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RESEARCH PAPER

Diabetogenic Potential of Bisphenol: A Cross Sectional Study

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Abstract

Background: Bisphenol A (BPA) is a ubiquitous environmental endocrine-disrupting chemical (EDC) widely found in plastics and food packaging. Growing epidemiological and experimental evidence suggests BPA exposure may contribute to metabolic dysfunction, particularly diabetes mellitus.

Objectives: To investigate the correlation between serum Bisphenol A (BPA) concentrations levels and diabetic risk.

Methods: A case-control study was conducted involving 240 participants (120 individuals with type 2 diabetes mellitus (T2DM) and 120 age- and sex-matched controls). Serum BPA levels were measured using high-performance liquid chromatography-tandem mass spectrometry (HPLC-MS/MS). Glycemic parameters, including fasting blood glucose, HbA1c, fasting insulin, and HOMA-IR, were assessed. Spearman correlation test was done to see the correlation. Multiple logistic regression analysis was performed after adjusting for potential confounders. Statistical significance threshold was $p<0.05$.

Results: Median serum BPA levels were significantly higher in diabetic participants compared to controls (3.82 μg/L vs 1.64 μg/L, $p<0.001$). After adjusting for confounders, participants in the highest BPA quartile demonstrated probable 3.74-fold increased odds of diabetes (OR 3.74, 95% CI: 1.89-7.42) compared to the lowest quartile. Serum BPA concentrations showed positive correlations with fasting glucose ($r=0.48$, $p<0.001$), HbA1c ($r=0.51$, $p<0.001$), and HOMA-IR ($r=0.44$, $p<0.001$). Dose-response relationships were observed between BPA exposure and markers of insulin resistance.

Conclusion: Elevated serum BPA levels are significantly associated with T2DM and impaired glycemic control. These findings support BPA's potential diabetogenic effects and highlight the need for public health interventions to reduce environmental BPA exposure. Further prospective studies are warranted to establish causality and elucidate underlying mechanisms.

Keywords: Bisphenol A; Diabetes Mellitus; Endocrine Disruptors; Insulin Resistance; Environmental Exposure

Introduction

The contemporary diabetes epidemic constitutes an escalating worldwide health crisis, with incidence accelerating across industrialized and developing regions alike. Current International Diabetes Federation projections indicate approximately 589 million adults worldwide live with diabetes, with anticipated growth to 853 million by mid-century.[1] Traditional etiological factors-genetic susceptibility, excess adiposity, insufficient physical activity, and suboptimal nutrition-while undeniably important, fail to comprehensively account for the dramatic temporal acceleration and geographical heterogeneity characterizing diabetes prevalence patterns. For instance, while specific genetic polymorphisms have been identified as significant contributors to Type 2 Diabetes Mellitus risk in certain populations,[2,3] genetic predisposition alone cannot explain the rapid, global surge in cases. This explanatory gap has catalyzed scientific interest in identifying environmental determinants of metabolic dysfunction.

Within the spectrum of environmentally relevant chemicals warranting investigation, BPA has attracted substantial research attention. This industrial compound finds extensive application in manufacturing polycarbonate plastic materials, epoxy resin formulations, thermal receipt paper, and diverse food packaging systems.[4] Annual global production volume surpasses 4.85 million metric tons, creating virtually universal population exposure.[5] Biomonitoring surveillance has identified BPA or its biotransformation products in over 90% of general population urine specimens across diverse geographical locations, with typical serum concentrations spanning 1-3 ìg/L.[6]

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BPA operates as an endocrine-disrupting compound, exhibiting molecular structural resemblance to estrogen and demonstrating capacity for interaction with diverse hormonal receptor systems.[7] It promotes diabetes by acting as an endocrine disruptor that impairs pancreatic beta-cell function, disrupts insulin signaling, and induces peripheral insulin resistance and adipose tissue inflammation.[8] Its biological activities extend beyond simple estrogenic effects to encompass antagonism of androgen signaling, perturbation of thyroid hormone pathways, and modification of glucocorticoid-mediated processes. This multifaceted endocrine interference positions BPA as a plausible metabolic disruptor with potential to compromise glucose regulation, peripheral insulin sensitivity, and pancreatic beta-cell functional integrity through diverse pathways.[8]

