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ANALYSIS OF COMORBIDITY IN ENDOCRINE DISRUPTORS OF POLYCARBONATE PLASTICS AND HYPERGLYCAEMIC AGENT IN MALE WISTAR RATS

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ABSTRACT

Polycarbonate plastics, widely used in consumer products, contains bisphenol A (BPA), a compound with endocrine-disrupting properties. BPA exposure has been implicated in exacerbating metabolic disorders, including diabetes. Understanding its histological impact on diabetic patients is critical, as chronic exposure may amplify cellular damage. This study employs an animal model to investigate the effects of BPA on diabetic tissues, focusing on histological alterations. Thus, the aim of this research is to elucidate the potential risks of polycarbonate plastic exposure for diabetic individuals. A total number of Twenty male Wistar rats which average weight was 185g were used and randomly splitted into four groups with each group comprising of five rats. Group A served as the control group, Group B were induced with Bisphenol- A (100mg/kg), Group C were induced with STZ (100mg/kg) which served as diabetic group, Group D were induced with Bisphenol-A (100mg/kg) and STZ (100mg/kg) which served as diabetic and bisphenol group. Bisphenol administration was done orally and STZ administration done intraperitoneally. The findings of this study revealed the histological damage caused by bisphenol-A (BPA), highlighting the serious health risks it poses upon entering the body. Specifically, chronic BPA exposure in adult rats was shown to have observable adverse effects, including significant reproductive alterations that could potentially result in infertility. Additionally, BPA exposure led to a marked reduction in body weight and an increase in fasting blood glucose levels. Chronic exposure to bisphenol-A induces histological damage, reproductive alterations, body weight reduction, and elevated fasting blood glucose, posing significant health risks.

Keywords: Polycarbonate plastic, Bisphenol, Streptozotocin (STZ), Endocrine disruptor

INTRODUCTION

Bisphenol-A (BPA) is a synthetic organic compound extensively used in the production of polycarbonate plastics and epoxy resins, which are key components in a wide range of consumer products. These include food and beverage containers, water bottles, dental sealants, and the linings of metal cans, among others (Malaise *et al.,* 2020). Due to its widespread usage, BPA has become a pervasive environmental contaminant, with its leaching potential posing significant risks to human and animal health, particularly under conditions of heat, acidic pH, or prolonged storage. This leaching leads to consistent exposure to BPA through dietary intake, inhalation, and dermal absorption, raising serious public health concerns (Shen *et al.,* 2020).

BPA is classified as an endocrine-disrupting chemical (EDC), meaning it can interfere with the body's hormonal systems by mimicking, blocking, or altering the natural actions of hormones. Its structural similarity to estrogen allows it to bind to estrogen receptors (ERs), disrupting estrogen-mediated processes (Malaise *et al.,* 2020). This disruption is particularly detrimental to the reproductive system, where BPA exposure has been associated with hormonal imbalances, impaired spermatogenesis, and oocyte dysfunction (Tomza *et al.,* 2018). The potential for BPA to induce reproductive toxicity and infertility has made it a focus of intense research and regulatory scrutiny.

The reproductive system is not the only target of BPA toxicity. Increasing evidence suggests that BPA exerts systemic effects, influencing metabolic pathways, glucose homeostasis, and overall body weight regulation. Chronic exposure to BPA has been shown to promote oxidative stress and inflammation, which are critical mediators of metabolic dysfunction (Schmucker *et al.,* 2022). Additionally, BPA exposure has been linked to glucose intolerance, insulin resistance, and alterations in fasting blood glucose levels in both animal models and human populations (Farrugia *et al.,* 2021) Such findings indicate that the impact of BPA extends beyond reproductive health, encompassing broader physiological systems that contribute to disease development. Histological studies provide valuable insights into the cellular and tissue-level changes caused by BPA exposure. Research has demonstrated that BPA can induce structural alterations in vital organs, including the testes, liver, and kidneys, often associated with oxidative damage and apoptotic pathways (Mentor *et al.,* 2020). These findings underscore the compound's ability to disrupt normal physiological functions,

even at low doses, which mimic environmental exposure scenarios. Furthermore, BPA-induced changes in testicular architecture, such as germ cell loss and Sertoli cell disruption, emphasize its role in compromising reproductive health (Rossi *et al.,* 2020)

