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Review Paper

Chemicals That Disrupt the Endocrine System and Their Effects on Behavior

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Abstract

Endocrine glands are significant human glands that conduct various activities and have distinct properties. The primary role of these glands is to control the whole system by creating hormones that they create on their own and releasing them directly into the bloodstream for specific action. All their actions are involuntary. They are ductless glands, and their function is controlled by a pea-sized pituitary gland known as the Master Gland. Little has been understood about these glands and how their activities or functions are halted or disrupted by chemicals or other environmental factors. Certain chemicals, such as chlorpyrifos, DDT, insecticides, pesticides, fungicides, and other everyday items such as plastics, paints, furniture, perfumes, toy polishes, electronic gadgets, and food packaging, have been reported to disrupt normal hormonal functions in humans, resulting in a variety of diseases caused by either a lack of production of a specific hormone or increased production of a particular hormone because of the action of these chemicals. Diseases commonly reported because of the action of the chemicals, and daily use items include neurological disorders, behavioral disorders, metabolic dysfunction leading to obesity or weakness, thyroid dysfunction, reproductive

disturbances, and a variety of others that can be fatal and lead to cancer. The researchers refer to all these substances and products as Endocrine Disruptors. Still, much remains to be done, and there is a vast potential for study in this area, which, if targeted appropriately, can be an agent of change in the medical and health sciences field. Humans can be saved from many ailments associated with these disruptors and treated promptly. Given the above, there is an urgent need for study in this field to get familiar with the dangerous and health-related concerns in humans caused by these disruptors, as well as to develop alternatives to the chemicals and objects to rescue mankind from the bad effects of these disruptors.

