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Review Article

Investigating the Effect of Phthalate Esters – Dimethyl Phthalate and Diethyl Phthalate (EDCs) in Waste Plastic Materials and Used Engine Oil

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Abstract

The increasing environmental presence of phthalate esters, particularly Dimethyl Phthalate (DMP) and Diethyl Phthalate (DEP), derived from waste plastic materials and used engine oil, has raised concerns about their potential health effects. This study investigates the routes of exposure, bio-metabolism, and toxicological impact of DMP and DEP, focusing on their endocrine-disrupting properties. Phthalates are semi-volatile organic compounds used in a variety of consumer products, leading to widespread human exposure via ingestion, inhalation, and dermal contact. These compounds have been found to leach into food, water, and personal care products, making exposure nearly inevitable. Once absorbed, DMP and DEP are rapidly metabolized and excreted, yet their disruption of hormone signalling pathways poses significant health risks, particularly to the reproductive systems of both males and females. The study highlights adverse reproductive effects, including reduced sperm quality, hormonal imbalances, and ovarian dysfunction, and emphasizes the teratogenic and carcinogenic risks associated with prenatal and chronic exposure. Furthermore, phthalates have been linked to developmental abnormalities, neurotoxicity, and a higher incidence of certain cancers. As DMP and DEP are commonly detected in the environment and human tissues, this research underscores the need for stringent regulatory measures and further investigation into their long-term health impacts.

Keywords: dimethyl phthalate; diethyl phthalate; endocrine disruptors; environmental contaminants; reproductive toxicity

Introduction

Phthalate esters, including Dimethyl Phthalate (DMP) and Diethyl Phthalate (DEP), are part of a class of endocrine-disrupting chemicals (EDCs). These compounds are commonly used as plasticizers in manufacturing a wide range of plastic products. Additionally, they find applications as additives in various industrial sectors, such as in engine oils [1]. Despite their extensive industrial use, recent studies have shown that DMP and DEP can leach from waste materials and enter the environment. This raises serious concerns about their potentially harmful effects on human health, wildlife, and ecosystems.

DMP and DEP, exhibit a notable tendency to leach from plastics and other industrial materials into the surrounding environment. This leaching occurs through several mechanisms, such as the degradation of plastics and improper disposal practices. When plastics containing phthalates break down due to environmental factors such as UV radiation, temperature changes, and microbial activity, these chemicals are released into the soil and water systems [2]. This process is exacerbated by the fact that phthalates are not chemically bound within the plastics but are rather mixed in, making them more susceptible to leaching when the matrix of the plastic deteriorates.

Phthalates are classified as persistent organic pollutants (POPs) due to their chemical stability and resistance to biodegradation. This persistence means that once phthalates enter the environment, they can remain for extended periods, leading to long-term contamination. According to Sathyanarayana [2], their stability arises from their molecular structure, which is resistant to the natural processes that typically break down organic materials. As a result,

even minimal exposure to phthalates can lead to significant and prolonged environmental contamination.

The persistence of phthalates such as DMP and DEP poses serious risks to both human health and ecological systems. These compounds can accumulate in soil and water, leading to potential health risks through direct exposure or the food chain. Aquatic ecosystems are particularly vulnerable; phthalates have been shown to affect the reproductive and developmental processes of aquatic organisms [3]. Terrestrial ecosystems are not immune, as phthalates can impact soil health and biodiversity by altering the composition of microbial communities and affecting plant growth [4].

Aim

The primary aim of this review is to examine the effect of phthalate esters (DMP and DEP) emanating from residential and industrial activities, specifically focusing on waste plastic materials and used engine oil.

Objectives

1. To determine the effect of DMP and DEP on the human metabolic system.
2. To determine the effect of DMP and DEP on male and female reproductive systems.
3. To assess the teratogenic effect of DMP and DEP.
4. To investigate the carcinogenicity of DMP and DEP.
5. To determine the effects of DMP and DEP on the general ecosystem.

