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Toxicity of Bisphenol A in Rats: A Study of its Accumulation and Effects on Brain, Kidneys, Liver and Blood Serum

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Annotation: This study investigates the harmful effects of Bisphenol A (BPA) bioaccumulation in the brain, liver, kidneys, and blood serum of albino rats. BPA, a chemical widely used in plastics and resins, is known to disrupt endocrine function and pose health risks. Twenty-four adult rats, including 12 males and 12 females, were divided into three groups, each receiving different doses of BPA over six weeks. BPA was administered orally with corn oil. Tissue samples from the brain, liver, kidneys, and serum were analyzed using High-Performance Liquid Chromatography (HPLC). Results showed a significant accumulation of BPA in the brain, liver, and kidneys, with higher concentrations observed compared to serum. The accumulation followed a dose-dependent pattern, with the highest BPA levels in the group receiving the maximum dose. Furthermore, differences in BPA accumulation were noted between males and females. The aim of this study is to understand the extent of BPA bioaccumulation in various organs and explore sex-specific variations in the effects of BPA exposure.

Keywords: Bisphenol A (BPA), Endocrine Disruptor, Toxicology, Accumulation, Organs, Albino Rats, Dose-Dependent Effects, HPLC.

1. Introduction

Bisphenol A, commonly abbreviated as BPA, is a man-made chemical compound that is extensively employed in the manufacturing processes of polycarbonate plastics and epoxy resins, both of which play crucial roles in the creation of a diverse range of consumer goods that are ubiquitous in modern society. Among these products are various food and beverage storage containers, essential medical apparatus, dental sealing materials, a plethora of children's toys, and even construction materials such as polyvinyl chloride (PVC), where the presence of BPA serves specifically as a polymerization inhibitor, effectively regulating the chemical reactions involved in the production process [1-3]. The chemical compound is particularly esteemed for its capacity to confer advantageous characteristics such as enhanced optical clarity and significant heat resistance to the polycarbonate plastics that are often utilized in daily life (Troiano & Goodman, 2008). However, it is important to note that not all of the BPA utilized in these manufacturing processes is completely consumed, resulting in the potential for this chemical to leach into consumable food and beverages, particularly when they are subjected to elevated temperatures and acidic environments [4]. This phenomenon of leaching raises critical health concerns, as BPA has been identified as an endocrine disruptor, possessing the ability to mimic estrogenic activity and thereby posing significant risks for reproductive and developmental health, especially among the most vulnerable segments of the population, including infants, young children, and pregnant women [5,6]. The widespread use of BPA in a variety of consumer products has resulted in increased regulatory scrutiny and advocacy for the identification and implementation of safer alternative substances, given that the measurable presence of BPA in both environmental samples and human biological specimens indicates widespread exposure among the general population [5,7]. Despite the practical benefits that Bisphenol A (BPA) provides in a variety of applications, increased awareness of the potential health risks associated with exposure has prompted an increase in research efforts and regulatory actions aimed at reducing interaction with this chemical, particularly in products specifically designed for infants and young children [5,8].

Bisphenol A, or BPA, exposure is multi-faceted and can occur via a variety of routes, such as the food we eat, our skin, and even our lungs. All of these ways add up to a significantly higher risk of exposure for people. The majority of people are exposed to bisphenol A (BPA) through the food they eat and drink. This is because many common food packaging and storage solutions use materials like polycarbonate plastics and epoxy resins, which contain BPA [10-12]. The widespread use of polycarbonate baby bottles exposes infants and toddlers to levels of BPA that are much higher than those experienced by adults [13], which is especially concerning because of their heightened vulnerability. The total amount of BPA exposure includes both dietary and non-dietary routes. Non- dietary routes include skin contact from things like thermal paper and cosmetics, and inhalation of dust and air that may contain BPA particles [10,13,14]. Surface water, sediments, and sewage sludge are only a few examples of the environmental media that contain BPA; as a result, there is a greater chance of exposure via contaminated soil and water sources [15,16]. The presence of BPA within household dust and indoor air serves to further highlight the potential for inhalation exposure, particularly in settings characterized by prolonged indoor occupancy [13][16]. In spite of the pervasive nature of BPA, current risk assessments indicate that the levels of dietary exposure are generally deemed to fall below the temporary Tolerable Daily Intake (t-TDI) set forth by regulatory agencies; however, there remains a considerable degree of uncertainty surrounding the estimates of non-dietary exposure [10,11]. This cumulative exposure, resulting from the various pathways mentioned, especially in populations deemed vulnerable such as infants, children, and pregnant women, underscores the urgent need for continuous monitoring and potential updates to existing risk assessment models to more accurately reflect the complex and diverse exposure pathways and their associated health implications [17-19].

