PBDE’s exposure in fishes and endocrine disruption and neurodevelopmental toxicity

**Abstract**

Polybrominated diphenyl ethers (PBDEs) are highly contaminated through polluted water, sediments, and food run through either industrial effluents or are released via domestic sewage. Thus, after some time, PBDEs degrade and are released during the recycling of appliances within consumer products. Under the concentration builds inside fish from their environment, PBDEs tend to interfere with biochemical systems, disrupting the thyroid hormones; leading low fertility, and reproductive organ, development and spawn behavior were impeded. Neurodevelopmental toxicity include physical deformities and low egg survival fidelity, thus stressing fish populations and ecosystem health. PBDEs disturb Thyroxin hormonal status, thus disrupting thyroid hormones, developmental functions, and reproductive functions increased brain dysfunction in fish feeding and swimming behavior that result in attentional problems and increased risk for learning impairments, particularly under situations of prenatal or very early life exposure. The management of PBDE pollution can involve recycling of plastic wastes; regulation on human populations and waste disposal would save aquatic lives.

**Key words:** PBDE bioaccumulation, Endocrine disruption, Neurodevelopmental toxicity.Waste management

**Introduction**

polybrominated diphenyl ethers (PBDEs) are flame retardant chemicals that are added in plastics and foam products to make them more difficult to ignite apart from this PBDES have also been added to many consumer and commercial products including textiles, carpeting, construction materials, and electronics to reduce their combustibility.

PBDEs are aromatic compounds that share a common structure of a diphenyl ether molecule with 1-10 bromine atoms(Figure 1); each variety is known as a congener. Theoretically, there could be up to 209 PBDE congeners, however the actual number is significantly lower. often detected in commercial PBDE combinations and assessments of PBDEs in humans also consider the environment. There are three commercial mixtures of PBDEs: Penta-BDEs, Octa-BDEs, and Deca-BDEs. Because of their ability to dissolve in lipids, bioaccumulate, and migrate over large distances, PBDEs are able to remain in the environment for an extended period of time. (Xiang et al., 2007) In addition, PBDEs do not react with polymer components and can be easily leached from a polymer throughout their life cycle (Cai et al., 2020). Polybrominated diphenyl ethers (PBDEs) are compounds that can accumulate in living organisms due to their biological enrichment and amplification. As these substances move through the food chain, they can be ingested by various organisms, starting from smaller prey and moving up to larger predators. This process can lead to increasingly higher concentrations of PBDEs in the tissues of these organisms. Eventually, humans can be exposed to these compounds through the consumption of contaminated food, particularly fatty fish and meat, which may contain significant levels of PBDEs that have bioaccumulated over time.

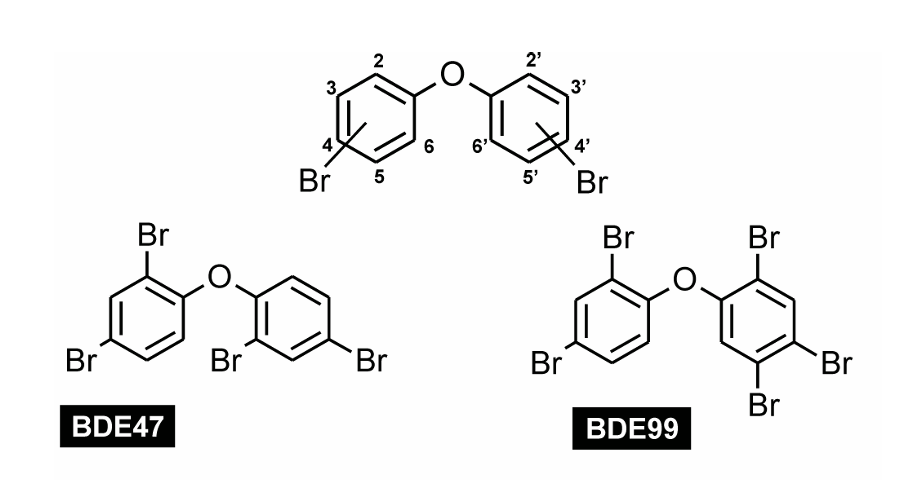


Fig.1. Chemical structures of PBDEs (Sheikh, 2021)

Increasing evidence has highlighted the impact of endocrine disruptors (EDs) in modifying hormonal signaling and function, originating from widespread used compounds in manufacturing and processing. As a result of their long half-life and persistence EDs can typically be detected not just in industrial goods but also in residences and in the ecosystem, therefore creating the prerequisites for long-term exposure. Polybrominated diphenyl ethers (PBDEs) are prevalent EDs commonly seen in industrial products like flame retardants, and emerging studies have increasingly shown

