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REVIEW

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Human metabolic effects of BPA and the application of a hybrid photocatalytic membrane for BPA contaminated water

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Abstract

Bisphenol A (BPA) is widely used in manufacturing various consumer products and detected in various water sources. Epidemiological studies reveal a correlation between BPA exposure and metabolic system disorders, including obesity, diabetes, and cardiovascular issues. Experimental studies further support these findings by demonstrating the adverse impact of BPA on physiological processes, contributing to the onset of metabolic disorders. Despite its detrimental health effects, removal of BPA poses a formidable challenge due to its intricate molecular structure, resistant to conventional water treatment methods. To address this, our review comprehensively summarizes human BPA exposure data and in vivo/in vitro mammalian studies, offering a comparative analysis of treatment technologies with a focus on documented health impacts. Biological treatment removes BPA efficiently, however, maintaining ideal bacterial populations and controlling biomass concentration provide difficulties that affect operational stability and scalability. In the meantime, despite the high removal rate of physiochemical treatment such as absorption and membrane technology, they consume significant amounts of energy and generate chemical residues that could retain toxicity. In this regard, a hybrid photocatalytic membrane emerges as a promising solution, forming the basis for our comparative evaluation in wastewater treatment and water purification. By effectively degrading BPA and mitigating the BPA toxicity, the photocatalytic membrane helps reduce human exposure to this harmful compound. This technology presents a viable approach to tackle BPA-related environmental challenges while shedding light on its intricate metabolic effects on human health.

Keywords Bisphenol A, Metabolic syndrome, Advanced oxidation processes, Photocatalytic membranes, Water treatment

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1 Introduction

Bisphenol A (BPA; 4,4'-dihydroxy-2,2-diphenylpropane) is widely used as a non-polymer additive in plastics manufacturing, in the production of polycarbonate and epoxy resins. BPA is a white, crystalline solid that is soluble in fats and almost insoluble in water with molecular weight of 228.29 g cm^{-3}. The melting and boiling point are 156 °C and 220 °C at a pressure of 5 MPa, respectively [1]. Figure 1 shows the molecular structures of BPA and of the similar 17β-estradiol based on the presence of OH groups and fused ring structures [2].

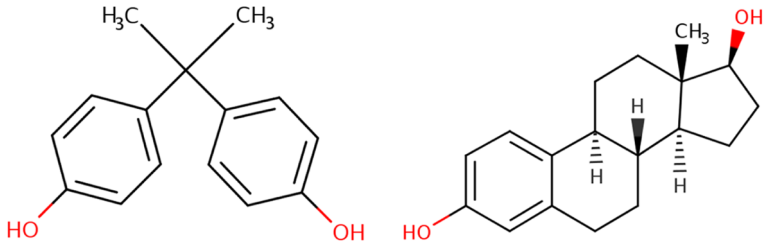

Fig. 1 Molecular structure of bisphenol A (BPA) appears to be similar to 17β-estradiol, allowing the chemical to bind to estrogen receptor Molecular structures were retrieved from United States Environmental Protection Agency, www.comptox.epa.gov)

Worldwide, BPA is commonly used as synthetic chemical in manufacturing various consumer products including infant feeding and drinking water bottles, plastic food containers and utensils, food packaging, toys, eyeglass lenses, dental and medical products, electronic appliances, beverage containers, water pipes [3], compact discs, impact-resistant safety equipment, detergents, flame retardants, food, cosmetics, and pesticides. Thus, the risks of BPA exposure among the majority of world population would be high. Dodds and Lawson [4] noted that BPA was first reported as a synthetic estrogen in 1890. Krishnan et al. [5] first recognized BPA as an endocrine disruptor chemical. Vandenberg et al. [6] have reviewed the level of BPA in human tissues and fluid and suggested that BPA levels were detected exceeding the safe dose in human due to use of products such as plastic products and containers, which result in increased in diseases and cancers. Studies have demonstrated that BPA has detrimental effects on the metabolism of adults, and a number of studies cited hereafter established the link between BPA and the development of metabolic diseases. The effects on fetuses, newborns, and young children are even more detrimental [7]. Specifically, the changes in endogenous estrogen gene expression after BPA exposure showed that the presence of BPA altered estrogen signaling [8]. Furthermore, changes in developing tissues and organs at an early age increase the risks of developing adult diseases in childhood and later in life [9]. Studies have linked BPA with the development of metabolic diseases that include obesity (ICD-10: E66.9) [10, 11], diabetes (ICD-10: R73.03) [12, 13], hypertension (ICD-10: I10) [14–16], hyperthyroidism (ICD-10: E05.90), hypothyroidism (ICD-10: E03.9) [17–19], and cardiovascular disease (ICD-10: I25.10) [20–22]. However, the mechanism of BPA effect towards human health is unique. Some tissues/organs may have been affected by very low concentration, and others may have been affected at higher concentration depending on the time of exposure, and duration. Therefore, there is no standard safe concentration besides the one that has been documented for low and no adverse effect level.

Chemical replacements such as bisphenol S (BPS), bisphenol F (BPF), and bisphenol AF (BPAF) have been used in some applications to replace BPA. The creation of bio-based polymers made from renewable resources is an additional approach. Although various options are available, each has a unique set of factors to take into account, and their effects on the environment and safety may differ. Furthermore, they might have comparable health risks as BPA. Harnett et al. [23] emphasize the harmful effects of BPA and its analogs, namely tetramethyl BPF (TMBPF), BPAF, and BPS, shedding light on their cytotoxic impact. Despite the fact that some of these compounds were made with the idea of "safety by design," current research and this study show that many of these compounds are, in fact, "regrettable substitutions," since they are more poisonous and potent on human cells than their parent compounds. Because of the unique qualities of BPA that make it advantageous in some production processes, finding a replacement for BPA that works in all applications is still difficult.

BPA is a pervasive environmental material due to its mass manufacturing and consumption. Many ecological media, including water, soil, biomass, and air, have been shown to contain BPA. The traces of BPA found in surface water, ground water, sea water, and other sources of water are difficult to detect and remove [24].The intricate and demanding removal process of BPA relies predominantly on current treatment methods, which involve biological and advanced oxidation process. Biological treatments are up to 90% effective, and some oxidation and photocatalytic treatments can remove BPA to less than the detection limits [25, 26]. For biological treatments, the pH must be regulated to control the rate of removal [27] and enzyme cultures are difficult to acquire, maintain, and apply or alternatively, the seeding with well-acclimated microorganisms [28] is difficult to maintain as indigenous consortia typically have competitive advantages.

The two important advanced oxidation treatments of BPA apply chlorination or photocatalysis. The chlorination of BPA usually increases human toxicity by

estrogenic disruption, accumulation in body fat, and maternal transfer to new-born compared to unchlorinated BPA [29]. Often investigated and applied to treat many water-borne contaminants, photocatalysis effectively transforms recalcitrant organic pollutants into less toxic metabolites in comparison with any other conventional treatment. The typical photocatalytic treatment involves a suspension of either nano or microscale photocatalytic particles in a reactor. Despite the efficiency of photocatalysis, the separation of the photocatalytic particles is a major problem. Beside reusability of the recovered photocatalytic particles, the separation is important to avoid the adverse biological effects of the photocatalyst even in the absence of light [30]. Therefore, the development of membranes that bind photocatalytic particles and permit light penetration is very promising in removing BPA from water.

This paper reviews epidemiological and laboratory investigations on the effect of BPA exposure to humans causing metabolic disorders. This BPA exposure assessment is necessary to evaluate treatments of BPA contaminated water from surface water, groundwater, seawater, and other sources. Other investigators have separately reviewed (1) the investigation of the occurrence and impact of BPA on human health and (2) the treatment technologies for the removal of BPA and other pollutants in wastewater treatment, particularly in engineering literature. In the meantime, some investigators have also reviewed on BPA occurrences, health effects and remediation mechanisms including membrane technology. However, those reviews did not synthesize the human health effects of BPA well enough to fully guide the selection of the better treatment methods to remove harmful amounts of BPA from water. This review emphasizes on the significance of UV and visible light radiation in the application of photocatalytic hollow-fiber membranes as these membranes demonstrate the capability to reduce BPA levels in contaminated water to below detection limits. Given that drinking water stands out as the primary source of BPA exposure to humans, the review extensively delves into sources from surface fresh waters and wells. Additionally, it examines variations in BPA levels associated with the household use of portable water. This approach ensures a comprehensive exploration of BPA exposure, particularly in the context of drinking water and its various sources.

2 Effect of bisphenol A exposure on human health

2.1 Epidemiological studies

Upon ingestion, BPA is swiftly absorbed by the stomach and intestines. The liver metabolizes it into the primary glucuronic acid-bound form, which is then excreted through urine with a short half-life. Urine is commonly used as a bio-monitoring matrix and reflects different BPA concentrations across various ages, occupations, and genders globally [31]. BPA affects the endocrine and reproductive systems by interacting with various receptors and enzyme pathways involved in steroid metabolism [31]. It disrupts normal body functions by binding to these receptors, either mimicking or blocking the effects of natural hormones [32]. The endocrine system plays a crucial role in regulating various physiological processes, including metabolism. Hormones, which are regulated by the endocrine system, influence metabolism and play a role in the development of metabolic syndrome. Many epidemiological studies link BPA exposure to impairments of human (1) metabolic [33, 34], (2) reproductive [35], and (3) neurodevelopmental health [36–38]. Figure 2 shows the association of BPA exposure with development of metabolic diseases and neurodevelopmental impairments.

Exposure to BPA *in utero* causes miscarriage and low birth weights [39–41], and increases the risk of obesity and cardiovascular disease during adulthood [42]. Study has demonstrated that BPA exposure *in utero* alters the plasma lipid profile by increasing triglycerides, which eventually leads to the accumulation of lipid droplets in fat tissues [11]. Hence, adult obesity prevails because of juvenile exposure to BPA. Altered glucose metabolism also increases the risk of obesity but further causes type 2 diabetes [43]. Obesity and diabetes both increase the risk of cardiovascular disease [42]. The accumulation of BPA in the body may affect thyroid gland functions, and eventually leads to impaired brain development in young children, thus, increases the risk of neurodevelopmental impairment [38]. The onset of any or all the following conditions associated with obesity leads to insulin resistance, glucose intolerance, dyslipidemia and cardiovascular disease [42, 43].

Among the most common sources of BPA exposure is occupational exposure. Study by Chu et al. [44], showed that direct contact with BPA daily induced skin allergy symptoms and the development of dermatitis for 22 workers at construction sites. Apart from physical skin symptoms, BPA exposure has been shown to elicit negative effects on reproductive system. Study by Li et al. [45] reported a significant risk of sexual dysfunction and low libido among BPA-exposed workers in a factory producing and packaging epoxy resins. The increased BPA concentration in worker serum was associated with the decrease in the mean testosterone concentration in serum.