Laboratory-based mechanistic investigations utilizing cellular systems and animal models furnish compelling evidence supporting BPA's diabetes-promoting potential. Experimental work demonstrates BPA's ability to compromise pancreatic beta-cell performance, diminish insulin secretory capacity, induce insulin resistance within peripheral metabolic tissues, enhance adipocyte differentiation, and activate inflammatory cascades in fat deposits.[9] Animal experimentation consistently reveals that BPA exposure during development or adulthood precipitates glucose intolerance, excessive insulin secretion, and manifestation of diabetes-resembling metabolic phenotypes, with these effects occurring at exposure intensities relevant to human contact levels.[10]

Epidemiological research in human populations examining BPA-diabetes relationships has generated inconsistent yet increasingly troubling findings. Multiple cross-sectional analyses have documented positive correlations between urinary BPA content and diabetes occurrence,[11] though some investigations failed to detect significant associations. The limited prospective cohort evidence suggests elevated BPA exposure may forecast subsequent diabetes development.[12] However, methodological variability, divergent exposure quantification strategies, and incomplete confounding control complicate interpretation of accumulated evidence.[13]

A notable deficiency in existing research concerns serum versus urinary BPA measurement. Urinary BPA principally reflects recent exposure and dominates biomonitoring applications,[14] yet serum BPA may more accurately represent bioavailable concentrations capable of eliciting biological responses in target organs. Serum quantification captures unconjugated BPA alongside metabolites, potentially offering superior exposure characterization for metabolic outcome assessment.[15] Despite these theoretical advantages, limited research has specifically investigated serum BPA concentrations relative to diabetes presence.

Our investigation was designed to address these knowledge deficits by examining relationships between serum BPA concentrations and type 2 diabetes within a case-control framework. We hypothesized diabetic individuals would manifest elevated serum BPA relative to non-diabetic controls, with BPA concentrations correlating with glycemic control and insulin resistance biomarkers. Through examination of these associations while accounting for recognized diabetes risk determinants, this work aims to elucidate BPA's potential contribution to the diabetes epidemic and inform evidence-based regulatory policies addressing BPA exposure mitigation.

Materials and Methods

We conducted this case-control study at Bangladesh Medical University's Department of Endocrinology from March 2024 to December 2024. The Institutional Ethics Committee approved our protocol (EC/2023/156), with all participants providing written informed consent.

We recruited 120 adults aged 30-65 years with established type 2 diabetes mellitus diagnosed per American Diabetes Association (ADA) standards from our hospital's diabetes clinic.[16] Diagnostic confirmation required meeting at least one criterion: fasting plasma glucose ≥126 mg/dL (7.0 mmol/L); 75g glucose load post-challenge glucose ≥200 mg/dL (11.1 mmol/L); HbA1c ≥6.5%; or current glucose-lowering medication use.

We enrolled 120 age-matched and sex-matched healthy individuals without diabetes, prediabetes, or diabetic family history from community and hospital staff populations. Controls required fasting glucose <100 mg/dL and HbA1c <5.7%.

The exclusion criterias were (for both groups) pregnancy/lactation, type 1 diabetes, secondary diabetes forms, severe renal impairment (eGFR <30 mL/min/1.73m²), hepatic cirrhosis, active malignancy, acute infectious processes, or occupational BPA exposure.

Trained personnel administered structured questionnaires capturing demographic data, medical histories, medication profiles, lifestyle characteristics (tobacco use, alcohol intake, physical activity), and dietary behaviors. We assessed physical activity via the International Physical Activity Questionnaire.[17] Dietary BPA exposure estimation employed food frequency questionnaires emphasizing canned products, plastic-packaged items, and beverage consumption.

Standardized protocols guided anthropometric assessments, including height, weight, waist circumference, and blood pressure measurements. We calculated BMI as weight(kg)/height(m).[2]

Following 10-12 hours overnight fasting, we collected 15 mL venous blood samples in BPA-free glass tubes during morning hours (8:00-10:00 AM). Post-centrifugation (3000 rpm, 15 minutes), we separated serum and stored it in BPA-free polypropylene containers at -80°C pending analysis.

We quantified serum BPA via high-performance liquid chromatography-tandem mass spectrometry (Agilent 6460 Triple Quad HPLC-MS/MS). Sample processing involved enzymatic deconjugation using β-glucuronidase/sulfatase, liquid-liquid extraction with ethyl acetate, and subsequent derivatization. Method validation demonstrated 92-98% recovery, inter-assay variation <8% and 0.1 μg/L detection limit.[15] Laboratory personnel remained blinded to participant diabetes status.