Polybrominated diphenyl ethers (PBDEs) are the extensive and prominent world widely used brominated flame retardants (BFRs) class of organic flame retardants , and they were included in the list of Persistent Organic Pollutants (POPs) in the addition to the Stockholm Convention on POPs in 2009 and 2017. The PBDEs have threatened human health and ecosystems in a great deal due to their prevalence in environment and toxicity. Polybrominated diphenyl ethers (PBDEs), a form of brominated flame retardant (BFR), are widely employed in furniture, fabrics, plastics, and electrical devices. Environmental pollutants known as PBDEs are common and can potentially affect ecosystems and human health. Here, we highlight new research on PBDEs' prevalence, level of contamination, and movement through soil, water/sediment, and the atmosphere. In-depth discussions are given of four topics:

1. the origins of PBDEs in the environment;
2. their presence and movement in soil;
3. their presence in aquatic ecosystems (water/sediment) and their water–sediment partitioning; and
4. their presence in the atmosphere and their gas-particle partitioning. Based on present scientific and practical requirements, prospects for the study of PBDE incidence are also considered (Wu et al., 2019)

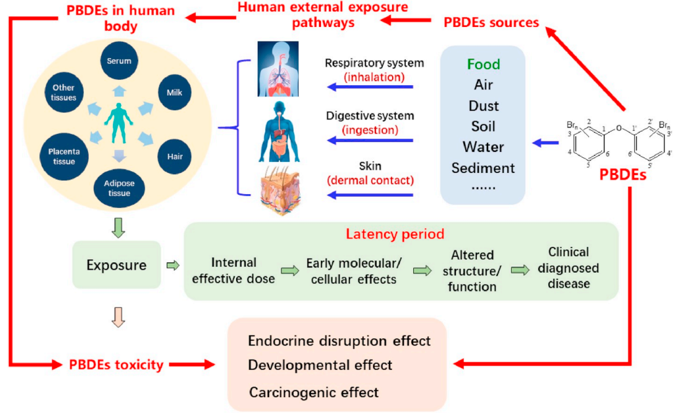


Fig.2.Sources,exposure pathway and toxicity of PBDEs to humans (Wu et al., 2020)

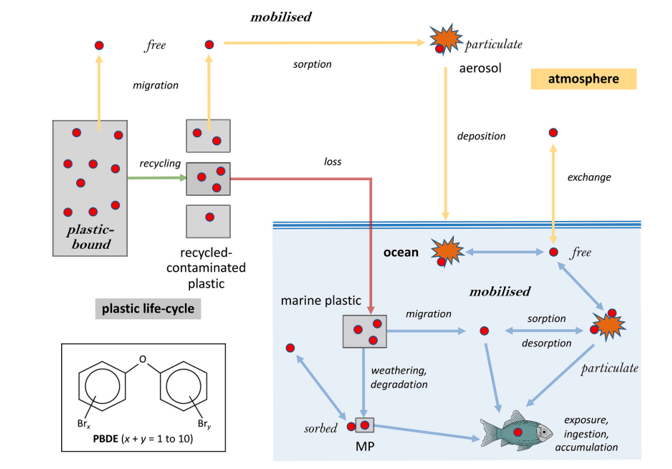


Fig.3. **.** Molecular structure of PBDEs and conceptualisation and definition of the terms, associations and processes referred to in the discussion. Red dots represent PBDE molecules which can be encountered in plastics (in grey boxes and “bound”), or occur as free or sorbed molecules that have migrated into the environment (mobilised) and are free or are sorbed to aerosols and particulate matter (orange stars) or microplastics (MPs).

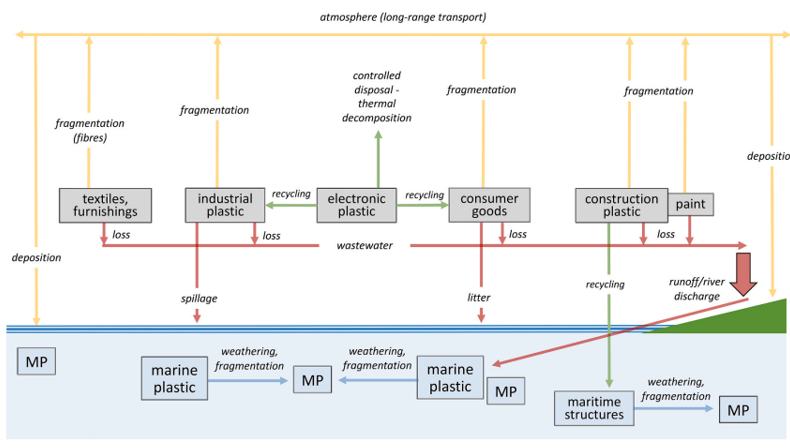


Fig .4. Environmental pathway of PBDE bearing plastic in different sectors and their inputs to the marine environment.MP= microplastic.

