individual, population and community levels, PAEs reduce nutrient cycling efficiency, disrupt energy transfer and impair key ecological functions such as biological control and population regulation³². Despite growing evidence of these impacts, most studies remain focused on organism-level mechanisms, with limited long-term field data at population and ecosystem scales. To improve ecological risk assessment, future research should adopt multi-scale approaches that integrate ecosystem modeling with species interaction networks, enabling a more comprehensive understanding of how PAEs affect aquatic ecosystem stability over time³².

Oxidative stress: In aquatic organisms, phthalate esters (PAEs) induce multiple toxic effects, including oxidative stress, metabolic disruption, endocrine interference, neurotoxicity, immunotoxicity and transgenerational impacts. A central mechanism underlying these effects is oxidative stress, characterized by excessive reactive oxygen species (ROS) production and weakened antioxidant defenses across trophic levels. In primary producers, PAEs rapidly disrupt cellular redox balance. The marine diatom *Phaeodactylum tricornutum* exposed to dimethyl phthalate (DMP) and diethyl phthalate (DEP) (20-200 µg/L) showed elevated ROS and

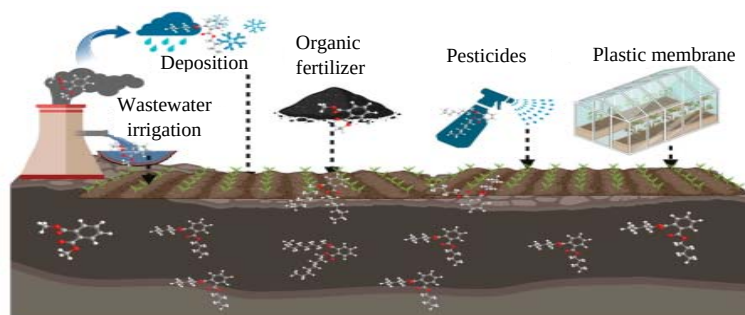


Fig. 3: Sources of PAEs in the soil³⁰

malondialdehyde (MDA), indicating lipid peroxidation+n. Comparable responses occur in lower trophic consumers; for instance, the rotifer *Brachionus plicatilis* exposed to benzyl butyl phthalate (BBP) showed dose-dependent oxidative stress and elevated antioxidant enzyme activity³². In fish, oxidative injury is consistently observed across species. Acute exposure to DEHP in goldfish (*Carassius auratus*) induces ROS accumulation, antioxidant imbalance and metabolic disruption linked to purine pathway dysregulation³². However, prolonged stress may shift autophagy toward apoptosis and cellular injury. Collectively, these findings demonstrate that PAEs consistently disrupt oxidative balance across aquatic trophic levels through ROS overproduction, suppression of antioxidant systems and lipid peroxidation, forming a unified mechanistic pathway of toxicity³³.

Neurotoxicity and behavioral toxicity: The neurotoxic effects of phthalate esters (PAEs) in aquatic organisms are well documented, highlighting their capacity to disrupt nervous system function. In primary consumers such as *Daphnia magna*, exposure to dibutyl phthalate (DBP) (0.5-2 mg/L) significantly inhibits acetylcholinesterase (AChE) activity, indicating disruption of cholinergic neurotransmission³⁴. Metabolomic analyses further show that low-dose PAEs mixtures (0.1-10 µg/L) impair amino acid and energy metabolism, including glutamine GABA synthesis, choline metabolism and tRNA aminoacylation, suggesting reduced neurotransmitter production and altered neural signaling. Developmental exposure also affects dopaminergic regulation, with reduced expression of *drd1b* and *th*, altered AChE activity and disrupted circadian rhythm pathways. In fish, neurotoxicity is associated with both molecular and behavioral impairments. In zebrafish (*Danio rerio*), exposure to di(2-ethylhexyl) phthalate (DEHP) (4-324 µg/L) induces oxidative imbalance in neural tissues, neuronal damage and behavioral alterations, including impaired spatial learning and increased aggression³⁴. Additionally, these findings

demonstrate that PAEs and their metabolites induce neurotoxicity through interconnected mechanisms, including neurotransmitter disruption, oxidative stress, altered gene expression and neuronal apoptosis, ultimately leading to measurable behavioral and functional impairments in aquatic organisms³⁴.