Routes of Exposure of Phthalates

Plastic waste has received scrutiny by governmental and regulatory bodies. Global plastic use consumes more than 3 million tons of phthalates per year [5]. Due to their ubiquity in the environment, human exposure to phthalates leached from waste plastics is virtually unavoidable. For instance, in China, plastic usage tripled over eight years between 2003 and 2011 and reached over 50 million tons of raw plastics produced and is estimated to keep increasing in the following years [5]. As a result, the relative higher exposure to phthalates was found in China due to the high usage of plastics. In the USA, more than 340 million pounds of phthalates are consumed every year and cause potential health and environmental risks [6]. Phthalates can be easily leached into food, water, and other products applied directly to the human body. The detrimental health and environmental effects have been increasingly studied to assess the extent of the impacts on society. An important phthalate exposure route could have consisted of ingestion, inhalation, and dermal contact mainly via PCPs [5]. Some dairy products, fish, seafood, and oils are found to have a high level of phthalates. For the residents who live near phthalates manufacturing industries, phthalates are more likely to enter the body through absorption via the skin and the polluted air due to fugitive emissions [6]. Phthalates are semi-volatile organic compounds (SVOCs). DEHP and DBP are the main compounds in both indoor and outdoor air phthalates [7]. Dermal absorption also occurs from the daily use of PCPs containing phthalates via plastic packages. Infants are exposed to phthalates by drinking breast milk with their mothers exposed to DEHP and DiNP, and sucking on toys containing DEHP, DBP, and BBP [6]. Phthalates are also found to cross the placenta-blood barrier, which is the major exposure route of the fetus [7].

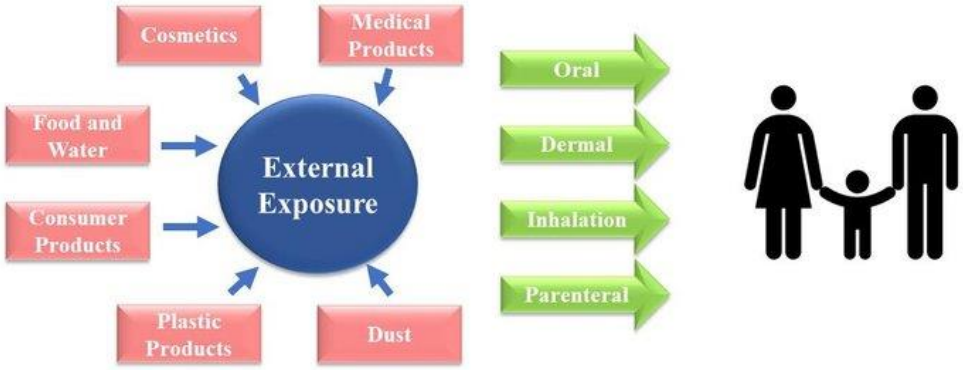

Figure 1: Routes of Exposure of Phthalates [5]

Bio-Metabolism of Phthalates in the Human Body

The bio-metabolism in the human body is very rapid since phthalates have short biological half-lives, about 12 h [9]. Figure 2 presents the metabolic patterns. The first step of metabolism is hydrolyzation after absorption into cells. The second step is conjugation to form the hydrophilic glucuronide conjugate, which is catalyzed by the enzyme uridine 5′-diphosphoglucuronyl transferase [10]. The type of phthalates determines its toxicological fate in the body. Short-branched phthalates are often hydrolyzed to monoester phthalates and then excreted in the urine, while long-branched phthalates mainly undergo several bio-transformations, such as hydroxylation and oxidation, and then excreted in urine and feces as phase 2 conjugated compounds [11]. For example, the DEHP, which has complex branched chains, may be hydrolyzed to mono(2-ethylhexyl) phthalate (MEHP), mono(2-ethyl-5-hydroxyhexyl) phthalate, mono(2-ethyl-5-oxohexyl) phthalate, mono(2-ethyl-5-carboxypentyl) phthalate (MECPP), mono(2-carboxymethylhexyl) phthalate (MCMHP) or other metabolites. The metabolites of DEHP above could also be found in serum. According to animal experiments, exposure to MEHP causes reproductive dysfunction in female zebrafish, which is possibly due to the alteration in endocrine activities (elevated cortisol levels) [12]. In addition, according to half-life and distribution pattern, previous studies indicated that MECPP in urine and MCMHP in serum could be used as suitable biomarkers [10]. Most of the phthalates and their metabolites can be found in urine and faeces, but some phthalates compounds (e.g., DEHP) and their metabolites can also be excreted in sweat [13]. Wittassek and Angerer [14] found that the oxidative

