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Research Article

Association of Organochlorine Pesticide Exposure with Dermatological Manifestations in Women with Polycystic Ovary Syndrome: A Study from North India

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Abstract

Objective: To investigate the association between organochlorine pesticide (OCP) exposure and dermatological manifestations in women with polycystic ovary syndrome (PCOS) compared to non-PCOS women in North India.

Methods: A prospective cohort study was conducted at S.N. Medical College, Agra, from January 2022 to December 2024, involving 220 women (110 PCOS, 110 nonPCOS) aged 18 - 40 years. PCOS was diagnosed using Rotterdam criteria. OCP exposure was assessed via a questionnaire on residential, occupational, and dietary history, pesticides levels quantified by gas chromatography-mass spectrometry (GC-MS) analysis of serum samples (ng/mL). Dermatological manifestations, including acne (Global Acne Grading System), hirsutism (Ferriman-Gallwey score), acanthosis nigricans, and alopecia (Ludwig scale), were evaluated by a blinded dermatologist. Statistical analysis done using Chi-square, t-tests, and logistic regression (adjusted for age, BMI).

Results: PCOS women exhibited 31.7 - 72.7% higher serum OCP levels ($p < 0.001$) across pesticide groups (cyclodienes, endosulfan, DDT derivatives, BHC isomers). Dermatological manifestations were significantly more prevalent in PCOS (acne: 63.6% vs. 9.1%; hirsutism: 43.6% vs. 0%; acanthosis nigricans: 42.7% vs. 0%; $p < 0.001$). OCP levels positively correlated with dermatological severity ($r = 0.51$, $p < 0.01$).

Conclusion: Elevated OCP exposure is strongly associated with aggravated dermatological manifestations in PCOS, emphasizing the need for environmental risk assessment in clinical management.

Keywords: *Polycystic Ovary Syndrome; Organochlorine Pesticides; Dermatological Manifestations in PCOS; Acne; Hirsutism; Endocrine Disruptors*

Introduction

Polycystic ovary syndrome (PCOS), affecting 5 - 10% of reproductive-aged women, is a prevalent endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology [1]. Its dermatological manifestations-acne, hirsutism, acanthosis nigricans, and alopecia-significantly impair quality of life and are driven by elevated androgens and insulin resistance [2]. While environmental endocrine-disrupting chemicals, such as organochlorine pesticides (OCPs), are hypothesized to exacerbate these features, critical gaps persist in understanding their role in PCOS pathogenesis, particularly in high-exposure regions like North India [3].

Despite evidence linking OCPs to PCOS risk [7,8], prior studies have primarily focused on metabolic or reproductive outcomes, neglecting dermatological severity-a key determinant of psychosocial burden in PCOS [2]. Moreover, no study has evaluated urban-rural gradients in OCP exposure and their differential impact on dermatological manifestations, despite stark variations in agricultural vs. industrial pesticide use [6]. OCPs, including DDT derivatives, BHC isomers, endosulfan, and cyclodienes, are persistent organic pollutants that interfere with steroidogenesis and insulin signalling [4,5]. Mechanistically, OCPs may exacerbate hyperandrogenism by activating androgen receptors (AR) in sebocyte and hair follicles, promoting lipogenesis and terminal hair growth [9], while also inducing insulin resistance via IRS-1 serine phosphorylation [14]. Recent studies further suggest that OCPs alter DNA methylation of genes regulating androgen synthesis (e.g. CYP11A1, CYP19A1), potentially amplifying hyperandrogenism in PCOS [3,15].

North Uttar Pradesh, a major agricultural region in India, reports high OCP exposure due to extensive pesticide use [6]. Residues like p,p'-DDE and β-HCH, detected in human serum, correlate with reproductive and metabolic disorders [7,8]. However, the combined effects of urban (e.g. industrial DDT) and rural (e.g. agricultural cyclodienes) OCP exposure on dermatological severity remain unexplored, despite their distinct chemical properties and biological targets.

We hypothesized that higher OCP exposure correlates with more severe dermatological manifestations in PCOS, with rural populations showing greater exposure to cyclodienes (linked to hirsutism) and urban populations to DDT derivatives (linked to hyperpigmentation). This study is the first in North India to:

1. Quantify urban-rural differences in OCP exposure patterns among PCOS women,
2. Evaluate dermatological severity (beyond prevalence) using standardized scales, and
3. Investigate mechanistic pathways (e.g. OCP-androgen-insulin resistance interactions).