Study has also reported that 28 workers at a semiautonomous epoxy resin factory who had inhaled BPA, had inadequate thyrotropin, the pituitary hormone that stimulates thyroid production of thyroxine (T4)

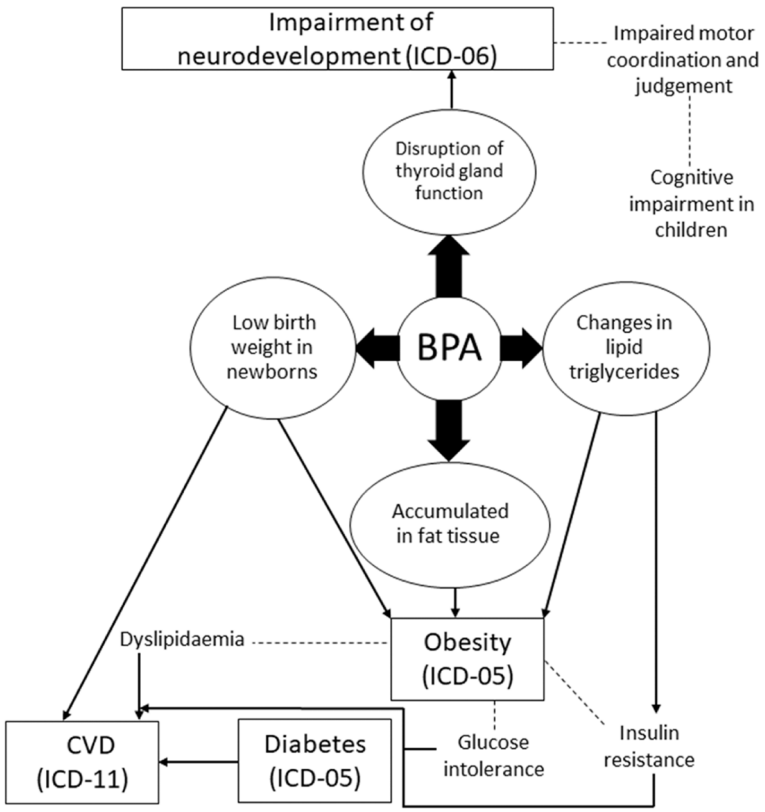

Fig. 2 Effects of bisphenol A (BPA) exposure on the metabolic and neurodevelopmental disorders (designated as rectangles): (ICD-06) impairment of neurodevelopment, (ICD-05) obesity, (ICD-05) diabetes, and (ICD-11) cardiovascular disease (CVD).– – – Dashed lines define the indicative linkage between the major disorders and possible causes of the impairment.→ Thin arrows define causation of the major disorders: obesity, diabetes, CVD, and neurodevelopmental impairment by the four health impairments known to be caused by BPA or intermediate conditions: glucose intolerance and insulin resistance caused by BPA accumulation in fat that causes obesity and elevated lipids and triglycerides, respectively. ➡ Broad arrows define the major health effects caused by BPA

and triiodothyronine (T3), which in turn stimulates the metabolism in most tissues of the human body [46]. To assess endocrine activity and hormone production of the thyroid, the authors have measured total and free T3, and free T4. Abnormal levels of the total and free T3, and free T4 were measured in the workers exposed to BPA. These thyroid hormones exceeded the normal range for adults [46]: total T3 (1200 to 2700 pM), free T4 (10.2 to 24.5 pM), and free T3 (2.76 to 6.45 pM). The function of the thyroid gland is of concern when free T3 exceeds 6.46 pM (diagnosis of hyperthyroidism is being established). In workers with the greatest BPA exposure, they also reported a positive association between free T4 and lactate dehydrogenase (Enzyme Commission or EC 1.1.1.27), and between insulin and thyrotropin (thyroid stimulating hormone). Thus, they also demonstrate that exposure to BPA disrupts the homeostasis of pituitary and thyroid hormones and leads to the development of type 2 diabetes as noted in Fig. 2.

Meanwhile, in children, studies conducted by [47, 48] estimated BPA exposure among children in day care facilities versus households. Surprisingly, BPA was detected in both liquid and solid foods, and the estimated BPA dose was 43 ng kg^{-1} d^{-1} for children of age 2 to 5 years [47]. The BPA samples were collected from environmental media: air, dust, and soil and personal media: liquid food, solid food, hand wipes, and urine [47]. The food samples were collected from both the day care facilities and homes where children live. In day care center, BPA contamination was typically higher in liquid food (average 384 ng kg^{-1}) compared to solid food contamination detected over a range of 196 to 932 ng kg^{-1}. In household samples, BPA contamination was less. However, the study did not adequately explain why BPA contamination varied between samples collected in day care centers versus homes.

Wilson et al. [48] found that 99% of the BPA exposure originated from the diet of children at an estimated dose of 52 to 74 ng kg^{-1} d^{-1}, and an estimated inhalation dose

of 0.24 to 0.41 ng kg^{-1} d^{-1} at day care facilities. The children of ages 1.5 to 5 years were randomly chosen from eligible day care centers and households [48]. The 2007 study detected BPA in more than half of the samples of indoor air and samples of hardwood floors and food preparation surfaces. In addition, nearly all the wipes used to sample the hands of the children had detectable BPA. More than 68% of the samples from the liquid food given to the children had detectable BPA, whereas 83% of the solid food samples contained detectable BPA. Interestingly, both studies by [47, 48] found that the exposure and doses of BPA were higher for children kept in day care centres compared to children stayed at home. Presumably day care centers serve more pre-processed, packaged food whereas households prepare meals from more basic foodstuff with less packaging in plastics.

Different investigators reviewed the U.S. National Health and Nutrition Examination Survey data of 2003 to 2006. The study reported there is no association between urinary BPA and diabetes [49]. Using the same data from the U.S. National Survey, however, another study found a significant relationship between the BPA in urine and diabetes [50]. In the reproductive organs, research discovered that the serum level of BPA increases with the level of androgen in women with polycystic ovary syndrome. Men normally have more androgen than women [51]. Perera and Herbstman [36], reported a negative correlation between androgen level and BPA in men who were exposed to epoxy resins. As of 2021, the authors of this review have not found epidemiological evidence to contradict Vandenberg et al. [6] who noted that whether the BPA effect on the androgen level being the pathway for BPA impact on metabolic diseases was unknown.

From chromosomal abnormalities research, [39–41] reported that higher BPA exposure is associated with complications during pregnancy. All three studies found that women carrying their pregnancy with chromosomal abnormalities had higher maternal serum levels of BPA, and higher BPA levels were associated with a history of recurring miscarriage [41]. These data are based on small sample sizes with limited information about each subject and about other causative factors.

2.2 BPA and metabolic disease

Disruption of normal metabolism is an important effect on the conversion of ingested food into energy. Metabolism involves thousands of enzymes involved in numerous pathways. When affected by metabolic diseases, the essential cellular enzymes involved in expressing, transporting, and processing proteins, carbohydrates, and lipids are disrupted. Investigation of the effect of BPA exposure on metabolism has been explored in vivo using mice, rats, and other rodents. Fetal and perinatal exposure of mice and other rodents to relevant doses of BPA affected organs and led to weight increases of the offspring in adulthood. The incremental weight increases to attain rodent adulthood depends on gender, doses of BPA, and duration of BPA exposure [52, 53], which is associated with the 55 [54].

In addition, perinatal BPA exposure in pregnant mice led to an increase in insulin resistance during the period of pregnancy. An individual who is diagnosed with insulin resistance usually develops type 2 diabetes or has a higher risk of cardiovascular diseases. Furthermore, insulin resistance is associated with an increase in triglyceride and leptin concentrations [55]. Surprisingly, BPA has a higher impact on human pancreatic β-cells compared to mice; Soriano et al. [56] also reported the adverse effect of BPA on glucose metabolism, which diminished the hormone adiponectin release from human adipocytes (fat cells). These findings suggested that BPA could be considered as a risk factor for metabolic disorder development in humans.

Both in vitro and in vivo investigations [11, 52, 53, 57] have reported the effect of BPA exposure on metabolism in children and adults. Several studies [21, 56, 58] conclude that limited exposure to BPA may have more impact on health compared to greater doses. Numerous studies have confirmed that alterations in the mechanism of BPA resulted in disruptions within the body, even at low levels of harm. Figure 3 shows the adverse effect of BPA exposure on vital organs. The study that was conducted on fetal lungs showed a similar hindrance in lung development between moderate and high amounts of BPA exposure [59]. Furthermore, researchers have also observed that BPA has an immediate effect on cardiac myocytes with a non-linear dose response [57] which supports the impact to human health from limited exposure of BPA as compared to high dosage. Women tend to have more estrogenic effects compared to men. Several cross-sectional studies [19, 60, 61] observed negative effects of BPA exposure, but these studies do not indicate acute effects that require immediate water or wastewater treatment. Further long-term investigations of molecular or genetic modifications are necessary to put the briefly documented cross-sectional effects into long-term chronic effects that require treatment standards. In particular, the programming of diseases that occur in adulthood due to BPA exposure prenatal, perinatal, or during younger childhood must be investigated.

The effect of BPA is particularly noticeable in the vital organs essential for survival. The vital organs affected by BPA include the brain [62], heart [63], kidneys [64], liver [65], and lungs [59]. Excessive levels of BPA have been correlated with obesity [58], diabetes [55, 59], cardiovascular diseases [18, 57], polycystic ovarian syndrome

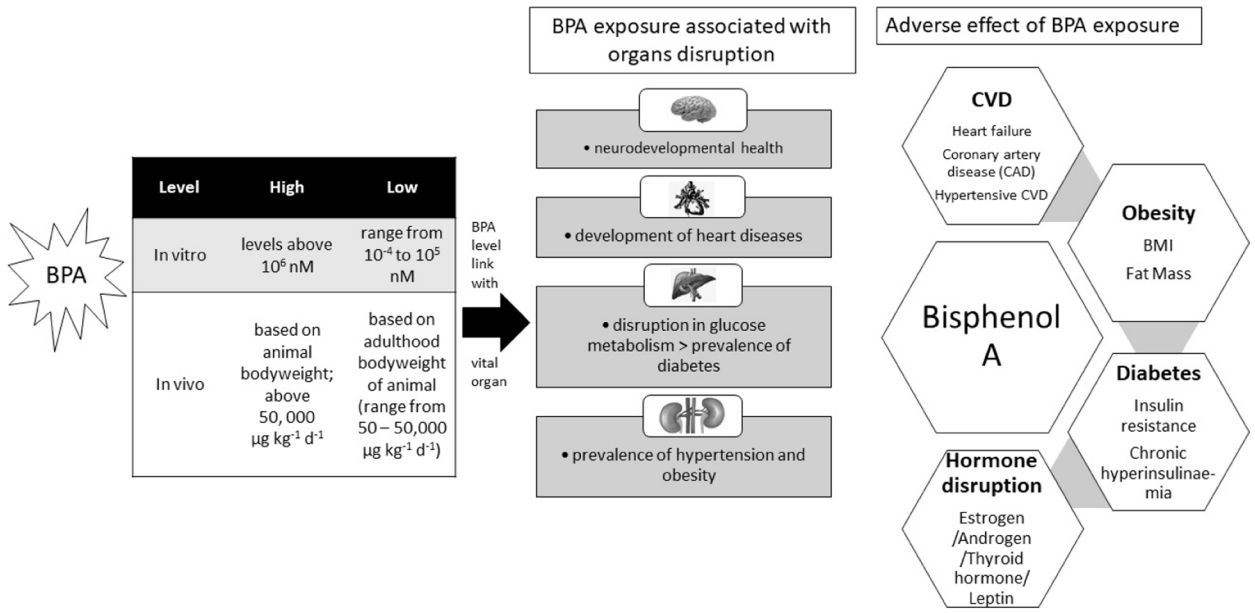

Fig. 3 Bisphenol A adverse effects on vital organs: brain (due to thyroid hormone disruptions), heart (heart failure and several heart diseases), liver (disruption of glucose and lipid metabolism, increased risk of type 2 diabetes and obesity), and kidney (interruption in leptin). Notes: CVD is cardiovascular diseases, body mass index is calculated by dividing the weight (kg) by height (m), chronic hyperinsulinaemia is a condition of excess level of insulin in the blood and usually promoted by obesity and associated with adipose dysfunction, and leptin is a hormone secreted by white adipose tissue that has an important role in the regulation of body weight; a larger amount of plasma leptin is associated with chronic kidney disease, and is thus, indirectly related to the kidney and the prevalence of obesity

[66], and low sperm count [67]. Depending on dose, BPA stimulates in vitro differentiation of murine fibroblasts into adipocytes and increases lipid storage [68]. Small doses of BPA inhibit adiponectin secretion by human adipocyte cell cultures in vitro and stimulate the secretion of interleukin-6 and a necrosis factor, which are two inflammatory adipokines, suggesting possible involvement in insulin resistance, obesity, and metabolic syndrome [54, 69, 70].

