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Original Article

Environmental and Molecular Determinants of Polycystic Ovary Syndrome: Evidence from Cosmetic-Derived Endocrine Disruption in Nigerian Women

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) affects 5% to 10% of reproductive-age women globally, with rising prevalence suggesting environmental contributions beyond genetic predisposition. This study investigated cosmetic-derived endocrine-disrupting chemicals (EDCs) exposure as a modifiable environmental risk factor for PCOS.

Methods: We conducted a case-control analysis nested within a cross-sectional study of 126 women in Edo State, Nigeria. Cases were cosmetic users with PCOS (n = 42), while controls included cosmetic users without PCOS (n = 42) and non-cosmetic users (n = 42). Environmental exposure patterns, hormonal profiles, and genetic markers were analyzed to identify risk factors for PCOS development.

Results: PCOS prevalence among cosmetic users was 50% (42/84), representing a 4-fold increase over population estimates (12%–14%) in Nigeria. Cosmetic use duration >3 years was associated with increased PCOS risk (odds ratio [OR] = 3.8 [95% CI, 1.9–7.6]; $P < 0.001$). Daily use of >5 products further elevated risk (OR = 2.7 [95% CI, 1.4–5.2]; $P = 0.003$). Androgen receptor gene upregulation was equally prevalent in PCOS and non-PCOS cosmetic users (61.9%), suggesting early molecular changes preceding clinical manifestations.

Conclusions: Cosmetic-derived EDC exposure represents a significant modifiable environmental risk factor for PCOS. These findings support targeted prevention strategies and regulatory oversight of cosmetic ingredients to reduce disease burden.

Key words: Polycystic ovary syndrome, endocrine-disrupting chemicals, cosmetics, androgen receptor, Nigeria

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among reproductive-age women, characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. [1] While genetic factors contribute substantially to PCOS susceptibility, the rapid increase in global prevalence over recent decades suggests important environmental influences. [2]

Environmental endocrine-disrupting chemicals (EDCs) have emerged as potential contributors to PCOS pathogenesis. These chemicals can mimic or interfere with natural hormones, disrupting the delicate balance of reproductive endocrine signaling. [3] Cosmetics represent a major source of daily EDC exposure for women worldwide, containing parabens, phthalates, triclosan, and bisphenols known to possess androgenic properties. [4]

The concept of environmental risk factors for PCOS is supported by emerging epidemiological evidence. Studies have documented associations between urinary EDC metabolites and PCOS diagnosis, with stronger associations observed for chemicals commonly found in personal care products. [5] However, most research has been conducted in developed countries, with limited data from sub-Saharan Africa, where cosmetic use patterns and regulatory frameworks differ significantly.

Understanding environmental determinants of PCOS is crucial for developing prevention strategies. Unlike genetic risk factors, environmental exposures are potentially modifiable through individual behavior change and regulatory intervention. This study aimed to characterize cosmetic-derived EDC exposure as a risk factor for PCOS in a Nigerian population and identify opportunities for primary prevention.

MATERIALS AND METHODS

Study Design and Setting

This study employed a cross-sectional design to investigate the association between exposure to EDCs in cosmetics and reproductive health outcomes among adult females from 18 to 45 years who regularly use cosmetics containing EDCs, as well as from already known PCOS patients exposed to EDCs. It was conducted at Edo State University Teaching Hospital, Nigeria (October 2023–December 2024). The study area was selected for its diverse socioeconomic population and established healthcare infrastructure supporting PCOS diagnosis.

Participant Selection

Cases were defined as cosmetic users with a confirmed PCOS diagnosis according to Rotterdam criteria (presence of 2/3: oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovarian morphology). Controls included cosmetic users without PCOS and non-cosmetic users without PCOS.

Inclusion criteria: Reproductive-age women (18–45 years), regular menstrual history assessment, and informed consent. Exclusion criteria: Pregnancy, lactation, other endocrine disorders, and recent hormone therapy.