Fig .3,4. (Turner, 2022)

PBDEs that have moved into the environment and are still in free molecular form or that have migrated to solids such as sediments, soils, and aerosols are referred to as "mobilized" throughout the paper. These are defined as different from PBDEs that are still physically "bound" (but not bonded) to the plastic itself. However, the relationships and interdependencies between bound and mobilized forms are discussed, with a special focus on the function of marine plastics and microplastics (which are operationally defined as having a maximum size of 5 mm). To support the study, Fig. 2 conceptualizes and specifies some of the key terminology, relationships, and procedures that are essential to the arguments and conversation, and Fig. 3 shows the environmental sources and channels of specific types (or use sectors) of PBDE-bearing plastics. The objective of this audit paper is to investigate the affect of polybrominated diphenyl ethers (PBDEs) introduction on endocrine disturbance and neurodevelopmental harmfulness in both angle and people. This incorporates looking at how PBDEs meddled with hormonal frameworks, influence generation, development, and neurological improvement, and assessing their potential dangers to oceanic biological systems and human wellbeing. The survey points to supply a clear understanding of the instruments of poisonous quality and highlight the broader suggestions for natural and open wellbeing security.

**Sources of PBDEs to the marine environment**

Evaporative or fugitive migration of BFRs into the atmosphere occurs during the production and use of plastics and during or after their disposal by landfilling, recycling, controlled incineration or open burning (Xu et al., 2021)The production, use, and disposal of brominated flame retardants (BFRs) can all result in evaporative and fugitive emissions that can leak into the atmosphere. BFR-containing polymers and materials volatilize, or release, these compounds into the air over time during manufacturing and usage, particularly when subjected to heat or mechanical stress. Consumer goods like electronics and furniture can accidentally release fugitive emissions due to leaks, abrasions, and wear. Air pollution is also caused by disposal methods such landfilling, controlled incineration, open burning, and recycling. Significant amounts of harmful byproducts are released during open burning, although controlled incineration may still produce incomplete combustion and airborne contaminants despite its efforts to reduce emissions. Long-term environmental hazards may result from recycling and landfill processes' progressive volatilization and leaching. Due to the extensive BFR pollution caused by these emissions, ecosystems, human health, and air quality are all impacted (Xu et al., 2021). The degree of unsaturation, glass transition temperature, additive presence, weathering-induced surface and structural changes, compound size (molar volume), and crystallinity all affect internal diffusion inside the matrix. All these parameters change the permeability and interaction of the matrix with the environment, which in turn affects the surface evaporation of evenly bound BFRs in polymers (Calò et al., 2011; Wu et al., 2019)Waste plastic itself may potentially release bound PBDEs into the environment. Microplastics are among the sources here, as are (Harrad et al., 2019)fibers released into wastewater or the environment during laundry; fibers produced by physical abrasion during disposal (Dalla Fontana et al., 2020; O’Brien et al., 2020) two and bigger plastics, including sporadic technological items, that result from spills, littering, and poor waste management (Shaw and Turner, 2019) Numerous factors related to the plastic and its environment influence the way PBDEs (polybrominated diphenyl ethers) stick to plastics, according to study. Surface area, roughness, and crystallinity all have a significant role in how well PBDEs may stick to a plastic. This process is also affected by the water's properties, including temperature and the kind and amount of organic materials. These factors combine to affect PBDE accumulation and mobility in aquatic environments, which can have a big impact on pollution and environmental health (Singla et al., 2020; Xu et al., 2019).

**Importance of studying PBDE exposure in aquatic ecosystems**

Inland waterways may be harmed by pollution from mining, transportation, industry, nuclear energy, power production, and the production of weaponry. Aquatic pollution can result from human use, misuse, and improper disposal of chemical products and byproducts. Businesses that use fossil fuels may be the source of metals, inorganic compounds, and pollutants that are aliphatic and aromatic hydrocarbons. Through spills, landfill overflow, and wastewater discharge, synthetic organic compounds—including plastics and plasticizers, flame retardants, pesticides, surfactants, and pharmaceuticals—can pollute inland waterways. While some pollutants are both toxic and persistent, they can bioaccumulate in aquatic systems' biota; fish and wildlife populations can be adversely affected by heavy metals and other toxic elements released by industrial and agricultural activities; radionuclide accidents from nuclear activities have contaminated inland waters; and warm industrial effluents have a detrimental effect on aquatic organisms' habitat (Echols et al., 2009) PBDEs are regarded as chemicals that are becoming more and more problematic for the environment. It is reasonable to assume that human exposure to these substances will continue given their usage patterns and enduring chemical characteristics. Human health impacts owing to such exposures also continue to be a subject of concern(Kodavanti and Loganathan, 2017) .Polybrominated diphenyl ethers (PBDEs) have been the most widely used brominated flame retardants across the globe for the past few decades. Due to their toxic nature and high occurrence in the environment, PBDEs are proving to be a threat to both ecosystems and human health. In aquatic systems, PBDEs accumulate in fish tissues. The ingestion of food such as fish and the inhalation or skin contact with house dust are the major routes of PBDEs exposure in humans. PBDEs exposure has been associated with developmental impairment of the nervous system in children, hepatotoxicity, disturbances in metabolism and endocrine function, carcinogenicity, and immunosuppression. With a functional bridge between innate and adaptive immune responses, the complement system, among the components of the immune system, has been evaluated after PBDE exposure in humans, mice, and fishes. The complement has emerged as an important immunological derailment possibly used as a biomarker to monitor effects from exposure to PBDEs, exactly due to its central role in immune response.