2.2.1 Cardiovascular disease

The studies of the relationship between human BPA exposure and cardiovascular disease are limited, but coronary artery disease in men and women exposed to BPA has been reported. Rodent exposure to BPA worsened cardiovascular disease morbidity and mortality [71]. Larger urinary BPA concentrations in humans has been associated with hypertension [15], heart attack [72], coronary heart diseases [73], and peripheral arterial disease [74]. Chronic exposure to BPA has shown to cause maladaptive changes in heart size and shape (cardiac remodelling), atherosclerosis, and altered blood pressure in female mice [72]. Patel et al. [72] also reported that BPA exposure resulted in alteration of cardiac calcium handling, which involved the expression of the protein sarco-endoplasmic reticulum calcium-ATPase where disruption of the protein would lead to electrophysiological dysfunction of the heart. The study demonstrated the negative impact of BPA exposure on heart was not limited to cardiac remodelling but caused cardiac conduction dysregulation occurring at protein level by alternating their expression level negatively. In addition, study by Jiang et al. [75] had evaluated (1) the structure of general heart tissue, (2) the morphology of cardiomyocytes (striated self-beating and cylindrical rod-shaped muscle cells), and (3) the mitochondria in Wistar rats (*R. norvegicus*) exposed to BPA at a dosage of 50 µg kg^{-1} d^{-1}. They observed that prolonged BPA exposure induced cardiac impairments by disrupting mitochondrial function in cardiomyocytes. The study has shown that disruption of cardiomyocytes mitochondrial function induced cardiac impairments by promoting myocardium hypertrophy and cardiomyocyte enlargement.

In addition, Gonzalex et al. [76] investigated whether BPA leads to myocardial interstitial fibrosis by inducing proliferation of cardiac fibroblast cells that increase collagen production. The study has demonstrated that BPA was able to induce myocardial interstitial fibrosis which contributes to chronic dysfunction of the left ventricular, eventually leads to heart failure. Cardiac impairment induced by BPA is not only limited to mitochondrial dysfunction and myocardial interstitial fibrosis. Hu et al.

[77] reported a decrease in cultured cardiac cells (isolated from healthy male Sprague–Dawley) upon exposure to BPA. Furthermore, BPA promotes the proliferation of cardiac fibroblasts via extracellular signal-regulated protein kinases 1 and 2 (ERK1/2; EC 2.7.11.1), which increases collagen production. Patel et al. [78] found that chronic oral BPA exposure at the beginning of gestation 11.5 days led to reduced survival of the mice after an experimental myocardial infarction was induced by isoproterenol when the mice reached adulthood, 16 weeks after birth. A substantial reduction was observed in survival among the chronically BPA-exposed C5bl/6n mice (*Mus musculus*) immediately after 3 weeks after the experimental myocardial infarction. The data presented by this group of researchers suggested that after the experimental myocardial infarction, cardiac remodeling and thus heart recovery was reduced by chronic exposure to BPA [78].

Yan et al. [79] investigated the effect of acute BPA exposure to the heart of rats subjected to global ischemia (inadequate blood supply), followed by reperfusion of the blood supply. They observed a period of sustained ventricular arrhythmias of the female Sprague–Dawley rats (*R. norvegicus*) heart exposed to 1 nM BPA compared to the male heart. The ventricular arrhythmias developed secondary to BPA exposure in female rats was due to a more sensitive female physiology response towards estrogen-like compounds compared to males [79]. They further demonstrated that acute exposure to BPA worsened the ventricular arrhythmia following ischemic-reperfusion injury or reoxygenation injury of the heart of female rats (*R. norvegicus)*. This worsening of ventricular arrhythmia is consistent with Posnack et al. [71] who found slowed electrical conduction in the hearts of BPA-exposed, female rats (*R. norvegicus*).

Moreover, higher total urinary BPA was associated with an increased incidence of hypertension [74]. A study of elderly Korean adults without a prior history of hypertension revealed similar findings in which BPA occurrence in urine significantly increased the onset of hypertension. Bae et al. [80] also found that BPA exposure is associated with reduced heart rate variability. Patel et al. [78] observed no differences in blood pressure of the male humans exposed to BPA. However, female suffered a small increase in systolic blood pressure associated with a BPA exposure at 5.0 μg kg^{-1} d^{-1}. Earlier Patel et al. [72] determined that BPA had a sex-specific impact on the blood pressure when they compared the systolic, diastolic, and arterial pressures between females and males. Kilic et al. [81] showed that more than a dozen genes and genetic loci were affected in BPA-exposed human subject. One of which is the sirtuin protein (SIRT1), a NAD (nicotinamide adenine dinucleotide)-dependent histone deacetylase (EC 3.5.1.98) located in the nucleus. This protein is expressed in many tissues. The protein SIRT1 is activated in distinct metabolic and stress response pathways. The expression of this protein is targeting many cellular proteins, such as PPAR-γ, PGC-1α, FoxO, AMPK, NF-kB, PTP, eNOS, and p53.

In summary, BPA exposure was proven to have an association with cardiovascular disease and hypertension development. The exposure impacts the expression of a number of cardiac proteins and seems to be more evident in exposed females; thus, increasing the possibility of women to experience cardiac crisis compared to men. Moreover, a longer duration of BPA exposure could modify and induce cardiac conduction. Acute exposure showed an impact on immediate observation such as arrhythmia compared to chronic. The dosage of BPA is also an important factor in the development of cardiac injuries. Most of the studies suggested that a small dose of BPA has more impact on cardiovascular disease development than a large dose [80, 82].

2.2.2 Obesity

Obese people usually have a 20% greater body weight than the person with a normal body mass index. In adults, a body mass index more than 30 kg m^{-2} is defined as obese. However, the calculated body mass index might reflect different physiology and metabolic health [82]. The energy expenditure should be greater than the energy intake to maintain or reduce weight gain. Obesity occurs due to overconsumption of fatty foods and having a sedentary lifestyle. Studies conducted have agree that exposure to endocrine disruptors (mainly from food and beverages) influence lipid metabolism thus causing obesity.

An epidemiological cross-sectional analysis carried out by Hong et al. [60] comprised of 296 healthy, reproductive-aged Korean women above 18 years old demonstrated that urinary BPA positively associated with the body mass index, waist circumference, and fasting serum insulin. In contrast, Do et al. [83] analyzed the Canadian Health Measures Survey (2007–2011) that monitored 4733 Canadian adults aged above 18 years old and reported that urinary BPA is not associated with increased waist circumference but was positively associated with obesity defined by the body mass index. However, Hong et al. [60] in the study of 11,293 Navajo (American Indian) and Alaska Native adults reported an association of waist circumference and of obesity with the urinary BPA concentration [84].

Another interesting study has described a positive correlation between BPA and an excessive body fat, and increased serum leptin. Zhao et al. [85] also examined the relationship between BPA and both osteocalcin

level and bone mineral density. Their cohort study of 246 healthy premenopausal women from Shanghai, China age 20 years and older with regular menstrual cycles showed that the exposure to BPA has an undetectable impact on bone mineral density. However, BPA in urine was positively associated with body fat mass and leptin. Bone mineral density was essentially unaffected by BPA, perhaps because the healthy participants were not overweight. Still, further investigations need to be conducted to observe the long-term effect of BPA exposure on bone mineral density due to reports of inconsistency in the effects of leptin signalling on neuroendocrine regulation and bone metabolism [83].

Vafeiadi et al. [10] reported that early life BPA exposure increased the risk of obesity and cardiometabolic disorders, based on a study involving 500 mother–child pairs from a pregnancy cohort in Greece. Larger BPA concentrations in the urine of children were associated with increased body mass index, waist circumference, and the sum of skinfold thickness at the age of 4 years. However, a larger cohort should be assembled to determine whether obesity later in childhood indeed resulted from prenatal or early childhood BPA exposure as they observed a negative association between body mass index and adiposity in girls whose mothers were exposed to BPA during early pregnancy.

In a cross-sectional study of a nationally representative subsample of 2838 randomly selected children and adolescents aged 6 to 19 years in the United States National Health and Nutrition Examination Surveys during 2003 to 2008, urinary BPA was significantly associated with obesity for Caucasians, but not for blacks or Hispanics [61]. An interesting finding from a study of 177 (120 normal and 57 obese) Saudi children aged 13 to 16 years revealed a positive association between serum BPA and obesity. Obese children had a larger serum BPA level compared to normal weight children. Al-Daghri et al. [86] also found a significant association of elevated low-density lipoprotein cholesterol with serum BPA level, but only in girls, thus, suggesting that effect of BPA is gender specific.

Xie et al. [11] found that microRNA-21a-5p regulated adipogenic differentiation. The results showed that overexpression of the biological regulator microRNA-21a-5p significantly decreased the red lipid droplets and triglycerides in BPA exposed 3T3-L1 cells (derived from mouse embryo), a cell line that differentiates into adipocytes. They also concluded that microRNA-21a-5p overexpression attenuated BPA-induced obesity in vivo by targeting map2k3 gene through map2k3 p38 mitogen-activated protein kinase (MKK3-p38 MAPK) in 3T3-L1 cells. These findings suggested a potential therapeutic strategy for BPA-induced obesity. Although BPA in urine was positively associated with body mass index, fat mass, leptin, and serum insulin (during fasting), further investigation is needed, especially regarding the range of ages of the people tested. Whether obesity is affected by prenatal or early life BPA exposure is still inconclusive as of 2021.

2.2.3 Diabetes

BPA has an intense effect on the mouse endocrine pancreas, an essential function involved in glucose metabolism. BPA disrupts pancreatic β-cells, which are crucial in producing insulin and that induce hyperinsulinemia and insulin resistance in male rats (*R. norvegicus*) [87]. Insulin is a peptide hormone, essential in maintaining the blood glucose level [88]. Excess glucose induces secretion of insulin from pancreatic β-cells. The cardiac muscles are one of the important tissues that exerted response within the presence of insulin, which in turn, responsible for transporting glucose to all living tissues within the blood circulation. Apart from cardiac muscles, adipocytes and skeletal muscles also exerted response towards presence of insulin [89]. Sivashanmugam et al. [13] assessed the effect of BPA on insulin signaling molecules in the cardiac muscles of adult-male, Wistar rats (*R. norvegicus*). The selected adult-male, Wistar rats (200 to 250 g body weight) were divided into four groups of which receiving four different 30-d doses orally of 0, 10, 100, and 400 mg BPA kg^{-1} body weight d^{-1}. They showed that the group of rats that received the largest dose (400 mg BPA kg^{-1} body $weight^{-1}$ d^{-1}) suffered significant protein decreases in insulin receptors and pIRTyr1162 proteins, with no significant changes in IRS1 (insulin receptor substrate-1) and kinase B or Akt proteins [13]. However, the authors observed a decrease in the three proteins that affect the cardiovascular function: (1) pIRS1Tyr632 (at BPA 30-d doses of 100 and 400 mg BPA kg^{-1} body weight d^{-1}), (2) pAkt Ser473 (400 mg BPA kg^{-1} body weight d^{-1}) and (3) glucose transporter 4 (GLUT4) at 100 and 400 mg BPA, which cycles between cell cytosol and the plasma membrane. They concluded that BPA exposure has an adverse effect on cardiac insulin signal transduction that may affect cardiac function.

Sakurai et al. [12] reveal that in adipocytes (fat cells), BPA exposure increases the basal and insulin-activated glucose transport by stimulating glucose transporter 4 expressions. Adult male mice (*M. musculus*) exposed to BPA developed both hyperinsulinemia and mild insulin resistance [90]. More prolonged exposure to BPA in rats (*R. norvegicus*), up to 4 days, resulted in chronic hyperinsulinemia and insulin resistance.