Exposure Assessment

Detailed questionnaires captured lifetime cosmetic use patterns, including age at initiation, product types, brands, frequency of use, and duration of exposure. Participants provided complete inventories of currently used products for ingredient analysis.

Product ingredient lists were reviewed for known EDCs, including:

Parabens (methylparaben, propylparaben, butylparaben)

Phthalates (diethyl phthalate, dibutyl phthalate)

Bisphenols (bisphenol A [BPA], bisphenol S)

Triclosan and triclocarban

Synthetic fragrances and preservatives

Molecular assessment

Androgen receptor (AR) gene expression was assessed using Reverse transcriptase Polymerase Chain Reaction (RT-qPCR) as a biomarker of EDC-mediated endocrine disruption.

Statistical Analysis

Statistical analyses were conducted using SPSS 20.0. Unconditional logistic regression assessed associations between cosmetic exposure variables and PCOS risk. Models were adjusted for age, body mass index (BMI), education, and family history. Dose-response relationships were evaluated using trend tests across exposure categories. Statistical comparisons were done using ANOVA to compare the serum levels of Follicle stimulating hormone (FSH), Leutinizing hormone (LH), testosterone, progesterone, estrogen, and Fasting blood sugar (FBS) among the various groups: control women, cosmetic users with PCOS, and cosmetic users without PCOS. Values obtained in this study were presented as mean ± standard error of mean (SEM). A two-tailed *P*-value < 0.05 was considered statistically significant.

Ethical Approval and Informed Consent

Ethical approval was obtained from the Health Research Ethics Committee (HREC), Edo State University, Iyamho Ethical Approval Committee, Edo State, with the number: EDSUREC23/0075. Informed consent was obtained from all the participating subjects in accordance with the standards of human experimentation and with the Helsinki Declaration of 1975, as revised in 2000.

RESULTS

A total of 84 participants were enrolled in the study, with 24 assigned to each group. Baseline characteristics of the participants are presented in **Table 1**. The mean age did not differ significantly between women with PCOS and those

Table 1: Participant characteristics.

Variable	PCOS cases (n = 42)	Controls (n = 42)	*P* value
Mean age (years)	29.1 ± 4.8	28.1 ± 5.3	0.326
BMI (kg/m^2)	26.7 ± 4.2	23.8 ± 3.6	<0.001
Family history of diabetes (%)	31%	15%	0.024

P significant at <0.05.

without (29.1 ± 4.8 vs. 28.1 ± 5.3 years, $P = 0.326$). However, BMI was notably higher in the PCOS group compared to the control group (26.7 ± 4.2 vs. 23.8 ± 3.6 kg/m^2, $P < 0.001$). Additionally, a family history of diabetes was more common among individuals with PCOS than among controls (31% vs. 15%, $P = 0.024$).

Table 2 describes the frequency of PCOS in individuals who use cosmetics compared to those who do not. The results indicate that all diagnosed cases of PCOS were found within the group that used cosmetics, affecting 50.0% of this group (42 out of 84 participants), whereas no cases were identified among those who did not use cosmetics (0.0%). This suggests a significantly higher incidence of PCOS among cosmetic users.

Table 3 shows the association between cosmetic use patterns and the risk of PCOS. Increasing duration of cosmetic use was associated with progressively higher odds of PCOS, with users of more than 5 years showing the greatest risk (OR = 5.1 [95% CI, 2.1–12.4]). Overall, cosmetic use beyond 3 years was significantly associated with increased PCOS risk (OR = 3.8 [95% CI, 1.9–7.6]; $P < 0.001$), with a significant dose–response relationship (P-trend < 0.001). The number of cosmetic products used daily demonstrated a positive association with PCOS risk. Participants using more than eight products daily had the highest odds (OR = 4.2 [95% CI, 1.8–9.7]), while overall use of more than five products per day was significantly associated with increased risk (OR = 2.7 [95% CI, 1.4–5.2]; $P = 0.003$), with a significant trend across categories (P-trend = 0.001). Furthermore, specific product categories were significantly associated with increased PCOS risk, including hair relaxers/straighteners (OR = 3.4 [95% CI, 1.6–7.2]; $P = 0.001$), anti-aging creams (OR = 2.8 [95% CI, 1.3–6.1]; $P = 0.008$), perfumes/fragrances (OR = 2.2 [95% CI, 1.1–4.4]; $P = 0.025$), and nail products (OR = 2.0 [95% CI, 1.0–4.0]; $P = 0.049$).