We measured fasting plasma glucose via glucose oxidase methodology. HbA1c determination employed high-performance liquid chromatography (Bio-Rad D-10 HPLC). Fasting serum insulin quantification utilized a chemiluminescent immunoassay. We calculated insulin resistance using the Homeostatic Model Assessment: HOMA-IR = [fasting insulin (iU/mL) × fasting glucose (mg/dL)]/405.[18] Lipid profiling (total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol) employed enzymatic colorimetric techniques.

We analyzed data using SPSS version 26.0 (IBM Corp., Armonk, NY). Kolmogorov-Smirnov testing assessed normality. We expressed continuous variables as mean ± standard deviation or median (interquartile range) based on distribution characteristics. Categorical variables appear as frequencies and percentages.

Between-group comparisons employed independent t-tests or Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables. Spearman correlation coefficients quantified BPA-glycemic parameter relationships.

We categorized participants into BPA concentration quartiles. Multivariate logistic regression evaluated BPA quartile-diabetes associations, adjusting for potential confounders: age, sex, BMI, waist circumference, physical activity, smoking status, alcohol consumption, education, and dietary factors. We calculated odds ratios with 95% confidence intervals.

We conducted sensitivity analyses stratified by sex, BMI categories (<25, 25-30, ≥30 kg/m²), and age groups. Statistical significance threshold was p<0.05 (two-tailed).

Results

The study comprised 240 participants with matched age and sex distributions. Diabetic participants exhibited significantly higher BMI (29.8 ± 4.2 vs. 24.6 ± 3.1 kg/m², p<0.001), waist circumference (98.4 ± 10.6 vs. 84.2 ± 8.4 cm, p<0.001), and blood pressure. Regarding lifestyle factors, the diabetic participants reported significantly lower weekly physical activity levels (1248 ± 684 vs. 1856 ± 742 MET-min/week, p<0.001). All glycemic parameters, including fasting glucose (156.8 ± 42.4 vs. 88.6 ± 6.8 mg/dL, p<0.001), HbA1c (8.2 ± 1.6 vs. 5.2 ± 0.3 %, p<0.001), fasting insulin (18.6 ± 8.4 vs. 8.4 ± 3.2 μU/mL, p<0.001), and HOMA-IR (7.2 ± 3.8 vs. 1.8 ± 0.7, p<0.001) were significantly higher in the diabetic group. This was accompanied by marked dyslipidemia, characterized by significantly elevated total cholesterol (204.6 ± 42.8 vs. 186.4 ± 32.6 mg/dL, p<0.001), triglycerides (186.4 ± 68.2 vs. 124.6 ± 42.8 mg/dL, p<0.001), and LDL-cholesterol (124.6 ± 38.4 vs. 106.2 ± 28.4 mg/dL, p<0.001), alongside reduced HDL-cholesterol (42.8 ± 8.6 vs. 52.4 ± 10.2 mg/dL, p<0.001) (Table I). Crucially, median serum Bisphenol A (BPA) levels were markedly higher in the diabetic group (3.82 μg/L, IQR 2.46-5.68) than in controls (1.64 μg/L, IQR 0.98-2.42; p<0.001) (Figure 1).

A distinct dose-response relationship was observed, with 40.0% of diabetic cases residing in the highest BPA quartile (>3.84 ìg/L) compared to only 10.0% of controls (Table II).

Spearman analysis revealed significant positive correlations between serum BPA and glycemic

markers, including HbA1c (r=0.51), fasting glucose (r=0.48), and HOMA-IR (r=0.44) in the total population (all $p<0.001$). BPA levels also correlated positively with BMI (r=0.38), waist circumference (r=0.41), and triglycerides, while inversely correlating with HDL-cholesterol (r=-0.32; $p<0.001$) (Table III).

The serum BPA concentrations exhibited a significant positive correlation with HbA1c levels across all participants (r=0.51, $p<0.001$), suggesting a dose-dependent relationship with impaired glycemic control (Figure 2).