In addition, Soriano et al. [56] found that BPA exposure to humans and adult female mice (*M. musculus*) interfered with glucose metabolism and may increase the possibility of diabetes type 2, cardiovascular diseases, and

insulin resistance. They found that in the human pancreatic β-cells, glucose-induced insulin secretion is stimulated at an environmentally relevant dose of BPA (1 nM), while for mice, exposure to increasing doses of BPA increased insulin secretion. Soriano et al. [56] also found reduced adiponectin (protein hormone and adipokine) in human adipocytes (fat storage cells), which confirmed a disruption of the glucose metabolism. Guan et al. [91] evaluated the effect of BPA exposure on lipid metabolism of the fish, Gobiocypris rarus. The authors found that BPA induced triglyceride increases in male fish suggesting that the fatty acid β-oxidation pathway, after BPA exposure, might be gender-dependent.

2.2.4 Other metabolic diseases

Several studies investigated the development of other metabolic diseases following BPA exposure, which include hyper- and hypothyroidism, as well as cancer. In addition, BPA binds to estrogen receptors and interferes with endogenous estrogen (demonstrated in vitro with cell lines). BPA also binds to androgen receptors, thus acting as an androgen antagonists or testosterone blockers in vitro. Furthermore, BPA binds to thyroid receptors, to disrupt the function of the two thyroid hormones. In a cross-sectional study from February to August 2012 involving 39 obese and overweight children aged 3 to 8 years from Dayton, Ohio in the United States, Khalil et al. [19] found that obese male children had significantly less free thyroxine as compared to female children. The thyrotropin was inversely associated with BPA. Earlier Braun et al. [17] suggested that BPA disrupts the thyroid function.

Chapalamadugu et al. [18] reported that BPA is a thyroid hormone receptors antagonist and able to inhibit the induction of gene *Myh6*, thus, interfering with the development and growth of the fetal heart into the postnatal heart. As an endocrine disruptor, BPA affects the nuclear hormone receptors, which bind steroids that act as hormones and regulate target gene transcription. Moriyama et al. [92] found that BPA reduced the thyroid hormone triiodothyronine (T3) binding to the nuclear thyroid receptors and inhibited the transcription process. They determined that BPA suppressed the activity of the transcription factor Gal4-TRα1 up to half of the respective control level. This finding also demonstrated that BPA impaired thyroid function by inhibiting the hormone triiodothyronine or T3 binding to nuclear thyroid receptors, thus suppressing transcriptional activity. Study by Heindel et al. [22] reported that BPA exposure disrupted thyroid hormone signaling during preclinical development and validation of an animal model that mimics human responses. The end point of the research program reported that two years of BPA exposure on Sprague–Dawley female rats (*R. norvegicus*) did not exhibit concerning health risks. However, the study investigated neoplastic changes rather than alteration on thyroid function secondary to BPA exposure suggesting that BPA exposure posed concerning threat on thyroid functions and metabolism [22].

BPA is a potential risk factor in breast cancer [93]. Two studies found in MMTV-erbB2 mice (*M. musculus*) that even small BPA doses could accelerate mammary tumorigenesis [94] and metastasis [95]. Small BPA doses induce aggressive tumorigenesis in breast cancer patients [96]. There is an association between BPA exposure and complex endometrial hyperplasia and endometrial cancer [97]. At very small doses (ng BPA kg^{-1} body weight d^{-1}), the level found in the blood of general populations, induced chemo-resistance in several malignant human breast cell lines, regardless of whether these cells are estrogen receptor positive or negative while it increases the expression of several anti-apoptotic proteins [98].

Another study on prostate cancer found that mice (*M. musculus*) fetal or perinatal exposure to BPA or diethylstilboestrol is associated with prostate hyperplasia and pre-malignant lesions in adults [99]. The perinatal exposure to BPA or estradiol (Fig. 1) increases the susceptibility to prostate cancer, either spontaneously or after a second estrogenic injection [100]. Heindel et al. [22] noted the possibility of whether BPA exposure would reprogram genes could be determined by comparing the methylome and transcriptome from rat (*R. norvegicus*) prostate cells with uterus cells. Interestingly, they found that BPA exposure also increases the chances of having testicular cancer. At low doses (nM or pM), BPA acted only through an intracellular seven-transmembrane G protein-coupled estrogen receptor (GPR30) by a promotive effect, while at greater doses (mM), BPA also acted through an estrogen receptor, which counteracted the promotive GPR30 mediated effect [101].

3 Distribution of BPA in the environment and occurrences of BPA in household water

Widely used as a liner in food and beverage packaging [102], as well as in other plastics, toys, and electrical equipment [103] BPA exposure ranges from a variety of sources, including direct contact with contaminated plastic materials, utensils, and equipment, swallowing contaminated food and water, and inhaling polluted air. Since meeting daily nutritional demands is necessary for survival, food exposure is the most significant factor for BPA contamination. BPA contamination through food exposure arises from the use of BPA in the production of various plastic food serving containers made of polycarbonate (PC) and polyvinyl chloride (PVC), where BPA seeps into food and drinks. Food cans are also made from

epoxy resins for the interior coatings, hence contribute significantly to food adulteration [104]. Furthermore, the bioaccumulation of BPA in the fish from the domestic pond occurs resulting from BPA leaching of the plastic storage tanks and other paraphernalia used in fish farming [105]. Interestingly, rivers, reservoirs, lakes, and estuaries are regarded as the main BPA sinks; thus explaining the uptake of BPA in the aquatic species. The level of BPA in seafood such as prawn, crab, blood cockle, white clam, squid, and ocean fish from the supermarket in Singapore was reported between 13.3 and 213.1 ng g^{-1} wet weight. BPA levels were undetectable (< 0.2 µg L^{-1}) in river water but ranged from 2 to 8.8 µg kg^{-1} and 0.3 to 12 µg kg^{-1} wet weight in periphyton and benthic organisms, respectively. As important food sources for invertebrates and some fish, benthos are aquatic species that live on the bottom of lakes, rivers and creeks or on the sea floor.

Apart from food, there are a few other potential sources that might influence the levels of BPA in humans, such as drinking water, air and dust, which receive lesser attention. Dermal exposure is the second most common way that BPA is absorbed which rises from direct contact with toys, medical equipment, and thermal paper. Thirdly, BPA exposure can occur via inhalation of BPA-containing vapors, mists, dust, and gasses [104]. Table 1 provides some concentrations of BPA based on the few published exposure assessments involving food wrapped in plastics, other food containers, air and dust, dental sealants, and paper currencies.

3.1 Occurrences in household water

Numerous water sources, including drinking water supplies, groundwater, and surface water, have been shown to contain BPA. The reason for this prevalent phenomenon is the pervasive usage of BPA, which is released into the environment during the production of plastics, epoxy resins, and other consumer products. BPA can enter surface water bodies like rivers and lakes by leaching from plastic garbage, runoff from cities, and industrial discharges. Contamination of groundwater can occur when BPA-containing materials seep into the soil. Furthermore, BPA may seep into sources of drinking water due to the deterioration of plastic pipes or the leaching of products containing BPA that are utilized in water treatment procedures. Household water is potable domestic water used indoor and outdoor for drinking, food preparation, and bathing, among others. Although BPA reacts rapidly in chlorinated water, this contaminant can persist to household taps [1] and beyond. BPA diglycidyl ether is used as a monomer in the synthesis of epoxy resins to manufacture polycarbonate products [112], and as a stabiliser in PVC products [113, 114]. In addition, epoxy coatings are used on the internal surfaces of water mains, and other connecting pipes, and household connectors. As a result, BPA can be released into drinking water and surface water from household water pipes and polyethylene tanks.

3.1.1 Tap water

Epoxy resins containing BPA are widely used to reline the interior surfaces of the water mains and water pipes [115] due to transparency, large impact strength, resistance to acids as well as stability in oil and intense temperatures (up to 145 °C) [113]. The widespread use of BPA in PVC pipe coatings has led to the emerging trace of BPA in household water. The contamination of BPA into tap water might happen due to the BPA leaching from the PVC hose as the water passing through [116]. The assessment of the BPA occurrence in Malaysia has reported BPA from 3.5 to 59.8 ng L^{-1} in 30 water samples collected directly from tap connected to the main distribution pipes [117]. Shockingly, large BPA concentrations of 56.4 and 59.8 ng L^{-1}, respectively, was detected in two samples collected from source fitted with filter devices. Meanwhile, samples collected from source without filter

Table 1 Bisphenol A concentrations in different media reported for 2010 to 2021

Sources of BPA	Bisphenol A concentrations	Locations or media sampled	References
Food	1.0 to 3.8 µg kg^{-1}	Cereals	[106]
	1.32 µg kg^{-1}	Milk sample	[107]
	233.8 ppb	Fruit samples	[108]
Air and Dust	4.55 ng m^{-3}	Urban regions of India	[109]
Cans	1.0 to 99.9 µg kg^{-1}	Canned seafood	[105]
	3.7 to 265.5 µg kg^{-1}	Canned vegetables	[105]
	21.3 ng g^{-1}	Canned beef	[110]
Denture equipment	3.3 to 30 µg mL^{-1} of saliva	Resin from dental sealant	[105]
Paper currencies	0.001 to 82.7 µg g^{-1}	Paper currencies collected from 21 countries	[111]

devices have been detected with BPA with a mean concentration of 9.6 ± 4.3 ng L^{-1}.

Previously, BPA was reported present in water samples from leaching test for epoxy resins [118] and materials and equipment used for water supplies [119]. Continuous test of tap water from pipes coated with epoxy resins A and B were performed in the presence of chlorine [120]. After 24 months, BPA was detected at all times in the retained tap water of both pipes A and B with the concentration varies from 3.9 to 130 ng L^{-1}, and 2.6 to 12 ng L^{-1}, respectively. A most recent investigation [116] to assess the occurrence of BPA in tap water reported that BPA was ubiquitous in the tested samples. For this assessment, the release of BPA from water supplied through pipes made of different materials such as PVC, stainless steel and galvanized pipes. Water samples were obtained during fall, winter, and spring for seasonal comparative purposes. The study findings indicated that BPA was found in two samples from Taipei and one from Kaohsiung, which were obtained from buildings using PVC pipes at concentrations of 12, 10 and 3 ng L^{-1}, respectively. As expected, the concentrations increase with contact times and ambient temperatures. BPA concentrations were also affected by ambient temperatures, which decreased in winter and increased again in spring.

A further investigation of BPA presence in tap water was conducted earlier in Guangzhou [121]. For this analysis, six samples of tap water were obtained from six separate drinking water sources and the findings revealed that five of the tap water sample plants were found to contain BPA concentrations ranging from 2.3 to 317 ng L^{-1}. The result also suggested that BPA levels were close to BPA levels in the drinking water survey results [122]. Like the previous study, this result also suggested that the concentration of BPA in tap water may vary in different seasons in which the concentration was lower in winter than in summer. In summary, different levels of BPA were found in most of the samples of tap water with the highest levels found in the samples obtained from taps connected to PVC pipes and water filter systems. To conclude, the reliability of the water treatment process does not guarantee the full elimination of BPA from drinking water, as pollutants can also be introduced by contamination of supply materials during contact with water.

3.1.2 Drinking water

Conventional drinking water treatment plants (DWTPs) typically provide coagulation, sedimentation, filtration, and chlorination. Although BPA quickly degrades in chlorinated water, DWTPs are not specifically designed to remove endocrine disrupting chemicals; thus, BPA in ambient surface or ground waters can persist through the water purification processes. BPA in drinking water has been recorded mainly because of lacquer coatings of concrete tanks and the lining of many fittings in contact with drinking water [102]. Other than water supplies, BPA may also leach from plasticisers and by-products during the manufacturing process, in addition to the potential for BPA presence in drinking water. The occurrence of BPA was previously investigated in a study in which they measured the water treatment efficiency in Taiwan [113] from 11 water treatment plants in various regions of Taiwan. Water samples were obtained three times a year from 2009 to 2011. The findings showed that BPA was found in most of the samples with BPA levels in treated water varying from below the quantification mark to 38 ng L^{-1}.