Despite extensive research on the acute and sub-chronic effects of BPA, the mechanisms underlying its chronic toxicity remain incompletely understood. Chronic exposure scenarios are particularly relevant because they reflect real-life conditions where low-level, repeated exposure to BPA is continuous over long periods. Such exposure conditions may amplify BPA's effects on endocrine function, oxidative balance, and histopathology, leading to cumulative damage that is difficult to reverse.

This study aims to investigate the histological and metabolic consequences of chronic BPA exposure in adult male rats, focusing on its impact on reproductive health, body weight regulation, and glucose metabolism. Specifically, we seek to elucidate the potential dose-dependent effects of BPA on testicular structure, serum hormone levels, and metabolic biomarkers. By bridging gaps in current knowledge, this study provides crucial insights into the health risks posed by BPA, which can inform public health policies and regulatory measures to mitigate exposure. Understanding the long-term effects of BPA is essential for developing preventive strategies to protect vulnerable populations, including pregnant women, children, and individuals with pre-existing metabolic or reproductive disorders.

MATERIALS AND METHODS

Compounds and Animals Procurement

All compounds used (Bisphenol and Streptozotocin) were pure compounds procured from Akol Pharmacy Osogbo. Experimental animals used for this research were procured from the Animal Holdings College of Medicine, Ladoke Akintola University Ogbomoso, Oyo State. The animals were allowed access to food ad libitum. They were given two weeks to get used to the lab condition before the study started. The study followed the rules set by the Health Research Ethics Committee at the College of Health Sciences, Osun State University, Osogbo, Nigeria, and complied with the National Institute of Health guidelines for caring for and using lab animals (Cadmus *et al*., 2024).

Experimental Design

Twenty male (20) adult Wistar rats which average weight was 185g were used and the animals were randomly split into four groups (A, B, C and D), each with five rats. Group A (served as the control) given distilled water and feed only, Group B rats were exposed to Bisphenol 100mg/kg, Group C rats were exposed to 100mg/kg of STZ which served as diabetic group, Group D were induced with Bisphenol-A (100mg/kg) and STZ (100mg/kg) which served as diabetic and bisphenol group. Potrebić *et al*., 2022

Measurement of Body Weight

The weights of the animals were obtained upon arrival and on weekly basis using digital weighing balance scale in order to account for possible results in physical changes in rats upon administration (STZ and chemical compounds) at regular intervals. The weights are checked for the comparison of possible changes from the initial weight and kept in record.

Sacrifice of Experimental Animals

Blood was withdrawn from the apex of the heart (left ventricle) of the twenty-five adult rats, which were first anesthetized with 80 mg/kg of ketamine hydrochloride, 12 hours after the last administration just according to Struyf *et al*., 2022 .

Sample Collection and Hormonal Assay

The blood was then dispensed into red-topped tubes for hormonal analysis. The pancreas was excised following an abdominal incision, and they were fixed in formosaline for histological analysis. It was then dehydrated progressively in graded alcohols, cleared in Xylene and infiltrated in paraffin wax, before being embedded in molten paraffin wax. A rotary microtome was then used to slice the paraffin block containing the tissue into 4 μm thick sections. The sections were then transferred to a glass slide, floated in a water bath set at 40 degrees Celsius, and stained with hematoxylin and eosin dyes.

Hormonal Assay

Serum samples were assayed for Insulin in batches with the control sera at both physiological and pathological levels by the standard Quantitative Enzyme-Linked Immunosorbent Assay(ELISA) technique with microwell kit which was manufactured by Syngenemed incorporated, India. The manufacturer instructions that accompanied the assay kits were strictly adhered to.