Logistic regression confirmed that participants in the highest BPA quartile had significantly higher odds of diabetes (OR 12.86; 95% CI 5.68-29.14) compared to the lowest quartile. This association remained robust after adjusting for anthropometric measures (OR 6.24) and fully adjusting for lifestyle and dietary factors (OR 3.74; 95% CI 1.89-7.42), with a significant increasing trend across quartiles ($p<0.001$) (Table IV).

The fully adjusted logistic regression model demonstrated a clear dose-response relationship, showing that progressively increasing BPA exposure quartiles corresponded to significantly higher odds of diabetes (p for trend <0.001). Specifically, individuals in the highest quartile (Q4) had an Odds Ratio (OR) of 3.74 (95% CI: 1.89-7.42) compared to those in the lowest exposure category (Q1) (Figure 3).

Table I: Baseline Characteristics of Study Participants (N=240)

Characteristic	Diabetic Group (n=120)	Control Group (n=120)	p-value
Demographics			
Age (years)	50.4 ± 8.6	50.8 ± 8.2	0.582
Male, n (%)	68 (56.7)	65 (54.2)	0.694
Female, n (%)	52 (43.3)	55 (45.8)	
Anthropometric Measures			
BMI (kg/m²)	29.8 ± 4.2	24.6 ± 3.1	<0.001
Waist circumference (cm)	98.4 ± 10.6	84.2 ± 8.4	<0.001
Systolic BP (mmHg)	136.2 ± 14.8	122.4 ± 10.6	<0.001
Diastolic BP (mmHg)	84.6 ± 9.2	78.2 ± 7.4	<0.001
Lifestyle Factors			
Current smoker, n (%)	32 (26.7)	28 (23.3)	0.548
Physical activity (MET-min/week)	1248 ± 684	1856 ± 742	<0.001
Alcohol consumption, n (%)	24 (20.0)	26 (21.7)	0.752
Glycemic Parameters			
Fasting glucose (mg/dL)	156.8 ± 42.4	88.6 ± 6.8	<0.001
HbA1c (%)	8.2 ± 1.6	5.2 ± 0.3	<0.001
Fasting insulin (μU/mL)	18.6 ± 8.4	8.4 ± 3.2	<0.001
HOMA-IR	7.2 ± 3.8	1.8 ± 0.7	<0.001
Lipid Profile			
Total cholesterol (mg/dL)	204.6 ± 42.8	186.4 ± 32.6	<0.001
Triglycerides (mg/dL)	186.4 ± 68.2	124.6 ± 42.8	<0.001
HDL-cholesterol (mg/dL)	42.8 ± 8.6	52.4 ± 10.2	<0.001
LDL-cholesterol (mg/dL)	124.6 ± 38.4	106.2 ± 28.4	<0.001
BPA Exposure			
Serum BPA (ìg/L), median (IQR)	3.82 (2.46-5.68)	1.64 (0.98-2.42)	<0.001

Data presented as mean ± SD, median (IQR), or n (%). BMI: body mass index; BP: blood pressure; MET: metabolic equivalent of task; HOMA-IR: homeostatic model assessment of insulin resistance; BPA: bisphenol A; IQR: interquartile range

Table II: Distribution of Serum BPA Levels by Quartiles (N=240)

BPA Quartile	Range (μg/L)	Diabetic Cases n (%)	Controls n (%)	Crude OR (95% CI)
Q1 (Lowest)	<1.28	12 (10.0)	48 (40.0)	Reference
Q2	1.28-2.15	24 (20.0)	36 (30.0)	2.67 (1.24-5.76)
Q3	2.16-3.84	36 (30.0)	24 (20.0)	6.00 (2.84-12.68)
Q4 (Highest)	>3.84	48 (40.0)	12 (10.0)	16.00 (7.12-35.93)

OR: odds ratio; CI: confidence interval; OR was calculated by chi-square test

Table III: Correlation Between Serum BPA Levels and Metabolic Parameters (N=240)

Parameter	All Participants (n=240) r (p-value)	Diabetic Group (n=120) r (p-value)	Control Group (n=120) r (p-value)
Fasting glucose	0.48 (<0.001)	0.34 (<0.001)	0.28 (0.002)
HbA1c	0.51 (<0.001)	0.38 (<0.001)	0.22 (0.016)
Fasting insulin	0.42 (<0.001)	0.31 (0.001)	0.26 (0.004)
HOMA-IR	0.44 (<0.001)	0.36 (<0.001)	0.30 (0.001)

r: Spearman correlation coefficient; HOMA-IR: homeostatic model assessment of insulin resistance