Fan et al. [123]. confirmed the identification of BPA and the chlorinated intermediates in Chinese drinking water from 62 DWTPs in 31 major cities across China using pre-chlorination, flocculation, sedimentation, filtration, and disinfection treatments. 61 DWTPS used chlorine or sodium hypochlorite as a disinfectant, one used chlorine dioxide. Of the 54 water samples from DWTPs, 31 were from reservoirs and 23 were from river water, while eight were from groundwater. BPA was detected in all 62 water source samples with concentrations ranging from 4.7 to 512 ng L^{-1}. Concentrations were generally higher compared to drinking water samples reported in the U.S. and Canada [122, 124]. BPA in drinking water samples from 62 DWTPs in 31 cities in China were detected at 60 of 62 plants [123]. The average concentrations of BPA collected from river were the highest followed by reservoir and groundwater with concentrations at 106, 79.8 and 62.8 ng L^{-1} respectively. As the water source for DWTPs in China is well protected, the concentrations of BPA recorded were lower (range between 2.2 and 1030 ng L^{-1}). BPA with concentrations less than 5 to 14 ng L^{-1} were also reported in 19 DWTPs used for the production of drinking water in the U.S. [122] as well as in surface water of south Germany [125], Canada [124], and France [126]. In France, the quantification of BPA from DWTPs located in the French Poitou–Charentes area was conducted [126]. Samples were collected from the surface and treated water from eight municipal DWTPs. The result shows that BPA was detected at concentrations ranging from 6.7 to 29.7 ng L^{-1} and from 2.0 to 16.9 ng L^{-1}, respectively.

Ironically, BPA level has decreased from 37 to 79% in most sampling sites which have been correlated with treatment processes. In the Poitou–Charentes area, different combinations of water treatment processes have been applied. Most BPA was extracted during the chlorination process of due to the intense chlorine reactivity. Next, the filtering phase was shown to eliminate BPA in most water treatment plant [127]. Ozonation [128] and

activated carbon have been proposed as an effective way of reducing BPA [129]. Different results were published with complete removal ranging from 70–100% [127].

Levels of BPA in China drinking water have been reported to be between less than 2 and 128 ng L^{-1}. Meanwhile, chlorinated BPA in drinking waters were reported to be an average concentration of 7.1 ng L^{-1} as compared to the sample collected from the reservoirs with an average concentration of 79.8 ng L^{-1} [127]. The reported levels of BPA in the drinking water were greater than those reported in southern Germany [125], the French Poitou–Charentes area [126], Malaysia [117], Canada [124], and United States [122]. Interestingly, significantly large concentrations of BPA ranging from 0.07 to 4.21 g dm^{-3} was detected in water stored in PC bottles with the greater BPA levels determined in newly manufactured bottles [130]. BPA with concentrations ranging from 17.6 to 324 ng L^{-1} was also detected in bottled mineral and drinking water from 21 brands at the local supermarket in Guangzhou. In Japan, bottled mineral water stored at 50 °C was detected with high concentrations of BPA in comparison with samples stored at 25 °C [131]. The minimal and maximal concentrations of BPA reported were 3.3 ± 2.6 and 11.3 ± 5.3 ng L^{-1}, respectively [117]. Similarly, BPA level at median 4.6 ng L^{-1} was detected in bottled water in Greece [132].

Table 2 shows that BPA has been detected in various surface waters ranging from less than the quantitation limit to 21 ng L^{-1} Greater levels of BPA found in some waters may be due to insufficient treatment and management of wastewaters from homes, villages, cities, and industries where typical wastewater treatment plants (WWTPs) are ineffective [117]. The highest BPA level in drinking water was found in samples obtained from taps connected to PVC pipes and water filters devices. In this regard, BPA are widespread as coatings for many fittings in contact with drinking water [102]. Trace levels of BPA have been found in most potable drinking water samples. Filtered mineral water had lower BPA levels, while samples processed in poor storage conditions had slightly high BPA levels. However, BPA consumption from drinking water is very low and is less than 0.01% of the tolerable daily intake. The study shows that BPA is a pervasive pollutant in soil, tap and bottled mineral water.

4 Removal of BPA from water

Experimental and epidemiological investigations (Sect. 2.0) have identified correlations between BPA exposure and diverse health issues, with a specific emphasis on the metabolic system, while the occurrence of BPA in the environment (Sect. 3.0), particularly in drinking water, has emphasize the significance of drinking water as a major route of BPA exposure for both humans and animals. The discussion underscores that the presence of BPA in drinking water directly aligns with the potential health impacts elucidated in the metabolic syndrome section. By highlighting the link between environmental exposure and health outcomes, the paper provides insight into how BPA, as an endocrine-disrupting compound, may contribute to

Table 2 Occurrences of Bisphenol A in different water sources worldwide

Surface and potable water sources	Bisphenol A concentrations	Location	References
All rivers country-wide	0.5 to 16 ng L^{-1}	Germany	[125]
Elba River	4000 to 92,000 ng L^{-1}	Germany	[133]
River water samples	21,000 ng L^{-1}	Netherlands	[134]
Houston Ship Canal	0.08 mg L^{-1}	Texas, United States	[135]
Taihu	8.5 to 97 ng L^{-1}	China	[136]
16 major rivers	0.01 to 44.65 µg L^{-1}	Taiwan	[137]
Inanam River	below detection level to 4.008 µg L^{-1}	Malaysia	[138]
Bentong River	1.13 ± 0.4 and 5.52 ± 1.7 ng L^{-1}	Malaysia	[139]
Langat River	1.4 to 87.5 ng L^{-1}	Malaysia	[117]
Tap water	3.5 to 59.8 ng L^{-1}	Malaysia	[117]
Tap water	3.0 to 12 ng L^{-1}	Taipei, Taiwan	[116]
Tap water	2.3 to 317 ng L^{-1}	Guangzhou, China	[121]
Drinking water	below detection level to 38 ng L^{-1}	Taiwan	[140]
Drinking water	2 to 128 ng L^{-1}	China	[123]
Bottled water	0.07 to 4.21 g dm^{-3}	China	[130]
Bottled mineral water	17.6 to 324 ng L^{-1}	Guangzhou, China	[121]
Bottled water	4.6 ng L^{-1}	Greece	[110]
Drinking water	0.00001 to 0.02883 ng L^{-1}	South Africa	[141]

adverse metabolic effects. Given the essential nature of water consumption, drinking water becomes a primary source through which individuals come into contact with BPA in their daily lives, therefore methods to efficiently remove BPA from the freshwater, wastewater, and soils are urgently needed [142, 143]. The aquatic assimilation of BPA is largely dependent on biological transformation due to the presence of microorganisms, bacteria, and other biological entities [144].

Through various enzymatic reactions and metabolic pathways, these microorganisms can initiate the breakdown and transformation of BPA into different compounds. The benefits of biodegradation of BPA may include the ability to operate at low concentrations of enzymatic reaction over a wide pH and temperature range, as well as the ease with which the process can be controlled. Therefore, in this section, the degradation of BPA by enzymatic treatment and bacteria transformation was reviewed to highlight the advantages and potential applications of biodegradation. These methods, inherently connected to the biological processes discussed in the exposure section, present promising avenues for environmentally friendly and sustainable BPA remediation. However, the costs of implementing such technologies at full-scale facilities, and data from studies on the treatment of solutions containing BPA at concentrations two to four orders of magnitude higher than the environmental occurrence is difficult to extrapolate to the real-world situation [145]. In the meantime, the complete elimination of BPA from drinking water cannot be guaranteed by the effectiveness of the treatment process because BPA is used so extensively to reline water mains and as coatings for numerous fittings that come into contact with drinking water, which can allow the contaminants to leak into the water supply [102]. Therefore, the inclusion of photocatalysis underscores the diversity of approaches, as it harnesses advanced oxidation processes to degrade BPA. The synergistic combination of photocatalysis and membrane filtration enhances the overall efficiency, ensuring that even trace amounts of BPA are targeted and eliminated. In this regard, the photocatalytic membrane system holds the potential to serve as the ultimate step in drinking water treatment, ensuring the thorough removal of BPA. Furthermore, photocatalytic membrane introduces an alternative and complementary strategy, broadening the toolkit for addressing BPA contamination in various environmental contexts. The comparison of biodegradation and photocatalysis underscores the necessity for a multimodal approach to properly handle BPA pollution, including both environmental sustainability and practical viability.

4.1 Biodegradation treatment

4.1.1 Enzymatic treatment

Enzymatic treatment has garnered significant attention from environmentalists as promising treatment for the removal of BPA as it offers a targeted and environmentally friendly approach to remediation. The breaking of chemical bonds within the BPA structure is mostly initiated by enzymes such as esterases and hydrolases [146]. The intermediate molecules produced by this enzymatic process are metabolized further into less complicated and maybe less hazardous chemicals [147]. The subsequent stepwise breakdown of these intermediates, facilitated by additional enzymes and metabolic pathways, ultimately transforms BPA into simpler and less toxic compounds. Xing et al. [148] proposed degradation pathways of BPA by dialysis membrane enclosed laccase catalysis (DMELC) as shown in the Fig. 4. The process initiates with laccase oxidization, leading to the breakage of the C–-C bond of isopropyl connecting two phenols. This results in the formation of intermediates 4-(2-hydroxypropan-2-yl) phenol through electron transfer. Subsequently, this intermediate is susceptible to further transformation into 4-(prop-1-en-2-yl) phenol (product 1). Concurrently, the loss of electrons and alkylation of 4-(2-hydroxypropan-2-yl) phenol generates either 3-methyl-2,3-dithtdrobenzofuran (product 2) or 2-methyl-2,3-dithtdrobenzofuran (product 3). Additionally, 4-(prop-1-en-2-yl) phenol has the potential to undergo methylation, leading to the formation of 1-methyl-4-(prop-1-en-2-yl)cyclohex-2-en-1-ol (product 4). With regard to the formation of the intermediates, product 1 was identified as Class I with low toxicity, products 2 and 3 were classified as persistent substances [148]. The possible pathway of degradation BPA will lead to complete mineralization to water and carbon dioxide. While these proposed pathways provide insights into the degradation process, the precise mechanism requires further elucidation in subsequent research endeavours. Furthermore, the exact degradation mechanism can vary among different enzymes involved in the breakdown of BPA. Enzymes are highly specific biological catalysts, and different enzymes recognize and interact with substrates in distinct ways. In the context of BPA degradation, various enzymes, such as laccases and hydrolases, may be involved, each with its unique mode of action.

Several oxidoreductases (e.g., peroxidases and polyphenol oxidases) expressed by microorganisms and plants transform aromatic compounds, such as BPA, nonylphenol, and other phenolic compounds. Previously, the degradation of BPA by manganese peroxidase (MnP) from white-red mycelia Basidiomycete (*Pleurotus ostreatus*) showed that approximately 80% of BPA was degraded within 12 days of incubation [149]. In-vitro

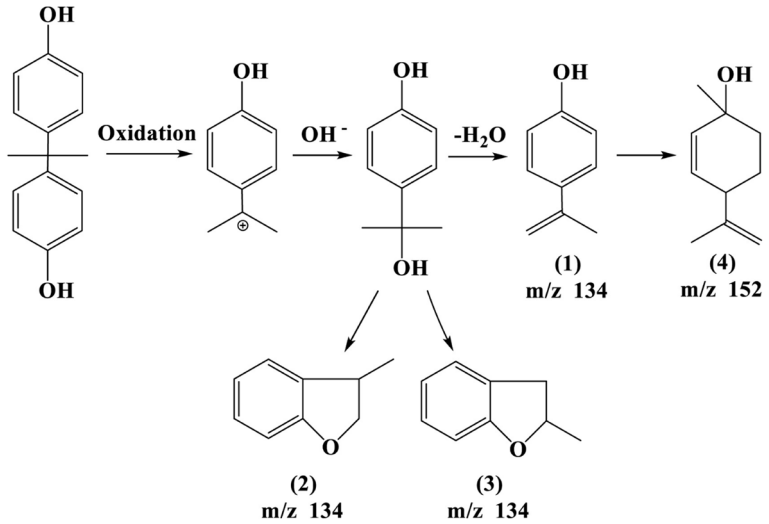

Fig. 4 Proposed degradation pathways of BPA by DMELC [148] (License No: 5790651247422)

study suggested that single-electron oxidation by MnP in the presence of H_2O_2 initiated the degradation of BPA followed by the metabolization into phenol, 4-isopropenylphenol, 4-isopropylphenol, and hexestrol [150].

