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Unveiling the Potential Risks: Long-Term Health Effects of Chronic Exposure to Bisphenol A

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Abstract:

The effects of bisphenol A (BPA) on estrogen levels, fertility, obesity, and liver injury were investigated. Through a series of control results (CR1 to CR16), this study examined the multiple effects of BPA on biological processes. The findings show that when exposed to BPA, estrogen levels rise, fertility decreases, obesity increases through changes in liver weight, and damage can be observed through confocal imaging. These results varied in different settings, suggesting partial or full support for the original hypothesis that BPA has various effects on estrogen, fertility, obesity, and liver damage. It is worth noting that in some cases, such as CR5, estrogen levels are not affected by BPA, but fertility, obesity, and injury are. In contrast, CR16 significantly refuted this hypothesis, suggesting that BPA may not have a substantial effect on estrogen, fertility, obesity, or impairment in specific situations. Overall, the study highlights the complex relationship between BPA exposure and biological systems, underscoring the need for further research to fully understand the effects of this compound on estrogen regulation, fertility, obesity development, and liver health. These findings provide valuable insights into the complex interactions between BPA and human physiology.

Keywords: Pharmaceutical personal care products, Bisphenol A, Endocrine disrupter, Human health

1. Introduction

1.1 Background

In recent years, a new pollutant which has become people's concern [1]. Since COVID-19, the increasing amount of usage of drugs and personal care products can cause a larger existence of Pharmaceutical and Personal Care Products(PPCPs), a new pollutant have pumped into people's views, as well as the endocrine disrupters that are also threatening people's life, especially babies [2]. These pollutants are created in people's daily lives because they are unable to dispose of garbage properly.

1.2 Introduction to Pharmaceutical and Personal Care Products (PPCPs)

PPCPs are new pollutants that mainly include human and veterinary medicine, such as several antibiotics and other chemical consumer products such as cosmetics. Another type of PPCPs also include musk substances but also include additives and inert ingredients used in the production and processing of pharmaceuticals and care products [2]. PPCPs may disrupt aquatic ecosystems, affect aquatic organisms, and enter the food chain [3]. In addition, these compounds persist in the environment, accumulate over time, and harm wildlife and humans through exposure [4]. The continued use and disposal of PPCPs can lead to the development of resistance to antibiotics, which can exacerbate public health problems. It has become particularly important to develop a comprehensive strategy to address the negative impacts of PPCPs. Possible solutions include improved wastewater treatment procedures, appropriate disposal methods, and increased awareness among consumers and health-care providers of the environmental and health risks posed by these substances.

1.3 Introduction to Endocrine Disrupter (EDCs)

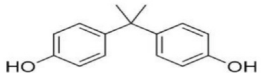

Figure 1: Basic molecular structure of EDCs

Endocrine disruptors are substances that can interfere with the normal function of the endocrine system [5]. Affects the endocrine system regulating hormones and signal transduction in the body. It can also be called an environmental hormone. EDCs can mimic or block hormones, resulting in disruption of developmental, reproductive, neurological, and immune processes. [6]

1.4 Introduction to bisphenol A (BPA)

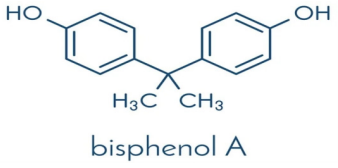

Figure 2: Molecular structure of BPA

Bisphenol A (BPA) is a synthetic compound that could be used in the production of polycarbonate plastics, epoxy resins, and thermal paper. BPA can leach into food and beverages, leading to ingestion and absorption by the body. Studies have shown that BPA exposure is almost ubiquitous and can be detected in human urine, blood and breast milk samples from the general population [7].

1.5 Limitations

Although BPA has been used in consumer products for at least 10 years and concerns about its possible long-term health effects have promoted extensive research, there are still the existence of some knowledge gaps that need to be filled [6]. For example, there is a paucity of research on the relationship between long-term exposure to BPA and specific diseases, such as cardiovascular or autoimmune diseases or even several cancers. The purpose of this introduction is to figure out the long-term effects and potential risks of exposure to BPA on human health by doing experiments on rats.

1.6 Research Purpose

This paper predicts that increasing concentrations and treatment durations BPA in rats will increase estrogen, decrease fertility, increase obesity, and lead to liver damage.