By addressing these gaps, this research aims to inform region-specific interventions to mitigate environmental risks in PCOS management.

Materials and Methods

This case-control study was conducted at the Department of Obstetrics and Gynaecology, S.N. Medical College, Agra, India from January 2022 to December 2024. We enrolled incident PCOS cases at first clinical presentation along with age-matched (± 2 years) controls from routine gynaecologic care, following all participants for 6 months to confirm diagnosis stability. The sample size of 110 cases and 110 nonPCOS was calculated based on prior studies showing an odds ratio of 2.1 for OCP-PCOS associations, with 80% power and 95% confidence to detect a minimum OR of 2.5 for dermatological outcomes, assuming 35% exposure prevalence in controls.

Exposure assessment incorporated both questionnaire data and biochemical measurements. A validated questionnaire assessed residential history (including urban/rural location and proximity to agricultural/industrial sites), occupational exposures, and dietary habits through face-to-face interviews. The questionnaire demonstrated good internal consistency (Cronbach's $\alpha = 0.78$ for exposure items) following pilot testing and cognitive interviews with 30 participants. Blood samples were collected throughout the year with

seasonal distribution (20% spring, 25% summer, 30% monsoon, 25% winter) to account for potential seasonal variations in pesticide exposure.

Serum OCP analysis employed gas chromatography-mass spectrometry (Agilent 7890B) using a DB-5ms capillary column (30 m × 0.25 mm, 0.25 µm film thickness). Quality assurance measures included analysis of ^{13}C-labeled internal standards showing 85 - 110% recovery rates, daily calibration curves ($R^2 > 0.995$), method blanks, and duplicate samples demonstrating < 15% coefficient of variation. Limits of detection ranged from 0.01-0.05 ng/mL across analytes. All OCP concentrations were lipid-adjusted using measured serum triglycerides and total cholesterol levels.

Potential confounders were addressed through study design and statistical adjustment. In addition to matching by age, we collected data on BMI, parity, socioeconomic status (using the Kuppuswamy scale), dietary fat intake (via 24-hour recall), physical activity (IPAQ-SF), and smoking exposure. These variables were included as covariates in regression models after confirming absence of multicollinearity (VIF < 5). We acknowledge potential residual confounding from unmeasured pollutants like phthalates, and conducted sensitivity analyses excluding women with occupational chemical exposure (n = 12).

Dermatological evaluations were performed by a blinded dermatologist using standardized scales: the Global Acne Grading System (GAGS) for acne severity, modified Ferriman-Gallwey score (mFG ≥8) for hirsutism, neck/axillary scoring for acanthosis nigricans (0-4), and Ludwig scale for alopecia grading. Statistical analysis included independent t-tests or Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables. Multivariate logistic regression models examined OCP-dermatological associations while adjusting for covariates. Additional sensitivity analyses stratified by urban/rural residence and excluded statistical outliers.

Results

The following sections details demographic characteristics, OCP exposure profiles, dermatological manifestations, and their associations, supported by statistical analyses.

Demographic and socioeconomic characteristics

The demographic and socioeconomic characteristics are summarized in table 1. The mean age was comparable between groups (PCOS: 26.4 ± 4.1 years; non-PCOS: 27.2 ± 3.8 years; P = 0.12). Women with PCOS had a significantly higher body weight (72.3 ± 8.5 kg vs. 62.1 ± 7.2 kg; P < 0.001) and BMI (28.5 ± 5.2 kg/m^2 vs. 24.1 ± 4.3 kg/m^2; P < 0.001), reinforcing metabolic disparities. Socioeconomic status distribution was similar (P = 0.23), with 34.5% of PCOS and 27.3% of non-PCOS women in the lower class, 56.4% vs. 61.8% in the middle class, and 9.1% vs. 10.9% in the upper class.

Residential pesticide exposure was more prevalent among PCOS women, with 68.2% residing in rural areas (vs. 54.5% in non-PCOS; P = 0.02), where agricultural pesticide use is common. Notably, parity was lower in PCOS women (1.3 ± 0.9 vs. 1.6 ± 1.0 in non-PCOS; P = 0.03), suggesting potential reproductive impacts linked to PCOS. Dietary exposure to pesticide-treated foods (e.g. conventional produce) was significantly higher in PCOS women (5.3 ± 2.1 times/week vs. 3.8 ± 1.9 times/week; P < 0.001). Contrary to the initial hypothesis, the duration of OCP exposure was comparable between groups (PCOS: 6.5 ± 2.7 years; non-PCOS: 6.2 ± 2.4 years; P = 0.34), though rural residence and dietary habits may contribute to cumulative bioaccumulation.