metabolism of DEHP is age-related. Younger children at the age of 6–7 years excrete more oxidative DEHP metabolites compared to mono-(2-ethylhexyl) phthalate (MEHP), one of the metabolites, than adults aged between 19 and 90 years.

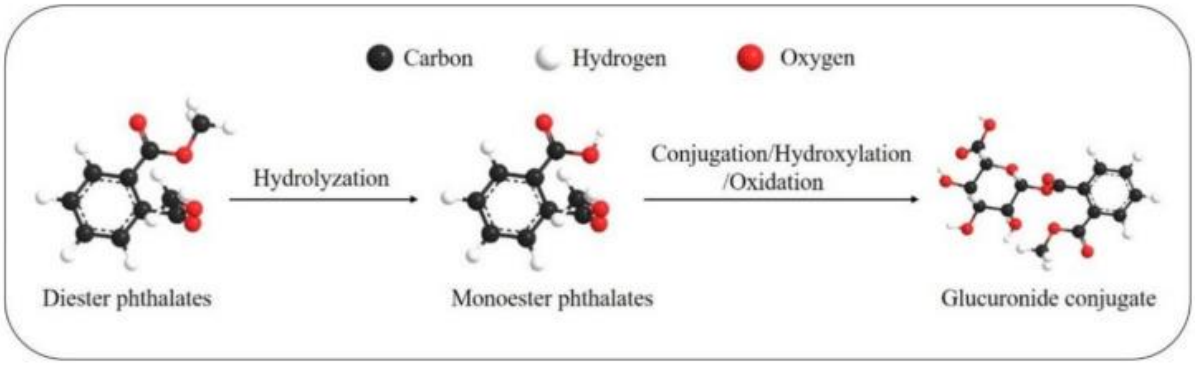

Figure 2: The metabolic pathway for phthalates [15]

Phthalates Toxicology and Risk Assessment

In rodent studies, it was found that phthalates have low acute toxicity with a median lethal dose (LD_{50}) of 1–30 g/kg bodyweight, and its toxicity is mainly concentrated in the liver, kidney, thyroid gland tissue, and testis [15]. Evidence for adverse effects on reproduction and development in animals and humans is ample. According to the laboratory experiment on pregnant animals, exposure to DBP at 100 mg/kg bodyweight/day is toxic to fetal development [15]. The no-observed-adverse-effect level for DEHP to humans is 4.8 mg/kg bodyweight/day and the tolerate daily intake (TDI) is 48 µg/kg body weight [16]. Studies found that low molecular phthalates, such as DEP, can acutely irritate the skin, conjunctiva, and mucous membrane of the oral and nasal cavities [17]. Phthalate exposure is associated with adverse developmental effects in terms of increased prenatal mortality, reduced growth and birth weight, skeletal, visceral, and external malformations in rodents [15]. Experiments on male rats found that the nervous system is rather sensitive to low doses of DEHP exposure during puberty [18]. The impacts of phthalates on human beings vary from gene expression to physiological changes. High molecular weight phthalates exposure is found to cause methylation status of imprinted genes, which could be directly related to androgen response, estrogen response, protein secretion, and spermatogenesis [19,20]. Human epidemiological studies have shown a significant association between phthalates exposures and adverse reproductive outcomes in both women and men, for instance, type II diabetes and insulin resistance, overweight/obesity, allergy, asthma [15]. Among all phthalates, DEHP was most frequently tested and had the highest concentration in food, except in beef where di-n-octyl phthalate (DnOP) has the highest concentration [21]. In household dust, DEHP (median contamination level in indoor air: 400–700 ng/m^3, (max. 410,000) mg/kg) has been found at high concentrations [15]. Evidence found that DEHP was significantly related to insulin resistance and higher systolic blood pressure and the reproduction system problems, including earlier menopause, low birth weight, pregnancy loss, and preterm birth [22]. During 2003–2004, the National Health and Nutrition Examination Survey (NHANES) found that the US population has been widely exposed to phthalates [23]. Women were found to be exposed at higher levels than men due to frequent use of PCPs (e.g., soaps and cosmetics) [23]. A systematic review and meta-analysis concluded that phthalates metabolites MBzP and MiBP were negatively associated with breast cancer among females [24]. Risk assessment of chemicals involves a comparison of the actual level of exposure to the acceptable level of exposure, mostly TDI values. But phthalates are a group of chemicals with individually different TDIs but with similar metabolites and impacts on the human body. Hence, the cumulative risk assessment is more appropriate to measure the risk of phthalates presented by summing the hazard quotient (HQ) as a hazard index (HI). Søeborg, et al. measuring the HQs and HIs of five phthalates, including DEHP, DBP, BBP, DINP, and DIDPA, found that DEHP and DBP contributed the greatest proportion of the HI. According to the NHANES data, the HI values of 10% of pregnant women exceeded 1, which means 10% of pregnant women were negatively impacted by phthalates, meanwhile, the Study for Future Families (SFF) found that the HI values of 4–5% of infants exceeded 1 [25].

