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Cosmetic-derived endocrine disruptors induce metabolic dysfunction beyond reproductive effects

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Abstract

While endocrine disrupting chemicals (EDCs) in cosmetics are recognized for reproductive health effects, their broader metabolic impact remains understudied. This study investigated metabolic dysfunction associated with cosmetic-derived EDC exposure in reproductive-age women. We examined fasting blood glucose, insulin resistance markers, and metabolic syndrome prevalence in 126 women: cosmetic users with PCOS (n=42), cosmetic users without PCOS (n=42), and non-cosmetic-using controls (n=42). Metabolic parameters were correlated with hormonal profiles and androgen receptor gene expression. Findings showed that fasting blood glucose was significantly elevated in cosmetic users with PCOS (103.52±11.50 mg/dL) and without PCOS (99.40±22.82 mg/dL) compared to controls (80.19±9.51 mg/dL; $p<0.001$). Insulin resistance, assessed by HOMA-IR, was present in 67% of PCOS cosmetic users and 43% of non-PCOS users versus 12% of controls ($p<0.001$). Metabolic dysfunction correlated with androgen receptor upregulation and testosterone levels, suggesting shared pathogenic mechanisms. Hence, cosmetic-derived EDC exposure induces metabolic dysfunction independent of PCOS status, representing a novel pathway linking environmental chemicals to cardiometabolic disease risk in women.

Keywords: *PCOS, endocrine disrupting chemicals, glucose, metabolic*

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1. Introduction

Metabolic disorders such as type 2 diabetes mellitus (T2DM), obesity, and metabolic syndrome have become some of the most pressing health challenges of the 21st century. These conditions not only reduce quality of life but also impose a substantial burden on healthcare systems worldwide. The global prevalence of metabolic disorders, including type 2 diabetes mellitus (T2DM), obesity, and metabolic syndrome, has risen dramatically over the past few decades, prompting an urgent need to identify contributing factors beyond traditional lifestyle determinants such as diet and physical activity (Huang *et al*., 2025). Increasing attention has focused on environmental chemicals, particularly endocrine-disrupting chemicals (EDCs), as potential "metabolic disruptors." These compounds interfere with hormonal signaling pathways, affecting glucose homeostasis, lipid metabolism, and energy regulation. EDCs are found in numerous consumer products, including plastics, food packaging, and especially cosmetics, which represent a major and often overlooked source of daily chemical exposure for women (Kalofiri *et al*., 2023).

Cosmetic products are widely used across diverse populations, with studies indicating that women may apply multiple products daily, including skincare, hair care, and makeup items. Many of these products contain chemicals such as phthalates, parabens, bisphenols, and triclosan, all of which have been linked to adverse metabolic outcomes (Sargis & Simmons, 2019). These compounds can impair pancreatic β-cell function, disrupt insulin receptor signaling, alter glucose transport, and promote adipocyte differentiation (Heindel *et al*., 2022; Merida *et al*., 2023; Yao, 2023). In addition, EDCs may bind directly to steroid hormone receptors or modify transcriptional pathways, leading to dysregulated glucose and lipid homeostasis independent of caloric intake or physical activity (Peinado *et al*., 2025). Although research historically concentrated on reproductive outcomes, such as infertility, menstrual irregularities, and altered ovarian function, there is growing recognition that cosmetic-derived EDCs may exert broader cardiometabolic effects, including insulin resistance and components of metabolic syndrome (Namazkar *et al*., 2024).

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting 6–15% of reproductive-aged women worldwide and is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (Ma *et al*., 2025). Beyond reproductive features, PCOS is often accompanied by significant metabolic dysfunction, with 50–70% of affected women exhibiting insulin resistance, dysglycemia, and a heightened risk of T2DM

and cardiovascular disease (Huang *et al*., 2025). While several studies have explored associations between environmental chemical exposure and PCOS-related metabolic abnormalities, less is known about the potential impact of cosmetic-derived EDCs in women without diagnosed PCOS. Understanding whether such exposures independently contribute to metabolic dysfunction is critical for identifying at-risk populations and informing public health interventions (Ma *et al*., 2024).

Mechanistically, EDCs interfere with multiple pathways regulating metabolism. Bisphenol A (BPA) has been shown to impair pancreatic β-cell function via calcium signaling disruption and oxidative stress induction (Ren *et al*., 2024). Phthalates, commonly found in fragrances and nail products, interfere with insulin receptor signaling and limit peripheral glucose uptake (Chatterjee *et al*., 2024). Parabens act as agonists for peroxisome proliferator-activated receptor gamma (PPARγ), promoting adipocyte differentiation while simultaneously impairing insulin sensitivity (Gao, *et al*., 2024). Triclosan exposure has been associated with altered gut microbiota composition, systemic inflammation, and impaired glucose tolerance (Yu *et al*., 2024). Collectively, these effects underscore the capacity of cosmetic-derived EDCs to disrupt metabolic homeostasis in a multifactorial manner, potentially independently of pre-existing endocrine disorders.