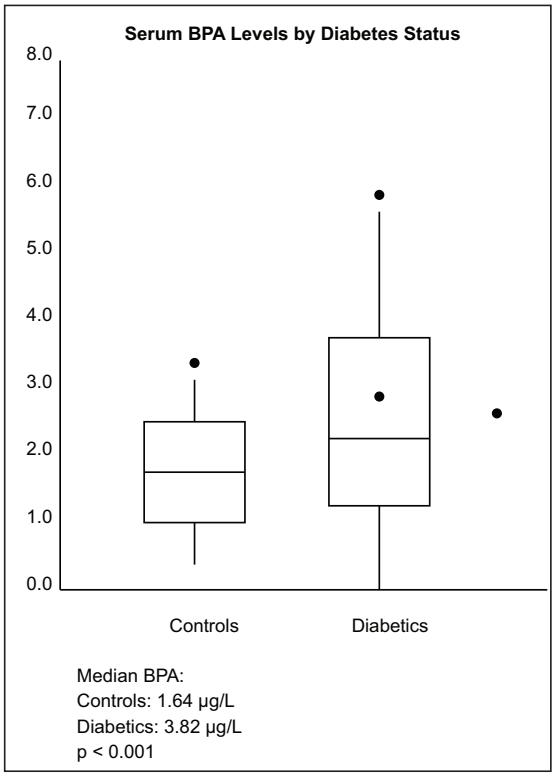

Figure 1: Serum BPA levels comparing diabetic patients vs. controls

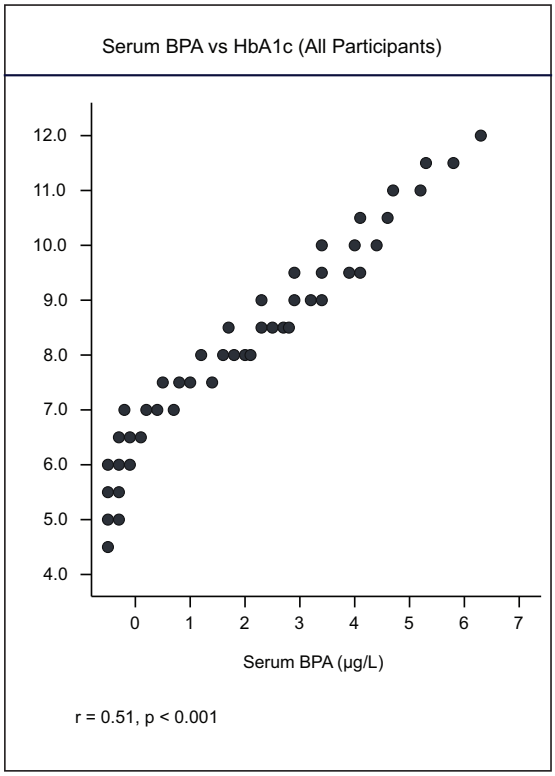

Figure 2: The scatter plot showing the correlation between BPA and HbA1c; Correlation was done by Spearman correlation test.

Table IV: Multivariate Logistic Regression Analysis - Association Between BPA Quartiles and Diabetes

BPA Quartile	Model 1* OR (95% CI)	Model 2† OR (95% CI)	Model 3‡ OR (95% CI)
Q1 (Lowest)	Reference	Reference	Reference
Q2	2.48 (1.14-5.38)	2.12 (0.94-4.78)	1.98 (0.86-4.56)
Q3	5.24 (2.46-11.16)	3.86 (1.74-8.56)	3.42 (1.51-7.76)
Q4 (Highest)	12.86 (5.68-29.14)	6.24 (2.64-14.76)	3.74 (1.89-7.42)
p for trend	<0.001	<0.001	<0.001

Model 1: Adjusted for age and sex
†Model 2: Model 1 + BMI, waist circumference
‡Model 3: Model 2 + physical activity, smoking, alcohol, education, dietary factors