Measurement of Fasting Blood Glucose

The blood sugars of overnight- fasted rats (for about 10-12 hours) were measured by using GLUCOMETER (Accu-check). Blood was obtained by tail vein puncture. The Glucose level was monitored weekly and record kept.

Statistical Analysis

The mean and standard error of mean (S.E.M) of all data's were calculated. Comparison of means was made by one way analysis of variance (ANOVA) using Graphpad Prism 5. Tukey's test was used to adjust for multiple comparisons. P value < 0.05 was considered to be statistically significant.

RESULTS AND DISCUSSION

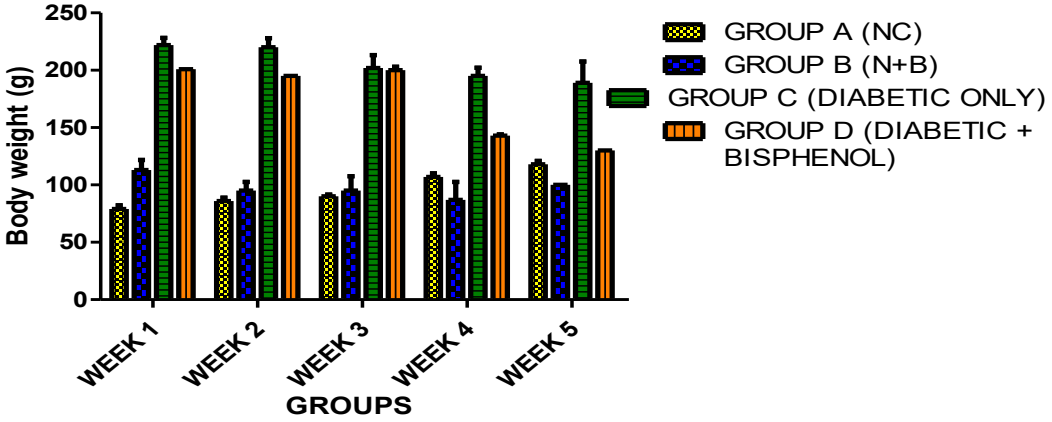

Figure 1: Bar chart showing mean weekly body weights of animals in four groups: normal control (A), normal + bisphenol (B), diabetic only (C), and diabetic + bisphenol (D). Diabetic groups (C and D) showed significantly lower weights compared to non-diabetic groups (A and B). One-way ANOVA revealed a significant difference among groups (***P < 0.0001), with post hoc Tukey's test confirming significant differences between diabetic and non-diabetic groups

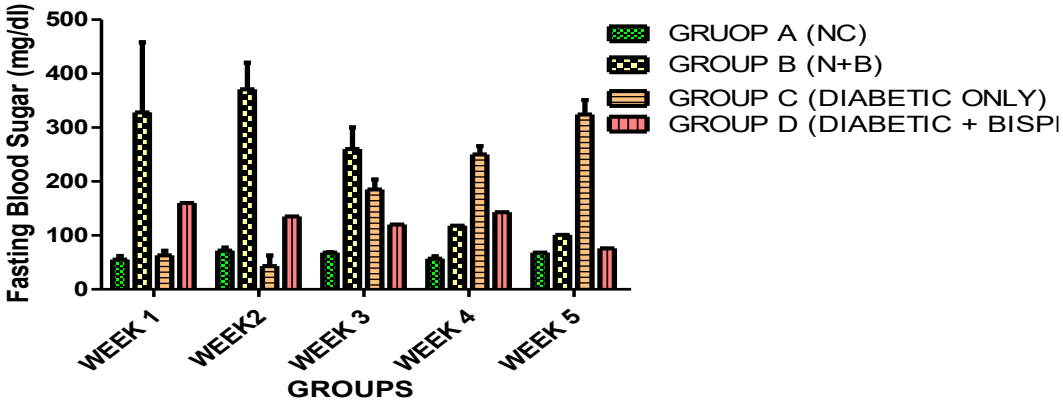

Figure 2: Bar graph showing mean fasting blood sugar (FBS) levels (mg/dL ± SEM) over five weeks in four groups: Group A (normal control), Group B (non-diabetic + bisphenol), Group C (diabetic only), and Group D (diabetic + bisphenol). Group B showed significantly elevated FBS compared to Group A (*P < 0.05, one-way ANOVA; Dunnett's post hoc test)