2. Material and Methods

2.1 Measurement of Estrogen by ELISA

Estrogen levels are measured using an enzyme-linked immunosorbent assay (ELISA) [8]. The equipment used is a microplate reader, ELISA plate and pipette. A blood or serum sample is first collected and appropriately diluted. The sample is added to the wells of the ELISA plate coated with an estrogen-specific antibody and a known concentration of an estrogen standard [7]. After incubation and washing to remove unbound material, an enzyme-conjugated estrogen-specific secondary antibody is added. A substrate solution is then added to initiate a color reaction, which is stopped with a stop solution. The absorbance of each well is measured using a microplate reader to determine estrogen concentration. Positive and negative controls consisting of samples with known higher and lower estrogen concentrations were used to test the accuracy of the assay, respectively. Estradiol will be used as a positive control, and Femara will be used as a negative control.

2.2 Fertility Assessment by Counting Offspring

To assess fertility, we used counts of offspring of rats [6]. Male and female rats were kept together in breeding cages. Natural mating was allowed and successful matings were recorded. Once successful mating was confirmed, the male rats were removed from the cage and the females were observed for signs of pregnancy. At the end of the gestation period, the birth of offspring was observed and the number of offspring produced by the female rats was counted. An experiment group in the BPA environment and an experiment group that are not exposed to BPA

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(consisting of known high and low fertility groups, respectively) were established as a baseline for comparison. Clomid will be used as a positive control, and DMSO will be used as a negative control.

2.3 Obesity Measurement by Weight

Obesity measurements consist of assessing the weight of the rats using electronic scales [8]. The weight of each rat is recorded. The rats are divided into groups by weight; one group will be exposed to BPA, the other group will not be exposed, and all other living conditions will be the same. The two groups will then be weighed to determine if BPA has any effect on obesity. Amitriptyline will be used as a positive control, and contrary will be used as a negative control.

2.4 Liver Damage Assessment by Confocal Microscopy

To assess liver injury, tissue samples are collected and fixed [9]. Thin sections of the sample are stained with a fluorescent dye specific for liver injury markers. After staining, the sections are mounted on slides and observed under a confocal microscope. Representative images are taken and analyzed using image processing software to measure markers such as fluorescence intensity and cellular appearance [8]. This analysis reveals the extent of liver damage. Damaged liver samples were used as positive controls and healthy liver samples were used as negative controls. Amiodarone will be used as a positive control, and Prednisolone will be used as a negative control.

2.5 Positive controls and Negative controls

Positive and negative controls are set as a guideline to compare with the test groups and intend to lower the possibility of the presence of bias. The level of estrogen will be measured by ELISA; obesity will be measured by electronic scales, fertility will be measured by the number of rats' offspring and liver damage will be measured by Confocal Microscopy [9].

2.6 Statistical Analysis

The experiments will be repeated at least three times, and the data will be reported in table format by showing the sign of ± [6]. For the treatment group, rats will be exposed to different concentrations of BPA circumstances, for example, 15, 25, 50, and 100 μM in 1.5 weeks to determine the results. This experiment will be done using student t-test to analyze the data. These tests usually follow the normal distribution where some values are known. The significance level will be available to indicate the statistical significance when $p < 0.05$.

3. Result

Table 1: Combination of possible results for test groups

Combination Result # (CR#)	BPA increases estrogen by ELISA	BPA decreases fertility by baby counting?	BPA increases obesity by weight?	BPA increases liver damage by confocal microscopy?	Support of hypothesis
1	+	+	+	+	Fully Support
2	+	+	+	-	Partial
3	+	+	-	+	Partial
4	+	-	+	+	Partial
5	-	+	+	+	Partial
6	+	+	-	-	Partial
7	+	-	-	+	Partial
8	-	-	+	+	Partial
9	+	-	+	-	Partial
10	-	+	-	+	Partial
11	-	+	+	-	Partial
12	+	-	-	-	Partial
13	-	+	-	-	Partial
14	-	-	+	-	Partial
15	-	-	-	+	Partial
16	-	-	-	-	Fully Contradicts

"+" indicates that the result of the experiment have similar influence or stronger influence on the test groups compared with the positive controls which support the hypothesis. "-" indicates that the result of the experiment have similar results compared with those negative controls which contradicts the hypothesis.