**Sources and Pathways of PBDE Exposure in Fish**

Absorption, distribution, metabolism, and excretion (ADME) patterns of PBDE toxicokinetics in fish have been found to differ according on the species, life stage, exposure route, and PBDE congener.

Table1. PBDE toxicokinetics measured in teleost fish species

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Species | Treatment | Route | Dose/Duration | Effects Observed | Reference |
| Common carp (Juv.; *C. carpio*) | BDE-99 | Gi,liver microsomes | 12–29 pmol/mg protein; 60 min incub | Reductive debromination: BDE-99 to BDE-47; Metabolism in liver > intestine; No debromination by Gi microflora | (Benedict et al., 2007) |
| Atlantic salmon (Juv.; *S. salar*) | PentaBDE; OctaBDE | Diet | 10, 50 mg/kg bw; 7 d | No significant hepatic CYP1A induction or protein expression | (Boon et al., 2002) |
| Chinook salmon (Adu*.; O. tshawytscha*) | BDE-99 | Liver microsomes, cytosol | 0.03 - 1.8 µM; 16 h incub | Reductive debromination: BDE-99 to BDE-49; negative GST/CDNB assay | (Browne et al., 2009) |
| Northern Pike (*E. Lucius)* | PCBs, PCNs, BDE-47, -99, -153 | Diet | 90 ng/µl lipid (10 µl injected into rainbow trout); 9 d | Uptake efficiencies: BDE-47 ~90%; BDE-99 ~60%; BDE-153 ~40% | (Burreau et al., 2000) |
| Northern Pike | 14C-BDE-47 | Diet | 16.2 µg/µl; 9, 18, 36, 65 d | 14C-BDE-47 uptake > 90%; Highest in liver, adipose tissue, spinal cord-surrounding tissue, eyes, gall bladder; Lowest in muscle, spleen, gills | (Cheng et al., 2012) |
| Crucian carp (*C. auratus)* | BDE-15 | Aqueous | 0, 10, 100 µg/l; 50 d | BDE-15 accumulation in gill, liver; 2 mono-brominated, 3 hydroxy metabolites | (Garcia-Reyero et al., 2014) |
| Zebrafish (Larv.) | BDE-209 | Spiked sediment | 12.5 mg/kg; 4 – 192 hpf | BDE-209 bioaccumulation | (Kierkegaard et al., 1999) |
| Rainbow trout (Juv.; *O. mykiss*) | DecaBDE (DowFR-300BA) | Diet | 7.5 - 10 mg/kg bw/day; 16, 49, 120 d; 71 d depuration | Reductive debromination: hexa- to nonaBDE formation (liver, muscle); BDE-154 dominant; BDE-209 uptake: 0.02–0.13% | (Kuiper et al., 2006) |
| Zebrafish (Juv.; *D. rerio)* | PentaBDE (De-71) | Aqueous | 0, 0.1, 1 mg/l; 4 wk | AhR-mediated effects linked to PBDD/F impurities; weak induction CYP1A; no DR-CALUX response (purified De-71) | (Kuo et al., 2010) |
| Lake whitefish (Juv.; *C.* *clupeaformis*) | BDE-209 | Diet | 0, 0.1, 1, 2 µg/g; 30 d | Accumulation: BDE-209 + nonaBDEs (BDE-206, -207, -208) in liver | (Nyholm et al., 2009) |
| Zebrafish (Adu.) | BDE-28, -183, -209 (mix) | Diet | 1 and 100 nmol/g ww food at 2% bw/day; 42 d with 14 d dep | Reductive debromination (high dose); Uptake: BDE-28 (100%) > BDE-183 (10%) > BDE-209 (< 1%) | (Munschy et al., 2011) |
| Common sole (Juv.; *S. solea L*.) | BDE-28, -47, -99, -100, -153, -209 (mix) | Diet | 82 - 184 ng/g ww food at 0.8% bw/day; 84 d, 149 d dep | Uptake efficiency: BDE-28 = 16%; BDE-47 = 15%; BDE-99 = 13%; BDE-100 = 14%; BDE-153 = 10%; BDE-209 = 1.4%. Reductive debromination: BDE-49; BDE-154; BDE-183; BDE-202 | (Munschy et al., 2010) |
| Common sole (Juv.) | BDE-28, -47, -99, -100, -153, -209 (mix) | Diet | 82 - 184 ng/g ww food at 0.8% bw/day; 84 d, 149 d dep | Oxidative metabolism: 6-OH-BDE-47; 4’-OH-BDE-49; 4’-OH-BDE-101; 4’-OH-BDE-103; No MeO metabolites detected | (Anastasiou et al., 2014) |
