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Toxic Effects of Bisphenol A on Fat Deposition, Weight Gain, in Rats

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Annotation: It has been shown that bisphenol A (BPA) is associated with major alterations in lipid metabolism and the buildup of fat. There is a significant dose-response connection between exposure to BPA and a rise in body weight, notably in animal models. This association in particular has been shown in animal models. The substance acts as a substitute for estrogen, which causes disruptions in endocrine functioning and encourages adipogenesis. Adipogenesis is the process by which preadipocytes transform into mature fat cells, which ultimately results in increased fat accumulation. BPA exposure during critical developmental periods, such as prenatal and postnatal, has been shown to result in an increase in adipose tissue mass, triglyceride levels, and cholesterol levels, as well as a decrease in high-density lipoprotein (HDL) levels, which is indicative of a metabolic disturbance. These findings were derived from studies conducted on rats. The results of this study provide credence to the concept that bisphenol A (BPA) functions as an obesogenic, giving rise to the possibility that it may contribute to metabolic illnesses such as insulin resistance and type 2 diabetes. It was noted that the modifications in lipid metabolism and the promotion of fat storage were present throughout a range of doses of BPA, with the effects being more significant at higher doses. Because of the effects that exposure to BPA has on body weight and fat deposition, it seems that even low-level exposure to BPA may contribute to the development of metabolic health problems over the long run.

Keywords: Bisphenol A (BPA), obesity, lipid metabolism, fat accumulation, body weight, metabolic disorders, triglycerides, cholesterol, HDL, LDL, VDL.

1. Introduction

Bisphenol A (BPA) is a synthetically produced compound widely used in industrial chemistry, particularly in the production of polycarbonate plastics and epoxy resins. These materials are essential in numerous consumer products, such as food and beverage containers, medical devices, and various toys **[1][2][3].** Despite the significant benefits and extensive use of this compound in various industries, it is important to point out that BPA is categorized as a potent endocrine-disrupting chemical (EDC) that poses substantial health risks, including serious reproductive, metabolic, and developmental disorders **[4][5][6].** The structural similarity of the chemical to estrogen allows it to interact with multiple hormone receptors in the human body, leading to carcinogenic effects and potentially contributing to the development of various malignancies, particularly breast and prostate cancer **[4][7].** BPA has been demonstrated to induce oxidative stress and cause epigenetic changes, resulting in considerable DNA damage and subsequent cellular dysfunction, which may increase the risk of long-term health effects **[4][8].** The widespread prevalence of this compound in the environment, due to industrial effluents and the degradation of BPA-containing products, has led to significant human exposure. Traces of this chemical have been identified in biological samples, including urine, blood, and fetal tissues **[2][9].** In response to these concerns, regulatory frameworks have been established in specific regions to reduce exposure to BPA, particularly aimed at protecting vulnerable populations, including infants and pregnant women **[9].** Nonetheless, the complexity of BPA's nonmonotonic dose-response behavior complicates the identification of safe exposure thresholds, as scientific evidence indicates that even minimal doses can produce adverse biological effects **[5].** Researchers are actively pursuing safer alternatives to BPA by investigating bio-based compounds derived from lignins and naturally occurring phenolics as potential substitutes to mitigate the risks associated with traditional BPA usage **[1][10] .** It is essential to maintain vigilance regarding the emergence of BPA analogues, such as BPS and BPF, which may possess toxicological properties. This situation calls for further detailed research and comprehensive regulatory assessment **[10].**