Effects of DMP and DEP on the Human Metabolic System

Phthalates, such as dimethyl phthalate (DMP) and diethyl phthalate (DEP) are known to be lipophilic, which allows them to be absorbed into the body through ingestion, inhalation, and dermal contact. Once in the body, DMP and DEP undergo metabolic transformations into their respective monoesters—monomethyl phthalate (MMP) and monoethyl phthalate (MEP), respectively [26]. These metabolites can interfere with critical metabolic pathways, leading to a range of adverse health effects, particularly concerning metabolic disorders.

One major concern regarding phthalate exposure is its link to metabolic syndrome, obesity, insulin resistance, and glucose homeostasis disruption. Metabolic syndrome, characterized by a combination of hypertension, hyperglycemia, dyslipidemia, and abdominal obesity, is a precursor to more severe conditions such as type 2 diabetes and cardiovascular diseases. The mechanisms through which DMP and DEP induce these disorders revolve around their interaction with key metabolic regulators. For instance, a study by Varshavsky et al. [27] found that even low-dose exposure to DEP increases the risk of insulin resistance and type 2 diabetes by affecting the function of adipocytes and pancreatic beta cells. Adipocytes, which store and release fat, and pancreatic beta cells, responsible for insulin production, are crucial in maintaining glucose balance. DEP exposure can impair these cells' function, leading to disrupted glucose metabolism and an elevated risk of diabetes.

Further research has highlighted the role of DMP and DEP in altering lipid metabolism. Lipid metabolism, which involves the breakdown and synthesis of fats such as cholesterol and triglycerides, is essential for maintaining overall metabolic health. According to Kim et al. [28], phthalates can alter the activity of key enzymes involved in cholesterol and triglyceride

regulation. This disruption primarily occurs through the interaction of phthalates with peroxisome proliferator-activated receptors (PPARs), which play a central role in the regulation of lipid and glucose metabolism. PPARs are nuclear hormone receptors that regulate the expression of genes involved in lipid storage, insulin sensitivity, and fatty acid oxidation. Phthalates' binding to these receptors disrupts their normal function, resulting in elevated levels of triglycerides and cholesterol in the bloodstream, further exacerbating the risk of metabolic syndrome.

Effects on Male and Female Reproductive Systems

One of the most studied effects of phthalates, including DMP and DEP, is their impact on the reproductive system. As endocrine disruptors, these compounds can interfere with hormone signalling pathways, leading to reproductive toxicity [29]. Phthalates have been shown to adversely affect both male and female fertility by altering the production and action of sex hormones such as testosterone and estrogen.