| Common carp (Adu.) | BDE-99 | Liver microsomes, cytosol | 354 pmol; 1 - 250 µM; 90 min incub | Reductive debromination (BDE-99 to BDE-47) more prevalent in liver microsomes than cytosol; THs (rT3, T4) and iodoacetate inhibited debromination | (Noyes and Stapleton, 2014) |
| Fathead minnow (Juv*., P. promelas*) | BDE-209 | Diet | 10 µg/g food at 5% bw/day; 28 d | BDE-209 bioaccumulation; BDE-209 uptake efficiency = 5.8%; Reductive debromination to penta-octaBDEs; BDE-154 dominant metabolite; BDE-101 lowest Mw metabolite | (Noyes et al., 2013) |
| Fathead minnow (Adu.) | BDE-209 | Diet | 95 ng/g ww food and 10 µg/g ww food at 3% bw/day; 28 d with 14 d dep | BDE-209 bioaccumulation; Reductive debromination to penta-octaBDEs; BDE-154 dominant metabolite; BDE-101 lowest Mw metabolite | (Olsvik et al., 2009) |
| Atlantic cod (Juv., *G. morhua*) | BDE-47 | Aqueous | 5 µg/l; 21 d | Liver: ↓ mRNA transcripts encoding CYP1A, CYP2C33-like, CYP3C1-like, UDPGT; No effects on antioxidant genes (GSH-Px, GR) | (Roberts et al., 2011a) |
| Rainbow trout, Common carp, Chinook salmon (*O. tschawytscha)* | BDE-28, -47, -49, -99, -100, -153, -154, -183, -203, -208, -209 | Liver microsomes | 1 µM; 24 h (hepta to BDE-209); 1 h (tri- to hexaBDEs) | Reductive debromination of BDE-99, -153, -183, -203, -208, -209; Carp: meta-position debrom dominated; Salmonids: meta- and para-position debrom; No metabolism of PBDEs lacking meta-substituted Br | (Nakari and Huhtala, 2010) |
| Rainbow trout, Common carp (Juv.) | BDE-209 | Diet, in vitro | 940 ng/g ww food, 1% bw/day; 5 mo; 15 pmol/mg protein; 1, 24 h (microsomes) | Reductive debromination (trout): Formation of BDE-207, -208, -188, -201, -202, unknown octa-heptaBDEs; BDE-209 uptake (trout): 3.2%; liver > serum > intestine > carcass (lipid-normalized) | (Roberts et al., 2011b) |
| Common carp (Juv.) | BDE-209 | Diet | 940 ng/day-fish; 60 d w/40 d dep | Reductive debromination: Formation of BDE-154, BDE-155, unknown hexa- to octaBDEs; No BDE-209 bioaccumulation | (Stapleton et al., 2004a) |
| Common carp (Juv.) | BDE-99, BDE-183 | Diet | 400 ng/day-fish (BDE-99); 100 ng/day-fish (BDE-183); 62 d w/37 d dep | Reductive debromination (Gi tract): BDE-99 → BDE-47; BDE-183 → BDE-154, unknown hexaBDE; Uptake: BDE-99 = 9.5%; BDE-183 = 17% | (Stapleton et al., 2004b) |
| Common carp (Juv.) | BDE-28, -47, -99, -153 (mix) | Diet | 470 ng/day-fish; 60 d w/40 d dep | BDE-47 accumulation, high assimilation; No BDE-99 bioaccumulation; No hydroxy metabolites detected | (Stapleton et al., 2004c) |
| Japanese medaka (Adu.; *O. latipes*) | 6-OH-BDE-47, 6-MeO-BDE-47, BDE-47 | Maternal | 21, 8, 0.9 µg/g dw food at 2% bw/day; 14 d | No OH-, MeO-BDEs in BDE-47 treated fish; In vivo and in vitro conversion of 6-OH-BDE-47 to 6-MeO-BDE-47 (and vice-versa); Maternal transfer to eggs | (Wan et al., 2010) |
| Common carp (Juv.) | Penta and DecaBDE mixtures | Diet | 100, 120, 150 µg/day/fish; 20 d | Reductive debromination facilitated by at least one meta- or para- doubly flanked Br; 11 OH-BDEs measured in serum of pentaBDE exposed fish; No OH-BDEs in decaBDE exposed fish | (Zeng et al., 2012) |
| Chinese sturgeon (Adu.; *A. sinensis*) | BDE-209 | Field collected | Liver microsomes; PBPK modeling | Reductive debromination; Formation of BDE-126, -154, -188, -202, -204, -197; Low partition coefficients from blood to tissues lead to higher bioaccumulation of hepta to BDE-209 in absorbing tissues | (Wan et al., 2013) |