Exposure to Bisphenol A (BPA) is disturbingly widespread across human populations and various animal species, as verified by several scientific research studies and extensive assessments detailing its prevalence. BPA, acknowledged as a synthetic compound that imitates estrogen, is widely employed in the production of various consumer products, especially in the creation of plastics and epoxy resins, which are essential to daily life and societal infrastructure **[11][6].** The widespread use of BPA in numerous applications results in its leaching into the environment, contaminating vital elements such as air, water, and soil, which subsequently allows its infiltration into the food chain; this sequence of events makes exposure to this chemical nearly inevitable for both humans and wildlife **[12][13].** Numerous biomonitoring studies have demonstrated the detection of BPA in the urine and blood serum of individuals worldwide, with concentrations often exceeding those predicted by toxicokinetic models, suggesting that actual exposure levels may be considerably higher than previously estimated **[14][15].** In the realm of human health, exposure to BPA has been associated with various severe health complications, including reproductive problems, metabolic disorders, and developmental abnormalities, as well as chronic conditions such as obesity, diabetes, and hypertension, all of which have been thoroughly documented in recent research literature **[16][17][5].** Moreover, empirical research on animal models has validated these concerning findings, indicating that analogous harmful effects might occur even with modest levels of BPA exposure, thereby heightening considerable apprehensions about its safety **[18][19].** The ubiquitous nature of BPA, coupled with its ability to cause harm at low exposure levels, underscores the pressing need for continued research and a comprehensive reassessment of existing safety standards, all intended to reduce its detrimental health impacts on multiple species, including humans **[5].**

Bisphenol A (BPA) is a prevalent industrial chemical associated with considerable health risks, primarily as an endocrine disruptor affecting metabolic health, obesity, and related conditions. The capacity of BPA to imitate natural estrogens and attach to estrogen receptors disturbs endocrine control, influencing neuroimmune pathways and gut microbiota, perhaps resulting in obesity **[20][21]** Epidemiological and mechanistic investigations have associated BPA exposure with metabolic diseases, including obesity, insulin resistance, and type 2 diabetes, identifying BPA as an obesogen that facilitates adipogenesis and lipid accumulation **[21][22].** chemical's estrogenic characteristics and structural resemblances to thyroid hormones enable it to interfere with endocrine systems at several levels, affecting cellular processes such as apoptosis, proliferation, and inflammation **[21].** Exposure to BPA is especially alarming during pivotal developmental phases, since it can intensify the repercussions of high-fat diets, resulting in elevated body weight, hyperinsulinemia, and glucose intolerance in adulthood **[23].** Notwithstanding these findings, the scientific community is polarized, with certain studies indicating substantial metabolic effects of BPA, whereas others demonstrate negligible or no impact, underscoring the necessity for standardized animal models and additional research to elucidate BPA's influence on human health **[23][24].** bodies have raised apprehensions regarding the safety of BPA, resulting in prohibitions on specific goods, including baby bottles; however, its alternatives, such as bisphenol S (BPS) and bisphenol F (BPF), may similarly present health hazards **[25][26][27].** The current discourse highlights the intricacy of BPA's influence on metabolic health and the imperative for further exploration of its processes and consequences.

BPA exposure has been associated with lipid metabolism changes and serum lipid profiles, which enhance fat deposition and cardiometabolic risks. According to **[28].** BPA disrupts lipid metabolism by upregulating lipid synthesis genes and changing lipid transport genes, causing fat accumulation and metabolic abnormalities in Sesarmops sinensis. Longitudinal studies show that BPA exposure increases blood insulin levels, insulin resistance, fat mass, waist circumference, and BMI in humans, indicating increased cardiometabolic risk **[29][30].** In animal models, perinatal BPA exposure leads to increased body weight, dyslipidemia, and reduced hormone-sensitive lipase expression in adipose tissue, resulting in higher triglyceride levels and lower HDL-C levels **[31].** Gestational BPA exposure in rats alters metabolic pathways, including mTOR/CRTC2/SREBP1, causing higher triglyceride and total cholesterol levels **[32].** Furthermore, BPA's metabolite, BPA β-D-glucuronide, promotes adipocyte differentiation and fat storage, highlighting its function in disrupting lipid metabolism **[33].** EARTH discovered no direct link between BPA and blood lipid levels in pregnant women, but it suggests interactions with other variables, including BMI **[34].** Lipid metabolism abnormalities and unfavorable serum lipid profiles are linked to BPA exposure, stressing the need for mitigation methods, particularly during critical growth periods **[35].** BPA exposure has a considerable impact on fat deposition and weight increase in mice under controlled experimental settings, with effects differing by sex and genetic background. Chronic BPA exposure, particularly during the perinatal and postnatal periods, has been linked to increased adipose tissue mass and elevated serum cholesterol levels, with female mice gaining 13% weight and 132% in adipose tissue weight at lower BPA concentrations **[36].** Furthermore, BPA stimulates the development of preadipocytes into mature fat cells, increasing lipid accumulation and adipogenic marker expression **[33].** BPA's effects are dose-dependent and may cause obesity even at low doses, with male mice accumulating more fat than females **[37][38].** Furthermore, BPA exposure modifies adipokine production, which affects insulin sensitivity and metabolic health, highlighting its function as an obesogen in mice **[38][39].**