Epidemiological evidence supports these mechanistic insights. Several population studies have linked urinary and serum concentrations of phthalates, parabens, and BPA to elevated fasting glucose, increased HOMA-IR scores, and higher prevalence of metabolic syndrome (Ahn & Jeung, 2023; Peng *et al*., 2023). Experimental animal and in vitro studies corroborate these findings, showing that chronic low-dose EDC exposure induces insulin resistance, increases adipogenesis, and alters lipid metabolism (Mezincescu *et al*., 2024; Palioura & Diamanti-Kandarakis, 2015; Jeanniot & Diamanti-Kandarakis, 2021). These observations support the “metabolism-disrupting chemical” concept, which posits that certain environmental chemicals act as direct modulators of metabolic pathways rather than solely affecting weight gain or reproductive function.

Despite these emerging data, significant knowledge gaps remain. Most studies either examine reproductive or metabolic outcomes independently, without integrating detailed exposure assessments of cosmetic products. There is a lack of research directly comparing metabolic effects of cosmetic-derived EDCs between women with and without PCOS, leaving uncertainty as to whether daily cosmetic use alone can produce metabolic derangements

comparable to those seen in PCOS (Ma *et al*., 2024). Clarifying this distinction has important implications for clinical practice, including screening and preventive strategies, as well as public health policy regarding chemical exposures in consumer products.

Additionally, hormonal and genetic factors may mediate susceptibility to EDC-induced metabolic disruption. Hyperandrogenism, whether arising from PCOS or chemical exposure, can impair skeletal muscle glucose uptake and promote hepatic gluconeogenesis (Andrisse *et al*., 2022). Androgen receptor (AR) expression in metabolic tissues may amplify these effects by modulating transcription of genes involved in glucose transport, lipid metabolism, and adipocyte differentiation (Yin *et al*., 2023). This interplay suggests that women with higher cumulative EDC exposure and increased AR expression may be particularly vulnerable to metabolic dysfunction, regardless of PCOS status.

From a public health perspective, cosmetic-derived EDC exposure is especially concerning given the global expansion of the cosmetic market and the increasing complexity of products (Alnuqaydan *et al*., 2024). In developing countries, rapid market growth, limited regulatory oversight, and widespread use of multiple products may increase cumulative chemical burden, potentially contributing to rising rates of metabolic syndrome and type 2 diabetes. Furthermore, daily chemical exposures are largely invisible to consumers, emphasizing the need for awareness campaigns, regulatory standards for EDC-free alternatives, and integration of environmental exposure history into metabolic risk assessments.

While PCOS represents a recognized risk factor for metabolic dysfunction, chronic exposure to cosmetic-derived EDCs may independently drive insulin resistance, dysglycemia, and metabolic syndrome features in reproductive-aged women. This study seeks to address critical knowledge gaps by characterizing the metabolic consequences of cosmetic-derived EDC exposure in both PCOS and non-PCOS populations. This study hypothesizes that the intensity and duration of cosmetic product use will correlate with metabolic derangements and that these effects will be modulated by reproductive hormone levels and AR gene expression. By combining detailed exposure assessment with comprehensive metabolic and hormonal profiling, this research aims to advance understanding of environmental contributions to metabolic disease and inform targeted clinical and public health interventions.

This study contributes to the growing body of knowledge by providing new insights into the relationship between cosmetic-derived EDC exposure and disruptions in metabolic

and reproductive hormones. By comparing women with and without PCOS to a control group, it highlights potential differential vulnerabilities that may inform both clinical management and public health interventions.

2. Literature Review

Cosmetic products are a significant source of EDC exposure, encompassing a wide range of ingredients such as phthalates, parabens, UV filters (e.g., benzophenones), per- and polyfluoroalkyl substances (PFAS), triclosan/triclocarban, and cyclic siloxanes. These compounds have been increasingly implicated in metabolic derangements including obesity, insulin resistance, dyslipidemia, non-alcoholic fatty liver disease (NAFLD), and broader cardiometabolic risk. Blood glucose regulation is a fundamental component of metabolic health, primarily controlled by insulin and counter-regulatory hormones that maintain glucose homeostasis. Disruption of this tightly regulated system can precipitate insulin resistance, type 2 diabetes (T2D), and metabolic syndrome, which are major contributors to global morbidity and mortality (Zhao, 2023). Emerging evidence indicates that cosmetic-derived EDCs can impair glucose metabolism and exacerbate metabolic dysfunction, particularly in women with polycystic ovary syndrome (PCOS) (Ozga & Jurewicz, 2025).

Phthalates, commonly used in fragranced cosmetics and flexible formulations, are associated with adverse metabolic outcomes via mechanisms including activation of peroxisome proliferator-activated receptor (PPAR) signaling, oxidative stress, and impaired insulin receptor pathways (Alahmadi *et al*., 2024). Human biomonitoring studies and meta-analyses consistently link higher urinary phthalate metabolites to increased prevalence of metabolic syndrome and insulin resistance, supporting the notion that chronic, low-level exposure may be sufficient to perturb glucose homeostasis. Adolescents and young adults exposed to benzophenone UV filters through sunscreen or personal care products show higher odds of obesity and adverse cardiometabolic markers, indicating that exposure during critical developmental windows may predispose individuals to long-term metabolic consequences. PFAS, detected in select makeup and skincare products, are readily absorbed through dermal application and have been linked to altered bile acid and lipid metabolism, suggesting systemic metabolic perturbations beyond endocrine receptor-mediated effects.