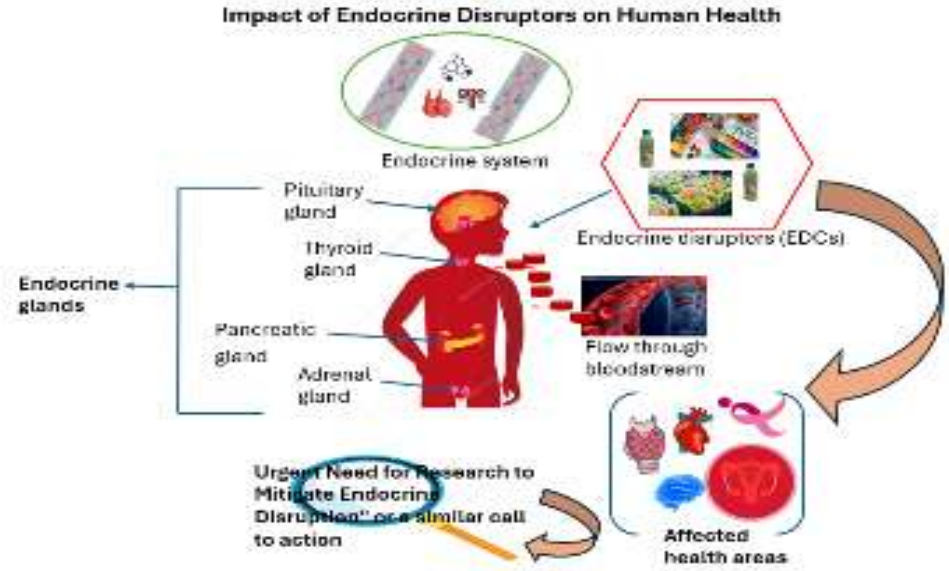


Figure 1. Endocrine-disruptive enter the body

Introduction

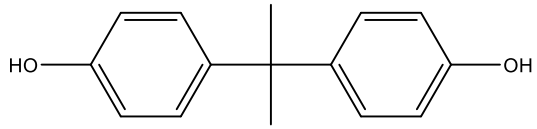
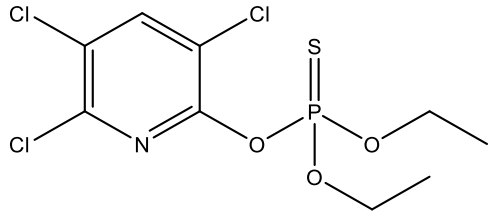
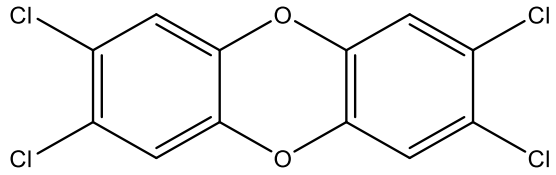
The endocrine and neurological systems interact to develop and exhibit behavior at the same time. Sexual dimorphism in behavior results from variations in mammalian brain structure and function caused by steroid hormones throughout early development. A comparable phenomenon, which may also be seen in animals, demonstrates the relationship in issue (Cooke et al., 1998, De Vries, 2004). Even though brain growth is tightly regulated and coordinated, exogenous drugs can interfere with the activity of endogenous hormones (Knudsen, 2004). Chemicals that disrupt the endocrine system (endocrine-disrupting chemicals or EDCs) occur in many different forms and have been found to have detrimental health impacts on both adults and their children. Because of these findings, EDCs have been classified as endocrine disruptors. Chemical compounds that can disrupt endocrine function include plasticizers, flame retardants, fungicides, pesticides, medications, heavy metals, and

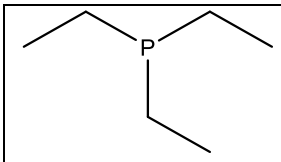
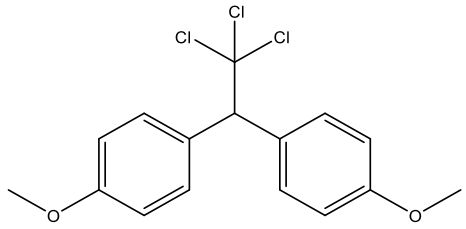
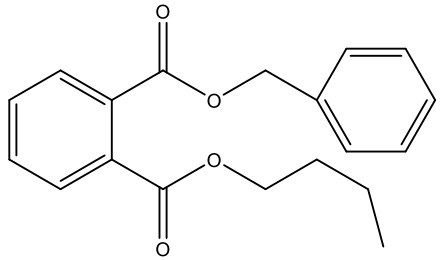
phytoestrogens. These and other EDCs can employ several direct and indirect ways to inhibit, block, or enhance endogenous hormone function as shown in **Figure 1**. For example, they might bind to hormone receptors (agonistically or antagonistically), limit hormone release, or decrease the number of hormone receptors (Gore et al., 2015).

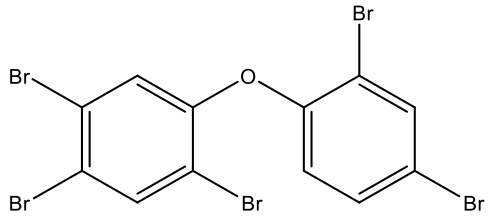
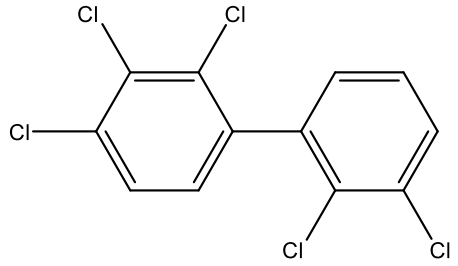
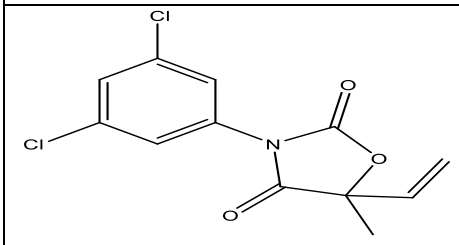
Several elements influence whether EDCs have long-term repercussions. A variety of parameters must be addressed, including the organism's genetic sensitivity, dosage, duration of exposure, and developmental window. Endotoxin-producing organisms (EDOs) have received a lot of interest from the public since their discovery in the scientific community (Hotchkiss et al., 2008). The global epidemic of sex-based disparities in disease prevalence may be linked to an increase in endocrine-related neurodevelopmental disorders like ADHD and ASD (McLachlan, 2016). This behavior cannot be explained merely via genetics. The brain's capacity to discriminate sexual cues may be compromised by developmental exposure to EDCs. Although many illnesses' causes remain unclear, this tendency continues. Several epidemiological and experimental research have found that exposure to environmental contaminants during early life is linked to aberrant neurobehavioral development. The goal of this essay is to look at the existing facts on how exposure to EDCs might influence behavior. Bisphenol A, popularly known as BPA, is likely the most widely used and researched EDC, despite the discovery of hundreds of other potentially harmful compounds. As an example, we shall discuss the repercussions of being exposed to this fatal poison at a young age.