Tsutsumi et al. [151] conducted a similar study on the elimination of BPA by MnP from lignin-degrading basidiomycetes and found that BPA was completely eliminated after a 1-h treatment. However, after 1-h, 40% of BPA's estrogen activity remained in the reaction mixtures. Extension of the treatment time to 12-h completed the removal of the estrogenic activity of BPA. *Phanerochaete chrysosporium*, the best-studied white-rot fungus, secretes two heme peroxidases under ligninolytic conditions, namely lignin peroxidase (LiP) and MnP [150]. 50% BPA removal was achieved after 1-h reaction of LiP and micellar solution, and the reaction was reactivated by adding H_2O_2 as the reversed micellar solution. However, the available literature on LiP is limited. Previously, the oxidation of BPA by horseradish (*Armoracia rusticana*) peroxidase (HRP) has also been studied [152]. HRP in the presence of a hydrogen donor catalyses the reduction of H_2O_2 to H_2O; followed the oxidation of BPA. In the study, the incubation of BPA with different concentration of HRP (0.3, 5.0, and 66.7 U mL^{-1}) show that the removal of BPA increased according to the enzyme level of activity with 99% achieved by 66.7 U mL^{-1}. Interestingly, the oxidation of BPA also removed the estrogen-like activity.

Table 3 shows various enzymatic treatments (peroxidise) for the removal of BPA. Peroxidases specifically HRP, bitter gourd peroxidises (BGP), soybean peroxidase (SBP), and polyphenol oxidase from various enzyme sources are useful in the removal of BPA under different conditions. The optimal pH can be varied from pH 5 to pH 10 depending on the enzyme types. Similarly, the optimal temperature was between 25 to 40 °C with complete removal of BPA estrogenic activity [12, 153]. Interestingly, BGP was quite effective in the degradation of BPA. About 90% BPA was removed by BGP at pH 7.0 and 40 °C [154]. The greatest enzyme activity was observed in the presence of polyphone oxidase from potato with more than 95% degradation for 60 min [155].

4.1.2 Bacterial transformation

BPA removal by bacteria is another prominent method. Using microbial communities with certain enzymes and metabolic pathways, BPA can be broken down and transformed through the process of bacterial remediation. The ability of this mechanism, called biodegradation, to lower BPA levels in a variety of environmental matrices, such as polluted soils and water systems, makes it especially noteworthy. Generally, BPA is recognized by bacteria which then carry it into their cells. Here, certain enzymes start the breakdown process which results in the development of intermediate chemicals. These intermediates are further metabolized through a variety of metabolic pathways and then gradually changing into less complex and possibly hazardous chemicals. Certain breakdown products might be incorporated into the bacterial cell, supporting other cellular functions or energy production. In the end, the breakdown products are either integrated into the bacterial biomass or released into the surrounding environment [162].

Various BPA-degrading bacteria have been identified and isolated to treat BPA in WWTPs including *Sphingomonas* sp., *Pseudomonas* sp., *Streptomyces* sp., *Bacillus* sp. and several more [163, 164]. Bacteria with greater

Table 3 Degradation of BPA by enzymatic treatment

Enzyme	Enzyme source	pH	Temperature, °C	Results	References
Peroxidase	Coprinus cinereus	9.0–10.0	40	Treatment removed all detectable acute toxicitytrogenic activity of BPA	[12, 153]
Bitter gourd peroxidise (BGP)	Bitter gourd	7.0	40	was removed in 4 h	[154, 155]
Horseradish peroxidise (HRP)	Horseradish	7.0	40	Efficient removal of BPA	[156]
	Horseradish	5.0,	25	80% BPA removal	[157]
Polyphenol oxidase	Mushrooms (Basidiomycota), and fruits and vegetables: potato (*Solanum tuberosum*), eggplant (*Solanum melongena*), burdock (*Arctium lappa*), and yacon (*Smallanthus sonchifolius*)	6.5	25	Enzyme oxidised BPA into monoquinone derivative	[157]
Soybean peroxidise (SBP)	Soybean	7.0	30	Huge reduction of BPA	[158]
Laccase	*Coriolopsis gallica*	5.0	40	Complete removal of BPA in 3 h	[159]
	Trametes versicolor	5.0	40	More than 95% removal of BPA	[160]
	Pleurotus sajor-caju CCB 019	5.0–7.0	20 to 40	Significant BPA removal rate (0.052 mg U^{-1} h^{-1})	[161]

BPA biodegradability are limited because the efficiency of the degradation process is very different from strain specificity in addition to the biomass amount, biological compounds, pH, and the presence of oxygen. *Sphingomonas* strains were identified from the sludge of WWTPs at a plastics manufacturing facility. This novel bacterium designated strain MV1 was Gram-negative, aerobic bacillus [165] and currently was the most frequently isolated BPA-degrading bacteria which use BPA as the sole source of carbon and energy. Strain MV1 consumed 10 g L^{-1} BPA within 4 d with the formation of 4-hydroxybenzoic acid, and 4-hydroxyacetophenone (4-HAP) as intermediate products. Greater BPA degradation was achieved by *Sphingomonas* sp. strains, which 115 mg L^{-1} BPA was consumed within 15-h [166]. Interestingly, *Sphingomonas bisphenolicum* strain AO1 from the soil of a vegetable growing field in Japan transformed 115 mg L^{-1} BPA after 6-h exposure [167].

In addition, *Pseudomonas putida* strains KA4 and KA5 were the two bacterial strains that were frequently isolated from the environment and showed greater BPA biodegradability using BPA as the sole carbon and energy source [168]. The degradation of BPA can reach up to 90% of BPA when the initial BPA concentration was 1 mg L^{-1}. Another BPA-degrading bacterial *Pseudomonas monteilii* strain N-502 was isolated [169]. 500 mg L^{-1} BPA was completely degraded within 10 d accelerated by folic acid, Mg^{2+} and Ca^{2+}. Ten more *Pseudomonas* strains could completely transformed 115 mg/L BPA [166]. Also, *Bacillus pumilus* strains isolated from Korean food, kimchi, were effective in removing BPA [170]. BPA with concentrations of 25 and 50 mg L^{-1} were successfully transformed without the detectible formation of 4-HAP in a medium supplemented with nutrients in the form of peptone, a beef extract, and a yeast extract in the presence of 10% NaCl [170].

Table 4 demonstrates different removal ratios for bacterial degradation of BPA. Recent studies found removal to nondetectable levels to 90% removal of BPA by bacterial strains tolerating high concentrations of this pollutant [171, 172]. Kang et al. [173] showed that *Streptomyces* sp. strain had greater BPA degradation potential with more than 90% in 10 d. Meanwhile several white-rot fungi removed BPA to undetectable levels in 12 h when the initial concentration of BPA was 50 mg mL^{-1} [174]. Interestingly, the transformation rate was higher in nutrient-rich YMG medium as compared to the minimal salts medium., *Sphingomonas* sp. strain BP-7 and *Sphingomonas yanoikuyae* BP-11R efficiently degraded 300 mg L^{-1} BPA without the formation of detectable BPA intermediates in the presence of activated carbon [175]. Furthermore, the degradation of BPA in the rhizosphere was enhanced in the presence of BPA-degrading bacterium, *Novosphingobium* sp. strain TYA-1 and a BPF-degrading bacterium, *Sphingobium yanoikuyae* strain TYF-1, isolated from the rhizosphere of *Phragmites australis.* BPA persisted in the soil in the absence of *P. australis*; hence, the interactions of *P. australis* in the rhizosphere efficiently removed BPA [176].

While bacteria and enzymes can facilitate the degradation of BPA, recycling or direct disposal of the resultant by-products, especially in landfills or sludge, may pose potential environmental risks. The process of BPA degradation may not always yield complete breakdown, leading to the formation of intermediates that could retain toxicity. Without a thorough analysis of these

Table 4 Transformation of BPA by different microorganisms

Microbe	Experimental Period	Result	References
Dracaena sanderiana endophytic bacteria	5 d	89.54 ± 0.8% removal of BPA	[171]
Pseudomonas putida YC-AE1	3 d	Transforms an extensive range of BPA concentrations (0.5 to 1000 mg L^{-1}) with complete transformation of 500 mg L^{-1}	[172]
Streptomyces sp. strain	10 d	> 90% BPA transformation	[173]
Irpex lacteus, *Trametes versicolor*, *Ganoderma lucidum*, *Polyporellus brumalis*, *Pleurotus eryngii*, *Schizophyllum commune*, and *T. versicolor* strains MrP 1 and MrP 13	12 h	BPA transformations of 76%, 98%, and nondetectable levelsusing *T. versicolor* strain MrP 1, *I. lacteus,* and *T. versicolor*, respectively	[174]
Sphingomonas sp. strain BP-7 and *Sphingomonas yanoikuyae* strain BP-11R	60 h	Transformation of BPA without formation of the intermediate 4-hydroxyacetophenone	[175]
Phragmites australis, *Novosphingobium* sp. strain TYA-1, *Sphingobium yanoikuyae* strain TYF-1	42 d	Enhanced removal of BPA from the rhizosphere	[176]
Pseudomonas aeruginosa PAb1 isolated from effluent of thermal paper industry	Not disclosed	Transforms 35 mM and less BPA in basal salt mineral growth medium	[177]

by-products, their introduction into recycling processes or direct disposal in landfills could result in unintended environmental contamination. Additionally, the potential for leaching of these compounds from landfills into the surrounding environment raises concerns about soil and water pollution. It is crucial to consider the long-term environmental impact and ensure compliance with environmental regulations to avoid adverse consequences and safeguard ecosystems. Advocating for a comprehensive approach that includes rigorous assessment, adherence to regulations, and sustainable disposal practices is essential to address the potential safety risks associated with recycling or direct disposal of BPA or its degradation by-products. Further research is warranted to better understand the fate and environmental implications of these by-products.