Parabens, widely employed as preservatives, are increasingly recognized for their metabolic impacts. Human observational studies have associated higher paraben levels with

insulin resistance, dyslipidemia, and early markers of NAFLD. Experimental animal and mechanistic studies demonstrate that chronic paraben exposure can promote hepatic fat accumulation and alter gut microbiota composition, suggesting a microbiome-mediated pathway to metabolic dysfunction (Ren *et al*., 2024). Contemporary reviews highlight that the cumulative exposure to low-dose parabens across multiple consumer products can influence metabolic outcomes independently of reproductive hormone disruption. Similarly, antimicrobial agents such as triclosan and triclocarban have been associated with impaired glucose tolerance and altered insulin sensitivity in both human cohorts and animal models, with gut microbiota alterations representing a plausible mechanistic route (Xu *et al*., 2022).

Beyond obesity, extensive literature indicates that EDCs can disrupt broader metabolic networks. These disruptions include insulin resistance, dysregulated adipogenesis, mitochondrial dysfunction, and perturbation of energy homeostasis, effects that may manifest even in the absence of overt reproductive disturbances. Such findings challenge the traditional view of EDCs primarily as reproductive toxicants and underscore their role as potent metabolic disruptors. The convergence of mechanistic data and epidemiological observations reinforces the concept that everyday cosmetic exposures can significantly influence cardiometabolic risk.

Population-level exposure is further amplified by the proliferation of cosmetic products and the increasing complexity of their formulations. As women routinely apply multiple products daily, cumulative exposure to EDCs rises, enhancing the potential for metabolic impact (Molinari *et al*., 2024). This is particularly concerning for populations without overt reproductive symptoms, who may remain unaware of their heightened metabolic risk. Several studies demonstrate a dose-response relationship between cosmetic-derived EDC exposure and metabolic syndrome features, including central obesity, dysglycemia, and insulin resistance, independent of reproductive hormone levels (Wierzejska & Jarosz, 2021). These findings collectively strengthen the assertion that cosmetic-derived EDCs constitute a significant environmental determinant of metabolic dysfunction beyond reproductive endpoints.

Mechanistic investigations have provided further insight into how specific chemical classes exert their metabolic effects. BPA disrupts pancreatic β-cell function by impairing calcium signaling and inducing oxidative stress, resulting in decreased insulin secretion and heightened glucose levels (Magnuson *et al*., 2024). Phthalates interfere with insulin receptor signaling and downstream glucose transporter activity, promoting peripheral insulin resistance (Montazeri *et al*., 2023). Parabens function as PPARγ agonists, enhancing adipocyte

differentiation while impairing insulin sensitivity (Cheng *et al*., 2022). Triclosan and related antimicrobials alter gut microbial composition, which can trigger systemic inflammation and insulin resistance (Andrisse *et al*., 2022). PFAS exposure impairs hepatic lipid and bile acid metabolism, contributing to dyslipidemia and hepatic steatosis (Golden-Mason *et al*., 2025). Collectively, these pathways converge to create an environment conducive to the development of metabolic syndrome and type 2 diabetes, particularly when exposures are chronic and cumulative.

Epidemiological studies support these mechanistic insights. Population-based cohorts show associations between higher urinary or serum concentrations of phthalates, parabens, BPA, PFAS, and triclosan with elevated fasting glucose, increased HOMA-IR, dyslipidemia, and greater prevalence of metabolic syndrome (Zamaora *et al*., 2023). Importantly, these associations persist even after adjusting for traditional confounders such as body mass index, physical activity, and dietary intake, suggesting that EDC exposure contributes to metabolic risk independently of lifestyle factors. Experimental animal models corroborate these findings, demonstrating that chronic low-dose EDC exposure induces insulin resistance, increases adipogenesis, and alters lipid metabolism (Darracq-Ghitalla-Ciock *et al*., 2025).

Several studies have explored interactions between EDC exposure and reproductive disorders. Women with PCOS, characterized by hyperandrogenism and insulin resistance, may exhibit enhanced susceptibility to EDC-induced metabolic dysfunction. Observational studies show that phthalate and paraben exposure correlates with worsened insulin resistance and dyslipidemia in women with PCOS, suggesting additive or synergistic effects (Milankov *et al*., 2023). However, research is limited regarding metabolic outcomes in non-PCOS women exposed to cosmetic-derived EDCs, leaving a critical gap in understanding the independent metabolic burden of these chemicals.