Bisphenol A (BPA), a common environmental contaminant, impairs hormone function and endangers human health. The growing data linking BPA exposure to metabolic diseases, reproductive issues, and developmental abnormalities raises public health and toxicological concerns. It is also recognized that bisphenol A (BPA) negatively impacts glucose metabolism, increasing the risk of diabetes, insulin resistance, and obesity. BPA alters endocrine-metabolic pathways, which govern physiological activities, including liver and pancreatic functions [20]. BPA's estrogenic properties cause metabolic abnormalities and increase the risk of various cancers, including breast and prostate cancers, by promoting cellular proliferation and disrupting the delicate hormonal balance needed for optimal health [21,22]. BPA exposure has been linked to severe neurological impairments like memory loss, neurodevelopmental disorders like ADHD and autism spectrum disorders, and neurodegenerative diseases like Alzheimer's and Parkinson's disease, complicating the public health context [23,24]. BPA may cause oxidative stress and lipid peroxidation, which impairs amino acid and lipid metabolism in animal models [25]. Concerns about the health of future generations are heightened by the fact that BPA poses serious risks to reproductive health, including infertility and several developmental problems in developing babies [26,27]. The health effects of bisphenol A (BPA) are already complicated, and there's more bad news: research shows that it might mess with your immune system, making you more prone to allergies and autoimmune diseases [4,12]. The European Food Safety Authority and other regulatory bodies have not yet designated bisphenol A (BPA) as a major health concern, even though there is a growing amount of evidence demonstrating the dangers this chemical poses to humans [28]. The rapid development of BPA-free alternatives and stricter regulatory frameworks are critical for reducing the extensive damage that BPA does to public health and safety, especially considering how common it is in consumer goods and the environment [29].

The investigation into the bioaccumulation of Bisphenol A (BPA) across different organ systems is highly important, given its widespread use and the possible negative health effects associated with its role as an endocrine disruptor. BPA is frequently present in synthetic polymers and has the potential to migrate into food and beverages, leading to human exposure. Empirical investigations have demonstrated that BPA can accumulate in various organs, including the renal system, hepatic tissue, and cardiac muscle, resulting in significant histopathological changes. Prolonged exposure to BPA has been associated with renal toxicity, marked by a reduction in glomerular structures and structural damage within the renal cortex, as demonstrated in animal models [30,31]. Concomitantly, exposure to BPA has been linked to hepatotoxicity, wherein it induces morphological changes and modifies gene expression in hepatic tissues, potentially culminating in severe metabolic disorders and carcinogenic outcomes [32]. Within the cardiovascular system, BPA exposure has been demonstrated to compromise cardiac architecture and functionality, thus potentially heightening the risk of cardiovascular diseases [33]. Moreover, the detection of BPA in dialysis apparatus has engendered apprehensions regarding its accumulation in individuals undergoing hemodialysis, as this exposure can markedly elevate serum BPA concentrations, potentially influencing endocrine and metabolic systems [34]. The propensity of BPA to accumulate within adipose tissues, coupled with its protracted elimination from the biological system, further complicates its risk assessment, indicating that non- dietary exposures and bioaccumulation may contribute to its persistence within the human organism [35]. Additionally, investigations have established a correlation between elevated BPA exposure and increased all-cause mortality, underscoring the necessity for further research to elucidate its long-term health consequences [36] The primary objectives of this comprehensive study are twofold: firstly, to meticulously assess and quantify the accumulation of bisphenol A (BPA) within particular biological tissues, specifically focusing on the brain, kidney, liver, and blood serum of murine subjects, and secondly, to rigorously evaluate the potential toxicological effects that BPA exerts on these vital organs in the aforementioned mice, utilizing high-performance liquid chromatography (HPLC) as the analytical technique for precise measurement and analysis.