Male Reproductive Health

Male reproductive health can be significantly influenced by environmental factors, including exposure to chemicals like Diethyl Phthalate (DEP) and Dimethyl Phthalate (DMP). These chemicals are commonly found in personal care products, plastics, and other consumer goods, which increases the likelihood of chronic exposure. Studies have shown that such exposure is associated with detrimental effects on sperm quality and male fertility.

In a comprehensive study by Wang et al. [30], it was found that men exposed to DMP and DEP exhibited decreased sperm quality, including lower sperm motility and count. Sperm motility and count are critical parameters in male fertility, as they influence the ability of sperm to reach and fertilize the egg. The same study also reported that exposure to these chemicals reduced testosterone levels, which can lead to hormonal imbalances and contribute to infertility. Testosterone is essential for the development of male reproductive tissues, sperm production, and overall sexual function, so any disruption in its levels can have far-reaching effects on reproductive health.

Furthermore, prenatal exposure to DEP has been linked to developmental abnormalities in male reproductive organs. Jiang et al. [1] conducted animal studies that highlighted the risk of cryptorchidism (undescended testes) and hypospadias (abnormal placement of the urethral opening) in male offspring exposed to DEP during gestation. These conditions can impair reproductive function later in life and are significant markers of disrupted reproductive development.

Epidemiological research has also reinforced the connection between phthalate exposure and male infertility. Wu et al. [31] found that men with higher urinary concentrations of phthalate metabolites, particularly those derived from DEP, had significantly lower sperm motility and sperm counts. Such findings underscore the importance of regulating phthalate exposure, especially in vulnerable populations like pregnant women and men of reproductive age.

Female Reproductive Health

Female reproductive health is particularly vulnerable to environmental contaminants, including phthalates like dimethyl phthalate (DMP) and diethyl phthalate (DEP). These compounds, commonly found in personal care products, plastics, and household items, have been associated with significant health concerns, especially regarding their impact on the menstrual cycle and ovarian function. Research has shown that phthalate exposure can disrupt the endocrine system, leading to hormonal imbalances that affect female reproductive health.

Phthalates are classified as endocrine-disrupting chemicals (EDCs) due to their ability to interfere with the hypothalamic-pituitary-gonadal (HPG) axis, a critical system that regulates reproductive hormone production and reproductive function in females. Disruption of the HPG axis can result in alterations in hormone levels, particularly estrogen, which is essential for regulating the menstrual cycle and supporting ovarian function. These disruptions can lead to a range of reproductive health issues, including polycystic ovary syndrome (PCOS), menstrual irregularities, and even early onset of menopause. The study by Ferguson et al. [32] highlights the connection between phthalate exposure and ovarian dysfunction, indicating that these chemicals can impair follicular development and reduce estrogen production, both of which are crucial for fertility and maintaining normal reproductive processes.

Furthermore, exposure to phthalates has been linked to adverse pregnancy outcomes, including reduced fertility. By impairing ovarian function and disrupting the delicate balance of reproductive hormones, DMP and DEP exposure can reduce the chances of conception and increase the risk of complications during pregnancy. Women exposed to higher levels of phthalates may face a higher likelihood of infertility, miscarriages, and other complications during pregnancy.

The role of environmental toxins like DMP and DEP in female reproductive health underscores the need for better regulation of chemicals in everyday products and further research into their long-term effects. As more studies emerge, it is becoming increasingly clear that reducing exposure to phthalates and other endocrine-disrupting compounds is vital for safeguarding female reproductive health, particularly in populations with high exposure risk.