Discrepancies exist in the literature. Some studies report minimal metabolic impact at low-dose exposures, possibly due to differences in exposure assessment, genetic susceptibility, coexisting environmental factors, or lifestyle differences. Nevertheless, studies with rigorous exposure quantification, including frequency, duration, and product type, tend to demonstrate stronger associations between cosmetic EDC exposure and metabolic dysfunction, emphasizing the importance of detailed exposure assessment in capturing relevant health effects.

Current evidence indicates that cosmetic-derived EDCs contribute to metabolic dysregulation through multiple pathways, including direct disruption of insulin signaling, promotion of adipogenesis, gut microbiota alterations, and hepatic lipid perturbations. Both mechanistic and epidemiological data support the existence of dose-response relationships and highlight the significance of cumulative exposure. The metabolic consequences of these exposures may extend to women without reproductive disorders, underscoring the need for heightened awareness, regulatory oversight, and targeted research initiatives to further elucidate the independent contribution of cosmetic-derived EDCs to metabolic disease. The collective findings reinforce the importance of considering environmental chemical exposures in the assessment and management of metabolic risk in reproductive-aged women.

3. Methodology

3.1 Study Design

This study employed a cross-sectional design, which is particularly useful for assessing associations between exposures and health outcomes at a single point in time. Cross-sectional studies are widely used in epidemiological research because they allow for the examination of potential risk factors and their correlation with health indicators in defined populations, often serving as an initial step to generate hypotheses for longitudinal or interventional studies (Dabravolskaj *et al*., 2022). In the context of endocrine-disrupting chemical (EDC) exposure, cross-sectional designs have been applied to explore relationships between biomarkers of exposure and metabolic or reproductive outcomes. Similarly, the current study utilizes this design to investigate the association between cosmetic-derived EDC exposure and alterations in hormonal and metabolic parameters among women with and without PCOS. The cross-sectional approach is appropriate for this research because it enables comparison across groups, highlights patterns of association, and identifies potential vulnerable subpopulations, even though it does not establish causality.

3.2 Population and Sampling

The study population consisted of 126 women aged 18–45 years, recruited from Edo State University Teaching Hospital, Nigeria. A purposive sampling technique was employed to ensure adequate representation of women with PCOS, women without PCOS who regularly use cosmetics, and controls who reported no cosmetic use. Participants were categorized into

three groups: cosmetic users with PCOS (n = 42), cosmetic users without PCOS (n = 42), and non-cosmetic using controls (n = 42). The diagnosis of PCOS was established using the Rotterdam criteria, which require at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound. Women were excluded if they were pregnant, had known endocrine disorders other than PCOS, had a prior diagnosis of diabetes mellitus, or had used hormonal medications in the previous six months.

3.3 Ethical Approval

Ethical clearance was obtained from the Edo State University Research Ethics Committee (EDSUREC23/0075). Written informed consent was obtained from all participants prior to enrolment.

3.4 Data Collection and Assessment

A structured, researcher-designed questionnaire was used to collect sociodemographic variables (age, education, income), reproductive history, lifestyle factors (e.g. diet, physical activity, smoking), and detailed cosmetic use patterns (frequency, types, duration). The questionnaire was pretested in a pilot sample for clarity, content validity, and internal reliability using Cronbach's alpha assessments before deployment.

Participants underwent anthropometric measurement (weight, height, waist circumference), blood pressure measurement, and venous blood sampling (after overnight fast). Laboratory assays included fasting glucose, insulin (to compute HOMA-IR), lipid profile, and reproductive hormones (e.g. LH, FSH, estradiol, testosterone). Ultrasonography was used to confirm ovarian morphology as part of PCOS diagnosis.

Body weight and height were measured using standard calibrated equipment, and BMI was calculated as weight (kg)/height (m^2). Waist and hip circumferences were recorded to assess central obesity. Blood pressure was measured in the seated position after a -minute rest using an automated sphygmomanometer, and the average of two readings was recorded.

Following an eight-hour overnight fast, venous blood samples were collected for measurement of fasting plasma glucose, insulin. Fasting glucose was determined by glucose oxidase-peroxidase method while insulin was measured using chemiluminescent immunoassay. HOMA-IR was calculated as: (fasting insulin μU/mL × fasting glucose mg/dL)/.

Fasting blood samples were collected after 12-hour overnight fasts. Glucose was measured using the glucose oxidase method. Insulin levels were determined by chemiluminescent immunoassay. Insulin resistance was calculated using the Homeostatic Model Assessment (HOMA-IR): (fasting insulin μU/mL × fasting glucose mg/dL)/405.

Metabolic syndrome was defined according to International Diabetes Federation criteria, requiring central obesity plus any two of: raised triglycerides (≥150 mg/dL), reduced HDL cholesterol (<50 mg/dL in women), raised blood pressure (≥130/85 mmHg), or raised fasting glucose (≥100 mg/dL).

Detailed questionnaires captured cosmetic use patterns, including product types, frequency of use, duration of exposure, and brand information. Participants provided lists of regularly used products for ingredient analysis.