dep = depuration; EROD = ethoxyresorufin-O-deethylase; dpf = days post fertilization; dph = days post hatch; dio = deiodinase; DR-CALUX = chemical-activated luciferase gene expression mediated by Ah-receptor activation; GI = gastrointestinal; HDT = highest dose tested; hpf = hours post fertiliza-tion; PBPK = physiologically based pharmacokinetic; UDPGT = uridine diphosphate glucuronosyl phosphate

Note: This table highlights the key species, treatments, and observed effects

Teleost and human PBDEs are metabolized differently. Reductive debromination of PBDEs has been acknowledged in numerous investigations as a significant metabolic route, as shown in Table 1. The subjects upon which information is collected include common carp (*C. carpio*), fathead minnow (P. promelas), rainbow trout, lake trout (*S. namaycush*),Chinook salmon (*O. tshawytscha*), and zebrafish (*D. rerio*). Despite PBDE reductive debromination seeming to represent a principal metabolic pathway in fish, the role played by some enzyme systems in catalyzing this biotransformation remains unknown. BFRs, Brownian Fluoride, is released to the atmosphere through production, use, and disposal of plastics. The rate-limited process for surface evaporation of BFRs includes properties like internal diffusion within the plastic matrix which relates to the following: crystallinity, glass transition temperature, degree of unsaturation, additives presence, modifications through weathering, and size of compound. Diffusion coefficients for PBDEs in the unweathered ABS used in electronic casings vary within 10 −20 to 10 −27 m2s −1 while showing a gradual decrease with enhanced bromination levels. Emission factors of varied PBDE species from polymers and textiles, in turn, cover the range around 10−2 to 10−7. In total, 0.1 – 0.4% loss of PBDEs is measured from the interior atmosphere by leaving domestic electronic devices based on surface evaporation after serving ten years in operation. Surface evaporation is expected to be increased in dismantling, recycling, or disposal of plastics in which thermal treatment or shredding-pulverization is used (Turner, 2022). Plastic debris known as PBDEs poses a serious threat to the environment. Soft furnishings and electronic plastics that are utilized for recycling or disassembly include them. However, it is anticipated that over the next ten or so years, their inputs of BFRs would decline as these goods are disposed of or destroyed. The varied pool of tainted and flame-retarded plastics and microplastics that have been left in the ocean or have particular marine applications is a more substantial source of PBDEs. The data utilized and the assumptions made determine the estimates for this source. Not with standing these doubts, it is anticipated that this reservoir would provide as a sustained supply of mobilized PBDEs with little overall effects. Research indicates that the movement of microplastics into some marine There is empirical evidence that digestive chemicals that speed up the breakdown of PBDEs may enhance migration from microplastics into components of the marine food chain. Microplastics that have seen substantial physical and chemical weathering are more likely to migrate.

There is increasing evidence that various endocrine-disrupting chemicals, such as PBDEs, affect genes related to steroidogenesis. While PBDEs mainly target the thyroid system, in vitro studies suggest they might also influence the expression or activity of steroidogenic enzymes, potentially affecting in vivo steroidogenesis, sex hormone levels, and reproduction. Currently, there is limited information on how PBDEs impact steroidogenesis in fish species. Research indicates that exposure to DE-71 significantly increases the expression of the brain aromatase gene in zebrafish during early embryonic development. Furthermore, exposure to DE-71 throughout their life cycle may alter the expression profiles of reproductive hormones that regulate genes and receptors along the brain-pituitary-gonad axis. Changes induced by PBDEs in several key genes related to egg and sperm quality could also lead to reproductive issues (Yu et al., 2015).