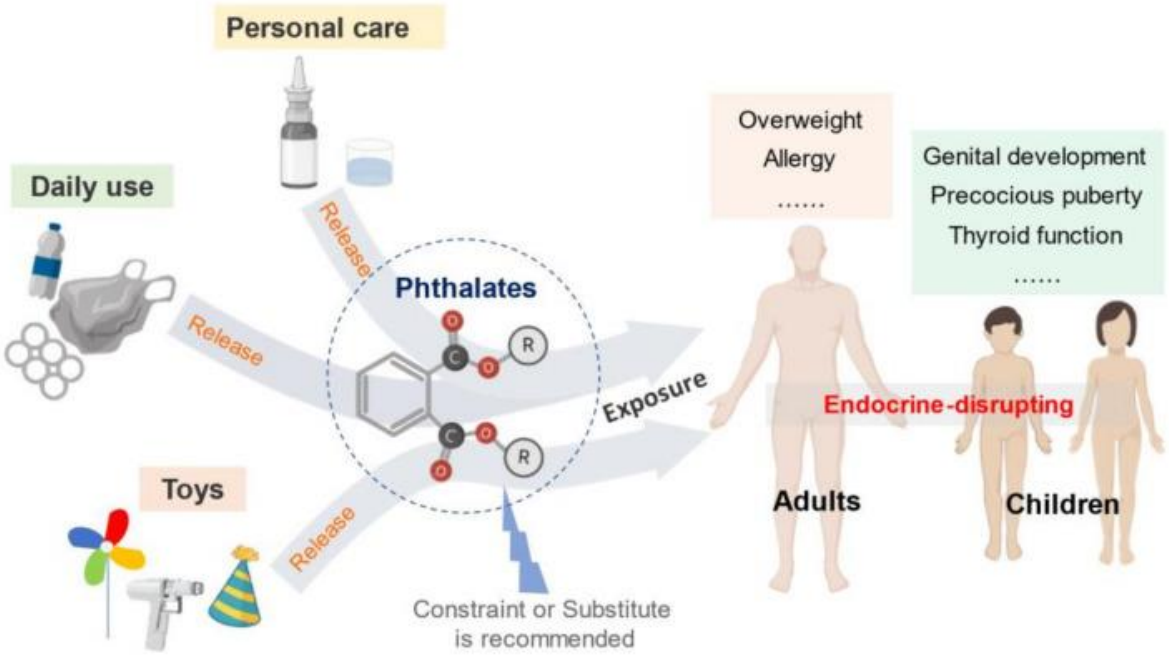

Figure 3: Phthalates application and the impacts on human health [15]

Teratogenic Effects of DMP and DEP

Teratogenicity refers to the potential of certain substances to disrupt fetal development, leading to congenital abnormalities or birth defects. Among these substances, Diethyl phthalate (DEP) and Dimethyl phthalate (DMP) have been reported to exhibit teratogenic properties, particularly when exposure occurs during critical stages of fetal development. These compounds belong to the phthalate family, which is widely used in consumer products like plastics, cosmetics, and personal care items. Phthalates can leach into the environment and are absorbed into the human body, raising concerns about their potential impact on fetal development.

Recent studies have demonstrated that prenatal exposure to phthalates, including DMP and DEP, can lead to structural abnormalities in vital organs such as the heart, liver, kidneys, and lungs [33]. Animal models have been used to investigate these effects, where findings suggest that phthalate exposure during pregnancy can result in malformations and functional impairments. For instance, Patel et al. [33] demonstrated that fetal rats exposed to high doses of DMP and DEP exhibited developmental delays, heart defects, and renal dysplasia. These adverse outcomes were particularly significant during the critical period of organogenesis, when the formation of major organ systems occurs.

In addition to structural abnormalities, studies have suggested that DMP and DEP can interfere with neurodevelopment. Exposure to these compounds during pregnancy has been associated with neurodevelopmental delays, impaired cognitive function, and behavioral abnormalities in offspring [34]. This teratogenic potential is not limited to physical malformations but extends to the brain's functional and behavioral aspects. For instance, research by Braun et al. [34] highlighted the relationship between prenatal phthalate exposure and attention-deficit disorders, reduced IQ, and social difficulties in children.

The underlying mechanisms of the teratogenic effects of DMP and DEP are believed to involve disruptions in gene expression and epigenetic modifications during fetal organogenesis. One major pathway implicated in phthalate-induced teratogenicity is the Wnt signaling pathway, which plays a critical role in cell growth, differentiation, and tissue patterning during embryonic development [35]. Disruption of this pathway by phthalates can lead to abnormal organ formation and functional deficits. In a study by [35], prenatal exposure to DEP dysregulated the expression of genes involved in the Wnt signaling pathway, leading to abnormal brain development in mice.