2. Methodology

2.1. Study Design

2.1.1. Experimental Animals

Rats from the College of Science, University of Al-Qadisiyah, operation with laboratories in The Ministry of Science and Technology Ethical approval No. ISO 17025, animal facility were used in this study. Rats aged 75–90 days weighed 145–200 grams for females and 85–150 grams for males. Plastic cages might expose the rats to BPA; thus, they were confined in metal cages. The animals had glass water containers in their cages. The animals spent 30 days at the animal facility before the experiment. The goal was to adjust to the animal house's 12–12-hour light-dark cycle at 20–25 degrees Celsius. The animals were kept at the University of Al-Qadisiyah College of Science facilities.

2.1.2. Components of The Diet

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

Table 1. Components of a kilogram of the diet.

No.	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.

- Control groups: Three female and three male rats were administered maize oil solely at 0.5 ml/kg of body weight daily.
- Groups (A): were divided into two groups: 3 female and 3 male rats received BPA orally daily in maize oil at 200 mg/kg.
- Groups (B): BPA was administered orally to 3 female and 3 male rats daily in maize oil at 100 mg/kg of body weight.
- Groups (C): Bisphenol-A was orally given to 3 female and 3 male rats daily in maize oil at 50 mg/kg of body weight.

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 dosing the control group. On a weekly basis, BPA was dissolved in corn oil and given to each group according to their body weight, with the control group getting corn oil [37,38].

2.1.5. Animal Sacrifice and Organ Collection

Before sacrifice, rats were weighed and anesthetized in a cotton-lid container tray filled with chloroform and closed with a rubber band. To access the study organs, the abdominal cavity was opened by cutting the midline (Fig . 1). The kidneys, liver, and brain were removed from the animal cavity and preserved at a high temperature until HPLC testing could be conducted.

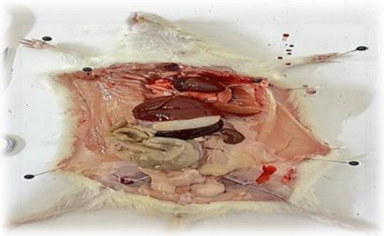

Figure 1. Open abdominal cavity to organ collection.

2.2. HPLC Analysis

2.1.1. Sample Preparation

The sample used for the experiment was homogenized tissue (170 mg). Mix serum samples (500 µl) or homogenized tissue with 3 ml hexane and shake for 3 min. Add 5 ml acetonitrile and mix for 3 min. This mixture was centrifuged for 15 minutes at 2500 rpm. The lower layer acetonitrile was transferred and filtered using a 0.2 um syringe filter. The filtrate was put into HPLC after being vacuum- concentrated to 500 µL [39,40].

2.2.2. HPLC System

The High-Performance Liquid Chromatography (HPLC) system used in this study was an HPLC, the components of this system are according to the table.

Table 2. The components of HPLC system.

No.	Component	Model or version	Company and origion
1	Binary high-pressure gradient pump	P6.1L	Knuaer, Germany
2	Diode array detector	DAD 2.1L	Knuaer, Germany
3	Sample loop (20 µl) and injector	D1357	Knuaer, Germany
4	Analyses and system control software	Claritychrom, V7.4.2.107	Dataapex, Czech Republic

The High-Performance Liquid Chromatography (HPLC) system used in this study was an HPLC - UV system. Column C18 Waters (300 mm x 4.6 mm i.d). Detection at 230 nm. Injection volume 10 µL. Mobile phase acetonitrile / water (60: 40, v / v) at flow 1 ml / min.