BPA, an Innovative Endocrine Disrupting Chemical

BPA is prevalent in polycarbonate plastics, epoxy resins, and many other common home objects, thus we must pay attention to it. In affluent nations, the great majority of people are exposed to BPA through the consumption of contaminated food and drink (Bushnik et al., 2010) More than 90% of adults in developed nations have detectable amounts of BPA in their systems (Calafat et al., 2005), (LaKind & Naiman, 2015). Childhood exposure to neurotoxic and neuroendocrine-disrupting medications is significant due to fast development and heightened susceptibility to their effects (Casas et al., 2013), (Von Goetz et al., 2010). The detection of BPA in fetal plasma (Ikezuki et al., 2002), amniotic fluid (Engel et al., 2006), fetal liver (Nahar et al., 2013), and placenta tissue indicates a high risk of BPA exposure during pregnancy (Schönfelder et al., 2002). The developing brain is also more exposed to environmental dangers because the blood-brain barrier has not yet been established (Adinolfi, 1985), (Perera & Herbstman, 2011).

Compounds Structure and Name		**EDCs Source**
Bisphenol A (BPA)  		Thermocouple sheets, medical implants, and plastic bags are just a few of the many products that make use of epoxy resins and polycarbonate polymers.
 **Chlorpyrifos**		The intake of insecticides in agroecosystems and the formulation of these chemicals for consumer use have an impact on chlorpyrifos production. After spraying, the residue was allowed to linger on items such as fruits and vegetables. During the preparation phase, however, the chimneys release fragments of themselves or residue into the air, which eventually settles on the edible items.
 **Dioxins (tetrachlorodibenzo-*p*-dioxin)**		The usage of high-fat foods, such as eggs, animal fats, and dairy products, results in these. These are also formed due to the burning of municipal waste during the production of industrial products.

 **Lead (Triethyl lead)**		Triethyl lead is another element that has several sources and is used in many products. Gasoline, for example, is cracked with a lead component to ensure a complete and uniform combustion in vehicles. A wide range of consumer goods contains triethyl lead. Lead is also a key component of paints used to embellish buildings and walls, and when released into the environment by cars, it reacts with dust. Each of them contributes to water pollution in the same manner as lead does. Lead traces have also been detected in jewelry and children's toys.
 **Methoxychlor (MXC)**		Pesticides, which are used to kill or destroy pests from crops, gardens, and animals, are another contributing factor. Air, land, and water are all immediately contaminated as a direct result of this.
 **Phthalates (Benzyl butyl phthalates)**		Food packaging, plastics, pharmaceutical waste, carpet and wall covering backing, vinyl flooring, and high-fat meals like animal fats, eggs, and dairy items may all contain similar quantities of benzyl butyl phthalates.

 **Polybrominated diphenyl ethers (PBDEs) (pentabromodiphenyl ether)**		Some potential sources of PBDEs are listed below: the air we breathe, flame retardants, pollutants present in home dust, sediments and soils, dairy products, seafood, and meat.
 **Polychlorinated biphenyls (PCBs)**		Disruptors like PCBs are often present in high-fat diets including animal fats, eggs, and dairy products. Besides capacitors, other potential sources of contamination include groundwater, air near industrial areas, electrical transformers, and transformer oil.
 **Vinclozolin**		Since vinclozolin is often included in fungicides, it's reasonable to assume that it serves a purpose.

A growing organism may be exposed to toxins that are invisible to an adult's nervous system but fatal to them. Despite dosages that are lower than the current US Food and Drug Administration NOAEL of 5 mg/kg body weight per day, BPA has been associated to serious neurobiological and mood-related behavioral repercussions (Blair et al., 2000). The FDA deems these amounts safe, even at lower levels. Despite extensive studies, there is no clear knowledge

of how BPA influences brain circuit development in humans (Kuiper et al., 1998). BPA was first designed as a synthetic estrogen, which led to the notion that it has estrogenic effects since its inception. Previously, it was believed that most BPA's negative effects were caused by its attachment to estrogen receptors (ERs). Nonetheless, BPA's affinity for ERa and ERb in cell culture experiments is 10,000-100,000-fold lower than that of endogenous estrogen, indicating that it is unlikely to exert its effects purely via traditional ER-dependent nuclear pathways (Waller et al., 1996). BPA exerts its rapid, nongenomic effects via membrane-bound ERs, with a very low affinity for other steroid receptors (Belcher & Zsarnovszky, 2001). According to current research, a person's gender and geographic location can impact estrogen receptor expression and DNA methylation patterns in the brain (Kundakovic et al., 2015). EDCs can alter the brain and behavior in many ways throughout one's life due to their numerous modes of action.