Insulin

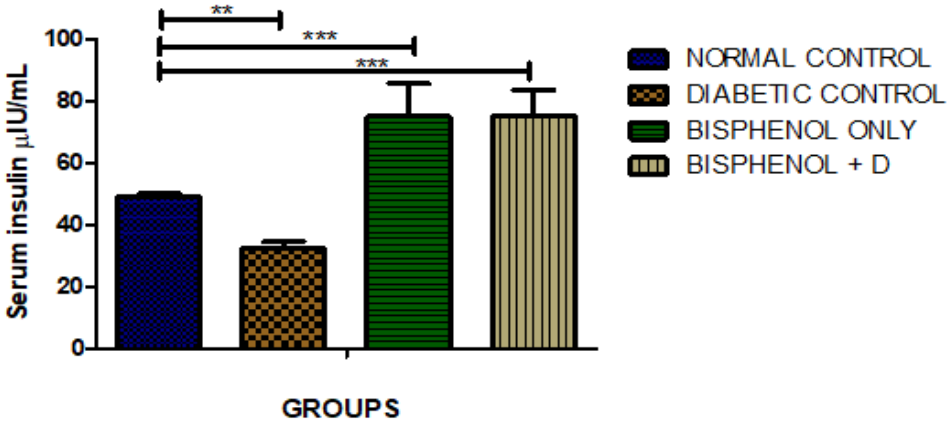

Figure 3: Bar chart representing the mean serum sodium levels (± SEM) across experimental groups. Statistical analysis by one-way ANOVA showed a significant difference among *groups (**P < 0.0001, F = 49.90, R^2 = 0.9034). Bonferroni post hoc test revealed significant differences between most group pairs, except between the Dioxin only and Dioxin + Diabetic groups (ns)*