Trans-generational transfer: Phthalate esters (PAEs) can be transferred across generations in aquatic organisms, affecting offspring survival and development. This occurs via direct transfer through gametes or embryos. In *Daphnia magna*, parental exposure to DEHP (1 µg/L) led to accumulation in ovaries and embryos, elevated ROS, gene dysregulation and reduced survival and reproduction in F0 and F1 generations. PAEs have also been detected in turtle eggs, confirming maternal transfer³⁵. In addition, PAEs induce heritable epigenetic changes. In zebrafish embryos exposed to MEHP (30 µM), 410 differentially methylated regions were identified that persisted into the F1 and F2 generations. DEHP exposure in adult males increased gonadal DNA methylation and affected offspring development, while DBP exposure caused germ cell disruption, embryonic defects and skeletal abnormalities in F1 progeny³⁴. Overall, trans-generational toxicity of PAEs is driven by two mechanisms: Direct chemical transfer and epigenetic inheritance (DNA methylation, histone modification and 5-hmC changes), disrupting development, metabolism and reproduction. These effects occur across aquatic taxa, linking molecular changes to observable phenotypes and raising concerns for long-term population stability under chronic environmental exposure³⁴.

Energy and metabolic disorders: Phthalate esters (PAEs) disrupt energy and metabolic processes across aquatic organisms by impairing photosynthesis, mitochondrial function and key metabolic pathways. In algae such as *Chlorella vulgaris* and *Phaeodactylum tricornutum*, DMP and DEP (10-5000 µg/L) reduce chlorophyll content and

mitochondrial membrane potential, leading to reduced photosynthetic efficiency and growth³⁵. Similar effects occur in aquatic plants like *Lemna minor*, where high DMP exposure decreases pigment levels and photochemical efficiency, indicating disrupted carbon and energy balance. In zooplankton, DBP and BBP impair TCA cycle intermediates and ATP production, reflecting disrupted energy metabolism³⁵. Metabolomic studies further show downregulation of amino acid biosynthesis and upregulation of glutathione metabolism, indicating oxidative stress-driven metabolic reprogramming. In bivalves such as *Mytilus coruscus* and *Mytilus galloprovincialis*, PAEs exposure induces ROS production, depletion of energy metabolites and biphasic metabolic responses affecting glucose, glycogen and amino acid pathways³⁵. Microplastics can further enhance DEHP bioavailability, worsening energy depletion and delaying physiological recovery³⁵. In crustaceans (*Penaeus vannamei*), PAEs disrupt lipid, amino acid and purine metabolism alongside inflammatory responses³⁵. In fish, PAEs alter lipid metabolism and endocrine regulation via PPAR and related signaling pathways. DEHP induces obesity-related energy imbalance in zebrafish and hepatic stress in catfish, while DINP disrupts endocannabinoid signaling, promoting lipid accumulation and hormonal disturbances. Overall, PAEs consistently impair energy homeostasis, metabolic regulation and physiological balance across aquatic taxa.

Immunotoxicity: The immune system is a key target of phthalate ester (PAEs) toxicity in aquatic organisms. In *Mytilus galloprovincialis*, DEHP exposure (4-324 µg/L, 21 days) upregulated NF-κB and downregulated HSP70, indicating disrupted immune-stress balance. In zebrafish, DEHP (1-5 mg/L, 28 days) increased serum complement C3 and its related gene expression. Early-life exposure to DBP (2 µg/L) induced persistent immune and inflammatory responses into adulthood, likely via disruption of the HPA axis and sustained cytokine activation. In *Labeo catla*, DEP exposure (1.6 mg/L, 30 days) significantly increased immune-related genes (igm-α, myd88) in liver tissue, with elevated myd88 also observed in the brain, suggesting systemic and neuro-immune effects³⁶.

Molecular pathway responses and associated adverse outcomes: The PAEs trigger molecular disruptions that cascade into physiological defects and ecosystem-level impacts. A key mechanism is oxidative imbalance: DMP and DEP increase ROS while suppressing antioxidant enzymes (SOD, CAT), causing lipid peroxidation, mitochondrial dysfunction, autophagy and apoptosis in microalgae such as *Phaeodactylum tricornutum* and *Chlorella vulgaris*³⁶. Similar effects occur in rotifers, where BBP impairs mitochondrial

function and reduces growth and reproduction³⁷. The PAEs also disrupt endocrine and neuro-signaling pathways. The DBP and DEHP suppress steroidogenic genes (cyp17a1, cyp19a, vtg), reducing sex hormones and causing gonadal damage and reproductive failure in fish. Thyroid disruption (tg, ttr, dio1/2) leads to developmental abnormalities in metabolism, while altered neural genes (ache, drd1b) impair behavior and locomotion³⁷.