Changes in Sexual Dimorphism Behavior May Reflect Hormonal Disruption

Sex differentiation is the process by which the physical and physiological differences between male and female brains emerge. Before the current study, it was considered that gonadal hormones played an important role in sexual identity formation (McCarthy, 2016). A male brain grows with gonadal hormones, but a female brain develops without them (McCarthy & Nugent, 2013). However, it was recently revealed that this mechanism is more sophisticated than previously imagined. A variety of hormonal, genetic, and epigenetic variables impact the development of sexually dimorphic physiology and behavior (Schwarz & McCarthy, 2008).

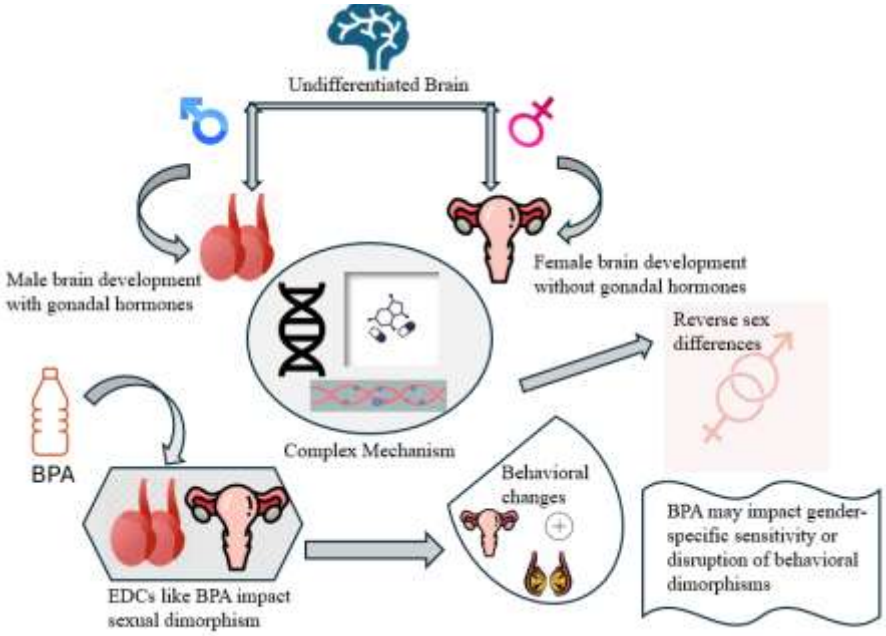


Figure 2. Sex Differentiation and the Role of Endocrine Disrupting Chemicals (EDCs)

Several studies have found that early-life exposure to EDCs like BPA can impair sexual dimorphism. BPA has been shown to have gender-specific effects on behavior in both animal research and human epidemiological investigations (Frye et al., 2012). Given the rich and diverse base of sex differences in behavior (Inadera, 2015), it is not unexpected that BPA might lessen, eliminate, or even reverse these differences as revealed in **Figure 2**. In certain circumstances, BPA appears to impact men and women differently, although it is unclear whether these variations are caused by gender-specific sensitivity or disturbance of behavioral dimorphisms.

Animal Testing Results

The behavioral and neurological consequences of prenatal and neonatal BPA exposure have mostly been examined in rats. These findings will be described here, with an emphasis on research that employed BPA levels at or below the FDA's NOAEL (5 mg/kg body weight per day).