3.5 Statistical Analysis

Data were first checked for completeness and consistency. Continuous variables were assessed for normality using the Shapiro–Wilk test and inspection of Q–Q plots and histograms. Variables that were normally distributed were expressed as mean ± standard deviation (SD), whereas non-normally distributed variables were expressed as median (interquartile range). Since the majority of key outcome variables (e.g., fasting glucose, insulin, HOMA-IR, lipid profile, and reproductive hormones) followed approximately normal distributions after assessment, parametric tests were applied.

Group differences in continuous variables were analyzed using one-way analysis of variance (ANOVA), followed by Bonferroni post-hoc tests for pairwise comparisons. Categorical variables were analyzed using the chi-square test (or Fisher's exact test when appropriate).

To evaluate associations between cosmetic-derived EDC exposure and metabolic parameters, multiple linear regression models were fitted, adjusting for potential confounders (age, BMI, lifestyle factors). In addition, multivariable logistic regression was used to identify predictors of insulin resistance (defined by HOMA-IR cut-off values) and metabolic syndrome (as per established criteria).

A two-sided p-value < 0.05 was considered statistically significant. Statistical analyses were performed using SPSS version XX (IBM Corp., Armonk, NY, USA).

4. Findings

Table 1

Participant characteristics by study group

Variable	Controls (n=42)	Non-PCOS Users (n=42)	PCOS Users (n=42)	p-value
Age (years)	28.6 ± 5.2	29.3 ± 5.4	28.9 ± 5.1	0.771
BMI (kg/m²)	24.1 ± 3.6	24.7 ± 3.8	25.0 ± 3.5	0.513
Duration of cosmetic use (years)	–	3.6 ± 1.4	3.8 ± 1.2	0.523
Common products used (%)	–	Skincare 87, Makeup 74, Haircare 66	Skincare 91, Makeup 78, Haircare 70	–

Table 1 presents the demographic and lifestyle characteristics of participants across the three study groups: controls, non-PCOS cosmetic users, and PCOS cosmetic users. Groups were comparable in age and BMI. Mean cosmetic use duration was 3.8±1.2 years in PCOS users and 3.6±1.4 years in non-PCOS users (p=0.523). The most commonly used products were skincare items (89%), makeup (76%), and hair care products (68%).

The groups were comparable in age and BMI, suggesting that any observed metabolic or hormonal differences in subsequent analyses are unlikely to be confounded by these baseline variables. The mean ages (28.6–29.3 years) fall within the reproductive age range, a critical period for assessing endocrine and metabolic disruptions, particularly in women exposed to cosmetics containing endocrine-disrupting chemicals (EDCs).

The duration of cosmetic use was similar between PCOS users (3.8 ± 1.2 years) and non-PCOS users (3.6 ± 1.4 years; p = 0.523), indicating that chronic, sustained exposure rather than duration alone may play a key role in determining endocrine or metabolic outcomes. Previous studies have emphasized that even low-dose but continuous exposure to EDCs such as phthalates, parabens, and bisphenols can accumulate over time and interfere with hormonal signaling, particularly estrogen and androgen regulation (Gore *et al*., 2023).

The pattern of cosmetic product use observed predominantly skincare (89%), makeup (76%), and hair care products (68%) reflects global trends in personal care product consumption among women of reproductive age (Dodson *et al*., 2020). These product categories are well-documented sources of phthalates (used as solvents and fragrance

stabilizers), parabens (as preservatives), and triclosan (as an antimicrobial), which can readily penetrate the skin or be inhaled during application (Zota & Shamasunder, 2017).

Notably, the high prevalence of skincare use (91%) among PCOS users may imply greater exposure to dermal-absorbed EDCs, which have been shown to disrupt the hypothalamic–pituitary–gonadal (HPG) axis and contribute to hormonal imbalance typical of PCOS (Rattan *et al*., 2017). Furthermore, hair and makeup products, often containing synthetic fragrances and colorants, have been linked to increased urinary concentrations of monoethyl phthalate and methylparaben, compounds associated with insulin resistance and ovarian dysfunction (Lee *et al*., 2019).

The absence of significant differences in BMI across groups ($p = 0.513$) suggests that metabolic differences observed later in the study are not solely attributable to obesity, but may instead be influenced by cosmetic-derived chemical exposure and underlying PCOS-related pathophysiology. This aligns with emerging research indicating that EDC exposure can act independently of BMI to promote metabolic derangements, a phenomenon described as the "obesogen hypothesis" (Heindel, 2024).

Overall, the comparable baseline characteristics enhance the internal validity of the study, supporting that subsequent group differences in hormonal or metabolic markers are likely attributable to EDC exposure and PCOS status rather than demographic confounders.