PBDE exposure in human

These chemicals are known as polybrominated diphenyl ethers (PBDEs), and they are often used in large quantities as flame-retardant ingredients in many commercial products. Biomonitoring data have recently shown that PBDE concentrations have increased sharply in human and animal bodies. The concentrations of PBDEs in North America have often been higher than those in Europe and Asia. Besides, the levels of PBDEs in newborns and toddlers are three to nine times higher than adults. Hence, this suggests that adults have been exposed to the highest levels of these chemicals through the pathways of dust and breast milk. The most frequently found isomers in humans are tetra-, penta-, and hexa-BDEs. Thyroid homeostasis disturbance, neurodevelopmental abnormalities, reproductive alterations, and possibly cancer are likely toxicological consequences of exposure to PBDEs, according to research on experimental animals. PBDEs may be developmental neurotoxicants, according to epidemiological evidence in people and experimental research in animals. Long-term behavioral impairments, especially concerning motor activity and cognition, may be caused by exposure to PBDEs either in the prenatal or postnatal periods. This review is based on recent research concerning the harmful effects of PBDEs on the environment and the serious negative impacts on health due to the current state of PBDEs in the environment (Linares et al., 2015). Any potential conclusions from animal evidence on the danger of adverse nervous system effects in humans exposed to PBDEs at any time during pregnancy or shortly after birth through breast milk or home dust can only be extrapolated. RfDs can be determined to range from 92 to 660 ng/kg/day by dividing NOEL values by common safety factors.

These values fall within the realistic range of infant exposure in the United States (via breast milk; approximately 300 ng/kg/day) and are comparable to the levels of exposure for toddlers (50 ng/kg/day) through food and household dust. Comparable results are achieved when body load is compared across species, that is, levels in animals that have been shown to result in adverse developmental behavioral effects lie within the same range of high human exposures (Gardos and Cole, 1976; Lorber, 2008; Schecter et al., 2006).

Though human tissues and environmental samples contain these chemicals, not much is known regarding human exposure through diet to PBDEs. In this study, we report the results of measuring levels of PBDEs in various food samples bought at Catalan supermarkets, Spain, during 2000. This entire population residing in this region was expected to intake PBDEs through their diets. The PBDE levels in fruits, vegetables, and tubers were lowest while oils and fats, fish and shellfish, meat and meat products, and eggs possessed the highest quantity. The total dietary intake of PBDEs per day through an adult male was either 81.9 ng (taking ND = 0) or 97.3 ng (considering ND = 1/2 limit of detection, LOD). Fish and shellfish contributed the largest share of around one-third of the total consumption. TetraBDEs and pentaBDEs were the homologues with the largest percentage contributions to the total PBDEs. The safety factor for PBDE exposure from food is more than five orders of magnitude when compared with the currently recommended lowest documented adverse effect level value of 1 mg/kg/day for the most sensitive endpoints for toxic effects of PBDEs (Bocio et al., 2003).

**Regulatory Measures and Risk Mitigation**

This section basically refers to some public and governmental pathologies to explain some regulation response that seems unaligned with technical risk assessments or some decision rule such as utility maximization. One of the most significant branches is about how psychological biases influence perception about danger and to what extent law should address this. It mainly borrows from Tversky and Kahneman's works on heuristics and biases, where the researchers probed into how the judgments of individuals were determined by intuitive rules of thumb while dealing with uncertainty much of the time in ways that contravened the principles of probability theory (Bocio et al., 2003). The fear in this study is that, in response to the (mis)fears of the people, democratic governments may perpetuate biased risk perceptions in laws, policies, and regulations, which would haunt the economy, environment, and public health in turn.(MacGillivray et al., 2011).The ecological impact of pollution on marine ecosystems has a myriad of adverse impacts on the environment and marine life. These range from long-term illnesses such as cancer to developmental disorders in children. These also get to the people through the food chain. The majority of the environmental pollutants present in fish products come from polluted waters. Options for minimizing the same are extremely limited. It is especially important to consider the pros and cons of consuming fish in relation to its healthy constituents. Environmental pollutants such as POPs, metals, and MPs that mainly threaten marine ecosystems have to be tackled immediately. Furthermore, the combined exposure to a number of substances that are detrimental to human health is an issue for risk managers and scientists alike. Knowledge and scientific evidence need to be continually updated for such pollutants as MPs, for example, where it is currently increasing in importance and for NPs, as these have yet to have any maximum values imposed on them. A combination of remediation techniques and legislation might do the best for increasing the obligation of both scientific and public communities (Visciano, 2024).