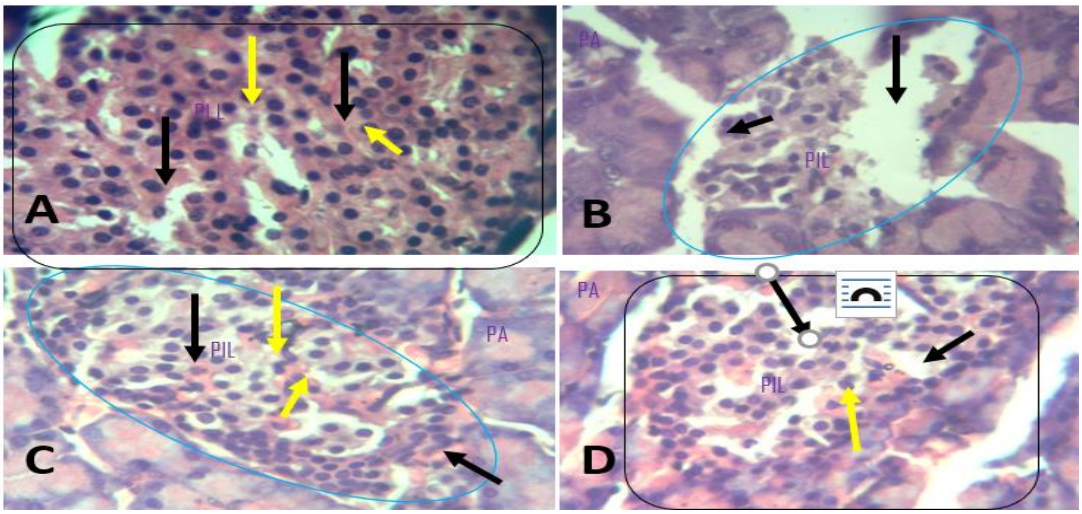

Plate 1: Hematoxylin and Eosin-stained sections of the pancreas (×400). **A**: Normal control showing intact islets of Langerhans with dense, uniform cells. **B**: Bisphenol-treated group displaying disrupted islet structure and reduced cell density. **C**: Diabetic group with shrunken islets, marked cell loss, and nuclear degeneration. **D:** Bisphenol + diabetic group showing severe islet atrophy and architectural distortion. PIL = Pancreatic Islets of Langerhans; PA = Pancreatic acini; black/yellow arrows indicate nuclei and islet boundaries, respectively

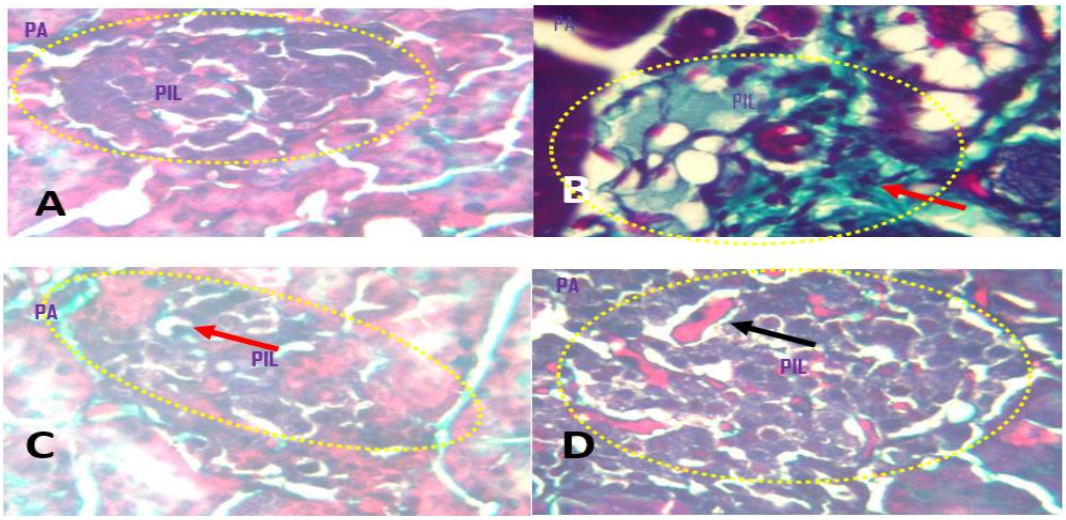

Plate 2: Masson's Trichrome-stained sections of the pancreas (×400). **A**: Normal control with intact islets and minimal collagen. **B**: Bisphenol-treated showing islet disruption and fibrosis (red arrow). **C**: Diabetic group with moderate collagen deposition (red arrow). **D**: Bisphenol + diabetic group with severe islet fibrosis (black arrow). Blue-green coloration indicates collagen; PIL = pancreatic islets of Langerhans, PA = pancreatic acini.