CR1

As BPA concentrations increase, estrogen levels increase, fertility decreases, and long-term exposure to BPA increases obesity and liver damage. This scenario is consistent with the hypothesis and suggests that BPA has multiple effects on estrogen, fertility, obesity, and liver health as concentration and duration increase. [8]

CR2

Increasing concentrations of BPA exacerbate effects on estrogen and fertility and may exacerbate obesity. Long-term exposure may increase effects on fertility and obesity but does not cause significant adverse effects. This subtle result partially supports this hypothesis and emphasizes the complex relationship between BPA concentrations, duration, and their effects on estrogen, fertility, and obesity.

CR3

High levels of BPA enhanced effects on estrogen and fertility but not obesity, whereas chronic exposure to BPA enhanced liver damage but did not significantly affect obesity. This result partially supports the hypothesis that BPA primarily affects estrogen, fertility, and liver health, with limited effects on obesity.

CR4

Higher concentrations of BPA enhanced effects on estrogen and obesity, and long-term exposure exacerbated obesity and liver injury, but had no significant effect on fertility. This result partially supports the hypothesis by highlighting the effects of BPA on estrogen, obesity, and liver health, with weaker effects on fertility.

CR5

Bisphenol A deficiency does not increase estrogen levels but decreases fertility, increases obesity, and causes disability, a result that partially supports the hypothesis and highlights the effects of BPA on fertility, obesity, and liver health, but has no significant effect on estrogen levels.

CR6

This hypothesis is supported in part by the fact that high levels of BPA lead to high levels of estrogen and lower fertility, but have little effect on obesity or liver damage. This suggests that BPA primarily affects estrogen and fertility, with limited effects on obesity or liver damage.

CR7

Exposure to BPA can cause elevated estrogen and liver damage, but fertility and obesity are barely affected. This finding partially supports the hypothesis that BPA primarily affects estrogen and liver damage, and does not have much effect on fertility and obesity.

CR8

BPA did not increase estrogen levels or decrease fertility, but it affected obesity and caused damage to the liver, partially supporting the hypothesis

CR9

Exposure to BPA increases estrogen levels and affects obesity but does not cause major damage to fertility or the liver. The study goes some way to supporting the hypothesis that BPA primarily affects the health of estrogen levels and metabolism, with no significant effect on fertility or liver damage.

CR10

Bisphenol A did not increase estrogen levels, cause liver damage, or have a significant effect on obesity, but it affects obesity. This partially supports the hypothesis.

CR11

This finding partially supports the hypothesis because it emphasizes the effects of BPA on fertility and obesity without causing significant liver damage or affecting estrogen.

CR12

The hypothesis is also partially supported by the fact that exposure to BPA increases estrogen levels but does not significantly affect fertility, obesity, or perceived liver damage. This suggests that although BPA affects estrogen levels, in this case its effects on fertility, obesity, and cellular health may be limited.

CR13

Exposure to Bisphenol A does not result in elevated estrogen levels, fertility is not affected, but obesity is affected, and liver damage occurs, which partially supports the hypothesis because it emphasizes that Bisphenol A has an effect on obesity and cellular health but no significant effect on estrogen or fertility.

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CR14

Exposure to Bisphenol A increases estrogen levels but fertility is not affected and obesity is affected and liver damage occurs, which partially supports the hypothesis because it emphasizes that Bisphenol A has effects on estrogen, obesity, and cellular health but no significant effects on fertility.

CR15

Exposure to Bisphenol A increases estrogen levels and decreases fertility, and obesity is not affected and liver damage occurs, which partially supports the hypothesis because it emphasizes that Bisphenol A has effects on estrogen, fertility, and cellular health, but no significant effects on obesity.

CR16

Exposure to BPA did not increase estrogen levels, fertility remained unchanged, obesity was not affected, and no increase in liver damage was observed, which completely refutes the hypothesis and suggests that BPA has an effect on estrogen, obesity, and cellular health, but no significant effect on fertility.

4. Discussion

The aim of this study was to investigate the effects of long-term exposure to bisphenol A (BPA) on human health. Previous studies have shown that BPA is an endocrine-disrupting substance that can adversely affect hormone regulation and various physiological processes. In the current study, it was hypothesized that increased concentrations and duration of BPA led to increased estrogen levels, decreased fertility, increased obesity, and liver damage.