Actions requiring both intellectual curiosity and emotional investment

Numerous studies on mice have found that BPA exposure during development may exacerbate anxiety and exploration-related behaviors (Rochester, 2013). It is crucial to note that the findings might vary greatly based on a variety of circumstances, such as the test subject's age, the animals utilized, and the test subject's gender. Despite the study's contradictory findings on the effects of BPA on developing teens, it indicates that BPA has distinct effects on males and women. According to two research done on young C57BL/6J mice, exposure to BPA during pregnancy or neonatal period enhanced anxiety in males but did not affect females (Cox et al., 2010; Matsuda et al., 2012A third research, however, showed the exact opposite outcome in CD-1 mice. More recent research of Sprague Dawley rats found that prenatal exposure to BPA did not affect their exploratory or anxious behavior (Rebuli et al., 2015). Women are often more vulnerable to the anxiety-inducing effects of BPA exposure in early life than men. It is worth noting that this applies even when males are exposed to the same quantity of BPA. Adult studies have more solid results (Gioiosa et al., 2013). Female CD-1 mice had a behavioral pattern identical to that seen in adult male control mice exposed to 10 mg/kg BPA (Hicks et al., 2016). The same outcomes were seen in adult mice. It is consistent with previous research indicating that prenatal BPA exposure may minimize or eliminate sex differences often observed in adult rats across several behavioral paradigms used to measure anxiety (Jones & Watson, 2012). Several animal models, including zebrafish, voles, and numerous other types of rodents, as well as nonhuman primates, have demonstrated that BPA exposure during embryonic development

affects anxiety-related behavior (Kubo et al., 2003), (Sullivan et al., 2014). According to the World Health Organization (FAO/WHO, 2011), developmental exposure to BPA has profound effects on the brain and behavior (Kundakovic et al., 2013), (Nakagami et al., 2009). This result was reached after examining several research papers and animal models (Arambula et al., 2016). In both the hypothalamus and amygdala, ER-related alterations in gene expression have been implicated in this behavioral disorder (Cao et al., 2013). A recent study has also discovered that anxiety-like behaviors vary by gender in adult BALB/c mice (Patisaul et al., 2012). These effects were associated with DNA methylation and ERa mRNA levels in the hypothalamus (Kundakovic et al., 2015).

The data lend credence to the concept that BPA affects anxiety-related behavior via an epigenetic mechanism (Baldwin et al., 2017). Phthalates, polychlorinated biphenyls, and certain flame retardants (thyroid disruptive) are examples of endocrine-disrupting substances (Gillette et al., 2014). Lead exposure during development may have led to a variety of negative neurobehavioral consequences (Cervantes et al., 2005). Rats, cats, and nonhuman primates have been reported to demonstrate increased hyperactivity, impulsivity, and aggression after being exposed to developmental lead (Li et al., 2003). These facts are consistent and significant . Even though lead and other metals are not EDCs in the usual sense, they can interfere with hormone function in some cases (Miao et al., 2015).

Learning and Memory

It has also been demonstrated that early exposure to BPA causes cognitive impairments. Male mice outperform female mice on spatial learning and memory tests under usual settings. Surprisingly, BPA exposure in early life has been repeatedly proven to minimize this gender gap (Carr et al., 2003). Numerous studies in rats and mice have revealed that prenatal and neonatal exposure to BPA impairs spatial memory in juvenile and adult men (Kumar & Thakur, 2014), (Tian et al., 2010). Male ICR mice exposed to prenatal BPA (0.5 and 5 mg/kg bw/day) showed substantial alterations in spatial memory as they matured into adolescents and adults (Xu et al., 2010). Despite this, little is known about how BPA affects spatial memory and learning in women, and available research is inconsistent (Xu et al., 2007), (Liu et al., 2016). Different studies have connected alterations in spatial memory to altered dendritic spine density and shape, as well as reduced NMDA glutamatergic receptor and ERb expression in the hippocampus after BPA exposure (Eilam-Stock et al., 2012), (Xu et al., 2010). Laboratory studies have linked environmental carcinogens (EDCs) such as chlorpyrifos, polychlorinated

biphenyls, polybrominated diphenyl ethers (PBDEs), and dioxin to cancer (Kakeyama & Tohyama, 2003). CD-1 mice exposed to PDBE during pregnancy developed weaker motor abilities as adolescents and adults. Studies on mice have shown that neonatal exposure to PBDE congeners reduces hippocampus cholinergic function and causes lifetime learning and memory deficits. To get these results, all these studies were merged (Viberg et al., 2003).