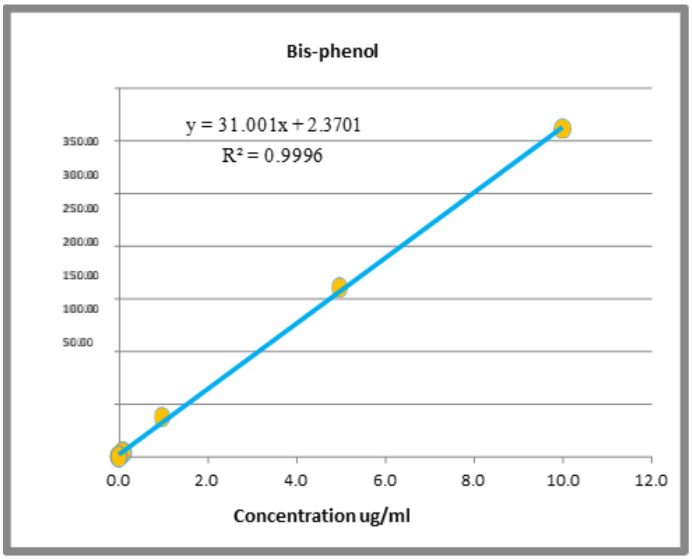

Figure 2. Linierity of BPA in acetonitrile by using HPLC - UV system. Column C18 Waters (300 mm x 4.6 mm i.d). Detection at 230 nm. Injection volume 10 µL. Mobile phase acetonitrile / water (60: 40, v / v) at flow 1 ml / min.

3. Results

3.1. Accumulation of BPA in Some Body Organ Tissues

The findings of the current investigation elucidated that, when juxtaposed with other cohorts, there existed a pronounced increase in significant variances regarding the accumulation of BPA within designated organ tissues across all treatment groups (groups A and B, groups B and C, and groups A and C), with a p-value of less than 0.05. The present research identified that across all treatment groups, there is a statistically significant elevation in discrepancies within the cerebral tissue of both male and female rats ($P < 0.05$). Our investigation revealed that in comparison to male cohorts, female cohorts demonstrate a statistically significant increase, and that obese subjects consistently exhibit lower body mass indices when contrasted with matched control individuals. The findings of the current study illustrated that there were considerable variances in the accumulation of BPA within hepatic tissue across all treatment groups, with heightened discrepancies noted between group A and group B, group B and group C, as well as group A and group C. These disparities were statistically significant ($P < 0.05$). Statistically significant alterations at $P < 0.05$ were observed in the hepatic tissue of both male and female rats across all treatment groups, as indicated by the results of the current study. In contrast to male cohorts, our investigation identified a statistically significant increase among females. The findings of the present study revealed that, relative to other cohorts, there was a marked increase in substantial variances concerning the accumulation of BPA in renal tissue across all treatment groups. These variances were most accentuated between groups A and B, B and C, and A and C. Statistically significant alterations at $P < 0.05$ were identified in the renal tissue of both male and female rats across all treatment groups, according to the results of the current study. In contrast to male cohorts, our investigation established a statistically significant increase among females. The results of the present study indicated that, in relation to the other groups, the significant variations in BPA accumulation within renal tissues were markedly elevated across all treatment groups. In comparison to the other cohorts, the current study's findings illustrated an increase in significant differences ($P < 0.05$) regarding the accumulation of BPA in blood serum across all treatment groups. These variances were most pronounced between groups A and B, B and C, and A and C. Statistically significant differences at $P < 0.05$ were identified in the serum of the blood of both male and female rats across all treatment groups, as evidenced by the findings of the current study. The results of our investigation denote a statistically significant increase in female cohorts when contrasted with male cohorts. The current study established that there were statistically significant variations in the concentrations of BPA accumulated in the blood serum across all treatment groups as show Table (3) and Fig (3,4,5) The table presents the effect of bisphenol A (BPA) accumulation in various organs (brain, liver, kidney, and serum) across male and female rats in three groups (A, B, and C). The concentrations of BPA are expressed in micrograms per milliliter (µg/ml) and micrograms per gram of tissue (µg/g tissue), with values accompanied by standard deviations. The statistical analysis indicates significant differences between groups based on the LSD (Least Significant Difference) values, which are provided for each organ.