Another critical concern with phthalates is their ability to cross the placental barrier, allowing direct interaction with the developing fetus. This permeability raises the risk of systemic exposure to the fetus, heightening the potential for teratogenic effects. The placenta's inability to block these compounds underscores the importance of understanding and mitigating prenatal exposure to phthalates, particularly during sensitive periods of gestation [19].

Carcinogenicity of DMP and DEP

The carcinogenic potential of phthalates, particularly dimethyl phthalate (DMP) and diethyl phthalate (DEP), has raised significant concerns due to their widespread use in consumer products and their persistence in the environment. Phthalates are known endocrine disruptors that interfere with hormonal balance, and their role in cancer development is a subject of ongoing research. Studies have demonstrated that DMP and DEP can induce carcinogenic effects through several molecular mechanisms, including oxidative stress, DNA damage, and interference with apoptosis.

One of the primary mechanisms by which phthalates like DEP promote carcinogenesis is through the generation of reactive oxygen species (ROS). ROS are highly reactive molecules that can cause oxidative stress, leading to DNA damage, chromosomal aberrations, and mutations. These alterations in the genome can disrupt normal cell function and promote tumorigenesis. Shen et al. [36] highlighted that phthalate-induced ROS production could lead to a cascade of cellular events, including inflammation and the activation of oncogenic pathways, which contribute to cancer development. Additionally, phthalates can impair the body's natural antioxidant defenses, exacerbating oxidative stress and further promoting DNA damage.

Experimental studies have provided strong evidence linking DEP to various forms of cancer. For instance, DEP exposure has been associated with an increased risk of breast, liver, and testicular cancers [37]. DEP may exert its carcinogenic effects by disrupting estrogen and androgen signaling pathways, both of which are critical in the regulation of cell proliferation and differentiation. Ji et al. [7] conducted a study on rodents, showing that long-term exposure to DEP significantly promoted the development of hepatocellular carcinoma. The researchers observed increased ROS production and suppression of DNA repair mechanisms, indicating that DEP may compromise the integrity of the genome, leading to tumor formation. Moreover, epidemiological studies have suggested a correlation between phthalate exposure and increased cancer risk in humans. Lee et al. [38] reported that populations exposed to higher levels of phthalates, including DEP, exhibit a higher incidence of breast and liver cancers. However, despite these associations, more research is necessary to establish a direct causal relationship between phthalate exposure and cancer in humans.

Effect of Phthalates on Children

When it comes to the effect on children, epidemiological studies about phthalates toxicity focused on pregnancy outcomes, genital development, semen quality, precocious puberty, thyroid function, respiratory symptoms, and neurodevelopment [39]. Table 1 summarizes the health impacts on children. Among the epidemiological studies, it was revealed that exposure to phthalates adversely affected the level of reproductive hormones (luteinizing hormone, free testosterone, sex hormone-binding globulin), anogenital distance, and thyroid function [39]. Altered thyroid function is found to be associated with thyroid cancer [40]. A recent Chinese study concluded that phthalates exposure is related to disrupted arginine and proline metabolism, resulting in the development of overweight and obesity among school-age children [41]. A 20-year birth cohort study found that prenatal phthalate exposure is negatively associated with height and weight during infancy and positively associated with height during childhood [42]. Another prospective study demonstrated that DiDP is associated with respiratory system health among boys aged under 5 years [43]. Phthalates have also been found to be linked to social impairment of children, the same as BPA [44]. Previous studies have found that infants and toddlers when contacting polymer toys may be exposed to levels of 5 to 44 μg/kg body weight/day of DiNP [15]. Another study reported that around 20% of the children have been exposed to higher levels of phthalates than the cumulative TDI for DEHP and DBP [45]. In 2013–2014, over half of the tests for phthalates for persons aged over 6 years found positive results for DEHP, and almost all women and children had DBP metabolites, according to the National Center for Health Statistics (NCHS) [6]. In Austria, few exceedances of TDI values of phthalates were observed among children, whereas the exceedances of TDI-based HIs for adults were in rare cases [46]. A study measuring the phthalates in air and dust in California (USA), found that 82–89% of children had DBP exposure exceeding the reproductive health benchmarks, and 8–11% of children aged less than 2 years were exposed to DEHP exceeding cancer benchmarks [47]. A study conducted in China found that the cumulative risk because exposure to phthalates was higher in preschool children aged 3–6 years compared to the reports in German and Danish [48,49]. Rice, vegetables, and flour are the main sources of DEHP in China [50]. Xu, et al. [51] reported that phthalates, mainly DEHP, DnBP, and DiBP, exist in commonly used plastic express packing bags, suggesting these bags may be the current main source of exposure of the population to phthalates. In addition, the intake of vegetables grown in plastic greenhouses made children experience higher (nearly 3 times) DEHP and DnBP exposure than adults [26]. Foods containing fat (e.g., dairy and meat) tend to be more likely to absorb phthalates from the packaging. From the review of the literature, we believe that the exposure pathway depends on the food, air, or products containing phthalates.