Organochlorine pesticide exposure profiles

Serum OCP levels, measured by gas chromatography-mass spectrometry (GC-MS), were significantly higher in PCOS women across all pesticide groups (Table 2). Detection limits were robust, with limits of detection (LOD) of 0.01 - 0.05 ng/mL and limits of quantification

Variable	PCOS (n=110)	Non-PCOS (n=110)	P-value
Age (years)	26.4 ± 4.1	27.2 ± 3.8	0.12
Body weight (kg)	**72.3 ± 8.5**	**62.1 ± 7.2**	**<0.001**
BMI (kg/m^2)	28.5 ± 5.2	24.1 ± 4.3	<0.001
Socioeconomic Status			0.23
- Lower class	38 (34.5%)	30 (27.3%)	
- Middle class	62 (56.4%)	68 (61.8%)	
- Upper class	10 (9.1%)	12 (10.9%)	
Living Locality			**0.02**
- Rural (pesticide-exposed)	75 (68.2%)	60 (54.5%)	
- Urban	35 (31.8%)	50 (45.5%)	
Parity (n)	**1.3 ± 0.9**	**1.6 ± 1.0**	**0.03**
Dietary Exposure (times/week)	5.3 ± 2.1	3.8 ± 1.9	<0.001
Years of OCP Exposure	6.5 ± 2.7	6.2 ± 2.4	0.34

Table 1: *Updated demographic and socioeconomic characteristics.*

Notes:

- *Data: mean ± SD or n (%).*
- *Significance: P < 0.05, P < 0.01.*

(LOQ) of 0.03 - 0.15 ng/mL, ensuring reliable quantification. Cyclodienes showed the largest disparity, with a 75.0% increase in PCOS women (6.75 ± 3.95 ng/g lipid vs. 3.86 ± 2.30 ng/g lipid; P < 0.001), driven by elevated aldrin (2.20 ± 1.15 ng/g lipid vs. 0.62 ± 0.35 ng/g lipid). Endosulfan compounds were 68.0% higher in PCOS women (3.02 ± 2.20 ng/g lipid vs. 1.80 ± 1.95 ng/g lipid; P < 0.001), reflecting rural agricultural exposure. DDT derivatives exhibited a 40.0% increase (6.72 ± 2.90 ng/g lipid vs. 4.80 ± 2.85 ng/g lipid; P < 0.001), with urban PCOS women showing significantly higher levels (7.10 ± 3.05 ng/g lipid vs. 4.95 ± 2.90 ng/g lipid), likely due to industrial and dietary sources. BHC isomers were 33.0% higher (5.58 ± 2.70 ng/g lipid vs. 4.20 ± 2.40 ng/g lipid; P < 0.001). Chlorinated pesticides, including methoxychlor, increased by 74.0% (5.56 ± 3.10 ng/g lipid vs. 3.20 ± 2.15 ng/g lipid; P < 0.001), with urban exposure prominent. These findings indicate significant bioaccumulation in PCOS women, amplified by urban-rural exposure differences and dietary habits.

Pesticide Group	PCOS (n = 110) (Mean ± SD)	Non-PCOS (n = 110) (Mean ± SD)	% Increase in PCOS	P-value
BHC Isomers (α, β, γ, δ)	5.58 ± 2.70	4.20 ± 2.40	33.0%	<0.001
Endosulfan Compounds (I, II, Sulphate)	3.02 ± 2.20	1.80 ± 1.95	68.0%	<0.001
DDT Derivatives (pp-DDE, pp-DDD, pp-DDT)	6.72 ± 2.90	4.80 ± 2.85	40.0%	<0.001
Cyclodienes (Aldrin, Dieldrin, Endrin, Endrin Aldehyde)	6.75 ± 3.95	3.86 ± 2.30	75.0%	<0.001
Chlorinated Pesticides (Methoxychlor, Heptachlor, Heptachlor Epoxide)	5.56 ± 3.10	3.20 ± 2.15	74.0%	<0.001

Table 2: *Serum organochlorine pesticide levels (ng/g lipid) in PCOS vs. non-PCOS women.*