The statistical analysis indicated a substantial positive link with the buildup of bisphenol A (BPA) in several organ tissues. The brain tissue had a significant positive association ($r = +0.97$) in both male and female participants. Renal tissue had a significant positive connection ($r = +0.97$) in men and ($r =$

$+0.90$) in females. Hepatic tissue had a significant positive association ($r = -0.98$) in men and ($r =$

$+0.88$) in females. In female patients, the serum exhibited a significant positive association ($r = -0.98$), whereas in male subjects, the connection was even more apparent ($r = +0.99$). This link is seen in Table (4) for female individuals.

Table 3. Effect of BPA through its accumulation in the tissues of the organs.

Organs	Groups	Males BPA	Males ug/ml	Males ug/g tissue	Females BPA	Females ug/ml	Females ug/g tissue
Brain	A	27.20±2.53 A	0.7438±0.53 A	2.19±0.56 A	22.42±2.13 A	0.6133±0.50 A	1.8±0.55 A
	B	19.67±1.96 B	0.5382±0.26 A	1.58±0.47 B	7.71±1.85 B	0.2119±0.21 A	0.62±0.45 B
	C	8.55±2.12 C	0.2347±0.12 C	0.69±0.39 C	7.68±2.05 B	0.211±0.10 A	0.62±0.31 B
LSD			0.532			0.78	
Liver	A	26.34±3.02 A	0.7203±0.55 A	2.12±0.52 A	16.18±3.11 A	0.443±0.50 A	1.3±0.50 A
	B	5.83±1.02 B	0.1603±0.02 B	0.47±0.01 B	12.52±1.14 B	0.343±0.01 A	1.01±0.02 A
	C	5.52±0.89 B	0.152±0.01 B	0.45±0.02 B	5.57±0.81 C	0.1535±0.03 B	0.45±0.05 B
LSD			0.35			0.57	
Kidney	A	20.9±2.11 A	0.5718±0.32 A	1.68±0.41 A	19.39±2.10 A	0.5306±0.30 A	1.56±0.46 A
	B	13.86±1.63 B	0.3796±0.11 A	1.12±0.36 B	13.86±1.69 B	0.3796±0.12 A	1.12±0.33 A
	C	6.35±0.95 C	0.1747±0.12 B	0.51±0.05 C	3.64±0.92 C	0.1008±0.15 B	0.30±0.02 B
LSD			0.46			0.55	
Serum	A	19.41±1.85 A	0.5312±0.25 A	0	5.95±1.80 A	0.1638±0.23 A	0
	B	13.76±1.66 B	0.3769±0.15 A	0	4.68±1.68 B	0.129±0.19 B	0
	C	2.96±0.58 C	0.0821±0.01 B	0	2.96±0.51 C	0.0821±0.02 C	0
LSD			0.24			0.36	

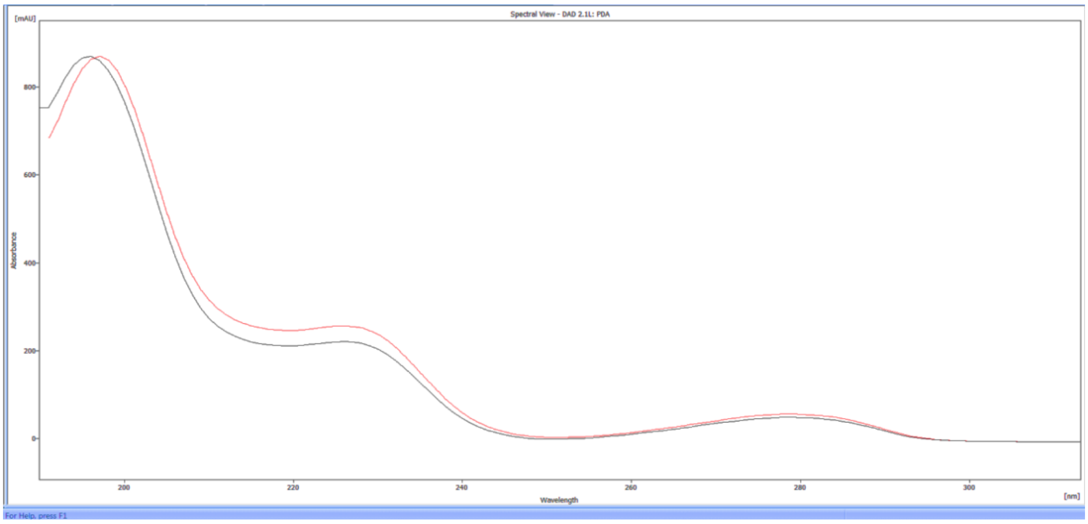

Figure 3. Shows the absorbance of BPA between the standard material of BPA and the sample during the specified time.