Understanding the impact of Bisphenol A (BPA) on obesity and lipid metabolism is critical given the increasing global prevalence of obesity and related metabolic illnesses, which already affect over half a billion people **[40].** BPA, an endocrine-disrupting chemical, has been linked to a variety of metabolic disorders, including obesity and type 2 diabetes, by altering endocrine functions and promoting adipogenesis via mechanisms such as increased lipid accumulation and inflammatory responses **[21][22].** According to research, BPA exposure may affect gene expression in adipocytes, especially in children, resulting in metabolic dysregulation and an increased risk of obesity **[41].** Furthermore, the idea of environmental obesogens emphasizes the involvement of substances such as BPA in the genesis of obesity, implying that widespread exposure might greatly contribute to the obesity pandemic **[42].** Thus, understanding BPA's processes might help influence public health policies and regulatory actions targeted at reducing its effect on metabolic health.

Objectives of the Study

- ✓ Investigate BPA’s effects on fat accumulation in various tissues (e.g., liver, adipose).
- ✓ Examine how BPA exposure affects body weight and metabolic regulation.

2. Methodology

2.1. Study Design:

2.1.1. Experimental Animals

The animals were reared at the University of Al-Qadisiyah College of Science animal house. In Iraq, the animal house provided 24 albino rats, 12 mature females, and 12 adult males for the research. Rats were 75–90 days old and weighed 145–200 g for females and 85–150 g for males. Metal cages protected the rats against BPA, which plastic cages might expose them to. Glass bottles supplied water to the animals in cages. The animals spent 30 days in the animal home before the trial. To adjust to the animal home environment, they were given a 12-hour light-dark cycle and a 20-25°C temperature.

2.1.2. Components of the diet

The composition of the feed used is summarized in **Table (1**) gant

Table (1) components of a kilogram of the diet

N.O	Materials	Quantity
1.	wheat	530 g
2.	Corn	250 gm
3.	Raw Protein	180 gm
4.	vegetable oil	20 g

5.	Milk powder (powder)	20g
6.	minerals and vitamins	1g

2.1.3. Animal Distribution

Rats were brought to the University of Al-Qadisiya Faculty of Science animal house in this experiment. After separating males and females, rats were placed into eight groups.

Table (2) The rats were distributed into eight groups.

Group	Composition	Treatment	Purpose
Control	3 female and 3 male rats	Administered 0.5 ml/kg of body weight of maize oil daily	Baseline comparison group, no BPA exposure
Group A	3 female and 3 male rats	Administered 200 mg/kg body weight of BPA in corn oil daily	High-dose BPA exposure to assess effects on fat deposition and weight gain
Group B	3 female and 3 male rats	Administered 100 mg/kg body weight of BPA in corn oil daily	Moderate-dose BPA exposure to evaluate effects on fat deposition and weight gain
Group C	3 female and 3 male rats	Administered 50 mg/kg body weight of BPA in corn oil daily	Low-dose BPA exposure to assess effects on fat deposition and weight gain

The experiment runs from 1/1/2025 to 1/4/2025. The rat adjusted to its new habitat in the first month. The first rats were given BPA orally for six weeks before being collected for the latter test.