**Future Research Direction**

The level of environmental PBDEs increased from their peak around 2000, following shortly after a manufacturing ban of the compounds; however, it appears that the rate of decrease is slowing down. Consequently, this paper again assessed a US riverine system found in 1999 to have the largest fish tissue loads (fillet) of PBDEs globally, with an objective of refreshing environmental PBDE trends. Fish tissues from the years 2018–2020 were analyzed for PBDEs, and the results were compared to those from 1999–2000 and 2007. Positively, over a period of 20 years, levels declined by more than 75%. However, PBDEs were still detected in 93% of the samples and at all sampling sites (n = 16). Fish from the Dan R., below the Hyco R. (where the highest level in the world was previously reported), had the highest PBDE level (16,300 ng g−1 lipid weight). Levels within Hyco R. fish have declined at an annual rate of 30% through 2007. However, reductions during the subsequent 12 years have diminished to only 1.2%. In fact, fillet levels since 2007 actually increased at an estimated annual rate of 8% immediately downstream from the Hyco R. The profiles among the species of the congeners differed, possibly by hepatic enzyme debromination, meaning single congener-based health risk assessments may not be protective enough. Within North America, PBDE fish levels in this freshwater system were twice those of North America's Great Lakes and exceeded by 10-fold those in carp examined from Illinois, USA, another historical hotspot. Average fish PBDE levels also exceed maxima observed in European and Asian riverine systems and were 1000's of times higher than the environmental quality standard (EQS) set by European Parliament (0.0085 ng g−1 wet weight, aquatic biota). Therefore, monitoring of PBDEs should continue to be a priority for regulatory agencies as part of a strategy aimed at determining and eliminating their source and evaluating their potential human health effects in combination with all other coexisting (emerging and legacy) contaminants(La Guardia et al., 2024). There is evidence to show that pregnant women exposed to different PBDE congeners may be at risk for injury to both themselves and their unborn children. Although not all of the studies of the associations between PBDE and maternal biomarkers or maternal/infant health outcomes have found statistically significant associations, some studies reporting on suggestive trends with similar directional associations lend support to the negative infant health outcomes—namely, birth weight—that are associated with PBDE exposure. However, few findings have been reported on PBDE exposures, such as BDEs 153, 47, and its link to the risk of preterm birth that needs further explanation. The studies in the review are heterogeneous, and future research is needed to confirm these findings. Understanding the risk associated with PBDE exposures during pregnancy can be improved by future research on large longitudinal prospective cohorts that include sophisticated statistical testing techniques to show causal relationship, a rich collection of confounders, maternal biomarker analysis, and dependable exposure monitoring. Further, toxicological research would help in clarifying the toxicity mechanisms responsible for the poorer pregnancy outcomes associated with PBDE exposure.(Gomes et al., 2024) In wastewater treatment plants, there are several treatment technologies specifically intended for the effective removal and breakdown of organic contaminants, and these can be classified under three broad categories: biological, physical, and chemical treatments. Biological treatments utilize a sequence of microorganisms, such as aerobic bacteria, fungi, algae, and protozoans, that are all involved in the natural process of removing organic contaminants in the wastewater. This is universally utilized in the majority of water treatment plants because of the effectiveness and efficiency of the treatment. Physical and chemical treatments are, however, utilized specifically to the intent of solving the problem presented by insoluble and toxic organic contaminants, particularly when these contaminants are relatively present in high quantities in the wastewater (Kim and Han, 2024)

**Conclusion**

PBDEs (Polybrominated diphenyl ethers) stand out as substances mostly used for fire protection in different products such as electronics, furniture, and textiles. Among other things, these chemicals become components of materials in order to lower the flammability of those materials, however, the downside of these chemicals is their resistance to degradation that purports environmental damage. The usual scenario is that PDBEs tend to manage to be bioaccumulated where they move up the food chain or spread in water bodies.

Due to their fat solubility, animals store PBDEs in their fattiest tissues, e.g., fish. The concentration of the toxic substances in the food chain consistently rises when minute organisms are consumed by larger animal species and, consequently, human beings can also get in contact those toxins. The contamination caused by these chemicals can lead to several health problems in people as they are known to bind to the thyroid hormone due to which various developmental issues are caused and further, they take part in the processes that can damage nerve cells. In addition, PBDEs can also induce animal tumor growth.

Despite all the attempts that have been made to bring the production and use of PBDEs to the lowest minimum, the presence of these substances in the environment remains substantial. More precisely, fishes' tissues in certain areas are still contaminated by a large amount of PBDEs. According to some reports, PBDE levels have decreased to a certain extent in the environment between the 1960s and 1970s and their peak was around 2000, but still, they are not below the threshold limit and they can potentially pose a health hazard.

To stop these dangers, one thing that needs to be done, is to decrease PBDE contamination by choosing the waste management practices that treat the chemicals in the most environmentally friendly way, using alternative formulations, and also check and limit those PBDEs released.

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**Author contributions**

All authors contributed to the design of this manuscript also conducted literature search and drafted the manuscript

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No potential conflict of interest was reported by the authors.

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**Data availability statement**

Data sharing is not applicable to this study as no datasets were generated or analyzed during the current study.

**Disclaimer**

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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