Category	Health Concerns
Endocrine systems	Weight (overweight and obesity) and height
	Type II diabetes and insulin resistance
	Thyroid function and increased risk of thyroid cancer
	Higher systolic blood pressure
	Anogenital distance
	Precocious puberty
	Males: genital development, semen quality
	Females: pregnancy outcome (pregnancy loss and preterm birth, low birth weight), reproductive hormones (including lueinizing hormone, sex hormone-binding globulin, earlier menopause)
Others	Respiratory system: allergy and asthma Nervous system: delayed neurodevelopment, social impairment

Table 1: Health impacts of phthalates on children

Effects on the General Ecosystem

The environmental impact of Di(2-ethylhexyl) phthalate (DHP) and Di-n-butyl phthalate (DNP) is significant, particularly due to their persistence and resistance to biodegradation, which makes them long-lasting pollutants in various ecosystems [50]. These phthalates are commonly released into the environment through the disposal of waste plastic materials and used engine oils, leading to contamination of soil, water, and air. The pervasive nature of these chemicals results in substantial ecological disruption, with far-reaching consequences for different environmental compartments.

Phthalates, once released into the environment, tend to accumulate in soil and aquatic systems, presenting a substantial risk to ecological health. In aquatic ecosystems, research has demonstrated that phthalates can accumulate in aquatic organisms, such as fish and amphibians, adversely affecting their growth, reproduction, and overall survival [30]. For instance, phthalates have been observed to interfere with the endocrine systems of

these aquatic species, leading to a range of detrimental effects, including altered sex ratios, delayed sexual maturity, and decreased fertility [50].

The endocrine disruption caused by phthalates extends beyond individual organisms to impact population dynamics and ecosystem health. In fish and amphibians, phthalates can cause feminization or masculinization of individuals, which disrupts natural reproductive processes and can lead to skewed sex ratios within populations. These disruptions can have cascading effects throughout the food chain, as altered reproductive rates and survival can affect predator-prey relationships and overall ecosystem stability [50].

Moreover, the bioaccumulation of phthalates in aquatic organisms poses risks to higher trophic levels, including humans. As these chemicals move up the food chain, they can concentrate in the tissues of predatory species, which can lead to higher exposure levels in humans who consume contaminated fish and other aquatic organisms. This bioaccumulation not only affects individual species but can also pose significant health risks to humans and other apex predators [30].

Conclusion

The evidence reviewed in this paper highlights the potential dangers of DMP and DEP, both to human health and the environment. These phthalate esters, commonly found in waste plastic materials and used engine oil, have been shown to interfere with metabolic processes, reproductive health, fetal development, and even carcinogenesis. Additionally, their persistence in the environment can have far-reaching consequences for ecosystems. It is imperative that further research is conducted to better understand the mechanisms of toxicity, and that regulatory actions are taken to limit human and environmental exposure to these hazardous chemicals.

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