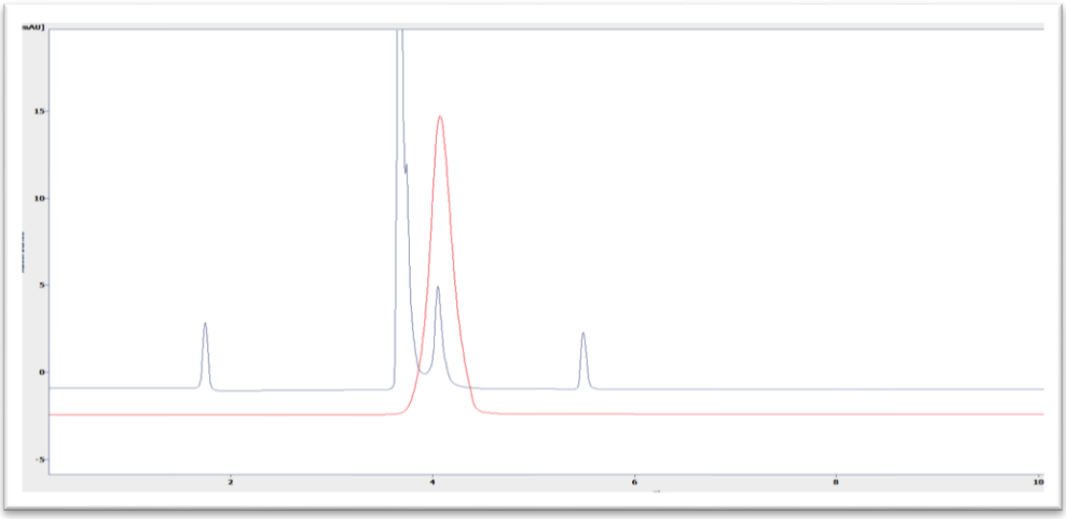

Figure 4. Shows the absorbance of BPA between the standard material of BPA and the sample during the specified time.

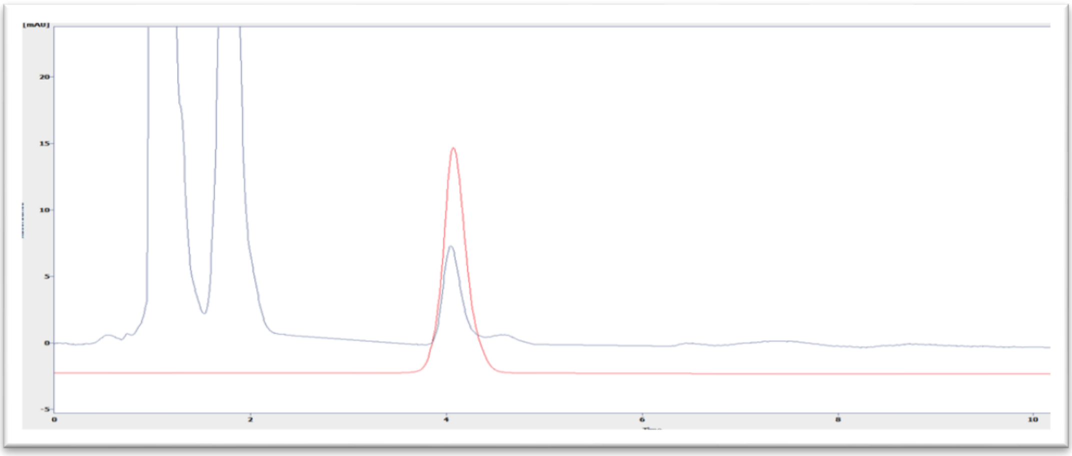

Figure 5. Shows the matching between the standard substance of BPA and the sample by ultraviolet rays.