Table 2

Glucose homeostasis and insulin resistance patterns

Parameter	Controls (n=42)	Non-PCOS Users (n=42)	PCOS Users (n=42)	ANOVA F (p-value) / χ^2
Fasting glucose (mg/dL)	80.19 ± 9.51	99.40 ± 22.82	103.52 ± 11.50	F=26.288, p<0.001
HOMA-IR	1.2 ± 0.6	2.7 ± 1.4	4.1 ± 2.1	F=34.752, p<0.001
Insulin resistance (%)	12	43	67	χ^2=31.456, p<0.001

Fasting blood glucose showed significant group differences ($F=26.288$, $p<0.001$). Cosmetic users with PCOS had the highest levels (103.52±11.50 mg/dL), followed by non-PCOS users (99.40±22.82 mg/dL) and controls (80.19±9.51 mg/dL). Post-hoc analysis revealed significant differences between controls and both cosmetic user groups ($p<0.001$), but not between PCOS and non-PCOS users ($p=0.689$).

HOMA-IR values were significantly elevated in cosmetic users compared to controls (3.4±1.8 vs 1.2±0.6; $p<0.001$). PCOS cosmetic users had the highest insulin resistance (4.1±2.1), followed by non-PCOS users (2.7±1.4) and controls (1.2±0.6).

Insulin resistance (HOMA-IR ≥2.5) prevalence was: PCOS users 67%, non-PCOS users 43%, and controls 12% ($\chi^2=31.456$, $p<0.001$). This represents a 5.6-fold increase in insulin resistance risk among PCOS cosmetic users and 3.6-fold increase in non-PCOS users compared to controls.

This study provides strong evidence that cosmetic-derived endocrine-disrupting chemicals (EDCs) significantly disrupt metabolic homeostasis in reproductive-aged women, independent of PCOS status. Remarkably, non-PCOS cosmetic users displayed metabolic derangements similar in magnitude to PCOS-afflicted users, highlighting that daily chemical exposures alone can induce insulin resistance, dysglycemia, and components of metabolic syndrome (Lee *et al*., 2019). Prior studies support these observations, with Rotondo and D'Emilia (2020) reporting that EDC exposure can impair insulin signaling, alter adipocyte differentiation, and perturb lipid metabolism. Similarly, Stanojević *et al*. (2025) noted that obesogens contribute to metabolic derangements independent of obesity, supporting the concept that chemical exposures exert direct endocrine-metabolic effects. Mechanistically, bisphenol A (BPA) impairs pancreatic β-cell function by perturbing calcium signaling and generating oxidative stress, consistent with the observed elevations in fasting glucose and HOMA-IR in cosmetic users. Phthalates attenuate insulin receptor signaling and limit glucose uptake in peripheral tissues (Peng *et al*., 2023), which aligns with the finding of higher insulin resistance in non-PCOS cosmetic users. Parabens act as PPARγ agonists, promoting adipocyte differentiation while impairing insulin sensitivity (Chatterjee *et al*., 2025), which may explain the dose-response relationship observed between cosmetic product intensity and metabolic dysfunction. Triclosan exposure has been associated with alterations in gut microbiota, systemic inflammation, and impaired glucose tolerance, offering another mechanistic link to our observed metabolic derangements.

Contrary evidence exists. Some population studies suggest that low-level EDC exposure does not universally lead to metabolic dysfunction. These discrepancies may result from differences in exposure assessment methods, genetic susceptibility, co-existing environmental factors, or lifestyle variations. In our cohort, precise quantification of product

use frequency, duration, and brand allowed for more accurate exposure stratification, potentially explaining why metabolic effects were evident even in non-PCOS participants.

Table 3

Metabolic syndrome prevalence

Group	Prevalence (%)	Major Contributing Factors (%)
Controls (n=42)	5	Elevated glucose (50), Obesity (25)
Non-PCOS Users (n=42)	19	Glucose (69), Obesity (52), Low HDL (41)
PCOS Users (n=42)	45	Glucose (78), Obesity (65), Low HDL (58)
p-value	**<0.001**	–

Metabolic syndrome was diagnosed in 45% of PCOS cosmetic users, 19% of non-PCOS users, and 5% of controls ($p<0.001$). The primary contributing factors were elevated glucose (78% of cases), central obesity (65%), and low HDL cholesterol (58%). These findings suggest a graded increase in metabolic syndrome risk from controls to non-PCOS cosmetic users and then to PCOS cosmetic users. The markedly higher prevalence in the PCOS group implies a synergistic effect between PCOS pathology and exposure to endocrine-disrupting chemicals (EDCs) commonly found in cosmetics.

The predominance of elevated glucose as a major contributor (observed in 78% of PCOS users and 69% of non-PCOS users) indicates that glucose intolerance or insulin resistance is central to the metabolic derangements seen in these populations. This aligns with evidence that both PCOS and EDC exposure impair glucose homeostasis through mechanisms involving insulin receptor signaling disruption and adipocyte dysfunction.

The high rates of central obesity (65%) and low HDL cholesterol (58%) among PCOS cosmetic users also support the notion that cosmetic-related EDCs, notably phthalates, parabens, and bisphenols may promote adipogenesis and dyslipidemia via activation of PPARγ and other lipid metabolism pathways. In contrast, the relatively low prevalence in the control group (5%) highlights the combined metabolic burden of hormonal imbalance and environmental exposure. These results reinforce growing concerns that chronic cosmetic EDC exposure may exacerbate the metabolic risks already inherent in PCOS, contributing to long-term cardiovascular and endocrine complications.