**Note: Values represent lipid-adjusted sums of compounds within each group. LOD: 0.01 - 0.05 ng/mL; LOQ: 0.03 - 0.15 ng/mL. Statistical significance: *P < 0.05, **P < 0.01.*

Dermatological manifestations

Dermatological manifestations were significantly more prevalent in PCOS women (Table 3). Acne was observed in 63.6% of PCOS women (70/110) versus 9.1% of controls (10/110; P < 0.001), with moderate-to-severe grades (GAGS score ≥ 19) in 45.7% of cases. Hirsutism was exclusive to PCOS women (43.6%, 48/110 vs. 0%; P < 0.001), with a mean modified Ferriman-Gallwey score of 12.3 ± 4.1, linked to 75% higher cyclodiene levels (OR = 3.7, 95% CI: 2.3 - 5.9; P < 0.001). Alopecia affected 60.0% of PCOS women (66/110) versus 13.6% of controls (15/110; P < 0.001), predominantly Ludwig grade I-II. Acanthosis nigricans was present in 42.7% of PCOS women (47/110) versus 0% in controls (P < 0.001), primarily affecting the neck and axillae. Hyperpigmentation was observed in 51.8% of PCOS women (57/110) versus 10.9% of controls (12/110; P < 0.001), with moderate Fitzpatrick scale scores, particularly associated with urban DDT exposure. Skin tags were noted in 40.9% of PCOS women (45/110) versus 5.5% of controls (6/110; P < 0.001). Seborrheic dermatitis was 5.6 times more frequent in PCOS (35.5%, 39/110 vs. 6.4%, 7/110; P < 0.001). These findings underscore the clinical burden of PCOS dermatological manifestations, driven by hyperandrogenism and insulin resistance.

Manifestation	PCOS (n = 110)	Non-PCOS (n = 110)	P-value
Acne	70 (63.6%)	10 (9.1%)	<0.001
Hirsutism	48 (43.6%)	0 (0%)	<0.001
Alopecia	66 (60.0%)	15 (13.6%)	<0.001
Acanthosis Nigricans	47 (42.7%)	0 (0%)	<0.001
Hyperpigmentation	57 (51.8%)	12 (10.9%)	<0.001
Skin Tags	45 (40.9%)	6 (5.5%)	<0.001
Seborrheic Dermatitis	39 (35.5%)	7 (6.4%)	<0.001

Table 3: *Dermatological manifestations in PCOS and non-PCOS women.*

**Note: Data are presented as n (%). Statistical significance: P < 0.05, P < 0.01.*

Associations between OCP exposure and dermatological manifestations

Logistic regression, adjusted for age, BMI, and parity, assessed associations between OCP exposure and dermatological manifestations (Table 4). Higher total OCP levels were significantly associated with increased odds of acne (OR = 3.0, 95% CI: 2.0 - 4.5; P < 0.001), hirsutism (OR = 3.3, 95% CI: 2.1-5.0; P < 0.001), and acanthosis nigricans (OR = 3.1, 95% CI: 1.9 - 4.8; P < 0.001). Alopecia showed a moderate association (OR = 2.5, 95% CI: 1.6 - 3.8; P = 0.001). Cyclodienes were strongly linked to severe hirsutism (OR = 3.7, 95% CI: 2.3 - 5.9; P < 0.001) and alopecia (OR = 2.9, 95% CI: 1.8 - 4.6; P < 0.001), clinically relevant for targeted dermatological interventions. Endosulfan compounds were associated with acne (OR = 3.1, 95% CI: 1.9 - 5.0; P < 0.001) and acanthosis nigricans (OR = 3.2, 95% CI: 2.0 - 5.1; P < 0.001). DDT derivatives, elevated in urban PCOS women, correlated with hyperpigmentation (OR = 2.7, 95% CI: 1.7 - 4.2; P < 0.001) and skin tags (OR = 2.6, 95% CI: 1.6 - 4.1; P = 0.002), highlighting urban-specific risks. BHC isomers were linked to seborrheic dermatitis (OR = 2.8, 95% CI: 1.7 - 4.5; P < 0.001). Total OCP levels correlated with dermatological severity scores (r = 0.53, P < 0.01), particularly for acne (GAGS score: r = 0.50, P < 0.01) and hirsutism (mFG score: r = 0.47, P < 0.01). BMI and dietary exposure were significant covariates, with stronger correlations in PCOS women (r = 0.52, P < 0.01).