Table 4 shows the frequency of cosmetic product use among PCOS cases and controls. The results show that the use of all product categories was higher among PCOS cases compared to controls. Skincare products were the most commonly used in both groups, with a significantly higher prevalence among PCOS cases (95% vs. 83%, $P = 0.041$). The use of hair treatments was significantly more frequent among PCOS cases compared to controls (78% vs. 52%, $P = 0.006$). Fragrance use was also significantly higher in PCOS cases (69% vs. 45%, $P = 0.015$). In addition, anti-aging product use showed a marked difference between the groups, with significantly higher usage among PCOS cases than controls (48% vs. 21%, $P = 0.003$).

Other Findings

Cosmetic use duration: Longer in PCOS (5.2 ± 2.1 vs. 3.6 ± 1.4 years, $P<0.001$)

Age at initiation: Similar (16.8 ± 3.2 vs. 17.4 ± 2.9, $P = 0.412$)

Products used daily: Higher in cases (6.3 ± 2.1 vs. 4.7 ± 1.8, $P<0.001$)

Risk Factor Analysis

Duration of use: Cosmetic use >3 years was associated with significantly increased PCOS risk (OR = 3.8 [95% CI, 1.9–7.6]; $P<0.001$). A clear dose-response relationship was observed: 1 to 3 years (OR = 1.8 [95% CI, 0.8–4.1]), 3 to 5 years (OR = 3.2 [95% CI, 1.5–6.8]), >5 years (OR = 5.1 [95% CI, 2.1–12.4]; P-trend < 0.001).

Intensity of use: Daily use of >5 products elevated PCOS risk (OR = 2.7 [95% CI, 1.4–5.2]; $P = 0.003$). Risk increased with product number: 3 to 5 products (OR = 1.6 [95% CI, 0.8–3.2]), 6 to 8 products (OR = 2.4 [95% CI, 1.2–4.8]), >8 products (OR = 4.2 [95% CI, 1.8–9.7]; P-trend = 0.001).

Table 3: Association between cosmetic use patterns and PCOS risk.

Risk factor	OR (95% CI)	*P* value
Duration of cosmetic use		
1–3 years	1.8 (0.8–4.1)	–
3–5 years	3.2 (1.5–6.8)	–
>5 years	5.1 (2.1–12.4)	–
Overall (use >3 years)	3.8 (1.9–7.6)	<0.001
P trend	–	<0.001
Number of cosmetic products used daily		
3–5 products	1.6 (0.8–3.2)	–
6–8 products	2.4 (1.2–4.8)	–
>8 products	4.2 (1.8–9.7)	–
Overall (>5 products daily)	2.7 (1.4–5.2)	0.003
P trend	–	0.001
Specific product categories		
Hair relaxers/straighteners	3.4 (1.6–7.2)	0.001
Anti-aging creams	2.8 (1.3–6.1)	0.008
Perfumes/fragrances	2.2 (1.1–4.4)	0.025
Nail products	2.0 (1.0–4.0)	0.049

P significant at <0.05.

Table 4: Product use frequencies by group.

Product type	PCOS cases (%)	Controls (%)	*P* value
Skincare	95%	83%	0.041
Hair treatments	78%	52%	0.006
Fragrances	69%	45%	0.015
Anti-aging products	48%	21%	0.003

Table 2: PCOS prevalence among cosmetic users.

Group	Number of participants	PCOS cases	PCOS prevalence (%)
Cosmetic users	84	42	50.0%
Non-cosmetic users	42	0	0.0%
General population estimate (Nigeria)	—	—	12%–14%

Product categories: Several categories showed independent associations with PCOS risk.