4.1.3 Limitation of biodegradation treatment of BPA

Although effective, biodegradation of BPA has certain drawback. In biodegradation, the breakdown of BPA is microbe dependent. These microbes can differ in their BPA specificity which could restrict the efficacy of the biological treatment since certain bacteria lack the enzyme machinery needed for successful BPA breakdown [178]. Previously, *Acinetobacter* sp. K1MN and *Pseudomona*s sp. BG12 have shown the capacity for BPA removal with reduction of its toxic effect [179]. In contrast, some strains of *Lactococcus* showed BPA adsorption but not degradation. In this regard, the hydrophobicity of BPA may be an important factor for its adsorption with lactococci where lactococci might adsorb BPA by hydrophobic-binding effect of proteins on cell surface [180]. Consequently, it is critical to find novel bacterial strains and look into how well they degrade BPA for effective biodegradation treatment. BPA may also have adverse effects on bacteria which may limit their capacity when attempting to process BPA. The toxicity of BPA might interfere with or disrupt the normal metabolic processes of the bacteria responsible for degradation which further hinder their ability to carry out efficient degradation processes [162]. Environmental factors such temperature, pH, and nutrient availability have a significant impact on how well biodegradation treatments work [181]. Microorganisms cannot function as well in suboptimal settings, which can lessen the efficacy of treatment [182]. Maintaining ideal conditions for microbial activity can be challenging when implementing biological treatment systems, and it may call for careful control. This complexity may increase the overall cost of the treatment procedure and create operational challenges. Besides that, extended periods of time may be necessary for biodegradation treatment to effectively remove BPA in certain situations, particularly when the concentration of BPA is high. When dealing with high BPA amounts, biological treatment may be less successful as microorganisms have the potential to degrade BPA at a comparatively sluggish rate. From the reported literatures, Sphingomonas sp. strain AO1 completely degrade 115 mg L^{-1} of BPA within 6 h, however *Sphingomonas* sp. strain MV1 completely degrade 10 g L^{-1} BPA within 4 days [183]. With regard to this, post treatment or combination of other techniques might be required to remove BPA efficiently. Another limitation of biodegradation treatment that is worth highlighting is the formation of intermediate compound during the BPA degradation process. These intermediates may possess distinct chemical properties and could potentially introduce new challenges, as their environmental fate and toxicity may differ from that of the original BPA [184]. Finally, the application of biodegradation treatment to BPA contamination in non-watery

matrices or solid substrates is limited because it is generally intended for aqueous conditions. When applied to solid substrates, the fundamental design of biodegradation treatment which is optimized for aqueous environments may result in diminished efficacy. Effective BPA removal or degradation is restricted by the unique physicochemical properties of solid materials, which calls for a sophisticated strategy catered to the unique features of these matrices. The complexity of implementing and managing biological treatment systems, coupled with their limited applicability to aqueous environments, adds operational challenges and considerations for optimizing BPA remediation strategies. Understanding all biodegradation treatment limitations is crucial for optimizing the use of biological treatment methods and for considering alternative or complementary approaches when addressing BPA contamination in different environmental settings.

4.2 Advanced oxidation processes

4.2.1 Photocatalysis

The development of advanced treatment techniques is now a subject of significant interest. Advanced oxidation processes have been commonly used for the treatment of various wastewaters because strong oxidants can easily transform a broad variety of organic and inorganic contaminants such as heavy metals, metalloids, and certain ions present in water [185]. Advanced oxidation processes use intensively reactive species such as hydroxyl radicals, superoxide anion radicals, and H_2O_2 as the main oxidizing agent. Generally, photocatalysis is a physical process of oxidation on the surface of catalyst excited by the source of light (Fig. 5) [186]. Photocatalytic degradation oxidizes complex organic compounds into smaller molecules, such as carbon dioxide and water, under light irradiation. In addition, photocatalysis may mineralize organic compounds to the harmless materials under sensible conditions due to several advantages include complete mineralization and up to 90% transformation efficiency in treating organic compounds at trace concentrations [187].

Among advanced oxidation processes, photocatalysis with titanium oxide (TiO_2), zinc oxide (ZnO), ferric oxide (Fe_2O_3), cadmium sulfide (CdS), and zinc sulfide (ZnS) has been reported with efficient degradation [188]. Table 5 demonstrates the degradation of BPA by various photocatalysts at various optimum conditions. For instance, in a study conducted by Ohko et al. [189], 99% of BPA at an initial concentration of 40 ppm was degraded by TiO_2 powder in the presence of Hg-Xe lamp after 15 h reaction. Similarly, 90% BPA degradation (initial concentration was 0.1 mM) was achieved in the presence of 0.8 g L^{-1} TiO_2 particles after 4 h of reaction under the same light irradiations [190]. Interestingly,

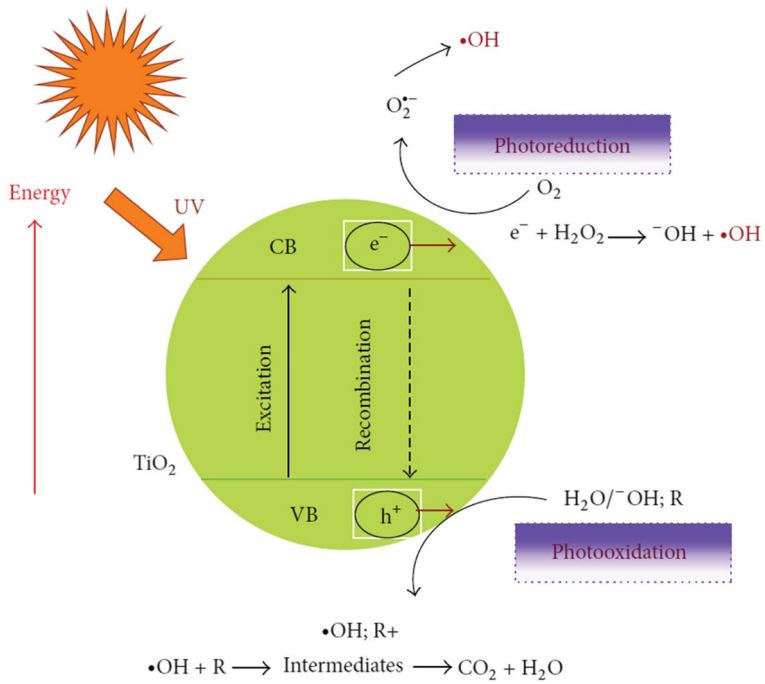

Fig. 5 principle action of photocatalytic degradation [186] *(Disclaimer: Copyright © 2013 Dipak Nipane et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.)

Table 5 Degradation of bisphenol A by different photocatalysis

Photocatalyst	Type of lamp	Initial concentration of BPA (ppm)	Time (h)	Efficiency (%)	References
TiO_2 powder	Hg-Xe lamp	40	15	99	[189]
TiO_2 particles	Hg lamp	22	4	90	[190]
Ce-ZnO powder, H_2O_2	UV Apelex	50	24	100	[191]
Titanium dioxide @ nanodiamond nanocomposite	UV lamps	10	1.7	100	[192]
TiO_2 suspension	UV lamps	20	60	97	[197]
TiO2 99 percent anatase	UVC	5	2	98	[198]
Zinc powder	Not mention	25	5	98	[199]
Ag_3PO_4	Xe lamp	10	0.17	100	[200]
Ag_3PO_4- graphene hydrogel	Xenon lamp	10	0.2	100	[201]
N, Co Codoped TiO_2 suspension	Solar light	Not disclosed	2.5	100	[202]
rGO–TiO_2 powder	Solar light	Not disclosed	0.5	100	[203]
Praseodymium doped TiO2 nanoparticles	Mercury lamp (UV-A light)	40	2.7	50	[204]

increased volume of photocatalyst would efficiently enhance the degradation of BPA where 98% degradation of BPA was reported in a 2 h of reaction in the presence of 120 mg L^{-1} TiO_2 99% anatase in the presence of UV light irradiations. Zn–O photocatalyst is another semiconductor oxide that has been widely studied due to the favorable photocatalytic properties. 98% of BPA at an initial concentration of 25 ppm was degraded by 0.1% w/w zinc powder after 5 h of reaction [190]. Bechambi et al. [191] found undetectable BPA after 24 h of reaction with 1 g L^{-1} Ce-ZnO powder, in the presence of 5.1 M H_2O_2 under UV irradiation. Recently, Hunge et al. [192] synthesised TiO_2-$_3$@nanodiamond (ND) composite photocatalysts which revealed that neutral and acidic conditions enhanced BPA degradation and complete removal was achieved with 8 mg of TiO_2-$_3$@ND composite at pH 5.1 under UV light after 100 min of experimental works. With regard to Table 5, although photocatalytic activity is efficient, ZnO based photocatalyst has a wide band gap and easily photocorrosive [193]. Silver-based photocatalyst such as oxoacid salt (Ag_3PO_4) is promising as an efficient photocatalyst [194]. There are materials less explored with pollutant management studies with literature on those materials scarce. All detectable BPA at an initial concentration of 10 ppm was transformed by 1.67 mg mL^{-1} Ag_3PO_4 and 150 mg Ag_3PO_4 graphene hydrogel in the presence of Xe lamp after 10 and 12 min of reaction, respectively. Similar to Zn–O based photocatalyst, Ag-based photocatalyst tends to undergo photocorrosion when exposed to a light source. This instability is the major limitation as a potential photocatalyst for pollutants removal. Anyway the photocatalytic reaction using TiO_2 has been extensively studied due to inexpensive, photostability, and long-term chemical stability against chemical corrosion [195]. Additionally, TiO_2 has become the focus of intense interest due to the capability to utilise UV light to efficiently catalyse reactions for the removal of a large variety of organic and inorganic contaminants such as heavy metals, metalloids, and certain ions [185, 196].

Based on an extensive evaluation of the literatures by Reddy et al. [205], substantial progress has been made with visible light-active photocatalysts especially in the case of titania-based materials. Black titania catalysts have shown excellent light absorption capacities and efficiencies in the treatment of aqueous pollutants. It is worth mentioning that titania, silver, and bismuth photocatalysts have excellent performance for the assessment of photocatalytic performance. Similarly, Nath et al. [184] describe application of several heterogeneous catalysts for the breakdown of BPA via Fenton-like processes. New materials for catalyst synthesis such as g-C3N4 and MXenes provide an interesting improvement in degradation efficiency [184]. While photocatalyst materials work excellently in BPA degradation, it is crucial to ensure that they can be readily separated from the reaction mixture in order to maximize their recycling and reusability. The overall sustainability and economy of photocatalytic processes are enhanced by the use of readily separable photocatalyst materials.

4.2.2 Hybrid process: photocatalytic membrane

Usually, a photocatalytic reaction is conducted in a suspension of catalyst particles; however, the separation of the TiO_2 particles is a major problem despite the efficiency of the photocatalytic activity. Fabrication of nanostructured TiO_2 hollow fibre photocatalytic membrane [206] demonstrated organic removal of 90% by filtration

and photocatalytic degradation of the membrane in the presence of UV irradiation. Recent fabrication of hollow fibre membranes from polyethersulfone (PES) and TiO_2 for water treatment was demonstrated to be photoactive in the degradation of Methylene Blue [207]. However, the selective layer was damaged during the UV-A irradiation of only 2 h at 0.6 mW cm^{-2}, which indicated that PES-TiO_2 ultrafiltration membranes are not resistant to UV radiation. Polyvinylidene fluoride (PVDF) hollow fibre membranes with nano-TiO_2 additives were fabricated previously [208] for dense algae cultivation and wastewater polishing. The nano TiO_2-embedded membranes improved the surface hydrophilicity with the total resistance of about 50% lower than that of the control. This study demonstrated that TiO_2 nanocomposite membranes had a better antifouling property for dense algae cultivation and wastewater polishing. Only a few were reported on the photodegradation of BPA by TiO_2 membrane as listed in Table 6. However, the degradation of BPA by photocatalytic TiO_2 hollow fibre membrane is still less explored, hence, available information is limited.

The incorporation of TiO_2 particles in the membrane matrix would eliminate the post-treatment process; however, it will decrease the photocatalyst active surface area [212]. This leads to the emerging in the exploration and fabrication of photocatalytic DLHF membrane for the excellent performance of immobilised photocatalyst. The outer surface layer of the membrane has embedded immobilised TiO_2 particles that catalytically react with contaminants in the presence of visible light and UV radiation, while the inner membrane layer will act as a separation layer. Interestingly, large loading of more than 50% by weight of photocatalyst immobilised at the outer layer membrane surface will ensure the fast transformation of the pollutant in the membrane photoreactor (MPR, Fig. 6). Briefly, MPR consists of submerged photocatalytic membrane modules fitted with light (LED and UV) source. The membrane modules will be immersed, and suction will be applied by the peristaltic pump for permeate collection. The compact design of photocatalytic membrane simultaneously transforms BPA and filter particulates, which allowed the membrane to be directly used for water treatment without leaving any photocatalytic particles in the water.