Discussion

This case-control study establishes a significant association between organochlorine pesticide (OCP) exposure and aggravated dermatological manifestations in women with polycystic ovary syndrome (PCOS) in North India. Below, we interpret the results, compare them with recent studies, explore mechanistic links, and discuss public health implications and limitations, referencing relevant tables.

Dermatological Manifestation	Pesticide Group	Odds Ratio (95% CI)	P-value
Acne	Total OCP	3.0 (2.0 - 4.5)	<0.001
	Endosulfan Compounds	3.1 (1.9 - 5.0)	<0.001
Hirsutism	Total OCP	3.3 (2.1 - 5.0)	<0.001
	Cyclodienes	3.7 (2.3 - 5.9)	<0.001
Alopecia	Total OCP	2.5 (1.6 - 3.8)	0.001
	Cyclodienes	2.9 (1.8 - 4.6)	<0.001
Acanthosis Nigricans	Total OCP	3.1 (1.9 - 4.8)	<0.001
	Endosulfan Compounds	3.2 (2.0 - 5.1)	<0.001
Hyperpigmentation	DDT Derivatives	2.7 (1.7 - 4.2)	<0.001
Skin Tags	DDT Derivatives	2.6 (1.6 - 4.1)	0.002
Seborrheic Dermatitis	BHC Isomers	2.8 (1.7 - 4.5)	<0.001

Table 4: *Logistic regression analysis of OCP exposure and dermatological manifestations.*

Note: Adjusted for age, BMI, and parity. Statistical significance: P < 0.05, P < 0.01.

OCP: Organochlorine Pesticide; PCOS: Polycystic Ovary Syndrome.

OCP exposure and PCOS pathogenesis

PCOS women exhibited significantly higher serum OCP levels, with increases of 33.0 - 75.0% across pesticide groups, as shown in table 2 [2]. Urban PCOS women had higher DDT levels (7.10 ± 3.05 ng/g lipid vs. 4.95 ± 2.90 ng/g lipid; P < 0.001), likely due to industrial sources, while rural women showed elevated cyclodienes (6.75 ± 3.95 ng/g lipid; P < 0.001), reflecting agricultural exposure [2]. Higher parity (1.8 ± 1.1 vs. 1.2 ± 0.9 live births in urban PCOS vs. non-PCOS) and dietary exposure (5.3 ± 2.1 vs. 3.8 ± 1.9 times/week), as reported in table 1, indicate lifestyle-driven OCP uptake [1]. The correlation between body mass index (BMI) and OCP levels (r = 0.52, P < 0.01) aligns with Yang., *et al.* (2021), who noted lipophilic OCPs accumulate in adipose tissue, worsening metabolic dysfunction [3]. Trabert., *et al.* (2020) reported a 2.1-fold increased PCOS odds with β-BHC exposure (P = 0.003) [11], and Toft., *et al.* (2016) linked p,p'-DDE to PCOS risk (OR = 2.4; 95% CI: 1.5 - 3.8) [12]. Our study highlights rural DDT exposure as a novel risk factor in North India [2].

Dermatological manifestations

The elevated prevalence of dermatological manifestations in PCOS women, including acne, hirsutism, and acanthosis nigricans, reflects hyperandrogenism and OCP exposure, as detailed in table 3 [3]. Logistic regression (Table 4) showed strong associations, with cyclodienes linked to hirsutism (OR = 3.7, 95% CI: 2.3 - 5.9; P < 0.001) and alopecia, and DDT derivatives to hyperpigmentation [4]. Keller., *et al.* (2019) demonstrated that DDT derivatives enhance sebocytelipogenesis by 40% via androgen receptor activation (P < 0.01), OCPs may upregulate androgen receptors in sebocytes, exacerbating acne and hirsutism [13]. Urban DDT prominence amplifies this effect [2]. Legro., *et al.* (2021) reported aldrin-induced IRS-1 serine phosphorylation (P = 0.008), supporting our endosulfan-acanthosis nigricans link [14]. These pathways highlight OCP-driven exacerbation of PCOS dermatological features.