CR1

The hypothesis that BPA would have significant effects on estrogen levels, fertility, obesity, and liver damage is well supported. This combined effect may be due to the strong influence of BPA on a variety of physiological processes, which is generally consistent with the expected results.

CR2

The CR2 hypothesis is partially supported by the fact that BPA affects estrogen, fertility, and obesity, but does not cause liver damage. One reason for the partial support of the CR2 hypothesis may be the different sensitivities of the assays for the different components. Effects on estrogen levels and fertility may be more pronounced and easier to detect, but imaging techniques used to study liver injury may not be sensitive enough to capture subtle cellular changes caused by exposure to BPA. Another reason may also be that BPA does not reach to liver properly. This difference highlights the importance of using comprehensive and sensitive methods to accurately assess the effects of BPA. In the future, the experiment should be using a more well-developed microscope in order to observe the liver sample more clearly. Scholars also need to consider whether they should directly attach BPA to the rats' liver to observe its impact on liver damage.

CR3

CR3 partially supported the hypothesis since it just shows the impact of BPA on estrogen, fertility, and liver damage but not obesity. The first reason may be that similar to CR2, the effects of BPA on estrogen levels and fertility may be more pronounced or more sensitive to short-term exposures, whereas effects on obesity-related processes may only become apparent after long-term or chronic exposures. In addition, BPA may also cause disease, lead to reduced food intake in test subjects, and even accelerate weight loss. In order to delve deeper into these complex issues, future experiments could explore the time-dynamic effects of short- and long-term exposure to BPA on various physiological parameters. Meanwhile, the vital signs of the experiment group should be carefully observed to guarantee that there are no other diseases that would interfere or disrupt the experiment to cause bias.

CR4

CR4 shows that BPA exposure will increase estrogen levels, increase obesity, and increase liver damage, but it shows no impact on fertility, which partially supports the hypothesis. One possible reason is that BPA affects complex hormonal interactions, which is partially supported by CR4. Although the effects of BPA on estrogen levels were as expected, the unexpected fertility results suggest that BPA may have complex effects on reproductive function. This discrepancy may be due to the ability of BPA to disrupt hormonal signaling pathways or alter reproductive physiology, as BPA is essentially an endocrine disruptor. Another factor partially supporting CR4 may be the potential effects of BPA on gut group composition and function. BPA has been shown to disrupt the balance of bacteria in the gut, causing biological disturbances that can have profound effects on metabolism, inflammation, and general health. To address these issues, hormone analyses should be performed in experiments and rats should be given beneficial fungi as antidotes to gut microbes.

CR5

This hypothesis is partly supported by the fact that CR5 has no effect on estrogen levels. One possible cause is oxidative stress and inflammation. Exposure to BPA is known to cause the formation of reactive oxygen species, promote inflammatory responses that disrupt reproductive function, contribute to metabolic processes of obesity, and cause liver damage. These effects can occur independently of estrogen regulation, providing an alternative mechanism for BPA to influence these physiological parameters. Another possible cause is mitochondrial dysfunction. BPA has been linked to damage to mitochondrial function, which is essential for a variety of cellular processes. Mitochondrial dysfunction can affect fertility and lead to obesity, metabolism, and liver damage. Therefore, the effects on these parameters observed in CR5 may be due to BPA-induced mitochondrial dysfunction, highlighting an underlying mechanism that affects many physiological systems rather than directly affecting estrogen levels. In future experiments, the intervention drug will need to be used to ensure that it does not create bias.

CR6

Part of the support for CR6 may be caused by differences in metabolism. Differences in metabolic responses in rats may help to partially support the observed CR6 support. Individuals may have different differences in BPA metabolism, which may affect obesity and liver health. Factors such as genetic predisposition, general health, or prior metabolic conditions may influence the outcome of obesity and injury. Genetic or metabolic analysis can help understand how individual differences in metabolism may contribute to the different effects of BPA on obesity and impairment.