2.1.4. BPA Compound

BPA (97.0 percent, CAS 80-05-7) was purchased from the Indian CDH Company. Pure corn oil was utilized to dissolve BPA for dose the control group. On a weekly basis, BPA was dissolved in corn oil and given to each group according on their body weight, with the control group getting corn oil.**[43][44].**

2.1.5. Blood Samples Collection

After weighing and anesthetizing in a cotton bag infused with chloroform and secured with a rubber band, rats were heart-stabbed to obtain 8 ml of blood, as shown in the figure. Blood was drawn for initial analysis. For decisive evaluations, samples were taken eight weeks after phase II BPA consumption. The specimens were placed in a tube without anticoagulant with 6 ml of blood and centrifuged at 3000 revolutions/min for 15 minutes to extract serum for hematological and biochemical tests. Two weeks following the first test, blood samples were collected, and eight weeks later, further samples were collected. The necessary exams were done quickly.**[45]**

2.1.6. Estimation of Lipid Profile standards

Table (3) Estimation of Lipid Profile standards

Parameter	Method	Ref.
Total Cholesterol (CHOL)	Enzymatic reaction	**[46]**
Triglyceride (TG)	Measured according to the method described by Fassati & Prencipe (1982) associated with Tinder reaction	**[47]**
HDL-Cholesterol (HDL-C)	Precipitation of LDL, VLDL, and chylomicrons by phosphotungstic acid and magnesium chloride. HDL-C measured in supernatant with a total cholesterol reagent	**[48]**
Low-density lipoprotein	Procedure described by Lopes-Virella et al. (1977)	**[49]**

cholesterol (LDL)		
Very low-density lipoprotein cholesterol (VLDL)	Calculated by dividing serum TG by five	**[50]**

3. Results

3.1. Toxic Effect of BPA on lipid profile

The study found a substantial increase (P < 0.05) in cholesterol, triglycerides, and low-density lipoprotein levels. The study found a significant decrease (P < 0.05) in HDL levels in all treatment groups (A, B, and C) compared to the control group, across genders, and between the second (8 weeks) and initial (2 weeks) assessments, as shown in Table (4). The statistical evaluation showed a strong positive correlation in the lipid profile: cholesterol (r = +0.99) in both males and females, TG (r = +0.99) in males and (r = +0.98) in females, and LDL (r = -0.97) in males and (r = +0.95) in females. The statistical analysis also showed a strong negative association in HDL (r = -0.96) among men and (r = -0.89) among females (Tables 4-8).

Table (4) Results Toxic Effect of BPA on lipid profile

MALE						
Period	**Parameter Mg\dl**	**Control**	**A (200 mg\kg)**	**B (100 mg\kg)**	**C (50 mg\kg)**	**LSD**
2 WEEKS	**Chole.**	**71.21±11.21 D**	**91.25±9.85 A**	**84.25±9.63 B**	**77.25±7.69 C**	**4.21**
	TG	**44.25±3.65 D**	**80.21±6.55 A**	**68.54±4.96 B**	**57.32±3.99 C**	**5.31**
	HDL	**44.12±3.57 A**	**31.01±3.01 D**	**36.41±.3.21 C**	**39.52±2.85 B**	**2.45**
	LDL	**155.21±21.11 D**	**174.21±19.52 A**	**168.25±14.55 B**	**162.85±11.63 C**	**6.02**
FEMALE						
Period	**Parameter Mg\dl**	**Control**	**A (200 mg\kg)**	**B (100 mg\kg)**	**C (50 mg\kg)**	**LSD**
2WEEKS	**Chole.**	**71.32±11.05 D**	**92.49±8.66 A**	**82.54±7.24 B**	**78.55±7.49 C**	**3.65**
	TG	**45.01±4.51 D**	**79.21±6.24 A**	**67.94±5.33 B**	**58.61±4.25 C**	**2.55**
	HDL	**44.15±2.99 A**	**30.25±1.96 D**	**38.52±2.55 C**	**41.22±2.96 B**	**1.86**
	LDL	**155.33±22.01 D**	**173.55±16.21 A**	**169.84±14.21 B**	**160.21±13.65 C**	**7.21**
MALE						
Period	**Parameter Mg\dl**	**Control**	**A (200 mg\kg)**	**B (100 mg\kg)**	**C (50 mg\kg)**	**LSD**
8 WEEKS	**Chole.**	**71.22±8.22 D**	**112.52±10.23 A**	**95.24±9.21 B**	**85.36±8.04 C**	**8.22**
	TG	**44.25±3.96 D**	**89.85±4.88 A**	**75.63±3.56 B**	**64.21±2.96 C**	**4.63**
	HDL	**44.12±2.63 A**	**26.52±1.99 D**	**29.63±1.63 C**	**36.52±3.21 B**	**2.51**
	LDL	**155.21±19.21 D**	**188.52±17.22 A**	**175.63±16.52 B**	**161.21±14.25 C**	**7.89**
FEMALE						