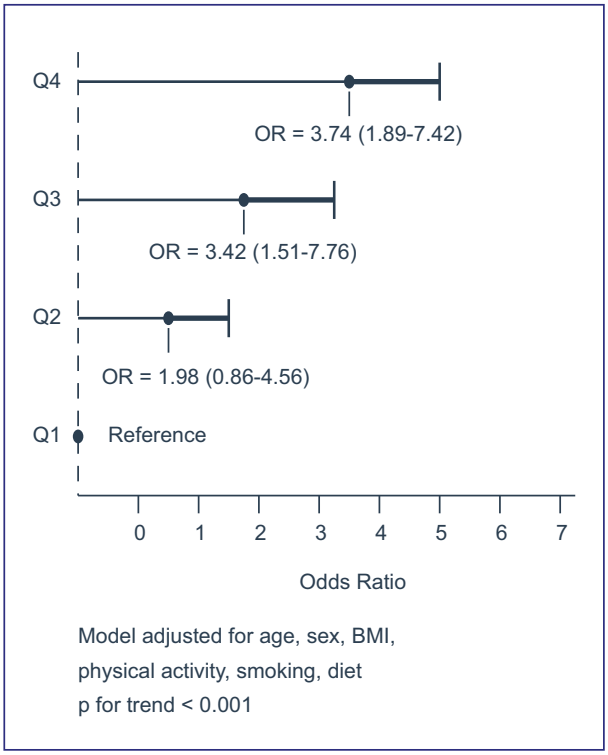

Figure 3: The dose-response relationship showing odds ratios by BPA quartile

Discussion

Our investigation furnishes substantial evidence documenting significant associations between elevated serum BPA concentrations and type 2 diabetes mellitus. Diabetic individuals exhibited approximately double the median serum BPA levels relative to non-diabetic controls, with this relationship persisting following extensive adjustment for conventional diabetes risk determinants. The exposure-response gradient observed across BPA quartiles, culminating in nearly quadrupled diabetes odds within the highest exposure stratum, reinforces causal relationship inference.

Multiple mechanistic pathways support biological plausibility for BPA's diabetes-promoting effects. BPA's endocrine-disrupting characteristics extend well beyond simple estrogenic activity, encompassing significant interference with insulin signaling networks.[19] Laboratory evidence demonstrates BPA's capacity to activate estrogen receptor-á within pancreatic beta cells, initially enhancing insulin secretion yet subsequently triggering oxidative stress, endoplasmic reticulum stress, and programmed cell death under chronic exposure conditions.[9,20] This biphasic phenomenon may elucidate the clinical progression from compensatory hyperinsulinemia toward beta-cell functional exhaustion characteristic of diabetes pathogenesis.

BPA additionally promotes insulin resistance through diverse mechanisms. It disrupts insulin receptor substrate phosphorylation processes, compromises glucose transporter cellular translocation, and activates inflammatory signaling pathways within adipose tissue.[18] BPA exposure stimulates adipocyte differentiation while modifying adipokine secretion profiles, elevating pro-inflammatory cytokine production while suppressing adiponectin release. These adipose tissue alterations contribute systemically to metabolic dysfunction.[8] Furthermore, BPA perturbs hepatic glucose metabolism by interfering with glucokinase expression and amplifying gluconeogenesis, producing elevated fasting glucose concentrations.[21]

Our correlation analyses demonstrating positive BPA relationships with HbA1c, fasting glucose, and HOMA-IR corroborate these mechanistic insights. The

correlation strength observed within both diabetic and control populations suggests BPA's metabolic effects operate continuously across the glycemic spectrum, potentially facilitating transition from normal glucose tolerance through prediabetes toward overt diabetes.[22] This continuum effect carries significant public health implications, suggesting population-level BPA exposure reduction could yield extensive metabolic benefits transcending diabetes prevention alone.

The exposure-response relationship documented in our study merits particular emphasis. The progressive diabetes odds increment across BPA quartiles, maintaining statistical robustness despite rigorous covariate adjustment, strengthens causal inference per Bradford Hill criteria.[23] Critically, BPA concentrations associated with elevated diabetes risk in our investigation fall within ranges routinely detected in general population biomonitoring,[24] suggesting widespread exposure intensities may confer metabolic consequences. This contradicts certain industry contentions that typical human BPA exposures are insufficiently intense to produce adverse effects.

Our sex-stratified analyses revealed comparable BPA-diabetes associations across male and female subgroups, though point estimates suggested marginally elevated female risk.[25] This pattern aligns with BPA's estrogenic properties and sex-specific metabolic vulnerabilities. Estrogen receptor tissue distributions differ between sexes, potentially modulating BPA's metabolic impact.[26] However, absent statistical sex interaction indicates both sexes face diabetes risk from BPA exposure, arguing against sex-differentiated regulatory strategies.