CR7

In CR7, the results showed that exposure to BPA resulted in elevated estrogen levels and liver injury but had no significant effect on fertility and obesity. This result partially supports the hypothesis by highlighting the effects of BPA on estrogen and injury, but suggests limited effects on fertility and obesity. One potential reason for this partial agreement may be the specific metabolic pathways affected by BPA. BPA may primarily target pathways associated with estrogen regulation and liver damage that do not have significant effects on reproduction and metabolism. Changes in the expression or activity of enzymes or receptors involved in these pools could explain the different responses observed in this study. To fill in these gaps, future experiments may focus on the epigenetic effects of BPA exposure. Studying BPA-induced changes in DNA methylation, or histone modification, provides insight into the long-term effects of BPA on genetic expression related to fertility, obesity, and estrogen signaling. In addition, the use of cell culture models in reproductive and metabolic tissues can help analyze specific responses to BPA affecting tissues and provide a more detailed understanding of how BPA affects various physiological processes.

CR8

The first reason partially supported by cr8 may be differences in the expression of hormone receptors. As can be seen from the previous results, differences in hormone receptor expression levels in different tissues can have an effect. In future experiments, BPA may detect estrogen receptors in the liver and reproductive organs. By comparing the expression patterns of the receptors, the researchers can explain how changes in the solubility of the receptors affect the response of specific tissues to BPA. This study could provide useful insight into the effects of BPA on estrogen signaling in a physiological setting. The second reason may be related to the metabolism and tissue distribution of BPA. Future studies will focus on the metabolism and distribution of BPA in various organs and tissues. Using techniques such as mass spectrum and image mass spectrum, researchers can track the fate of BPA in vivo and assess whether BPA accumulates in liver and reproductive tissue. Understanding the dynamics of BPA metabolism and distribution can reveal the physiological responses observed at exposure levels in specific tissues and fill the gaps in understanding the effects of BPA on estrogen, obesity, and injury.

CR9

The first reason to partially support cr9 is that postsexual genetic changes caused by BPA exposure affect estrogen observation and obesity but not reproduction and damage. Future experiments will be able to investigate the genetic changes in appearance caused by bisphenol in a variety of tissues, especially the inherited genes related to estrogen signaling, metabolic regulation, and reproductive function. By analyzing DNA methylation patterns or histone deformations, researchers can pinpoint how BPA induces genetic changes in appearance that affect the response of specific tissues. Understanding the cosmetic genetic mechanisms behind the other effects of BPA can provide a more comprehensive understanding of the effects of BPA on estrogen, obesity, fertility, and injury. The second reason is inflammation. Future studies could explore signs of inflammation in the liver, genitals, and other target tissues after BPA exposure. By evaluating inflammatory markers, such as cytokine discovery and immune cell infiltration,

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researchers can assess how inflammatory responses in specific tissues cause other effects of BPA. The observed physiological results may allow us to better understand the complex mechanisms that partially support CR9.

CR10

Some responses to BPA exposure support the CR10 hypothesis, which may be due to sex differences that lead to different effects on fertility and obesity. Future experiments could explore the different responses of males and females to BPA in terms of reproductive function and metabolic regulation. Comparative studies analyzing the effects of BPA on fertility and obesity in male and female animal models may reveal sex-specific patterns of response. By studying hormone levels, organ weight, and tissue histology in men and women exposed to BPA, it is possible to understand the underlying mechanisms that lead to sex-specific outcomes. By considering gender in experimental design and data analysis, researchers can better elucidate the complex interplay between BPA exposure, sex, and observed physiological effects by addressing one part of the CR10 support problem.

CR11

Part of the reason CR11 supports this hypothesis may be that BPA has the potential to disrupt the neuroendocrine system, thereby affecting fertility and obesity, without affecting liver damage and estrogen levels. The effects of BPA on the hypothalamic-pituitary-gonadal axis and hypothalamic-pituitary-adrenal axis neuroendocrine signaling pathways will be investigated in future experiments. The use of specific neuroendocrine knockout models or administration of targeted receptor antagonists of key neuroendocrine pathways in BPA-exposed animals could elucidate the role of neuroendocrine disruption in the fertility and obesity effects observed in CR11. By studying the effects of BPA on neuroendocrine function and its effects on reproductive and metabolic outcomes and revealing new mechanisms for the effects of BPA exposure.

CR12

CR12, which partially supports the hypothesis, may be because of the effect of BPA on hormonal crosstalk pathways. Future experiments may explore the interaction between estrogen signaling and other hormonal systems, such as androgens or thyroid hormones, in the context of BPA exposure. In a BPA exposure model, multi-hormone tests and cross-regulation studies of different hormone pathways can reveal how bisphenol A-induced changes in estrogen levels interact with other hormone signals to affect reproductive and metabolic function. Understanding the complex interactions between hormones and BPA exposure may help to understand the mechanisms underlying CR12 outcomes.