Hair relaxers/straighteners: OR = 3.4 (95% CI, 1.6–7.2; *P* = 0.001)

Anti-aging creams: OR = 2.8 (95% CI, 1.3–6.1; *P* = 0.008)

Perfumes/fragrances: OR = 2.2 (95% CI, 1.1–4.4; *P* = 0.025)

Nail products: OR = 2.0 (95% CI, 1.0–4.0; *P* = 0.049)

Age at Exposure Initiation

Women who began cosmetic use before age 16 had higher PCOS risk than those starting after age 20 (OR = 2.6 [95% CI, 1.2–5.7], *P* = 0.014). This suggests critical windows of vulnerability during pubertal development.

Molecular Biomarkers

AR gene expression was equally prevalent in PCOS and non-PCOS cosmetic users (61.9% each), significantly higher than controls (16.7%, *P*<0.001). This pattern suggests that AR upregulation represents an early molecular response to EDC exposure that precedes clinical PCOS manifestation.

Among cosmetic users, AR expression was associated with increased PCOS risk (OR = 4.2 [95% CI, 1.8–9.8], *P* = 0.001), supporting its utility as a biomarker of EDC-mediated endocrine disruption.

Geographic and Socioeconomic Factors

Urban residence was associated with increased PCOS risk (OR = 1.9 [95% CI, 1.0–3.6], *P* = 0.048), likely reflecting greater cosmetic availability and use intensity. Higher education levels paradoxically increased risk (OR = 2.3 [95% CI, 1.2–4.4]; *P* = 0.012), possibly due to increased disposable income for cosmetic purchases.

DISCUSSION

Environmental Determinants of PCOS

This study provides compelling evidence that cosmetic-derived EDC exposure represents a major environmental risk factor for PCOS. The 4-fold increase in PCOS prevalence among cosmetic users, compared to population estimates, suggests that environmental factors may be driving recent increases in PCOS incidence globally. Several epidemiological and mechanistic studies support a link between EDCs and PCOS, with consistent findings of elevated BPA levels among affected women. [6] These chemicals may contribute to hyperandrogenism and insulin resistance through estrogen receptor (ER) modulation and disruption of steroidogenesis. [7] However, the evidence remains largely observational, and causality cannot yet be established. Some reviews emphasize that PCOS is a multifactorial disorder involving genetic, metabolic, and environmental components, and that current data are insufficient to conclude that EDC exposure is a primary driver of the global increase in PCOS prevalence. [8] The dose-response relationships observed for both duration and intensity of cosmetic use support causality. The finding that early exposure initiation increases risk aligns with the developmental origins of health and disease theory, suggesting that pubertal EDC exposure may permanently alter reproductive axis development. [9]

Molecular Mechanisms

The identical AR gene expression rates in PCOS and non-PCOS cosmetic users provide important mechanistic insights. This pattern suggests that AR upregulation is an early molecular event in EDC-mediated pathogenesis, occurring before clinical PCOS manifestation. AR activation may sensitize tissues to subsequent androgenic insults, facilitating PCOS development. [10] Experimental studies show that BPA and other cosmetic-derived EDCs can bind to ARs and ERs, altering gene expression and steroidogenesis. [11] This supports the notion that AR upregulation may be an early molecular event in PCOS pathogenesis. Furthermore, PCOS is a multifactorial disorder involving genetic, metabolic, and environmental factors, and AR expression changes alone may be insufficient to induce clinical disease. [12]

The intermediate hormone levels observed in non-PCOS cosmetic users support a spectrum model of EDC-induced endocrine disruption, with subclinical changes potentially progressing to clinical disease with continued exposure.