BMI-stratified findings demonstrating BPA associations spanning weight categories challenge reductionist interpretations ascribing metabolic dysfunction exclusively to adiposity.[27] While obesity amplifies BPA effects, significant associations persisted even among normal-weight individuals, indicating BPA contributes independently to diabetes pathogenesis. This observation bears implications for understanding "metabolically unhealthy normal-weight" phenotypes and emphasizes environmental chemicals as underrecognized diabetes risk factors beyond traditional lifestyle considerations.

Literature comparison reveals both concordances and distinctions. Several prior cross-sectional investigations employing urinary BPA detected positive diabetes prevalence associations[9-10,12] though effect magnitudes varied substantially. Our serum BPA measurements may furnish more stable exposure metrics, less susceptible to day-to-day variability characterizing urinary measurements.[15,28] Limited prospective evidence generally supports our findings, with elevated baseline BPA predicting incident diabetes across follow-up intervals.[28]

Our findings' public health implications warrant serious deliberation. Given BPA's ubiquitous environmental presence and robust metabolic disruption evidence[29], population-level exposure reduction represents a viable diabetes prevention approach. Regulatory interventions including infant product BPA prohibitions have occurred across numerous jurisdictions,[30] yet broader restrictions encounter industry resistance. Our data support expanding such restrictions, particularly for food contact applications representing major exposure sources.[31]

Individual exposure reduction strategies merit promotion pending comprehensive regulatory action. Avoiding BPA-containing epoxy-lined canned foods, minimizing plastic food container use, limiting thermal receipt handling, and preferring fresh/frozen over packaged foods can substantially decrease BPA intake.[14] Healthcare providers should incorporate BPA exposure assessment and reduction counseling within diabetes prevention programs, particularly for high-risk populations.

However, replacement chemicals warrant scrutiny. Bisphenol S (BPS) and bisphenol F (BPF), increasingly deployed as BPA substitutes, demonstrate comparable endocrine-disrupting properties in preliminary investigations.[32] This "regrettable substitution" phenomenon underscores comprehensive chemical safety assessment necessity prior to widespread commercial deployment, rather than reactive replacement following harm documentation.[33]

This study has several inherent limitations. Firstly, the cross-sectional design prevents definitive causal determination, as reverse causation, where diabetes alters BPA metabolism, remains theoretically possible.[12] Secondly, single-timepoint BPA quantification may not adequately reflect long-term cumulative exposure due to BPA's brief half-life (6 hours), though repeated temporal measurements were precluded by resource limitations.[28] Thirdly,

unmeasured or residual confounding cannot be entirely excluded despite extensive covariate adjustment, as factors like specific dietary patterns, psychological stress, or other unmeasured environmental chemicals may influence both BPA exposure and diabetes risk;[29] furthermore, exposure assessment lacked detailed source characterization. Fourthly, the study population derived from a single urban hospital setting, potentially limiting generalizability to other geographic regions with varying exposure profiles, dietary habits, and regulatory environments[34]. Fifthly, we were unable to evaluate BPA exposure during critical developmental periods (prenatal, early childhood) which may program adult metabolic dysfunction[35], as retrospective assessment was infeasible. Finally, the analysis focused exclusively on BPA without assessing other bisphenol analogues (BPS, BPF) or other endocrine-disrupting chemicals[7], which may contribute to diabetes risk through mixed, potentially additive or synergistic, chemical exposure impacts.[36]

Conclusion

In conclusion, our study demonstrates that elevated serum Bisphenol A (BPA) concentrations are independently and dose-dependently linked to Type 2 Diabetes Mellitus (T2DM). This association is supported by positive correlations with markers of impaired glucose regulation (HbA1c, fasting glucose) and insulin resistance, confirming BPA's role as a metabolic disruptor.

For clinical recommendations, providers should incorporate BPA exposure reduction into diabetes counseling, emphasizing patient education and promoting fresh, unpackaged food. Counseling should prioritize high-risk groups (e.g., pregnant individuals, prediabetics). Policy actions require expanded BPA restrictions in food packaging, mandatory safety testing for alternatives, and public biomonitoring programs. Future research must focus on prospective cohort studies to establish causality, mechanistic studies to clarify pathways, and the role of prenatal exposure.

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