Beyond individual effects, PAEs induce heritable epigenetic changes. MEHP causes persistent DNA methylation changes across generations in zebrafish, while paternal DBP exposure leads to developmental defects in offspring³⁷. In *Daphnia magna*, DEHP effects persist across generations, reducing survival and reproduction. At the ecosystem level, reduced algal productivity limits energy flow to higher trophic levels, while impaired survival and reproduction in consumers reduce population stability. Over time, these effects simplify community structure and weaken ecosystem functions such as nutrient cycling and biological regulation, threatening aquatic ecosystem resilience³⁷.

EXPOSURE PATHWAYS OF AQUATIC ORGANISMS TO PAEs

Direct exposure: The hydrophobic nature of phthalate esters (PAEs) promotes their uptake into aquatic organisms via passive pathways. In aquatic animals, dissolved PAEs enter mainly through respiratory diffusion, while in microalgae, uptake occurs through surface biosorption driven by equilibrium processes. At the cellular level, passive membrane diffusion dominates, with higher hydrophobicity enhancing permeability and bioaccumulation. Extracellular polymeric substances (EPS) in microalgae further influence adsorption and bioavailability³⁸. In fish, gills are the primary uptake route for lipophilic PAEs, especially during early life stages. Studies on *Etroplus suratensis* show higher DEHP accumulation and oxidative stress in gills than muscle, consistent with field observations reporting elevated PAEs levels in gill tissues, including DIBP up to 1061 ng/g³⁹.

Indirect Intake: Aquatic organisms can also be exposed to phthalate esters (PAEs) indirectly via trophic transfer and leaching from ingested microplastics (MPs). MPs act as secondary contamination sources by continuously releasing PAEs and additives. Feeding studies show that ingestion of PAE-containing MPs increases hepatic PAE levels in fish⁴⁰. Field evidence supports this pathway, with microplastics carrying PAEs detected in yellowfin tuna (*Thunnus albacares*) and associated with high tissue burdens, while similar correlations between MP ingestion and PAEs accumulation have been reported in Pacific sharks⁴¹. Even with biomagnification factors

below one, continuous dietary exposure can lead to significant accumulation at higher trophic levels. This is demonstrated in tri-trophic systems where DEHP transfers from algae to zooplankton and fish, accumulating in fish tissues. Additionally, physicochemical properties influence distribution, with low-molecular-weight PAEs showing higher accumulation in lower trophic organisms due to greater solubility and slower metabolism⁴².

Phthalate esters impacts on aquatic ecosystems: Aquatic systems, particularly in urban and industrial regions, are major sinks for phthalate esters (PAEs), largely from anthropogenic sources such as wastewater, agriculture and landfill leachates⁴³. Their hydrophobicity and low volatility promote persistence, sediment accumulation and bioavailability. PAEs enter organisms via water and diet, enabling trophic transfer and widespread toxicity, including endocrine, developmental and metabolic disruption. Compounds such as BBP, DEHP and DBP are especially harmful, causing deformities, reproductive impairment and organ damage⁴⁴. (Toxicity occurs even at low (ng-µg/L) concentrations and varies with molecular structure and life stage, with juveniles generally more sensitive. While some microorganisms can degrade PAEs, many persist and accumulate in sediments, acting as long-term contamination reservoirs. Evidence of transfer into food chains highlights aquatic systems as key pathways of exposure, emphasizing the need for continuous environmental monitoring⁴⁴.

Bioaccumulation in high-trophic-level organisms: Algae form the primary entry point for phthalate esters (PAEs) into aquatic food webs, where their hydrophobic nature promotes strong association with algal biomass and facilitates transfer to higher trophic levels. Accumulation is influenced by molecular properties, with higher-molecular-weight PAEs (high log K_{ow}) preferentially retained. Field observations confirm that phytoplankton can act as major contaminant sinks, sometimes exhibiting higher PAEs concentrations than organisms at higher trophic levels. While passive adsorption dominates uptake, emerging evidence suggests that active transport mechanisms and interactions with extracellular polymeric substances (EPS) and co-contaminants such as microplastics can further enhance accumulation⁴⁵.

The trophic transfer of PAEs is governed by a balance between uptake, metabolism and elimination. High-molecular-weight PAEs are primarily acquired through diet, whereas low-molecular-weight compounds are more readily absorbed via gills but are also rapidly excreted. Consequently, most PAEs exhibit limited biomagnification, with biomagnification factors generally below one and instead

display trophic dilution in many aquatic systems. This pattern is supported by field studies across both freshwater and marine environments. However, despite this limited magnification, continuous exposure and the formation of potentially toxic metabolites sustain ecological risks, indicating that PAEs remain significant contaminants within aquatic food webs⁴⁵.