Behaviors Relating to Parents, Social and Sexuality

A few studies have found changes in parental, social, and sexual behaviors in mice following developmental exposure to BPA, although the evidence is weak (Adewale et al., 2011). There have only been two research studies that look at the link between prenatal BPA exposure and subsequent mother behavior. An experiment found that exposing pregnant CD-1 mice to 10 mg/kg body weight per day of BPA reduced the amount of time they spent huddling or nursing their young (Palanza et al., 2002). Second, research on Wistar rats reached similar results concerning the effects of BPA exposure throughout pregnancy and the animal's lifetime at a level of 5 mg/kg . The effects of prenatal BPA exposure on adult paternal conduct are uncertain. Conventional rat toxicity models are unlikely to provide biparental care for the young. Despite limited and inconsistent evidence, it appears that female social behavior is more vulnerable to disruption than male social behavior following prenatal exposure to BPA (Boudalia et al., 2014). During prenatal development, C57Bl6J mice were administered 1.25 mg of BPA to improve their social play behaviors (Wolstenholme et al., 2011). Prenatal exposure to BPA at a dose of 40 mg/kg decreased social play in female Sprague-Dawley juvenile rats (male rats were not examined in this study) (Porrini et al., 2005). Prairie voles, a more cooperative animal than laboratory rats, were used to study gender and age-related differences in social behavior. During pregnancy, women exposed to daily dosages of 5 and 50 mg/kg body weight did not acquire mate preferences, but male adolescents exposed to the same level displayed decreased interest in novel social circumstances. Dopaminergic neurons in the bed nucleus of the stria terminalis, oxytocin-producing neurons in the paraventricular nucleus of the hypothalamus, and vasopressin-producing neurons in the hypothalamus were represented differently in male and female subjects. There is some, however ambiguous, evidence that early exposure to BPA may influence sexual behavior later in life (Adewale et al., 2009). Two animal studies found a slight decrease in male sexual performance (delay, introduction, and ejaculation), although human trials have not confirmed this (Picot et al., 2014). Although there has been some discrepancy in female-specific research, most studies have concluded that BPA exposure during pregnancy or early infancy has no substantial effect on a woman's sexual behavior following

pregnancy or childbirth. The lordosis reaction, as well as the hopping and darting reactions, are commonly employed to evaluate female perceptive and receptive behaviors in rats. It was discovered that 0.05 mg/kg BPA exposure during pregnancy decreased female adult Wistar rats' hopping and darting activity while leaving lordosis behavior unaffected (Monje et al., 2009). Sprague-Dawley rats treated to 40 mg/kg of BPA prenatally demonstrated a little increase in lordosis behavior as adults (Farabollini et al., 2002). Furthermore, previous research has found no link between prenatal exposure and a woman's risk of becoming pregnant or taking birth control (Ryan et al., 2010). EDCs reported in animal research include methoxychlor, polychlorinated biphenyls, and phthalates (Suzuki et al., 2004). The data support the idea that PCBs cause cancer. Exposure lowers lordosis and mating chance in developmentally susceptible rats by inhibiting both receptive and preceptive sexual behavior (Steinberg et al., 2007).

Table 2. BPA (bisphenol A) exposure and its impact on parental, social, and sexual behaviors in various animal studies

Study Type	Species	BPA Dosage	Behavioral Effects Observed	Evidence
Parental Behavior	CD-1 Mice	10 mg/kg/day	Reduced time spent huddling or nursing their young	Evidence is weak
	Wistar Rats	5 mg/kg/day	A similar reduction in maternal care observed	Limited research
	Conventional Rats	Not specified	Uncertain effects; biparental care unlikely	Lack of evidence

Social Behavior	C57Bl6J Mice	1.25 mg/kg	Improved social play behaviors	More sensitive than male behaviors
	Sprague-Dawley Rats	40 mg/kg	Decreased social play observed in female juvenile rats	Male rats not examined
	Prairie Voles	5 and 50 mg/kg/day	Females did not develop mate preferences; males showed reduced interest in novel social situations	Gender and age differences observed
Sexual Behavior	Wistar Rats	0.05 mg/kg	Decreased hopping and darting activities; lordosis behavior unaffected	BPA affects some female sexual behaviors
	Sprague-Dawley Rats	40 mg/kg	Slight increase in lordosis behavior as adults	Limited evidence