Table 4. Correlation Coefficient of Dosing BPA through its accumulation in the tissues of the organs.

	Males			
	Brain	**Liver**	**Kidney**	**Serum**
Correlation Coefficient	0.9774	0.9895	0.9796	0.9889
		Females		
	Brain	**Liver**	**Kidney**	**Serum**
	0.9733	0.8899	0.9085	0.9985

4. Discussion

Bisphenol A, commonly referred to as BPA, is a man-made chemical that has found widespread application in the manufacturing processes of polycarbonate plastics and epoxy resins, and the extensive incorporation of this compound into a multitude of consumer products has catalyzed considerable apprehension regarding its implications for both human health and the environment. cancers, as evidenced by the research of [12,41,42,43]. From an environmental perspective, BPA represents a consequential pollutant, frequently detected across diverse environmental matrices such as aquatic systems, terrestrial soils, and atmospheric air, thereby posing significant risks to wildlife, particularly in marine ecosystems where it is capable of bioaccumulating in aquatic organisms, resulting in detrimental hormonal disruptions and reproductive challenges. The widespread prevalence of this chemical, coupled with its detrimental effects observed even at low levels of exposure—levels that regulatory agencies often deem to be safe—renders BPA a persistent and formidable threat to the well-being of both human populations and the broader environment, as highlighted by the findings of [44]. Albino rats are frequently employed in scientific investigations to assess the deleterious effects of Bisphenol A (BPA) owing to their physiological and genetic resemblances to humans, which render them an appropriate model for toxicological inquiry. BPA is a prevalent industrial chemical recognized for its endocrine-disrupting characteristics, which have the capacity to mimic estrogen and potentially result in adverse health consequences. Empirical studies in toxicology and pharmacology have clearly shown that exposure to bisphenol A (BPA) in albino rat models leads to numerous significant biochemical, histological, and reproductive changes, establishing these animals as an ideal model for understanding the substantial effects of BPA on mammalian biological systems. [45,46,47]. The strategic use of albino rats as research subjects enables scientists to closely observe and evaluate the various effects of BPA exposure in a controlled experimental environment, thereby providing critical insights into the potential risks and harmful effects that BPA may present to human health and well-being. The ability to systematically control key variables, such as the dosage of BPA and the duration of exposure in studies with albino rats, greatly improves the understanding of the dose- dependent toxicity of BPA and its biological effects. [48,49]. The extensive investigations highlight the crucial importance of utilizing albino rats as a model organism for the critical evaluation of health risks linked to BPA exposure, thereby enhancing the understanding of its complex effects on biological systems and subsequently informing regulatory frameworks regarding its appropriate use in various consumer products. [50,51].