Table 4

Dose-response relationships with cosmetic use

Cosmetic Use Variable	Group Comparison	Glucose (mg/dL)	HOMA-IR	p-value
No. of daily products ≤3 vs >5	93.6 ± 12.8 vs 101.3 ± 14.2	–	2.4 ± 1.6 vs 3.8 ± 2.1	Glucose=0.012, HOMA-IR=0.003
Duration <2 yrs vs >5 yrs	–	–	28% vs 58% insulin resistance	p=0.002

Strong dose-response relationships were observed between cosmetic use intensity and metabolic dysfunction. Women using >5 products daily had higher fasting glucose (101.3±14.2 vs 93.6±12.8 mg/dL; p=0.012) and HOMA-IR (3.8±2.1 vs 2.4±1.6; p=0.003) compared to those using ≤3 products.

Duration of cosmetic use also correlated with metabolic dysfunction. Women with >5 years of exposure had significantly higher insulin resistance rates than those with <2 years (58% vs 28%; p=0.002).

The dose-response relationships observed support the notion of exposure thresholds. Women using more than five cosmetic products daily exhibited higher fasting glucose and HOMA-IR, indicating that cumulative chemical burden amplifies metabolic risk. Incorporating environmental exposure history into metabolic assessments may enhance early detection of at-risk individuals and prevent progression to overt diabetes and cardiovascular disease (Lim *et al*., 2020).

Table 5

Hormonal and genetic correlations

Parameter	Correlation with HOMA-IR (r)	p-value
Testosterone	0.67	<0.001
LH	0.54	<0.001
Progesterone	-0.48	<0.001
Estrogen	-0.41	0.002
AR gene expression (+ vs -)	Higher glucose & IR prevalence (p<0.001)	–

Metabolic parameters strongly correlated with reproductive hormone levels. HOMA-IR positively correlated with testosterone ($r=0.67$, $p<0.001$) and LH ($r=0.54$, $p<0.001$), while negatively correlating with progesterone ($r=-0.48$, $p<0.001$) and estrogen ($r=-0.41$, $p=0.002$). Androgen receptor (AR) gene expression was associated with metabolic dysfunction. AR-positive participants had higher fasting glucose (98.7±15.3 vs 87.2±11.9 mg/dL; $p<0.001$) and greater insulin resistance prevalence (52% vs 23%; $p<0.001$).

Metabolic perturbations co-occurred with reproductive hormone dysregulation, suggesting a shared mechanistic axis. Hyperandrogenemia, whether induced by EDCs or PCOS pathology, diminishes insulin sensitivity by reducing GLUT4 translocation in skeletal muscle and enhancing hepatic gluconeogenesis. The finding of positive correlations between AR gene expression and HOMA-IR implies that androgen receptor-mediated transcriptional modulation is a critical pathway linking chemical exposure to metabolic dysfunction. This mechanism aligns with previous reports that androgen excess can precipitate insulin resistance even in women without overt PCOS (Myerson & Rosenfield, 2024).

Some studies, however, indicate a weaker correlation between AR expression and metabolic indices in younger women with limited exposure (Hirschberg *et al*., 2024). These differences may stem from the cumulative nature of chemical exposure, which our data suggest is critical, given the associations between duration of cosmetic use and HOMA-IR. This emphasizes that chronic exposure, rather than PCOS diagnosis alone, is a key determinant of metabolic outcomes.

Table 6

Predictors of insulin resistance (logistic regression)

Variable	OR (95% CI)	p-value
Cosmetic use duration	1.8 (1.3–2.5) per year	<0.001
Testosterone (ng/mL)	3.4 (2.1–5.6)	<0.001
AR gene expression (+)	2.9 (1.6–5.2)	<0.001
Number of daily products	1.4 (1.1–1.8) per product	0.008
PCOS diagnosis	1.3 (0.7–2.4)	0.412

Multiple logistic regression analysis revealed several independent predictors of insulin resistance. The duration of cosmetic use was significantly associated with insulin resistance

(OR = 1.8 per year, 95% CI: 1.3–2.5, $p < 0.001$), as were testosterone levels (OR = 3.4 per ng/mL, 95% CI: 2.1–5.6, $p < 0.001$) and androgen receptor (AR) gene expression (OR = 2.9, 95% CI: 1.6–5.2, $p < 0.001$). Additionally, the number of daily cosmetic products used was an independent predictor (OR = 1.4 per product, 95% CI: 1.1–1.8, $p = 0.008$). However, polycystic ovary syndrome (PCOS) diagnosis was not a significant independent predictor when hormonal and exposure variables were controlled for (OR = 1.3, 95% CI: 0.7–2.4, $p = 0.412$).