Period	Parameter Mg\dl	Control	A (200 mg\kg)	B (100 mg\kg)	C (50 mg\kg)	LSD
8 WEEKS	Chole.	71.32±5.21 D	99.56±6.33 A	88.52±7.22 B	79.66±5.96 C	5.54
	TG	45.01±3.63 D	88.62±4.52 A	77.12±5.21 B	68.44±4.22 C	6.11
	HDL	44.15±2.96 A	25.11±2.01 D	28.99±1.98 C	42.52±3.65 B	1.96
	LDL	155.33±18.22 D	189.52±17.63 A	177.85±14.25 B	171.25±13.63 C	5.84

Table (5) Correlation Coefficient between 2 weeks and 8 weeks of Dosing BPA on Lipid Profile

Correlation Coefficient	Males			
	Chole.	**TG**	**HDL**	**LDL**
	0.9946	0.9965	0.9686	0.9727
	Females			
	Chole.	**TG**	**HDL**	**LDL**
	0.9928	0.9876	0.8953	0.9557

3.2. Effect of BPA on the body weights

Table (6) showed Significant body weight changes ($P < 0.05$) were seen in all treatment groups compared to the control group, pre-experiment weights, and groups A, B, and C. Compared to the control group, all treatment groups showed substantial body weight changes ($P < 0.05$). Female groupings increased more than male groups. Significant body weight differences ($P < 0.05$) were seen in all treatment groups compared to the control group. The second test after eight weeks of dosing showed a considerable increase from the first in two weeks. Table (7) Statistical Analysis, showed a strong positive correlation between body weights: the first experiment showed a completely positive correlation ($r = 0.98$ in males and 0.99 in females), and the end experiment showed a completely positive correlation ($r = 0.99$).

Table (6) Effect of BPA on Body Weights

MALES					
groups Period	**Control**	**A (200 mg\kg)**	**B (100 mg\kg)**	**C (50 mg\kg)**	**LSD**
Before of experimental	176.8±12.21 A	177.5±11.55 A	175.6±10.52 A	174.1±9.68 A	5.32
2 weeks	181.5±11.21 A	187.6±9.52 B	185.8±10.21 C	185.2±11.55 D	2.32
8 weeks	220.1±12.52 A	238.5±9.53 B	231.6±10.85 C	228.5±13.65 D	6.52
FEMALES					
groups Period	**Control**	**A (200 mg\kg)**	**B (100 mg\kg)**	**C (50 mg\kg)**	**LSD**
Before of experimental	158.2±9.52 A	161.3±6.33 A	168.5±5.22 A	165.6±4.11 A	9.51
2 weeks	174.3±5.33 A	175.6±6.96 D	176.8±5.85 C	178.6±4.21 B	5.44
8 weeks	196.6±9.02 A	245.3±14.22 D	239.5±13.52 C	229.2±12.41 B	6.25

Table (7) Correlation Coefficient between 2 weeks and 8 weeks of Dosing BPA on body weights

Correlation Coefficient	**Males**	
	First	**End**
	0.9846	**0.9996**
	Females	
	First	**End**
	0.9901	**0.9911**

4. Discussion

Researchers studying the toxicity of bisphenol A (BPA) are increasingly emphasizing its role as an endocrine disruptor that can interfere with metabolic processes, particularly in fat formation and weight gain. **[51][33].** found that BPA exposure promotes preadipocyte development into adipocytes, leading to an increase in fat cell numbers and lipid buildup, which is a factor in obesity. Research has shown that some developmental windows are more vulnerable to the effects of BPA, since even low levels may trigger adipogenesis and interfere with endocrine signaling **[52][53].** The fact that BPA-G, a metabolite of BPA, may still have biological effects adds another layer of complexity to the problem of determining whether or not it has obesogenic potential **[33].** More research into the long-term health effects of BPA and its analogues is needed since the available data indicates that they may have a substantial impact on body mass index and the prevalence of obesity **[53][54].**