The present study evaluated the effects of bisphenol (BPA), diabetes, and their combined impact on body weight, fasting blood sugar (FBS), serum insulin, and pancreatic histology in experimental animals. Both biochemical and histological assessments provided insights into how these conditions independently and collectively affect metabolic and pancreatic health. Clear differences in body weight were observed among the groups(Fig.1). The non-diabetic groups (A: normal control and B: bisphenol-treated) maintained relatively higher body weights throughout the five-week study period. In contrast, the diabetic groups (C: diabetic only and D: diabetic + bisphenol) showed consistently lower weights. Among them, the diabetic-only group had the lowest values, whereas the diabetic + bisphenol group exhibited slightly higher weights, though still markedly reduced compared to the non-diabetic groups. Statistical analysis using one-way ANOVA confirmed that these differences were highly significant ($P < 0.0001$, $F = 34.58$). The R^2 value of 0.8664 indicated that 87% of the variation in body weight could be attributed to group differences. Bartlett's test showed no violation of the equal variance assumption ($P = 0.0898$). Tukey's post hoc test revealed no significant difference between Groups A and B, confirming that bisphenol alone did not significantly influence body weight in non-diabetic animals. However, significant differences were found between non-diabetic and diabetic groups ($P < 0.001$), underscoring the strong effect of diabetes on weight reduction. No significant difference emerged between Groups C and D, suggesting bisphenol did not exacerbate diabetic weight loss. Weekly monitoring of FBS revealed distinct patterns(Fig 2). Group A maintained stable, low levels throughout the study. In contrast, Group B exhibited markedly elevated FBS, particularly in Weeks 2 and 5, suggesting that bisphenol disrupts glucose regulation even in non-diabetic animals. Groups C and D showed moderate to high glucose levels, with Group D tending to have higher values than Group C, especially in later weeks. One-way

ANOVA confirmed significant differences (P = 0.0414, F = 3.460), with 39.4% of variability explained by group differences. Bartlett's test indicated unequal variances (P = 0.0001). Dunnett's post hoc analysis revealed significantly higher FBS in Group B compared to the control (P < 0.05), whereas differences between the control and diabetic groups (C and D) were not statistically significant. Serum insulin levels varied markedly among the groups(Fig 3). ANOVA results showed a highly significant difference (P < 0.0001, F = 49.90), with an R^2 value of 0.9034, meaning about 90% of the variation could be explained by treatment differences. Bartlett's test revealed unequal variances (P = 0.0007). Post hoc Bonferroni analysis showed that diabetic animals had significantly lower insulin compared to controls (P < 0.01). Interestingly, both the bisphenol-only and bisphenol + diabetic groups displayed significantly higher insulin compared to the diabetic-only group (P < 0.001), approaching values seen in the normal controls. However, there was no significant difference between bisphenol-only and bisphenol + diabetic groups. Pancreatic histology revealed progressive damage across treatments (Plate 1). In controls, the islets of Langerhans appeared intact, densely populated, and well-structured. Bisphenol exposure caused reduced cellular density, irregular nuclei, and mild vacuolation, indicating early toxic changes. Diabetic animals displayed severely compromised islets with marked cellular loss, nuclear degeneration, and poorly defined architecture. In the combined bisphenol + diabetic group, islet disruption was extensive, with significant cellular loss, vacuolation, and atrophy, suggesting bisphenol intensified diabetes-related pancreatic damage. Masson's Trichrome staining highlighted collagen deposition and fibrosis(Plate 2). In controls, collagen was minimal, with intact islets and normal acinar tissue. Bisphenol exposure induced marked collagen deposition around and within the islets, alongside adipocyte infiltration. The diabetic group showed moderate fibrosis and disrupted islet architecture. The combined bisphenol + diabetic group displayed the most severe fibrosis, with extensive collagen accumulation and distorted islet boundaries, suggesting a synergistic effect of bisphenol and diabetes in promoting pancreatic fibrosis.

Discussion

The histological analysis of pancreatic tissues in this study reveals critical insights into structural alterations resulting from bisphenol-A (BPA) exposure, diabetes, and their combined effects. Each experimental group exhibited distinct histopathological changes, highlighting the impact of these conditions on pancreatic integrity. In the normal control group (Group A), the pancreas displayed a well-preserved histoarchitecture, characterized by clearly defined Islets of Langerhans, orderly arranged acinar cells, and intact blood vessels. This observation represents the expected morphology of a healthy pancreas, providing a reference point for comparison with the other groups. In Group B, which was exposed to BPA, early signs of pancreatic damage were evident. These included slight disorganization of the acinar cells and initial structural alterations in the Islets of Langerhans. Such changes underscore the toxic effects of BPA on pancreatic tissues. BPA is known to disrupt cellular structure through mechanisms involving oxidative stress and inflammation, potentially impairing insulin production and secretion over time (Malaise *et al.,* 2020). The observed histological changes suggest that even low-level BPA exposure can compromise pancreatic function. Group C, comprising diabetic subjects, demonstrated significant pathological changes. These included inflammation within the interstitial spaces and a reduction in the size of acinar cells. These findings align with the established effects of diabetes on pancreatic tissues, where chronic hyperglycemia and oxidative stress contribute to cellular atrophy, fibrosis, and inflammation (Mentor *et al.,* 2020). The reduced size of acinar cells may indicate diminished exocrine function, a common feature of diabetes-related pancreatic damage.The findings of this study reveal that bisphenol-A (BPA), a key component of polycarbonate plastics, exerts significant adverse effects on diabetic animal models. BPA exposure led to observable histological alterations, metabolic disruptions, and exacerbation of diabetic symptoms, highlighting its toxicological impact on vulnerable systems.