CR13

In CR13, the results showed that BPA did not increase estrogen levels but reduced fertility without affecting obesity or causing liver damage. This supports the above hypothesis to some extent. One potential reason for this partial support could be that BPA indirectly regulates fertility pathways without directly affecting estrogen levels. BPA may disrupt major signaling pathways or cellular mechanisms involved in regulating fertility, leading to the observed decline in fertility without affecting estrogen levels. To address these gaps, future experiments may focus on the molecular mechanisms underlying BPA's effects on fertility in the absence of changes in estrogen levels. Gene or proteomic expression analysis can reveal specific pathways or biomarkers associated with BPA-induced fertility changes. In addition, longitudinal studies evaluating the long-term effects of BPA exposure on reproductive function may provide more information about the mechanisms behind the CR13 results.

CR14

In CR14, the results showed that BPA did not alter estrogen levels or fertility but affected obesity and did not cause serious liver damage. This result was partially consistent with the hypothesis, suggesting a significant effect on obesity, but no effect on estrogen, fertility, and liver damage. One possible explanation is that BPA affects metabolism and storage mechanisms in fat tissue, leading to increased obesity, but does not directly affect estrogen or fertility. BPA can alter hormonal signaling related to metabolism, thereby promoting weight gain without altering reproductive function. To address these gaps, future experiments could further investigate the effects of BPA on specific metabolic pathways that contribute to obesity. Conducting metabolic profile studies or studying gene expression related to lipogenesis and lipid metabolism can provide help in understanding the molecular mechanism of BPA-induced obesity. Longitudinal studies that track metabolic markers over time could also reveal the long-term effects of BPA exposure on the development and progression of obesity, as well as their role in metabolic disorders.

CR15

In CR15, the results showed that BPA had no effect on estrogen levels, fertility, or obesity, but increased liver damage, more consistent with the hypothesis of induced

damage, and had no significant effect on other parameters. This may be due to cytotoxic mechanisms. One possible explanation for this inconsistency is that BPA acts primarily through direct cellular damage pathways rather than hormonal or metabolic pathways. BPA can damage cell structure or function, leading to observable liver damage, but does not directly affect estrogen levels, fertility, or mechanisms associated with obesity. To fill this gap, future experiments may focus on cellular mechanisms that bypass BPA liver damage. Studying markers of oxidative stress, inflammatory responses, or apoptotic pathways can give us insight into how BPA causes damage at the cellular level. In addition, studying the interaction of BPA with specific cell receptors or signaling pathways could reveal the molecular mechanisms of the damage caused by BPA and contribute to a better understanding of its toxicological effects. Long-term studies that monitor changes in cells over time may also shed light on the progression and persistence of liver damage caused by BPA.

CR16

In CR16, BPA did not have much effect on estrogen, fertility, obesity, and liver damage. One possible paradox observed in CR16 is the presence of confounding variables that were not accounted for in the study. Genetic variation in the researcher population, different metabolic responses to BPA exposure, or interactions with other environmental chemicals may mask the expected effects of BPA on estrogen levels, fertility, obesity, and tissue damage. The amount used in this experiment was not sufficient to have an observational effect on the measured parameters. In addition, exposure time may not be sufficient to cause substantial changes in estrogen levels, fertility, and tissue integrity. This suggests that the effects of BPA on these physiological aspects are dose-dependent and may be influenced by exposure time, which leads to the contradictory results observed in CR16. Future retests are needed, using higher concentrations of BPA and tests lasting longer on the test group.

5. Conclusion

The study is looking at the long-term effects of BPA exposure on human health. The results showed adverse effects of BPA exposure on estrogen, fertility, obesity, and liver function, partially supporting this hypothesis [6]. However, because the study was conducted in rats, people should be careful when experimenting with the results in humans [9]. Overcoming known knowledge gaps and exploring potential threat factors will help people better understand the complex relationship between BPA and human health [4]. Future research should focus on the mechanisms and observed effects of a particular disease. All in all, this study adds to the existing understanding of the risks of BPA exposure and highlights the need for further research [3].

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