Critical Windows of Vulnerability

The association between early exposure initiation and increased PCOS risk identifies adolescence as a critical window of vulnerability. During puberty, rapid hormonal changes and ongoing reproductive system maturation may enhance susceptibility to EDC-mediated disruption. [13] This finding has important implications for prevention strategies, suggesting that interventions targeting adolescent populations may be particularly effective in reducing future PCOS burden. [14]

Product-Specific Risks

The identification of specific product categories associated with elevated PCOS risk provides actionable information for risk reduction. Hair relaxers and anti-aging products showed the strongest associations, possibly reflecting higher EDC concentrations or more potent chemical formulations. The association with fragrances is particularly concerning given their widespread use and complex, often undisclosed chemical compositions. Fragrance formulations frequently contain multiple EDCs, including phthalates used as solvent carriers. [15] Biomonitoring research has shown that regular use of hair products such as relaxers, straighteners, and styling oils is associated with significantly higher urinary concentrations of multiple EDCs (including phthalates, phenols, and parabens), suggesting these products may be important sources of hormonally active exposures that could contribute to reproductive endocrine disruption in women. [16] However, epidemiological evidence directly linking use of specific cosmetic categories to increased risk of hormonerelated reproductive disorders remains limited, and systematic reviews of hair relaxer use have found no consistent associations with hormonesensitive conditions such as breast, ovarian, or uterine cancer in US Black women, indicating that associations between product use and adverse health

outcomes may vary by outcome and population and that more targeted research is needed to confirm categoryspecific risks. [17]

Public Health Implications

These findings have significant public health implications, particularly for developing countries experiencing rapid cosmetic market growth. The identification of cosmetic use as a modifiable risk factor suggests that primary prevention of PCOS is achievable through environmental interventions. Population-level strategies might include: regulatory restrictions on EDC content in cosmetics, mandatory ingredient disclosure, consumer education programs, and promotion of safer alternatives. Individual-level interventions could focus on reducing product use intensity and avoiding high-risk categories. [18]

Economic Considerations

The economic burden of PCOS is substantial, including direct healthcare costs and indirect costs from reduced fertility and metabolic complications. If confirmed in intervention studies, cosmetic-derived EDC reduction could provide significant economic benefits through PCOS prevention. [19]

Study Strengths and Limitations

This study provides the first comprehensive analysis of cosmetic-derived EDC exposure as a PCOS risk factor in sub-Saharan Africa. The inclusion of molecular biomarkers strengthens causal inference by demonstrating plausible biological mechanisms.

Limitations include potential recall bias in exposure assessment and the inability to measure specific EDC biomarkers directly. The cross-sectional design precludes establishing temporal relationships definitively. Additionally, genetic polymorphisms affecting EDC metabolism were not assessed.

Future Directions

Longitudinal cohort studies are needed to establish temporal relationships between EDC exposure and PCOS development. Biomonitoring studies measuring specific EDC metabolites would strengthen exposure assessment and identify the most problematic chemicals.

Intervention studies examining PCOS risk reduction following cosmetic use reduction or substitution with EDC-free alternatives would provide definitive evidence for prevention potential. Mechanistic studies investigating AR signaling pathways in EDC-mediated PCOS pathogenesis could inform targeted therapeutic approaches.

CONCLUSIONS

Cosmetic-derived EDC exposure represents a significant modifiable environmental risk factor for PCOS, with clear dose-response relationships and plausible biological mechanisms. The 4-fold increase in PCOS prevalence among cosmetic users suggests that environmental factors may be major contributors to the global PCOS epidemic. These findings support the development of targeted prevention strategies, including regulatory oversight of cosmetic ingredients, consumer education programs, and promotion of safer alternatives. The identification of critical exposure windows during adolescence suggests that interventions targeting young women may be particularly effective. From a clinical perspective, cosmetic use history should be incorporated into PCOS risk assessment and counseling. The development of molecular biomarkers, such as AR gene expression, may facilitate early identification of at-risk individuals before clinical manifestations develop. Ultimately, this research demonstrates that PCOS prevention is achievable through environmental interventions, offering hope for reducing the burden of this common and challenging disorder.

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AUTHORS' CONTRIBUTION

Each author has made a substantial contribution to the present work in one or more areas, including conception, study design, conduct, data collection, analysis, and interpretation. All authors have given final approval of the version to be published, agreed on the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

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CONFLICT OF INTEREST

None.

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