LIMITATION AND CHALLENGES

Phthalate esters (PAEs) typically occur in aquatic environments as components of complex contaminant mixtures, coexisting with other plastic additives, microplastics and heavy metals. These interactions create multifaceted exposure scenarios where pollutant behavior and toxicity can be altered. Microplastics, for instance, can adsorb PAEs onto their surfaces, increasing persistence and bioavailability and potentially amplifying toxicity beyond single-compound effects. However, the mechanisms underlying these combined effects, particularly regarding metabolism, accumulation and organismal responses, remain insufficiently understood. Environmental conditions further shape PAEs dynamics and toxicity. Elevated temperatures enhance their release from plastics, hypoxia can increase uptake through intensified respiration and salinity changes may disrupt osmoregulation and membrane permeability. These stressors often act together, influencing both exposure pathways and physiological responses, though their combined effects are not yet fully resolved. At the ecosystem level, PAEs can disrupt trophic interactions by impairing the survival, growth and reproduction of key species. This may alter community structure, reduce energy transfer efficiency and destabilize aquatic food webs over time. Despite these risks, most research remains focused on single-species toxicity. A more comprehensive understanding will require integrated approaches that incorporate mixture effects, environmental variability, field observations and ecosystem-level modeling.

RECOMMENDATION

Future efforts should prioritize stricter regulation and phased replacement of high-risk phthalates with safer, biodegradable alternatives. Advanced monitoring programs integrating molecular biomarkers, environmental surveillance and long-term ecological assessments are needed to detect early biological effects and improve risk evaluation. Further research should focus on chronic low-dose exposure, mixture toxicity and the combined impacts of PAEs with other emerging pollutants under changing environmental

conditions. Strengthening wastewater treatment technologies, promoting sustainable plastic management and enhancing international collaboration will be critical for reducing PAEs contamination and safeguarding aquatic biodiversity, ecosystem resilience and public health.

Possible ways to address the problems associated with the molecular disruptive potential of phthalate esters (PAEs) involve a combination of environmental regulation, scientific innovation, public awareness and sustainable industrial practices. One of the most effective solutions is the reduction or gradual elimination of phthalate-based plasticizers in industrial products. Industries should adopt safer and biodegradable alternatives that possess lower toxicity and reduced environmental persistence. Governments and environmental agencies should establish and enforce strict regulations on the production, use, discharge and disposal of phthalate-containing materials to minimize environmental contamination.

Therefore, addressing phthalate ester (PAEs) toxicity requires integrated regulation, innovation and sustainable practices. Key actions include phasing out phthalate-based plasticizers and replacing them with safer biodegradable alternatives, supported by strict government regulations on production, use and disposal. Improved waste management, recycling and public education can reduce plastic pollution and environmental release. Wastewater treatment should be upgraded with advanced technologies such as activated carbon, membrane filtration and biodegradation systems. Continuous environmental monitoring and biomarker-based assessments are needed for early detection of toxicity. Ongoing research and multi-sector collaboration are essential to protect aquatic ecosystems and human health.

CONCLUSION

Phthalate esters (PAEs) are pervasive emerging contaminants that pose significant threats to aquatic ecosystems through endocrine disruption, oxidative stress, metabolic dysfunction and altered gene expression. Their persistence, bioaccumulation and trophic transfer adversely affect growth, reproduction, immunity and survival of aquatic organisms, ultimately threatening biodiversity and ecosystem stability. Environmental factors further influence their mobility, bioavailability and toxicity, complicating ecological risk assessment. Despite growing evidence of their harmful effects, critical knowledge gaps remain regarding chronic exposure, mixture toxicity and long-term ecosystem impacts. The continued release of PAEs, particularly in regions with weak regulatory frameworks, highlights the urgent need for

strengthened environmental policies, improved waste and wastewater management, advanced monitoring strategies and safer alternatives to conventional plasticizers. Effective mitigation of PAEs pollution is essential for protecting aquatic biodiversity, ecosystem resilience and human health while ensuring long-term environmental sustainability.

SIGNIFICANCE STATEMENT

Phthalate esters are widespread environmental contaminants that pose substantial risks to aquatic organisms and ecosystem stability through endocrine disruption, oxidative stress, metabolic dysfunction, neurotoxicity, immunotoxicity and reproductive impairment. This review integrates current evidence on their sources, environmental occurrence, exposure pathways, molecular mechanisms, bioaccumulation and ecological consequences. It highlights critical knowledge gaps concerning chronic exposure, mixture toxicity, interactions with microplastics and other stressors and long-term ecosystem effects. The findings emphasize the need for improved environmental monitoring, stronger regulatory measures, safer alternatives and integrated risk-management strategies to protect aquatic biodiversity, ecosystem resilience and human health.

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