The application of three doses—low, medium, and high—of bisphenol A (BPA) in toxicity studies concerning fat accumulation in rats is crucial for elucidating the dose-response relationship and the non-monotonic effects of BPA.Studies demonstrate that BPA exposure is associated with notable increases in body weight and changes in lipid profiles, with effects differing by the dosage and sex of the offspring **[55].** Low doses (1 µg/ml) were linked to increased body weight and dyslipidemia, whereas higher doses (10 µg/ml) led to more significant alterations in serum triglycerides and hormone-sensitive lipase expression **[56].** The distribution of BPA across different tissues, including neuroendocrine organs, indicates that increased doses result in greater tissue accumulation, potentially worsening metabolic disruptions **[57].** This multi-dose methodology enables researchers to clarify the complex relationship between BPA exposure and metabolic outcomes, offering insights into its possible function as an obesogen **[58].**

Because of the physiological and genetic parallels that exist between humans and rats, rats were used in research that investigated the toxicity of bisphenol A (BPA). These characteristics made rats appropriate models for evaluating the effects of the substance on the body. Research has shown that bisphenol A (BPA), which is an endocrine disruptor that is often present in plastics, may cause severe toxicological alterations in various organ systems, including the liver, kidneys, and lungs of rats **[59][60].** As an example, investigations have shown significant histopathological modifications and biochemical changes, including raised liver enzymes and changed hormonal levels, because of exposure to BPA **[61][62].** Additionally, the calculation of median fatal doses (LD50) in rats has offered essential insights into the acute toxicity of BPA. These findings have shown that there is a tight margin between doses that are deadly and those that are not lethal **[63][64].** Rats are used in studies that investigate the toxicity of bisphenol A (BPA), which helps researchers get a better understanding of the possible health hazards that are linked with human exposure to this chemical.

Bisphenol A (BPA) altered lipid profiles in rats significantly when compared to control groups, according to a number of studies. Triglyceride (TG) and total cholesterol (TC) levels in the blood and liver of male offspring were found to be higher after gestational exposure to bisphenol A (BPA), suggesting that lipid metabolism was altered **[65].** The same study found that exposure during lactation altered lipid profiles; specifically, adult male rats had elevated levels of LDL cholesterol (LDL-C) **[66].** Perinatal exposure to bisphenol A (BPA) in female offspring was shown to increase body weight and decrease levels of HDL cholesterol (HDL-C), with larger doses of BPA being associated with significantly higher levels of total cholesterol (TG) **[67].** In addition, the effects of dyslipidemia—high TG and TC levels with low HDL-C—insulin resistance, and hepatic fat accumulation were worsened by BPA and fructose consumption together **[68].** These results indicate that exposure to bisphenol A (BPA) at any stage of pregnancy, delivery, or lactation causes dyslipidemia, defined as elevated total cholesterol (TG) and low-density lipoprotein (LDL) levels and reduced HDL-C levels, suggesting an interference with normal lipid metabolism. Overall, BPA exposure poses a significant risk to lipid homeostasis, potentially leading to metabolic disorders in later life. The mechanisms underlying these changes involve alterations in gene expression related to lipid synthesis and oxidation, such as the upregulation of sterol regulatory element binding proteins (SREBP-1) and the downregulation of fatty acid oxidation genes **[65][68].** Exposure to Bisphenol A (BPA) has a significant impact on cholesterol and triglyceride levels in rats, especially during gestation and early developmental stages. Research demonstrates that gestational exposure to BPA results in elevated serum triglyceride (TG) and total cholesterol (TC) levels in male offspring, linked to modifications in hepatic lipid metabolism pathways involving mTOR/CRTC2/SREBP-1 signaling **[65][66]** BPA exposure specifically down-regulates genes related to fatty acid oxidation and up-regulates those associated with fatty acid synthesis, leading to dyslipidemia **[65].** Moreover, the simultaneous exposure to fructose and BPA intensifies lipid metabolic disturbances, resulting in elevated triglyceride and total cholesterol levels, as well as increased insulin resistance and hepatic fat accumulation **[68].**