Epidemiological Studies

Certain epidemiological research undertaken over the previous decade has revealed that infants exposed to BPA throughout their youth exhibited problematic behaviors. BPA exposure is still

being contested (Bellinger et al., 2007), as are its health repercussions. BPA levels in pregnant women's urine were assessed for the first time in the Health Outcomes and Measures of the Environment Study (HOMES) during 16- and 24-week gestation, as well as after birth. We analyzed children's conduct in the neighborhood and at home using a questionnaire prepared for 2-year-olds. The kids were handed a questionnaire. Increased externalizing behaviors, such as hyperactivity and aggressiveness, were linked to prenatal exposure to greater levels of maternal BPA in girls but not boys. These relationships were only observed in females (Braun et al., 2009). A future investigation looked at the HOMES cohort's third-year behavior and executive performance. There is evidence that moms who are exposed to BPA while pregnant are more prone to experience anxiety, hyperactivity, and depression. Interestingly, two follow-up investigations of the HOMES cohort found no link between maternal BPA levels during pregnancy and autistic behaviors or visual-spatial skills in children ages 4-5 or 7. This is crucial because the HOMES cohort was the focus of this investigation. Based on this research, it is concluded that, while there are measurable and significant effects of chemical exposure on behavior and cognition with long-term consequences, it is extremely unlikely that a clinically recognized condition such as autism will develop, which is notoriously difficult to prove as shown in **Figure 3**. The reason for this is that proving autism is a time-consuming task. Previous longitudinal cohort studies have found that boys are more prone than girls to exhibit bad behavior because of higher BPA levels in fetal urine.

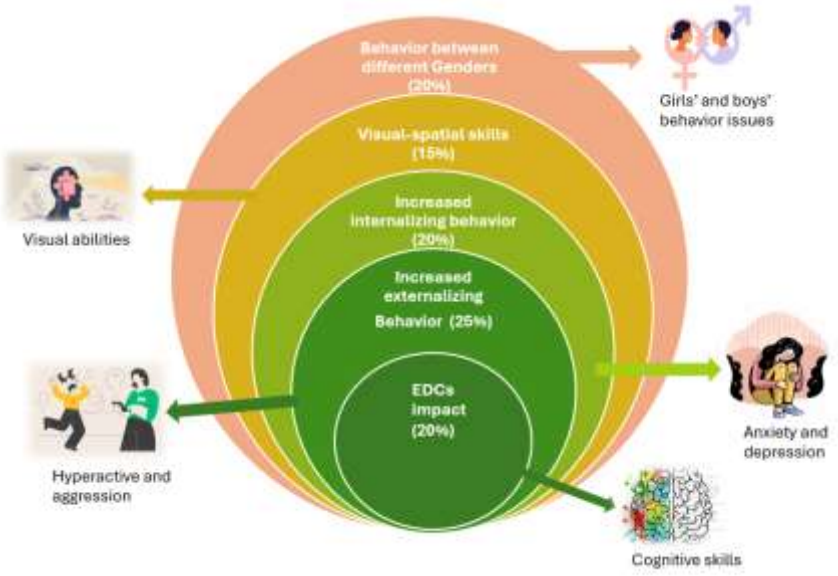


Figure 3. Effects of BPA and other endocrine-disrupting chemicals (EDCs) on child behavior and cognitive development

According to the Center for Children's Environmental Health Cohort (CCCEH), a study of mothers and their African American and Dominican children, higher levels of BPA in maternal urine are related to increased aggressiveness and emotional reactivity in preschool boys. This was demonstrated in a study of men aged 3 to 5 years old (Perera et al., 2012). Researchers in the CCCEH cohort discovered that high levels of BPA exposure during pregnancy were linked to both internalizing difficulties (such as worry and melancholy) and externalizing issues (such as aggression and rule-breaking). Prenatal exposure to BPA is highly connected with behavioral difficulties in boys aged 6 to 10. These problems include emotional discomfort, impatience, and poor attention. In recent epidemiological studies, greater levels of BPA in pregnant women have been associated to cognitive impairment and introverted behavior in toddler males. These epidemiological studies suggest that BPA exposure can have a deleterious influence on neurobehavioral development in children, albeit the effects may differ across sexes. While we don't completely understand what causes the impact, the large amount of animal evidence backs up this conclusion. The study's conclusions are supported by large cohorts of women and their children, BPA examinations throughout numerous trimesters, and documentation of diverse behavioral repercussions. The contradictory findings, however, must be seen in light of the limitations inherent in longitudinal cohort research. A significant issue is that various cohorts have distinct demographics. The scientific literature has also documented disparities due to variances in cognitive testing and interindividual differences in urine BPA levels (Roen et al., 2015). Epidemiological studies have proven the neurobehavioral consequences of prenatal exposure to EDCs. Several persistent organic pollutants (POPs), including polychlorinated biphenyls (PCBs), have been related to impaired cognition, memory, and social skills during prenatal development (Thayer et al., 2015). PCBs may have a favorable effect on brain development. A meta-analysis of prospective cohort studies found that PCB exposure can influence processing speed, language ability, and visual recognition memory. Lead and methylmercury exposure during development may cause executive dysfunction comparable to what is seen in adults. People are routinely exposed to many EDCs and other toxins; therefore, the cumulative effects of repeated exposures are a serious worry. People are continuously in danger of developing health problems because of their ongoing exposure to potentially toxic substances.