Studies on Bisphenol A (BPA)'s harmful effects usually use male and female albino rats. This strategy seeks to capture BPA's complete spectrum of effects, which vary across sexes owing to its estrogenic characteristics, which influence biological systems differently. [52]. BPA's effects on gene expression and apoptotic processes in the brain's neural structures show significant sex-specific variations, with male rats showing a significant decline in social preference behaviors and unique changes in prefrontal cortex gene expression profiles that are absent in females, highlighting the importance of considering sex as a biological variable in such research. [53]. Both men and women have shown histopathological changes in organs including the liver and lungs and genotoxic consequences. This shows that BPA's systemic toxicity affects more physiological systems than reproductive organs, highlighting its widespread health effects [54,55]. Researchers must include male and female subjects in their experimental designs to accurately assess BPA exposure's biological responses and develop health regulations that address each gender's vulnerabilities. This comprehensive methodology clarifies BPA's health risks and accurately reflects male and female physiological responses in research findings, enhancing our understanding of the compound's effects on health and disease dynamics. [56]. Albino rats, which are tiny to medium-sized and vary in age, are used in a study to determine the health risks of bisphenol A (BPA), a chemical molecule. These lengthy investigations use juvenile or moderately aged rats to properly depict the effects of BPA exposure on growth and reproductive health. Academic literature shows a considerable loss in sperm quantity and motility and changes in reproductive organ histology [57]. According to prior research, these rat models allow for the careful administration and systematic observation of dose-dependent effects, which are necessary to determine the threshold levels of BPA-induced toxicity and its systemic effects [55,58]. It uses a well-structured strategy to study this compound's toxicological effects on biological systems, notably rodent models. Methodology involves using a triadic dosage schedule with low, medium, and high bisphenol A (BPA) concentrations. This sophisticated approach allows researchers to identify the threshold of exposure at which BPA triggers a cascade of toxicological effects and the unique characteristics and severity of these effects across various dosage levels, improving our understanding of BPA's impact. Numerous studies have shown that high BPA levels might cause serious side effects. Such exposures may cause severe testicular atrophy, spermatogenesis suppression, and endocrine disturbance in rats. At even greater exposure levels, reproductive harm becomes apparent [59]. However, lower doses are often used to study subtle biochemical and physiological changes that may have long-term health effects, such as body mass changes and hematological indices [60]. Please consider this. Intermediate doses are crucial to understanding complicated dose-response interactions and detecting non-linear effects between low and high exposure [61]. Given the widespread exposure to BPA from consumer products, this stratified dosing framework is essential for the construction of a comprehensive toxicological profile of BPA, which is crucial for regulatory policy development and understanding of human health risks [62,63]. Multiple dosing regimens enable the determination of the no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL), which are essential for rigorous risk assessments and safety benchmarks [62].The kidney, brain, and liver tissues of both male and female specimens have significantly higher concentrations of bisphenol A (BPA), a synthetic chemical compound, due to various factors related to its unique chemical properties and complex biological interactions with various physiological systems. BPA, a man-made substance that can disrupt endocrine function, is found in polycarbonate plastics and epoxy resins, which are used in food and beverage containers, dental materials, and various consumer products [64,65]. The extensive utilization of these materials in modern society leads to a continuous and pervasive human exposure to BPA through multiple routes, including ingestion, inhalation, and direct dermal contact, culminating in the ubiquitous presence of this chemical compound within the human biological system, as noted by [66,67]. Upon entering the human body, BPA undergoes metabolic processing, primarily in the liver where it is subjected to glucuronidation and sulfation; however, despite this rapid metabolic degradation, it does not effectively prevent the accumulation of BPA in various tissues, a finding corroborated by [65,68]. The liver, recognized as the central organ for detoxification processes, is continuously exposed to BPA, which can precipitate hepatotoxic effects and result in liver injury through mechanisms involving oxidative stress and inflammatory pathways, [68]. In a similar vein, the kidneys play a crucial role in the excretion of BPA, and empirical studies have demonstrated that exposure to BPA can induce renal pathologies, such as podocytopathy, which may facilitate the compound's accumulation in renal tissues, as noted in the study titled [69]. BPA may cross the blood- brain barrier and interact with estrogen receptors, which may affect neurodevelopmental and neuroendocrine processes [70,71]. The estrogenic properties of bisphenol A (BPA) allow it to efficiently bind to estrogen receptors in various tissues in the human body, including the intricate networks of the brain, the vital functions of the liver, and the essential processes of the kidneys, thereby profoundly affecting their normal physiology. Blood serum is a key indicator of systemic BPA exposure; hence, its presence is concerning. Numerous studies show that blood BPA levels typically exceed toxicokinetic model predictions, indicating a worrying increase in bioavailability and health risks [66]. BPA can profoundly disturb tissue homeostasis and modify essential physiological functions vital to health by inducing epigenetic modifications and interacting with many transcription factors, as shown in reference [70].

Conclusion

The inquiry's results indicate that BPA accumulates in vital organs, including the kidneys, liver, and brain, in a dose-dependent manner. The results show the substantial risks associated with BPA exposure. They illustrate the need for instituting more rigorous regulatory measures and doing more study to get a comprehensive understanding of the long-term health impacts of BPA on both human and animal populations. The observations indicate potential gender-based variations in BPA accumulation, necessitating additional investigation into the molecular mechanisms underlying these differences.

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