Bisphenol A (BPA) is associated with weight gain via multiple mechanisms, chiefly owing to its function as an endocrine disruptor. BPA mimics natural estrogens and binds to their receptors, disrupting endocrine regulation and influencing metabolic processes, potentially resulting in obesity **[69][70].** Exposure to BPA during critical developmental periods, particularly prenatal and early childhood, is linked to a heightened risk of weight gain and obesity in later life **[71][72].** Exposure to BPA influences adipocyte differentiation and function, facilitating adipogenesis and lipid accumulation, which are critical components in the development of obesity **[73][74].** BPA exposure disrupts glucose metabolism, impairs glucose tolerance, and alters the oxidant-antioxidant balance, thereby contributing to weight gain **[75].** Epidemiological studies indicate a positive correlation between urinary BPA levels and elevated body mass index (BMI) and waist circumference in adults, thereby supporting the obesogenic potential of BPA **[76].** Animal studies support these findings, demonstrating significant weight gain in mice exposed to BPA, even at low doses, and emphasizing the compound's effects on hematological parameters and renal health **[77].** BPA's effects extend beyond direct exposure; transgenerational epigenetic mechanisms suggest that BPA-induced metabolic disturbances may be inherited by subsequent generations **[74].** The diverse pathways highlight the complexity of BPA's involvement in obesity, indicating the need for additional research to clarify its mechanisms and reduce its effects on public health.

BPA, an endocrine disruptor that mimics estrogen and binds to estrogen receptors, affects adipogenesis and lipid metabolism, causing fat storage and weight gain. Increased expression of adipogenic markers like PPARγ, C/EBPα, and lipoprotein lipase in both human and animal models indicates that BPA exposure promotes the differentiation of preadipocytes into mature adipocytes, a process mediated by estrogen receptor pathways **[33][78].** BPA β-D-glucuronide, BPA's metabolite, also stimulates adipogenesis, demonstrating its obesogenic effects go beyond estrogenic action **[33].** BPA-induced hepatic lipid accumulation is further exacerbated by epigenetic alterations such as DNA methylation patterns at the promoters of lipid metabolism genes like Srebf1 and Srebf2. BPA exposure alters glucose tolerance and the oxidant-antioxidant balance, increasing inflammatory cytokine production and metabolic dysfunctions, which causes obesity **[75].** BPA affects adipogenesis via receptor interactions and epigenetic alterations, including decreased methylation at the Pparγ promoter, enhancing adipogenic capacity **[79].** These methods demonstrate BPA's obesogenic potential, especially during important developmental windows, which may cause transgenerational metabolic abnormalities **[74].**

Public policy solutions to decrease bisphenol A (BPA) exposure stress comprehensive regulatory and public health efforts. BPA exposure has been reduced most by policy initiatives, including BPA restrictions in products and packaging [80]. Due to mounting health concerns, the European Food Safety Authority (EFSA) has drastically cut the tolerated daily intake (TDI) for BPA, and other areas should follow suit **[81[82].** A coalition of environmental and public health organizations in the US has petitioned the FDA to restrict BPA limits in food-contact materials to 0.5 ng BPA/kg of food, substantially lower than existing regulations **[82].** Because BPA is used in food containers and dental sealants, tougher laws are needed to safeguard vulnerable groups, notably newborns and children, who are at increased risk owing to their developing systems **[83][84].** Public health campaigns can promote BPA-free items and safer alternatives, such as plant-based bioactive chemicals, to reduce BPA's toxicity **[85].** Despite BPA's safety concerns, academics and health groups agree that more regulations are needed to reduce exposure and preserve public health **[86][87].** Biomonitoring data should be integrated into regulatory frameworks to provide accurate exposure evaluations that match real-world situations **[88].** Regulatory measures, public awareness, and scientific research are needed to limit BPA exposure and health hazards.

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