Comparative studies

Recent studies (2019 - 2023) corroborate our findings. Sathyanarayana., *et al*. (2021) found endosulfansulfate disrupts steroidogenesis at ≥ 1.2 ng/mL, near our PCOS levels (3.02 ± 2.20 ng/g lipid) [2,15]. Randall., *et al.* (2022) reported heptachlor epoxide reduces hair shaft elongation by 58% (P = 0.002), supporting our cyclodiene-alopecia association [3,4]. Koual., *et al.* (2022) showed combined OCPs exert 3.7-fold greater endocrine disruption (P < 0.001), explaining our grouped OCP results [2,16]. Vandenberg., *et al.* (2023) noted nonmonotonic

OCP dose responses, suggesting low-dose urban exposures (e.g. DDT derivatives) have disproportionate effects, a limitation of our single-timepoint data [2,17]. These studies affirm OCP's global relevance in PCOS, with our data emphasizing urban-rural gradients [1,2].

Public health recommendations

The association between OCP exposure and dermatological severity, evidenced in table 3 and 4, necessitates environmental risk assessment in PCOS management [3,4]. Higher parity and dietary exposure in PCOS women suggest urban lifestyle factors amplify OCP uptake [1]. Fucic., *et al.* (2020) reported similar OCP levels in Eastern European farmworkers (P = 0.21 for cross-study comparison) [18]. Yang., *et al.* (2021) showed a 6-month detoxification program reduced OCP levels by 38% and improved menstrual regularity (P = 0.04) [3]. We recommend screening for OCP exposure in high-risk PCOS cohorts, particularly in urban and rural North India, and promoting dietary interventions (e.g. organic produce) to reduce exposure. Urban-focused regulations on industrial DDT sources, alongside rural pesticide bans, as proposed by Teede., *et al.* (2023), are critical [1]. Clinicians should integrate environmental and reproductive histories into PCOS care.

Study Limitations and Future Directions

The single-timepoint OCP measurements, as reported in table 2, introduce potential temporal bias, as levels may fluctuate with dietary or seasonal changes [2]. Vandenberg., *et al.* (2023) advocate longitudinal sampling to capture exposure variability, a critical need for future studies [17]. Unmeasured confounders, such as dietary OCP residues in dairy, noted by Petrakis., *et al.* (2019), may affect results [18]. The regional focus (Table 1) limits generalizability to non-agricultural populations [1]. Future research should employ longitudinal designs to assess cumulative OCP exposure, particularly urban DDT sources, and investigate dietary and reproductive factors like parity [1]. Mechanistic studies on OCP-induced epigenetic changes could clarify dermatological effects. Intervention trials, building on Yang., *et al.* (2021), are essential to evaluate OCP reduction benefits [3].

In conclusion, our study demonstrates a robust association between OCP exposure, particularly urban-driven DDT derivatives, and aggravated dermatological manifestations in PCOS, with significant implications for environmental and clinical strategies in North India, as supported by table 3 and 4 [3,4].

Conclusion

This study evaluated the association between organochlorine pesticide (OCP) exposure and the severity of dermatological manifestations in women with polycystic ovary syndrome (PCOS) compared to non-PCOS controls in North India. The findings reveal that elevated OCP exposure, particularly to cyclodienes, endosulfan compounds, and urban DDT residues, significantly exacerbates dermatological symptoms such as acne, hirsutism, acanthosis nigricans, and alopecia in women with PCOS. The higher serum OCP levels observed in PCOS participants reinforce the role of environmental endocrine disruptors in aggravating hyperandrogenism and insulin resistance, both of which are central to PCOS pathophysiology.

The strong correlation between OCP burden and dermatological severity underscores the importance of integrating environmental risk factors into clinical evaluations. This is especially critical in high-risk areas like North India, where agricultural pesticide usage and urban DDT exposure remain prevalent. Incorporating environmental exposure histories can help clinicians tailor more effective, individualized treatment strategies that address both hormonal and cutaneous manifestations of PCOS.

These findings have vital public health implications. Mitigating OCP exposure through policy regulation, community awareness, and safer agricultural practices may reduce symptom severity and disease burden. In addition, future studies should explore OCP detoxification strategies in PCOS management, aiming to lower systemic toxin levels and improve clinical outcomes.

In conclusion, OCP exposure is a modifiable environmental risk factor that significantly influences the dermatological presentation of PCOS. Addressing this intersection between environmental health and endocrine dysfunction can lead to more comprehensive and effective management, particularly in pesticide-prone regions.

Recommendation

Screening for OCP exposure in PCOS patients may guide targeted interventions.

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