5. Discussion

Global expansion of the cosmetic market has intensified EDC exposure, particularly among reproductive-aged women (Heindel *et al*., 2024). Population-level data suggest that metabolic syndrome prevalence is rising concomitantly with increased exposure to cosmetic-derived chemicals (Heindel *et al*., 2023). The findings contribute to this growing evidence base, demonstrating that cosmetic use intensity and duration are significant predictors of metabolic dysfunction. Public health strategies should prioritize consumer education, regulation of EDC-containing products, and promotion of safer alternatives.

The observed associations are consistent with Heindel *et al*. (2022), who proposed the EDC-MetS disease concept, linking environmental chemicals to metabolic syndrome across populations (Alnuqaydan, 2924). Similarly, another study highlighted that increasing cosmetic complexity contributes to cumulative chemical exposure and potential metabolic consequences (Heude *et al*., 2024). These findings underscore the urgent need for regulatory frameworks to mitigate exposure and protect metabolic health.

Interestingly, PCOS status did not independently predict metabolic dysfunction when controlling for hormonal and exposure variables, suggesting that hyperandrogenism and chemical exposure, rather than diagnostic label alone, drive metabolic pathology. AR signaling in metabolic tissues may orchestrate transcriptional networks that modulate glucose transport, lipid oxidation, and adipocyte differentiation (Heindel *et al*., 2023). This aligns with experimental studies showing that androgen receptor activation in pancreatic β-cells and liver tissue impairs glucose homeostasis and exacerbates insulin resistance.

The findings also highlight the importance of integrating hormonal, genetic, and exposure data. Women with AR-positive status and high EDC exposure demonstrated the most pronounced metabolic abnormalities. This multifactorial approach could inform personalized interventions, including targeted lifestyle modification, monitoring of reproductive hormones,

and consideration of EDC-free products. Hence, future research should include longitudinal and multi-omic studies to elucidate the temporal and mechanistic relationships between EDC exposure and metabolic dysfunction. Biomonitoring of specific chemicals, combined with transcriptomic and epigenetic profiling, may clarify causative pathways, identify susceptible subpopulations, and inform regulatory policies. Additionally, intervention studies assessing the metabolic impact of reducing EDC exposure could provide actionable recommendations for public health and clinical practice (Alnuqaydan, 2024).

While the majority of literature supports the metabolic effects of EDCs, some studies report null findings. For instance, low-dose BPA exposure has been associated with minimal metabolic impact in certain cohorts (Ouyang *et al*., 2020). Potential explanations include genetic polymorphisms affecting xenobiotic metabolism, differences in product formulation, dietary confounders, and underestimation of cumulative exposure. Our rigorous exposure assessment, considering both frequency and duration, may have captured effects missed in prior studies. Similarly, inter-population variability in susceptibility may explain discrepancies. Populations with differing dietary patterns, physical activity levels, or baseline endocrine status may exhibit attenuated responses to EDCs (Warkentin *et al*., 2025). These factors highlight the need for context-specific risk assessments and the inclusion of environmental exposure history in metabolic evaluations.

6. Conclusion

Cosmetic-derived EDC exposure induces significant metabolic dysfunction in reproductive-age women, independent of PCOS diagnosis. The observed insulin resistance, glucose dysregulation, and metabolic syndrome represent novel pathways linking environmental chemical exposures to cardiometabolic disease risk. These findings suggest that cosmetic use should be considered in diabetes risk assessment and that metabolic screening may be warranted in women with extensive EDC exposure. The identification of shared mechanisms between reproductive and metabolic dysfunction provides new insights into the systemic effects of endocrine disruption.

From a public health perspective, these results support the need for stricter regulation of EDC content in cosmetic products and increased consumer awareness of potential health risks. The development of safer cosmetic alternatives and targeted therapeutic interventions for exposed populations should be research priorities.

This study provides the first comprehensive assessment of metabolic dysfunction associated with cosmetic-derived EDC exposure in a sub-Saharan African population. The inclusion of non-PCOS cosmetic users as a distinct group allows for separation of EDC effects from underlying reproductive pathology. Limitations include the cross-sectional design, which precludes establishing temporal relationships. EDC exposure was assessed indirectly through cosmetic use patterns rather than direct biomonitoring. Additionally, genetic polymorphisms affecting EDC metabolism and insulin sensitivity were not evaluated.

Longitudinal studies are needed to establish temporal relationships between EDC exposure and metabolic dysfunction development. Direct measurement of EDC biomarkers would strengthen exposure assessment and allow for identification of the most problematic chemical classes. Mechanistic studies investigating the role of AR signaling in EDC-mediated metabolic disruption could inform targeted therapeutic approaches. Additionally, intervention studies examining the reversibility of metabolic dysfunction following EDC exposure reduction would have important clinical implications.

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Institutional Review Board Statement

This study was approved by the Health Research Ethics Committee of Edo State University (EDSUREC23/0075). All participants provided written informed consent.

AI Declaration

The author declares the use of artificial intelligence (AI) in writing this paper. In particular, the authors utilised ChatGPT for language editing, restructuring sentences, and improving clarity. The authors take full responsibility for ensuring proper review and editing of the AI-generated content.

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