It will be possible to synthesize and bio-explore heterocyclic compounds for the treatment of various diseases that are affecting the world today. This study aims to evaluate the biological activities of heterocyclic compounds by developing new and efficient synthetic methods, which

is the basic approach to synthesizing oxadiazoles and thiadiazoles. Infectious diseases can be effectively treated with these molecules. Additionally, these scaffolds have shown that they can be useful in the fight against bacteria, fungal diseases, cancer, depression, as well as anti-urease and anticholinesterase activity. Alzheimer's disease is a progressive disease that affects the brain, leading to cognitive decline and deterioration in memory, thinking, and behavioral abilities. Most of the symptoms of dementia are caused by this condition, which can interfere with daily activities and affect mental ability. Genetics, environment, and lifestyle factors are believed to contribute to Alzheimer's, but its exact cause is unknown. Brain plaques and tangles cause nerve cells to die as a result of interference with normal functioning. It is usually associated with a slow progression of mild forgetfulness, followed by more profound cognitive and behavioral impairments. As the disease progresses, the symptoms of Alzheimer's disease usually worsen over time. It is common for people with dementia to struggle remembering recent events or conversations, to be confused or disoriented, to change their mood, or to have difficulty expressing themselves.

Future Challenges and Directions

Exposure to EDCs during pregnancy or early childhood may impact the development of a variety of neurological and behavioral illnesses. However, there are various difficulties to establishing direct causal links. Neurodevelopmental abnormalities might take a long time to cause behavioral difficulties. Genetics, upbringing, and environment may all have a long-term impact on a person's behavior. Chemical exposures might be difficult to determine. People are frequently exposed to several environmental poisons throughout their lifetimes, sometimes in synergistic or antagonistic combinations with one another. Efforts to imitate "real world" human exposure (low-dose mixtures and chronic exposure) are enhancing the translational value of animal studies in the field of EDC. Ethical and practical concerns restrict research into the neurobehavioral effects of early exposure to EDC. One apparent limitation is the difficulty of reaching human brains in some locations. In vivo and in vitro models can be used to identify peripheral biomarkers of EDC exposure and related diseases. Once found, these biomarkers may be useful in both current and future investigations. Using precise biomarkers might substantially help identify susceptible groups. Approximately 30 years after endocrine disruption was initially proposed, scientists have uncovered numerous unique routes by which EDC affects the brain. More study is needed to determine the influence of brain development on behavior. Additional study is required to have a better understanding of complex behaviors

including activity, sociality, and executive function. As we get a greater knowledge of the biological mechanisms that cause EDCs and other developmental neurotoxins, we will be able to predict and avoid their negative consequences.

Conclusions

According to prior experimental findings, prenatal exposure to BPA can interfere with sexual differentiation of the brain system even at concentrations lower than the present NOAEL, impacting both reproductive and nonreproductive behaviors. During early infancy and later adolescence, the brain is more vulnerable to environmental damage. It suggests that if we take extra precautions to limit our exposure to chemicals, we may be able to reduce BPA levels in children whose parents were concerned about environmental disease-causing substances (EDCs). Despite some indications suggesting children exposed to BPA and other EDCs during development are more likely to have neurological disorders, there is no definite relationship between the two. More research is needed to better understand the relationship between EDC exposure and human health, both mechanistically and epidemiologically. Further research is needed to investigate the combined and cumulative effects of repeated exposure at different crucial periods.

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