One of the most striking observations was the histological damage caused by BPA in the examined tissues. The endocrine-disrupting nature of BPA has been well-documented, particularly its ability to mimic estrogen and disrupt normal hormonal signaling (Malaise *et al.,* 2020). In diabetic conditions, where metabolic and oxidative stress are already heightened, BPA exposure likely compounds tissue damage through the generation of reactive oxygen species (ROS) and inflammation (Mentor *et al*., 2020). These findings align with previous studies that have reported BPA-induced oxidative damage and apoptosis in vital organs, including the pancreas, liver, and reproductive tissues (Wang et al., 2019).

The metabolic effects observed in this study, including alterations in fasting blood glucose levels, further support the notion that BPA interferes with glucose homeostasis. BPA has been shown to impair insulin signaling, contributing to glucose intolerance and insulin resistance (Farrugia *et al*., 2021). This is particularly concerning for diabetic individuals, as BPA exposure may exacerbate hyperglycemia and hinder glycemic control. These results are consistent with epidemiological studies linking BPA exposure to an increased risk of type 2 diabetes in humans (Schmucker *et al*., 2022)

BPA's structural similarity to estrogen enables it to bind to estrogen receptors, disrupting the delicate hormonal balance necessary for reproductive health (Tomza-marciniak *et al*., 2018). This study also highlights the cumulative impact of chronic BPA exposure, which reflects real-world conditions where individuals are continuously exposed to low levels of BPA over time. Unlike acute exposure, chronic exposure scenarios provide a more accurate representation of the long-term risks associated with polycarbonate plastics in daily life. These findings emphasize the need for stricter regulatory measures to limit BPA exposure, particularly for high-risk groups such as diabetic patients and individuals with pre-existing metabolic disorders.

The combined exposure to BPA and diabetes in Group D resulted in more pronounced histopathological alterations, including vacuolation and disorganization of the Islets of Langerhans. The vacuolation suggests cellular degeneration, likely due to the synergistic effects of BPA toxicity and diabetes-induced oxidative stress. The disorganization of the Islets of Langerhans further underscores the additive impact of these conditions on endocrine pancreatic function. Previous studies have reported that BPA exacerbates diabetic pathology by interfering with glucose homeostasis, insulin signaling, and cellular integrity (Farrugia *et al*., 2021). The findings

in this group highlight the heightened vulnerability of diabetic individuals to environmental toxins like BPA.

CONCLUSION

Polycarbonate plastic, through its primary component bisphenol-A (BPA), demonstrates significant histological effects in diabetic models. Chronic BPA exposure exacerbates tissue damage, metabolic imbalances, highlighting its potential to worsen diabetic complications. These findings underscore the urgent need for stricter regulation of BPA-containing materials to safeguard diabetic patients from its harmful effects.

RECOMMENDATION

To reduce the harmful effects of bisphenol-A (BPA), stricter regulations should limit its use in consumer products, especially those in contact with food. Public awareness campaigns are essential to educate individuals on BPA's risks and promote safer alternatives. Diabetic individuals, being particularly vulnerable, should minimize exposure.

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