Dzinun et al. [213] has successfully fabricated a photocatalytic DLHF membrane with surface TiO_2 immobilised for the removal of nonylphenol. The DLHF membrane has a sandwich-like structure where both layers were filled with sponge like structure with the presence of finger-like void in the inner and outer space. The

Table 6 Degradation of BPA by photocatalysis immobilised in a membrane matrix

Photocatalyst	Nature of Lamp	Initial concentration of BPA (ppm)	Time (min)	Efficiency (%)	References
Nitrogen-doped TiO_2 DLHF	Visible LED lamp	10	360	82	[142]
TiO_2 thin films	UVA	10	2880	100	[209]
TiO_2/Ti film	Xenon lamp	0.3	180	85	[210]
TiO_2/Ti film	Xenon lamp	0.3	180	100	[211]

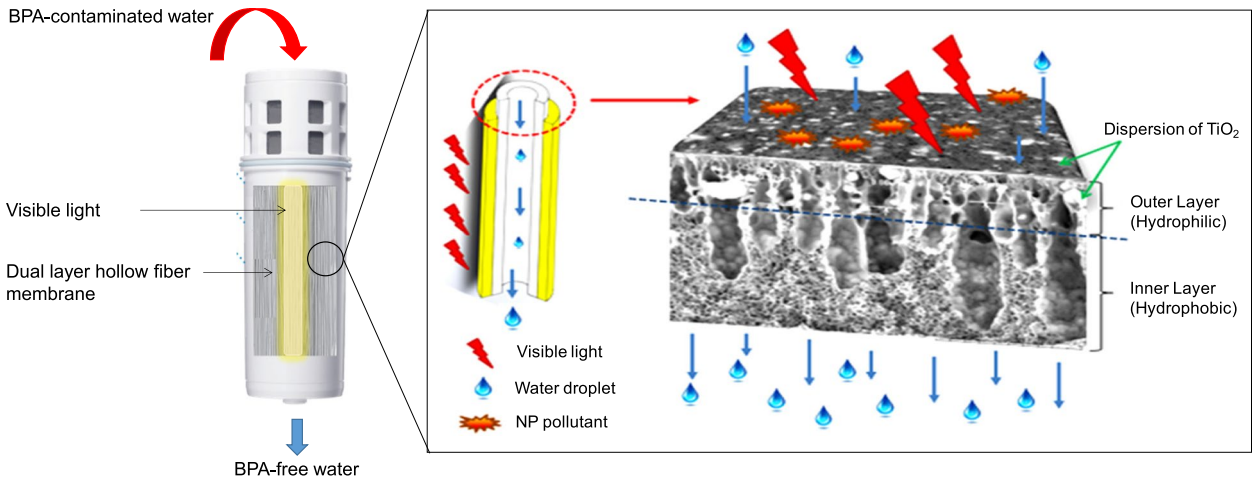

Fig. 6 Photocatalytic dual layer hollow fibre membrane in the MPR system

distribution of TiO_2 on the outer layer membrane surface has improved the membrane hydrophilicity, pore size, and permeability performance with almost 85% of nonylphenol removal was achieved under UVA irradiation. Previously, the fabrications of TiO_2/ZnO DLHF membranes were proposed as a superior sensor material in comparison to regular single layer hollow fibres (SLHFs) for the detection of reducing gases, such as CO [214]. The study reported that TiO_2/ZnO DLHF membranes demonstrated a better behaviour compared to SLFHs when detecting reducing gases but showed a lower response to oxidising gases such as NO_2. In this regard, the inner TiO_2 layer is responsible for electron absorption, enabling the use of a thicker outer layer than the one that would be required to achieve the same sensing properties in the case of SLHFs. Hence, TiO_2/ZnO DLHF membranes were proven to be advantageous over regular SLHFs for the detection of reducing gases.

Recently, Fu et al. [215] summarized the progress in photocatalytic degradation of BPA. According to the literature, a few issues still need to be addressed to improve the efficiency of photocatalytic degradation include: (1) low efficiency of photogenerated electron–hole pair recombination and visible light utilization; (2) large mass transfer resistance; and (3) poor selectivity of photocatalysts. To enhance photocatalytic efficiency, expanding the visible spectrum response range and minimizing the recombination of photogenerated electron–hole pairs are crucial. Different approaches, like using conductive materials as photocatalysts, have been suggested to shift the spectrum bands from UV to visible light, enhancing electron–hole separation and charge transfer speed. Interestingly, Kamaludin et al. [142] successfully fabricated visible light driven photocatalytic DLHF) membrane for the removal of BPA from water. Removal efficiencies of 88 and 87% of BPA were achieved after 360 min of visible and UV light irradiation, respectively. Interestingly, N-doped TiO_2 DLHF membrane effectively mitigates the effect of BPA. This finding shows that N-doped TiO_2 DLHF membrane is a promising option for the removal of BPA from contaminated water sources. Recently, Mohamad Noor et al. [216] successfully developed a Cu_2O composited PVDF DLHF membrane. Under visible light irradiation, about 75% of the 10 ppm BPA was successfully removed in 360 min, without the copper element being leached. Similarly, Sukaini et al. [217] reported that Cu_2O/WO_3/PVDF DLHF membrane efficiently remove 98% of 2-ppm BPA without copper leaching into the water sample. Interestingly, under dark conditions, the DLHF sample showed the capability of energy storage performance and could drive certain degradation after lighting off up to 71% of 2-ppm BPA. This finding showed that the development of photocatalysts with energy storage capability can provide a significant contribution toward more practical photocatalytic membranes, so photodegradation can occur even in dark conditions. In the meantime, Zakria et al. [218] successfully photodegraded about 74% of 2 mg/L BPA in synthetic wastewater while outstanding 91% removal of BPA in real treated sewage wastewater achieved using Cu_2O/PVDF thin film hollow fiber membrane after 360 min light irradiation. Despite the excellent photodegradation performance, the fabrication of DLHF membranes for photocatalytic degradation of BPA is still less explored. Similarly, the mitigation of BPA health effect on the treated water was not assessed. Therefore, relevant literature is limited.

To further emphasize the selection of photocatalytic membranes for BPA removal, comparison between biodegradation treatment and photocatalytic membrane is summarized into Table 7. This comparison will encompass factors such as efficiency, cost-effectiveness, environmental impact, scalability, and practical feasibility to provide valuable insights into the unique attributes of each method. This comparison will also provide a

Table 7 Comparison between biodegradation treatment and photocatalytic membrane

Factors	Biodegradation treatment	Photocatalytic membrane
Efficiency	Up to 90% effectiveness, dependent on microbial activity	Efficient degradation with the potential to bring BPA levels below detection limits
Cost-effectiveness	Generally cost-effective, but operational and maintenance costs may vary	Initial setup costs might be higher, but energy-efficient and potentially cost-effective in the long run
Environmental impact	Generally, environmentally friendly, but by-products and sensitivity to environmental conditions can be factors	Minimal environmental impact with proper design, as it relies on light activation for catalysis
Scalability	Can be scalable but may require optimization for different water sources	Can be designed for scalability with consistent performance across various water sources
Practical feasibility	Generally practical but may require careful monitoring and adjustment	Practical and potentially more versatile, especially with the integration of membrane technology, offering concurrent oxidation and separation

comprehensive view to support the preference for photocatalytic membranes in BPA degradation.

Photocatalytic membranes, while offering notable advantages in the degradation of BPA, are not without significant drawbacks. One well-known limitation is their reliance on energy-intensive UV light for optimal performance, introducing potential operational cost concerns and limiting energy efficiency. However, photocatalytic membranes benefits, such the capacity to effectively break down persistent pollutants like BPA and act as a physical barrier for membrane-based separation underscore their potential as a multifaceted solution for water treatment. The ability to simultaneously address chemical degradation and physical separation enhances their overall efficacy. For instance, while photocatalysis may face challenges like energy consumption and potential catalyst fouling, the integration of photocatalytic membranes combines the benefits of membrane filtration and advanced oxidation processes. This integration enhances the overall efficiency of BPA removal by addressing both physical separation and degradation aspects in a single step. Comparing photocatalytic membranes with other treatment options, such as biodegradation and enzymatic treatments, it reveals a nuanced landscape. Biodegradation and enzymatic treatments offer eco-friendly solutions leveraging natural processes but may face limitations in terms of scalability and the need for optimal environmental conditions [219]. Photocatalytic membranes, on the other hand, provide a continuous and scalable treatment method that operates under various environmental conditions [220]. However, the energy requirements and potential catalyst fouling must be carefully considered. In summary, the advantages of photocatalytic membranes, particularly their dual functionality, can offset certain drawbacks. When compared with other treatment options, the choice between them should be contextual, considering factors such as energy efficiency, scalability, and the specific environmental conditions in which the treatment will be applied. The holistic evaluation of these factors will guide the selection of the most suitable and effective treatment approach for addressing BPA contamination.

5 Conclusions

This paper provided an outline on the presence of BPA in surface water, household water, and drinking water and adverse health effect associated with BPA exposure. Numerous studies on metabolic diseases have proven that metabolic diseases in adults can originate *in utero* and during infancy. Furthermore, experimental research animals support the concepts of a causal link between adverse events *in utero* and early life with the development of cardiovascular and metabolic disease in later life. In addition, BPA levels detected in the umbilical cord, foetal cord serum and amniotic fluid indicate that BPA can cross trans-placentally and potentially impact the developing foetus. Multiple studied rodents and primate demonstrated the long-term detrimental effects of BPA exposure on foetal organs. Therefore, BPA exposure during pregnancy should be given a better emphasis to lower the burden on the development of metabolic diseases in the future.

BPAs could be successfully removed by enzymatic and microbial treatment in the presence of suitable mediators. AOPs, including photocatalysis, ozonation, and Fenton reactions, have shown effectiveness in breaking down BPA and its byproducts through the generation of highly reactive species. Integrating AOPs with photocatalytic membranes could further enhance the removal efficiency and ensure complete mineralization of BPA, addressing both chemical degradation and physical separation in one step. The appeal of photocatalytic degradation stands out due to its unique properties, including anti-fouling, superhydrophilicity, and concurrent photocatalytic oxidation and separation. Photocatalytic membranes emerge as a superior solution for degrading BPA in drinking water and its sources when compared to biodegradation treatments. The continuous and scalable nature of photocatalytic membranes ensures uninterrupted BPA degradation, making them suitable for both small-scale applications and large-scale municipal water treatment. Their robustness and ability to operate under diverse environmental conditions further enhance their reliability. Importantly, photocatalytic membranes provide a dual functionality by simultaneously physically separating contaminants and chemically degrading BPA. This distinguishes them from biodegradation methods, which may face challenges in scalability and robustness, relying on microbial activity that can be sensitive to fluctuations in environmental parameters. Photocatalytic membranes, with reduced dependence on specific microbial conditions, offer a versatile and effective approach to address BPA contamination in drinking water, presenting a holistic solution that combines chemical degradation with efficient physical separation. However, some technical issues still need to be resolved such as increase the contact area between photocatalyst, and UV for more effective photocatalytic activity. Furthermore, solar irradiated photocatalytic membranes need to be explored, since most of photocatalytic membranes in the literature are excited only by UV. Finally, there is still a lack of durability data of these photocatalytic membranes. It is inevitable that some photocatalyst may leach off the membranes, hence shortening their lifespan. As the use of BPA is expected to increase in the future, more deliberate efforts to develop diverse yet effective treatment

options are imperative to manage water quality. Further research is urgently needed to investigate the transformation pathways of BPA and the intermediates as well as to determine the effect and fate of the intermediates in the environment.

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Authors' contributions
RK and ZR wrote the main manuscript text. MHDO and SK are responsible for editing the article. All authors reviewed the manuscript.

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Not applicable.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
We confirm that the manuscript has been read and approved by all named authors. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We understand that the Corresponding Author is the sole contact for the Editorial process. He is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

Competing interests
The authors declare they have no competing interests.

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